STRUGGLE of LIFE

or

The Natural History of Stress and Adaptation

by Martial Rossignol
Line Rossignol
Roelof A.A. Oldeman
Soraya Benzine-Tizroute

TREEBOOK 1
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Martial and Line Rossignol
Roelof A.A. Oldeman
&
Soraya Benzine-Tizroutine

with contributions by A. Ambroise,
E.A.P. de Bruijn & C. Caisne

1998

Treemail
Prins Bernhardlaan 37
6866 BW Heelsum
The Netherlands
email: info@treemail.nl
This book is dedicated to Wil Oldeman-Helder, who sealed our friendship and kindled our collaboration. She generously provided huge amounts of human warmth and practical help for this transfer of knowledge and so, like our Sun, exercised this noble task at the top organization level, far above leaves, plasmids, or birds energizing biological information transfer.

Once all ten thousand things are seen in their unity
We return to the beginning
And remain where we have always been
(Chinese proverb)

There is no conception more fallacious
than the sense of cosiness implied by “Mother Nature”
(John Wyndham, 1957, The Midwich Cuckoos)

La cible de cette symbiose entre l’inné et l’acquis
est non pas la structure, mais sa recomposition
(Jean-François Kahn, 1994, Tout change parce que rien ne change)

Loin de l’exclure du monde qu’elle décrit, la science retrouve
comme un problème l’appartenance de l’homme à ce monde

(Ilya Prigogine & Isabelle Stengers, 1979, La Nouvelle Alliance)
TREEBOOK 1

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About the Authors

As a professional oceanographer, Dr. Martial Rossignol since the ‘fifties studied ocean currents, marine biology and fisheries in the Gulf of Guinea and the Caribbean, particularly the interactions between climatic and oceanic circulation, like El Niño in the Pacific and its counterpart, the Guinea stream in the Atlantic. From 1977 to 1994 he was associated to the Laboratory for Experimental Plant Morphogenesis of Paris University, in Orsay, where with his wife Dr. Line Rossignol he worked on *in vitro* clones of various plant species, and their explants. Prof. Roelof A.A. Oldeman studied architecture, ecology, and dynamics of tropical trees and rain forests since 1963, adding other trees, forests and ecosystems, as well as their culture, since 1977. Dr. Soraya Benzine-Tizroutine worked on the genetics of *in vitro* cultures in the team of M. and L. Rossignol. Dr. Rossignol, Dr. Rossignol and Dr. Oldeman started cooperating 25 years ago where the Ocean meets the Rain Forest in Cayenne (Fr. Guyana), and in Orsay. Important technical and editorial contributions to this book were due to A. Ambroise and C. Gaisne from Orsay and E.A.P. de Bruijn from Wageningen.
FOREWORD

The present book is the result of more than a hundred years of human research, moved by our great wonder before the awesome treasurehouse of our marvelous planet and our deep desire to understand its mysteries. The Guyanas, where under the impact of the sun the ocean meets the tropical rain forest, were one of its poles. The other pole was the laboratory of Orsay in France, where the basic stuff of life was exposed to the extreme stresses of losing the coherence of its tissues and meeting the raw environment of a test tube in a lab. The struggle of life to adapt, always and everywhere, was the core of everything we observed and put to the test.

The present book, outcome of this quest for knowledge, inherently differs from current reports covering some research project paid for a few years and with aims dictated by science politicians. This is why we did not follow the usual path, which is to send a manuscript to peer review and publisher. We are no official team of some official organization, but what a team we are! We do not belong to some official network, but how nice is our human network! In short, we may seem a bit old-fashioned and freely admit it. We indeed think that there is an immaterial scientific heritage largely worth the trouble to be saved, hand in hand with our results. It was handed over to us by appreciated masters, such as the oceanographers Dr. Jean Furnestin, Prof.Dr. J.-M.Pérès and Dr. Tregouboff and the botanists Prof.Dr. H.C.D. de Wit, Prof.Dr. C.A.Reijnders-Gouvefont and Prof.Dr. R. Nozeran.

This heritage is manifold. There is the source of inspiration, open-minded and wide amazement at the wonderful world that surrounds us all. Mind you, we are not just curious but we marvel before the wonders of Creation. This inspiration clashes with today's usual reasons for research, commercial or political. We also inherited a different modus operandi. Senior scientists now use to unite around them a team of younger people working on the ideas of le patron. This is all right as long as young people flock towards such teams moved by their passion of discovery. However, the mechanism has become very different. Young people are bought too often, under insulting conditions imitated from soviet science, bullied by orthodox academic monopolists of diplomas.

The senior authors of the present book lived the years between 1946 and 1966 when scientists were free to wonder and select their own subjects, ways and methods, and were paid and treated as free and respected people. We strongly believe that the new generation deserves to inherit this dignity instead of a position in society that inexorably slides downhill to that of a brain slave.

The third jewel in the heritage of science is free discussion. Scientific discussion is older than any of us authors and readers can remember, and it is public. From the 17th Century mathe-
maticians who defended their theses in the market place, it goes to the original Doctor's thesis, printed and published independently by the candidate before it might be publicly defended. Only this guarantees full independence of the scientific mind, balanced by honest, because public, discussion in the scientific community.

The tainted contemporary "peer review system" on the contrary rests on the "party spirit". Decisions about what is scientifically correct are reached in backrooms, equivalent to party cells. Intentions there are screened in advance by project evaluations, and results by acceptance or rejection of manuscripts. What is rejected in the backroom or by some council or other is never made public and forever hidden. The reader be aware of the Russian word for council. It is "soviet".

The present book hence is published privately. It has not been reviewed by peers, whoever these may be. We hope that it will elicit a lively discussion in public, particularly among young scientists who still can feel that shiver of excitement for things free and wonderful. Please be convinced of our true respect. This book also is more accessible to you because of its distribution by Internet, for the moment still a bulwark of freedom. Internet is of course abused, but is still vital in this time and age tending to suppress risky Liberty and to replace it by a few riskless liberties.

The price is set to make the book accessible without ruining either you or us. Please accept our apologies for being obliged to make you pay taxes for the upkeep of the Republic of France, the Kingdom of the Netherlands and the United Kingdom.

We thank the members of what once was Professor R. Nozeran's team in Orsay for discussions and help. Wageningen University provided funds for travel and important technical support for this venture outside its official research programme, which is very meritorious. Thank you. We also are obliged to Mmes M.J. Boudewijn and D.E. Boeijink of the Hutan Lestari team for their help. The free researchers of ORSTOM Cayenne during J.-M. Brugièrè's directorship were valued discussion partners whom we thank, like those in the networks around Professor F. Hallé in Montpellier and around the Hladik family in Brunoy. Special thanks to Professor P. Blandin of the Museum National d'Histoire Naturelle for permission to borrow figure 5-2, and to Dr.G. Remmers for our figure 6-1, from his 1998 book. All those we met and all who gave us their insights during all-but half a century can not be mentioned here. We feel very privileged to have known you all, and are grateful.

Now let the reader beware of this peerless and fearless book. We hope it is useful and pleasant at least, and that discussion will be public and fruitful.

The Authors
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Chapter 1  Introduction: the new dilemmas

Science may be conceived as the human venture to define problems and to provide their solution by way of answers that can be proven. There are new discussions today as to what is proof (Horgan 1993). Still, most scientists admit that proof rests on potential repetition of data gathering by observation or experiment, data processing by some method of abstract calculation or graphic visualisation, and concluding. In short, proof is in the principle “you can repeat it and see for yourself” (Oldeman 1990).

These preoccupations are in the margin of society. Scientific problems are tackled in a businesslike way, it being taken for granted that a solution does exist. The context indeed is assumed to be known. In science this context is modern physics, in a garage it is the blueprint of a car which has to be repaired. The solution of any problem hence can be reached by anyone who knows the rules. Scientists have always claimed and some still claim that a basic “law of everything” is forthcoming and will be perceived by some scientist soon. This would be the end of science as an important human activity (cf. Horgan 1996).

The newspapers, and many a general scientific journal show that the deep human preoccupation’s are not problems. They are dilemmas. A dilemma is fear-laden, heartfelt, existential, dramatic. There is no known way out of a dilemma. The last thoughts of passengers just before a plane crash, the reflections and feelings at the sickbed of a loved one, the mental agony of the starving and the poor, the dismay of those who discover the broken relics of the landscape of their childhood, they all bear upon dilemmas. They have several things in common. There always is a dimension of deep, existential emotion in a dilemma. Dilemmas always occur in apparently complex situations which do not seem to be explained by simple blueprints or natural laws. Finally, dilemmas can not be resolved like problems, because dilemmas require knowledge and problems only need information to be resolved.

Which are the principal dilemmas of today? They concern both health and environment. As biological scientists we recognise this as a composite field of evolutionary preoccupations. Health is close to “fitness” of an organism and environment may be conceived as a set of “selection pressures”. However, this realisation is of little help if one sees for instance in recent history the failure to eradicate infectious diseases (Garrett 1994) or the bankruptcy of half a century of genuine efforts to halt deforestation, erosion and pollution (cf. Weinschenk 1994, Woodword & al. 1996, FAO 1997, Péroux et De Framond 1997). There was information galore, but the insight was lacking to unite it into coherent knowledge.
Can a transition be managed, so as to transform a dilemma into a problem, which can be solved? Let us first consider some examples of the hot issues of the day.

“The Children are Dying” says Gibney Jr. (1996) in Time. He writes on an epidemic of food poisoning, which struck Japan since May 28 1996, when it became manifest in the fishing village of Oku, West of Osaka. It killed at least 8 people and made more than 8,500 persons ill all over Japan. The pathogen was an unusually virulent strain of Escherichia coli, known under the number O157:H7. Most forms of this bacterium are harmless intestine dwellers of animals and humans. However, the runaway virulent strain produces acute illness and occasional death.

The same bacterium was reported a year earlier in Newsweek to be able to mutate very fast, in a direction favouring survival (Begley 1995). Such “oriented” mutations were neo-Darwinian heresy. Nonetheless, teams at MIT and the University of Utah experimented with Escherichia coli strains lacking a lactose-digesting enzyme, culturing it on a substrate with nothing but lactose. Nearly all colonies died by starvation, but a few did not. Instead, these developed a lactose-digesting enzyme, which they did a hundred times quicker than expected if one assumes random mutations. The key was a sexual process, i.e. the transfer of a plasmid, which is a DNA ring. In the case of this E. coli strain, the plasmid contained a gene for digesting lactose. Such sexual transfer systems were shown to become active when a population is starving.

The book by Garret (1994), “The Coming Plague”, written for a broad, informed readership, has widely been cited to substantiate dilemmas. It includes, for instance, the history of E. coli research till the year of publication. In First Class, the magazine of the International Airline Passengers Association, Burne (1997) warns air travellers for “superbugs”. The theory was, he says, that drug-resistant bacteria like Staphylococcus aureus were simply random mutations. But they are not only that, Burne says, citing Garrett and others. Bacterial strains of different origin may swap genetic information via plasmids, so transferring blueprints for antibiotic resistance in the absence of the drug (also see Amable-Cuevas & Chicurel 1993). Worse, “molecular chaperons” protect bacteria against reactions of the human immunity system.

Burne also evoked the 1991 cholera outbreak in Peru, due to shrunk En Tor bacteria that crossed the Ocean from Bangladesh on floating algae. This is Vibrio cholerae 01 biotype El Tor (Garrett 1994). They were transmitted by shellfish to human consumers, there to return to their normal size and properties, including immunity to most antibiotics. Modern air travel favours this kind of diseases, says the article, to conclude that an airborne superbug, like a new strain of the Ebola virus, possibly might not even leave us months to prepare ourselves.

These examples among many from general magazines, show that new and old diseases once more have become existential dilemmas. They are often linked to infection by old or new microbial pathogens. The Netherlands consumer organisation “Consumentenbond” links such infections to food. For instance, Campylobacter sp. belongs to the bacteria known to make chicken meat unsafe for consumption unless fully sterilised by long cooking, says Consum-entenbond (1996). It cited “...enrofloxin by which poultry-farmers treat campylobacter, a normal commensal in the intestinal tract of the chicken. Meanwhile, campylobacter has become resistant to a host of comparable antibiotics (the quinolones) which include remedies for humans. Hence these can be used no more against campylobacter.”
Many predominant, mass-produced foods today are felt to be dangerous, particularly meat. Meat from diseased animals has caused a quandary. After the chickens, 1996 witnessed the panic in Europe due to the mad cow’s disease broadly reported in the media to be linked to the syndrome of Kreutzfeldt Jacob, a degenerative human ailment. Recently, the Consumentenbond (1997b) mentioned bovine paratuberculosis to be linked to a human intestinal disorder, Crohn’s disease. The year 1997 then saw the coming of swine fever in the Benelux. European meat consumption and exportation decreased temporarily, due to both ignorant fear and scientific knowledge. Meat prices rose because of massive liquidation of livestock.

Cattle being fed with products containing recycled animal products, e.g. bone flour of diseased sheep, is widely cited in the media as a cause of the dilemmas: “herbivores should not be raised as cannibals”. This is linked to prions, proteinaceous infectious particles, discovered by Prof. Stanley Prusiner in 1981, says Köhler (1996) in an article in the weekly “Science and Education” appendix to the Dutch newspaper NRC-Handelsblad. Healthy prion protein (PrP0), according to the interview given by prof. Prusinger months before his Nobel prize, is made by many cells, particularly brain cells. PrP0 is folded in α-helices and takes another architecture, to wit a β-sheet, in PrPsc, the foreign pathogenic form. Sc stands for the sheep’s disease scrapie. Such unhealthy prions would travel to the cow with fodder containing sheep-flour, and go with the steak to the human intestine. It is a mystery how PrPsc can travel in humans from the intestine to the brain. Strong misgivings are indeed widespread as to the food chain being tainted more generally, including many human foodstuffs.

Public apprehension is also growing with regard to green produce. Overselection of crop plants is becoming an issue in densely populated industrial countries. There is increasing opposition, in the form of political action against biotechnologically manipulated basic foods, like soya. Ships bringing the first loads to Europe in 1997 met with manifestations and boycotts in the harbours. Once more, the key issue implicitly is mistrust of the genetic properties of plants and animals selected in wholly artificial lab environments. The treatment of fruits by mild gamma radiation to prolong their consumable period now is forbidden by Dutch law. A less recent but persisting dilemma regards over-fertilisation. Vegetables contain high amounts of nitrates nowadays, which are converted into nitrites in the human body. The Consumentenbond (1997a) found amounts far exceeding legal bounds in eight common Dutch vegetables. The organisation emphasises that overdoses of nitrite may hamper oxygen absorption and with proteins can form cancerogenous nitrosamines.

Other diseases also are assumed to be linked to genetic defects. An anonymous source in La Recherche (1996a) reports on a case of premature ageing because of a specific gene. This is the Werner syndrome, studied by Yu & al. (1996). Patients are struck before age 50 by atherosclerosis, cancers, diabetes, osteoporosis and cataracts, but not by Alzheimer’s disease. The researchers identified the gene as containing a protein of 1432 amino-acids showing similarities with helicases. The job of such enzymes is, to open the double helix of DNA, i.e. to separate its two strands in preparation of the molecule being duplicated before cell division. The enzymes also act in DNA reparation processes. Mutation of the enzyme induces errors in gene expression and cell division. Once more, the article concerns a dilemma, that of ageing and senescence, rather than a mere scientific problem.

Spontaneous reparation of genetic material followed by the unexpected recovery of an American boy was reported anonymously in 1996(b), under the title “A good mutation”. The patient lacked a vital enzyme dubbed ADA, so that the resistance of his organism was seriously undermined. At age 11, most symptoms of the disease disappeared and the physicians found
that a defective gene inherited from the mother had spontaneously mutated, healing some white blood corpuscles which then prospered at the cost of the diseased white blood cells. De Telegraaf, a Dutch daily newspaper (June 5, 1997, p. TA3), reported on damage to skin cell DNA by exposure to UVA and UVB light from the sun or from sun lamps. According to the dermatologist interviewed, Dr. Hoekzema from Maastricht University, in a few hours the cells carry out repairs due to the activity of a special gene known as p53.

In the industrial world, psychological disorders have become so frequent as to worry the public. In another issue of De Telegraaf, De Kromme (1996), in a whole page, reported on research at Utrecht University linking schizophrenia to genetic factors, among other causes. In this article, researcher Clarine van Oels tells about twins, one of them being schizophrenic. The chance that monovular twins are schizophrenic due to identical genotypes proved not to be the expected certitude (100%), but only 50%. In bivovular twins this chance was expected to be the same as in two arbitrary brothers and sisters, which on the average is 9%. But it was nearly twice as high. According to researcher Joke Kalkman, quoted in the same article, another genetic puzzle is that people conceived in the stressful Dutch war winter of 1944-45 had 50% more chances to become schizophrenic than those conceived in easier years.

One predominant dread in the world of today is radioactivity. A straightforward case which can not be denied is the spread of radioactive matter in the wake of the Chernobyl nuclear catastrophe of April 26, 1986. It still remains actual in the press, which shows that it is a dilemma touching profound emotions. One example among many is the article in the popular French magazine Sciences et Avenir (Anonymous 1996c) on the state of health of the hundreds of thousands of “extinguishers” having been active in 1986 and/or still living on and around the site today. There is no exceptionally high mortality in this group, claims the Centre of Environmental Medicine in Saint Petersburg (Russia). However, their health is feeble. They are ill thrice as often as non-irradiated persons. Contrary to expectations, cancer did not play the leading part, but many digestive troubles did, as well as malfunctions of the liver, the hormonal system, the heart and blood vessels, and the nervous system. This is summarised as premature ageing, caused by the accumulation of free radicals. Cancers indeed were found, but outside Ukraine in faraway White Russia. Instead of the expected leukaemia, however, thyroid cancer in children had multiplied thirty six-fold. This form of cancer is ascribed to radioactive iodine deposition originating from rain clouds blown in from Chernobyl.

Major worries also concern the quality of the environment. They are so strong that a major world top conference convened to place this issue on all political agendas; UNCED in Rio de Janeiro in 1992. It decided on principles that have to sustain the solutions compactly formulated in Agenda 21. This agenda, as usual in such documents, makes no difference between a dilemma and a problem. However, neither the 1992 dilemma, nor earlier ones could be solved by contemporary knowledge although this was not recognised explicitly.

The environmental issue is the counterpart of land and sea use. It is broadly admitted that such use is not sustainable as practised today. On the one hand, industrial agriculture, forestry and fishery were invented in Europe some one and a half-century ago. They treat land and sea as if they were inorganic substrates, which can be made to permanently produce unprecedented quantities of organisms and bio-products by applying chemical and mechanical tools. On the other hand, traditional land use, based upon the architecture and dynamics of natural organisms and ecosystems, proved to be sustainable by surviving over a millennium
or more in and outside Europe. However, their updating and development still meet resistance from various established “modern” interests, so they can not as yet express their full potential to face the challenges and dilemmas of today’s overpopulated world and its inhabitants.

Concrete dilemmas are pollution by acid rain, climate deterioration by greenhouse gases, and land degradation due to over-farming with excessive use of chemical fertilisers, herbicides and pesticides. Since the early XXth Century, social movements did question these matters and proposed “healthy” alternatives. Many were bundled since the end of the Second World War in the International Federation of Organic Agricultural Movements (IFOAM), founded upon the axiom “…the health of soil, plant, animal and man is one and indivisible…” (Balfour 1944 ex Woodword & al. 1996). This view is not popular in politics. Garrett (1994) tells how the El Tor cholera story was known to need “ecological medicine” since 1970, but was only acted upon after the major Peruvian epidemic in 1991. The epidemic made it into a dilemma!

Amanor (1993, p. 156) wrote in a general book on genetic diversity, farmer experiments and crop research: “.... commercial interests and development institutions frequently act to downgrade the creative capacities of farmers, and to reinforce industries’ control over genetic materials and the marketing of seeds.” He claimed that conservation and development of genomes based on local knowledge and design require a new and critical view on the monopoly commercial interests of agricultural research leading to standardisation. The latter imperils both farmer’s creativity and the genetic diversity it begets.

Industrial tinkering with genetics opens the way to unpredictable natural hazards. Palfreyman (1995) wrote in Newsweek on carpets of dead sardines washing up the shores of Australia from Perth in the West to Sidney in the East. The dilemma is illuminated by the comments of fishermen, that it was “bloody terrible......never seen anything like it”. One more mystery was, that only adult sardines died, not young ones. Either all sardines were born different and infection-prone, or there was a new strain of pathogens. The sheer extent of the epidemic made fishery experts state that its surrounding a whole continent was absolutely without precedent in the world.

In 1995(a), an anonymous reporter in L’Express (A.K.) referred to the fears of infertility in human males of the last few years. Citing a report by Prof. Jouannet from Paris, published in the New England Journal of Medicine, he calls the results disquieting. In between 1972 and 1992, the number of human spermatozoids per sample had lowered by 30%. The article smells of a dilemma by raising questions of critical lower thresholds of male fertility and the causes of the decline. They are unknown - perhaps pollution? Sterility of the unfortunate alligators of the Florida lakes, known to be due to the dumping of chemicals, is cited as a possible parallel.

Jégou (1996) gave a new overview of this matter in the French colleague of the Scientific American, La Recherche. He cited research done by Carlsen showing that in between 1940 and 1990 the mean volume of human ejaculate lowered from 3.4 ml to 2.75 ml, whereas over the same period the mean sperm concentration sank from 113 million to 66 million spermatozoids per millilitre. This is a fall of some 1% per year over half a century. In between 1973 and 1992, healthy Parisians even showed a decrease of almost 2% annually. Moreover, in the latter group the normal motility and normal form of the spermatozoids respectively lowered
by 0.6% and 0.5% per year. In this case, the volume of the ejaculate remained equal. In Belgium the gist of these results was confirmed by Comhaire, said Jégou, and in Scotland by Irvine. Many, but not all studies in this field confirm the general tendency. Moreover, there seems to be an increase of the diseases of the human male genitals, as well as in certain animals. The causes once more are ascribed to lifestyle, eating habits, stress and radiation.

In a vague way, all these dilemmas are thought to be linked to loss of “biodiversity” by an accelerating extinction of biological species (Myers 1979, de Boef & al.,1993). In summary, the popular verdict is “bad environment kills good organisms”. This is neither entirely false, nor entirely correct. The above examples from widely distributed media show several facts which do not support such a blanket judgement. If, like in Escherichia coli or the sick American boy, there is evidence of new “biodiversity” being born facing the new stresses, hope remains that we may not be living in an increasingly hostile environment, made so by our own hands, and paying us back by disease and sterility.

Dilemmas and problems are separated by a moving border. Knowledge in general, and the scientific knowledge which is the signature of our times in particular, converts one dilemma after another into a problem. New solutions to new problems then are found. The ambition behind the present book is to contribute a synthesis of recent scientific results contributing to a further advance along this road of human destiny.
Chapter 2  Some experiments on plants *in vitro* and *ex vitro*

The present chapter describes the experiments which were the eye-opener for the present book. They were done in the Laboratory of Experimental Plant Morphogenesis at the University of Paris VII in Orsay, some 30 km South of Notre Dame de Paris. The investigations were carried out by members of the team of the late Professor René Nozeran. They concerned the processes occurring during *in vitro* culture of several plant species, and the properties of the resulting cells.

This study, conducted over many years, concerned the phenotypes of subcultured plants belonging to different species. It showed the appearance of atypical specimens at regular, not random moments in the vegetative progenies that were monitored. The phenological observations were part of a larger research project on the origin of somaclonal variation. Hence a search was initiated into comparable and correlated quantitative variability at the level of the mass of DNA molecules in the nuclei. Such non-random variability indeed existed.

The mass of the DNA molecules hence could be taken as a parameter of nuclear behaviour in different cells. A second behavioural marker at the whole cell level was thought to be the number of chloroplasts, because chloroplasts depend immediately on nuclear DNA functioning. Indeed, the chloroplasts, like mitochondria, possess their own replication system. They transcribe DNA and translate messenger RNA in proteins. However, most proteins found in chloroplasts are coded by nuclear genes, synthesised in the cytoplasm in the form of precursors which are imported into the chloroplasts (Weil 1988). Hence a correlation should exist between the metabolic activities of the chloroplasts, those cornerstones of the metabolic cell activity, and the activity of the nucleus. Indeed “biogenesis and functioning of a chloroplast require control of the expression of genes in the nucleocytoplasmic and chloroplastic compartments”.

Those who are not interested in the technical laboratory set-up of these experiments may skip the present chapter. However, they should be aware that the results reported here are used in the next chapters. The illustrations for most people will be sufficient to understand most of these results. Still, all experimental details are indispensable in this book. Science, as the system developed by our civilisation to order our knowledge, rests on the availability of proof. The hardness of proof depends upon the demand that everybody should in principle be able to repeat experiments and observations. This can only be done if a chapter is consecrated to a detailed explanation of the work done and the tools used.
2.1 - The experiments

2.1.1 - The first set of experiments concerned modulated mitotic activity in proliferating potato cells in vitro. They used the following materials and methods.

The plant material. This originated from the semi-early dihaploid Solanum tuberosum clone BF15. This clone has 2n = 24 chromosomes. It was cultivated at the laboratory for ten years, always on a MS culture medium (Murashige & Skoog 1962) with additional vitamins according to Morel & Wetmore (1951) and a further 20 g l\(^{-1}\) saccharose and 7 g l\(^{-1}\) agar.

Leaves, or unbudded nodal cuttings were taken for callus production in vitro. They were placed at different moments on a C\(_3\) medium with growth regulators as prescribed by Quraishi & al. (1979). Eight calli were obtained in this way. Two of these were cut in five portions 36 days later and were then subcultured on a fresh C\(_3\) medium. The physical conditions during the initiation and growth of the callus were a temperature of 25\(^\circ\) C, a photoperiodicity of 12 dark and 12 light hours, a light intensity of 80 nEm\(^{-1}\)*sec\(^{-1}\). and a relative humidity of 50%.

![Diagram of the classical stages of the mitotic cycle](image)

Figure 2-1: The classical stages of the mitotic cycle (m\(_1\) = mitosis; G\(_1\) = interkinesis; S = DNA and histone replication; G\(_2\) = end of interkinesis, preparation of mitosis) and the phases of mitosis (P = prophase; M = metaphase; A = anaphase; T = telophase). **Mitosis** is division of somatic cells, not becoming sexual. Every mitosis produces two daughter nuclei equal to the original nucleus. **Meiosis** is a sequence of two successive nuclear (and cellular) divisions producing animal **gametes** or plant **spores**, both sexual and with half the parental genetic material. The mitotic m\(_1\) period generally is the shortest phase of the cycle, some 5 to 10%. During the **interphase**, wrongly called “resting period” by some authors, many cell functions are activated, e.g. DNA synthesis. Our observations were made at the end of the prophase, when chromosomes are well visible, with their two sister chromatid strands united in the middle by the **centromere**.
Sampling. Protoplasts were obtained in the following way. Calli and roots were taken from ten
days old control cuttings (code T) and macerated with proteolytic enzymes, so as to dissociate
their cellular components. Samples of protoplasts were isolated from calli at increasing ages
in order to test the effect of ageing. Both these protoplasts and the ones that originated from
roots were fixed on microscopic slides by using smear techniques, and were stained with
Schiff reagent.

Observations. DNA mass was measured using a Leitz MPVI cytophotometer. The measure-
ments were evaluated according to the two-wavelength-method of Patau (1952). All values
cited concern nuclei in the prophasic period of the mitotic cycle (Fig. 2-1). In that period, the
DNA molecules have achieved replication, their mass is doubled, and so is the volume of the
cells. For each callus, 75 nuclei were observed. Two measures (X₁ and X₂) were taken for
each nucleus. All data were expressed in “arbitrary units” (u.a.). Although they had been gath-
ered from replicated nuclei (4C), for convenience the data have been converted to 2C values
in the present chapter.

The arbitrary character of the units noted is due to the fact that light intensities appear in a
different way if measured with different measurements and different methods. Patau (1952)
found it useless to express these values in standard units if using one instrument for compara-
tive purposes only. Arbitrary units since then are a usual tool in this field.

Calculations. The data were processed using different tests (cf. Dagnélie 1975). A modified
Student-Fisher bilateral equality test (U-test) compared mean mass values two by two. Normal-
ality was tested with Pearson & Geary coefficients. Frequency diagrams were analysed us-
ing probit paper, on which the ordinate represents cumulated relative frequencies and the ab-
scissa shows mean mass values, as calculated per 50 u.a. class. Given the plurimodal character
of the frequency curves, each value of X (x = \( \frac{X_1 + X_2}{2} \)) was transformed into a decimal
logarithm \( \log x \). If groups were represented by near-straight lines delimited by breaks, they
were expressed by their own mean with a correlation coefficient \( \alpha = 0.05 \) (Fig. 2-3).

Correlations between the synodic lunar cycle and the periodicity of mitotic activity were
tested by using a simplified “nearest neighbour analysis”. For this purpose, we divided the
synodic lunar cycle in six parts, each of five days approximately. The five days before the new
moon formed the first period. Differences between successive periods, i.e. between time t and
t-1, were expressed as percentages. In case there is no trend, all positive and negative differ-
ences show a Gaussian distribution around a mean value. In fact, regression parameters of the
log% values were calculated within security limits of \( \alpha = 0.05 \), taking into account the resid-
ual variance of \( \sigma^2 \). The regression parameters, needed to assess the relation between two
types of callus cells found (see 2.2, and Figs 2-5, 2-6) were calculated in the same way.

Abbreviations. The nature and age of each callus will be coded in the text as follows:

\[
\begin{align*}
CA_6 & = \text{callus (original), 6 days old} \\
CR_{21} & = \text{callus (subcultured), 21 days old} \\
T_{10} & = \text{control, 10 days old}
\end{align*}
\]

2.1.2 - The second set of experiments

This concerned the mean number and distribution of chloroplasts in control plants and ad-
ventitious diploid clones, as well as the same values for polyploid (tetraploid, hexaploid etc.)
clones. We used the following materials (Fig. 2-2) and methods.
Figure 2-2: *In vitro* experiments on potato, *Solanum tuberosum*, diploid variety BF 15 (2x). A, B - Adventitious clones from calli originating either from leaves, or from de-budded nodia. C, D, E - Samples out of the many, diverse chromosome and chloroplast arrays. C1 - Diploid control, 24 chromosomes; C2 - Diploid control, 11 chloroplasts; D1 - Tetraploid control, 48 chromosomes; D2 - Tetraploid control, 23 chloroplasts; E1 - Polyploid clone, 84 chromosomes; E2 - Polyploid clone, 43 chloroplasts.
Plant material. The same plant material was used as described in 2.1.1, i.e. the diploid clone BF15(2x)(H1). Adventitious diploid and polyploid clones were obtained from calli on the same clone, on leaves or debudded nodes. They were taken either directly on the primary callus or on a callus having been subcultured one to four times.

Culture. Both the controls and the adventitious explants taken on calli have been multiplied in vitro by node cuttings every six weeks, according the the methods developed at Orsay (Nozeran & al. 1977; Rossignol-Bevalin & al. 1980). The culture medium was identical to that in the other experiments (see 2.1.2). The conditions were a little different. Temperature was 19°C<1<21°C, photoperiodicity was maintained at 18 light and 6 dark hours, light intensity was 55 µEm^-2*sec^-1 and relative humidity 50%.

Obtaining calli and adventitious explants of BF15(2x). The techniques for obtaining calli are described in 2.1.2. To produce adventitious explants, the temperature and photoperiod were changed to t ≈ 20°C instead of t ≈ 25°C and 18 light hours instead of 12 light hours per day.

Obtaining plants for the study of stomata. Plants from in vitro cultivation, controls as well as adventitious explants, were cultivated as cuttings in earthen pots of 20 cm Ø in which clay and compost were mixed in equal proportions. The observations and measurements were made on seven weeks old plants.

The leaf epidermis was lifted off from its lower surface with tweezers and mounted between microscopic slides in a drop of water, or a drop of Lugol if chloroplasts were very numerous. The chloroplasts in pairs of stomatic cells were counted under an optical microscope with an 40x objective and a 10x eyepiece (Fig. 2-2).

Sampling levels and experimental protocol. There are two tests.

Test 1 - * One sampling level at the fifth open leaf under the apex.

- Number of observed clones: 24 adventitious ones, 2 controls (dihaploid and tetraploid)
- Number of plants per clone is five
- Sampling territories per leaf are five
- Repetitions per territory are ten
- Total sampled: 250 stomatal cell pairs per clone

Test 2 - **Two sampling levels, at the fifth open leaf under the apex and the fifth open leaf above the stem base

- Number of observed clones 32 adventitious ones and 2 controls (dihaploid and tetraploid)
- Number of plants per clone is 6 or 8
- Repetitions per leaf are 15 or 30
- Total samples per clone are either 120 (8 plants, 15 repetitions), or 180 (6 plants, 30 repetitions), or 240 (8 plants, 30 repetitions) stomatal cell pairs

Testing the method. We made a preliminary analysis of variance with three classification criteria, i.e. clone, sampling level and leaf territory, so as to determine the part of the eigen variance. This yielded the following results (Benzine-Tizroute 1989).

- A highly significant clone effect (α = 0.001)
- A non-significant territorial effect on the end foliule of the fifth open leaf below the apex
- an effect of the sampling level on the stem which is significant with a safety factor of α = 0.05 but not so if α < 0.05
The present analysis hence principally rests on the results of the first test with the highest sampling density of 250 samples per clone. Some clones from the second test provided a comparison at the sampling level of the fifth subapical open leaf. The second test also allowed to study changes in the frequency distribution of cell populations in the dihaploid control case. These cell populations were compared in the order of leaf formation, the oldest being the fifth open leaf above the plant foot, and the youngest studied open leaves were in the fifth position below the apex.

All in all, the sampling schedule of this experiment hence included:

- 11 adventitious diploid clones of test 1;
- 13 adventitious tetraploid and mixoploid clones, 10 of which in test 1;
- 1 dihaploid control including 3 clones (Ds1, Ds2, Ds3), 2 of which in test 1;
- 1 tetraploid control including 3 clones (Ts1, Ts2, Ts3), one of which in test 1.

**Ranging the data.** The table of frequencies was calculated in classes separated by one-unit intervals. For instance, there were 6, 7, 8, ..., 15, 16, chloroplasts. In both the dihaploid and the tetraploid clone, one look at their frequency distributions shows that these do not follow a Gauss curve. Dissymmetry is the rule. Although the number of observations is as high as 120 to 150 per sample and 610 or 730 for all observations per control, there are clear peaks or modulations, principally in the median part of the curve. This is an indication that the curve is not normal and that the populations of stomatal cells are heterogeneous. Hence we have expressed the value of the parameter “number of chloroplasts” by its logarithm.

Like nuclear DNA mass in the first experiment, the population analysis has been couched on probit paper. The ordinate here represents cumulated relative frequencies and the abscissa the decimal logarithm of the rank of the classes in the sample studied. For instance this gives for the sample cited above:

- classes: 5, 6, 7, ..., 15, 16 chloroplasts;
- class ranks: 1, 2, 3, ..., 10, 11 chloroplasts

### 2.2 - The level of the nucleus or the new deal

The results of the first experiment, concerning nuclear DNA, are reported here. The further interpretation of these data will be undertaken in chapter 3.

The first result is the conspicuous absence of correlation’s in some cases, and their presence in others. The U-test and the normality test showed the existence of four groups of calli, correlated neither with age, nor with the *in vitro* conditions. However, there is a correlation between the occurrence of each one of the groups and the lunar phase at the moment when mitotic division started in its explant culture or subculture. This correlation shows the following pattern.
Figure 2-3: Relation between nuclear DNA mass (2C, diploid cells) and lunar phases (CA12, CA6, \ldots\ldots CA13 = calli in culture, CR = callus planted out). A, B - Frequency diagrams with smoothed curves. C - Nuclear DNA mass (2C) of new moon calli, analysed on probit paper. •••••• = mean per class of 50 a.u. for sample \{CA22 + CR13\}; \textbullet\textbullet\textbullet\textbullet\textbullet\textbullet\textbullet = same for sample CA22; □□□□ = same for sample CR13. \{Z, B and A\}, \{z, b and a\}: supergroups of cells; capitals indicate amplified nuclear mass, being 1.5 times the non-amplified mass (Table 1-1), small cast non-amplified nuclear mass.
• Groups of calli with low DNA mass, 660 u.a < X < 714 u.a and a normal distribution
• New moon group (CA₆, CA₁₂) with a unimodal distribution
• Full moon group (CA₂₂, CR₁₃) with a bimodal distribution
• Groups of calli with high DNA mass, 722 u.a. < X < 801 u.a and a symmetrical distribution with a curve flattened around the mean values (CA₆', CR₂₁, T₁₀)
• First quarter group, not behaving differently from the
• Second quarter group
• Intermediate group of calli with low DNA mass, like in the new moon group, but with an asymmetrical distribution, observed 5 days after the new moon (CA₃₃, CA₅₅), with a mixed character

The next result was the establishment of the probability of the preceding correlation’s representing random events. Let us suppose that, like in a card deal, in one particular moon phase one member of one of the above groups be randomly dealt. This we call “event (A)”. If there are four groups, the probability that any one group appears in any one phase is Pr(A) = ¼ = 0.25. This is found, for instance, in the cases of CA₆ and CA₁₂, the explants of which were cultured during a new moon phase.

In the case of the events of the first quarter phase, the cards were dealt differently. Explant T10 was placed in the culture medium in April (event A) and CR21 was subcultured in May (event B). Both events were very different. The probability of (B) is independent of the probability of (A), because it is conditioned by the replacement of the initial CR21 by a subculture, as if it were a fifth card in the deal, a joker. In that case

\[
\begin{align*}
Pr(B) &= ¼ * 1/5 \\
Pr(A \land B) &= (1/4 * ¼* 1/5) = 0.0125
\end{align*}
\]

The probability hence is only a little over 1% that we may find the same conditions during event A and B, i.e. that we may find the same distribution in T₁₀ and CR₂₁. The same reasoning is valid for CR₁₃ and CA₂₂ at full moon.

The log-normal distributions were analysed in more detail on probit paper (Rossignol & al. 1990). This revealed the existence in callus or root meristems of heterogeneous populations, consisting of cell groups differing from each other because their mean DNA mass was not the same (Table 1-1 and Fig. 2-3).

| Table 1-1: Groups of proliferating cells in a callus or a root meristem, according to their mean nuclear DNA mass (α = 0.05) |
|---|---|---|---|
| z | Xₑ = 507.3 u.a ± 12.4 (n = 70) | Z/z = 1.55 |
| Z | Xₑ = 783.9 u.a ± 7.5 (n = 77) |
| b | Xₑ = 594.8 u.a ± 17.8 (n = 78) |
| B | Xₑ = 903.1 u.a ± 9.4 (n = 74) | B/b = 1.52 |
| a | Xₑ = 694.3 u.a ± 7.4 (n = 73) |
| A | Xₑ = 1022.8 u.a± 14.4 (n = 75) | A/a = 1.47 |

There exist two supergroups, (z, b and a) and (Z, B and A), differing in mean DNA mass. In the first one, called “primary group” a difference of the same order exists within the pair [b - z] as in the pair [a - b]. This amounted to approximately 15% of the DNA mass in the pair [b - z] and 17% in the pair [a - b]. Both values represent a quantum of B.DNA. Three “secondary
groups (z, b, a), respectively. These regular excesses in DNA mass point to an “amplification” or endo-replication of some part of the genome. Therefore we coined the name *amplified cells* for cells displaying this particular property, to be explained further in chapter 3.

![Diagram](image)

Figure 2-4: Relations between diploid cell groups (z, b, a with DNA mass m) and amplified cells (Z, B, A with nuclear DNA mass 1.5 m). ★ = calli 22 to 55 days old; grey areas = interval with α ≤ 0.05. Frequency diagram shows two groups. In G1 with regression coefficient β = 0, regression is linear, i.e. the relative share of \{z,b,a\} is high when culture starts and diminishes with the age of the callus, whereas \{Z,B,A\} starts low and does not vary with age. In GII, \{z,b,a\} and \{Z,B,A\} are linked by a curvilinear regression, i.e. the number of amplified cells \{Z,B,A\} rises rapidly towards an asymptotic value after proliferation starts. These amplified cells have a “new” nature, differing from that of the original mother cells. Because they activate division of G1 cells with a new character, the latter gradually obtain the majority and the new character takes the place of the old one. This proliferation mode may be compared to cancer cells. *In vitro*, the cells become as it were “immortal”.

15
A significant but weak correlation (r_{obs} = 0.44 as against r_{0.975} = 0.38 with 25 ddl) proved to exist between the amplified cells and common cells. The frequency diagram (Fig. 2-4) explains the low correlation coefficient. In one and the same callus, two categories of protoplasts exist, differing by the kinetics of their mitotic division. In the first category of protoplasts, which we will call \( G_h \) the null-hypothesis tested by the regression coefficient \( \beta \) is

\[
H_0 = \beta_{x/y} = 0
\]

This may be accepted with a safety factor \( \alpha = 0.05 \). In this case, the relative frequency of amplified, active Z, B and A cells remains constant and low, whereas the numbers of z, b and a cells decrease with the ageing of the callus. The numbers z, b, and a decreased from a value between 14 and 22 in 6 to 13 days old calli, and fell to four in 22 to 55 days old calli.

In the second category of protoplasts, which we will call \( G_h \), the z, b, and a cells are shown to be linked to Z, B and A cells by a curvilinear regression. Here it is the curvilinear part of the curve which falls within the zone of safety defined by \( \alpha = 0.05 \). In this second case, the number of z, b, and a cells increases exponentially, both in comparison with the number of Z, B and A cells and with the age of the callus.

The frequency diagram (Fig. 2-4) explains these correlation’s. The \( G_h \) cells have to be considered as the mother cell type, i.e. as representatives of the initial meristematic or other explant tissues at the start of the culture on that medium. Following the DNA condition synchronous with the synodic lunar cycle, new cell types are preferentially activated. Their mitotic divisions are sped up from the outset during proliferation, by multiplication of cells belonging to the same type, but having an amplified DNA molecule.

Among our results, three observations illustrate this interpretation.

**Case 1.** - We observed 6 days old CA_6 calli. In \( G_h \), the relative frequency was of 20 b mother cells for one mother cell of the B type (CA_6). In \( G_h \), there were 5 z mother cells for 12 Z cells of the new type.

**Case 2.** - In a 55 days old callus (CA_{55}), the z cells of a new type in \( G_h \) were more numerous than the a cells of the mother type in \( G_h \). New cell types from \( G_h \) became dominant and organogenetic tissues issued from the callus in time showed a z character, although they originated from a b cell type.

**Case 3.** - The correlation between the fluctuation in mitotic activity and the synodic lunar cycle is particularly well illustrated by CA_{22}. This rather old callus was placed in culture at full moon. Examining the frequency diagram (Fig. 2-4) and taking into account diploid cells only, the a cells of the new type never replaced the b type cells, at least not in the same proportions. In fact, part of the new cells had doubled their chromosome numbers and formed tetraploid, more rarely octoploid cells. This behaviour is discussed in chapter 3.

These cases point to the importance of the variation in relative frequency of amplified cells as compared with the periodicity of the synodic lunar cycle.

When considering the total number of amplified cells of types Z + B + A (Fig. 2-5), a significant positive trend was observed during the first and second quarter periods of the lunar cycle. A significant depression marked the days around the full moon. In figure 2-5, mean values in the shaded zones of safety (\( \alpha = 0.05 \)) are estimated to be distributed at random. Trends observed, e.g. both peaks at the first and second quarters and the depressions at full moon and new moon were mainly due to the mitotic activity of B cells.
The particular frequency of Z cells during new moon is to be noted (Fig. 2-6). A relative peaking of Z characteristics (ca 85%) was observed in the week around the new moon. Around full moon, there was a relative minimum. In the week before the full moon, the A cells showed a passing peak of activity, preceding an increase of B cell activity.
Figure 2-6: Trends in mitotic cell activity, related to the synodic lunar cycle divided in 6 parts of approximately 5 days each. ▲ = means observed for amplified Z cells, □ = means observed for amplified A cells, grey zone delimited by α = 0.05. Note the peak at new moon for the Z cells and the one at full moon for A cells. To be compared with figure 2-5.

2.3 - The level of the cell

The present section presents the results of the second set of experiments, concerning chloroplasts as markers of different cell behaviour caused by different DNA properties. Chapter 3 gives the further interpretation of these results.

2.3.1 - Results in near normal cases

The first group of results concerns the control plants and the adventitious clones which are diploid or differ little from the dihaploid control. These results concern, in this order, the chloroplast number in two controls, the variation in chloroplast number with the level of sampling in the plant and the mean numbers of chloroplasts in adventitious diploid clones.
Figure 2-7: Chloroplast numbers per stomatal guard cell pair and control ploidy. Mean number and frequency distribution of chloroplasts in BF15 diploid control explant. A functional haploid cell is a diploid cell with one mute set of chromosomes, and a functional dihaploid cell is a tetraploid cell with two mute sets (see Ch. 3). Values analysed on probit paper for three controls, Ds1, Ds2 and Ds3. There is one, trimodal, central diploid cell group including 82.6 % of the cells, with a mean chloroplast number in between 10 and 15. This group is surrounded by two smaller ones. One includes mainly functional haploid cells, 12% of the total and with 6 to 10 chloroplasts. The second one includes mainly functional dihaploid cells, 5.4% of the total and with 14 to 19 chloroplasts.
Figure 2-8: Chloroplast numbers per stomatal guard cell pair and control ploidy. Mean number and frequency distribution of chloroplasts in B/F15 tetraploid control explant. A functional haploid cell is a diploid cell with one set of chromosomes, and a functional dihaploid cell is a tetraploid cell with two sets (see Ch. 3) Values analysed on prohit paper for three controls, T_s1, T_s2 and T_s3. There is one, trimodal, central tetraploid cell group including 67.7% of the cells, with a mean chloroplast number in between 20 and 25. This group is surrounded by two smaller ones. One includes mainly functional dihaploid cells, 17.2% of the total and with 14 to 19 chloroplasts. The second one includes mainly functional tetraploid cells, 15.1% of the total and with 25 to 35 chloroplasts.
• Chloroplast number in two controls (BF15, dihaploid and tetraploid)

The frequency distribution, as shown in figures 2-7 and 2-8), shows comparable results for both controls. There is one trimodal central group with a mean chloroplast number ranging between 10 and 15 for the dihaploid control (2n = 2x) and between 20 and 25 for the tetraploid control (2n = 4x).

The majority of this group rests on the following score of cells belonging to it:

# 82.6% of the observed cells for the dihaploid (Ds1 + Ds2 + Ds3);
# 67.7% of the observed cells for the tetraploid (Ts1 + Ts2 + Ts3).

The group is surrounded by two minority groupings. The chloroplast numbers in the first one range in between 6 and 10 with a relative mean frequency of 12%. In the tetraploid control this is respectively from 14 to 19, and 17.2%. The chloroplast numbers in the second group range in between 14 and 21 with a relative mean frequency of 5.4%. In the tetraploid control the range is from 25 to 35, and the frequency is 15.1%. The second group has a higher chloroplast number than the central group.

What do these groups represent, among our results?

At first sight the central majority group, with mean values very close to the means of the whole sample (11.87 chloroplasts against a mean of 11.76 for dihaploids, 21.65 against 21.82 for the tetraploids), represents cells with diploid (2n = 2x) or tetraploid (2n = 4x) nuclei. The group with values between 2n = 2x and 2n = 4x would be formed by cells with 3x. The group with lower values, between 6 and 10 chloroplasts, would contain haploid cells with n = x. This takes into account that the chloroplast number is proportional to the chromosome number. This is the very reason that we chose this number as a marker at the cellular level. In chapter 3 this assumption will be discussed very thoroughly.

This matter can not be taken up, however, without the second batch of results. This allows to better understand the meaning of the three modulations of the central group. These are the results of individual variations per plant showing one principal and one secondary modulation, both distinguished by a mean chloroplast number of either 10, or 12, or 14 in the dihaploid control.

• Variation linked to the sampling level on the stem.

The activity of the end meristem of the stem varies in time. In our model, both the fifth leaf under the apex and the fifth leaf above the stem base possess ripe chloroplasts. They are distinct by the moments of their formation by the terminal meristem. This was earlier for the lower of the two (moment t₁) and later for the upper one (moment t₂). The differences between t₁ and t₂, if any, represent variations in the nature of the proliferating meristematic tissue. Indeed, chloroplasts do not become active before having matured during some period, but their genetic characteristics are expressed in an undifferentiated way as proplasts as soon as the embryonic or in vitro cells start their divisions.
Figure 2-9: Variation in meristematic activity in one set of three 7 weeks old plants originating from *in vitro* explants. Criterion is the chloroplast number per stomatal guard cell pair. Observations at two leaf levels on the stem, the oldest near the base (time t1) and the youngest near the apex (t2). The results obtained with chloroplast numbers as markers confirm those with nuclear DNA-mass as a marker (Fig. 2-3). The contents of the stomatal cells show 3 modes, a central one with 12 chloroplasts and two lateral ones with 10 and 14 chloroplasts, respectively, i.e. differing by ~ 17%. Both markers, chloroplast number and nuclear DNA-mass, hence are indeed correlated. The nature of the stomatal cells depends on the nature of the nuclear DNA. During stem growth, the principal mode observed at t1, the initial character, may have changed at t2, because a new kind of meristematic cells has come to determine growth. In this case there is occurrence of *chimerae* (see text).
Some dihaploid plant samples (D2) provide an example. The kinetics of the variation are shown in the frequency diagrams of figures 2-9 and 2-10. There are three types of variation.

# In the first case, the main modulation at t₁ in the fifth leaf above the stem base is shown by cells with 10 chloroplasts, the relative frequency of which decreases simultaneously with the increase of the proportion of cells with 12 chloroplasts in the fifth leaf from the apex, where they form the majority mode at t₂. This was checked in plants 1, 2 and 3 (Fig. 2-9) and plant 8 (not shown). The general means per plant, each resting on examination of 30 stomates are:

- Plant 1: level t₁: 10.5 chloroplasts
- Plant 2: level t₁: 10.6 chloroplasts
- Plant 3: level t₁: 11.4 chloroplasts

# In the second case, plants 4 and 5 (Fig. 2-10) and 4 plants not shown here showed the principal mode to be formed by cells with 12 chloroplasts at t₁. For t₂ it is replaced by a mode of cells with 11 chloroplasts, showing characteristics intermediary between those with 10 and those with 12 chloroplasts. The general means per plant are:

- Plant 5: level t₁: 12.0 chloroplasts
- Plant 6: level t₁: 10.7 chloroplasts

# In the third case, concerning plant 7 (Fig. 2-10), the cells with 12 chloroplasts form the principal mode, like in the former case. However, they are supplanted at moment t₂ by cells with 13 chloroplasts, being intermediary between those with 12 and 14 chloroplasts. The general mean is:

- Plant 7: level t₁: 11.0 chloroplasts

These results show the same rules of proliferation as the differences in DNA mass of the first experiments (see Sect. 2.2). There are three modes, i.e. two variants and a specific mode. The two variants we will call Vₘ₁, the minor one, and Vₘ₂, the major one. The main mode is Mₙ. Both variants differ from the main mode by two chloroplasts. Hence Vₘ₁ is by \( \frac{2 \times 100}{12} = 17\% \) poorer in chloroplasts than Mₙ and Vₘ₂ is richer by the same amount. This validates the results of the both first set of experiments (see Sect. 2.2.) and the choice of the chloroplast number as a marker.

Moreover, the above results demonstrate the existence of intermediate cells between Mₙ and Vₘ or Mₙ and Vₘ. Hence there is recombination potential. This is a subject tackled in chapter 3.

- **Mean number of chloroplasts in adventitious diploid clones**

The results of the study of cell populations have been grouped in classes 6 to 21. The group of plants used for this classification includes 11 diploid adventitious clones and 3 dihaploid controls. The notes used concern the number of observed cells per clone, the general mean Mₙ (see above) and its possible fluctuation (\( \alpha = 0.05 \)), the mean cell populations clone for different modes and, between parenthesis, their relative frequency, as well as the mean of all cells (2x) and the relative frequency of the group (2x).
The adventitious clones and the three control clones (2x) show a great similitude in both distribution and means of cell populations. Four clones, D_{111}, D_{37}, D_{99} and control D_{53} are distinct from the other ten by having on the average ca 13% less of diploid cells (2x). This low amount is accompanied by a different character of the V_m and V_M modes. Their means take intermediate values in between their own and those of the specific mode M_s. Moreover, this is accompanied by a strong increase of the relative frequency of supposedly “haploid” cells, which is 9% in barely disturbed clones and 16 to 25% in the four clones examined here.
Here follows the comparison of mean values of cell populations.

# (2x) cells

Excluding the intermediate values of D_{63} (V # 11; V # 13) for the V_m mode, the mean values are close to:

- 10 chloroplasts for V_m
- 12 chloroplasts for M_i
- 14 chloroplasts for V_M

# Supposedly haploid (x) cells

Excluding an extremely rare cell with 6 chloroplasts, observed once in each of two clones, three modes were found:

- ca 8 chloroplasts most frequent and most abundant in 160 cells (10 clones; x \approx 8.4)
- ca 9 chloroplasts very abundant, but less frequent in 179 cells (5 clones; x \approx 8.9)
- ca 7 chloroplasts rather rare, observed in 3 cells in one clone, D_{99}

# Supposedly triploid (3x) cells

The mode which is clearly in the majority is the most frequent one, observed in 10 clones, of a chloroplast number around 16. This is the equivalent of twice the majority value in supposed haploids. (mean value over 85 cells x \approx 15.80)

It seems likely that two somewhat lower means may be joined to the above mode (clones D_2 with 15.43 and clone 111 with 15.25). The same applies to part of the cells with means around 17. This means that the chloroplast number of the cells in question varies between 15 and 17, with a large majority possessing 16 chloroplasts.

Two other cell populations may be distinguished. The first one has a modal chloroplast number of 18, varying between 17 and 19, the second has a modal number of 20 varying between 19 and 21. The latter case is that of the tetraploids.

# Some remarks

The overview of this set of results shows several uncertainties remaining in our analysis. It is necessary to go deeper into the matter, especially as regards our marker. Before being ready for the general discussion of chapter 3, three essential questions require an answer.

First, if the chloroplast number per pair of stomatal cells indeed were proportional to the ploidy of the plant and mirrors only this property, then why is the mean chloroplast numbers in the tetraploid control not the double of that in the dihaploid control? This question arises for all values, the general mean, the mean for all (4x) cells and the mean for each mode of (4x) cells. The number of chromosomes, on the contrary, did double.

Second, we saw that in diploid clones the difference between the two modes V_m and V_M and the specific mode M_i is 2 chloroplasts, around 17% of the value of M_i. Why, then, does the change in ploidy have no influence on this difference, which remains of 2 chloroplasts, be it in the control or in tetraploid adventitious clones, or also in hexaploid, octoploid decaploid or higher ploid clones?

Third, what is the real nature of the cells supposedly haploid and triploid?
2.3.2 - Results in complex cases

The second group of results concerns the adventitious mixoploid and tetraploid clones. These results concern, in this order, depressed clones and tetraploid clones close to the tetraploid control. The data are not reproduced here.

Fifteen clones were selected to illustrate how the change occurs in the spread of cell populations that starts with a strong disturbance of bivalence in diploid cells. Indeed, the original tissue that was placed in vitro came from a diploid plant. We will examine seven mixoploid [(2x)to(4x)] clones in a depressed state and with a strong tetraploid majority, six tetraploid clones close to the tetraploid control, and two mixoploid [(6x to 8x), (3x to 4x)] clones.

# Depressed [(2x) to (4x)] clones

A depressed clone has a deficient metabolism in comparison with other clones.

The seven clones used to observe chromosome numbers were of the same descent as those having served for the study of chloroplasts. The chromosome numbers belonged to tetraploids. These were euploids (2n = 48 chromosomes) like T_{71}, or aneuploids (2n = 46 chromosomes) like T_{176}, or having 44 to 48 chromosomes in still other clones. In fact, according to the plant observed, that is according to the change which occurred in the cells going to build its meristems, one studies either a depressed diploid clone, or a tetraploid clone. Results pertaining to the latter case are presented later.

The results are divided in four groups according to the impact of the disturbance.

# Three clones, T87, T13 and T176, strongly depressed as compared to other controls

There is a weak relative frequency of diploid cells, ca 20%, and a comparable frequency of tetraploid cells, except in T_{13}, where it is lower, i.e. 14.2% (2x) against 20.6% (4x). This group is marked by a high proportion of functional dihaploid cells, between 58% and 65%.

# One clone, T71, medium depressed as compared to diploid or tetraploid controls

The relative frequency of (2x) cells decreases to 13.3%. Simultaneously, a relative increase is found of the number of tetraploid cells, which becomes 40%, and a decrease in functional dihaploids to 41.3%.

# Three clones, T132, T137 and T146, barely depressed as compared to controls

The relative frequency of diploid cells is low, from 4.4% to 2.4%, whereas the proportions of tetraploid cells becomes significantly larger, from 51.2% to 58%, than the proportion of functional dihaploids, 36.8% and 34.8% respectively.

The response to desequilbreria due to stress in vitro is clear. There is an increase in the level of ploidy, both among diploid cells and functional haploids. The latter transform themselves into functional dihaploids and tetraploids.

Whatever the clone, the impact of disturbance is greater on M_{s} and V_{M}, the specific mode and major variant, than on the minor variant V_{m}. In strongly depressed clones, the proportion of tetraploid M_{s} or V_{M} cells is small, if any. A strong majority of cells, or all cells of this level of ploidy are functional dihaploids. For instance, in T_{87}, no tetraploid M_{s} cell was observed and less than 1% of V_{M} cells. The relative frequencies of functional dihaploid cells here are 34.8% for M_{s} and 26.8% for V_{M}.
Tetraploid (4x) clones similar to the tetraploid clone

These were the clones T_{127}, T_{26}, T_{29}, T_{45}, and T_{25}. The spread of the cell populations closely resembles the one of the tetraploid control. The only small difference is a slightly lower proportion of dihaploid cells in the control. There are some 10 to 20% in the control and 22 to 25% in the adventitious clones. However, in both cases cells appear that belong to populations of a higher level, with more than 30 chloroplasts. Their number may be small, but their presence is an additional indication allowing to distinguish them from the mixoploid clones mentioned earlier, which are [(2x) to (4x)] chimeras (also see Figs 2-9, 2-10).
Chapter 3  Cells under stress follow rules

The simple results reported in the previous chapter provide simple and clear answers to questions in the specialised field of cell biology. In particular, statistical values for DNA mass and chloroplast numbers in potato plants obtained under precisely defined conditions in vitro were elucidated, as well as their change over time, due to processes of mitosis and cell division. These results, up to that level of scientific questioning, speak not only for themselves. They speak a clear language too.

But there is more. The field of life is vast. It ranges in practical terms from the information contained in macromolecules to the information contained in the solar system. The scientific evidence brought together by Lovelock (1979, 1988) in his much-abused "gaia theory" has force of proof. In the previous chapter, we produced new data proving that a correlation exists between the lunar cycle and the behaviour of DNA in the nuclei of cells cultured in vitro (also see Rossignol & al. 1990). However, the numbers and proportions found in DNA and chloroplast values strongly remind one also of the studies in tree architecture (Hallé & al., 1978; Édelin, 1984, 1991), in particular when thinking of phyllotaxis and the Fibonacci series, or the way in which trees may show depauperate architecture. They also recall phenomena of fragmentation and fusion in ecosystem units (Oldeman 1990).

Where do the results of our in vitro experiments fit into natural history in general? The quest into this field of knowledge fills the rest of the present book. We adopted a system hierarchy of nested living systems and subsystems to define the levels of organisation, each of which requires its own research techniques in terms of experiments, observation and data processing. The present chapter concerns the levels which were experimentally explored in chapter 2.

3.1 Data processing

The data were processed in the normal way for cell and cell nuclear research. Their mathematical ordering rests on safety assessments by the use of ordinary statistics. These are focused, however, on the understanding of the spread of values around normal regression curves, and their dynamics in time, rather than upon the calculation of average values and regressions as an end product. They are used only as tools of reference.
Figure 3-1: Comparison between three types of cellular division. Mitosis is a somatic process, meiosis is a sexual process (see Fig. 2-1). At the close of meiosis, after two successive divisions, chromatic reduction yields four cells, two paternal and two maternal ones (7). They also possess DNA-sequences of mixed paternal and maternal origin, due to crossing-over during the pairing of chromosomes in the prophase (3). Somatic meiosis is like meiosis up to the anaphase I, except for two facts. First, no crossing-over occurs because strongly oxidised cell membranes prevent chromosome pairing (asynapsis). However, there is a substitute for crossing-over, limited to A cells issuing from Z cells, possessing minute, extra, circular chromosomes (to the left of (2)). Second, twisted and prematurely separated daughter chromatids prevent chromosomal reduction in anaphase I. The last division (telophase: Fig. 2-1) restitutes the chromosome pair number of the somatic cell. Meiosis and somatic meiosis have in common that paternal and maternal DNA is exchanged, but in very different ways.
This research orientation, scrutinising variation rather than means, was of course reflected in our choice of instruments. The cytophotometer of Patau (1952), in particular, provides us with a precise image of the mitotic cycle at a precise moment. This is the moment, the prophase, when the two chromosomes of paternal and maternal origin have just doubled and the membrane around the nucleus has ruptured. Patau’s instrument indeed has the advantage to show this particular phase, whereas the modern flux cytometers display the end of the cycle when mitotic dynamics are at rest again. Patau looks at the building activities, whereas the other instruments often look at the completed house.

In the next parts of the present chapter, the results will be discussed as is usual, by comparing them with the existing knowledge in this field. We will pay particular attention to the definition of stress factors modulating those inherent in the disruption of living structures in vitro.

3.2 Cytogenetic processes implied in somatic meiosis

3.2.1 - DNA masses under stress and the lunar cycle

Among the many reported cases of correlations between the lunar cycle and the periodicity of biological or physiological processes, those of Mikulecky & Zemek (1992) are noteworthy here. A comparison between the more sophisticated Eucaryotes, the predatory mites of which the activity was studied by these authors, and our simple potato genomes has explanatory virtues.

As to the part played by amplified cells in a callus, we know that in spite of constant in vitro conditions the initial character of cells can be changed by chimaerization. This is the final activation of one of three nuclear DNA types, characterised by their mass (chapter 2). Their mitotic divisions speed up because amplified cells with new properties were formed from the outset in the cultures.

Moreover, primary cell permeability regulates nutrient diffusion out of cells. Hence amplified cells seem to be ideal nurturing cells. Nurturing cells can activate mitotic divisions by rapid diffusion, as needed to double the cell volume during the interphase of mitosis. Indeed, amplified Z, B and A cells show some analogy with albumen cells in seeds. The cell’s nuclear DNA mass is close to that of a triploid cell, although it shows (2x + x) in amplified cells against (2x + y) in the albumen.

This context makes less surprising the comparable results from fields as different as predatory mites and mitosis of potato cells in vitro. The phenomenon is basically the same, only the research method is different. Mikulecki & Zamek (1992) state that “the rate of food intake appeared to be positively correlated with the ovipositional rate in a remarkably linear pattern”. This tells us that fluctuations in predatory activity reflect fluctuations in the need for nurturing reserves for the eggs during the period of oviposition.

Some reports mention “saltatory” variation of nuclear DNA mass (e.g. Bennett & Smith 1976; Essad & al. 1975, Essad 1987). The work on flax by Evans & al. (1966) or Alberts & al. (1986) proves that usually silent parts of the genome, i.e. repetitive and dispersed DNA sequences, can be activated at intervals. This happens when mother plants grow under stress, e.g. due to increased amounts of phosphorus in the medium. Changes in progeny included
modified physiological responses, changed growth properties or phenotypical characteristics like L- and S-forms. These modifications are linked to a difference of ca 16% in nuclear DNA mass, added or lost. This is the “DNA quantum” of chapter 2. It closely corresponds to the difference in nuclear DNA mass found between pairs of our z, b and a cells.

These data show such processes to be adaptive and triggered by stress, e.g. in plants having been subcultured in vitro. What is more, comparison of our results with those of Mickulecki & Zemek (1992) shows the physical mechanism to be the same as in stress-less conditions. In proliferating potato cells there is stress, in mites there is none during predation. Nevertheless, both mites and b-cells of potatoes follow the periodicity of the synodic lunar cycle. Only when environmental stress is applied to mother organisms do z and a cells appear to replace the b cells in potato cultures. Our monitoring results for vegetative progenies (chapter 2) show fast growth and fast ageing to characterise the a form, slow and retarded development the z form.

The mechanism of the synchrony between lunar cycle and biological periodicity has lead to much speculation. However, a coherent model based on electromagnetic diffraction is available (Rossignol & al. 1990). At dawn and dusk, our planet forms a round screen intercepting the sun’s rays. At the border of the screen, rays interfere and energy is redistributed according to the Principle of Fresnel. Interference leads to rhythmic increase and decrease of energy.

Three conditions have to be met before diffraction occurs. First, light rays must be polarised, as is the case when they enter the atmosphere in the above conditions. Second, they must be coherent, which means that they are “in phase” and this is so because of their common source, the sun. And, third, they have to be simple and monochromatic, which they only are during two moon phases, new moon and full moon.

This is due to another physical factor, the “thermic tides”, occurring when the earth and the moon are in conjunction with the sun. During these two periods and at the sun’s vertical, thermic waves enter in resonance with the gravitational wave, the two having the same period. Their resonance initiates strong heating in the high atmosphere. The moon causes atmospheric tides. However, its gravitational forces are too weak to be felt, except where the atmosphere is densest and coldest. Hence the tides are effective only at the periphery of the overheated zone, so increasing the thickness of the layer to be crossed by the solar radiation. This occurs perpendicularly above an observer, at sunrise and at sunset during the periods of full and new moon.

Another influence is exercised by numerous, small diffusing particles in the middle and lower atmosphere. They reduce the part of the solar radiation which is restituted with the same momentum as the initial radiation. The weakened part includes the longest wavelengths. The thicker the layer to be crossed, the nearer to the red are these wavelengths. By this mechanisms, sun rays are selected which are all-but monochromatic. This happens both at the full moon and the new moon, when sun, moon and earth are aligned. These rays generate a weak beat at the “edge” of the round earth screen in their way. However, they are amplified by the ionised air at the confines of the stratosphere, receiving an impulse from electrically charged particles coming in with the electromagnetic solar wind.

Signals produced by these processes can be captured on the ground by sensitive sensors in the two passing bands. Their wavelengths, $\lambda = ca 660$ nm and $\lambda = ca 730$ nm, are in the red and the far red. This agrees with comparable observations made with semi-conductor gear by Krasnogorskaya & Parkhomov (1989).
At the level of the cell, and only under stress, bioreceptors probably are capable of capturing such signals too and to transform them in selective impulses to produce the co-enzymes accompanying stress proteins (cf. Van Hinsberg 1997). Perhaps they function like phytochromes in seed germination. In the absence of unusual stress, biological and mitotic activity depend on the characteristics and intensity of light at the time preceding dusk or following sunrise.

The red and far red monochromatic radiation explains the different responses at the DNA level under stress. That these responses are different is due to the far red signals during the full moon (λ = ca 730 nm) compared to the red during the new moon (λ = ca 660 nm). Indeed, the far red radiation from the round border of our planetary screen interferes with far red lunar radiation coming from the opposite side. This can initiate a resonance in the far red.

### 3.2.2. - From DNA mass to chloroplast number: two parameters compared

What is the precise meaning of the number of chloroplasts, to be used as a marker of cell behaviour which is steered by DNA activity? And how is this meaning best expressed to link it to matters of ploidy, depression of clones, or stress behaviour?

The meaning of the chloroplast number follows from the work by Benzine-Tizroute (1990). These studies concerned the meiosis in Solanum tuberosum L var. BF15, both as a dihaploid and a tetraploid. Bivalents are pairs of homologous chromosomes of paternal and maternal origin. The mean number of bivalents observed in metaphase I was 9.1 in the dihaploid control and 16.8 in the tetraploid control. The ratio between (4x) and (2x) hence is 1.85.

The same ratio, assessed with the parameter of chloroplast numbers, using general mean values, indeed is exactly 21.82 / 11.75 = 1.86.

The chloroplast number hence is proportional to the number of bivalents, not to the total number of chromosomes at every level of ploidy. Functionally, a tetraploid therefore turns out to be a diploid of which the number of homologous paired chromosomes has increased, even doubled in the strongest case.

What do the modes with 7, 8 and 9 chloroplasts represent? If they were haploids, why do their respective values not correspond to half the values of the corresponding diploid modes? Before tackling these questions, the haploid function will be compared to the diploid one.

In a diploid cell, both sets of functional parental chromosomes, paternal and maternal, in the stage of meiotic pre-division are able to form pairs. For instance, the diploid control has 2*9 functional chromosomes. In the case of a haploid cell originating from a diploid cell having completed the double reductive division of meiosis, there is only one set of 9 chromosomes which regulate the life of the organism.

This state of affairs is expressed by chloroplast numbers, which after our results are the following.

# (2x9) chromosomes are expressed phenotypically by 12 chloroplasts for the M5 mode in a diploid cell, 10 for the Vm mode and 14 for the VM mode.

# half of (2x9) functional chromosomes are expressed phenotypically by 12 / 2 = 6 chloroplasts in the M5 mode of a haploid cell, 5 in Vm and 7 in VM.

The last haploid cell type, with 6 chloroplasts in the M5 mode, we will call reduced diploid, as the opposite of the functional haploid cell (Alberts & al. 1983). This in fact is a diploid having lost its bivalence. Its functions are ensured by only one of both complete of chromosomes.

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(here n = 12). This happens when a recessive gene undergoes a mutation which is lethal for the organism and so endangers the life of the cell.

Because 6 chloroplasts correspond to 9 chromosomes, our diploid cell which is a functional haploid has the following phenotypical expression:

- $6/9 \times 12$ = 8 chloroplasts in the specific mode $M_s$, or
- $8 - \{2 / 2\} = 7$ chloroplasts in the minimal version $V_m$, or
- $8 + \{2 / 2\} = 9$ chloroplasts in the maximal version $V_M$

In the case of a tetraploid cell which is a functional diploid, the phenotypical expression is doubled, i.e. there are 16 chloroplasts ($M_s$) or 14 ($V_m$) or 18 ($V_M$).

However, in that case, why isn’t the amplification or deletion double or quadruple in a tetraploid or an octoploid cell, in view of the twofold or fourfold multiplication of maternal chromosomes? The logic answer to this question is, that all polyploids with regard to bivalence are nothing else than assemblages of diploids, so that the amplification or deletion process does not need to be multiplied.

We may now attempt an overview of the somatic meiosis of the mother-cells in a proliferating environment. This is an incomplete meiosis. The daughter cells are diploid, not haploid. The two cells issued from division possess both sets of chromosomes from the original cell. However, contrary to classic mitosis, this division conserves a unique meiotic feature. This is a crossing-over which causes DNA sequence exchange between the two daughter chromatids during the prophase before the metaphase and the first meiosis or anaphase I (Fig. 3-1). In section 3.3. we will see what precise kind of crossing-over causes this to happen.

This particular behaviour was observed in cytological studies in vitro on cells originating from various plant species (Nuti Ronchi 1990, Nuti Ronchi & al. 1990, Nuti Ronchi 1991). These authors state: “...the second equational mitotic division is lacking in somatic meiosis, which only proceeds up to anaphase I stage...........In fact, the precocious separation, at anaphase I, of the centromeres of the bipartite chromosomes may restore the diploid number at 2C level”. This is exactly what is shown in figure 3-1.

Which are the mother-cells capable of accomplishing this incomplete meiosis? In suspension culture, they belong to the population “...(which) enters, at every subculture, a meristematic phase which lasts only a few days (with a remarkable similarity for all species when grown in optimal conditions): approximately from day two to day seven.” (Nuti Ronchi, 1991, p.4).

Our own observations on proliferating cell populations of a callus (Chapt. 2) and on cells characterising the meristematic activity of a plant at two growth moments (section 2.3) provide a striking parallel between the two images of the transformation of the original cell in vitro and in vivo.

In vivo, under conditions without stress and especially in Angiosperms, the meiotic transformation of the mother cell yields four megaspores. In most dicotyledons, one of those four, a haploid, fully ripens after meiosis and after many divisions constitutes an embryo sac of eight or more cells. One of those cells, at the side of the micropyle, is the female gamete or oosphere which, after fertilisation by the male gamete, will become diploid and yield embryo cells.
*In vitro*, the image is comparable, but the sequence is contracted. The chromosomes studied initially were autosomes, i.e. asexual chromosomes. Nevertheless, the fact of somatic meiosis implies a momentary “feminization” of the mother cells, even if the chromatic reduction has aborted. Feminization is triggered by inactivation of the zones determining the male sex. In plants building both autosomes and heteromorphic sexual chromosomes, such as *Melandrium dioicium*, the region which determines the sexuality is in the middle of the Y chromosome, on both sides of the centromere (Gorenflo 1983). The restitution of diploidy in the anaphase I after the crossing-over of two pairs of sister chromatids in the prophase is a shortened, but similar version of the fertilisation of the oosphere and the beginning of proliferation of diploid embryo cells.

This sheds new light upon the $X_{ist}$ gene discovered by two British teams (Rastan, and Willards, ex La Recherche, 1994). This gene has original features. In mammals, females are distinct from males not so much by the two sexual chromosomes, which are quite similar with an xx pair in the female chomosome instead of an xy pair in the male one. The main difference is, that the genetic functions in females are ensured by only one of the two chromosomes of paternal or maternal origin. The other one is disabled. This happens three days after fertilisation. It is triggered by the activity of one disabling gene, $X_{ist}$ in mice, $X_{ist}$ in *Homo sapiens*. This gene is situated in the middle of the disabled x-chromosome.

It has strange properties. It is transcribed upon a RNA molecule larger than the usual RNA molecules, obtained from other genes. The gene in question further apparently has no code for any protein and stays put in the nucleus, whereas usually RNA lecture and translation take place outside the nucleus in the cytoplasm. These observations led the British teams to formulate the hypothesis that “...the RNA of $X_{ist}$ is itself a component of the disabling signal which is propagated along the chromosome.” The RNA is thought to become bound to the proteins forming the heterochromatin and so to form a complex which fixes itself to the chromatine near $X_{ist}$. From there it would form the primer for the propagation of heterochromatin, particularly towards the sites corresponding to the genes for ribosome DNA.

The odd properties and behaviour of this RNA do remind one those of a ribozyme, which is a RNA molecule with enzymatic attributes and also a carrier of genetic information. If this were so for our RNA, its behaviour in conditions without stress would be that of an inactive enzyme in a state of rest. To be enabled it would need a coenzyme, e.g. a stress molecule. It would then play an inhibiting role towards heterochromatin proteins. This would open “windows” on the chromatine allowing the enabling of usually mute genes. According to our study, the choice of windows is specific. It depends on electromagnetic environmental conditions influencing ionic reactions. This implies participation by a second coenzyme with a mitochondrial origin.

Let us now go back to our proliferation schedule (Fig. 3-2). Our hypothesis remains that the function of the disabling gene of the x-chromosome has been conserved in the course of the evolution, and that it is present in plants in general and the potato in particular.

In our model, the founding cell which was blocked in stage G2 of the mitotic cycle provides, after the cycle has started up again in the culture medium, two daughter cells with different destinations.

The first one, brought forth by the parental chromosomes already formed or prepared on the original plant before it was cultured, has the characteristics of the original tissue. After suc-
cessive mitoses and cell divisions it will constitute a lineage, the primary cell of which is the founder cell.

The second one, brought forth by the daughter chromatids of the founder cell, emits stress proteins and becomes the seat of somatic meiosis. It will be the mother cell of the lineage of secondary cells having a variant nature. On the chromosome of maternal origin, the $X_{1st}$ gene is enabled and its transcribed RNA is enabled by its links with the two coenzymes.

Observation of nuclear DNA mass in cell populations (Chapt. 2.2) shows the proliferation of the meristematic type to be preceded by the establishment of four or five cells by successive divisions. Each of these has a chromosome amplified by duplication (see Sect. 3.3). The DNA mass is amplified 1.5 times. These are the “nurturing cells”, probably linked together by cytoplasmic bridges like in some invertebrates. One of these cells will divide and initiate era
tion. The additional DNA is the provider of enzymes, ATP, RNA and diverse proteins needed for cell development and doubling of volume before dividing. This DNA hence activates proliferation. In section 3.3 the nature of the “nurturing cells” and the mechanism driving the “character” change of the entity called “variant” will be examined more closely.

![Diagram of cell proliferation](image)

Figure 3-2: Proliferation at the cell level, schematically showing what happens in apical shoot meristems when exposed to environmental stress. The same happens in every subculture and in proliferating cell populations of a callus. Also see figure 2-4.

Other authors since long had seen and described this phenomenon under different names: “metabolically labile DNA” (Sampson & al. 1963), “metabolic DNA” (Roels 1966), “DNA métabolique de synthèse” (Essad & al. 1975, Vallade & al. 1978, Essad 1987). It is an interphasic DNA, synthesised in stage G₂, which does not come further than the prophase.
chapter 3.3. this additional DNA will be seen to be a synthetic DNA, which is used temporarily as a control measure to mark DNA in specific sites as well as in genetic recombination.

Enabled RNA permits the reading of new genes. This reading occurs at the same time as the changing of nuclear DNA mass. In Chapt. 3.3 this changing of mass will be seen to correspond to a modification or rearrangement of chromatine.

Hence, the first division of the mother cell of the secondary lineage is slower than that in the primary lineage. Our observations show the meristem-like proliferation to be preceded by the positioning of about 16 nurturing cells with amplified DNA, just like in the primary lineage but in greater number. Though delayed, secondary cell proliferation accelerates relatively to primary cell proliferation, which leads to a changed nature of the future tissues.

In the apical meristem of the plant to be, the cells of the primary type will build the first leaf initials, whereas the following initials originate from proliferation of secondary cells of the variant type (see Figs.2- 9 and 2-10). We only show one pair of homologous chromosomes, a bivalent, per diploid cell, so as not to overload the figures. In fact, the average diploid potato cell of clone BF15 has 9 bivalents. During the succession of accelerated mitoses a recessive gene may be duplicated which is lethal for the organism. If this occurs in one bivalent only, the X, gene of paternal origin is enabled after “feminization” of the autosome. It starts the advancement of heterochromatine along the chromosome. Only one out of every two chromosomes remains active, but it ensures the continued functioning of the organism. In case of the bivalents affected being too numerous to continue ensuring the vital cell functions, asynadesis occurs. This disables the whole set of chromosomes by the same process. The cell then becomes a functional haploid, conserving its variant nature as shown by the indicator “number of chloroplasts”.

The group of cells “supposedly” 3, with the exception of the nurturing cells, is the set of functional di-haploids. These are functional haploids having doubled their chromosome number. In fact these cells are tetraploids (2n = 4x = 48 chromosomes), in which only one set of homologous x-chromosomes is functional.

With this information, table 3-1 now is seen to reveal both classes of haploid cells, i.e.

# reduced diploids (n = x), which are very rare and are observed only in two cases, control Ds1 and clone D3;

# functional haploids which are diploids of which only one of both x-chromosomes, of paternal or maternal origin, is functional. The mode best represented is M5, with 8 chloroplasts, followed by V4 with 9 chloroplasts.

Overall, the mode M5 with 16 chloroplasts (= 2 * 8) is most strongly represented.

Clones with a beginning desequilibrium, like D35, D37 and D99 have a higher share of functional haploid cells, from 16% to 25%, than control. Phenotypically, this results in a slight depression, visible by a smaller size of the internodia and the whole plant. In the following pages it will be shown that the relative share of functional haploids increases with the degree of desequilibrium in a clone. The plant strategy then is to increase the number of bivalents by upping the level of ploidy. This happens in diploid cells as well as in functional haploids, transforming themselves into tetraploids and into functional dihaploids respectively.
Table 3-1: Cell population buildup, cell modes, chloroplast numbers and ploidy

<table>
<thead>
<tr>
<th>Modes</th>
<th>Means</th>
<th>Chromosome number</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>6.3 ± 0.169 chloroplasts</td>
<td>nx = 1x = 12</td>
</tr>
<tr>
<td>12</td>
<td>12.241 ± 0.039 chloroplasts</td>
<td>2n = 2x = 24</td>
</tr>
<tr>
<td>18</td>
<td>17.69 ± 0.259 chloroplasts</td>
<td>nx = 3x = 36</td>
</tr>
<tr>
<td>22</td>
<td>22.123 ± 0.239 chloroplasts</td>
<td>2n = 4x = 48</td>
</tr>
<tr>
<td>32</td>
<td>31.7 chloroplasts (mean over 2 clones)</td>
<td>2n = 6x = 72</td>
</tr>
<tr>
<td>41</td>
<td>41.486 ± 1.025 chloroplasts</td>
<td>2n = 8x = 96</td>
</tr>
</tbody>
</table>

In all clones, the upheaval is more important in the specific mode M₅ and its major variant V₉ than in the minor variant V₉. In strongly depressed clones, the share of tetraploids in M₅ or V₉ is minute or nil. A large majority or all cells at this level of ploidy are functional dihaploids. In T87, for instance, the relative frequencies of functional dihaploids are 34.8 for M₅ and 26.8% for V₉, without any tetraploid having been observed in M₅ and less than 1% in V₉.

### 3.2.3 - The evolutionary strategy of proliferating cells

To finish section 3.2, we will now reconnoiter the direct bearing of our results on the evolutionary strategy of proliferating cells.

The build-up of cell populations is logical. To demonstrate this, table 3-1 shows the chloroplast number against the level of ploidy. In this way, the characteristics of the cells composing every population is defined and their potential in an environment of proliferation can be assessed. The image evoked by table 3-1 is quite similar to the correspondence between chloroplast numbers and chromosome numbers established by Frandsen (1968). His means were calculated from data he obtained from hybrid potatoes *Solanum tuberosum* x *S. phureja*. These plants had diverse genotypes and diverse levels of ploidy. Here are the results of his calculations.

These means are very close to those we found as general means and in the specific mode M₅ for the diploid and tetraploid controls. The value of both variants, V₅ and V₉, can be easily deduced at every level of ploidy by subtracting or adding two chloroplasts per level. For the hexaploid level we accept Frandsen’s mode of 32. As to the octoploid level, our analyses of cell populations show that the mode M₅ is 40 rather than 41. Indeed, the ratio $M_{5(8x)}/M_{5(4x)}$ of 1.82 and the ratio $M_{5(4x)}/M_{5(2x)}$ of 1.83 then are very close.

Let us now examine the different cell populations. First are the somatic cells proper. They are linked by the relation

$$y = n (2x)$$

in which n is a whole number and x equals one set of chromosomes.
• diploids, $2n=2x=24$ chromosomes, marked by chloroplast numbers $M_s = 12$, $V_m = 10$, $V_M = 14$
• tetraploids, $2n=4x=48$ chromosomes, marked by chloroplast numbers $M_s = 22$, $V_m = 20$, $V_M = 24$
• hexaploids, $2n=6x=72$ chromosomes, marked by chloroplast numbers $M_s = 32$, $V_m = 30$, $V_M = 34$
• octoploids, $2n=8x=96$ chromosomes, marked by chloroplast numbers $M_s = 40$, $V_m = 38$, $V_M = 42$

Figure 3-3: Architecture of the three different DNA forms. The only active DNA-form under normal conditions, without major stresses leading to blocked chromosome pairing (asynadysis, Fig. 2-1), is dexter B.DNA. When its regulation is strongly compromised, one of the two other forms, A.DNA and Z.DNA, may be activated as a "chaperon" of B.DNA (see Sect. 3.3.2.3). DNA has two strands rolled up in a double helix, the early strand 5'-3' serving as a matrix to the late strand 3'-5'.
In the second place we examine the functional haploids and the nurturing cells.

At every level of ploidy, chloroplast numbers of functional polyhaploids are a multiple of that of the functional haploid \((x_f + x_m)\) or \((x_{rf} + x_m)\), \(x_f\) being the set of functional chromosomes and \(x_m\) of mute chromosomes. Its chloroplast numbers equal 8 \((M_f)\), 7 \((V_m)\) and 9 \((V_M)\).

* functional dihaploids \(2(x_f + x_{rm})\) \[2 * 8 = 16\] chloroplasts \((M_f)\)
* \[2 * 7 = 14\] chloroplasts \((V_m)\)
* \[2 * 9 = 18\] chloroplasts \((V_M)\)
* functional trihaploids \(3(x_f + x_{rm})\) \[3 * 8 = 24\] chloroplasts \((M_f)\)
* \[3 * 7 = 21\] chloroplasts \((V_m)\)
* \[3 * 9 = 27\] chloroplasts \((V_M)\)

............. and so on.

At every level of ploidy, the chloroplast numbers of nurturing cells is easy to calculate when remembering that nurturing cells possess unstable interphase DNA. This is shown by sticking the superscript + to the ploidy indicator.

* nurturing diplloid \((2x)^+\) \[12 + 6 (or 12 * 1.5)\] = 18 chloroplasts \((M_f)\)
* \[10 + 5 (or 10 * 1.5)\] = 15 chloroplasts \((V_m)\)
* \[14 + 7 (or 14 * 1.5)\] = 21 chloroplasts \((V_M)\)
* nurturing tetraploid \((4x)^+\) \[22 + (2 * 6)\] = 34 chloroplasts \((M_f)\)
* \[20 + (2 * 5)\] = 30 chloroplasts \((V_m)\)
* \[24 + (2 * 7)\] = 38 chloroplasts \((V_M)\)
* nurturing hexaploid \((6x)^+\) \[32 + (3 * 6)\] = 50 chloroplasts \((M_f)\)
* \[30 + (3 * 5)\] = 45 chloroplasts \((V_m)\)
* \[34 + (3 * 7)\] = 55 chloroplasts \((V_M)\)

At every level of ploidy, chloroplast numbers of functional polyhaploids are a multiple of that of the functional haploid \((x_f + x_{rm})\) or \((x_{rf} + x_m)\), in which \(x_f\) or \(x_m\) is the set of functional chromosomes and \(x_{rf}\) or \(x_{rm}\) that of mute chromosomes. Its chloroplast numbers equal 8\((M_f)\), 7\((V_m)\) and 9\((V_M)\). In the reduced diplloid \((x)\), the chloroplast number equals 6 chloroplasts \((M_x)\), 5 chloroplasts \((V_m)\) and 7 chloroplasts \((V_M)\), respectively.

Why, finally, do these cell populations show the above logic in composition? The next section of this chapter (3.3.) will dwell on this aspect in particular. For indeed the whole build-up of these cell populations make sense only if cell proliferation is seen as a tool to implement an evolutionary strategy.

Here also lies the origin of chimaerae. Let us recall once more the disorder, the effect of which must be corrected if the organism is to survive. This disorder was described above as asyndesis or defective matching of homologous chromosomes. If the number of mismatched chromosomes passes a certain threshold, the main cell functions cease to be ensured in the diploid state. The first response of the cell is to become haploid by rendering mute one of the two sets of chromosomes. However, the return of this operation is marginal because of the depression caused by the switch-off of half the genome. The second way out of the chaos of asyndesis is the suppression of mutation by genetic recombination. This is done by crossing-
overs during somatic meiosis, which initiate a rapid meristem-like proliferation during the first week of in vitro culture.

In vitro culture, followed by explants as cuttings, lead to the formation of a new plant. The second process described above sometimes leads to parts of the final organism having distinct genotypes, as if they were parts of one plant grafted on a quite different plant. Such plants combining characteristics of different genotypes are known from old as chimaerae. In view of evolutionary strategies it is important to know that chimerae are not horticultural freak organisms, but that they represent a route to survival of organisms under stress.

The next section of this chapter traces a systematic image of these and other mechanisms for survival and describes the precise ways in which they work. Survival strategies have to be played according to rules, otherwise the consequence is death and extinction.

3.3 The rules of cell and tissue behaviour under stress

In the present part of the present chapter two questions are paramount. The first one concerns the variants, rather the supports of variation. Do DNA molecules show physical and chemical change? The second question concerns the architectural homology of different DNA conformations. Such a homology is indispensable to ensure a correct expression of the genome during replication (DNA), transcription (RNA) or translation (proteins). This second question is linked to the advent of mutations, some being conserved if they embody a selective advantage.

3.3.1 DNA molecules as supports of variation

DNA molecules are generally found in the dexter form of DNA-B (Dickerson 1963). As to physico-chemical DNA changes the hypothesis was formulated by Rossignol & al. (1989) that cell DNA may be found, under certain circumstances and in part, in two other configurations, i.e. A, which is dexter like B but more compact, and Z which is sinister.

The issue of homologous sequences is complex. Several authors contributed in this field. Nuti Ronchi (1991: 4) says: “Durante et al. (1989) have demonstrated a selective DNA synthesis of specific sequences (satellite and/or repetitive sequences) during the first days of subculture in Nicotiana cultured cells: variations in the endogenous methylation pattern of these sequences could induce changes in gene expression and in the differentiation phase”.

Satellite DNA shows up as repetitive elements inside specific sequences. “All known telomeres possess similar tandem sequences” (after Watson & al., 1989, p.620). These elements appear as fragments, the sizes of which are multimeres of the basic elements. In this way, a series is produced of repetitive units like boxcars in a train “...(with) a unique restriction site in each unit, except in some where it has disappeared after punctual mutations” (Delseny & al. 1983).

According to the same authors, direct comparison of such sequences among different species, displaying homologous blocks which sometimes are sizeable, might reveal a concerted evolution of these repetitive units. This concept is supported by the observation of repeated tandem
sequences which contain spacers like the “great spacemaker” in between two transcription units of ribosome genes (18S and 25S). These spacemaker sequences, which can not be subjected to direct natural selection, are nonetheless automatically co-homogenized with selection-prone sequences (of coding and regulation) to which they are attached” (Watson & al. 1989).

Homogenisation can indeed occur through processes well-known in molecular biology, like genetic conversion, genetic amplification, or exchange between sister chromatides. However, the effect of such processes is limited. It can never warrant the simultaneous homology of all parts of the whole genome. In order to manage this, a mechanism of control and regulation is needed, acting on the whole set of nuclear components including all chromosomes. We postulate that the seat of this regulation is none other than the satellite DNA, localised in the heterochromatine associated to the centromere regions of the chromosomes.

This hypothesis rests on the observation that “the size of one unit of satellite DNA often is very close to the size of a unit of DNA wrapped up inside a nucleosome” (Delseny & al. 1983). Hence this unit may be used as a measuring rod. In view of this assumption the nucleosome will be examined more closely below.

3.3.1.1 - The nucleosome

In Eucaryotes, the DNA is folded in chromatine and wrapped up in compact complexes, the histone DNA-proteins. The latter are strongly conservative proteins containing a high proportion of amino-acids, lysine and arginine. This allows them to become firmly attached to the double DNA helix with its negative charge. The quantity of DNA associated to the packing unit is called the nucleosome, a more detailed description of which will be given later.

Experiments on nucleosomes have been conducted by applying DNase I. This is one of the enzymes of the group of nuclease which cut phosphodiester links in polynucleotid chains. The active chromatine regions are characterised by sensitivity to DNase I (Watson & al. 1989 p.758).

Application of high concentrations of DNase I showed, that nucleosomes had their sites most sensitive to cutting of both DNA strands by this nuclease on the free exterior side of the double helix, away from the histones of the “heart”. Two sensitive sites on each strand are about 10 nucleotides distant from each other (Watson & al. 1989).

This information is important. It forges a link between DNA forms observed by diffraction profiles of X-rays (Dickerson & al. 1982) and the forms found from data on nuclear DNA mass (chapter 2.3; Rossignol & al. 1989). In view of the strong conservation of the boxcar configuration within telomeres or along the centosome, like the grand spacemaker of the units of precursor 35S, they teach us the patterns of the sequences in the genome, be they ancient or newly selected.

3.3.1.2 - DNA forms, crystallography and measures of nuclear DNA mass

Table 3-2 summarises the main features of the three configurations detected by crystallographic methods (Watson & al. 1989: 272).
Table 3-2: Comparison of the architecture of B.DNA, Z.DNA and A.DNA. Sizes in Ångstrom (Å)

<table>
<thead>
<tr>
<th>Helix type</th>
<th>B to the right (dexter)</th>
<th>Z to the left (sinister)</th>
<th>A to the right (dexter)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Helicoidal rotation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Base pair number</td>
<td>≈ 10</td>
<td>≈ 12</td>
<td>≈ 11</td>
</tr>
<tr>
<td>(per helix turn path)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Helix turn path</td>
<td>33.2 Å</td>
<td>45.6 Å</td>
<td>24.6 Å</td>
</tr>
<tr>
<td>Average individual length between two bases</td>
<td>3.32 Å</td>
<td>3.80 Å</td>
<td>2.24 Å</td>
</tr>
</tbody>
</table>

The criteria for comparison will be the helix turn path (Fig. 3-3) and nuclear DNA mass. The three forms of DNA will be compared in pairs, to wit A.DNA with B.DNA, and Z.DNA with B.DNA. The numerator in the quotient is defined as the largest value between members of the pair. We find that:

- **Mean helix turn path:** Z.DNA / B-DNA = 1.37 and B.DNA / A.DNA = 1.35  
- **Nuclear DNA mass:** m(B.DNA) / m(Z.DNA) = 1.17 and m(A.DNA) / m(B.DNA) = 1.17

The same ratios exist between Z.DNA and B.DNA as between B.DNA and A.DNA, but in the compared couples the terms of the fraction are inverted according to the criterion given above.

This supports the hypothesis that

- **the b-form (see 2.2) or the specific mode \( M_b \) of chloroplast numbers corresponds to the B.DNA configuration (Dickerson & al. 1982);**
- **the a-form (see 2.2) or the major variant \( V_M \) of chloroplast numbers corresponds to the A.DNA configuration;**
- **the z-form (see 2.2) or the minor variant \( V_m \) of chloroplast numbers corresponds to the Z.DNA configuration.**

There are other elements of observed proof. Thus an indication of the nature of Z.DNA is provided by its properties. In a solution, “puric and pyrimidic residues only take the sinister configuration if strong concentrations of positive ions are present, for instance \( \text{Na}^+ \), to neutralise the negatively charged phosphate groups...” (Watson & al. 1989). Jaffé ex Côme & al. (1982) reports the results of an experiment on responses by root fragments of bean plants to radiations brought to bear through optical fibres, and found stress wavelengths in the red (\( \lambda = 660 \text{ nm} \)) to induce a positive bioelectric potential in cells. These correspond to the stress wavelengths found in our DNA mass research, as reported in section 3.2.

Indeed, the response to red through phytochromes and by phenotype change like chromosome number modification, is a change of ion flux through cell membranes. This flux engenders a positive bioelectric potential. In chapter 2 (2.2) and in 3.2 we saw that the z form and the chloroplast numbers in minor variant modes \( V_m \) were observed only during the new moon phase, marked by a near-monochromatic red light at dawn and in the evening twilight. The same causes have the same effects, so that at least a fraction of DNA formed during this period must have the Z.DNA configuration. This is an important finding, but it is also a puzzle because it leads to a paradox.
A.DNA and Z.DNA forms in fact have never been found otherwise than as traces in the nuclear DNA of Eucaryotes. On the one hand, one has to ask how such traces can influence the total DNA mass? On the other hand we have the certitude of observation of the effects of environmental factors causing changes in DNA mass. The situation reminds us of the PR-person having found a liquid that looks like beer, that tastes like beer, but that is no beer. The only solution of such apparent contradictions is to find out how things work in reality.

The first element of explanation is the certitude that the aggression inherent in in vitro culture induces a strain in the cells, manifesting itself as a variation in nuclear DNA mass. This involves two new processes, occurring at the limit between two phases in the somato-miotic cycle, the interphase and the prophase (Figs. 2-1 and 3-1). These processes are:

- synthesis, next to B.DNA, of a labile DNA strand with helix characters showing it to possess either an A.DNA or a Z.DNA configuration;
- the onset of chromatine pricking out with the newly synthesised DNA as a standard measure for specific molecular rearrangement in both daughter chromatides. Recombined DNA initiates mutations.

An initial state can change over to another state, like the specific mode $M_s$ can change over to the major variant $V_M$ or the minor one $V_m$. However, this is possible only if the rearrangement is made by way of homologous elements.

### 3.3.1.3 - Chromatine pricking out

The model pattern for synthesis in monocatenarian polymerisation is comparable to that observed in a retrovirus, i.e. a tumoral RNA virus, during viral infection. A complementary DNA molecule of one strand is built, proceeding from a labile RNA strand used as a matrix. The mechanism is worked by a reverse transcriptase enzyme (Watson & al. 1989:92).

Moreover, we already made a comment earlier as to “windows” being activated in chromosomal regions which usually remain silent. Adding this to the above, we formulate two propositions.

a) The first proposition is that such windows are represented by satellite DNA. This affects heterochromatin associated to centromeric parts of chromosomes which in normal conditions remain silent in the sense that no transcriptions are made. This is due to their lacking all enhancing or promoting sites. However, satellite DNA can be used as a fingerprint by a specific ribozyme (RNA). With the help of the reverse transcriptase enzyme, this ribozyme polymerises monocatenarian DNA. The helix turn path of these single chains corresponds to one out of both configurations other than B.DNA. There are thus three fingerprint types, the choice of one among them depending on the signal transmitted through mitochondria by way of a co-enzyme.

b) The second proposition says that synthetic, labile DNA is used as a standard measure to co-homogenize the different DNA structures. It is emphasised that B.DNA is the only configuration which is always present, with or without stress. Chromatine pricking out then yields not one but three B.DNA forms, as follows:

** B.DNA$_{(M_s)}$ is the specific mode, which under normal stress-free circumstances characterises the only form in proliferating, non senile cells. Under stress it continues to appear
predominantly during the first and last quarter of the synodic lunar cycle. The DNA-configuration then is a dexter B.DNA double helix.

** B.DNA_{(VM)} is the *early variant*, occurring under stress during the full moon conditions. The configuration neosynthesized as a standard measure is a dexter double helix of A.DNA.

** B.DNA_{(Vm)} is the *delayed variant*, occurring under stress during the new moon conditions. The configuration neosynthesized as a standard measure is a sinister double helix of Z.DNA.

Let it be emphasised once more that *the stress is not due to the moon*, but to the conditions *in vitro*. The *in vitro* response induced at DNA level by a coenzyme is determined by the quality of the light, the variation of which is timed by the lunar cycle as explained above.

To become operational, the adaptation process has to go through an initial step of substitute matching, previous to the crossing-over characterising the somato-meiotic prophase. Such a mechanism was observed in numerous lower organisms, e.g. bacteria, bacteriophages and others. It allows a hybrid construction as a transition between two DNA molecules acting as enzymes which catalyse the biochemical recombination processes specific for the site. The best known among these enzymes is Rec A protein, a product made by *Escherichia coli* genes. It specifically recognises the monocatenarian DNA and makes a homogeneous duplex in combination with a complementary sequence. The process is described in “Biologie moléculaire du gène”, a French book by Watson & al. (1989, IV-Chapt.11: 346-347). Rec A protein first fixes itself on the monocatenarian DNA, yielding a DNA-protein filament. Assisted by ATP, Rec A protein then progresses along the molecule to the homologous spot where it meets the complementary sequence. “.... one expects that higher organisms demand the presence of sophisticated enzymes like Rec A protein for the search of homologous sequences...” (transl. R.A.A.O).

According to Howard & al. (1984, ex Watson & al. 1989:347) it is important that a monocatenarian DNA tied to Rec A protein can establish specific contacts, even in a bicatenarian target DNA groove, so recognising its complementary sequence “from the outside”. Watson & al. (1989, IV-11, p. 348) say “...It is evident that such matching at the origin of a helix with three strands has to *speed up the search for homology, not each and every segment examined needing to be unrolled beforehand.*” (Transl. and italics R.A.A.O).

The singularity of precocious matching distinguishes somatic meiosis from mitosis and meiosis, as far as the adaptational aspects are concerned (see Fig. 2-1).

Association between a monocatenarian, not a bicatenarian DNA strand and one of the two daughter chromatides is not only part of a hypothesis, it is observed fact. The proof is given in chapter 2.2 and table 2-1, in which the measured DNA mass of amplified cells is shown to be 1.5 m, not 2m, m being the DNA mass of a normal-sized cell.

With the above, the first conceptual flooring has been laid for the solution of the paradox of seemingly negligible traces of variant DNA playing a cytogenetic role they are too rare to assume. Now can we establish a theoretical simulation, aided by the present knowledge of molecular biology, of the successive events leading to our experimental results?

Such a simulation has to show that the pricking-out of chromatine is both supported by secure data from molecular biology and functional in adaptation and evolution. The simulated process should yield the same results as our experiments, i.e. a loss of some 15% of total nuclear B.DNA_{(M5)} mass for B.DNA_{(Vm)} , and a surplus of some 17% for B.DNA_{(VM)}.
3.3.1.4 - Chains of regulation, rearrangement of B.DNA in homologous structures, specific site recombination

* List of operative elements in the model
  
  - The unit is represented by one nucleosome, expressed in base pairs (bp)
  
  - The basic operative element, or “mesh” or “stitch” (boe) is a bicatenarian B.DNA segment which has to be excised or duplicated in order to adjust B.DNA to Z.DNA or A.DNA standards.

  - The operative standard measure, abbreviated osm, is an initial nucleosome number which allows to calculate the reshaped standard measure, abbreviated rsm, and so establish B.DNA length once boe duplication or excision is achieved. Both osm and rsm, as links of the chain of regulation, are repetitive elements distributed all along the chain. Indeed they are nothing more than specific replication units, ru. They are expressed as nucleosome numbers or bp.

This kind of regulation presents the advantage in the real biological world of being rerun and achieved at the same time along the whole chromosome length.

3.3.2 - Description of the operative strategy

First, it is worth recalling the leading role of labile synthetic DNA in both homogenisation of the three B.DNA forms and in recombination. In fact, labile DNA is a marker, replacing at certain chromatine sites certain nucleotides from the initial B.DNA form by labile DNA nucleotides. However, the logic of the strategy behind this replacement can not be understood in every particular situation without first listing the following topical DNA features and/or structures.

3.3.2.1 - Eucaryote DNA and its molecular properties

The DNA chain is a very long nucleotide polymere, each nucleotide being connected to the next one by a phosphodiester bridge and consisting of a pentose with a nucleic acid base bound to it. The pentose is desoxyribose in DNA and ribose in RNA. The matching of bases depends on the specific “hydrogen bond” between two base pairs. The matches are G (Guanine) with C (Cytosine) and A (Adenine) with T (Thymine) in DNA. In RNA the matches are G (Guanine) with C (Cytosine) and A (Adenine) with U (Uracyl).

DNA molecules with histone proteins constitute the basic chromosome architecture. Two strands of complementary sequences appear as a dexter double helix with some 10 pairs of bases per turn path. The matching of bases between a newly starting desoxyribonucleotide strand and a pre-existing DNA strand, the matrix, controls the making up of a replicated strand. New strand growth is achieved in the 5’-3’ direction. Desoxyribonucleotide addition to the 3’DNA chain extremity is the fundamental reaction by which new DNA is built.

3.3.2.2 - DNA superwinding and packing in a cell nucleus.

In view of the enormous size of the DNA molecule and its monopoly as the only occupant of a chromosome it is obvious that DNA must be neatly folded and compacted in the cell nucleus. Different steps of this process take place mainly during the last phases in a mitotic or
meiotic cycle, from G2 to the telophase (see Fig. 2-1). They are implemented by specific nuclear proteins. Thick complexes resulting from the association between these proteins and DNA are called chromatin. The most abundant and the most conservative proteins in chromatin are histone proteins. They contain a large share of amino-acid particles, i.e. arginine and lysine, the positive charge of which helps the negatively charged DNA helix to become tightly bound to them.

When observed under the electronic microscope, chromatin shows to be no rope, but rather a string of granules or spherical particles (D = 100 Å), linked together by fine fibres. This is the elongated chromatin configuration usually called “string of pearls” It corresponds to a first-order packing of chromatin (Fig. 3-4). Its particles include two elements, which are:

![Diagram of chromatin packing]

**Figure 3-4**: First-order packing of chromatin in “string of pearls” configuration. In this first-order packing, volume occupied by DNA is reduced by 5. The string of pearls achieves that sequence of intrinsic operations of the adaptive response which takes place in the prophase (see Fig. 2-1 and text). Thus packed, the new, rear ranged reading of the coded genetic programme is “sealed off” by means of histone proteins. Packing is pursued later by successive stacking of chromatin, leading to a highly condensed form of the metaphasic chromosome.
** a “nucleosome core particle” in which DNA is wound around the exterior of a discoid or ellipsoid histone octomer built by H₂A, H₂B, H₄, H₃, H₄, H₂B, H₂A proteins.

** a “bond” between two adjacent nucleosomes.

The quantity of DNA associated to each nucleosome, including the core and the bond, amounts to circa 200 pairs of bases, 146 bp in the actual nucleosome core and 54 bp in the bond. The latter quantity is decomposed as 54 = 2 + (5 * 10) + 2 or 5 B-DNA helices + 4 bp.

Due to the superwinding singularity, only the inside of the DNA helix is strongly protected by virtue of its association with histones. The outside is more sensitive to attack by desoxyribo-nuclease (DNase I). When treated with high concentrations of the DNase I enzyme, the two DNA strands show cuts mainly on the outside, falling apart in segments which are the monomeric equivalent of 10 nucleotides. This also is the equivalent of one turn of the path (Watson & al., 1989: 747).

As a second feature, DNA accomplishes circa two turns around the outside of the histone octomer, so that it gets packed with a reduction factor of 5. Its elongated double helix chain of 146 bp equals one nucleosome DNA quantity and is approximately 500 Å long, compared to only some 100 Å for the nucleosome core diameter. One more property introduced by the process of superwinding and packing is a supplementary counterclockwise turn which induces a constraint, particularly at the level of the bond. It is known that specific enzymes, belonging to the topoisomerases, can convert one topological DNA-form into another by modifying the number of turns made by two DNA chains winding around each other. These enzymes induce minute changes. For instance, topoisomerase II, together with other proteins, like helicase in Eucaryote organisms and ATP energy, brings about negative superw windings. The outcome of two negative turns is counterbalanced by the effect of the action of topoisomerase I, which is one clockwise positive turn. A concerted enzymatic action of this kind results in the loosening of a good deal of the initial tensions. Still, some constraints remain which are due to the leftover of one counterclockwise turn. However, this tension is necessary to energise the cleavage needed in contingent operations of regulation or rearrangement of DNA. An example of such a contingency is shown by the establishment of a heteroduplex. A heteroduplex is a double helix of two B.DNA strands supplemented by a third strand of synthetic DNA.

On every turn on the inner face of the double helix there is interaction between histone proteins and the DNA phosphodiester skeleton. At sites adjacent to points of strong contact between DNA and H₁-H₄ proteins, the regular configuration of the double helix is symmetrically deformed. These apparent anomalies can be used by other, non-histone proteins to put nucleosomes “in order” or “in phase” for the second-order packing to take place by the piling up of nucleosome fibers (D ≃ 300 Å). In the next pages, the key role of this particular process in B.DNA marking, specific site recombination and chromatine pricking out will be emphasised.

3.3.2.3 - Heteroduplex setting and B.DNA marking

Different stages of this process are outlined in figures 3-5 and 3-6. Figure 3-5 represents a hybrid structure with three strands:

* On the one hand there is a bicatenarian B.DNA fragment with a loosened double helix at a specific site (ss).
Figure 3-5: Heteroduplex setting. Hybrid structure with 3 strands, i.e. two complementary B.DNA strands plus one 5'-3' neosynthetised DNA strand (here A.DNA). - Step 1: Setting of the heteroduplex by means of RecA protein, responsive to the positive or negative charges of hydrogen bonds between nucleotid bases of two complementary strands. - Step 2: The various filaments composed by synthetic nucleotides and RecA protein have pivoted around the hydrogen "liaison" axis. They have taken place opposite the complementary nucleotides of the B.DNA strand (3'-5'), playing the part of the ancient matrix B.DNA strand (5'-3'), the nucleotides of which are either neutralised, or cleft by anti-B antibody action.

* On the other hand, there is a monocatenarian neosynthesized A.DNA matrix (5'-3' strand) running in the major B.DNA double helix groove.

In stage 1, the synthetic strand is a [Rec A protein + A.DNA] filament which is sensitive to positive or negative electric charges in the hydrogen bonds between the two complementary sets of nucleotid bases.

In stage 2, the labile synthetic 5'-3' [Rec A + A.DNA] fragment, turning around the heteroduplex pivot which is a hydrogen "liaison" line, positions itself in front of the complementary (3'-5') B DNA fragment. Its nucleotides then in fact compete with those from the (5'-3') B.DNA matrix strand, which are momentarily neutralised or cleft because of an anti-B antibody enzyme action.

In stage 3 (Fig. 3-6), the neosynthesized (5'-3') fragment is used as "chaperon" basis for the complementary (3'-5') B.DNA strand, the eventual defect in base complementarity of which is corrected in this way.

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Figure 3-6: B.DNA marking in two steps (cont’d from Fig. 3-5). - Step 3: Nucleotide correction in a (3’-5’) B.DNA strand, defective for pairing with their neosynthesised opposites of the (5’-3’) A.DNA matrix. After correction and upon degradation of the labile matrix “chaperon”, two things may happen. If the B.DNA fragment was degraded before being replaced by the neosynthesised “chaperon” (shown in the figure), a new fragment is formed by using the 3’ end of B.DNA as a primer. This is caused by the DNA polymerase enzyme. If nucleotides in the B.DNA fragment were neutralised, they are reactivated. In both cases, the complementary B.DNA strand, its bases corrected as to pairing capability by the DNA “chaperon”, will become a matrix itself. – Step 4: Defective (5’-3’) A.DNA pairing capabilities are corrected by the new B.DNA matrix strand. This ends the marking process. The figure shows the specific site (ss), marked with A.DNA as a chaperon. However, this result depends upon compatible bases of B.DNA and neosynthetic A.DNA or Z.DNA, with quite different sizes. Precautionary adjustments hence occur before heteroduplex setting (e.g. see Sect. 3.3.2.4 and 3.3.2.5).

In stage 4, when the neosynthesized fragment is degraded, the “corrected” complementary (3’-5’) B.DNA fragment is used in its turn as a matrix to initiate the correction of complementary defects in the reactivated, temporarily neutralised (5’-3’) B.DNA fragment. If the (5’-3’) B.DNA fragment has been cleft, it may also initiate a new, corrected (5’-3’) B.DNA chain polymerisation, with the preceding 3’ extremity of the old chain as a primer.

Once the marking process is achieved, the flux of information is the same as the initial one. This is not always the case, as the following pages will show, when the heteroduplex is made up by a mononacatenarian Z.DNA strand as a standard measure.

In short, DNA marking may be compared to a replication with an interposed neosynthesized ADNA or Z.DNA strand used as a temporary chaperon to mark a specific site. However, there are differences. We have to admit that the two synthetic helices, Z.DNA and A.DNA,
have other sizes than the B.DNA helix as well as being different in size from each other. Furthermore, the helices of A.DNA and B.DNA turn clockwise, but the Z.DNA helix turns counterclockwise (Fig. 3-3). The process described above is therefore feasible only after previous adjustments of the participant macromolecules and their way of interacting.

3.3.2.4 - Compatibility of base matching between B.DNA and neosynthetic A.DNA or Z.DNA.

As we saw, differences between the Z, B and A forms concern length and numerical characteristics of the helix (Fig. 3-3).

Their values are larger in Z.DNA. Its total length is 45.6 Å, instead of 33.2 Å in B.DNA, and it stacks 12 bases in a turn path of the helix against 10 for B.DNA. The average interbase space (ibs) in Z.DNA equals 3.8 Å. Hence the sum of all these spaces per turn path is

$$\Sigma \text{ibs} = 0.5 \text{ibs} + 11 \text{ibs} + 0.5 \text{ibs} = 1.9 + 41.8 + 1.9 = 45.6 \text{Å}.$$  

In A.DNA, the characteristics are smaller. Its total length is of 24.6 Å. It stacks 11 bases in one turn path of the helix. Its average ibs measures 2.24 Å. The other calculations are not repeated.

The heteroduplex setting is most likely to succeed if aided by the intervention of a specific enzyme, which effects the appropriate adjustments during the polymerisation of DNA chains. To do so, this protein must be able to pinch or pleat a monocatenarian chain in the way a ribbon is pleated or pinched (see Figs 3-5 and 3-6). On account of its specific capability we call this enzyme a pinching or pleating protein enzyme. It is probably associated to a repressive protein akin to the CrO protein and to enzymes like integrase, helicase, replicase and others.

3.3.2.5 - Compatibility between B.DNA and A.DNA. Precautionary adjustment before heteroduplex setting.

Adjustments pace along with the “inventory” made by the [Rec A protein + A.DNA] complex, which may be compared with an eye “visualising from the outside” the major groove in between the strands of the B.DNA helix (cf. Fig. 3-6, Table 3-3.). Visualisation takes place during the polymerisation of B.DNA and starts from each replication unit origin (ruo).

There is a directly observed pairing compatibility limit at the rank of each 5th nucleotide. After number five, the visualisation process rests. This is a sign of heterobasic matching incompatibility. On and after the 6th nucleotide, a process with a dual purpose is required to overcome the pause. It implies both the pleating protein and the associated proteins which we just discussed. In total there is an enzymatic response.

This involves a pleating protein action at every 6th A.DNA nucleotide or every 5th B.DNA nucleotide, i.e. at every half-helix turn path. However, the second half turn path of each double B.DNA helix shows an important difference from the first half turn path. The first helix, as schematised in figure 3-7, after the data of table 3-3, presents the following case.

* In the first half-helix turn path, the logic behind the strategy is to bring the 6th A.DNA nucleotide by pleating protein action at the level of the 6th B.DNA nucleotide, after having centred the heteroduplex corresponding to the first five A.DNA bases. Indeed, the 6th B.DNA
nucleotidic is the first base of the second B.DNA half-helix turn path. Hence there are two tasks operated by the pleating protein, i.e.

- \textit{a)} \textit{the heteroduplex centering operation}, implemented by the transfer by the pleating protein of the 6th A.DNA base towards the level of the 5th B.DNA base,

- \textit{a}2) \textit{the 6th A.DNA base shift towards the second half-turn path} of the first helix, implemented through a transfer by the pleating protein after sectioning the (5'-3') A.DNA filament in between the 5th and the 6th nucleotide.

* \textit{In the second half-helix turn path}, the operation (a1) takes place like in the first half-turn path. The 11th nucleotide becomes the first one of the second A.DNA helix and is used as a primer for its polymerisation.

Why does it happen this way? The necessity of different processes rests on the principal part played by the heteroduplex, to wit the marking of chromatine at specific sites. The main task of regulation proteins hence is to preserve and to conserve the coded print. This task is apparent in figures 3-5 and 3-6, in which the A.DNA helix is shown with the following coded print:

\[
5' - A A A T A T A A A T A - 3'
\]

with 1 2 3 4 5 6 7 8 9 10 11-as bases

After enzymatic correction, the print of the A.DNA used in the heteroduplex of each helix will always remain the same, i.e.

<table>
<thead>
<tr>
<th>first half turn path</th>
<th>second half turn path</th>
</tr>
</thead>
<tbody>
<tr>
<td>5' - A A A T A T A A A T A - 3'</td>
<td></td>
</tr>
<tr>
<td>with 1 2 3 4 5 6 7 8 9 10 -as bases</td>
<td></td>
</tr>
</tbody>
</table>

This does explain why the logic of life requires and selected an asymmetrical helix. Why indeed are there (6 + 5) nucleotides and not (6 + 6) like in the Z.DNA helix or (5 + 5) like in the B.DNA helix? The answer is, that this is so by simple \textit{necessity}.

This first overview teaches us, that B.DNA is one nucleotide short on six as compared to A.DNA (standard). This is a shortage of

- \{1/6\} \* 100 = - 16.7%

of chromatine mass to be corrected.

\[\text{3.3.2.6 - Compatibility of B.DNA and Z.DNA (Table 3-4 and Fig. 3-8).}\]

Mean interbases spaces are shorter in A.DNA than in B.DNA, but larger in Z.DNA. Another major difference between A.DNA and Z.DNA is their way of setting up a heteroduplex with B.DNA. Above, we discussed the capacity of a [Rec A protein + A.DNA] filament to visualise “from outside” the paired basic sequences during B.DNA chain polymerisation and its setting up a heteroduplex structure when running along the visualised B.DNA strand like a sniffing tracker dog.

With Z.DNA as a standard measuring stick, however, the strategy is quite different because of its helix turning to the left, and not to the right as in B.DNA or A.DNA.

\textit{Hence it is impossible for Z.DNA to build a heteroduplex longer than half a turn-path with one and the same strand of B.DNA.}\n
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RecA protein progress: 1rst B.DNA helix (1rst 1/2 helix
"visualization"

(a) = heteroduplex centring operation
(a) = 6th A.DNA basis alignment on 6th B.DNA basis

Figure 3-7: Base complementarity test between B.DNA and A.DNA before marking. Preventive adjustment prepared by "visualisation from the outside" of the major groove in between the B.DNA helix strands (also see Fig. 3.8). - a2 = re-lodging the 6th A.DNA base at the site opposite the 6th B.DNA base, after section of the strand between the 5th and 6th A.DNA bases. Last diagram below: The 11th bases of A.DNA and B.DNA are placed to face one another by an a2 re-lodging. The 11th A.DNA nucleotide then is the primer for the formation of the 2nd A.DNA helix, chaperon of the 2nd B.DNA helix.
Figure 3-8: Base complementarity testing between B.DNA and Z.DNA. Adjustment before marking (also see Fig. 3-7 and Table 3-4).
Figure 3-3 schematically represents the RecA protein at work, associated to a neosynthesized (5' - 3') Z.DNA strand. Its task is to search for possibilities of contact and pairing between the bases of B.DNA and Z.DNA. Beyond the 6th nucleotide, the two nucleotide chains of Z.DNA and B.DNA clearly become incompatible because they take opposite directions. This handicap is overcome by an energy-saving strategy, i.e. pairing of a synthetic (5' - 3') Z.DNA strand with, alternatively, the complementary (3' - 5') B.DNA strand and the (5' - 3') B.DNA strand. The processes involved are, successively, the following ones.

- base pairing between a first matrix (5' - 3') Z.DNA strand and the five first bases of the complementary (3' - 5') strand of the first half-turn, first double B.DNA helix,
- base pairing between a second matrix (5' - 3') Z.DNA strand with the last five bases of the second half turn, (5' - 3') B.DNA strand. It will be explained below that this second base pairing can only take place after a Z.DNA strand twisting.
- base pairing between a third matrix (5' - 3') Z.DNA strand and the first five bases of the complementary (3' - 5') strand of the first half-turn, second double B.DNA helix,
- repetition of this heterostructuring pattern *ad infinitum* or at least *ad helicis finis*.

Energy is saved because cleaving of the 6th nucleotides in the successive Z.DNA and B.DNA helices liberates energy, which is recycled. This recycled energy partly drives the starting of neosynthesis of the next Z.DNA helices. A complement of energy is provided by ATP. The successive operations drawn in figure 3-8 and numerically expressed in table 3-4, just like the adjustments analysed earlier, need the aid of a helix pinching protein, the length of which equals the length of six B.DNA nucleotides.

Like in heterostructures with A.DNA, heterostructures with Z.DNA show hetero-base pairing incompatibility on and after the 6th nucleotide of the half-turn. However, here we can observe one B.DNA nucleotide *in excess* on six as compared to Z.DNA, i.e. a chromatine *surplus* of

\[ + \{1/6\} \times 100 = + 16.7\% \]

which has to be corrected and which is indeed suppressed during the adjustment in the form of an excision of one B.DNA nucleotide on six.

Having examined both processes involving synthetic DNA forms (A.DNA and Z.DNA) as standards in chromatine pricking out, their role as “chaperons” and more precisely as markers of specific sites demonstrates that the processes involving one or the other of these DNA molecules indeed follow specific strategies. These strategies are put under a magnifying glass in the following paragraphs.

### 3.3.3 - B.DNA marking of specific sites

#### 3.3.3.1 - Marking with Z.DNA as a chaperon.

Specific sites are located on chromatine molecules, in places easily accessible to regulation proteins. Such a place is for instance ss... in the middle of the bond between two nucleosomes where DNA double helices can be easily loosened (see Fig. 3-9). Loosening is implemented by appropriate enzymes like *helicase* and the *ss.b. protein*.

Figure 3-5 and 3-9 show the heteroduplex, placing the strands of neosynthesised, chaperon DNA and B.DNA side by side to keep the drawing legible. In reality the first one lies in the middle of the major groove of the second one, where it can not be easily seen on a drawing.
Figure 3-9: Marking process with Z.DNA as a chaperon
Heteroduplex setting (also see Figs. 3-10 and 3-11).

(2) B.DNA complementary strand linearization (Rec.A protein +
topoisomerase I + synthetase enzyme + helicase action) - S.S. loosening
Table 3-3: Base matching compatibility between B.DNA and A.DNA. \textit{ruo} = replication unit origin; \textit{ibs} = inter-base spacing.

\textbf{• First B.DNA double helix from \textit{ruo}}

* First half helix turn path; nucleotide number per helix turn path B.DNA=5+5=10; A.DNA=6+5=11

\textbf{A. RecA protein visualization during DNA polymerisation}

<table>
<thead>
<tr>
<th>Base number (= rank)</th>
<th>(\Sigma \text{ibs}^*) B.DNA</th>
<th>(\Sigma \text{ibs}^*) A.DNA</th>
<th>(\Delta = \Sigma \text{ibs} (\text{A.DNA}) - \Sigma \text{ibs} (\text{B.DNA}))</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0</td>
<td>0</td>
<td>(\Delta_1 = 0)</td>
</tr>
<tr>
<td>2</td>
<td>3.32</td>
<td>2.24</td>
<td>(\Delta_2 = -1.08)</td>
</tr>
<tr>
<td>3</td>
<td>6.64</td>
<td>4.48</td>
<td>(\Delta_3 = -2.16)</td>
</tr>
<tr>
<td>4</td>
<td>9.96</td>
<td>6.72</td>
<td>(\Delta_4 = -3.24)</td>
</tr>
<tr>
<td>5</td>
<td>13.28</td>
<td>8.96</td>
<td>(\Delta_5 = -4.32)</td>
</tr>
</tbody>
</table>

\begin{footnotesize}

********** base pairing compatibility limit: \(|\Delta| \leq 4^*\Delta_2 = 4^*1.08 = 4.32\)

\end{footnotesize}

<table>
<thead>
<tr>
<th>6</th>
<th>(16.60)</th>
<th>11.20</th>
<th>(\Delta_6 = )</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td></td>
<td>13.44</td>
<td></td>
</tr>
</tbody>
</table>

\textbf{B. Enzymatic response}

Pleasing protein action on A.DNA strand:

\textit{a} \textsubscript{1} \textbf{Heteroduplex centering operation}

alignment of the 6th A.DNA base on the 5th B.DNA base

\(\Delta\) after \(\text{a} \textsubscript{1}\) operations

\begin{center}

<table>
<thead>
<tr>
<th>Base number (= rank)</th>
<th>(\Sigma \text{ibs}^*) B.DNA</th>
<th>(\Sigma \text{ibs}^*) A.DNA</th>
<th>(\Delta = \Sigma \text{ibs} (\text{A.DNA}) - \Sigma \text{ibs} (\text{B.DNA}))</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0</td>
<td>2.08</td>
<td>(+2.08)</td>
</tr>
<tr>
<td>2</td>
<td>3.32</td>
<td>4.32</td>
<td>(+1.00)</td>
</tr>
<tr>
<td>3</td>
<td>6.64</td>
<td>6.72</td>
<td>(-0.08) \textbf{heteroduplex axis}</td>
</tr>
<tr>
<td>4</td>
<td>9.96</td>
<td>8.80</td>
<td>(-0.16)</td>
</tr>
<tr>
<td>5</td>
<td>13.28</td>
<td>11.04</td>
<td>(-2.24)</td>
</tr>
</tbody>
</table>
| 6                   | (16.60)              | 13.28 \(\Rightarrow\) = 11.20 + 2.08 | \\
|                      |                      | transfer \(\downarrow\) 13.28 + 3.32 = 16.60 |

\textit{a} \textsubscript{2} \textbf{Transfer of the 6th A.DNA nucleotide to the 6th B.DNA position after cutting of the A.DNA strand between the 5th and 6th nucleotide}

6th A.DNA base and 6th B.DNA base are aligned

<table>
<thead>
<tr>
<th>Base number (= rank)</th>
<th>(\Sigma \text{ibs}^*) B.DNA</th>
<th>(\Sigma \text{ibs}^*) A.DNA</th>
<th>(\Delta = \Sigma \text{ibs} (\text{A.DNA}) - \Sigma \text{ibs} (\text{B.DNA}))</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>16.60</td>
<td>16.60</td>
<td>(\Delta_1 = 0)</td>
</tr>
<tr>
<td>7</td>
<td>19.92</td>
<td>18.84</td>
<td>(\Delta_2 = -1.08)</td>
</tr>
<tr>
<td>8</td>
<td>23.24</td>
<td>21.08</td>
<td>(\Delta_3 = -2.16)</td>
</tr>
<tr>
<td>9</td>
<td>26.56</td>
<td>23.32</td>
<td>(\Delta_4 = -3.24)</td>
</tr>
<tr>
<td>10</td>
<td>29.88</td>
<td>25.56</td>
<td>(\Delta_5 = -4.32)</td>
</tr>
</tbody>
</table>

\begin{footnotesize}

********** base pairing compatibility limit: \(\Delta > 4^*\Delta_2\)

\end{footnotesize}

| 11                  | (33.20)              | (27.80)              | \\
|---------------------|----------------------|----------------------|----------------------------------|

\(\text{(cont'd next page)}\)

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Table 3-3 (cont’d from previous page)

<table>
<thead>
<tr>
<th>B. Enzymatic response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pleating protein action on A.DNA strand:</td>
</tr>
<tr>
<td>( a_1 )  <strong>Heteroduplex centering operation</strong></td>
</tr>
<tr>
<td>alignment of the 11th A.DNA base on the 10th B.DNA base</td>
</tr>
<tr>
<td>( \Delta ) after ( a_1 ) operations</td>
</tr>
<tr>
<td>6</td>
</tr>
<tr>
<td>7</td>
</tr>
<tr>
<td>8</td>
</tr>
<tr>
<td>9</td>
</tr>
<tr>
<td>10</td>
</tr>
<tr>
<td>11</td>
</tr>
</tbody>
</table>

A.DNA strand cutting between the 5th and 6th nucleotide

\( \Rightarrow \) transfer of 11\(^{th}\) A.DNA nucleotide to 11\(^{th}\) B.DNA site

- **Second B.DNA double helix from ruo**

1. 11\(^{th}\) A.DNA nucleotide becomes a primer for……
2. a new A.DNA helix, built to operate in the groove of the second B.DNA helix.
3. This second A.DNA helix operates like the first one (see above).
4. It functions identically, with the help of RecA protein and a pleating protein.

- **Third B.DNA double helix from ruo**

The pattern is rerun in all following double helices.

**end of table 3-3**

It may also be observed that the second part of the site, which is the second half-turn around the helix, displays prolonged polymerisation of the whole Z.DNA helix after excision of the 6\(^{th}\) nucleotide which leaves the end of the strand free and unbound. This is explained in the next passage (Fig. 3-9).

The phased progress of the marking goes as follows.

Before the start of the marking process *sensu stricto*, the strands of complementary B.DNA must be lined up so that both complementary strands face each other correctly (Fig. 18-2).

The succession of the marking phases proper in the first half-turn of the helix (Figs. 3-10 and 3-11) is similar to and was described above for A.DNA as a chaperon. The figure represents reality, with its repeated pair of Z.DNA nucleotides in a sequence which was discovered in 1985 by X-ray diffraction (Watson & al. 1989: 275). Its written forms looks like this:
Figure 3-10: Marking process with Z.DNA as a chaperon. Details of the process on the first half-helix (also see Figs. 3-9 and 3-11).
| matrix strand | \( 5' - m^5 \text{C G A T} m^5 \text{C G} - 3' \) |
| complementary strand | \( 3' - G \text{C T A G} m^5 \text{C} - 5' \) |

The ability of the substance shown in this sequence to crystallise under the Z.DNA configuration was demonstrated, provided that cytosine residues be methylated at the 5’ position as shown above.

When the marking has proceeded along the first half-turn of the helix, the information flow goes in the right direction, i.e. from 5’ to 3’.

The second half-turn in the marking process can not go in the same direction, due to a fundamental reaction in DNA polymerisation, in which an obligatory deoxyribonucleotide must be added to the 3’ end of a DNA chain. This makes that the synthetic 5’-3’ Z.DNA fragment serving as a “chaperon” must by necessity be twisted when turning around the heteroduplex axis (Fig.3-11). This necessity leads to a reversal of the information flow (Fig. 3-11 and 3-12).

When both steps have been repeated so as to have the whole turn path marked as required, the successive bases are read as follows:

\[ \text{matrix strand} \quad 5' - \text{CGATC}; \text{GATCG} - 3' \]
\[ \quad \equiv \quad - \cdot - \cdot \equiv \]
\[ \text{complementary strand} \quad 3' - \text{GCTAG}; \text{CTAGC} - 5' \]

In literature, the Ancients called a word which can be read from left to right as well as from right to left a palindrome. The short sequence above, of twice repeated bases is a genetic palindrome, left to right as well as top-down. This has not escaped Watson & al. (1989, p.302): “...Inverted sequences (....) mark the end of certain mobile genetic elements, whereas others mark places where site-specific crossing-over occurs, which allows the mobile genetic elements to function by virtue of a balancing act in inverted directions. “ (transl. and italics RAAO).

This singularity confers to Z.DNA a major role in the adaptive process, in so far as it is acting as a “chaperon”. This role is to organise resistance against many causes of nuisance, be they physical, chemical, biological or physiological.

It can only play this role because it manufactures an extra chromosome setting, the plasmid. This is a bicatenaionary DNA circle or disk. Its job is also to maintain preserved sequences, although it is located outside the nucleosome sensu stricto and its conservative histone proteins.

How is the B.DNA marking , with Z.DNA as a chaperon, followed up?

**Once the markings are in place, the follow-up operations proceed according to a strategy involving the enhancement of two enzymatic operations of correction.**
twisted (5'-3') Z.DNA strand fragment used as matrix "chaperon" for (5'-3') B.DNA complementary base

Z. DNA strand fragment

(5'-3') B.DNA strand base complementarity correction

base complementarity correction with corrected (5'-3') B.DNA strand as matrix

cleft (3'-5') B.DNA replaced temporarily by twisted (5'-3') Z.DNA untwisting as (3'-5') Z.DNA

Figure 3-11: Marking process with Z.DNA as a chaperon. Details of the marking process on the second half-helix (also see Figs. 3-9 and 3-10).
Table 3-4: Base pairing compatibility between B.DNA with a dexter helix and Z.DNA with a sinister helix. Feasibility limit inherent in specific helicoidal form, defined by 5 nucleotides on the same strand, i.e. half a B.DNA helix turn path. Heteroduplex setting strategy is in the alternating confrontation by RecA protein "visualisation" between half-turn paths of Z.DNA and B.DNA. One B.DNA half-turn path (5'-3') has six nucleotides (5+1). So has the next one, but in a (3'-5') direction. rnu = replication unit origin; ibs = inter-base spacing.

- **First B.DNA double helix**

  * complementarity with the (3'-5') B.DNA strand, the first half-helix turn path

<table>
<thead>
<tr>
<th>Base number (= rank)</th>
<th>B.DNA</th>
<th>Z.DNA</th>
<th>( \Delta = \Sigma \text{ibs} ) (Z.DNA) - ( \Sigma \text{ibs} ) (B.DNA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \uparrow 1 )</td>
<td>0</td>
<td>0</td>
<td>( \Delta_1 = 0 )</td>
</tr>
<tr>
<td>first</td>
<td>2</td>
<td>3.32</td>
<td>( \Delta_2 = + 0.48 )  compatibility: RecA</td>
</tr>
<tr>
<td>half</td>
<td>3</td>
<td>6.64</td>
<td>( \Delta_3 = + 0.96 )  (</td>
</tr>
</tbody>
</table>
| B.DNA               | 4    | 9.96 | \( \Delta_4 = + 1.44 \)  | "visual-
| helix               | 5    | 13.28| \( \Delta_5 = + 1.92 \)  | ization"

************ base pairing compatibility limit: ************

\[ (16.60) \quad (19.00) \quad \Delta_6 = + 2.40 \quad |\Delta| > 4\Delta_2 \]

** Enzymatic response**

* Pinching protein action

\( a_1 \) - **positive transfer**: alignment of 5th B.DNA base on 5th Z.DNA base
\[ (15.20 - 13.28 = 1.92) \]

\( a_2 \) - **negative transfer**: alignment of 6th B.DNA base on 5th Z.DNA base

** results:** - looping of 6th B.DNA nucleotide which is excised
- at the same time the 6th Z.DNA nucleotide is cleft

** New configuration of first heteroduplex**

<table>
<thead>
<tr>
<th>Base number (= rank)</th>
<th>B.DNA</th>
<th>Z.DNA</th>
<th>( \Delta = \Sigma \text{ibs} ) (Z.DNA) - ( \Sigma \text{ibs} ) (B.DNA) (after enzyme action)</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \uparrow 1 )</td>
<td>1.92</td>
<td>0</td>
<td>( \Delta_1 = + 1.92 )</td>
</tr>
<tr>
<td>first</td>
<td>2</td>
<td>5.24</td>
<td>( \Delta_2 = + 1.44 )</td>
</tr>
<tr>
<td>half</td>
<td>3</td>
<td>8.56</td>
<td>( \Delta_3 = + 0.96 )</td>
</tr>
<tr>
<td>B.DNA</td>
<td>4</td>
<td>11.88</td>
<td>( \Delta_4 = + 0.48 )</td>
</tr>
<tr>
<td>helix</td>
<td>5</td>
<td>15.20</td>
<td>( \Delta = 0 )</td>
</tr>
</tbody>
</table>

* The situation being symmetric, the pinching protein with a helicoidal form and a length equivalent to 6 B.DNA nucleotides executes the same a1 and a2 operations on the (5'-3') strand of B.DNA.

** cont'd next page**
Table 3-4. (cont’d from previous page)

* complementarity with the (5’-3’) B.DNA strand, the second half-helix turn path

<table>
<thead>
<tr>
<th>Base number</th>
<th>Σ ibs (before pinching)</th>
<th>Δ</th>
</tr>
</thead>
<tbody>
<tr>
<td>Z.DNA/B.DNA</td>
<td>B.DNA</td>
<td>Z.DNA</td>
</tr>
<tr>
<td>Z.DNA</td>
<td>↑1 / 6(7)</td>
<td>0</td>
</tr>
<tr>
<td>fragment, twisted</td>
<td>2 / 7(8)</td>
<td>3.32</td>
</tr>
<tr>
<td>for hetero-duplex</td>
<td>4 / 9(10)</td>
<td>6.64</td>
</tr>
<tr>
<td>setting</td>
<td>5 / 10(11)</td>
<td>9.96</td>
</tr>
<tr>
<td>half</td>
<td>↓6 / 11(12)</td>
<td>(16.60)</td>
</tr>
</tbody>
</table>

** Enzymatic response like in the first half turn path

** New configuration of second heteroduplex

<table>
<thead>
<tr>
<th>Base number</th>
<th>Σ ibs (before pinching)</th>
<th>Δ</th>
</tr>
</thead>
<tbody>
<tr>
<td>Z.DNA/B.DNA</td>
<td>B.DNA</td>
<td>Z.DNA</td>
</tr>
<tr>
<td>↑5</td>
<td>6(7)</td>
<td>1.92</td>
</tr>
<tr>
<td>second</td>
<td>4</td>
<td>7(8)</td>
</tr>
<tr>
<td>half</td>
<td>3</td>
<td>8(9)</td>
</tr>
<tr>
<td>turn path</td>
<td>2</td>
<td>9(10)</td>
</tr>
<tr>
<td>helix</td>
<td>↓1</td>
<td>10(11)</td>
</tr>
</tbody>
</table>

- Second B.DNA double helix
  * complementarity with the (3’-5’) B.DNA strand, the first half-helix turn path
    same processes as in the first half-turn path of the first B.DNA double helix
  * complementarity with the (5’-3’) B.DNA strand, the second half-helix turn path
    same processes as in the second half-turn path of the second B.DNA double helix

- Further B.DNA double helices
  same processes and architecture as in the first two are rerun.

end of table 3-4

- The first one is switched on according to the information provided by (Rec A protein + an integrase enzyme) This specifies the “visualising” of the B.DNA base arrangement during the successive heteroduplex arrangements. Acting upon this information, a systematic cleavage occurs of one nucleotide on six, equivalent to a reduction of - 16.7% in chromatine mass.

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- The second and final corrective operation is the setting of the kingbolt of the adaptation process, which is a marked bicatenarian plasmid. This is the bicatenarian minute circular chromosome, Bmcc for short. This is not included in the chromosome, but it is standing by near the level of each specific site, the ss we saw earlier.

This is how a plasmid is born. Figure 3-9 and 3-12 show that one of the two nucleotid B.DNA chains, the matrix 5'-3' strand, is prolonged at its 3'-end by a segment containing six Z.DNA nucleotides. This strand is brought outside the chromosome by the “rolling circle method” (Campbell 1977; Watson & al. 1989: 210-211, their figs 7-17 and 7-18). When rolling up, the excised segment is used as a matrix for the replication of its complementary, matched, strand, after the setting of RNA primers. Once the circle is achieved excisase enzymes, collaborating with an integrase enzyme, catalyse the cleavage or excision of the matrix strand at its 5’ end. The plasmid is completed by the action of a polynucleotid ligase. Plasmid diameter ( R ) amounts to 17.8 Å and the perimeter (2πR), which was the former length of the strand, to 56 Å.

In the chromosome, the excised B.DNA segment is replaced by polymerisation of a new chain, using the 3'-OH end of the old 5'-3’ strand as a primer.

3.3.3.2 - Marking with A.DNA as a chaperon.

This follows the rules explained earlier. Now let us remember the following sequence of 11 bases as the “footprint” of one standard A.DNA helix among others.

5’ - A A A T A T A A A T A - 3’
with 1 2 3 4 5 6 7 8 9 10 11 - as base ranking numbers

As explained (Fig. 3-8; Table 3-3), the heteroduplex setting requires some adjustments. The new synthetic A.DNA fragment then looks as follows.

5’ - A A A T A T A A A T {A} - 3’

As explained, the first base of the second helix

Base ranking now concerns 10 nucleotids, as follows.

half double helix turn path half double helix turn path
1 2 3 4 5 6 7 8 9 10

When the whole ss double B.DNA helix is completely marked, the successive bases are read as follows.

Matrix

5’ - A A A T A | T A A A T - 3’

Complementary

3’ - T T T A T | A T T T A - 5’

At the ss site, the information flux proceeds to the right-hand side, both in the second half of the helicoidal turn path and in the first half.
Like in the Z.DNA marking procedure, there is a follow-up for correction purposes.

In the case of A.DNA, it corrects a shortage of nucleotides instead of the excess found with Z.DNA as a standard. This B.DNA deficit is of one nucleotide per half turn, i.e. \[ \frac{1}{6} = -16.7\% \], as compared with A.DNA. This deficit must be corrected. Expressed in nucleotide numbers or bp to be added for every B.DNA nucleosome, the correction amounts to

\[ 200/6 = 33 + 2 = 35 \text{ nucleotides or bp} \]

For B.DNA this is 3.5 turn paths for each whole B.DNA nucleosome of 200 bp. A de novo DNA mass therefore has to be added to each daughter chromatid. The only parts which can possibly do this are the two telomeres, located at each chromosome end. This process is due to an enzyme, telomerase, found at the University of California by Carole Greider, in the laboratory of Elisabeth Blackburn, the discoverer of telomeres (ex Chambon, 1992). Telomerase has a matrix capable of stringing telomeric sequences to chromosome ends. Our assumption as to the significance of telomeres is supported by a report from another source altogether, i.e. the science writer Chambon (1992, p.28, transl. RAAO): “...in normal cells, this enzyme seems to remain inactive......However, it was recently discovered that it is reactivated in cancer cells. ...... For that matter, cancer cells are used since many years in cell biology because of their unlimited capacity for proliferation.” Now this indeed also is a characteristic of the cell masses in vitro, the study of which was the point of departure of the present chapter.

In our experiments (Chapt. 2), this prolific tissue originated in vitro from a first cell showing the specific mode B.DNA\(_{(M_S)}\), which continued to proliferate in the form of B.DNA\(_{(VM)}\) or B.DNA\(_{(VM)}\). This was due both to the stress conditions in vitro, i.e. in the immediate environment of the cultured cell mass, and to the electromagnetic signals arriving from the planetary environment. The latter determine the kind of correction the genome needs.

Indeed we just saw that the adaptive response was translated at the cellular level into an increased level of potential energy. This energy can be used, if need be, for occasional DNA-repairs. In this way, the limits of senescence due to accumulation of genetic errors are shifted further into the future. The adaptive response also results in the marking of DNA at specific sites, causing an increased cleavage of chromosomes and genes. These then can act as a “promotor” region, i.e. a regulatory region acting as a privileged site for transitory RNA-polymerase binding.

These responses constitute the underlying logic of the process of founder cells in proliferating environments giving rise to a daughter cell, possessing for instance the characteristics of the early variant B.DNA\(_{(VM)}\), and being capable of initiating a new lineage. Now due to its B.DNA\(_{(VM)}\) nature, this daughter cell can also function as a founder cell and initiate a cell lineage with monospecific B.DNA\(_{(M_S)}\) or a lineage of a delayed variant with B.DNA\(_{(VM)}\).

3.3.4 - From cell to tissue

3.3.4.1 - Founder cell with B.DNA\(_{(VM)}\) and daughter cell lineage with B.DNA\(_{(M_S)}\)

In this case, the specific form is found back by cleavage of both chromosome ends, where there is an exceedent of telomere DNA. The chromosomes of the cells belonging to the daughter lineage then conserve the specific markings of the founder cell with its B.DNA\(_{(VM)}\) The cleavage of the terminal parts of the telomeres liberates increased amounts of free energy.

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This boosts the cell metabolism and so shortens the mitotic timespan. Mitosis hence is also boosted.

On the longer term, the result of so boosting mitotic activity is the generation of cell groups, tissues, organs and organisms which in their turn display a boosted metabolism (cf. Crabbé 1987 on rhythmic meristems - section 4.2.1 of the present book). Moreover, the marked site may act as a promotor site for a neighbouring gene strongly responding to some selection pressure, and so be able to amplify the effect of that gene.

3.3.4.2 - Founder cell with B.DNA\textsubscript{(VM)} and daughter cell lineage with B.DNA\textsubscript{VM}

This case is more complicated and requires three operations to arrive at the B.DNA\textsubscript{VM} variant.

- **First, like in the preceding case, excess telomeric DNA is cleft. Free energy becomes abundant.**
- **Second, one on six base pairs along the chromosomes is cleft.**
- **Third, a heteroduplex is set, the markings at the specific sites (ss) are changed, and at each of these sites a plasmid is installed.**

The adaptive response in this case is more flexible in comparison with the preceding one. It has the advantage of recombination because of a different lecture of the codon sequence. Moreover, the increase of free energy provides this delayed form of variant with a substantial supply of regulator protein-enzymes at the genome level. At the level of the cell it provides a boosted metabolism which can lead to a strong growth of the whole organism and/or an increase in weight, which is the equivalent of abundant reserves. This may be combined with or replaced by a stronger response of resistance to selection pressure, owing to the presence of the plasmid. Together with the following sequences, this one offers the ideal tool to respond to stress conditions.

3.3.4.3 - Founder cell with B.DNA\textsubscript{(VM)} and daughter cell lineage with B.DNA\textsubscript{(Ms)}

This advantage is explained by two series of experimental facts from molecular biology.

* **The first series** concerns the stable insertion in a yeast chromosome of circular DNA made linear, i.e. a plasmid of a sectioned Paramecium. This experiment was carried out at the University of California in San Francisco by Elisabeth Blackburn & Jack Szostak (ex Chambon 1992). They showed that such linearized plasmids degrade and disappear quickly once they are inserted in the nucleus of the yeast. Contrariwise, if the extremities of the paramecial chromosome are cut off and spliced to the linearized plasmid, the plasmid remains perfectly stable in the cell nucleus of the yeast.

* **The second series** was established after the discovery of jumping genes by Barbara McClintock (1948, 1978) in the ‘forties and ‘fifties (also see Vedel & Delseny 1987), i.e. inserted elements like plasmids and episomes. The key to these studies are the transposons or transposable genetic elements (see Suzuki & al. 1989: 517-542). The part they play in the transformation of genetic programs by rearrangement of chromosomes was most thoroughly studied in Procaryotes (bacteria and viruses). However, some Eucaryotes were studied too (e.g. corn, black corn, Drosophila). The experiments were designed especially to understand
the mechanism conveying to these transposons the \textit{capacity to transfer genes which specify certain forms of resistance}, for instance to antibiotics (also cf. Garrett 1994). The transfer indeed carries genes towards unrelated sites on the same or another chromosome.

The work by McClintock was followed by numerous experimental studies, a few examples of which we mention. Watson \& al. (1984) cited the contributions of S.N. Cohen in 1977 to the Cold Spring Harbor research on DNA insertion elements, and the publication by F. Heffron, McCarthy, M. Ohtsubo and E. Ohtsubo in 1979. Other data and insights were contributed by Cohen \& al. (1980), and Fedorov \& al. (1984).

In the context of the present book, these writings show that in exceptional and particular circumstances, under selection pressure or stress, cells use a recombination process leading to the rearrangement of nucleotide sequences. \textit{DNA-segments so can be moved and recombined at target sites, where different genes or sets of genes are then expressed} (Watson \& al. 1984: 357). Two types of transposons exist. The first one is a \textit{simple transposon}, called \textit{insertion sequence}, the second one is a \textit{complex transposon} which also includes one or more genes. Complex transposons often possess insertion sequences at their ends, which suggests (Watson \& al. 1984) that insertion sequences “\textit{jump}” on both sides of a gene, so that the whole aggregate can travel outside the outer limits of the insertion sequences.

These experimental facts are relevant for the particular founder cell and lineage discussed here.

The DNA of the founder cell is the delayed variant, B.DNA\(_{\text{VM}}\), displaying the following characteristics. In comparison with B.DNA\(_{\text{MS}}\), this DNA shows a mass deficiency of -16.7\% being the equivalent of one lacking nucleotid pair on six or 35 nucleotid pairs (= 35 \text{bp}) per nucleosome. Strands of this DNA present marked sites (ss) in the proportion of one site per nucleosome. At every such site, an extrachromosome plasmid is standing by, i.e. a small, circular chromosome of 16 \text{bp}, as described above.

The DNA transformation towards the specific mode Ms in the daughter cell lineage requires a preliminary correction. The stages of the process implementing these corrections are shown in figures 3-13 and 3-14. They illustrate how the complement of 35 nucleotid pairs (c = 17 + 18) comes to be added to the DNA at the level of every marked site ss, and so serves as an insertion sequence at the target site. They show how the particular properties of the plasmid allow the addition of stabilised DNA sequences to the chromosone. In certain circumstances, this allows these same parts to be \textit{transformed into transfer elements} which can place themselves \textit{on both sides of genes which are “circumstantially” useful}, i.e. which are capable to exert a useful action upon the \textit{stress effects performing as inducers}.

\textbf{a) First stage (ST I). Stable insertion of plasmid sequences in the chromosome}

A DNA copy with the same nucleotide sequence and paired base number as the plasmid (\(\Sigma \text{bp} = 16\)) is coined by the method of the rolling circle. This plasmid copy is cleft by a restriction enzyme located at either end of the symmetry axis (S.A.) of the plasmid (Fig. 3-12).
Figure 3-12: Plasmid setting up following Z-DNA marks. b.m.c.c. bicatenarian minima circular chromosome. S.A. = symmetry axis. The drawing is self-explanatory.
Figure 3-13: Stable insertion of a plasmid cop) sequence in chromosomal DNA in a daughter cell lineage B.DNA_{cop} from a founder cell B.DNA_{nuc}. (A) "lollipop structure" integrated in chromosomal DNA at the level of a ss target site. To each end of the cleft and stretched plasmid (2*8 = 16 nucleotides per strand) telomeric nucleotides are added, in total |17+18| = 35 bp. The complex (plasmid + telomeric sequences) is inserted in the chromosomal DNA in a stable way by its ends, so forming a "lollipop structure". The insertion takes place at a specifically coded site of the V_{nuc} variant, sectioned midway by a restriction enzyme (also cf. DNA marking). (B) insertion complete and stretched out.

Figures 3-12 and 3-13 show the nucleotid sequence of the stretched DNA fragment resulting from these processes and which presents a double symmetry once it is stretched. This twofold rotational symmetry has a sequence of n = (8 bp)^2, the plasmid sequence, in relation to a pivot in between the bases A and T or T and A in the first case and in between Cand G or G and C in the second case.

First case:

\[3' - C TAG CG CT - 5' \]
\[5' - G AT CG CG A - 3' \]

Second case:

\[3' - A GC G CT AG \]
\[5' - T CG C G AT C - 3' \]
Below, arguments will be set out for natural selection rather favouring the first case in the present situation of “B.DNA(Vm) founder cells ⇒ B.DNA(Ms) daughter cells” and the second case in the next situation “B.DNA(Vm) founder cells ⇒ B.DNA(VM) daughter cells”.

For their stable integration in the chromosome DNA at the target site, both strands of the plasmid have to present at each end a certain number of repeated telomeric sequences of the chromosome. The repeated base pairs plus those of the plasmid should total per nucleosome a number of base pairs of 35, indeed:

Telomeric DNA is known to be built by stringing together repeated, identical sequences of 6 nucleotids (6 bp). As a desk experiment, let us assume our telomeric sequence to be the same as the one observed in the above-mentioned paramecium, *Tetrahymena thermophyla*, i.e.

\[ \begin{align*}
\text{T} & \text{T}\text{G}\text{G}\text{G}\text{G} \\
\text{A} & \text{A}\text{C}\text{C}\text{C}\text{C}\text{C}
\end{align*} \]

Now we may outline the second operation, i.e. the splicing by the enzyme ligase, to each end of the sectioned plasmid, of a more or less equivalent number of telomeric nucleotides. This would amount to $6 + 3 = 9$ nucleotides at one end and $6 + 4 = 10$ nucleotides at the other. All is inserted at the target site ss, sectioned midway by a restriction enzyme (Fig. 3-13).

The similarity is striking between the insertion architecture of figure 3-13 and the architecture observed by electron microscopy and described according to its form as a “lollipop-shaped structure” (Suzuki & al. 1989: 522). Indeed a lollipop is a round structure on top of a stick, as mirrored by a DNA loop on top of two inverted repeat DNA sequences (IR sequences). Subsequent studies have demonstrated that “the genes for drug resistance and other genetic abilities ..... are located between the IR sequences” on the “lollipop head”.

Once the plasmid DNA with its two telomeric tails has entered into the chromosome DNA, in between both inverted sequences of 5 nucleotides at the target site, it becomes integrated there in a stable manner (cf. Blackburn, cited above). But that is not all. Together with itself, the plasmid DNA also has introduced a repeat sequence characteristic of the target site, reading **GATC**, just like the *promotor* of the transposase of *Tn10*, a transposon of the λ bacteriophage. Indeed, in the DNA of *Tn10*, “...the site on which the transposase acts contain **GATC**” (Watson & al. 1989:364-367).

The results of the first corrective stage (Fig. 3-14-(a), “nuclear enzyme action”) show that the integrated DNA, consisting of the plasmid and two telomeric sequences, can be divided by a quincunx cutting in two all-but equal sub-plasmids by the action of two nucleases, being exonuclease and endonuclease. The two sub-plasmids are “insertion sequences”.
Figure 3-14: Preliminary enzyme action leads to first copies of complementary insertion elements both “above” (left side) and “below” (right side) a gene. Cells studied showed specific mode Ms, originated from a founder cell Vm, late variant “marked” by a sinister Z.DNA helix, their nucleus containing extrachromosomal plasmids. (A) Rolling circle process integrates a 6 bp fragment of the plasmid “above” the (5’-3’) DNA strand, to the left of the original target site (5 bp). (B) Replication of a (5’-3’) DNA strand segment formerly used as rolling circle to insert the plasmid fragment of 6 nucleotides in chromosomal DNA “above” the original target site of 5 nucleotides (CGATC); polymerisation -. Replication of lacking part of (3’-5’) strand, after temporary cleavage of (3’-5’) strand and unrolling of the helix, with the (5’-3’) fragment as a matrix and the 3’ end of the late (3’-5’) strand as a primer; polymerisation -. Because each possesses the promotor of transposase and both can place themselves at the sides of the transposase gene or transfer enzyme, we will designate these two sub-plasmids as transfer elements or insertion elements.

The complex formed by transfer elements+transposase will be designated as primary transposable element.

The latter can move either along a chromosome or from one chromosome to another. In this way it can take place directly next to a gene which offers a selective advantage against stress. Both transfer elements then jump at both sides of the gene or set of genes.

The complex formed by primary transposable element+resistance gene(s) will be designated as the transposon. Its formative modalities and process are described below in detail.

b) Second stage (ST.II). Nuclease enzyme action and setting of insertion or transfer elements (Fig. 3-14 (a) and (b))

In one operation, a DNA fragment of the plasmid on both sides of the axis of symmetry (S.A.), three to the left and three to the right, is cut in a quincunx by an endonuclease. The length of the fragment corresponds to 6 nucleotides. In a second operation at the same moment, two nucleases do the same job at both ends. This occurs to the left of the target site over a DNA length of 6 nucleotides (or 6 bp) and to the right of the target site over the same DNA length. (6 bp).
Figure 3-15: End of preliminary enzyme action. Two complementary insertion elements formed as double-stranded, motile, circular plasmids. (A) Double cleavage and DNA extraction from the double-stranded heteroduplex fragment. Its (5'-3) filament then is spliced to the single-stranded sub-plasmid (+ telomeric elements) and made circular. Late strand to the right follows the process of A, but delayed. (B) Polymerisation of the complementary (3'-5) fragment with the (5'-3) single-stranded ss. Plasmid serves as a matrix and (3'-5) heteroduplex fragment as a primer.
Figure 3-16: First "jumping" element facilitating the transfer of a R-factor gene - also see next figures. (A) Relocating of the two insertion elements in the chromosome, after producing a first copy. Duplicated target sites show at each end a double repeat of of the transposase promotor GATC. So do the inverted repeat (I.R.). (B) Setting of the primary transposable element. First copying from the plasmid integrated in the chromosome provided two plasmids becoming circular, travelling to the transposase gene. They flank the gene, the ends of which have been cut in a quincunx by a nuclease. A "primary transposable element" results, which becomes circular and can travel along a chromosome, or from one chromosome to another, finally to a R-gene, aided by sub-plasmids of a second generation of copies (see Fig. 3-17).
These operations set free two DNA fragments, each one having two “sticky ends” in a quincunx, as shown in figure 3-14 (a). Both fragments can function as sub-plasmids, if their monocatenarian ends can be “glued” together as complementary strands. This operation requires the participation of the proteinaceous enzyme RecA plus the integrase enzyme. After this enzyme has bound itself, as a first step, to the plasmid’s monocatenarian axial strand of six nucleotides (5’ - CGATCG - 3’), it moves to the left till it reaches the left end of the target site ss, CGATC. The terminal nucleotide is recognizes its counterpart C and binds itself to it. In the heteroduplex which is formed with the (3’-5’) sequence of the “sticky end” to the left, the sequence 5’ - CGATCG - 3’ becomes the matrix for the correction of the bases at the “sticky end” to the left. So the two “sticky ends” become complementary. An analogous operation has the same result at both “sticky ends” of the fragment to the right.

The required recognition of its own base or of its complementary base by virtue of the “tracking head” of the protein RecA dictates the pinpointing by the enzyme of the location of the cleavage causing the linearization of the plasmid, prior to its splicing into the chromosome.

The process has a double result.

In the first place, the two inverted sub-plasmids are set free as transfer elements, also named insertion sequences, able to move to a gene that codes for transposase (Fig. 3-15-(a),(b)).

In the second place, the old marks on the chromosome at the former target site level ss are overwritten and corrected. The former target site was built by two sequences of 5 nucleotides, the one to the right being the inverted complement of the one to the left, as follows:

\[
\begin{align*}
\text{sa} & \\
3' & \text{G C T A G} \mid \text{C T A G C - 5'} \\\n5' & \text{C G A T C} \mid \text{G A T C G - 3'}
\end{align*}
\]

Meanwhile, the two sequences of the new, recombinant target site at both ends of the above sequences form a palindrome of 2 * 11 nucleotides (cf. Fig.3-15-(a)). The axes of the palindrome are S.A. and the line separating the bases of each main (5’-3’) strand from its complementary (3’-5’) strand.

\[
\begin{align*}
\text{sa} & \\
\end{align*}
\]

The restriction enzyme recognizes the same bases C and G prior to cutting the DNA strands at the level of the symmetry axis sa.

In the third place, the two DNA strand fragments used for the sub-plasmids are replicated again at the 3’ end of the intact strand, used as a primer. The complementary strand is used as matrix (Fig. 3-14-(a), “polym.1 and polym. 2”). In this way, the plasmid stays inserted in the chromosome and can provide copies of insertion elements on demand.

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c) Third stage (ST.III; Figs. 3-16 and 3-17). Primary transposable element and transposon setting (trp.x(4))

The two insertion elements, each with monocatenarian ends of 11 bp, move in on the two sides of the transposase gene (Fig. 3-16-(b)). The ends of the latter are also cut in a quincunx. Their “sticky ends” are 6 bp long. This is how the primary transposable element is obtained. It contains the gene which codes for the transposase enzyme, stuck in between two inverted repeat sequences (I.R.) and being tailed at both ends by identical duplicated sequences of 6 target site nucleotides each. This sequence is nothing else but the sequence of Z.DNA, which is the matrix structure of the heteroduplex. Fitted out in this way, the primary transposable element can now take place near a “R-factor gene”, the activation of which triggers the resistance to the particular stress of the moment.

This operation takes place in two steps (Fig.3-18-(A),(B)) and requires a second pair of insertion elements, provided as “second printings” on demand.

As a result of these two steps, a transposon has been positioned in the chromosome. The architecture of this transposon is characterised by its ends both being formed by the same, duplicated but imperfectly inverted sequence of 11 bp. This is like the A_c element of corn, of which Suzuki & al. (1989: 540) state: "An A_c element (........) has 11 b.p. imperfect inverted terminal repeats." The two inverted repeat sequences total 35 bp in two R.I. The R.I. sequences contain 17 bp at the left of the transposase gene and 18 bp at the right.

The next case shows still another, additional advantage because of the positioning of a promotor offering a site for the initiation and the end of transcription of the whole transposon.

3.3.4.4 - Founder cell with B.DNA(Vm), daughter cell lineage with B.DNA(Vm)

The steps of the process ending with the setting of a transposon in the DNA of a chromosome are essentially the same in the present case and the former one. However, there is a shade of difference which does bring about a considerable selective advantage. Indeed, the daughter cell in the present case carries a second promotor or TATA box. This provides the lecture framework from the beginning to the end of the whole transcription of the transposon to the enzyme RNA-polymerase.

In what respect do the daughter cells B.DNA(Vm) differ from the founder cell B.DNA(Vm)? They differ from the latter by a nuclear DNA mass increased by 33.4%, being 2*16.7%. This amounts to the equivalent of two nucleotid pairs on six, totalling 2*35 excessive nucleotid pairs or bp per nucleosome as compared with B.DNA(Vm). The first operation hence is the work of the “lollipop structure”, as in the previous case. Because the number of nucleotides or base pairs of the plasmid is constant, it is the number of telomeric bp which is to be augmented as shown below.

```
          sa
         /
       27       8       8       27
       \         \         \         \   ( = 35 \ 35)
      telomere   plasmid  telomere
             |                |
             sa             sa
```

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Figure 3-17: From primary transposable element to transposon \( x_{\text{m-c1}} \): (A) Second copying of insertion elements (sub-plasmids) allows the copies to "jump" to both sides of the R-gene, flanking it and linking the whole to the primary transposable element. (B) Transposon \( \text{Tn} x_{\text{m-c1}} \), built by the Inverted Repeat sequences (I.R., occupying \( (18 + 17 \) + ( 17 + 17 ) = 72 bp) plus the transposase and R-factor genes.
There is a loss of nucleotides, taken away from the DNA of the telomeres and used for framing and stabilising the insertion of the plasmid DNA in the chromosome. This loss is compensated by polymerisation of telomeric DNA fragments from both ends of the chromosome, having nucleotid pair or bp numbers equivalent to $2 \times 35 = 70$ bp. In other words, the increase of the potential energy is twice that of the precedent situation.

The double sequence of 5 bp (or “specific site” ss) which functions as the original target site can be read as follows:

\[
\begin{align*}
3' & \text{- TTTAT} \quad | \quad \text{ATTTA} - 5' \\
5' & \text{- AAATA} \quad | \quad \text{TAAAT} - 3'
\end{align*}
\]

The restriction enzyme therefore must recognise adenine (A) or its complement thymine (T). Section of the plasmid occurs in between T-A or A-T and the symmetry axis sa passes between the bases cytosine (C) and guanine (G) or G and C. This indeed causes the central plasmid sequences to obtain the configuration of the second case above, which is the following.

\[
\begin{align*}
3' & \text{- A G C G C T A G} \quad | \quad \text{C T A G C G C T} - 5' \\
5' & \text{- T C G C G A T C} \quad | \quad \text{G A T C G G A} - 3'
\end{align*}
\]

In the “lollipop structure”, 4.5 repeat telomeric sequences are added at each end of the plasmid sequences. Each of these sequences has 6 bp, their sum being $4.5 \times 6 = 27$ bp. In this way, the plasmid is stabilised in the chromosome by $2 \times (27 + 8) = 2 \times 35$ telomeric nucleotids (Fig. 3-18-(A)).

As in the preceding situation, the sectioning in a quincunx of DNA segments by nuclease enzymes is followed by duplication of the central plasmid sequence by protein RecA. This leads to the establishment of insertion elements or transfer elements (Fig. 20-2). The latter are shaped as sub-plasmids and frame both the transposase gene and the factor gene providing a selective advantage under the impact of a stress factor. This still is the same sequence as in the previous case.

Figure 3-18-(B) schematises the characteristics of the insertion elements or transfer elements framing the transposase and resistance factor genes so as to form the transposon t.r.p.$X_{(A,2)}$. Both insertion elements consist of inverted repeat sequences (I.R), each 35 pb long and prolonged at their ends by inverted, duplicated hybrid target sites of 11 pb, as follows.

\[
\begin{align*}
3' & \text{- T A G C T A T T T A T} - 5' \\
5' & \text{- A T C G A T A A A T A} - 3'
\end{align*}
\]

\[
\begin{align*}
3' & \text{- A T T T A T A G C T A} - 5' \\
5' & \text{- T A A A T A T C G A T} - 3'
\end{align*}
\]

In the resulting transposon Tn.$X_{(A,2)}$ the start of transcription and the framework of lecture by the RNA-polymerase enzyme have been summarised in figure 3-19.
Figure 3-18: Preliminary enzyme action, like in Figs. 3-14 and 3-15. However, cells studied here also originated from a late variant founder cell V_m, but are characteristic early variant cells V_m, showing the markings of A.DNA (not Z.DNA like in Fig. 3-14 and 3-15).
In general, transposons can be integrated in any region whatsoever of a new genome. Beyond the part they play in the setting up of defences against lethal stress effects, they can be significant tools in the crossing-over of daughter chromatids. In any case, they are important players in the game of gene mixing and stirring.

3.4 - The struggle of life at the cellular level

The whole complex assembly displayed above has an amazingly high performance. The amplification or mass production of certain sequences by plasmids is a highly efficient mechanism to “design” or “inform” the reaction of a cell to selection pressure. Moreover, the orderly programmed passage of the specific DNA form to and from whichever of both variants as required in view of the prevailing environmental conditions is a smooth and flexible process to face stress of all kinds.

The transmission of the acquired adaptive cell properties to higher organisation levels and whole organisms, and their expression higher up in the system hierarchy will be considered in later chapters. The present chapter leaves us with two general questions concerning the adaptation of cells in view of the phenomena explained above.

The first question addresses the language of the genome. What happens when one nucleotide on six is suppressed in the genetic sequences, including those writing the coded language of the genes that rule resistance factors, in other words the language of transposons? Our analysis has shown that a prerequisite for the existence of a transposon is the presence in the cell nucleus of an extrachromosomal structure, the plasmid. A prerequisite for the presence of a plasmid in its turn is the existence of the delayed DNA variant B.DNA_{(V_m)}, with Z.DNA as a chaperon. The delayed variant can characterise two types of cells.

Considering daughter cells, their founder cell can represent the specific mode B.DNA_{(M_S)} or the early variant B.DNA_{(V_M)} with A.DNA as a chaperon. In both cases, the plasmid is extra-chromosomal. During transcription and translation it only participates in polymerisation of amino-acids and proteins, if the splicing of its sequences to chromosomal, i.e. genetic sequences, provides a selective advantage. This advantage is no hereditary character which can be transmitted to descendents by hybridisation.

Considering the founder cell, its daughter cells may be either delayed variants B.DNA_{(V_m)} like itself, or belong to the specific mode B.DNA_{(M_S)}, or finally show the early variant B.DNA_{(V_M)} with an A.DNA chaperon. The two latter modes allow the integration of the plasmid in chromosomal DNA and the positioning of transposons. The adaptive character with survival value, selected at the DNA level to be amplified by the transposon is hereditary. It is transmitted by hybridisation.

The second question addresses the number of variants. Why did two and only two variant DNA forms during the evolutionary process escort the specific DNA mode? Might not a broader variation be expected when observing the many diverse forms, colours, sounds and smells of the organisms around us? On the one hand, this contrast suggests that two variants may not be enough. On the other hand, it raises the question of redundancy. Is not one variant enough?
Figure 3-19: Transposon Tn \( x_{\alpha, \beta} \). Characteristics and modes of transcription. All mRNA messenger molecules synthesised by RNA polymerase II. Synthesis is optimised by two control sequences outside the transcribed zone. One is a promoter like the TATA box, which seems indispensable to position the start of transcription. The other is one of the sequences enhancing the transcription rate of early or late mRNA. These operate "upstream" or "downstream", often far away from the promoter they activate (cf. Suzuki & al., 1989). In the case of our transposon Tn \( x_{\alpha, \beta} \), the TATA box (median part) sits at 38 to 40 bp upstream from the codon starting transcription, UAG. The action of RNA polymerase into a strong binding by a transcription factor ATCGAT upstream, read in the correct direction by RNA polymerase.
3.4.1 - Purity of language?

Taking into account that replication and development of organisms are impossible unless coherently informed, the mechanisms described in the present chapter include the risk of gibberish being introduced in such well-organised speech. In later chapters we will examine this at other levels of organisation. The next paragraphs concern semantic disorder at the genetic level.

Such disorder, wrought among the well-arrayed codons by the systematic elimination of one nucleotide pair on six, influences the whole organism. Indeed, the steering system is built to do exactly that. The blurring of the genetic language can not but exceed the capacity of an organism to conserve its general balance. This can only be avoided by the action of a correction mechanism.

In all cases examined with Z.DNA as a chaperon in the cell, the mutations generated by the adaptation mechanism are of the FCO-type, a proflavin-induced mutation. The proflavin molecule is thought to act by adding or deleting single nucleotide pairs in DNA (Crick 1966 ex Suzuki & al. 1989: 336-337). The FCO-mutation can be counteracted by a suppressor mutation, inducing “reversions”. Proflavine-induced mutations are also called sign mutations. If the original mutation was a deletion, designed as “minus”, like in the deleted variant B.DNA$_{(V_m)}$, it can only be nullified by a “plus”. Let us present a concrete example, which is the following sentence built up exclusively by three-letter words symbolising codons.

**YOU DID NOT SEE HIS HUT FOR YOU HAD ONE EYE**

Now one character on six is deleted from this eleven-word sentence. The result is this:

**YOU DIN OTS EHI SHU FOR YOH AD NEY E.**

The sentence now contains only nine words and one letter and lacks all meaning.

Now let us suppose that at the end of the prophase (Fig. 2-1), before the DNA is compactly wrapped up during the metaphase of the mitotic cycle, a process would allow to insert one letter on six into the new text. This would be a suppressor mutation. In the present case we assume it to work by doubling the 5th, the 10th, the 15th letter, and so on.

In our model, the adaptation mechanism is portrayed mainly according to the means of survival of procaryotes and retroviruses. It is probable that the process examined in the above paragraphs is similar to the one in the cells of the immune system of vertebrates, as demonstrated by molecular biologists. In antibody genes, Leder (1987, transl. RAAO) found that “...in certain conditions, a RNA sequence which is the complement of one strand of the double DNA helix can hybridise itself with this strand in a tighter way than the complementary DNA strand itself.” Indeed the end of the RNA strand in the polymerisation phase is linked to a RecA protein, carrying in its memory the number 6 for Z.DNA. Hence the RNA-polymerase enzyme duplicates the 5th, 10th, 15th ...nucleotides while at the same time cutting the DNA strand in between the 5th and 6th, the 10th and 11th .......... nucleotides. The DNA repair mechanism then allots a complementary nucleotide to the vacant spots on the DNA strand. The reverse transcriptase enzyme acts to transform the ribonucleotide into a deoxyribonucleotide.

Now if the same fast-change trick is applied to our above sentence, the final recombined and rearranged version is the following revertant.

**YOU DII NOT SEE HIS HUU FOR YOO HAD ONN EYE**
Except for some spelling errors, the meaning has been restituted to the sentence. In terms of organic semantics, one may say that except for some errors of detail, the *phenotype of the revertant restitutes to the organism a phenotype quite similar to the natural phenotype*. In the next chapter we will come back to the distinction between similarity and identity. Let us say here that the errors which denote a changed array of amino-acids in the proteine coded by the gene or genes, can indeed generate a different quality of the organism and a different response to stress.

These differences are not so much numerical differences as architectural differences at the molecular level.

3.4.2 - Two variants only?

There are two and only two DNA variants except for the specific mode. Is this both enough and not too much to respond to the countless environmental challenges? In short, the answer to this question is that these variants are complementary and that this complementarity is both necessary and sufficient.

It is necessary, because the delayed variant \( B.DNA_{(V_m)} \) is a “double agent”. On the one hand, it is the cardplayer having to shuffle the pack, that is to say to thoroughly change the order of the cards. On the other hand, as discussed above, it has to be able to unshuffle the pack again, i.e. to nullify more or less imperfectly the process of change. In computer jargon these instructions would be “undo delete” or “undo”, but the *organic* response is less precise.

Shuffling cards or nucleotides is surrendering to randomness or coincidence (cf. Javary 1994). The rules of probability in this context of increased entropy determine the chance that some new array of molecules comes out, which favours survival in the given circumstances. It counteracts the effects of prevailing stress. This may be due to plasmid action, an individual defence tool that can not be transmitted by hereditary means. It also may be due to transposon action, which is a very effective defence mechanism because it is both selective and transmissible along hereditary pathways to descendants. However, the latter opportunity requires both a sharp definition of the lecture framework by the TATA box, and the input of supplementary energy. Both can only be provided by the participation of the second, early DNA variant.

This participation is sufficient, because the early variant, \( B.DNA_{(V_m)} \), also is a “double agent”. On the one hand, it maximises the response to stress by selection and amplification of the gene or genes with optimal resistance potential. On the other hand, it efficiently manages the demand for free energy.

The co-operation between the two variants allows to create numerous possibilities of stress resistance, to undo the chaotification with a few errors if none of the possibilities is likely to succeed, and to select and boost the ones which are most promising. The whole operation depends on the ongoing purification of the genetic language illustrated above with the three-letter words and eleven-word sentence. It is driven by plasmids and transposons acting with a surplus of energy from telomeres. Its secret is molecular architecture.
3.4.3 - The struggle for the best response

The present chapter offered a model or portrait of the struggle of cells and genotypes, generating the best response to the challenges of selection pressures and stress. One proof of its contents is the inner consistency of the portrait, explaining all facts. Another element of proof is over ten years of experimental support found in our own laboratory at Orsay. Still other components of scientific proof are facts borrowed from literature in a wide array of research fields, including for instance cytogenetics, somaclonal variation and molecular biology.

The meaning of the title of the present book is partly unveiled by this body of knowledge. Indeed, all facts uncovered falsify the “struggle for life” which implicitly goes against other things. Quite to the contrary, cell life struggles to make use of all that suits survival rather than warring against other cells or organisms which might endanger survival. The struggle of life is the effort to find optimal pathways to survival, rather than to open new roads by contending with others. We have used the competitive term “resistance” in the text only because it is familiar to many readers.

To close the present chapter, topical elements of proof are summed up below.

* One *formal* proof is the direct observation of differences in DNA mass and chloroplast numbers between cells belonging to the specific mode Mₙ and the two variants, early (Vₘ) and delayed (Vₘ). Expressed as mean chloroplast numbers in stomatal guard cells, the differences between the specific mode and the two variants are equal but of opposite signs, -16.7% between Vₘ and Mₙ, and +16.7% between Vₘ and Mₙ. Expressed in DNA mass, the same difference of +16.7% is measured between Vₘ and Mₙ, whereas that between Vₘ and Mₙ is -14.7%. This deviation of 2% between the chloroplast and DNA markers baffled us a long time. Was it an observation error? A bad choice of markers?

We know the cause now. The 2% deviation is due to the traces of the genetic extrachromosomal element carried by plasmid DNA in the delayed variant. Davis & al. (1990) for instance, write on Agrobacterium-mediated gene transfer to Populus hybrids by transfer-DNA originating from a Ti plasmid. Such an extrachromosomal constituent probably was also observed in animals by Watson & al. (1989), who coined the name “double minute chromosomes”. These implement the *unstable amplification* of genes influenced by an inhibitor of an enzyme they coded. This enzyme, essential for the survival and cellular growth, is dihydrofolate reductase in mammals (DHFR; see Watson & al. 1989:718-719, transl. RAAMO). The genes manifest themselves here on small, paired extrachromosomal elements without centromeres. These are “...known under the name of double-minute chromosomes...”. Contrariwise, the DHFR genes *amplified in a stable manner* appear as prolonged chromosomal bands and “...are directly incorporated in one of the cell’s chromosomes; ...[the extended chromosomal bands] can sit in the natural site of the unamplified DHFR gene ... or in other chromosomes.” Here indeed we encounter the incorporation of plasmid parts and the transposon action in the early variant Vₘ as a daughter cell generated by a Vₘ founder cell.

* There are several *indirect* supporting pieces of proof. These are observations made elsewhere, some important ones being cited below.

The nucleotide numbers per turn of the helix path and the helicoidal rotation, to the left or to the right of the Z and A helices involved in the heteroduplex and the marking process (Figs.3-5 and 3-6) were observed by others, cited earlier. Our model of adaptation shows
the functional inevitability of these characteristics. Indeed it is a helix turning to the left, a Z.DNA with 12 bp, which allows the delayed variant $V_m$ to function, whereas the early variant $V_M$ requires a helix turning to the right. A.DNA with 11 bp only.

An old and unexplained mystery was the quincunx cutting of double DNA strands by the enzyme endonuclease. This was demonstrated as early as 1973 by genetic manipulation in vitro (Cohen & al. 1973; Morrow & al. 1974; Cohen 1975). In these experiments, DNA of plasmid pSC 101 was cut in a site where the complementary nucleotides form a palindromic structure (see p. 59). The strands were cut in two places. Foreign DNA was inserted at that site. The fragment of foreign DNA and the plasmid then were introduced into that acme of experimental organisms in genetic labs, the bacterium Escherichia coli (see Garrett 1994). Inside the bacteria, the hybrid DNA “.....replicates due to the replication capability of the plasmid”. This too completely supports our model.

Another indirect proof is the theoretic structure of the transposon Tn $X_{(A,B)}$ characterising the early variant B.DNA$_{(VM)}$ issued from B.DNA$_{(VM)}$. Its properties indeed closely resemble the characteristics of the Ac transposon in corn (Zea mays), described in Suzuki & al. (1989:539-541) as displaying “.....11 bp imperfect inverted terminal repeats.”

Still another indirect element of proof is the discovery of T.DNA. (various authors) This is a portion of a Ti plasmid from Agrobacterium tumefaciens. It is inserted in the vegetative cells of a host plant. This discovery allowed the production of transgenic organisms.

Finally, the rules of interaction in stress and adaptation would be quite comparable to the ones of the so-called SOS system in bacteria. SOS is put to work when the integrity of the genome is threatened. This occurs for instance when DNA replication is distorted under stress, as described by Tadei, Matic & Radman, writing under the collective pseudonym of TaMaRa (1996) and citing, among others, Friedberg & al. (1995). The stepped functional sequence of SOS as given by these authors runs parallel to our adaptive model: 1) appearance of a simple DNA strand as in the heteroduplex; 2) activation of some 20 genes with strong expression, among which the gene coding for the protein RecA; 3) increased genetic variability by mutagenesis, chromosomal rearrangement, transposons moving and recombination. According to the authors cited, all this happens after exposure of the bacteria to physical and/or chemical stress agents, such as UV rays, heat or nutrient deficiencies.

Our theoretical framework has been set out carefully in such a way that it always is falsifiable by experimental checking. Hence it is as certain as contemporary science allows to make it, that at the genetic and cell levels stress and adaptation interact according to rules not too different from those explained in the present chapter.

These rules now open the road to higher integration levels in the natural system hierarchy. The next chapter is on organisms, with emphasis on plants, particularly trees. It goes from meristems to organs, from organs to organ-bearing axes, from axes to branched arrays and thence to whole organisms versus populations. The fifth chapter then leaves the individual organism behind, to cover collective and physical scales ranging from the solar system to the living environment closely enveloping an organism. This allows us to use a sliding scale, the meeting place between stress and Life being the precise system level where they interact. Between the Sun and Life we will search for recurrent rules fitting in with the physical laws of matter and energy as known today (Prigogine & al. 1973, Prigogine 1980, Prigogine & Stengers 1984).
Chapter 4  The architecture of plants under stress

Around the year 1980, one of us had the pleasure to travel in Surinam with a professor of organic chemistry Dr. H.C van der Plas, then rector magnificus of Wageningen Agricultural University. When visiting one of the rain forest experiments in Suriname, in the Kabo region, we had the occasion to look at the architecture of the young trees growing up in recently cleared sites. The regular patterns of branching gave many of these trees – smaller species of families like Rubiaceae, Annonaceae or Flacourtiae – the physiognomy of giant organical molecules (Fig. 4-1). This analogy struck us both.

The branches with leaves arranged in neat rows like atoms in macromolecules, the branches in neat spirals, or in storeys, on the vertical stem like the turn paths along the imaginary central axis of a DNA helix, suggested fundamental patterns of nature, recurrent at widely different scales. This recalled discussions with Dr. F. Hallé, when working on our books on tree architecture (Hallé & Oldeman 1970; Hallé & al. 1978). We remarked upon the curious geometrical correspondences between tree growth and the growth of crystals in a saturated solution. Later, Dauget (1985) established fascinating similarities between tree architecture and that of corals. Some years ago, a thesis from Amsterdam analysed the architecture of corals with fractal geometry (Kaandorp, 1992). An overview from Minnesota provided many entries in the fractal geometry of forest organisms (Leary, see Lorimer & al.1994).

Hence it was no great surprise that the same message was strengthened by the combination of the silvological rules at the level of forest ecosystem dynamics (Oldeman 1990, chapter 7) and the results of the work at microlevel summarised in the preceding chapters of the present book. We discussed this in 1991 among the co-authors and decided that each of us would coordinate a chapter or two in which he or she felt at home. This is in essence what we did.

The jump from the architecture of the cell nucleus to the architecture of organisms may seem vast. However, the point of view is quite similar, because both the famous double helix and the famous giant forest tree, to be understood, must be sketched, mapped, and measured so as to calculate images rather than formulae. The present chapter portrays individual organisms.

The living communities and their majestic abiotic environment are in chapter 5.
Figure 4-1: A young tree built like a giant organic molecule. Example of Casearia sp. (FLACOURTIACEAE, Roux's model) at a roadside in French Guiana, 1977. Note vertical trunk bearing horizontal branches in a spiral. Photograph Oldeman.
4.1 - On plant architecture

Human beings orient themselves in the world they inhabit by assessing forms, weights and numbers. Recognising patterns and forms is the most important humans survival trait. Indeed visual recognition of the components of one’s environment is the first step in evaluating risks of life and death. Assessing weight is the second important capability, because things heavy or light may mean arms or tools, food or hunger, prey or predator. Counting is the third important capability. Indeed it comes last, because knowing numbers alone has no existential meaning without knowing first what is being counted. Architectural analysis follows this order and starts by investigating forms.

Recognising and understanding forms is one leg on which biological architectural analysis stands. The second leg is forging links between forms and organisation levels, because a form has relevance only at its own scale. The third leg of the architectural tripod is understanding of architectural dynamics, i.e. viewing three-dimensional form against the dimension of time.

4.1.1 - Criteria for architectural analysis

Architecture of organic systems is the complex of forms grown per well-defined organisation level in the hierarchy of living coherent assemblages, if one is to avoid the eroding term of “living systems”. Architecture differs conceptually from “structure”, because structure is neither limited to form, nor expressed exclusively per hierarchical system level. Architecture requires its own particular kind of analysis, being a particular case of structure.

The isolated consideration of only one level of organisation is not enough, as this book makes abundantly clear. In general, we consider the system level studied as if it were the cheese level in a sandwich. The two explanatory, neighbouring levels are the slices of bread. The upper one is the supersystem in which our system is at work as an active member. The lower level defines subsystems working together as active members of our system.

The following paragraphs show precise architectural criteria to analyse the growth of whole plants. However, they are not precise in the sense that they may be quantified by means of numerical values with many decimals. A building block of a plant or animal may be said to belong to a certain category, e.g. it is green or not green. Yet, variegated leaves show that the limits of these categories are not absolute: the borders between green and not green are vague. The categories are fuzzy sets (e.g. Kosko 1993; Ross 1995; Oldeman & Vester in press).

There is no break with the preceding chapters. The precision used in the present book is a precision linked to scale of observation, distinct per chapter.

Now the criteria listed in the present chapter concern organs and complexes of organs, built by organisms in order to survive. In general, they are built for individual survival, but they are hereditary too. This explains their fuzziness. They must have a general, inherited capability to make all individuals of their species survive. However, their architecture is flexible enough to respond to the close environment surrounding one particular individual organism.

This is true in so far as the exchange processes through the membranes or interfaces of the cells remain normal. Normal exchanges ensure the energetic balance providing for mitosis and meiosis. This ceases to be so under important stress impacts, such as X or UV radiation, radioactivity, toxic chemical impacts or attacks by free radicals. Such major stress factors pierce
all organic shields at higher levels and cause cellular membranes to deteriorate (Chapt. 5.2). The damages require counteraction, reserved to cells apt to transmit hereditary characteristics, which may augment the variability of these characteristics by mutation.

Mutation by the adaptive mechanism, explained in chapter 3, is close to sexual processes in primitive organisms like mutualistic or parasitic symbiotic bacteria (Procaryota) living with higher organisms (Eucaryota). When conditions regain a normal balance, Eucaryotic sexuality reclaims its rights and restores its own laws, Mendel’s laws. New alleles so are distributed haphazardly in the population. This happens either randomly or by using intermediate messengers. The flexibility of individuals belonging to such a population so is enhanced.

In all arrangements of parts of living systems, the utmost economy is predominant. Organs are “packed” or “wrapped” in such a way, that their energy cost is minimised and their profit maximised. Here again, the parallel with the stacking of nucleotides and double helices is striking. In terrestrial plants, this means that the organs of exchange, root hairs and leaves, have each to be positioned at a spot where their output of water and gases and their uptake of light and/or gases can be optimised at the least cost of building vessels, i.e. transportation tissue. The same can be said of the organs of procreation.

At the level of the organs this explains why most leaves are flat, having a large surface of exchange against a relatively short trajectory of the sapstream via vessels in the veins. This is not so for plants growing in dry or physiologically dry climates, where the leaf surface has to be reduced so as to reduce transpiration. They become near-spherical (cf. some Crassulaceae) form needles (cf. Conifers) or skip leaf expansion altogether (cf. Cactaceae). The organ level of organisation is treated more thoroughly in a later section (4.3.1.).

We will continue at the level of the whole plant.

The following list shows five fundamental criteria to distinguish organ complexes which are the building blocks of trees (Fig 4.2). These building blocks are called axes and are per definition assemblages of organs. The term “axis” is sometimes also used for strings of axes. This is careless wording, comparable to the helix we met in chapter 3, a term sometimes used to indicate the turn path which is only part of the helix.

a) Long axes, modular axes, short axes, pauperised axes (Fig. 4-2A) A long axis generally is built by an undefined (“infinite”) number of internodes, each with a leaf and its axillary products. A short axis is built by few, severely programmed, often small, specialised internodes. In between the long and the short axis sits the modular axis (or module; Figs. 4-2C; 4-31), which may have a considerable number of large or small internodes, is severely programmed and is one element in a string or fan of axes. Finally, a pauperised axis (not drawn) is unable to express its whole growth programme, due to some deficit of resources.

Most often it is easy to identify an observed axis as a member of one of the above fuzzy sets. Modules with many internodes may be confused with long axes and, if they have few, small internodes, with short shoots. However, the vagueness of the limits does not hinder the overall precision needed to assess the axis. The pauperised axis is no “trashcan” category wherein to drop difficult cases, but most often includes axes which have remained so small in terms of internodes, that no leaf spiral or other differentiation mark has been completely expressed.
Figure 4-2: Axial differentiation. The axis to the left possesses all the distinctive characters contrasted to the cases at the right side of the figure, e.g. A - (left-hand side) long shoot versus A (to the right), short shoot or brachyblast. B - pure axis versus mixed axes (for ortho/plagio see D). C - indeterminate axis versus determinate (modular or finite by flowering) axes. D - orthotropic versus plagiotropic axis. E - rhythmic versus continuous axis.
The axes per definition are assemblages of organs. In the same way, the cell nucleus may be seen as an assemblage of RNA, DNA and proteins. Both the organisation of organs and the organisation of macromolecules in assemblages follow rules at their respective system levels. The rules at the level of axial organ assemblages are shown in the following categories.

b) Pure axes versus mixed axes (Fig. 4-2B). A pure axis is architecturally similar over its entire length, a mixed axis changes architecturally somewhere in between its base and its top. Mixed axes are almost exclusively long axes. If the orientation of the axis (see d) is taken into account only, there are four kinds of mixed axes:

- orthotropic below/orthotropic above (common; Hallé & Oldeman 1970; Hallé & al. 1978)
- orthotropic below/plagiotropic above (rare; Hallé & Oldeman 1970; Hallé & al. 1978)
- plagiotropic below/plagiotropic above (abundant; Hallé & Oldeman 1970; Hallé & al. 1978)
- plagiotropic below/orthotropic above (rare; Oldeman & Hallé 1980).

This character can be assessed as easily as the differentiation between orthotropic and plagiotropic. Ortho/ortho and ortho/plagio mixed axes most often are found in shrubs, plagio/plagio axes characterise woody plants from very big trees to minute shrubs, and the plagio/ortho mixed axis is particularly apt to be a building block of branches and creepers. The assessment of frequency in nature of the four kinds of mixed axes, however, may be due to some extent to an insufficient number of observations to cover the plant kingdom evenly.

In analogy with Hallé’s reasoning on continuous, rhythmic and relayed growth (1986; see e below), we might remark here that a long axis which becomes overly long may switch to branch behaviour at its extremity (the case of Guyanese Mouriri species, Oldeman 1974). Here we may have the sequence long axis (branched), unstable long axis (branched), determinate long axis (sympodially branched; see c below and Fig.4-3F) versus mixed axis.

c) Determinate axes versus indeterminate axes (Fig. 4-2C). The end meristem of a determinate axis ceases to be vegetatively active at set, predictable moments. For instance, terminal flowering arrests axial extension regularly, so generating a determinate axis. The end meristem of an indeterminate axis remains vegetatively active until an accident kills it.

Again, these categories seem very clear. Still, determinate axes may form strings, in which the splicing of successive elements is camouflaged. This often results in practical difficulties to distinguish between an axis and a camouflaged sequence of axes. Usually, however, a good hand lens solves the puzzle, so that no doubt needs persist. The position of scars, leaves or flowers betrays the hidden configuration (for herbs see Blanc 1989). The criterion itself is not fuzzy, but according to some authors it barely has functional significance if hidden.

Doubts indeed appear when plant architecture is confused with biological function. A trunk may be built by one indeterminate axis or by a string of determinate axes. Such a trunk is seen as one physiological unit, often and confusingly called an axis. As said above, this is not done in the present book. The distinction is particularly important if other functions, i.e. adaptation and regeneration of the plant body, are considered.

d) Orthotropic axes versus plagiotropic axes (Fig. 4-2D). Orthotropic axes are vertical, plagiotropic axes horizontal. Moreover, orthotropic axes have leaves arranged in spirals, sometimes in double spirals when there are leaf pairs. Plagiotropic axes have leaves arranged in a plane, sometimes by forming them in that plane from the outset, sometimes by torsioning them into that plane later (Roux, 1968; Hallé & al. 1978; Vester 1997).
These categories are very clear at first sight (e.g. Fig. 4-1). However, when studied more precisely (Bancilhon 1965, 1969; Bancilhon & al.1963; Roux 1968; Nozeran 1978, Nicolini 1997) they are fuzzy. Mixed axes are distinct (see b above). Pure plagiotropic axes may slant. When slanting strongly they may be confused with leaning vertical axes. Plagiotropic axes rarely display two perfect rows of leaves. They often show visible traces of spiral phyllotaxis.

However, it usually is not overly difficult to attach a leaf-bearing axis to one of these two fuzzy classes. Seen next to the three other classes, the image is quite precise.

e) Rhythmic axes versus continuous or diffuse axes (Fig. 4-2E). A rhythmic axis shows an alternation of zones with long internodia and large leaves, and zones with short to very short internodia and scale leaves. The latter are the traces of a resting period. A continuous axis only shows one pattern with a similar internode length from one end to the other, and adult leaves of similar size and form. A diffuse axis is like a continuous axis without any regularly alternating pattern, but with wide and seemingly random variation in internode size and leaf form and size.

The terms rhythmic, continuous and diffuse are not quite correct because they indicate a growth process, not its result, an axis. This must be accepted as no word exists to say it better. The categories are fuzzy. No objective, crisp limits exist between rhythmic and diffuse, between diffuse and continuous. Often, particularly in tropical rain forests, the distinction is easily seen. Rhythm in temperate regions usually causes bud formation in winter. However, irregular weather in all climates may provoke diffuse growth in plants with continuous axes.

Hallé (1986) argued that rhythmic growth might be an intermediate with a resting, surviving end meristem, in between a determinate axis with a programmed death of the end meristem and a continuous axis without endogenous limits to vegetative extension growth. This vision has a direct bearing on notions regarding survival strategy, adaptation and adjustment.

As shown below, the five criteria can be combined in a diagram to visualise the conceptual resolution power of this diagnostic procedure. When criteria are no longer important or relevant, i.e. after a certain degree of either differentiation or pauperisation is reached, they are shown between parentheses. This does not mean that they play no role at all, but that their role is no longer structural at that organisation level. Hence such a structural differentiation would only count at a more detailed level, which we do not want to consider here.
Figure 4-3: Branched arrays. The branched array a bit to the left of the centre possesses all distinctive characters contrasted to the cases shown at both sides, e.g. F - (centre) branched array versus F (to the right) unbranched array. G - (center) core array versus G (to the right) edge array, i.e. building the edge of the plant crown. H - monopodial array versus sympodial arrays (modules in fan, umbrella) or modules in strings (I - vertical versus horizontal). J - continuous versus diffuse (to the left) or rhythmic (right upper corner) arrays.
One level of organisation higher, the above axes can combine in different patterns due to five branching processes. The next list of criteria hence concerns branching patterns. The architecture of the plant is an attribute of a concrete and visible object, the term “branched array” is introduced here. A branched array is a building block for more complex plants, like giant forest trees or extended creepers.

Here are the elements to distinguish between different branched arrays.

f) Branched array versus unbranched array (Fig. 4-3). All aboveground organs (e.g. leaves, flowers) of the unbranched plant are formed directly on the one and only axis. In big structures like Palms this leads to very complex leaf and/or flowering systems. Some minute herbs also show this structure, such as saprophytic miniature chlorophyll-less Gentianaceae found in the neotropical rain forests.

The distinction between branched and unbranched building blocks is crisp, i.e. barely fuzzy. It is mentioned here for the sake of completeness, because it can be seen immediately in an estimated 99% of all cases. Rare doubts arise mainly in sympodial strings with hidden relay branching.

g) Branched arrays in the core versus branched arrays at the edge (Fig. 4-3G). This is the architectural view of trunk versus branches. Any physiological bias has been eliminated. The centerpiece of a plant is its trunk, or a creeping central array in a prostrated shrub or herb (Jeannoda-Robinson 1977). Usually, a trunk is branched. A branch mostly is branched too.

The assessment of a core array can be astonishingly precise, as soon as the observer sheds the habit of seeing trunks as long and thick with roots at the base. The core is not fuzzy. Contours do become fuzzier towards the edge of the green structure and also the root system (see Atger’s illustrations, 1992). Edge arrays often show a delicate, minute outer architecture at a finer scale, although it only seems a fuzzy blur at the scale of the tree (cf. Vester 1997).

h) Sympodial branched arrays versus monopodial branched arrays (Fig. 4-3H). Sympodial arrays mostly consist of an assemblage of determinate axes, monopodial arrays surround a monopodial core. Here, the physiological trunk may be either a monopodium or a sympodium, without our set of definitions close to nature being falsified by this fact.

The distinction is not always easy, due to fuzzy transitions within a plant from one to the other array (e.g. Aesculus hippocastanum). There are large sympodia among the famous tropical umbrella trees (Leeuwenberg’s model, Hallé & Oldeman 1970, Hallé & al. 1978), and minute monopodial branched arrays in the genus Phyllanthus (Euphorbiaceae; Bancilhon 1965, 1969; Roux 1968; Nozeran 1978). Sympodial branched arrays possess linear strings or horizontal fans of axes, or umbrella-like crowns (Fig. 4-3I). A string array forms a fuzzy line. Its axes are often camouflaged, the place where an axis takes over from a previous one is hidden to the untrained eye. A fan-shaped array lies in a fuzzy plane and axes often appear more clearly, although their ends may be disguised as lateral organs, e.g. inflorescences. In umbrella-like crowns, occupying fuzzy volumes, axes are clearly visible and often flower terminally.

The branches of monopodial arrays surround a central axis, from which they grow laterally. They either build plagiotropic arrays in the form of feathers or fishbones, or they build green columns, cones, pagodas or “hatracks” if at least the central axis is orthotropic (Fig. 4-3I). It is emphasised, that these words are no new standard terms. They just evoke the general shapes built by sympodial and monopodial arrays, like the “lollipop” in plasmid architecture (Chapt.
3). They can not be used in formal diagnosis. Contrariwise, the next set of branching arrays concerns analytical properties which are very important determinants of plant architecture.

j) **Continuous, rhythmic and diffuse branching arrays** (Fig. 4-3j). Like arrangements of leaves in axes (see e above), lateral branching patterns may be a main axis bearing side axes in a continuous spiral, in tiers separated by unbranched segments, or at irregular places along the leaf spiral of the main axis. The latter shape indeed reminds one somewhat of a hatrack. Like the continuous, rhythmic and diffuse architecture of axes, sets of branching arrays with the same disposition have fuzzy limits. They can most often be recognised, but in some cases, some 10 to 20 % as estimated from numerous field observations by one of us (RAAO), these form complexes are transitional between categories. In our experience this does not obscure the whole image, which agrees with the tenets of fuzzy logic (cf. Kosko 1993a,b)

Here follows the same type of diagram for branched arrays as the one given above for axes.

![Diagram](image)

Tree architecture is given above in a way and in words quite distinct from the original books by Hallé & Oldeman (1970, 1975), Oldeman (1974), or Hallé & al. (1978), and also distinct from newer vocabularies like those developed by Édelin (1984,1986, 1991, [in press]) or Blanc (1989). For several reasons the present book uses other terms.

First, it is useful to express the same knowledge in a different way, so as to avoid *idées fixes*, i.e. ideas immobilised by words. Second, it is useful because these terms are a simple tool to explain matching biological rules at vastly different scales, e.g. cell nuclei and whole plants. Third and not least, different words used by past authors on plant architecture, including the classical German morphological schools (e.g. see Goethe 1790, Hofmeister 1868; Roloff 1989; Gleißner 1996, 1997 [in Édelin, in press]), here are shown not to represent irreconcilable viewpoints (cf. Froebe & Gleißner 1995; Caraglio & Barthélémy 1997).

The following step in the explanation of plant architecture concerns the assembling of branched arrays as building blocks for larger, higher or wider plants. This part is largely a rewording of the concepts developed at the international colloquia “L’Arbre” in Montpellier (Proceedings edited by Édelin, 1986, 1991, [1997 in press]). However, the concepts of levels of architectural flexibility versus levels of architectural severity are due to Oldeman & Vester (in Édelin 1997, in press) and Vester (1997).
For this last step into the analysis of plant architecture a list is given here.

**k) Uni-array plants versus multi-array plants** (Fig. 4-4). This set is the higher level expression of the unbranched and branched axes we saw one level lower (Fig. 4-3F). Uni-array plants never show more than one branched array in their life cycle. Multi-arrayed plants show the same branched array more than once in a similar if sometimes imperfect pattern.

If in a multi-array the branched arrays are all of the same kind, and their axes also are all of the same kind, it is hard to see the difference between uni-array and multi-array. This happens in the many-fanned trees of the families of Leguminosae (Oldeman 1974, 1989; Oldeman & Sieben-Binnekamp 1994, in other terms) or Fagaceae (Oldeman & Sieben-Binnekamp 1994). The key feature is the core array. Each new trunk axis mimics the original trunk. If a core array displays clear differentiation between trunk and branches, the diagnosis is much easier.

One way in which a uni-array can change compared with the original architecture issued from the seed is by architectural metamorphosis (Édelin 1977). This happens by the intercalation of new and different kinds of axes in between the trunk and the edge axes during the branching process, later in life but not always very late (e.g. Dipterocarpaceae). This architecture marks trees rather than other plants. As said, in the present book we distinguish the intercalation of axes, *metamorphosis*, from the intercalation of branched arrays, *hypermorphosis*.

Mixed and/or determinate axes complicate things because they mostly form sympodial arrays. In small herbs with this architecture the sympodia occur near or below the ground. Multi-array plants with sympodial axes are usually difficult to analyse. The links between arrays often are well camouflaged, like those between axes in a sympodial string. It took many years of careful work by Édelin (1991) to show that a surprisingly large number of large forest tree trunks of the Guyanas are built by a succession of arrays (“architectural units” says Édelin). Earlier in the XXth Century, European forest trees had revealed this after long years of painstaking work by German teams (cf. Roloff 1989, Gleißner 1996). Their traditional terms sometimes obscure the universality of their findings due to an excess of detailed latin terms.

The distinction between uni-arrays and multi-arrays is not very fuzzy. It can be made clearly and with certitude in an empirical proportion of well over 90% of the cases examined. However, there are few easy and clear criteria, if any, to arrive at a diagnosis. It costs nearly as much trouble to make sure of these patterns as it takes to analyse chromosome build-up.

**l) Controlled versus loose multi-arrays** (Fig. 4-4L). Traces of regulation or control in the architecture of uni-arrays, are left in the disposition of the axes. At different steps in the development process, many multi-arrays show metamorphic architecture (e.g. *Betula* spp.). In uni-arrays there is repetition of such edge arrays, but not of the whole branched array.

Multi-arrays are stacked branched arrays. *Controlled* multi-arrays are stacked in a neat pattern. *Loose* multi-arrays show no regular pattern and mainly are opportunistic (“réitération”, Oldeman 1974, Hallé & al. 1978). Controlled multi-arrays resemble uni-arrays. On closer inspection, however, their branches and trunk consist of many similar branched arrays deceptively well spliced together. Their organisation is clear and simple. Their parts in turn are as highly organised. Controlled multi-arrays show high-level monopodial organisation, not with axes but with arrays. Intercalation of axes leads to *metamorphosis*. For the intercalation of arrays we coined the term *hypermorphosis* (Fig. 4-4) in an earlier paragraph.
Figure 4-4 : Multi-arrays. From left to right: seedling (*), sapling, “Oldman’s tree” (K), “Hallé & Oldman’s tree” (K), “Blanc’s tree” (L, mobs) and “Édelin’s tree” (L, teams). K (center) - Uni-array: Hallé & Oldman defined branched arrays (architectural models), which according to Hallé may show gigantism. K (left-hand) - Multi-array: Oldman showed piles of models, growing smaller and less complete in successive waves showing size and behavoiur like trees, treelate, shrubs and herbs, and constituting “subcrowsns” by reiteration. L (extreme right) - Controled multi-array: Édelin with his metamorphosis concept sketched trees built by teams of units which might be parts of models, and show intercalation of branching (see Fig. 4.9). L (right of center) Loose multi-array: Blanc regarded the outcome of all tree growth like a mob of more or less regularly interlinked axes. Édelin’s tree (hierarchic) and Blanc’s tree (polyarchic) were seen as two faces of tree architecture by Édelin. Super-arrays not drawn, see Fig. 4-5.
A loose multi-array is a kind of high-level symposium. The arrays are stacked on top of each other, or next to each other, each a complete or partial repetition, from a vegetative meristem, of the first array from the seed (“réiteration”, Oldeman 1974, 1990). This is the acme of flexibility. A new branched array of the right size is built wherever needs be, e.g. in a spot in the sun, in wet soil where rooting is good, or in places where parts of the organism must be regenerated. In ramets (Begon & al. 1986, p.126) of vegetation-forming herbs, e.g. grasses, cohesion is so loose that branched arrays finally lead an individual life forming roots. In controlled multi-arrays, control mainly is endogenous. In loose arrays, it is mainly exogenous.

Loose multi-arrays and plant fragments cultured in vitro show a noteworthy correspondence. In in vitro tissues, cohesion among adventitious cell groups is as loose as among the branched arrays in whole plants. Moreover, the most unruly loose multi-arrays are found in stressed biotopes, e.g. subboreal or desert regions, or the hot and dry canopy of the tropical rain forests, or again the deep and irregular shade under the forest (e.g see Blanc 1989). Many examples of such plants are in Ericaceae (Oldeman 1990).

m) Superarrays versus populations. This pair of classes is only illustrated by an example of the well-known phenomenon of giant fig trees in the tropics and subtropics (Fig. 4-5A). This is a super-array, in that it is a quite well-organised assemblage of multi-arrays, each forming its own adventitious roots. For the tourist, these many roots resemble many trunks of the tree, so that travellers wrote on “one giant fig tree forming a forest”.

This architecture, with adventitious roots, forms a near-population of multi-arrays remaining one individual tree, but only just. The other way round, trees with many root suckers, each a multi-array, form a giant superarray cohering below the ground by a huge root network (e.g. Robinia pseudo-acacia). The parts are very independent, so the potential for genetic variation in such giant and loosely cohering pieces of plant mass should be huge, too. This assumption rests on the comparable situation of near-independant cells in vitro under stress.

To finish these pages on the basic image of plant architecture, below is the third organogram of architectural features, at the organisation levels above the branched array.

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M. SUPER-ARRAYS (POPULATIONS)
Figure 4-5: A - Super-array, example of *Ficus parvifolia* (MORACEAE) in Yangshou, China, 1991. Note tourists for scale. Each reiterated multi-array makes its own adventitious, aerial root system, so the super-array indeed is a transition between a giant individual, with multi-arrays as parts, and a clone-like organism built by the cohabitation of, essentially, a clone-like population of multi-arrays. B - Example of reiteration in multi-arrays. *Symphonia globulifera* (GUTTIFERAЕ), Ile de Cayenne, Guyane Française, 1977. Note strong similarity among architectural patterns around each vertical core axis ("trunk") and Roux's model (shown in Fig. 4-1 in another genus). Photograph F. Hallé.
The terms by Hallé (1986) “gigantism and repetition” are not restricted to this last diagram. They apply to each of the three summing-ups at the level of the axis (A-E), the branched array (F-J), and the multi-array (K-M). The present section was kept severely architectural. Only rarely allusions were made to dynamics and growth, when necessary for the sake of clarity.

4.2 - Architectural dynamics implied in plant building

The previous section was a selective reminder of criteria to judge plant architecture. A choice was made among many, because no full handbook on plant morphology or plant architecture is needed to establish links between tree biology and architecture and those of the cell and its nucleus. The matter has a long scientific history (see Sattler 1982; Froebe & Gleißner 1995).

However, many phenomena in the double helix and the cell are closed or limited in space and time. This is not generally so in the growth of whole plants. Organs like indeterminate axes can in principle grow on without any endogenous brake or stop. In a Wageningen greenhouse, a plagiotropic branch of an Indonesian Dipterocarp tree (Shorea sp) was grown from a rooted cutting. It grew on for 5 years. Though several meters long it remained a rooted, plagiotropic branch. This behaviour was studied thoroughly by Bancilhon & al. (1963) in herbaceous Phyllanthus species (Euphorbiaceae). For a recent, thorough study of plagiotropic branches in European beech (Fagus sylvatica) see Nicolini (1997).

We are not able to conceive any conditions causing a DNA helix to grow a centimeter or longer, or inducing a cell to grow into the size of a football. These living assemblages do not grow but replicate, divide or multiply. So do other plant parts, for instance determinate axes as standard modules in a sympodial array, or branched arrays like Roux’s model (Fig.4-5B) in the Guyanese marsh forest tree Symphonia globulifera (Guttiferae). This indeed is the same model as shown in figure 4-1, here duplicated in an adult tree, exemplified by another species.

Still, sizes and masses of the different arrays are open, not constant. Duplicates of the original tree with its unique array, however, are always smaller as long as they can not make their own root system and have to share the sapstream with the rest of the whole tree crown.

4.2.1 - Growth rhythms, DNA pinching, and water

Rhythmic architecture and growth are due to rhythmic meristem activity. Rhythmic growth was studied experimentally, e.g. by Hallé & Martin (1968) in flushing rubber trees (Hevea brasiliensis, Euphorbiaceae). The results were cited and illustrated by Hallé & al. (1978).

Rubber trees have compound, digitate, rather large leaves with three leaflets. They flush six times a year, so their axes show traces of resting every two months. Hallé & Martin cut away two leaflets in every young leaf of a few mm long appearing during a flush. The axes grew continuously, not resting as long as they had artificial unifoliolate leaves. Cellular and meristematic activity were monitored by microscopic staining. Periods of rapid leaf and internode growth and strong meristematic cell division alternated with periods of leaf maturation and expansion and meristematic differentiation. The key was water.
The tree’s water balance was alternately in favour of leaf expansion, consuming vast amounts of water, and meristematic building activities, which also consume much water. A third water glutton is subapical protoxylem ripening into functional xylem, destined to conduct sap to the apex. It is not only water that counts, but also the energy, nutrients and hormones it carries. In the xylem, water ascends as a weak solution of nutrients and nitrogenous rhizosynthesates. The descending, concentrated solution in the bark with sugars and other carbon photosynthates completes the sapstream. Following most plant physiology handbooks, the high water consumption by the ascending sapstream is due to huge transpiration sucking the ascending mass movement up through the woody capillaries. The descending sapstream is driven by electromagnetic interactions between dissolved ions and phloem cells.

Hence the water balance is an energy balance, but it is certainly regulated by macromolecules too. Crabbé (1987) gave an integrated image of the cross-action by meristematic activity and energy balance including metabolites and/or phytohormones (Fig. 4-7). We added four hierarchical system levels to his figure, i.e. cell nuclei, cells, organs and axes. The enormous complexity hidden behind the apparent simplicity shown can be understood intuitively by imagining the countless interactions within each system level and between levels. Often, such interactions would each require a diagram like tables 3-3 or 3-4. Such diagrams do not exist today at all classic physiological levels (but see Crabbé 1987). Champagnat & al. (1986) and later Parmentier & al. (1991) provided drawings based on experiments, showing rhythmic sequences of differentiated leaf-bearing internodia. They bear some resemblance to drawings of sequences of nuclear bases (e.g. Chap. 3). Leaf spirals according to a Fibonacci algorithm may be compared to double helices in a sort of general way. However, the shape of the parts of the sequence can be well defined in a chromosome today, but less well in an axis or a root. Probably, such definitions are inherently fuzzier than the genetic ones.

On the contrary, Nozeran (e.g. 1978, 1986) zooms in from the organisation of the axis towards the nature of its constituents, as far as their nature is important to explain their role in the axis. Both approaches will undoubtedly meet and enrich each other. Both essentially consider energy flows that feed and inform axial differentiation, but seen from distinct viewpoints.

In chapter 3, we saw that the marking of the double helix at regular intervals also involved an energy transfer, particularly by the cut-and-paste operations operated by the plasmids (Fig. 3-16 and 3-17). The high sap consumption by growing meristems and Crabbé’s figures (1987, cf. Fig. 4-7) show the sapstream to be linked to the mitotic events noted by Hallé & Martin (1968). In tissue or cell cultures in vitro there is no sapstream. This lack of a biologically organised water input certainly explains one part of the stress brought to bear upon the explants in this artificial environment. Indeed, the remaining regulation of water movements is abiotic.

In a later section, more examples will be shown in which all kinds of stress work out on the sapstream, the architectural dynamics and the mobilisation of adaptive mechanisms.
4.2.2 - Orthotropy, plagiotropy and molecular arrangements

In the architecture of whole plants, orthotropic and plagiotropic differentiation are important. Vertical orthotropic spirals are as spectacular in small versions (Fig. 4-6) as in whole trees (Fig. 4-1). Beech trees (*Fagus*: Peters 1992, 1997, Cao 1995, Nicolini 1997) and many legume trees have plagiotropic, mixed axes (cf. Fig. 4-2B,D), many herbs consist of pure plagiotropic, prostrated axes, creeping on the soil they cover (Jeannoda, 1977). The original concept of plagiotropy, due to German botanists in the early XXth Century, was vegetative differentiation induced by gravity. It first was called diageotropy, an orientation across the Earth force.

A few researchers wrote on photogeotropy, flattening of green plant surfaces across the light direction. Massart (1923) tested diageotropy by slowly revolving a trunk placed horizontally, so exposing its plagiotropic branches to gravity from all directions. The branches remained plagiotropic. In later experiments, Roux (1968) also showed plagiotropy to be obstinate. Once an organ has become plagiotropic, changing its differentiation is very hard.

This strong position of plagiotropy in plants is intermediate between plagiotropy in animals, predominantly plagiotropic (F. Hallé, pers. comm., ex Oldeman 1990), and the constituents of the cell nucleus, essentially orthotropic. As shown by the double helix, radial or spiral architecture marks living macromolecules. Even such a vital body as the plasmid is round and its molecular structure has the shape of a fat disk. When straightening out along DNA molecules, the plasmid enters once more in the spiral world. Several factors explain the dominance of radial symmetry at this system level.

On the one hand, the minute size of the cell nucleus and its components is striking. The latter are wrapped up as economically and as safely as possible (see chapter 3). They are twice rolled up, both on the double helix and as chromosomes in the nucleus. Now small size in general goes together with a short lifespan. The dynamics of cell division are measured in hours or minutes. The dynamics of the nucleus are so fast that nobody paid much attention to rates and time sequences. For most scientists, nuclear cell dynamics are all-but instantaneous.

The lifespan of macromolecules, particularly those with a quick job like plasmids or enzymes, is very short. However, compared with the general dynamics at that level, the relative lifespan of the macromolecules and molecular architectures involved perhaps would give the lie to our assumption that no important plagiotropic elements exist at that level. They might be simply so short-lived that we forgot to observe them. We might forget because in whole plant architecture, as a rule-of-thumb, stronger plagiotropy goes together with a shorter lifespan. The extreme is a leaf-like axis or phylloform (Hallé 1967; Roux 1968; Hallé & Oldeman 1970), and at the organisation level of the organs, a minute leaf (*nanophyll*).

On the other hand, plagiotropy and other differentiation’s like shortshoots are due to adaptive growth, determined by the immediate environment (cf. ch. 5). It is commonly assumed that light, gravity or coevolution with insects mainly select the organs, axes and branched arrays able to survive. However, these factors vary in an important aspect, to wit, their orientation. Light, gravity and wind are more or less oriented and provide a sustained, reliable pressure, always from the same average side or direction. Insects or water are little or not oriented.
Figure 4.7: Diagram of the physiological dynamics regulating the growth of plant axes. Inspired by Crabbé (1987), hierarchical levels and rewording by the present authors.
We insisted above on the watery environment of the components of cell and nucleus, resting on the biological organisation of water transport inside organisms, at all levels. A first survey of specialised plant architecture at the molecular level supports this. Nothing we know today suggests other ecological forces to influence architectural dynamics at that level. Correlations between the synodic lunar cycle and biological cell behaviour indeed not only are linked to electromagnetic, but also to hydrodynamic influences of the moon.

In a living aqueous environment there are biological tides. Tides are stronger when their bed is narrower, as shown by inundation’s of the deltas of Bangladesh or the Netherlands by the sea. Narrow tubes are epitomised in capillary xylem vessels, conducting the ascending sap-stream. An intact plant hence has intense biological tides, with strong vertical vectors during full moon and weak ones during quarter moons. Towards full moon, end meristems of orthotropic axes receive more sap. However, maxima in end meristems of plagiotropic axes occur during the first and second quarters, when the orthotropic apices are at their minimum.

A strong ascending sapstream at biological high tide carries to the meristems large amounts of nutrients, phytohormones like auxins and cytokinines, enzymes and regulatory proteins. This leads to activation of mitotic processes causing an increased meristematic activity. Indeed, gardeners know empirically since times immemorial that one should prune hedges for growth in height during the first quarter and full moon, and for growth in width during the last quarter and new moon. On the contrary, cell clusters in vitro barely have any tidal mechanism left.

4.2.3 - Architectural metamorphosis and intercalation of macromolecules

The marking, cutting and splicing of molecular structures at the level of the double helix find a strong parallel in architectural metamorphosis. In this matter we follow Édelin (1977, 1984, 1991), who discovered the metamorphic branching patterns so common in tree architecture.

Metamorphosis is due to intercalary branching (Fig. 4-8). Germination yields an unbranched seedling with spiralling leaves (Fig. 4-8 SL). The same principle shows up in axes with double spirals like those with leaf pairs, or in plagiotropic seedlings. Seedlings start to branch from axillary buds which are positioned according to the leaf spiral (Fig. 4-8 ST.1).

When the tree grows up, the first branches have the architecture of the first branch. In the figure, plagiotropic branches were chosen so as to make the illustration clear. After the first branches, the next ones in the same leaf spiral abruptly cease to be plagiotropic and become orthotropic (Fig.4-8 ST.2). In their turn, they bear branches which are plagiotropic like the first branch of the tree (Fig.4-8 ST.3). After some time and after a number of plagiotropic branches the branches also replace plagiotropic by orthotropic branches in the spiral (Fig.4-8 ST.4).

In metamorphic architecture we may speak of the rule of constancy of edge arrays.

Édelin (1984) showed that monopodial trees (see Fig. 4-3F) have a limited number of branch orders, often five (Par. 4.3.1.2). After metamorphic intercalation of five branch orders, one after the other (Fig. 4-8), the sixth order reverts to the differentiation of number one. A new trunk is formed, so the whole architecture is recurrent from a bud. The architectural path so is followed, step by step, from one axis to an uni-array to a multi-array including many times the pattern of figure 4-8 SL, to a super-array in which multi-arrays are rerun. Computer simulation of these events yields patterns similar to those observed in vivo, (Barthélymi & al. 1995, 1997) which at least shows numerical consistency in plant architecture.
Figure 4-8: Intercalary branching. The tree can form branches of six orders (O1 to O6, above to the left), the leaf considered as the last order. Regular intercalary branching proceeds in 4 steps following the seedling. SL - Seedling composed by O1 (axis) and O6 (leaf). ST.1 - Sapling composed by O1, O5 and O6. ST.2 - Sapling composed by O1, O4, O5 and O6 (leaf). ST.3 - Older sapling composed by O1, O3, O4, O5 and O6 (leaf). ST.4 - Oldest sapling (in other species this is the adult phase) composed by O1, O2, O3, O4, O5 and O6 (leaf). The order of each axis is recognized by precise morphological markers.
Architectural metamorphosis has some features in common with dynamics in the cell nucleus. The first is intercalation. Instead of stacking ever more complex components on top of each other, both systems automatically react to certain configurations of their bodies by initiating and activating morphogenetic processes of an ambiguous nature. The paradox is, that in both cases architecture has to change in order to sustain itself. One solution is procreation of the body by forming a population. The other is acting upon the first architectural sequence by using a second architectural sequence for purposes of architectural repair, mutation, or both. Populational as well as individual architectural flexibility rest on intercalation cq. insertion.

In whole plants, the whole architectural sequence indeed must be followed to sexuality with its fructification and procreation. Intercalary branching allows jumping from seedling to fructification on early formed, “late” axes without passing through a complex branching sequence. Under heavy stress, populations so contain small, precociously fruiting individuals. Under lesser stress, organisms grow larger by intercalary sequences, but still do not lose their precocious procreation. This explains the well-known small, little-branched individuals of many widespread species, when growing in less hospitable parts of their geographical area.

Genetic architectural dynamics take intercalary steps by marking, pinching, cutting and splicing. The whole plant does it by the mobilisation of meristems which from one leaf axil to the next show different behaviour (Nozeran 1978, 1986). These form side axes, on which the lateral meristems may again revert to earlier dynamics. Nozeran’s morphogenetic sequences along axes are embodied in strings of meristems, each with a potential slightly different from both earlier and later meristems in the same string. In the same way, there is a clear sequence of complex molecular structures, each with a different potential, along a chromosome. In both sequences, certain steps are triggered under stress. Meristems (buds), transposition elements and chromosomes are tools in the differentiation sequences at different hierarchical levels.

The second feature common to architectural metamorphosis and genetic dynamics is the component number per architectural element. DNA-bases number 5 per half-helix turnpath in B.DNA, 6 in A.DNA. Number 6 indeed is the restart of a new half-turn of the DNA-helix (Table 3-3). Often, a complete branched array has 5 branch orders (Edelin 1977, 1984). The sixth vegetative differentiation order is automatic reiteration, the rerun of a branched array. The Latin term reiteration indeed means “taking the same road again” (Oldeman 1974).

However, we know that living systems never take exactly the same road twice. The second road may be similar, but selection stress never is quite identical the second time. Finding a decision point at five differentiation steps at two hierarchical levels so widely divergent as tree architecture and the genetical helix hence is at least a coincidence worthy of wonder.

4.3 - The rules of organ and whole plant behaviour under stress

In comparison with the relatively few, elegant steps of genetic molecular dynamics under stress (Fig.3-7, 3-8), the steps in morphogenesis of whole plants are more numerous and less elegant. There nonetheless is evidence to show that whatever the variation in stress, plants draw upon relatively few inherited growth reactions, leading to an architecture allowing them to prolong life. Chronic lethal stress of course in the end causes chaos and death. This is clear
both from the experiments in chapter 2 and from newspaper photographs showing whole forest dieback in Central Europe a few years ago, assumed to be due to acid rain stress.

However, stress peaking during a short moment, selectively killing members of populations, is more interesting. Forms of such acute stress were discussed by Crabbé (1987; Fig. 4-7: “prime movers”). They often are periodical, like seasonal cold and/or drought. The next chapter will show that most forms of natural stress do not arrive at most organs or organisms in their original, crude, chaotic state. They are previously filtered or organised by ecosystems.

Organised stress is met by organised architecture of the organisms, i.e. the organism is adapted. Its architecture dictates standard growth, maintenance, and exchange of matter and energy with its direct environment. Satisfying architecture is passed on through procreation and characterises all members of a well-adapted population. In an individual life cycle, it ordains rebuilding that architecture, with a certain fuzziness allowing for small incidents like warmer winters or unexpected dense insect populations. Fuzzy rhythm in oak generates no identical flushes, only similar build-up of all flushes (Parmentier & al. 1991, their Figs 4 to 6). Differences are buffered by a subapical ATP pool as shown by the in vitro experiments by physiologists like Barnola, Champagnat or Crabbé on oak in nutrientless distilled water.

However, except for organised, more or less periodical stress, there are “new” forms of stress, which do not fit into the historically observed nature and patterns of stress, its force and its periodicity. They appear to be chaotic. Strong chaotic stress can not be met by mechanisms of architectural physiology as they are. Whole architecture and whole growth behaviour have to change. This again is the paradox of section 4.2.3. The architecture has to change if the same architecture is to survive. Are there any features in whole plant architecture which anticipate this need, with a task like that of A.DNA and Z.DNA in the realm of the chromosome?

4.3.1 - Reaction to stress and organisation levels

Organic architecture and architectural dynamics can only be understood well if they are examined at their proper scale or level. Hence we first regard some known mechanisms for adaptation and resistance per general level. After the genotype level of the earlier chapters, the scales considered below in ascending order are the organs, the axes and arrays, and the organisms themselves. The latter are directly linked to populations (cf. schedule K.-M., end sect. 4.1.1.).

4.3.1.1 - Organ mass, size and shape.

We know of no study on the comparative organ biomass (e.g. leaves, fruits) of plants in biotopes with and without stress. Leaf classifications according to size and shape described by Dansereau & Lems (1957) are useless in our study, because they concern mean values for whole species, in which variation due to stress is averaged out. Leaf class diagrams by these authors (“Dansérogrammes”) are used in syntaxonomical classification of vegetation types.

Most studies on organ mass are agricultural or silvicultural and usually do not separate biological mass from commercial mass. We hence can not compare the studies on DNA and chloroplast mass of chapters 2 and 3 with studies on organ mass. However, we will try to sketch the situation by parametric assessment of organ behaviour under stress.

Organ size is a fuzzy parameter of organ mass. Big, massive organs often owe their stress resistance to excellent overall adaptation. Massiveness always is due to biological investment of
matter and energy. Massive organs hence are often able to function for a long time. Tiny organs not seldom function over short periods, then to be shed. This is discussed below for “throwaway organs”, named by Givnish (1978), whose working definition of a compound leaf was “a collection of leaflets arranged about a decidual twig”. This indeed pinpoints an essential property of massive organs, which beyond a critical size develop an inner, plurilevel hierarchy. Givnish rightly distinguished “leaflets” and “rachis” from the whole leaf system.

Big organs usually are systems with multi-level control and build-up.

Their subsystems may be anatomically differentiated as tissues, e.g. wood and bark in a tree. Morphologically they may appear as suborgans, e.g leaflets in a compound leaf, fingers in a hand, or anthers in a stamen. As usual in system analysis, the system limits, the subsystems, and with these the length of the appropriate standard measure, are chosen by the scientist involved (cf. Lorimer & al. 1994). The above systems-with-subsystems are inherently fuzzy in their definition. The rule concerning big organs indeed is true, but its expression is fuzzy.

The organisation of both large and small organs is expressed by their architecture, visible as a complex geometrical shape (cf. Hallé & al. 1978). There are very complex small leaves and very complex big leaves. Size or massiveness are no absolute but relative properties, related to the axis bearing the organs. The same leaf or flower would be large if borne by a thin twig, and small if borne by a fat branch. Corner’s rules (1949 ex Hallé & al. 1978) rest on what we now might see as a scaleless, fuzzy rule of similar proportions between any axis and its appendices. Corner postulated two rules, capable of mathematical treatment. The stouter the branches of a tree, the stouter and the more complicated are their appendages (first rule), and the more branched a tree, the slenderer are its branches and their appendages (second rule).

The fuzzy rule can be experimentally tested by checking whether or not there are forms of stress causing the appearance, on a similar twig in the same plant, of leaves, inflorescences or flowers in several distinct weight classes. Hypothetically, there might be one average or “normal” weight class and two classes differing from it by x% more or less weight. This hypothesis can be proven by checking whether regular categories of leaf weight variation exist, linked to stress. If so, the hierarchical range of this rule extends from DNA and chloroplast levels to the organ level.

One indication that this might be a regular feature are the classical sun leaves and shade leaves in one and the same plant (Schiper 1903; Thibaut & Comps 1991). The rule of thin shade leaves with a large blade versus thick sun leaves with a reduced blade also can have a morphological aspect. Horn (1971) provided drawings of discretely zigzagging lobes in leaves of “a seedling” and “a shaded branch near the ground” in American black oak (Quercus velutina, Fagaceae), versus strongly jagged and complex lobes in leaves “progressively higher in the tree”. The leaf forms may well be characterised by the fractal dimension of their perimeter, as was done by Vlcek & Cheung (1986, ex Lorimer & al. 1994).

Horn discerned them by comparing a circle equalling the whole leaf surface with the largest circle that can be drawn inside the leaf contour. Treetops are much stressed due to radiation and drought. Stressed leaves higher up in Horn’s black oaks show strongly increasing values of the circles’ quotient, indicating an increasingly lobed leaf. At increasing stress, the total leaf surface first decreases compared with the seedling leaf. When stress continues to increase with height in the tree, first the total leaf surface increases again, then decreases once more. The leaves in the forest canopy, assumed to be most stressed, are smallest and most divided.
Horn (1971, his Fig. 2.1) also provided light transmission spectra of leaves from various heights in an oak-hickory forest. Over the whole photosynthetic range of 400 nM < λ < 800 nM, between 0.1 and 0.4% of the total light passes through the leaves in the 500 nM < λ < 640 nM range. Much is absorbed at λ ≈ 660 nM in the red, with only some 0.02% passing through. Then a sharp transition occurs to higher transmission of some 0.8% at λ ≈ 730 nM in the far red. These are the stress wavelengths timed by the lunar cycle (Sect. 3.2.1). The small, complex leaves in the forest canopy absorb much red for photosynthesis. The large, simpler leaves in the undergrowth certainly receive a relatively abundant share of far red light.

Research by the Netherlands Institute of Ecology in Heteren on garden and forest populations of Plantago lanceolata (PLANTAGINACEAE) indeed showed that “Plants from two contrasting habitats both react strongly to the light intensity and the red to far-red (R/FR) ratio of the ambient light. Light intensity mainly affected plant size, whereas light quality affected the growth habit. Populations differ in their mean response rather than in the level of plasticity (i.e. the slope of the reaction norms). Experiments show that genetic factors (population effects), R/FR ratio, and hormone treatments (GA or CCC) have similar effects on morphology, and are largely additive and interchangeable” (Van Tienderen & Van Hinsberg 1996:87).

The light conditions at the forest floor were known, for instance by Pardé (1974), and confirmed by many, e.g. Muñoz Pastor & De Amo R. (1985: 102) in México. They wrote: “The intensity and quality of light is altered by the canopy of a tropical rain forest. The most important changes are the total transmittance of ultraviolet, blue and far red wavelengths.” In the undergrowth of the rain forest they found a red/far red ratio of 0.01 to 0.29, in thinned forest this was 0.5 and in secondary bush 0.51 to 0.66. Germination of pioneer tree seeds is triggered in the same Mexican forest by an increased ratio (Vazquez-Yanes & Orozco Segovia 1984).

The quality of light even permits to establish a diagnosis of the whole vegetation from optical satellite images. Reflectance by a forest cover in the near-infrared and visible red wavebands allows the calculation of a “Normalised Difference Vegetation Index” expressing variations in green leaf mass, a parameter closely related to above-ground biomass (Box & al. 1989 ex Van der Sande 1997). Stressing light colours are filtered and reflected by the canopy, a mirror image of the observations in the field cited above.

Follow-up experiments of our research on potato clones (Solanum tuberosum, see Ch. 2) showed that orthotrophic stem growth is triggered by increased red/far red ratios. In low ratios, axial growth is slowed down and the formation of reserve substances is boosted.

Finally, compound leaves show two neat organisation levels, that of the parts, leaflets and rachis, and that of the whole leaf. In some species of Meliaceae, the young apex of the rachis unrolls slowly, spreading one pair of leaflets after the other. This apex is not meristematic (Roux pers. com. 1981). Under stress the process stops. This is a direct, opportunistic, response by the individual tree, not linked to the genome by biological clocks or sequences. In the words of Crabbé (1987, p. 96, transl. RAAO): “From a morphogenetic viewpoint, leaf surface measuring rather is to be considered as evaluating leaf expansion”.

Indeed, adaptation rests on new growth patterns induced by the genome in organogenesis. The process in the above compound leaves is the unrolling of an organ already generated, not organogenesis. Organogenesis causes initiation of leaves by a meristem.
4.3.1.2 - Leaf formation as the terminus of the architectural diagram.

The notion of “throwaway branches”, coined by Givnish in 1978, rests on energy and nutrient economy in building compound leaves rather than leaf-bearing branches. Massive organs are apt investments for protracted functioning. Unpredictable stress makes heavy and durable organs into a liability rather than an asset, so lighter and shorter-lived axes are built. This is behind Givnish’s definition of a compound leaf. His concept is functional. Morphologically, however, a twig bears leaves with axillary meristems, whereas the rachis of a compound leaf bears leaflets without axillary appendices.

Givnish was not the only scientist comparing the two. Édelin (1977:62), in his “remarks on plagiotropy” also rather sees plagiotropy in a functional way. He regards it as a differentiation leading to “exploiting axes”, contrary to orthotropy which favours exploratory behaviour.

Hallé (1967) examining “phyllomorphic branches”, Bancelhon & al. (1963), Bancelhon (1965, 1969) or Roux (1968) studying plagiotropic morphogenesis, are among the many investigators of plagiotropy since centuries (e.g. Sattler 1982). However, the above authors rather studied the morphogenesis instead of the physiological economy of leaf-like organs.

Crabbé (1987, p. 97, transl. RAAO) expressed this perfectly: “All organs we have to study (internodia, leaves, inflorescences, fruits) are initiated during a mitotic phase within a meristematic zone before they start growing in the proper sense. Because these mitoses at the outset install a certain number of cells, they literally fix a growth potential of the organ, which is only limited by the possibilities of individual expansion of each of these cells”. This agrees with Nozeran’s summary of decades of experimental research in his laboratory, in terms of morphogenetic sequences (1986).

By definition, every lateral organ is initiated by an apical meristem one plastochrone after the preceding primordium. A plastochrone hence is a time unit, a kind of biological second. The mitotic process involved is influenced at every plastochrone by the results from all previous plastochrones. This interaction generates a differentiation sequence. The influence of the earlier organs arrives via the sapstream, a highly organised watery transport system carrying nutrients and phytohormones (cf. 4.2.1). It also carries a translation of the momentary stresses, if only because the sapstream concentration varies in various environments.

One more element needed to understand the rules of reaction to stress at the organ level is the architectural diagram (Édelin 1977: 84). He defined it as “the expression of the architectural model by any one species”. Édelin presents it as a table with as many columns as there are different types of axes in a branched array (“architectural model”). The definition of each axis, given in the rows of the table, is morphologically very precise, using more criteria than our figure 4-2. The order of the columns originally was the branching order in time (1977) until Édelin discovered intercalary branching (1984). Hence the diagram shows the apparent, spatial branching order in a larger tree when all types of axes are present.

In table 4-1 we summarise the architectural diagrams of 6 coniferous tree species after Édelin (1977). The table is representative for many other species, including hardwoods with monopodial branching and trees with plagiotropy (Édelin 1984, 1986,1991). We added two items to the differentiation sequences. If the last vegetative axis bore axillary inflorescences we added these as an extra branch order. All inflorescences are axes with strongly specialised appendages, but all have some kind of leaves. Flowers also are axes, contracted, specialised and often simplified (Corner 1964). In all cases we added, as a last order of “branching”, a
leaf with its axillary potential, abbreviated as leaf-plus. Sexualised leaves are part of the flower, so purists may add them two columns to the right of the “inflorescence” order.

The above defines the rules of adaptation at the organogenetic level in plants, explaining the sequential architectural dynamics by Hallé & Oldeman (1970). Stress factors are translated both in sapstrem impulses, and into electromagnetic signals to the leaf. In the differentiation steps of the initium and the primordium, mitoses occur in the subapical meristematic “initial ring”, induced by those stresses, transmitted by the intact plant. Somatic meiosis may then yield a specific lineage of daughter cells, following some mechanism described precisely in section 3.3. The leaf-plus formed under these conditions is adapted to the prevailing stress. Its axial products are determined by the rate of biomass formation, growth rate, and activation of certain resistance genes. Until today it is not known how one can see that such a gene is active. Quantitative markers found in somatic chloroplasts allowed to find the rules of intracellular adaptation (Sect. 3.2.2). Precise architectural markers in organs can not yet be linked up in a regular way to the behaviour of DNA or the activation of specific genes, except perhaps in the Mendelian way.

4.3.1.3 - Between the leaf-plus and the branched array

The summarised architectural diagrams in table 4-1, supplemented by data from the original sources, allow us to sketch a sequence of axial sequences. Together, these sequences interact to determine the body of the branched array. As usual in system analysis, this body is considered as the sum of its branches plus their interaction. The sequence of the array hence is not a mere summing up of the sequences of its parts. This is shown by the following list of gradients, the principle of which has been clearly announced by Hallé & Oldeman (1970).

<table>
<thead>
<tr>
<th>Attribute</th>
<th>axis 1</th>
<th>axis 2</th>
<th>axis 3</th>
<th>axis 4</th>
<th>axis 5</th>
<th>axis 6**</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mass</td>
<td>large</td>
<td>☐☐☐☐☐☐☐☐☐☐ intermediate ☐☐☐☐☐☐☐☐☐☐ tiny</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lifespan</td>
<td>long</td>
<td>☐☐☐☐☐☐☐☐☐☐ intermediate ☐☐☐☐☐☐☐☐☐☐ short</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cambium*)</td>
<td>essential ☐☐☐☐☐☐☐☐☐☐ useful ☐☐☐☐☐☐☐☐☐☐ vestigial ☐☐ none (leaf)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Internodes</td>
<td>numerous ☐☐☐☐☐☐☐☐☐☐ few ☐☐☐☐☐☐☐☐☐☐ transformed ☐☐ none (leaf)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Differentiation</td>
<td>weak ☐☐☐☐☐☐☐☐☐☐ clear ☐☐☐☐☐☐☐☐☐☐ strong ☐☐☐☐☐☐☐☐☐☐ strongest</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sexuality</td>
<td>none ☐☐☐☐☐☐☐☐☐☐ sometimes ☐☐☐☐☐☐☐☐☐☐ always ☐☐☐☐☐☐☐☐☐☐ none (leaf)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*) of course not in Monocots and Cryptogams

**) seventh axial sequence, which if it exists is either a leaf-plus or a sexualised leaf sequence, omitted

In their first book on tree architecture, Hallé & Oldeman (1970: 8) already implied, that not all axes are architectural in the strict sense of producing the framework of a tree. Only the early vegetative axes in the “ramified sequence” determine tree architecture. Indeed, these are the axes 1 to 3, perhaps 1 to 4 in the above list. They are progressively shorter-lived, less massive, hence less dependent on secondary thickening. They are vegetative in that they flower late in life if at all, but in plagiotropic axes sexuality comes earlier. The later axes are so short-lived,
so light and so specialised that they become mere appendages of the structural axes, at least from a strictly architectural viewpoint.

On the one hand, the presence of numerous or at least several leaf-plus complexes on every axis does not change this view fundamentally. It gives an orderly explanation of some traditionally intriguing phenomena, like caudiflory. This is the appearance of inflorescences and fruits “on the tree trunk”. We see it now as intercalation of an inflorescential axis 5 or 6 in between axis 1, the trunk, and axis 6 or 7, being a leaf on that trunk. However, the framework of the tree still is built by the earlier vegetative axes, in such perfect order that one can recognise and largely understand without great errors, for instance, an Indonesian meranti (Shorea sp., Dipterocarpaceae) or a Christmas tree (Picea abies) by its architecture.

On the other hand, adaptation to the forest, the natural environment of a tree, can not be explained by architecture alone. The collection of properties of a tree, by which it is adapted to its native forest, such as shade and drought tolerance, nutrient requirements or reproduction strategy is called temperament, an old European forester’s term. Oldeman & Van Dijk (1991) examined the temperaments of some tropical woody plants, the architecture of which was known. Except for architecture, temperament rests on leaf properties and sexual reproduction.

The sequence of sequences, the “branched morphogenetical sequence” of many authors, shows the links between the integration level of branched arrays and the organ level. Axial adaptation is a result of adaptive organogenesis, and results in a well adapted branched plant architecture in the broad sense.

4.3.1.4 - The role of axes in adaptation of the branched array

At the level of a meristem, every plastochrone interacts with the results of all previous plastochrones of that meristem. This interaction is often seen as a set of physiological correlations. Scaling of physiological correlations, for instance “...the closer the command is to the bud, the more stable it is...” (Champagnat & al. 1986: 298) fits in with the hierarchical systems’ approach used here. At the level of the axes in a branched array, correlations are among those axes, classically known as basitony versus acrotony, or apical dominance. During its growth, every axis is in interaction with all other axes in the same array.

This can lead to very regular, inherited plant architecture (cf. Fig.4-1). Such configurations were thoroughly studied by Hallé & Oldeman (1970), Oldeman (1974), Hallé & al. (1978), and others. Until today, however, no strict correspondence between vegetation type, plant architecture and temperament was found (e.g. Vester 1997). It is impossible to link precisely any particular branched array to any particular ecosystem. This has two reasons.

First, the properties of axes are subject to evolutionary change, operated by the stressed leaf-plus mechanism described above. Modification of the leaf-plus complex leads to formation of an axis deviating from the original sequence. This influences physiological correlations code-terminating axial polymorphism in the branched array. It also may influence ovuli formed by flowers issued from leaf axils on the modified axis and its laterals. The modified seeds hence transmit to the next generation the ability to react to stress in the same way. If the stress is recurrent or permanent, it acts as selection pressure and the ability turns into a dominant standard property. If the stress was incidental, the ability will remain recessive during some generations then to be sooner or later “overwritten” by other DNA-sequences.
The branched array only rarely shows abrupt architectural change. The structural axes to the left of the architectural diagram embody a long evolutionary history. This can not be easily overwritten in one fast set of new genetical instructions. The bigger these arrays, the longer it takes to build them, and the more stable the environmental conditions must be to permit this build-up to be completed. Giant Douglas firs of the NW United States (Pseudotsuga menziesii, see Kuiper 1988, 1994), nearly a hundred meters high and over a thousand years old, resist recurrent stresses like forest fires, protected by their thick bark. Their architecture remains true to a simple branched array up to a height of several tens of meters, so both the axes and the array may be considered to be perfectly adapted to the local ecosystems on the long term.

From 1970 on, Hallé & Oldeman emphasised that such inherited arrays (their “architectural models”) only could change “quantitatively, not qualitatively”. Nozeran (1986) added that the accumulation of quantitative change finally abuts at qualitative change. In table 4-1 this can be seen as the stepwise invasion of the massive, unwieldy axes at the right side by genetical change generated by the specialised axes and leaf-plus complexes at the right side.

Meanwhile, axes can strongly adapt to changing environments by quantitative mechanisms without disturbing the fundamental organisation of the array, its quality. These mechanisms have been discussed by Oldeman (1989a), Vester (1997) and Oldeman & Vester (in press). They are briefly sketched in the paragraphs below.

- a) Synchronisation and postponement
Most trees show a regular calendar of synchronous activities, some keyed to the seasons, like leaf fall or rhythmic growth. Flowering also may be simultaneous on all axes, like in the New Caledonian tree Cerberiopsis candelabrum (Apocynaceae; Veillon 1971). Flowering once is called hapaxanthy or monocarpy (see Corner 1964; Hallé & al. 1978). Well known instances are Corypha and Metroxylon palms, once flowering massively on their solitary axis before dying. In C. candelabrum, the whole branched array flowers at the same time. The array is vegetatively built in steps, one set of rhythmic, orthotropic axes after the other, but terminal flowering is simultaneous on all axes.

Another spectacular example is periodic flowering in bamboo’s. With intervals of decades, whole clones start flowering and do not cease until all leaf-plus complexes are used up and the whole clone dies. Corner (1964: 273) described this, adding that the huge mass of fruits feeds rats and mice whose numbers then explode, so that farmers about twice a century face both bamboo shortage and rodent pests. Historical documents over two millennia confirm this.

As shown earlier, growth rhythms are physiologically determined by the plants themselves. In species from regions with a winter or a pronounced dry season, however, the flushes are synchronised. Figure 4-7. shows how this is thought to work. Rhythmic growth is determined endogenously by physiological induction of a diapause in a vegetative meristem. Exogenous stress, conveyed directly or by some endogenous carrier like the sapstream, then provokes a quiescent period in rhythmic growth, which may be converted later into dormancy. Dormancy is self-maintained in the meristem and, to be lifted, requires specific external signals different from the mere return of previous growth conditions (Crabbé 1987).
<table>
<thead>
<tr>
<th>Species</th>
<th>axis 1</th>
<th>axis 2</th>
<th>axis 3</th>
<th>axis 4</th>
<th>axis 5</th>
<th>axis 6</th>
<th>axis 7</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Chamaecyparis lawsoniana</em></td>
<td>monopode, ortho, diffuse branching, indefinite growth, vegetative</td>
<td>monopode, ortho, diffuse branching, indefinite growth, vegetative</td>
<td>monopode, no precise orientation diffuse branching, definite growth, vegetative</td>
<td>monopode, no precise orientation diffuse branching, definite growth, vegeative</td>
<td>monopode, no precise orientation diffuse branch, definite vsh, deciduous female infl TERMINAL</td>
<td>monopode, no precise orientation diffuse branch, definite vsh, deciduous male infl TERMINAL</td>
<td>leaf +</td>
</tr>
<tr>
<td><em>Cunninghamia lanceolata</em> RAUH's model</td>
<td>monopode, rhythmic, ortho, indefinite growth, narrow leaf vegetative</td>
<td>monopode, rhythmic, ortho, indefinite growth, broad leaf vegetative</td>
<td>monopode, rhythmic, no precise orientation def. growth, decid, broad leaf, female infl TERMINAL</td>
<td>monopode, rhythmic, no precise orientation def. growth, decid, broad leaf, male infl. AXILLARY+TERM</td>
<td>monopode, ortho, diff. branch, def. vsh, deciduous male infl TERMINAL</td>
<td>monopode, ortho, diff. branch, def. vsh, deciduous male infl TERMINAL</td>
<td>leaf +</td>
</tr>
<tr>
<td><em>Cupressus sempervirens</em> ATTIMS' model</td>
<td>monopode, ortho, diff. branching, indefinite growth, vegetative</td>
<td>monopode, ortho, diff. branch, definite growth, vegetative</td>
<td>monopode, ortho, diff. branch, definite growth, vegetative</td>
<td>monopode, ortho, diff. branch, def. in vsh, deciduous female infl TERM</td>
<td>monopode, ortho, diff. branch, def. in vsh, deciduous female infl TERM</td>
<td>monopode, ortho, diff. branch, def. vsh, deciduous female infl TERM</td>
<td>leaf +</td>
</tr>
<tr>
<td><em>Metasequoia glyptostroboides</em> RAUH's model</td>
<td>monopode, ortho, rhythmic, indefinite growth, vegetative</td>
<td>monopode, ortho, rhythmic, indefinite growth, vegetative</td>
<td>monopode, no precise orientation rhythmic, definite growth, vegetative</td>
<td>monopode, no precise orientation rhythmic, definite growth, vegetative</td>
<td>monopode, rhythmic, no precise orientation definition gr. decidu, broad leaf, female infl. TERMINAL</td>
<td>VERY SPECIALIZED SHORT SHOOTS on all other axes (female, male, photosynth)</td>
<td>leaf +</td>
</tr>
<tr>
<td><em>Picea abies</em> RAUH's model (Figs.4-13,4-15)</td>
<td>monopode, ortho, rhythmic, indefinite growth, vegetative</td>
<td>monopode, ortho, rhythmic, indefinite growth, vegetative</td>
<td>monopode, ortho, definite growth, shs. female infl. TERMINAL</td>
<td>monopode, ortho, definite growth, shs. female infl. TERMINAL</td>
<td>monopode, ortho, definite growth, shs. short shoots/ male</td>
<td>monopode, ortho, definite growth, shs. short shoots/ male</td>
<td>leaf +</td>
</tr>
<tr>
<td><em>Pinus sylvestris</em> RAUH's m'l (4-16) like <em>Picea abies</em> + shortshoots</td>
<td>like <em>Picea abies</em> + shortshoots</td>
<td>mono, ortho, definite growth, shs. short /female</td>
<td>mono, ortho, definite growth, shs. short shoots/ male</td>
<td>mono, ortho, definite growth, shs. short shoots/ male</td>
<td>mono, ortho, definite growth, shs. short shoots/ male</td>
<td>mono, ortho, definite growth, shs. short shoots/ male</td>
<td>leaf +</td>
</tr>
<tr>
<td><em>Thuja plicata</em> ATTIMS' model</td>
<td>monopode, ortho, diffuse branching, indefinite growth vegetative</td>
<td>monopode, ortho, diffuse branching, indefinite growth vegetative</td>
<td>monopode, ortho, diffuse branching, definite growth vegetative</td>
<td>monopode, ortho, diffuse branching, definite growth vegetative</td>
<td>monopode, no precise orientation diff. branch, def. in vsh, deciduous fem.infl TERMINAL</td>
<td>monopode, no precise orientation diff. branch, def. in vsh, deciduous female infl TERMINAL</td>
<td>leaf +</td>
</tr>
</tbody>
</table>
Now the architectural diagrams given above allow us to understand the synchronisation of specific meristematic jobs over the whole plant body to be linked to all leaf-plus complexes present. They can be simultaneously activated either by a specific state of the whole plant, or by a direct incentive from its immediate environment, or by both interacting. The fuzzy character so induced in periodic or mass flowering is illustrated well by the case history of the Central American dry forest tree *Ateleia herbert-smithii* (Leguminosae) by Janzen (1989:292). He observed its behaviour during eleven years and states: “Flowering and fruiting was synchronised within the population at two-year intervals from 1974 to 1984, but this perfect synchrony was disrupted by and odd-year and population-wide flowering in 1985”.

Exogenous incentives vary. Non-lethal drought is always linked to chemical factors, as it influences concentration and speed of the sapstream. Light has three impacts. Light intensity codetermines photosynthetic production and so influences the chemical load of the sapstream. Photosynthesis puts photosynthates into the sapstream and uses its outputs of rhizosynthates and water. Daylength is a signal of season, synchronising photoperiodic behaviour of plants, e.g. autumnal leaf fall. The light spectrum is a signal we met earlier in connection with the synodic lunar cycle (Sect. 3.2.1) and as a determinant of the forest microclimate, influencing seed germination. Since the 1950’s the existence of photoreceptors in the far red in seeds of several trees, keying their germination is known (e.g. Vazquez-Yanes 1976).

Average heat over longer periods, or acute heat waves, also are important. The parameter of heat is temperature. Heat too is a stress factor synchronising seasonal behaviour in many climates. Like drought, it has lethal potential. Classical plant physiology lets gravity guide the spatial orientation of axes, plagiotropic or orthotropic, but not the axial sequences in time.

However, tides are due to the interaction between the moon’s gravity and that of the earth, so they certainly induce biological tides in the sapstream (see 4.2.2). Atmospheric pressure and humidity and vibrations due to earth shocks or earthquakes are incidental, not involved in synchronisation of plant growth. However, they can be a starter pistol synchronising animal behaviour, for instance flight.

Synchronisation of meristematic building of either flowers or shoots ensures an ongoing adaptation of axes, both to their “inner environment” of a branched array and their “outer environment” closely surrounding the plant. Chapter 3 showed how fast mobilisation of genes can take place, dictating the nature of mitotic daughter cell lineage’s in meristems. The trigger of the mobilisation of the new potential at least has two sources. The leaves transform light signals, and the sapstream transforms hydrochemical signals into physiological drivers. Depending on the architectural diagram, in particular on the differentiation of axes 3 to 5 to the right, specific meristems then starts behaving in the same way at the same time. Building leaf abscission layers may be seen as a very specialised meristematic differentiation.

This highly organised behaviour of axes within branched arrays under stress is to be contrasted with the havoc played by *in vitro* culture upon such organisation.

Synchronised flushing of leafless plants after a leafless stress period, in springtime or after a dry season shows an unusual feature. The awakening meristems have no regulating leaves. These were shed in the season before. In most cases there is no total absence, because there are buds. These yield proleptic axes, defined by a development after resting. The new axis has scale leaves and very reduced internodes at its base (Hallé & al. 1978). The bud hence contained many specialised leaf-plus complexes with leaves reduced to scales.
Scales mostly are not photosynthetic. Sugars needed for flowering or axis building are delivered by the sapstream from reserve stocks. The role of scale leaves regulating their axillary meristems should be closely inspected, also because they are laterals on axillary meristems having lost their original leaf. One might speak of leaf-minus situations. The anatomical structure of adventitious meristems seemingly without leaves or scales should be examined too.

As a contrast to synchronisation, there are postponed growth or abscission dynamics.

Woody Leguminosae provide many examples of postponed cambial activity and postponed flowering (Oldeman 1989a). When the larger axes, at the left hand side of the architectural diagram, postpone cambial thickening they remain slender. Extension makes them lianescent. This is shown by genera like Bauhinia, Pterocarpus or Mimosa (Leguminosae), the famous tropical apocynaceous garden plant Allamanda cathartica. Another instance is a liane, Paulinia cupana (Sapindaceae, Fig.4-9), the fruits of which are the base of Brazil’s most popular softdrink Guaraná. These plants show a sympodial branched array, composed by axes which would be mixed plagiotropic or mixed orthotropic in trees (see Fig. 4.2).

Here, an intimate mix of endogenous and exogenous regulation occurs. Under different kinds of stress, many of the plants mentioned form either a small tree, a shrub, or a liane (Oldeman 1989a). In cultivated plants this property is used for pruning, particularly to keep plants bushy so as to concentrate fruits or flowers in small, convenient volumes. In nature postponement of secondary thickening is clearly induced by environmental factors. For instance, Pterocarpus officinalis (Leguminosae-Papilionoideae, local name montouchi) is a big Guinean riverside tree with spectacular buttresses It had a fully-fledged architecture, but a shrubby physiognomy in the brackish water of the estuary of the Rivière de Cayenne. According to Stiriton (pers. com. ex Oldeman 1989a), Spirotropis longifolia (Leguminosae) produces both lianas and trees, and in a Kew greenhouse the legume Xanthocercis zambesiaca was experimentally transformed from a tree into a liana by pruning back the main axis at two years of age.

In all arrays permitting individuals of a same species to behave facultatively as trees, shrubs, or climbers, all axes are normal members of one of the branching orders of the architectural diagram. They were built by a leaf-plus in a well defined spot in the array. Postponement of secondary thickening means three things. First, the morphogenetic potential of the meristem is very flexible, pointing to stress behaviour of its ring-shaped initial zone just below the tip. Second, the leaf soon loses its physiological grip on cambial activity. Third, axes not being self-supporting because of postponed thickening can not be trunks of treelike core arrays.

This behaviour is very common in weeping trees, where it is regulated by the leaf-plus as an inherited standard process. Weeping willows (Salix vitellina var. pendula; S. babylonica), beeches (Fagus sylvatica var. pendula Fig. 4-10) and ashes (Fraxinus excelsior cv."Pendula") provide classical examples. These trees are sympodial multi-arrays (Fig. 4-4), the components of which are core arrays in which only a small basal portion of the central axis has thickened soon and fast enough to become a trunk segment of the whole plant organism.

Sites and circumstances around lianescent and weeping behaviour suggest that the original adaptation addressed drought stress. This is supported by the behaviour of trees growing in a heavily polluted atmosphere, like the spruces, silver firs (Fig. 4-14C,D) and beeches observed by one of us (RAAO) in the Black Forest in 1984. Acid and other toxic rain upsets soil life.
Figure 4-9: *Paullinia cupana* (Sapindaceae), a liane (grapnel lower left, seeds upper left), source of Guaraná, the most popular softdrink of Brasil. To cultivate this liane easier, postponed secondary thickening is manipulated by pruning. The plant becomes a shrub (upper right). Brasil, Una, June 1985. Photographs R.A.A. Oldeman.
Figure 4-10 Weeping beech. *Fagus sylvatica* var. *pendula* (Fagaceae), Wageningen, 1978. Note multi-array built by reiteration of simple array. The simple array is built by plagio-plagio mixed axes (Fig. 4-2). These axes have postponed thickening, so they remain very slender and hang down. Photograph R.A.A. Oldeman.
Figure 4-11: Precocious flowering or neoteny. *Dipterocarpus hasseltii* (Dipterocarpaceae) from Kalimantan, Indonesia, grown in a greenhouse, 1988 (also see Smits 1994). The seedling axis behaves like an axis of the 4th or 5th order, not a trunk, but is orthotropic like a trunk. Unknown stress causing this extreme form of intercalation. Photographs Guy Ackermans.
It particularly hinders the roots hosting mycorrhizal fungi, responsible for large parts of water absorption by tree roots. Trees with a shortage of such symbionts suffer internal drought.

The whole image of such behaviour is that of circumstantial or major environmental stress inducing the setting up of a programmed response expressed morpho-physiologically at the levels of organs, axes or arrays.

In the stressed trees, this is observed as postponement of secondary thickening in the axes of the second and third orders, accompanying the arrest of apical growth of the primary axis. The cambium is fed by the sapstream, so it is particularly hit by drought. This is compounded by the plant giving priority to the feeding of meristems over cambium. Meristems indeed are vital, the formation of new leaf-plus complexes being essential for survival. New leaves of course are crucial to refuel photosynthesis, diminished by precocious leaf abscission due to chemical stress. However, new lateral meristems also mobilise genetic resistance by somatic meiosis (Sect. 3.4). The latter aspect, being a programmed response, could not be perceived before chapter 3 was written.

Silver fir (*Abies alba*, Fig.4-14) shows the architectural result. Trunk development (axis 1) is arrested. Plagiotropic branches (axis 2) are slender and very prolonged, with young needles clumped at their ends. This stress architecture is known among foresters as a “stork’s nest” (Fig. 4-14C; Schütt in Hatzfeldt 1982). If atmospheric stress persists, the next reaction is abundant reiteration (Fig. 4-14D), as discussed in a later section. Other tree species under air pollution stress are illustrated in Oldeman (1990, his Fig. 3.33).

Postponed flowering contrasts with postponed secondary thickening. Architectural dynamics of thick axes emphasise the orders to the left (axes 1 to 3 or 4). Only after the build-up of a big vegetative body the leaf-plus complexes start intercalating inflorescences. This usually results in large plants flowering late in life, such as big trees or large woody lianes which do not flower before arriving in the forest canopy. Big lianes then combine postponed thickening and postponed flowering. This often is an adaptation to high stress in the juvenile stage, such as shade and/or drought due to root crowding in the forest undergrowth.

Advanced flowering is more common. Early flowering is the differentiation of inflorescences by leaf-plus complexes on axes to the left of the diagram. An extreme example is neotenic terminal flowering on seedlings, reported by Scarrone in Mango (*Mangifera indica*) and observed by one of us (RAAO) in a seedling of *Dipterocarpus hasseltii* Bl. (of the Smits-Schalk-Oldeman Indonesian dipterocarp collection in Wageningen; Fig. 4-11; also see Smits 1994). In a natural SE Asian forest, such seedlings are quickly outshaded, so neoteny is lethal.

Indeed there also is a clear tendency in tree populations under pollution stress to flower early, to build smaller but more numerous organisms, and to die early. This favours population build-up rather than individual build-up. An example are the populations of Douglas fir (*Pseudotsuga menziesii*, Pinaceae, NW America) planted in Europe, subjected to atmospheric pollution. We observed these stressed trees in the Netherlands on sandy soils with low water retention. The firs are small-sized and look senescent at an early age. They engender huge populations of seedlings, “like hairs on a dog” as the saying goes among foresters.

A completely different example of size differences in the same organism, linked to stress and function is *Entamoeba histolytica*, a frequent commensal unicellular organism in mammal intestines. This omnipresent component of the intestinal flora is the small form, *E. histolytica* f. *minuta*. Under stress, e.g. excessive dosages of alcohol or antibiotics, the population starts to show more and more large variants, *E. histolytica* f. *hystolitica*, a pathogen causing the
Figure 4-12: Multi-array, distributing sapstream to countless tiny arrays. *Parinari excelsa* (CHRYSOBALANACEAE), ca 45 m high, Piste de St Elie, Guyane Française, 1989. Every array built by mixed plagio/plagio axes (Fig. 4-2), superrows are non-synchronous multi-arrays; i.e. some have young leaves, some old leaves, some are leafless. Photograph R.A.A. Oldeman.
Figure 4-13: A - *Pinus cembra* (Pinaceae). Intercalated reiteration under heavy climatic stress in high mountains. Reiteration of completely orthotropic, rhythmic branched arrays originating from dormant (or adventitious?) meristems, turning back to the start of the branching sequence, an order 1 axis. B - *Picea abies* (Pinaceae). Young, dense plantation for wood production. Note different sizes of saplings (pocket knife ca 8 cm long). Delvaux claimed that the highest ones often maintain their advance over the whole life cycle. Austria, 1977. Photographs R.A.A. Oldeman.
terrible disease of amoebiasis. We assume that the stresses mentioned upset the whole intestinal ecosystem (cf. Ch. 5.1) which in its turn causes unbalanced food conditions for *E. hystolitica.*

- **b) Playing the numbers**

In the third chapter the adjustment to stress by virtue of lineages of mutant meristematic cells was explained. They provide the organism with the capability of fast, *direct response* to stress by mobilising resistance genes. Moreover, they sometimes guide a more far-reaching change in plant building dynamics, so that new architectural properties are acquired as *second- or third-line responses.* As shown, transmission of the effects of genetic mutations to organs, axes and branched arrays is operated by mitotic initial rings with cells capable of somatic meiosis under stress and playing a key role in building differentiated meristems.

We also saw how growth phenomena at the levels of organogenesis, axial development and the building of branched arrays can be synchronised, delayed or advanced in time, with spectacular consequences as to the adaptation of their architecture and dynamics.

In the present section, a third property is quickly regarded. It was not thoroughly researched as far as we know. It concerns *numbers* of organs in axes and numbers of axes in arrays.

The number of needles and leaves on a branch is the balance between the number of leaves shed and the number of leaves formed. The rate of leaf formation is not constant. It oscillates regularly in axes with rhythmic growth, and irregularly if adjusting to unpredictable impacts from outside. Stress often causes reduced leaf numbers per axis or per plant crown. Estimates of “leaf occupancy” often were used as routine indicators of forest vitality, e.g. during the great dread of acid rain in the early ‘eighties (cf. Bauer 1985:122-160).

Indeed, if the number of new *leaf-plus* complexes formed lags behind the number of stressed leaves shed, both photosynthesis and genetic flexibility of adaptation decrease, because the probability of potentially “other” meristems being formed decreases too. Figure 4-14C not only shows axial adjustment by *ad hoc* postponement of secondary thickening in stressed *Abies alba,* but also allows to assess needle numbers by ranking them in classes from abundant to absent. Early and fast functioning of the apical meristem of the axis, indispensable to replace the needles as quickly as possible, is hardly explained without instructions associated with B.DNA heteroduplex setting during somatic meiosis in the meristematic region.

Except for decreased leaf numbers, branched arrays under stress often show a diminished number of axes per branch group or tier. This was observed but not studied. In most rhythmic arrays, tiers contain a standard number of axes, five in many tree taxa (e.g. Myristicaceae, Gymnosperm genera *Picea, Pinus* or *Pseudotsuga*). In others, there are three (e.g. the genus *Cordia* and some euphorbs). Pauperate architectural forms (see Hallé & al. 1978), caused by stress, usually show incomplete tiers. The same phenomenon occurs in dwarfed arrays.

Axial numbers in branching patterns can also diminish in a more diffuse way. Continuously branching arrays are defined by a regular branch spiral following the leaf spiral, because the meristem of every *leaf-plus* complex becomes immediately active. It is *sylleptic,* which is defined as functioning without any preceding resting period. Stressed conditions first become apparent by divergent size patterns among branches that usually have a highly standardised development (Fig. 4-1). Small and large branches are mixed without regularity. Stronger
stress causes some axillary meristems not to develop at all. Irregular lacunae appear in the branch spiral. Finally, the plant is barely branched if at all.

Unbranched pines, called foxtails by foresters (e.g. Hallé & Oldeman 1970, their Fig. 77), are often mentioned as unwanted monstrosities in plantations. Forestry literature ascribes them to genetic deviations, the innate growth and branching rhythms being out of tune with seasonal periodicity. This explanation rests upon the exotic origin of many if not most planted pines, dragged from one part of the world to the other. The phenomenon deserves deeper study.

4.3.1.5 - The branched array: gigantism, dwarfing and reorganisation

Expansion or reduction in size of a plant body leads to either populations of small organisms or to single, large organisms. “Miniaturisation” leads to herbaceous life say Hallé & Oldeman (1970) and Hallé & al. (1978). Indeed, in the above list of sequences, secondary thickening in herbs is delayed ad infinitum, even in first order axes, and flowering is advanced by early intercalation of inflorescences between leaves and early axis. The ultimate dwarf form shows neoteny (Fig. 4-11). If neoteny becomes normal, the species has become herbaceous, with numerous, small individuals without secondary thickening, richly flowering and fruiting.

In 1986, Hallé pondered upon dwarf forms growing together in one organism, “repeated” over and over again from vegetative meristems. He wrote about what we call “branched arrays” in the present book. If these become giant, they build trees conforming to one of Hallé & Oldeman’s “tree models” (1970) over most of their individual life cycles, i.e. with a standard build-up of branched arrays. This he called gigantism. If standard arrays become smaller and are formed time and again by vegetative meristems, they form a stack, like the colony-tree of earlier authors comparing trees to corals: “A leafy wood, a grassy sward, a piece of sponge, a reef of coral, are all instances of a like phenomenon” (D’Arcy Thompson 1917, ex Bonner 1961:35). Hallé (1986) called the stacking of dwarf arrays in one plant repetition.

Both strategies, bound to similar architecture of different size, have no direct counterpart at the level of the double helix. As shown in chapter 3, there are size differences among B.DNA, longest and most slender, A.DNA, shortest and stoutest, and Z.DNA longest and most slender of all, but turning to the left. This does not correspond, however, to size differences in whole plant architecture, which may differ an order of 10^4 as between a tiny herb of 1 cm and a Californian Redwood tree of 100 m high. This is so at the level of organs, axes and arrays too, excluding microscopical plants. However, the repetition of “standard” patterns of course is a point we found all organisation levels to have in common, even if relative size ranges do not correspond.

• a) The role of the branched array in adaptation of whole plants

The trick of the tree is to make available the right branched array at the right moment in a period with a given type of stress. This is so from the system level of the whole tree to that of cell nuclei involved in building one of the tree’s meristems, where the right gene is provided. How do the many arrays making a tree work together? This is outlined in the alineas below, with our excuses to all authors we could not cite in this limited context.

Édeline’s architectural diagram links axial and organogenetic sequences to sequences building branched arrays. The sequence of sequences, explained above (Par. 4.3.1.3), links sequences of branched arrays to sequences building whole multi-array trees, the arrays being building blocks. Figure 4.4 shows how branched arrays aggregate into multi-arrays, an oval shape
depicting a branched array. The former expansion of arrays with one surviving axis is shown by stippled contours. The trees drawn represent successive views of tree development.

Hallé & Oldeman’s tree (1970; Hallé & al. 1978) is a mono-array (Fig. 4-4K, to the right). It has a very regular growth pattern, named “architectural model”, and is assumed to be adapted to “ideal” conditions with “optimally” balanced growth factors.

Oldeman’s tree (1974) grows in the far from ideal tropical rain forest (Fig. 4-4K left), and is a pile of recurrent Hallé & Oldeman sequences. Oldeman (1974) coined the name reiteration for this recurrent process, i.e. “following again (re) the path (iter) of the differentiation sequence”. Reiteration is stepwise downsized, treelike at the tree base to herblike in the crown, the sapstream having to feed ever more arrays. The myriads of arrays at the crown edge are tiny and stressed because they nearly starve, their portion of sap becoming marginal.

Édelin’s tree (1977; Fig. 4-4L right) has intercalary branching (Fig. 4-8). This explains downsized and partial reiteration patterns. However, the importance of the arrays, particularly the early, large ones, is reduced. Édelin emphasises regular teamwork by many arrays building a big tree, programmed perhaps, but not so by the straightforward differentiation sequences of Hallé & al. (1978 - Fig. 4-4K, both cases). More studies originated from the tropical forest canopy campaigns with Hallé’s “canopy raft” lowered on the giant tree crowns by an outsized balloon (Hallé & Blanc 1990).

Blanc’s tree (1989) is an unruly mob of small axes, fragments or paupered forms of regular axes, and displaying barely any branching regularity (Fig. 4-4L right of center). The tree of Édelin & Blanc (Fig. 4-4L extreme right) shows the unifying concept of hierarchic versus polyarchic tree patterns Édelin (1991). Polyarchic trees resemble loose populations of arrays rather than a closely knitted organism. Torquebiau (1979) wrote on “a demographic approach to the tree”, including selection pressure on reiterated arrays.

Each of these images carries part of the truth, and none carries the whole truth. They range from perfect organisation, like Hallé & Oldeman’s tree, also assumed by other authors, to the acme of chaos, such as the tree considered by Blanc, with Édelin and others in the middle.

Anyhow, in nice conditions a tree clearly expresses its nature by building a large-sized organism. However, in the usual stress of forest or human landscape, a long-lived tree applies small “packages of organisation” in order to use ephemeral resources for survival. Édelin (1991) associates strongly regulated growth with rapid but vulnerable development to occupancy space, and loosely organised growth with survival in an occupied space, facing successive stress periods successfully.

We saw how the architectural diagram, completed with the concept of the leaf-plus, explains the mechanism by which the right kind of axis is initiated and energised inside a branched array. Both sequential dynamics and intercalation play their part at all levels ranging from the DNA to the branched array in a tree. Is this plausible also at the system level of multi-arrays?

The list below remains quite speculative, because no data prove it formally. Nobody counted the orders of branched arrays or architectural models which a single tree can form. Oldeman (1974:86) assessed the number of “trunks” of large and small reiterated arrays against height in rain forest. This shows relatively few big arrays and countless minute ones at specific levels in a mature forest patch. Our hypothesis is that the inherited pattern has discrete versions, from a maximal expression of the branched array, through 4 intermediary orders (2, 3, 4 and
5) to its minimum expression. The minimum expression would be one internode with one leaf
and two meristems, i.e the end meristem and the meristem in the sole leaf axil.

However, the real number of reiteration orders is not essential. We may suspect that perhaps it
conforms to the numbers 5, 6 and 7 found at well studied levels. However, it is much more
important that the present list demonstrates a tendency. The number of meristems without a
surviving leaf increases automatically and inescapably. After the timespan needed to build a
tree with three reiteration orders, all surviving meristems on the old arrays are either dormant
or adventitious, and none has its leaf left. Resting meristems are important in all long-lived
plants and their equivalent in long-lived animals would be an interesting item to discover.

We saw that the leaf-plus complex unites a source of genetic activity, the meristem, and an
energiser, the leaf. In purely sequential branching inside an array, even if interrupted by
seasonal stress effects, the absence of the leaf is compensated fast. However, in dormant or
adventitious meristems, no leaf is present any more. These meristems have access only to
leaves if generating leaves themselves. Their first mobilisation hence is a low-energy leaf-
minus variant, fed only with starch from reserve stocks, without their own generator-leaf.

Therefore low-energy adaptation is particularly probable in multi-arrays. Juvenile plants most
commonly only face a challenge to occupy a forest volume offering all facilities for survival,
relatively poor in stress or at least rather constant. In older age, a contrast develops between
the ageing crown above and the oldest axes below (Fig. 4-14) In the crown, the endogenous
energy level is low, due to countless axes sharing the one sapstream (Fig. 4-12), but there are
leaves, so leaf-plus are available. In the leafless parts of the lower crown, and on the trunk,
the situation is leaf-minus. The minimum array of the hypothetical “seventh order” is
expected to be most often intercalated between the leaf-plus and whatever axis it grows on.
Even unbranched arrays, like the papaya tree, may so reiterate under stress (Fig. 4-6A).

The minimum axis has only few options. All show a strong genetic bias. Its meristems may
either become both vegetative, or both floral, or one may become vegetative and the other
floral. Because of the frugal sapstream, it is improbable that either meristem forms more than
one leaf-plus, but also that none forms a leaf. In any case there is energy to boost mutant

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*) of course not in Monocots and Cryptogams

**) seventh array reduced to one axis without branching potential, perhaps a complex of one or a few leaf-plus
or sexualised leaves? Hypothetical, particularly because the number of array orders never was studied.
meristems. Moreover, variation is broadened because both sexual and vegetative variation occur. A great genetic variability hence is to be expected in the crowns of giant forest trees.

This is illustrated by the medium-sized Guyanese forest tree *Mabea piriri* (Euphorbiaceae, Oldeman 1968). Its *trunk* is orthotropic. The *seedlings*, however, do not have an orthotropic but a plagiotropic seedling axis. Later, an orthotropic axis growing from an axillary meristem at the base of the horizontal seedling becomes the trunk of the young tree. The flowers sit terminally on strongly plagiotropic axes (Fig. 4-2). The differentiation of the branch hence persists in the seed and in the seedling. Oldeman (1968) postulated a time lag between the start of the life cycle and the differentiation sequence. This case also shows the reality of “vertical” genetic transfer (Ch. 6) of remnant axial differentiation to the next generation.

European beeches under acid rain stress may produce *juvenile*, toothed leaves in the crown periphery after premature yellowing and abscission of the leaves of the season (Schütt in Hatzfeldt 1982: 71). Here, the start of the new seedling cycle lags behind a juvenile symptom.

These examples shed new light on the old issue of “sexual transmission of acquired traits” to offspring. Genetically fixed characters like branch plagiotropy in *old* trees survive in *young* seedlings, or *juvenile* characters like toothed leaves appear in *old* trees. Both help survive. Plagiotropic axes are excellent captors of light in sun spots on the forest floor. Juvenile leaves are excellent photosynthesis boosters. However, the moment of “acquisition” and the moment of “transmission” show a time lag and are situated in different generational life cycles.

Foresters (Heybroek 1985) know that, even within monospecific seedling populations, there is a broad gamut of inherited properties. Lavender & Newton (1985: 219) write: “...that variable stock makes efficient selection of site, site preparation method or planting method difficult or impossible”. Significantly, seedlings overtopping other seedlings in a nursery or a forest generally conserve their lead as long as they live (Fig. 4-13B; Delvaux 1964, 1966, 1975). A literature review by Lavender & Newton (1985: 218) shows that survival is often linked to either *extra large* size of the surviving seedlings....but also to *extra small* size.

In nurseries of Douglas fir (*Pseudotsuga menziesii*), the isozyme spectra of spontaneously germinated seedling populations from the forest and seedling populations sown in the nursery were found to be quite different (ex Beuker 1988). In the forest, the ratio between red and far red light certainly differs strongly from that in the nursery (Vazquez-Yanes & Orozco-Segovia 1984). High mortality is common in young plantation trees after transplantation in the field. Their adaptive package can not face the change in conditions from nursery to plantation.

**Young parts of old tree crowns are sites of strongly boosted genotypical variation**, transmitted by seeds and possibly reinforced by somatic meiosis in seedling meristems. Adaptation to the precise site of germination occurs by selection among these highly varying genotypes. Freak forms like the neotenic dipterocarp (Fig. 4-11) are weeded out.

On the surviving parts of *older* arrays, meristems may awaken or be formed adventitiously after a long time. This is shown by a reiterated array on a trunk segment cut from a big *Dipteryx odorata* (Fig. 4-14B), blown over by a storm in Bogor’s great Kebun Raya (King’s Garden). Formation and building of adventitious meristems like the one at the origin of this reiteration, are both energised exclusively by residual starch from the tissues of the trunk fragment. In living trees, adventitious or awakening meristems may drain the sapstream. Such meristems are exactly in the right position, with their base in the cambium in between the ascending and the descending sapstreams, which feed the cambium too.
Meristems without their own energy supply, exposed to far red light in the deep shade under the forest (Sect. 3.2.1), would somewhat resemble cell masses in vitro. One would expect to find more stress DNA forms, more unadapted, random morphogenetic variation and broader variation in both axial differentiation and organisation patterns of the branched arrays. Indeed, it is well known among plant taxonomists that tree species should never be described after adventitious shoots collected near tree bases. Such “suckers” usually display a broad range of morphological shapes, far beyond the range characterising the species.

Old woody parts of trees are sources of weakly boosted phenotypical variation. Quite often, the reiterated arrays are of a low order, often as low as the seedling array. In that case a full tree is reiterated from the tree base.

Arrays of different orders so have different adaptive functions. Differences are emphasised with age in the surviving parts of older arrays. However, intercalation remains possible. A spectacular case was recently described by Sanoja (1992) in Qualea species (Vochysiaceae), big late pioneer trees of the Guyanas. In trees of more than 30 m high, dieback of the enormous crowns is followed by intercalation of low-order (“trunk”) arrays at the lowest branching fork. They reconstitute the whole crown, attaining sizes up to 15 meters high.

These spectacular reconstitution’s of whole trees are of course well known from coppice, as in the Castanea sativa plantations (Chestnut) in the Cévennes, or European oaks (Quercus sp. pl.; Fig. 4-14A), or eucalypts, or wattles (Acacia sp.pl.) of the tropics. Once more there is a time lag involved. It is used in coppice silviculture for economical reasons. In the wild it has the potential to bridge periods between the occurrence of different kinds of stress. The capacity to reiterate arrays hence exists and is hereditary, but its expression is individual. It allows plants to use an opportunistic mechanism to face one stress after another, perhaps different, one in the course of their life. The morphogenetic mechanism is called adjustment. It does not involve hereditary change and can be completely explained by reiteration.

On the contrary, hereditary change is involved in adaptation (e.g. see Hallé & al. 1978; Oldeman 1990). The two can not be strictly separated, because meristems are involved. The case of the European chestnut indeed has an extra complication. During the second World War, chestnut blight was imported from the New World, a fungal disease caused by Endothia parasitica, which had already infected all chestnut trees (Castanea dentata) in the Eastern US. Many survived by basal reiteration, but before flowering age the reiterated arrays were killed again by the fungus. The American species now only exists as a low, thin coppice.

The imported fungus also wrought havoc in the chestnut plantations in the Mediterranean. The trees in the Cévennes, at more than 600 m above sea level, were saved from destruction because their reiterated stems survived and thickened again. Their survival is fully explained if we assume that the following processes have occurred.

First, the chestnuts were routinely coppiced in the past to boost commercial fruit production. Hence trees with a high reiteration capacity were selected by farmers for economic reasons. Second, abundant reiteration raises the chances of somatic meiosis occurring in meristems, particularly first-order meristems near the ground. Hence mutated founder cells probably yielded some lines of daughter cells carrying a resistance transposon against blight. Contrarily to the American case, the reiterated arrays in C. sativa could reach flowering age, being indeed selected for fruit production. Blight resistance could therefore invade the chestnut population gradually, by reaching some seeds and spreading in the Mendelian way.
Individual survival by reiteration is called adjustment, invasion by hereditary resistance is adaptation. Both co-operated in the European case. In the American case adaptation was neutralised.

The mono-array and different kinds of loose and controlled multi-arrays now can all be understood as versions of reiteration of the same patterns, the right kind of array being intercalated in between the minimal array and the first-order array as required by the prevailing environmental conditions. Arrays indeed are both highly adjusting and adaptive in the life of plants, each order of array having a specific adaptive job to do.

- **Reiteration and organism: treelike, shrublike, herblike**
The array orders in multi-arrays are based upon Oldeman’s description (1974) of “reiteration waves” in trees. He found successive treelike, shrublike and herblike waves of reiteration (Fig. 4-4K). In his text, he sometimes inserted transition classes in between the other three, e.g. “like a treelet” or “like a bush”. The words “tree”, “shrub” and “herb” from colloquial English, like their translations in other languages, have no sharp definition. This and the fact that intermediate classes are necessary, points to the fuzzy character of these classes, which do allow one to follow and assess the process in terms that make sense.

The number of classes with intermediaries is some five or six at most. Further detail serves no useful purpose. This empirical classification indeed points to a limited number of array orders in a multi-array. Detailed descriptions of arrays are still needed, like those made by Edelin (1977) for axes. However, “reiteration waves” give insight in tree development under stress.

Except for their lacking roots, reiterated arrays behave biologically like their individual plant analogues. Treelike arrays produce much wood and few leaves, so that the leaf/wood ratio is low. Only rare, late flowers appear at its edge. On the contrary, herblike arrays produce nearly no wood and ample leaf mass, so leaf/wood ratio is high. They flower and fruit abundantly. If they had roots, they would spend most energy on sexual multiplication by flowering.

These facts once more confront “multi-array” with “population”. This option exists at every system level. A gene is a viruslike “reiteration”. A cell is a microbelike reiteration. A tissue may be a funguslike reiteration. Viruses, microbes and fungi are free living organisms having much in common with their counterparts bound in larger organisms.

This provides an important element for ecosystem analysis (Oldeman 1990). For instance, “the interaction between a bacterium and a leaf” is operational at the cell level, not at the leaf level. It is an interaction between a bacterium and a leaf cell. Mycorrhizae are no symbiotic interactions between a fungus and a root, but between fungal mycelium tissue and root tissue, parenchymatic or vascular. The other way round, the interaction “between an aphid and a potato plant” in reality is between a small organism and a leaf tissue. In chapter 5 this is fundamental.

- **Telescoping and dwarfining**

Finally, the properties of axes, branching and arrays are linked to what J. Roux (pers. comm. 1979) calls “telescoping”. This is intercalation seen from another viewpoint. Instead of looking at figure 4-8 (ST.1) as an array with axes 5 directly inserted upon axis 1, one might see this as if the axial orders 2 and 3 and 4 were “telescoped into invisibility”. Taking into account the hereditary potential for forming these axial orders which is present in all cells of
such a plant, the telescoping image is not wrong. Intercalation explains what “telescoping” is, telescoping explains what we see when observing the adult plant (ST.3).

Norway spruce (Picea abies) is known to show three physiognomic forms in the mountains or in the North, where a heavy snowload falls every winter. These forms are so constant that they are described as “genetic”. Translated from German, these varieties are called “plate spruce”, “comb spruce” and “bristle spruce”. Figure 4-15A shows the “normal” image of the Christmas tree, i.e. the “plate spruce”, in which the plagiotropic edge arrays form horizontal plates. The edge arrays of the “comb spruce” show much shorter and thinner central axes and hanging, prolonged, rather slender side axes. The “comb spruce” supports heavy snow loads without breaking its branches. The tree is adapted to mechanical snow stress.

The comb spruce looks as if the “normal” axes of the second order were “telescoped away” (Fig. 4-15A/B). The first branches are of the third order. The “orders” are defined in Êedlin’s architectural diagram (Table 4-1). The secondary or tertiary branches, at the crown edge of the comb spruce, correspond to orders four and five in the architectural diagram. They bear cones and are reproductive. The architectural diagram shows the plate spruce. The comb spruce diagram is a variant lacking intercalation of the solid axes of the second order.

One of us (RAAO) observed Picea abies, plate variant, in an acid rain demonstration zone in the Black Forest (Germany, 1984). The individual adjustment of these trees was an imperfect version of telescoping towards the comb version. Figure 4-15C shows the same behaviour under still another kind of stress in a 40 years old and 1.20 meter high Norway spruce having survived in the shade under a heavy natural forest canopy in Finland.

We saw Abies alba, white fir (Fig. 4-14C), react to atmospheric pollution by a similar change in architecture, forming a “stork’s nest” built by long, slender, fast-growing plagiotropic axes, rhythmically branching in closely stacked tiers on a near-arrested trunk.

The common ground of these individual adjustments, perhaps hereditary adaptations in some cases like comb spruces in snow country, is the morphogenetic emphasis shift to the right of the architectural diagram, from massive, long-lived axes towards shorter-lived, more slender, reproductive axes. The process described above is the “telescoping” of axes in a mono-array.

The same occurs in multi-array architecture, shown in figure 4-14D. The reiterated array in stressed Abies alba is so small that it does not even express its architecture completely. In the list of array orders in section 4.3.1.4 (a), this mini-array sits far to the right. No intermediate, big arrays have been reiterated, and hypermorphosis has taken place while telescoping away everything in between the trunk of the tree and one of the minimal array orders.

Telescoping by skipping intercalation of orders of axes or arrays is a universal adjustment and adaptation mechanism at all morphogenetic scales (e.g. see Oldeman 1990, his fig. 3.33). The way in which it is done in DNA helices is quite different, but intercalation is there under the name insertion, and intermediate sequences can be left out or inactivated during adaptation dynamics (Chap 3). Moreover, there are boosted versions versus low-energy versions of adaptation at all scales, as shown with the leaf-plus in comparison with the transposon.

In whole plant architecture, a shift to higher orders of either axes or arrays necessarily and automatically goes hand in hand with a decrease in size, mass and volume. This results in an increased leaf mass / stem mass ratio, in other words a decreased investment in long-lived tissues like wood. Adaptation to stress so diminishes the life span of the organism. The first,
somatic consequence of this mechanism is dwarfing or miniaturisation.

In trees, this shift is automatic, as soon as stress occurs. It may lead to the adjustment of organisms in a reversible way as we saw in the above case of the American chestnut. If stress is chronical, it may become incorporated in the hereditary package like in the case of the European chestnut, where it probably can be transmitted by sexualization of the meristems in leaf-plus complexes. Axes and arrays of higher orders were shown to be sexualization-prone.

Seen at an evolutionary timescale and for organisms in general, both responses to stress, adjustment and adaptation, are directed by genetic forces. Their roles depend upon the gamut of adaptive flexibility in trees, insects, mammals or other living beings. This gamut is defined by the set of genetic sequences of resistance to earlier major stress events of the same order that were incorporated in the genome and can still be mobilised. The genome is incessantly enriched with such sequences as organisms pass through one upheaval after the other.

The processes described explain automatic architectural miniaturisation and the evolution of larger ancestors towards herbaceous species. This supports one evolutionary line from trees to herbs, discussed by Hallé & Oldeman (1970) and earlier by G. Mangenot and E.J.H. Corner.

Douglas fir, *Pseudotsuga menziesii*, is an example well studied by foresters and biologists. Its area is in Western North America, between Canada and México. Introduced in plantations in Europe, it was barely acclimatised when atmospheric pollution struck. In its own area, it may reach 90 m high and live more than 1,000 years. Kuiper (1994) compared the architecture and size of trees in the region of origin around Washington State with those in Dutch, French and German plantations. Douglas firs of 106 years old in the Netherlands, some 40 m high, were slightly lower and more irregularly built than 55 years old ones in Washington.

American and Dutch sites of widely differing quality were compared (Kuiper 1994, his Fig. 2.12). Douglas fir stands on the best sites pass through four development phases, i.e. second-growth, mature, old-growth and degradation. This takes 1,000 years or more. On poor sites, second-growth phase are longer, mature and old growth phases shrink, and the degradation phase is the same. This takes some 750 years. In the Netherlands, development was partly extrapolated because the species there has not achieved a complete life cycle as yet. However, a well-informed prediction is 150 years of second-growth, absence of mature and old-growth phases, and a century or so of degradation. The total cycle would barely cover 300 years.

The mature phase is “telescopied away” under stress. The trees never reach maximum Douglas height on the poor, dry sands of the central Netherlands. The spectacular arrays reiterated in old American trees never occur. The organism is smaller. It rarely shows reiteration other than for repair purposes, and its lifespan is a third of that in its original region. It still took over 30 years before the planted Dutch stands started seeding. Then, in some five years only, the whole central Dutch region became covered in Douglas fir seedlings. Their abundance since the beginning of the nineteen-eighties coincides with increased atmospheric pollution and silvicultural soil treatments boosting germination (see Sect. 5.2 for climate in the early ‘80’s)

The maximum height and the lifespan of the trees have decreased by two thirds, whereas their seed production has become very abundant. Hence *Pseudotsuga menziesii* provides a very convincing case of telescoped development phases, dwarfing of the organism and strong sexualization under the stress of poor growing conditions after migration to a new biotope.

Atmospheric pollution is not necessarily a key stress factor. Van der Sleesen (1994) diagnosed the impact of industrial emissions on two Siberian forest plots, close to and far from the
industrial zones near the Baikal Lake. She weighted the stress factors using a simple fuzzy set evaluation. One conclusion was, that stress due to poor sites and chemical ones were difficult to untangle, particularly because her comparative plots had very different site qualities.

Long-term results of the same adaptation process are seen in *Pinus nigra* populations planted in the barren, windy Dutch sands dunes close to the North Sea since the eighteen-twenties. The small, telescoped, short-lived trees planted ca 50 years ago West of Alkmaar markedly contrast with the enormous, regularly built trees shown to one of us (RAAO) by the Dutch forester P. Szabo in the windy, barren zone of original introduction near Haarlem, some 170 years ago. The latter, big trees were standing amidst carpets of their abundant descendants.

After dwarfing of trees under stress, these trees or their descendants bounce back to gigantism once the mode of survival has been initiated. Apparently, it is not so easy to overwrite the tree building instructions, i.e. sequences of determinate numbers of axes an/or arrays. Everything looks as though a rather thorough and lasting suspension of the instructions for intercalation of large building blocks keeps the plants small until conditions improve. It would take a long time to completely delete these instructions instead of merely filing them away. Individual adjustment of parents and natural selection of descendants would be enough to explain this adaptation without hereditary change in fundamental properties.

In regions with permanently stressed sites, however, the instructions to build trees wear down and disappear with time. We will return to this deletion mechanism. The result is a pullulation of herbaceous species in regions, like the Sahel or the Siberian tundra’s. A significant remark made by botanists is, that little or no “true” herbs exist in the wet tropics. Their stem bases are usually woody. Invasion of the aerial parts of trees by wood formation might indeed come from the tree base. This fits in with Hallé’s hypothesis (1991:108, transl. RAAO) that:

- “the juvenile wood would be of caulinary origin and constitute the stem of the young tree prior to reiteration.
- the adult wood, being the vast majority, would have a rooty nature resulting from the accumulation and integration of successive reiterated root and shoot systems.”

This image fits in also with Corner’s (1964:53) vivid image of the first successful land plants: “the primarily photosynthetic and autotrophic plant, building its protoplasm from inorganic food supplies, becomes differentiated into an autotrophic skin and a heterotrophic interior supplied with organic food elaborated by the skin.”

The thorough analysis of tree root systems by Atger (1992:186 ff.) shows that plant parts below the ground also display “branching” orders. There are seven orders in the architectural root diagrams of each of the five hardwood species she studied. The last three orders (root 5, 6 and 7) are ephemeral, with “...natural pruning in the short term.” Moreover, root branching proceeds by intercalation of root orders in between the taproot and the root hairs, and there is root reiteration. Telescoping and dwarfing of root systems hence are of the same nature as in the aerial parts of plants. They provide us with the last concepts and data needed to sketch our theory of adaptation also at the levels between the tissue and the whole organism.
4.4 - The struggle of life at the levels of the organism

The complex assembly of life, at these levels like at subcellular ones, has an amazingly high performance. Amplification or mass production of certain sequences by leaf-plus complexes is strikingly efficient. A “greenprint” is transmitted from cells, as dictated by their genomes, and by coding the right mix of phytohormones supervises the building of right kind of organs and axes. Davis & al. (1990:213) wrote on Agrobacterium-mediated gene transfer in hybrid Populus biotechnology (cf. Sect. 3.4): “In the plant cell, the [bacterial] T-DNA is transcribed and translated into enzymes that lead to the synthesis of phytohormones including indoleacetic acid, an auxine, and isopentenyladenosine monophosphate, a cytokinin.”

Further along the system hierarchy, the orderly prescription of a specific axis in between whichever couple of branch orders, in view of prevailing environmental conditions, is a smooth and flexible process to face stress of all kinds. The transmission of newly edited adaptive cell properties to higher levels and whole organisms, and their expression higher up in the system hierarchy so were explained in plants. The present chapter closes with some general questions as to the adaptation of organisms in view of the issues explained above.

The first question addresses the “syntax” of the combinations of organs, of axes and of arrays. The American chestnut and the comb spruce illustrated what happens when one or more elements are “telescoped away” from the morphogenetic sequence, including those carrying the organ complexes that either ensure resistance, like the chestnut fruits, or enhance risk, like stiff fir branches breaking by snow overload. In other words, morphogenetic sequences are either regular and hammer out similar, repetitive series of organs, axes or arrays, or the quality of the series is changed by change in leaf-plus complexes.

At the levels of plant organ, axis or array the elements functioning like codons or instruction-carriers are the leaf-plus and leaf-minus. It is unclear as yet where such carriers should be sought in animal organisms. A leaf-plus works by virtue of the inframeristematic initial ring under the apical dome. This ring initiates local mitosis and/or somatic meiosis. The chances of somatic meiosis to occur there are rather scant and if it does, so are its chances to become included in reproductive transfer.

The question now is, can we understand morphogenetic syntax in organs, axes and arrays in the same way as the grammar of DNA-coding in section 3.4.1? Is there a clear build-up with clear mechanisms to change the grammar or to delete the changes when necessary?

We tried in vain to find a clear linguistic analogue like YOU DID NOT SEE HIS HUT in the double helix (Sect. 3.4). The connection between DNA functioning and plant build-up is a ladder with increasingly complex steps, as shown by architectural diagrams and sequences of architectural diagrams above. Their syntax is not nearly as elegant as those in the previous chapter. The semantics of plant architecture are studied today as a basis of the AMAP software (see Bouchon & al. 1997).

A second question addresses the number of variants. We saw in chapter 3 that two and only two variant DNA forms are both enough and necessary to escort the specific DNA mode during the evolutionary process. In the present part of the many-layered system hierarchy, with its command levels of the organ, the axis, the array and the multi-array, such simplicity is not found. Broader variation is expected here. Direct observation indeed shows countless diverse forms, colours, sounds and smells in the organisms around us. The sentences written
with the words of chapter 3 are now arranged in alineas, chapters and texts. In our overview of
the architectural dynamics of plants, the numbers five, six and seven were very frequent. Are
they limitative numbers of organogenetic orders. Do they have any biological meaning?

A third question occupies biologists since Goethe postulated the “phyton”, a leaf-bearing
internodium, as the basic building block of plants (Frobe & Gleißer 1996). Our leaf-plus is
no phyton, but the carrier of a messenistic package of architectural building “instructions”.
This opens a new view on regular architectural deployment of plants, guided by transmission
of genetic instructions from one hierarchic command level to the next. However, several cases
of barely organised architectural dynamics balancing on the brink of chaos were shown above.
How organised and how flexible is morphogenesis?

A fourth question then asks whether the dynamics of growth and development are equally
severe or loose at all organisation levels, and at all places in the architecture of a living being?
Does this influence fulfillment of biological functions? The very concept of system hierarchy
hangs on a correct answer to these questions. Indeed, if the answer did not matter we would
all be monocellular or viral.

A fifth question arises because this chapter is on plants. Their responses to the stresses they
face are dictated by the fact that most plants are rooted in one place. How is this in animals?

4.4.1 - The fuzziness of language

The precision of the adaptation model in chapter 3 is high and its performance can be
demonstrated very precisely. One determinant of this precision is, that DNA mutations occur
in biological points. These are structures with a fractal dimension close to zero (for fractals
e.g. see Barnsley 1988:172ff.; Lorimer & al. 1994). Only mathematical points possess a zero
dimension. For this reason, genetic precision closely approaches mathematical precision. Still,
from the outset the living architecture at all levels has a certain degree of fuzziness.

This is expressed as follows in the stochastical terms of mathematical statistics. The basic
mechanism of Evolution, being an adaptive process, is informed by the genome, a coded text
characterising a living entity. This includes an increasing number of alineas, the genes. The
number and variety of genes increase for the simple reason that over the geological timespans
since the origin of life on this planet, the environmental conditions varied ceaselessly.

Every desequilibrium is followed by a new equilibrium in the midst of increasing entropy
(Prigogine & Stengers 1984). Hybridisation by crossing-overs during meiosis indeed causes
responses to the same stress factors to vary a little. This adds a certain degree of stochastical
incertitude to the observed responses when noting these in mathematical language.

From the outset the present chapter emphasised that limits between architectural categories in
biology can not be sharp (Vester 1997). Any biological point wavers and so at least subtly
suggests a line. Every line zigzags a bit, thus subtly intruding into the dimension of surfaces.
Biological lines, like in our drawings, so have a fractal dimension higher than one. Biological
surfaces have a certain thickness, at least that of a layer of molecules, and so trespass subtly
beyond dimension two, towards a volume. The dimension of biological volumes slightly
exceeds three. The fourth dimension is linked to time, so there always is a bit of dynamics.
The use of sharp limits in the study of biological systems indeed means little, unless well scaled. A “sharp” limit at one system level then is read by using the patterns one level lower. For instance, the limits between the parts of the axes shown in figure 4-2 are at the level of tissues, such as abscission layers between leaves and axes. In the figure, they are shown as lines, i.e. sections of surfaces. However, studying leaves at their own level, these lines are bands with a surface. They are sections of a volume filled and structured by cells of a specific kind. This shift is made visible by a common microscope, changing the magnification.

Understanding the syntax of the language at each level and its translation to languages at other levels depends on the choice of system levels and, per level, choice of compartments. If the present chapter is right, the “instruction manuals”, i.e. the sequences of differentiation, are clear, stable, and liable to be corrected if syntactic elements drop out or change place.

Genetic instructions were compared in section 3.4.1 to words forming sentences. Alines as edited from these sentences inform chromosome action. After many intermediary aggregation steps of ever more complex sequences, the composition of the whole “text” instructs organism development. This semantic image is borrowed from Koestler (1967), who used it in his pioneer discussion of system hierarchies versus linear sequences.

In section 3.4.1, a sentence corresponds to three turns of an 10 bp B.DNA helix plus 3 bp. This is \( 3 \times 10 + 3 = 33 \) bp, or 11 words of 3 letters each. In each half-turn of the helicoidal path, five/six dynamics have to be solved before continuing on the next half-turn. Moreover, the number of five axial states, the “branch orders” in Édelin’s architectural diagrams (1977, 1984 etc.), is remarkably recurrent. Analysing tree crown formation, Froebe & Gleißner (1992/1993) postulate five development “cycles” in shrubs, each involving construction of a “Gerüstmodell” (German for “framework”), i.e. our reiteration of a branched array. The leaf-plus and the minimum axis may be at their levels what the sixth or seventh nucleotide is at the level of the helix, to wit final words of sentences, alineas, paragraphs, etc. The Fibonacci series in leaf spiral studies shows that other numbers may be involved than 5, 6 or 7 (Van der Linden 1996). However, the syntactical rules remain the same.

**In all cases there is a limited sequence, terminated by a biological clasp.**

A clasp works like a belt buckle. In plant morphogenesis, a clasp is a miniature structure closing off one sequence and hooking it to a new, perhaps cyclic beginning, so that it may start over again in a more or less similar way. Biological claspers are protein molecules in chromosome building. In axis building they are meristems or in vitro other prolific cell heaps. In array building, they are leaf-plus. In multi-array building, they are minimum axes.

In the semantic image, the clasp is the point separating one sentence from the next one. The difference is, that the biological point is not a passive punctuation mark. It actively closes off one morphogenetic word, sentence or other “text” sequence and initiates the next one.

For those being used to great precision of genetic mechanisms, macroscopic architecture and dynamics may seem overly approximative. Therefore it is once more emphasised that the fuzziness of systems becomes more perceptible when moving towards larger sizes and longer time periods. In fact, guided plant construction and architectural fitting designs exist at all scales visited, but their precision and biological efficiency are similar relatively to their level.
4.4.2 - Elements of architectural flexibility versus organisation

The branched array is the basic inherited pattern in trees, used to confront all environmental conditions (cf. Froebe & Gleißner 1992/1993, 1995). A plant dies if its “organisation unit”, as those authors call it, provides insufficient response to its environment. However, the job of organisation often shifts from the branched array to finer structures or to larger ones (cf. Hallé 1986). Oldeman & Vester (1997, in press) discussed this shift of the focus of organisation in plants. Such shifts remained underexposed in biology, so methods to examine regulation were apt to be restricted to some one or two levels between macro and micro. Moreover, small and large branching and differentiation patterns interact. This hampers categorisation still more.

The terms "crisp" and "fuzzy" were used above in their everyday meaning. Their scientific definitions are: "A classical set is defined by crisp boundaries; i.e., there is no uncertainty in the prescription or location of the boundaries of the set. (...) A fuzzy set, on the other hand, is prescribed by vague or ambiguous properties; hence its boundaries are ambiguously specified." (Ross 1995:17). Fuzzy logic mathematically deals with fuzzy sets (see McNeill and Freiberger 1993, Kosko & Isaka 1993).

Trees are often considered as solid building blocks of an eternal forest. However, the above sections show that no forest building blocks of any size are as fuzzy and ceaselessly changing as trees. This inherent fuzziness, linked to fractal dimensions (see 4.4.1) is indispensable for adjustment or adaptation to a fuzzy environment. It gives the tree the architectural flexibility it needs to survive (cf. Oldeman 1989a).

Plant development and adjustment can not be understood without flexibility. Some axes and arrays have a flexible nature, like all mixed plagio/plagio axes (Fig. 4-2) and the arrays they build (Oldeman 1989). Other plants, like unbranched ones, are not so flexible. Many react in a balanced way to environmental variations, their large appendices allowing the growth programme to vary but hold. An instance are Palms with flexibility at the axial level allowing adjustment of leaf numbers. As described by Oldeman & Vester (1997 in press), the potential of metamorphosis and reiteration gives the tree flexibility as a multi-array.

The mirror image of the preoccupation with the focus of organisation leads to the question: “Is there also a focus of flexibility in a tree?”

As said, in quite a few trees the focus of organisation is one near-cyclic process, i.e. recurrent production of branched arrays (cf. Froebe & Gleißner 1995). For every tree a moment comes in which its cyclic architectural recurrent sequence deviates far from its well-defined "reference" architecture (Fig. 4-3). The thermodynamical term would be "a state far from equilibrium" (see bifurcation diagrams, Prigogine and Stengers 1984). This state depends on both ontogenetic and external influences that may be close to or far from the average pathway of their behaviour. "Quite a few", "near-cycle", "close to" or "far from" are fuzzy concepts.

In big tree architecture there are foci of flexibility at certain levels and foci of organisation at others. Due to both adaptation and ontogenesis they together determine tree organisation in an actual situation. The miniaturisation sequence from trees to herbs (see above; also Gamalei in Édelin 1998), may be conceived as an instance of organisation versus flexibility.

Flexibility at and above the level of axes interferes with classification efforts among branched arrays. A different name may be attached to every observed shape, but this is paid by loss of dynamic understanding. Another option is the use of architectural diagrams at every system level, as done in the present chapter. Flexibility at those levels is then comprehended by
differentiation, intercalation, number of orders and the nature of clasps. However, fuzziness is inherent in descriptions of flexibility. The database is inherently incomplete because only a limited number of cases can be observed out of an infinite number of existing ones. We also saw in the preceding chapter that stress may induce somatic meiosis in meristem cells, so unpredictable change is inherent in morphogenesis. The levels of the foci of flexibility hence are less easy to pinpoint than the levels where foci of organisation are manifest.

Figures 4-2, 4-3, 4-4, 4-8 and others show strongly organised system levels. We saw that this organisation can deviate to a certain extent from “true form” at each level, in a flexible way not easily defined. The drawings by Frobe & Gleißner (1995) of NW-European trees under stress illustrate this point. Only a very precise morphological analysis allowed them to separate orderly branched arrays from panicky heaps of branched and rather dissimilar axes.

Some authors, like the Russian morphologists in Édelin (1998, in press) hence analysed plant architecture as if all axes were metameres, like the recurrent segments building the bodies of worms and arthropodes. Metameres are modular. However, functional modification of such segments also is so strong and so frequent that flexibility is paramount. In the northern vegetation belt of Asia and Europe, plants indeed not only suffer the burden of an irregular climate including man-made chemical stresses (van der Sleursen 1994). They also carry the heritage of the generations of stressed plants having survived the Glaciations (cf. Chapt. 5).

Differentiation of structural elements in plants, such as chromosomes, intracellular particles, cells, tissues, organs, axes, arrays and such is evident from the preceding pages. Figure 4-17A shows the spectacular example of *Acacia drepanolobium.* However, the next question now is: “Are the system levels all strictly equivalent, or are they specialised for certain tasks too? Can adaptation or adjustment take place by shifting functions from one level to the next?”

4.4.3 - Transfer of functions from one level to another

Transfer of functions from one organisation level to another is classical. It was observed since long ago in the inflorescences of Compositae, functioning as flowers, or in the green axes of American Cactaceae and African euphorbs (Fig. 4-17B) photosynthesising as leaves.

In the Orsay laboratory, we studied a case of functional transfer in potato, *Solanum tuberosum* (Blanc 1983; Charles 1992; Charles & al. 1992, 1994; Blanc & al. 1995). This important crop plant was introduced from the Andes in Europe by Spanish explorers in the late XVIth Century. Since the XVIIth Century European potato production unceasingly increased.

*S. tuberosum* branches aboveground and also branches below the ground by stolons at the base of the stems. Under certain circumstances of “induction” these stolons extend and initiate tubers. The induction of tuberisation is operated by metabolic stress causing a decrease in the activity of the apical meristem, a slowing and finally an arrest of growth of the stem. The metabolic stress is due to a deficient activity of ATP-ase, caused by insufficient sugar concentration in the cells. This is a photoperiodic response to European short days.

The metabolic deficiency causes poor fixation of the IAA growth hormone on the inner cell membrane, the plasmolemma. This brings about accelerated ageing of the plant and, at the same time, a trophic inversion. Indeed, the sugars produced by the leaves now are transported downwards by the descending sapstream to the basal parts of the plant including the stolons, whereas in vigorous plants their transport is upwards in the ascending sapstream to the apex. The adjutive response is made in three steps.
The adjutive response is made in three steps.

1 - As an *initial* step, an ephemeral increase is observed in the conductance of the stomata and cells at the level of non-senescent leaves. This passingly boosts photosynthesis.

2 - As a *second* step, an inversion of the demand for sugars and phytohormones occurs along the axis. During this period, the activity of an enzyme linked to growth, cytoplasmic invertase acid, gives way to saccharose-synthase activity. Tuberisation so is induced.

3 - As a *third* step, reserves of substances such as sugars and proteins are stocked in the appropriate tissues of the tuber.

Carbon management hence is transferred from the level of the leaves to the level of the axes. Stolons and tubers indeed possess an axial nature.

Another case to study would be C₃- and C₄-photosynthesis. This is the fixation of CO₂ from the air by two distinct biochemical cycles (e.g. see Larcher 1976:39-44). C₃-photosynthesis functions at the level of one cell, follows the cell-physiological CALVIN-BENSON cycle and has an endogenous asymptotic curve of light intensity versus production. C₄-photosynthesis functions at the tissue level of co-operating mesophyll and sheath cells in grasses. It follows the HATCH-SLACK-KORTSCHAK cycle and has an endogenous production curve without any asymptote, the production being limited by non-light factors, mainly heat. There is still another photosynthetic pathway followed by succulents, which is only mentioned here.

Chapters 2 and 3 showed the chloroplasts to be direct markers of DNA dynamics. The performance of cells with C₃-chloroplasts is the normal version. The superior performance of C₄ photosynthesis occurs at the tissue level. So at least three distinct levels of photosynthetic maximisation are required by environmental conditions, i.e. the increase of the chloroplast number (Chapt 3), the increase of the number of green cells, and the qualitative modification of the chloroplasts which lifts the improvement from the cellular to the tissue level. Cactaceae or many euphorbs (cf. Fig. 4-17B) represent the next shift, from organ to axis level. The photosynthetic function so is transferred step by step from chloroplast to branched array. One set of stress factors driving this transfer is increasing environmental heat load and water stress. C₃ mainly occurs in forests, C₄ in savannas and the Cactus system in arid places.

Oldeman (1989b, 1990, 1994) gave several overviews of the matter at the macroscopical level. We now can see this as the macroscopic part of a transfer series starting at microscopic levels. An important rule of adaptation and adjustment hence is the extension of Oldeman's Third Silvological Rule (1990: 562), i.e. the set of rules referring to transfer of functions:

“All biological functions within a living system are carried out either by subsystems or at levels that ensure a minimal cost-benefit ratio in terms of matter, energy and information.”

This rule can be applied to biological communities as living systems, as a prelude to the next chapter. The transfer most apparent and best studied is the mycorrhizal association between fungi and plant roots. It indeed is vital for both the mycobiont, a fungus, and the phytobiont, a green plant (e.g. Smits 1994, Smith & Douglas 1987, Oldeman 1990). They may be compared to a large firm, a green plant, and its small ancillary suppliers, i.e. fungi and often mutualistic bacteria. In particular, the fungal hyphae may have such a large absorption surface that they can increase the water supply to the green plant by several hundreds of percent. They also provide metabolites, particularly nitrogen-containing molecules.
Is this also a stepwise transfer from cell nucleus to tissue level? Plasmids travel to and fro between micro-organisms and larger ones (e.g. Garrett 1994, Davis & al. 1990), dealing in nitrogen, especially complex proteins. We know that many mycorrhizal tree species do not form root hairs, their function being transferred to the mycelium of mycorrhizal fungi.

The analogy of the large firm and its ancillaries hence is faulty. The phytobiont and mycobiont are linked much more intimately than by water, nutrients and vitamins alone. Interlinked at the DNA level by travelling plasmids, they can not be said to be “either two distinct species or one species with genetically distinct organs”. The inherent fuzziness of their limits precludes separation. The truth is double-faced, like in light waves versus photons. This completes the serial endosymbiosis theory of Margulis (1970 ex Smith & Douglas 1987:238), postulating the incorporation by proteocaryote cells of aerobic bacteria which evolved into mitochondria, of spirochaetes which first became cilia and flagella, and of cyanobacteria at the origin of plastids.

Transfer of root functions is fuzzier than the cases of photosynthesis and carbon management. Its complexity may be unravelled by studying nitrogen processing. Our hypothesis is transfer of metabolical command functions by travelling plasmids, between the nuclei of fungal and green plant cells. Phytobiont nitrogen synthesis goes to fungal cells. The transfer would reach the tissue level if water or soil oxygen became limiting.

The phyllosphere (Ruinen 1974; Chapt. 5) may reveal an analogous transfer of functions when better studied. The phyllosphere is the interface between tree crowns, primarily their leaf surface, and the atmosphere. Like the rhizosphere, this interface hosts many microorganisms and meso-organisms forming a kind of symbiotic community colonising the leaf canopy.

4.4.4 - Animals and motility

This chapter nearly exclusively dealt with plants, whereas most genetical studies emphasise the zoological side of the medal. We expect to read about animal architecture and life soon in a book by Francis Hallé from Montpellier. Animals therefore will only be shortly mentioned here. A fundamental difference with plants of course is animal motility (Oldeman 1990).

One of the stress reactions in plants is the shift towards sexual behaviour. This is well known by gardeners, who, for instance, apply stress to shrubs by root pruning to provoke abundant flowering. Flowering means production of pollen and seeds. Both travel (Fig. 4-17C). Indeed, plants are constrained by their fixed position to face stress, to die, or to form diaspores.

Animals take flight as soon as the stresses they perceive become too threatening. However, many severe stresses are insidious and can not be perceived. Such major stresses are exemplified by UV or X rays, radioactivity, the far red irradiation mentioned earlier, chemical pollution, or biotic impacts by viruses, bacteria, fungi or other tiny but powerful agents. From such invisible threats, animals can not flee. They show the same adaptive response as plants, ensuring the procreation of numerous descendants with high mutation rates by means of boosted sexual and somatic meiosis. Somatic meiosis as a general response to stress indeed is the tool of the last chance in the struggle of Life to perdure.

Animal motility is optimised by their finite size and their general dorsiventrality, i.e. bilateral symmetry with a back and a belly. Animals also show a general lack of reiteration, although metameric insect architecture might be explained as the result of a programmed sequence in
which a regular metameric repeat sequence occurs in strongly different versions. However,
this regular process may rather be a branching differentiation sequence than reiteration.

Animals lack reiteration, but a specific and close interrelationship exists, at the chromosome
DNA level, between zones called “homeotic genes”. These take care, e.g. in insects, of the
setting of “imaginal disks”, small cell groups in insect larvae, each group destined to develop
into a particular, entire, adult structure like a leg or wing. Homeotic genes are marked by
“homeoboxes”, homologous sequences like those found at the 3’ exon of homeotic genes in
Drosophila fruit flies. “Similar sequences have been found in other organisms......possibly
involved in DNA-binding during gene regulation” (Suzuki & al. 1989: glossary).

These homeotic genes, aided by homeoboxes, so initiate the different parts of an animal
embryon. In the same way, plant seedling cells initiate seedling parts; the apical meristem, the
epicotyl with its tissues and the cotyledons above the ground, the hypocotyl with its tissues
and epidermis plus the root meristem below the ground. Functionally, an animal egg then may
be compared to a spermaphyton seedling. In the seedling, the axis and the cotyledons
energize meristematic activity. In a hen’s egg, the albumen feeds embryonic development.

Ecologically, animals are carriers. They carry biomass and information. Biomass often is
digested, i.e. it is first transformed, then dropped elsewhere in the ecosystem. The spread
of information includes information contained in pathogens or symbionts travelling by contagion
Information is also carried by spreading certain pheromones interacting either with sexual
mates, or with other species. Moreover, optical and acoustic signaling occurs in a truly multi-
media way. An important information highway is formed by transported seeds, spores and
pollen. An example of the complex, precise and refined interactions involved is provided by
Bruneau (1997) in her thorough study on the bird pollination syndromes in the leguminous
tree genus Erythrina. In the conclusion of this impressive study covering hundreds of species,
she states (p. 65): “overall, the analysis indicates that pollination systems are not
evolutionarily constrained, even at lower taxonomic levels.”

This once more demonstrates the fuzziness of the distinction between plants and animals. Are
we a distinct human species, or part of a fuzzy species including , for instance, our intestinal
fauna and flora as members? In the next chapter we will examine the ecological viewpoint.
Chapter 5  Ecosystems under stress

In 1948, A.E. van Vogt wrote a classic science-fiction bestseller, “The world of null-A”. Among other things it gave an implicit and largely unnoticed caricature of a basic tenet in classical evolutionary science. Null-A means “null-abstraction”. In summary, the thesis from Van Vogt’s book is “memory is identity” (also see Van Vogt 1970). His hero, Gilbert Gosseyn, can memorize some spot, eliminating abstraction by attaining a precision of tens of decimals. He then can return bodily to that spot by evoking it in his mind. Excepting Gosseyn’s physical displacement, this resembles a recurrent idea in genetics concerning internal cell memory.

Cell memory is basic to adaptation. Every organism is assumed to have a genetic memory perfectly reflecting its environment. If this memory were imperfect, and if the imperfections were passed on to descendants, these would be unadapted and become extinct under selection pressure. Now let us consider both terms in this balance, “environment” and “memory”.

On the one hand, the environment most often is considered as the sum of many factors. The literal translation of the Latin factor is maker. However, in complex ecosystems, like forests, tropical rain forests in particular, coral reefs, even rural ponds or root systems with their myriads of micro-organisms, individual factors cease to be clear and become unrecognizable in the countless interacting giga-, mega-, macro-, meso-, mini-, micro- or nano-environments. Vester (1997) has shown this for the light climate in Amazonian forests in Colombia.

On the other hand, the genetic cell memory, assumed to mirror the environment, may not be conscious like the memories in our brain which can be expressed in words. Hence there is no rigorous, explicit proof of the contents of a cell memory. It only can be inferred from cell behaviour, like the stress-induced reactions described in earlier chapters. These all lead to doubt whether indeed the genetic memory of organisms inhabiting complex ecosystems offers a faithful image of the above welter of mutually interacting and interdependent environments which, to make it worse, change every second.

A much longer “recollection trajectory” must be explained between the “selfish gene” (coined by Dawkins 1976) and the whole organism. How is the genetic memory linked, for instance, to preference of squirrels for nuts, to construction of hanging nests by weaver birds, or to the principles of mechanical repair common to broken bones and broken trees (Matteck 1993)?
Still, cause and effect chains straight from the molecular to the macroscopic are often taken for
granted in current science. Such confusing masses of memory, information, environment and
milieu are better understood by the use of a system hierarchy, as in the previous chapters.

Let us take the case of the yellow flowers of the dry and coastal tropics. Such flowers often
dominate the visual impression of these vegetations, whereas tropical rain forest canopies are
visually defined by a mixture of white, purple, yellow and red flowers. Tropical biologists
usually recognize this when it is pointed out. However, discussion of the phenomenon quite
often returns to "the one environmental factor" having "selected this trait" in the flowers.

Dr. P. Raven, director of the Missouri Botanical Garden, pointed out to one of us that selection
was probably due to bee pollination. Different bee taxa, with different colour vision,
predominate in dry and in humid neotropical tree biotopes. This does explain both the yellow
predominance and other observed facts (see Oldeman 1990: 442), particularly so because no
one general environmental factor is used, but an interaction delimited very precisely in time and
space. Flowers and bees both have lifespans of days. The selection occurs in a space of
decameters (10 m) for colour perception and cubic centimeters for pollination.

This raises another crucial question. On the one hand, did a hot, dry climate select xanthophyll
producing plants, causing yellow flowers to dominate and did it so offer an advantage to bees
with colour vision in the yellow? Did feedback then advantage yellow flowers because they
were pollinated selectively by such bees? This explanation is classical (see Janzen 1967). On
the other hand, colour vision in insects is an acquired character. Did bees have a vision in
yellow-grey first, and did plants produce flowers with a mixture of coulours, yellow among
them, in dry climates in remote geological periods like the Secondary or the Tertiary? Did bees
then start servicing yellow flowers by selective pollination, the flowers rendering the service to
the bees in a self-reinforcing feedback loop, by advantaging bees with a yellow colour vision?
Both hasard, i.e. acquired characters, and necessity, i.e. living architectural heritage, are
players here.

Precisely defined architectural and temporal criteria give us a solid grip on the discussion of
both Van Vogt’s idea and the notion of adaptation. Russian dolls illustrate a good, simple
spatial hierarchy, a set of nested systems with organic forms. A larger one always is the
environment of the next smaller one. A hierarchy in time is illustrated by cogwheels of different
sizes, turning at different velocities, like in mechanical clocks or gear-shifts. Short and long life

Often the hierarchy of life is seen as a set of communities built by species, species built by
individuals, individuals built by organs, organs built by tissues, tissues built by cells, and so on.
It is also tacitly assumed that such systems are simpler or more “primitive” if smaller and
shorter-lived. However, in reality the organization of a forest stand is as complex as that of a
tree, a tree as complex as a branch, a branch as complex as a leaf, ad infinitum.

The word scale derives from the Latin scala, “ladder”. Organic hierarchical system ladders are
not parallel in space, time and complexity. Something very complex is not automatically very
long-lived and/or very large-sized. Nature has no preference for certain scales.
5.1 On nested ecosystems

5.1.1 A system hierarchy

Can “the environment” be better explained by using a hierarchy of systems, like we did for organisms in the above chapters? An analogy is the conceptual hierarchization of a Paris borough or quartier. This is the environment of a street, for instance the Rue des Écologistes. This street is the environment of an antiques shop, an antiquariat. The shop is the environment of a set of differentiated spaces, like corridors, stairways, salons, packing and selling rooms, toilettes, and the show-window or étalage. Each room is the environment of a set of diversified furniture. Several pieces of furniture are the environment of the bric-à-brac of small belongings of the owners. And so it goes on.

Claiming that yellow flowers are predominantly selected by a dry climate is as empty of meaning as claiming that rocking-chairs are mainly found in red-roofed houses. Even if faultless statistics testified to such a fact, it would be a useless fact. It is meaningless as long as the selection mechanism can not be explained (cf. Thomson 1997). Said otherwise, the whole hierarchy of organic system levels must be examined to explain biological change in response to stress.

Like a nest of bowls, the following is a nested system hierarchy.

- The environment of a nucleotide is a chromosome
- The environment of a chromosome is a cell nucleus
- The environment of a cell nucleus is a cell
- The environment of a cell is a tissue
- The environment of a tissue is an organ
- The environment of an organ is an organ complex (axis: Fig. 4-2; organ in animal)
- The environment of an organ complex is a branched array (Figs. 4-3, 4-8)
- The environment of a branched array is a multi-array (Fig. 4-4)
- The environment of a multi-array is an organism
- The environment of an organism is an eco-unit (Oldeman 1990)
- The environment of an eco-unit is a vegetation mosaic (Oldeman 1990)
- The environment of a vegetation mosaic is an abiotic mosaic (see Sect. 5.1.2)

Scientists may and do chose levels or scales as they see fit for the understanding of what they observe. Sometimes they make systems. The “ideal gas” concept emerged from the “closed vats” of XIXth Century physicists. Otherwhiles, scientists think that their systems exist, being “naturally defined” by “natural limits”, independently of the observer. However, no scientist can deny having chosen in person the kind of system to be studied, by the very act of choosing, i.e. delimiting the object of examination.

The above system hierarchy contains many system levels, very close to each other (Oldeman 1990). For instance, the plant cell level is not explained as a direct subsystem of a much larger system, e.g. a river basin. This is not self-evident in present-day ecological literature, which often explains the very small in a very wide context. Biomass production by a whole plant cover is often explained by the surface of all leaves in m² over one m² of soil. This yields the dimensionless leaf area index (e.g. Lächer, 1976:31ff; Landsberg & Gower 1997:68ff). In this way, a corn field or forest stand is explained in terms of sums of thousands of individual
leaves. Calculation of relations between levels so far apart demands estimation of numbers and use of statistical averages to create scientific order in such a sea of details.

Next to such widely spaced system levels, the above hierarchy is like a fine-toothed comb and requires fewer stochastical mathematics. Compared with the Russian dolls, this set has more nested dolls, fitting more closely together with less rattle. The environment of a tree leaf here is no forest stand, but a leaf-bearing branch and its direct surroundings. The whole hierarchy is built in this way, a sequence of systems with their direct or close environments. The differences are fundamental with the usual “environment”, i.e. a set of factors expressed as average values, assumed to select a whole organ or organism, down to its germplasm.

* First, the factors in the close environment differ from those in the far environment because they have been filtered. Rain, arriving at a tree leaf, for instance, has been sifted;

- by the planetary climatic system hierarchy (see sect. 5.2),
- by the landscape, e.g. the exposition of the relief to the dominant winds,
- by the local vegetation, i.e. the faraway surrounding vegetation mosaic,
- by the architecture of the closely surrounding vegetation patch (eco-unit) in the mosaic,
- by the branched array of its tree crown,
- by the surrounding organs of its axis

As concerns the rain arriving at the leaf, its rate of descent, the size of its drops, its chemical composition and its caloric load hence are not at all the same as those of that rain when it started to fall from its cloud

* Second, the direct environment regulates the living conditions of the organism, organ, cell or virus it so closely surrounds. It functions like clothing, by optimizig heat, humidity, aeration or mechanical support. However, contrary to clothing, the direct plant environment can not be changed by the plant inside. A vegetable system either dies or adapts itself, if it happens to grow in a stressed direct environment. This is the base of many phytotronic or in vitro experiments like those in our first two chapters. It also is one of the important distinctions between human, animal and plant adaptation in general (see Sect. 4.4.4). Most animals can flee, humans can often modify their direct environment.

Plants can only migrate by seeds, spores or cuttings. Their vectors are abiotic, such as gravity, wind or rivers, or biotic, like birds, insects or mammals. Positional change by reiteration yields root suckers that form clones after the original tree dies. This often is a blind, short-distance mechanism like seed dispersal by gravitation, but sometimes it allows plants to cover larger distances, e.g. grasses or bamboos. Migration is not flight, because flight works by feedback, triggered by danger signals and initiating a quite precisely perceived and oriented flight path “away from danger”. Moreover, migration is not at all the equivalent of conversion of an environment by an organism. Plant reactions indeed differ much from animal reactions.

* Third, the direct environment does not need to be a very close fit. Lovelock’s proof (1988) of some plasticity in the marginal conditions of life on Earth may be expected to apply proportionally over the whole living system hierarchy. The fuzziness of axial differentiation shown in Chapter 4 emphasizes this point. Seen in physical space, how thick is the layer that forms the direct environment of an axis? Which are the marginal values of ecological factors defining this layer? Do thickness and fuzzyness of a direct environment increase or decrease with the scale of the system considered? Such a proportionality must exist. The direct environment of a virus can not have the same size as that of a tree, and the other way round.
*Fourth,* very thin direct environments do exist too. They often consist of films formed by symbionts living at the outer surface of organs, organisms or ecosystem compartments, and have obligate mutualistic relations with the larger system. The clearest examples of such filmy direct environments are our intestinal flora, the rhizosphere and the phyllosphere.

The phyllosphere, named by Ruinen (1953, 1956), is the interface between leaves and the atmosphere. It supports a mini-ecosystem originating from colonization of wet, exuding leaf surfaces by nitrogen-fixing bacteria. These bacteria are succeeded by other organisms like fungi, algae and small epiphytic mosses and seed plants over the life span of the leaf (e.g., see Andrews & Hirano 1991). Ruinen defined the phyllosphere strictly as the interface between plant leaves and atmosphere. Some 30% of nitrogen recycling in forests is phyllospherical.

As such, the phyllosphere is the counterpart of the rhizosphere, discovered in the early XIXth Century by German researchers who found N-fixing bacteria in root nodules. It is now recognized not only bacteria, but also mycorrhizal fungi and a whole retinue of minute plant and animal species, e.g., nematodes (cf. De Goede 1993). The rhizosphere may seem thicker than the phyllosphere, but this can be due to soils being supportive and not so the atmosphere.

The gastrointestinal flora and fauna in animals and people have long been recognized as an important disease factor, before being seen as a health factor. Indeed, the gastrointestinal tract formally and biologically is the interface between the animal skin surface, differentiated for water and nutrient exchange, and the further environment. As stated above, interactions are bound to one organization level. For instance, they are between an intestinal cell and an *unicellular* organism, not between an unicellular organism and a tissue, organ or organism. Another interface is in the lungs, their cells interacting with airborne microbes. We do not know any mutualistic unicellular organisms in this environment as yet.

The outer animal skin also is an interface with the atmosphere. Hairy skins in particular host small organisms, from bacteria, fungi (skin diseases) and mites (scabies), to insects (fleas, lice). The pelt of the South-American sloths (e.g., Bradypus tridactylus) is colonized by many symbiotic organisms, among which primary producers such as green algae. To wash sloths with desinfec ting soap is to kill them, as disappointed “owners” of pet sloths well know. This at least is one case of an animal counterpart of the phyllosphere in which small mutualistic symbionts are rife. However, it is indeed well known that intensive, antiseptic washing of domestic animals like dogs causes loss of resistance too (e.g., Méry 1959).

All this fits in with interactions through travelling plasmids, explaining the “epidemic of plasmids” found recently by O’Brien at Harvard, when cataloguing numerous diseases assumed to be bound to plasmids (ex Garrett, 1994:431). The transfer of information-carrying DNA fragments by plasmids was analyzed and explained in chapter 3 of the present book. In section 4.3.1.4a, the fuzzy limit between large organisms and populations was explicit.

This viewpoint is particularly botanical. There are few such cases in animals. Corals are often mentioned as such, and their treelike architecture (Dauget 1985) supports this view. Mutualistic symbiosis may lead to incorporation by larger organisms of small organisms, first in the role of “privatized” ancillary “pseudo-organs” (see 4.4.3 and Oldeman 1990: 386). Again, we stress the fuzziness of the limit between organism and population, as well as between independent species. If plasmid transfer proves to be as common as it appears today, it would rarely act to spread disease, but on the contrary keep living systems healthy within the complex of interacting assets and stresses in their direct environment.
5.1.2 - Ecosystem architecture

The term ecosystem, coined by Tansley (1935 *ex Deléage 1991:119), conveys a general meaning and originates from study of species populations. “Ecology” was formally defined by Haeckel (1886 *ex Deléage 1991:63) as “the relations between organisms and the exterior world”. Haeckel, although a morphologist, hence based ecology strongly on population numbers and food chains, the “exterior” world being partly inorganic, partly biotic.

The present book does not see ecosystems from this viewpoint. The architecture built by organisms, not their numbers, explained our observations. The base number in variant DNA or the chloroplast number in variant leaf cells were parametric, not explanatory markers of general structure. Biological architecture expresses four-dimensional biological complexity. Numbers of organic building blocks are dimensionless, dependent variables.

The next pages summarize ecosystem architecture in general terms, extending the hierarchy of the whole book. This conveys to ecosystems two more attributes than population numbers and frequencies alone, i.e. spatial dimensions and a lifespan. All living systems germinate or are born, grow, become adult or mature, and finally degenerate and die. Ecosystem frameworks are built by plants, in the seas by corals or algae, so ecosystem life cycles have geographical coordinates. Next to a lifespan, they have the area of soil or seabottom on which they are born as a bottom surface. Finally, they have a specific organization, built by their growth dynamics and expressed at every instant of their life by their momentary architecture.

One feature, says Boyce (1978), precludes this viewpoint. He claimed that ecosystems, especially forests, have no organs to process information because they lack a nervous system, and hence should be analyzed numerically in terms of their component species.

This hypothesis was rejected by Oldeman (e.g. 1985, 1990). He defined the information base of an ecosystem as its stock of eggs, seeds, cuttings, meristems and other information carriers. The transmission of information in biological communities varies. It may be by sunshine sifted by leaves resulting in shade patterns, or aqueous solutions of minerals, or particles sus-pended in the atmosphere, or biotic architecture inducing wind turbulence. Other information channels are biotic, e.g. information-carrying propagules, streams of biological solutions like those from the phyllosphere to the organic soil horizon, or seasonal litter decomposition products carrying physiological instructions (e.g. phenolic acids, Kuiters 1987).

In ecosystems, like in organisms and in chromosomes, energized conveyance of information exists. *Common boosters of information are seed-dispersing and pollinating animals* (cf. 4.4.4). A striking case is the occurrence of two pioneer tree species of the genus *Cecropia* (MORACEAE-CECROPIOIDEAE) in large Guayanese forest openings. One set of species grows on the borders of such openings, another set in the center. This was attributed to microclimatic patterns. However, Charles-Dominique (pers.com. 1989) found that the seeds of border species came with droppings from defecating birds seated on branches, whereas the seeds of central species were dropped by bats flying over the opening.

To some extent, in biotic architecture “the medium is the message”. Plant architecture mirrors the tubes conducting the sapstream inside plant bodies. It also mirrors the direct impacts of plants on redistribution of ecological forces, such as diffusing light, or grouping drip patterns of phyllosphere solutions loaded with nitrogen on the ground, or exposing their information
Figure 5-1: Hierarchical organisation levels of communities. Read from e) to a) to go from the smallest ecosystem (eco-unit) to the planet, from a) to e) to go from the whole Earth level back to communities (cf. Sect. 5-2). a) Biosphere, planetwide, in interaction with the solar system (Sect. 5-2). b) Continental level. Units are biomes, in interaction by macroclimatic and relief factors. c) Landscape level, perhaps equivalent to Blandin & Lamotte’s eco-complexes (cf. Fig. 5-2). Units are vegetation types (eco-mosaics), interacting by mesoclimatic and toposequential factors. d) Eco-mosaic level. Units are smallest terrestrial ecosystems, eco-units, interacting by reciprocal shading, wind-protection, rain sharing etc. Note different age and architecture of eco-units, conceived either as discrete units composed by organisms (left hand) or folds of a green interface, respiring, photosynthesizing and transpiring (right hand). Both concepts differ in the fuzzy limits between folds, rather discrete ones between eco-units. e) Smallest ecosystem, eco-unit. composed by organisms. Organisms of widely divergent sizes usually organized in smaller eco-units within larger eco-units, e.g. herb layer, rhizosphere, phyllosphere. Note that in the “folded vegetation” concept the crowns from folds of folds, reiterated crowns one more fold, and so on. The image is loosely fractal-like.
carriers, the seeds, to animals or wind. This double face of organic architecture, to the inside and the outside, can be exemplified at all system levels examined above.

**The arrangement of plants in an ecological community is as semantic as the arrangement of axes in a branched plant or nucleotides in a chromosome.**

We saw (Sect. 5.1.1) that this interactive, semantic arrangement of parts has coordinates and a lifespan. This makes it necessary to consider *ecosystem size and life cycle* as a determinant aspect of ecosystem architecture. In the following paragraphs we will therefore review small, simple relatively short-lived ecosystems (eco-units) as distinct from large, composed, maybe immortal, mosaic-like ecosystems (eco-mosaics) and their supersystems (eco-complexes).

These ecosystems will be regarded in two distinct ways (Fig. 5.1), omitting their root zones which we know nearly, but not quite, precisely enough. The first image shows the structural organisms composing an ecosystem, as exemplified by forest. The second image displays the “folded green blanket”, characterizing each ecosystem by its surface of exchange. The latter image rests upon the well-known property of organic systems to adapt or adjust their inputs and outputs of energy, nutrients, gases and water by folding the surface of exchange. This happens at the cell level (amoebas), at the tissue level (our spongy lung tissues), at the organ level (leaf shapes), and at the organismic level. We will find it in ecosystems too, the canopy and its exchanging phyllosphere being folded by the architectural framework of the component plant crowns. Both images are true, complementary and not mutually exclusive.

Examining ecosystem size and organization, telescoping emerges again. It is related to the degree of dependence of tiny ecosystems on large ones, e.g. of epiphyte communities on a forest canopy. Two similar shrub communities may exemplify this too. The one domineers herbs, the other is dominated by high forest. Van Rompaey (1993) indeed justified and designed a method for separated treatment of the giant tree community in Côte d'Ivoire. Symbiotic ecosystems, e.g. phyllosphere systems, hence are considered in the present book as *symbiosystems* of a field crop, forest, savanna or other ecosystem.

**A symbiosystem is a biotic community acting as a collective symbiont.**

Finally, ecosystem dynamics are incomprehensible without closely relating their origin to environmental impacts. This is a recurrent issue at all hierarchic levels of Life, from the close relation between genetic dynamics and stress to the relation between reiteration and traumatism and finally that between ecosystem dynamics and macro-impacts like storm, fire, inundation or earthquakes. Most environmental forces are natural. Factors manufactured by humans should not be overrated, as proven without any doubt by Lovelock (1988).

The words “disturbance” and “catastrophe” so are not used in these pages. Their meaning only derives from a comparison of “things as they ought to be” following some human text or plan, with “things as they are”. Our present text does not pretend to command the universe.

In the last chapter of the present book the consequences of this fact will be considered and linked to the title “Struggle of Life”.

**5.1.2.1 - The smallest ecosystems: eco-units and their phasic development**

In the present section, figure 5-1. retraces *growth and dynamics* of ecosystems and is to be read from bottom (5-1e) to top (5-1a). One so arrives at global climate and biogeographic zonation in figure 5-1a. In section 5.2. the figure retraces *environmental impacts and stresses*
and is to be read from top to bottom, from the sun’s impact (5-1a) to the level of a tiny, special site originating from a strictly local sequence of events (5-1d).

Figure 5-1e shows what happens, in general lines, on a surface of some 2 to 4 ares (1 are = 1 square decameter = 1 dam$^2 = 100m^2 = 0.01$ ha), cleared in some precise manner within a forest vegetation. In other words, there is one surface, and there is one impact at one moment.

**An eco-unit is defined as one ecosystem, developing on one surface cleared by one impact, from one specific moment on, and by one development process.**

As soon as one or more criteria change, the eco-unit ceases to exist. If there is more than one surface, or if the original surface splits up, the eco-unit is gone. If a surface is cleared by more than one event at more than one moment, no one, single eco-unit is born. If more than one process unrolls on the one surface cleared at one moment, the one eco-unit is no more. Time, surface and process are defined by fuzzy limits, in line with the rules of chapter 4.

The **surface** of the eco-unit in literature is called a “vegetation gap”. This is roughly similar to the early state of an eco-unit just after clearing. Much was written on exact limits of “gaps”. However, neighbouring eco-units interact, so gaps inherently lack sharp, “crisp” limits. These limits are biologically fuzzy, as shown in figure 5-1d and 5-1c and proven by Vester (1997). For mathematical treatment see Lowell (1994), Lowell & al. (1996) and Edwards & Lowell (1996). Any method to determine “exact limits” of vegetation gaps hence is empirical and can only be used for comparative purposes, like the arbitrary units of Patau used in our chapter 2.

The development of an eco-unit is presented in four conventional steps (Fig. 5-1e). These are youth, adolescence, maturity and decay, four phases distinguished by people since the dawn of history. These phases have an intrinsically fuzzy duration in time. For eco-units, they are outlined here on the base of more detailed descriptions by Oldeman (1990, 1994, 1997).

1 - **Youth**, or "seedling phase", or "initiation phase". Architecture is ephemeral and chaotic, built by herbaceous plants including herbaceous seedlings of woody species.

The canopy is short-lived, holed and highly dynamic. The fauna is rich in herbivores eating and exporting the fresh, young and tasty herbaceous mass, and importing seeds, spores, eggs and other packages full of biotic information. The wind also brings information carriers, directly and on the wings of birds and bats.

After an ecologically short period, often less than a year, the information bank of the young eco-unit stabilizes. It includes a heritage from the cleared-away former eco-unit, which often lost information by the clearing impact, particularly if fire, inundation or soil disruption did the job. However, the new information bank is enriched too by carriers like seeds and eggs, the travel of which was energized by vector organisms or abiotic vectors like wind or water.

Indeed, the eco-unit adjusts. Its direct environment **does not equal** that of the preceding eco-unit. The new eco-unit adjusts by selective interaction of actual vectors with the old propagule bank, inherited by the new unit. Key forces are selective animal attraction by "tasty" biomass (herbivore/plant; bat/insect; bee/nectar etc.) and propagule selection by the direct environment, allowing some few of them to germinate or hatch, to share in eco-unit building.

At this level, this mechanism at the same time **conserves** very ancient information and **adapts** the whole information bank by overwriting certain sequences carried by certain species. The young phase of an eco-unit is similar to, not identical with that of the preceding eco-unit.
2 - Adolescence, "growing phase" or "aggradation phase". Architecture with a low, closed canopy of mature shrubs and growing trees, other plants and animals in between, soil shaded.

The chaotic architecture of the young eco-unit here becomes more orderly, with only one structural ensemble of mature woody plants. This instructs all other organisms, except those trees potentially able to overgrow it and form a higher canopy. Germination is rare. So are animals. The cards have been dealt. The eco-unit obeys the instructions mobilized at the outset of its life. There usually is a lot of dead organic matter from former eco-units, decaying underneath the growing phase of the new one. Decomposer organisms like bacteria, mycorrhizal fungi, insects or worms, often in association, are numerous and active.

Decomposition deletes a lot of ancient genetic information, but not all, by the destruction of eggs, cuttings, spores and seeds. The low canopy filters light, rain and wind. The resulting microclimate is always heterogeneous (Koop 1989; Vester 1997). In the changing patterns, places remain where a few small plants and animals find enough resources to live.

In sum, the growing phase builds itself in compliance with an adapted set of building orders, and stabilizes the information bank by deletion of surplus reproductive material.

3 - Maturity, "adult phase" or "biostasis". Architecture complete, local maximum number of structural crown ensembles ("storeys"), ordered, long-lived and offering highly diverse biotopes to other organisms.

The longest-living tree components have come to maturity and attain maximum deployment of their crowns. They have built a pattern of highly varied sub-biotopes hosting countless biological species. The biological compartment "forest roof", structured by multi-arrayed, giant trees, and the compartment "root basement", structured by abundantly branched giant root systems and the organic layer covering them, are immensely rich in species.

Plant and animal assimilation is balanced by decomposition. Nutrients and water are mostly recycled, although no perfect cycle can exist due to the fuzzy nature of forest architecture and the rapid demographic change in partaking organisms, notably tiny ones. Forest architecture, its crown layers of mature trees interspersed with potential trees, climbers and other creatures forms a complex sieve for light (Vester 1997). Decomposition of specific litter liberates regulating molecules ("impellors", Oldeman 1990) triggering germination, or herb flowering, at specific dosages. Except for organisms in the outer canopy, none capture direct, unsifted abiotic stress impacts. "Major stresses", like radioactivity or polluted air may bypass the filter.

Abundant interweaving of ecosystems of different sizes and lifespans marks the mature phase. Inside the skeleton built by the largest trees supporting a high eco-unit, there are ecosystems built by shrubs, by patches of associated herbs, by epiphytes. Communities exist in epiphytes, like the algae, bacteriae, insects, frogs and snakes inside bromeliad leaf sheaths full of water. Rhizosphere and phyllosphere ecosystems are tiny, patchy ecosystems inhabiting a leaf or a few roots. Van Rompaey (1993) proved in W-Africa, that no analysis can deal with all species of all communities, so vastly different in size and lifespan but coexisting in one forest.

Altogether, the mature phase shows a fully adapted and fully grown architectural framework, many smaller ecosystems telescoped within, and an explosive increase of information in associated, more or less symbiotic, organisms or mini-ecosystems over a vast size range.
Decadence, "decaying phase" or "degradation". Forest architecture breaks down, exploding or dissolving into a loose assortment of diverse, smaller ecosystems.

The architecture of the mature eco-unit is lost for various reasons. There simply is the death of old tired trees, unable to respond well to stresses any more, except by spotwise somatic or sexual meiosis, as a last recourse. There are many impacts too, from fire, wind, soil cohesion weakened by rain, to epidemics, which cause a slow or rapid collapse of a mature eco-unit. Conforming to the rule of transfer of functions (Sect. 4.4.3) the organization of the remains of the eco-unit is transferred to the assortment of loose, small ecosystems left. Figure 5-1e (right-hand side) shows some of these as more or less anarchical green folds.

The longer the decadent phase, the stronger genetic diversity is boosted. The increase of dead organic soil mass stimulates soil flora and fauna. The number of epiphytes, herbs and lianes is passingly larger and their species composition richer. Food is more diverse, animal diversity augments, and with it the range of “passengers” on animals, such as seeds, insects, or spores. In a Brazilian case, a third of the insects in decaying phases were omnivorous ants (Akker & Groeneveld 1984, ex Oldeman 1990). The earlier filtering functions of the mature eco-unit become disorganized. Hence, many remaining organisms are subjected to new, strong forms of stress. The propagule bank of the next young phases is largely prepared during decay.

In summary, the decaying phase shows disrupted architecture, transfer of functions to smaller, loosely connected ex-component ecosystems, and much dead biomass. Component species in increasing numbers add information to instruct the next eco-unit (or eco-units!) to be built.

5.1.2.2 - Large, compound ecosystems: eco-mosaics

The level including eco-units as building blocks marks the organization of larger ecosystems or “communities of communities”. Figure 5-1d shows different kinds of eco-units, one being displayed in all its phases. The latter is similar to the kind of smallish eco-unit shown in 5-1e.

An eco-mosaic is an ecosystem inhabiting one class of similar sites and is composed by various kinds of interacting eco-units in various development phases.

An eco-mosaic can be observed as a snapshot at any one moment, but human lifespan falls short of observing its complete development. Koop (1989) found traces of clearcuts in the XIVth Century, still visible in today's forest architecture of the 200 years old Forest Reserve of Fontainebleau. In the Polish “virgin forest" of Bialowieza traces still remain of the armies of Napoléon (1812) and Wilhelm II (1914-1919). In French Guyana, architecture and flora of the forests on the Orapú River, on sites once covered by XVIIIth Century Jesuit plantations, provoked a logging spree 200 years later, aimed at the abundant kwalli wood from vochysiaceous late pioneer trees (Oldeman & Fundter 1986), indicators of long ago secundarization.

Moreover, millenary developments always take place in changing environments. The sun is no constant energy supplier over millennia (cf. Sect. 5.2), people alter the fauna (Vera 1997) and tinker with synthetic chemicals, and the composition of the air is liable to be readjusted, for instance by ecosystem fires (Lovelock 1988). The impetus of change on land is carried by rain and rivers to the continental shelves, stressing marine ecosystems, also affected directly by oceanic currents (Sect. 5.2), like terrestrial ecosystems by the atmosphere.
Eco-mosaics never are identical to earlier eco-mosaics, although often similar. The concept of succession as currently used hence is left out of this book. “Succession” would lead to some predictable ecosystem state(s). This is shown in chapter 6 to be inherently impossible.

The long-term development of eco-mosaics can only be tentatively pieced together from the snapshots we may observe during our short lives. It can not be experimentally investigated. Thomson (1997) points to such lack of satisfactory scientific proof of evolution, comparing it to a murder investigation where the evidence of “the smoking gun” is wanting. Perhaps we need a monastic order with a millenary charter. Its members would certainly find that there is no “undisturbed development” at this mega-level, just as there is none at the micro-levels of nucleotides and chromosomes.

Reconstructions over thousands of years are attempted by using historical texts (Koop 1989; Vera 1997) and pollen analysis (Van der Hammen & al 1991), and extrapolated to the future as hypothetical “potential natural vegetations” (Van der Werf 1991). Clearly, such fragmented, incomplete and maybe lopsided databases lead to different reconstructed images. All are true to some extent and all have elicited differences of opinion. A typical discussion concerned Vera’s book (1997). The lack of computer modelling of the millenary processes he described was criticised. In fact, computer models are no less determined by inference (Thomson 1997) than descriptions. Neither can be validated experimentally or by independent observation.

However, we can be sure of some main lines in eco-mosaic development. More visibly than at other scale levels it depends on the initial state of the system (Prigogine & Stengers 1984). During periods of similar bioclimatic conditions, probably no more than a century or two at most (Sect. 5.2), eco-mosaics can conserve a fuzzy state of dynamic constancy. And finally, the essential processes in eco-mosaic dynamics are fragmentation and fusion of eco-units.

* The initial state of an eco-mosaic. Eco-mosaics may start on sterile, lifeless terrain, or they may inherit the site of an earlier vegetation. The first condition is rare. Instances are montane subsoils denuded by landslides, lava deposits, or the seafloor pumped dry in the Dutch polders. Colonization here starts with micro-organisms, followed by small green plants. On pure rock, a low vegetation with leathery leaves slowly develops, for instance the Bromeliads on Guyanese inselbergs (De Granville 1978). On normal but virgin soil, and with people as vectors of diaspores, quite a rich pioneer forest may grow up in decades, like in Flevoland (The Netherlands), out of which the Zuiderzee water was pumped in the 1950’s.

However, eco-mosaic development currently occurs on incompletely cleared, huge sites. Even after fire, bulldozers and heavy tropical rains, there always is biotic remanence in the form of seeds or spores of very hardy plants, and eggs of hardy insects. Nomad species blow in on the wind, or are flown in by animals. Nomad species grow rapidly, individually, as “weed trees”, or collectively, like insect populations. They reproduce prolifically, their seeds or eggs thrown everywhere. Thorough clearing makes a huge eco-unit built by pioneer trees as a first step in eco-mosaic development (cf. Kahn 1982, on abandoned fields in Côte d’Ivoire).

Figure 5-1c shows toposequences in a hilly landscape. These are sequences of land types more or less “hospitalable” to vegetation. The fuzzy notion of hospitality is empirical, but there is no realistic alternative. Unless one site factor dominates, like water in a marsh, many “site
factors” are apt to interact. Measuring them all, computing all interactions, monitoring their values, and designing a computer-aided dynamic simulation model costs time, brainpower and money, against little gain in meaningful precision (cf. Van der Sleesen 1994).

The toposequence of figure 5-1c shows plains, slopes, tops and valleys of different hospitality. This is due for instance to slope, available water, erosion-proneness, soil depth and exposition to the sun during its daily round, under an angle depending on season and latitude. Each “forest type” in the figure is composed by a slightly different retinue of species, due to seedling selection in each site. However, pioneer eco-units and pioneer trees are flexible and can adjust to ample differences within a site class (De Rouw 1991, her Chapt.10; Vester 1997).

The initial state of an eco-mosaic then depends upon its fuzzy site class and its past history.

The site class, if analyzed too precisely, is a single geographical point. Only if analyzed less precisely, or assessed by ranking of “hospitality”, it englobes points with similar but unidentical properties. Crisp site classes hence can not exist, because they are inherently fuzzy. Soil taxonomy or forestry site classifications remedy this. If a class is too fuzzy, it is divided in several smaller classes. This procedure is the same we saw in the last chapter (4.4.1). In terms of fuzzy logic it is called “defuzzifying” (but also see Kosko 1993).

The past history of the cleared surface, especially the information carriers inherited from that past, often tells more about eco-mosaic development than site class. Eco-mosaic architecture depends on the variability of the inherited diaspore set. A heterogeneous set usually includes bunches or patches of propagules which germinate or hatch together. Each bunch initiates a young eco-unit. As said, each development phase of an eco-unit contains distinct information. Below, we will see the same for different kinds of eco-units, large pioneer eco-units having a flora and fauna which strongly differ from small intruder eco-units.

The usual initial state of an eco-mosaic is determined by its biotic history rather than by the abiotic site properties.

The normal initial state of an eco-mosaic already contains a mosaic of young eco-units, large or small, fast or slowly growing, species-rich or species-poor. Their initial state is very unstable. Some of these eco-units are selected to share in the building of the adult mosaic.

From the initial state on, when the mosaic “does not know what it wants to do”, it develops towards a more stable state, the form of stability of which is increasing resilience. Once the initial eco-units start developing, the architectural eco-mosaic pattern (“texture”, cf. Barkman 1979; Edwards & Lowell 1996) is fixed for a longer period. In the next paragraph we will follow the generation of an ever more stable pattern, oscillating around a fuzzy average state.

* The state of dynamic eco-mosaic constancy or equilbrium (spelled with c).

The initial state of eco-mosaic development contains different kinds of young eco-units, as seedling patches combining herbaceous, shrubby and woody plants. At the very start one can only check the mosaic-like nature of composition and local development forecast by seedling identification at a quite detailed scale. Biological homogeneity of pioneer vegetations is a myth, not borne out by the facts, except in monocultures made to be monotonous.

Normally, the mosaic emerges in a year or so. Many of its eco-units are dominated either by herbaceous species - grasses, forbs - or by young pioneer shrubs or trees. These grow fast, so
the patchy architecture of this highly dynamic mosaic is accentuated soon. In the wet tropics, the first pioneer eco-units mature in five to ten years and may be five to twenty meters high. The trajectory from young to mature in this kind of woody eco-unit hence occupies a decade or two only. This is the kind of eco-unit in the middle of Figure 5-1d, in the middle.

The kinds of eco-units are summarized in the following Table 5-1

Table 5-1. - Kinds of eco-units, summarized from Oldeman (1983, 1990). Figure 5-1e illustrates some kinds. Short and long pioneer eco-units have a short or long lifespan. Borders are relative to total surface. Plug eco-units plug themselves in medium to small openings. A narrow plug eco-unit is a minimal unit, its fuzzy borders reaching to its center. Structural layers in mature eco-units are sets of fully expanded (mature) tree crowns.

<table>
<thead>
<tr>
<th>Eco-unit ⇒ Attribute</th>
<th>short pioneer</th>
<th>long pioneer</th>
<th>large plug</th>
<th>plug</th>
<th>narrow plug (minim. unit)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Surface</td>
<td>large</td>
<td>large</td>
<td>medium</td>
<td>small</td>
<td>minimum</td>
</tr>
<tr>
<td>Height</td>
<td>low</td>
<td>later very high</td>
<td>medium</td>
<td>high</td>
<td>various</td>
</tr>
<tr>
<td>Lifespan</td>
<td>short</td>
<td>very long</td>
<td>medium</td>
<td>long</td>
<td>(very) long</td>
</tr>
<tr>
<td>Relat. border</td>
<td>crisp</td>
<td>rather crisp</td>
<td>fuzzy</td>
<td>very fuzzy</td>
<td>small border</td>
</tr>
<tr>
<td>Initial condit.</td>
<td>open</td>
<td>open</td>
<td>large gap</td>
<td>smallish gap</td>
<td>small gap</td>
</tr>
<tr>
<td>Growth rate</td>
<td>very fast</td>
<td>fast, slowing</td>
<td>intermediate</td>
<td>slow</td>
<td>very slow</td>
</tr>
<tr>
<td>Struct.layers</td>
<td>one</td>
<td>two or three</td>
<td>three</td>
<td>two or three</td>
<td>various</td>
</tr>
<tr>
<td>Herbs</td>
<td>many</td>
<td>few</td>
<td>some</td>
<td>few</td>
<td>very few</td>
</tr>
<tr>
<td>Lianes</td>
<td>herbaceous</td>
<td>herb/woody</td>
<td>woody</td>
<td>woody</td>
<td>woody</td>
</tr>
<tr>
<td>Animals</td>
<td>insects‡</td>
<td>generalists</td>
<td>varied</td>
<td>very varied</td>
<td>specialists</td>
</tr>
<tr>
<td>Sp. richness</td>
<td>low</td>
<td>rather low</td>
<td>rich</td>
<td>rich</td>
<td>very rich</td>
</tr>
<tr>
<td>Biomass</td>
<td>low</td>
<td>high</td>
<td>medium</td>
<td>rather high</td>
<td>rather high</td>
</tr>
<tr>
<td>Stresses</td>
<td>rather direct</td>
<td>indirect</td>
<td>filtered</td>
<td>strongly filt’d</td>
<td>strongly filt’d</td>
</tr>
</tbody>
</table>

An initial forest eco-mosaic is composed mainly by short and long pioneer eco-units, some plug units plugging in somewhat later in gaps where no pioneer trees occurred, or where some died. Participation by trees depends on their temperament (Oldeman & Van Dijk 1991, Oldeman & Sieben-Binnekamp 1994, Vester 1997), i.e. their set of reactions to their direct biotic and abiotic environment. Pioneer trees love light, resist stress, and produce abundant seeds, often carried by wind, birds or bats. Many grow fast and have a short life, e.g. “weed trees” like Eurasian birches or S-American balsa. They also may show fast early growth and a slower growth later, like Californian *Pinus aristata*, the oldest tree on Earth, or Asian teak.

The different growth rates, the genetic variability, the lifespans that vary normally as in all species, make that the eco-mosaic rapidly displays a very diverse composition. The height, architecture, species composition and biomass of the component eco-units soon become widely different. With this architectural diversity of the mosaic, the interaction between eco-units becomes more important. They filter light, wind, precipitation and migrating animals, and so put their neighbours in sun or shade, in the wind or alee, in seed scarcity or abundance.

Flying low over the Guyanese rain forest in a small airplane, it is indeed visible that narrow chimneys are open from the canopy level to the ground, without any plants occupying them. Narrow openings usually are filled with species belonging to “late plug” eco-units, with a struggler temperament characterized by slow growth, resistance to crowding, regular sparse fruiting and interaction with specialized animals. If such openings are too narrow, however, they remain empty and are sometimes filled by expanding crowns from adjacent eco-units.
**We define the minimum eco-unit** as the eco-unit which contains the smallest central zone which can still allow and select germination and hatching processes.

The minimum eco-unit was often conceived as a phenomenon at the end of succession when climax forests have tree-wise replacement or notions to that effect (e.g. Oldeman 1983). We may see now that at this community level, like in trees, **bordering** by minimum units may occur from the outset at the periphery of large units (Fig. 5-2d). Such small eco-units are stressed, e.g. because of obscurity, far red light, water interception or dense root mats. They indeed become foci of adaptation and speciation, due to activation by stress of mechanisms at all levels described earlier. We may speak of **eco-adaptation**, because the community adapts.

We may include *long and narrow eco-units* ("shoestring eco-units") among the minimum eco-units at the borders between larger pioneer eco-units. This explains the richness generally found in "mantle vegetations" or "border vegetations", even between plantations or along roads. Along roads or fields the stress factors of course differ from those in the forest. In historical land use by Central American Mayans or Carolingian farmers in Europe, hedges fulfilled this function, in between epotomic pioneer vegetations, i.e. agricultural fields.

Shortly after its initial state, an eco-mosaic hence often is composed by interacting eco-units of both quite large and very small sizes. Agents of interaction are, as usual in biological communities, the animals. The state of the eco-mosaic changes during its development. It passes through two states, the *preequilibrium* and the *equilibrium*, defined by Oldeman (1990).

**Precequilibrium** means pre-eco-unit-equilibrium and **equilibrium** is eco-unit-equilibrium.

Both terms are intentionally written with the *c* of eco-unit.

They denote states of an eco-mosaic, so in the present book they are stripped of their former temporal dimension as "stages" (Oldeman 1990). They become purely architectural, dynam-ics being a series of states at progressive moments. The preequilibrium is roughly comparable to an adolescent state, leading from the initial state to the equilibrium.

This happens by the eco-mosaic changing under the unremitting impact of a series of heterogeneous abiotic forces. These are sometimes classified as "disturbances" in geometrical size classes (e.g. Shugart 1984, his Fig. 10.1). However, an eco-mosaic undergoes in any year large and small impacts out of a continuum of intensity, focus, energy involved, duration and abiotic causes. A thunderstorm is a unique, momentary combination of wind, electricity and rain, focussed upon a unique spot of fuzzy size, with a certain soil, during an unpredictable timespan. Eco-mosaics develop under an unceasing bombardment by ecological grenades loaded with highly diverse explosives and impacting over a wide range of surface and mass.

This bombardment is *not a disturbance*, it is a normal force of nature, just like the bombardment with ions from the sun at another scale (Sect. 5.2). All organic systems live under stress.

Eco-mosaics develop from an initial state to a fuzzy state of dynamic balance, the equilibrium. The first state is caused by a rare, giant impact and the transition is towards a state, being the *preequilibrium*, marked by a quite long period of small and medium impacts. This is a dynamic process, but no succession in the usual sense of the word, because *everything may succeed to everything* (Hallé & al. 1978, their Fig. 110 on "silvigenetic cycles"; Oldeman 1983)

In each opening caused by some impact, a plug eco-unit develops. The size of the opening requires a large plug, a plug or a narrow plug. The plugging unit never is one organism, e.g. a tree. An eco-unit is no organism, but one small ecosystem built by many organisms of many
species, even if there is only one tree among them. The limit between a minimum eco-unit and a tree species with its symbionts is as fuzzy as that between a DNA strand and a plasmid.

The processes in the precultivarium state often are the ecological equivalent of intercalation in branching. There is a plug eco-unit in an eco-mosaic for every ecological requirement, like a branch order in trees. In case of the minimum eco-unit biological variation builds up that can be mobilized if acute stress occurs. With time, the number of minimum eco-units increases, as small-scale impacts create small openings very frequently. Medium-sized plug eco-units also increase in number, in two ways. First, medium-sized impacts, regular but not very frequent, create medium-sized openings, plugged by medium-sized eco-units. Second, adjacent small plug eco-units, once grown up and mature, merge into one functional eco-unit (e.g. Peters 1992, 1997, Northern beech). The surface covered by pioneer eco-units hence decreases.

Huge pioneering impacts, like the earthquakes in Panamá and Papua New Guinea (Garwood & al. 1979), the eruption of Mt. St. Helen in 1980 (Findley 1981) or the cyclones of Eastern Australia (Webb 1958), periodically create an initial eco-mosaic state. Balancing out of this eco-mosaic under the usual assault of lesser factors is the essence of the precultivarium state. The resulting eculvilibrium is a mosaic composed by different kinds of eco-units in rather constant proportions and always with fuzzy limits. Constancy is a mean. If the average abiotic environment remains the same, every decadent eco-unit is replaced by a young eco-unit of the same kind elsewhere in the eco-mosaic. An elegant term is kaleidoscopic stasis.

- The precultivarium is the state of an eco-mosaic of which the eco-unit composition is not yet balanced out by the abiotic environmental impacts.
- The eculvilibrium is the state of an eco-mosaic of which the eco-unit composition is in balance with the abiotic environmental impacts.

With this image of forest development we are close to the “canopy types at different stages” of whole forests (in our terms: eco-mosaics), as advanced by Webb (1977). However, this author placed these canopy types in a context of “succession”. In the present book, the different kinds of eco-units do not appear in any privileged order in time. All may be present from the initial state on, or different kinds may appear in any order during precultivarium. The ecological dynamics involved hence have little to do with species composition. The forces building complex environments on the contrary open or close niches for species. In the following section an outline of these processes is given.

* Eco-mosaic dynamics: eco-unit fragmentation and fusion, and transfer of functions.

The challenge of ecosystem study is that ecosystems are no organisms, but nonetheless they are organic systems. Particularly at the level of the eco-mosaic, their direct environment is very little filtered. Eco-mosaics undergo direct impacts of raw external macroclimatic factors. They are immediately exposed to harsh radiation, untamed winds, crude rain, earthquakes and the like. Still, their manner of reacting to major and minor stresses follows the lines we saw at other organic levels in the preceding chapters.

At the nuclear level, the onslaught of all impacts is met by the activation of the right set of nucleotides in the chromosome. At the level of the whole plant, the right set of meristems is activated, or the right set of branched arrays in big trees or corals. At the eco-unit level the awakening of selected species ensures the building of a collective organic system in a direct environment well filtered by surrounding eco-units. Finally, the eco-mosaic mobilizes the right eco-units to face the music.

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We saw in the preceding paragraph that the initial state of the eco-unit contains many large pioneer eco-units, with smaller ones plugged in here and there. This principle is similar to the big trunk with a few twigs in a tree sapling, although the architecture and ecophysiology are quite different. Even if we admit that the various species are less isolated than we once thought, being linked for instance in symbiosystems, *an ecosystem is no organism.*

Are ecosystems subjected to internal stresses?

This question is inescapable, because of the notions of pests and diseases. Food pyramids are taken for granted, but the concept of illness caused by pathogens and parasites is deep-seated in our medicinal and agricultural thinking. The next example suggests another viewpoint.

In the Northern-German sand plains, a storm had downed most trees of an old *Pinus sylvestris* stand, shaking loose the roots of the surviving trees. The next year, they were massively attacked by a well-known pine pest, the insect *Tomicus piniperda* (Scolytidae). This animal “shaves” the pine crowns by hollowing out and breaking off the young needle-bearing shoots. Said Brünig (pers. comm.) who showed us this stand: “Fortunately, the insects reduced needle transpiration, so the roots could recover and the trees survived well”. Indeed, an insect species here boosted resistance of the *eco-unit* to the aftereffects of the storm. Indeed, the eco-unit under stress responded by shifting from wood production to insect biomass production.

In the process, the eco-unit did not remain one eco-unit, but is was divided in a few surfaces under old, surviving trees, and several other, large surfaces with young pioneer characteristics. In other words, the original eco-unit became an eco-mosaic. The ecological adjustment of the older pine eco-units was operated by *T. piniperda.* The development of the young eco-units was operated by seeds, including many from grasses (Gramineae) or heather (Ericaceae).

When roaming in ecosystems faraway from people, including some tropical rain forests or coral reefs, dead or weak trees, corals or other animals are common, their sickness induced by micro-organisms, fungi or insects. Barkman & al. (1983, p. 58) stated “In a healthy forest there also are diseased trees”. This leads us to remark, first, that a scale aspect is evident, and second, that the vision of diseases and pests as something wrong is a human artefact.

The list of direct environments in section 5.1.1 is one element to explain the scale issue. The second element is, that the interactions we call “diseases” are shifted in the hierarchy and take place between cells, i.e. body cells of a large organism and unicellular or paucicellular bodies of microbes. The third element is, that *at its own scale* an eco-unit grows as its environment requires. Life and death of component species are mere tools in ecosystem dynamics.

Eco-mosaics and eco-units hence have *no internal stresses.* Only their components, or the components of components may be stressed at their own level. If the adaptation to stress is to be understood, the notion of “horrible” disease is too anthropocentric to count in biology.

As a rule, natural command substances are biodegradable and specific. They are regulators at one level, poisons one level lower (Oldeman 1990: 372 ff.). Sesquiterpenes, for instance, like *abscissin* and *dormin* drive leaf abscission in plants under seasonal stress. These substances are regulators of the whole plant, but poisons for the leaves. In autumn, or when a dry season begins, plants systematically poison their leaves, which become “ill” and fall off.

Synthetic, man-made molecules usually are little or not biodegradable, too often are barely specific, and are overdosed, so becoming killers or stressors instead of regulators. They kill agrosystems, rather than adjusting them by selective, timely thinning or culling of precise
components. Moreover, when introduced by \textit{H. sapiens} they are \textit{external} stress factors, because they have no natural equivalent at the particular site, time and biological scale level.

\textit{With the above we demonstrated the unity of the hierarchy of living systems, because at any level the statement is true that chemical and other command factors produced by a system regulate that system, whereas one level lower the same factors put systems under stress.}

The level from which immediate stress falls upon eco-mosaics is, as we saw, macroclimatic. Some filtered impacts do exist, as outlined in the next section on landscapes. However, the stresses eliciting an immediate response from an eco-mosaic are the climatic ones.

Their main effect is to eliminate parts of the mosaic, making large or small gaps in the green blanket. Every gap is a potential young eco-unit of one kind or another. The climatic impacts hence \textit{fragment} part of an existing eco-mosaic, one eco-unit often being replaced by several eco-units. The impact factors are not incidental in that they are just a regular property of the direct environment of any eco-mosaic. However, the energy, size and duration show wide variety, which may be analyzed as either stochastic or fuzzy variability.

The fragmentation of the eco-mosaic is a continuous process which may be visualized as a green carpet being eaten by moths (Oldeman 1978a), every moth hole being mended with a cross-stitch. The carpet at first is quite homogeneous, but with time the frequency of smaller and larger cross-stitches augments. They display a pattern similar to an increasingly complex piece of embroidery. Later their density increases. Some bigger holes also are eaten in the carpet by whole moth families. The embroidery starts being self-covering and repetitive, the untouched parts of the carpet disappear altogether. In this dynamically constant state, the carpet lies on the ground until someone throws it in the fire, an analog to some large, intense, climatic factor sweeping a large tract of land clear once more.

\textit{Fragmentation of a living system is its being divided up by outside influences in parts which are viable in themselves as old or young living systems.}

The presence of “outside” influences excludes sexual multiplication from this definition, unless the role of a mate or pollinator is considered to be exterior. Only in this form is the definition universal. Including sexual multiplication would exclude ecosystems. The present definition so differs from earlier ones (Hallé & Oldeman 1970, Hallé & al. 1978).

Fragmentation is universal in Life, because no two system \textit{components} are ever absolutely identical and because the \textit{impacts} from the direct environment never are absolutely regular.

Eco-unit fragmentation transforms an eco-unit in an eco-mosaic. \textit{Component} organisms differ
- in their genetic buildup, also intraspecific, e.g. their responses to stress and architecture
- in their age expectation, no population having members with identical lifespans
- in their growth rate, no population having all members growing equally fast or slowly

Eco-unit fragmentation patterns differ because the environmental \textit{impacts} on ecosystems are
- of unequal energetic impact, e.g. storms (Beaufort scale) or earthquakes (Richter scale)
- of unequal impact in space, e.g. lightning (small surface) or snow (large surface)
- of unequal impact in time, e.g. cold (frost from one night to one winter) or light (moon phase)

To make the image more complex still, both the variations “below” in component species and the variations “above” in climatic factors interact. There are thunderstorms and snowstorms, depending on the factor interacting with wind. However, everything is not liable to interact
with everything else. A thundersnowstorm, for instance, may perhaps exist but certainly is not
common. Plants of the same species but with a flexible genotype, for instance those able to
grow into either a tree or a climber, were exemplified above (Fig.4-9). In either case they
interact with other species in their eco-unit, but in a different way.

The above description emphasizes eco-mosaic dynamics, suggesting fast change throughout,
the equilibrium being distinct only because changes balance each other out. Calibration of
these processes can be done against a simple variable in time, i.e. the average period needed by
a structuring plant to grow up to maturity. In seasonal vegetations like arctic steppes this basic
time span is one summer, i.e. a few months only. The same timescale applies to crops. In
pioneer forests it ranges from 5 years in the humid tropics to 50 years in the temperate North.
In tropical rain forests most authors concur with a timespan in between 80 and 120 years for
total average turnover (see Oldeman 1990). A quarter or a third, i.e. 20 to 50 years, would be
the time needed by an average tree to grow to maturity.

Now this fits in well with the frequency of larger impacts upon eco-mosaics in the tropics. In
Indonesia, historical documents suggest that there are major droughts or fires every 40 years
or so (Van Eijk-Bos in prep.). The same frequency of drought and fire we encountered in parts
of Southern Venezuela and in South Carolina (USA). Oldeman & Van der Meer (unpubl. data)
point to a cyclone impact frequency in between 20 and 40 years in Northern Queensland
(Australia). In section 5.2 such fuzzy periodicities are examined from the side of the climate.

However, the eco-mosaic does not only fragment.

Eco-units are growing. They have the time to grow up to maturity and they often may then
remain in that state and of that size during numerous years. Peters (1992; 1997.70) found that
there were huge age differences in one Japanese beech stand between similarly-sized Fagus
crenata trees. He analyzed the mechanism of these differences by counting periods of
suppressed growth and free growth, and showed that trees and groups of trees had different
chronologies. Their ages ranged between 120 and 280 years.

The oldest of such groups, i.e. the oldest eco-unit (Oldeman 1990) reached the mature size
first. The surrounding eco-units perhaps started later as plug eco-units in the eco-mosaic, then
to overtake the first large eco-unit later. The result is, that in an 80 years old forest with eco-
units needing 40 years to grow up, there are eco-units with ages in between 40 and 80 years
which together compose one canopy at the same height. The eco-units have merged together in
a kind of super-eco-unit which unavoidably falls apart later in smaller eco-units again.

Fusion of living systems is their being merged together by their own growth dynamics into one
larger living system which is functionally viable but architecturally unstable.

Ecosystem levels are not the only ones with fusion of living systems. A good example at the
level of branching systems in plants is provided by the inflorescences of many Compositae
(Asteraceae), of which the miniflowers fuse into one functional “superflower”.

Eco-unit fusion transforms an eco-mosaic in one functional eco-unit. Component organisms

• as species, belong in the same eco-mosaic, e.g. by their architecture and response to stress
• as structural organisms, have ages that allow long crown coexistence when mature
• as builders, vary in growth rate at one ecological moment so the one may overtake the next

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An ecological moment is defined by the time gone by since the initiation of an ecosystem. Eco-unit fusion temporarily homogenizes patterns of the upper eco-mosaic canopy, under an environmental impact régime

- in a low-energy impact range, not capable of opening new eco-units in the mosaic
- with small impact sizes in space, not capable to eliminate structural organisms or crowns
- showing calm impact dynamics over long years, allowing long-term crown development

A fused forest eco-mosaic, seen from the ground, shows widely divergent heights of the first big branch forks among the canopy trees. Trees with low first forks are younger than those with first forks high up in the crown. Large age differences among the trees correspond to a wide height range of the forks, often indicating previous fusion. Canopies originating from fusion are not too stable and relatively short-lived. The oldest eco-units start to die back and so the original fragmented nature of the eco-mosaic becomes visible again.

However, functional, merged eco-units may remain fused during several decennia, which is long in human terms. This explains why fused eco-mosaics, with their all-but indiscernable eco-units, contributed to the myth of the “monotonous climax”. European beech forests, the prototypes of Rousseau’s “green cathedrals”, are typical cases of a misleading image of eternal stability. Another example are old coppiced oak stands in the French Gâtinais region (Fig. 4-14A), where bark structure often indicates trunk age.

The state of the organic soil is a diagnostic property of eco-units too. In detailed studies of the humification process during forest development, Bernier & Ponge (1994) found this to be the case in montane forests of Norway spruce in the French Jura, and Ponge & Delhaye (1995) in the “virgin” beech reserves in Fontainebleau. The organic horizons change regularly with the development of an eco-unit. The kind of unit they allow to grow up depends on the phase of organic soil development at the moment of the impact clearing the old one away.

This information may serve foresters planning to log and regenerate timber stands at the right moment. Logging impacts indeed resemble heavy, rare impacts at the macroclimatic scale.

However, the state of the organic soil ecosystem as an eco-unit compartment is particularly significant in order to grasp the coherent processes of life binding together the organisms building the eco-units. The ecological moment of interruption of an eco-unit building process determines the following step in the sequence of life. The selection of information carriers after the clearing impact largely is operated by the organic soil system. More precisely, it is determined by the state of this subsystem at that precise ecological moment. Ponge and his coworkers clearly demonstrated that the transfer of information in these cases is boosted by earthworms. The information mobilized is quite different at different development phases.

Introducing his Doctor’s thesis, Bernier (1995) gives master diagrams of the development paths initiated in this way in the marshy Norway spruce forests of the high Jura. At each altitudinal level in these mountains, the mechanism is slightly different. However, at all three altitudes studied, a logged-over forest follows one out of two pathways. First, the organic layer may allow initiation of the same spruce forest. Second, however, if the organic layer is opened up at another ecological moment, a low, shrubby bilberry heathland grows up. The first sequence is the recurrent sequence of the forest. The second sequence is a memory from the past. Heathland is a biologically poor pioneer mosaic on exhausted or otherwise barren lands. Everything happens as if there existed either a repair sequence, or a sequence activating
an ancient response to stress, two options we encountered before at the DNA level. A third possibility, considered by Lamotte & Blandin (1985) is a wholly new sequence.

The movements of fragmentation and fusion of eco-units represent a swing between the eco-unit and the eco-mosaic level. This swing is linked to transfer of functions between both levels, a phenomenon we encountered earlier (Sect. 4.4.3; see glossary).

In an eco-mosaic, microclimate is filtered at the level of the eco-unit by the horizontally arranged sets of crowns at different heights. At the mosaic level, filtering works by interaction between eco-units acting as light screen, windbreak, rain shadow etc. Fusion shifts the whole function of microclimate filtering to the “functional eco-unit” level, that of the eco-mosaic. Transfer of microclimate regulation so occurs from the eco-unit level to the eco-mosaic level. Fragmentation transfers microclimate regulation back again to the eco-unit level.

However, the information functions remain at their original level, as we saw above. This is caused by the organic soil compartment, hosting the roots and their symbiosystems. It also is due to all other compartments of non-structural plants and animals having become installed in every eco-unit in the course of its history. At the level of structuring organisms, often trees, there may be fusion among eco-units, causing transfer of certain functions. The same is not necessarily true for other organisms and their mini-ecosystems (e.g. see Wolf 1993 for beautiful descriptions), which differentiate the eco-units, also when the latter are fused.

The level of the eco-mosaic is probably the highest level at which the distinction between a living system and its environment makes sense. One still can tell rather well where the limits of the ecosystems are, although they are quite fuzzy. Hence it can be determined rather precisely which are the structures and processes inside and outside an eco-mosaic. Once this can be told, selection paths can be traced down, from organization level to organization level.

From the propagule bank, with its great diversity of genetic instructions stocked in diaspores to be selected, through the selection of minimum branched arrays and, next level down, leaf-plusses or leaf-minuses, down to the initial ring in the meristem, to cell bodies like chloroplasts and finally the genetic material the sequences, biological clasps and selection mechanisms can now be recognized as the authentically and purely biological mechanisms they are.

The next paragraph spans the gap between the biotic and the abiotic by the fuzzy bridge of landscape ecology, where the situation is partly biotic, partly abiotic, without much inherent distinction between the stress and the stressed.

5.1.2.3 - From eco-mosaic to eco-complex, biome and biosphere

A classical dilemma in forestry is forest site definition. Which are the “growth factors” to be treated as permanent and which are the fickle ones in planning silviculture (cf. Fanta 1986)? This dilemma was expressed as a scientific problem by physical geographers like Bertrand (1982), using the Russian notion of geosystem to tackle the problems at that wider scale. Their discussion is largely the same as the one questioning the dimensionless ecosystem, launched in the 1970’s by Oldeman (1974:14-15) and continued by authors like Lamotte & Blandin (1985), Blandin & Lamotte (1988, Fig. 5-2) or Blandin (1992).

The climatic impacts, which are direct eco-mosaic stressors, indeed vary at a time scale close to human life. Human societies are exposed to the weather in the same way as eco-mosaics,
without the means to influence the impacts and without other recourse than continuous building of their home (οικος) by reproducing, under stress, their community structures.

A *landscape*, say the dictionaries, is the composite of the land forms in a region, or is our surrounding world as seen from one spot. Landscape stress factors are in space rather than in time, i.e. the mountains and valleys, the riverbeds and sedimentsed plains, the land and the sea. Of course these features change, but so slowly that for all *practical* purposes we may consider them to be constant. This pattern hierarchy starts at the small end with the toposequences we examined earlier (Fig. 5-1c) and leads to regional geographies (Fig. 5-2).

As stressors upon life, landscape factors are constant over long periods. They operate by regulation of atmospheric and marine dynamics. Water goes downhill, eroding the landscape upstream and sedimenting new lands downstream. Abiotic transport of propagules tends to go downhill, so propagule bank dynamics are asymmetrical. The effects of mountain chains on the unequal distribution and varying nature of precipitations are asymmetric too (Cao 1995).

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**Figure 5-2**: Eco-complex, example in the savanna of Lamto, Côte d’Ivoire, West Africa, at the border between the rain forest and savanna biomes. Each vegetation type is an eco-mosaic (see Fig. 5-2d). Courtesy Dr. Patrick Blandin, Muséum National d’Histoire Naturelle, Paris.
Ecosystems built by several eco-mosaics, coexisting and interacting within a creek or river basin, which is the tridimensional expression of a toposequence, form an kind of super-ecosystem called an eco-complex by Blandin & Lamotte (1985:554). These authors define it simply as “an ecosystem of ecosystems”, specifying that an eco-complex shows emergent properties, i.e. properties not present in a simple sum of ecosystems Figure 5-2 taken from these authors shows, however, that human land use added heavily to the emergent properties, as confirmed by the analysis of forest land use in Québec by Bouchard & Domon (1997). However, the present authors prefer to limit this to one or a few levels above the eco-mosaic.

The hierarchy of collective living systems then is marked by the eco-unit, the eco-mosaic, the eco-complex and higher levels like biome and biosphere. It may be convenient to delimit collective levels in between the eco-complex and the biome, but for the moment we can not define them. Biomes (Fig, 5-1b) are eco-complexes of interacting eco-complexes, the size of which is defined by a huge bioclimatic zone, like the humid tropics or the mountains above the snow limit. Finally, the biosphere (Fig. 5-1a) is the largest system, composed by all interacting biomes. Modifications of the chemical composition of the whole atmosphere and oceans are due to biosphere dynamics.

One of the constant features of discussions concerning such vast living systems always is the inherent fuzziness of their limits and their definitions. A biome can not at all be mapped with great precision, because it has no clear limits. This has been clearly shown by Hallé (1993:18ff) when discussing the definition of the Tropics, the limits of which can only be described as a wide, fuzzy band. The biosphere itself is the largest living system on Earth.

The surface covered by this system is rather clear. Its lifespan is unknowable. Its architecture displays infinite structural diversity, in which some shimmery patterns are assumed to be recurrent. This subject is out of the scope of the present book. As to the recognition of Life, Lovelock (1979:3ff) wrote beautiful pages which can not be improved. An approach aiming at the technical imitation of Life (“artificial life”) is thoroughly discussed by Levy (1992).

In this realm, phenomena are giant and have a duration outside our scope. However, they are determined by a system which is still larger. This is the solar system, with its periodicities and swinging energetics. We will have to read Figure 5-1 in the other sense now, starting with the whole planet (Fig. 5-1a), bathing in the rays and fields of our star, the Sun.

5.2 Ecosystem dynamics and the environment of the Earth

Life would have been unable to colonize our planet and to provide the major component of its atmosphere, were it not for the biological mechanism of converting the energy of sunlight in chemical bonds. Energy so bound is necessary for the assimilation of carbon originating from atmospheric carbon dioxide. In plants, the carbon is stocked in sugar and starch. This conversion is due to a series of chemophysical reactions common to all chlorophyllous plants, not only from the sea, like marine Algae, but also land plants with green leaves. During these photosynthetic reactions water molecules are broken up in their constituents, i.e. protons or hydrogen nuclei, molecular oxygen and electrons.

Photosynthesis takes place in chloroplasts, which are chlorophyllous organelles provided with a system that transports electrons. When chloroplasts capture light, molecular oxygen is set

The following are among the oxidants distinguished.

- **Superoxide ions** (O$_2^-$) are formed during oxygen reduction in chloroplasts. Because of its single extra electron it is a free radical with the power to break chemical bonds in organic molecules. Superoxide ions are the forerunners of other oxydants, such as the next ones.
- **Hydrogen peroxide** (H$_2$O$_2$) “is toxic for cells because it inactivates certain enzymes of the CALVIN-BENSON cycle [see 4.4.3] which operates the fixation of carbon from the air. However, it also reacts with other superoxide ions and produces other free radicals, the hydroxylous radicals OH$^-$, which are among the most toxic oxidants known” (Foyer 1993).
- **Singular oxygen** (O$_2^-$) is formed in the centres of light capture by excess radiation inputs.

The excitation energy is tranferred to the oxygen and may generate singular oxygen.

In circumstances normal for growth, plant cells have enzymatic and non-enzymatic defense systems. They are adapted to avoid the toxic effects of these derivatives of oxygen. As said by Foyer (1993) in her excellent overview: “......however, if the pressure exercised by outside conditions (light, cold, drought, pollution) becomes too strong, [the defense systems] are outclassed and can not ensure the detoxification of the organism any more”.

Lethal pollution pressure can be brought into existence by the combined forces of climate and people. In this way, herbicides may cause plants to die by blocking photosynthesis. Ozone (O$_3$) reacts with organic molecules and so may provoke oxidation, particularly of lipids of the cellular membrane. The same kind of oxidation is caused by such industrial sulfurous oxides as occur in acid rains.

Human activities hence are one of the components of the pressure called major stress in the preceding pages. The human component becomes ever less negligible - from an optimistic viewpoint - or ever more ominous - from a pessimistic one - if human beings do not become aware of their responsibilities. In the next paragraphs, the climatic impacts are examined.

### 5.2.1 - The climate, its nature and forecasting

As we saw above, the behaviour of an organism or other living system can be analysed as if it were the set of live reactions to environmental impacts. The environment produces two classes of impact. The abiotic ones have a physical or chemical nature and originate from the climate, the soil or the waters. Biotic ones are all forms of interaction between living entities, e.g. consumption, crowding (see Vester 1997) or symbiosis.

Some key concepts in the ecological analysis of these phenomena were formulated by Prenant and Mondchaskey (1958, ex Dajoz 1975, transl. from French RAAO). Prenant states that “The essential idea of ecology is adaptation, i.e. a certain correlation between the organism and its environment.” Mondchaskey adds that “adaptation occurs in the first place in relation to environmental factors varying with a regular periodicity, diurnal, lunar, seasonal, or annual, all resulting directly from the spinning of the Earth around its axis, or its rotation around the Sun, or the succession of lunar phases. These are the primary periodical factors. Heat, light and tidal rhythms are such factors.”
Secondary periodical factors are set in motion by the primary ones. Atmospheric humidity, the precipitation regime, currents in sweet and salt water are examples.

Non-periodical factors include all factors resulting from complex interactions with pseudo-chaotic results (Gleick 1987). In the abiotic range there are storms, thunderstorms or fires and in the range of biotic factors there are, for instance, finding food, meeting a sexual partner, dropping a seed in the right or wrong place, having an accident, or many human activities. In section 5.1, reasons were given why we do not use such terms as disease or competition. The latter fit in the XIXth Century concept of struggle for life, not in struggle of life.

However this may be, no concept of Life can neglect the fact that organisms and other life forms are struggling in the compelling context of their environment (cf. Sect. 5.1).

5.2.1.1 - What is a climate?

Climate can be confounded with an unchanging, average, meteorological state of a point or an area on the surface of the Globe, as suggested by superficial examination of climatic maps. However, the weather changes incessantly, so the dimension of time should be considered in any definition of climate. Therefore we follow Pédelaborde (1970, transl. from French RAAO): “Climate and weather both result from a combination of dominant and permanent tendencies, i.e. the most general elements, of the atmosphere above a place”. The general elements are familiar from the weather forecast, i.e. heat and its parameter temperature of the air, atmospheric pressure, humidity, precipitations, wind speed and direction, evaporation, electric charge, solar radiation and nebulosity.

The most important element is heat, being proportional to the amount of incoming solar radiation. This energy is transformed in the atmosphere and at ground level by diffusion, absorption and reflection. Temperature is the parameter used to assess heat. At any point of the Earth’s surface indeed it reflects the balance between the local effect of irradiation and the heat accumulated in an earlier state by moving air masses.

However, for plants the duration and diurnal or seasonal variation often are more important than the intensity of radiation. This has to determine the particular use of temperature as a parameter. For instance, one should use the annual mean of the daily temperatures with great circumspection, as the same average value may hide widely different amplitudes between day and night temperature. In this way, an oceanic climate with day and night temperatures of moderate values may be erroneously lumped together with a continental climate showing high day temperatures and low night temperatures. Variations at all scales hence have to be studied in order to understand the mean values and to chose the right parameters.

5.2.1.2 - Climatic variations

“Since nearly a century, the world’s mean temperature has risen by 0.5°C and nothing points to a future reversal of this trend” wrote Jones of the University of East Anglia, Norwich, UK in 1990 (transl. RAAO). “Has the sun gone crazy?....New outburst of fury. Since last March, our star gets excited and treats itself to formidable eruptions. And Earth pays the bill by disrupted telecommunications, unsettled climates and disturbed satellites. All this could persist during several years.....” claims Harrois-Monin in the French Weekly L’Express (1989, transl. RAAO).
Quite some confusion exists as to climatic variations and their causes. There is consensus that the Earth underwent a climatic revolution at the end of the Tertiary, changing over from a hot and homogeneous regime to a heterogeneous Ice Age with its alternating glaciations and hot periods. However, there is no communis opinio as to the extension and causes of these oscillations. Saint Blanquat (1970) wrote “The summers seem fresher than before and the winters less severe. Is this a statistic fluctuation of the climate? Experts are divided on this issue, but numerous are those that predict an approaching glaciation.” (transl. RAAO).

Many scientists claim that a climatic warming-up took place between the early XXth Century and 1940, followed by a cooldown and then another warming-up from some moment between 1965 and 1970 on. Other scientists see no proof of this and even postulate the opposite in certain regions of the world. Still others base themselves upon recently increasing amounts of atmospheric CO$_2$ and forecast a warming-up due to a greenhouse effect instead of a cooldown towards a glaciation. Finally some researchers, retracing atmospheric CO$_2$ over past periods, showed that there is “...no evidence allowing to conclude that the great climatic warming-up between 10,000 and 15,000 years ago was caused by increased amounts of CO$_2$ in the atmosphere. The data rather point to the opposite, since the temperature and CO$_2$ curves happen to show a temperature rise before CO$_2$ started to increase”.

Although most scientists today take for granted the existence of global warming due to greenhouse effects, they recognize that the explanation still escapes us. For instance, referring to the increase in plant biomass and the longer growing season recently found in northern regions, Pitelka & al. (1997:473, italics RAAO) state: “Scientists can not be sure what is causing the bloom, but it is certainly possible that the satellites have spotted one of the first signs of the effects of global warming on the biosphere.” Proof, even a coherent picture, are still lacking of the direction of climatic variation and the nature of local or regional effects.

However, there are reliable historic data. LeRoy-Ladurie (1967) demonstrated the warming-up by an average + 0.5°C temperature rise from 1850 to somewhere between 1940 and 1950. This warmer period put an end to the “little ice age” from 1590 to 1850. The amplitude of the heat variation seems tiny, particularly when compared to its effects such as the “great high tide of the Alpine glaciers” which was never interrupted between 1590 and 1850. Since LeRoy-Ladurie published his data, the warming-up, after a dip of two decades around 1950, has been shown to continue to the present years between 1990 and 2000.

The subject of these articles, like for instance the popularity of the work by Lovelock (1979, 1988) spotlight one of the great preoccupations of our times. Does a simple hiccup of our Sun suffice for the Earth to tremble, the climate to deteriorate and the seasons to get jumbled? What were the origins of events like the pluricentennial high tide of the Alpine glaciers? And even if we knew this, adding our present knowledge in general, could we make long-term forecasts of the planetary climate? Let us examine the general atmospheric circulation first.

5.2.1.3 - The general circulation of the terrestrial atmosphere

As a first step to understand the circulation patterns the troposphere is presented. This is the lower layer of the atmosphere, showing horizontal and vertical movements. It has an average depth of 12 km. The mean altitude of its superior interphase, the tropopause, declines from 17 km at the equator to 6 km at the poles.

The general atmospheric circulation is driven by dynamic factors and heat factors.
The heat load arriving at the tropopause from the high atmosphere is distributed unequally over the planet, the intertropical regions receiving most radiation. The atmospheric pressure at the poles is lower than the tropical pressure, because in the cold the same change in altitude causes a faster weight decrease of the air. Hence a current of warm air is generated from the equator to the poles. This current is under the influence of the Coriolis forces due to planetary rotation. In the northern hemisphere they push to the right, in the southern hemisphere to the left. The air has tendency to stream along lines of equal pressure, the isobares, so on the polar belt and in the temperate zones the circulation goes from West to East.

At the equator, a vertical compensation force sucks the air upwards like a vacuum cleaner. At ground level in both hemispheres it activates a double circulation with contrary directions, with the hands of the clock in the northern hemisphere, against the clock in the South. Their interface is the ICZ or intertropical convergence zone. Its movement causes the monsoons.

At the North pole, the air masses cool down and become heavier. They accumulate on the ground, forming a high pressure center which generates an East-West circulation. This meets the northern component of the West-East circulation ring. The encounter of both currents breaks down the polar ring in several action centers. The circulation so becomes cellular at this location, called the polar front. The southern component deviates to the South-West and the West in the intertropical zone, generating the trade winds. These are dry and stable on the continents, but become charged with humidity over the oceans.

As a second step to understand circulation patterns, jet stream dynamics and climate change are presented. “...Zonal movement of the free atmosphere is an average movement. In reality, the upper belt of westerlies does not follow a simple curve around the hemisphere, but it undulates to the North and the South.” (Pèdelaborde 1970, transl. RAAO). The distance between two wave tops or troughs, i.e. the wavelength is of the order of 2,000 to 5,000 km at intermediate latitudes. The sinusoidal path of the western flow represents the “planetary wave” with a period of ca eight days, not to be confused with shorter undulations linked to waves in the lower reaches of the atmosphere.

Rossby & al. (1946 to 1947, ex Pèdelaborde 1970) studied the air masses above the polar front during a stable western weather regime over the North American continent. They found that normally there exists, at an altitude between 5 and 15 km, a narrow band of strong western winds, the jetstreams, being most intense and narrowest at the tropopause level. Evidently, both jetstream and polar front go along with the whole zonal flux and both have a seasonal swing. These atmospheric currents run between 55° and 60° latitude in the summer and between 30° and 35° in the winter. The force of the jetstream and the delimitation of the polar front are maximal during the cold season.

Lamb (1969) recognized two main types of climate linked to two main jetstream types.

- The slow circulation type, extended in latitude and marked by short, ample waves, allows cold polar air masses to penetrate very far to the South. To the North and the South of the main current, eddies are formed when cold drops surrounded by warm air become depressions and warm drops surrounded by cold air produce anticyclones. In this pattern the jetstream is slow and halting. The circulation has a meridian type and may spread over the temperate regions. If this process persists or recurs, cold periods happen and over the whole northern hemisphere five large valleys filled with cold air exist in summer.

- The fast, western circulation type, with increased jetstream speed and marked by very long, narrow waves, generates a climatic optimum, meaning optimal conditions for people,
their crops and their animals. A strong, regular flow of tepid, humid air runs towards Europe and the Mediterranean. The number of valleys filled with cold air during summer is reduced to four. During such periods, glaciers are melting.

The alternation of both circulation types explains, according to Pédelaborde (1970), “climatic variations at all scales and in all eras.”

Relations were assumed between the variations of the western flow and the activity pattern of the Sun. In this view, the speeding up or slowing down of the jetstream would be caused by strong variation of air pressure due to varied heating of the high atmosphere. However, no correct physical solution of this problem has been advanced. It can not exist for two reasons.

First, none of today’s and yesterday’s concepts and models integrate the energy exchange of the troposphere into its environment.

Second, insufficient attention was paid to the major distinctive feature of a cold episode in comparison with a warm episode. Indeed, during the cold episode an enormous quantity of water is fixed as snow and ice at the polar front. This causes an increasing water deficit in the intertropical convection systems. These constraints are due to a predictable event in the middle atmosphere. Before this issue can be raised, however, we have to examine the entire physical Sun-Earth system, starting with the prime cause of its development, the Sun.

### 5.2.2 - The Sun and its variations

To say it with Sadourny (1996, transl. RAAO): “The sun is the unique energy source of the climate of our planet. With the modulations of the orbital parameters of the Earth, the variations of the solar flux represent the principal outside causes of natural climate change.” Indeed, as stipulated by Hoyle’s classic (1962), “The tiny fraction of solar energy reaching the Earth - about one five thousandths of a billionth - is nearly 100,000 times larger than all energy used by the world’s industries. The total energy emitted by the sun in one single second would suffice to feed a one kilowatt electric radiator during a billion billions of years. In other words, the energy emitted by the Sun in one second is more than the sum of energy absorbed by humanity during its whole history.” What energy is this, and how is it produced?

Our Sun is one of the countless stars in the galaxy. Astrophysically, it belongs to the class of G2 stars of rather modest dimensions in between the red giants and the white dwarves. From its center to its envelope, the lower atmosphere or photosphere, its radius is 695,000 km. Its structure is layered.

- **The core** produces energy. At pressures up to some 2*10^9 atm and with heat driving the temperature up to some 15*10^6 °K, a chain of nuclear fusions every minute transforms 6*10^8 tonnes of hydrogen into helium. As said by De Graaff (1990:14): “These conditions result from gravitational forces tending to pull matter inward until a pressure is reached capable of withstanding further contraction. When the proper conditions for fusion are reached it will start spontaneously, and the resulting energy production will help to oppose the gravitational contraction. This energy will then be transported outward, and emitted from the Solar surface at a temperature of about 6,000 K.”

- **A radiating zone** surrounds the core. It is crossed by the energy produced at the core on its way to the outside.

- **A convection zone** surrounds the radiating zone at the exterior. Here, cyclic currents carry the burning gases to the surface.
Above the photosphere, the Sun possesses an atmosphere with several layers somewhat similar to the terrestrial atmosphere.

- The chromosphere, first layer above the photosphere, ca 15,000 km thick, first shows a lowering of the temperature by more than 1,000° K to a minimum of ca 4,500° K.

- The corona is the thin outer part of the Sun’s atmosphere, named after the great halo surrounding our star and making it shine. This is due to overheating with temperatures up to 35,000° K at the outer reaches. Inside the corona itself they may attain 1.6* 10^6 ° K.

The hot gases in this atmosphere are far too tenuous to emit much light. However, the little light emitted has properties due to extreme heat, i.e. spectral rays in the helium range. In the solar corona, atoms collide violently and lose their electrons in the process. Processes here occur similar to those in the core, where atoms are broken down by thermic agitation. Indeed, the photosphere consists of solar plasma, a kind of hot slush of broken hydrogen atoms, a mix of hydrogen nuclei, protons and loose electrons. This environment conducts electricity.

We pointed to similarities between the solar and the terrestrial atmospheres. These are not in their composition but in their temperature profiles upwards from the solar convection zone and the terrestrial sea level. Both show a comparable thermic reversal when ascending above the zero level. On Earth, temperature above sea level first descends to a minimum and then rises again between some 25 and 55 km altitude, where it once more reaches values like those at the planetary surface but sometimes may rise over 100° C.

The cold layer in between 10 to 15 km and 40 to 45 km, considered as isothermic, is the stratosphere. It is severely lacking in water vapour, showing a relative atmospheric humidity like the Sahara, and its gases are very tenuous.

Because it is also a compartment where the major part of the atmospheric ozone concentrates, its synonym is ozonosphere. This ozone is generated by absorption of ultraviolet radiation in the Schuman band, with wavelengths in between 0.12 and 0.20 μ, by the oxygen present. Above 40 km the oxygen also absorbs radiation from the Schuman band, but the ozone formed then absorbs ultraviolet from the Hartley band with wavelengths from 0.29 to 0.40 μ.

This double absorption is the key to life on Earth. The amount of ozone is not constant. Strong seasonal and irregular variation exists (cf. Vassy, 1956, 1959, 1960). Says Pédelaborde (1970, transl. and italics RAAO): “Seasonal variation is explained by the balance of generation and destruction which are a function of the solar radiation. The irregular variation generally follows the undulating disturbances (planetary wave with a period of 8 days) which also affect the troposphere.” We will come back to these disturbances later, and on the part they may play in “natural pollution” of the troposphere.

Above the stratosphere lies the mesosphere. It reaches from 40 to 45 km upwards to 80 km altitude, and shows a temperature maximum around 55 km above sea level. Here, ozone also absorbs ultraviolet rays from the Hartley band.

In the stratosphere and mesosphere, violent winds blowing at speeds up to 250 km*h^-1 are generated by strong atmospheric dynamics. Once more quoting Pédelaborde (1970, transl. RAAO): “Eastern winds dominate between 20 and 80 km altitude in summer. In the winter, the winds are blowing, contrariwise, from the West. This seasonal reversal is due to the enormous temperature variation between summer and winter, both in the warm layer and the ozonosphere. Hence it is called stratospheric monsoon.”
Above ca 80 km stretches the high atmosphere, where diluted air absorbs short UV radiation and so drives its temperature up to 1,000° C. This part of the atmosphere hence also is called thermosphere. It is named ionosphere too, because its rarefied air is strongly ionized by the breakdown of hydrogen and helium atoms, resulting in high amounts of free electrons. The ionized air at this altitude conducts electricity well and reflects radio waves to the ground.

Coming back to the Sun, its surface displays dark stains, the sunspots. Periodically, these have a maximum of activity when they become more numerous and drift closer to the solar equator. These sunspots originate from gigantic streams of hot, ionized gases caught between the lines of the Sun’s magnetic field, forming a kind of tubes in the solar atmosphere. Just like bundles of isobars in the earth atmosphere cause air currents to become storms, the magnetic tubes conduct intense currents. Sunspots appear where these tubes emerge from the surface of the photosphere.

Sunspots generally appear in pairs. If, for example, the magnetic field has a negative polarity where the tube emerges at the outside of the sun, the polarity of the tube turned towards the inside of the star is positive. Hence pairs of sunspots are nothing else but immense magnetic dipoles, the orientation of which is inverted in the northern and southern hemispheres. The existence of a magnetic field is something else that the Sun and the Earth have in common.

5.2.3 - The dynamo effect and the magnetic cycle of the Sun

There is a magnetic cycle of the Sun, commanded by the differential rotation of the star and the convection streams below the photosphere. The regions of the surface of the sun do not rotate at the same speed. At the equator the angular velocity is one rotation in 25 days, at the middle latitudes in 28 days. These differences cause a deformation of the magnetic field. The lines of that field, which were oriented North-South initially, change shape after several rotations because of the Coriolis force and are finally drawn out parallel to the equator, where their power is intensified. The circulation in “tubes” or “rolls” parallel to the equator was discovered by Elisabeth Nesme-Ribes in 1985, in collaboration with Pierre Mein and André Mangeney of the Observatoire de Paris.

The migration of these magnetic tubes towards the poles causes the general solar dipole to change polarities every 11 years. “The circulation of fluid associated to these tubes contributes naturally to the transport of nuclear energy leaving the Sun's core. Because these tubes appear every two years, with increased vigour during the active phase of the 11 years cycle, the luminosity and apparent diameter of the Sun will vary during the cycle .... The apparent diameter is less (and the luminosity higher) when the sun is most active.” (E. Nesme-Ribe 1990, transl. and italics RAAO).

The maximum of solar activity is correlated both to the migration of sunspots from the Sun’s middle latitudes to the equator and to the number of sunspots. The mean cycle of these events is 11.11 years, the solar cycle. After every maximum of activity, polarity is inverted, so each hemisphere of the Sun finds its original polarity back after 22.22 years, the magnetic cycle.

Following Foukal (1990), the magnetic eruptions cause the inversions of polarity in between sunspot cycles. He says: “Coming out from the Sun, the magnetic tubes would generate regions of strong polarity, from where the magnetic field would spread. At the same time, the field would be pushed back towards the atmosphere by the new magnetic fields which the differential rotation forces drive up from the interior of the star. During this expansion the polarity of the field would weaken and make place for the new, inverted field polarity.”
5.2.4 - Solar activity, the solar wave and the magnetic cycle

In his pertinent review of theories on the control of terrestrial climates, Fairbridge (1972) wrote (transl.RAAO): “One minor variable of solar radiation has capital importance. Indeed, the infrared radiation of the Sun, vector of most of the caloric energy, is recognized to be constant. However, the transfer of weak energy by ultraviolet radiations shows a variation linked to the solar activity. ...... Their cycles in their turn seem to result from a “tidal effect” spurred by the movements and “stellar cycles” of the principal planets. Therefore they can be forecast, the last tidal maximum having occurred in aD 1433. The periods of 556 and 1668 years seem to be the most significant ones for the long cycles.”

This periodicity attracted the attention of one of us (MR) some years earlier. He examined the external causes, particularly an “exceptional solar activity”, that could be at the origin of the piling up of warm waters, poor in salt, at the surface of the western Atlantic and deeper below, the compensating “Guyana countercurrent” running in a south-easterly direction against the general circulation (Rossignol 1977).

What do these periods of 556 and 1668 years represent?

* The basic cycle of 556 years coincides with the opposition between Jupiter and Saturn in line with the Sun as well as with the Earth. Indeed, the opposition of Jupiter to Saturn with the Sun in the middle occurs every 19.859 years. The Earth fits into this line every 139.01 years, i.e 7*19.859 years. And 4*139.01 = 556.04 ≈ 556 years.

The last solar tidal maximum took place in 1433 aD. It was certainly the cause of numerous, very strong solar eruptions, at least as strong as those observed in 1989 and 1990, 556 years later. These eruptions dissipated much energy at the surface of the Sun. They also caused important radioactive and electromagnetic disturbances following the shock wave at the level of the magnetosphere around the high Earth atmosphere. This dissipation of energy could well be at the origin of another important event on Earth, the minimum of Maunder. It occurred between 1645 and 1715, i.e. 220 years or 10 magnetic cycles of 22 years later, and showed typical rarity or absence of sunspots. This period is usually called the “little Ice Age” because of the unusual cold, and the growth of glaciers and polar ice.

It is useful to bring to mind that the solar atmosphere may be conceived as a gas, but a gas which conducts electricity due to free electrons and a varying magnetic field. It therefore obeys to laws both of magnetism and flow. Taking into account this double command, one may understand the significance of the smallest component of 11.11 years, both in the maximum tidal cycle of 556 years and the magnetic cycle of 22.22 years.

* The eleven years cycle and the solar wave.

The lines of the solar magnetic field traverse the layers of the solar atmosphere from the convection zone. The magnetic field is “frozen” in photosphere matter, where it moves slowly. It traverses the chromosphere after crossing the coldest zone of the solar atmosphere where the temperature inversion occurs, just above the photosphere. Finally, it traverses the corona. When the system is in a balanced state, slow plasma movements give rise to electric currents in the corona and the magnetic field exerts force upon these currents. This force can be decomposed in two vectors. The first one is a magnetic tension, which conveys to the magnetic field properties similar to an elastic cord, and the second one is a magnetic pressure. Nonetheless, the gas maintains a very low density, so tension and pressure can balance each other out (Amari & Démoulin 1993).
What is the probable impact of the tidal effect on the “cold” temperature inversion zone at the base of the chromosphere when Jupiter and Saturn are in opposition to the Sun, particularly so when the Earth is aligned too? The balance of forces is broken, the “elastic cord” starts to vibrate and the surrounding matter, i.e. photospheric magma and gases in the chromosphere and corona, becomes hotter. The “feet” of the magnetic lines of force start moving more rapidly, entailing an increased intensity of the electric currents. Starting at the feet of the lines of force, at the bottom of the photosphere, solar magma carried upwards from the Sun’s core by the convection cells is thrown outward. This generates a wave front at both sides of the solar equator. At the equator both fronts meet and enter in resonance, turning around the Sun in 11.11 years.

This wave is called here the solar wave, with a period of 11.11 years. At the solar scale, it is the equivalent of the terrestrial planetary wave encountered above, with its period of 8 days. This fits in with the proportionality of both waves inside their respective scales. Indeed, the solar chromosphere is ca 15,000 km thick and the stratosphere around 30 km. Their ratio is 15,000/30 = 500. Now 500*8 = 4,000 days ≈ 11 years. The solar and planetary waves hence are directly proportional to the depth of the layers upon which tidal impacts are acting.

* The magnetic solar cycle and the variations in radiation are further explained and proven by recent physical research. An anonymous reporter (1996a) wrote in La Recherche on experiments by the team of Serge Haroche and Jean-Michel Raimond at the Ecole Normale Supérieure in Paris: “The probability of transition of the atomic \( g \) state towards the \( e \) state is a periodic function of time. And this function is characterized by frequencies of oscillation, related directly to the number of photons present......An experience revealing in a glaring manner that the magnetic field is granular.”(also cf. Brune & al. 1996).

These elegant experiments support our model of the variation of solar energy, based on the idea that not only the electromagnetic radiation could be quantified, but also the time needed by a hydrogen atom to pass from one energy level to the next. Indeed, a hydrogen atom caught in the electromagnetic field of the Sun must pass from one energy level to the next, which can in principle be calculated as a periodic quantum function of Time.

Figures 5-3 a,b and c represent the different energy levels of a hydrogen atom at the solar surface during an augmentation of the intensity of the electromagnetic field of the star.

We start from a low level, level 1, characteristic of a hydrogen atom during a period of “solar rest” with a feeble electromagnetic field (Fig. 5-3a). The field is activated by the tidal impact of the solar wave, with a period of 11.11 years. This may be called the “ignition time”, i.e. the time of the installation of the first convection tubes which lift the hydrogen atom to a certain energy level.

- If we call this initial level \( n_1 \), we need \((11.11) + (1*2*11.11) = (11.11 ) + (2!*11.11) = 33.33 \) years to lift the hydrogen atom one energy level higher, to \( n_2 \).
- The third level \( n_3 \) is reached after \((11.11) +(1*2*3*11.11) = (11.11) + (3!*11.11) = 77.77 \) years.
- The hydrogen atom is brought up to level \( n_4 \) after \((11.11) + (1*2*3*4*11.11) = (11.11)+ (4!*11.11) = 277.75 \approx 278 \) years.
Figure 5-3: Relation between the magnetic cycle and tidal waves on the solar surface, and the tidal impact on the Sun, when aligned with Earth and the two heaviest planets Jupiter and Saturn. (a) – Fluid circulation associated with “magnetic tubes” helps transport of nuclear energy from the solar core outwards. In the resting phase of the sun this energy is at level $n_1$. After one period of the solar wave, i.e. 11.11 years, the magnetic tube has obtained a certain polarity. To recover the same polarity, the tube must wait for $11.11 + (2 \times 11.11) = 11.11 + 22.22 = 33.33$ years, i.e. two temporal quanta more. The energy returning from $n_2$ to $n_1$ is then dissipated by emitting a photon. (b) – Level $n_3$ is reached after $11.11 + 3 \times 11.11 = 77.77$ years. (c) – Level 4 is reached after $11.11 + 4 \times 11.11 \approx 278$ years. Photonic density has increased. The period of 278 years is twice the period separating two alignments of Sun, Earth, Jupiter and Saturn, occurring every 139 years. Over longer periods, the energy level is high and fits the first long cycles of Fairbridge (1972), i.e. $2 \times 278 = 556$ years; $3 \times 278 = 1,668$ years, and $4 \times 278 = 6,672$ years. “Nuclear cycles” of $3 \times 6,672 = 20,016$ years and $3 \times 4 \times 6,672 = 80,064$ years lie at still higher levels.

The period of 278 years is twice the period of 139 years in between two alignments of Jupiter, Saturn, Earth and the Sun.

- Twice this period of 278 years is 556 years, the first long stellar cycle of Fairbridge (1972), due to long cycles coinciding with the tidal impact at the Sun’s surface.
- The second long stellar cycle then is $3 \times 556 = 1,668$ years.
- The third long cycle equals $4 \times 1,668 = 6,672$ years.

This chronology could be checked over a long enough period. This was done by reading the famous ice core from the glaciers of Groenland, conserving yearly ice layers from between 100,000 BP and 1970. This core is 1.400 m long and for each of the 100,000 years the temperature of the air can be calculated from the gases dissolved in the water when it froze. The Danish oceanographers Johnsen, Dansgaard, Clausen and Longway lifted the core and did the calculations, using the ratio between the isotopes $\text{O}^{16}$ and $\text{O}^{18}$ in the air as a parameter for temperature (ex Saint Blanquat 1973). Our figure 5-4 contains the smoothed curve established after the mean curve of temperature against time published by Saint Blanquat.

The curve shows some remarkable features. First, both in 1989 and in 91,419 BP, an abrupt and significant warming-up of the globe occurred, as shown by a strong rise in temperature. Second, this heat, probably with minor fluctuations, marks the period of 6,672 years in between the first event in 91,419 BP and 84,747 BP, when a maximum of solar activity heralded the first episode of the Great Würm Ice Age. The temperature fell inexorably. This episode, Würm I, was the harbinger of an agitated period of the history of the Earth which was going to take 80,064 years altogether. The first climatic optimum after the Great Würm Ice
Age, quite comparable with the one between the two Great Ice Ages Riss and Würm, appeared only in 4,683 BC.

What is this great cycle of 80,064 years? It equals 4 times 20,016 years, and 20,016 years equals 3 times the third cycle of 6,672 years.

- \(3 \times 6,672 = 20,016\) years
- \(4 \times 20,016 = 80,064\) years.

What distinguishes these long cycles from the other long cycles, except the fact that they are longer? The answer to this question lies in the “memory” of the glacier in Groenland and the terrestrial climatic variation and solar activity as observed since centuries. To compare both data sets, figure 5-4 superposes the set of long cycles upon the smoothed Groenland curve. The countdown goes from 1989-1990, the most recent solar maximum, to 100,000 BP.

The resulting diagram, coupled to the more or less stable state of the solar environment and the causes of destabilization, leads to the concept of cycles of great solar instability and strong dissipation, or cycles of nuclear fusion. These have their impact upon the high terrestrial atmosphere, where the magnetic and electrical fields are activated. This activity in its turn is handed down to the stratosphere, the high troposphere and the oceans, where the strong instabilities generate the dissipative structures needed to find a new balance. Such dissipative structures are for instance the major event of the high troposphere discussed later, or in the ocean the subsiding waters and the forced deep compensation currents like the countercurrent of the Guyanas in the western Atlantic or an analogous phenomenon in the Pacific, El Niño.

*The cycles of great solar instability or nuclear fusion cycles* can be read from the temperature curve (Fig.5-4), two important points of which draw our attention.

First, at both extremities of the curve around 1980 and around 80,000 to 90,000 BP, more precisely the 84,747 and 91,419 BP events, temperature is high. They are warm episodes. The earlier episode is the end of the Interstadial or climatic optimum which closed off the Great Riss Glaciation Cycle. This warm episode lasted 6,672 years, before the situation turned around and lead, after the dissipation of the heat energy, to the installation of a cold climate, with snowfall and growing glaciers in the polar zones and temperate mountains.

This was the beginning of the Würm Super Ice Age. As we saw, it lasted a little over 80,000 years before a new balance was struck on Earth, starting with the Bölling episode (11,355 years BP) and becoming stabilized with the climatic optimum of 4683 BC. The Würm itself was marked by alternating episodes of intense cold and warming up. One of these warmer episodes, between the great climatic optima and more or less comparable, draws our attention. It occurred after Würm I and before Würm III, from 51,387 BP to 31,371 BP, when a climatic stabilisation took place, but at lower temperatures than during the two other ones. Its “warmer” episodes are indicated as (a) and (b) on our main line in figure 5-4, (a) corresponding to the Amersfoort and the Brörup and (b) to the Gottweig. During the Gottweig, the “New Human” Homo sapiens sapiens relayed Homo sapiens neanderthalensis.

Two of several factors which in Milankovic’s theory cause very long periodic variations of the solar activity, may be considered as the main factors explaining the phenomena observed.
Figure 5-4: Is there a relation between the cycles linked to the movements of our planet around the Sun (Milankovitch’s theory), the dissipation pattern of solar energy by tidal maxima, and the climate variation on Earth? Time sequences according to these periodicities are confronted with the smoothed curve of atmospheric mean temperatures over the last 10³ years, following the results obtained by Johnsen, Dansgaard, Clausen & Longway. These authors analysed the oxygen isotopes dissolved in successive growth layers of ice in a 100 centuries old cylinder taken from the polar ice in Groenland, as a parameter of temperature of the air. H.R.S.T.M. = high rank solar tidal maximum, occurring every 92,000 years.
The eccentricity cycle of the terrestrial orbit has a period of 92,000 years. The Earth describes an ellipsoid orbit around the Sun, the latter being in one of the foci of the ellipse. In the phase of the perihelion, the Earth occupies the end of the long axis closest to the Sun focus (Fig. 5-5). This perihelium distance varies in time with the eccentricity of the orbital ellipse. The eccentricity is defined as the ratio c/a between half the interfocal distance and half the longest axis. The eccentricity of the Earth is on the increase since 22,000 years (ca 20,000 BC). Indeed, the c/a ratio shrinks to its minimum value, expressing a maximum eccentricity, in approximately 24,000 years. The ratio then starts increasing, and will reach three times its actual value 46,000 years later, then to start decreasing again. In the past, the perihelium distance was minimal in 20,000 BC, maximal in 66,000 BC and minimal in the year 112,000 BC.

The eccentricity cycle of the terrestrial orbit causes the tidal impacts at the Sun’s surface, when the Earth is at that end of the long axis which is closest to the Sun. During this maximal activity of the Sun the thermic agitation and the intensified magnetic field cause an abrupt temperature rise due to the shocks between particles. At the same time that hydrogen fusion inside the sun has become possible, the antigravitational force kicks part of the generated energy to the surface. It is dissipated in the form of radiation and particles “frozen” in the mass during solar eruptions.

The impact of such a solar wind on the terrestrial magnetosphere and its effects on the atmosphere of the Earth have been conserved in the form of isotopic oxygen traces in the “glacial memory” at the North Pole.

The inclination cycle of the terrestrial axis to the orbital plane has a period of 40,000 years. It determines the width of the Sun’s apparent, seasonal, latitudinal movement. This swing causes the perpendicular position of the Sun at the summer solstice over some spot at a latitude near or distant from the polar circle. The stronger the inclination, the closer that spot is to the pole. This factor accounts for the puzzling intermediary warming-up of the climate in
mid-Glaciation, starting in 51,387 BC between Würm II and Würm III. Figure 5-4 shows indeed that, according to the memory of the Groenland glacier, the event between Würm II and III is comparable to that of Würm IV, ca 40,000 years later in 11,355 BC (the Bölling episode) and to the one in the Riss-Würm Interstadial, 40,000 years earlier in 91,419 BC.

The two factors discussed above are important. Indeed, they created the conditions favouring the insemination, the proliferation, the evolution of “living matter” on our Earth. This happened through two dissipative structures. The first one acted at the level of the Sun and we just discussed it. The other one acted at the level of the Earth, due to the broadcasting of particles, carried by the solar wind, on the high atmosphere.

The latter factor is a “modulator” of the angle of impact of the solar radiation upon our planet. Hence it determines the larger or smaller part of the terrestrial surface touched by this radiation. As a first consequence, summer temperatures on Earth have a longer or shorter duration. In chapter 6, a second modulator in the opposite sense will be evoked, causing winter temperatures to perdure over a shorter or longer period.

5.2.5 - Unstability and dissipative structures in the atmosphere and the oceans

These energetic phenomena can be observed in the two terrestrial surface layers that possess freedom of resonance and hence show tides. These are

- The temperature inversion layer in the middle atmosphere which forms the limit between troposphere and stratosphere and is rather cold;
- The interphase in the tropical oceans between the superficial, warm waters poor in salt and the deeper layer of rather colder and denser waters with a salinity at maximum.

In 5.2.1.3 we introduced the jetstream and the two types of climate linked to this stream:

- a type of slow circulation and with wide waves spread in latitude, allowing the cold polar air masses to penetrate to latitudes as low as the temperate and subtropical regions;
- a type of rapid western circulation and with long waves of feeble amplitude, characterizing the climatic optimum.

We also saw that speeding up and slowing down of the jetstream depend upon the strongly varying air pressure, driven by heat as shown by temperature change in the high atmosphere. Kinetic energy and turbulent wind speed in the ionosphere are the stronger as the environment is richer in free energy. This happens at the maximum of solar activity, the vector being the solar wind. Winds then have been measured peaking at 10 km·sec⁻¹ at 250 km altitude.

During the maximum of solar activity, the stratospheric or ozonospheric circulation goes from East to West, clockwise, against the direction in the ionosphere. The easterlies of the stratosphere then often reach speeds of the order of 250 km·h⁻¹.

What happens if these stratospheric easterlies are accelerated?

This was explained by Rossby (ex Pédelaborde 1970) as a result of the centrifugal effects of the rotation of the Earth upon the movements of the air relatively to the surface of the globe. When an atmospheric ring moves to the East more slowly than the planet, which is the case of
the easterlies, the ring obtains a deficient centrifugal force and “it tends to get closer to the axis of the Earth, i.e. to be rejected to the pole.” In this way it compresses the air masses to the North and generates a North-South pressure gradient. At the same time, in the southern hemisphere and during the boreal summer, the acceleration of the western winds conveys to the atmospheric ring an excessive centrifugal force. The ring then tends to get farther away from the terrestrial axis, i.e to be driven to the equator. This generates another North-South gradient of atmospheric pressure, but closer to the equator.

The outcome of this increased tropospheric pressure is predictable, because the fast western jetstream reaches its maximal speed at the lower limit of the stratosphere. The jetstream can indeed be seen as a fast flux of inertia, for which the order of magnitude of the advection of the vorticity is given by the term of subsidence

$$\begin{align*}
\begin{pmatrix}
\frac{\delta}{\partial t} & D_x \\
\delta & \ast & \delta_t
\end{pmatrix}
\end{align*}$$

which contains two components, a barotropic and a baroclinic one. The first one is linked to the differential variations of the surface level. The second one depends upon the changes obtained, occurring in the density field below the surface. The barotropic component also is a function of the force of lateral strain.

The increasing stratospheric North-South pressure gradient produces a negative lateral strain $\delta_n/\delta_s$ in the high troposphere of the polar front. The system of natural coordinates is used. One may image this force as a wedge that opposes the flux at the left side of the current. Air accumulates and subsidence of warm air occurs at the left side of the jetstream, the axis of which deviates to the South. In its wake, cold air is sucked towards the lower latitudes.

If the strain is weak, the jetstream shows long undulations with cold air in the troughs and warm air travelling North in the crests. In western Europe, for instance, this produces a mild western marine flux.

If the strain is strong, circulation in the northern hemisphere is strongly disturbed, turbulent, divided in cells, with cold air invasions to the South. The meeting of cold air masses with warm ones generates gravity waves which, together with the waves of inertia, spawn undulations in the form of the polar front waves. When these formations are sufficiently advanced, a movement of ascending turbulence by warm air spiraling upwards is originated following the process of the polar cyclone described by the Norwegian school (Bjerknes & Solberg 1960). Cold air currents or cold polar air “drops” can reach the domain of subtropical, or tropicalised polar air following the contours of the five “cold valleys” of the northern hemisphere in summer (cf. Sect. 5.2.1.3).

This type of strain, as we said, is due to the impact on Earth of the instabilities of the Sun and the dissipation of excess energy freed in periods of maximum solar activity. This happens especially at the end of “long stellar cycles” inducing the tides at the Sun’s surface. The shortest of those is the 556 years cycle, which may be considered as the minimal significant “rhythmic beat” of a change in the level of entropy of our planet. Such change or “major event” can be expressed as a “little Ice Age” on the polar borders and in the temperate regions. It can also be expressed by what human beings experience as catastrophes causing deep
dilemmas, such as physical desequilibria causing torrential rains and heavy snowfall in some spots and at the same time drought in other places. The catastrophes can also be biological, the oxidation of cellular membranes of higher organisms leading to viral and bacterial epidemics.

The major atmospheric event of the polar belt has been schematized in figure 5-6, using three perpendicular component axes. The southern and eastern components form a horizontal plane, on which are represented the lines of equal pressure (isobares) and the wind circulation of the northern hemisphere at soil level. The eastern and altitudinal components form a vertical plane perpendicular to the previous one, with the isobares in transverse view. Behind the vertical plane the dynamics of the system to the West of the polar cyclone are shown, whereas the foreground visualizes the curved lines of the cyclonic turbulence. The event has been shown during the summer, in the northern hemisphere, above the Atlantic. A similar image could have been given for the Pacific, only the surfaces covered being larger.

We saw that the excess pressure in the high troposphere, at the transition layer towards the stratosphere which is the temperature inversion layer, generates a dissipative structure that will correct the instability and desequilibrium of the existing local structures. It is expressed in two ways, by kinetics and undulations. The kinetic expression is the acceleration of the turbulent winds of the polar cyclone. The undulating expression is the generation, to the left of the jetstream with warm "tropospheric" winds, of gravity and inertia waves at the origin of valleys through which cold, exotic air can intrude in the South (Fig. 5-6: c.e.a.i.). This current to the South, later to the South-West because of the Coriolis forces, obtains its energy from the polar cyclone and the whirling acceleration of its winds. The air that circulates through the "valleys" lost its original character. It contains a more or less important fraction of stratospheric or ionospheric air. This explains the term "exotic air". The system is self-maintained and its periodicity corresponds to the eight days of the planetary wave.

The cold exotic air intrusion, either to the South and West in the form of "cold drops" at high altitude, or to the South and East as sunken and reheated cold air, has great consequences.

In the zone of lower atmospheric pressure of the western Atlantic, the high-altitude "cold drops" bring forth tropical cyclones in the Caribbean and Florida. These carry destruction not only because of the strong winds but also by the torrential rainfalls. During its voyage over the Ocean, the turbulent air is indeed charged with humidity. In the eastern Atlantic, the air heavy with humidity on the left flank of the anticyclone of the Açores, travels either to quite high latitudes causing depressions in Great Britain, the Netherlands, Danmark and Norway, or sends a branch to the East with the Mediterranean depression. During a major solar event, this can cause havoc and have unusual outcomes. There may be strong inundations and precocious snow in full summer in the temperate regions of Europe and Africa, rain in the northern Sahara and drought in the Sahel to the South.

These phenomena all are the physical and dynamic aftermath of a major solar event.

Less striking are its insidious impacts on Life on land and in the seas. There is a natural pollution of the aqueous and gaseous elements, brought in by the "exotic air". Indeed we saw that it contains not a little stratospheric air and hence carries ozone and free radicals imported by the turbulence of the lower stratosphere. We see this as the origin of the "hole in the ozone layer". The cause is physical, not chemical. Indeed, the last thirty years we have seen the widening of the "hole in the ozone layer" concurring with a series of solar events similar to those having caused the "little Ice Age" 556 years earlier.
Figure 5-6: Instability and dissipative structures in the terrestrial atmosphere and ocean, example of the Atlantic. The situation shown occurs during a tidal solar maximum, which is a major atmospheric event at the polar belt. These structures, typical for the Northern hemisphere, are schematized in the figure. The Equatorial Counter-Current, an important oceanic structure which would have to be visible at the base of the figure, has been omitted for the sake of clarity. Ca.C. = Canary current. c.e.a.l. = cold exotic-air intrusion. G.C.C. = Guiana counter-current. GD = Guinea dome. G.S.C.C. = Guinea-Senegal counter-current. G.St. = Gulfstream. HP = high pressure. LP = low pressure. UpW = Upwelling waters.
Section 5.2 started with an enumeration of the damage and mortality caused in organisms by ozone and free radicals, inducing for instance the oxidation of lipids in cellular membranes. This shows why under such circumstances Life could not continuously exist without its own kind of dissipative structures.

However, we mainly examined the Atlantic part of the northern hemisphere up to this point. The moment has come to survey the consequences of major solar events on the intertropical oceanic environment.

→ The oceanic instabilities in the intertropical zone originate from centers of climatic activity. Above the Atlantic Ocean there are two high pressure cells (HP) separated by a low pressure cell (LP) as shown in figure 5-6. In the western Atlantic and in the northern hemisphere, low pressures occur in the Carribean, whereas in the eastern Atlantic the situation differs in the northern and southern hemisphere. In the figure, it is the auroral winter in the southern hemisphere, so the atmospheric North-South gradient has come closer to the tropics. The southern tradewind, pushed by high pressure, blows to the NNW along the African coast between Cape Frio and the Gulf of Guinea. It turns off to the right (NNE) when passing the equator, particularly off the coasts of Guinea and Sierra Leone, where the pressures are low. This tradewind so contributes to install in these regions a regime similar to the monsoon, just like in the eastern part of the Gulf of Guinea in the Cameroons.

In the western part of the Atlantic, the monsoon effect can be felt to the latitudes of the Caribbean Sea. A branch of the southern tradewind turns off to the North-West, i.e. towards the Carribean low pressure zone.

Now let us consider the outcome of this particular disposition of the centers of atmospheric activity. In the western parts of the Ocean as well as in its eastern part, the Gulf of Guinea, the superficial waters of the intertropical zone are warm and desalinized by the abundant rains and the sweet water from rivers and creeks. This is so mainly along the Brazilian and Guyanese coasts, due to the great capacity of the local rivers, particularly the Amazon. These superficial waters are separated from the colder and denser waters of the northern and southern Atlantic by a thermic and saline gradient, the thermocline.

The wind system, at the moment of the solar event in the northern hemisphere, particularly in the West on the Brazilian and Guyanese coasts, piles up the warm and desalinized surface waters. Under the thermocline, but with a certain delay due to the inertia of the dense and cold intermediate waters, the cold and salt upwellings emerge below the water piled up at the surface. With the lateral strain of the coast to the left of the current, there then exists a disposition similar to the origins of the atmospheric instability caused by excessive pressure during the event. Like in the atmosphere, the ocean installs a dissipative structure by the buildup of a countercurrent or “undercurrent of forced compensation”, the “Guyanese countercurrent” off the coasts of the Guyanas (Rossignol 1977, Inst. Acad.Sci. Ukr. SSR 1975), and the “countercurrent of Guinea” off the Guinean and Senegalese coasts (Inst. Acad. Sci. Ukr. SSR 1975). In figure 5-6, the Guyanese countercurrent is indicated as G.C.C. and the Guinean one as G.S.C.C.

A comparable dissipative structure is observed in the Eastern Atlantic of the Northern Hemisphere. It originates from “forcing” by SSW monsoon winds. Cold Intermediate and salt waters emerge below the warm waters piled up at the surface. With the lateral, coastal strain to the left of the current, a dissipative structure is installed, which embraces two flow systems.
The first is the Guinea Dome in the southern part of the continental shelf along Senegal and Guinea (Fig. 5-6, G.D.). Below its thin, warm surface layer, its waters are Intermediate waters from the South Atlantic. A second flow system, running to the NE beyond the continental shelf, incorporates maximum salinity, belonging to the Intermediate North Atlantic Waters (Fig. 5-6, G.S.C.C.).

These waters are rich in nutrients, which mix with the warm surface waters above the Guinean Dome and so increase their biotic productivity. Phytoplankton booms and with it the grazers of the zooplankton. The boost is transmitted over the whole food chain to all larger marine animals and plants (Rossignol 1965; ca 1970).

These physical and dynamic summer conditions occur whatever the intensity of the solar event may be. However, during a great solar event, with a long cycle of 556, 1,668, 6,672 .... 20,016 years, it is not only the intensity of the phenomena which is increased, but they also are prolonged beyond their season.

In a period of solar rest, the barometric pressure descends from September to November in the intertropical zone at both sides of the Atlantic, i.e. the coasts of Senegal, Guinea, Gulf of Guinea, Gabon and Congo to the East and North-eastern Brazil, the shore North of the Amazon, the Guyanas and the Caribbean to the West. In the central part of the Atlantic, the thermocline subsides, the thickness of the warm water layer increases and so it does too on the continental shelves. In the atmosphere of this region, the zone of intertropical convergence (ZITC) descends to the equator, carrying rainclouds. This causes the little rainy season in northern Brazil, the Guyanas, the Caribbean as well as Senegal, Guinea, western Africa, Gabon and Congo.

In a period of solar activity corresponding to a solar event with a long cycle, the instability of the polar front keeps exotic cold air running to the South and South-West. In the western Atlantic the "cold drops" in the atmosphere activate and fatten tropical cyclones with very humid air. Suction by the turbulent winds in the high atmosphere establishes a divergence in the equatorial belt of the western Atlantic. It splits the tradewind in two branches, one to the North-West, the other to the South-West. At the latitude of northeastern Brazil, where the split takes place, the warm surface waters are pushed either North or South. Their place is taken by upwelling colder and saltier waters, which cool down the air so that it cannot ascend. This causes dry weather spells from the North of Brazil to the Guyanas. More to the North, the air gets warmed up and is progressively loaded with humidity, which rains out of the clouds when the cyclones pass over the Antilles and Florida, for example.

Later in the season, from October to December when it is autumn in the North and spring in the South, a high pressure regime is installed from the South of Angola to Congo, with winds blowing to the North or North-West. The warm and desalinized surface waters are chased away from the coast. A coastal upwelling then occurs during a quite fresh local little dry season. The situation in the eastern Atlantic now is similar to the earlier one at the West side. The air leaves the African coast, warms up, becomes loaded with humidity when crossing the sea, and arrives in the Northern part of the Ocean after crossing the equator. The winds then join up with the tradewinds on the western flank of the anticyclone of the Açores, carrying heavy rain clouds that can be mixed with the exotic air. The outcome is violent and may show tornades and diluvian rains on the Açores themselves, on African coastal regions where the climate is usually arid, like Mauritania or the Spanish Sahara, and on the European and North
African periphery of the Mediterranean.

During the boreal winter and austral summer, the situation is the other way round. High pressures prevail on the eastern Atlantic from Cape Blanc to Guinea, and low pressures at both sides of the intertropical Atlantic basin. The coastal upwelling takes place between Cape Blanc and Guinea, whereas heavy rains fall around the Gulf of Guinea and from Gabon to Angola, and in the West from the Amazon to the Caribbean in the large or main rainy season.

The phenomena described here for the Atlantic may be summarized as a seasonal seesaw from East to West along the equator and from South to North and North to South in the eastern Atlantic. They are very similar to "El Niño" in the Pacific basin. The only difference is the scale of magnitude of the extension of both oceanic basins. El Niño is literally the Child who granted Peruvian fishermen a free Christmas because the fish had gone elsewhere.

In view of the important disturbances which were the birthday presents of the last Niño, the question was raised whether or not it might provoke a radical change of the global climate. A climate characterized by a temperature optimum and mild winters in the temperate zone would then change over to a Glaciation, with falling temperatures and mobilization of waters from the tropical Oceans. These waters would be stocked as snow and ice around the Poles and the high mountains, by fattening of the polar land ice and alpine glaciers.

El Niño has even been seen by some as the chief agent causing a coming worldwide Ice Age.

In our view this is neither entirely true, nor completely false. In order to make an Ice Age, water is indeed needed in great quantities, the provider being the Ocean. However, this water can only be mobilized by instability and acceleration of winds, and these conditions can not maintain themselves over a long enough period if there is no extra solar energy available to keep the machine turning. In other words, the prime agent is the Sun, not the Child.

This is confirmed by a “Report to the Nation on our changing planet” (1997, http://www.atmos.washington.edu/gcg/RTN/rtnt.html) under the heading “El Niño and climate prediction”, which states: “In contrast to the march of the seasons which is regular and therefore highly predictable, El Niño recurs at irregular intervals ranging from two years to a decade, and no two events are exactly alike.” These clear irregularities should be related to a link established by Karin Labitzke of the Free University in Berlin, i.e that the relations between the Sun and the climate can be linked to the eleven years cycle of the solar activity, on condition to integrate the near-biennial inversions of the stratospheric winds. Nesme-Ribes (1990) also made a relevant remark when writing on the work on sunspots of the team mentioned earlier, at the Observatoire de Paris, she stated that “the apparent maxima and minima of the solar diameter coincide with the onset of the stratospheric winds”. This means that the Sun presents two recurrent phases during the minimum of activity in between two maxima in the eleven years’ cycle.

5.2.6 - Between the tiny and the enormous

With the present section, our text arrived at the highest scale it considers. The immense planetary system with its immense power generator in the middle beats the drum to the rhythm of which everything and everybody marches on, willingly or not. The signals are captured, read, and converted to behaviour of Life at the tiniest scale of complex molecules.
Within the welter of entropic, abiotic forces of cosmic dimensions, a spark of enthalpic, biotic pushes is answering. Expanding and incorporating more and more matter, the spark grows into the mighty biosphere, filtering out the crude drumming to a large extent, until it is perceived as no more than a faraway signal. The dance of the two partners, biotic and abiotic, is a local, terrestrial expression of a universal duality in which the extremes of life and death are states never completely reached. We will consider the dilemmas of our own, human position in this cooking pot once more in the final chapter.
Chapter 6  Struggle of life

The present book was started in 1991. Since then, a growing number of publications appeared that described experiments and observations agreeing fully with our analyses and syntheses. An article by Weindruch (1996) in the Scientific American, on animal aging, was brought to our attention by Prof. R. Hoekstra, geneticist of Wageningen University. Remmers (1997) submitted a surprising paper on biodiversity to a congress of rural sociologists. Amabile-Cuevas & Chicurel (1993), in the American Scientist, used a title which directly links their article to our chapters 3 and 4, i.e. “Horizontal gene transfer. Gene flow from parent to child is the basis of heredity. Gene flow from unrelated organisms, even across biological Kingdoms, may be a cornerstone of evolution”. All these examples and data, added to ours, perfectly demonstrate the behaviour of organisms under the influence of their environment.

This behaviour of organisms or communities may follow one out of two principles, depending on their state. One state is endogenous order, the other is exogenous disorder. Living, organic systems are open and always exchange matter and energy with their surroundings. Non-living order and disorder were defined by physicists for closed, lifeless thermodynamic systems. Physical disorder is linked to entropic energy, order to enthalpic energy. Enthalpy is useful energy in a wound-up clock, useless entropy is produced when running the clock down.

Living systems maintain order by strict control of the exchange between the open systems and their surroundings. The importance of boundary filters at all levels of Life was emphasized in section 5.1. The state in which such filters maintain strict order inside system limits and allow strictly regulated growth of the system itself was called biostasis by Oldeman (1974). An example is a large tree, living three centuries in a balancing metabolic state inside the same ecological volume, another case is the strict way of life of nucleotides in a chromosome.

The onslaught of external stress causes breakdown of biological boundary filters, disorder. This is not merely entropic, as in physics, because it is no mere entropization of energy in a closed vat. In living systems, stress undermines many-leveled architectures and degrades their structuring energies. However, living systems have responses. They can confine the damage (cf. Shigo 1986,1989), or repair biostasis, or again establish a new and different biostasis.
6.1 - Order, enthalpy and biostasis: organisms and Mendel’s laws.

A system keeping its organisms and their direct environment in balance is ordered, in biostasis. It is stationary, because inputs and outputs through the filtering interface balance each other out. It has a thrifty energy budget, because any increase of output due to wasting of energy inside leads to net losses and increasing disorder. Indeed, it maintains order inside the filter with great care. It finally displays a harmonic set of interactions among all components, cells in a tissue or organisms in an eco-unit.

This modus vivendi includes commensalism between organisms, when reciprocal advantages are exchanged between host and commensal. It also contains certain forms of regulated parasitism, like in the witch’s broom, a version of branching due to a tissue response to local impacts on dormant buds by fungi or viruses. Still another form of symbiosis is the healthy and controled interaction between mammal intestines and their intestinal flora (Sect. 5.1).

Indeed, “good neighbourly relations” at all levels, between communities, organisms, organs, tissues and cells depend on correctly functioning interface filters. The cellular membrane is a privileged filter. It controls exchanges affecting many other, higher and lower organization levels with vital or lethal effects. These membranes also owe their special power to their filtering the transmission of genetic information from parents to descendants, i.e. vertical transmission. If and when they function correctly and are in balance, transmission is orderly. The claspers at all levels function regularly, the code of their locks remains the same over long timespans, and the architecture of Life expresses this regularity following Mendel’s Laws.

6.2 Disorder, entropy, degradation : the Laws of Adaptation

A system unable to keep its organisms and their direct environment in balance is disordered, in degradation. It is running down, since either outputs through the filtering interface exceed inputs, or inputs are fouled. Its energy budget is irregular, since inputs are no longer upgraded and disorder increases. The system struggles to reestablish interior order. It finally displays dissonant interactions among all components, e.g. cells in a tissue or organisms in an eco-unit.

Examples of unbalancing of systems are very numerous and are known of old under names such as diseases, pests, intoxication, cancer, ageing, pollution, or desertification.

Major stresses were defined above (Sect. 5.2). Radioactive radiation, non-photosynthetic light, free radicals, bypass all filters at higher organization levels, like ecosystem canopies. They hit cell membranes directly, oxidize or modify them, and so damage their filtering function. This partially or wholly blocks meiotic pairing of homologous chromosomes in proliferating cells (asynidesis). Vertical transmission of genes is arrested. The biological claspers at the genetic level do not open at the usual codes, and neither do the claspers at higher levels which depend on healthy cells. The architecture of Life makes this irregularity visible at all levels as malformations, incomplete structures, and limping morphogenetic rhythms.
6.2.1 - Elimination and encystment

If such damage is localized at the cell level, it is dangerous for surrounding cells and is beyond repair, cells commit programmed suicide (apoptosis). It operates in animals by selective unblocking of cytoplasmic caspase-activated DNase (CAD), becoming active by the removal of its inhibitor ICAD (Enari & al. 1998). Wyllie (1998:21, abridged) speaks of a mechanism that destroys DNA effectively (host as well as viral) in the process of removing virally infected or otherwise unwanted cells; an endonuclease restricted to apoptosis but not to any particular target sequence, and a superbly designed, highly efficient and tightly controlled removal kit that is able not only to kill unwanted cells but to bury the evidence, and fast.

Cell killing, and the cutting up of their long DNA strands (cf. Sect. 4.2.2) in bits so tiny as to be unable to wreak genetic havoc in neighbouring cells, rather fits in life cycles of organisms with a fixed volume, such as animals. This is supported by an article on cancer by Van den Hooff (1996). Based on extensive literature, he describes how cancer nearly always originates in epithelium, i.e. the outer cell layer of animals, also “outside-in” like the lining of intestines or lungs. Epithelium sits on connective tissue (stroma) containing an extracellular matrix (ECM), veins, collagen fibres holding things together, and cells including fibroblasts. The latter are hyperactive when cancer develops in adult tissue, may perhaps secrete demolition enzymes, and sometimes move like embryonic fibroblasts engaged in morphogenesis. Is cancer a disturbance of tissue architecture? - asks Van den Hooff, using the word architecture.

Connective tissues, he says, have been recently shown to play a critical role in the interactions that determine the architecture of animal tissues and even organs and bodies. He calls ECM the “immediate environment” of cells (cf. Chapt.5.1). Among the cells suspended in the ECM, free organic molecules are found, carrying information needed for organized buildup. The ECM influences the behaviour of the genome of surrounding cells. Sound cells grafted experimentally upon cancerous stroma fall ill, whereas cancerous cells grafted on healthy stroma often heal. The author links the architectural instructions to the 90% of DNA shown not to code for proteins. This vast majority was called “junk DNA” in earlier literature.

These processes have been described in much greater detail, including chromosome disorganization and the role of apoptosis, by Van Noorden & al. (1998).

The whole image fits into the concept of fixed-volume animals. Elimination of corrupt cell clusters at tissue level is their only way to maintain a healthy architecture. We mostly know this process today by its mistakes, under the common name of “cancer”. Cancerous clusters are not eliminated but start colonizing. Central to this image is the concept of architecture. Hallé (1978:217, abridged), in his study on architectural variation and mutation in plants says “In most cases, the form of the mutant is not arbitrary; it belongs to a known model, normal in other species. Plant species must conform, over and over again, to a few basic designs.”

In proliferating organisms like most plants, biomass indeed is eliminated at other organisation levels, e.g. by hormone-driven leaf or branch abscission or by “compartimentalization”, i.e. encystment, within “boundaries that resist spread of pathogens” (Shigo 1986:24). Plant cells, tissues or organs to be eliminated often indeed are “embarrassing” because of “healthy” processes being denatured. The “horizontal transmission” of genetic material, for instance, can go awry by transfection, transfer to a cell of pathogenic matter causing DNA to dysfunction. Unrestricted woody outgrowths on stems of branches of trees are implicitly and correctly compared with such phenomena by the name of tree cancers. So are leaf cancers.
6.2.2 - The adaptive response

There is only one solution “of the last hope” to keep cells alive, to wit an alternative way of gene transmission in the impaired condition, by an adaptive response. A dissipative structure is built at the DNA level to reroute the impact of stress. Part of the enthalpy in the cell is used in this way, establishing an adaptive, two-route mechanism.

- **The first route leads to short-term results.** It substitutes a more primitive form of gene transmission for the orderly, sexual, vertical gene transmission of the Eucaryotes.
- **The second route leads to results on the longer term.** It cancels the effects of membrane oxidation due to the major stress, by the emission of an anti-oxydant. This is an enzyme coded by new genetic sequences which are either transferred to the genome from outside, or due to in situ mutations of genetic sequences. Combating oxidation of course also is an ancient mechanism in plants, operational since the air contains a large share of oxygen.

The adaptive mechanism must bring the following properties to follow this double road:

- **Like a retro-virus,** it should possess the ability to integrate its own RNA in a DNA molecule due to the action of the transverse transcriptase enzyme.
- **Like a procaryote bacterium,** it should possess a “fertility factor”, transferred from donor cell to recipient cell by plasmids, the circular two-stranded extrachromosomal DNA elements we encountered above (Figs. 3-12, 3-13). These are the “tools” of horizontal gene transfer from one organism to another one, of the same or another species. They include all paraphernalia in a rich, new toolbox full of regulatory proteins.

These two properties, favouring genetic flexibility, were probably acquired by organisms very early in the history of Life spreading over our planet. Without them, the evolution of the Eucaryotes is very difficult to explain. At the outset, it requires a double contamination, to wit a first infection of a bacterium by a retrovirus, both procaryotes, followed by a second infection of an procaryote cell, e.g. a protozoic one, by the hybrid bacterium carrying retroviral DNA. The other way round, a protozoan carrying retroviral DNA already infected by DNA of a bacterium, may receive bacterial DNA by such horizontal transfer.

The adaptive mechanism is preceded by the activation of centromeric sequences, mute in ordinary conditions, in the chromosomal DNA. These sequences play the part of chaperon molecules in the rearrangement of the initial DNA molecule. Two kinds of DNA molecules, one dexter and the other sinister, act in this process in addition to the initial DNA molecule which is dexter. Activation is triggered by an electromagnetic signal from the high atmosphere, i.e. diffraction of the sun’s photonic radiation at sunrise and sunset. Its wavelength is modulated in time following the moon phases at the moment of first cellular division (Chapt. 2), marked by three situations, i.e. full moon, new moon and first-or-last quarter.

If the first divisions occur during full moon, the wavelength of the signal is in the far red ($\lambda \approx 730$ nm) and the DNA chaperon molecule becomes dexter (molecule A). If they occur during the new moon, the wavelength is in the clear red ($\lambda \approx 660$ nm) and the chaperon DNA is sinister (molecule Z). During either one of the moon’s quarters the wavelength is shorter ($\lambda < 600$ nm) and the chaperon molecule is similar to the specific original DNA (molecule B). In this way, three classes of individual organisms, with different molecular DNA mass, coexist in the same population. The first class has an original DNA molecule in what we called the specific mode B.DNA$_{(M)}$ in chapter 3. The second is the delayed variant with B- DNA$_{(V_m)}$, its
molecular mass being about 15% lighter than in the specific mode. The third class is the early variant, its B.DNA_{VM} being some 17% heavier than in the specific mode.

The two variants play complementary parts (Sect. 3.4.2). The delayed variant is written in an imperfectly corrected coded language, so enhancing the amount of mutations by genetic drift. It also contains extrachromosomal circular DNA elements which are information vectors, the plasmids.

The early variant carries energy and transposable elements, making possible the insertion of DNA originating from a plasmid copy, so enriching the chromosome with stress-resistant genes.

With so many “jokers” in the card pack, there will always be a fortunate winner of the “ten million Euro” prize. With time, the number of winners increases until the stress factors barely affect the population anymore. This is so for all stress factors, from the major atmospheric ones to the acute impacts of virulent toxins, parasites or pathogens.

Albert Einstein declared in The Observer in 1954: “I can not believe that God plays dice with the cosmos” (ex Van den Hoff & Van den Hooff 1995:15). In the context of the present pages, one comment could be: “Perhaps God does not play dice ..... However! in our world falling apart at a terrible rate, a little biased sleight-of-hand was certainly indispensable to force the creation of some havens of peace or niches of balance in a disorderly universe”.

6.3 - Order in the midst of disorder : the secret of Life and diversity

Stress conditions quiet down. Systems return to a more or less balanced state. The damages due to stress have been repaired and the living system rendered immune. Mendel’s laws take the upper hand again. Vertical gene flux by the classical rules of genetics once more prevails.

This regularity is no simple mechanical beat, as expressed by Life’s architecture, each higher level of organization being the direct environment of the next lower one (Sect. 5.1.1). Mendel did not discover his genetic laws by studying genes. He found them with the naked eye in plant morphology or in the colours of flowers. A balanced organic state recurs at each level, as filtering interfaces expel entropy and keep enthalpy in. This seems to contradict the second principle of thermodynamics stating that every physical system runs down by entropization.

However, this architecture on the contrary is a literal biological version of a nested dissipative structure, sensu Prigogine & Stengers (1984: 143 abridged): “...the close association, at first paradoxical, between structure and order on the one side, and dissipation or waste on the other.” In fact, the system hierarchy of chapter 5 reflects the bifurcation diagrams by May (ex Gleick 1989:77 ff.), where at a point far from equilibrium a system may produce order at a higher energy level than the “window of order” preceding it.

If a system is stressed, maintenance of order can be delegated to a lower or a higher level. We met this earlier as the rule of transfer of functions (4.4.3). The above two routes to adapt to stress are exactly that. The function transferred is the task of the genetic ordering system under prevailing circumstances. A transfer to a lower energy level is operated by shifting to
primitive, asexual mechanisms of gene transmission. A transfer to a higher level of energy is operated by shifting to larger carriers, e.g. plasmids, for horizontal gene transmission.

Amábile-Cuevas & Chicurel (1993) cite two cases of horizontal transmission in Eucaryotes.

First, Doolittle & al. (1990), at the University of California, working on close associations between organisms, studied *Escherichia coli* (see Chapt. 1). In one of its metabolic bacterial enzymes, Gapdh or Glyceraldehyde-3-phosphatedehydrogenase, they discovered a striking particularity. *E. coli* possesses not one, but two genes coding for Gapdh. The first is close to the Gapdh gene in other bacteria. The second, however, strongly resembles the Gapdh-coding gene in Eucaryotes. “In addition to *E. coli*, more than a dozen other intestinal bacteria contain the eucaryotic variant, which suggests that the initial transfer was from a eucaryotic cell in the intestinal lining of the host to the ancestral bacterium.”

The second example is a transfer the other way round, from a bacterium to an eucaryotic cell. Certain bacteria live in the cytoplasm of amoebas, which are Eucaryotes. The bacteria convey to the amoebas a pathogenic capability, so they can cause a form of dysentery, amoebiasis (cf. Sect. 4.3.1.4.(a)). However, in its association with the bacterium, the amoeba has acquired a new gene too. This codes for iron-containing superoxide dismutase, an antioxidant enzyme which usually occurs in bacteria only. “It is interesting to note that under stressful conditions, several bacterial species survive by living within amoebas and other protozoa...”, note Amábile-Cuevas & Chicurel (1993).

This well illustrates exchanges which are not yet full fusions of organisms, but already exceed mutualistic symbiosis somewhat (cf. Sect. 4.4.3). The partners and their environments stem from quite different levels of organization. Under stable conditions, the bacterium happily proliferates in the eucaryotic amoeba, its immediate environment. When the environment of the amoeba worsens, one level up, the bacterium, one level down, lends the amoeba a defense against oxidation disorder. The bacterium so ensures the sustenance of its energy provider.

However, this demands one essential condition. Both the bacterium and the amoeba must possess the same password, a coded sequence, for their type of biotic union to be possible. The bacterium has to belong to a lineage which, in the near or remote past, was invaded by a parasitic retrovirus similar to the one having keyed the adaptive mechanism of the amoeba.

Weindruch (1996) studied the effect of calorie-poor diets on aging of animals, from protozoans to insects, fishes and mammals. Experiments show that animals, ingesting ca 30% calories less than normally fed control animals, lose about a third of their body weight and show postponed aging. They attain an average age of 1½ to 2 times the species average. Calorie-poor maximum age is in excess by 10% to 50%, exceptionally more. The cause is caloric. In no experiment the quality of the food carrying the calories made any difference.

In low-calorie animals, 90 out of some 300 age-marking properties stayed “younger”. For instance, decline of blood glucose control, of DNA repair and of learning ability were delayed, increases in oxidation damage to tissues or cross-linking of long-lived proteins were slowed down, and the onset of diseases common in later life, such as autoimmune disorders, cancers, or hypertension was delayed. Weindruch (1996) and the authors he cites found the cause of these remarkable changes in a factor that we already pinpointed at the start of section 5.2, to wit free radicals causing oxidation of cell components. In the present case, however, they come from components of the cell itself, the mitochondria, not from the atmosphere.
Mitochondria are powerhouses, synthesising ATP (Sect. 3.2.2; 3.3.1.3). The inner membranes of mitochondria carry complex chains of chemical stations transporting electrons from one to the other. ATP is produced at the end by the enzyme ATP-synthase. Waste products along the way include free radicals, particularly abounding at the ubiquinone station. Free radicals accumulate in the mitochondria, degrading the production chain. Production of free radicals then increases, whereas ATP production decreases. The mitochondria degrade and die, their decomposition freeing products noxious to the whole cell, which gradually starves.

High levels of energy conversion speed up these processes. Now mitochondria are said to have evolved from a symbiosis between eucaryote cells and aerobic bacteria (Sect. 4.4.3). The process appears similar to the coded gene transfers above. It is significant that highly energized cells risk trouble with the processing of so much energy, whether or not the decay of mitochondria is the main cause of cell aging in general.

The energetic status of plant cells is boosted by increasing numbers of chloroplasts when DNA mass rises (Chapt. 3). This resembles high food intake by animals. Larger sizes and shorter lifespans may be expected in such cells, both properties relayed physiologically to the tissue, organ and organism levels.

Anxolabéhère & al. (1989) wrote a survey “The history of a genetic invasion”, on higher organization levels. They illustrated the stress responses at those levels by a genetic infection and the advent of new Drosophila species due to mobility of genetic elements. Drosophila, a cosmopolitan genus of winged insects, “fruit flies”, consumes fermenting fruit. Its prolific procreation and easy culture make it into an ideal lab animal, also with countless mutations compared with its wild relatives. Mutations are visible by morphological markers and eye colour, so Drosophila genetics were studied intensively over many decennia.

Anxolabéhère & al. (1989) showed the part played by transposable elements in the evolution of its populations. “In fact it clearly shows that certain transposable elements, called P-elements, are invading the species Drosophila melanogaster at a worldwide scale” (Transl. RAAO).

These particular P-elements show four regions coding for transposase. Quite in line with the present study, “it is very improbable that the P-elements transpose themselves by literally jumping from one place to the next, but it is probable that this rather happens by what is called replicative transposition. In this case, the DNA of mobile elements probably is “doubled” at each transposition, one copy remaining at the original site, whereas the other one is integrated elsewhere in the genome.”(Transl. and italics RAAO).

We add that this happens only, either in the genome or outside, under stress conditions, if the DNA of the receptor genome has similarly coded sequences (Sect. 3.3.3.2; 3.3.4). The conclusions by Anxolabéhère & al. (1989) support the adaptive process as described in the present book.

They wrote: “...The data obtained are perfectly compatible with the hypothesis of a recent invasion of the species D. melanogaster by P-elements, advanced in 1979 by M.G. Kidwell (Kidwell & Sang 1986 ex Anxolabéhère & al. 1989). This invasion would have started before 1950 in America, the SE zones of the USA or Central America being possible epicentres. The invasion of Europe and the other continents would have happened over the next decade and this process would not be totally at an end [in 1989, RAAO]. This phenomenon of a cosmopolitan species being invaded is unknown for classic chomosomal genes. It indeed
reminds one because of its dynamics ..... of the present spreading of the HIV virus (responsible for AIDS) in the human species.” (Transl. & ital. RAAO).

The worldwide distribution, according to UN reports, of a total of 30.6 million cases of AIDS, is enlightening. The hotspots of the epidemic lie South of the Sahara in Africa (20.8 million infected adults), followed by South and South-East Asia with 6 million, and tropical America with 1.3 million. Compared with these explosive epidemics, numbers of infected adults to the North of the Southern Sahara latitudes seem ludicrous. North America has 860,000 cases, Western Europe 530,000, North Africa and the Middle East 210,000, Eastern Europe and Central Asia 150,000 and Eastern Asia with the Pacific has 440,000 cases. Elsewhere in the South Pacific, Australia and New Zealand show a rather modest 12,000 infected adults.

It is often claimed, that such unfair disparity occurs because human populations North of the Sahara live in the most favoured regions of the planet. This is largely true. Huge means were mobilized to wipe out the epidemics and we owe high credit to the medical teams who have selflessly dedicated their lives to combating illness, often in hostile milieus (Garrett 1994).

Still, the HIV explosion in the two main focal points, Central Africa and South-East Asia, evidently is in another class altogether. This is underscored by the fact that the much-publicized epidemic started in North America, where Public Health Services were rather taken by surprise and needed several years to set up an adequate response. So the HIV virus multiplied with a headstart and, by a boomerang effect, invaded Central Africa and South-East Asia to do its terrible job there. The backlash was less violent in Europe, the rest of Asia and the other Southern continents. All is very similar to the invaded Drosophila fruit flies.

In both cases we may assume a boomerang effect. Due to genetic drift away from the original genome, this effect causes populations far from the region of origin of the species to be at a great “genetic distance” too.

Now let us consider in the region of origin, somewhere in the world, an immigrant from faraway, the cells of whom show an adaptive response to climatic stress, to be infected by a local retrovirus. In that case, the very exoticness of the DNA introduced in the cells of another host than its usual one probably reinforces HIV virulence. This is wont to provoke a severe immunodeficiency syndrome in the victim.

Everything suggests that HIV and certain related forms are endemic, parasitic retroviruses in human and other primates of Central Africa and South-East Asia. Their biotic union with humans certainly is not recent, perhaps as far back as the appearance on Earth of Homo sapiens sapiens during the last Super Ice Age, the Würm. Living passively in human cells when climatic conditions are quiet, the mute DNA of HIV is regularly replicated during cell division. During a major stress period after a solar event, however, the pathogenic properties of this DNA awaken, due to the adaptive response by the cells of the host. Even then, many diseased cells are probably killed and reduced to dust by apoptosis.

The boomerang effect supports the thesis that all humans living on Earth today originate from one African lineage that swarmed out all over the planet. Travelling from Southern and Southeastern Africa, one branch went down the Nile and crossed the Sinai desert to the East. Part of the people then voyaged North and West and invaded the Mediterranean region and Europe. Two branches went East and ended up in the Americas by two different routes.

The first branch crossed the whole of Asia, traversed the frozen Bering Strait and spread out over North America. The second one crossed the Ocean following the Pacific Counter-current
and settled in South America. A third one much more recently crossed the Atlantic in successive waves coming from Europe and Africa. Iberians with African slaves colonized South and Central America after 1,500 AD. Between 1600 and 1800 AD, other European nations successively did so in North America where they imported Africans too, until 1865 saw the Abolition of slavery. These migrations were discrete waves and had little in common.

This is how it works:

### Diagram:

1. *Homo sapiens sapiens*, genetically farther from the Super Ice Age Würm person than 2; genetic distance larger still because of the adaptive response to climatic stress

2. *Homo sapiens sapiens*, genetically closer to Würm Man than 1; with endemic, not very virulent HIV chromosomes

Stress semi-permanent or contingent, which by sexual transmission to 1 becomes more virulent and turns into a severe selection factor.

DNA of 1 has become exotic in 2 cells and the other way round

Contamination of 1 by 2 AIDS gene

1. $^+_s \delta$

2. $^+_s \delta$

**BOOMERANG EFFECT** → AIDS EPIDEMIC

with the unruly crowds of tourists crossing the planet today in all directions by the billions.

It is certainly the much closer contact between American travellers and the inhabitants of the original human territories in Africa and Asia which, during the solar events of the 1980’s culminating in 1989/1990, triggered HIV virulence in infected Americans. Some years later and unintentionally, these travelers sent the boomerang back to the other continents.

### 6.4 Species, ecosystems, succession, competition and biodiversity

In the light of the above, some notions must be reviewed here now, especially in ecology and population biology. Otherwise, a flaw will persist in our logic regarding the struggle of Life. This flaw exists since the early studies of biological communities. From organic molecule to organism, Life was structurally or morphologically regarded. Reproduction, implying two organisms at most, also was structurally interpreted. However, as soon as more than two
organisms entered the spotlight, scientists started to count. Their discourse became numerical instead of structural. Individual plants or animals with high structural similarity were counted and grouped into species. These inseparably linked high similarity between individuals to high similarity between parent and offspring and exclusive reproduction within the species.

This species concept is in line with an ancient human task, told in Genesis 2:19,20: “And out of the ground the LORD God formed every beast of the field, and every fowl of the air; and brought them unto Adam to see what he would call them: and whatsoever Adam called every living creature, that was the name thereof. And Adam gave names to all cattle, and to the fowl of the air, and to every beast of the field;...” Species were recognized after visible form and colour also when fine criteria became visible later, with lenses, optical microscopes and stronger magnifiers. Taxonomy is the oldest profession, notwithstanding other rumours.

Today, micromorphological criteria have not only been refined without precedent, but also cast a new light upon species’ reproduction. Lewis Thomas, famous New England medical doctor, philosopher, and columnist of the New England Journal of Medicine (1992: 100 ff.) points out that the oldest known ancestor of Life is a fossil, 3.7 billion year old, bacterial cell from Australia. He retraces Life from that faraway past, emphasizing the unbroken genetic link with ourselves (also Dean 1998). Bacteria, we saw, “invented” DNA transmission by plasmids or viral “transfection” across species limits. Nature’s November 1997 issue depicts the entire genome of Bacillus subtilis, indeed with ten bacterial viruses among some 4100 genes. Thomas (1992) cites Sonea and Panisset claiming that all bacteria together form one giant kind of clone englobing the majority of the biomass on our planet.

Indeed, limits between monocellular species are fuzzy. The combinations of properties defining these species today may have shifted tomorrow. Earlier chapters show this to be the same at other levels of organization, due to stepwise selection by nested direct environments (Sect. 5.1). Recently, Gao (1998) reduced five species of the Chinese shrub genus Exochorda (Rosaceae) to one species with three fuzzy subspecies, complementing classical taxonomy by wood anatomy, chromosome numbers, DNA analysis and floral biology.

A general term for the transfer of information-bearing parts in whole multicellular organisms is grafting. Plasmid or retrovirus transfer can be seen as grafting at molecular level. Changed cells are “grafted” upon the tissue of which they are a part and change the properties of that tissue. Tissue and organ grafting, yesterday mostly horticultural, today are known most widely from medical surgery. The changes in architecture and behaviour of the body they may cause by information transfer mostly still remain worried speculation in daily newspapers (Chap. 1). The debate is exacerbated in 1998 by potential transplantation of animal organs to humans.

Horticultural grafting is comparable to reiteration. Horticultural chimeras, made by grafting one tree species upon another, find a parallel in vitro (Chapt. 3). Chimeras are organisms with changed architecture and behaviour, built by cells from different genetical lineages. Natural grafts between tree branches and among roots are common (Oldeman 1990). Communication between the sapstreams of grafted trees causes exchange of hormonal information. The Dutch elm disease shows another transfer of information. The fungus Ophiostoma ulmi, brought in the sapstream by coleoptera of the genus Scolytus, can travel from one elm tree to the next by the sapstream in grafted roots. Such fungi often carry bacteria (cf. Hiemstra 1995). Bacteria carry information, or dysinformation when pathogenic, at the cell-cell interaction level.
In branching and reiteration, new information can originate from somatic meiosis in stressed meristems (Chapt. 4). Solid evidence of the existence of such processes was published just before the present book went to the press. Murawski (1998) substantiated the coexistence of Random Amplified Polymorphic DNA (RAPD) and morphological polymorphism in giant, old rainforest tree crowns, studied from the Canopy Raft. This can also be due to horizontal transmission by bacteria in infected meristems. In ecological communities, information carriers are seeds, spores, eggs, cuttings or clones. In quiet times, they repetitively build successive, similar eco-units, so the average eco-unit composition, species composition and biomass distribution of a known vegetation mosaic remain similar over longer periods. Ecological fragmentation dynamics change under stress (Chapt. 5). With them architecture and behaviour of the vegetation mosaic change, including all its species, from microbes to trees and animals. “Tuning” of larger information carriers like seeds always does start at the genetic level (Oldeman 1990), horizontally or vertically.

Taxonomic species descriptions are instant pictures. Over vast timespans, they are frames of a movie. Herbaria and collections of plants and animals in conservation liquid, labeled with notes and the dates of collection, provide a reference to these timespans for multicellular plants and animals. Those specimens remain valid during centuries at least. Archeological drawings show species still quite similar to our larger plants and animals (Attenborough 1989). Even on stone age murals, species are recognized. However, they are not the species of our times, as proven also by the fossil record. The European Douglas Fir of 40.000 years ago is recognized as a *Pseudotsuga*, but is quite distinct from today’s North American species, *P. menziesii*. Other species have disappeared altogether, like the mammoth.

Three general statements now can be made about species;

- Species inherently have no crisp, but fuzzy limits in biological time, they are crisp sets of organisms only in a point in mathematical time.
- Species, when described by a fuzzy definition, are born and die and have a natural lifespan, some being longlived, others being shortlived.
- Species only have the reality of their definition by Adam, i.e. by those characteristics that remain constant when perceived over a human lifespan.

*A species is a cluster (population) of clusters (organisms) of genetic information, its material expression being recognizable by the human senses, and persisting, by various means, over a longer or shorter timespan while living in a direct environment that feeds it.*

Feeding reigns supreme in much current work on physiological ecology, emphasizing fluxes of matter and energy, and biomass accumulation. The ways and means, and the life histories of species adapting to different “food and stress niches” lead to sharp, fundamental discussions, e.g. between Grime and Tilman as analyzed by Grace (1991). The latter showed that much conflict was due to basic viewpoints, laid down in definitions. This is emphasized here because our definitions are much different too. The ecophysiological image indeed does not conflict with the architectural one, which depicts the canals and structures carrying food and energy, but usually ecophysiology neglects the transient nature of species and ecosystems.

Species are not constant but transient elements in ecological systems, even if some species have long lifespans and other species have very short ones. The contours of the definition of ecosystems so become uncertain, because the composition of their participating species is not constant. Let us recall the general lack of crisp natural spatial and temporal limits apt to be used in defining ecosystems (Sect. 5.1). *The picture of “competition” then shows neither a*
crisply defined arena, nor crisply defined competitors. The usual competition models assume
too many structures and processes to be constant. It is as if the sea were portrayed by a very
short-lived observer, who would see the waves standing still and the fishes in fixed positions.

With a biosphere perspective of 3.7 billion years, the view shows an alternatingly growing
and receding carpet of land and sea Life. In this carpet, bubbles of enthalpy of increasing sizes
interact with a surrounding, chaotic matrix of entropic forces. The bubbles interact mutually
too. Higher organized ones often embody lesser systems by interactions ranging from feeding
to symbiosis to incorporation. Both parties change irreversibly even if one simply feeds upon
the other. Overly complex interactions beget chaos (Gleick 1987). This can be reorganized at
a higher energy level only, shown by the bifurcation diagrams in Prigogine & Stengers

These energy levels correspond to the kind of levels defined in this book and earlier (cf.
Oldeman 1990). In the present state of biological and ecological sciences, however, the
prigoginian laws at the higher, ecological levels of the organization of Life can not be defined.
Prigogine & Stengers (1984:191), in their chapter “Order through fluctuations”, indeed state
on feedback between small and large in evolutionary theory: “Such interrelated processes
generate very complex situations, the understanding of which is needed before any kind of
modelization.” This is why they describe only very simple cases.

The present book shows that no case in Life is simple. The inherent fuzzyness of the limits of
organisms, populations and communities causes a slight dissimilarity in interactions in similar
situations among similar life forms. The players are not precisely the same, from the level of
nucleotides to the biome level and from one moment to the next. Morawetz (pers. com. 1995)
had a rain forest model made, in which the computer generated forty or so tree species, each
with a different temperament. Temperament is the package of reactions of a plant towards its
environmental stimuli. The computer-generated “trees” were “sown” at arbitrary places in a
computer-generated “forest plot”, and made to “grow and interact”. After fifty “computer
years” the screen showed a convincing picture, resembling a tropical rain forest.

The scientists then discovered that they had not saved the initial situation of the plot and the
“seed coordinates” in the computer memory. They tried to retrieve this by calculations to
backtrack the situation from 50 to zero years of “computer-age”. This proved impossible. Here was mathematical proof that complexity of interactions leads to a form of chaos that is
neither cyclic, nor predictable. Direct proof is the “high salvage rate” in Germany’s annual
wood harvest. Much German wood is harvested from dead or dying trees that did not behave
according to regular, cyclic model predictions made by foresters (Büting 1984:351).

In the four “real” dimensions of direct observation, space and time, neither “competitors” nor
“communities” can be defined as crisp, constant or regular. The assumptions of “succession”
and “competition” therefore must be revised. The present tenets are linked to the concept of
the ecological niche, defined by Hutchinson (1957 ex Begon & al. 1986) as “a species’ true
ecological niche is a n-dimensional hypervolume within which a species can maintain a viable
population”. Begon & al. treat factors like humidity or availability per mineral nutrient, and
temperature, a parameter of heat, as “dimensions” of that volume. Numbers and frequencies of
species and those of “factors” then are linked up in a wholly numerical virtual ecosystem.

The computers of today visualize such virtual ecosystems on screen. Soon the use of sensors,
earphones and smells will perfect the sensation of reality in the observer of that screen. However, such a virtual reality is generated by mathematical rules and physical means at the
scale of the electron, having little in common with biological rules and means. Both are
separated by many levels of biological organization, in which identity, fine limits and
erring processes are stepwise replaced by similarity, fuzzy limits and shifting dynamics.

The degrees of freedom indeed are much more numerous and larger in biological systems than
in systems generated by computers. Computer certitudes can never constitute proof of
biological buildup and functioning. Computers give excellent help in mapping and portraying
Life, but not in explaining it conceptually or mathematically. Computers are programmed in
such a way, that any deviation from the program is an error per definition. On the contrary,
the struggle of Life in an entropic universe rests on the frequently recurrent option of either
putting such deviations to use or discarding them (Sect. 3.4.1; 6.2).

Computers are restricted to evoking the past engraved in electronic memories. Living systems
evoke the past too but, under stress, have an option to save new, unexpected information in
their genetic memories. Both function independently of the future. Computers do so because
they are denied access to innovation. Life does so because living is living today. The present
book hence does away with any future “aim” of life. The explanation of Life does not require
any plan, aim or other teleological future. There is no “race” to run “against” a “competitor”.
The racetrack stops here and now in the present, nobody knows where it leads in the future,
and today’s “competitor” may be tomorrow’s “symbiont”.

Computers are difficult to program for meaningful incorporation or rejection of deviating
instructions, as proven by long and disappointing studies in artificial intelligence and expert
systems. The difficulty is compounded by the hierarchy of ten or more stacked organization
levels without other than historical references, the Prigoginian initial states. “Artificial life”
simulations (e.g. see Levy 1992 or Dawkins 1986) are clever portraits from the outside. Their
generation by computer has nothing in common with the biological genesis of the portrayed
organisms. Dawkins’ “electronic genes” are no biological genes.

A better image shows the whole mass of organized Life, in all its diversity, pressing the wall
of the present into the future, in all its diversity. This requires an image of a diverse past, a
fuzzy present and an open future, as proposed for human civilization by Remmers (1998, our
fig. 6-1). The best forces for the pushing job are unceasingly mobilized out of the past. At all
organization levels shown (Sect. 5.1), diversity reflects the indispensable biological “library”
of the past with all deletions and additions needed today. Deletions are extinctions, ranging
from a genetic sequence being “overwritten” (Sect. 4.3.1) to the whole information package of
a species becoming extinct. However, this is compensated by additions such as new
sequences, new transfections, new forms of symbiosis or new species.

The biological present then is no physical moment with a dimension of zero (Fig. 6-1). It has
a fractal dimension a little above zero. Material evidence was recently discovered. A team
lead by Prof. R.M. Buijs from the Netherlands Institute for Brain Research in Amsterdam, by
“...anterograde tracing in the adult human postmortem brain ...of efferent connections of
the biological clock....in the human hypothalamus”, found that “...with suitable in vitro treatment,
survival of human brain cells is possible up to 8 hours postmortem delay, in such a way that
they still have the potential of recovering their axonal transport” (Dai & al. 1997; also see
Buijs 1998). The 8 hours’ delay appears to be a first indication that the “fuzzy present” exists.
Is this "the end of predictability" (Broer & al. 1995)? It is, as far as the precise prediction of future structures and events is concerned. It is not, in as far as vertical gene transmission and its results at higher levels tend to reproduce the past. It is not, in as far as we now also know the rules of adaptation to situations where past solutions are not supportive of Life.

The industrial world fashioned by us humans in our ending XXth Century is built following the principles of the computer. Factories are organized for the more or less automatic making of one or a few products, without deviating from product specifications laid down in the past. Such products, from inorganic cars to organic crops, sooner or later become obsolete. For plants and animals used as agricultural tools, deprived of natural adaptation, extinction will overtake speciation. Wild plants and animals, seen as useless or harmful, suffer heightened extinction risks. Great worries about biodiversity loss indeed are pertinent to this situation.

Conservation of biodiversity by creating sanctuaries where species are conserved like floppy disks in a plastic box, is no solution. Living species, transients in Life, can not be conserved indefinitely. Remmers (1997, 1998) states correctly that biodiversity can not be conserved, it must be reproduced. He found old, efficient ways to reproduce biodiversity in Andalucian agriculture of Moorish origin and ancient Cretan land use. Remmers defined the principle of cultivation stops, like bus stops. Farmer Lopez is widely known to have the best beans, so he sells seed beans to people from many kilometres away. After 10 or 20 years, beans from this stock have become excellent on three other farms, ecologically slightly different. These seeds are sold again. Diversity continues to originate from reproduction from one "cultivation stop" to the next, providing quiet waiting periods for the adaptive response to take place.

This way of reproducing biodiversity does not owe its success only to ecological variation of cultivation stops. It also uses the founder effect, alleles in the newly founded population differing from the alleles in the old population, so the resulting plants are inherently different.
(cf. Wilson 1992: 81). This contributes to many cultivation stops being needed to produce only a few resounding, widely reputed successes in crop selection. Another consequence of the founder principle is the above boomerang effect, in which the HIV virus, after a "cultivation stop" in the North went back to the South in a new and more virulent form.

The human task as a steward of this treasurehouse of exceedingly diverse life forms then is perhaps best defined as the supervision and management of the organic planetary memory, or in the biblical words "to dress and to keep the garden of Eden".

6.5 The Sun, the Earth and the struggle of Life

In the above, time and again evolution was seen to spawn new races or species during more or less periodic accelerations of genetic drift, often with species migration. These periods match the caprices of the climate on Earth, ruled by the Sun (Sect. 5.2). In the same periods, species disappeared due to cataclysmic epidemics. This happened particularly during the Ice Ages.

Australian scientists proved this by the analysis of DNA fragments of fossil birds and lizards from different forest refugia. Refugia are forests areas once isolated due to climatic heating and cooling in the far past. The refugia theory originated in the New World. It postulated dry periods during the last Glaciations, with a withdrawal of rain forests to refugia remaining wet enough to support rainforest species (Van der Hammen 1986). Others explain refugia by hydrological regimes of rivers, varying with climatic oscillation (Irion 1989). In view of chapter 5.2 both explanations are largely compatible. The Niño events in Indonesia in 1982-1983 and 1997-1998 suggest that Asian refugia were more to the North and the South.

The refugia studied by the University of Queensland were said to be separated in time by several tens of millions of years. According to the anonymous author in Sciences et Avenir reporting it (Anonymous 1995b:20, transl. RAAO): “all species studied, with one exception, diverged into two distinct populations, every time when an Ice Age occurred”.

In paragraph 6.4 we gave a qualified answer to the question of predictability of biological events. However, can we predict the climate along the lines of section 5.2?

Climatic variation on Earth depends on the instability of the Sun. In its turn this is related to the movements of the planets. The chief planetary movements in this context are those of the Earth, its moon, and the heaviest planets, Jupiter and Saturn. Prediction of their movements depends on regular cycles. Now recent studies, for instance by astronomers at the Paris Bureau des Longitudes (Laskar 1996), showed Earth to move chaotically. So do Mars, Venus and Mercurius. Laskar (1996:26, transl. R.A.A.O.) states: “However, over a timespan of 10 million years, the irregularities of the planetary movements are barely perceptible, and it is possible to calculate precisely the evolution of their orbits over this timespan”.

Figure 6-2 shows a tentative prediction of the climate over the next 10,000 years. Two cycles of major solar instability were mentioned above (Sect. 5.2), the cycle of eccentricity of the Earth’s orbit and the cycle of the inclination of the terrestrial axis towards the orbital plane. Figure 6-2. shows one more cycle from Milankovic’s theory, the precession of the equinoxes.
Figure 6-2: Planetary factors in the solar system, instability of solar behaviour, and climatic variation on Earth. Forces determining the close environment of Earth, seesawing between climatic optima and Super Ice Ages. Tentative climate forecast in the geologically near future. Except for cycles shown above (Figs 5-3,4,5), the precessional cycle of the equinoxes, 25,920 years, is involved (third factor of Milankovic). Main cause of solar instability and dissipation of energy are the solar tides. Figure 5-5 showed minimal orbital eccentricity of Earth every 92,000 years. This causes a High Rank Solar Tidal Maximum (H.R.S.T.M.). Shock is transmitted by solar wind to planetary magnetosphere and high atmosphere. Most important among dissipative structures formed is the polar, or “Norwegian”, cyclone. Two modulators regulate solar impact on our climate. Linked to terrestrial axial movement relative to orbital plane, they are the cycles of the precession of the equinoxes and of the axial inclination of the Earth. The first has a period of 25,920 years. It entails longer periods of winter temperature; i.e. planetary cooling. The second has a rotation of 40,000 years. It causes extension of periods with summer temperatures; i.e. planetary heating. A third modulator is “solar background noise”. It follows Kepler’s Law of Harmonics: “orbital periods (P) squared of the planets are proportional to the third power of their mean distance to the Sun (a) ** expressed in “astronomical units”, or P² = a³. Jupiter is at 5 astronomical units from the Sun, so a³ = 5³ = 125 years. The exact rotation of Jupiter indeed is √125 ≈ 11.18 years (cf. Sagan 1980). The solar wave takes 11.11 years (Sect. 5-2). The disparity of 0.07 y, or 26 days, per year is tiny, but cumulative. Both movements slowly get out of phase, obtain contrasted phases, then slowly get back in phase. The period is of (4,055.1 : 26)*11.11 = 1.733 years. When both cycles are in phase the tidal wave and the solar wave resonate and the “elastic fibre” of the solar chromosphere vibrates. These vibrations are buffered when both cycles are out of phase.

It is due to a gyroscopic movement. Children used to play with tops in the early XXth Century. A top provides a good image of a gyroscope. It spins on the ground, turning around a spinning axis. This axis slowly turns around the vertical, describing a cone with its point standing on the ground. So does the axis of our planet, describing a slow cone around a mean, normal direction with reference to the ecliptic plane. The precession of the equinoxes, timed by this movement of the Earth, retrogrades by a value γ defining the annual advance of the spring equinox. This γ follows the duration of winter. The duration of the precessional cycle
differs slightly according to its calculation. One value takes into account the gravitational forces of sun and moon only, the other is corrected for disturbances by the other planets.

- The solar-lunar precession shows a cycle of 25,920 years
- The general precession follows a cycle of 25,720 years

The precession cycle is nowadays considered “...to be at the origin of the appearance of the Quaternary Ice Ages” (Laskar 1996, transl. R.A.A.O.).

Figure 6-2 integrates all factors discussed earlier. The successive precession cycles do center upon the solar event of 18,027 BC (20,025 BP), which corresponds to Würm IV, the coldest episode of the Würm Super Ice Age.

An important solar event is the maximum tidal effect at the Sun’s surface linked to the cycle of eccentricity of the Earth’s orbit. Every 92,000 years the Earth is in its perihelium phase, i.e. it arrives closest to the sun in its course around it. This leads to an excess of energy, dissipated by the solar wind into the Earth’s atmosphere as heat. Its dissipation proceeds stepwise.

The first step covers $2 \times 6,672$ years of stepwise warming-up, until it reaches an asymptotic temperature curve, the temperature optimum known from palaeobiology.

- The second step takes 6,672 years. The temperature then oscillates regularly around the mean optimum value with a gradually increasing amplitude, until the oscillations become “little ice ages”, one of which occurred some centuries ago (Sect. 5.2).
- The third step finally covers 6,672 years of obvious temperature rise, between the years 1989/90 and 7,893, accompanied by an increase of the CO$_2$ level. Probably, this third period is interrupted by increasingly violent “little ice ages”, the ultimate one being the precursor of the future Super Ice Age.

The cycle of eccentricity of the Earth’s orbit, with a period of 92,000 years, hence is the principal cause of the solar tidal maximum, hence the terrestrial climate change. This is especially so during the warm Interstadials between Super Ice Ages. However, the impact of this cyclic force is modulated by two other factors, working in opposite directions.

- The precession of the equinoxes, with a period of 25,920 years, works towards a cooling-off, because the cold winter periods are extended.
- The inclination of the terrestrial axis to the orbital plane, with a period of 40,000 years, works towards a warming-up, because the warm summer periods are extended at high latitudes. This factor caused the little Interstadial called Amersfoort-Brörup in the midst of the Würm Super Ice Age.

Figure 6-2 shows that, unless physical laws change, the next Great Ice Age is not yet knocking at the door. However, Life on our planet will soon enter into the “zone of turbulence” preceding it. The dilemmas discussed in the introductory chapter 1, caused by such horrors as epidemics, anxiety, agitation, cancer, new diseases, aggression, cataclysmic weather, volcanic eruptions or changes of sea-level announce it. They show the struggle of Life, at all levels, incorporating and transforming entropy such as heat. It becomes organized within the enthalpic “bubbles” evoked above. The struggle of Life so follows its strict rules demonstrated in the present book, under the whiplashes of the sun.
One spectacular, final proof of the whole set of rules is gigantism. We saw that cells under stress contain a proportion of variants (Chapt. 3). The early variant is shorter-lived and ca 17% heavier than the main B-DNA-driven “normal cells”, and the late variant is longer-lived and ca 15% lighter. We saw the reasons for a shorter lifespan of cells living on the high burner (Sect. 6.3). Their mitochondria produce earlier and more abundant free radicals as a side-effect, so the cells of which they are part die earlier. However, this applies not only to the scales of the cell and its parts. Whole animal organisms of very different animal groups show shorter lifespans in well-fed individuals and spectacularly longer ones in case of low calorie diets (Weindruch 1996). There is a syndrome from deviant mass of DNA to deviant body mass, with variants that are smaller and live long lives against the giant ones that die young.

One case is so close to us that it escapes our daily vision. It appears in news items, such as a series of miscellaneous scoops on the size of average Dutchmen now being the largest of the world and so having difficulties finding cars in which they fit. This reality unfolds itself right under our noses since the XIXth Century. It is the appearance in growing numbers of large-sized men and women in human lineages. They are larger than their parents, and also may be more muscular or more obese. The phenomenon started in North America and Scandinavia, to continue South towards the Mediterranean. Nowadays, in barely one generation, young Frenchmen are catching up in the matter of size with their American and Northern European homologues. The same tendency has been found in Japan.

This uncontested phenomenon was loosely explained by better food and/or a less cramped lifestyle. Food and lifestyle were conceived as limiting factors before the XXth Century. No proof of this exists. Did Spaniards or Italians eat less than Danes or Finns? The present book places increasing body size and lifespan in a new, proven context. Variation in lifespan and size are part of the adaptive response by human organisms under stress, caused by instability of the middle atmosphere in boreal regions. The early variant, a highly energized, big and shortlived human organism, rich in enthalpy, provides a logical explanation. These variants often replace the “modern” sexual way of gene transfer by “antique” methods of horizontal transmission and somatic meiosis. With a wink, today’s unisex fashions might be evoked.

There is at least one very antique, serious indication of a phenomenon of this order. This is the biblical passage in Genesis 6:1, 2 and 4, relating an apparently mythical fact which is not mythical at all. Only the wording may convey this impression; “And it came to pass, when men began to multiply on the face of the earth, and daughters were born unto them, that the sons of God saw the daughters of men that they were fair; and they took them wives of all which they chose. .... There were giants in the earth in those days; and also after that, when the sons of God came in unto the daughters of men, and they bare children to them, the same became mighty men which were of old, men of reknown.”

Indeed, the Bible refers to a recurrent phenomenon which inflamed human imagination to such an extent that it was transmitted over the generations in strong images by word-of-mouth. The phenomenon itself exists. It is linked to the dissipation of solar energy periodically producing atmospheric pollution, in the form of increasing concentrations of ozone and free radicals close to the surface of Earth in the troposphere and the hydrosphere (Chap. 5.2).

The adaptive response of organisms to these stressors is to replace their impaired vertical transfer of recombined genes (Chap. 3) by horizontal transfer. The share in a population of organisms with this particular genetic makeup depends upon the number of organisms having
integrated these genes in their genome by means of transposons. Organisms belonging to the early variant possess this trump card, so their proportion augments. The closer their biotope is to the polar circle where pollution is strong, the faster their share in the population increases.

Everything points to this adaptive phenomenon being universal. It links all organisms, from the primitive PROCARYOTAE such as viruses and bacteria to the more complex organisms in EUCHARYOTAE, including the sophisticated Vertebrates. The adaptive response described in the present book indeed is a universal tool to ceaselessly rearrange the organic potential to fit the ceaselessly changing environment. In brief, Life so had to struggle and evolve without respite to become implanted on and to spread over our planet.

As is so often the case, a formal element of proof rested in old research files, without having been seen for what it was at the time. It emerged from a biological and ecological study conducted thirty years ago by Rossignol (1968), on the yellow-fin Atlantic tunafish {Thunnus (Neothunnus) albacores (Bonnaterre)}. The yellow-fin is a fish of the warm intertropical seas. It occurs in important, dense shoals at the front between warm waters and upwellings of more saline, colder waters. This exists on the African coast at both sides of the Equator, in the Guinean Complex from Cap Blanc in Mauritania to Cap Frio in southern Angola. It also occurs along the tropical American coast from the Mexican Gulf and the Caribbean in the northern hemisphere to the Brazilian coasts in the southern hemisphere. It exists in the area of equatorial currents and counter-currents too (cf. Fig. 5-6).

Among the biometric data in the files were the number of branchial spines per fish. This is a fish-head character which stabilizes very early in life after the larval stage. So does the number of chloroplasts in the stomatal guard cells in plant seedling leaves. With thirty years of hindsight, it is astonishing to note that both markers are comparable in that they both refer to central tools for energy processing in cells. Chloroplasts in green plants indeed provide photosynthetic sugars and expulse oxygen gas. Branchiae provide respiratory oxygen as a complement to the glucose from nutrients, so allowing metabolic breakdown of glucose, chemical fixation of energy in ATP and expulsion of respiratory carbon dioxide gas.

Now in both plant and fish, groups of specimens occur with a mean weight either 17% superior or 15% inferior to the mean of the larger number. In the case of the yellow-fin tunafish, these deviations occur in classes of young fishes of one and two years old, living in waters enriched in nutritious salts and plankton, because of upwellings of cold water. This water is a mixture between the layer of maximum salinity and the Intermediary Antarctic or Arctic waters. At certain moments and in certain places, movements of wide amplitude occur, causing exchanges with superficial waters. This causes pollution of these waters in close analogy to the atmospheric circumpolar phenomena mentioned. Epidemics result, causing massive die-back of plankton and hence of planktrophic fish or mammals (e.g. see Chap. 1).

Now expanding potato cells, tunafish and human bodies have something in common. They all occur under major stress causing hyperoxidation of cell membranes, reported in the beginning of this chapter. In potato cells in vitro, this stress is caused by stripping the cellulose membrane off tissues before culturing. In the case of humans and tunafish, the stress is due to the entry into the lower atmosphere in the polar belt of “exotic air” originating from the poisonous stratosphere. We saw that the adaptive response consists in replacing the blocked Mendelian processes by horizontal transfer mechanisms.

This adaptive response in all three cases is the mechanism of the last chance, before apoptosis intervenes (see Sect. 6.2.1). Perhaps the same mechanism is unleashed automatically at
clinical death of a whole organism, a moment defined with respect to the fuzzy biological present (Fig. 6-1). At the moment of death, the enzyme integrase would cease to function, so that no more genetic splicing can occur. DNA would fall apart in bits and pieces. This process should be considered when studying the origin of prions (Chap. 1) found in the mad cow’s disease, scrapie in sheep and the disease of Kreutzfeldt-Jacob in humans. An aspect to be pondered is active apoptosis by DNase, cutting up harmful DNA fragments, versus passive apoptosis by ceasing of integrase activity, perhaps leaving a precise kind of harmful pieces intact, such as ptomaines, which do not matter for a dead organism itself, but explain part of its toxicity and of the traditional taboos surrounding the handling of “unclean” dead bodies.

The whole present-day situation trembles under the serious dilemmas linked to a new balance becoming established between the titanic forces of solar behaviour and biotic organization. It is vain for us to look for total human responsibility, with our well-known mixture of arrogant presumption concerning our powers, and bad conscience about our acts damaging Life. No human action is so powerful that it can cause the heating-up of a planet, the disappearance of huge sheets of atmospheric ozone between atmospheric compartments, the spectacular rises of atmospheric CO₂ rates (e.g. see Lovelock 1979, 1988), or the increasing share of giants in populations of Homo sapiens or other organisms. The sun can and does this, all alone, with a seemingly small shrug of its radiant shoulders.

Of course these facts are no charter for human licentiousness in handling our beautiful planet. All who impress upon people not to contribute to the stresses already building are good counsel. Indeed it is important to continue organizing international cooperation with the aim to deflect the impacts of stress, to protect Life and to counteract if and when possible the forces of chaos now building up. We trust that the present book contributes to the arsenal of knowledge needed to convert at least some of our dilemmas into manageable problems.

Let us always join in the struggle of life, not struggle against it.
Glossary

Many of the definitions given in the present glossary are inspired by other authors, e.g. Crabbé (1987), Hallé & al. 1978, Suzuki & al. (1989)
The form in which they appear below, however, is the sole responsibility of the present authors.

**Acrotony**
Branching mode characterized by the preferential appearance of shoots at the upper extremity of the axis and, by extension, its physiological control mechanism.

**Annealing**
Rebuilding a double-stranded nucleic acid from single strands.

**Antibody**
Protein molecule made by the immune system to recognize a particular foreign antigen and to attach itself to it for its destruction.

**Apical dominance**
Control by an apical meristem over all meristems on axes generated by the lateral meristems (part of the leaf-plus) initiated earlier by the same apical meristem.

**Array**
Architectural complex of organic structures, organised by regular, more or less strict inter-actions among these structures, their scale being specified by an adjective ("branched array") or prefix ("multi-array").

**Axillary**
Placed in leaf axil.

**Basalony**
Branching mode characterized by the preferential appearance of shoots at the base of the axis and, by extension. its physiological control mechanism.

**Biodiversity**
Epiphenomenon of biocomplexity expressed as diversity of biological instruction carriers at one moment in biological time and at one biological scale level.

**Biological clasp**
Structure, at the end of a limited carrier sequence of biological instruction, which actively ensures the coded link to another such sequence.

**Biological instruction**
Set of instructions, either genetic or derived from the genetic, expressed by a material structure and driving the development of organic systems at one biological scale level.

**Biological instruction carrier**
Material body containing biological instruction.

**Biome**
Ecosystem uniting all interacting ecosystems at all levels in one large biogeographic region.

**Biosphere**
Ecosystem uniting all interacting ecosystems at all levels on a planet.

**Cambium**
Thin, hollow, secondary meristematic cylinder inside an axis, ensuring its diameter increment and renewal of transport tissue by forming wood inside and bark outside.

**Clasp (biological)**
Often minimal biological structure, at whatever system level between the molecular and the whole organism, which ends a development sequence and initiates another sequence of a similar or different nature.

**Codon**
Section of DNA, 3 nucleotide pairs long, coding for one single amino-acid.

**Crossing-over**
Exchange of corresponding chromosome parts between homologues by breakage and reunion.

**Diapause**
State of temporary, self-induced cessation of growth which is lifted when the interior stresses causing it are discontinued.

**Diaspore (= propagule)**
Any part of an organism capable to reproduce that organism when moved to another place.

**DNA (deoxyribonucleic acid)**
Double chain of linked nucleotides with deoxyribose as sugars, basic substance by which genes are built.

**DNA-polymerase**
Any of several enzymes able to synthesise new DNA strands using a DNA-template.

**DNase**
Enzyme breaking down DNA strands into their nucleotides.

**Dormancy**
State of temporary, self-maintained cessation of growth in a meristem, needing a specific exterior incentive, i.e. not a mere return to previous circumstances, to be lifted.
Eco-complex  Ecosystem at the landscape level, composed by various kinds of eco-mosaics
Ecological moment  Time passed since the initiation of an ecosystem.
Eco-mosaic  Ecosystem living on sites within one similarity class, composed by various kinds of interacting eco-units in various development phases.
Eco-unit  One ecosystem, developing on one surface cleared by one impact, from one specific moment on, and by one development process.
Equilibrium  The state of an eco-mosaic the eco-unit composition of which is in balance with the abiotic environmental impacts (spelled with e, not q).
Endonuclease  Enzyme cleaving phosphodiester bonds within a polynucleotide chain.
Enthalpy  A measure of the available energy in a closed thermodynamic system, considered as a measure of the state of certitude, order, organization or structure of that system.
Entropy  A measure of the unavailable energy in a closed thermodynamic system, considered as a measure of the state of incertitude, disorder, desorganization or breakdown of that system.
Exonuclease  Enzyme cleaving nucleotides one at a time from the end of a polynucleotide chain.
Flexibility (biological)  The capacity to organise a life cycle in part or completely by very fuzzy development sequences inside a rather broad general "pathway" of adaptive sequences (also called plasticity, Vester 1997)
Genome  The complex of genetic material in an organism
Grafting  The transfer of information-bearing parts in whole multicellular organisms, by extension in all living systems.
Heteroduplex  DNA double helix formed by annealing single strands from different sources.
Interaction  Mutual or reciprocal action between two or more elements, here biological subsystems.
Leaf-minus  Meristematic zone, carrying genetic information minus an axillating energizing leaf, adventitious (extra-axillary), or its axillating leaf having been shed.
Leaf-plus  Complex of a leaf as energy provider and functional trigger plus the axillary meristematic zone of that leaf carrying genetic information and regulated by its leaf.
Meiosis  Reductive nuclear division yielding daughter nuclei each containing half the number of chromosomes of the parent nucleus, followed by sexual cell processes.
Mendel's Laws  Basic laws of genetics formulated by Gregor J. Mendel (1822-1884), the first law stating the segregation of alleles, the second one the independent assortment of genes.
Minimum eco-unit  The eco-unit which contains the smallest central zone which can still allow and select germination and hatching processes.
Mitosis  Reproductive nuclear division yielding daughter nuclei each containing the same number of chromosomes of the parent nucleus, followed by somatic cell division.
Nuclease  Any of several enzymes breaking down DNA by breaking its phosphodiester bonds.
Nucleotide  Basic molecular building block of nucleic acids, composed of a nitrogen base, a sugar and a phosphate group.
Phosphodiester bond  Bonds holding together the sugar-phosphate backbone of DNA by linking a sugar group to a phosphate group.
Pioneer plant (tree)  Species that is adapted to grow in empty biotopes, often linked to early development phases of eco-mosaics (the term "succession" is banned from this book).
Plasmid  Autonomously replicating extrachromosomal DNA molecule.
Plastochrone (real)  Temporal interval separating the initiation of two successive leaf primordia in an apical stem meristem.
Plastochrone (apparent)  Value in measured time units of a temporal interval separating the arrival at a certain development stage (to be defined precisely) of two successive primordia.
Pre-equilibrium  The state of an eco-mosaic the eco-unit composition of which is not yet balanced out by the abiotic environmental impacts.
Proleptic (meristem activity)  Meristematic activity starting after a period of meristem-atric rest, i.e. after a period in which no cells divide but cell differentiation may occur.
Promoter  Regulatory region close to the 5' end of a gene, acting as binding site for RNA-polymerase
Propagule  see Diaspore
Prophase  Early phase in nuclear division, when chromosomes condense and become visible.
Quiescence  State of temporary cessation of growth in a meristem imposed by exterior incentives (stress), which is lifted when the stress stops.
Regression coefficient  Slope of the straight line correlating two variables most closely.
Restriction enzyme  Restriction endonuclease.
**Reverse transcriptase**  Retroviral DNA-polymerase synthesising DNA on a viral RNA template.

**RNA (ribonucleic acid)**  Large, linear, single-stranded nucleic acid variously composed of ribonucleotide sub-units, transcribed from chromosomal DNA templates, found in cells as transfer RNA, ribosomal RNA or messenger RNA.

**RNA-polymerase**  Enzyme catalysing synthesis of a RNA-strand from a DNA-template.

**Species**  Cluster (population) of clusters (organisms) of genetic information, persisting as such over a longer or shorter timespan, by various means, its material expression recognizable by the human senses, and which lives in a direct environment feeding it.

**Syleptic (meristem activity)**  Meristematic activity starting after meristem formation without any intermediate rest, i.e. without interruption in cell division and differentiation.

**Synopsis**  Close pairing of homologues in meiosis to form a bivalent.

**Telomere**  Tip, literally “end-part”, of a chromosome.

**Temperament**  The package of reactions of a plant towards its environmental stimuli.

**Template**  Molecular “mold” for the structure or sequence of another molecule (also see RNA).

**Transduction**  (genetic meaning) Horizontal transfer of genes, carried by viruses, among bacteria.

**Transfer (of function)**  Transfer of a biological function within a living system from one subsystem or organization level to another, ensuring a minimal cost-benefit ratio in terms of matter, energy and information.

**Transgenic organism**  Organism in which genes from other species were introduced by genetical engineering, in plants often by the use of carrier *Agrobacterium tumefaciens*.

**Transposon**  Mobile piece of DNA, flanked by terminal repeat sequences and usually carrying genes coding for transposition functions, larger bacterial transposons also have genes coding for antibiotic resistance.

**Upwelling**  Oceanic vertical compensation movement in stratified water masses of unequal density, i.e. superficial, warm, little saline, light waters separated by a highly saline layer from deeper, heavier and colder waters, caused by seasonal persistent trade or monsoon winds chasing warm surface waters far from the coast, creating a “vacuum” compensated by upwelling of cold, deep or deeper waters boosting production such as planktonic blooms.
References

- Anonymous, 1996c. Une bonne mutation. L’Express, August 8: 36
- Benzine-Tiziroute, S., 1989. Apports de la morphologie de la cytogénétique et de la caryologie à la compréhension de la variation somaclonal chez la pomme de terre diploïde BF.15. Orsay, Dr. thesis, Université de Paris-Sud, 247 p


• Buïjs, R.M., 1998. De hypothalamus: balans van het Leven [in Dutch: “The hypothalamus, balance of Life”]. Amsterdam, inaugural address at the Faculty of Biology of the University of Amsterdam.


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• D'Arcy Thompson, 1917. On growth and form. [see Bonner 1961].

De .... for names starting with De see under the other name (e.g. De Granville under G)
• Dickerson, R.E., 1963. The DNA helix and how it is read. Scientific American 249:100-104
• Hooff, A. van den & Hooff, P. van den, 1995 (ed.). A propos of Science: quotations and aphorisms. Doetinchem (NL), AmstelScience. ISBN 90-9007630-1
• Jones, P.D., 1990. Le climat des mille dernières années. La Recherche 21,219:304-312
• Kaandorp, J., 1992. Modelling growth forms of biological objects using fractals. Doctor’s Thesis Univ. of Amsterdam, publ. by the author

216
- Nesme-Ribes, E., 1990. [Comments added to an article by Foukal, P.J. Revue Pour la Science, 150:32-41


Prigogine, I. 1980. From being to becoming - time and complexity in the physical sciences. San Francisco, W.H. Freeman & Company


Remmers, G., 1997. Towards a theoretical understanding of the generation of diversity in the countryside. Chania (Crette, Greece), XVII Congr. Eur.Soc.f.Rural Sociology, Paper, manuscript to be publ., 18 pp. (E-mail author gaston1@dds.nl)


Scarrone, F., 1969. Recherches sur les rythmes de croissance du Manguier et de quelques végétaux ligneux malagasy. Clermont-Ferrand University, Dr. thesis


*Van*, *Van der* (cf.) ....(see under the first letter of the second part of the name, e.g. *Van Maanen under M; Van der Wef under W*)


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Yu, C.E. & al.. 1996 (ex La Recherche 1996, Vieilles prématurée pour cause de gêne.) Science 272: 258
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- Prélude à la Nuit -

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Embryons d’Univers,
Volute de feu surgies
des entrailles de la Terre, ...
Quel message de l’Esprit,
Quelle menace de Lucifer,
- en arabesques dorées,
en doux reflets grisants -
viennent un instant troubler
la quiétude des gisants?...

***

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***

» C’est tout cela, je crois, la Vie!...

***

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Romain Marti
Mars 1997