Micro-evolution according to the Poisson distribution[1]

Summary
You expect the genetic changes in a population are described as random or non random and thus selective, but this appears not to be the case. Random genetic changes are nearly always described as genetic drift with changes in the heterozygosis, but the gene frequencies and so the random genetic changes are not to be reduced from the heterozygosis. So it is tried to develop a uniform theory with the random expected genetic change as the neutral theory and the zero hypothesis for the selection. For the procreation and gene transfer from the individual in a large, relative unlimited population the Poisson distribution is the obvious method for calculating the random expected genetic change. Yet also in small populations this distribution appears accurate and well applicable. The Poisson distribution appears very flexible in the sense that the parameters determining the intensity can describe well the small populations and the population dynamics (change in size and allele selection). By means of the parameters you can also find indications for the complicated effect of the selection in the procreation on the allele transfer. Even with a very simple application of this theory there are immediate indications that the selection by people has been suddenly stopped with the entrance of the modern society. The relevancy in the appearance of the mutations for the dynamics of the organism and the species is checked. By making simple distributions the transfer of the alleles through the generations is followed in coherence with the random variation in the effective procreation. These distributions are superposed over some generations. This superposition of the distributions is possible by working systematically with the Poisson distribution, but it is very laborious. It appears than that the accumulation of the distributions over many generations can easily be calculated, only for the exponential part of the Poisson distribution The result of it the P0, the extinction of the neutral alleles is essential for the random theory. The calculations of the extinction with the exponential recurrence formula is also possible with population specific parameters determining the cumulated exponential intensity are interesting. They give information over the random neutral path and the non random selection. This is showed by describing the decay of the alleles over many generation in tables for populations, large and small, increasing and decreasing in size, with and without selection, with and without inbreeding. The random path of larger quantities of the alleles and thus allele frequencies is also described. Important is however that these simple distributions do describe primarily the decay of the absolute quantities, the "quanta", of the offspring and the alleles. In literature investigation later I found that these extinction is also described plain by Motoo Kimura, but he reduced them not in this basic way. There is some evidence that the conclusion of Kimura's and others ware: the calculations of the extinctions are not relevant, because they are not applicable in a limited population. Nowise this, the extinction is essential in a logic consistent theory.

Points of attention are:
The neutral theory as the zero hypothesis for the genetic selection concerns exclusively the direct changes in numbers or frequencies of alleles and/or descendants of individuals as described here. Changes in the heterozygosis are an indirect basis for the neutral theory.
The direct neutral change or allele extinction is also described in the limited and small population and gives here besides the genetic drift extra information over the genetic changes.
Selection always is the result of non random differences in the parities, the offspring of the individuals.

Preface

After my study medicine and some years later the training in the epidemiology in tuberculosis and similar things until 1976 I did lose all the tangible connections with the organisations of science and research. Moreover I do not anymore practice the profession of a doctor already since my 40th. Now I am 61 and I looked after my children for a long time, while my wife was working. My curiosity and interest in different fields and the increasing quantity of spare time results in a number of hobby-studies. Magazines, books and later on the internet did provide me afterwards plentiful in information The study of the topic evolutionary biology was one of my most favourites. No official training or studies were followed in this. My study activities consist of collection here and there interesting data and than thinking about it endless with my super critical dialectical customs, or you may can call it also addiction. Anything you read than is denied and that negation follows a laborious constructed meaning, but that meaning again is denied, etc. So the negation of the negation in order to find at last the all synthesis, the logic, the unity, the truth in which anything is participating. This in principle is the rational method of Spinoza, which was described later on by Hegel. By these negations points of view mostly are not simply accepted or rejected, because often a synthesis is possible so than the and/or is the best solution. Study in this way is an endless ruminating, destructing and constructing of meanings and theories. Using these radical dialectics you do not need a teacher, but it can result in a stomach ulcer, for with this method of negation you are not a nice teacher for yourself. The advantage of these primary negations is that it makes you independent of other people like teachers and authors. Their information it is not followed and taken over, but negated. So I tried to be no man's follower and an open minded searcher to the uniform principles.

Publication by internet is for me the most convenient way to share my ideas about the evolution with other people. Perhaps it can help starting discussions and deepening studies to the stirring, interesting and in many aspects so important topics of

the evolution. Furthermore I do hope I also will be able to publish here more ideas about the macro-evolution and the religious-philosophical aspects of the evolution as a logical integral.

Some principles of the evolution theory

The evolution of the organisms and their species still is somewhat disputed. It is nevertheless without doubt that anything we can observe is changing and that nothing is able to remain the same forever. If the changes in this are irreversible there always is a development or evolution. So the a-priori statement is plausible: **the changeable and evolutionary characteristic of the nature, living and not living, is nothing more than its very existence in the time or properly the space-time.** There are indeed observations that confirm the evolution. Already in 1859 Charles Darwin did describe and prove that the similarities and differences between the yet living species and the died out fossils indicate evolution, as well as the frame of the embryo's. Although there now is much more knowledge and we can fill now probably a bookshelf of more than 1 km with books and magazines with data relevant for the evolution theory about genetics, biochemistry, and palaeontology that ware unknown by Darwin, his book "the origin of the species" yet is in our days an important source which often is cited in manuals. So this does indicate the scientific and nature-philosophical grandiosity of Darwin. On the other hand this also indicates that we after 150 years do not know much more about the fundaments of the evolution theory than Darwin did, in spite of all the details of the genetics, the molecular properties of the DNA, the cell structure, the physiology etc. We do have now much more evidence from the palaeontology and by the molecular biology the genetic similarities and differences can be studied by DNA research very good now, so that the descent of the species, their common ancestors and thus their evolution can be followed directly. There now is from the different disciplines a lot of evidence for the existence of the evolution in the way it has been described in essential by Darwin. So now we do know very well that new species do arise, but how and why the genetic changes arise and how different species do arise, we do not know much more than Darwin did: hazard and selection by the survival of the fittest. Many researchers in our time are thinking that is all and there is nothing more essential to be discovered. Probably the scientists of the 19th century had a more optimistic view on this in the sense of: evolution is a natural process and thus a regular process and the natural laws that underlie it will soon also been discovered. The very important discovery of Georg Mendel however, the discrete transfer of genetic variations in pairs with their segregation in the gametes in 1866, did not make the theory of evolution more clear initially, on the contrary. When this discovery was at last accepted in 1900 within the official biological science as an important tenet, after the research of Hugo de Vries, Carl Correns and Erich von Tschermak, it was difficult to unify this law of Mendel with the

evolution theory. The difficulty was that the material steady genes could not change apropos of nothing or as a direct consequence of the circumstances in the life surroundings of the plants and animals, like Darwin thought as did Lamarck. Nevertheless science has accepted now both the laws of Mendel and the evolution theory of Darwin. The idea now is that the discrete genes change by accidental mutations. These mutated genes are passed through the generations as genetic variations or alleles. These changed genes can increase or decrease within the populations by hazard or by selection because they may be advantageous or unfavourable in the circumstances of the living individual. In the accepted view the accidental mutations are the primary and the only possible causes for changes in the genetic DNA of the organisms. It now is again disputed that all the changes and mutations of the genes do arise in events that are hazardous, so not logical lied with biological functions of living individuals. For the micro-evolution however the starting point is the observed variations (alleles) on the loci of the genes and their changes in number and disappearances within populations.

Micro-evolutionary principles

Within a population is a knock-out competition between the different allelic variations on the gene loci. By two factors all the gene variations or alleles are not transferred through the generations of descents and so the numbers and frequencies of the alleles will increase or decrease in the following generations. These 2 factors are:

1st the distribution of the reproduction. The individual organisms of the parent generation F0 do have different numbers of effective descendents, that reach adultness and are able to reproduce themselves.

2nd The endowment of the alleles to the effective descendants.

The organisms of the F0 that have been able to reproduce effectively in this way, will pass at most the same, but on the average fewer different alleles to their total offspring than they do have themselves. Even if all parents should have an equal number of offspring they will pass different parts of their genetic variations to the next generations or otherwise not. There are many mutations and often an individual has a number of seldom mutations. Also are many mutations seldom and are they in small frequencies in large populations, or a total species. However the absolute numbers of seldom mutations are large in large populations, of in the whole species. One percent of 10^8 yet ever is 10^6. So it is obvious a-priori that seldom mutations practically never will vanish in large populations, unless they are ultimate seldom and occur in immeasurable small frequencies, or are very unfavourable. In this it also is obvious that alleles will be practically never be fixed in large homogeneous populations. In small populations seldom alleles do have small absolute numbers and by this they can vanish or increase in number and sometimes be fixed in small populations. One percent of 100 yet only is 1. That this is a-priori at

random to be expected may appear from the following:
Pose a bag with 100 marbles. They have a number of different colours, some colours are singular, some occur on 2 marbles, some on 3 or more. The marbles all are drawn under replace. The results of the total turn of 100 drawings under replace are recorded and a new bag is composed, so that the colours of the marbles are distributed following these results. It than appears that the composition has been changed: Some colours have been disappeared and some colours that were singular in the first bag now are present in twofold or more. At the second turn, starting from the results of the first bag again the composition changes evidently. If these drawing turns are ever repeated more and more colours will disappear (extinction) and ultimately after a big number of turns only one colour will remain in the bag (fixation). The same experiment can be executed as well with the help of a computer in a bag with 10^8 marbles, in which some colours are present on 10^6 marbles or on two or more times 10^6. It will be evident that in this bag the composition hardly will change in the drawing turns; 10^6 may become 9.10^5, but not easy 2.10^6 and practical never 0. So the frequencies will hardly change here and can at most fluctuate somewhat in the turns. Yet is the change, that a singular allele (marble) is not drawn and will disappear in a population (bag) of 10^8, nearly equal to that in a population (bag) of 100 and so the change that all the 10^6 alleles will disappear, is practically zero. This is in principle the model of the random or neutral genetic change in a population. Essential in this however is that the non random genetic change, the selection, comes upon to this as a parameter of the chance distributions. As well in the case of selection are these drawing turns valid in the model, but the drawings than are not 'honest'. In the selection for instance the red marbles will have a smaller chance to be drawn and the green ones a larger chance than at random, because the red 'marbles' are unfavourable alleles and the green ones are favourable for the survival and the reproduction of the individuals. This is the essence of the micro-evolution that is elaborated here further.

Genetic Drift

This process by which the alleles will vanish or settle totally in a close population with limited size is called in literature genetic drift. So the allele frequencies always become 0 (in extinction) or 1 (in fixation) and after a longer period this also occurs in larger populations. The heterozygosis and thus the genetic variation within a population is getting smaller and smaller by this genetic drift. By the genetic drift arise ultimately a population that is genetic total identical, which is of course also total homozygote if there were no mutations. Theoretically the population becomes even identical exclusive by descent, after it was already a long time homozygote and identical in general occurring alleles, but in practice this event is not likely because the population will dye out before. From the binomial distribution **Sewell Wright** deduced there is a

decrease in the **heterozygosis** [2] by the drift with the average factor (1- 1/2n) per generation. In this is n the size of the population and so 2n the number of alleles on the loci in a diploid population with sexual procreation. This decrease is to be calculated with the formula **H g +1 = H g [1 – 1/2n]**, in which Hg is the heterozygosis in generation g. This means for instance that in a population with 50 animals participating in the procreation is a decrease of 1% per generation. So this is an important problem for many threatened species. This decrease does not mean however that such a population will be total homozygote and genetic identical by descent already after 100 generations. It yet is an exponential decrease; in general the heterozygosis changes by a factor e^-1 = 0,3679 after 2n generations, so the decrease than is 63,2%. After a x 2n generations the heterozygosis changes with a factor e^-a. In this is e the logarithmic base, so e = 2,7183.. This decrease in the heterozygosis at the genetic drift is based on random inbreeding. The drift to extinction or fixation of the alleles can be intuitively a-priori approached in two ways:

1st By the inevitable or random inbreeding in a close population arises homozygosis, so that the heterozygosis decreases, being its complement. This process implicates imperatively the vanishing of some alleles and the increase of their alternatives on the loci until it remains only one, but now is it not easy to guess how this will happen.

2nd By random sampling there ever is fluctuation of the numbers of the alleles, but if the decrease goes incidentally to zero there is no way of return. This makes the curve of the chances for the smaller numbers asymmetrical. This vanishing of some alleles means the increase of their alternatives on the loci and so also the increase of the homozygosis and decrease of heterozygosis. This happens in a population with limited size as well as in the unlimited population. This vanishing or extinction of the alleles however is limited in a pool with a limited number of alleles, because not all the alleles can disappear here. There must remain in the limited pool one of all the possible variations and in the unlimited pool are infinite variations and so there will remain nothing. If this happens there is fixation in the limited pool, with a fixation chance 1/2n.

Which allele will be fixed by the drift and which will vanish is of course not to be predicted. You can pose the allele with the largest frequency on the datum date at start should have the greatest probability. The differences however in the probabilities often are very small, because there are many events with random fluctuations between the datum date and the real fixation. In a population of some size it will last a very long time till an allele is fixed, but the increases and decreases of the allele frequencies can go fast temporarily. Conditions for the genetic drift as it is described by the formula: H g +1 = H g [1 – 1/2n] are:

1st The close population without genetic exchange. 2nd The constant size of the population. 3rd Random breeding, so no more or no fewer inbreeding than at random. 4th There is no selection. 5th There arise no new mutations after the datum date. 6th

[2] This is to be described more exactly as the total heterozygosis of all the allelic variations on the gene loci. This makes it impossible to derive the allele frequencies from this parameter. See also table 11 and 12.

There is no mating between the generations. 7th Self-fertilisation is possible, because the individuals are fertile in both genders.

Criticism on this model, Disadvantages

1st The great drawback of this formula is: it describes how the **heterozygosis** decreases in a close population, but unfortunately **not** how do **allele frequencies** change in populations, as it is sometimes suggested indeed. The in- and decreases of the frequencies and numbers of the alleles also is not to be derived from this formula or from this model. Insight in the random and non random changes however is essential for insight into the micro evolution.

2nd This formula $H_{g+1} = H_g [1 - 1/2n]$ only is valid in very restricted situations, because of the above called conditions. Further on it is, I think, disputed if this formula and model fulfil if the population has more than 2 allelic variations on the locus. If for instance 4 different alleles a; b; c and d are at start on the locus, the extinction of any of these alleles should be described as a separate process. Yet the vanishing of the first allele is not lied with the fixation of the last allele. Evident further is that a number stochastic processes independent of each other can not be described as one process with one formula. So this should mean that another condition for the formula is: *there should be only two allelic variations for the locus in the population.*

3rd The formula appears than also not applicable in many real situations. It can not describe for instance how a new arisen and thus very seldom mutation often disappears very fast from a large population. Also the fast genetic changes that arise in populations shortly after they got isolated can not be explained well by this formula. These fast genetic changes arise for instance in animals that got isolated in small populations after people did disturb the ecology of their old life area. If so a mother population splits into a number of deems there will be initially in these deems alleles singular, in twofold, in threefold etc and by the small numbers of these alleles many of them will disappear in a little generations. Also the stocks in descent of the domestic animals are models of these very isolated populations, that underwent impressive genetic changes in the course of a restricted number of generations. The different races of the domestic animals may origin from source populations of minimal 50 to about 1000 of animals. According to the formula $H_{g+1} = H_g [1 - 1/2n]$ the heterozygosis should decrease in these effective populations with ca 1% to ca 0,5‰, per generation, while thus the observation indicates us that the changes in the genes in these populations must have taken place much faster.

The large advantage of the formula however is that it is simple and gives good and easy insight in the important aspects of

the genetic changes: the decrease in the heterozygosis and the increase of the homozygosis. This easy calculation of the heterozygosis in this model, means thus a reduction, which restricts the flexibility of this model in the different situations. By this it only is possible to get more specific information with very complicated further calculations, that than again do not give any more at all the simple intuitive insight in the biologic events. So it could be useful and is any way harmless trying to approach this matter in another way with models primary describing what will happen in general with the allelic variations in a close, limited population and in the theoretical unlimited population of Hardy and Weinberg.

In search of another model

In this model is started from the generation F1, that is born, or arises and receives at random alleles from the former generation F0 on a distinct locus in the genome. The size of the population in generation F0, F1, F2, etc is constant on n examples. Thus are 2n alleles on the diploid loci, so that the chance that a distinct allele of F0 comes into the zygote of F1 is 1/2n and the chance that this allele does not come into the zygote is 1-1/2n. In this way for all the n zygotes in F1 are 'drawn' 2n alleles for the locus from the generation F0 alleles. Standard should be drawn in this way all the alleles or gametes of F0 and so should be passed the total set of alleles from generation F0 to generation F1. This standard event however is in reality as likely as a long street in a poker game with a lot (2n) of different cards. Always are drawn a number of alleles two times or more and an accordingly number are not drawn. We can follow with the aid of a game with marbles or a computer module of it what are exactly the fortunes of the genes with their potential and real allelic varieties in a population. We start with a bag of 2n marbles, that all have a singular number 1; 2; 3; …2n. These numbers represent the separate, in generation F0 singular alleles, or potential variations of the genes. Further on the marbles do have colours so that some marbles have the same colour. The colours indicate the real existent gene variations. 2n marbles are drawn under replace and the drawings are recorded. After a turn of 2n drawings the contents of the bag is replaced by the results of the 2n drawings as recorded. So after the first turn of drawings the first bag, F0, is replaced by the second, F1, and so on. It than appears from the recordings that already in the first turn a lot the of numbers on the marbles is not drawn and that many numbers are drawn 2x and some 3x or more. Also the colours did change in number in this way and some very seldom colours were not drawn. At the second turn from bag F1 is formed bag F2. Now also many numbers are not drawn, but less than at the turn from bag F0, because in F1 not all the numbers are singular. If these turns are ever repeated the singular numbers the singular numbers of F0 will vanish ever more giving rise to increase of the frequencies of the remaining numbers and colours. The numbers on the marbles and their colours are fluctuating in the further turns The numbers and

colours will decrease some turns and than again increase, but if they go to zero no return is possible and it vanishes. By these vanishing the remaining numbers and colours are ever increasing and by this the vanishing becomes ever more seldom in the later turns. After a large number of turns will remain only one colour and later also only one number. If the bag contains a small number (2n) of marbles this process of fixation goes very fast and if there are many marbles the fixation is slow and is only possible after a huge number of turns. In the beginning however will the singular numbers on the marble vanish in the large bag as fast as in the small one. If the remaining numbers on the marbles become somewhat larger to about 50 or 100 than they will only very seldom disappear. The colours will be present in the large bags already at the first turn mostly in numbers of more than 100, so that they will scarcely disappear from the beginning.

This model of the marble game is a simplification, a reduced principle, that does not describe the total biological reality. This hazard game model than also does not intend to describe the total biological reality of the genetic changes, but only the hazardous events in this. That is why it must have indeed intrinsically restrictions: The possible gene changes caused by the biological functions are to be excluded in the model. Unfortunately the model must have also extrinsically restrictions: For reasons of clarity and survey-ability not all possible extrinsic events can be included in a model. It is convenient to describe a model within a standard situation with exclusion, or freezing of all possible events. Later on some events may be included into the model as a new parameter. These restrictions are mostly the same as in the general model of the genetic drift, as they are:
1st The close population without genetic exchange. 2nd The constant size of the population. 3rd Random breeding, so no more or fewer inbreeding than at random. 4th There is no selection. 5th No new mutations do arise after the datum date. 6th There is no mating between the generations. 7th Self-fertilisation is possible, because the individuals are fertile in both genders.
The conditions random mating and no selection are largely or totally intrinsically, as they are causal lied with the biological functions. The events that may open the population and will change its size can be both extrinsic and intrinsic. The arise of mutations is seen mostly as an extrinsic factor, but this may be disputed. The mating between the generations is intrinsic. The fertility in the genders and their participation in the procreation is a biological or intrinsic factor.

Most of these restrictions are described as a parameter further on here. This is not the case in the mating only within the same generation. The possibilities of allele transfer in genealogic studies indicate however the influence of mating between the generations on the allele transfer may be small, because this mating does not cause inbreeding. It can cause however

some fluctuation of the effective size of the population while the real size remains constant. Making models and calculations with this mating is difficult. The measure of the mating between the generations depends from the length of generation time in relation to the period of fertility and reproduction of the individuals. Mice can mate with much more generations than people. The random possibility of self-fertilisation itself in somewhat larger populations is very small and thus unimportant. If the individuals of the species have separated genders and can be fertile in only one gender this also is of no influence if these both genders participate equally in the procreation. The problem however is that in practical live the genders do not equal participate. The observations learn us that more individuals of the gender that 'invests' the most in the next generation participate in the procreation than those of the minor investing gender, thus mostly the masculine. The phenomenon of biological functions by which is caused this unequal participation of the genders in the procreation is called sexual selection. It is possible and often practised to correct for this intrinsic factor in calculating the effective population size for these cases, but it is disputed if it always is useful to correct in the biological data to get the random situation.

The potential most important restriction of this model however is that *at 8th the transfer of the alleles from generation Fn to F(n+1) must be one uniform event*. In reality it yet is a composed event. So this restriction is very important, but it is not generally acknowledged in the literature I guess. As pointed out before there is in fact a drawing or distribution of:
1st The reproduction. The individual organisms of the parent generation F0 draw different numbers of effective descendents, that reach adultness and are able to reproduce themselves.
2nd The effective descendants can draw different alleles from their parents and further ancestors.
It is pointed out here **further on**[3] that it is possible and in many situations necessary to make a model with specification of these two drawing events.

Further more it is possible to extend this model by putting more data into it in order to get more information about the changes of the genes in the course of time. So the marbles can have besides their number and colour also other marks by means of which can be reed for instance which individual is carrier of the allele and which was the carrier in the former generation. With data like these the genealogy within the population can be followed and so you can have much more information about random genotypic distributions, the measure of homozygosis and especially the important random

[3] See Table 9

changing linking as there is between the allele on a distinct locus and many other loci of the genes of the ancestors. Many interesting computer models can be made for the study of these problems. Primary however is this simple reduced model. But besides of the models it is necessary to describe in algebraic terms what happens at random to the genes and what happens in essential in the biological reality:

Deduction why the Poisson-exponential distribution is appropriate.

In a population are n individuals, so 2n diploid alleles are in the model and they are seen as singular, so that they represent the potential variations on the loci of the genes in the total population. If the size of the population remains constant, the chance that one distinct allele is drawn in one fertilization, so in one descendant, or is transferred from F0 to F1, is 1/2n and its complement, the chance that this allele is not drawn thus is 1-1/2n. This means that this distinct allele is not drawn on the average in 2n draws, so in one generation (1-1/2n)^2n and so it is than not transferred in one generation. It appears now that this relation (1-1/2n)^2n converges fast to 1/e, for if n→∞ becomes (1 - 1/2n)^2n = 1/e, in this is e the base of the natural logarithm, so e=2,7183.. The conversion goes fast, if the size of the population n=10, it is (19/20)^20=0,3585 so that the 'base' than already is 2,7895, only 2,6% more than e. So is 1/e, or e^-1 the proportion of the singular alleles that is not transferred from generation F0 to F1. The alleles however only can be transferred in this way at random in a population with individuals that are fertile in the both genders. In a population with ½ n individual of the masculine and ½ n of the feminine gender the alleles are 'drawn' or transferred separately for the genders. In both of the genders n alleles are present and are drawn. At one fertilization one allele is drawn in both of the genders. In this are the proportions 1/n and 1-1/n transferred respectively not transferred and so the proportion not transferred is nearly 1/e in somewhat larger populations. So is indeed this principle also valid in separated genders, if the participation in the reproduction is equal. In somewhat larger populations (n>ca 10) singular alleles are not transferred and will vanish in the proportion or in the rate 1/e=0,3679. In the smallest possible population, if n=1, so in self fertilizing, this rate is (1- ½)^2=0,25. Further it is obvious in a population with 2n alleles that if the different alleles are not singular but are present in absolute numbers 1; 2; 3; or q they are transferred or not transferred to one descendant in proportions q/2n and 1- q/2n respectively. In 2n drawings, so in one generation they are not transferred in the proportion (1- q/2n)^2n. This is if n→∞ (1/e)^q=e^-q. It is evident to that if the effective size of the population is not constant, but changes by a factor p and the alleles do occur in the number q, these alleles are transferred of not transferred with chances, or in proportions pq/2n and 1- pq/2n respectively. So in one generation are (1- pq/2n)^2n alleles not transferred. This is for n→∞ e^-pq. This formula P0 = e^-qp is easily to be deducted and is than also applied in

many specialities. If a number of events occur in a period of time and the events appear ‘memory less’ in general is valid: P0(t) = e^-qt. In this is P(0)t the chance on no observation or hit of any event within period of time t. The complement of this, Pi(t) = 1 - e^-qt, is the exponential distribution. So it is the chance on one or more ‘events’ ‘arrivals’ or ‘hits’ within period of time t. This period may be a constant, for instance the time of one generation. The events or arrivals can be drawn or transferred alleles, if they are transferred memory less at random. **This is the case in this biologic field if any individual has any moment the same chance on effective reproduction.**

This (negative) exponential distribution is used generally in science. It is a statistical distribution, but you can see it also as an essential natural law. It also is supplied for instance in the field of epidemiology. An unfortunately realistic instance for illustration: a group of 10 young people goes to Ghana for development aid. They are handling careless their malaria prophylaxis and 6 of them acquire malaria. So now is measured here an average disease and infection prevalence of 0,6. But if 60% of the population has been infected more infections than 60 on 100 will be present in this population, because some people did probably acquire more than one infection. So what is here the infection incidence or infection load, or how many mosquito stings with the parasite plasmodium has been distributed in average to this population. Following this formula the time t is given and no infection in 4 on 10 people is given, so P0 =0,4 =e^-q and in this the negative quantity –q is to be calculated as –q= ln 0,4=−0,9163..So there were more than 9 infections in 10 persons. So here is the quantity q=0,9163 the proportion of events, or drawings, or hits per population. This proportion can also be expressed as a ‘chance’ or a ‘probability’, but than it is essential different from the change to be hit, to be infected, etc. The exponential distribution does however not describe how the events in the time, as infections, or drawings, or hits further are distributed into the space. In this example how many people do have in average 1; 2; 3, or more infections. The complete description of the distribution however is given in the Poisson distribution. Already in 1838 this complete theory has been posed by Siméon-Dénis Poisson, but only after 60 years (!) it was for the first time applied by Ladislaus Bortkiewiecz in the practical calculus of probabilities. This complete theory is given in the simple notion: **P(i) = e^-q . q^i/i!** In this formula P(i) is the expected, or average proportion of event in number i, in this i always is a natural number as 0; 1; 2; 3 etc and i! means i factorial. In the example it is from the observation certain that 60% has been infected and 40% has been not and with aid of the formula of Poisson than is to be calculated from this that on the average 36,7% did acquire one infection; 16,8% two infections; 5,1% three infections; 0,2% four etc. This generally well-known Poisson distribution is often describe by the formula **Pt(i) = e^-λt . λt^i/i!** In this **λt** is the parameter of the intensity of the Poisson distribution. The **t** in this is the variable period of time in which the events take place. In a system with a constant standard time like the generation time the **t** can be let away.

In the Application of the Poisson-exponential distribution in this field are taken as example of events, drawings, etc the ‘arrivals’ of the numbers of descendants or alleles into the next generation. The starting lemma’s as condition at this application are the lemma’s of the neutral hypothesis, the hypothesis of the negation or the zero-hypothesis of the evolution by selection:

1st Any individual has any moment the same chance on effective reproduction and any allele has any moment the same chance on transfer.

2nd Differences in reproductive success between parents and differences in the transfer between alleles are caused by

accident.
These a-priori and a-posteriori conditions, which differ only in meaning concerning the time of observation: before or after the event, are apparently not present in the actual life of the organisms. The reproduction is yet in many species seasonal, so that at once are born a number of cubs as a litter. So the chances on mating and reproduction are apparently not memory less, because a large part of the year the animals are not for mating disposed and not fertile as do the plants in most climates. This however concerns the actual reproduction we can easily observe in nature, **but for the study of the genetic changes and the evolution we have to observe the effective reproduction in the nature which is much more difficult**. In the effective reproduction are counted only the cubs that grow adult and get a litter themselves. The observation of the effective reproduction of course is necessary, because the genes and their variations are transferred only via individuals of the following generations that survive. The effective reproduction as the balance of birth minus juvenile mortality and infertility is much more or perhaps total time independent, because the death hazards are memory less and the juvenile mortality takes mostly a large part of the birth figures. The above conditions concern for instance the question if an animal that becomes a grandparent will have after this event the same chances to get effective descendants than before this. Further on are differences in chances if smaller scales are concerned which cause fluctuations as is pointed out here later, but this may be averaged on the larger scale. For instance in an area or in a period of drought the litters are smaller than in an area or period of abundance and this may effect even the effective reproduction and so by this it is fluctuating. On the larger scales however this may be averaged and than the condition ‘the same chances in any time’ should also be taken larger. The neutral hypothesis of equal chances and thus random causes for the differences in the reproductive results and ultimately for the evolution can be tested by observations of the results of the effective reproduction. Primary are equal chances on effective reproduction and not equal reproduction. Real equality of effective reproduction and allele transfer is excluded, because in nature does not exist something like rationing systems for mating, birth and dying. So not real or potential existing equality, but equal chances in effective reproduction and transfer of the genes should be the (negative) basis for the evolutionary theory.

The Poisson distribution is used here in the explication of the neutral random theory for the general populations. This because it concerns here at first the descendants of one person or the transfer of one or a very small number of alleles in a relative large population. In very small populations as pointed out later on here the binomial distribution is taken. The Poisson distribution can be used best in this cases with small numbers in a large space and it also has great advantages, I

think: its flexibility and its simplicity. The Poisson intensity does describe the average events in time and this intensity, or '**λ**' can easily be defined by parameters as is done here: For the distribution can be used the formula **P(i) =** e**^-λ . λ^i/i!** in the standard generation time. In this field the Poisson intensity **λ** apparently is determined by some factors, **q**, or **Q**, **p**, **r** and **s**, so that **λ=qprs** or **λ=Qprs.** In this is Q the primary quantum the absolute number of ancestors in the parent generation F0 (which always is 1 in the calculations here) or the absolute numbers of alleles in F0 that are transferred to the following generations. The further quantities of the absolute numbers in the generations from F1 are indicated with **q.** The factor indicating the change in the size of the population is **p.** If **p**=1 the size is constant; if **p**>1 the size is increasing; if **p**<1 the size is decreasing. The **s** faces the **theoretical** selection on account of the supposed fitness of the allele. Alleles with **s**=1 are neutral, with **s**>1 are advantageous and with **s**<1 are noxious for the survival and the reproduction. The factor **r** is the replacement ratio in neutral population dynamics, so if the population remains constant in size and if the alleles are neutral, so that the only changes are by hazard. At the reproduction in random mating with absolute out breeding **r**=2 and by inbreeding 1≤**r**<2; at the allele transfer always **r**=1. The validity of these factors determining the average numbers and so the Poison intensities in this way is pointed out further on here, but a-priori it is self-evident.

So are explained some basic conditions for the neutral theory, which is elaborated further in the chapters about the calculations of the Poisson distributed reproduction and allele transfer. At first however some observations and practical implications are concerned to show the relevance of the random neutral theory as the basis of the total evolutionary theory.

Observations

It is possible to examine in what extend the participation in the effective reproduction and thus the transfer of genetic variations is indeed Poisson distributed. It is possible but not easy to count numbers of effective descendants of the living animals and plants in the nature. This is a lot of work, but the statistic data of the numbers of children people do have in the different counties are direct available:

Table 1

Table H2. Distribution of Women 40 to 44 Years Old by Number of Children Ever Born and Marital Status: Selected Years, 1970 to 2004

Source: U.S. Census Bureau
Internet release date:

(Years ending in June. Numbers in thousands)

Year	Women 40-44 yr x1000	Women by number of children ever born in %								Children ever born per woman
		Total	None	One	Two	Three	Four	Five and six	Seven or more	
All Marital Classes										
1976	5684	100	10,2	9,6	21,7	22,7	15,8	13,9	6,2	3,091
Poisson λ=3,091		100	4,546	14,051	21,715	22,374	17,289	16,195	3,827	
1982	6336	100	11	9,4	27,5	24,1	13,8	10,4	3,9	2,783
Poisson λ=2,783		100	6,185	17,214	23,953	22,22	15,46	12,596	2,373	
2004	11535	100	19,3	17,4	34,5	18,1	7,4	2,9	0,5	1,895
Poisson λ=1,895		100	15,032	28,485	26,99	17,049	8,077	4,028	0,34	
Women Ever Married										
1970	5815	100	8,6	11,8	23,8	21,4	14,6	12,9	6,8	3,096
Poisson λ=3,096		100	4,523	14,003	21,168	22,371	17,315	16,254	3,858	
1976	5455	100	7,5	9,6	22,4	23,4	16,4	14,4	6,3	3,19
Poisson λ=3,190		100	4,117	13,134	20,948	22,275	17,764	17,359	4,396	
1982	6027	100	7,6	9,6	28,7	25,1	14,3	10,8	4	2,885
Poisson λ=2,885		100	5,585	16,114	23,245	22,354	16,123	13,776	2,804	
1985	6836	100	8	12,9	34,2	24,1	11,4	7,4	2	2,548
Poisson λ=2,548		100	7,823	19,935	25,397	21,571	13,741	9,976	1,557	
1988	7543	100	10,2	14,7	37,3	22,1	9,5	5,2	0,9	2,28
Poisson λ=2,280		100	10,228	23,321	26,586	20,205	11,517	7,247	0,896	
1998	9995	100	13,7	18,1	38,7	19,6	6,2	3,2	0,6	2,002
Poisson λ=2,002		100	13,506	27,04	27,067	18,063	9,04	4,828	0,456	
2004	10036	100	13,2	17,4	38	19,9	8,2	2,9	0,4	2,046
Poisson λ=2,046		100	12,925	26,445	27,053	18,45	9,437	5,179	0,513	

The US Census Bureau did collect in her table H2 the data of all the women from the total American population. The figures of the US Census Bureau are given here in **Table 1** and they show that particular in the short period from 1980 to 1990 has been a sharp decrease from about 3 to 2 in the average number of children a women of 40 to 44 years of age had in those years and also, what is important here, how this was distributed over the numbers of children, the parities, the have. Under a row with the data of the US Census Bureau I did ever make for comparison a Poisson distribution of the parities with, of course, the average number of children as its intensity λ. It concerns here figures of women that nearly all did complete their family (99%). In this way however are compared in **Table 1** the figures of the **actual** reproduction with the Poisson distribution and only the **effective** reproduction is the basis of the genetic transfer. There are indications that in this population the figures of the juvenile mortality are relative low and that the infertility, the younger generation will have later, should be distributed about equal over the parities. So the expectation here is that the failure by using the actual reproduction instead of the effective is not large, but there will be some differences. The point of interest is that these figures of the reproduction, despite these are actual here, give some direct indication for answers to the questions: are the genetic differences between the generations here larger, smaller, or as large as they are in at random expectation as the Poisson distribution indicates. Another problem is that the various differences may strengthen each other, but may also weaken and even neutralize each other. So further analysis and the calculation of the effective reproduction will give more specific evidence, but these raw figures already may give a realistic indication, which is described here.

We see in **Table 1** that the initial figures of the years ’70 and begin ’80 show some resemblance between the observation and the expectation following the Poisson distribution. Also in the group ever married are the figures under no children however larger than expected, probably because there have been always a minimum number of families that keep childless by biological infertility of one of the partners. In the later years the childlessness is more in accordance with the Poisson distribution and this is an indication for the small difference between the actual and the effective reproduction especially in the later years. In general are in the observed figures fewer women with 1 child, more with 2 children and fewer in the higher parities, although initial were the observed figures the very high parities larger. These general tendencies are increasing in the years and all the high parities than have lower figures later on. The aspect than in the later years of the observed figures in relation to the Poisson is that of a shift from the extreme values to the average value (=2). So the divergence in the distribution of the natural parities becomes obvious smaller than in the distribution of the parities following Poisson. So the genetic differences between the generations have become smaller the at random. **This is evidence**

for the view there is no (more) selective evolution in the modern American population. These differences are large and the divergences are so much smaller than random that it is very unlikely that figures from the effective reproduction should give another indication. The observed figures also did differ initially from the Poisson figures. The figures here are somewhat conflicting: the average values (=3) are about equal, but there was initial a shift from the moderate to the more extreme values in the observed distribution. So this is indicative for a little larger divergence and thus is likely a larger genetic difference between the generations than at random.

The divergence in the observed distributions in relation to the random Poisson distribution is an important datum, which directly indicates the changing of the genes and so the evolutionary intensity of a population. To learn the relevance of this you should inquire some more characteristics of the population. A population that is very heterogenic in the reproduction will have a large divergence, but such a population may be not a real existent social or natural group of individuals and than its evolutionary intensity is not so relevant. Such a population here in **Table 1b** is for instance the population women never married with 0,88 children per woman on average and a large divergence. The descendants of this population will of course be genetically different in the next generation, but that may be not relevant. This population must consist of women that are real singles and get only a little children and a group that have about average children and a family life, but they only are not married for the law and there may be others. So it is necessary to inquire the composition of the populations and the US Census Bureau gives than also the figures of the typical American subpopulations as they are called: Whites, Blacks, Asians and Hispanics of any race. Some of their data are given in Table 1b and I do compare these also with the Poisson distribution. In trying to make a better comparison and to estimate the evolutionary intensities I used a provisory coefficient in the right column. This is an instance, this method is not satisfying, I think, but is must be possible to develop here good exact methods. There may be relative small differences between the American subpopulations in these aspects of reproduction and genetic evolution. It is possible that these differences between the European subpopulations are larger but their data are not, or more difficult available.

Table 1b

Table 1. Women by Number of Children Ever Born by Race, Hispanic Origin, Nativity Status, Marital Status, and Age: **June 2004**
(Numbers in thousands. For meaning of symbols, see table of contents.)

(leading dots indicate sub-parts)
(Column B is in persons, all others are percents)

		Women by number of children ever born									
age	total x1000 women	Total women %	None %	One %	Two %	Three %	Four %	5 and 6 %	≥ 7 %	children ever born per woman	coëf MP
ALL RACES											
All Marital Classes											
.40 to 44	11.535	100	19,3	17,4	34,5	18,1	7,4	2,9	0,5	1,895	0,868
Poisson λ=1,895		100	15,032	28,485	26,99	17,049	8,077	4.028	0,34		
ALL RACES											
.Women Ever Married											
..40 to 44	10.036	100	13,2	17,4	38	19,9	8,2	2,9	0,4	2,046	0,723
Poisson λ=2,046		100	12,925	26,445	27,053	18,45	9,437	5,179	0,513		
ALL RACES											
.Women Never Married											
40 to 44	1.498	100	59,8	17	11,2	6,2	2,1	2,9	0,9	0,88	1,973
Poisson λ=0,88		100	41,478	36,501	16,06	4,711	1,036	0,042	0,001		
WHITE ONLY											
.Women Ever Married											
40 to 44	8289	100	13,4	16,8	39,3	19,7	8	2,4	0,3	2,02	0,694
Poisson λ=2,02		100	13,266	26,796	27,064	18,223	9.203	4,97	0,478		
WHITE ONLY, NOT HISPANIC											
.Women Ever Married											
40 to 44	7206	100	14,1	17,2	39,8	19,6	7,1	1,9	0,2	1,959	0,718
Poisson λ=1,959		100	14,1	27,622	27,056	17,667	8,653	4,498	0,406		

HISPANIC (of any race)											
.Women Ever Married											
40 to 44	1179	100	8,1	14,3	36,1	20,5	14,6	5,2	1,2	2,437	0,854
Poisson λ=2,437		100	8,742	21,305	25,96	21,088	12,848	8,8056	1,251		
BLACK ONLY											
.Women Ever Married											
40 to 44	1.054	100	12,3	20,3	27,9	24,7	9,5	4,2	1,1	2,198	0,928
Poisson λ=2,198		100	11,102	24,403	26,819	19,65	10,797	6,485	0,743		
ASIAN ONLY											
.Women Ever Married											
40 to 44	470	100	12,9	20,5	40,1	13,4	6,3	6,1	0,7	2,052	0,711
Poisson λ=2,052		100	12,848	26,364	27,049	18,502	9,491	5,227	0,519		

Source: U.S. Census Bureau, Current Population Survey, June 2004.

A situation different from the data of the US Census Bureau shows the picture of the historical data on **Table 2**. This population is describes more detailed at **Table 9b**. In this population is described concretely the effective reproduction. Used are data from a genealogy of a family of fishermen and skippers living in the South-western of the Netherlands in the 18th and 19th century. They are 72 parents with 220 effective children, all descendants of one person. As is shown on Table 2 the variations of the parities, so the observed divergence in the distribution, are larger than the divergence of the Poisson distribution. This is an indication that this people, different from the modern one, did have unequal chances on reproduction and survival and that by this the genetic differences between the generations must be larger than random expected. Thus this is an evolutionary population. If larger and more of these populations are inquired in this way and these results should be confirmed, this should give direct evidence for the evolutionary character of these populations and also parameters of the evolutionary intensity of the different populations are easily to be deducted by experts in this study. It is probable, I think, that the populations of the animals and the plants in nature and also of people mostly are evolutionary, but some populations or species may be not evolutionary and we as modern living people are one of them. The non-evolutionary species may be exceptions, but possibly they occur more often than we think. There may be indeed different reasons by what animals can be non- or very slow evolutionary.

Tabel 2

→0	→1	→2	→3	→4	→5	→6	→7	→8	→9
Poisson, λ=3,05556									
0,0471	0,14391	0,21986	0,22393	0,17106	0,10453	0,05324	0,02324	0,00888	0,00301
populatie n=72		gemiddeld 3,0555 kinderen per ouder							
→0	→1	→2	→3	→4	→5	→6	→7	→8	→9
0,09722	0,194444	0,22222	0,11111	0,06944	0,13889	0,08333	0,06944	0	0,01389
7/72	14/72	16/72	8/72	5/72	10/72	6/72	5/72	0	1/72

There are many publications about birth and fertility figures in the various countries. Data about the parities are however much more difficult to be found and are apparently not collected in most counties, but there are other countries than the USA that do so, see the literature. These diagrams of the parities do also show obvious how was the development in Russia from more variation to less variation than at random in the parities.

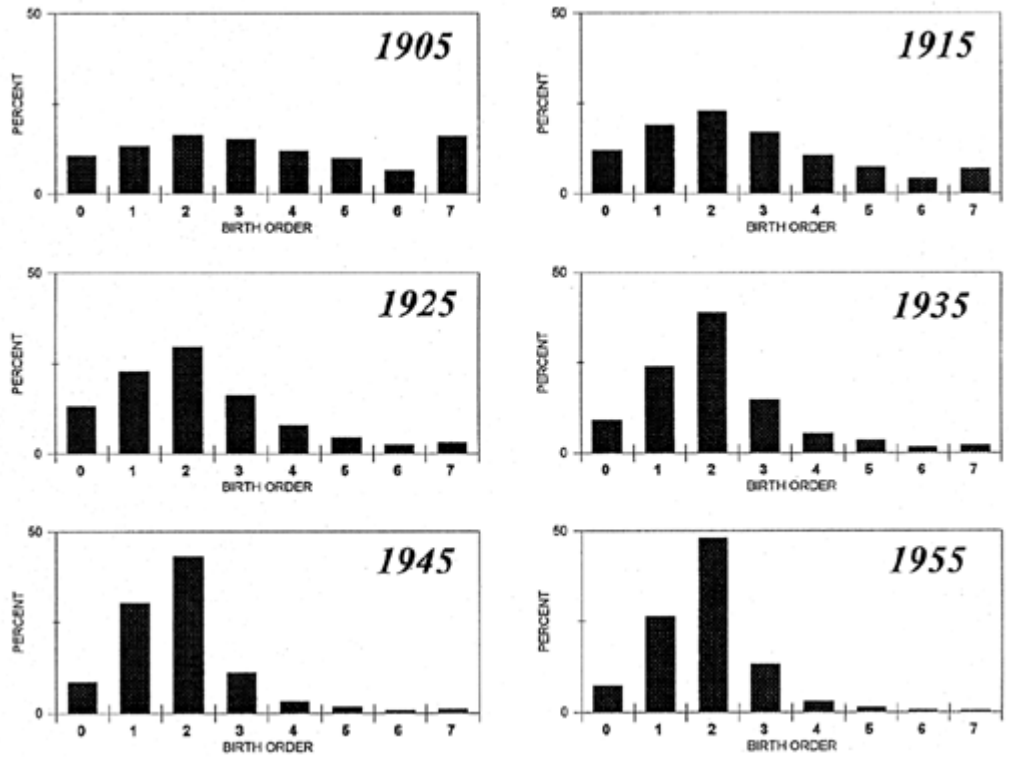

Figure 2.9--Distribution of Women by Number of Children Born by Age 50 in Different Birth Cohorts in Russia (estimates for birth cohorts of 1945 and 1955)

Random or[4] selective changes

The premise of random mating, which often is pointed in literature, can be described in plain English as: any individual has any moment the same chance on mating. This mating can better be specified to its consequence we are interested in: the effective reproduction. This random effective reproduction does exist actually in real existing biological populations, if the condition equal chances have been fulfilled and if they have not been fulfilled, as is mostly, the random effective reproduction will exist only potentially in the population. The dialectical neutral theory starts from the idea: there are possibly random differences and non-random differences in the reproductive results individuals have and in the transfer of the individual alleles. It is possible with the modern technical tool to observe in all kind of levels and situations in actual populations differences in these both processes: the effective reproductive results and the transfer of the gene variations (alleles). As pointed out further here (page 55) reproduction and allele transfer are causal linked and in the random form they (or it) even exist uniformly. The observational results are of course the sum of random and non-random differences. The random differences are well-known, as they are given by the Poisson distribution**.** This also is ascertained in literature. The observed distributions of the effective offspring thus will mostly differ from the Poisson distribution. This fact thus should nowise induce the meaning that the statistical distributions are of none or very limited importance in the inquiry of genetic changes in the micro-evolution. The random events are of course physical present, but what we observe on living beings never is the result of hazard alone. Children of people and animals are not born and do not dye only by accident. The results we can observe like products of effective reproduction and so gene transfer always are a combination of hazard and biological action or function. The latter of this, the organic skill to procreate and survive, can be defined as selection. So selection than also is the causal unity of the non-random differences, which is the dialectic complement of hazard in the struggle for life and so has here a somewhat more specific meaning than in the pure Darwinist sense. Although.., there is survival of the fittest and there is survival of the luckiest, but only the survival of the fittest is survival by selection. It ever is important I think, in philosophical and scientific research to find the essential detail in the background of the noise of accidental events.

[4] In this the meaning of ‘or’ is inclusive in the sense of or/and.

Random transfer or selection at work

In the data of Table 1 is for instance an about 30% smaller variation than Poisson-random in the parities in the data, the US Census Bureau collected in the later years, of women getting children in the years about 1970-2000. In the data of Table 2 from some small number of Dutch people getting children in about 1740-1890 is a larger variation in the parities than the Poisson distribution gives. This does not mean that the Poisson distribution gives invalid information, or it should be not appropriate here. It does mean that people in both populations did not procreate by accident. This point of course is very well known, but interesting to this may be the exact specification of the total different deviation from the random. This complement of the accident is what makes a researcher curious in many fields. The modern women with the smaller variation in the parities appears to have little chance (smaller than average) to get anymore children, if they have already 2 or 3 children. If they have only one child their chance to become mother again is larger than of the average other women. As we know the cause of it is a strong selection on the numbers of children itself. In the social context of their society modern American people do prefer to have 2 or 3 children. They do have possibilities to do so and of the children that have been born only about 3% dies before adulthood. However more than 100 years ago people lived in total different situations in most countries, as did my ancestors, the people of Table 2. They did live in another social context, with also less possibilities and a large (ca 50%) and variable number of their children died before adulthood. So especially by the high infant mortality they also had their 'family planning', so determining the number of their adult (or effective) children, but effectuate it more by caring the children well and preventing their dead than by contraception. They had much more differences in the numbers of their adult, effective children than the modern people do, by difference in good luck, but also by their different talents, their motivations and possibilities to keep their children alive. **By these large variations in their effective parities and also by inbreeding in combination with more competition our ancestors were biologic evolutionary and we are not anymore**. More evidence for this conclusion is pointed out further here. By this conclusion however is no reason to worry about our modern way of family planning as being non-evolutionary and bad for our genes, on the contrary we have to worry about the many ill-functioning genes acquired into our genomes by the evolution of our ancestors! Interesting also are the US figures of birth in the parities, collected in the former years, so the women getting their children in the years about 1940-1970. In those years the infant mortality was a bit higher than now, but still so low that it already was no more a substantial part of the effective reproduction. The women in this time did differ more from each other in their family planning. The culture was not yet so uniform in this as it is now and this issue may be more important than the lesser possibilities for contraception, because this already was sufficient possible. These more different choices in the

family planning are showed by the variety in the distribution of the actual children over the parities they had, being nearly at random, or somewhat larger.

Another picture gives the reproduction of salmons. A population of salmons is reproducing far in the upper reaches of a distinct river. The circumstances in the spawning-grounds are rather uniform. The salmons seldom reproduce more than once in their lives and dye afterwards. The next generation hatches from the egg and grows in the river and further in the sea. A small part of the young salmons return later into the source river as an adult for reproducing and dying. As for concerning the parent generation the young salmons will apparently behave as real equal luck 'Poissons' (Fr: fishes). The parent salmons did yet produce about the same numbers of fertilized eggs and in this river the young's became all about the same change on growing and survival from their parents, that brought them hitherto. It is all up to the youngsters now. The eventual non-random or selective differences and so non random allele transfer may appear in the possible differences in chances to survive and to grow up to adulthood and to swim at last all the way back to this source river. If some salmons have more chances on success than others, this will give rise to more variations in the distribution of the offspring and in the larger genetic differences between the generations in the population. This possible selection than will also strengthened by the inbreeding the salmons will probably have by mating in relative small populations for many generations in the source rivers.

By birds and many other species again is the situation different. Many species of animals and even plants will give indeed some form of parent care to their living children or give them something to survive better. The salmons were particular in this because they do invest into the offspring already before mating by swimming up to grounds that are favourable for the survival of the brood. The real parent care birds give come after mating, brooding and hatching. The survival of the young generation than depends on the dedication and the possibilities of the parent(s) and the young itself, so of some more genetic different individuals. The difference in the success the parents have at the raising of their young's often is based on good luck or blind evil. It so can happen that at one year there is a drought in their living area, so that the parents can find less food for the young's than do parents in other living area's. Such incidental differences are not important, for the non-random distributions of the population in somewhat larger scales may stay valid, because in more litters and more generations the incidental differences will compensate each other. It is important that the differences do exit systematically as capacities in the care and in finding the food some parents may have more than other parents. Because the young's

themselves are present at this care the better caring capacities can be transferred in two ways through the generations: by the genetic transfer and by the imitation, because the young's will imitate their parents behaviour later when they get litters themselves. This imitation can make some stocks of birds systematically more successful in survival and reproduction. This however may happen without regard of the characteristics of some genes. The genes of these more successful individuals, because of their acting well by imitation will also be transferred more than at random, although these genes did not attribute to the success. In this can the transfer of some ill-functioning genes accidentally be promoted it they occur in smart acting birds. This, however may cause drawbacks later on, because physical defects in the birds can develop by the accumulation of less functioning genes. So on the larger scales the non-random differences are more consistent if the are transferred indeed by the genes.

Some evolutionary problems in people

From the beginning some million years ago the human (hominid) species have had to give - in relation to average animal species - a very intense and lasting care for their children and this further is increased during their evolution. They did have than also more abilities in this care than the animals had and could transfer their abilities more effective through the generations than the other species. They were able to do this and many other things, because their brain is very large in relation to individuals of all other species with about the same bodyweight. This brain makes people capable to use their sense organs more efficient and that improvement of their sensory perception was very useful for the people in their care for the children, their defence against predators and their search for food. The possibilities of the brain however go much further, we do know now as modern people. Your brain makes also possible to perceive the things behind the things. This deeper perception however of the causes etc behind the things was a huge problem for the primitive people in prehistory and they generally avoided to gather information about the things behind the things. If they did inquire these or should invent new possibilities this ever brought themselves and their tribes in large difficulties. Although the brains of our direct ancestors, homo sapiens and perhaps also of the other hominids as homo rectus and his later form the homo Neanderthal could work as well as ours or perhaps even better, those people ware not able to use their brains as we do, because of their social situation. An important problem is yet that people do observe the world indeed much deeper than animals do, because much more efficient receivers are opened for the information from the outside, but people do have the same anxious cautiousness animals have for survival in dangerous surroundings. This excess of info about potential dangers makes ancient people, but often also modern people, very anxious and also very aggressive. The problems are much aggravated by the

consciousness people have of the things behind the things and the communication about these with each other. People will so, in lack of modern knowledge, experience various threatening phenomenon's and see a whole threatening world as causes for different events. They can be very anxious for the thunder, for the shining of the moon, for the strange behaviour of other people being friends of evil ghosts, etc. The destructive consequences of magic and fear were moreover not the only disadvantages of the peoples brains. In this matter also important is the interference between the transfer of the genes - the genetic information - and the transfer of the 'neuronic' information through the generations: More than at the birds the problem here was if your tribe or stock of people have success by their smart solutions they may gather ill-genes. Drawbacks by this must have happened, but this problem will be prevented mostly by a smash with the knout on the smart brain that should not let people behave strange and thus evil. The human evolution has been a very complicated process, which is only partly unravelled by the scientific research, I think. The micro-evolution in this, so the genetic transfer through the homo sapiens generations in about 200000 years, of course is of special importance. A question that rises is how fast was the evolution in the sapiens generations, or how large are the genetic differences between the generations. The problem is that the indications for the answer to this question are conflicting:

A-priori there is indication for fast evolution within sapiens, because the non-random or selective differences are made by the biological, social and whatever functions of the organisms selves. Humans are relatively well equipped to achieve their targets and do often use aggressive and radical modes to do so. For the things they can prefer, as are the appearances, it thus must have gone very fast. So, many people in our part of Europe do have **fair or red hair and white skins**[5] chiefly because once that was the most sexy trend for our ancestors. Also the invisible interference of the 'neuronic' and genetic information may increase the selection. For instance this situation: an epidemic comes over 3 groups consisting of a few genetic resistant people and a variable number of people that also are able to survive because they are smart. The 1st group with none smart people may easily dye out, because the resistant ones probably die of starvation. The 2nd group with some smart people will survive, because they have more and smarter survivors of the epidemic. The 3rd group has only smart people, so that the total group survives. By the interference only in the second group is selection on the resistance gene and this is the situation at the primitive people. The first group is more the situation at the animals and the third group is at the modern people, both

[5] The selection on these genes must have been very strong because they mostly are recessive, were probably seldom present in the source populations that came from the South, while they now are nearly fixed in many populations in the North of Europe.

without selection. Also the archaeological data indicate a fast evolution. That data shows a picture of the different homo species the arose and died out, following each other in a short time in relation to the evolution of many animal species. On the other hand apparently is against the fast evolution that the genetic differences between in our time living people are small, so that the people of the different continents did not yet grow much different in such a long time, which is not consistent with the fast evolution. It however is possible in two cases that the human and particularly the sapiens evolution has been much faster than average in the nature in spite of the small genetic differences between people now. Than there should have 1st been much migration between the continents, or 2nd the evolution should have been in the same direction at the continents, so that the same genetic differences are transferred by prefer in the different continents. Both motives seem improbable to me, but the later is of course consistent with the multiple regionality theory. In this theory the homo sapiens should have arisen not only in Africa but in different places on the world and the different stocks of these sapiens like mutated former homo erectus people must than have converged to our modern people, with their relative small genetic differences. This multiple regionality theory, with some things more, will indicate than also there must be something like an internal organisation of the **genes** that is **self-regulating** them. That this exists is not totally evident by what is known in the molecular biology, I guess, although there may be some hints in this. Nevertheless there is evidence for the existence of self-regulation in the physiology of the organisms and broader in their biological functions and by different reasons this implicates imperatively also self-regulation within the genome. This is a logic a-priori premise, which yet is to be checked and specified a-posterior by the molecular characteristics of the DNA and some primary biologic functions. By self-regulation the allelic characteristics will not only diverge, but also converge in the evolution and this is a very important issue. Self-regulation in the bio-evolution is not generally taught, but it is by Prof. Francis Heyligen in Brussels.

Also what we know from the historical and prehistorical data about the reproduction possibilities in the live of the people is indicative for fast evolution. The more different possibilities will cause probably more variations in not modern people than average in most species. You can imagine that when people became somewhat more knowledge together with increasing social inequalities more chances for the privileged groups will arise. Also the often very aggressive tendencies in the social life of people (also in modern!) as in war making in combination with killing the conquered enemies, burning down their possessions and violating their wives may cause strong selective differences, although in the larger scales many of the effects by these inequalities may be balanced. An important issue however in the historical view at the human evolution is that the fitness in ancient times was mostly very different from what fits in our present society and in our present biological

situation. It often was fit to be an aggressive man raging at people of other ethnicity and violating their women. This now is very criminal behaviour, but it is no wonder that you can watch this kind of behaviour everyday in the streets of our cities. These problems were still worse if behaviour was total inheritable and people could not correct by intelligence for their natural tendencies. In other situations the historical–evolutionary problems are still more evident. In the past many alleles have accumulated in the genomes of people by selection at fitness towards situations that do not exist anymore. Examples of this are the alleles that make people resistant for specific infectious diseases that are easily to control now. The sickle cell anaemia is a famous example of this. There are found many more of these cases and some will never be found because the infectious agent has been disappeared for a long time. The cause of the systematic occurrence of this phenomenon is very simple: The pathogen needs a key to come into some specific cell of the host organism and it is specialized by genetic selection to use the key, which often is a protein on the cell wall. If the key does not function the pathogen has a problem, but also the host organism. The less functioning key at the heterozygote does increase the resistance, but this does ever mean also a less functioning protein or total cell, which means non functioning in the case of homozygosis. This problem is evident in some monogenetic diseases in people as the different haemoglobin disorders, cystic fibrosis and others. The problem may be much broader: also many polygenetic inheritable diseases are possibly induced by genetic selection. Our ancestors did live close together without any form of hygiene and also got many traumata and this did them so suffer a lot from all kinds of infections during many ten thousands of generations. No wonder from the evolutionary view that we now posses a very aggressive immune system that easily deregulates giving rise to auto immune diseases, in which the immune system attacks the cell of the own body and also to allergies with the exaggerated reactions on harmless vectors.

This comes to the conclusions: People may have had a relative fast evolution. The evolution now suddenly has stopped, but we have definitely not to worry about this ceasing. We are on the contrary in big troubles by the genetic variations accumulated by selection in our ancestors. The genes are of course now not to be removed by natural evolution, also not by sharp artificial evolution, or as it is called radical eugenics. The results of artificial evolution is to be seen at the sad genetic state of our house animals. The races of the house animals have been bred mostly under veterinarian control, but there are still gigantic genetic problems. There are also very important ethical objections against eugenics. A free medical advise for a family in specific cases is of course something else.

Random transfer or sexual selection

The distribution of descendants and alleles is, as a-priori expected, in the masculine gender different from the feminine. The masculine and feminine organisms do reproduce with different properties and these differences will result in different chances on reproduction and so in a different distributions of their descendants. The more general differences between feminine and masculine is not always corresponding with the biological sex even in the field of reproduction. So it may be better to use other words for describing the sexual characteristics more general and more typical: yin for the feminine and yang for the masculine reproduction. In the yin the woman's part is uniform within the mother's part and the pure yin will receive the both parts. The yin does invest **maximal** into the child to create **maximal** effective numbers of children. This is attended with few sexual competition and even with cooperation between the females. The yin will limit to the minimum the variation in the numbers of children the different females have. A more unequal distribution of the numbers of the descendants would be unfavourable for the survival. It yet is not 'efficient' human economics would say if the hard work of motherhood is not borne by all the females in proportion to ability. This minimal variation in the effective children is given by the Poisson distribution, if the abilities are equal and if there is large juvenile mortality. This last condition is mostly present in nature, but in our modern human populations the infant mortality is very small and so than the most efficient distribution of the children among the mothers is with a smaller variation than Poisson. By differences between the females in abilities for the motherhood the yin wants a larger variety than Poisson and it so creates selection. Particularly in more intelligent organisms there may be systematic differences in abilities.

In the yang the man's part is distinct from the father's part and so in the pure yang can present only the man's part. The yang invests **minimal** in the child for the possibility to create **maximal** numbers of children. This brings much competition between the males and even with females. In that eternal male-female conflict the ever heard female argument is: you always are thinking of the one thing and the male argument is: you cannot make well more than one thing simultaneously. The yang is working on distance the effectiveness of his procreation (care) is via the yin. The number of the partners and their possibilities are the results of the yang, whereas the numbers of the children and their possibilities are the results of the yin. So the yang has to follow the yin in the minimum variation augmented by the variation the yang has in the numbers of partners. This extra variation the yang has in the parities is created by the differences between the male individuals for mating in competition with each other. These extra variations are called the sexual selection. The sexual selection generally is present in nature. The sexual selection however probably is larger in species that do have large individual differences, as

is in intelligent creatures. If there also is a large social inequality, as it was in historical human societies, sexual selection can become extreme large. Some men with much power and high distinction did have a lot of descendants. So nearly everymen in the neighbourhood descents apparently from the old celebrities as Charlemagne or Dzengis Khan. It may be obvious that the selective variation by the fatherhood (or sexual selection) in general is larger and also has other qualities, because it has been selected on other characteristics (mating ability) than by the selective variation by the motherhood (care ability). These differences in quality may be of evolutionary importance: systematic selection on characteristics. In the larger scales may exceed furthermore the female selection for instance also the quantity of the male selection, if the larger differences by the male selection in more generations can be more neutralized in the allele transfer, while the female selection can be more systematic.

So because yin and yang are different biologic functions it is useful to observe and calculate the male reproduction distinct from the female. This however is not done in the genetic drift theory; in the literature the distinct yin and yang selection always are equalized by the formula of the effective population size. Oh, girls did not I say that you can not do your work well if you try to fix the different things at the same.

Calculation of the Poisson distributed reproduction.
Suppose the size of the population is constant on **n** individuals in the generations F0; F1 and F2. The reproduction population in study consists of parents of effective children. Only descendants in the first generation that have become parents themselves are counted as individuals of the population. So parents with 0 children are parents that did not become grandparents. There is random reproduction. So any individual of F0 has the same change of 1/**n** to be the parent of the new individual of F1 and 1-1/**n** to be not the parent. For **2n effective children**[6] of F1 the change to be not the parent is (1−1/2**n**)^2**n.** This is 1/e for larger values of **n**. In this is e the natural base 2,7183.. In the case **n**=10 this "base" already is 2,7895.. On this account the distribution of the effective descendents in the next generation is essentially according to the Poisson-exponential distribution, if the population is not very small. So the average proportions of the individual organisms in F0 with **i** descendants in F1 are to be calculated by substitution in **P(i) = e^-λ . λ^i/i!** In this the intensity **λ** can be determined by parameters, so that **λ=Qprs** or **λ=qprs.** In this is **Q** the primary quantum; **q** is the general quantum; **p** the

[6] The n individuals of F0 do have 2 children on the average together with their sexual partners in the population of constant size.

change in the size of the population; **s** the theoretical or virtual selection and **r** is the replacement factor by neutral population dynamics. Because the children and further descendents of 1 individual in the generation of the first parents, the F0, are studied in **Table** 3 **Q**=1. This individual has in many cases (proportions) more than 1 descendant in the F1, These will than also participate in a number (quantum) >1 participate in the new parent generation. That is why q is indeed variable and has natural values as 1;2;3, etc. In this example the population size is constant, so **p**=1. By sexual reproduction the individual organisms have on the average 2 descendants in the next generation in neutral population dynamics, so **r**=2. There is random mating with equal chances, without selection, so **s**=1. So it is obvious that **p**, **s** and thus **λ** can have in principle any values ≥0.

The average random or Poisson distributed offspring of the individual organisms of the primary parent population F0 is given in **Table 3**. If you calculate by substitution in the formula with **λ**=2 it appears that the proportion of F0 with 0 descendents in F1 is e^-2 = 0,1353.. This proportion participates active in the reproduction but will have no descendants by random mating with equal chances. The proportion 2.e^-2 = 0,2707 of F0 has 1 descendant on the average in F1. The proportion 4/3.e^-2 = 0,1804 has 3 descendants. 2/3 .e^-2 = 0,0902 has 4 descendants etc. So the average of 2 descendants is in this way Poisson distributed and the sum of the descendants, calculated in this way indeed is 0,2707x1 + 0,2707x2 + 0,1804x3 + 0,0902x4 +… = 2. These are descendants that individuals in the F0 will have together with their different sexual partners so that the population keeps the same size.

In the distribution of the descendants of F0 in F1 there is only one intensity **λ** of the expected number of children of F0 in F1. This intensity is in random mating only determined by the population dynamics, so what is necessary for maintaining of changing the effective population size. But if we consider the descendants of F0 in F2, the grandchildren, there must be different intensities. The children the F0 individual has in F1 determine by their numbers **q** the expected number of their grandchildren of F0 in F2 and so determine the **λ** of the distribution for the new generation together with any possible changes. So, because of the different numbers of children in the F1, the distribution of the descendants of the primary parents, the F0, in the further generations has no uniform intensity. As a symbol for this variable intensity is used **λ***, so that : **λ*=qprs.** By 1 descendant in F1 the expected number of descendants in F2 is in neutral dynamics : **λ*=qprs**=1x1x2x1=2. By 2 children there are 4 grandchildren on the average, because than q=2, so that **λ***=2x1x2x1=4. By 3 children there are 3x1x2x1=6 descendants in the F2 etc. So it is possible to calculate the 4 descendants the individual from F0 has on the

average in F2 in a further Poisson distribution. In this would be not right to consider the originate of the generations F1 and F2 out of F0 straight away as one process and do so calculating this as a Poisson distribution with intensity 4. That is not right, because the origin of the F1 and the F2 are two processes all within its own space of time. In these the individuals of the F1, the parents and not the grandparents from F0, are concerned in the events, the "arrivals" that give rise to the F2. The only overlap of the two processes is that individuals in F0 that have no descendents in F1 will also have no descendents in F2.

The proportion 2e^-2=0,2707 of F0 has one descendant in F1. The size of the population in F0 and F1 is constant on **n.** So there are **n**2e^-2 individuals coming in this way. You can calculate the proportion of this, so the way of 1 descendant from F0 to F1 and zero descendants from F1 to F2 (notice →1→0) by substitution with **λ***=2 and i=0, than it is 2e^-2 . e^-2 = 2e^-4. So is (→1→1): 2.e^-2 . 2.e^-2 = 4 e^-4. (→1→2) is 2.e^-2 . 2.e^-2 = 4 e^-4. (→1→3) is 2.e^-2 . 4/3. e^-2 = 8/3 . e^-4. (→1→4) is 2.e^-2. 2/3 .e^-2 = 4/3 . e^-4. (→1→5) is 2.e^-2 . 4/15 .e^-2 = 8/15 . e^-4. (→1→6) is 2.e^-2 . 4/45 . e^-2 = 8/45 . e^-4. (→1→7) is 2.e^-2 . 8/315 .e^-2 = 16/315 . e^-4, etc. Simply used is as substitution in the Poisson formula is **q**=1; **r**=2; **λ***=2 en **i**=0,of **i**=1, of **i**=2, etc and multiplied with the factor 2e^-2, the proportion of the one descendant in the F1.

So has also the proportion 2e^-2 of F0 two descendants in the F1. These parents in F0 expect however to get here 4 grandchildren, so **q**=2; **r**=2 and **λ***=4 and (→2→0) is 2.e^-2. e^-4=2e^-6. (→2→1) is 2.e^-2. 4.e^-2 =8.e^-2. (→2→2) is 2.e^-2. 8.e^-2 =16.e^-2, etc. In these calculations continually **λ***=4 en **i**=0, **i**=1, **i**=2, etc. The total distribution of the descendants of F0 in F1 and F2 is given in **Table 3**. The way of descend is showed with the arrows.

Table 3

Descendants of F0 in F1								
→0	→1	→2	→3	→4	→5	→6	→7	→8
e^-2	2.e^-2	2.e^-2	4/3. e^-2	2/3 .e^-2	4/15e^-2	4/45e^-2	8/315e^-2	2/315e^-2
0,135342	0,27067	0,27067	0,18045	0,09022	0,03609	0,01203	0,00344	0,00086
x2/1	x2/2	x2/3	x2/4	x2/5	x2/6			
Descendants of F0 in F2								

→0→0
e^-2

→1→0	→1→1	→1→2	→1→3	→1→4	→1→5	→1→6	→1→7	→1→8
2.e^-4	4. e^-4	4 e^-4	8/3 e^-4	4/3 e^-4	8/15 . e^-4	8/45 e^-4	16/315e^-4	4/315e^-4
→2→0	→2→1	→2→2	→2→3	→2→4	→2→5	→2→6	→2→7	→2→8
2e^-6	8e^-6	16e^-6	64/3e^-6	64/3e^-6	256/15e^-6	512/45e^-6	2048/315e^-6	1024/315e^-6
→3→0	→3→1	→3→2	→3→3	→3→4	→3→5	→3→6	→3→7	→3→8
4/3e^-8	8e^-8	24e^-8	48e^-8	72e^-8	86,4e^-8	86,4e^-8	2592/35e^-8	1944/35e^-8
→4→0	→4→1	→4→2	→4→3	→4→4	→4→5	→4→6	→4→7	→4→8
2/3e^-10	16/3e^-10	32/3e^-10	512/9e^-10	1024/9e^-10	8192/45e^-10	242,736e^-10	277,401e^-10	277,401e^-10
→5→0	→5→1	→5→2	→5→3	→5→4	→5→5	→5→6	→5→7	→5→8
4/15e^-12	8/3e^-12	40/3e^-12	44,44e^-12	111,11e^-12	222,22e^-12	370,37e^-12	529,10e^-12	661,38e^-12
→6→0	→6→1	→6→2	→6→3	→6→4	→6→5	→6→6	→6→7	→6→8
4/45e^-14	16/15e^-14	6,4e^-14	25,6e^-14	82,29e^-14	184,32e^-14	368,64e^-14	631,95e^-14	947,93e^-14
→0→0				→→	→→	→7→6	→7→7	→7→8
0,135342						265,59e^-16	531,18e^-16	929,57e^-16
∑ 0 F2	∑ 1	∑ 2	∑ 3	∑ 4	∑ 5	∑ 6	∑ 7	∑ 8
0,042068047	0,09604	0,12155	0,1207	0,10737	0,09086	0,074075	0,05832	0,04447
as e functon	X 1	X 2	X 3	X 4	X 5	X 6	X 7	X 8
[(e^2^(e^-2)-1]/e^2	0,9604	0,2431	0,3621	0,42948	0,4543	0,44445	0,40824	0,35576

∑ 0 F1+F2
0,177412
[(e^2^(e^-2)]/e^2
X 0

→9
0,00141093e^-2
0,00019

→1→9

→2→9 →2→10 →2→11

1,4448e^-6	0,5779e^-6	0,2102e^-6	0,07005e^-6					
→3→9	→3→10	→3→11	→3→12	→3→13	→3→14	→3→15		
1296/35e^-8	22,217e^-8	12,118e^-8	6,059e^-8	2,797e^-8	1,199e^-8	0,479e^-8		
→4→9	→4→10	→4→11	→4→12	→4→13	→4→14	→4→15	→4→16	→4→17
246,579e^-10	197,263e^-10	143,464e^-10	95,643e^-10	58,857e^-10	33,633e^-10	17,937e^-10	8,969e^-10	4,221e^-10
→5→9	→5→10	→5→11	→5→12	→5→13	→5→14	→5→15	→5→16	→5→17
734,86e^-12	734,86e^-12	668,06e^-12	556,71e^-12	428,24e^-12	305,89e^-12	203,92e^-12	127,45e^-12	74,97e^-12
→6→9	→6→10	→6→11	→6→12	→6→13	→6→14	→6→15	→6→16	→6→17
1263,91e^-14	1516,69e^-14	1654,57e^-14	1654,57e^-14	1527,30e^-14	1309,11e^-14	1047,29e^-14	785,47e^-14	554,45e^-14
→7→9	→7→10	→7→11	→7→12	→7→13	→7→14	→7→15	→7→16	→7→17
1446,0e^-16	2024,40e^-16	2576,51e^-16	3005,93e^-16	3237,16e^-16	3237,16e^-16	3021,34e^-16	2643,68e^-16	2172,65e^-16
∑ 9	∑ 10	∑ 11	∑ 12	∑ 13	∑ 14	∑ 15	∑ 16	∑ 17
0,0329265	0,0238454	0,0168737	0,0116831	0.00788	0,0052613	0,00343908	0,00217468	0,00135777
X 9	X 10	X 11	X 12	X 13	X 14	X 15	X 16	X 17
0,2963385	0,238454	0,1856107	0,1401972	0,10244	0,0736582	0,0515862	0,034795	0,02308

→4→18					
1,876e^-10					
→5→18	→5→19	→5→20	→5→21		
41,65e^-12	21,92e^-12	10,96e^-12	5,22e^-12		
→6→18	→6→19	→6→20	→6→21		
369,63e^-14	233,45e^-14	140,07e^-14	80,04e^-14	43,66e^-14	22,78e^-14
→7→18	→7→19	→7→20	→7→21	→7→22	→7→23
1693,33e^-16	1247,72e^-16	873,40e^-16	580,27e^-16	370,54e^-16	225.55e^-16
∑ 18	∑ 19	∑ 20	∑ 21	∑ 22	∑ 23
0,00083899	0,00046922	0,000249	0,000164	0,00008	0,00004
X 18	X 19	X 20	X 21	X 22	X 23
0,0151	0,00892	0,00498	0,00344	0,00176	0,00092

In **Table 3** are mentioned the numbers of descendants as a product of e under the field of the arrows. So under example →3→6 is noticed in proportions of F0 through 3 descendants in F1 to 6 descendants in F2. Notice that the numbers of the sums under ∑, so 0,17740 ; 0,09604 ; 0,12155; etc are proportions of F0 with totally 0; 1; 2; 3; etc descendants in F2. The further Poisson-like distribution that is given here is thus more asymmetric than the normal primary Poisson distribution. Of that totals under ∑ only the sums for 0 descendants can be expressed fully as products of e. The total of all the proportions or change intensities is indeed 1. The sum of the descendants in F2 to an ancestor in F0, so 0x 0,17740 + 1x 0,09604 + 2x 0,12155 + … is indeed in total 4. In this secondary distribution F1→F2 there is no uniform **λ** for the different ways of descent, because of the variable quantities, **q**. However there can be a constant **μ=prs** through the generations. Than you can use **λ*=qμ** by the substitution in the formula **P(i) = e^- λ* . λ*^i/i!** of the Poisson distribution. There is also a sum Poisson intensity **ς** up to the level of the distributions, here generations. This **ς** here is simply the average number of descendant in a generation Fg to an ancestor in the F0. If **μ** is constant through the generations **ς=μ^g** for F0→Fg. The calculations in Table 3 are restricted. It is stopped with 8 descendants in the F1 and with 23 in the F2. So there has been made a calculation over about 99% of the descendants over the two generations. Notice as a conclusion of **Table 3**: there are great differences in the efficiency of the reproduction even if there are equal changes. Even if any individual has the same reproductive possibilities and there are no differences between the organisms themselves or their living surroundings you see that 21,2% of the organisms has the half of the progeny in the F2 and that 17,7% has no descendants in the F2.

The Poisson distributed allele transfer

In **Table 4** are described the fortunes of the allelic variations. So how many of the unique alleles or possible gene variations are transferred on the average to the next 3 generations according to the continued Poisson like distributions. Pose an individual has on a locus the alleles a and b. The change on transfer of allele a by 1 descendant in F1 is 0,5. By 2 descendants, the replacement in neutral population dynamics, the transfer of allele a is on the average 2x0,5=1. This the same for allele b. So the parameters for the intensity of the transfer of one allele to the next generation in a neutral population are **Q** =1 **p**=1, **s**=1 en **r**=1. The distribution of the alleles thus is with Poisson intensity λ=1, so that in the F1 the proportion 1/e=0,368.. has been disappeared, also 1/e=0,368 occurs singular, the half of this 0,184 occurs in twofold, etc. Also at the transfer from F1 to F2 and the further generation there will be on the average 2 descendants and 1 allele in the distribution. To make the distribution for F2 and F3 you must for all the proportions, those in singular, in twofold etc from the former generation, separately calculate how their further Poisson distribution is, as indicated with the arrows. In this are

each of these proportions different distributed with the intensities **λ*=q**=1; **λ*=q**=2; **λ*=q**=3, etc. The total intensity μ of these 2nd; 3rd and further degrees Poisson distributions remains 1 in **Table 4.** For instance in the 2nd distribution, F1→F2, the total intensity, the sum of all the different λ*, so **μ**= 0x(e^-1) + 1x(e^-1) + 2x[½(e^-1)] + 3x[1/6(e^-1)] + .. The sums of all these distribution form than the distribution for the new generation, always in proportions of the alleles in F0. Because the total intensity here keeps constant **μ**=1, is valid **λ=μ=ς=1.** Notice that **only the sums under ∑0**, the proportions of alleles that disappear in the further generations **can be expressed as a function of** e. The cause of this is that the Poisson distribution with the formula **P(i) = e^-λ . λ ^i/i!** gets the particularity of the complementary exponential distribution, if i=0 than P(i)= **e^-λ.** So in the F3 62,6..% of the singular alleles in F0 has been vanished. In this continuing distribution with intensity 1 there are of course as many alleles disappeared as there have come alleles to in the two folds, three folds etc, so that the total number of the alleles in all the generations is equal. The number of the alleles in the F0, 1x2n, is of course equal to the number of the alleles in the F3, so 2n x (0 x 0,626.. + 1 x 0,122 + 2 x 0,134 etc).

Table 4

F0									
Q=1 λ=1									
F1									
1→0	1→1	1→2	1→3	1→4	1→5	1→6	1→7	1→8	1→9
e^-1	e^-1	1/2.e^-1	1/6.e^-1	1/24.e^-1	1/120.e^-1	1/720.e^-1	1/5040.e^-1	1/40320.e^-1	2,76.10^-6 e^-1
F2	μ=1								
→0→0	→0→1	→0→2	→0→3	→0→4	→0→5	→0→6	→0→7	→0→8	→0→9
e^-1	0	0	0	0	0	0	0	0	0
→1→0	→1→1	→1→2	→1→3	→1→4	→1→5	→1→6	→1→7	→1→8	→1→9
e^-2	e^-2	1/2.e^-2	1/6.e^-2	1/24.e^-2	1/120.e^-2	1/720.e^-2	1/5040e^-2	1/40320.e^-2	2,76 10^-6.e^-2
→2→0	→2→1	→2→2	→2→3	→2→4	→2→5	→2→6	→2→7	→2→8	→2→9
1/2.e^-3	e^-3	e^-3	2/3.e^-3	1/3.e^-3	2/15.e^-3	2/45.e^-3	4/315.e^-3	1/315.e^-3	7,06.10^e^--4
→3→0	→3→1	→3→2	→3→3	→3→4	→3→5	→3→6	→3→7	→3→8	→3→9
1/6.e^-4	1/2.e^-4	3/4.e^-4	3/4.e^-4	9/16.e^-4	27/80.e^-4	81/480.e^-4	81/1120.e^-4	243/8960.e^-4	9,04.10^-4e^-4
→4→0	→4→1	→4→2	→4→3	→4→4	→4→5	→4→6	→4→7	→4→8	→4→9
1/24e^-5	1/6.e^-5	1/3.e^-5	4/9.e^-5	4/9.e^-5	16/45.e^-5	32/135.e^-5	128/945.e^-5	64/945.e^-5	0,0301e^-5
→5→0	→5→1	→5→2	→5→3	→5→4	→5→5	→5→6	→5→7	→5→8	→5→9
1/120.e^-6	1/24.e^-6	5/48e^-6	25/144.e^-6	125/576.e^-6	125/576.e^-6	0,18084e^-6	0,12618e^-6	0,08073e^-6	0,04485e^-6

→6→0	→6→1	→6→2	→6→3	→6→4	→6→5	→6→6	→6→7	→6→8	→6→9
1/720.e^-7	1/120.e^-7	1/40.e^-7	1/20.e^-7	3/40/.e^-7	0,09.e^-7	0,09.e^-7	27/350.e^-7	81/1400.e^-7	0,03857.e^-7
→7→0	→7→1	→7→2	→7→3	→7→4	→7→5	→7→6	→7→7	→7→8	→7→9
1/5040.e^-8	1/720.e^-8	7/1440.e^-8	0,01134.e^-8	0,01985.e^-8	0,02779e^-8	0,03242e^-8	0,03242e^-8	0,02837e^-8	0,02206.e^-8
→8→0	→8→1	→8→2	→8→3	→8→4	→8→5	→8→6	→8→7	→8→8	→8→9
1/40320e^-9	1/5040e^-9	1/1260e^-9	2/945e^-9	0,00433e^-9	0,00677e^-9	0,00903e^-9	0,01032e^-9	0,01031e^-9	0,00917e^-9
∑ 0 F2	∑ 1 F2	∑ 2 F2	∑ 3 F2	∑ 4 F2	∑ 5 F2	∑ 6 F2	∑ 7 F2	∑ 8 F2	∑ 9 F2
0,163584164	0,195514535	0,13372015	0,07295863	0,036145345	0,016973463	0,007630948	0,003299023	0,001378136	0,000357693
as e function	as e function								
[e^(e^-1) 1]/e	e^(1/e-2)								
∑ 0 F0 - F2	or e^(1/e)/e^2								
0,53146305									
≈e^(1/e-1)									
or [e^(e^-1)]/e									

F3

→0→0	→0→1	→0→2	→0→3	→0→4	→0→5	→0→6	→0→7	→0→8	→0→9	→0→10
0,53146305	0	0	0	0	0	0	0	0	0	0
→1→0	→1→1	→1→2	→1→3	→1→4	→1→5	→1→6	→1→7	→1→8	→1→9	→1→10
0,07192577	0,07192577	0,035962888	0,011987629	0,002996907	0,000599381	0,000099896	1,43E-05	1,78E-06	1,98E-07	1,98E-08
→2→0	→2→1	→2→2	→2→3	→2→4	→2→5	→2→6	→2→7	→2→8	→2→9	→2→10
0,018097054	0,036194108	0,036194108	0,024129405	0,012064702	0,004825881	0,001608271	0,000459608	0,000114902	2,55E-05	5,11E-06
→3→0	→3→1	→3→2	→3→3	→3→4	→3→5	→3→6	→3→7	→3→8	→3→9	→3→10
0,00363234	0,010897188	0,016345783	0,016345783	0,012259337	0,007355603	0,003677801	0,001576201	0,000591075	0,000197025	5,91E-05
→4→0	→4→1	→4→2	→4→3	→4→4	→4→5	→4→6	→4→7	→4→8	→4→9	→4→10
0,000662025	0,0026481	0,005296201	0,007061601	0,007061601	0,00564928	0,003766187	0,002152107	0,001076054	0,000478246	0,000191298
→5→0	→5→1	→5→2	→5→3	→5→4	→5→5	→5→6	→5→7	→5→8	→5→9	→5→10
0,000114366	0,000578315	0,001429579	0,002382631	0,002978289	0,002978289	0,002481907	0,001772791	0,001107994	0,000615552	0,000307776
→6→0	→6→1	→6→2	→6→3	→6→4	→6→5	→6→6	→6→7	→6→8	→6→9	→6→10
1,89E-05	0,000113491	0,000340474	0,000680948	0,001021422	0,001225707	0,001225707	0,001050606	0,000787954	0,000525303	0,000315182
	→7→1	→7→2	→7→3	→7→4	→7→5	→7→6	→7→7	→7→8	→7→9	→7→10
	2,11E-05	7,37E-05	0,000171976	0,000300958	0,00042134	0,000491564	0,000491564	0,000430118	0,000334536	0,000234175
			→8→3	→8→4	→8→5	→8→6	→8→7	→8→8	→8→9	→8→10
			3,95E-05	7,89E-05	0,000126242	0,000168323	0,000192369	0,000192369	0,000170995	0,000136796

				→9→4	→9→5	→9→6	→9→7	→9→8	→9→9	→9→10
				1,21E-05	2,17E-05	3,26E-05	4,19E-05	4,71E-05	4,71E-05	4,24E-05
∑0 F3	∑ 1 F3	∑ 2 F3	∑ 3 F3	∑ 4 F3	∑ 5 F3	∑ 6 F3	∑ 7 F3	∑ 8 F3	∑ 9 F3	∑ 10 F3
0,09445047	0,122378031	0,095642736	0,062799424	0,038774185	0,023203445	0,013552238	0,007751407	0,004349379	0,002394518	0,001291877
∑0 F0-F3										
0,625917694										∑ 11 F3
≈e^[e^(1/e-1)-1]										6,88 E-4

The number of the events as “arrivals” of descendants and genes in generation F1 is Poisson distributed with the known primary Poisson distribution. This means a distribution of the primary quantum Q with the uniform intensity λ into proportions for the quanta i=0; i=1; i=2; i=3, etc, so that the distribution results in quanta and proportions. The result of the former distribution, these proportion can of course be distributed Poisson again. Then however the proportions must be distributed separately each with its own intensity **λ*=q.μ**, so the product of the quantum **q** of the proportion in the former generation and **μ**. In this way also in the further generations the arrivals of the alleles remain to be Poisson distributed in the same generation time **t** and this distribution can be calculated by the same substitution in the formula **P(i) = e^-λ*. λ*^i/i!** through the generations. So the proportions of the old generations are always distributed into the new generations. In this way the 2nd degree Poisson distribution arises out of the general known primary distribution, the 3rd degree out of the 2nd degree and the nth degree out of the (n-1)th degree Poisson distribution. These further distribution all originate from the normal primary Poisson distribution with the uniform intensity **λ**. I do call these 2nd , 3rd and further degree Poisson distributions, because the same primary quantum Q is distributed here primary, secondary, tertiary and further. In Table 4 this happens with a constant total Poisson intensity **μ=λ**=1 for F(g-1)→Fg. The μ in this is the intensity in which all the proportions will decrease or increase in total at the distribution F(g-1)→ Fg. The μ is the proportional total intensity of the degree g, so that: **μ=0x[P(i=0)] + 1x[P(i=1)] + 2x[P(i=2)] + …qx[P(i=q)],** in this is **P(i=q)** the result of the distribution according to **P(i) = e^-λ*. λ*^i/i!** of degree g-1. This μ of the continued Poisson distribution is constant in these examples, but the Poisson distribution of the quanta can also be continued in the next degree with another intensity. The calculation of a large number of degrees are easily practicable, I guess, with a computer and the right software, but not in this way.

So there are in the graduated Poisson distributions levels of quantities and the distributions are from the former to the next

level of the quantities. In this application the levels of the quanta are called the generations F0; F1; F2;..Fg. The degrees of the Poisson distributions are between these levels or generations, so that degree Gg distributes the quanta of Fg into those of F(g-1). See on **Table 5**.

The accumulating exponential distribution.
The peculiarity of the P(i=0), this is the negation or the complement of the Poisson event or arrival, the zero-proportion is exponential distributed, according to **P(i=0)** = e^-**λ** at the primary and further Poisson distribution and it is the complement of the exponential distribution, **P(i=n) = 1 -** e^-**λ**. The intensity λ of these exponential distributions is also within the next degrees equal to the **μ** of the continued Poisson distribution, of which it is a part. With **λ*=μ.q** and the quanta **q** the P(i=0) can be calculated with the superposed Poisson distributions in the way of Table 3 and 4. If you express than the P(i=0) as an algebraic function of e it just appears that the remaining quantity, so 1-P(i=0) just is negative exponential distributed through the degrees or generations. So P(i=0) of generation g simply is e^{1-P(i=0)} of the former generation g-1. The exponential distribution of the non arrival accumulates in this way. Through the generations is the intensity **λ** or **σ** of the exponential distribution equal to the remaining quantity and decreases, while the non arrival, the extinction of the allele increases. In this is **λ** the intensity of the primary distribution and is **σ** the accumulated intensity of the distributions in the further degrees. The superposition of the exponential part of the Poisson distribution can be calculated in following the recurrence and this is noticed in **-σ(Fg)=ν-1.** In this is **σ** the accumulated intensity of generation Fg and **ν** is the P(i=0) or the extinction accumulated up to the former generation F(g-1). The extinction for generation g than is P(i=0)=e^**(ν-1**). This is the most convenient formula for making the recurrence table, as given here on **Table 5**. This is easy to be done with a simple calculator. The allele survival in the generations or degrees of the exponential distribution goes as an exponential ladder with the steps t, so t1=1 t2=1-e^-1 t3=1-e^-(1-e^-1) t4=1-e^-[1-e^-(1-e^-1)] t5=1-e^-{1-e^-[1-e^-(1-e^-1)]}, etc.

In Table 5 the extinct alleles, the P(∑i=0), shortly P0, is calculated from the intensities **λ**, or **σ** for the generations F0-F200, starting from **λ=μ**=1.

Table 5

F0	F1	F2	F3	F4	F5	F6	F7	F8	F9
λ=1	σ=0,6321	σ=0,4685	σ=0,3741	σ=0,3121	σ=0,2681	σ=0,2352	σ=0,2095	σ=0,1890	σ=0,1723
P0=0,368	P0=0,531	P0=0,626	P0=0,688	P0=0,732	P0=0,765	P0=0,790	P0=0,811	P0=0,828	P0=0,842
F1	F2	F3	F4	F5	F6	F7	F8	F9	F10

F10	F11	F12	F13	F14	F15	F16	F17	F18	F19
σ=0,1582	σ=0,1464	σ=0,1361	σ=0,1273	σ=0,1195	σ=0,1127	σ=0,1066	σ=0,1011	σ=0,0961	σ=0,0916
P0=0,854	P0=0,864	P0=0,873	P0=0,880	P0=0,887	P0=0,893	P0=0,899	P0=0,904	P0=0,908	P0=0,912
F11	F12	F13	F14	F15	F16	F17	F18	F19	F20

F20	F21	F22	F23	F24	F25	F26	F27	F28	F29
σ=0,0876	σ=0,0838	σ=0,0804	σ=0,0773	σ=0,0744	σ=0,0716	σ=0,0692	σ=0,0668	σ=0,0646	σ=0,0626
P0=,0916	P0=0,919	P0=0,923	P0=0,926	P0=0,928	P0=0,931	P0=0,933	P0=0,935	P0=0,937	P0=0,939
F21	F22	F23	F24	F25	F26	F27	F28	F29	F30

F30	F31	F32	F33	F34	F35	F36	F37	F38	F39
σ=0,0607	σ=0,0589	σ=0,0572	σ=0,0556	σ=0,0541	σ=0,0526	σ=0,0513	σ=0,0500	σ=0,0487	σ=0,0476
P0=0,941	P0=0,943	P0=0,944	P0=0,945	P0=0,947	P0=0,949	P0=0,950	P0=0,951	P0=0,952	P0=0,954
F31	F32	F33	F34	F35	F36	F37	F38	F39	F40

F40	F41	F42	F43	F44	F45	F46	F47	F48	F49
σ=0,0465	σ=0,0454	σ=0,0444	σ=0,0434	σ=0,0425	σ=0,0416	σ=0,0407	σ=0,0399	σ=0,0391	σ=0,0384
P0=0,955	P0=0,956	P0=0,957	P0=0,958	P0=0,958	P0=0,959	P0=0,960	P0=0,961	P0=0,962	P0=0,962
F41	F42	F43	F44	F45	F46	F47	F48	F49	F50

F50	F51	F52	F53	F54	F55	F56	F57	F58	F59
σ=0,0376	σ=0,0369	σ=0,0363	σ=0,0356	σ=0,0350	σ=0,0344	σ=0,0338	σ=0,0332	σ=0,0327	σ=0,0322
P0=0,963	P0=0,964	P0=0,964	P0=0,965	P0=0,966	P0=0,966	P0=0,967	P0=0,967	P0=0,968	P0=0,968
F51	F52	F53	F54	F55	F56	F57	F58	F59	F60

F60	F61	F62	F63	F64	F65	F66	F67	F68	F69
σ=0,0317	σ=0,0312	σ=0,0307	σ=0,0302	σ=0,0298	σ=0,0293	σ=0,0289	σ=0,0285	σ=0,0281	σ=0,0277
P0=0,969	P0=0,969	P0=0,970	P0=0,970	P0=0,971	P0=0,971	P0=0,972	P0=0,972	P0=0,972	P0=0,973
F61	F62	F63	F64	F65	F66	F67	F68	F69	F70

F70	F71	F72	F73	F74	F75	F76	F77	F78	F79
σ=0,0273	σ=0,0269	σ=0,0266	σ=0,0262	σ=0,0259	σ=0,0256	σ=0,0252	σ=0,0249	σ=0,0246	σ=0,0243
P0=0,973	P0=0,973	P0=0,974	P0=0,974	P0=0,974	P0=0,975	P0=0,975	P0=0,975	P0=0,976	P0=0,976
F71	F72	F73	F74	F75	F76	F77	F78	F79	F80
F80	F81	F82	F83	F84	F85	F86	F87	F88	F89
σ=0,0240	σ=0,0237	σ=0,0235	σ=0,0232	σ=0,0229	σ=0,0227	σ=0,0224	σ=0,0221	σ=0,0219	σ=0,0217
P0=0,976	P0=0,977	P0=0,977	P0=0,977	P0=0,977	P0=0.978	P0=0,978	P0=0,978	P0=0,978	P0=0,979
F81	F82	F83	F84	F85	F86	F87	F88	F89	F90
F90	F91	F92	F93	F94	F95	F96	F97	F98	F99
σ=o,0214	σ=0,0212	σ=0,0210	σ=0,0208	σ=0,0205	σ=0,0203	σ=0,0201	σ=0,0199	σ=0,0197	σ=0,0195
P0=0,979	P0=0,979	P0=0,979	P0=0,979	P0=0,980	P0=0,980	P0=0,980	P0=0,980	P0=0,980	P0=0,981
F91	F92	F93	F94	F95	F96	F97	F98	F99	F100
F100	F101	F102	F103	F104	F105	F106	F107	F108	F109
σ=0,01935	σ=0,01917	σ=0,01898	σ=0,01881	σ=0,01863	σ=0,01854	σ=0,01829	σ=0,01812	σ=0,01796	σ=0,01780
P0=0,98083	P0=0,98102	P0=0,98119	P0=0,98137	P0=0,98154	P0=0,98171	P0=0,98188	P0=0,98204	P0=0,98220	P0=0,98236
F101	F102	F103	F104	F105	F106	F107	F108	F109	F110
F110	F111	F112	F113	F114	F115	F116	F117	F118	F119
σ=0,01764	σ=0,01749	σ=0,01733	σ=0,01718	σ=0,01704	σ=0,01689	σ=0,01675	σ=0,01661	σ=0,01647	σ=0,01634
P0=0.98251	P0=0,98267	P0=0,98281	P0=0,98296	P0=0,98311	P0=0,98325	P0=0,98339	P0=0,98352	P0=0,98366	P0=0,98379
F111	F112	F113	F114	F115	F116	F117	F118	F119	F120
F120	F121	F122	F123	F124	F125	F126	F127	F128	F129
σ=0,01621	σ=0,01608	σ=0,01595	σ=0,01582	σ=0,01570	σ=0,01557	σ=0,01545	σ=0,01533	σ=0,01522	σ=0,01510
P0=0,98392	P0=0,98405	P0=0,98418	P0=0,98430	P0=0,98443	P0=0,98455	P0=0,98467	P0=0,98478	P0=0,98490	P0=0,98501
F121	F122	F123	F124	F125	F126	F127	F128	F129	F130
F130	F131	F132	F133	F134	F135	F136	F137	F138	F139
σ=0,01499	σ=0,01488	σ=0,01477	σ=0,01466	σ=0,01455	σ=0,01445	σ=0,01434	σ=0,01424	σ=0,01414	σ=0,01404
P0=0,98512	P0=0,98523	P0=0,98534	P0=0,98545	P0=0,98555	P0=0,98566	P0=0,98576	P0=0,98586	P0=0,98596	P0=0,98606
F131	F132	F133	F134	F135	F136	F137	F138	F139	F140

F140	F141	F142	F143	F144	F145	F146	F147	F148	F149
σ=0,01394	σ=0,01385	σ=0,01375	σ=0,01366	σ=0,01356	σ=0,01347	σ=0,01338	σ=0,01329	σ=0,01320	σ=0,01312
P0=0,98615	P0=0,98625	P0=0,98634	P0=0,98644	P0=0,98653	P0=0,98662	P0=0,98671	P0=0,98680	P0=0,98688	P0=0,98697
F141	F142	F143	F144	F145	F146	F147	F148	F149	F150
F150	F151	F152	F153	F154	F155	F156	F157	F158	F159
σ=0,01303	σ=0,01295	σ=0,01286	σ=0,01278	σ=0,01270	σ=0,01262	σ=0,01254	σ=0,01246	σ=0,01238	σ=0,01230
P0=0,98705	P0=0,98714	P0=0,98722	P0=0,98730	P0=0,98738	P0=0,98746	P0=0,98754	P0=0,98762	P0=0,98769	P0=0,98777
F151	F152	F153	F154	F155	F156	F157	F158	F159	F160
F160	F161	F162	F163	F164	F165	F166	F167	F168	F169
σ=0,01223	σ=0,01216	σ=0,01208	σ=0,01201	σ=0,01194	σ=0,01187	σ=0,01180	σ=0,01173	σ=0,01166	σ=0,01159
P0=0,98784	P0=0,98792	P0=0,98799	P0=0,98806	P0=0,98813	P0=0,98820	P0=0,98827	P0=0,98834	P0=0,98841	P0=0,98847
F161	F162	F163	F164	F165	F166	F167	F168	F169	F170
F170	F171	F172	F173	F174	F175	F176	F177	F178	F179
σ=0,01153	σ=0,01146	σ=0,01139	σ=0,01133	σ=0,01126	σ=0,01120	σ=0,01114	σ=0,01108	σ=0,01102	σ=0,01096
P0=0,98854	P0=0,98861	P0=0,98867	P0=0,98873	P0=0,98880	P0=0,98886	P0=0,98892	P0=0,98898	P0=0,98904	P0=0,98910
F171	F172	F173	F174	F175	F176	F177	F178	F179	F180
F180	F181	F182	F183	F184	F185	F186	F187	F188	F189
σ=0,01090	σ=0,01084	σ=0,01078	σ=0,01072	σ=0,01066	σ=0,01061	σ=0,01055	σ=0,01050	σ=0,01044	σ=0,01039
P0=0,98916	P0=0,98922	P0=0,98928	P0=0,98934	P0=0,98939	P0=0,98945	P0=0,98950	P0=0,98956	P0=0,98961	P0=0,98967
F181	F182	F183	F184	F185	F186	F187	F188	F189	F190
F190	F191	F192	F193	F194	F195	F196	F197	F198	F199
σ=0,01033	σ=0,01028	σ=0,01023	σ=0,01018	σ=0,01012	σ=0,01007	σ=0,01002	σ=0,00997	σ=0,00992	σ=0,00987
P0=0,98972	P0=0,98977	P0=0,98982	P0=0,98987	P0=0,98993	P0=0,98998	P0=0,99003	P0=0,99008	P0=0,99013	P0=0,99018
F191	F192	F193	F194	F195	F196	F197	F198	F199	F200

If the population dynamics are not neutral.

If the population **dynamics**[7] are not neutral, because **p≠1** or/and **s≠1**, the population size or the number of the alleles will change with a constant factor **μ=p.s**. The population size and the alleles will increase exponential, if μ>1 and will decrease exponential, if μ<1. In this μ can have any value ≥0. Starting from single alleles in F0 (Q=1) the intensity of the primary Poisson and exponential distribution has the value λ=μ. Also for the further grades or generations the distributions can be found with λ*=qμ and μ=ps. If μ=2 the distributions for the next generations are found in the way of **Table 3**. That is because the absolute numbers of the alleles will increase in the generations with a factor 2 on the average and will be Poisson distributed as well. These calculations can also be continued with only the exponential part of the distribution. The accumulated exponential distribution is easy to be calculated with **–σ(Fg)=μν-μ**. In this is again **σ** the accumulated intensity of generation Fg and **ν** is the P(i=0) or the extinction accumulated up to the former generation F(g-1). The general intensity **μ** over the generations is determined by the parameters of the population dynamics. In the neutral population there is no change in size and no selection the general intensity **μ**=1. As show non **Table 5** the accumulating intensities **σ** of the of the exponential distribution are converging through the degrees or generations. If **μ**=1 the σ converges slowly from 1 to 0 in the increasing generations, while the P(∑i=0) converges from 0 to 1. If μ<1 converges the σ faster from the value of **μ**<1 to 0. If **μ**>1 the σ converges from the value **μ**>1 to a value >0. For instance if μ=2 the σ converges from 2 to 1,593624261.. in about 10 generations and so the extinction or the P(∑i=0) converges to 0,203187869…so ultimately 20,32% of the ancestors will have no descendants with two effective children on the average. If μ=1,1 the σ converges from 1,1 to 0,193747555.. and P(∑i=0) to 0,823865858.. The hypothetic selection or allele selection can be studied with s>1 or s<1 in **–σ(Fg)= sν-s**, but in general **–σ(Fg)=μν-μ**. The Table 5a show prominent differences in the transfer of the alleles through the generations at the different intensities **μ**. The same Table is described by Motoo **Kimura**[8]**.** However his deduction is different and he calculates the complement of the extinction, the probability of survival of the alleles. The application of the recurrence tables dates probably back to a publication of JBS Haldane from 1927, also **RA Fisher**[9] has published about it in 1922. Mostly it was thought however these extinction tables should be not relevant, because you could not calculate the fixation process of the alleles in general, in small or in limited populations. So more complicated calculations ware used by Kimuro, but I wonder if these can accurately describe the elementary simplicity of the concrete biologic substrate. I will emphasize

[7] Increase in population size has the same effect as positive selection on the **absolute** numbers of alleles. In this there is increase on specific alleles by the selection and on all the alleles by the increase in population size.
[8] Kimura: an introduction to population genetics theory blz 422
[9] On the dominance ratio blz 326

the simple theory of extinction by recurrence is general applicable also in small populations as pointed out at **Table 5c**. These **Tables 5, 5b and 5c** give a global impression of what you can call the theoretical or virtual selection. By **s**=**μ**=1 the allele is neutral, by s=μ=0,97 there is 0,03 negative selection and by s=μ=1,05 there is 0,05 positive selection. The problem however is there are in the concrete biologic reality no constant factors that determine the transfer of genetic variations. Selection is physical only present as differences in the effective reproduction. By that the intensities of the allele transfer are not constant but fluctuating and variable. Calculations can nevertheless be made of the allele transfer with fluctuations and variations in the intensity caused by selection at the reproduction of the individuals. **Table 8** gives a somewhat better impression of the reproduction selection on the allele transfer than does **Table 5** and **5b**, but the reality is of course yet much more differentiated. The primary Tables 5, 5b and those of Kimura are of essential importance, because the intensity **μ** can describe many different properties of the populations also random and non random inbreeding. See Table 9c and 5c.

Table 5b

F0	F4	F9	F14	F19	F49	F99	F199	F299	F399	F∞
μ=0,97	σ=0,28263	σ=0,1436	σ=0,0917	σ=0,0645	σ=0,0156	σ=0,0028	σ=0,000	σ=0,000	σ=0,000	σ=0,000
P0=0,37908	P0=0,75380	P0=0,8662	P0=0,9124	P0=0,9375	P0=0,9845	P0=0,9972	P0=1	P0=1	P0=1	P0=1
F1	F5	F10	F15	F20	F50	F100	F200	F300	F400	F∞
F0	F4	F9	F19	F49	F99	F199	F299	F399	F499	F∞
μ=1	σ=0,3121	σ=0,1723	σ=0,0916	σ=0,0384	σ=0,0195	σ=0,00987	σ=0,00652	σ=0,00491	σ=0,00391	σ=0
P0=0,368	P0=0,732	P0=0,842	P0=0,912	P0=0,962	P0=0,981	P0=0,99018	P0=0,9935	P0=0,9951	P0=0,9961	P0=1
F1	F5	F10	F20	F50	F100	F200	F300	F400	F500	F∞
F0	F4	F9	F19	F49	F99	F199	F299	F399	F499	F∞
μ=1,01	σ=0,32215	σ=0,18241	σ=0,10197	σ=0,04919	σ=0,03117	σ=0,02298	σ=0,02096	σ=0,02029	σ=0,02006	σ=0,01993
P0=0,36422	P0=0,72459	P0=0,83326	P0=0,90306	P0=0,95200	P0=0,96931	P0=0,97728	P0=0,97926	P0=0,97991	P0=0,98014	P0=0,98027
F1	F5	F10	F20	F50	F100	F200	F300	F400	F500	F∞
F0	F4	F9	F19	F49	F99	F199	F299	F399	F499	F∞

μ=1,03	σ=0,34270	σ=0,20364	σ=0,0,12451	σ=0,07544	σ=0,06238	σ=0,05955	σ=0,05941	σ=0,05941	σ=0,05941	σ=0,05941
P0=0,35701	P0=0,70985	P0=0,81576	P0=0,88293	P0=0,92734	P0=0,93953	P0=0,94219	P0=0,94232	P0=0,94232	P0=0,94232	P0=0,94232
F1	F5	F10	F20	F50	F100	F200	F300	F400	F500	F∞
F0	F4	F9	F14	F19	F29	F49	F99	F199	F499	F∞
μ=1,05	σ=0,36380	σ=0,22605	σ=0,178533	σ=0,14942	σ=0,12412	σ=0,10655	σ=0,09901	σ=0,09838	σ=0,09838	σ=0,09838
P0=0,34994	P0=0,69503	P0=0,79768	P0=0,83918	P0=0,86121	P0=0,88327	P0=0,89893	P0=0,90573	P0=0,90630	P0=0,90630	P0=0,90630
F1	F5	F10	F15	F20	F30	F50	F100	F200	F500	F∞
F0	F4	F9	F14	F19	F29	F49	F99	F199	F499	F∞
μ=1,1	σ=0,41874	σ=0,28672	σ=0,24130	σ=0,22020	σ=0,20283	σ=0,19496	σ=0,19374	σ=0,19374	σ=0,19374	σ=0,19374
P0=0,33287	P0=0,65787	P0=0,75072	P0=0,78560	P0=0,80236	P0=0,81642	P0=0,82287	P0=0,82387	P0=0,82387	P0=0,82387	P0=0,82387
F1	F5	F10	F15	F20	F30	F50	F100	F200	F500	F∞
F0	F2	F5	F10	F15	F19	F29	F49	F99	F499	F∞
μ=1,5	σ=1,03226	σ=0,90753	σ=0,87729	σ=0,87450	σ=0,87426	σ=0,87421	σ=0,87421	σ=0,87421	σ=0,87421	σ=0,87421
P0=0,22313	P0=0,35620	P0=0,40352	P0=0,41591	P0=0,41707	P0=0,41717	P0=0,41719	P0=0,41719	P0=0,41719	P0=0,41719	P0=0,41719
F1	F3	F6	F11	F16	F20	F30	F50	F100	F500	F∞
F0	F1	F2	F3	F4	F5	F6	F8	F12	F16	F∞
μ=2	σ=1,72933	σ=1,64519	σ=1,61405	σ=1,60184	σ=1,59695	σ=1,59497	σ=1,59385	σ=1,59363	σ=1,59363	σ=1,59363
P0=0,13534	P0=0,17740	P0=0,19298	P0=0,19908	P0=0,20153	P0=0,20251	P0=0,20291	P0=0,20314	P0=0,20319	P0=0,20319	P0=0,20319
F1	F2	F3	F4	F5	F6	F7	F9	F13	F17	F∞

The primary quantity Q.

The allele **a** or the ancestor in F0 has as the primary quantity Q. If Q is not 1 but 2; 3 or 1000, there are 2; 3 or 1000 distributions of the quantity 1, which is in the calculation the same as one distribution of the quantities 2;3 or 1000 in the classical exponential and Poisson distribution. This is not different in the exponential distribution through the degrees, but here is the quantity decreasing by the accumulating intensity. In fact: the essential quantity of the classical exponential distribution is always 1, because this distribution describes to be hit of not, and to this 1 may added or multiplied. According with **Table 5** the quantity 1 has been diminished from 1 to 0,00987 in the 200^{th} degree or generation and in general the

quantity 1 diminishes to a small value **u** after many generations. So if the Q ancestors or alleles should have all the same number of descendants or of alleles through the generations the combination of Q primary quantities should diminish from Q to 1-{(1-**u**)}^Q, so to nearly **u**Q for small values of **u**. However if the Q ancestors or alleles have the same changes, but none equality of events, they have Poisson distributed descendants and allele transference. This negative exponential transfer can be calculated from the recurrence ladder. So if the former generation (g-1) is calculated with **σ(g-1)=e^{ν(g-2)-1}** the average transfer to the generation g is calculated as **σ(g)=e^{Qν(g-1)-Q}**, so the primary quantity must be counted in the last step of the ladder. This is because the accumulating exponential distribution describes in fact only the recurrence of the quantity 1. It is yet essentially a qualitative distribution: to be hit or not to be hit. The different distribution(s) of 1 can nevertheless be added, multiplied or divided exponentially with this formula, of course after the calculation of the distributions it selves.

With the quantitative Poisson distribution you can calculate the superposition of the degrees in the distributions for different values of **Q** and **μ**. That is a tough job when you have only a calculator. I did make only a modest begin for Q=2 and μ=1 in 2 generations and only for the P(∑i=0) in 3 generations. This is shown on **Table 7**. If you will study the extinction you can use only the quantitative part of the Poisson distribution, the exponential one with is accumulation through the generations.

Table 7

F0									
F0→F1; Q=2; λ=2									
2→0	2→1	2→2	2→3	2→4	2→5	2→6	2→7	2→8	2→9
e^-2	2.e^-2	2.e^-2	4/3. e^-2	2/3 .e^-2	4/15e^-2	4/45e^-2	8/315e^-2	2/315e^-2	0,00141093e^-2
0,135342	0,27067	0,27067	0,18045	0,09022	0,03609	0,01203	0,00344	0,00086	0,00019
i=0 F1	i=1 F1	i=2 F1	i=3 F1	i=4 F1	i=5 F1	i=6 F1	i=7 F1	i=8 F1	i=9 F1
F1→F2; Q=2; μ=1									
→0→0	→0→1	→0→2	→0→3	→0→4	→0→5	→0→6	→0→7	→0→8	→0→9
0,135342	0	0	0	0	0	0	0	0	0
→1→0	→1→1	→1→2	→1→3	→1→4	→1→5	→1→6	→1→7	→1→8	→1→9
2.e^-3	2.e^-3	e^-3	1/3.e^-3	1/12.e^-3	1/60.e^-3	1/360.e^-3	1/2540.e^-3	1/20160.e^-3	5,5115E6.e^-3
2→0	2→1	2→2	2→3	2→4	2→5	2→6	2→7	2→8	2→9

2.e^-4	4. e^-4	4 e^-4	8/3 e^-4	4/3 e^-4	8/15 . e^-4	8/45 e^-4	16/315e^-4	4/315e^-4	0.0028219.e^-4
→3→0	→3→1	→3→2	→3→3	→3→4	→3→5	→3→6	→3→7	→3→8	→3→9
4/3.e^-5	4.e^-5	6.e^-5	6.e^-5	4,5.e^-5	2,7.e^-5	1,35.e^-5	0,578571.e^-5	0,21696.e^-5	0,072321.e^-5
→4→0	→4→1	→4→2	→4→3	→4→4	→4→5	→4→6	→4→7	→4→8	→4→9
2/3.e^-6	8/3.e^-6	16/3.e^-6	64/9.e^-6	64/9.e^-6	256/45.e^-6	512/135.e^-6	2048/945.e^-6	1,0836.e^-6	0.48156.e^-6
→5→0	→5→1	→5→2	→5→3	→5→4	→5→5	→5→6	→5→7	→5→8	→5→9
4/15.e^-7	4/3.e^-7	10/3.e^-7	50/9.e^-7	125/18.e^-7	125/18.e^-7	625/108.e^-7	4,1336.e^-7	2,5835.e^-7	1,43528.e^-7
→6→0	→6→1	→6→2	→6→3	→6→4	→6→5	→6→6	→6→7	→6→8	→6→9
4/45e^-8	8/15.e^-8	1,6.e^-8	3,2.e^-8	4,8.e^-8	5,76.e^-8	5,76.e^-8	4,93714.e^-8	3,70286.e^-8	2,46857.e^-8
→7→0	→7→1	→7→2	→7→3	→7→4	→7→5	→7→6	→7→7	→7→8	→7→9
8/315e^-9	8/45.e^-9	28/45.e^-9	196/135.e^-9	343/135.e^-9	2401/675.e^-9	4,14988.e^-9	4,14988.e^-9	3,63114,e^-9	2,82422.e^-9
→8→0	→8→1	→8→2	→8→3	→8→4	→8→5	→8→6	→8→7	→8→8	→8→9
2/315.e^-10	16/315.e^-10	64/315.e^-10	512/945.e^-10	1,0836.e^-10	1,73376.e^-10	2,31168.e^-10	2,64191.e^-10	2,64191.e^-10	2,34837.e^-10
∑i=0 F2	∑i=1 F2	∑i=2 F2	∑i=3 F2	∑i=4 F2	∑i=5 F2	∑1=6 F2	∑ i=7 F2	∑i=8 F2	∑i=9 F2
0,147118255	0,207817487	0,180359676	0,129835019	0,084822699	0,051674392	0,029717982	0,0162779	0.00854897	0,004325

∑i=0 F0-F2
0,282453538
als e function
≈e^(2/e-2)
F2→F3; Q=2; μ=1

→0→0					
0,282453538		0,02170.e^-5	0,00592.e^-5		
→1→0		→4→10	→4→11	→4→12	→4→13
0,07645178		0,192640.e^-6	0,07005.e^-6	0,02335.e^-6	0,00718.e^-6
2→0		→5→10	→5→11	→5→12	→5→13
0,024409027		0,71764.e^-7	0,3262.e^-7	0,13592.e^-7	0,05228.e^-7
→3→0		→6→10	→6→11	→6→12	
0,006464105		1,48114.e^-8	0,8079.e^-8	0,40395.e^-8	
→4→0					
0,000883308		1,9767.e^-9	1,25806.e^-9	0,73387.e^-9	
→5→0					
0,000348179		1,8787.e^-10	1,36632.e^-10	0,91088.e^-10	
→6→0		∑i=10 F2	∑i=11 F2	∑i=12 F2	∑i=13 F2
0,000073664		0,00210423	0,00099929	0,00044925	0,00006547
→7→0					

0,000014835
→8→0
0,000002868
∑1=0 F3
0,108647765
∑i=0 F0-F3
0,391101303
≈e^[2e^(1/e-1)-2]
≈2.e^(1/e-2)

The Poisson-exponential distribution continued in degrees is not only a law of the small numbers, because by the endless iteration also the large numbers come into the picture. The primary distribution of for instance Q=λ=100 is nearly symmetrical. The P(i=0) is yet very small, e^-100 and that is why this distribution is not to be discerned from a normal distribution. However if the distribution of the Q=100 is continued in the further degrees the P(i=o) will increase gradually. Such a large large superposed distribution is to be calculated with a computer and the right software. The calculation of the extinction only is much easier: In the data for the Table 5 was the not rounded value of σ(F198) = 9,922274724.10^-3, so the P(0) for F199 = e^-0,009922274724 = 0,990126788. Following **σ(g)=e^{Qv(g-1)-Q}** this x100 and afterwards -100 is -0,987321136. The is the accumulated intensity σ for the 200th degree in which Q=100 and λ=μ=1. So the P(0) on the 200th level here is e^-0,987321136 = 0,372573428. So the 100 alleles, present at the parent generation F0 all are extinct in 37,3% of the cases at the 200th generation. In this way it also to be calculated from the data that if Q=10 the σ of the F200 is 0,0982 so that the 10 alleles are extinct in 90,65% of the cases. So the trend is that after 2Q generations the total extinction is in a little more than e^-1 or 37% of the cases and after 20Q generations the extinction is in more thane^-0,1 or more than 90% of the cases total. So if there are 1000 alleles, not much in a large population, the extinction up to more than 90% should last as many as 20000 generations. This is not quite sure as far as these calculations, but evident is that for any positive small value of x $\left|[(e^{-x}) - 1]\right| < \left|x\right|$ is valid, so that the σ(Fg) of **table 5** converges to 0 by increasing g and any value of Q.

The exponential extinction in the Hardy-Weinberg population.
Because single and very rare alleles can disappear in one generation they are evidently not in equilibrium in the Hardy-

Weinberg population. Also large but finite quantities are not in equilibrium in the infinite large population and will ultimately disappear from it in a stochastically process. In an infinite large population the frequencies and quantities of the alleles are only constant if the quantities them selves are also infinite large. The limited quantities always will fluctuate by random sampling. If someone describes allele **frequencies** in an infinite H-W population, the **absolute quantities** of the alleles must also be infinite large in the H-W population. The frequencies of alleles with distinct finite quantities would of course always be zero in an infinite large population. So in allele frequencies in the H-W population, as often described in the literature, it always concerns infinite quantities. There are nevertheless also finite absolute quantities in an infinite large H-W population and there are stochastic processes describing the changes of these quantities. In this way is a quantity of for instance 10^6 is small in relation to ∞ and the superposed Poisson-exponential distribution shows how this quantity disappears ultimately.

If the population size or the selection fluctuates around a constant.
It generally occurs that populations on account of the ecologic equilibriums are about constant in size at the long term. By all kinds of changes in the life surroundings as droughts and epidemics the size of the population often fluctuates as well. Also the selection on fitness fluctuates mostly together to the fluctuating size. Favourable or fit properties are fitting in distinct life surroundings. So for instance in a time of great drought with low and more specific food offer other properties are favourable than in a time of abundance and so are in the course of the time always different circumstances that distinct the measure of the fitness of the genes and their combinations. For instance the varying occurring of different species of predators and competitors results also that sometimes these and than again the other properties are favourable for the survival. The selection also fluctuates by the linkage of the genes. Somewhat nearby on the same chromosome localized genes are transferred linked during many generations and are often only after long time independent of each other by the recombination. The linked genes are passed through the generations as bigger or smaller DNA fragments and the resultant of the different selective properties of all the genetic varieties (alleles) within such a fragment determines of course the selection factor for all the genes the fragment consists of. By the cutting and fixing of the recombination there are fluctuating linkages and so there is fluctuating selection on the distinct alleles. This means that alleles being selectively neutral on the basis of their own properties are ever lifted by their neighbours to positive and negative and alleles being positive them selves transfer temporally with neutral, negative or strong positive selection. The fluctuation in the selection of the alleles occurs generally and is so of great importance.

The accumulating distribution and extinction of the alleles is also fluctuating selection and population dynamics easily to be found with **–σ(Fg)=μν-μ**. In this is μ=ps, so that μ has alternating values by the fluctuating and the average value of it here is pointed out as λ= μ average. The extinction of the alleles for generation Fg than is **∑p(i=0)=e^-σ** for **(Fg-1→Fg).** The particularity of the recurrence is in this to be taken into account: the events should be calculated recurrent in the time. If the population changed in the first generation with the factor 0,5 and in the second with the factor 2; in the third again with 0,5 and in the forth 2, etc than the real historical chronology is turned. So at first −σF0=(2x0)-2=-2 and afterwards −σF1=0,5**(**e^−2**)**−0,5=−0,4323 than −σF2=2e^**[**0,5**(**e^-2**)**-0,5]-2=−0,7020 and at last −σF3=0,5e^{2e^**[**0,5**(**e^−2**)**-0,5]-2}-0,5=−0.2522. The P0 values of the extinction than are to be found with P(0)F1=e^(2x0−2)=0,1353 and P(0)F2=e^**[**0,5**(**e^−2**)**−0,5]=0,6490, etc. It is evident that in the calculations in this way the P0 values at the distribution to the uneven generations →F1; →F3, etc as showed on **Table 8a** in italics, are not the real values of the extinction for this generations. The calculation for this extinction again is turned. The extinction to the uneven generations can be found from the calculation of the values found in this way at the reverse phase. So the false values in italics **μ**1=1 and **μ**2=0,5 on Table 8a are the right values for the extinction to the uneven levels for **μ**1=0,5 en **μ**2=1. This is showed on **Table 8b** and **c**. On these tables it is evident that the extinction in the increasing phases are smaller than on the phases with deceasing population dynamics. In **Table 8d** are the accumulating intensities **σ** also shown like on **Table 5**. On the Table 8d are reproduced only the extinction values to the even levels, so after the total binary phases. The extinction values at the fluctuation between μ1 and μ2 can be compared with the average extinction according to the constant λ.

The proof for the correctness of these estimations can be found by the superposing and the full elaboration of the Poisson distributions. The superposed distributions for **μ**=2 and **μ**=0,5 are elaborated in a very restrictive measure in **Table 11**. The bold printed values in **table 8 b** and **c** are affirmed by **Table 11**. This by itself is definitely not a general deduced proof, but it is possible to produce unlimited much evidence, inductively and experimentally, by elaborating these distributions. Besides it may be easy for a mathematician to find the exact deductive proves by infinitesimal calculus, but I do work in a more philosophical way: In this view it is a consequence of the qualitative property of the exponential distribution. The exponential distribution according to **P(i=0) = e^-λ** and **P(i=n) = 1 - e^-λ** is in principle a distribution of the quantity 1, to be hit or not to be hit, so that the intensity λ should be always λ=1. The number n distributions of the quantity and intensity λ=1 gives in the calculations however the same result as 1 distribution with λ=n at the normal primary exponential distribution and this is much easier to work with. In the accumulated exponential distribution of the further degrees the principle difference between n distributions of λ=1 and 1 distribution of λ=n of also of practical importance. This induces a perpetual turning of the intensities as a consequence of the dialectical

negation. That is why the distribution on the one side with the intensity μ2 and on the other hand the exponential summation of the primary quanta Q, which is here μ1 as the intensity and also the result of the former distribution, alternate each other continuous in the fluctuation. In this is, as in the primary quant Q, at first is to be calculated the distribution with **μ**2 and afterwards follows the summation of **μ**1 of these distributions. In this elaboration are calculated only the real values after the total binary phase of the fluctuation, because the values for the uneven levels in **Table 8a** are the distributions of the even levels before (and without) the summations. The complement of this, the alternate phase than appears to give the right data for the uneven levels This again is the dialectic mirror.

So by the fluctuation between the intensity **μ**1 and **μ**2 in the extinction of the neutral allele (λ=1) there is no change in the point of convergence, so that the extinction remains complete. However if the fluctuation begins with a decrease in the population the extinction proceeds faster and if it begins with an increase, thus in the rising phase, it proceeds slower than the constant extinction. The point of convergence of the extinction and so the chance on fixation however is changed by the theoretical positive allele (λ>1, because ps>1). The change of fixation is smaller by start in the sinking phase and larger by start in the rising phase than in the constant population. So new mutations have a larger change to be absorbed by the population if they initiate during a phase with increasing population or with temporal positive selection. The problem is however that we often do not know which phase is primary. Sometimes we have an impression of it: natural populations do have some decided size in the ecologic equilibrium, which than is repeatedly broken by "catastrophes". This kind of bottleneck situations do indicate lower minima in the population size and thus on a start in the sinking phase. This distinction is more difficult by the fluctuating selection and at the non poissonic parities the phases are in fact at the same time. So than we do have in fact than 2 or more points of convergence and fixation by the fluctuation cause by the non poissonic parities.

In general a population grows exponential, with a constant factor, until limitations come from internal (physiological) or external (ecological) factors. The increase is decelerating by this and the population comes at last in equilibrium with its limitations. That is why the growing curve of for instance a bacterial colony in the laboratory has a s-form aspect. In the nature there are yet often changes in the external factors, occurring as disasters like droughts, inundations, epidemics etc. The growth of the population often is snapped off in the phase with the constant growing factor and the population decreases than sharply in short time. The curve of the turning size of the population has by this probably often a sewing teeth aspect. Starting from the minimal population size the points are upwards with two dual phases like this:  . The sew must be

turned horizontal and vertical if you start from the maximum population size, like  . One dual phase with start from the about average population size looks like  . This is the most probable situation at the varying population size in the actual nature. However the transfer of the descendants and alleles is decided by the smallest population size and so the minimal population size with  and start in the rising phase is the real starting point for the theory of the neutral population dynamics. In **Table 8e** the extinction is calculated in an unlimited population with fluctuation **μ**1=100 and **μ**2=0,01. At the top of **Table 8e** are again the unreal calculation values in italics. In the first two columns of **Table 8e** this fluctuation is symmetric. In this the increase and decrease with factors respectively **μ**1=100 and **μ**2=0,01 are each within one generation, so there is fluctuation around the average **λ**=1. In the following 11 columns of **Table 8e** the increase is divided over 10 generations each with **μ**1=1,585 so that the total increase also is **ς**1=1,585^10≈100. The fluctuations in the allele transfer as a result of the variations in the population size do have probably often this sewing teeth aspect of **Table 8e**. Fluctuations by variations in the selection will have mostly a more symmetrical aspect. The asymmetrical fluctuations are important because the population has an increased capacity by it to absorb mutations in the prolonged rising phases. This also is shown on **Table 8**e, compared with the constant extinction of **Table 5.**

The importance of the fluctuation is getting even more important owing to non Poisson distributed reproduction. As pointed out the parities often are not Poisson distributed this is partly or total caused by the non random selection. These larger varieties in the parities can however also (partly or total) be caused by random differences. These random differences in the parities can be approximated as extinction with fluctuating intensity. So the expectation is that the fluctuation of the intensity occurs very general in natural population by a combination of different causes. **The selection introduces itself as the resultant of a lot of fluctuations and variations**.

Tabel 8a

F0 λ=1	F1; P0=	F2	F3	F4	F5	F6	F7	F8	F9	F10
μ1=2 P0=0		0,455236288		0,620721229		0,70787681		0,76201347		0,799017493
μ2=0,5	*0,606530659*		*0,761563398*		*0,827257401*		*0,864104477*		*0,88781378*	

F0 λ=1	F1	F2	F3	F4	F5	F6	F7	F8	F9	F10
μ1=0,5	*0,135335283*		*0,495586824*		*0,640291569*		*0,719487576*		*0,769719626*	
μ2=2 P0=0		0,648993642		0,777084185		0,835391989		0,869135523		0,891241194

Tabel 8b

F0 λ=1	F1	F2	F3	F4	F5	F6	F7	F8	F9	F10
μ1=2	**0,135335283**		0,495586824		0,640291569		0,719487576		0,769719626	
μ2=0,5 **P0=0**		**0,455236288**		0,620721229		0,70787681		0,76201347		0,799017493

F11	F12	F13	F14	F15	F16	F17	F18	F19	F20	F21
0,804513437		0,830077154		0,849672275		0,865180565		0,877765529		0,888185996
	0,825955852		0,846465125		0,862612599		0,875662272		0,886431324	

F22	F23	F24	F25	F26	F27	F28	F29	F30	F31	F32
	0,896958347		0,904446376		0,910913877		0,916556861		0,921524046	
0,895471941		0,903170885		0,909807246		0,91558754		0,9206679		0,925168586

Tabel 8c

F0 λ=1	F1	F2	F3	F4	F5	F6	F7	F8	F9	F10
μ1=0,5 **P0=0**		**0,648993642**		**0,777084185**		0,835391989		0,869135523		0,891241194
μ2=2	**0,606530659**		**0,761563398**		0,827257401		0,864104477		0,88781378	

F11	F12	F13	F14	F15	F16	F17	F18	F19	F20	F21
	0,906881687		0,918547718		0,927591476		0,934812114		0,940712948	
0,904393022		0,916656861		0,926105205		0,933612601		0,939724187		0,944797804

F22	F23	F24	F25	F26	F27	F28	F29	F30	F31	F32
0,945627073		0,949783869		0,953346538		0,956434414		0,959136796		0,961521862
	0,949078249		0,95273874		0,95590535		0,958672053		0,961110348	

Tabel 8d

F1	F3	F5	F7	F9	F11	F13	F15	F17	F19	λ=1
σ=0,6321	σ=0,3741	σ=0,2681	σ=0,2095	σ=0,1723	σ=0,1464	σ=0,1273	σ=0,1127	σ=0,1011	σ=0,0916	μ1=1
P0=0,531	P0=0,688	P0=0,765	P0=0,811	P0=0,842	P0=0,864	P0=0,880	P0=0,893	P0=0,904	P0=0,912	μ2=1
F2	F4	F6	F8	F10	F12	F14	F16	F18	F20	

F1	F3	F5	F7	F9	F11	F13	F15	F17	F19	
σ=0,4323	σ=0,2522	σ=0,1799	σ=0,1403	σ=0,1151	σ=0,0977	σ=0,0850	σ=0,0752	σ=0,0674	σ=0,0611	λ=1
P0=0,649	P0=0,777	P0=0,835	P0=0,869	P0=0,891	P0=0,907	P0=0,919	P0=0,928	P0=0,935	P0=0,941	μ1=0,5
F2	F4	F6	F8	F10	F12	F14	F16	F18	F20	μ2=2

F1	F3	F5	F7	F9	F11	F13	F15	F17	F19	
σ=0,7869	σ=0,4768	σ=0,3455	σ=0,2718	σ=0,2244	σ=0,1913	σ=0,1667	σ=0,1478	σ=0,1328	σ=0,1206	λ=1
P0=0,455	P0=0,621	P0=0,708	P0=762	P0=0,799	P0=0,826	P0=0,846	P0=0,863	P0=0,876	P0=0,886	μ1=2
F2	F4	F6	F8	F10	F12	F14	F16	F18	F20	μ2=0,5

F1	F3	F5	F7	F9	F11	F13	F15	F17	F19	
σ=1,165	σ=0,9657	σ=0,9075	σ=0,8869	σ=0,8791	σ=0,8761	σ=0,8749	σ=0,8745	σ=0,8743	σ=0,8743	λ=1,5
P0=0,311	P0=0,381	P0=0,404	P0=0,412	P0=0,415	P0=0,416	P0=0,417	P0=0,417	P0=0,417	P0=0,417	μ1=1,5
F2	F4	F6	F8	F10	F12	F14	F16	F18	F20	μ2=1,5

F1	F3	F5	F7	F9	F11	F13	F15	F17	F19	
σ=1,5829	σ=1,3469	σ=1,2777	σ=1,2535	σ=1,2445	σ=1,2411	σ=1,2398	σ=1,2393	σ=1.2392	σ=1,2391	λ=1,5
P0=0,205	P0=0,260	P0=0,279	P0=0,286	P0=0,288	P0=0,289	P0=0,289	P0=0,290	P0=0,290	P0=0,290	μ1=0,75
F2	F4	F6	F8	F10	F12	F14	F16	F18	F20	μ2=3

F1	F3	F5	F7	F9	F11	F13	F15	F17	F19	
σ=0,7127	σ=0,5874	σ=0,5522	σ=0,5400	σ=0,5355	σ=0,5338	σ=0,5332	σ=0,5329	σ=0,5328	σ=0,5328	λ=1,5
P0=0,490	P0=0,556	P0=0,576	P0=0,583	P0=0,585	P0=0,586	P0=0,587	P0=0,587	P0=0,587	P0=0,587	μ1=3
F2	F4	F6	F8	F10	F12	F14	F16	F18	F20	μ2=0,75

Tabel 8e

F0	F1	*F0*	*F1*	*F2*	*F3*	*F4*	*F5*	*F6*	*F7*	*F8*	*F9*	F11
σ=0,01	σ=0,995	*σ=0,01*	*σ=0,0158*	*σ=0,0248*	*σ=0,0388*	*σ=0,0604*	*σ=0,0928*	*σ=0,1405*	*σ=0,2078*	*σ=0,2974*	*σ=0,4077*	σ=0,5307
P0=0,990	PO=0,368	*P0=0,990*	*P0=0,984*	*P0=0976*	*P0=0,962*	*P0=0,941*	*P0=0,911*	*P0=0,869*	*P0=0,812*	*P0=0,743*	*P0=0,665*	P0=0,588
F1	F2	*F1*	*F2*	*F3*	*F4*	*F5*	*F6*	*F7*	*F8*	*F9*	*F10*	F12

F0	F1	F0	F1	F2	F3	F4	F5	F6	F7	F8	F9	F11
μ1=100	μ2=0,01	μ1=1,585	μ1=1,585	μ1=1,585	μ1=1,585	μ1=1,585	μ1=1,585	μ1=1,585	μ1=1,585	μ1=1,585	μ1=1,585	μ2=0,01

σ=100	σ=0,995	σ=1.585	σ=1,260	σ=1,135	σ=1,076	σ=1,044	σ=1,027	σ=1.018	σ=0,012	σ=1,009	σ=1,007	σ=0,5307
P0=0,000	PO=0,368	P0=0,205	P0=0,284	P0=0,321	P0=0,341	P0=0,351	P0=0,358	P0=0,361	P0=0,363	P0=0,365	P0=0,365	P0=0,588
F1	F2	F1	F2	F3	F4	F5	F6	F7	F8	F9	F10	F12

Discussion about Table 4 and the specified distributions.

Table 4 shows the transfer of the alleles to the next generation as one uniform process, but there may be two processes in the biologic reality. There is yet one distribution of the numbers of offspring among the parents, the parities, and the second distribution is the endowment (0 or 1) of the alleles from the parent to the individual offspring. **Table 9** describes the random Poisson distributed parities with the average of 2 children and superposed over this is the endowment of the alleles to the real children. The distribution of the alleles of the heterozygote parent of F0, with genotype ab, is calculated with the binomium (a+b)^q. By means of the arrows the superposition of the binomium is easily to understand. For instance: ¼ of the parents with 2 children does not transfer the allele a to F1, ½ transfers the allele a singular and ¼ transfers the allele a in twofold. The sums of the columns are products of e on behave of converging and it appears that the total distribution of the alleles over the offspring is a Poisson distribution with intensity the half of the intensity of the distribution of only the offspring. The sum of one divided by all the faculties, so 1+1+1/2+1/6+1/24.. etc is equal to e, so that e^−2(1+1+1/2+1/6+1/24.. etc)=e^-1. If the series begins with ½ and it goes further on with 1/2x1/1; x1/2; x1/3, etc. than the sum is ½e. If the series begins with 1/b, the sum is 1/b x e. More convenient notations are $\sum 1/n!=e$ and $b\sum(1/n!)=be$. So in the general the random allele transfer can be considered as one uniform process. In special situations however it may be better to be described as two processes. In **table 9a** is the same distribution of the parities as in **table 9**, but in this population is inbreeding with ***f***=0,2. The change that the parent pass any of their alleles to the next generation respectively do not pass their alleles is in out breeding populations equal on 0,5 and 0,5. Because the alleles of the partners are in this inbreeding population in 20% of the cases identical, these chances here are respectively 0,6 and 0,4. Yet the child receives besides the Mendelian 0,5 of allele a from its father here also 0,5x0,2=0,1 of allele a from its mother, so in total 0,6 of the cases allele a is transferred to the child and this is true for allele b as well. So the Poisson distribution is in **table 9a** superposed by an asymmetrical binomium based on (0,6+0,4)^q. The superposition is marked by the arrows and it appears now indeed that this asymmetrical binomium over the Poisson distribution with λ=2 results in a Poisson distribution with **λ**=1,2. This is also the case in general for other values of ***f*** according to the converging series $b\sum(1/n!)=be$.

You should possibly pose the statement **Table 9a** describes also the situation of selection for the allele that is selective advantageous with the factor **s**=1,2. This however is not true in the light of the physical biological events, because selective

differences in the endowment of the alleles to the real individuals in the reproduction population are impossible. The children always receive the **Mendelian** half of the alleles from their parents, so the endowment (0 or 1) of the alleles to the existent individuals is always at random. The only source of selective or non-random differences is the reproductive distribution of the parities. The real existing reproductive selection will cause many fluctuations and variation in the allele transfer, that result ultimately in the allele selection. How the allele selection is effected from the physical observable reproduction selection here is described only superficially and partly on the tables. The allele selection is nevertheless described in the classical theory as in **Table 5b** and **9a**, but it seems better to call this the theoretical or virtual allele selection.

Table 9

Descendants of F0 in F1

→0	→1	→2	→3	→4	→5	→6	→7	→8	→9
e^-2	2.e^-2	2.e^-2	4/3. e^-2	2/3 .e^-2	4/15e^-2	4/45e^-2	8/315e^-2	2/315e^-2	4/2835e^-2
0,135342	0,27067	0,27067	0,18045	0,09022	0,03609	0,01203	0,00344	0,00086	0,00019

→0n→0a	→0n→1a	→0n→2a	→0n→3a	→0n→4a	→0n→5a	→0n→6a	→0n→7a	→0n→8a	→0n→9a
e^-2	0	0	0	0	0	0	0	0	0
→1n→0a	→1n→1a	→1n→2a	→1n→3a	→1n→4a	→1n→5a	→1n→6a	→1n→7a	→1n→8a	→1n→9a
e^-2	e^-2	0	0	0	0	0	0	0	0
→2n→0a	→2n→1a	→2n→2a	→2n→3a	→2n→4a	→2n→5a	→2n→6a	→2n→7a	→2n→8a	→2n→9a
1/2.e^-2	e^-2	1/2.e^-2	0	0	0	0	0	0	0
→3n→0a	→3n→1a	→3n→2a	→3n→3a	→3n→4a	→3n→5a	→3n→6a	→3n→7a	→3n→8a	→3n→9a
1/6.e^-2	1/2.e^-2	1/2.e^-2	1/6.e^-2	0	0	0	0	0	0
→4n→0a	→4n→1a	→4n→2a	→4n→3a	→4n→4a	→4n→5a	→4n→6a	→4n→7a	→4n→8a	→4n→9a
1/24.e^-2	1/6.e^-2	1/4.e^-2	1/6.e^-2	1/24.e^-2	0	0	0	0	0
→5n→0a	→5n→1a	→5n→2a	→5n→3a	→5n→4a	→5n→5a	→5n→6a	→5n→7a	→5n→8a	→5n→9a
1/120.e^-2	1/24.e^-2	1/12.e^-2	1/12.e^-2	1/24.e^-2	1/120.e^-2	0	0	0	0
→6n→0a	→6n→1a	→6n→2a	→6n→3a	→6n→4a	→6n→5a	→6n→6a	→6n→7a	→6n→8a	→6n→9a
1/720e^-2	1/120e^-2	1/48e^-2	1/36.e^-2	1/48.e^-2	1/120e^-2	1/720e^-2	0	0	0
→7n→0a	→7n→1a	→7n→2a	→7n→3a	→7n→4a	→7n→5a	→7n→6a	→7n→7a	→7n→8a	→7n→9a
1/5040e^-2	1/720e^-2	1/240e^-2	1/144e^-2	1/144e^-2	1/240e^-2	1/720e^-2	1/5040e^-2	0	0

→8n→0a	→8n→1a	→8n→2a	→8n→3a	→8n→4a	→8n→5a	→8n→6a	→8n→7a	→8n→8a	→8n→9a
1/40320e^-2	1/5040e^-2	1/1440e^-2	1/720e^-2	1/576e^-2	1/720e^-2	1/1440e^-2	1/5040e^-2	1/40320e^-2	0
→9n→0a	→9n→1a	→9n→2a	→9n→3a	→9n→4a	→9n→5a	→9n→6a	→9n→7a	→9n→8a	→9n→9a
1/362880e^-2	1/40320e^-2	1/10080e^-2	1/4320e^-2	1/2880e^-2	1/2880e^-2	1/4320e^-2	1/10080e^-2	1/40320e^-2	1/362880e^-2
+…	+...	+...	+...	+...	+...	+...	+...	+...	+...
∑ 0a	∑ 1a	∑ 2a	∑ 3a	∑ 4a	∑ 5a	∑ 6a	∑ 7a	∑ 8a	∑ 9a
e^-1	e^-1	1/2e^-1	1/6e^-1	1/24e^-1	1/120e^-1	1/720e^-1	1/5040e^-1	1/40320e^-1	1/362880e^-1

Table 9a

→0	→1	→2	→3	→4	→5	→6	→7	→8	→9	
e^-2	2.e^-2	2.e^-2	4/3. e^-2	2/3 .e^-2	4/15e^-2	4/45e^-2	8/315e^-2	2/315e^-2	4/2835e^-2	
0,135342	0,27067	0,27067	0,18045	0,09022	0,03609	0,01203	0,00344	0,00086	0,00019	λ=2

→0n→0a	→0n→1a	→0n→2a	→0n→3a	→0n→4a	→0n→5a	→0n→6a	→0n→7a	→0n→8a	→0n→9a
e^-2	0	0	0	0	0	0	0	0	0
→1n→0a	→1n→1a	→1n→2a	→1n→3a	→1n→4a	→1n→5a	→1n→6a	→1n→7a	→1n→8a	→1n→9a
0,8e^-2	1,2e^-2	0	0	0	0	0	0	0	0
→2n→0a	→2n→1a	→2n→2a	→2n→3a	→2n→4a	→2n→5a	→2n→6a	→2n→7a	→2n→8a	→2n→9a
0,32e^-2	0,96e^-2	0,72e^-2	0	0	0	0	0	0	0
→3n→0a	→3n→1a	→3n→2a	→3n→3a	→3n→4a	→3n→5a	→3n→6a	→3n→7a	→3n→8a	→3n→9a
0,085333.e^-2	0,384.e^-2	0,576.e^-2	0,288.e^-2	0	0	0	0	0	0
→4n→0a	→4n→1a	→4n→2a	→4n→3a	→4n→4a	→4n→5a	→4n→6a	→4n→7a	→4n→8a	→4n→9a
0,017067.e^-2	0,1024.e^-2	0,2304.e^-2	0,2304.e^-2	0,0864.e^-2	0	0	0	0	0
→5n→0a	→5n→1a	→5n→2a	→5n→3a	→5n→4a	→5n→5a	→5n→6a	→5n→7a	→5n→8a	→5n→9a
0,002731.e^-2	0,02048.e^-2	0,06144.e^-2	0,09216.e^-2	0,06912.e^-2	0,020736.e^-2	0	0	0	0
→6n→0a	→6n→1a	→6n→2a	→6n→3a	→6n→4a	→6n→5a	→6n→6a	→6n→7a	→6n→8a	→6n→9a
0.000364.e^-2	0,0032768e^-2	0,012288.e^-2	0,024576.e^-2	0,027648.e^-2	0,016589.e^-2	0,004147.e^-2	0	0	0
→7n→0a	→7n→1a	→7n→2a	→7n→3a	→7n→4a	→7n→5a	→7n→6a	→7n→7a	→7n→8a	→7n→9a
4,161E-5.e^-2	0,000437.e^-2	0,001966.e^-2	0,004915e^-2	0,007373e^-2	0,006636e^-2	0,003318e^-2	0,000711e^-2	0	0
→8n→0a	→8n→1a	→8n→2a	→8n→3a	→8n→4a	→8n→5a	→8n→6a	→8n→7a	→8n→8a	→8n→9a
4,16E-6.e^-2	4,993E-5.e^-2	2,621E-4.e^-2	7,864E-4e^-2	0,001475.e^-2	0,001769e^-2	0,001327.e^-2	5,688E-4.e^-2	1,066E-4.e^-2	0
→9n→0a	→9n→1a	→9n→2a	→9n→3a	→9n→4a	→9n→5a	→9n→6a	→9n→7a	→9n→8a	→9n→9a

3,699E-7.e^-2	4,438E-6e^-2	2,996E-5e^-2	1,04877E-4e^-2	2,3593E-4e^-2	3,53894E-4e^-2	3,53894E-4e^-2	2,28773E-4e^-2	8,5314E-5e^-2	1,4219E-5e^-2	
+...	+...	+...	+...	+...	+...	+...	+...	+...	+...	
∑ 0a	∑ 1a	∑ 2a	∑ 3a	∑ 4a	∑ 5a	∑ 6a	∑ 7a	∑ 8a	∑ 9a	
2,2255756e^-2	2,6706482e^-2	1,602386e^-2	0,6409423e^-2	0,19223393e^-2	0,0460839e^-2	0,0091509e^-2	0,0001509e^-2	0,0001919e^-2	1,4219E-5e^-2	
0,3011989	0,3614329	0,2168594	0,086721	0,026016	0,0062368	0,0012384	0.000204163	2,597E-05	1,92E-06	population
0,30119	0,36143	0,21686	0,08674	0,02602	0,00625	0,00125	0,00021	3,20E-05	4,30E-06	λ=1,2

Superposition binomium on the non random reproduction

Also if the reproduction is not exact Poisson distributed, as is mostly the case in the practice of the nature because the changes are not total equal and the events are not totally at random in the time, the calculations with the specified distributions of **Table 9** should be used instead of the uniform distribution of **Table 4**. The endowment of the alleles (0 or 1) always is total at random, but the distribution of the reproduction - the parities over the parents - may be non random. As pointed out the non Poisson, or non random, distribution of the reproduction in natural populations can be observed and measured. If you want information over the transfer and distribution of the alleles in these populations you can superpose the binomium of the endowment of the alleles over the observed non Poisson distributed reproduction. It than is to be expected that the allele distribution is more like the Poisson distribution, because of the random endowment of the alleles. In the **Tables 9b** and **9c** is the distribution of the reproduction according to the measurement at first compared with the Poisson distribution. Further is the endowment of the alleles posed on these observed natural distributions. **Table 9b** concerns a population with a variety in the distribution of the reproduction smaller than the Poisson distribution does. In this is an excess in the parities with about the average number of children of the population and there is a shortage or equal in the extremes on **both** sides in relation to the Poisson distribution. The data of Table 9b are taken from the date of the US Census bureau. These are data of 7,2 million women from the American subpopulation: all the English speaking white women in the age of 40-44 years in 2004. So women that have accomplished their families to 99%. But they are data from the actual reproduction. In **Table 9c** the population has distribution with yet a larger variation than the Poisson does. Here is a shortage in the about average values and an excess in the extremes on both sides. This is a population of 72 parents that lived in the past in the Netherlands and got 220 children, between the years about 1740 until 1890. The 220 children are the effective reproduction. In fact the parents got much more children, but only these 220 got children of themselves. This

population is described on page…

Table 9b

WHITE ONLY, NOT HISPANIC			Women Ever Married		n = 7,2 E6				
Numbers of children par woman 40-44 jr			Average number of children par woman is 1,959						
0	1	2	3	4	5	6	7	8	
0,141	0,172	0,398	0,196	0,071	0,01425	0,00475	0,00156	0,00044	
Poisson λ=1,959									λ=1,959
0,141	0,27622	0,27056	0,17667	0,08653	0,04498	0,01107	0,0031	0,00076	
→0n→0a	→0n→1a	→0n→2a	→0n→3a	→0n→4a	→0n→5a	→0n→6a	→0n→7a	→0n→8a	
0,141									
→1n→0a	→1n→1a	→1n→2a	→1n→3a	→1n→4a	→1n→5a	→1n→6a	→1n→7a	→1n→8a	
0,086	0,086								
→2n→0a	→2n→1a	→2n→2a	→2n→3a	→2n→4a	→2n→5a	→2n→6a	→2n→7a	→2n→8a	
0,0995	0,199	0,0995							
→3n→0a	→3n→1a	→3n→2a	→3n→3a	→3n→4a	→3n→5a	→3n→6a	→3n→7a	→3n→8a	
0,0245	0,0735	0,0735	0,0245						
→4n→0a	→4n→1a	→4n→2a	→4n→3a	→4n→4a	→4n→5a	→4n→6a	→4n→7a	→4n→8a	
0,0044375	0,01775	0,026625	0,01775	0,0044375					
→5n→0a	→5n→1a	→5n→2a	→5n→3a	→5n→4a	→5n→5a	→5n→6a	→5n→7a	→5n→8a	
0,00045968	0,002298	0,0045968	0,00459677	0,00229839	0,00045968				
→6n→0a	→6n→1a	→6n→2a	→6n→3a	→6n→4a	→6n→5a	→6n→6a	→6n→7a	→6n→8a	
0,0000742	0,00044531	0,00111328	0,0014844	0,00111328	0,00044531	0,0000742			
→7n→0a	→7n→1a	→7n→2a	→7n→3a	→7n→4a	→7n→5a	→7n→6a	→7n→7a	→7n→8a	
1,21875E-05	0,00008531	0,0002559	0,00042656	0,00042656	0,0002559	0,00008531	1,21875E-05		
→8n→0a	→8n→1a	→8n→2a	→8n→3a	→8n→4a	→8n→5a	→8n→6a	→8n→7a	→8n→8a	
0,00000172	0,00001375	0,000048125	0,00009625	0,00012031	0,00009625	0,000048125	0,00001375	0,00000172	
∑ 0a	∑ 1a	∑ 2a	∑ 3a	∑ 4a	∑ 5a	∑ 6a	∑ 7a	∑ 8a	
0,351485	0,379092	0,205639	0,048853	0,008396	0,0012571	0,0002076	0,00002594	0,00000172	∑p=0,994957
Poisson λ=0,9759									∑a=0,978226
0,3755	0,3664	0,1795	0,0584	0,0142	0,00278	0,00045	0,00006	0,000008	

Table 9c

→0	→1	→2	→3	→4	→5	→6	→7	→8	→9
Poisson, λ=3,05556									
0,0471	0,14391	0,21986	0,22393	0,17106	0,10453	0,05324	0,02324	0,00888	0,00301
population n=72		Average 3,0555 children per parent							
→0	→1	→2	→3	→4	→5	→6	→7	→8	→9
0,09722	0,194444	0,22222	0,11111	0,06944	0,13889	0,08333	0,06944	0	0,01389
7/72	14/72	16/72	8/72	5/72	10/72	6/72	5/72	0	1/72
→0n→0a	→0n→1a	→0n→2a	→0n→3a	→0n→4a	→0n→5a	→0n→6a	→0n→7a	→0n→8a	→0n→9a
7/72	0	0	0	0					
→1n→0a	→1n→1a	→1n→2a	→1n→3a	→1n→4a	→1n→5a	→1n→6a	→1n→7a	→1n→8a	→1n→9a
7/72	7/72	0							
→2n→0a	→2n→1a	→2n→2a	→2n→3a	→2n→4a	→2n→5a	→2n→6a	→2n→7a	→2n→8a	→2n→9a
4/72	8/72	4/72	0						
→3n→0a	→3n→1a	→3n→2a	→3n→3a	→3n→4a	→3n→5a	→3n→6a	→3n→7a	→3n→8a	→3n→9a
1/72	3/72	3/72	1/72	0					
→4n→0a	→4n→1a	→4n→2a	→4n→3a	→4n→4a	→4n→5a	→4n→6a	→4n→7a	→4n→8a	→4n→9a
0,3125/72	1,25/72	1,875/72	1,25/72	0,3125/72	0				
→5n→0a	→5n→1a	→5n→2a	→5n→3a	→5n→4a	→5n→5a	→5n→6a	→5n→7a	→5n→8a	→5n→9a
0,3125/72	1,5625/72	3,125/72	3,125/72	1,5625/72	0,3125/72	0			
→6n→0a	→6n→1a	→6n→2a	→6n→3a	→6n→4a	→6n→5a	→6n→6a	→6n→7a	→6n→8a	→6n→9a
0,09375/72	0,5625/72	1,40625/72	1,875/72	1,406256/72	0,5625/72	0,09375/72	0		
→7n→0a	→7n→1a	→7n→2a	→7n→3a	→7n→4a	→7n→5a	→7n→6a	→7n→7a	→7n→8a	→7n→9a
5,42535E-4	3,79774E-3	0,01139332	0,0189887	0,0189887	0,01139332	3,79774E-3	5,42535E-4	0	
→8n→0a	→8n→1a	→8n→2a	→8n→3a	→8n→4a	→8n→5a	→8n→6a	→8n→7a	→8n→8a	→8n→9a
0	0	0	0	0	0	0	0	0	0
→9n→0a	→9n→1a	→9n→2a	→9n→3a	→9n→4a	→9n→5a	→9n→6a	→9n→7a	→9n→8a	→9n→9a
2,71267E-5	2,44141E-4	9,765625E-4	2,278646E-3	3,417969E-3	3,417969E-3	2,278646E-3	9,765625E-4	2,44141E-4	2,71267E-5
∑ 0a	∑ 1a	∑ 2a	∑ 3a	∑ 4a	∑ 5a	∑ 6a	∑ 7a	∑ 8a	∑9a
0,274442	0,300917	0,198568	0,121962	0,06798	0,026964	0,007378	0,001519	0,000244	0,000027
In this distribution ∑p=1 en ∑a=1,527775=3,05555x0,5									
Poisson λ=1,527778									
0,217017	0,331554	0,253271	0,12898	0,049263	0,015053	0,003833	0,000837	0,00016	0,000027

At **Table 9c1** is made another distribution with the historical population of 72 parents. The data are somewhat mutated, so that the parents now have in total 216 children, thus exact 3 on the average. Further is superposed for this calculation an asymmetric binomium over the distribution of the reproduction. The change the allele is transferred or not is posed here not on ex aequo ½ as in the **Tables 9** and **9c** but on respectively ⅓ yes and ⅔ no. So the binomium is (⅓+⅔)^q. The distribution further is described with the arrows. The result here is an hypothetic allele distribution with ∑p=1 and so the average intensity of the reproductive distribution with λ=3 is reversed into an allele distribution λ=1. So the allele distribution in this natural growing population is reversed into a population with constant size and the larger variation in the parities can now be compared with the neutral population dynamics by λ=1. In this natural population the distribution of the alleles can be regarded as a distribution with different Poisson intensities for any of the quanta (values of i=n) and these different Poisson intensities can also be calculated for comparison. So we see in the **tables 9c** and **9c1** a shift to smaller averages and larger extremes compared with the random distribution, but there is a limitation in the ultimate large values especially in **Table 9c1**.

Table 9c1

Poisson λ=3

→0	→1	→2	→3	→4	→5	→6	→7	→8	→9
0,04979	0,14361	0,22404	0,22404	0,16803	0,10082	0,05041	0,0216	0,0081	0,0027
→0	→1	→2	→3	→4	→5	→6	→7	→8	→9

Populatie n=72 gemiddeld 3,0 kinderen per ouder

0,09722	0,194444	0,22222	0,11111	0,06944	0,152778	0,08333	0,06944	0	0
7/72	14/72	16/72	8/72	5/72	11/72	6/72	5/72	0	
→0n→0a	→0n→1a	→0n→2a	→0n→3a	→0n→4a	→0n→5a	→0n→6a	→0n→7a		
7/72	0	0	0	0					
→1n→0a	→1n→1a	→1n→2a	→1n→3a	→1n→4a	→1n→5a	→1n→6a	→1n→7a		
7/54	7/108	0							
→2n→0a	→2n→1a	→2n→2a	→2n→3a	→2n→4a	→2n→5a	→2n→6a	→2n→7a		
0,098765432	0,098765432	0,024691358	0						
→3n→0a	→3n→1a	→3n→2a	→3n→3a	→3n→4a	→3n→5a	→3n→6a	→3n→7a		
0,03292181	0,049382716	0,024691358	0,004115226	0					
→4n→0a	→4n→1a	→4n→2a	→4n→3a	→4n→4a	→4n→5a	→4n→6a	→4n→7a		

0,013717421	0,027434842	0,020576131	0,006858711	0,000857339	0			
→5n→0a	→5n→1a	→5n→2a	→5n→3a	→5n→4a	→5n→5a	→5n→6a	→5n→7a	
0,020118884	0,05029721	0,05029721	0,025148605	0,006287151	0,000628715	0		
→6n→0a	→6n→1a	→6n→2a	→6n→3a	→6n→4a	→6n→5a	→6n→6a	→6n→7a	
0,007315958	0,021947873	0,027434842	0,018289894	0,006858711	0,001371742	0,000114312	0	
→7n→0a	→7n→1a	→7n→2a	→7n→3a	→7n→4a	→7n→5a	→7n→6a	→7n→7a	
0,004064421	0,014225473	0,02133821	0,017781842	0,008890921	0,002667276	0,000444546	3,17533E-05	0	0
∑ 0a	∑ 1a	∑ 2a	∑ 3a	∑ 4a	∑ 5a	∑ 6a	∑ 7a		
0,403755771	0,326868354	0,16902911	0,072194278	0,022894122	0,00466774	0,000558858	3,17533E-05	∑P=1	

Populatie; verdeling over de quanta met onderstaand de intensiteiten van de quanta.

λ=0,90694511	λ=0,5891883 λ=1,568	λ=0,9219	λ=1,087057	λ≈1,146831	λ≈1,112461	λ≈1,018122	λ≈0,871774	
0,36787944	0,36787944	0,18393972	0,06131324	0,0153283	0,00306566	0,000510944	0,000072992	Poiss λ=1
Poisson λ=1								

The **Table 1a; 1b**; **2 and 3** that describe random or in the case non random and than thus probably selective distributions of the reproduction can be converted in a simple way into allele distributions, as described in the **tables 9; 9b** and **9c(1)**. The differences between these **tables 9b** and **9c(1)** now are prompt an indication for an important trend. These simple data indicate namely the probability of a natural selection by people until the recent past, which is almost suddenly disappeared with the entrance with the modern industrial society. The existence of the natural selection in the past is however not proven by these data. It is yet obvious that the historical data of **table 2** and **9c(1)** are not representative for all the people of for instance the Netherlands of those years. All these people were from one family, that had at least one descendant in the 21st century describing this study. So there is bias and the population also is too small for conclusions. Much more data are to be analysed to make this study evident and this is quite possible. Many genealogical data are available to make these studies about the reproduction during some centuries by people of European descend and perhaps even over a longer time in countries with a much older culture like China. From what we experience globally about differences in chances on survival and reproduction in many human populations and in the nature it seems probable that further studies will affirm the general existence of the larger varieties, but more research of course is essential to scientific conclusions at modern criteria. The data of the **tables 1,1b** and **9b** make together with the other data from the US census bureau a very large representative population. Although these are data from the **actual** reproduction in the total **actual** population, it is obvious that the

effective reproduction within the reproductive population will give about the same distribution with a smaller variation than at random, but than by a somewhat smaller average parity. So these data give evidence for the concept there is no natural selection (anymore) in the modern American population. Selection is of course excluded if the variation in the numbers of children, parents have, is fewer or equal to random expected. More genetic differences between the generations than at random only are possible by larger variations in the parities.

Some other specific situations:

Asexual reproduction

Other conditions for Table 4 are diploid genes and sexual reproduction. By asexual reproduction the genes are transferred 1on 1 to the next generation. In this way of reproduction there is another reproductive population, or in other words another effective size of the population. The replacement ratio **r** is yet the ratio that defines the intensity of the reproduction en so describes at which average parity the population of reproduction and the total number of the alleles remains constant. The inverse 1/r indicates which part of their genome individuals pass to individuals of the next generation. Furthermore is by the asexual reproduction the distribution at the reproduction alone is decisive for the distribution at the allele transfer, because the endowment of the alleles to the children is 1 on 1 and so no random distribution is at the endowment here as it does contrary at the sexual reproduction. Now have the various species different ways of asexual reproduction. In accordance with this the distribution of the asexual reproduction will take shape different at the various species and the differences of these distributions with the Poisson distribution be larger in some cases than generally at the sexual **reproduction**[10]**.** This expectation however is not right in some cases. The unicellular organisms are for instance particular in this respect, because they always have two descendants in the actual reproduction. During the start of the growing phase of a bacterial colony there is an exponential increase in size for the actual reproduction as well as for the effective one In the phase with a constant number of the bacteria and an equilibrium between the bacteria that die off by internal and external factors and the bacteria that can split anymore is a Poisson distributed effective reproduction of the individuals. The condition for this is however that the bacteria all have the same change on dividing and survival. If the bacteria do have different changes on survival in the environment, as can happen after administration of antibiotics, they are different susceptible to, the variation

[10] So in those cases the asexual reproduction is more susceptible for selection than then the sexual one.

in the distribution of their effective reproduction will be larger than according to the Poisson distribution. So than there will be more genetic differences between the generations than Poisson expected.

By many plants is a combination of sexual and asexual reproduction. The aspect of the distribution of the total reproduction is more complicated by this. Specially by plants the asexual part of the reproduction induces generally augmentation the aspect of the total reproduction is complicated. In instance of this are the tuberous plants like the potatoes. Without asexual augmentation, because just one bulb in made generally, as in the tulip, you can regard this as a prolonged survival of the organism, reproducing only sexually. It is however to be taken into account that not only plants but also animals and even people do have the combination of sexual and asexual augmentation! Also the birth of uniovular twins, triplets etc yet is a form of asexual augmentation. The distribution of the descendants and the transferred alleles has been made different by this from the aspect of the exclusive sexual reproduction. Although these differences may be small, as in people, they are of fundamental importance.

Haploid genes

The distributions of Table 4 and 9 have of course also as a condition diploid (twofold) genes. Some genes however are haploid (singular) and are transferred 1 on 1 to the next generation. In many animal species and in people these are a (large) part of the genes on the Y-chromosome and the genes of the mitochondrial DNA. The genes on the Y-chromosome only are transferred from fathers to sons and the mt-DNA is only transferred through mothers. That is why the distribution of the transfer of these genes should be specified to the gender, so that only the masculine, respectively the feminine descendants are counted at the effective reproduction. The replacement factor here also is **r**=1 and for this part the whole genome is transferred to the next generation. Also here is no endowment of the alleles superposed upon the distribution of the reproduction. It is an important point that the transfer of the non recombinant alleles is in neutral population dynamics distributed with the Poisson intensity $\lambda=1$ with only source of variation in this the variation in the reproduction. As pointed out at Table 9 has the neutral allele transfer of the recombinant diploid genes the same intensity $\lambda=1$. At the recombinant genes this intensity $\lambda=1$ however is constructed of a part reproduction with intensity $\lambda=2$ and a part endowment of the alleles to the offspring. Only the first part of this, the reproduction, may have a non random distribution as a consequence of the selection, but the endowment always is at random and buffers the influence of the reproductive selection on the allele transfer. So it is obvious that the consequences of the selective differences are not buffered for the transfer of the non

recombinant haploid genes at the sexual reproduction. That is why the non recombinant genes are 2x as susceptible for selective differences than the diploid genes. So for general stochastic reasons the non recombinant genes will differ more in place and time through the populations than the “normal” recombinant genes. This furthermore is much more the case at the genes of the Y-chromosome than the mt-DNA on account of another reason: The alleles of the Y-chromosome will have of course an extra large variation in the transfer caused by the sexual selection in evolutionary populations. This is very important if you will use these genes for studying genetic changes in the whole genome.

Inbreeding

Inbreeding in the population is a complex datum. The most important reason for this properly is that the conception inbreeding is ill-defined in the literature. This seems curious and even incredible, because inbreeding is yet a simple idea, already known in the grey antiquity. The problems at the quantification of incest are however the differences in its forms and aspects, which are delicate and difficult to be defined. The aspect of the inbreeding does yet determine the size of the reproductive population and the effective reproduction. These aspect are: allele transfer in a defined population at an average inbreeding; allele transfer at the descendants of related parents; allele transfer at the descendants of the common ancestors of the related parents; allele transfer at lines of inbreeding with accumulating relationship between the parents, etc An essential aspect is the study of inbreeding at allele transfer out of the common ancestors. Study of inbreeding in this aspect results in the important conclusion that inbreeding is in the population genetics a hybrid of the sexual and the asexual reproduction. The ratio r, the replacement ratio, is in this aspect of inbreeding smaller than in out breeding. In inbreeding is yet a larger part of the whole genome transferred to the next and further generations than in out breeding. This implies also that the size of the population of the effective reproduction decreases in this aspect of inbreeding. An example as an illustration: In a family is a marriage between first cousins. The common grandparents of these partners will get on the average (**Table 3**) 2 children and 4 grandchildren in neutral population dynamics. In this situation however did 2 of these grandchildren become partners of each other and so have these grandparents now to expect 6 great-grandchildren on the average instead of the 8 in out breeding. The grandparents of these partners can on the other hand rejoice in the fact that they will pass their genes along two ways to two of their grandchildren. The great-grandchildren now will receive a double portion of genes from these great-grandparents and are in genetic respect practical grandchildren from them. The consequence of this is that the common ancestors do transfer in this example to al their descendants as many genes as in the out breeding. If all the common ancestors should have in this way fewer descendants in the reproductive population, but always should transfer

their alleles to these descendants equal to the out breeding situation, the allele transfer at inbreeding should also be described on Table 4 and 5. This however appears to be nearly but not totally right if you make more complicated genealogies. That is because the common ancestors can transfer maximal 2 alleles to their diploid descendants, while there may be more than two lines of descent. So you can see a-priori that the extinction of the alleles of the common ancestors goes faster in inbreeding than in out breeding.

The picture of **Table 10** affirms this idea indeed. **Table 10** shows the allele transfer by selfing in an unlimited population. This population is composed of an unlimited number of populations of reproduction with selfing, so with n=1. The extinction within this selfing population n=1 is given on **Table 5c** *n=1*, page… The total of an (relative) unlimited number n of such populations is for instance a population of n plants, all having different, so 2n unique alleles. The reproduction by selfing is started at F0. The descendants of the plants now are Poisson distributed with the intensity **λ**=1, on account of **r**=1. The plants do have yet one descendant on the average at selfing in the neutral population. The endowment of the alleles to the offspring is according to an incomplete binomium. The possibilities here are different from the situation in out breeding (**Table 9**). In the case of for instance 1 descendant now the allele a of the heterozygote parent will be singular transferred in ¼ of the cases, in ½ it will be transferred in twofold and in ¼ it will not be transferred, etc. Shortly the allele transfer now is described by the binomium (a+b)^2n, superposed on the distribution of the reproduction (λ=1). In **Table 10** the average intensity of the distribution remains λ=1, but the distribution now has become irregular and has no more a constant Poisson intensity. The P0 or extinction now is in accordance with a distribution at λ=0,75 and the multiples are corresponding more increased as compared with the out breeding, with regular Poisson λ=1. There is obvious extra extinction and in proportion extra multiplication of the alleles by the increase in the homozygosis. The elaboration of this distribution in the further generations is complicated by the increasing homozygosis, but can be deduced in this case (n=1 in the part population) from the random increase in the homozygosis. **Table 10a** shows this extinction according to **$-\sigma(Fg-1)=\mu\nu(Fg-2)-\mu$,** in this is for F0→F1 μ=0,75; for F1→F2 μ=0,875; for F2→F3 μ=0,9375,etc. In comparison with the situation by out breeding goes the extinction in this aspect of the inbreeding at first faster, but is gained up later on by the out breeding, so that both by the development of total homozygosis are going equal after a number of generations. In the complete homozygosis the total population here is of course not genetic identical. In the situation of homozygosis is similarity with the out breeding in the random extinction but not in the potential non random extinction by selection on account of the smaller average parity at inbreeding. The buffer by the endowment of the alleles disappears at total homozygosis. The random and non random

extinction than go both similar to the situation in the asexual reproduction.

Table 10

F0							
Q=1 λ=1							
F1 descendants							
1→0	1→1	1→2	1→3	1→4	1→5	1→6	
e^-1	e^-1	1/2.e^-1	1/6.e^-1	1/24.e^-1	1/120.e^-1	1/720.e^-1	
Alleles a							
→0n→0a	→0n→1a	→0n→2a	→0n→3a	→0n→4a	→0n→5a	→0n→6a	
e^-1	0						
→1n→0a	→1n→1a	→1n→2a	→1n→3a	→1n→4a	→1n→5a	→1n→6a	
1/4.e^-1	1/2.e^-1	1/4.e^-1	0				
→2n→0a	→2n→1a	→2n→2a	→2n→3a	→2n→4a	→2n→5a	→2n→6a	
1/32.e^-1	1/8.e^-1	3/16.e^-1	1/8.e^-1	1/32e^-1	0		
→3n→0a	→3n→1a	→3n→2a	→3n→3a	→3n→4a	→3n→5a	→3n→6a	
1/384.e^-1	1/64.e^-1	5/128.e^-1	10/192.e^-1	5/128.e^-1	1/64.e^-1	1/384.e^-1	
→4n→0a	→4n→1a	→4n→2a	→4n→3a	→4n→4a	→4n→5a	→4n→6a	
1/6144.e^-1	1/768.e^-1	7/1536.e^-1	3/768.e^-1	35/3072.e^-1	3/768.e^-1	7/1536.e^-1	
→5n→0a	→5n→1a	→5n→2a	→5n→3a	→5n→4a	→5n→5a	→5n→6a	
1/122880.e^-1	1/12288.e^-1	3/8192.e^-1	1/1024.e^-1	21/12288.e^-1	63/30720.e^-1	21/12288.e^-1	
→6n→0a	→6n→1a	→6n→2a	→6n→3a	→6n→4a	→6n→5a	→6n→6a	
1/2949120.e^-1	1/245760.e^-1	11/491520.e^-1	11/147456.e^-1	11/65536.e^-1	11/40960.e^-1	77/245760.e^-1	
∑P=0	∑P=1	∑P=2	∑P=3	∑P=4	∑P=5	∑P=6	∑a=0,9841
0,4723665	0,2361832	0,1771370	0,0669690	0,0307483	0,0080384	0,0033785	∑p=0.996
1,2840254.e^-1	0,6420127.e^-1	0,4815095.e^-1	0,1820407.e^-1	0,0835825.e^-1	0,0218506.e^-1	0,0091837.e^-1	x e^-1
e^-1	e^-1	0,5.e^-1	0,1667.e^-1	0,04167.e^-1	0,0083.e^-1	0,0013889.e^-1	Poiss. λ=1
e^-0,75	2/4.e^-0,75	12/32.e^-0,75	0.14177.e^-0,75	0,06502.e^-0,75	0,01702.e^-0,75	0,00715.e^-0,75	x e^-0,75
e^-0,75	3/4.e^-0,75	9/32.e^-0,75	0,07031.e^-0,75	0.01318.e^-0,75	0,00198e^-0,75.	0,00025.e^-0,75	Poiss. λ=0,75
λ=0,75	λ≈0,3278	λ≈0,96365	λ≈1,045	λ≈1,275	λ≈1,283	λ≈1,485	Intensities of the selfing dist

1→7	1→8	1→9		
1/5040.e^-1	1/40320.e^-1	2,76.10^-6 e^-1		
→0n→7a	→0n→8a	→0n→9a		
→1n→7a	→1n→8a	→1n→9a		
→2n→7a	→2n→8a	→2n→9a		
→3n→7a	→3n→8a	→3n→9a		
0				
→4n→7a	→4n→8a	→4n→9a		
1/768.e^-1	1/6144.e^-1	0		
→5n→7a	→5n→8a	→5n→9a	→5n→10a	
1/1024.e^-1	3/8192.e^-1	1/12288.e^-1	1/122880.e^-1	
→6n→7a	→6n→8a	→6n→9a	→6n→10a	
11/40960.e^-1	11/65536.e^-1	11/147456.e^-1	11/491520.e^-1	
∑P=7	∑P=8	∑P=9	∑P=10	∑a=0,9841
0,0009371	0,0002563	0,0000574	0,0000112	∑p=0.996
0,0025472.e^-1	0,0006968.e^-1	0,000156.e^-1	0,0000305.e^-1	x e^-1
0,000189.e^-1	0.000025.e^-1	2,76.10^-6 e^-1		Poiss. λ=1
0,00198.e^-0,75	0,00054.e^-0,75	0,00012.e^-0,75		x e^-0,75
0,00003.e^-0,75	0.0000006.e^-0,75	0,0000000		Poiss. λ=0,75
λ≈1,56	λ≈1,645	λ≈1,69	λ≈	Intensities of the selfing dist

Table 10a

Selfing									
F0	F1	F2	F3	F4	F5	F6	F7	F8	F9
σ=0,75	σ=0.4617	σ=0,3467	σ=0,2838	σ=0,24322	σ=0,2142	σ=0,1921	σ=0,1741	σ=0,1594	σ=0,1472
P0=0,4724	P0=0,6302	P0=0,7070	P0=0,7529	P0=0,7841	P0=0,8071	P0=0,8252	P0=0,8402	P0=0,8526	P0=0,8631
F1	F2	F3	F4	F5	F6	F7	F8	F9	F10
Outbreeding									
F0	F1	F2	F3	F4	F5	F6	F7	F8	F9
λ=1	σ=0,6321	σ=0,4685	σ=0,3741	σ=0,3121	σ=0,2681	σ=0,2352	σ=0,2095	σ=0,1890	σ=0,1723
P0=0,368	P0=0,531	P0=0,626	P0=0,688	P0=0,732	P0=0,765	P0=0,790	P0=0,811	P0=0,828	P0=0,842
F1	F2	F3	F4	F5	F6	F7	F8	F9	F10

These calculations at selfing are relative simple because here is a close subpopulation of reproduction with n=1 and the population also is close in the case of mating between 1st degree relatives, than with n=2. Furthermore the mating parents in n=1 also are the common ancestors. The populations with continued inbreeding in the lower degrees of relationship are open and that is why the calculations in these populations of reproduction are much more complicated. The population of the descendants than is namely not constant, but is increasing through the generations and the increase is slower than in out breeding. This increase of the descendants in neutral dynamics in out breeding is according the series 2-4-8-16-..and this is in half-brother half-sister mating in a linear genealogy the series 2-3-4-5-.. Furthermore accumulates the factor of inbreeding ***f*** in closed populations always to complete ***f***=1 and in this ultimate situation the population of reproduction is genetic identical. Within such an identical population no extinction is possible anymore. So within the population of reproduction the extinction is limited by the chance of fixation 1/n. From the aspect of a collection populations of reproduction the extinction is yet continued, because populations can dye out, see **Tables 9** and **9a.** In the open populations with inbreeding in the lower degrees the ***f*** accumulates in the half-brother half- sister according 1/8 – (1/8+1/16) – (1/8+1/16+1/32) - ..etc according the geometric series to ***f***=1/4. So the ***f*** accumulates at the first cousin continued in the generations in the neutral dynamics from ***f***=1/16 to ***f***=1/8, etc. So in the open populations the completed inbreeding does arise and the extinction remains to be continued in this hybrid of sexual and asexual reproduction with 1>**r**>2. This makes these calculation too much complicated.

So it is easy to conclude that the factor ***f*** of inbreeding accumulates in selfing fast and in the larger close populations slower

to complete, f=1, in the ultimate population with identical individuals. The existence of these identical sexual subpopulations in a population of reproduction is effectively equal to asexual reproduction in the subpopulations. So asexual reproduction properly is the highest form of inbreeding with f=1.

If in a large population are various lines of inbreeding or of asexual reproduction, this has globally no influence on the **random** changes in the genes of the total population. The random changes in the different lines will annul each other. This however is not the case for the influence of inbreeding in the subpopulations on the **non random** changes in the population. The smaller average numbers of descendants in the further generation by inbreeding makes a population more susceptible for selection, if there is inbreeding in the subpopulations. So the genetic changes by selection are accelerated by inbreeding. By inbreeding and asexual reproduction the same alleles remain random in the whole population, but their presence in the population is less homogeneous. Inbreeding is the accumulation of alleles. The population by inbreeding is divided up into groups with different genotypic and phenotypic characteristics. Those groups also will react different on factors in their environment determining their fitness. So the results of the reproduction in the groups with the various lines of inbreeding will be different by selection and some lines may dye out. It is obvious that the selection of recessive alleles for some more reasons is more effective by the inbreeding. Namely because the expression of recessive alleles mainly is possible by inbreeding, so that only than the positive or negative fitness of these alleles can come to light. Furthermore the selection by inbreeding is more effective by the distribution of the alleles with the multiple lines of descent over smaller numbers of descendants in the neutral dynamics. Yet if an ancestor has fewer or more descendants by the selection the consequences of it for the allele transfer are amplified by the multiple descent. That is why a population is by inbreeding more susceptible for selection than a population in panmixia of alleles. This selection strengthened by inbreeding so gives important and fast non random genetic changes in a population.

So by these **indirect consequences** of inbreeding a population is by inbreeding more susceptible for selection than a population in panmixia of alleles. Furthermore inbreeding may have a **direct effect** on the selection, so that the inbreeding itself is selective. This is the case if the related parents do have more children than on the average and the common ancestors so have more descendants than at random, as described in the neutral dynamics. A systematically larger than random expected success in the procreation at inbreeding in fact is inbreeding selection. This should increase the amplifying force of inbreeding on the selection even more. Indications a-priori for the selection on inbreeding are: Inbreeding has been for

various reasons opportune in the human societies and is this also generally in the nature, if both parents have to cooperate at the care for the next generation. It brings a better oneness and team-spirit between the parents if they are relatives or at least acquaintances of each other. To this latter has been developed a deep-rooted custom of inbreeding by people and some animal species: namely **monogamy**[11]**.** At inbreeding are fewer territorial problems within the reproductive population, etc. So it is interesting to investigate if indeed is affirmed by observation that inbreeding gives larger numbers of effective children.

How inbreeding is present in a human population, the allele transfer influences and interferes with the selection can also be studied on the basis of genealogical data as they are on this site and more complete on the geneanet site http://gw.geneanet.org/wschot. Particularly in our branch of the family there were many marriages between relatives and that often during some generations. Consanguinity was by the way in the past much more frequent in the autochthon Dutch population. Not only by noblemen but also by common citizens ware often practical and material circumstances that facilitate consanguine marriages, as there were in the serfdom and later in the system of the guilds. The anchovies fishery within the guild in Bergen op Zoom may have initiated this and afterwards it also was no taboo. In the Protestantism there were also no principal objections against consanguinity until the degree of first cousin. Important was that a possible disadvantage as diseases or dying of the children was in those days evidently or certainly not present so that there was no damage. The infant mortality was yet already about 50% and was by various factors very variable. Now we see indeed an increase in the chance on serious problems by the children from 3% general to about 5% in a first cousin marriage, but in the past that increase was not present.

Interesting thus is that consanguinity, or inbreeding, the companion of the selection, has been nearly disappeared out of the modern human populations together with the selection. The inbreeding is as pointed out an important amplifier of the efficiency of the selection and it appears also a frequent companion of it. Inbreeding in different measures do occur yet very often in natural populations, although not every species reproduces with some inbreeding. The species that don't inbreed are active, mobile organisms that all come together all at the same place, at the same time, where in a gigantic orgy the total

[11] This affinity is an indirect cause of inbreeding: descendants of full brothers and sisters have a higher relationship and will give a larger degree of inbreeding than descendants of half-brothers and half-sisters.

species reproduces at once. So the squids are doing it for instance. This policy of reproduction giving panmixia of the genes can decrease and possibly stop the selection and will preserve the properties of the species. The species doing so however are in the minority and this policy of reproduction is impossible for many species and it is not evolutionary. Mostly the species do reproduce with some measure of inbreeding, that together with the sexual selection determines the intensity of the species' non random genetic changes and so the velocity of their micro-evolution. So it come to the point that the sexual and social behaviour of the animals and the **plants**[12] determines the measure of inbreeding the species has and so its evolution. That was also what Charles Darwin had in view when he wrote that the nature procreated the species in the way that people did breed their domestic animals and their improved plants. The intuitive knowledge of the people in the past about the consequences of inbreeding is functioning also as a system throughout the nature. The hypothesis that the speed of the micro-evolution of the species may be well ruled in the chain: genetic decided reproductive behaviour → measure of inbreeding and sexual selection → genetic changes → behaviour at the reproduction etc, however is yet a bridge further. This hypothesis is not generally known I think and it seems worthy to be studied.

This concerning the interaction between inbreeding and selection, which should be of great importance according to global observation, but about which only can be concluded after analysis on the basis of comparison with the random changes by inbreeding in neutral dynamics. In these random changes **Sewall Wright** did provide clarity in the different aspects of inbreeding by turning the matter in that he let decide the size of the population the measure of the inbreeding. This construction of the random inbreeding by itself is a really good one, which has provided its value for many years. The genetic drift is described in this theory as random inbreeding and is defined as: **H(g+t) = Hg x [1−1/2n]^t** and ***f*** **= 1/2n**. In this ***f*** is the inbreeding factor in the primary, original generation g (or F0). So this formula describes the measure of the heterozygosis as an exponential function in the time in relation to the population size. The great benefit of this formula is its efficiency, giving in a simple way good insight in the most important genetic changes in a close population. The restrictions however are an disadvantage: It is not applicable in variable population size or in non random inbreeding, but the most important problem is that it only describes the heterozygosis **extinction**[13] and not the primary and more essential allele extinction. The description of the allele extinction is yet indeed more complicated and in general not used as a theory for

[12] Also plants do have sexual behaviour or sexual communication by their flowers mostly in the symbiotic relations with insects.
[13] Properly its complement the heterozygosis survival.

genetic drift and random genetic changes. Nevertheless the allele extinction is necessary in the neutral theory and so as a basis for the micro-evolution theory. The use of the Poisson distributions does simplify substantially the application of the theory of the allele extinction. Also in limited and even in very small populations the allele extinctions are to be calculated in different ways and may give important information besides to the well-known heterozygosis extinction.

In the population with inbreeding the transfer of the singular allele follows the superposed Poisson distributions with intensities **μ**=1+***f,*** as described at **Table 9c**. This is the case in the open population with relative (more than average) inbreeding as well as in the close population with random inbreeding because of the limited size n. The inbreeding factor ***f*** is not constant in (random) continued inbreeding, but increases through the generations because of the increase of the consanguinity and the number of identical alleles of the parents in the population. In the open and unlimited H-W population the inbreeding factor in the same line increases from ***f***=1/2n in the F0 to ***f***=1/n in the F∞, but within the close, limited populations this will increase from ***f***=1/2n in the F0 to complete, ***f***=1 in the F∞, when the population is at last complete homozygote and genetically identical. In a population of somewhat larger size n ***f***=1/2n is small in relation to 1 and so the chance on survival (=1-F0) in the F∞ will be about 1/n according to **Table 5b.** Then it will be about 2x as large as the a-priori chance on fixation, 1/2n. This seems curious but in **Table 5** and **5b** extinctions are calculated of one allele on one locus of one individual in F0 and the complement of it is the survival on one locus in a proportion of individuals in F∞. This all in the H-W population. Within the limited population is survival in F∞ also complete fixation. This then is however a survival on 2 loci within the population n. So the chance on fixation in a limited population is about the half of the chance on ultimate survival in an unlimited population.

As described on page 19 the extinction in the primary population is 1/e in the (relative) **unlimited** population, because P0= (1−1/2n)^2n →1/e, if n→∞. In the **limited** population the primary extinction depends than however on the population size and is to be calculated as P0= (1−1/2n)^2n. The general negative exponential intensity can be calculated here in the limited population as well by **μ**=−ln(1-1/2n)^2n, or **μ** = −2n.ln(1−1/2n). So this is **μ**=1, if n→∞, because (1−1/2n)^2n →1/e, if n→∞. If n=3, as in the example of Table **5c** *2n=6***,** it is obvious that the **μ** in the general recurrence formula **−σ(Fg)=μν-μ** is simple to be calculated as P0= (1-1/6)^6 en **μ**=−6.ln(5/6)= 1,093929.. The extinction calculated with the accumulated exponential distribution as in **Table 5c** does give the a-priori expected ultimate survival and fixation. In this example the fixation is 1/6, the complement of the extinction, p0 in F∞, thus 1-5/6. So if the right intensity is used the extinction can be

calculated in any population. For a population of **unlimited and limited size** the intensity simply is to be calculated as **μ=−2n.ln(1-1/2n).** The practical proof of the rightness of this formula follows from the application of it with trials by observation of drawings, as described on page 17. In this case can be started with a bag of 6 marbles with different colours. The 6 marbles ever are drawn and placed back into the bag. Than after 6 drawings a new bag is composed with the results of these 6 drawings from the former bag. After a number of these experiments the marbles in the bags will have ever more the same colours, because the other colours disappear. The velocity of this fixation and vanishing of the colours of the marbles however is different in all these experiments. The average velocity of this extinction and fixation is indicated in **Table 5c** *2n=6*. The theoretical deductive proof follows directly out of the deduction of the negative exponential distribution. Different from the Kimura positions the recurrence formula also is applicable in a population of limited size and is the recurrence a good general theory. Further it may be important to explore the compatibility of these calculations of the allele extinctions with the calculation of the decrease or extinction of the heterozygosis in accordance with the Wrights' formula. It is impossible in general the calculate the allele extinctions starting from the heterozygosis extinctions. Only in an average population with n=1 the allele extinction can be calculated the heterozygosis extinction.

Because there is just one individual in each generation, there is also only one possibility to heterozygosis and describes the heterozygosis extinction in n=1 the complete course of the allele distributions through the generations. From Wrights' formula namely is direct deducible that the genotype ab occurs in the generations with proportions or chances following the series 1 - 0,5 – 0,25 -0,125 etc and both homozygotic types aa and bb each following 0 - 0,25 – 0,375 – 0,4375 etc. However if n>1 there are more possibilities of heterozygosis and are the courses of the genotypes and the allele extinctions no longer deducible from the heterozygosis extinction. Already at n=2 there are unlimited many possibilities of distributions that comply with the data, that for instance the heterozygosis is decreased in two generations from complete, Het=1 to Het=(3/4)^2=0,5625. To be able to calculate average expected distributions of the original 4 alleles you must return to the complete statistical distribution, of which the Wrights' formula has been deduced.

In **Table 5** and **5c** only is showed the accumulating exponential distribution and not the superposed total Poisson distributions. The Tables 5b and 5c are the same in the way of the calculation, at both the extinction is calculated with the intensities **μ** and **σ** of the cumulating exponential distribution, as e^**−σ.** The particularity of Table 5c however is that these exponential intensities here are determined by the limited or small population size and in this is the number of the parities in the distribution of course also maximized to 2n. Because of this limited population size the accumulating exponential distribution can not be extended to the complete Poisson distributions in superposition. At the Tables 5c P0 again is the allele extinction and the complement of it the survival is indicated as Su, so that Su=1-P0; **α** is de survival van of all the alleles, so that **α** =2n.Su. Het is the heterozygosis of the old and the new generations calculated with the Wright's formula.

The generations are indicated as F0; F1, etc. The population with 51 alleles, **Table 5c** *2n=51,* is not quit exact, but an approach. This table has been made with some data of Kimura, see Table 5b, but 51ln(50/51)=−1,009934 and so gives **μ**=1.009934, here this is now rounded to **μ**=1.01 and with this intensity was already made a table.

In the example of table 5c *2n=6* is indicated: In a population with 3 individuals (in both genders fertile) are vanished of the 6 allelic variations that (possibly) are in F0 on the loci, after 1 generation about 2, after 3 generations nearly 3, after 5 generations nearly 4, after 70 generations nearly 5 and after ∞ generations exact 5 of the 6 alleles on all the loci.

So the exponential accumulation and recurrence seems excellent applicable in small populations. Yet the extinction P0 converges ever to the a-priori expected value, the complement of the fixation., so that in F∞ always rests one allele, **α**=1.

Table 5c *2n=6*

F0	F1	F2	F3	F4	F5	F6	F7	F8	F9
μ=1,093929	σ=0,72757	σ=0,56548	σ=0,47247	σ=0,41192	σ=0,36933	σ=0,33781	σ=0,31360	σ=0,29447	σ=0,27903
P0=0,334898	P0=0,48308	P0=0,56809	P0=0,62346	P0=0,66238	P0=0,6912	P0=0,71333	P0=0,73081	P0=0,74493	P0=0,75652
Su=0,6651	Su=0,5169	Su=0,4319	Su=0,3765	Su=0,3376	Su=0,3088	Su=0,2867	Su=0,2692	Su=0,2552	Su=0,24348
α=3,99	α=3,10	α=2,59	α =2,26	α =2,026	α =1,853	α =1.72	α =1,62	α =1,531	α =1,461
Het=1	Het=0,8333	Het=0,6944	Het=0,5787	Het=0,4823	Het=0,4019	Het=0,3349	Het=0,2791	Het=0,2326	Het=0,1938
Het=0,8333	Het=0.6944	Het=0,5787	Het=0,4823	Het=0,4019	Het=0,3349	Het=0,2791	Het=0,2326	Het=0,1938	Het=0,1615
F1	F2	F3	F4	F5	F6	F7	F8	F9	F10

F10	F11	F12	F13	F14	F15	F16	F17	F18	F19
σ=0,26635	σ=0,25580	σ=0,24690	σ=0,23934	σ=0,23284	σ=0,22723	σ=0,22236	σ=0,21809	σ=0,21436	σ=0,21107
P0=0,76617	P0=0,77430	P0=0,78122	P0=0,78715	P0=0,79228	P0=0,79674	P0=0,80063	P0=0,80405	P0=0,80706	P0=0,80972
F11	F12	F13	F14	F15	F16	F17	F18	F19	F20

F20	F21	F22	F23	F24	F25	F26	F27	F28	F29
σ=0,20814	σ=0,20556	σ=0,20082	σ=0,20121	σ=0,19938	σ=0,19774	σ=0,19627	σ=0,19496	σ=0,19377	σ=0,19269
P0=0,81209	P0=0,81419	P0=0,81806	P0=0,81774	P0=0,81924	P0=0,82058	P0=0.82179	P0=0,82287	P0=0,82385	P0=0,82474
F21	F22	F23	F24	F25	F26	F27	F28	F29	F30

F30	F31	F32	F33	F34	F35	F36	F37	F38	F39
σ=0,19173	σ=0,19086	σ=0,19007	σ=0,18936	σ=0,18871	σ=0,18813	σ=0,18760	σ=0,18713	σ=0,18669	σ=0,18629
P0=0,82553	P0=0,82625	P0=0,82690	P0=0,82749	P0=0,82803	P0=0,82851	P0=0,82895	P0=0,82934	P0=0,82970	P0=0,83003
F31	F32	F33	F34	F35	F36	F37	F38	F39	F40

F40	F41	F42	F43	F44	F45	F46	F47	F48	F49
σ=0,18593	σ=0,18561	σ=0,18532	σ=0,18504	σ=0,18480	σ=0,18458	σ=0,18438	σ=0,18419	σ=0,18402	σ=0,18387
P0=0,83033	P0=0,83060	P0=0,83084	P0=0,83107	P0=0,83127	P0=0,83145	P0=0,83162	P0=0,83178	P0=0,83192	P0=0,83204
F41	F42	F43	F44	F45	F46	F47	F48	F49	F50

F50	F51	F52	F53	F54	F55	F56	F57	F58	F59
σ=0,18373	σ=0,18361	σ=0,18349	σ=0,18339	σ=0,18330	σ=0,18321	σ=0,18313	σ=0,18306	σ=0,18300	σ=0,18294
P0=0,83216	P0=0,83226	P0=0,83236	P0=0,83244	P0=0,83252	P0=0,83259	P0=0,83266	P0=0,83272	P0=0,83277	P0=0,83282
F51	F52	F53	F54	F55	F56	F57	F58	F59	F60

F60	F61	F62	F63	F64	F65	F66	F67	F68	F69	F∞
σ=0,18287	σ=0,18283	σ=0,18278	σ=0,18273	σ=0,18270	σ=0,18266	σ=0,18264	σ=0,18261	σ=0,18258	σ=0,18255	σ=0,18232
P0=0,83287	P0=0,83291	P0=0,83295	P0=0,83299	P0=0,83302	P0=0,83305	P0=0,83307	P0=0,83309	P0=0,83312	P0=0,83314	**P0=0,83333**
F61	F62	F63	F64	F65	F66	F67	F68	F69	F70	F∞

Table 5c 2*n=2*

F0	F1	F2	F3	F4	F5	F6	F7	F8	F9
μ=1,386294361	σ=1,03972	σ=0,89617	σ=0,82050	σ=0,77603	σ=0,74828	σ=0,73033	σ=0,71845	σ=0,71046	σ=0,70505
P0=0,25	P0=0,35355	P0=0,40813	P0=0,44021	P0=0,46023	P0=0,47318	P0=0,48175	P0=0,48751	P0=0,49142	P0=0,49409
α =1,5	α =1,2929	α =1,1837	α =1,1120	α =1,0795	α =1,0536	α =1,0365	α =1,0250	α =1,0172	α =1,0118
F1	F2	F3	F4	F5	F6	F7	F8	F9	F10

F0	F1	F2	F3	F4	F5	F6	F7	F8	F9
Het=1	Het=0,5	Het=0,25	Het=0,125	Het=1/16	Het=1/32	Het=1/64	Het=1/128	Het=1/256	Het=1/512
Het=0,5	Het=0,25	Het=0,125	Het=0,0625	Het=1/32	Het=1/64	Het= 1/128	Het=1/256	Het=1/512	Het=1/1024
P0=0,25	P0=0,375	P0=0,4375	P0=0,46875	P0=0.48438	P0=0,49219	P0=0,49609	P0=0,49805	P0=0,49902	P0=0,49951
α =1,5	α =1,25	α =1,125	α =1,0625	α =1,0312	α =1,0156	α =1,0078	α =1,0039	α =1,0020	α =1,0010
F1	F2	F3	F4	F5	F6	F7	F8	F9	F10

Table 5c 2*n=51*

F0	F4	F9	F19	F49	F99	F199	F299	F399	F499	F∞
μ=1,01	σ=0,32215	σ=0,18241	σ=0,10197	σ=0,04919	σ=0,03117	σ=0,02298	σ=0,02096	σ=0,02029	σ=0,02006	σ=0,01993
P0=0,36422	P0=0,72459	P0=0,83326	P0=0,90306	P0=0,95200	P0=0,96931	P0=0,97728	P0=0,97926	P0=0,97991	P0=0,98014	P0=0,98027
α =32,4	α =14,0	α =8,5	α =4,9	α =2,45	α =1,57	α =1,16	α =1,06	α =1,0246	α =1,013	α =1,006
Het=1	Het=0,9238	Het=0,8368	Het=0,6864	Het=0,3790	Het=0,1408	Het=0,0194	Het=0,0027	Het=0,0004	Het=0,00005	Het=0
Het=0,9804	Het=0,9057	Het=0,8203	Het=0,6730	Het=0,3715	Het=0,1380	Het=0,0191	Het=0,0026	Het=0,0004	Het=0,00005	Het=0
F1	F5	F10	F20	F50	F100	F200	F300	F400	F500	F∞

The binomial superposition and extinction

The binomial distribution will describe the random changes in a small population better than the Poisson distribution does. Like in the example of the 6 drawings under replace in the bag with 6 marbles and followed by the composition of the new bag by the result of the former bag. The binomial distribution will describe the total distribution of all the drawing events within this small space or population and not only the P0, the complement of the drawing as does the exponential distribution. The Poisson, binomial and normal distributions however are continuous in each other and compose properly one stochastic distribution. One can pose that the Poisson distribution is a limit case in the binomial distribution for n→∞ and that the binomial distribution is a specific case in the Poisson distribution for the limited n. In the Tables 11 the superposition of the binomial distribution is made. This could be for instance the chances to cast in 2n=2 throws 0x; 1x or 2x six with the dice, or the chances to cast in 2n=6 throws 0x; 1x; 2x…6x six with the dice or as well to draw so many times the right marble or the right allele. In the well known primary binomial distribution this distribution of chances is calculated as the product of the binomial coefficient and the chance to draw the right allele and the complement of this. At the second and further degree binomial distributions the proportions of the former distribution are further distributed, following the arrows. It is obvious that the chances will change in this: So for instance the right white marble is at first singular in the bag of 6 that white marble is drawn with the change 1/6 and so it is drawn twice in the proportion of 0,2009.. following the distribution. In the second turn this proportion is not drawn with the chance 1/6 but with 1/3, because it now occurs in twofold. Of course marbles can not be drawn that were not drawn in the former turn (extinction) and always is drawn 6x the right marbles from bags that contain exclusively the right marbles (fixation).

Table 11 *2n=2* shows the binomial superposition at 2n=2, so in selfing. Mind the increase of the extinction under∑=0 and of the fixation under ∑=2. Also the alternative allele has the same distribution and so the percentage of identical populations increases here as 2x the fixation. The binomial allele extinction is in 2n=2 also to be calculated direct from the heterozygosis extinction following the Wrights' formula as is done on **Table 9c** *2n=2.* This **Table 9c** also shows the difference between the binomial and exponential extinction.

Table 11 *Binomial 2n=2*

→0	→1	→2					
F0							
0	1	0					
1→0	1→1	1→2		F3			
1.0,5^0.0,5^2	2.0,5^1.0,5^1	1.0,5^2.0,5^0	Binomial	→0→0	→0→1	→0→2	
0,25	0,5	**0,25**	**F1**	0,4375	0	0	
F1			50%pop id	→1→0	→1→1	→1→2	
→0→0	→0→1	→0→2		0,03125	0,0625	0,03125	
0,25	0	0		→2→0	→2→1	→2→2	
→1→0	→1→1	→1→2		0	0	0,4375	
0,125	0,25	0,125		∑0	∑1	∑2	
→2→0	→2→1	→2→2		**0,46875**	0,0625	**0,46875**	**F4**
0	0	0,25		F4			93,75% pop id
∑0	∑1	∑2		→0→0	→0→1	→0→2	
0,375	0,25	**0,375**	**F2**	0,46875	0	0	
F2			75% pop id	→1→0	→1→1	→1→2	
→0→0	→0→1	→0→2		0,015625	0,03125	0,015625	
0,375	0	0		→2→0	→2→1	→2→2	
→1→0	→1→1	→1→2		0	0	0,46875	
0,0625	0,125	0,0625		∑0	∑1	∑2	
→2→0	→2→1	→2→2		**0,484375**	0,03125	**0,484375**	**F5**
0	0	0,375		F5			96,9% pop id
∑0	∑1	∑2					
0,4375	0,125	**0,4375**	**F3**				
F3			87,5%pop id				

The table 11 2n=4, so as by inbreeding of first degree relatives in the F1? These distributions always are without mixture of the generations, so here is in the first generation the possibility of full brother and sister indeed, but also is possible the random selfing in this model with random mating of two individuals, fertile in the both genders. In the further generations the genetic relationship increases fast as is evident yet more in the next **Table 12** *2n=4*. Distributions of populations with mixture of the generations and separate genders are more complicated. Although the picture of these tables give general insight in the principle the distributions in very small mammal populations will deviate substantially from these numbers. Mind the increase of the extinction at ∑0 and the fixation at ∑4. The percentage identical populations will increase with 4x the fixation.

Table 11 *Binomial 2n=4*

Binomial 2n=4. Distribution alleles, gametes of **F0** four times singular.

→0	→1	→2	→3	→4
0	1	0	0	0
1→0	1→1	1→2	1→3	1→4
1.(1/4)^0.(3/4)^4	4.(1/4)^1.(3/4)^3	6.(1/4)^2.(3/4)^2	4.(1/4)^3.(3/4)^1	1.(1/4)^4.(3/4)^0
0,3164063	0,421875	0,2109375	0,046875	0,0039063
F1				1,6% pop id
→0→0	→0→1	→0→2	→0→3	→0→4
0,3164063	0	0	0	0
→1→0	→1→1	→1→2	→1→3	→1→4
0,13348389	0,1779785	0,0889893	0,0197754	0,00164795
→2→0	→2→1	→2→2	→2→3	→2→4
0,0131835	0,0527344	0,0791016	0,0527344	0,0131835
→3→0	→3→1	→3→2	→3→3	→3→4
0,0001831	0,0021973	0,0098876	0,0197754	0,01483154
→4→0	→4→1	→4→2	→4→3	→4→4
0	0	0	0	0,0039063
∑0	∑1	∑2	∑3	∑4
0,4632567	0,2329102	0,1779785	0,0922852	**0,03356929**
F2				13,4% pop id

F3				
→0→0	→0→1	→0→2	→0→3	→0→4
0,5484354	0	0	0	0
→1→0	→1→1	→1→2	→1→3	→1→4
0,0465369	0,0620492	0,0310246	0,0068944	0,0005745
→2→0	→2→1	→2→2	→2→3	→2→4
0,0084586	0,0338345	0,0507517	0,0338344	0,0084586
→3→0	→3→1	→3→2	→3→3	→3→4
0,0003685	0,0044224	0,01990092	0,03980184	0,0298514
→4→0	→4→1	→4→2	→4→3	→4→4
0	0	0	0	0,07480275
∑0	∑1	∑2	∑3	∑4
0,6037994	0,1003061	0,10167722	0,08053064	**0,11368725**
F4				45,5% pop id
→0→0	→0→1	→0→2	→0→3	→0→4
0,6037994	0	0	0	0
→1→0	→1→1	→1→2	→1→3	→1→4

→0→0	→0→1	→0→2	→0→3	→0→4	0,0317375	0,0423166	0,0211583	0,0047018	0,0003918
0,4632567	0	0	0	0	→2→0	→2→1	→2→2	→2→3	→2→4
→1→0	→1→1	→1→2	→1→3	→1→4	0,0063548	0,0254193	0,038129	0,0254193	0,0063548
0,07369442	0,098259	0,0491295	0,0109177	0,0009098	→3→0	→3→1	→3→2	→3→3	→3→4
→2→0	→2→1	→2→2	→2→3	→2→4	0,0003146	0,0037749	0,0169869	0,0339738	0,0254804
0,0111237	0,0444946	0,0667419	0,0444946	0,0111237	→4→0	→4→1	→4→2	→4→3	→4→4
→3→0	→3→1	→3→2	→3→3	→3→4	0	0	0	0	0,11368725
0,0003605	0,0043259	0,0194664	0,03893282	0,02919996	∑0	∑1	∑2	∑3	∑4
→4→0	→4→1	→4→2	→4→3	→4→4	**0,6422063**	0,0715108	0,0762742	0,0640949	**0,14591425**
0	0	0	0	0,03356929	F5				58,4% pop id
∑0	∑1	∑2	∑3	∑4					
0,5484354	0,1470795	0,1353378	0,09434512	**0,07480275**					
F3				29,9% pop id					

In **Tabel 12** *2n=4 Binomial homozygosis* the increase in the homozygosis through the generations is calculated direct from the superposed binomial distribution. Its complement the heterozygosis survival than is of course also given. The results are equal to the calculations with the well known Wrights' formula. That is no wonder because Wright deduced his formula from the binomial distribution. The niceness of this inductive derivation however is that it gives easier insight. It also gives a specification of the homozygosis over the fixation parity and the other parities. The calculations are simple: as is described in Table 11 the singular allele in the F0 is in the next generation also singular, or in twofold, in threefold, in fourfold or it is vanished. The average expected proportions of the n-folds of all the four alleles are of course found by multiplication with 4.

So the principle is simple: The n-folds or parities to for instance the F1 are calculated with the binomial distribution. These are than properly the alleles in the gametes of F0, which are transferred to the F1. Out of this are calculated the genotypes of F1 in the sense of heterozygote or homozygote, as is showed in this **Table 12** at F0→F1. From F1 is for any generation to be calculated in the stochastic process the expected measure of homo and heterozygosis. The structure of the F0 however is defined as starting population and is not determined in the stochastic process. The potential homozygosis starts from a free stochastic cause of F0 and so can have another value than the real one. The heterozygosis is of course complete (Het=1) in the starting population with its 4 unique alleles. The potential homozygosis of 0,25 in F0 however has risen by the random

chance on to F0, but the genotype of F0 has been given in the data and reproduction to F0 did not take place within this population 2n=4.

In F1 the mating individuals are from a global view full brothers and sisters, but their genetic relationship is much larger and they procreate much more homozygotes (0,44) than these relatives do in an open population (0,25). This here is obvious caused by the chances on homozygosis by random selfing in F0 and F1. In these of course are differences with the situation at the actual mating in the nature or in the laboratory in very small populations. In larger populations this random selfing is much smaller and in the Poisson distributions it is fallen off. It also appears from this **table 12** *2n=4* that the random selfing decreases fast in the further increasing generations. The global estimation thus is that the allele and heterozygosis extinction is some generations slower in real populations with separated gender in 2n=4. It however is obvious that the possibilities of such population are else and so the distributions are. So it yet is a challenge to make specific distributions for these natural populations.

Tabel 12 *2n=4F0 singular. Binomial homozygosis*

Binomial 2n=4. Distribution alleles, gametes van F0 four times singular

→0	→1	→2	→3	→4	∑	
0	1	0	0	0		F0 distribution Q=1
0	4	0	0	0	4	F0 distrubution all 4 alleles
	0,25				**0,25**	Hom. in F1

According to Wrights' formula is the Homozygosis in F1 1-0,75=0,25

0,31640625	0,421875	0,2109375	0,046875	0,00390625	1	F1 distribution Q=1
1,2656252	1,6875	0,84375	0,1875	0,0156252	4,0000004	F1 distribution all 4 alleles
x(0/4)^1	x(1/4)^2	x(2/4)^2	x(3/4)^3	x(4/4)^4		
0	0,10546875	0,2109375	0,10546875	0,0156252	**0,4375002**	Hom. in F2

According to Wrights' formula is the Homozygosis in F2 1-(0,75)^2=0,4375

0,4632567	0,2329102	0,1779785	0,0922852	0,03356929	0,99999989	F2 distribution Q=1
1,8530268	0,9316408	0,711914	0,3691408	0,1342772	3,9999996	F2 distribution all 4 alleles
0	0,05822755	0,1779785	0,2076417	0,1342772	**0,57812495**	Hom. in F3

According to Wrights' formula is the Homozygosis in F3 1-(0,75)^3=0,578125

0,5484354	0,1470795	0,1353378	0,09434512	0,07480275	1,00000057	F3 distribution Q=1
2,1937416	0,588318	0,5413512	0,3773804	0,2992108	4,000002	F3 distribution all 4 alleles
0	0,03676988	0,1353378	0,21227648	0,2992108	**0,68359495**	Hom. in F4

According to Wrights' formula is the Homozygosis in F4 1-(0,75)^4=0,68359375

0,6037994	0,1003061	0,10167722	0,08053064	0,11368725	1,00000061	F4 distribution Q=1
2,4151976	0,4012244	0,4067088	0,3221224	0,4547488	4,000002	F4 distribution all 4 alleles
0	0,02507653	0,10167722	0,18119385	0,4547488	**0,7626964**	Hom. in F5

According to Wrights' formula is the Homozygosis in F5 1-(0,75)^5=0,7626953

0,6422063	0,0715108	0,0762742	0,0640949	0,14591425	1,00000045	F5 distribution Q=1
2,5688252	0,2860432	0,3050968	0,2563796	0,5836568	4,0000016	F5 distribution all 4 alleles
0	0,0178777	0,0762742	0,14421353	0,5836568	0,82202223	Hom. in F6

According to Wrights' formula is the Homozygosis in F6 1-(0,75)^6=0,8220215

Starting from a population with 6 singular alleles is made **Tabel 11** *2n=6 six times singular*. Mind the increasing extinction at ∑0 and the increasing fixation at ∑6. In this table the Poisson distributions are given for comparison at the F1, F2 and F3. With these binomial distributions only the neutral population dynamics can be described. As for this aspect the Poisson distribution with λ=1 is comparable with the binomial distribution. So mind the differences between the Poisson and binomial distributions are not large, even in this vary small population and they decrease in the further increasing generations. Also interesting is comparison with the Poisson distribution with intensity λ=6ln(5/6)=-1,093929, because the P0 (extinction) of this intensity is equal to the binomial extinction.

Tabel 11 *2n=6 six times singular*

Binomial 2n=6. Distribution alleles, gametes of F0 six times singular

→0	→1	→2	→3	→4	→5	→6	∑	
0	1	0	0	0	0	0	1	F0 distribution Q=1
F0						(1/6)^5 pop id		
1→0	1→1	1→2	1→3	1→4	1→5	1→6		
1.(1/6)^0(5/6)^6	6.(1/6)^1(5/6)^5	15.(1/6)^2.(5/6)^4	20.(1/6)^3.(5/6)^3	15.(1/6)^4.(5/6)^2	6.(1/6)^5.(5/6)	1.(1/6)^6.1	Binomiaal	
0,33489797	**0,40187757**	**0,20093878**	**0,05358368**	**0,00803755**	**0,000643**	**0,0000214**	**0,99999995**	**F1 Binomial Q=1**
0,367879	**0,367879**	**0,1839397**	**0,06131324**	**0,0153283**	**0,00306566**	**0,00051094**	**0,99991584**	**F1 Poiss λ=1 Q=1**

0,33489797	0,3663547	0,2003831	0,0730683	0,0199829	0,004372	0,000797	0,99985597	F1 Poiss λ=6ln(5/6)
F1								
→0→0	→0→1	→0→2	→0→3	→0→4	→0→5	→0→6		
0,33489797	0	0	0	0	0	0		
→1→0	→1→1	→1→2	→1→3	→1→4	→1→5	→1→6		
0,13458798	0,16150558	0,08075279	0,02153408	0,00323011	0,00025841	8,613E-06	0,40187756	
→2→0	→2→1	→2→2	→2→3	→2→4	→2→5	→2→6		
0,01764072	0,05292215	0,06615269	0,04410179	0,01653817	0,00330735	0,00027564	0,2009385	
→3→0	→3→1	→3→2	→3→3	→3→4	→3→5	→3→6		
0,00083725	0,00502347	0,01255868	0,01674491	0,01255867	0,00502347	0,00083725	0,05358369	
→4→0	→4→1	→4→2	→4→3	→4→4	→4→5	→4→6		
1,10E-05	0,00013231	0,00066153	0,00176408	0,00264612	0,0021169	0,00070563	0,0080376	
→5→0	→5→1	→5→2	→5→3	→5→4	→5→5	→5→6		
1,38E-08	4,13E-07	5,17E-06	0,0000345	0,0001292	0,00025841	0,00021534	0,000643	
→6→0	→6→1	→6→2	→6→3	→6→4	→6→5	→6→6		
0	0	0	0	0	0	0,0000214	0,0000214	
∑0	∑1	∑2	∑3	∑4	∑5	∑6	0,66510175	
0,48797495	**0,21958392**	**0,16013086**	**0,08417931**	**0,03510228**	**0,01096453**	**0,00206387**	**0,99999972**	**F2 Binomial Q=1**
∑0 Poiss	∑1Poiss	∑2Poiss	∑3Poiss	∑4Poiss	∑5Poiss	∑6Poiss	∑≥7 Poiss	
0,53146305	**0,19551454**	**0,13372015**	**0,07295863**	**0,03614535**	**0,01697346**	**0,00763095**	**0,00503485**	**F2 Poisson Q=1 λ=1**
F2						1,24% pop id		
→0→0	→0→1	→0→2	→0→3	→0→4	→0→5	→0→6		
0,48797495	0	0	0	0	0	0		
→1→0	→1→1	→1→2	→1→3	→1→4	→1→5	→1→6		
0,0735382	0,08824585	0,04412293	0,01176612	0,00176493	0,00014119	4,699E-06	0,21958391	
→2→0	→2→1	→2→2	→2→3	→2→4	→2→5	→2→6		
0,01405813	0,04217439	0,05271799	0,03514533	0,0131795	0,0026359	0,00021966	0,1601309	
→3→0	→3→1	→3→2	→3→3	→3→4	→3→5	→3→6		
0,0013153	0,0078918	0,0197295	0,026306	0,0197295	0,0078918	0,0013153	0,0841793	
→4→0	→4→1	→4→2	→4→3	→4→4	→4→5	→4→6		
4,82E-05	0,00057782	0,00288908	0,00770421	0,01155631	0,00924505	0,00308168	0,0351023	
→5→0	→5→1	→5→2	→5→3	→5→4	→5→5	→5→6		
2,35E-07	7,05E-06	8,81E-05	5,88E-04	0,00220319	0,00440639	0,00367169	0,01096421	
→6→0	→6→1	→6→2	→6→3	→6→4	→6→5	→6→6		
0	0	0	0	0	0	0,00206387	0,00206387	

∑0	∑1	∑2	∑3	∑4	∑5	∑6	0,51202448	
0,57693497	**0,13889692**	**0,11954765**	**0,08150921**	**0,04843345**	**0,02432034**	**0,01035689**	**0,99999944**	**F3 Binomial Q=1**
∑0 Poiss	∑1Poiss	∑2Poiss	∑3Poiss	∑4Poiss	∑5Poiss	∑6Poiss	∑≥7 Poiss	
0,625917694	**0,122378031**	**0,095642736**	**0,062799424**	**0,038774185**	**0,023203445**	**0,013552238**	**0,01647518**	**F3 Poisson Q=1 λ=1**
F3						6,2% pop id		
→0→0	→0→1	→0→2	→0→3	→0→4	→0→5	→0→6		
0,57693497	0	0	0	0	0	0	0,57693497	
→1→0	→1→1	→1→2	→1→3	→1→4	→1→5	→1→6		
0,0465163	0,0558195	0,0279098	0,0074426	0,0011164	0,0000893	0,000003	0,1388969	
→2→0	→2→1	→2→2	→2→3	→2→4	→2→5	→2→6		
0,0104953	0,0314858	0,0393572	0,0262382	0,0098393	0,0019679	0,000164	0,1195476	
→3→0	→3→1	→3→2	→3→3	→3→4	→3→5	→3→6		
0,0012736	0,0076415	0,0191037	0,0254716	0,0191037	0,0076415	0,0012736	0,0815092	
→4→0	→4→1	→4→2	→4→3	→4→4	→4→5	→4→6		
0,0000664	0,0007973	0,0039863	0,0106301	0,0159452	0,0127561	0,004252	0,0484335	
→5→0	→5→1	→5→2	→5→3	→5→4	→5→5	→5→6		
5,21E-07	0,0000156	0,0001955	0,0013032	0,0048869	0,0097738	0,0081448	0,0243203	
→6→0	→6→1	→6→2	→6→3	→6→4	→6→5	→6→6		
0	0	0	0	0	0	0,01035689	0,01035689	
∑0	∑1	∑2	∑3	∑4	∑5	∑6		
0,63528706	**0,09575972**	**0,0905525**	**0,07108568**	**0,0508915**	**0,03222859**	**0,02419431**	**0,99999936**	**F4 Binomial Q=1**
F4						14,5% pop id		
→0→0	→0→1	→0→2	→0→3	→0→4	→0→5	→0→6		
0,63528706	0	0	0	0	0	0		
→1→0	→1→1	→1→2	→1→3	→1→4	→1→5	→1→6		
0,0320697	0,0384837	0,0192418	0,0051312	0,0007697	0,0000616	0,0000021	0,0957597	
→2→0	→2→1	→2→2	→2→3	→2→4	→2→5	→2→6		
0,0079497	0,0238492	0,0298115	0,0198743	0,0074529	0,0014906	0,0001242	0,0905525	
→3→0	→3→1	→3→2	→3→3	→3→4	→3→5	→3→6		
0,0011107	0,0066643	0,0166607	0,0222143	0,0166607	0,0066643	0,0011107	0,0710857	
→4→0	→4→1	→4→2	→4→3	→4→4	→4→5	→4→6		
0,0000698	0,0008377	0,0041886	0,0111696	0,0167544	0,0134035	0,0044678	0,0508915	
→5→0	→5→1	→5→2	→5→3	→5→4	→5→5	→5→6		
6,91E-07	2,07E-05	0,000259	0,0017269	0,006476	0,012952	0,0107933	0,0322286	
→6→0	→6→1	→6→2	→6→3	→6→4	→6→5	→6→6		

0	0	0	0	0	0	0,02419431	0,02419431	
∑0	∑1	∑2	∑3	∑4	∑5	∑6	0,3647123	
0,67648775	**0,06985562**	**0,07016171**	**0,06011632**	**0,04811364**	**0,0345719**	**0,04069242**	**0,99999935**	**F5 Binomiaal Q=1**
F5						24,4% pop id		

On **Tabel 12** *2n=6 F0 singular* the homozygosis also is calculated out of the binomial distribution and the results here are also compared with the method of Wright. The character of this calculus is showed in the F1. The equality here also supports evidence that Wrights' formula is compatible with the binomial distribution and extinction, but not with the exponential extinction.

Tabel 12 *2n=6 F0 singular. Binomial homozygosis*

2n=6. Distribution alleles, gametes of F0 6x singular. Heterozygosis F0, Het=1								Real heterozygosis Het=1
→0	→1	→2	→3	→4	→5	→6	∑	
0	1	0	0	0	0	0	1	F0 distribution Q=1
0	6	0	0	0	0	0	6	F0 distribution all 6 alleles
0	0,166666	0	0	0	0	0	0,166666	Potential Hom. in F0
By binomial allele distribution in F0→ Het in F0=1-0,166..=0,833.. By Wrights' formula Het in F0=1x(5/6)^1= 0,8333..= Potential Het.								
0,33489797	0,40187757	0,20093878	0,05358368	0,00803755	0,000643	0,0000214	0,99999995	F1 verdeling Q=1
x6	x6	x6	x6	x6	x6	x6		
2,00938786	2,4112656	1,2056328	0,3215022	0,0482256	0,003858	0,0001284	6,00000046	F1 distribution all 6 alleles
x0	x(1/6)^2	x(1/3)^2	x(1/2)^2	x(4/6)^2	x(5/6)^2	x1		
0	0,0669796	0,1339592	0,08037555	0,0214336	0,00267917	0,0001284	0,30555552	Hom. in F1
By binomial allele distribution in F1→ Het in F1=1-0,30555=0,69444. By Wrights' formula Het in F1=1x(5/6)^2=0,6944..								
0,48797495	0,21958392	0,16013086	0,08417931	0,03510228	0,01096453	0,00206387	0,99999972	F2 distribution Q=1
2,92785	1,3175034	0,9607854	0,5050758	0,2106138	0,065787	0,0123834	5,9999988	F2 distribution all 6 alleles
0	0,03659732	0,10675393	0,1262690	0,09360613	0,04568542	0,0123834	0,42129515	Hom. in F2
By binomial allele distribution in F2→ Het in F2=1-0,4212951=0,5787049. By Wrights' formula Het in F2=1x(5/6)^3=0,5787037								
0,57693497	0,13889692	0,11954765	0,08150921	0,04843345	0,02432034	0,01035689	0,99999944	F3 distribution Q=1
3,46161	0,8333814	0,7172856	0,4890552	0,290601	0,1459218	0,0621414	5,9999964	F3 distribution all 6 alleles
0	0,02314948	0,0796984	0,1222638	0,129156	0,10133458	0,0621414	0,51774367	Hom. in F3

By binomial allele distribution in F3→ Het in F3=1-0,5177437=0,4822563. By Wrights' formula Het in F3=1x(5/6)^4=0,4822531

0,62346								
0,63528706	0,09575972	0,0905525	0,07108568	0,0508915	0,03222859	0,02419431	0,99999936	F4 distribution Q=1
3,8117226	0,5745582	0,543315	0,4265142	0,305349	0,1933716	0,1451658	5,9999964	F4 distribution all 6 alleles
0	0,01595995	0,06036833	0,10662855	0,13571067	0,13428583	0,1451658	0,59811913	Hom. in F4

By binomial allele distribution in F4→ Het in F4=1-0,5981191=0,4018809. By Wrights' formula Het in F4=1x(5/6)^5=0,4018776

0,67648775	0,06985562	0,07016171	0,06011632	0,04811364	0,0345719	0,04069242	0,99999935	F5 distribution Q=1
4,0589262	0,4191336	0,4209702	0,3606978	0,2886816	0,2074314	0,2441544	5,9999952	F5 distribution all 6 alleles
0	0,0116426	0,04677447	0,09017445	0,12830293	0,14404958	0,2441544	0,66509843	Hom. in F5

By binomial allele distribution in F5→ Het in F5=1-0,6650984=0,3349016. By Wrights' formula Het in F5=1x(5/6)^6=0,334898

In **Table 11** *2n=6* in F0 *two times in threefold* is a symmetric distribution with allele frequencies 0,5 in the F0 generation. Here the extinction chances are equal to the fixation chances like in table 2n=2.

Table 11 *2n=6* in F0 *two times in threefold*

Binomial 2n=6. Distribution alleles, gametes of F0 two times in threefold

→0	→1	→2	→3	→4	→5	→6	∑	
0	0	0	3	0	0	0	3	**F0 binomial Q=3**
F0								
3→0	3→1	3→2	3→3	3→4	3→5	**3→6**		
0,015625	0,09375	0,234375	0,3125	0,234375	0,09375	**0,015625**	1	**F1 binomial Q=3**
F1								
→0→0	→0→1	→0→2	→0→3	→0→4	→0→5	→0→6		
0,015625	0	0	0	0	0	0		
→1→0	→1→1	→1→2	→1→3	→1→4	→1→5	→1→6		
0,0313966	0,03767602	0,018838	0,00502347	0,00075352	0,00006028	2,009E-06	0,0937499	
→2→0	→2→1	→2→2	→2→3	→2→4	→2→5	→2→6		
0,02057613	0,0617284	0,07716049	0,05144033	0,0192901	0,00385802	0,0003215	0,23437497	
→3→0	→3→1	→3→2	→3→3	→3→4	→3→5	→3→6		
0,0048828	0,02929688	0,07324219	0,09765625	0,07324219	0,02929688	0,0048828	0,31249998	
→4→0	→4→1	→4→2	→4→3	→4→4	→4→5	→4→6		
0,0003215	0,00385802	0,0192901	0,05144033	0,07716049	0,0617284	0,02057613	0,23437497	
→5→0	→5→1	→5→2	→5→3	→5→4	→5→5	→5→6		

2,009E-06	0,00006028	0,00075352	0,00502347	0,018838	0,03767602	0,0313966	0,0937499	
→6→0	→6→1	→6→2	→6→3	→6→4	→6→5	→6→6		
0	0	0	0	0	0	0,015625		
∑0	∑1	∑2	∑3	∑4	∑5	**∑6**	0	
0,07280404	0,13261959	0,1892843	0,21058385	0,1892843	0,13261959	**0,07280404**	0,99999971	**F2 binomial Q=3**
F2								
→0→0	→0→1	→0→2	→0→3	→0→4	→0→5	→0→6		
0,07280404	0	0	0	0	0	0		
→1→0	→1→1	→1→2	→1→3	→1→4	→1→5	→1→6		
0,044414	0,05329684	0,02664842	0,00710625	0,00106594	8,5275E-05	2,8425E-06	0,13261956	
→2→0	→2→1	→2→2	→2→3	→2→4	→2→5	→2→6		
0,01661755	0,04985266	0,06231582	0,04154388	0,01557895	0,00311579	0,00025965	0,18928429	
→3→0	→3→1	→3→2	→3→3	→3→4	→3→5	→3→6		
0,00329037	0,01974224	0,0493556	0,06580747	0,0493556	0,01974224	0,00329037	0,21058389	
→4→0	→4→1	→4→2	→4→3	→4→4	→4→5	→4→6		
0,00025965	0,00311579	0,01557895	0,04154388	0,06231582	0,04985266	0,01661755	0,18928429	
→5→0	→5→1	→5→2	→5→3	→5→4	→5→5	→5→6		
2,8425E-06	8,5275E-05	0,00106594	0,00710625	0,02664842	0,05329684	0,044414	0,13261956	
→6→0	→6→1	→6→2	→6→3	→6→4	→6→5	→6→6		
0	0	0	0	0	0	0,07280404		
∑0	∑1	∑2	∑3	∑4	∑5	**∑6**	0	
0,13738845	0,1260928	0,15496473	0,16310772	0,15496473	0,1260928	**0,13738845**	0,99999967	**F3 binomial Q=3**
F3								
→0→0	→0→1	→0→2	→0→3	→0→4	→0→5	→0→6		
0,13738845	0	0	0	0	0	0		
→1→0	→1→1	→1→2	→1→3	→1→4	→1→5	→1→6		
0,04222823	0,0506739	0,0253369	0,00675652	0,00101348	0,0000811	2,7026E-06	0,12609283	
→2→0	→2→1	→2→2	→2→3	→2→4	→2→5	→2→6		
0,01360458	0,04081375	0,05101718	0,03401146	0,0127543	0,00255086	0,00021257	0,1549647	
→3→0	→3→1	→3→2	→3→3	→3→4	→3→5	→3→6		
0,00254856	0,01529135	0,03822837	0,0509712	0,03822837	0,01529135	0,00254856	0,16310775	
→4→0	→4→1	→4→2	→4→3	→4→4	→4→5	→4→6		
0,00021257	0,00255086	0,0127543	0,03401146	0,05101718	0,04081375	0,01360458		
→5→0	→5→1	→5→2	→5→3	→5→4	→5→5	→5→6		
2,7026E-06	0,0000811	0,00101348	0,00675652	0,0253369	0,0506739	0,04222823		

→6→0	→6→1	→6→2	→6→3	→6→4	→6→5	→6→6		
0	0	0	0	0	0	0,13738845		
∑0	∑1	∑2	∑3	∑4	∑5	**∑6**		
0,19598509	0,10941096	0,12835023	0,13250716	0,12835023	0,10941096	**0,19598509**	0,99999971	**F4 binomial Q=3**
F4								
→0→0	→0→1	→0→2	→0→3	→0→4	→0→5	→0→6		
0,19598509	0	0	0	0	0	0		
→1→0	→1→1	→1→2	→1→3	→1→4	→1→5	→1→6		
0,0366415	0,04396983	0,0219849	0,00586264	0,0008794	0,00007035	2,3451E-06	0,10941096	
→2→0	→2→1	→2→2	→2→3	→2→4	→2→5	→2→6		
0,01126806	0,0338042	0,04225521	0,02817014	0,0105638	0,00211276	0,00017606	0,12835023	
→3→0	→3→1	→3→2	→3→3	→3→4	→3→5	→3→6		
0,00207043	0,01242255	0,03105638	0,0414085	0,03105638	0,01242255	0,00207043	0,1325072	
→4→0	→4→1	→4→2	→4→3	→4→4	→4→5	→4→6		
0,00017606	0,00211276	0,0105638	0,02817014	0,04225521	0,0338042	0,01126806		
→5→0	→5→1	→5→2	→5→3	→5→4	→5→5	→5→6		
2,3451E-06	0,00007035	0,0008794	0,00586264	0,0219849	0,04396983	0,0366415		
→6→0	→6→1	→6→2	→6→3	→6→4	→6→5	→6→6		
0	0	0	0	0	0	0,19598509		
∑0	∑1	∑2	∑3	∑4	∑5	**∑6**		
0,24614348	0,09237969	0,10673968	0,10947406	0,10673968	0,09237969	**0,24614348**	0,99999977	**F5 binomial Q=3**
F5								

At table 12 *F0 in threefold* the calculation of the homozygosis with the binomial distribution again are compared with the calculation in accordance with Wright. Also here the results are equal. This now is more interesting than at the former table, because the start here is at F0 in a population with already some measure of homozygosis. The homozygosis in the F1 now is larger than in the former population, that started with singular alleles and the homozygosis in all the generations here is not exclusively caused by identity of the alleles by descent but also by general identical alleles. That is different in all the populations that start with singular alleles (Q=1). So this supports evidence for the point that **Wrights' formula describes the binomial extinction of the heterozygosis in general and not only for alleles that are identical by descent**, as it is suggested by some people. Different from the population with singular alleles here are many genotypes possible for F0, all with 2x3 alleles, or frequency 0,5 in a population with n=3. On the average however these genotypes here have a proportion

homo or heterozygosis of 0,5 according to the binomium (a+b)^2. The potential homozygosis should be here than this average homozygosis plus the potential selfing (0,5x1/6). That however is now not confirmed by the elaboration of the binomial distribution. The potential selfing in the F0 here is not expressed in the F1, which is different from the events at the singular population. You can pose however that the application of Wrights' formula simply is the operating with a geometric progression with the terms ar^0; ar^1; ar^2..ar^n, with the constant ratio r and the primary or scale factor a. If the value of a is 1 the series effectively begins at the second term ar^1 as is the situation in the singular allele population. If the value of a ≠1 as in the population with multiple alleles the series begins with ar^0. So it follows the factor between the terms of the first and second series is not a, but a/r. So the heterozygosis can be calculated beginning from the population with singular alleles in the F0 as Het={(2n-1)/2n}^g, whereas n= the size of the population and g= the generation number, so that in F0 g=1. Starting in the population with multiple alleles with average **heterozygosis**[14] H as Het = H.(2n-1/2n).{(2n-1)/2n}^g

Table 12 *2n=6 F0 2x in threefold. Binomial homozygosis*

2n=6. Distribution alleles, gametes of F0 2x in threefold.								Real Het is unknown
→0	→1	→2	→3	→4	→5	→6	∑	
0	0	0	3	0	0	0	3	F0 distribution Q=3
0	0	0	6	0	0	0	6	F0 distribution all 6 alleles
0	0	0	0,5	0	0	0	0,5	Gemiddelde Het. = 0,5
	0,5x1/6		0,5				0,5833…	Potential Hom. in F0
By the Wrights' formula Het in F0=0,5x(5/6)^0=0,5								
0,015625	0,09375	0,234375	0,3125	0,234375	0,09375	0,015625	1	F1 distribution Q=3
x2	x2	x2	x2	x2	x2	x2		
0,03125	0,1875	0,46875	0,625	0,46875	0,1875	0,03125	2	F1 distribution all 6 alleles
x0	x(1/6)^2	x(1/3)^2	x(1/2)^2	x(4/6)^2	x(5/6)^2	x1		
0	0,005208	0,052083	0,15625	0,208333	0,130208	0,03125	0,583333	Hom. in F1
By the binomial distribution in F1→ Het in F1=1-0,58333=0,4166667. By the Wrights' formula Het in F1=0,5x(5/6)^1=0,416667								
0,07280404	0,13261959	0,1892843	0,21058385	0,1892843	0,13261959	0,07280404	0,99999971	F2 distribution Q=3
0,14560808	0,26523918	0,3785686	0,4211677	0,3785686	0,26523918	0,14560808	1,99999943	F2 distribution all 6 alleles
0	0,00736776	0,04206318	0,10529193	0,16825271	0,18419389	0,14560808	0,65277754	Hom. in F2

[14]In this the homozygosis in F0 and the further generations is not exclusively by common descent.

By the binomial distribution in F2→ Het in F2=1-0,6652777=0,3472222. By the Wrights' formula Het in F2=0,5x(5/6)^2=0,347222.

0,13738845	0,1260928	0,15496473	0,16310772	0,15496473	0,1260928	0,13738845	0,99999967	F3 distribution Q=3
0,2747769	0,2521856	0,30992945	0,32621544	0,30992945	0,2521856	0,2747769	1,99999934	F3 distribution all 6 alleles
0	0,00700516	0,03443661	0,08155385	0,13774644	0,17512889	0,2747769	0,71064785	Hom. in F3

By the binomial distribution in F3→ Het in F3=1-0,7106478=0,2893522. By the Wrights' formula Het in F3=0,5x(5/6)^3=0,289352.

0,19598509	0,10941096	0,12835023	0,13250716	0,12835023	0,10941096	0,19598509	0,99999971	F4 distribution Q=3
0,39197018	0,21882191	0,25670046	0,26501432	0,25670046	0,21882191	0,39197018	1,99999942	F4 distribution all 6 alleles
0	0,00607839	0,02852228	0,06625358	0,11408911	0,15195965	0,39197018	0,75887318	Hom. in F4

By the binomial distribution in F4→ Het in F4=1-0,7588732=0,2411268. By the Wrights' formula Het in F4=0,5x(5/6)^4=0,2411265

0,24614348	0,09237969	0,10673968	0,10947406	0,10673968	0,09237969	0,24614348	0,99999977	F5 distribution Q=3
0,49228696	0,18475938	0,21347937	0,21894813	0,21347937	0,18475938	0,49228696	1,99999954	F5 distribution all 6 alleles
0	0,00513221	0,02371993	0,05473703	0,09487997	0,12830514	0,49228696	0,79906123	Hom. in F5

By the binomial distribution in F5→ Het in F5=1-0,7990612=0,2009388. By the Wrights' formula Het in F5=0,5x(5/6)^5=0,2009388.

Binomial and exponential extinction

As pointed out there is difference between the binomial extinction and the exponential extinction. The binomial extinction is found by complete elaborating of the superposition in the binomial distributions, but the exponential extinction can be found much easier and is calculated here in two ways. The first way of calculation is described at **Table 5c.** In this the primary exponential extinction is calculated from the primary intensity **μ** by **P0=**e^**-μ** and **μ** as **μ=−2n.ln(1-1/2n**). The recurrence of it is followed by the exponential accumulation and the intensities in the following generations are calculated by**−σ(Fg)=μν-μ** and the extinctions by **P0=**e^**−σ.** Different from **Table 5b** is the accumulating exponential distribution at **Table 5c** not a part of the Poisson superposition. This calculation only can be applied (by me) at the singular alleles at the F0 (Q=1). The extinctions of the multiple alleles (Q>1) cannot be summed in the recurrence in the way pointed out in the text at **table 7** of the Poisson distributions. In small populations the courses of the extinctions and multiplications of the various alleles are not independent of each other, as it does in the infinite population. The second way of calculation starts from the complementary chance. This is the chance that a marble, an allele will not been drawn in one drawing. This calculation simply takes this chance as a base and the allele survival, **α**, as an exponent. If for instance there are 6 different coloured marbles in the bag at the first drawing (2n=6 Q=1), the extinction chance for the total first drawing turn is (5/6)^6, because the 6 original marbles

are in the bag. At the second drawing turn the extinction chance is (5/6)^3,9906 now, because on the average for drawing were left 3,9906 of the original 6, namely 6{1-(5/6)^6}. Also if we start with the multiple alleles in the F0 (Q>1), for instance 2x3 marbles (2n=6 Q=3) the resting marbles do decide the exponent of the recurrence. So the formula is **{Q(1-1/2n)}^α**.. Both calculations of the exponential extinction are giving the same results, but I only can apply the second formula generally.

The differences between the exponential and binomial values are obvious not very large in the first generations, but they increase and will decrease afterwards, because they will converge to the same points. These differences probably are due to differences in taking the averages in the total line at the superposition of the distributions. The successive drawing turns or generations are described in the binomial superposition as separate processes and the problem then is that they are not independent of each other. In the binomial superposition the average of the events in one generation is ever taken as the basis for the calculations for the next generation. In this is not taken into account how deviations from the averages within the separate generations may change the total average through the course of all the generations as is the extinction and fixation process. This binomial extinction so is a compound process. In the exponential approach on the contrary the events through all the turns or generations are described within one uniform process, according to the fact the exponential accumulating distribution just is one distribution through the generations (see at **Table 5**). The deviations from the average within the uniform exponential distribution also are not described here, but they are simply determined within this distribution itself. The uniformity of the exponential approach will describe probably better what can be measured as extinction velocity in computer simulations.

Besides of the exponential extinctions also can calculated the exponential (or recurrence) fixations. These exponential fixations probably are easy to be calculated by Q.**(1/2n)^α.** Also the exponential heterozygosis extinctions can be calculated. The values of the exponential heterozygosis extinctions will differ somewhat from the binomials of Wright. These exponential values may have a better approach, but they probably are to be calculated more difficult than the binomial ones do with the simple Wrights formula. I did not yet found a way to calculate the exponential extinctions of the heterozygosis. So this remains yet as a challenge as well as the proofs for the exponential fixations and a good uniform description of the total exponential theory for the small population.

Table 5d

Table 5d *2n=2*

F0	F1	F2	F3	F4	F5	F6	F7	F8	
μ=1,38629	σ=1,03972	σ=0,89617	σ=0,82050	σ=0,77603	σ=0,74828	σ=0,73033	σ=0,71845	σ=0,71046	
P0=0,25	P0=0,35355	P0=0,40813	P0=0,44021	P0=0,46023	P0=0,47318	P0=0,48175	P0=0,48751	P0=0,49142	exponentiial
0,25	0,375	0,4375	0,46875	0,484375	0,49219	0,49609	0,49805	0,49902	binomial
F1	F2	F3	F4	F5	F6	F7	F8	F9	

Table 5d *2n=4 Q=1*

F0	F1	F2	F3	F4	
μ=1,15072829	σ=0,78663	σ=0,62671	σ=0,53584	σ=0,47735	accumulating exponential intensity
(3/4)^4	(3/4)^2,7344	(3/4)^2,1785	(3/4)^1,8626	(3/4)^1,6593	accumulating exponential extincton
P0=0,31641	P0=0,45538	P0=0,53434	P0=0,58518	P0=0,62043	accumulating exponential extincton
0,3164063	0,4632567	0,5484354	0,6037994	0,6422063	binomial extincton
F1	F2	F3	F4	F5	

Table 5d *2n=6 Q=1*

F0	F1	F2	F3	F4	
μ=1,093929	σ=0,72757	σ=0,56548	σ=0,47247	σ=0,41192	accumulating exponential intensity
(5/6)^6	(5/6)^3,9906	(5/6)^3,1015	(5/6)^2,5915	(5/6)^2,2593	accumulating exponential extincton
P0=0,334898	P0=0,48308	P0=0,56809	P0=0,62346	P0=0,66238	accumulating exponential extincton
0,334898	0,487975	0,576935	0,635287	0,676488	binomial extincton
F1	F2	F3	F4	F5	

Table 5d *2n=6 Q=3*

F0	F1	F2	F3	F4	
(3/6)^6	(3/6)^3,9906	(3/6)^3,1015	(3/6)^2,5915	(3/6)^2,2593	accumulating exponential extincton
0,015625	0,062909	0,116506	0,165918	0,208878	accumulating exponential extincton
0,015625	0,072804	0,1373885	0,1959851	0,2461435	binomial extincton
F1	F2	F3	F4	F5	

Some Practical conclusions

It is obvious that -for the absolute numbers Q- the total sum of the extinctions and fixations together of the alleles in a population of **limited** size n converges through the generations to the values the extinction has in the **unlimited** population

according to **table 5** and as described further on in the application of the recurrence if Q≠1. In somewhat larger populations (ca n>50), so with small fixation chances the extinctions do differ little from the extinction by **Table 5** in the unlimited H-W population. This however is applicable for the **quanta, the absolute numbers** and in this it is obvious that in a large and in a practical unlimited population only in relatively extreme small numbers of alleles talk could be of observable random extinction. In the unlimited H-W population the extinction of the neutral singular allele goes rather slow, as is pointed out in table 5: At the 2nd generation 1/e, so already 37% of the alleles has been vanished, but only after 200 generations 99% has been disappeared. Alleles that occur in multiplicity, Q>1 in F0, will have after 2Q generations an extinction of about 1/e, so 37% and after about ca 200Q generations they will have an extinction of about 99% and a survival of 1%. This mostly is a very, very long time because this all is about the absolute quantities Q of alleles in the populations and alleles, neutral or of some biologic importance in general do occur in large quantities within large population. So the expectation is that the alleles, which occur in measurable frequencies, never will vanish from large populations, even if the frequencies are very small. Also in a feeble selection against this allele it will disappear very difficult or not from a large population. This is general the case at recessive alleles, that are observed in the large population because of the very seldom occurrence of homozygous organisms.

Suppose an allele occurs homogeneous in the world population, n=5.10^9 and once a year one homozygous child is born on 10^8 births with the lethal marks of the recessive allele. The absolute quantum of the allele now is easy to be calculated, it is 500000, by the allele frequency (10^-8)^0,5. By the selection every year will vanish perhaps 2 alleles, which is practical nothing. So globally the extinction will last about 5.10^7 generations to reach a change of 99%. So from the perspective of the survival times of individuals, species or even total planets such an allele will never disappear.

In large homogenous populations extinction only is to be expected for alleles with very small quanta if they are neutral or **recessive**. In large populations the very small quanta do occur only the case of new mutations and as **dominant** alleles and further observable genetic changes are excluded in large populations at homogeneous reproduction. **Important changes in frequencies of genes with limited or none expression are excluded in large homogeneous populations, this concerning changes by selection as well as at random.** Total recessive alleles only are sensitive for selection, if they occur in the population in such high numbers, that the presence of homozygosis is evident. Changes in the frequencies of genes with an obvious expression in the functions of the organisms at their changes on survival and behaviour in the reproduction are yet possible, but this concerns a very limited number of genes, so that there is no talk of a general micro-evolution in large homogeneous populations. That is why evolution within large populations only is possible if they are not homogeneous by place and so exist out of demes, or by time and so are fluctuating strong in number.

The flexible small quanta do occur of course in small populations. In this is the extinction of the singular neutral allele within the limited population only is a little slower than in the unlimited H–W population and there only are observable differences in very small populations. **So by the neutral theory it easily is to be seen how fast genetic changes (by selection or random) can take place within small populations and demes or by inbreeding. So the extinction is of great importance in small populations and the study of it as the zero-hypothesis of the selection is indispensable.** The allele extinction are to be calculated very well within limited and small populations and at non random inbreeding, but why this is not be done? Why is the extinction not described in the manuals and why she is considered as irrelevant? Are even the sciences for a big part fashionable parroting and a product of a narrow-minded and dependent way of thinking?!

The conclusion that micro and so also macro-evolution only can take place by reproduction within small demes or small populations, by reproduction with non random inbreeding and with selection by sexual behaviour leads also to the conception that the living organisms themselves actively are concerned in their evolution. So in the evolution does not exist something like a sieve that is screening the organisms in their struggle for live and decides which organisms or alleles can go through the meshes and which can not. No, the struggle for live is not suffered by the organisms, but is pursued by them and they decide how it is executed. The evolution is an integral part of the existence of the organisms. The chemical functioning of the DNA in joining with all the processes of live let the organisms themselves decide and regulate how their evolution is, yet:
An organism exists, because it can survive in its life surroundings, otherwise it could not exist.
An organism exists, because it can procreate, otherwise a mortal organism could not exist.
An organism exists, because it can change itself through the generations, otherwise it could not exist in the ever changing life **surroundings**[15]**.** So there is evolution because the organisms **can change**[16] their genes, for if they could not do so…**.** So the organisms do participate actively in the evolutionary changes and these are regulated within the processes of the living

[15] By a lot of micro and macro factors the live surroundings and so the life conditions are changing ever: By the annual alternation of summer and winter or wet and dry seasons and on the longer term the periodical climate is changed by astronomical factors as the precession of the earth's axis and the variable elliptic form of the earth's orbit. There also are sudden changes in the surrounding by irregular and unpredictable factors as are droughts and floods, but also by global factors as an impact of a meteor and a mega volcanic outburst. For all the living organisms on earth the circumstances of live are changing ever more and they will have to change by this, but they are affecting each other in this process, so giving a snowball effect to the changing surroundings..

[16] This is a global conclusion on the basis of generalisations, but the fact that we do not know yet **how** the organisms can change their genes nowise is an guilty negation of this lemma that they do so, because ignorance never is an argument.

organisms, in their organic functions. The micro-evolution is an integral part of the life functions by joining as: genetic decided behaviour at the procreation → the measure of inbreeding and sexual selection → genetic changes → behaviour at the procreation, etc. The evolution takes place within the many small and large ecological unities, so the small and large syntheses of the many species of organisms on the one side and on the other hand the changing existence of the vehicle of their life, the planet Earth.

That active participation of the living organisms in their evolution makes the study of the macro-evolution so very fascinating. Although there are yet many challenges in the micro-evolution I now will make a study of especially the macro-evolution to find out the possibilities to get here also somewhat more clarity in the vagueness.

Some notions

Out breeding: The complement of inbreeding. So this can be relative but often the meaning here is absolute in the sense of: Random mating in the infinite Hardy-Weinberg population, or absolute no common ancestors.

Inbreeding, absolute (and relative): Relationship (more than average) between the parents, because they have common ancestor or they themselves are genetically identical.

Effective size population of reproduction: The number of individuals participating effectively in the reproduction. So they have children that get children of themselves. Individuals having no children are not counted and parents that have children but get no grandchildren are parents with 0 children etc. Elucidation: Inbreeding determines the average expected number of descendants of the common ancestors, So inbreeding determines the effective population size. However, different from S. Wright and M. Kimura I think that a variation in the parities larger of smaller than random expected can not determine the size of the effective population. This should be than an ideal or virtual population, but you can only work in science with **perceptible** populations, so real or possible physically existent populations. Non random differences in the results by individuals participating at the procreation is yet in the picture as the only possible source of the selection. Also the inequality men and women in the participation of the procreation is usually equalized in the formula for the effective reproduction. In this study this also is not a matter of course or necessity. The population of reproduction here is the concrete or possible progeny of one or more individuals.

Parity: In obstetrics the number of deliveries an (expectant) mother has passed through. In this context the number of (effective) children of one parent and also the number of exemplars of one allele, transferred from one individual in one generation.

Replacement factor: This ratio **r** gives the number of descendants that replaces one individual in the following generation(s) in neutral population dynamics. The value of **r** per generation is $1 \leq \mathbf{r} \leq 2$. This value of **r** is decided by the form of reproduction. In asexual reproduction $\mathbf{r}=1$. In absolute out breeding $\mathbf{r}=2$. In inbreeding $1 \leq \mathbf{r} < 2$.

Elucidation at the literature.

So I tried to develop a simple uniform system that can easily be followed and that give some insight in the figures at the complicated steps of the biologic substrate. This elementary system that uses the versatile abilities of the transcendent number e can extend to more applications. The sciences and mathematics are uniform so there are of course many ways leading to the same results. In this it is not necessary and sometimes even not desirable to consult and pursue always the ideas of other people in the literature. So I did not do this, but if I try to compare afterwards this calculus with the literature I get the idea this is a particular way and it describes new possibilities. I did make some study of the literature of particular Kimura after a useful advise of dr Gerdien de Jong of the Utrecht University. I can definitely not work out the discussion between this and the different calculations in the literature. My knowledge of the maths is much too small for this job and is not prone to improvement because I am in the age of 60 and have other ambitions. Nevertheless, glancing through the literature I found some evidences this system should give more and easier results. So you should see this list of the literature as superficially consulted for their results and sometimes for their essentials and also for you as a possibility to link to interesting information. As pointed out I used as a source for this study only the manual of Scott Freeman with other general knowledge and the data of the US census bureau.

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