somes which impairs the survival of the gametes and affects the threshold of genes.

It is significant to note that a high level of hyperdiploidy (tetrasomy?) was achieved in the progeny of a desynaptic mutant induced by gamma irradiation. Although the production of trisomics and occasional tetrasomics has been recorded in a variety of plant types from desynaptic lines[4-7], the increase in chromosome number has been limited to the addition of 1–2 chromosomes. Gottschalk and Milutinovic[5], however, recorded a few hyperploid cells reaching near triploidy in the cell population of an otherwise trisomic plant in *Pisum sativum*. The present study records a case going much further. Thus, there is reason to believe that the original mutant might generate progeny of different chromosomal races and various degrees of fertility.

Frequency and pattern of chromosome distribution at anaphase I

	Sum of chromosome Nos at both the poles				
	48	50	52	54	others between 48–54
Frequency of occurrence	22.3%	37.2%	18.1%	16.1%	6.3%

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Fluctuating rates of evolution

O. Mayo and T.W. Hancock

Biometry Section, Waite Agricultural Research Institute, The University of Adelaide, Glen Osmond (Australia 5064), 9 July 1984

Summary. Constant evolutionary rates are possible only in very large populations, where natural selection does not exhaust variation because mutation supplies fresh variability. In a small population where a small number of genes influence an integrated system like brain and body size which have an allometric relationship, variation is removed rapidly under natural selection, This occurs even when the final fitness of the population is not optimal.
Key words. Evolution; evolutionary rate; stasis; brain; encephalization; body size; fitness.

The concept of 'punctuated equilibria'[1] is a misconception in at least one way. It is based on a rejection of the idea that observed or apparent slow, gradual evolutionary change must be at a constant rate. Given the precision with which evolutionary rates may be measured[2], it would be a rash person who would suggest either that constant rates had been observed or that rates fluctuating about zero had been observed, with large steps at intervals. What does not seem to have been considered in attempting a new 'modern synthesis' is the very straightforward idea that when mutations arise which are favorable, they will be selected steadily until they are fixed, after which stasis may occur, if there is no further genetical variability available, or further change may occur.

This can be the case whether the changes are major or minor. Furthermore, slow change may occur or rapid change may occur without affecting the fossil phenotype, giving rise to the assessment of stasis when changes was the case, or change may occur in a relatively trivial attribute detectable in the fossil record, giving rise to the idea of important change, on account of the inability to assess the importance of the change.

In this note, a model is developed of evolutionary change affecting an integrated system, and it is shown how relatively rapid change may occur within the system, all change occurring by standard Darwinian natural selection, but with breaks in between periods of change, these breaks occasioned by the absence of genetical variability.

The model. Consider the evolution of the human brain. Whatever one's view of man's place in nature, one cannot but agree that man's ability to record his introspection for posterity differs in kind from that of any other living organism. While such records cover no more than perhaps $\frac{1}{10}$ of 1% of man's evolution, man's increasing encephalization is documented imperfectly in the fossil record for perhaps 10 million years. This represents perhaps half a million generations, and there appears to have been no significant change over the last 10% of this time. Given the slowness of even this very rapid change, of course, there may have been undetectable changes during this most recent period. Such changes are not relevant to the present argument.

As has frequently been mentioned by many authors[3], evolutionary change in one organ must be accompanied by accommodation in other organs. Let us therefore set down arbitrarily a simple model for the ontogenetic determination of human brain and body size, and see how this changes under natural selection.

Suppose that there are 15 genes which determine various aspects of size. Three affect the brain only. Three affect brain and body equally. Three affect overall size. Three affect body and pelvis size equally. Three affect pelvis size only.

Now suppose that there is stabilizing selection on overall size. Then if directional selection on brain size occurs, this will interact with the stabilizing selection on overall size.

In the model (table), we shall ignore factors like development instability[4-6] as direct influences but such factors could clearly be of great importance, for example in contributing to selection against extremes.

There have been extensive analyses of the single locus model of the interaction between artificial directional selection and natural selection favoring intermediates, whether through an intermediate optimum or heterozygous advantage; see Nicholas and Robertson[7] for details. These analyses, while generally illuminating, are not directly relevant to the present case where the hypothesized genes have distinctive functions.

Hence, stabilizing selection intensity $= k_1 (1 - \exp(- (n_s - 9)^2))$ where n_s = total number of genes increasing phenotype in categories 2, 3 and 4,

Gene type	Phenotype influenced by a gene	Number of genes	Type of selection
1	Brain	3	a) Directional; advantage proportional to phenotype
2	Brain × whole organism	3	b) Truncation; a mismatch of brain (and head) size × reproductive tract size leads to death of parent and offspring
3	Whole organism	3	Stabilizing; deviants disadvantageous proportional to square of departure from optimum phenotype
4	Whole organism × pelvis	3	
5	Pelvis	3	Truncation; as above

i.e. fitness $= 1 - k_1(1 - \exp(-n_s - 9)^2))$,
directional selection intensity $= k_2 n_d$,
where n_d = total number of genes decreasing phenotype in categories 1 and 2,
i.e. fitness $= 1 - k_2(n_d - 6)/b$,
truncation selection occurs when
$a + k_3 n_d > b + k_4(n_4 + n_5)$,
when n_4 and n_5 are the total numbers of genes increasing phenotype in categories 4 and 5.
It is also possible to incorporate random environmental variation in any trait.

Results. Values of population size, N, were 50 and 100. The parameters k_1 and k_2 took the values 0, 0.01, 0.05, 0.1 and 0.2. Initial gene frequencies of 0.5 were used throughout for all 15 genes because 0.5 lies in the part of the gene frequency space where modification of major gene effects by minor genes is likely to be most effective[8]. With 15 genes initially segregating, each one is individually of modest effect.
Extensive simulation trials were carried out, using the methods of Mayo[9]. Simulations were carried out for 1000 generations or until fixation of all 15 genes occurred, whichever was the sooner. The results may be summarized as follows.
In all cases, the genetical and phenotypic correlation for brain and body sizes were in the range 0.7–0.9, reasonable values for such traits (see Cheverud, Rutledge and Atchley[10] for references).
For $k_2 = 0.2$, brain and pelvis sizes increased on average but body size did not increase for $k_1 \geqslant 0.1$. For $k_1 \leqslant 0.05$, there was an increase in body size, but not in brain or pelvis size.
For $k_2 = 0.1$, there was no change in brain size but increase in body and pelvis sizes.
For $k_2 = 0.05$, results were very similar to those with $k_2 = 0.1$.
For $k_2 \geqslant 0.01$, a decline in brain size occurred, with modest increases in body and pelvis size.
All these results are assessed by the mean number of alleles fixed which increased the trait in question relative to the initial intermediate state.
The time to fixation excepted for a neutral allele at a frequency p is given by $T = -4N(p \ln p + (1 - p)\ln(1 - p))$. For N = 50, $T \simeq 138$ and for N = 100, $T \simeq 275$. In most cases, mean time to fixation was accelerated relative to the appropriate expectation, i.e. it was of the order of 100 generations for N = 50, 200 generations for N = 100, except where one unfixed gene was strongly influenced by the stabilizing selection, in which case segregation persisted. Thus, the overall effect of selection was to eliminate variability where this allowed an intermediate optimal phenotype to be fixed[11,12].

Discussion. In most cases, the simulated populations evolved to optimal fitness as regards stabilizing selection, but only achieved optimal fitness as regards brain size where directional selection was strong. This is in agreement with the results of Lande[13], who showed that strong and sustained selection is necessary for a major gene affecting a quantitative trait to be fixed, where there are significant deleterious pleiotropic effects. Pacquin and Adams[14] showed that such epistatic interactions in fitness can lower mean fitness even in asexual populations of the yeast *Saccharomyces cerevisae*. Thus, if major genetical changes occur by natural selection as a result of a change in the environment, directional selection for some other traits may halt on account both of the low mean fitness and the low numbers of offspring among whom selection can occur and of the bulk of selection being of modifiers of defective but necessary genes.
Since the amount of genetical variance in a quantitative trait attributable to recent mutation has been estimated as no more than 0.1% of the environmental variance, artificial selection response attributable to new mutants is likely to be small over time intervals less than 20 generations[15]. Hence, fixation of a major gene or a number of major genes, which is rapid in a small population, even where there is considerable stabilizing selection, may be expected to be followed by a period of stasis, as mutation and recombination regenerated the variance in the trait. Under natural selection, the process will be similar but much slower. Depending on population size and environmentally induced variability, the alternation of periods of stasis and rapid change will appear more or less smooth in the fossil record. However, since the periods of rapid change may be expected to last less than 1000 generations and the periods of stasis to be of similar or greater duration, the chance of observing a very smooth continuous sequence of 50,000 generations or more is small. Over longer periods, the change of scale may make a series of the type predicted appear smooth.
For hominid brain size, the fossil record is by no means complete, but there has certainly been no simple single lineage with gradual increase in brain size from the australopithecines to *Homo sapiens*[16,17]. (Indeed, *Homo* was simultaneous with some of the australopithecines.) Now we know of interacting systems of genes which would allow change in some components of the phenotype but not others, or allow some change in a component of interest, yet leave genetical variation[18].
Hence, we can accept either change within a lineage or at a bifurcation in a lineage as a possibility in the evolution of the human brain.
Brain size increase and brain structural reorganization are both aspects of brain evolution, and there is evidence that they were not necessarily simultaneous. Holloway[19], for example, has suggested that some degree of human cerebral organization occurred 3–4 million years ago. Lande[20], from a theoretical investigation of brain-body allometry, has concluded that short-term brain-body size differentiation in closely related taxa results mainly from selection for altered body size but that in the long term, selection directly affecting brain size has been more important. As a specific example of the latter process, Lande suggests that selection for increased encephalization in primates will have decreased the genetical correlation between brain and body sizes, hence facilitating further encephalization. Our results shed little light on these conclusions but do not disagree with them.

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Decomposition of toxic and environmentally hazardous 2,3,7,8-tetra-chlorodibenzo-p-dioxin by gamma irradiation

H.R. Buser and H.J. Zehnder

Swiss Federal Research Station, CH-8820 Wädenswil (Switzerland), 4 September 1984

Summary. The decomposition of the toxic and environmentally hazardous 2378-TCDD by gamma irradiation was studied and successfully used to decontaminate laboratory wastes containing small quantities of this chemically and biologically stable compound. The method makes use of gamma irradiation from a commercial ^{60}cobalt facility at high dose levels (1000 kGy) to break down the compound into nontoxic products. Irradiation also decomposed 2378-TCDD in contaminated soil from the Seveso accident.
Key words. 2,3,7,8-Tetrachlorodibenzo-p-dioxin; hazardous waste; ionizing radiation; gamma irradiation; decomposition.

2,3,7,8-Tetrachlorodibenzo-p-dioxin (2378-TCDD) belongs to a group of toxic and environmentally hazardous compounds, the polychlorinated dibenzo-p-dioxins (PCDDs). There are 75 such compounds ranging from the mono- to the fully chlorinated octachloro species. The symmetrically substituted 2378-TCDD is apparently the most toxic congener of this group[1,2]. 2378-TCDD is an unwanted byproduct in the synthesis of certain industrial chemicals. It is one of the most toxic man-made chemicals and has no technical use or value. Several industrial accidents have occurred in which 2378-TCDD was formed and sometimes released into the environment, i.e. in the accident in Seveso, Italy, in 1976[3]. In all these incidents the synthesis of 2,4,5-trichlorophenol was involved. This industrial product is used for the production of herbicides, bactericides and wood preservatives. The safe disposal of residues and wastes from these productions poses a formidable toxicological and environmental problem. PCDDs have recently also been found as trace contaminants in emissions from incineration or combustion sources. In the case of municipal incinerators the main contaminants were not 2378-TCDD but other PCDDs[4].

Incineration or deposition of wastes containing 2378-TCDD or other PCDDs may involve significant risks because of the possible emission of these hazardous compounds into the environment. Additionally, incineration or combustion is a problem in such cases where highly stable compounds are involved and toxic by-products can be formed. This is the case with the polychlorinated biphenyls (PCBs) which can be thermally converted into the polychlorinated dibenzofurans (PCDFs), an other group of toxic and environmentally hazardous compounds, closely related to the PCDDs[5].

Irradiation of these organochlorine compounds has been investigated previously. Photolysis using UV- or sunlight was found to dechlorinate 2378-TCDD and other PCDDs in pure solutions but was largely unsuccessful when these compounds where absorbed on soil or in the presence of large amounts of other materials[6,7]. Gamma irradiation has previously been applied for the decomposition of PCDDs, PCBs and other organochlorine compounds using solutions of the pure substances[8-12]. Considerable irradiation doses (up to 1000 kGy) were required, and the degree of success varied; practical applications were not documented. In this report we show the successful use of gamma irradiation for the decomposition of 2378-TCDD and other hazardous compounds in laboratory wastes and in small quantities of contaminated soil from Seveso.

Materials and methods. A commercial gamma irradiation facility (Sulzer, Winterthur, Switzerland) consisting of 14 ^{60}Co-rods with an activity of 10^{15} Bq was used. The rods were arranged in circles of 35 or 90 cm diameter. The incident dose was measured with Clear Perspex HX dosimeters, 3 mm (Gillette UK Ltd, Reading, England), based on Fe-sulfate dosimetry. The dose rates ranged from 1.6 to 8.0 × 10^3 Gy/h. Exposure periods were up to 280 h with doses up to 1148 kGy. Samples were exposed to different radiation doses by varying their distances from the ^{60}Co-rods.

Synthetic 2378-TCDD (96%, Givaudan Ltd, Dübendorf, Switzerland) was dissolved in n-hexane at a concentration of 0.5 µg/ml and portions of 1 ml exposed to gamma radiation (dose rate 1.6 kGy/h) in sealed borosilicate vials (3 ml volume, air not removed). The vials were placed inside an additional screw cap flask for safety reasons.

Laboratory waste solution containing 2378-TCDD, other PCDDs as well as additional components was carefully concentrated to about 200 ml and exposed to gamma radiation (dose rate 5 kGy/h) in a 250-ml Sovirel flask. This flask was placed in a plastic bag and a metal container filled with an absorbing mate-