Memory Effects, Maladaptation and the Evolutionary Advantage of Nostalgia.

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Abstract

Through consideration of the integrity of information transmission between generations as described by genetic and cultural transmission mechanisms, we construct a notion of temporal domains over which selection mechanisms can naturally act. Adapting a phenotype centred dual inheritance model of guided variation, we incorporate the action of the fitness measure over these domains as effective memories of previous optimum phenotype values in addition to augmenting traditional learning objectives. On simulating the evolution of a population, the arising conflicting pressures are shown to guide a population between the specified optimums, appearing under Natural Selection assessment as maladaptation. The evolutionary advantage of genetically determined learning goals is found to be advantageous in special cases under cultural pressures and Natural Selection.

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1 Introduction.

Evolution, the variation of one quantity w.r.t. time, is in the context of human genetic, individual and social evolution, an object of fascination and study. Today we have the multi-faceted Modern Evolutionary Synthesis representing decades of work building on that of Charles Darwin and Gregor Mendel. It focuses on the mechanisms that describe the variation of sets of genes carried by individuals across generations, identifying several preconditions and drivers of genetic evolution such as mutation, drift, flow, recombination and Natural Selection. Darwin recognized the necessity of phenotypic variation within a population upon which Natural Selection(henceforth N.Selection) could act as a differential selector. He observed the correlation between phenotypic traits of parents and offspring and while having no knowledge of genetic structure as sets of Mendelian units, he grasped the concept of heredity and thus considered the phenotype to be the unit of selection under this differential selector, simply understanding that through heredity there was some mechanism responsible for the transmission of phenotypic traits from parent to offspring. Through the recognition of heredity, sources of variation and differential selection taken, this was a remarkable leap in the understanding of human evolution, but was far from complete. Today the Mendelian unit of the chromosome and allele has taken the place of the biologically heritable unit which determines an individual's genotype and we often consider the phenotypic degeneracy of the genotype to be raised by cultural and environmental 'pressures' upon which N.Selection can act. The distinction between genetic and cultural effects has given rise to Dual Inheritance Theories (DIT's) that incorporate the action of culture and genetics on the distribution of either continuous or Mendelian phenotypic traits in a population.

Motivated by the observation that N.Selection appears to promote seemingly, at least temporally local maladaptation in individuals, we seek to examine potential explanations for the evolution of maladaptive traits of the individual's phenotype. An issue with DIT's is that they tend to be constructed, not necessarily naturally I think, to consider explicitly only the generational transmission mechanisms of culture and genetics on the phenotype so that N.Selection can act on its natural generational domain. While there is no intrinsic problem with this if we consider N.Selection to be the dominant selection mechanism, we argue that it does neglect the action of the biological selection hierarchy, under which it is possible to consider selection mechanisms acting across the biological/ecological scale and the wealth of new phenomenon that these can bring at the scale of the individual phenotype that is in no way manifest in traditional DIT's.
In this thesis we adapt a guided variation DIT process to incorporate cultural and genetic memory effects into the learning goals/individual's perception of optimum phenotype and hence perception of fitness at this scale. Running a simulation of this process we find the action of N.Selection forms pressures conflicting with the pressures caused by the genetically and culturally determined perceived optimums. Under cultural selection pressures and natural learning goals we find that during selection events will almost always favour those that rely most heavily on social learning, while for a population who rely equally on social and asocial learning we find that both cultural selection **and** N.Selection, can favour geneti-

cally defined learning goals with greatest adaptive advantage measured by both cultural and natural fitness given to those whose effective genetic memory has greatest temporal domain.

Part I

Building a DIT Model.

2 The Chaotic Problem.

As part of an ecological system, the human civilization exhibits rich, dynamical and chaotic behaviour from the scale of a chromosome, through that of the individual, community to that of the civilization. We often want to relate the macro to the micro scale when modelling systems such as these, but the frequent problem here is that by scale, we often mean that we want to both relate large to small space-time scales and simultaneously find a relation through the increasingly complex behaviour of the system as we go to the smaller scale necessary to describe it.

We may have rules that describe the behaviour of a system at one scale and its constituents at a smaller one, but the constituents at the smaller scale when taken together become chaotic and relations between these rules are often evasive - like trying to model a planets' orbit quantum mechanically. The increasingly complex behaviour considered at the smaller scale necessary to describe behaviour at the higher in greater detail often make modelling complex phenomenon in a meaningful manner impossible. For models describing behaviour at different scales this means that simply through relative loss of degrees of freedom then found at the larger scale model it only makes sense to talk of a description of the larger in terms of the smaller and not the other way around. It is not that actual physical degrees of freedom have been lost, but we choose to consider the larger as a constrained system when modelling. This could be considered a notion of 'scale causality' when modelling. In attempting to relate even simple macro human behaviour to the micro such as genes $\rightarrow$ genotype and phenotype $\rightarrow$ population, It may be fair to say represent the biggest challenge to modelling cultural dynamics in a deterministic sense.

There are however many examples of chaotic systems that allow a few useful quantitative statistical measurements to be related across scales; the application of statistics to Thermodynamics is an excellent one, allowing a relation between the microscopic variables of $m, N, \bar{v}$ and the macroscopic state variable S, P, T and V[1]. This is the kind of relation that when building a DIT model we will be forced impose on our system. If we did not have the problem of chaos then we could simply describe all behaviour at all scales with a set of micro initial conditions and we would be done with meaningful evolutionary theory (amongst many other things). The partial motivation for the consideration of selection mechanisms acting at specific physical and biological scales is that, foregoing an arguments on self-similarity, the only set of rules that we have that is simple, elegant and consistent to some extent with

[1] m = article mass, N = no. of particles, $\bar{v}$ = mean particle speed,S = entropy, P = pressure, T = temperature and V = volume.

either observation or established conjecture -'selfish' gene model and group selection are not new concepts. We speculate that the application of rules such as these through scales has potential for removing some of the chaotic behaviour of models when trying to find relations across scales.

3 The Principle of Natural Selection, Group Selection and Genetic Selection.

The principle of Natural Selection as the term coined by Darwin in his 1859 book 'On the Origin of Species' describes a selection mechanism under which evolutionary forces act, culminating in the generational reproduction process under which successful biological traits can be passed from parent(s) to offspring increasing the frequency of the most successful, or fittest genotypes through this form of differential selection. Under conditions of the introduction of genetic variation this will result in a non-static evolutionary process. This mechanism's domain/unit of selection is the set of phenotypes within a group; it describes a single genetic iteration and it implicitly refers to a measure of temporally local phenotype fitness. It assumes a correlation between genotype and phenotype, otherwise it would have an evolutionarily neutral action. We do observe this correlation of course; without it Darwin would have had no notion of heredity. The important point here is that N.Selection can be considered to act on a generational timescale and its unit of selection is the phenotype - the variation of the genotype's vertically transmitted gene program is induced by this action. What this represents is the association of both a unit of selection and also temporal domain within which the selection mechanism acts - the phenotype is selected for over one generation. There is no selection considered here as occurring at the scale of the genome unless we are referring to the *induced* inter-generational genetic variation. Often group level behaviour is considered as being induced by the operation of N.Selection on the individual as well. Of course genome and group scale behaviour is not independent of that of the phenotype, but considering the action of only one selection mechanism on one unit of selection, we speculate risks omitting a wide range of behaviour at the all scales when model building. For reference here we will call this type of model in which genome and group scale behaviour, a 'phenotype centred model' (PCM).

It would be especially convenient for the purposes of modelling a chaotic system to have a rule analogous to N.selection that could be applied to groupings across scales of human society as a guiding mechanism. If this could be done, it could reduce the problem of relating evolutionary civilisation level behaviour to an individual's genetic behaviour to one of relating the former to the latter through processes governed by selection rules acting at scales in between.

Group selection behaviour is indeed observed on many scales, from the family unit, community to nation and species as examples. It seems natural to extend the principle of selection to include corresponding such selection units as groups and species for this purpose - the fittest should always be favoured?! Athough when modelling, the fitness measures may have to exist in different spaces corresponding to each scale mechanism. Richard A. Watson dis-

cusses the issue of selection acting at scales in his paper 'On the Unit of Selection in Sexual Populations' [7] where he discusses competing processes of fitness optimization at different scales in the genome region. The essence of this generalisation means re-interpreting selection as more of a generalized physical principle acting with the same differential selection function on groupings across scales and temporal localities more than solely the biological principle of Natural Selection.
Part of the motivation for the consideration of this generalisation of selection and unit is that due to the inductive effect of behaviour through scales specified in our case by genetic and cultural transmission rules, we would expect that measures of fitness for a unit under one selection mechanism could cause a maladaptive behaviour of another unit at another scale under another selection mechanism. This sets the stage for the consideration of a human ecology as a system of interacting selection units under scale dependant selection mechanisms.

4 A Conceptual Construction of Effective Memory

When considering the action of N.Selection we implicitly refer to its cumulative action across an individual's life cycle. We do not refer to it acting on a specific moment in this cycle though we understand it to be the case that its action is continuous across the life cycle; we say that on a generational time scale, an individual is operated on under N.Selection. If we applied a measure of fitness as considered by this mechanism to the daily activities of an individual, we would first and foremost become confused as to how this would be done as we have no functional notion of the unit of selection being acted upon in such a continuous manner. Conceptually, we might be able to say that there is an associated temporal domain to the unit; we understand that a phenotype is able to adapt, learn and improve or reduce their fitness throughout their life cycle - the selection mechanism acts on the unit's history, culminating in a measure of success defined by an individual phenotype's eventual action in passing on their genetic material. In the case of the individual's phenotype under N.Selection, there is a convenient line to be drawn; an individual is born, lives, and passes or does not pass on this material and we talk of their fitness in the context of model building as the probability of surviving and success in pass on their genetic material. Looking at the speculative corresponding group selection process acting on a pre-cultural species, we could consider the group unit, but there is no such natural temporal domain. If we consider humanity, we possess a cultural capacity; the ability to *transmit information* that we have acquired through some learning process socially. This does give us a notion of a temporal domain of the cultural group unit. Cultural knowledge and information, perhaps important to the survival of the group can be transmitted horizontally and vertically generation by generation. We can speak of an effective vertical/temporal cultural range in terms of the rate of degradation of this information as it is transmitted between phenotypes within the group. This is the basis of the *memory effect*; the ability of a unit of selection to adjust its fitness across time scales - the temporal domain of the unit. In analogy with the action of N.Selection acting across a phenotype's life cycle where they have the ability to use their experiences or history to adapt and improve their fitness, we might be able to speak of a cul-

ture's ability to adapt on a timescale of many generations while being acted upon by group selection. This gives at least some notion of the temporal domain of the cultural group unit. So in the same way, the single or multi-generational memory capacity used by the individual and the cultural group respectively gives the unit of selection the ability to adapt to the action of selection across its temporal domain. In the case of the individual, we make decisions at times that we might judge to reduce our immediate fitness in the belief that it will improve our fitness in the long term, such as migration perhaps. The point is that we have the ability by some means to assess based on experience and conjecture. We speculate that the cultural group unit possesses the same ability, but now over a multi-generational time scale. One function of cultural tradition is to just this; to adjust the daily habits of its members to use historically successful traits. From the perspective of N.Selection which operates over a much shorter time scale with corresponding local optimum phenotype, this might be considered nostalgic maladaptation. This might seem rather obvious, but we frame it in the language of units of selection and temporal domains because of the proposal to extend this memory effect to the scale of the genome.
Selection at the genome scale, acting on individual alleles, like group selection, is not a new idea, first addressed by R. Dawkins [2]. We would like to consider the alleles as the units of genome scale selection. We also know that the genome can be replicated under recombination and this information transmitted and exchanged through the gene pool (in analogy with the phenotype and culture) a great many times without significant degradation. This gives us a notion of the possible temporal domain upon which selection can act at this scale. The analogous question now is over what temporal domain are these alleles selected for under the direct and induced effect of all three mechanisms. Following through with the phenotype $\mapsto$ cultural group analogy we will need to consider the concept of fitness in this context.

To summarize; what we have described, at least conceptually is a three layered system of selection in which we have three units of selection, all with a different effective range of viable information transmission or effective memory range, but all interdependent, with genetic, phenotypic and cultural selection pressures acting across scales. Many more are conceivable, but these are plenty to concern us for the time being. This effective memory is trivial to conceive at the phenotype scale, being an individual's lifetime experiences, reasonable to consider at the group scale with the effective cultural memory and becomes slightly more speculative when talking about the effective memory represented by the gene pool. One thing that we can assume, is that while all the above pressures may not be acting with the same effect across scales, it should indeed be the case that they support one another in general as what harms success at one scale will in general also harm the others at another as the success of the genome, phenotype and group are intimitely linked. Because of this, for practical purposes it seems plausible that the action of these three mechanisms may appear as the action of only one - N.Selection, hence we speculate that PCM's under Darwinian N.Selection appear as effective first approximations up to the seemingly anomalous appearance of locally maladaptive traits.

It should be stated that the above construction here is the preferred conceptual one as it

describes a symmetry through scales, but it is possible to consider these effective memories to be the temporal domain of the generational action of N.Selection due to the requirement that in general processes at all three scales support one another if this is preferred. In any case, the adaptation of a PCM to come will make use of these memory effects at different scales that can if it is preferred be considered as operated on solely by N.Selection such that the employment of the different memory resources are independent of all other selection mechanisms, but still contribute to multiple temporal domains of N.Selection.

5 A Basic DIT-PCM.

Given the concepts of selection acting at scales described above and that of effective memories as a starting point, together with the aim to examine success and conflict of genetic and cultural traits in a dual inheritance PCM, it is important to examine some simplified aspect of the consequences of such a generalisation. What we are especially interested in is the speculated natural action of the effective memory on the fitness measures at each scale.

5.1 Different perspectives of Fitness.

Boyd & Richerson[3] use simple deterministic PCM models, to describe qualitatively the iterative generational processes of genetic and cultural transmission. A Gaussian fitness function $W(Y, H)$ is used as a weight for the normalized statistical population distribution of mature phenotypes $f(Y, \bar{Y})$, where $\bar{Y}$ are representations of the mature phenotypic traits.

$$W(Y, H) \propto e^{-\frac{(Y-H)^2}{2S}} \tag{5.1.1}$$

$$\bar{W}(\bar{Y}, H) = \int W(Y, H) f(Y, \bar{Y}) dY. \tag{5.1.2}$$

$W(Y, H)$ is the probability of an individual of phenotype Y becoming a parent and sets the optimal mature phenotype Y at the value of H characterizing the environment and optimal phenotype under N.Selection, for simplicity to be a function only of time - or in this case generation number. This fitness function represents the action of N.Selection at the phenotype scale, hence it acts on the space of Y and H.

It is important to note that the concept of fitness that 5.1.1 represents is a Darwinian one. It assigns a measure of fitness that is a function only of properties of the phenotype and environment at their values at a particular generation. Y and H take discreet values for each generational iteration and each individual here and couple to the natural selection mechanism through this fitness function which is treated as the probability of an individual of trait Y reproducing. The fitness measure is treated in part as a probability of survival and part as an observable of the phenotype here, i.e. one individual can observe to some degree of accuracy, the values of Y and H that belong to another individual and the environment and make an assessment of another's suitability to reproduce under N.Selection.
Building on this Darwinian measure of fitness, we add sets of $\bar{W}(\bar{Y}, H)$'s to represent a

Darwinian measure of the fitness of phenotypic traits associated with various alleles within which Natural Selection can act. This is the first step in building a model of dual inheritance at the phenotype scale.
If the generalisation of the selection mechanisms acting on our system are to be made, then there will need to be a generalisation in the concept of fitness used in any PCM. Ideally we would use fitness landscapes operating in different spaces corresponding to different scales and selection mechanisms, build a model of the entire system's dynamics across scales and read off the observables at the phenotype scale. As we have limited time though, we choose to adapt the PCM used by Boyd & Richerson. This means that we are restricted to inferring effects of the memory effects on the phenotype scale and phenotypic fitness measure - specifically by adjusting the perception that individuals in a population will have of both the local optimum (local optimum, here meaning the temporal limit to an individual's ability to assess optimum without the genetic or cultural memory effect) phenotype as it impacts on their own learning goals, and their perceived fitness of others. Considering how an individual would perceive the fitness of another is then a natural issue to raise when considering the effects that culture and genetics could have on an individual's assessment of the local optimum for the purposes of choosing a learning goal and mate.

Addressing the simplifications of perceived fitness under N.S represented by the H environment/optimum phenotype; this makes some sense in a species not capable of social learning or genetic pre-disposal as it is then reasonable to assume that an individual's assessment of optimum phenotype is indistinguishable from the representative function we specify with H, but if we recognise the distinction between sources of perception, then treating the genome and culture as sources of behavioural traits, value/priority system and hence assessment of H, then we have three notions of fitness at the phenotype scale, one under a form of cultural 'pressure', one under natural pressure and one under genetic pressure. The consideration of the source of perception in the genetic and cultural transmission rules of a P.C.M. will allow incorporation of the concept of individual, cultural/group and genetic memory to act on individual's local optimum assessment in the PCM by use of three representative functions of the 'local' phenotype optimum H. This is the speculated source of local maladaptation.

To elaborate on the motivation of the use of three representative functions of the optimal phenotype - memory functions; if we exclude genetic and cultural assessment bias, then an individual's assessment of H at generation n has no dependence on its values at generation $n-1$. It is not unreasonable to expect that an individual will not be able to recall past values of $\bar{W}(\bar{Y}, H)$, Y and H. In a cultural species, it could be expected that knowledge of previous values of observables would affect an individual's assessment of the observable aspects of H. This is a possible effect of cultural memory and could allow a re-evaluation of the phenotype fitness measure. While we have described culture as the unit of selection under group selection, we only use its memory effect in the PCM that we are building. At the other end of the scale we have the analogous concept of genetic memory, which could conceivably have a far greater effective range in generations, perhaps limited only by the age of a species.

The most important point here is that the memory functions represent the temporal domains of the corresponding units of selection acted upon by the three selection mechanisms. This taken together with the earlier stated requirement that in general these selection mechanisms would 'support' one another through mutual interaction, we speculate will allow us to represent these memory functions at the phenotype scale as recorders of optimum phenotype over a history of generations. We require this mutual interaction and support between mechanisms because genome and cultural optimums do not themselves exist on the space of phenotypes and so we require that whatever interaction we specify in our PCM via transmission rules between them maintain a general beneficial correlation, i.e. what is bad for the genes is bad for the genotype and culture. This of course cannot always be true otherwise we would not observe local maladaptation, but the correlation allows a representation of all three scale memory effects at the phenotype scale in the PCM.

Motivated by the temporal range of the units of selection, we construct the memory functions as recorders of local optimum history at the phenotype scale and construct an ansatz modification to $W(Y, H)$ to incorporate measures of fitness at all three scales for the case of individuals characterized by a phenotype and allele index i as they impact on an individual's phenotype fitness:

$$\tilde{W}_i(Y, H(n)) = \frac{1}{\lambda + \sigma + \chi_i}\left[\lambda e^{-\frac{(Y_i - H(n))^2}{2S_N}} + \sigma e^{-\frac{(Y_i - c[H(n)])^2}{2S_C}} + \chi_i e^{-\frac{(Y_i - p_i[H(n), Y_j, p_j, n])^2}{2S_P}}\right] \tag{5.1.3}$$

where

- i is the allele index. We consider a set of N_g alleles that will characterize learning propensities and genetically determined perception of phenotype optimum.
- $c[H(n)]$ is the cultural/group measure of optimum phenotype fitness manifest as an individually perceived optimum.
- $p_i[H(n), Y_j, p_j, n]$ is the optimum fitness as determined by an individual with Y_j phenotype of the individual with phenotype Y_i as determined by Y_j's genetically determined pre-disposition . Then in a P.C.M. it is also dependant on the proportion of a population at generation n carrying allele i, $P_i(n)$.
- n is the generation number.
- S_N is the strength of Natural Selection on the phenotype.
- S_C is the strength of cultural selection on the phenotype.
- S_{P_i} is the strength of genetic selection on the i'th phenotype.
- λ, *sigma* and χ_i represent the weightings of Natural Selection, cultural and genetic selection in the phenotype fitness measure.

To examine the full effect of the interactions of different selection mechanisms acting on the phenotype at different scales would be enthralling, but for the sake of simplicity here, we

only examine one possible consequence, that of memory and its effect on an individual's culturally perceived fitness and learning goal. Accordingly we define $c[H(n)]$:

$$\begin{aligned} c: \qquad & H(n) \mapsto c[H(n)], \\ & \mathbb{R}^{N_C} \mapsto \mathbb{R}. \end{aligned}$$

where N_C is the number of environment/optimum phenotype histories that a culture can record/recall and

$$\begin{aligned} c[H(n)] &= \frac{\sum_{n\prime=0}^{n\prime=N_C} e^{-\beta n\prime} H(n-n\prime)}{\sum_{n\prime=0}^{n\prime=N_C} e^{-\beta n\prime}} \\ &= \frac{1-e^{-\beta}}{1-e^{-\beta(N_C+1)}} \sum_{n\prime=0}^{N_C} e^{-\beta n\prime} H(n-n\prime). \end{aligned} \tag{5.1.4}$$

Here $c[H(n)]$ is an average over the N_C generational iterations preceding n weighted by an exponential and $\beta i = ln(100)/N_i$ specifies the relative strength of the N_C'th generation in the weighting. The σ term in 5.1.3 refers to the cultural measure of fitness, incorporating a history of environments. The λ term is the classical Darwinian fitness measure under Natural selection similarly used by Boyd & Richerson[3]. Noting the index structure we use, only the χ_i term has an index associated to its optimum in phenotype space where there is a dependence on the distribution of other alleles in a population and their perception of fitness. Strictly speaking as we are considering the effects of dynamics at the three scales and we only treat the phenotype as an observable in the P.C.M. we should really have 5.1.3 as a tensor map such that $\mathbb{R}^{N_g} \otimes \mathbb{R}^{N_{groups}} \otimes \mathbb{R}^{N_p} \mapsto \mathbb{R}^{N_p}$, but as we are only considering the effect of one culture $N_{groups} = 1$ and one dimensional phenotypes, $N_p = 1$ and we are actually going to neglect the χ_i the mixing term due to a shortage of time, we can just treat 5.1.3 as a 1-vector of allele maps from $\mathbb{R}^1$ phenotype space redefined at each generation.

5.2 Guided Variation as the Learning Rule.

Constructing a dual inheritance theory incorporating this memory effect and seeking a relation between observables at each iteration at the population level that embody learning processes in its dynamics, we take the process of guided variation as a simple approach to the learning process for this test case. This was chosen because it incorporates explicitly the two processes by which an individual can learn and adapt its phenotype to an environment - socially/culturally or asocially. This allows us to compare traditional adaptive learning processes acting within a generation to their action when we adjust cultural transmission mechanisms and asocial learning goals to incorporate genetic and cultural memory effects that span generations.

We construct the following learning rule that incorporates social and asocial learning.

$$Y_i(n) = a_i X_i(n) + (1 - a_i)(T_i[H(n)] + \epsilon_i) \tag{5.2.1}$$

where

$$T_i[H(n)] = \zeta_i H(n) + \kappa_i g_i[H(n)] + \tau_i c[H(n)] \tag{5.2.2}$$

and

- i labels the allele carried by the individual as with the fitness function.
- ϵ_i is a random variable with Gaussian probability distribution $G_i(\epsilon)$ and $\bar{\epsilon}_i = 0$. representing estimation errors on the optimum when relying on asocial learning.
- $a_i = \frac{V_{\epsilon_i}}{V_{\epsilon_i} + L_i}$; V_{ϵ_i} is the variance of $G_i(\epsilon)$.
- L_i are learning parameters describing the propensity to rely on social or asocial learning.
- $X_i(n)$,$Y_i(n)$ are the immature/mature phenotypes at generation n
- $H(n)$ characterizes the learning goal. This goal could be quite general in its nature, but we take it to represent the environment independant of the observables of the individual. Under N.Selection this represents an optimum phenotype.
- ζ_i, κ_i and τ_i are weighting parameters s.t. $\zeta_i + \kappa_i + \tau_i = 1$ and characterize the reliance on different memory resources in perturbing the individual's perception of the learning goal.

and

$$\begin{aligned} g_i : \qquad & H(n) \mapsto g_i[H(n)], \\ & \mathbb{R}^{N+N_{Gi}} \mapsto \mathbb{R}^{N_{Gi}}, \end{aligned}$$

$$\begin{aligned} g_i[H(n)] &= \frac{\sum_{n\prime_i=0}^{n\prime_i=N_{Gi}} e^{-\alpha_i n\prime_i} H(n - n\prime_i)}{\sum_{n\prime_i=0}^{n\prime_i=N_{Gi}} e^{-\alpha n_i\prime}} \\ &= \frac{1 - e^{-\alpha_i}}{1 - e^{-\alpha_i(N_{Gi}+1)}} \sum_{n_i\prime=0}^{N_{Gi}} e^{-\alpha_i n\prime_i} H(n - n\prime_i). \end{aligned} \tag{5.2.3}$$

Here $g_i[H(n)]$ is an average over the N_{G_i} local optimums preceding the n'th weighted by an exponential and $\alpha_i = ln(20)/N_i$ specifies the relative strength of the N_{G_i}' th in the memory range. This is the genetic memory function and it is defined for each allele. For each value of N_{G_i} of each allele it describes a different definition of the temporal unit of genetic selection. This is not entirely consistent with our idea of the natural domain of the unit of genetic selection as the generational range in which information can be reliably transmitted across generations in the gene pool, but allowing different alleles to observe different effective ranges allows us to use a range of ansatz's across alleles to examine the

different successes within one of the simulations to come. We can easily fix it as a constant across alleles if we want to be more quantitative.

Many simplifying assumptions are made here, which although unrealistic by themselves, will allow the qualitative affects of the retarded transmission mechanism to be seen more clearly. One key assumption is that an individual carries a single allele and this single allele the transmission rules described by its $g_i[H(n)]$, L_i and V_{ϵ_i}.
This learning process incorporates both social and asocial learning. It states that a mature phenotype with trait and allele $Y_i(n)$ is the culmination of a combination of individual learning and copying of cultural parent(s) or encultrination. We have followed Boyd & Richerson here in assuming that the bulk of encultrination takes place during youth as an immature phenotype. The balance between these two processes is described by the a_i parameters. At the individual level the ϵ_i variables describe asocial learning errors that can be taken to represent the risk to mature phenotype fitness of this type of learning. In the limit of high epsilon, this rule for learning can be seen to favour encultrination. The learning parameters L_i play off against the variance V_{ϵ_i} describing the propensity of the immature individual to rely on learning. In the limit of high L_i learning is characterized more by individual learning with the goal characterized by the environment.

By requiring the probability/population level distribution of $X_i(n)$ and $Y_i(n)$ to be Gaussian, finding a relation for $\bar{X}_i(n)$, and the variance of $X_i(n)$, $V_{X_i}(n)$ between generations will specify the relation between generations.

$$\bar{Y}_i(n) = a_i \bar{X}_i(n) + (1 - a_i) T_i[H(n)], \tag{5.2.4}$$

$$V_{Y_i}(n) = a_i^2 V_{X_i}(n) + (1 - a_i)^2 V_{\epsilon_i} \tag{5.2.5}$$

are found from 5.2.1 and relate the phenotype distributions within generation n. Using 5.1.1 with 5.1.3,

$$\bar{\tilde{W}}_i(\bar{Y}, H) = \int \tilde{W}_i(Y, H) P_i(Y|X) dY_i, \tag{5.2.6}$$

where $P_i(Y|X)(n)$, the mature phenotype distribution after guided variation is found which is specified by another normalised Gaussian such that

$$P_i(Y|X)(n) = \frac{1}{\sqrt{2\pi V_{Y_i}}} e^{\frac{-(Y_i - \bar{Y}_i)^2}{2V_{Y_i}}}. \tag{5.2.7}$$

A genetic transmission rule, weighted by $\bar{\tilde{W}}_i$, a $\bar{Y}_i(n) \to \bar{X}_i(n+1)$ can be defined giving the effect of the fitness measure described as

$$P_i(n+1) = \frac{P_i(n) \bar{\tilde{W}}_i(\bar{Y}, H)}{\sum_{j=1}^{j=N_g} P_j(n) \bar{\tilde{W}}_j(\bar{Y}, H)}. \tag{5.2.8}$$

$$\tag{5.2.9}$$

This gives the proportion of immature phenotypes in generation $n+1$ with allele i. This is a linear transmission rule giving the probability of an offspring of pair mating being characterized by the i'th allele. It is not derivable solely from mating and genetic transmission probabilities as information about both is contained in $\bar{\bar{W}}_i(\bar{Y}, H)$, but is imposed. It can be shown though that if $\bar{\bar{W}}_i(\bar{Y}, H)$ were a measure only of fitness under natural selection and 5.2.8 were the probability of a an 'i' individual randomly pair mating, with all alleles having equal transmission probabilities after mating, then 5.2.8 would be the statistical probability of a random offspring then carrying allele i. So the combined mating and genetic transmission rule 5.2.8 should not seem unreasonable

We then construct a transmission rule for the offspring trait is here by

$$X_i(n+1) = \sum_{j=1}^{j=N_g} \gamma_{ij} Y_j(n) + \epsilon_{trans_i}, \tag{5.2.10}$$

where ϵ_{trans_i} is another random variable with $\bar{\epsilon}_{trans_i} = 0$ normal population level distribution and variance $V_{\epsilon_{trans_i}}$. As we are still dealing with Gaussians and require the variance and the mean, the mean is then given by a linear cultural blending rule with similar weighting

$$\bar{X}_i(n+1) = \sum_{j=1}^{j=N_g} \gamma_{ij} \bar{Y}_j(n) \tag{5.2.11}$$

$$= \sum_{j=1}^{j=N_g} \gamma_{ij} \left(a_j \bar{X}_j(n) + (1-a_j)\{(\zeta_i H(n) + \kappa_i g_i[H(n)] + \tau_i c_i[H(n)]\} \right), \tag{5.2.12}$$

$$\tag{5.2.13}$$

where

$$\gamma_{ij} = \left(\mu \frac{P_j(n) \bar{\bar{W}}_j(\bar{Y}, H)(1-\delta ij)}{\sum_{a=1}^{a=N_g} P_a(n) \bar{\bar{W}}_a(\bar{Y}, H)(1-\delta ia)} + \rho \delta ij \right) \tag{5.2.14}$$

$$\tag{5.2.15}$$

$$= \begin{pmatrix} \rho & \gamma_{12} & \cdots & \gamma_{1\beta} \\ \gamma_{21} & \rho & \cdots & \gamma_{2\beta} \\ \vdots & \vdots & \rho & \vdots \\ \gamma_{\beta 1} & \gamma_{\beta 2} & \cdots & \rho \end{pmatrix}. \tag{5.2.16}$$

$$\tag{5.2.17}$$

This is a vertical cultural transmission rule and gives $\bar{X}_i(n+1)$ as the average of the mature phenotypes of the previous generation with a weighting γ_{ij} which is the weighted blending rule such that $\rho + \mu = 1$ giving a weighting of ρ for the genetic parent whose allele the offspring carries and gives the unweighted average over the rest of the population. This is a way of accounting for the heavy reliance on encultrination by parents.

We now define the variance for the i'th's immature cultural trait between generations:

$$V_{X_i}(n+1) = \sum_{j=1}^{j=N_g} (\gamma_{ij})^2 * V_{Y_j}(n) + \tilde{V}_{trans_i} + 2 \sum_{a,b|a\neq b}^{N_g} \gamma_{ia}\gamma_{ib} Cov\left(Y_a(n), Y_b(n)\right), \quad (5.2.18)$$

where $\tilde{V}_{trans}$ is the variance introduced to the distribution of immature phenotypes with the i'th allele due to estimation errors on the cultural means. The second term above vanishes so long as the distributions for a and b are.

The cultural transmission rule 5.2.11 says that the i'th immature phenotype adopts the average of the distributions in a population with a weighing of ρ for the parent that it received its allele from. The variance of the i'th distribution of immature phenotypes given by 5.2.18 says that the population level variance is the statistical error on the measurement of the mean in addition to an error due to the method of estimation and transmission. This is a slightly artificial simplification for the transmission of variance as it assumes that an immature phenotype can identify the different phenotypic trends in a population, but will have to be suffered for the sake of simplicity here. It should be noted from the form of the dependence of $\tilde{\tilde{W}}_i$ on V_{Y_i} and V_{ϵ_i} that the variance of the distribution of cultural traits in a population will have a serious effect on average fitness of an allele. As such, it would be sensible to consider more carefully the dependence of parameters such as V_{ϵ_i} and L_i on some other measure of environmental, cultural or genetic variation in a population. Mark Lake argued the importance of the variability of environments in predicting evolutionary processes in [5] amongst many others, but we assume that they take constant values while building a PCM as we risk clouding the effects of the processes we are focusing on by adding too much complexity into the model.

Part II

Simulations of Guided Variation.

In order to examine the dynamics of the guided variation model described in the previous section, we have written a program to simulate the process. The aim of these simulations is to investigate our speculation that the additional memory effect can differentially advantage an individual associated with a particular allele and phenotype in certain environments. Different simulations consider $N_g = 4, 5, 9$ alleles, each characterizing different memory strengths and we run a variety of simulations over 100-50000 iterations displaying the most interesting of those. Because there are several dozen couples variables within and between each generation, the dynamics of the process through generations, while deterministic is still less than transparent for anyone restricted to conceptual constructions in only three or four dimensions. As such, we will begin the analysis with the simplest case of pure Natural Selection and local measure of optimum phenotype defined generationally by H with $\kappa_i = \tau_i = \sigma = 0$ and $\zeta = \lambda = 1$ and then continue to include cultural and genetic memory effects later. As will be shown the form of variation in the environment, the learning parameters, learning errors and memory effect will all have great effect on which allele gains a decisive advantage over the others in the short and/or long term.

The first thing to do is make the distinction between two general types of environmental variation, what we will call here, a selection event and a fluctuation. A selection event can be characterized by a sudden shift in $H(n)$ w.r.t. n to some new value where it remains. A local fluctuation is then the same form of sudden shift where $H(n)$ later returns to a value similar to the one it held before the variation began. In nature, the distinction is less clear. There seems to be a long term environmental equilibrium on a scale such that many local selection events appear as small fluctuations. This is in fact part of the motivation for the examination of the memory concept as it seems natural that there be positive and negative effects of memory for the two scenarios. With these forms of environmental variation in mind, it might also be interesting to note that in a particular choice of coordinates, the selection events and fluctuations are symmetric and anti-symmetric respectively, so we can describe all possible environments with linear sums of the functions describing these (we will use a gassian and an arctan). Selection events such as major earthquakes, water shortages etc representing a sudden cause of change of optimum phenotypes may on a longer timescale look like a local fluctuation. The distinction in the context of evolution between the two types of variation can be made a number of ways, but a seemingly sensible distinction might be that a selection event can be described as a variation such that the ranking of fitness before the event is significantly different after the event if the event is a selection event.

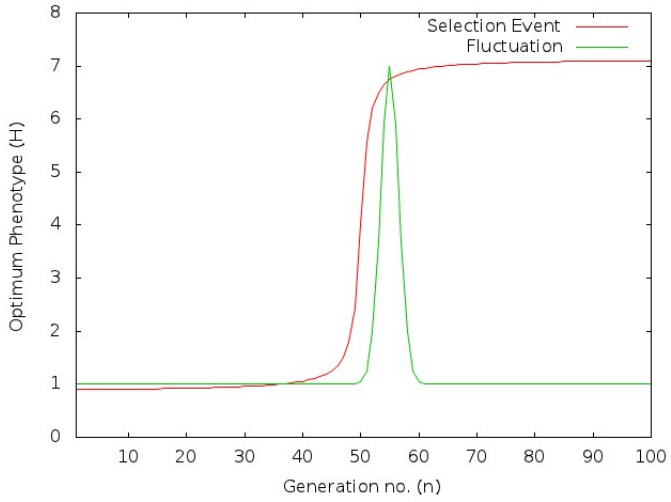


Figure 1: Two forms of environmental variation.

6 Darwinian Simulation.

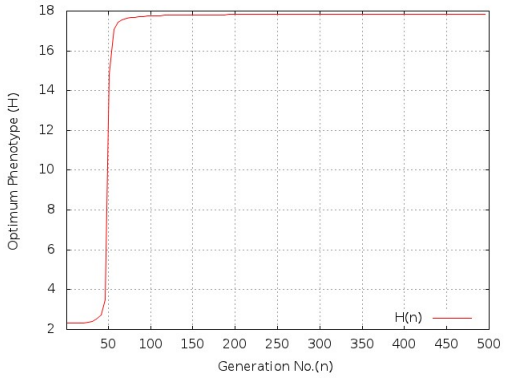
Even in the simplest case there are many interesting dynamics evolving under Natural selection. Here we focus on the effect of the propensity to rely on learning L_i, learning cost represented by V_{ϵ_i}, encultrination cost represented by V_{X_i} with a high dependence on V_{trans_i} and form of environmental $H(n)$ variation.

6.1 Selection Events.

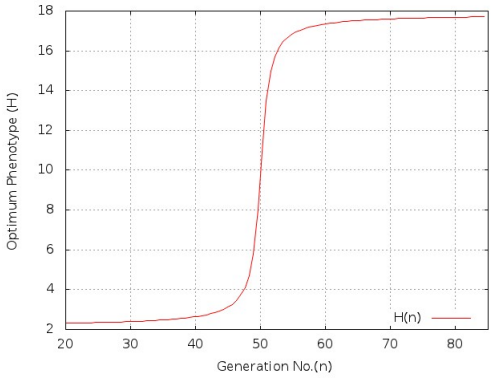
Table 5 are the parameters used for a simulation of a population evolving through a selection event shown in Figure 5.

Table 1: Parameters.

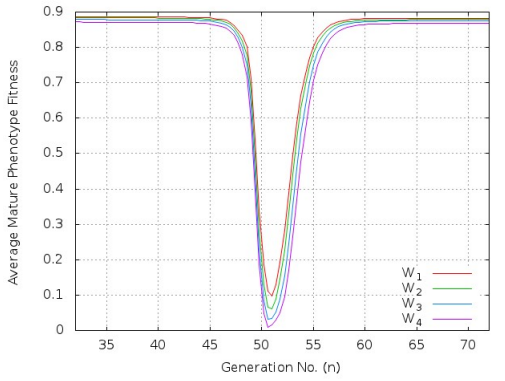
Allele	V_{trans_i}	S_{N_i}	V_{ϵ_i}	L_i	$a_i = \frac{V_{\epsilon_i}}{V_{\epsilon_i}+L_i}$	ρ	μ	λ	σ	ζ_i	κ_i	τ_i
1	1	2	1	1.00	0.50	0.5	0.5	1	0	1	0	0
2	1	2	1	0.81	0.55	0.5	0.5	1	0	1	0	0
3	1	2	1	0.67	0.60	0.5	0.5	1	0	1	0	0
4	1	2	1	0.54	0.65	0.5	0.5	1	0	1	0	0



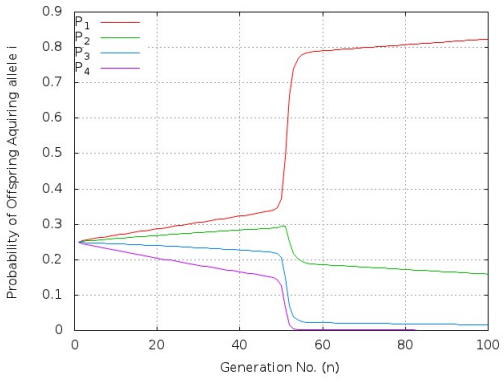
(a) Selection event.



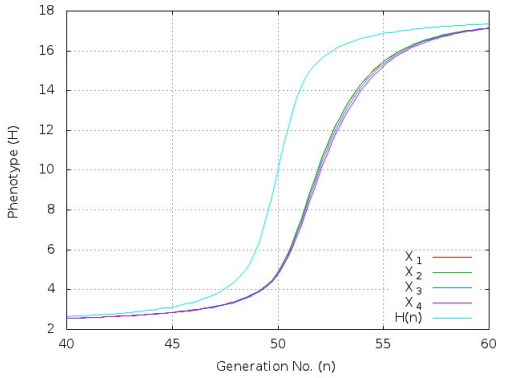
(b) Selection event.



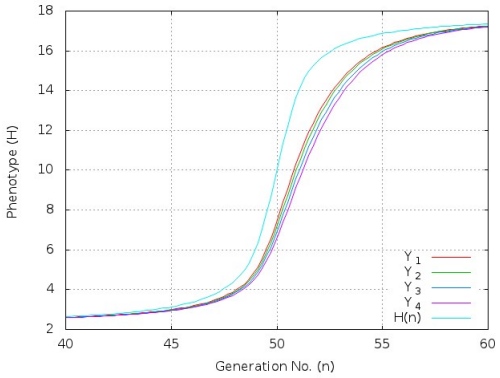
(c) Average fitness of i'th allele.



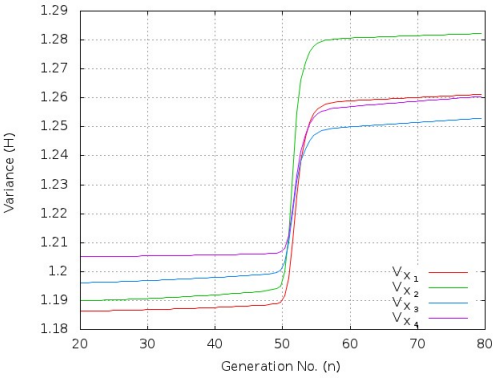
(d) Probability of individual receiving i'th allele.



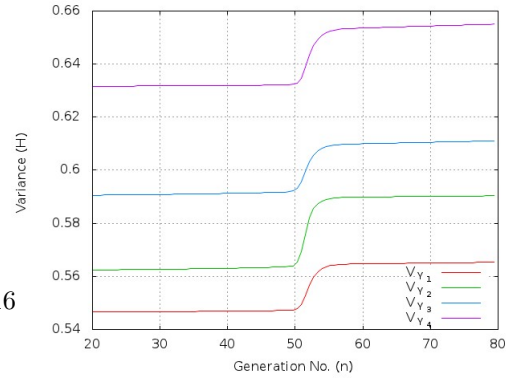
(e) Mean immature phenotype of i'th allele.



(f) Mean mature phenotype of i'th allele.



(g) Variance of i'th immature phenotype.



(h) Variance of i'th mature phenotype.

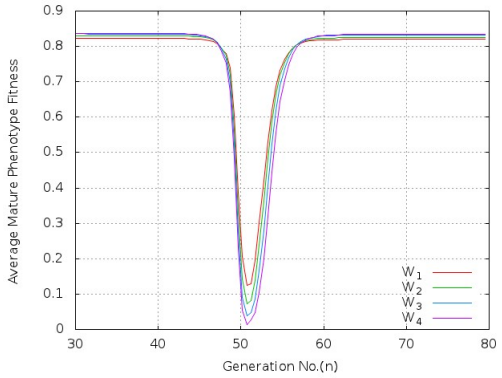
Figure 2: 400 generations through a selection event.

Looking at Figure 6.1 the effect of the selection event on the population can be seen in subfigure 2(d) to accelerate the trend of all alleles, but this was inevitable from the first generation. Looking at subfigure 2(c) it can be seen why; the fitness levels of each allele clearly drop with decreasing propensity to rely on learning. In so far as the acceleration of this trend goes during generations 40-60, the greater the learning propensity L_i, the greater the fraction $(1 - a_i)$ in the guided variation process, the better able an individual is able to track the optimum phenotype, relying more on individual learning over encultrination. This does however assume no change in learning cost V_{ϵ_i} w.r.t. change in learning propensity. The seemingly intrinsic lower level of fitness associated with lower L_i relates to the variances of the mature phenotype distributions V_{Y_i}, learning costs and selection strength parameters S_i.

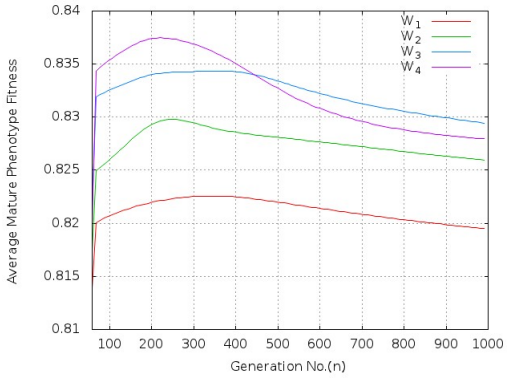
From subfigure **??** for V_{Y_i} it can be seen that increasing the value of the L_i's will either raise or lower the variance of the i'th mature phenotype depending on whether $V_{\epsilon_i} < V_{X_i}$ or $V_{\epsilon_i} > V_{X_i}$. If the former then larger L_i decreases V_{Y_i} if the latter, then V_{Y_i} increases with L_i within a given generation. There are then two resulting effects depend on the level of environmental variation. If the environment is relatively steady as it is in the first 40 or so generations, Y_i tracks H well no matter the variance of the mature phenotype and $\tilde{\bar{W}}_i(\bar{Y}, H)$ will in fact be larger for larger L_i because of the normalisation factor in the Gaussian involving a $1/\sqrt{V_{Y_i}}$. The second effect is in a varying environment such as that occurring in generations 40-60 where the decreased V_{Y_i} negatively affects the relative fitness of the alleles, but this effect on fitness plays off against the increased tracking ability that goes with the larger L_i's.

From subfigure 2(h) it can be seen that the case dealt with here is $V_{\epsilon_i} < V_{X_i}$ (but only marginally). We do see the effect described above in subfigure 2(c) and in the varying environment the better tracking of the larger L_i's wins out against the cost to the variance and at no point does the order of fitness change. In fact the tracking effect of the stronger learning strengths wins out so well that the relative fitness's of the learner increases by many times w.r.t the worst during the selection event, which is exactly why the sudden shift in subfigure 2(d) occurs. Looking at subfigure 2(f) though it can just about be seen that the worse learners track the optimum worse. Looking at subfigure 2(e) the encultrination effect 5.2.10 can be seen to have the effect of reducing the clustering of the N_g mean phenotypes which is expected as this kind of parental bias has an averaging function on the phenotypes. There is also apparent interesting phenomenon relating to the variances of the mature and immature phenotypes in subfigures 2(h) and 2(g). The reordering of the size of the variances for the immature phenotypes is in fact just a temporary occurrence relating to the squaring of the γ_{ij} term and the proportions within in the cultural transmission rule 5.2.10 mentioned earlier. While it cannot be seen on the graph, they do regain their original order later. In any case, the scale may be deceiving as the effect of the reordering is very small given the relative values involved. The disparity with the form of the variance of the mature phenotypes is another effect of the averaging of the cultural transmission rule with effects represented by subfigure 5.2.18.

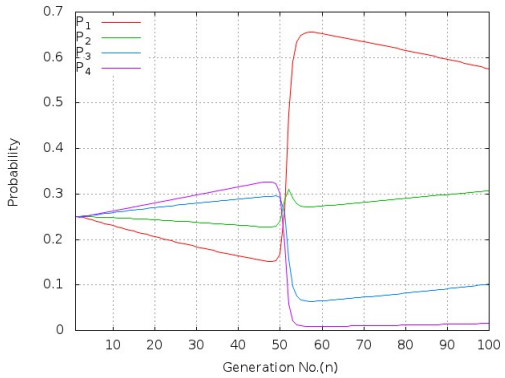
The second interesting case in which $V_{\epsilon_i} > V_{X_i}$ presents even more interesting results:



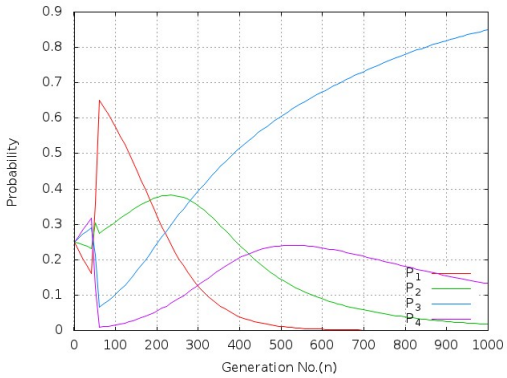
(a) Average phenotype fitness associated with i'th allele.



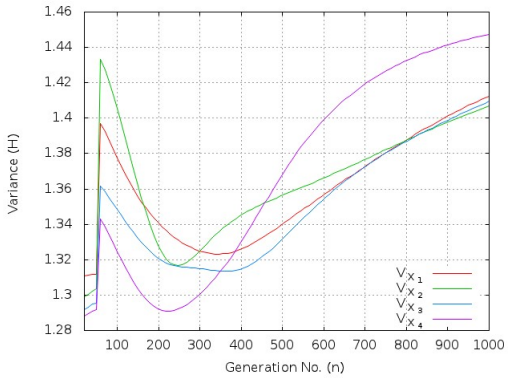
(b) Average phenotype fitness associated with i'th allele.



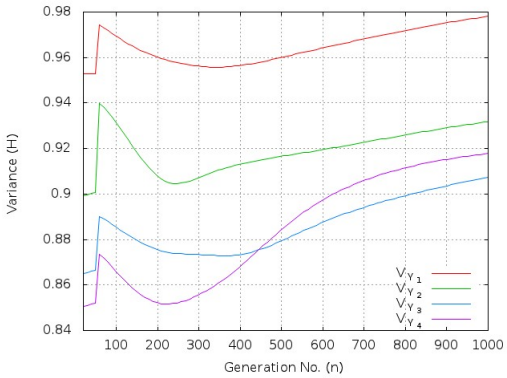
(c) Probability of individual receiving i'th allele.



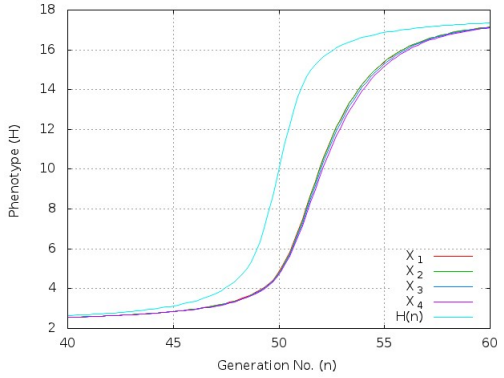
(d) Probability of individual receiving i'th allele.



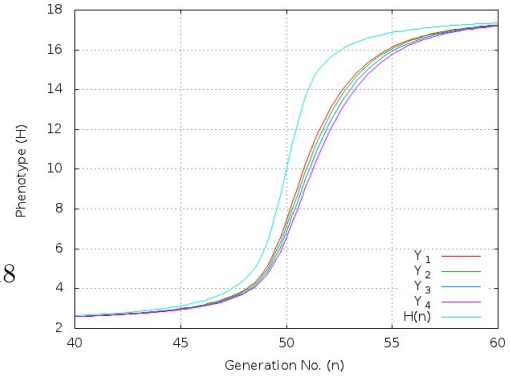
(e) Variance of i'th immature phenotype.



(f) Variance of i'th mature phenotype.



(g) Mean immature phenotype of i'th allele.



(h) Mean mature phenotype of i'th allele.

Figure 3: 1000 generations through selection event, now with $V_{\epsilon_i} = 2.5$.

We now have a complete reversal in correlation between the steady environment fitness and learning strengths as increasing L_i now increases V_{Y_i} as seen in subfigure 3(f). This also increases the adaptivity of the allele; this combined with the improved tracking of H in the varying environment results in the sudden reordering of fitness in subfigure 3(a) during the selection event resulting in the change in probability of an infant being born with allele i in subfigure 3(c).
After the selection event once environmental conditions stabilize, fitness levels take the order and approximate values that they had before hand, for another three hundred generations or so; then they fluctuate once again (subfigure 3(b)) due to the long term effects of the selection event on V_{X_i} and V_{Y_i}, in figures 3(e) and 3(f) respectively.
One simplification here that has allowed the dominance of the worst learners in steady environments is the assignment of only two values for both V_{ϵ_i} and V_{trans_i} for all alleles, which does not directly reflect the increased cost of heavier reliance on either social or asocial learning, though V_{ϵ_i} does affect the propensity to learn. This seriously affects the long term proportions of each allele in the population, but given that it is the varying environment that we will be focusing on, the very different dynamics that can come about by adding unique costs is largely neglected.

In both figures 6.1 and 6.1 The effect of dispersion of phenotype distributions has had long lasting effects on fitness under N.Selection and in the case of the worst learner, critically damaged the pre-selection dominance of allele 4 (3(d)). However given that most realistic environments and learning objectives vary significantly over these timescales, it appears that, at least in this simple case where we only look at varying learning parameters and one value for learning and encultrination costs, the most realistic sensible case to consider has $V_{\epsilon_i} > V_{X_i}$, the learning variance is greater than the cultural variance(for a given allele), otherwise the dominance of those who favour cultural learning seems certain in any environment which does not seem feasible given the likely result being an entire maladaptive culture as there would be no variation induced by the guided variation process(the learners would become extinct) which would probably mean elimination under group selection. This makes sense given that the alternative would be for evolution to favour a species without culture. This is considering current parental bias cultural transmission however. It is possible that prestige or success bias could lead to a situation in which a minority of adaptive learning phenotypes and alleles are favoured by some means and maintain overall population level cultural adaptability through encultrinating others, but this will unfortunately not be considered here.

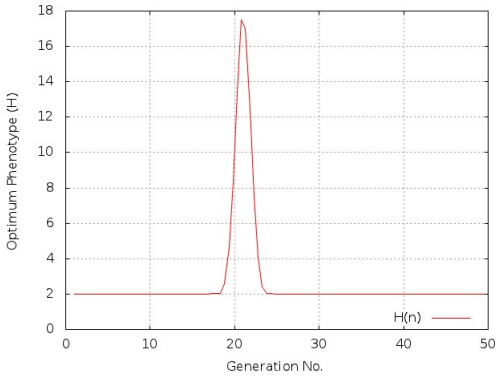
6.2 Fluctuations.

The motivation for looking at fluctuations is two-fold; firstly, on a large enough timescale, many naturally occurring selection events appear as fluctuations, and secondly it will allow an examination of the consequences for the most adaptive on having to adapt to one extreme and then back again. This is in some ways just a double selection event, but will have special significance on fitness when other forms of selection and objective are turned on.

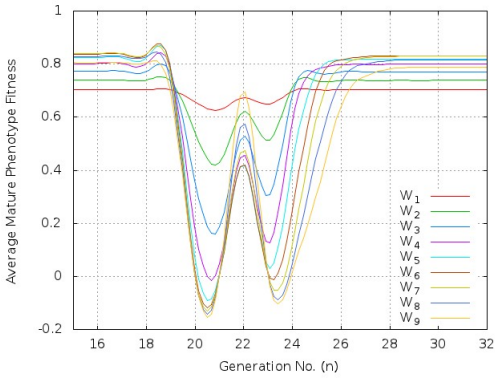
Now that some of the qualitative behaviour of has been seen in for the selection event, we add another five alleles to gain better resolution and range of behaviours for parameters. Looking first at the single fluctuation:

Table 2: Parameters.

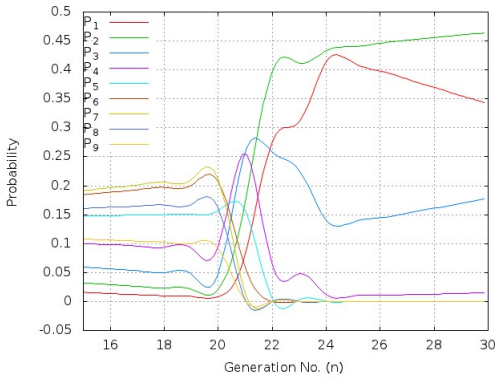
Allele	V_{trans_i}	S_{N_i}	V_{ϵ_i}	L_i	$a_i = \frac{V_{\epsilon_i}}{V_{\epsilon_i}+L_i}$	ρ	μ	λ	σ		ζ_i	κ_i	τ_i	
1	1	2	2.5	22.50	0.1	0.5	0.5	1	0	0		1	0	0
2	1	2	2.5	10.00	0.2	0.5	0.5	1	0	0		1	0	0
3	1	2	2.5	5.83	0.3	0.5	0.5	1	0	0		1	0	0
4	1	2	2.5	3.75	0.4	0.5	0.5	1	0	0		1	0	0
5	1	2	2.5	2.50	0.5	0.5	0.5	1	0	0		1	0	0
6	1	2	2.5	1.60	0.6	0.5	0.5	1	0	0		1	0	0
7	1	2	2.5	1.07	0.7	0.5	0.5	1	0	0		1	0	0
8	1	2	2.5	0.63	0.8	0.5	0.5	1	0	0		1	0	0
9	1	2	2.5	0.27	0.9	0.5	0.5	1	0	0		1	0	0



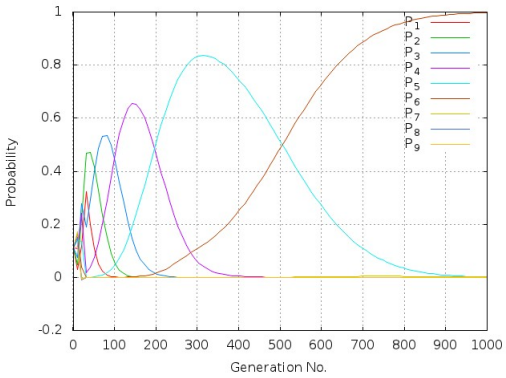
(a) Fluctuation.



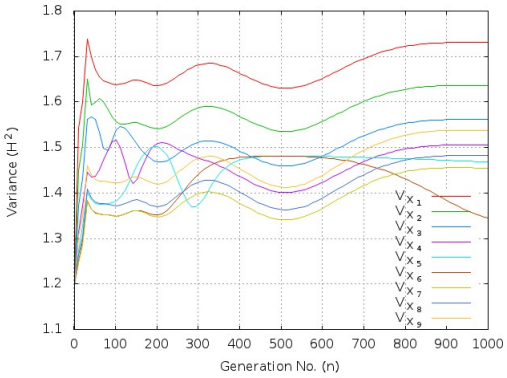
(b) Average fitness of i'th allele.



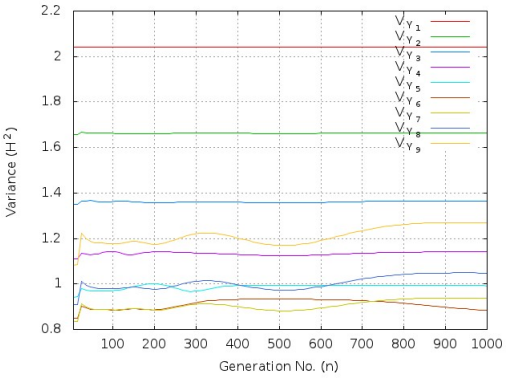
(c) Probability of individual receiving i'th allele.



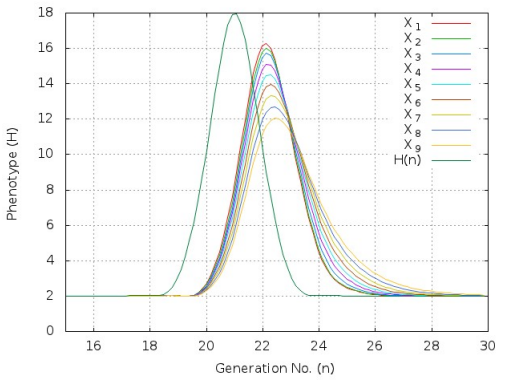
(d) Probability of individual receiving i'th allele.



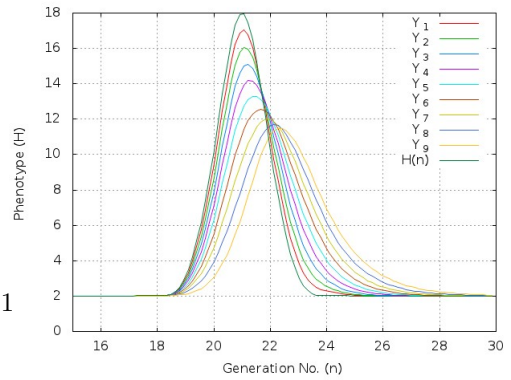
(e) Variance of i'th immature phenotype.



(f) Variance of i'th mature phenotype.



(g) Mean immature phenotype of i'th allele.



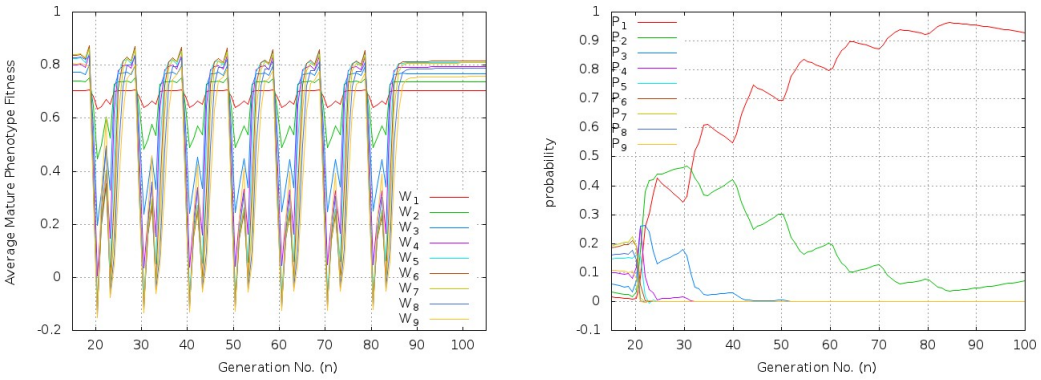
(h) Mean mature phenotype of i'th allele.

Figure 4: 1000 generations through fluctuation.

A broad and complex range of behaviours can is described here. In subfigire 4(b) the effect of the single peak fluctuation can be seen to cause an overall double dip in fitness. Notice the asymmetry in the reversal of fitness across learning strength through the fluctuation though. Now that we have more alleles across a larger range of learning strengths it we can see that up until allele 5(the genotypes most reliant on learning) they follow the same order of fitness as seen in the simulation of figure 6.1; due to their increased mature phenotype variance they are less fit in the static environment. Alleles 5-9 exhibit less ordered pre and post-fluctuation behaviour though. The issue here is that the worse learners here will rely more on encultrination, which means the mature variance transmission rule relies more on V_{X_i} than V_{ϵ_i} which means higher dependence for V_{Y_i} through V_{X_i} on the γ matrix describing the cultural transmission rules. This of course makes sense, but then the increasingly dominant γ brings with it another layer of complexity - the first four alleles dynamics are dominated by the learning parameters and their distinguishing behaviour can be seen to be easier to follow in subfigures 4(e) and 4(f).
During the fluctuation, the dominant distinguishing 'force' on fitness for all alleles is their learning strengths and they order perfectly by their ranking of L_i's(this will not always be the case). The cause for this is seen most clearly by the separation between optimum and mature phenotypes by allele in subfigure 4(h). The clustering effect of cultural transmission versus the dispersive effect caused by the varying asocial learning propensities can be observed as the vertical spacing of immature phenotypes in subfigure 4(g) relative to mature phenotypes of subfigure 4(h) along with the crossover of phenotypes in both graphs as those with higher values of L_i first overtake then undertake the those with lower L_i.
Finally, looking at the probability distributions of subfigures 4(c) and 4(d) we see the short term effect of the fluctuation's ordering of fitness as alleles 1-4 climb and the rest decline. At first appearance it is the second allele that comes out on top, but looking at an alleles pre-fluctuation fitness relative to its fitness immediately after, the increase is in fact in order of learning strength. For this reason it might have been best to take a numerical derivative of probability w.r.t generation no. here, but this relative measure can still be visually approximated here. Because allele 1 was reduced in proportion so much during the first 18 generations, it never reaches a dominant position before the fluctuation ends and its relative fitness plummets. Allele 2 seems to represent the best short term fitness balance though. As its fitness level never drops to that of allele 1 it does not need to increase in relative magnitude so much to reach absolute dominance (this is the motivation for the memory effect). Figure 4(d) displays an elegant after effect of the fluctuation in the static environment as one after the other the worst learners regain numbers and gain dominance one after the other. This is entirely a result of the bias of the cultural transmission and its impact on V_{Y_i} represented by *gamma*. It would be tempting to draw conclusions from this, but the model is too simplistic to do anything but reflect qualitatively an immediate response to specific circumstances, but it is interesting to see how in an unrealistically static environment, our transmission rules direct the population after a fluctuation.

We saw in subfigure 4(d) that allele 1 responded well to a single fluctuation, but allele 2 was the one taking the dominant position. Below in figure 6.2 several of these events have

been modelled in sequence:



(a) Average fitness of i'th allele.

(b) Probability of individual receiving i'th allele.

Figure 5: 2000 generations through fluctuation.

The effect on fitness is very similar to that of the single fluctuation, but the repeated sequence can be seen to 'build' the proportion of individuals with alleles 1-4 relative to the others. Absolute proportion only increases for allele 1 as its growth out strips the others so well that alleles 2,3 and 4 still drop, but can none the less be seen to be promoted by each fluctuation.

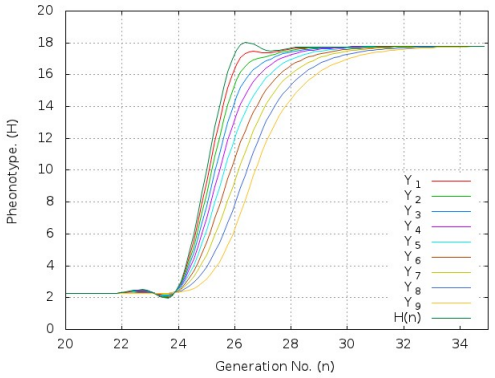
7 Cultural Memory Selection Pressures and Optimums.

In a previous section on selection events and fluctuations, we have seen that an individual equipped with an adaptive(L_i) allele often have a fitness under N.Selection optimized for one of two general types of environment corresponding to social learning in static environments and asocial learning in varying environments. We held equal the costs represented by V_{ϵ_i} that would naturally vary with L_i V_{X_i} which could have made a significant difference, but allowed us to examine the adaptive consequences for a class of alleles additionally characterized by the condition $V_{X_i} > V_{\epsilon_j} \quad \forall i, j$ with more clarity than would otherwise been possible given the already non-trivial evolutionary dynamic.
We have seen how under N.Selection, the environmental guiding function $H(n)$ and its average fitness function $\bar{\tilde{W}}_i(\bar{Y}, H)$ choose to promote different learners in different environments while we held all other constants equal across alleles. Now we add the cultural memory function to the learning goal and vary objective parameters τ_i instead. We would like to see how, given a particular cultural transmission rule (γ in this case), those who use their cultural optimum as a learning goal and as a basis for assessment of fitness of one another compare with those who rely on the Darwinian measure of objective and fitness used so far. We look at a mixture of possibilities ranging from the extremes of learning goal being purely cultural while fitness being entirely Darwinian through to the reverse. The motivation for

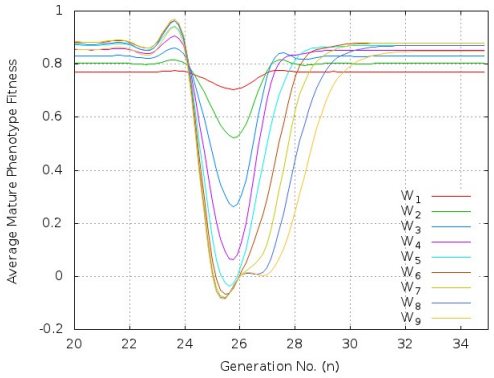
this range of considerations is the observation in society that at extremes an individual's personal objectives may be personally(rather than culturally) defined, while at the same time they might seek a 'normal'/encultrinated mate. And of course the reverse.
With the previous simulations we were able to test for the most favoured approach under N.Selection. Given that now some of the model's parameters that we wish to vary are the selection mechanisms themselves, we instantly lose the measure of absolute fitness that we had before, and gain those of fitness under different selection pressures. Given that this is a PCM operating on a generational cycle, we refer now to the combined effect of the memory pressures as the 'absolute reproductive fitness'. Now the question of whether the associated memory functions are evolutionarily advantageous is not asking anything so simple as what traits, carried by what alleles are fittest, most successful, in what environment over what timescale, as our memory functions now individually define their own success measures in the form of optimums over different temporal domains. While this adds complexity the measure of adaptation, these distinguishing measures of maladaptation are what we were looking for. Of course when we talk of maladaptation normally we are referring to local maladaptation under N.Selection, and this is still possible to examine here simply using the local optimum. The local optimum is now not a measure of absolute maladaptation though but rather a measure associated with a temporal domain - the generational timescale.
From the beginning, we had a main hypothesis; that these selection mechanisms would, from the perspective of the phenotype centred models, act with separate pressures on the individual and that each memory resource would couple to every other selection mechanism through the PCM such that the memory effect associated with one unit of selection would in general advantage another unit under a different selection mechanism while individual's with different coupling strengths to the cultural and genetically defined optimums (represented by λ, σ, ζ_i, κ_i and τ_i) would vary their phenotype in a potentially locally maladaptive manner. This is where we start to look at the different effects of cultural memory.
Before we proceed, we would like to address the possibility the cultural memory effect might even be advantageous as it impacts on learning objective under N.Selection. This seems like a remote possibility especially given the model that we have constructed, but we have checked none the less. As it happens, we have indeed been unable to find this potential effect in the case of cultural optimums.
Apart from looking at cultural objective under natural selection , we consider the cultural fitness measure. The following is a set of results from simulations comparing the effect on learners whose objective is defined under N.Selection with different mixes of cultural and natural fitness. We have the two forms of environmental variation to consider; first we take a brief look at the selection event we saw before, but look at the impact on the two fitness measures this environmental optimum represents.

Table 3: Parameters for first of three figures under N.Selection objective.

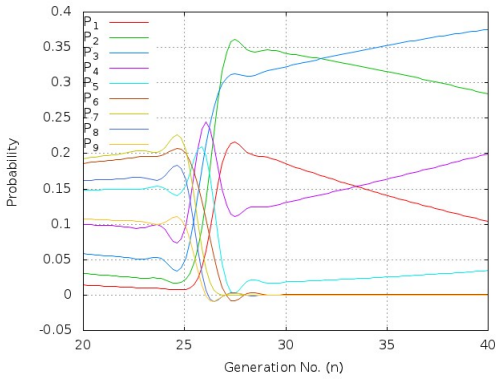
Allele	V_{trans_i}	S_{N_i}	V_{ϵ_i}	L_i	$a_i = \frac{V_{\epsilon_i}}{V_{\epsilon_i}+L_i}$	ρ	μ	λ	σ	ζ_i	κ_i	τ_i	N_C
1	1	2	2.5	22.50	0.1	0.5	0.5	2	0	1	0	0	20
2	1	2	2.5	10.00	0.2	0.5	0.5	2	0	1	0	0	20
3	1	2	2.5	5.83	0.3	0.5	0.5	2	0	1	0	0	20
4	1	2	2.5	3.75	0.4	0.5	0.5	2	0	1	0	0	20
5	1	2	2.5	2.50	0.5	0.5	0.5	2	0	1	0	0	20
6	1	2	2.5	1.60	0.6	0.5	0.5	2	0	1	0	0	20
7	1	2	2.5	1.07	0.7	0.5	0.5	2	0	1	0	0	20
8	1	2	2.5	0.63	0.8	0.5	0.5	2	0	1	0	0	20
9	1	2	2.5	0.27	0.9	0.5	0.5	2	0	1	0	0	20



(a) i'th Phenotype .



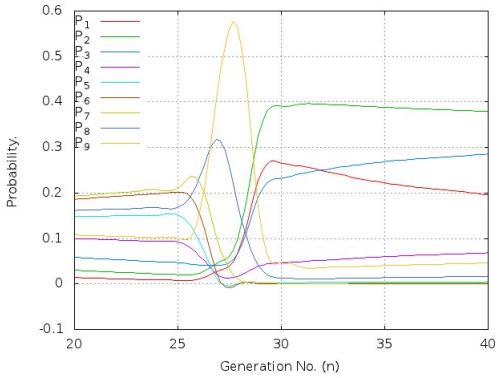
(b) Average fitness of i'th allele.



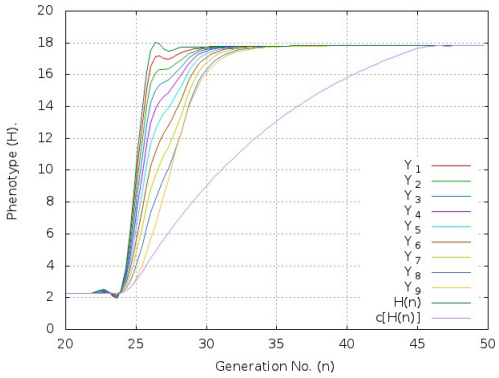
(c) Probability of individual receiving i'th allele.

Figure 6: Pure N.Selection and objective through selection event.

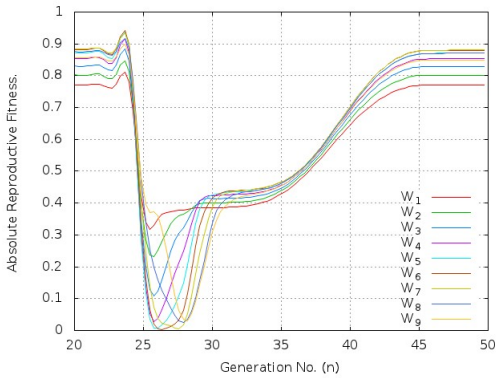
Here we see the natural and cultural selection pressures impact on the absolute reproductive fitness of the different learners through the selection event and the reordering of this fitness as we did earlier with the fluctuation under the N.Selection measure of fitness. The key observation here being the order of fitness going by learner strength during the event - as expected from our experiences with the fluctuation earlier.



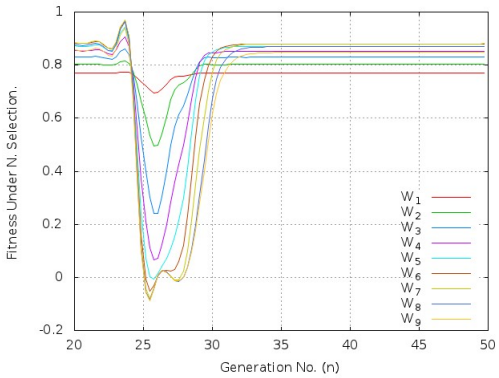
(a) Probability of individual receiving i'th allele.



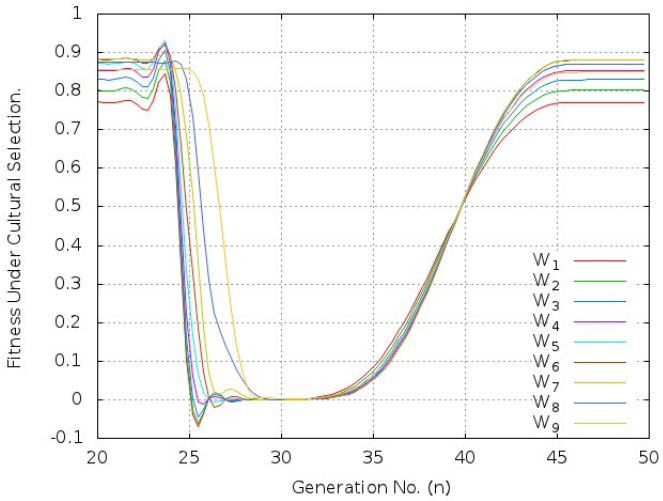
(b) i'th mature phenotype .



(c) Absolute reproductive fitness of i'th allele.



(d) Average fitness Under N.Selection of i'th allele.



(e) Average fitness Under Cultural Selection of i'th allele.

Figure 7: Selection event with $\lambda = \sigma = 1$.

The effect of the introduction of equally weighted cultural and natural selection pressures can be seen to instantly favour the worst two learners in terms of absolute reproductive fitness in subfigure 7(e) at the event as the those able to adapt fast, guided by the N.selection optimum pull away from the cultural optimum through generations 24-45. The weighted average of fitness between the two measures of fitness now negatively impact those who rely on learning over encultrination in a clear manner through this generational range.
We could look at the case of pure Cultural Selection, but the trend is clear - with selection events such as this where all learners(except the worst who have been excluded) will pull away from the cultural optimum as in subfigure 13(d), a society whose constituent members rely more heavily on cultural or traditional measures of fitness will always see an increase in fitness for those that most rely on encultrination with selection events such as these. This is a very different result to that described by Darwinian fitness. It represents a clear conflict between cultural and Darwinian selection pressures and shows how those who rely on encultrination will be favoured by the cultural fitness measure - which seems quite natural, as does the favouring of the best learners by Darwinian fitness here. The key result of this mixing of pressures through the selection event is that it brings out the conflict most clearly as seen by the disparity in fitness of the worst learners through generations 25-30 in figures 7 and 7.

The question now is how do these results change when we consider a fluctuating environment?

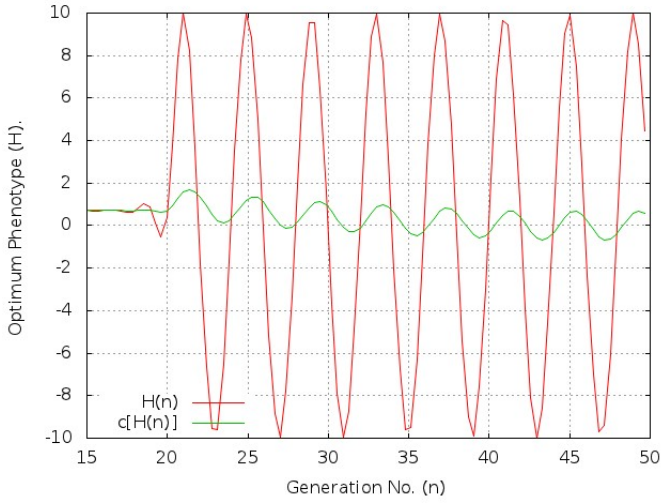
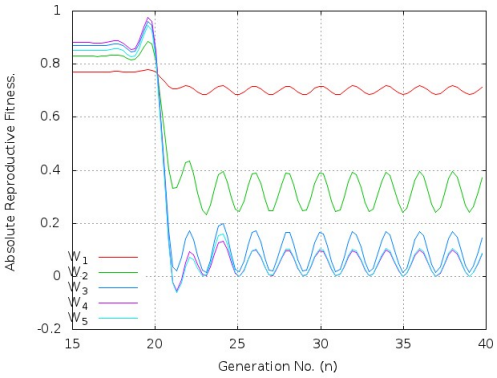
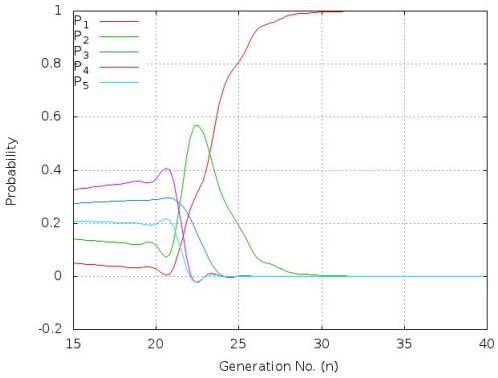


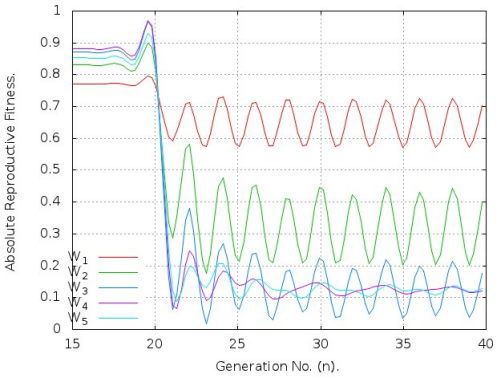
Figure 8: Natural and cultural environmental optimums.



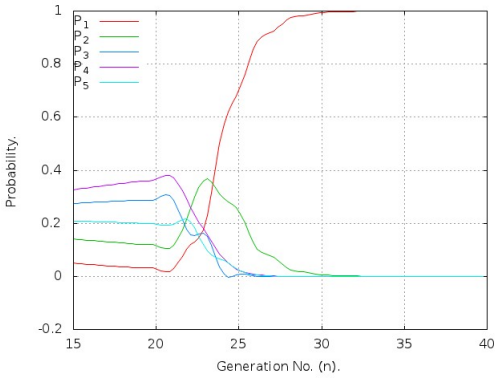
(a) Absolute reproductive fitness: $\lambda = 3, \sigma = 0$



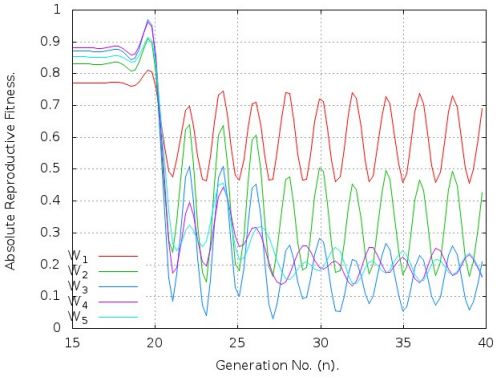
(b) Probability of individual receiving i'th allele: $\lambda = 3, \sigma = 0$



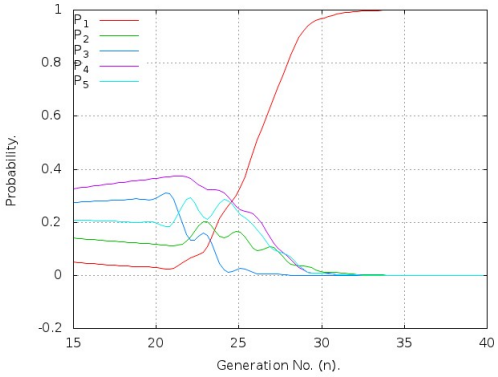
(c) Absolute reproductive fitness: $\lambda = 2.5, \sigma = 0.5$



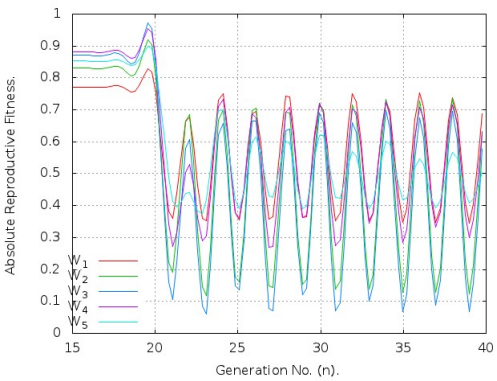
(d) Probability of individual receiving i'th allele: $\lambda = 2.5, \sigma = 0.5$



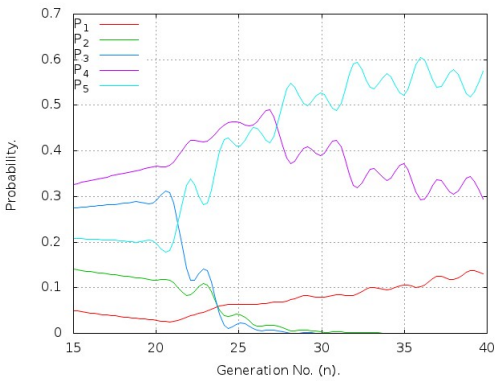
(e) Absolute reproductive fitness: $\lambda = 2, \sigma = 1$



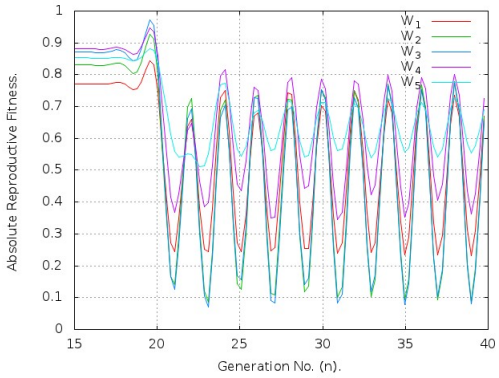
(f) Probability of individual receiving i'th allele: $\lambda = 2, \sigma = 1$



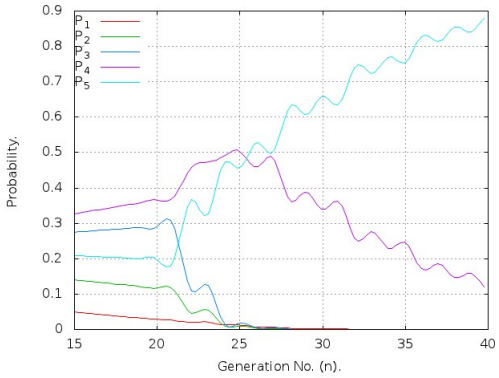
(g) Absolute reproductive fitness: $\lambda = 1.5, \sigma = 1.5$



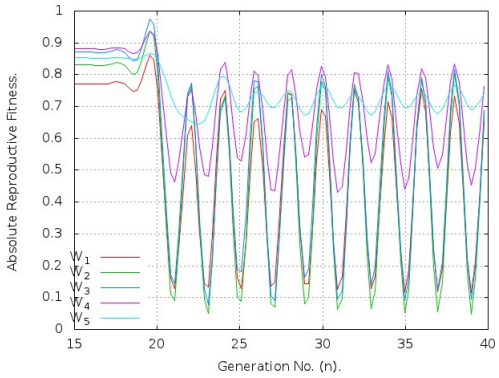
(h) Probability of individual receiving i'th allele: $\lambda = 1.5, \sigma = 1.5$



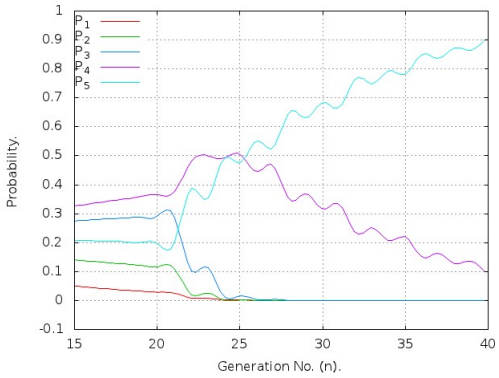
(i) Absolute reproductive fitness: $\lambda = 1, \sigma = 2$



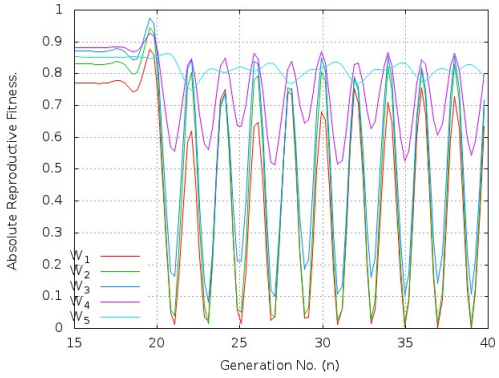
(j) Probability of individual receiving i'th allele: $\lambda = 1, \sigma = 2$



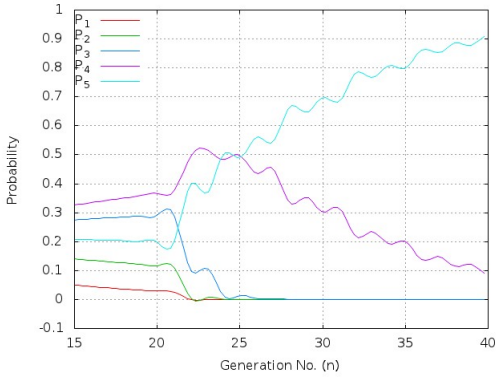
(k) Absolute reproductive fitness: $\lambda = 0.5, \sigma = 2.5$



(l) Probability of individual receiving i'th allele: $\lambda = 0.5, \sigma = 2.5$



(m) Absolute reproductive fitness: $\lambda = 0, \sigma = 3$

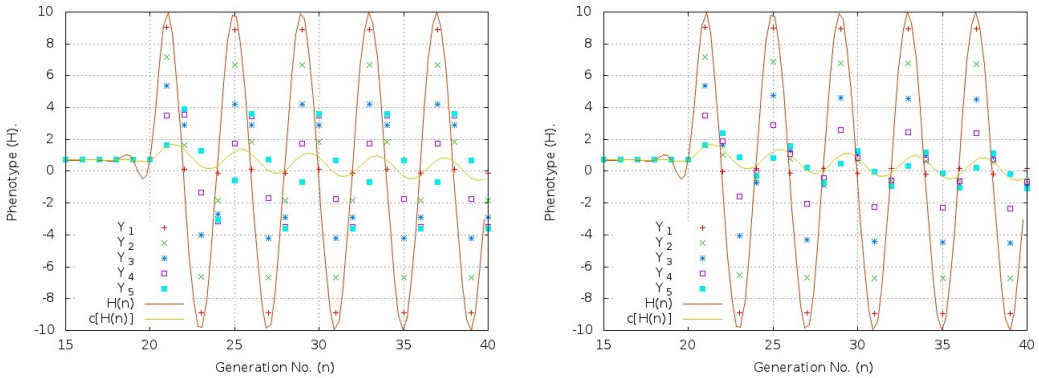


(n) Probability of individual receiving i'th allele: $\lambda = 0, \sigma = 3$

Figure 9: Fluctuations

There are several competing processes here. Firstly, the similarity between the selection

event in figure 7 and fluctuation in figure 7;as we add cultural fitness to the weighting in the case of the selection event, the increasingly dominating effect of decreasing cultural fitness over natural fitness has the relatively ordered effect of harming absolute fitness most with the best learners and least with the worst. In the case of fluctuations, we see the same overall effect over each fluctuation, but not at each point in a fluctuation where we see the fitness lines of different learners crossing as the cultural fitness dependence is increased. Comparing all the above absolute fitness measures above, starting from 9(c) and working down, we see a now well observed phenomenon of better learners being fitter in erratic environments, but we also see a clustering of the fitness of the worst three learners through 9(e) to 9(g). To see why this is happening, we pick the case in which this seems clearest; that of pure natural fitness shown in subfigure 9(c), compare to the mean's of 9(i) and look at the N_g means of the mature phenotype distributions:



(a) $\lambda = 3, \sigma = 0$.

(b) $\lambda = 0 \sigma = 3$

Figure 10: i'th mature phenotype with N_g line fits removed.

So what we see is the culmination of the guided variation, encultrination rule and environment causing this clustering. The actual cultural measure of fitness seems to have little direct effect on this particular occurrence. If we change the proportion of an individual's phenotypic trait that they receive from $\rho = 0.5$ to $\rho = 0.1$, while not shown here we do lose much of this clustering but this means reducing the connection between genetic and phenotypic traits to very low levels. This is quite interesting as this clustering is then representative of a symmetry of the environmental variation - allele's 4 and 5 are increasing their phenotypic value as allele 3 is reducing its own and they meet in the same region every cycle of the environment. This clustering is lost if the period of $H(n)$ is altered significantly, but raises the question as to whether under certain learning conditions, this could occur on larger timescales over a broader range of learners? What would happen if there were a drastic selection event at a time when this clustering were occurring? It seems likely that distinct sets of alleles would be selected(or de-selected depending on the fitness measure) because they happened to be in the right phenotypic location at the right time. This is a phenomenon impossible with selection events.
Coming back to the cultural fitness measure, it can be seen looking through the graphs that

as we increase the cultural weighting, the reversal of absolute fitness for each allele does not occur in the clear cut way in which it did for the selection event. Looking at the behaviour of allele 5 in particular as we increase cultural fitness weighting, we see its fitness magnitude fluctuating above and below the magnitudes of the other alleles in subfigures 9(i), 9(k) and 9(m). . While looking at the probability distributions, the relatively sudden effects of the fitness reversal start to favour the worst learners. The key point to note though is that although the effect on the overall order of fitness follows in a similar manner to the selection event, it is not certain - in this case we still see allele 5's fitness fluctuating between highest and lowest.

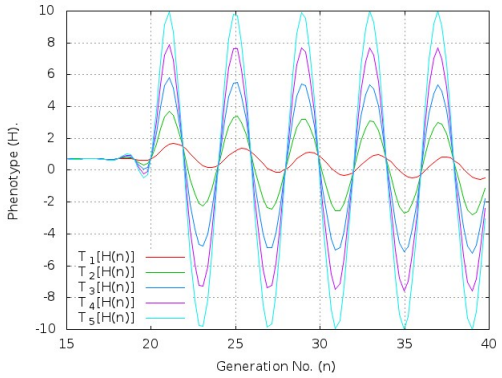
In summary; after incorporating a relative memory function $c[H(n)]$ associated with the group unit of selection under group selection -culture, we have inferred a measure of fitness in the phenotype centred DIT model at the reproduction phase and examined qualitative effects on the distribution of alleles as a measure of impact. For simplicity, we have assumed for the most part that an individual will always aim to match their traits to the local environmental optimum, but assumed that an individual's measure of fitness will be affected by a cultural memory of past fitness measures manifesting in the memory function. What we have found is that when considering this cultural imperative alongside that associated with Darwinian fitness, we can generally expect very different evolutionary directions for a group then when this cultural pressure is turned off.
In simplifying the analysis by making off the cultural memory's effect on learning objective, we made a slightly artificial separation between cultural fitness and cultural learning goal. It seems natural that a culture would impose/encultrinate concepts of learning ideal and fitness ideal together on its constituent individuals, as a special case, it does not seem unreasonable under the present set of assumptions to speculate that an individual within a society might prioritize their own 'selfish' learning objective over that of the culture to improve their natural fitness at the cost of their cultural fitness. While not shown in figure 7, in figure 7 we do see an ordered increase in natural fitness with those who rely less on encultrination and more on asocial learning.

8 Genetically determined cultural and natural learning goals.

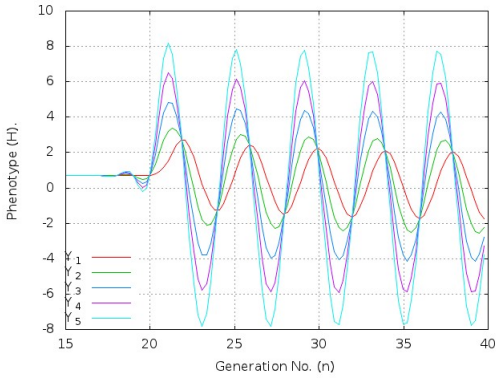
We would now like to see how varying the cultural and natural pressure weights of the individual's learning goal with κ_i and τ_i affect their fitness. We run three simulations, each with a spread of learning goals across alleles, but all with $L_i = 0.63$, all other parameters as before except $\rho = 0.2$(to allow more cultural diversity across alleles), $N_g = 5$ and we vary the weightings of the cultural and natural pressures:

Table 4: Parameters for figures 9, 8 and 8.

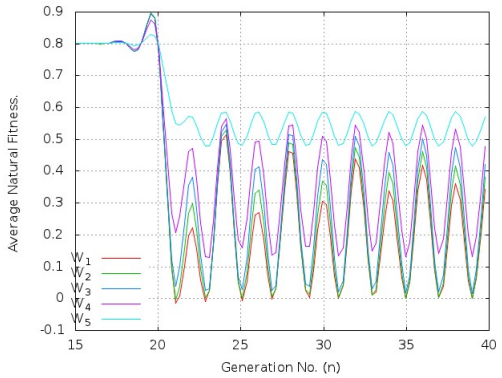
Allele	V_{trans_i}	V_{ϵ_i}	L_i	$a_i = \frac{V_{\epsilon_i}}{V_{\epsilon_i}+L_i}$	ρ	μ	ζ_i	κ_i	τ_i	N_C
1	1	2.5	10	0.2	0.5	0.5	1	0	0	20
2	1	2.5	10	0.2	0.5	0.5	0.66	0	0.33	20
3	1	2.5	10	0.2	0.5	0.5	0.5	0	0.5	20
4	1	2.5	10	0.2	0.5	0.5	0.33	0	0.66	20
5	1	2.5	10	0.2	0.5	0.5	0	0	1	20



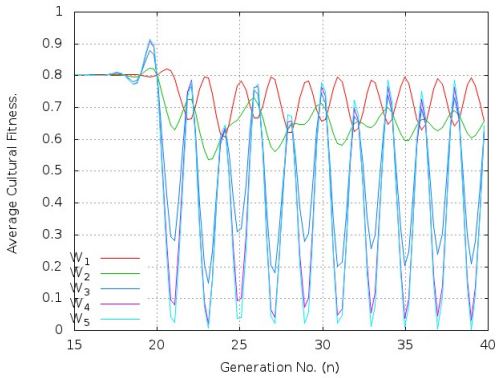
(a) i'th Learning goal



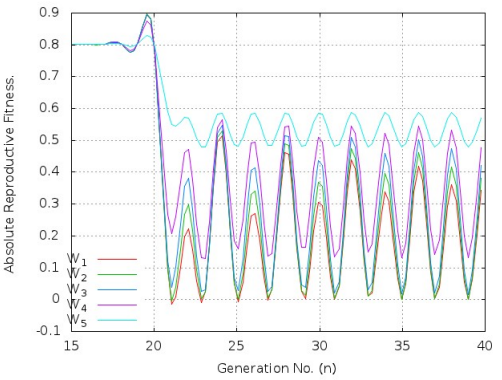
(b) Average mature phenotype.



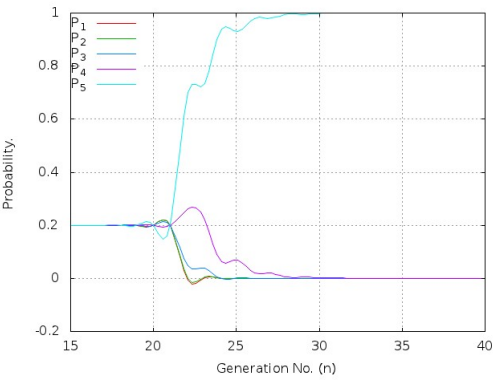
(c) Average natural fitness.



(d) Average cultural fitness.

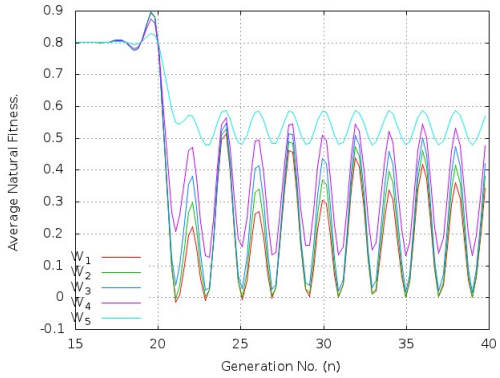


(e) Absolute reproductive fitness.

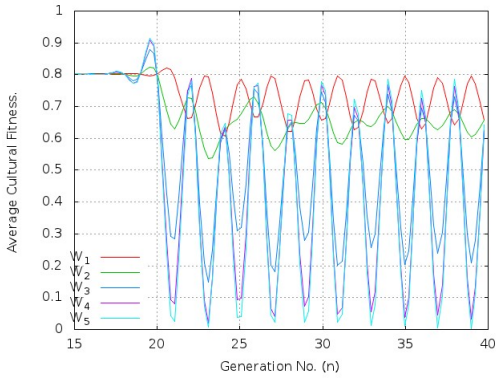


(f) Population allele distribution.

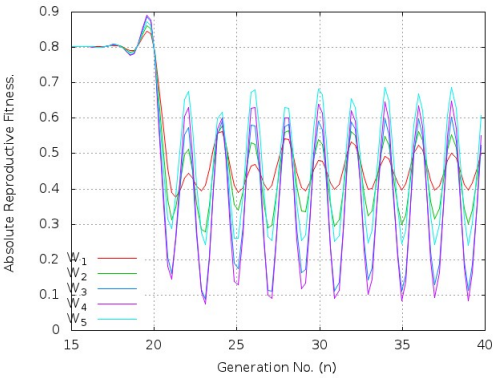
Figure 11: $\lambda = 1\sigma = 0$



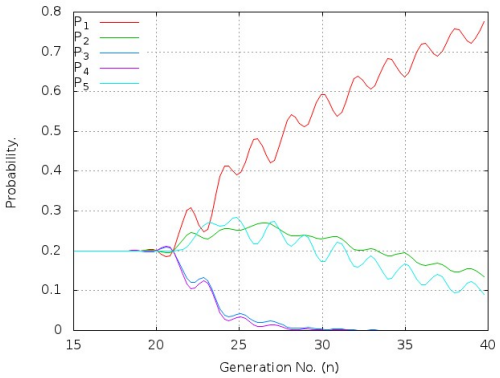
(a) Average natural fitness.



(b) Average cultural fitnes.s

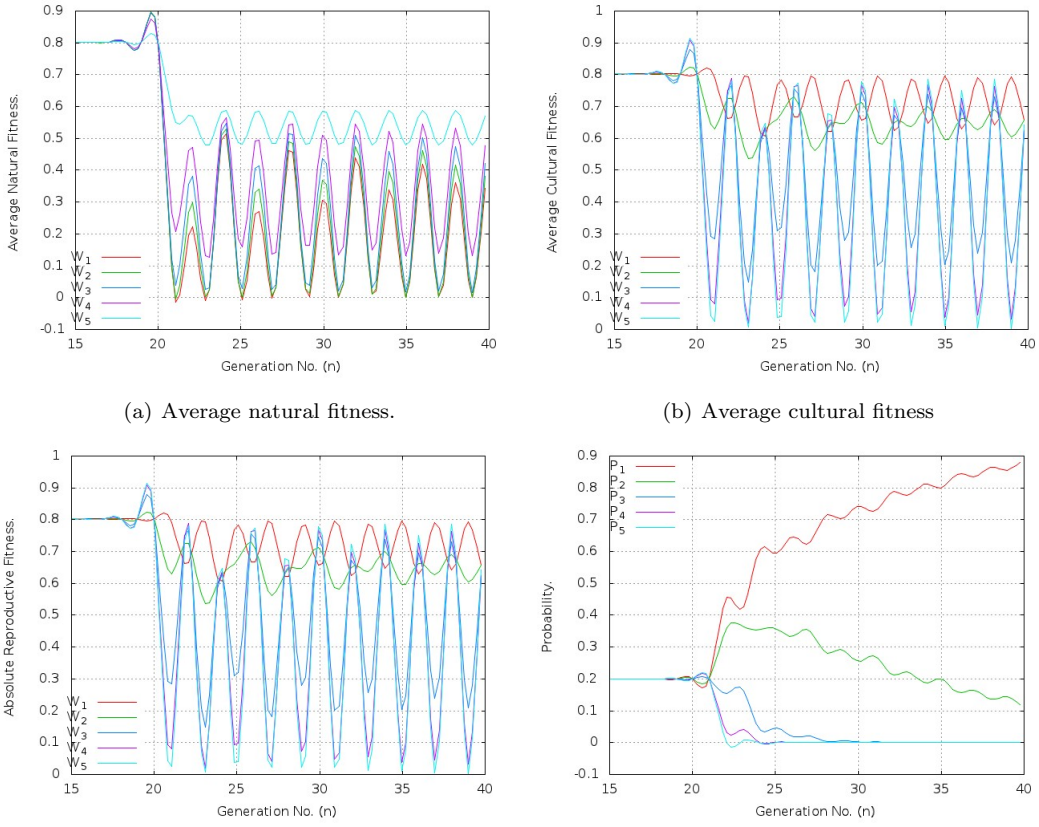


(c) Absolute reproductive fitness.



(d) Population allele distribution.

Figure 12: $\lambda = 1\sigma = 1$



(a) Average natural fitness.

(b) Average cultural fitness

(c) Absolute reproductive fitness.

(d) Population allele distribution.

Figure 13: $\lambda = 0\sigma = 1$

in 9, 8 and 8 we see very much what we would intuitively expect to see. With each allele now determining the propensity of an individual to follow the natural or cultural learning and the cultural selection pressures increased w.r.t the natural pressures through the three simulations, the trend established is that individuals described by an allele favouring the cultural learning objective are increasingly favoured when cultural selection is strongest and those with alleles favouring the natural learning goal are favoured under natural selection pressures.

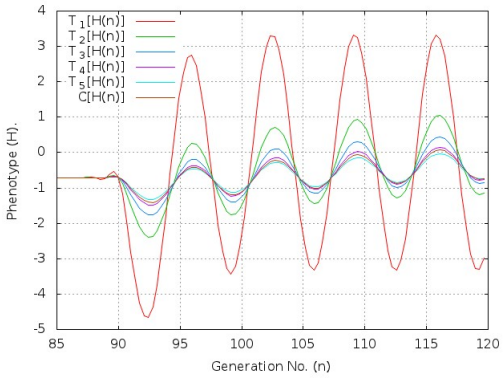
9 Simulations of Genetically Determined Learning Objectives Under Cultural Selection.

In the previous section we added a cultural memory measure to Darwinian fitness and simulated the evolution of the society under cultural and Darwinian selection pressures. On considering the cultural unit of selection we found an intuitive way to incorporate its memory effect into the Darwinian model that we adapted, originally designed to demonstrate

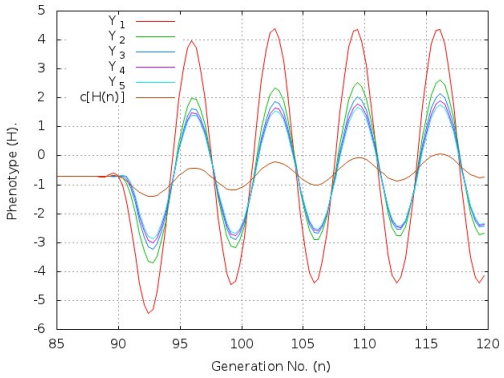
natural selection acting on the phenotype unit. Now we would like to look at the analogous speculated genome scale selection effect on the individual's learning goal through the genome scale selection mechanism. While it is not so intuitive to propose this genetic memory effect as it was for its cultural counterpart, from the perspective of the scale selection mechanism and the integrity of information transmission, it is in principle no more unreasonable. It would be nice to consider the effect on perceived fitness, but we have not had time to construct this measure and code it. without a fitness measure incorporating this memory effect that might favour this genetic memory, we therefore aim to explore the possibility of cultural pressures favouring the genetic transmission of learning goal. This differs from the previous section in that now the genetic memory describes the learning goal itself rather than describing the propensity of an individual to rely on the cultural goal. With this in mind we assign cultural selection as the sole selection mechanism and define the learning objective entirely genetically; un realistic but allows the exploration of the question as to what happens if cultural pressures coincide with genetic goals.

Table 5: Parameters for first genetic objective and cultural selection simulation.

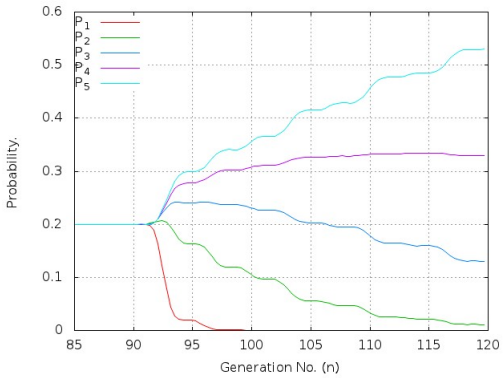
Allele	V_{trans_i}	V_{ϵ_i}	L_i	$a_i = \frac{V_{\epsilon_i}}{V_{\epsilon_i}+L_i}$	ρ	μ	λ	σ	ζ_i	κ_i	τ_i	N_C	N_{G_i}
1	1	2.5	10	0.2	0.5	0.5	0	1	0	1	0	50	10
2	1	2.5	10	0.2	0.5	0.5	0	1	0	1	0	50	30
3	1	2.5	10	0.2	0.5	0.5	0	1	0	1	0	50	50
4	1	2.5	10	0.2	0.5	0.5	0	1	0	1	0	50	70
5	1	2.5	10	0.2	0.5	0.5	0	1	0	1	0	50	70



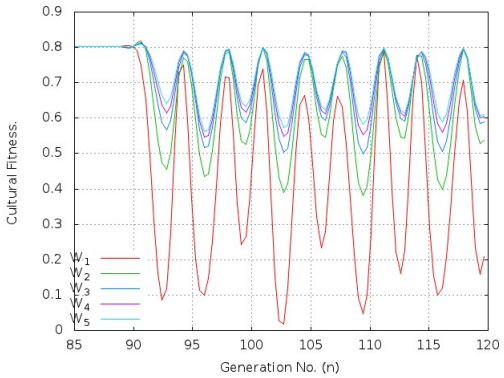
(a) i'th Learning goal



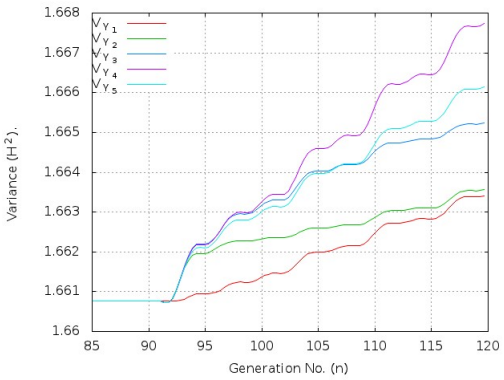
(b) Average mature phenotype with allele i.



(c) Proportion of population with allele i.



(d) Average cultural fitness



(e) Variance of i'th phenotype.

Figure 14: Simulation of genetic learning goal and cultural selection.

What we see here is quite unexpected an I think remarkable. We expected here that the cultural selection pressure would favour allele 3, the allele whose genetic memory length in its learning goal matched the cultural memory length, but in fact, it is favouring allele 5! This is the allele whose genetically determined learning goal is characterized by the longest

memory length, while all other parameters are held constant across alleles. This is the strongest evidence so far that a longer memory effect in learning goal is favoured under a single selection pressure acting identically over all phenotypes, at least in certain environments. This is what we were looking for earlier in section 6, where we were looking for an evolutionary advantage of cultural memory acting on the learning goal under N.Selection with simple fluctuations, selection events and different learners. Because both cultural selection and natural selection act on all alleles equally(they both act on the phenotype) this means that in similar environments this model describes an evolutionary advantage under N.Selection for a population **do not** seek to adapt their phenotype to the local environment, but rely on unique genetically driven learning that do not vary in such an extreme manner as the natural learning goals varying more rapidly on a generational time scale. Referring back to the distinction between the two types of variation discussed earlier, this memory effect will only give an advantage under natural selection when we are dealing with a fluctuating environment; during selection events this will have infinitely lasting negative impact on fitness under N.Selection and cultural selection so long as $N_{G_i} \geq N_C \forall i$.

To understand the reasons for the fitness advantage in more detail, we have to look at how the mature phenotype tracks its learning goal. Because the variance across relevant generations and alleles varies so little, we can approximate this to be a function of $|\bar{Y}_i(n) - c[H(n)]|$ where the smaller this value, the greater the fitness will be.

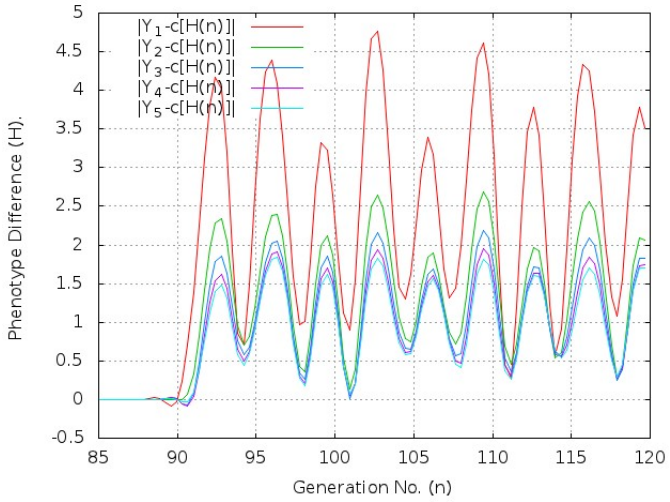


Figure 15: $|\bar{Y}_i(n) - c[H(n)]|$.

Here we see that the lowest values for $|\bar{Y}_i(n) - c[H(n)]|$ are indeed held by allele 5, but not constantly and there is certainly little in the difference, with the allele 3 holding very similar values here followed roughly by alleles 4, 2 and 1.

Part III

Summary.

10 Conclusions.

The initial aim of this thesis was to examine the apparent evolutionary promotion of locally maladaptive genetic and phenotypic traits. We started by conceptually constructing temporal domains of selection and then built a phenotype centred model of a dual inheritance theory described by the guided variation process. The corresponding temporally defined measures of perceived optimum phenotype and average fitness induced at the generational phenotype scale at which Natural Selection would usually act were incorporated by using functions representative of the environment/learning goal $H(n), c[H(n)]$ and $g_i[H(n)]$, for which the latter two averaged over a history of local optimums.

Constructing a model and simulating this generational process, first looking at the traditional action of Natural Selection and entirely locally defined measures of optimum phenotype and learning goal, a high sensitivity to the relative magnitudes of cultural and asocial learning errors was found. When cultural learning errors were greater than asocial learning errors the static and varying environments tended to favour those who favoured encultrination over asocial learning. When asocial learning errors were greater than those associated with encultrination, alleles describing a predisposition to asocial learning were again not favoured in the static environment but tended to become dominant in the varying environment. We also found that the cultural transmission rule representing parental bias encouraged greater dispersion of mean phenotypes associated with each allele when the weighting for the genetic parent's importance in the process ρ was reduced.

We found that conflicting natural, cultural and genetic pressures were caused by different temporally defined optimums that acted to promote alleles and cultural traits differentially between them over different timescales. Traits that were considered optimally fit under the fitness measure corresponding to the traditional action of Natural Selection were often, though not always considered unfit under fitness measured by cultural and genetic optimums; alleles were also promoted differentially under the action of these different pressures. Distinguishing between fluctuating environments and selection events, we found that during selection events increasing the relative strength of the cultural fitness pressure when a population would seek to increase their fitness locally rather than through the same cultural measure of optimum would favour those in the population who relied more strongly on encultrination while during fluctuations, results were far more mixed.

The evolutionary advantage of genetically determined optimums appeared under cultural pressures and it can be trivially inferred to also be advantageous under the action of traditional Natural Selection, at least in certain environments, as described in the guided variation process used in the simulations. This is the strongest evidence found within the construction used that genetically determined phenotype optimums - instinct and predisposition, are advantageous even under Natural Selection in spite of the pressure that they can

cause to drive an individual's perceived optimum/objective away from the local optimum and become maladaptive by fitness measures under this selection mechanism.

11 Further Work.

Through the construction of the phenotype centred guided variation model we were able to qualitatively analyze the effects of introducing non-local optimums described by the memory effect. This I think has had some qualitative success, but was ultimately an adaptation of the phenotype centred model approach and while producing the local mal-adaptation that we were looking for employed the speculative memory effect that we justified on the grounds that information at the scale of the culture and the genome in the form of cultural transmission and genetic transmission respectively could be transmitted with low levels of degradation between generations. We removed the possibility of clear clashes between the actions of the selection mechanisms at all scales by only employing this memory effect as well as treating genetic and cultural transmission processes as induced at the phenotype scale - hence phenotype centred model. What might be interesting is to attempt to relate models of selection at these different scales through constructing a purpose built model incorporating corresponding fitness landscapes along with a far more thorough examination of natural origins of the selection process.

11.1 Cultural Transmission.

Many assumptions were made in the guided variation model to allow the qualitative effects of retarded transmission to show themselves most clearly at the population level, not least the manner in which population level determinism was required to result from the stochastic processes associated with the individual represented by random variables. This requirement, implemented largely by the demand that all distributions be Gaussian removed much of the potential for interesting cultural dynamics to evolve. The assignment of one allele and a one dimensional space of cultural traits removed the possibility of considerations of selection landscapes [6]and further restricted the potential for dynamics, as well as a more thorough examination of sources of cultural and genetic variation. This was done deliberately in this case and the guided variation model allowed a particular feature, the memory effect act, but in so far as cultural transmission processes are concerned the weighted average of cultural traits representing a parental bias was very simplistic. It did feature the two main sources of individual learning though, social and asocial, but together with the above restrictions does not reflect the complexity of encultrination or the basic rationale behind any learning process, trial and error learning.

Part IV

Appendix & References.

12 Appendix.

Appendix (A) - The following is the fortran source code used to simulate the guided variation process.

```
program gene

integer,parameter::number_of_timesteps=200, number_of_alleles=5, integration_steps=20000
integer,dimension(number_of_alleles)::n_memory_steps,ngs_memory_steps
integer::i,j,m
real(8)::a, num
real(8), parameter::pi=3.1415926535898
real(8),dimension
(number_of_timesteps,number_of_alleles)::p_n_i,x_n_i,v_x_n_i,y_n_i,v_y_n_i,g_n_i,group_n_i,tp_n_i
real(8),dimension(number_of_timesteps)::H_n,H_n_dummy,H_n_dummy2
real(8),dimension(number_of_alleles)::wbar,xprevious,a_parameters,V_epsilon,V_transmission,S_parameters
real(8),dimension(number_of_alleles)::beta_i,NS_parameters,alpha_i,GRS_parameters
real
(8)::denom_p,denom_gam,sum,h_previous,output,sum3,lower_limit,upper_limit,sum5,sum4,sum8,sum9,sum10,sum11,
real(8),dimension(number_of_alleles,number_of_alleles)::gamma
real(8), dimension
(integration_steps)::w_integrand,Wbar_integrand_sel,Wbar_integrand_grs,w_integrand_grs
real(8), dimension(integration_steps)::w_integrand_ns,w_integrand2,Wbar_integrand_nats
real(8),dimension
(number_of_alleles)::sel,nat_sl,wbar_sl,wbar_ns,wbar_grs,group_sl,seltp,nat_sltp,group_sltp

open (unit = 4, file = "w.dat")

open (unit = 7, file = "w_nat.dat")
open (unit = 8, file = "w_group.dat")
open (unit = 9, file = "w_genetic.dat")
open (unit = 10, file = "w4.dat")

rho=0.5
mu=1-rho

!limits_of_wbar_integral
lower_limit=-200.d0
upper_limit=200.d0

!a_parameters
a_parameters(1)=0.2
a_parameters(2)=0.2
a_parameters(3)=0.2
a_parameters(4)=0.2
a_parameters(5)=0.2
!a_parameters(6)=0.6
!a_parameters(7)=0.7
!a_parameters(8)=0.8
!a_parameters(9)=0.9

!relative target phenotype group, genetic and natural optimum phenotype strengths
seltp(1)=01.d0
seltp(2)=01.d0
seltp(3)=01.d0
seltp(4)=01.d0
seltp(5)=01.d0
!seltp(6)=0!1.d0
!seltp(7)=0!1.d0
!seltp(8)=0!1.d0
!seltp(9)=0!1.d0

nat_sltp(1)=0.d0
nat_sltp(2)=0.d0
nat_sltp(3)=0.d0
nat_sltp(4)=0.d0
nat_sltp(5)=0.d0
!nat_sltp(6)=01.d0
!nat_sltp(7)=01.d0
!nat_sltp(8)=01.d0
!nat_sltp(9)=01.d0

group_sltp(1)=0!1.d0
group_sltp(2)=0!1.d0
group_sltp(3)=0!1.d0
group_sltp(4)=0!1.d0
group_sltp(5)=0!1.d0
```

```
!group_sltp(6)=0!1.d0
!group_sltp(7)=0!1.d0
!group_sltp(8)=0!1.d0
!group_sltp(9)=0!1.d0

!relative selection versus natural selection strength parameters
sel(1)=0!1.d0
sel(2)=0!1.d0
sel(3)=0!1.d0
sel(4)=0!1.d0
sel(5)=0!1.d0
!sel(6)=6.d0
!sel(7)=0!7.d0
!sel(8)=0!8.d0
!sel(9)=0!9.d0
nat_sl(1)=0!2.5d0
nat_sl(2)=0!2.5d0
nat_sl(3)=0!2.5d0
nat_sl(4)=0!2.5d0
nat_sl(5)=0!2.5d0
!nat_sl(6)=2.d0
!nat_sl(7)=2.d0
!nat_sl(8)=2.d0
!nat_sl(9)=2.d01
group_sl(1)=01.d0
group_sl(2)=01.d0
group_sl(3)=01.d0
group_sl(4)=01.d0
group_sl(5)=01.d0
!group_sl(6)=1.d0
!group_sl(7)=1.d0
!group_sl(8)=1.d0
!group_sl(9)=1.d0

!!memory parameters
n_memory_steps(1)=10
n_memory_steps(2)=30
n_memory_steps(3)=50
n_memory_steps(4)=70
n_memory_steps(5)=90
!n_memory_steps(6)=20
!n_memory_steps(7)=20
!n_memory_steps(8)=20
!n_memory_steps(9)=20

ngs_memory_steps(1)=50
ngs_memory_steps(2)=50
ngs_memory_steps(3)=50
ngs_memory_steps(4)=50
ngs_memory_steps(5)=50
!ngs_memory_steps(6)=20
!ngs_memory_steps(7)=20
!ngs_memory_steps(8)=20
!ngs_memory_steps(9)=20

!S_parameters, NS_parameters
S_parameters(1)=3
S_parameters(2)=3
S_parameters(3)=3
S_parameters(4)=3
S_parameters(5)=3
!S_parameters(7)=10
!S_parameters(8)=10
!S_parameters(9)=10

NS_parameters(1)=3
NS_parameters(2)=3
NS_parameters(3)=3
NS_parameters(4)=3
NS_parameters(5)=3
!NS_parameters(6)=3
!NS_parameters(7)=3
```

```
!NS_parameters(8)=3
!NS_parameters(9)=3

GRS_parameters(1)=3
GRS_parameters(2)=3
GRS_parameters(3)=3
GRS_parameters(4)=3
GRS_parameters(5)=3
!GRS_parameters(6)=3
!GRS_parameters(7)=3
!GRS_parameters(8)=3
!GRS_parameters(9)=3

!V_epsilon constants
V_epsilon(1)=2.5
V_epsilon(2)=2.5
V_epsilon(3)=2.5
V_epsilon(4)=2.5
V_epsilon(5)=2.5
!V_epsilon(6)=2.5
!V_epsilon(7)=2.5
!V_epsilon(8)=2.5
!V_epsilon(9)=2.5

!V_transmission constants
V_transmission(1)=1.d0
V_transmission(2)=1.d0
V_transmission(3)=1.d0
V_transmission(4)=1.d0
V_transmission(5)=1.d0
!V_transmission(6)=1.d0
!V_transmission(7)=1.d0
!V_transmission(8)=1.d0
!V_transmission(9)=1.d0

!Initial conditions
num=number_of_alleles

p_n_i(1,1)=1/num
p_n_i(1,2)=1/num
p_n_i(1,3)=1/num
p_n_i(1,4)=1/num
p_n_i(1,5)=1/num
!p_n_i(1,6)=1/num
!p_n_i(1,7)=1/num
!p_n_i(1,8)=1/num
!p_n_i(1,9)=1/num

v_x_n_i(1,1)=1.2d0
v_x_n_i(1,2)=1.2d0
v_x_n_i(1,3)=1.2d0
v_x_n_i(1,4)=1.2d0
v_x_n_i(1,5)=1.2d0
!v_x_n_i(1,6)=1.2d0
!v_x_n_i(1,7)=1.2d0
!v_x_n_i(1,8)=1.2d0
!v_x_n_i(1,9)=1.2d0

!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!^^^^^PARAMETERS/INITIAL CONDITIONS^^^^   _ _ _MAIN PROGRAM_ _
_ _!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!

!H-functions

do i=1,number_of_timesteps
!
!H_n_dummy(i)=10*cos(0.25*i*2*pi-1.5)!+1.0*cos(0.01*i*pi)+3*cos(0.04*i*2*pi)+10*cos(0.01*i*2*pi)+20*cos
(0.004*i*2*pi)+50*cos(0.001*i*pi)
H_n_dummy(i)=10*cos(0.15*i*2*pi-1.5)!+1.0*cos(0.01*i*pi)+3*cos(0.04*i*2*pi)+10*cos(0.01*i*2*pi)+20*cos
(0.004*i*2*pi)+50*cos(0.001*i*pi)

!a=i
!H_n_dummy(i)=5*(2+atan(10*(a-25)))
```

```
!H_n_dummy(i)=10.d0

!H_n_dummy(i)=2+15*( &
!exp(-(i-n_memory_steps(number_of_alleles)-10)**2.0/1.5) &
!+exp(-(i-n_memory_steps(number_of_alleles)-20)**2.0/2) &
!+exp(-(i-n_memory_steps(number_of_alleles)-35)**2.0/2) &
!+exp(-(i-n_memory_steps(number_of_alleles)-60)**2.0/2) &
!+exp(-(i-n_memory_steps(number_of_alleles)-100)**2.0/2) &
!+exp(-(i-n_memory_steps(number_of_alleles)-150)**2.0/2) &
!+exp(-(i-n_memory_steps(number_of_alleles)-220)**2.0/2) &
!)

if (i .LE. n_memory_steps(number_of_alleles)) then
H_n=H_n_dummy(n_memory_steps(number_of_alleles))
else
H_n(i)=H_n_dummy(i)
end if
end do

!x_n_i initial conditions
x_n_i(1,1)=H_n(1)
x_n_i(1,2)=H_n(1)
x_n_i(1,3)=H_n(1)
x_n_i(1,4)=H_n(1)
x_n_i(1,5)=H_n(1)
!x_n_i(1,6)=H_n(1)
!x_n_i(1,7)=H_n(1)
!x_n_i(1,8)=H_n(1)
!x_n_i(1,9)=H_n(1)

!!g_n_i function initial conditions
do k=1,number_of_alleles
do m=1,n_memory_steps(k)
g_n_i(m,k)=H_n(m)
end do

!!g_n_i function
do i=n_memory_steps(k)+1,number_of_timesteps
alpha_i(k)=LOG(20.0)/n_memory_steps(k)
!end do

sum5=0.d0
do m=0,n_memory_steps(k)
sum5=sum5+exp(-alpha_i(k)*m)*H_n(i-m)
end do

g_n_i(i,k)=(1-exp(-alpha_i(k)))/(1-exp(-alpha_i(k)*(n_memory_steps(k)+1)))*sum5

end do
end do
!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!1
!!group_n_i function initial conditions
do k=1,number_of_alleles
do m=1,ngs_memory_steps(k)
group_n_i(m,k)=H_n(m)
end do

!!group_n_i function
do i=ngs_memory_steps(k)+1,number_of_timesteps
beta_i(k)=LOG(5.0)/ngs_memory_steps(k)

sum9=0.d0
do m=0,ngs_memory_steps(k)
sum9=sum9+exp(-beta_i(k)*m)*H_n(i-m)
end do

group_n_i(i,k)=(1-exp(-beta_i(k)))/(1-exp(-beta_i(k)*(ngs_memory_steps(k)+1)))*sum9

end do
end do
```

```
open (unit = 15, file = "g.dat")
do i=1,number_of_timesteps
write(15,*),i,H_n(i),(g_n_i(i,j),j=1,number_of_alleles)
end do

!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!

!target optimum phenotype

do j=1,number_of_alleles
tp_n_i(1,j)=(nat_sltp(j)*H_n(1)+seltp(j)*g_n_i(1,j)+group_sltp(j)*group_n_i(1,j))/(nat_sltp(j)
+group_sltp(j)+seltp(j))
y_n_i(1,j)=a_parameters(j)*x_n_i(1,j)+(1-a_parameters(j))*tp_n_i(1,j)
v_y_n_i(1,j)=a_parameters(j)*a_parameters(j)*v_x_n_i(1,j)+(1-a_parameters(j))**2*V_epsilon(j)
end do

do j=1,number_of_alleles

!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!

do k=1, integration_steps
dummy_Y=lower_limit+k*(upper_limit-lower_limit)/integration_steps
w_integrand_ns(k)=exp(-(dummy_Y-H_n(1))**2/2.0/NS_parameters(j))                    !1.d0/
((2.d0*pi*NS_parameters(j))**0.5)*
w_integrand2(k)=1.d0/((2.d0*pi*v_y_n_i(1,j))**0.5)*exp(-(dummy_Y-y_n_i(1,j))**2/2.0/v_y_n_i(1,j))
Wbar_integrand_nats(k)=w_integrand_ns(k)*w_integrand2(k)
end do

sum4=0.d0
do k=1, integration_steps-1
sum4=sum4+0.5*(Wbar_integrand_nats(k+1)+Wbar_integrand_nats(k))
end do

sum4=sum4*(upper_limit-lower_limit)/integration_steps

wbar_ns(j)=sum4


!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!


do k=1, integration_steps
dummy_Y=lower_limit+k*(upper_limit-lower_limit)/integration_steps
w_integrand(k)=exp(-(dummy_Y-g_n_i(1,j))**2/2.0/S_parameters(j))                    !1.d0/
((2.d0*pi*S_parameters(j))**0.5)*
w_integrand2(k)=1.d0/((2.d0*pi*v_y_n_i(1,j))**0.5)*exp(-(dummy_Y-y_n_i(1,j))**2/2.0/v_y_n_i(1,j))
Wbar_integrand_sel(k)=w_integrand(k)*w_integrand2(k)
end do

sum8=0.d0
do k=1, integration_steps-1
sum8=sum8+0.5*(Wbar_integrand_sel(k+1)+Wbar_integrand_sel(k))
end do

sum8=sum8*(upper_limit-lower_limit)/integration_steps

wbar_sl(j)=sum8

!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!

do k=1, integration_steps
dummy_Y=lower_limit+k*(upper_limit-lower_limit)/integration_steps
w_integrand_grs(k)=exp(-(dummy_Y-group_n_i(1,j))**2/2.0/GRS_parameters(j))                    !1.d0/
((2.d0*pi*GRS_parameters(j))**0.5)*
w_integrand2(k)=1.d0/((2.d0*pi*v_y_n_i(1,j))**0.5)*exp(-(dummy_Y-y_n_i(1,j))**2/2.0/v_y_n_i(1,j))
Wbar_integrand_grs(k)=w_integrand_grs(k)*w_integrand2(k)
end do

sum10=0.d0
do k=1, integration_steps-1
sum10=sum10+0.5*(Wbar_integrand_grs(k+1)+Wbar_integrand_grs(k))
end do
```

```
sum10=sum10*(upper_limit-lower_limit)/integration_steps

wbar_grs(j)=sum10

!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!

wbar(j)=(nat_sl(j)*wbar_ns(j)+sel(j)*wbar_sl(j)+group_sl(j)*wbar_grs(j))/(nat_sl(j)+sel(j)+group_sl(j))

end do
!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!

do i=1,number_of_alleles
print *, wbar(i)!,wbar_ns(i),wbar_sl(i),wbar_grs(i)
end do

!do timesteps
do i=2, number_of_timesteps

!define previous x vector
do j=1,number_of_alleles
xprevious(j)=x_n_i(i-1,j)
end do

!denominator for P(n+1)
denom_p=0.0
do j=1,number_of_alleles
denom_p=denom_p+wbar(j)*p_n_i(i-1,j)
end do

!calculate gamma
do j=1,number_of_alleles
denom_gam=denom_p-p_n_i(i-1,j)*wbar(j)
do k=1,number_of_alleles
if (j .ne. k) then
gamma(j,k)=mu*(p_n_i(i-1,k)*wbar(k))/denom_gam
else
gamma(j,k)=rho
end if
end do
end do

!H_thistimestep
h_previous=H_n(i-1)

do j=1,number_of_alleles
p_n_i(i,j)=p_function(denom_p,wbar(j),p_n_i(i-1,j))

!calc new x
call x_function(gamma,xprevious,a_parameters,h_previous,number_of_alleles,j,output)
x_n_i(i,j)=output

sum3=0.d0
do k=1,number_of_alleles
sum3=sum3+gamma(j,k)*gamma(j,k)*v_y_n_i(i-1,k)
end do
sum3=sum3+V_transmission(j)
v_x_n_i(i,j)=sum3

tp_n_i(i,j)=(nat_sltp(j)*H_n(i)+seltp(j)*g_n_i(i,j)+group_sltp(j)*group_n_i(i,j))/(nat_sltp(j)
+group_sltp(j)+seltp(j))
y_n_i(i,j)=a_parameters(j)*x_n_i(i,j)+(1-a_parameters(j))*tp_n_i(i,j)
v_y_n_i(i,j)=a_parameters(j)*a_parameters(j)*v_x_n_i(i,j)+(1-a_parameters(j))**2*V_epsilon(j)

!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!

do k=1, integration_steps
dummy_Y=lower_limit+k*(upper_limit-lower_limit)/integration_steps
w_integrand_ns(k)= exp(-(dummy_Y-H_n(i))**2/2.0/NS_parameters(j))                  !1.d0/
((2.d0*pi*NS_parameters(j))**0.5)*
w_integrand2(k)=1.d0/((2.d0*pi*v_y_n_i(i,j))**0.5)*exp(-(dummy_Y-y_n_i(i,j))**2/2.0/v_y_n_i(i,j))
Wbar_integrand_nats(k)=w_integrand_ns(k)*w_integrand2(k)
```

```
end do

sum4=0.d0
do k=1, integration_steps-1
sum4=sum4+0.5*(Wbar_integrand_nats(k+1)+Wbar_integrand_nats(k))
end do

sum4=sum4*(upper_limit-lower_limit)/integration_steps

wbar_ns(j)=sum4

!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!

do k=1, integration_steps
dummy_Y=lower_limit+k*(upper_limit-lower_limit)/integration_steps
w_integrand(k)=exp(-(dummy_Y-g_n_i(i,j))**2/2.0/S_parameters(j))                    !1.d0/
((2.d0*pi*S_parameters(j))**0.5)*
w_integrand2(k)=1.d0/((2.d0*pi*v_y_n_i(i,j))**0.5)*exp(-(dummy_Y-y_n_i(i,j))**2/2.0/v_y_n_i(i,j))
Wbar_integrand_sel(k)=w_integrand(k)*w_integrand2(k)
end do

sum8=0.d0
do k=1, integration_steps-1
sum8=sum8+0.5*(Wbar_integrand_sel(k+1)+Wbar_integrand_sel(k))
end do

sum8=sum8*(upper_limit-lower_limit)/integration_steps

wbar_sl(j)=sum8

!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!

do k=1, integration_steps
dummy_Y=lower_limit+k*(upper_limit-lower_limit)/integration_steps
w_integrand_grs(k)=exp(-(dummy_Y-group_n_i(i,j))**2/2.0/GRS_parameters(j))          !1.d0/
((2.d0*pi*GRS_parameters(j))**0.5)*
w_integrand2(k)=1.d0/((2.d0*pi*v_y_n_i(i,j))**0.5)*exp(-(dummy_Y-y_n_i(i,j))**2/2.0/v_y_n_i(i,j))
Wbar_integrand_grs(k)=w_integrand_grs(k)*w_integrand2(k)
end do

sum11=0.d0
do k=1, integration_steps-1
sum11=sum11+0.5*(Wbar_integrand_grs(k+1)+Wbar_integrand_grs(k))
end do

sum11=sum11*(upper_limit-lower_limit)/integration_steps

wbar_grs(j)=sum11

!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!

wbar(j)=(nat_sl(j)*wbar_ns(j)+sel(j)*wbar_sl(j)+group_sl(j)*wbar_grs(j))/(nat_sl(j)+sel(j)+group_sl(j))

end do
!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!!

print *, 'timestep',i,'wbar='
print *,i,(wbar_sl(j),j=1,number_of_alleles)

!open (unit = 4, file = "w.dat")

write(4,*),i,(wbar(j),j=1,number_of_alleles)

write(7,*),i,(wbar_ns(j),j=1,number_of_alleles)
write(8,*),i,(wbar_grs(j),j=1,number_of_alleles)
write(9,*),i,(wbar_sl(j),j=1,number_of_alleles)
write(10,*),i,wbar_ns(4),wbar_grs(4),wbar_sl(4)

end do

open (unit = 14, file = "tgni.dat")
do i=1,number_of_timesteps
```

```
write(14,*),i,(tp_n_i(i,j),j=1,number_of_alleles)
end do

open (unit = 20, file = "grp.dat")
do i=1,number_of_timesteps
write(20,*),i,(group_n_i(i,j),j=1,number_of_alleles)
end do

open (unit = 40, file = "gni.dat")
do i=1,number_of_timesteps
write(40,*),i,(g_n_i(i,j),j=1,number_of_alleles)
end do

open (unit = 3, file = "hy.dat")
do i=1,number_of_timesteps
write(3,*),i,H_n(i),(y_n_i(i,j),j=1,number_of_alleles)!,g_n_i(i,1),g_n_i(i,2),g_n_i(i,3),g_n_i
(i,4),y_n_i(i,1),y_n_i(i,2),y_n_i(i,3),y_n_i(i,4)
end do

open (unit = 11, file = "yg1.dat")
do i=1,number_of_timesteps
write(11,*),i,tp_n_i(i,4),y_n_i(i,4)
end do

open (unit = 18, file = "x.dat")
do i=1,number_of_timesteps
write(18,*),i,(x_n_i(i,j),j=1,number_of_alleles)
end do


open (unit = 17, file = "y.dat")
do i=1,number_of_timesteps
write(17,*),i,(y_n_i(i,j),j=1,number_of_alleles)
end do

open (unit = 5, file = "p.dat")
do i=1,number_of_timesteps
write(5,*),i,(p_n_i(i,j),j=1,number_of_alleles)
end do

open (unit = 16, file = "vy.dat")
do i=1,number_of_timesteps
write(16,*),i,(v_y_n_i(i,j),j=1,number_of_alleles)
end do

open (unit = 17, file = "vx.dat")
do i=1,number_of_timesteps
write(17,*),i,(v_x_n_i(i,j),j=1,number_of_alleles)
end do

open (unit = 60, file = "v.dat")
do i=1,number_of_timesteps
write(60,*),i,(abs(y_n_i(i,j)-group_n_i(i,j)),j=1,number_of_alleles)
end do

end program gene

real function p_function(denom,w_bar,p_prev)
real(8)::p_prev,w_bar,denom
p_function=P_prev*w_bar/denom
end function

subroutine x_function(gamma,x_prev,a,hfunction,num_alleles,allele,answer)
real(8),dimension(num_alleles,num_alleles)::gamma
real(8),dimension(num_alleles)::x_prev,a
real(8)::hfunction,sum2,answer
integer::allele

sum2=0.0
do i=1,num_alleles
sum2=sum2+gamma(allele,i)*(a(i)*x_prev(i)+(1-a(i))*hfunction)
end do
```

```
answer=sum2
end subroutine
```

References

[1] Boyd & Richerson *Transmission Coupling Mechanisms:Cultural Group Selection.* 2010: Philosophical Transactions of the Royal Society.

[2] Richard Dawkins *The Selfish Gene.* 1976: Oxford University Press.

[3] Boyd & Richerson *Cultural Evolution* 1985: The University of Chicago Press.

[4] J. M. Halley, S. Hartley, A. S. Kallimanis, W. E. Kunin, J. J.Lennon and S. P. Sgardelis *Uses and abuses of fractal methodology in ecology* 2004: Ecology Letters.

[5] Mark Lake *Archaeological inference and evolution* 1996: .....

[6] Dolph Schluter, Douglas Nychka *Exploration of Fitness Surfaces* 1993: The American Naturalist.

[7] Richard A. Watson *On The Unit of Selection in Sexual Populations* 2005: Advances in Artificial Life, Eighth European Conference (ECAL 2005).