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PARENTAL AND OBLIQUE TRANSMISSION AND INTERACTION BETWEEN TRAITS IN THE EVOLUTION OF NON-GENETIC TRAITS: SUGGESTIONS FOR A CULTURAL ECOLOGY

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Abstract: A model of the evolution of a trait, which is under phenotypic transmission, originally proposed by Feldmann and Cavalli-Sforza, is further investigated under the additional assumption the probability of oblique (non-parental) transmission being dependent on the previous diffusion of the trait in the population. This model is then applied to the contemporaneous diffusion of two or more traits interacting each other and influencing their transmission probabilities and/or the relative viabilities of the phenotypes carrying them.
The analysis is conducted algebraically in simple cases and *via* computer simulation in cases more complex. Relations between traits, and evolution in time of their diffusion, turn out similar to relations between living organisms in an ecological context (*e.g.* competition for the same niche, succession in a trophic chain) and evolution in time of their diffusion.
An analogy between cultural and biological evolution is then proposed, in wich the analogue of a living organism is applied to a cultural trait rather than to a whole culture. The analogue of the evolution of a whole culture is, under this view, the historical succession of climatic climaxes.

Introduction

Analogies between cultural and biological evolution have been suggested since the first appearance of Darwin's «Origin of species». Herbert Spencer, in his «Principles of Sociology», compared explicitly the growth of a society with that of an organism. In 1889, Sir Francis Galton proposed an analogy between biological mutations and technological innovation. The so-called «social darwinism» encompassed the idea of a struggle for life between cultures being analogous to the competition between different phenotypes to propagate their genetic trait. These theories have been rejected, not only because of the unacceptable social and ethical consequences they suggest, but also because the proposed analogies did not stand up to a deeper analysis (Childe 1947).

Hitherto, the analogue of the living organism in the cultural context, was applied to a whole social group. In 1975, Feldman and Cavalli-Sforza (Feldman and Cavalli-Sforza 1976) looked at the question from a novel point of view in two ways. In the first place, a quantitative approach to the problem was attempted, to overcome the inadequacy of purely qualitative analyses. In the second place, the evolution analysed was not that of a whole culture, but that of a single non-genetic trait. A non-genetic trait is a phenotype's characteristic that influences its behavior, and hence its viability, as a genetic trait does, but differs from the latter in its mode of transmission. By this definition, infectious diseases (and intestinal flora) are non-genetic traits transmitted by contagion; behaviors, attitudes and technologies are non-genetic traits transmitted *via* imitation, conditioning or learning. Feldman and Cavalli-Sforza apply the same quantitative analysis to both these kinds of traits, calling an individual having the trait «infected» or «skilled» interchangeably.

Models proposed by Feldman and Cavalli-Sforza assume implicitly that only one trait diffuses into a population over a period of time covering many generations or, if other traits are diffused contemporaneously, the different traits do not interact with each other. When these models are applied to human cultural traits, these assumpions must be abandoned: to have a complete picture of the process of cultural evolution, we are forced to study the contemporaneous diffusion of two or more traits that somehow interact.

In this paper, one of these models is considered. In the model, the transmission of a non-genetic trait can occur vertically, from parents to offspring, or obliquely, from adults to young people, with probabilities independent of genotypes: selection is also genotype-independent. In the first part this model, as originally proposed, is slightly modified to take into account the obvious fact that probabilities of oblique transmission should increase with the diffusion of a trait in the population. In the second part, the modified model is applied to contemporaneous diffusion of two traits that interact in some particular ways. The results are then discussed.

1) Diffusion of a single trait

The starting point of this analysis is eq. (34) of (Feldman and Cavalli-Sforza 1976). Feldman and Cavalli-Sforza consider the transmission of a non-genetic trait (skill or infection) that may come both from a parent and from a member of the social group. After transmission, a selection occurs: it depends only on the presence of the trait, and, following the Feldman and Cavalli-Sforza symbolism, the relative viability of an infected to an uninfected phenotype is $(1+s):1$ $(s \geq -1)$. Transmission does not depend on genotype, and the probability of any offspring being infected is

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b_3 if both parents are infected
b_2 if the «teaching» parent alone is infected
b_1 if the «non-teaching» parent alone is infected; and
b_0 if both parents are uninfected
$b_0 > 0$ implies that some form of oblique transmission is active.

Calling K the rate of infected phenotypes to the total before selection, Feldman and Cavalli-Sforza derive the following recursion formula:

$$K' = \frac{b_3(1+s)\ K\ +\ (b_1+b_2)\ (1+s)K(1-K)\ +\ b_0(1-K)}{1\ +\ sK} \quad (1)$$

which they discuss, in particular cases, assuming that b_0, b_1, b_2 and b_3 are constant. In order to account for an increased probability of contagion (or for social pressure toward learning) it seems appropriate to assume that the oblique transmission probability depends on the previous diffusion of the trait K.

To write explicitly $b_0 = b_0(K)$, some assumptions about the transmission process must be made. Schematically, the model proposed here is as follows: offspring are born, are subject to a given amount of parental care, during which vertical transmission may occur, reach adulthood living socially, subject to selection, and then reproduce. During social life, each offspring has a fixed number (say n) of random contacts with some adult, that, if infected, can infect (or influence behavior) with probability t: so after n contacts, the probability of an offspring staying uninfected is

$$1-b_0 = \left[1 - \frac{(1+s)K}{1+sK}\, t\right]^n \quad (2)$$

The same considerations apply to the interaction with *one* parent, with the difference that the parent is always or never infected. If both parents are uninfected, the probability of their offspring being infected during parental care is zero. If one parent is infected, the probability of any offspring being uninfected is

$$(1-t_p)^{n_p}$$

A complete picture should take into account both the existence of a «teaching» parent (for which t_p is larger) and the fact that one parent may interact with the offspring more than the other (n_p larger). Let us assume no difference between parents. Expression $(1-t_p)^{n_p}$ can then be rewritten as $(1-T_p)$, where T_p is the global probability that an infected parent will infect an offspring during the period of care.

The probabilities of an offspring being uninfected before selection become

$$\begin{aligned} 1-b_0 &= \left[1 - \frac{(1+s)K}{1+sK}\, t\right]^n \\ 1-b_1 &= 1-b_2 = (1-b_0)\,(1-T_p) \\ 1-b_3 &= (1-b_0)\,(1-T_p)^2 \end{aligned} \quad (3)$$

Substituting b_1, b_2 and b_3 given by (3) in (1), we obtain

$$1-K' = (1-b_0)\left[1 - \frac{(1+s)K}{1+sK}\, T_p\right]^2 \quad (4)$$

The interpretation of (4) is straightforward. $1+sK$ is, neglecting one constant, the surviving population, and hence, neglecting another constant, the number of offsprings. $(1+s)KT_p$ is, neglecting the same constants, the number of offsprings infected by the father. The expression in parenthesis is hence the probability of any offspring *not* being infected by one of his or her parents; its square is the proportion that remains uninfected after interaction with both parents. $(1-b_0)$ of this proportion will remain uninfected after social life.

Let $H = 1-K$. The equilibrium condition of (4) is then $H' = 1-K' = H = 1-K$. $K=0$ (all the population is uninfected) is always an equilibrium point. For $K=1$ to be another equilibrium point, it must be true that

$$(1-t)\,(1-T_p) = 0, \quad s > -1$$

That is, $t=1$, or $T_p=1$, or both ($s=-1$ entails the disappearance of the whole populatio). To keep the whole population infected, one of the transmission modes must be complete.

To search for other points of equilibrium, it is advantageous to compare the functions $H=1-K$ and $H'=H'(K)$ given by (4) in the interval (0,1), looking at intersection points. Let us start with two special cases.

A) Incomplete familial transmission alone

Let $t=0$ (or $n=0$). No transmission occurs outside the family. $b_0=0$, b_1, b_2 and b_3 become constant, so that general considerations made by Feldman and Cavalli-Sforza in (Feldman and Cavalli-Sforza 1976) apply here. At most three equilibrium points can exist (*). One of them is $K=0$. (4) reduces to:

$$H' = \left[1 - \frac{(1+s)\ K}{1\ +\ sK}\ T_p\right]^2 \quad (5)$$

The graph of $H=1-K$ is a straight line going from (0,1) to (1,0) (Figure 1), while H' goes from (0,1) to (1,H'(1)). Since $H'(1)=(1-T_p)^2$ is positive as long as $T_p<1$, it is true that, if for $K=0$ the slope of the curve is less than the slope of the straight line, one (and only one) intersection point at $\hat{K}$ must exist between 0 and 1; and, since in the open interval $(0,\hat{K})$ it is true that $H'<H$, $K=0$ is an unstable equilibrium point, while $K=\hat{K}$ is a stable one. This happens if

$$\partial H/\partial K(K=0) = -2(1+s)T_p < -1 \quad (6)$$

that is

$$T_p > T_p^* = 1/2(1+s), \quad \text{or} \quad s > s^* = \frac{1}{2T_p} - 1 \quad (7)$$

If the trait is severely disadvantageous ($s<-1/2$) condition (7) cannot be fulfilled ($T_p^*>1$). For advantageous or

moderately disadvantageous traits ($s>-1/2$), with each value of s is associated a threshold value of T_p, T_p^*, such that if $T_p>T_p^*$ an infection newly introduced in the population diffuses up to an equilibrium $\hat{K}$. Since $\partial H/\partial T_p$ is always negative in the open interval $0<K<1$, $\hat{K}$ increases with T_p and becomes 1 when $T_p=1$ (Figure 1). Conversely, a threshold of s, s^*, is associated with any valid value of T_p , such that for $s>s^*$ a stable equilibrium is reached. $\hat{K}$ increases with s and tends to $(1-T_p)^2$ as s tends to infinity (Figure 2).

If, instead, the slope of the curve at K=0 is higher than −1, it may intercect the straight line at either two points or none. Three cases can be distinguished.

1) $-.5<s<-.25$

In this range, the curve defined by s and $T_p=T_p^*$ is tangential to the straight line at K=0, but its second derivative is negative (*), so that it goes beyond the straight line and then intercets it at a stable equilibrium point. For T_p values slightly less than T_p^*, as $\partial H'/\partial T_p$ is always negative for $0<K<1$, all points of the curve (except for K=0) are slightly raised, and the number of equilibria becomes three: K=0 (stable), $K=\hat{K}_1$ (unstable, and bounding the domain of attraction of the other two) and $K=\hat{K}_2$ (stable). The threshold in T_p necessary for a stable equilibrium is (therefore slightly) less than T_p^*, but associated with a threshold in the initial value of K beyond which the trait disappears from the population instead of spreading (Figure 3).

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10 T=    .00 N=    .00 TP=   1.00 S=    .30
 9 T=    .00 N=    .00 TP=    .90 S=    .30
 8 T=    .00 N=    .00 TP=    .80 S=    .30
 7 T=    .00 N=    .00 TP=    .70 S=    .30
 6 T=    .00 N=    .00 TP=    .60 S=    .30
 5 T=    .00 N=    .00 TP=    .50 S=    .30
 4 T=    .00 N=    .00 TP=    .40 S=    .30
 3 T=    .00 N=    .00 TP=    .30 S=    .30
 2 T=    .00 N=    .00 TP=    .20 S=    .30
 1 T=    .00 N=    .00 TP=    .10 S=    .30
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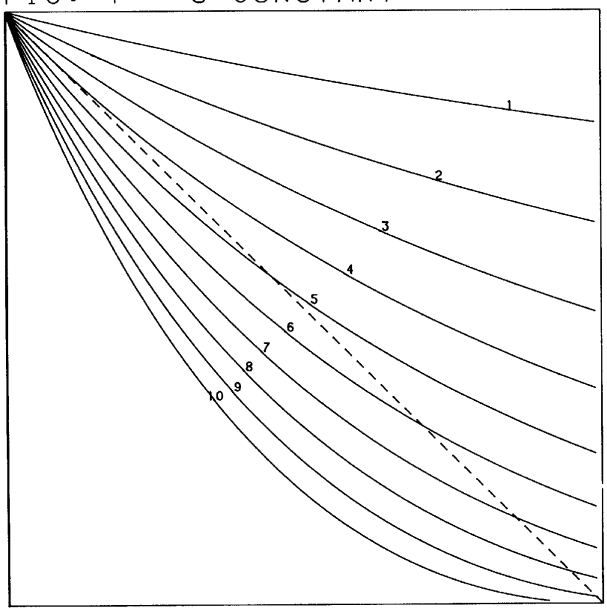
FIG. 1 - S CONSTANT

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10 T=    .00 N=    .00 TP=    .50 S=1000.00
 9 T=    .00 N=    .00 TP=    .50 S= 100.00
 8 T=    .00 N=    .00 TP=    .50 S=  10.00
 7 T=    .00 N=    .00 TP=    .50 S=   1.00
 6 T=    .00 N=    .00 TP=    .50 S=    .50
 5 T=    .00 N=    .00 TP=    .50 S=    .00
 4 T=    .00 N=    .00 TP=    .50 S=   -.30
 3 T=    .00 N=    .00 TP=    .50 S=   -.50
 2 T=    .00 N=    .00 TP=    .50 S=   -.70
 1 T=    .00 N=    .00 TP=    .50 S=   -.90
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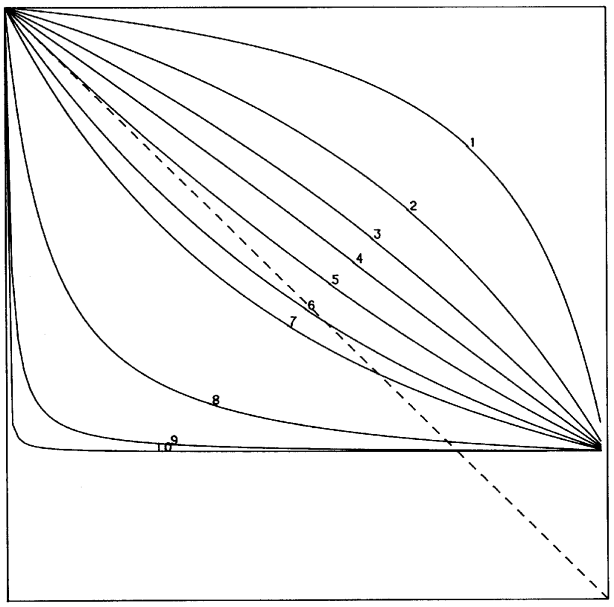
FIG. 2 - TP CONSTANT



2). $s \leq -.5$

For severely disadvantageous traits, K=0 is always a stable equilibrium point. However, if T_p is large enough, H'(K) intercects the straight line in two more points, establishing a three-equilibria situation much the same as that seen before. It has been found numerically that, fr s=−.5, the second stable equilibrium point exists if $T_p>.86$. For s=−.6, it must be true that $T_p>.87$ (Figure 4). For s=−.75 it must be true that $T_p>.92$.

3). $s>-.25$

For advantageous or moderately noxious traits, the curve H'(K) defined by s and $T_p=T_p^*$ runs above the straight line in the whole open interval (0,1) (*). Being in the same inter-

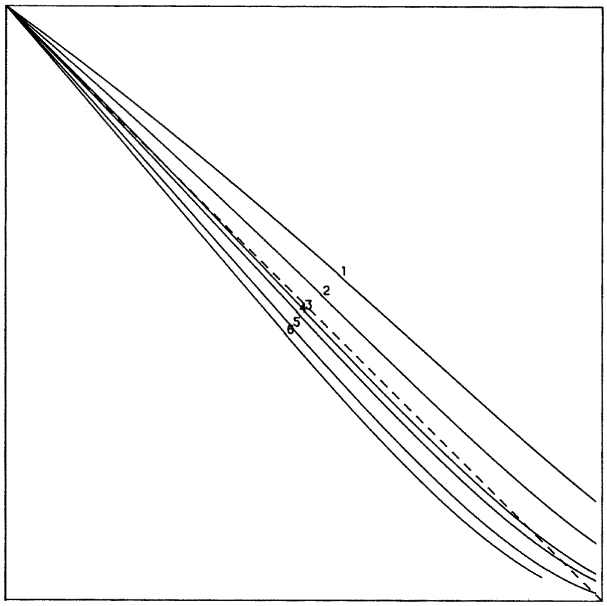
(*) Notice that, for s>-1, the denominator of (4) is nonzero in the close interval (0,1) of K. Multiplying both sides of (4) by its square, we obtain a condition of the form f(K)=0, where f is a cubic polynomial in K. f happens to be 0 if K=0, always non-negative at K=1, and its first derivative at K=0 is negative if (6) holds. To have three equilibrium points in the interval (0,1), f(K) must have three zeroes in the same interval, and this implies that its second derivative must have one in it, that is,

$$6s^2K - 2[s^2 - 2s - (s - T_p - s\,T_p)^2\,] = 0.$$

For $T_p^* = T_{p^*} = 1/2(1+s)$, K>0 implies $s<-1/4$, so that, if $s>-.25$, the flex point of f falls outside the interval (0,1). For $-.5<s<-.25$ and $T_p=T_p^*$, at K=0 the second derivative of f is negative, and, as f(1) is non-negative, a second equilibrium point must exist between 0 and 1 (or at K=1).

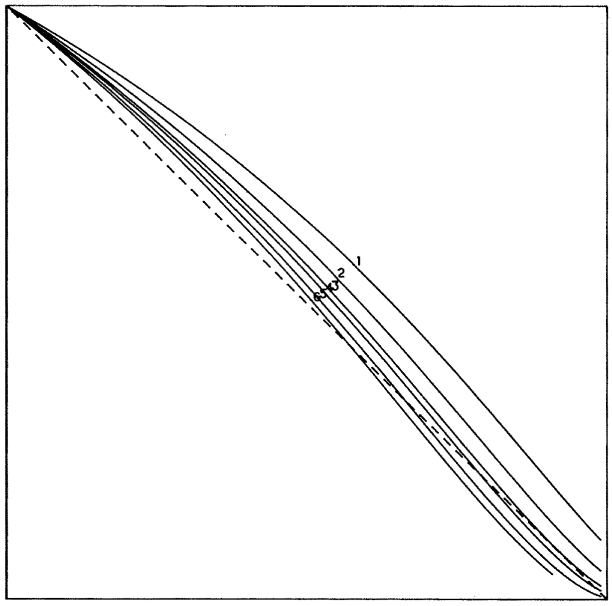
6 T= .00 N= .00 TP= .95 S= -.40
5 T= .00 N= .00 TP= .90 S= -.40
4 T= .00 N= .00 TP= .83 S= -.40
3 T= .00 N= .00 TP= .80 S= -.40
2 T= .00 N= .00 TP= .70 S= -.40
1 T= .00 N= .00 TP= .60 S= -.40

FIG. 3 $-.5 < S < -.25$



6 T= .00 N= .00 TP= .99 S= -.60
5 T= .00 N= .00 TP= .95 S= -.60
4 T= .00 N= .00 TP= .90 S= -.60
3 T= .00 N= .00 TP= .87 S= -.60
2 T= .00 N= .00 TP= .80 S= -.60
1 T= .00 N= .00 TP= .70 S= -.60

FIG. 4 $-S < -.5$



val $\partial H'/\partial T_p$ always negative, if $T_p<T_p^*$ $K=0$ is the only existing (and stable) equilibrium: such a trait diffuses into the population if, and only if, $T_p>T_p^*$ (Figure 1).

A1) Incomplete familial transmission, 1 «teaching» parent

If parental care is given to offsprings by one parent only, the quadratic exponent disappears from (5). The resulting function H'(K) can intersect the straight line $H=1-K$ in two points at most, so that no three-equilibria situation can exist. Condition (6) reduces to

$$-(1+s)\,T_p<-1$$

that is

$$T_p>T_p^*=1/(1+s).$$

In such a situation, no disadvantageous trait can spread into the population. The transmission coefficient T_p needed to diffuse an advantageous trait is double the one needed for diffusion with two «teaching» parents. The equilibrium point $\hat{K}$ is always lower than the equilibrium point that would be reached by halving the transmission coefficient and doubling the number of «teaching» parents.

B) Incomplete oblique transmission alone

Let $T_p=0$. No privileged interaction occurs with regard to this trait between parents and their offsprings; these may be equally infected by (or learning from) any adult of the population. The recursion formula (4) becomes

$$H'=1-K'=\left[1-\frac{(1+s)K}{1+sK}t\right]^n \qquad (8)$$

which simply states that the fraction of uninfected offsprings in the next generation equals the probability of any of them remaining uninfected after n contacts with any adult.

An interesting point to notice is that (8) is obtained under the hypothesis that selection acts on the survival of young adults, so that individuals eliminated by the selective filter cannot act as parents, nor as infecting adults. A different hypothesis may be made: selection acts only on the prolificity of phenotypes. Under this hypothesis, (8) becomes

$$H'=(1-K\,t)^n$$

in which the fitness parameter s has disappeared. In such a situation, the diffusion of a trait is independent of its selective value, and depends only on its chances of transmission.

Turning back to (8), $H'(K=0)=1$, $H'(K=1)=(1-t)^n$. The behavior of this function depends on the value of n. If $n=2$, (8) reduces to (5), where T_p i substituted by t, and all the considerations of pharagraph A) apply. If $n=1$ the considerations of paragraph A1 apply.

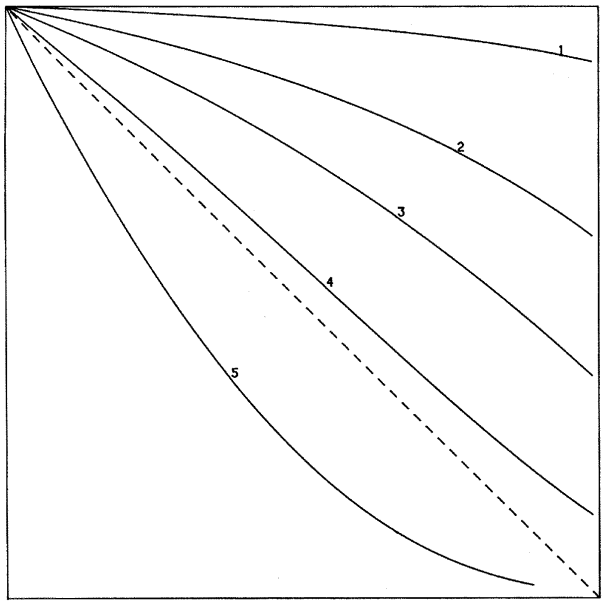
If $n>2$, $K=0$ is an unstable equilibrium point if $\partial H'/\partial K<-1$, that is

$$(1+s)\,n\,t > 1. \qquad (9)$$

As can be seen, oblique transmission allows for the diffu-

5 T= .01 N= 500.00 TP= .00 S= -.60
4 T= .01 N= 200.00 TP= .00 S= -.60
3 T= .01 N= 100.00 TP= .00 S= -.60
2 T= .01 N= 50.00 TP= .00 S= -.60
1 T= .01 N= 10.00 TP= .00 S= -.60

FIG. 5 - OBLIQUAL TRANS ALONE



sion of very disadvantageous traits ($s<-.5$), provided that n and/or t are large enough (Figure 5). The situation is complicated by the fact that, for some values of s and t, n equilibria other than $K=0$ are possible, of which $n/2-1$ are unstable and $n/2$ stable. These precarious equilibria, however, may arise only for disadvanageous traits. If $s>0$, it can be proved that if (9) is *not* satisfied, $K=0$ is the only (stable) equilibrium point; otherwise, $K=0$ is unstable, and only one other stable equilibrium point $\hat{K}$ exists (**).

A more complete analysis, not given here in detail, takes into account the fact that n may be not the same for all offsprings. If the dispersion of the distribution of n is non-zero, this does not affect the condition (9) – where n is understood to be the mean value – but lowers the value of $\hat{K}$.

A particular extreme case of indifferent equilibrium is $n=1$, $t=1$: any initial value of K is preserved, but not restored if modified.

C) Incomplete vertical and oblique transmission

Since in this case H'(K) is the product of the two functions seen in the preceding paragraph, and for $0<K<1$ both are less than 1, the resulting function is always lower than both of them taken separately. Moreover, since both of them assume the value 1 for $K=0$, its initial slope is the sum of the initial slopes:

$$\partial H'/\partial K(K=0)= (1+s)\,(-n\,t-2T_p) \qquad (10)$$

so a stable equilibrium at $\hat{K}$, other than zero, is ensured if

$$P= (1+s)\,(n\,t+2T_p) >1 \qquad (11)$$

$(1+s)\,(2T_p+n\,t)$ can be viewed as the penetrability factor that determines whether a trait can spread into a population where few individuals are initially infected. Let us call this P for later pourposes.

As was to be expected, familial and oblique transmission reinforce each other, and together may combine to establish an equilibrium that neither of them could reach separately. In particular:

If an oblique tranmision alone ensures a stable nonzero equilibrium point, an additional familial transmission increases both the value of $\hat{K}$ and the speed of convergence, and *viceversa.*

If the oblique transmission alone is insufficient, an additional familial transmission can lead to a stable equilibrium, or, if $s>-.25$, to a three-equilibria situation, with T_p less than the threshold required by familial transmission alone. These three-equilibria situations may explain the diffusion of «kuru» disease with no need to postulate complete familial transmission (Feldman and Cavalli-Sforza 1976).

A stable equilibrium point, with $K=0$ unstable (that is, no threshold in K) can be obtained even where $s<-.5$, if a small oblique transmission reinforces the existing familial one (Figure 6).

A similar result, under different assumptions, has been obtained by Uyenoyama, Feldman and Cavalli-Sforza for the contemporaneous action of familial and horizontal (within-generation) transmission (Uyenoyama, Feldman and Cavalli-Sforza 1979).

2) Contemporaneus diffusion of two interacting traits

Let A and B be two traits diffusing at the same time into the same population. Possible phenotypes are, with obvious symbolism, 0, A, B and AB. The analogue of (4)

(**) To prove this, let us write

$H'(K) = f(K)^n$

so that

$$\frac{\partial H'}{\partial K} = n\,f^{n-1}\frac{\partial f}{\partial K} = n\,f^{n-1}\left[-t\,\frac{1+s}{(1+sK)}\right]$$

At $K=0$, $f=1$ so that the condition $\partial H'/\partial K<-1$ becomes

$-n\,t\,(1+s) < -1$

that gives (9).

If $s>0$ and $K=0$ is a stable equilibrium point, a three-equilibria situation implies that an unstable equilibrium exists at $K>0$, in which it should be the case that $n\,t\,(1+s)\,f^{n-1}/(1+sK) > 1$.

Since $n\,t\,(1+s)<1$, $(1+sK) >1$, $f<1$, this is absurd.

If $s>0$, and $K=0$ is unstable, a three-equilibria situation would require $\partial H'/\partial K>-1$ at K(1), and $\partial H'/\partial K<-1$ at $K(2)>K(1)$, that is

$n\,t\,(1+s)f(1)^{n-1}/(1+K(1)s) < 1$ and

$n\,t\,(1+s)f(2)^{n-1}/(1+K(2)s) > 1$

Dividing term by term, $(1+K(1)s)\,f(2)^{n-1}/(1+K(2)s)\,f(1)^{n-1} > 1$.

Since $K(2) > K(1)$, this implies $f(2)>f(1)$: but, as $\partial f/\partial K$ is always negative in the interval (0,1), this is absurd.

```
7 T=   .40 N=   2.00 TP=   .95 S=   -.60
6 T=   .10 N=   2.00 TP=   .95 S=   -.60
5 T=   .10 N=   2.00 TP=   .90 S=   -.60
4 T=   .10 N=   2.00 TP=   .85 S=   -.60
3 T=   .00 N=    .00 TP=   .85 S=   -.60
2 T=   .00 N=    .00 TP=   .80 S=   -.60
1 T=   .00 N=    .00 TP=   .70 S=   -.60
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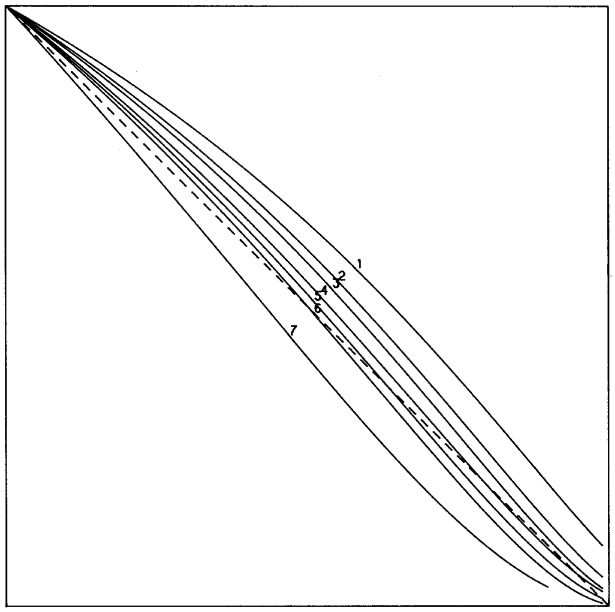
FIG. 6 - S < -.5, ENFORCED



becomes a system of three recursion formulae, into which many additional parameters must enter. The relative viability of phenotype AB may, in fact, differ greatly from the product of relative viability of phenotype A and that of phenotype B: contemporaneous possession of traits A and B can help an individual to survive situations in which neither A, nor B would suffice alone; on the other hand, contemporaneous presence of two advantageous traits could be harmful. Parental and oblique transmission of each trait depend on whether or not the possible transmitter and the candidate receive are infected by the other trait: the transmission probabilities of the simple form (3) cannot be written, since the phenotypic composition of offsprings changes dynamically during education and social life. This makes algebraic treatment of the general case prohibitive.

For these reasons, the analysis of the contemporaneous diffusion of two traits has been conducted heuristically. First, a numerical simulation program was written, to plot the diffusion in time of four phenotypes, knowing diffusion parameters, relative viabilities and initial frequencies: some promising and «reasonable» inputs were tried, varying one parameter at a time along its validity field. Interesting results were obtained with particular parameter values, corresponding to restrictive assumpions on trasmission modes and interaction between traits. These assumptions simplify the recursion system in some cases so as to allow for algebraic analyses, that are presented below. The results of algebraic analyses were then verified using the simulation program.

Case 1. Independent diffusion, synergic effects

Assume that A and B are two traits that diffuse into a population independently: that is, the probabilities of contagion by each of them do not depend on the transmitter and/or the candidate receiver already being infected by the other. Let K_a be the frequency of trait A in the population (total of phenotypes A and AB to the whole), K_b the frequency of trait B, and K_{ab} the fraction of the population infected by both. Let $(1+s_a)$, $(1+s_b)$ and $(1+s_{ab})$ be the relative viability of phenotypes A, B and AB respectively, to the uninfected phenotype.

After selection, the frequencies modify as follows:

$$K_a \to (1+s_a)(K_a - K_{ab}) + (1+s_{ab})K_{ab} = (1+s_a)K_a + (s_{ab} - s_a)K_{ab}$$

$$K_b \to (1+s_b)K_b + (s_{ab} - s_b)K_{ab}$$

$$K_{ab} \to (1+s_{ab})K_{ab}$$

$H = 1 - K_a - K_b + K_{ab}$ remains unchanged. (1.1)

and the whole population becomes, forgetting the absolute viability of the uninfected phenotype,

$$1 + s_a K_a + s_b K_b + (2s_{ab} - s_a - s_b)K_{ab}.$$

Posing

$$f_a = \frac{(1+s_a)K_a + (s_{ab} - s_a)K_{ab}}{1 + s_a K_a + s_b K_b + (2s_{ab} - s_a - s_b)K_{ab}} \quad (1.2)$$

The following recursion formula can be written:

$$1 - K'_a = (1 - f_a T_{pa})^2 (1 - f_a t_a)^{n_a} \quad (1.3)$$

where T_{pa}, t_a, n_a are the transmission parameters T_p, t and n for trait A. A symmetric recursion formula holds for trait B.

If A is introduced into the population, it can diffuse only if

$$\frac{\partial(1-K'_a)}{\partial K_a} < -1 \quad \text{at } K_a = 0.$$

That is, since for $K_a = 0$ f_a is also equal to 0,

$$P_a\left[1 + \frac{(s_{ab} - s_a)}{1 + s_a}\frac{\partial K_{ab}}{\partial K_a}\right]/(1 + s_b K_b) > 1 \quad (1.4)$$

where $P_a = (2T_{pa} + n_a \cdot t_a)(1+s_a)$ is the penetrability factor of trait A alone. The remainder of the left side of (1.4) can be seen as an accelerating factor of penetration (if >1) or a retarding factor (if <1). The symmetry of (1.4) holds for B.

The term $\partial K_{ab}/\partial K_a$ needs additional assumptions if it is to be evaluated. A particularly simple situation arises if one of the two traits (say B) transmits only obliquely: in this case, it is $K_{ab} = K_a K_b$ from the second generation on, and (1.4) becomes

$$P_a\left[1 + \frac{(s_{ab} - s_a)}{1 + s_a} K_b\right]/(1 + s_b K_b) > 1 \quad (1.5)$$

The multiplying factor of P_a acts as an accelerating factor if

$$\left(\frac{s_{ab}-s_a}{1+s_a}-s_b\right)K_b>0 \qquad (1.6)$$

that is

$K_b>0$ *and*

$$(1+s_{ab})>(1+s_a)(1+s_b) \qquad (1.7)$$

If $(1+s_{ab})=(1+s_a)(1+s_b)$, the diffusion of A is completely independent of the presence of B, and *viceversa.* This may happen if the selective process involving trait A is quite separated from that involving trait B. In such a situation, the diffusion of A and B simply overlap. This remains true even if both A and B are transmitted vertically.

Suppose $P_a>1$. A alone can diffuse into the population up to an equilibrium value $\hat{K}_a$. If (1.7) holds, its diffusion is accelerated (and $\hat{K}_a$ raised) by the presence of trait B. If (1.7) is false, there exists a threshold K_b^* (not necessarily <1) such that, if $K_b<K_b^*$, (1.5) holds and A can diffuse, but $\hat{K}_a$ is lowered; if $K_b>K_b^*$, A cannot diffuse.

On the contrary, if $P_a<1$, A cannot diffuse; if (1.7) does not hold, A cannot diffuse *a fortiori.* If (1.7) is true, there exists a threshold K_b^* (not necessarily <1) such that, if $K_b>K_b$, (1.5) becomes true and A can diffuse.

If both P_a and P_b are less than 1, $K_a=0$ and $K_b=0$ is a stable equilibrium point; if both traits are contemporaneoulsy introduced into the population in small quantities, they cannot diffuse. But, if the initial frequencies K_a and K_b are large enough and (1.7) is true, the presence of each trait may allow the other to maintain its level of diffusion. Conversely, if both P_a and P_b are greater than 1, but (1.7) is false, the eventual equilibrium may comprise both traits, at frequencies lower than $\hat{K}_a$ and $\hat{K}_b$, but it may happen that one of them increases to expel the other from the population.

Case 2. Trait A is a prerequisite to receive trait B

In this case, only individuals already infected by trait A may be infected by trait B. The only possible phenotypes are thus 0, A and AB. The symbolism is the same used in the previous case, but, since s_b has here no meaning, it is legitimate to put

$(1+s_b)=(1+s_{ab})/(1+s_a)$.

The algebra is greatly simplified if we assume that susceptibilities to traits A and B are limited to distinct and subsequent ages. After selection, frequencies of traits are modified:

$$K_a \to (1+s_a)(K_a-K_{ab})+(1+s_{ab})K_{ab}=(1+s_a)(K_a+s_bK_{ab})$$

$$K_{ab} \to (1+s_a)(1+s_b)K_{ab}$$

$H=1-K_a$ remains unchanged

and the whole population becomes, neglecting the absolute viability of the uninfected phenotype,

$1+s_aK_a+(1+s_a)s_bK_{ab}$.

Putting

$$f_a=\frac{(1+s_a)(K_a+s_bK_{ab})}{1+s_aK_a+(1+s_a)s_bK_{ab}}$$

and

$$f_b=\frac{(1+s_a)(1+s_bK_{ab})}{1+s_aK_a+(1+s_a)s_bK_{ab}}$$

the following recursion formulae can be written:

$$1-K'_a=(1-f_aT_{pa})^2(1-f_at_a)^{n_a} \qquad (2.1)$$

$$\frac{K'_a-K'_{ab}}{K'_a}=(1-f_bT_{pb})^2(1-f_bt_b)^{n_b} \qquad (2.2)$$

If $s_b=0$, or $K_{ab}=0$, (2.1) reduces to (4), and A diffuses independently of the presence of B. For $K_{ab}>0$, if $s_b>0$ the diffusion of A is accelerated; if $s_b<0$, it is retarded, and the corresponding equilibrium value $\hat{K}_a$ raised or lowered respectively.

The diffusion of B is restricted to offspring already infected by A, but transmission probabilities depend on the diffusion of B in the whole population. The diffusion of B is thus always slower than the diffusion of a trait with same penetration factors that need no prerequisite, as the diffusion of B would be in a population totally infected by A.

To study the dynamics of diffusion processes, let us suppose K_a varies slowly (e.g., because it is near equilibrium). Trait B, if introduced into the population, diffuses only if

$$\frac{\partial}{\partial K_{ab}}\left(\frac{K_a-K'_{ab}}{K_a}\right)<-\frac{1}{K_a} \quad \text{at } K_{ab}=0$$

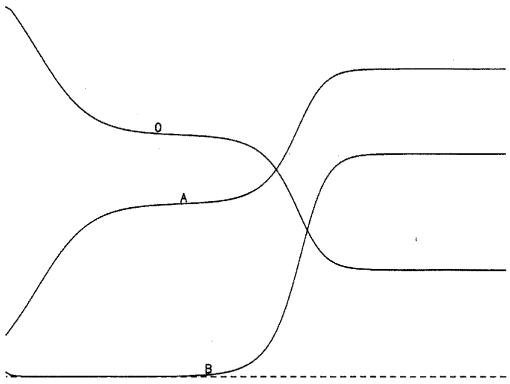
that is,

$$(2T_{pb}+n_bt_b)(1+s_b)\frac{(1+s_a)K_a}{1+s_aK_a}>1 \qquad (2.3)$$

This implies $P_b>1$. Since the remainder of the left-hand side of (2.3) is zero for $K_a=0$ and 1 for $K_a=1$, there exists a threshold K_a^* in the open interval (0,1) such that, for $K_a>K_a^*$, (2.3) holds and B can diffuse; for $K_a<K_a^*$, K_{ab} decreases. If the equilibrium frequency of A, $\hat{K}_a$, is less than K_a^*, B can never enter. If not, an interesting evolution can be observed simulating numerically diffusions of two traits linked by this relationship. While K_a increases toward $\hat{K}_a$, K_{ab} initially decreases, but, if B is not expelled (that may happen in a finite population) when K_a goes over the critical value K_{ab} begins to increase. A stable equilibrium is reached if $s_b>0$ (Figure 7). If $s_b<0$, the new equilibrium value of K_a, lowered by the presence of B, may go beyond

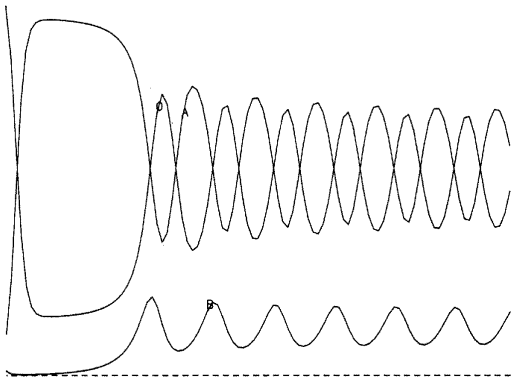
B T= .150 N= 10 TP= .000 S=1.000 KINIT= .01000000000
A T= .000 N= 10 TP= .500 S= .200 KINIT= .10000000000

FIG. 7. B NEEDS A



B T= .900 N= 10 TP= .000 S=-.850 KINIT= .01000000000
A T= .000 N= 10 TP= .500 S=2.000 KINIT= .10000000000

FIG. 8. B NEEDS A



the critical value of K_a needed for K_{ab} to increase. The result is the oscillation of both K_a and K_{ab} in time, that may or may not damp to a stable equilibrium (Figure 8). As K_{ab} is always less than K_a, K_a never zeroes: but statistical fluctuations may expel A, and hence B, from a finite population.

Case 3. Incompatibility between traits

Let us consider two traits incompatible A and B: that is to say, that an individual infected by one of them is immune from the other. Possible phenotypes are thus 0, A and B, with relative viabilities 1, $(1+s_a)$, $(1+s_b)$. Let us suppose again that susceptibilities to traits A and B are limited to distinct and subsequent ages. After selection, the relative frequencies of phenotypes are modified simply:

$K_a -> (1 + s_a)K_a$
$K_b -> (1 + s_b) K_b$
$H = 1 - K_a - K_b$ remains unchanged

and the whole population becomes, neglecting the absolute viability of the uninfected phenotype,

$$1 + s_a K_a + s_b K_b.$$

Posing

$$f_a = \frac{(1+s_a) K_a}{1 + s_a K_a + s_b K_b}$$

and

$$f_b = \frac{(1+s_b) K_b}{1 + s_a K_a + s_b K_b}$$

we can write

$$1 - K'_a = (1 - f_a T_{pa})^2 (1 - f_a t_a)^{na} \tag{3.1}$$

$$\frac{1 - K'_a - K'_b}{1 - K'_a} = (1 - f_b T_{pb})^2 (1 - f_b t_b)^{nb} \tag{3.2}$$

The situation is, in a sense, the reverse of the previous one. If $s_b=0$, or $K_b=0$, (3.1) reduces to (4), and A diffuses independently of the presence of B. For $K_b>0$, if $s_b>0$ the diffusion of A is retarded, and its equilibrium point lowered; the reverse is true if $s_b<0$. In this case, even a trait A that, if alone, cannot diffuse, may spread into a population in which B is already present. Equation (3.2) reduces to (4) only if $K_a=0$; the presence of A always has some influence on the diffusion of B.

Suppose that A alone is at equilibrium, with frequency $\hat{K}_a$. Trait B, if newly introduced into the population, could diffuse if

$$\frac{\partial}{\partial K_b}\left(\frac{1 - \hat{K}_a - K'_{ab}}{1 - \hat{K}_a}\right) < -\frac{1}{1 - \hat{K}_a}$$

that is

$$P_b(1-\hat{K}_a)/(1 + s_a \hat{K}_a) > 1 \tag{3.3}$$

On the contrary, the conditions for A to diffuse into a population in which B is nearly at equilibrium is

$$P_a/(1 + s_b \hat{K}_b) > 1 \tag{3.4}$$

As can be seen, condition (3.4) is less restrictive than (3.3): new traits diffuse more easily if transmitted earlier than their competitors.

Suppose both P_a and P_b to be greater than 1. In this case, both traits can diffuse in an uninfected population.

If both (3.3) and (3.4) hold, each trait can also diffuse in a population already infected by the other. Any combination of initial frequencies leads eventually to a stable equilibrium in which A and B coexist, with phenotype frequencies lower than $\hat{K}_a$ and $\hat{K}_b$ respectively (Figure 9).

If one of them, say (3.4) is false (this implies $s_b>0$), as $\partial K'_a/\partial K_a$ is positive for $K_b=0$ and negative for $K_b=\hat{K}_b$, there exists a value K^*_b such that, for $K_b>K^*_b$, $\partial K'_a/\partial K_a$ is always negative. Introducing A at $K_b>K^*_b$, K_b continues to increase up to $\hat{K}_b$ and K_a decreases to zero.

Introducing A at $K_b<K^*_b$, K_a begins to increase, and the presence of A influences the diffusion of B. What happens then depends on (3.3). If (3.3) is true, K_b continues to in-

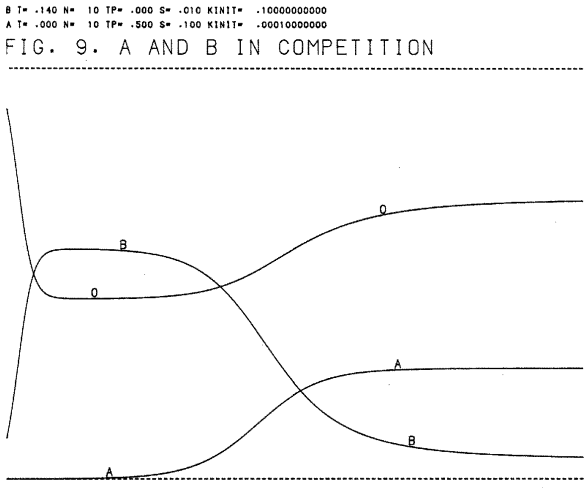


FIG. 9. A AND B IN COMPETITION

crease anyway, and the eventual equilibrium may be either the expulsion of A from the population, or a coexistence of A and B which is stable but precarious for A. It is precarious because, if K_a falls by fluctuation under a critical value, A is expelled from the population. If (3.3) is false, there are three possible outcomes: A is expelled, B is expelled, A and B coexist, stablely but precariously for both.

An interesting case arises if $P_a<1$ but (3.4) holds: this is possible if $s_b<0$. A cannot infect an uninfected population, but can infect a population already infected by B if K_b is greater than a given threshold K^*_b ($<\hat{K}_b$). However, as K_a increases in time, K_b must decrease, and, if K_b falls under K^*_b before a new equilibrium is established, K_a begins to decrease in turn, allowing K_b to increase. Again, we find an oscillatory behavior of both K_a and K_b, with or without damping to equilibrium. If the amplitude of oscillation of K_b is high enough, in a finite population K_b may reduce to zero, thus making A also disappear (Figure 10).

The case in which susceptibilities of offsprings to A and B is not age-dependent was simulated numerically. The general picture does not change. Conditions (3.3) and (3.4)

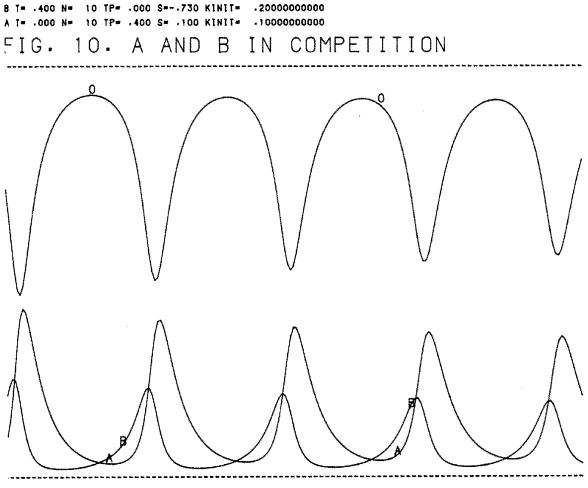


FIG. 10. A AND B IN COMPETITION

reduces to two intermediate symmetric conditions which are difficult to compute algebraically: say

$$P_a \frac{(1-\hat{K}_b)^{\varkappa}}{1+s_b K_b} > 1$$

where x is some number in the range (0,1). Figure 11 is the result of a simulation involving 4 differente incompatible traits with increasing penetrabilities and decreasing initial frequencies. A lower initial frequency gives, in an infinite population, the same effect of a retarded introduction. As can be seen, each trait substitutes the previous one in sequence.

Discussion

The model proposed in the first part seems appropriate to explain the diffusion of epidemics, endemic diseases ($s<0$) and of symbiotic flora ($s>0$) into a population. When applied to cultural evolution, some remarks must be made.

The basic assumption is that the transmission process of a cultural trait (skill, custom, behavior, belief etc.) is much the same as that of a parasite or of a symbiont. This model assumes that only one trait is diffusing into a population over a period of time covering many generations. This holds true if the mean time between innovations is long compared to the lifetime of the host animal, as happens today for some species (e.g., titmice or macaques) into which new skills can diffuse only by imitation. In this hypothesis, it has been proved (Feldman and Cavalli-Sforza 1976, Uyenoyama et alt. 1979) that if the probability of learning is determined by the individual's genotype, selection on a cultural character can cause changes in gene frequencies. That is, a selective advantage given by a skill (e.g. the ability to tie knots) increases the genetic aptitude to learn it (e.g. manual ability) in the population.

Feldman and Cavalli-Sforza demonstrated also (Feldman and Cavalli-Sforza 1976) that gene frequencies can change without genetic difference in learning ability; when an advantageous skill is completely transmited only vertically, the genotype of individuals initially skilled will eventually dominate in the population. This remains intuitively true if a skill (or a complex of skills) is transmitted only to the offspring of members of a group, without interchange between groups.

Such a situation was probably in the mind of those authors who treated cultural evolution, speaking of the struggle for life and competition between *cultures*. In 1947, Gordon Childe (Childe 1947) approached this problem from an antropological point of view, studying mediterranean civilisations, and concluded that such an assumption in untenable, because of the high rate of diffusion of traits between different cultures.

This does not mean that such a skill-based genotype selection was not active at earlier stages of human evolution, when communication between groups was poor and exogamy (a surely advantageous cultural trait) was not yet adopted by most of them.

In history, however, innovation rate has always been far

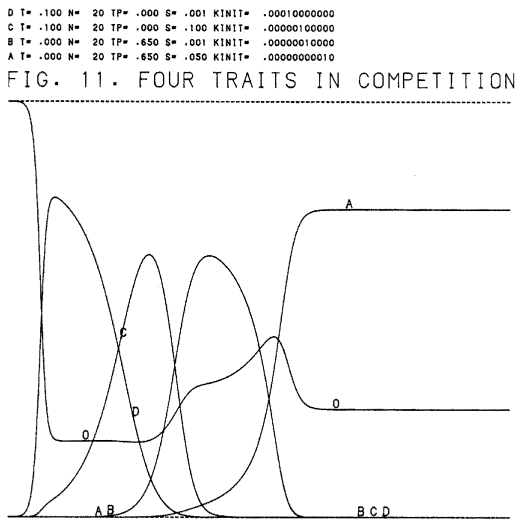


FIG. 11. FOUR TRAITS IN COMPETITION

greater than one innovation per generation, so that at any time a number of cultural traits has always been diffusing into any human society in all directions (including the ascending vertical one).

Rapid propagation of innovations requires mostly horizontal and oblique transmission, and in such cases, as seen in the first part, the high penetrability of a trait may promote its diffusion even if it is severely disadvantageous. If only (or mostly) advantageous traits spread into the whole population, a previous (genetic and/or cultural) selection must have affected the receivers' susceptibility. This selective learning may fail for behaviors whose long-term effects are difficult to forecast.

It has been shown in the secon part that when different traits diffuses contemporaneously, interactions between traits play a dominant role in determining if one of them can or cannot spread into a population.

Two different traits may interact with each other in many ways. If, for instance, they dictate two different

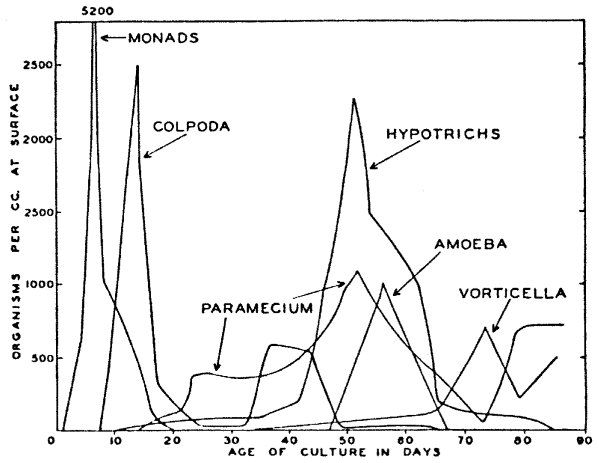


Fig. 12. Succession in a protozoan culture with dominance by successive species. (After Woodruff, 1912). (Reproduced from Odum, 1965).

behaviors in the same situation, they are mutually exclusive. If any individual that has learned one of them becomes refractory to the other (examples spring immediately to mind), case 3 applies. If a mutual substitution is possible during education, the most invasive trait can diffuse more quickly at the expenses of the other.

Let us compare Figures 11 and 12. The latter is the result of a classic experiment by Woodruff (Odum 1965). The succession of protozoan populations with successive dominants in a culture medium made by boiling hay is shown. The similarity between these figures is not a coincidence. If two analogous measurable quantities behave over the time in the same way, this implies a similarity between the underlying laws that govern their evolution. This enables us to say that the relation between the traits of Figure 11 is the same as that between the protozoa of Figure 12: they are in competition for the same ecological niche.

In our culture, competition between ethical values is known to be very high. Competition between skills is usually lower, for different skills help normally to solve different sets of problems. Their niches may overlap, but seldom coincide.

Competition between scientific theories has been treated by Popper (Popper 1934). In his opinion, if a theory is «falsified», it must be substituted by another. In fact, when a theory fails, its field of application is dramatically reduced, and a competitive one can take its place even though it may be more difficult to understand and apply. However, if the difference between learning (transmitting) difficulties is large enough, and/or the old theory allows for predictions reasonably correct which are easier to obtain in some frequently met situations, it may continue to survive within a reduced niche (Figure 9).

In case 2, the presence of one trait in the receiver is the prerequisite for the transmission of another trait. In our culture, this is the case with elementary knowledge that is required for the efficient learning of higher and more useful skills. One of the results of that algebraic analysis has an immediate interpretation: high technology cannot be maintained in a population in which the diffusion of the basic knowledge needed is below a critical value, if it is not continuously reintroduced.

A similar situation is found in nature when one species follows another in the trophic chain. If the relationship between them is predatory (or parasitical), too massive a presence of predators reduce the size of prey population down below the critical threshold. The cyclic density variation of both species, often observed in nature, is the analogue of the oscillations encountered in case 2. The difference is that in the latter case, the second trait destroys resources needed by the first one instead of acting directly on its diffusion (Figure 8).

The oscillations found in case 3 are of a different kind. In this case, two vicariant species are in competition. The more succesful of them, over a given density, destroys its resources and starts to decline, creating gaps that give way to the other (Figure 10).

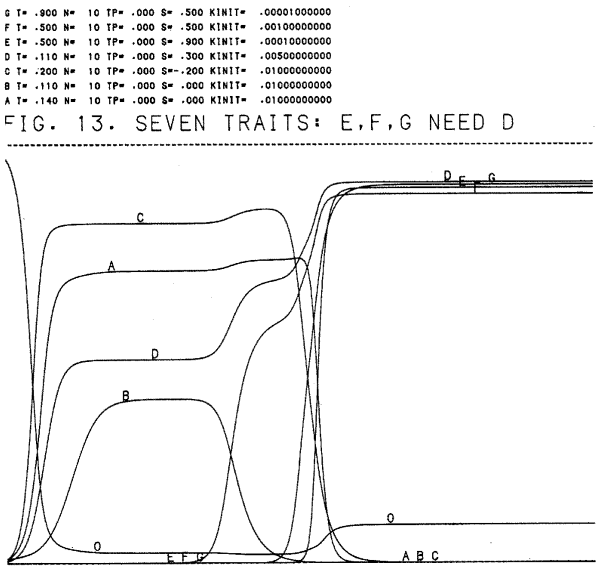


FIG. 13. SEVEN TRAITS: E,F,G NEED D

Figure 13 represents the simulated evolution of 7 traits. A, B and C are in competition with E, F and G respectively, but the latter three require the presence of D to be trasmitted. Cases like this one are well known in ecological successions (Odum 1965). Traits A, B, C and D may be seen as pioneer species that colonize a new territory. The presence of D changes the environment, so successive species can take root and replace some or all of the pioneer ones. The left-hand side of the figure represents a first seral stage. The final self-perpetuating equilibrium state is known as the climax.

The transmission probability of many different traits can be highly influenced by the presence of other traits. What to learn, how to learn, from whom, and communication between individuals are (partly) cultural traits themselves, and act as selective channels or vehicles of diffusion for important skills and customs. Case 2, with s_a zero or slightly negative,can be viewed as an extreme case in which a communication mode A, culturally transmitted, is needed to learn a skill B. In such a complex situation, the fitness s of a single trait is hard even to define. A trait may be of no importance, or be moderately noxious by itself, but symbiont with or commensal of an higly useful skill. Some skills (e.g., medical competence) may be advantageous even if they are not carried by their beneficiary, provided that they are diffused enough in his social group. Figuratively speaking, the relation between the viability of a human being and the culture he and his social group carry is much the same as that between the stability of a substrait and the biotic community living on it and in the neighbourhood in a general environment. The study of case 1 is only a first attempt to find which way the diffusion of two traits is influenced by their combined action in consolidating or making barren their humus.

Human social groups are seldom insulated. New traits can be introduced into them from other groups (the classical «diffusion» of antropology). It may be that some traits cannot spread because they cannot compete with some already existing traits, or because the environment in which *the group* is living makes them unnecessary or inadequate. It makes sense to speak of many climatic cultural climaxes, varying from place to place.

A climatic climax may be modified by new traits. I.e., innovations. The analogue of the evolution of a whole culture is, in this conceptual framework, the historical evolution of a climatic climax. Under the proposed analogy, the mathematical instruments for a quantitative analysis of the evolution and diffusion of cultural traits are already available, and are those of deductive ecology. Some of the descriptive knowledge of the ecological interaction between different traits necessary for such an approach can be drawn from existing studies in sociology and antropology, if they are looked at from this point of view.

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