CHAPTER 5
EVOLUTIONARY IMPLICATIONS

Science, particularly systems science, has been dominated for three decades by an emphasis on the common thermodynamic properties of diverse systems. This nearly exclusive viewpoint has been particularly pervasive in biology, ecology, and socio-economics. Certainly, in some cases the mathematical properties of these diverse systems are identical to that of thermodynamics. This has been particularly brilliantly demonstrated by Kimura (1964) for the mathematics of population genetics, and by Demetrius (1974) or by Keyfitz (1968) for the mathematics of demography of age-dependent fertility and mortality schedules.

None the less, certain problems eluded treatment as thermodynamic systems. Especially important was the description of cultural systems, and their relationship to demography and historico-demographic process. Thus, the successful treatment of this subject by Ballonoff (1982, 1982b, 1983) raises serious questions on scientific direction. The noted work is outside of the thermodynamic tradition in at least three ways: (1) it does not depend on thermodynamical or statistical-mechanical formulations and cannot be derived from them; (2) it does not depend on "statistical sampling" research designs based on "probability methods" or other "research methods" so religiously taught in graduate schools, and indeed implies these are fundamentally wrong for cultural theory and applications; (3) it particularly <u>does</u> depend on recognizing the uniqueness of the individuals. (These three points essentially just restate the Stirling Number result discussed in earlier chapters). My point in particular is not that thermodynamics is "wrong", "irrelevant to" or even contradicted by minimal structure theory. Rather, that the theory is simply something quite different from and not encompassed by thermodynamics, and certainly not by thermodynamics alone.

The above is not an isolated example of behavioral research which does not depend on or follow from thermodynamic models. Understanding the behavioral biochemistry of ecosystems depends upon knowing how the structural chemistry of plant products interacts with various predators. When such interaction affects evolution of predator species, this could be an example of a thermodynamic result, with a non-thermodynamic cause. Other useful examples of ecological biochemistry have been detailed by Harborne (1977).

The purpose of the next section is thus in part to demonstrate another realm in which unique structural properties (studiable by physical laws) determine individual behavior and potentially even certain cultural properties. The subject is the biochemical foundation of allergy and anaphylaxis. Relying on the work of medical researchers plus supporting evidence from medical and non-medical principles, I will note that: (A) there are identifiable common physical biochemical properties of these conditions; (B) there are identifiable features of these properties which

enable clinical treatment of various conditions by similar procedures; (C) none the less these same procedures imply (and result from) the biochemical uniqueness of individuals, demonstrable clinically and implied theoretically. These results tie to certain problems of biological evolution, and of the relation of certain evolutionary mechanisms to possibility of cultural capacities. Finally the chapter relates possible forms of cultural biological evolutionary mechanisms, to the forms predicted by minimal structure theory.

A Medical Example

The essential mechanism for allergy and anaphylaxis was detailed by Godlowski (1962). The principles he discussed follow from general principles of pharmacology; of enzyme chemistry; of the stereochemistry and biochemistry of cholinesterase (and acetylcholine), peptides and terpines; and from properties and effects of fluorides and other acids on bio- and stereochemistry. These mechanisms turn out to be basic to many "ideopathic" syndromes including Tourette and related psycho-neurotic seeming disease, possibly autism, and others; parallel mechanisms in diabetis were implied by Godlowski (1963).

The purpose here is not a medical history, but rather to demonstrate certain points about medical knowledge and practice. My basic reference to the nature and mechanism of allergy and anaphylaxis is to Sherwood (1951). This somewhat aged work is useful since it was a standard reference, and embodies what may be termed a classical and still largely prevalent view.(1) The generally accepted properties of allergic reactions, which they share with anaphylatic reactions, are the following: each is tissue specific (that is, effects of stimulation by an antigen are specific to a single tissue or group of tissues); "dosage" of the antigen appears related to effect; changes in dose, particularly in studies of anaphylaxis, affect degree of reaction in the animal; "sensitization" of an animal to second and following doses occurs; reactions are specific to particular animals within a species (and generally differ between species).

A long and not too successful history of attempting to explain clinical and experimental allergic effects by operation of the immunological system can be documented. It seemed to most authors that some form of antigen/antibody reactions was involved, but the exact nature of this was not easily specified. Discussion on the relation of allergic mechanism to the chemistries of histamine and acetylcholine particularly pervades the literature.(2)

The contribution of Godlowski was to recognize that the underlying mechanism is application of the biochemistry of enzyme/substrate reactions, in connection with understanding configurational chemistry. This seems so simple and clearminded that one is left wondering why the mechanism should even have proven mysterious to other

authors. Certainly the basics of enzyme action were known to medicine prior to the 1962 publication date of Godlowski's work.[3]

Summarizing the widely known properties of enzyme reactions[4] these are: reaction begins when the enzyme and its substrate (the specific chemical with which the enzyme interacts) are added together; this reaction generally begins immediately and continues at a constant rate; as the substrate is used up, products of the reactions may accumulate, and reverse the reaction; all such reactions are highly sensitive to the acidity and temperature of the environment. Photosensitivity[5] of reactions is also known. Indeed, once it is understood as something affecting the energetic background, in a similar manner to changes in acidity (changes in proton/electron balance), photosensitivity should not be surprising.

Principles of pharmacology may be easily seen as extensions of the basic knowledge of enzyme chemistry.[6] The further detail needed to extend basic enzyme chemistry into general pharmocology is to recognize that specific drug (chemical) action on cells depends upon the spatial configuration (stereochemistry) of specific compounds or their close relatives (including isomers, and others with strongly related structures). Examples abound of two phenomena: chemicals with generally dissimilar structures except for particular subgroups and/or spatial configurations of groups of molecules, which affect similar mechanisms in similar ways; and, very structurally similar chemicals whose grossly (or even slightly) different actions can be related to their highly specific differences in structure.[7] Thus, specific individuals can have highly ideopathic, unique, reactions, with very similar chemical mechanisms as their cause.

Godlowski recognized that knowledge of enzyme chemistry is sufficient to understand both the general mechanisms of allergy and anaphylaxis (to with subject Vol. I. of his work is dedicated) and specific applications in diagnosis and treatment of diverse metabolic disorders (to which subject his Vol. II is dedicated). In a normal person, protein substances entering the body are broken down into their constituent protein parts by enzymes of the body, in a process known as proteolysis. (The specific version of this studied by immunologists is often referred to as "antigen-antibody reactions"). In a normal reaction, the result of proteolysis is creation of small constituents which may be either consumed or excreted by the body; the products themselves are seldom harmful except in unusual circumstances (such as following inactivation of some organ).

In simplified form, what happens in an allergic person is incomplete proteolysis. For any of a number of chemical (stereochemical, isomeric, or genetically induced) causes, some specific enzyme/substrate reaction does not run to its normal completion. Instead, the action is to create some chemical by-product other than the usual; in general, this "unintended" by-product will not be constructive to the

organism. In may in fact be specifically harmful and in extreme cases toxic even in small doses.

The steps from this knowledge to understanding allergic and anaphylactic behavior is simple. The specificity of allergic reactions has already been noted.[8] Since each tissue has in fact its own particular chemistry, the observation of tissue specificity of allergic reactions is not surprising. The physiological effects of the chemistry that results from the products of the incomplete proteolysis, with "normal" body chemistry, is what is described as the allergic or anaphylactic reaction. Though generally described clinically by means of effect on gross behavior or physiology of an individual, the chemical foundation is well described by Godlowski. Thus in specific individuals there are specific (to that individual) reactions as a result of exposure of the individual to a specific chemical (the intruding antigen). Various medical researchers have demonstrated that allergic reactions may be described specifically for a great variety of organs.[9]

Because of the specificity of chemical reactions, the incomplete proteolysis of some specific antigen (including as a possible antigen, natural chemical products of the body itself -- e.g., self-allergy to certain hormones) may have specific effects at any possible site in the body of the individual, but will have consistent effects within any one individual. The commonality of all allergy patients is the general mechanics of chemistry, not a specific chemical agent; thus attempts to "explain" all allergy by some specific agent cannot be successful.

The two most commonly cited "causes" or predicted causes of allergic biochemistry, histamine and acetylcholine, are therefore not universal "causes". Instead, both are chemicals either relatively easily identified in their site of action or important in their physiological effect. Thus, if the allergy or anaphylaxis is defined in terms of its physiological effect, and for example gross neurological effects are taken as the defining conditions, then disorders of the cholinesterase mechanisms will almost certainly be implicated.[10] None the less, this is but an example of the essential mechanism outlined by Godlowski (1962). A similar explanation holds for histamine. Being structurally "almost" a terpene, histamine would be expected to be chemically related to the proteolysis of many plant generated compounds, in particular the terpenes. Since terpenes are produced, by and large, as irritating products as part of the warfare of plants with their various predators (particularly insects, but not exclusively so)[11], it is not in the least surprising that errant histamine chemistry is implicated in many of the common pollen-related allergies and folk medicines including off-the-shelf commercial allergy drugs. (Note that while the specific effects of any incomplete proteolysis will be unique to any given individual, certain properties are common to all individuals. It is these common properties which make possible the forms of allergic patient treatment described in Godlowski (1965). But it is the lack of chemical specificity between patients that means folk remedies do not work universally and in fact often carry label warnings

for various "side effects", which effects do not occur under a Godlowski type treatment.)

A further generalization of the mechanism is useful. The body system implicated in metabolic disorders of allergy is the endocrine system: the endocrine system in particular regulates the stability of body chemistry by production of various hormones.(12) This would be true even for allergic disorders that affect the chemistry of the brain. It is thus the endocrine system which deals with the bodily insults from the chemical environment (including both the natural and man-made environment).

These facts lead to totally different conclusions on the rise of human intelligence, perhaps even human cultural/ecological co-adaptation, than that provided by Lumsden and Wilson (1981), the "socio-biologists". Lumsden and Wilson hypothesized that culture arises coincident with cognitive ability, as a specific genetic adaptation to specific environmental circumstances. Therefore, they conclude culture as such (or, abilities successful within a given culture) are selected for, coincident with rise in intelligence and brain capacity.

The reality implied by an understanding of allergy is totally different. The one environmental fact shared by all humans in this planetary environment is exposure to the forces of natural evolution. These "forces" most particularly include the actions of the plant world, to defend itself in a continually adapting fight with its predators. This results in a continually changing and progressively more challenging exposure to plant produced products, each of which is a potential pathological allergen to some portion of any animal population. All of these plant products are exposed to and potentially affect <u>every</u> member of <u>all</u> local animal populations. Add to this the relatively slower geological changes in ecological biochemistry, and the much faster human-created changes in local physical chemistries. Each of these sources produces potential irritants exposed to all members of each population; within each individual they all affect the endocrine-regulated balances of many body chemistries simultaneous. The insult to the normal body must be incredible; an omnivore in particular could be exposed to a possibly devastating variety of partially disabling and continually changing irritants, should an allergic reaction to any one of them occur.

<u>Thus, the rise of one of the most telling aspects of culture, self awareness, may well be ascribed to a very prevalent and highly non-mystical cause: survival of the individual in the face of continually-threatened breakdown of body defenses normally controlled by the endocrine system</u>. The neurological system grows in importance, once its ability to reason in any abstract form at all becomes established. The greater this ability to reason and act independently of the actions and particularly <u>failures</u> of the endocrine system, the greater the survival capability of the individual.(13) This mechanism for selection of greater neurological abilities is

universal to all humans, indeed all animals. Since cognitive abilities are associated with abilities to act within symbol systems (i.e., particular cultures) the inherently biological process of natural survival in the face of biological risk, has the by-product of increased ability to deal in the abstract systems known as cultures, by selecting for development of an organ which makes this possible. This explanation is quite different from that of Lumsden and Wilson (1981).

With an additional fact, the explanation above also shows why certain species may evolve more individual intelligence than others. One major difference between mammals and insects is that insect cognitive and social behavior is more controlled by chemical action outside of the body of an individual, in particular by pherormones, than is mammalian behavior. In particular, the mammalian body must deal with the by-products of errant allergic chemistry of all forms, in particular with those that affect its cognitive system: the body is after all an enclosed system, the animal can not escape its own chemistry. Further, the reproduction of each animal can affect the biologic survival of the entire population. When however much cognitive or social behavior is controlled by external chemistry subject, in particular, to air flow (as are pherormones subject to vagaries of the movement of air) there is no necessity that any individual ever react to the accidental "allergic" chemistry that may change a particular hormonal signal. Further, even if some individual does so react, because most individuals do not engage in reproduction, the chances that the particular reaction can favorably affect genetic evolution of the hive are greatly reduced. Thus, while many insect individuals may be poisoned by successful plant chemical adaptation, the possibility that this same adaptive process will accidentally produce additional "cognitive" effects by allergic-chemical mechanisms is absent (or reduced) in insects, compared to mammalian type animals. This conclusion is used at the end of the next section.

Relation of Minimal Structure Theory to Evolutionary Biology

At several places, I have argued that the various population measures of marriage theory provide demographic but not genetic information. This position may be counterintuitive to someone familiar with the kinship diagrams used in genetic studies of inbreeding or of studies of family inheritance, etc. Anthropologists know, and geneticists ought to know, that such diagrams, if based on social representations, may not precisely reflect the specific biology (the actual matings) of genetic inheritance.

Simply stated, people "cheat". Diagrams of minimal structures seem so regular, and the statistics associated with them so appealingly available, one is obviously tempted to ask "surely there is some relationship of these to the purposes of population genetics theory?" The answer is that there may be, provided one is very careful on what question is asked, and how it is answered. For example, the network theory of these structures (such as found in Appendix I or Ballonoff, 1976b) might

be treated as a theory of regular systems of mating. These regular systems were exploited for example by Wright (1921) who evidently had no systematic device for enumerating structures, though he clearly knew how to compute genetic coefficients associated with following them biologically. From that viewpoint the theory of minimal structures may have value to genetics studies.

A quite different viewpoint results however if one accepts the social reality that, outside of controlled breeding experiments, kinship diagrams do not reflect biology. In this case, to gain genetic results from the present theory, one must essentially ignore the literal matings in minimal structures, and ask about genetic models that depend on population statistics. There are a number of such models. In Ballonoff (1974c) I explored one of them, with some results of interest here. The interest of this model is primarily for populations near their minimal size (for structural number theory), or where actual population statistics can be used (as in lineage theory).

The genetic model used was developed by Crow and Kimura (1970:102) to study the probability of identity by descent of two genes uniting in a randomly mating population, of known size. (I do not study the question here, but it does not require much "leakage" in a regular system of mating for the system to become effectively random; random mating is therefore a good model to study population size effects of marriage rules with undisclosed cheatings).

We do however know, at least in minimally structured systems, that the numbers of males and females per generation will be approximately constant. (In the minimal structure itself, of course, these numbers will be identical each generation, both to each other in each generation and with each sex over time). This permits estimation of the genetic effective size of the population, through a formula provided by Crow and Kimura (1970:351). At generation T, this formula is:

$$N_{e(T)} = \frac{2N_T - 2}{\bar{k} - 1 + \frac{V_k}{\bar{k}}} \qquad (1)$$

where $N_{e(T)}$ is defined as the effective size of the breeding population per generation; N_T is the actual size of the breeding population, $\bar{k}$ is the average number of gametes contributed by individuals of generation T to individuals of the following generation; and V_k the variance about $\bar{k}$.

The most obvious first question is on how to relate N_T to the values L and N used in this work. Since L is the number of lines reproducing and each act of reproduction requires 2 individuals, 2L seems a minimum bound for N_T. Clearly, N is an upper bound, so $2L \leq N_T \leq N$. Dividing by N produces the result $p \leq N_T/N \leq 1$ since $p = 2L/N$. In other words, it seems reasonable to use p, the proportion of ascribed

reproducers, as an estimate of the minimum bound of the proportion of actual reproducers. This results in $pN = 2L = N_T$ as an estimate of N_T for present purposes.

In the development of Ballonoff (1974c:175-180) then assumed (after reviewing several alternatives) that $\bar{k} = 2$, and had a Poisson variance, so that $V_k = \bar{k}$. Placing these values into the expression for $N_{e(T)}$ gave $N_{e(T)} = 2L - 1$.

With this estimate of the genetically effective size of a population for which the value L could be known from the marriage rule, the next problem is to estimate how this rule (this value of L) affects the amount of genetic identity that can occur in a population, as a result of "drift" or random sampling effects. The table below shows this, computed from the formula from Crow and Kimura (1970:102):

$$f_T = f_{T-1} + \frac{(1 - 2f_{T-1} - f_{T-2})}{2N_{e(T)}} \qquad (2)$$

where f_T is the amount of "inbreeding" (identity by descent) in the population at generation T.

Table 1 shows that if a population with small structural number remained both stable in size and isolated for 10 generations, that up to 84% identity by descent would occur if $s = 2$, but that this drops off to 54% for $s = 3$, to 28% for $s = 6$ and still less for higher structural numbers (and, similarily for more complicated lineage systems). Note nowever tnat a system witn structural number 4 acnieves in 7 generations approximately the same amount of drift effect as a system with structural number 10 (or a larger lineage system) achieves in 20 generations.

What this says is that, due completely to drift effects, the diversity between populations near minimal sizes is affected by the structural number of the rule followed by the system. The amount of such drift is severely lessened by using larger rather than smaller structural numbers (and/or using lineage systems of relatively greater complexity). If there is population interchange between local populations, then the effect of drift is smaller, since the effective size of the breeding population is then much larger than indicated by the size of the local cultural unit.

However, marriage rules are not therefore ineffective for evolutionary (drift) effects: a population whose total size is all in one group following a rule with high structural number (or complex lineage organization) with say $s = 10$, would drift about 2/3 more slowly for the first 20 generations, than would a population of the same total size, broken into units each following rules with structural number 4, if all these units are near their minimal sizes.

TABLE 1

f VALUES FOR $2 \leq s \leq 10$, FOR GENERATIONS

t = 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 20, 30, 40 and 50.

	s								
t	2	3	4	5	6	7	8	9	10
1	.207	.081	.056	.042	.033	.028	.024	.021	.018
2	.329	.149	.105	.080	.065	.054	.047	.041	.036
3	.443	.213	.153	.117	.095	.080	.070	.060	.054
4	.535	.271	.198	.152	.125	.105	.092	.080	.071
5	.612	.326	.240	.187	.153	.029	.113	.099	.088
6	.677	.376	.280	.219	.181	.153	.134	.117	.105
7	.730	.423	.318	.251	.207	.176	.155	.135	.121
8	.775	.466	.354	.281	.233	.198	.175	.153	.137
9	.812	.506	.388	.310	.258	.220	.195	.171	.153
10	.843	.543	.421	.337	.282	.241	.214	.188	.168
20	.974	.789	.664	.560	.484	.424	.382	.340	.309
30	.995	.903	.805	.708	.630	.562	.514	.464	.425
40	.999	.955	.886	.806	.734	.668	.618	.565	.522
50	.999	.979	.934	.871	.809	.748	.699	.640	.603

Consider also the effect of drift on evolution for 50 generations. It is hard for any population shown to have less than 60% inbreeding in that time, even if the structural number is high. If the structural number is low, then the amount of inbreeding is greater still, easily approaching 100% for the smallest structural numbers (simplest marriage rules). If the generation interval is say 20 years, then 50 generations is about 1000 years.

In other words, no matter what the cultural rules, almost any fairly small population will have a significant amount of drift occur in a 1000 year period. ("Small" means per-generation size of local group of 44, the N value for s = 10. This would mean a total group size of at least 2N = 88. This easily includes many "primitive" populations taken to represent the human condition up to the neolothic). Thus the observation of Lumsden and Wilson (1981) that the 1000 year period has some special relationship to culture is true but trivial: cultures must always exist in populations. If these populations have any amount of subdivision then drift can occur. Since much of evolution is the effect of drift, therefore evolution could be measured in 1000 year intervals and found to have "significant occurrence". This fact however has essentially nothing or at best very little to do with the cultural system, particularly over the 1000 year time span. All cultures would exhibit similar drift.

However, I do not argue that there are no relationships between culture and biological evolution. Important relationships exist but are more subtle. I presented one

such argument in the first half of this chapter: the capacity for culture results (quite coincidentally) from biological selection for individual survival in face of errors of the endocrine system. There is another relationship more directly connected to the structure and results of marriage theory. This result follows from the equation for empirical growth per generation unit T, noted in Chapter 4. This equation was

$$R(t) = \frac{2p(t)r(t)}{p(t)^2 + 2r(t)} \qquad (3)$$

where R(t) is empirical growth predicted, r(t) is the value found from equation (10) of chapter 3, and p(t) is the average value of the p values of all rules in use at time t.

With unit generation interval, the dependence of R(t) on the associated p(t) and r(t) values is reflected in the next table, Table 2. The equation is general, but the p values above .5 in the table are those of the more common structural numbers in human cultures.

The table of R(t) values has certain very interesting characteristics. For lower values of r(t), the predicted empirical growth is greatest for some mid-range of p values, and lowest for both very high and very low p values. As r(t) increases this characteristic disappears: growth is greatest for large p values near 1.0, and smallest for smaller values. Indeed, as r(t) increases, R(t) and p(t) approximate each other, as is obvious from the bottom rows of this table.

TABLE 2

SUMMARY OF PREDICTED R GROWTH RATES FROM

EQUATION (3) FOR SELECTED VALUES OF r AND p.

r =	.01	.10	.20	.30	.40	.50
p = 1.0	.019	.167	.286	.375	.444	.500
.92	.021	.176	.296	.382	.447	.498
.82	.024	.188	.306	.387	.446	.490
.73	.026	.198	.313	.387	.439	.477
.68	.028	.205	.315	.384	.431	.465
.63	.030	.211	.316	.379	.421	.451
.50	.037	.222	.380	.353	.406	.400
.40	.044	.222	.285	.316	.333	.345
.30	.054	.207	.269	.261	.270	.275
.20	.066	.167	.190	.186	.190	.192
.10	.066	.095	.098	.098	.098	.098
.01	.009	.010	.010	.010	.010	.010

This fact has an evolutionary implication: keeping in mind that (from chapter 3 appendix) any growth rate computed has a corresponding and equal decline risk, species can only be long run stable if they have combinations of p and r values at the upper left, or toward the lower and lower right edges of the table. One would expect that if a species is "hard wired" by specific genes for its "culture" or social organization, that it would be so for a very stable form. The social systems most stable for widest ranges of variations of internal group actions (that is, for changes in r(t) per interpretation of this in chapter 4) must therefore have a very low value of p(t).

A low value of p(t) means in effect that local groups are offspring of relatively few adults compared to the local population size. The social insects fits this description. In a bee hive, the entire local population are generally the offspring of a single female. For social wasps, a single "queen", or several mutually nested females, produce the entire next generation. Thus, when evolution produces a species with a social system that will not change under its own internal pressures, evolution will produce a species with a low value of p. Something rather like the social insects!

On the other hand, if a species requires a great deal of plasticity in its structure, particularly to provide occasional high rates of population growth (R(t) values) as a result of internal actions of the system through cultural choices (r(t) values), then evolution must permit a social system with high p(t) values. In such systems due to the range of risked rates inherent in the combinations of marriage rules (per Chapter 3 above), actions within the range of p and n values compatible with their structural numbers or lineage organizations can temporarily drive up the r(t) and hence R(t) values, without requiring a change in p(t), thus without requiring a change in social structure. Similarly, to the degree that changes in social structure is part of a species adaptation to its ecological or economic environment, then species plasticity of p(t) in local groups is an expected part of such species.

Or, simply stated, if a species is capable of surviving in a great diversity of environments, requiring diverse ecological adaptations, then "cultural plasticity" is to be expected. Man's cultural diversity and geographic dispersion are part of the same phenomena, and both are related to the fact that all of man's cultures have have (compared to social insects) high values of p. This parallels the result of the last section of this chapter on evolution of intelligence. (The reader may also find it useful to compare the p values listed on the current table, to the p values for various structural numbers in table 1 of the appendix to chapter 3.)

Note that a culture, or local population, can regulate its value of r(t) by changes in its average family size per ascribed reproducer; but it regulates its value of p(t) by changes in its (mix of) culture(s). One would not expect to find any species or cultures existing (for more than short periods) with mid-range values of

p(t), since all entries in table 2 of this chapter could just as easily be negative; they are, after all, risked rates per chapter 3. Several successive generations with these negative values, with even moderate value of r(t), would eliminate the population. In structural numbered systems, if the minimal structure conditions are violated by insufficient population size, the decline risk associated with a higher structural number could force the culture to adopt a smaller structural number, and correspondingly smaller R(t) risk. On the other hand, a culture could obtain a greater growth rate, without change in its growth efforts (constant r(t)) by shifting to a higher structural number with lower p(t), provided it has sufficient population to maintain the minimal size of the higher ordered rule.

Cultural plasticity is intimately connected to the evolutionary characteristics of a species. In humans, unlike ants, populations are not genetically hard wired for any specific cultural form. From the above analysis, one would expect just that result.

Marriage theory is a useful tool for evolutionary biology. The fact that it is not the tool expected by socio-biologists in no way detracts from its value.

Chapter Summary:

A mathematical theory which successfully predicts the properties of human social demography, from knowledge of culture, now exists. A totally independently derived line of research on physiological biochemistry suggests that evolution of capacity for culture is a product of evolutionary biology.

Together, these separate lines of research imply that human evolution and especially evolution of human intellectual (cognitive) ability may be explained by processes of natural selection. This natural selection will favor those individuals with cognitive ability at any given time, sufficient to "override" specific internal failures particularly of the endocrine system. Since the environment of chemical insult to the endocrine systems of members of population is constantly changing (itself from processes of natural selection particularly in the plant kingdom) all human descent lines are eventually subject to this environmental risk.

Thus, natural selection acting on a independent biological process (plant predator/prey co-adaptation) has as a consequence greater, but universal human, abilities for manipulation of cognitive systems. Since we generally recognize these systems as human cultures, natural selection for overcoming failures of the endocrine system is a primary evolutionary cause for the rise and maintenance of human capacity for culture in all planetary human populations. Furthermore, in distinction to mechanisms that posit literal "genes" for specific cultural adaptations, this mechanism results in a universal human ability for culture: it accounts for successful human transcultural adoption. such adoption would generally fail were

culture or the ability for specific human cultures literally transmitted via specific genes.

Footnotes to Chapter 5

1. I particularly rely on chapter 27 and 30.
2. See Freiden (1979), Greenberg and Harper (1960), and Nakamura (1954).
3. For example, Macleod (1922) is not in the least shy to describe enzymatic reactions and their properties.
4. See as examples Baldwin (1965) pages 40-41 or Greenberg (1960) pages 3-25.
5. As example, Blum (1964).
6. For example. Chapter 8 of Krantz and Carr (1954). This work is particularly interesting because it pre-dates the publication of work by Godlowski (1962).
7. Examples of stereochemistry and structural chemistry can be found in nearly any text on biochemistry. The more useful may be in Smith (1976), Richards (1977) and of course Godlowski (1962), Vol. I. The idea of control of proteolysis as a possible metabolic mechanism has been rediscovered, Holzer and Heimrich (1980). For a detailed example of how stereochemical and isomeric variations affect metabolic properties, see Vida (1977), or many articles in Vessel (1971). On the subject of tissue specificity, particularly note the conclusions of the article by Gillette in Vessel (1971) pages 43-63.
8. This in not only true of allergic chemistry but of tissue chemistry generally. The work of Landsteiner (1936) is classic on this subject. Nor is the conclusion restricted to human chemistry. Nakamura (1954), working on guinea pigs, was aware of this. Consider the following quotations from Rybak and from Landsteiner:

> Biological individuality would appear to be related to morphology (an example of this being finger prints) but is also related to function, corresponding to the correlation between gene, enzyme and structure. This individuality is apparent in the composition of different animal species. ...the value of comparative biochemistry is that it is a molecular expression of genetic development from which similarities and differences in structure and functions of different lines, which are related morphologically, can be established... Numerous examples of biological specificity can be cited. We have already seen that the food requirements (vitamins, amino acids, etc.) of one group of animals differ from those of another. In addition to apparent experimental variations, individuals of the same species show differences in, for example, the amount of compound secreted and such a compound expresses an aspect of biological individuality.
>
> For example, the concentrations of the various constituents of human blood (are) such that a given concentration can not be described as pathological...
>
> Thus, the level of glucose in blood can vary between 84 and 125 mg. per cent, these extreme levels being an expression of biochemical individuality and not of any abnormality.
>
> (Rybak, 1968, page 205)

> if in trying to form a definite idea of the chemical basis of protein specificity one adopts the current view that proteins consist of peptide

chains, or, at any rate, are made up of amino acids, then their specificity obviously depends in some way on the nature and arrangements of these components...

(Landsteiner, 1936, page 19)

9. The behavioral effects of the daily chemical environment have been described in Randolph and Moss (1980). Molecular foundations of many other disorders were described (in an unfortunately mystical manner in evident ignorance of Godlowski's work) by Philpott and Kalita (1980). The most comprehensive body of clinical research, to my knowledge, was amassed through the brilliant clinical work of Dr. William Cox, M.D. of Independence, Missouri. Unfortunately, Dr. Cox was unable to publish the results of over 15,000 provacative antigen tests on clinical patients using essentially the methods described by Godlowski.

10. For example, Nakamura (1954). The chemistry of acetylcholine is also described in Greenberg (1960). It is very useful to compare the chemistry of cholinergic drugs and neurotransmitters to understand how allergic reactions, being tissue specific, can also produce many "neurological" including "mental" effects.

    The gross pathology described as "anaphylactic shock" is essentially recognized, it seems to me, due to such disorientation of medical research, not to absence of other pathological effects related to the syndrome. If the effort of medical research is to determine a "just barely lethal dose", then the "subtle" effects resulting from other does will almost certainly not be noticed.

    The human brain, for example, is a complex of peptides and polypeptides acting in various neurotransmittal roles. (See Marx, 1979 or Mendez, 1980 as examples). With small stereochemical changes in any of these, the more surprising result would be that no behavioral effect is notices. The more likely result is that small changes in rates of isomeric reactions and of changes in configurational stability (i.e., of the stationarity of the vector of rates among configurations) will produce profound effects. As a simple example, consider the effects of various salts (e.g., "alkaseltzer") in treating "hangovers" (by changing the pH of the environment). As a less simple example, consider the effects of electrochemical activity of the body of fluoride intake. Fluorine being one of the most chemically (electrochemically) active of elements, changes in rates of configuration flux are to be expected. The brain, being so complex in compounds which are highly sensitive to con- figuration, is a place one might expect to look first for behavioral and "allergic" effects of changes in fluoride concentration.

11. See Harborne (1972 and 1977) and Smith (1976). Also, note that while histamine is not normally classified as a terpene, recognizing its structural similarity to terpenes is but an application of standard pharmacological research practice. Allergy can only be "explained as related to histamine" if allergy is defined by those gross physiological effects caused by plant mediated substances containing terpenes. A great deal of medical ignorance has been produced by inadvertently doing exactly that. This may be one reason why, in

the U.S., the term "clinical ecology" is used to describe the practice of physicians interested in all metabolic disorders related to incomplete proteolysis; histamine/terpene related disorders, the classical realm of "allergy" are but one class of these.

12. From this fact alone, as well as a reading of Godlowski, one should suspect that many heretofore untreatable and poorly understood syndromes (such as autism, Tourette, etc.) will very likely be found to be either allergic disorders due to environmental antigens, or very possibly auto-allergy, perhaps to an individuals' own hormones. This could only be proven by undertaking provocative testing as described by Godlowski and developed practically by Cox.
13. Indeed, the widely repute "greater intelligence" or "allergic people" may be taken as evidence of this mechanism. It results in part because those with the more disabling disorders (e.g., autism) may never even be diagnosed as "allergic" and likely never tested. Thus the true population mean is not that observed in those diagnosed; and in part because allergic people are forced to depend more obviously on cognitive abilities to a greater extent than do "normal" people. However, because the background of biochemical insult is continually changing, over time, as a consequence those with greater intelligence will more likely survive and reproduce. No human population, nor descent line within a population, is exempt from this risk and therefore all are subject to this same differential selection favoring intelligence. Indeed, the same argument also implies abilities related to "intelligence", such as play, which involve greater "cognitive" control over the body, would all be favored by such selection.