# ASCAP NEWSLETTER

Across-Species Comparisons And Psychopathology Newsletter
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"A marriage aged five/ Is coming alive./ Watch it wither and thrive;/Though it's coming alive,/ You must guess,/ No or yes,/ If it's going to survive." Ogden Nash[1]

The ASCAP Newsletter[2]
is
a function of the

**International Association for the Study of Comparative Psychopathology (IASCAP)[3]**

Newsletter aims: 1. A free exchange of letters, notes, articles, essays or ideas in whatever brief format.
2. Elaboration of others' ideas.
3. Keeping up with productions, events, and other news.
4. Proposals for new initiatives, joint research endeavors, etc.

IASCAP Mission Statement; The society represents a group of people who view forms of psychopathology in the context of evolutionary biology and who wish to mobilize the resources of various disciplines and individuals potentially involved so as to enhance the further investigation and study of the conceptual and research questions involved. This scientific society is concerned with the basic plans of behavior that have evolved over millions of years and that have resulted in psychopathologically related states. We are interested in the integration of various methods of study ranging from that focusing on cellular processes to that focusing on individuals to that of individuals in groups.

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Planning: John Pearce and Leon Sloman have been thinking about our 1993 meeting; Leon suggests that Binghamton, NY, should be preferred as that is where the State University of New York branch is hosting the Human Behavior and Evolution meeting (HBES) meeting 4-7 Aug 1993. John is looking into that instead of Boston prior to HBES. But Bostonians Steve Heisel and Dan Wilson are thinking ahead to 1994: might a confluence of meetings occur there and then?

Announcement: Harold Pincus editorializes in the December issue of Psychiatric Research Report that the U.S. Congressional Office of Technology Assessment (OTA) has just

released a report on "The Biology of Mental Disorders." OTA prepared this at the request of Congress.

OTA is an independent respected body, and the report is timely given the NIH reorganization (NIH now includes research components of NIMH and drug/alcohol institutes). Congress learns in it that funding in this research area has lagged behind heart disease and cancer research. Also, it states that "Development of animal models, brain/tissue banks, elimination of fiscal and practical barriers to clinical research, and increased training of clinician researchers are also badly needed."

As expressed in a reply to Professor Boris Dashevsky just below, this is also an exciting time conceptually for the sciences fundamental to psychiatry. Funding for patient care and the prestige of the specialty for medical students may be at all time lows in the U.S., but perhaps this is preliminary to better times.

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Letters: *12 December 1992*

*In the beginning let me introduce myself. I am formerly from Moscow State U but am now a political refugee (after 11 years of refusenik experience in the Soviet Union) I have lived in the US since May 1991.*

*I am a member of the International Society of Human Ethology (after the 22nd meeting in Kyoto, where I presented). I have extensive experience in comparative study of cognitive behavior and its disorders in a wide range of species including primates and also human infants and children. I have the honor to be a student of the most prominent Russian anthropologist, the late Professor Yakov Roginsky.*

*Now I am invited to be at U Cincinnati as Adjunct Professor of Psychological Department (a temporary position) . I am in the process of preparing a course for undergraduate psychology majors entitled "The Introductory Human Ethology" where I would like to cover and conceptually connect main ideas of "evolutionary psychology" being presented by Barkow, Buss, Cosmides, and Tooby on the one hand, and properly "human ethology" being worked out by Eibl-Eibesfeldt* et al *on the other hand. As a matter of fact, these fields of knowledge [that are] so genially interconnected have been developed almost independently so far. I think it is not a formal fortuity that the both sides hardly mention each other in their studies. Besides I'd like very much to include in this course the important approaches of yours and your colleagues from ASCAP Newsletter for I do absolutely agree that "a mature understanding of human behavior rests on cross-species comparisons." I send a check for ASCAP Newsletter, 1993.*

*So I am eager to get your advice both in regard to the best (text)books (and articles) on introductory level in Human Ethology/ Psychiatry and your personal notes and point of view about teaching such a course.*

*I will be so grateful for getting your letter and possibly some materials for lecturing.*
*Boris Dashevsky, U Cincinnati, U.S.*

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Congratulations on your success in behaving in the refusenik mode! What an incredible human ethological laboratory has resulted from the extraorcnary and sometimes tragic convulsions of the Soviet world. In-group and out-group behaviors have become highlighted for all the world to observe with great variety in their resolutions.

I hope that readers will respond to the above call for references, books and papers. I send you material I have personally authored (especially proofs of a chapter for a forthcoming

comprehensive textbook of psychiatry) as well as other ASCAP Newsletter issues that provide you with key references. For example, these include the bibliography of John Price, a landmark volume edited by Michael Chance and the books of Paul Gilbert.

Let me say a few words about distinctions among ethology, Darwinian psychology and the central philosophy of ASCAP, which I would like to think of as "sociophysiology" though I am unsure that other **IASCAP** members agree on the term. "Biopsychosocial model" is a possible alternative.

Biopsychosocial model was coined in 1965 by George Engel (a physician-psychoanalyst though not psychiatrist) who worked with the emotional issues of medical and surgical patients. He intended this expansion of the medical model to heighten medical concern with social and individual factors. But Kathryn Hunter asserts: "The reception of Engel's potentially revolutionary position in medicine has been distinctly odd; no one disagrees, yet little changes."

For our purpose, it also fails to capture the basic plan assumptions of the central defining features of **IASCAP**'s mission: "This scientific society is concerned with the basic plans of behavior that have evolved over millions of years and that have resulted in psychopathologically related states." It fragments the three realms of experience and investigation (bio-, psycho- and social are each their own—different things that may link together but that are not truly integrated). The three levels should instead be different views of the same thing, not artificially separated and distorted. We need conceptions that provide connecting links that may not be yet there although we know eventually they must be; search for the missing links will proceed better if they are looked for. A mind prepared will see things perhaps there but otherwise not visualized.

Sociophysiology means that many physiological mechanisms in the individual organism have been evolutionarily adaptive because the individual can define and connect to others of its kind. Perhaps this occurs via in-born registrations of conspecifics and reactions to them. Or via memories of early existence: Mark Erickson has noted that there is evidence of familial bonding in humans and other mammals that results in incest barriers later.

Other areas of sociophysiology include sexual and mating mechanisms, and (prominently featured in these pages) the biology of social rank. In humans, language and other motor communications are included along with sensory systems; a neurological disorder in which people cannot recognize famous or familiar faces (prosopagnosia) indicates a cortical system is usually present for this.

Physicians and psychologists—clinicians—have been important for **IASCAP.** In the preface to The Man Who Mistook His Wife For A Hat, neurologist Oliver Sacks provides two epigraphs, one stating "The physician is concerned [unlike the naturalist] ...with a single organism, the human subject..." while another epigraph (from Osier) states that patient stories *are* like what a naturalist does. Sacks goes on to say (in a statement that echoes issues of **IASCAP** clinicians) that he feels himself *both* a naturalist and a physician.

Of course, another feature is that concern with body mechanisms anchor **IASCAP** preoccupations. Indeed, some psychiatric disorders may give a purchase on the investigation of normal states similar to them—in parallel to the insight provided by prosopagnosia. We need to establish some method to dissect communicational systems which appear seamless and whole when operating normally. Pathology can help, but our diagnostic schemes

have problems. For a superb critical review of psychiatric diagnoses see Dan Wilson's contribution below.

Ethology and Darwinian psychology are adjunctive — not at all contradictory — to the central focus of **IASCAP.** We refer to them constantly but they haven't been enough — they attend to some facets only of **IASCAP** concerns; they are basically naturalistic, not medical nor do they conceptually root adaptive patterns in physical cells — at least to the same degree. They focus less on individuals and more on populations.

Sociophysiology on the other hand would examine (eventually if not now) the workings of the genome and brain/body in specified detail, relating facets and determinants of behavior to mechanisms of molecules, cells and bodies. What genes on what chromosomes are there to mediate communication? What proteins stem from their operation? Where in the brain and other body parts do such proteins get expressed? Functionally, how do they govern/cause/influence behaviors and communicative states?

To illustrate the point with a question: despite the fact that the 1973 Nobel Prize in *Medicine* and *Physiology* was awarded to three ethologists (Lorenz, Tinbergen and von Frisch), has ethology yet related itself to the central models of pathogenesis characteristic of medicine and descriptions of the normal workings of the body implied by the term physiology? In ethology the cross-species comparisons are those of havior, not those of neural and other body mechanisms. Comparisons stemming from ethology have allowed speculations about the evolutionary history of the behavior in question and represent important hypothetical formulations. I would see ethology as part of sociophysiology: it examines the "socio-" component; others benefitted from ethological insights may be better equipped to explore physiological components using blotting paper/electric currents of chemistry or the imaging techniques of PET and MRI.

Evolutionary psychologists are concerned with adaptation assumptions and population models in conjunction with Darwin's central tenets of natural selection, diversity and inheritance. They rely heavily on mathematical models and examine complex psychological attributes with an eye to calculations of relatedness with 'selfish gene' assumptions built in. Again these are important hypotheses needing physiological exploration. Below (this issue) John Pearce considers the ideas of Helen Fisher that explain an empirically registered timing for divorce 4 or 5 years after marriage. Sexual passion uniting couples wanes over time, for adaptive purposes perhaps, with other reasons for attachment operating after this.

What systems, neuronal areas, and endocrine systems are involved with sustained passion *vs* its withering? Where are such pacemakers and how are they transduced from the person's life history? In commentary in Nature on a documented change in bird migration (see below p 13), "selection pressure must have been strong." But of course it is premature to consider yet which are the involved genetic and neuronal structures. Premature *yet* but on the horizon! Let us prepare our minds and those of our students! *Vive* Dashevsky!

Also, considering a separate point, John Pearce in his evolutionary psychology article this issue notes that the gatekeepers of the fabulous imaging machines are as yet unaware or uninterested in our hypotheses. With Frank Sulloway, he forwards a specific hypothesis that they would like to test. This is exemplary! Now we have to talk those gatekeepers into using their machines for this.

Persuasion of these potential collaborators will be enhanced with the publicity and acceptability of our

ideas; only with teaching and distribution will that happen. I am therefore immensely pleased at your interest in developing these groups of thought and data into an integrated course. And I believe that it is no accident that you are creatively open to this after being oppressed in the former Soviet Union.

Back again to what we as a group or groups need to do to convince the Pearce/Sulloway gatekeepers: Paul Gilbert notes that we must publish, publish, publish to get our point of view across. We need to brainstorm about what it is we wish them to look at (the function of ASCAP), but major review articles are also needed in widely read and respected journals. Gatekeepers are more likely to respect our requests if they are backed by peer-reviewed and supported scientific hypotheses. In the meantime, the pages of the Newsletter can feature drafts, first versions, ideas to toss up and be debated.

Fundamentally, we need to isolate behavioral states that have hypothetical central counterparts that can be looked at. Is there a distancing gauge with the gain turned up in the prefrontal lobes influencing schizoid behaviors? If so, where? right? left? orbital frontal cortex? How might it be differently parlayed in the subcortical *vs* cortical neurons? Optimally, of course, would that there were genetic correlates too (or even better *genomic* correlates — DNA structures that exist in many species — for basic plan insights).

I hope this is helpful in class preparations: please challenge your classes themselves to come up with hypotheses. This is an extraordinary time in neurobehavioral (or, as I would prefer to say it, socio-physiological) science. Those of us in university medial centers will be working on the gatekeepers but we need all help we can get! Please keep us posted. Contribute your results.

Letters (cont). *December 14, 1992*

*... I'm surprised that the APA symposium was not accepted - [but] I think It's a matter of time until projects like this are routine.*

*Did you see the evolutionary arguments concerning trichotillomania In the Dec '92 Issue of The Psychiatric Times? Apparently recent studies indicate the prevalence is much greater than previously thought. Writers like Richard O'Sullivan of Harvard and Rappaport and Swedo of the NIMH now seem to favor evolutionary explanations— which to me seem plausible.*

*Mark Erickson, San Francisco, U.S.*

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Abstract : Gilbert P: Defense, safe(ty) and biosocial goals in relation to the agonic and hedonic social modes. World Futures 1992;35:31-70.

This paper outlines why sociobiological efforts to move from gene to behavior without consideration of the intervening, evaluative systems that tag stimuli as threats or rewards is misleading. ...[Rather] social behavior [may] signal threat (sending and responding to threats) or investment (social rewards and reassurance).. .two mental dimensions. The paper explores the role of various species-typic behaviors for care eliciting, care giving, cooperating, and competing, distinguishes between authoritarian and authoritative hierarchies and considers the evolution of self-awareness which arises from various role enactments. These aspects of human nature can be integrated in Chance's theory of the two social modes of relationship, agonic (threat-based) & hedonic (affiIiative-reassurance based). In the final sections, the implications of the ideas are considered in relation to cultural and political value systems.

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Fundamentals of Evolutionary Psychology by John K Pearce

It seems to me that our enterprise is, at bottom, applying ethnological and other biological concepts to human beings. We try to use the same concepts that have proved to be powerful with other species to understand people. (As I see it, a key mediating

concept makes this possible: The neuroscience concept of hierarchical systems of parallel processing networks. These networks make possible the variety and complexity of human behavior.)

As a method, we pick out typical behaviors, track their cross species parallels, and speculate about their neural substrate. Neuroscience speculations are essential to our ambitions; we hope to get to the true heart of things, as Freud did when he began his neuro-psychological conjectures. The problem is, as Frank Sulloway points out, we don't do neuroscience; we are not the keepers of the fast MRIs — the tool that seem most promising for mapping the neural substrate of behavior.

We should propose experiments for using MRIs to clarify evolutionary psychology concepts, particularly in connection with the use of medications. Obsessive-Compulsive disorder (OCD) is an obvious candidate. OCD is thought to be a disorder of the neural system that mediates grooming. Serotonin blockers are clearly effective in moderating OCD. Sulloway points out that we need experiments with fast MRIs that compare, with a cross-over design using medication, the brain when involved in grooming, obsessing, and in a control state.

Leon Sloman has expressed concern that evolutionary psychologists might split into several groups. That is surely true, simply because people like to do that sort of thing, at least they do if there are enough people to form a number of separate bands. (I know I hate crowds.)

Splits in the psychotherapy world are partly diated by taking up different cases as paradigms—Psychoanalysis/hyiceria, Feminist Therapy/male dominance and exploitation, Bowen family system/lack of differentiation, Cognitive Therapy/depression, and so on. It is not that these therapies are not successfully applied to many different problems, they are, but the paradigmatic case is important to the identity of each school (and can even be a procrustean bed). What corresponds to this paradigmatic choice in our field is the selection of particular behaviors to which we apply our evolutionary analysis. Since people do all kinds of things, there are lots of behaviors to choose from. A sensible alternative would be to list the <u>many</u> behaviors we would like to explain, as biochemists list metabolic reactions. This has to be done in schematic form, since there are too many for narrative presentation. When we undertake narratives, we find our vision narrows, and what we describe fills the world—at least as the world as we see it when we construct narratives.

This methodological warning is important. Understanding human limitations is our specialty; we should not forget these limitations when we are doing our science, our ambitious science.

I believe my clinical work is enriched by my evolutionary framework. But in this belief I am no different from any other therapist, shaman, or believer. I award myself points for my cross-species perspective, but I know that other people will consider our cross-species approach crude reductionism. It is the link to neuro-science (or other experimental procedures like those of Cosmides and Tooby, or David Buss) that is essential to legitimacy of evolutionary psychology as a scientific movement. We have a potential role contributing good ideas to the keeper of the MRIs. As Frank Sulloway points out, and almost any scientist would agree, science is a method. It is a collaborative, systematic, controlled effort to get things straight. We must keep this in mind; we must constantly think about how we can put our ideas to the test.

Lumpers *vs* Splitters in Evolutionary Psychology by Daniel R Wilson

This refers to Paul Gilbert's response in Nov ASCAP to RG's review of Paul's Depression and Powerlessness book in Sep ASCAP; Paul synthesizes so much so well that I think it a bit harsh to judge him for his possible "sins of omission". Still, his "confession" is a good read.

While I share the reviewer's keen interest in genetics, receptors and all the other basic medical science so germane to evolutionary and clinical research, I think Paul, sensibly, hoes a different row. His is, afterall, the work of a practicing clinical psychologist and it shows: kind, thoughtful, yet bold ideas masterfully built on the firm foundation of his exceptional knowledge of the literature of his discipline. I think Paul's books are a strong contribution to his evolutionary psychology. He writes with clarity, style and substance.

Basically, I think the two of you are looking at the same edifice from slightly different perspectives of neighboring intellectual "guilds." As it has been said that medicine has no foundational theory while psychology has too many, we all enthuse about the day when evolutionary sciences will remedy such issues. Yes, the incorporation of more medicopsychiatric 'blood and guts' would be a plus, but one cannot be a master of all trades! Moreover, anyone who, as Paul does so well, can use evolutionary theory to invigorate complex notions of theoretical psychology must be doing [1] the Lord's work'. That he does so at the end of long clinical days is all the more remarkable. Indeed, how good it would be if more of our colleagues, say those in the laboratory, tried as hard to think through and explicate the evolutionary consequences of their work and to do so moreover with such skill.

As to Paul's response, I was especially interested in his critique by way of faint praise for the Neo-Kraepelinian classification agenda ("DSM phenomenologies"). I am now taking this matter up in a paper for the June '93 meeting on "Evolution and Human Sciences" at London School of Economics. Beware, I hope to spike the waters of both Kraepelinians and Freudians with a few friendly polemicisms! I must confess that I was once—before I got the 'religion' of clinical Darwinism—a neo-Kraepelinian. I imagined that DSM pointed the way to synapses, genes and all the rest. Later and more briefly, I trod the more arid path of Fruedian apologetics. For what it is worth, I think Paul only half-heartedly gives taxonomy its due and this I do not understand. Yes, neo-Kraepelinism is riddled with problems. Still, it is: (1) really all we have that is reliable, (2) rather workable in its basics, (3) cut of a better cloth than all that Freudianism and other abstruse chatter! In any case, there is no escaping the fact that taxonomy, good and true, is an absolute prerequisite to deeper knowledge of a more dynamic nature. Just try to imagine Darwinism without taxonomy. No Paul, it just will not do to gloss over reductionism. After all, as Sirs Karl Popper and Peter Medawar remind us, deduction with its necessarily reductive insights, remain *the* only basis for valid scientific insights. See also the words of Kant below.

Interestingly, Paul cited work by my friends and colleagues, Skip Pope and Jim Hudson. I assume it was their superb 1990 review of "Affective Spectrum Disorder" in the American Journal of Psychiatry. Without using such language, they noted how whole generic clusters of DSM-IIIR categories are little more than academic differentiations without natural validity. They went on to catalogue how a great many arbitrary

'taxons' (that is, properly put, what diagnostic categories are) can be lumped together on a variety of phenomenological grounds. For example, several of the mood disorders can be lumped with some of the eating disorders; so too, bipolar and obsessive-complusive disorder overlap. I have read this work in from its prepublication form onwards and have regularly talked with both Skip and Jim about it. I believe they would agree their groupings are essentially "phenotypic equivalents," ie, they represent variant epigenetic expressions of the same genes. It is such natural taxa as these that eventually will be amenable to the analytic techniques of molecular and evolutionary science.

Such a lumping approach is a way to begin to clean up the mess that DSM-IIIR and other operationalized taxonomies are fast becoming. Having participated in the field trials for both DSM and its sibling International Classification of Diseases, I know a bit about how these things are put together and split apart—by committee! Of course, historians of medicine will someday note that psychiatry passed through an adolescent phase replete with fadmongering. Such fads overemphasize the clinical differences and thereby "split" observations up into ever more numerous if narrow 'species' of disorders. This, in turn, gives rise to: (1) false claims of expertise, (2) published arcana of dubious worth and, (3) a tendency to miss the forest, not for the trees, *but for the leaves themselves!*

Leaves are not species any more than some of these entities are cogent disorders. Yet, whole careers are now manufactured of such base materials. Witness a most recent example or two. It is mentioned here and there that Body-Dysmorphic Disorder and Late-Luteal Phase Disorder are hovering in the wings trying to sneak into DSM-IV. I do not mean to pick on these two entries, but they do have their lobbyists for inclusion in the DSM nosology. Fortunately, there are some skeptical heads prevailing who prefer diagnostic taxa with a bit of a track record in the history of ideas. That sceptics have also noted an incongruence between the narrow basis of validation and the gross overlap with other categories into which such disorders could better be lumped adds to the hollow ring. If only common sense were less rare in the Councils of the APA!

It is all quite reminiscent of the early days of fossil hunters when a bone, nay, even a tooth, was enough to define new taxa. Of course, such seemingly brilliant advances were memorialized by their "discoverers" with eponymous names for "their" species. Such ambitious researchers have been described as "splitters". They were countered by "lumpers" who, properly, emphasized the unity and reason that flows from a good taxonomy. Lumpers also were not so egotistical as to put the prophet ahead of the prophesy. I suppose we perhaps ought to be thankful that DSM-IIIR does not name categories after the personages who have pushed them forward. Too often, such researchers have made up for thin data and thinner sense with insistent committee wrangling that would do a Washington lobbyist proud. Like fossil hunters of old, there is a career "paydirt" in naming a 'new' disease and trumping it up as a unique clinical taxon. Clinical lumpers make less academic hay, but will be proved right in due course. Surely, there is an intellectual promised land with hell to pay for miscreants!

I would propose, like Paul and others such as Kalman Glantz, that a tentative diagnostic taxonomy be developed based on evolutionary first principles. In such a taxonomy, a key

point would be to "lump" together clinical phenomena that are likely phenotypic variants of the same or similar genetic systems. This would immediately point to important aspects of developmental genetics—the epigenetic socioecologic means by which human behavioral phenotypes are elicited. A few leading questions. Why does antisocial disorder in males covary with borderline personality in kindred women? Hagop Akiskal notes a thirty percent overlap in Bipolar/OCD pedigrees. Why do some family members express manic-depression while others exhibit obsessionality? Why do genes linked to bulimia in women covary with affective illness in kindred men? Is this a sex steroid effect, a social psychological effect or some combination? Indeed, cannot these clinical sexual dimorphisms, themselves, be linked more directly to sexual strategies. If so, this angle might simultaneously appeal to both Freudians (sex!) and Kraepelians (classifications!) .

Moreover, it is this very mess of psychiatric classification that fouls the efforts to delineate genes expressing psychopathological phenotypes. Currently, direct genetic studies of molecular biology hinge on techniques of LOD-scoring. A detailed discussion of LOD-scoring is beyond the scope of this missive. It is well treated by Miron Biron in his chapter in Goodwin and Jamison's Manic-Depressive Illness. Suffice to say, this crucial technique bases calculations wholly on clinically defined diagnostic terms. Diagnostic terms of course have great validity in many areas of medicine (eg, metabolism, oncology, etc). In psychiatry this is less so. It too quickly becomes tautological. Direct genetic searches for genes defined by a flimsy psychiatric nosology will be thwarted until schemes of diagnosis are established that have a validity in nature. That is, findings from the direct genetic study of psychopathology will remain inconsistent until psychiatry comes to grip with how its hodge-podge of clinical phenotypes are often simply the expressions of a genome that is more coherent and unified than is now appreciated. The "splitters" are holding back progress with their introduction of spurious nosological distinctions. Theirs is bad sport in itself, and only worse when slogged through the playing field of contemporary genetics analytic techniques.

As John Price mentions, both heritable and non-heritable features are subsumed in the complete Darwinian view (an idea splendidly developed in Dawkins' Extended Phenotype). Therefore, a classification system built on evolutionary first principles will incorporate the "softer" ecological stuff, too. Thus, may we avoid the dusty extremes to which psychiatric reductionism is sometimes taken (as bad, in its own way, as the worst examples of Freudian "just-so" story telling).

*Very important* is John's further point about how psychiatry ought to basically ignore recent squabbles which have erupted over the proper interpretation of direct genetic linkage studies within psychiatry. It is a tempest in a teapot. After all, a century of sound psychiatric epidemiology exists (twin, adoptee and family studies). This remains the most solid foundation for current discussions of psychiatric genetics. The direct studies will work out eventually and, I am sure, largely corroborate the vast body of 'old fashioned' epidemiology. They will be icing on a cake that has long ago been baked.

For this reason, I find it curious that social constructivist ideologues have found such joy in the technical complexities which beset the Amish genetic studies. Their relief is illusory. The issues inherent in these

studies are nothing more than problems of terminology writ large. Translating LOD-scoring techniques to the realm of psychopathology requires subtle skill. It is delicate largely because psychiatric diagnosis is highly idiomatic as compared to 'real medicine'. So then, what are we to make of Amish who are not diagnosed as "real" cases although they manifest bipolar spectrum phenotypes (eg, cyclothymes, dysthymes, etc)? The inclusion of such persons among the "normal" group defies naturalistic sense even though it is consistent with the fine print of DSM-IIIR. The upshot of the Amish study is that one (of a variety) of genes linked to bipolar disorder has basically been localized. This appears to be a largely mendelian dominant single gene with variable penetrance. We have, from the standard works of psychiatric genetical epidemiology, known all this for years. John is right to remind us that we can talk up genetics even as the 'wet' geneticists fuss.

Back to the splitters. I mentioned them not with true rancor, but with some annoyance as to how this "Disorder of the Month Club" psychiatry undermines more serious efforts to establish a naturally valid, as opposed to an academically hip, taxonomy. Kant, as ever, seems to have said it first and probably best. As he noted in A Critique of Pure Reason; *Academic taxonomy deals with classes; it merely arranges by similarity; while a natural taxonomy arranges by kinships of generation. The former supplies a school system for the sake of memorization; the latter supplies a natural system for the sake of understanding; the former has fcr its purpose only to bring creatures under a system of labelings; but the latter seek to bring them under a system of law.*

John Price, in his indefatigable fashion, cranked out yet another first rate review of theory central to our common work. His piece on "Behavior and Genetics" in the Nov ASCAP is superb especially in how he extends solid evolutionary concepts toward new horizons. As it happens, I am spending the better part of this sabbatical year delving into sources of variation within psychiatric populations. With time away from my busy practice and medical school duties, I have an opportunity to collect my thoughts and transmit them to old friends via ASCAP. In so doing, I recall the proverb that it is better to remain silent and be thought a fool than to speak and remove all doubt. Ah well.

John asks who 'discovered' the phenomenon of frequency dependent selection. It is difficult to assign priority for, like so much in modern evolutionary theory, this concept was adumbrated long before it was finally worked out in detail. Even Grandpa Erasmus Darwin, in Zoonomia implies that environmental conditions can shape contingent, heritable forms and behaviors in predictable ways. So, too, Chambers implied in his Vestiges of Creation.

Truly rigorous explanations have been hammered out only much more recently. Perhaps the first to note that it may be advantageous for a species to utilize subniches via differinq varieties was Ludwig in 1950. In fact, this idea is occasionally called "Ludwig's theorem". More precisely, Ludwig's paper is the first to calculate the means by which traits which would be of inferior viability in the classic niche are maintained when subspecies varieties occupy atypical subniches. This is, to my knowledge, the first formal expression of what appears to be the central idea of frequency dependent selection. Dr. Moment in "Reflexive selection: a possible answer to an old puzzle" (Science 1962) came closer to the current ecological

geneticists' concept of frequency dependent selection, although I do not believe he coined the term as such.[13] Perhaps the term itself is one of those purely descriptive coinages which arise in the University lecture hall as teachers try to explain things in alternative language.

I should mention that the authors of the 'classic synthesis' seem not to have said much about anything resembling frequency dependent selection as such. However, Haldane did mention the dimorphic effect of selection intensity as it relates to competition within predictably alternate environs. But Haldane did not amalgamate all the ideas so central to frequency dependent selection.

There are few contributions of an explicitly clinical-psychiatric nature along these lines. I believe the article published in Nature in 1963 by Sir Julian Huxley, Ernst Mayr and others titled "Schizophrenia as a Morphism" is likely the first. I had the pleasure of discussing this with Professor Mayr lately. The technical arguments remain sound. But psychiatry has (with its shifting diagnostic criteria, faddishness, etc) proved a fickle suitor in this early marriage of what I call "Evolutionary Epidemiology." As I recall, we agreed that mood and anxiety disorders are more likely candidates for this line of reasoning today.

Also, John mentioned his old readings on variation in populations. This, as I said, is an area of great relevance to those of us keen on seeing clinical insights and practice derive from evolutionary theory. Still, it seems most experts in basic population genetics regard balanced polymorphisms, or heterosis, as the main cause of significant population variation, at least as seen in nature. I think this is where most of the action is, since frequency dependent selection is thought to be more unusual. I can, of course, readily appreciate the relevance of frequency dependent selection as it applies to John's signal work in alternative strategy theory.

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He/She Used To Be So Attractive

by John K Pearce

Do men and women, after five years or so of monogamy, tend to get rid of their spouses by becoming progressively critical and shutting down sexually? It is possible.

Helen Fisher has long argued that women's genetic best interests have, on an evolutionary time scale, been served by serial monogamy with marriages lasting about five years. She cites data on divorce in cultures without fixed property; cultures where divorce does not mean loss of resources. Most divorces occur after five years. Her scenario is: passionate love, mating, birth of a child, cooling love, and divorce when the child is big enough to travel fast. This is not inevitable. Some married people shift from passion to attachment and do not part.

Fisher's view has found indirect support in the findings of Baker and Bellis, behavioral biologists who publish in the journal Animal Behavior.[18] They have demonstrated physiological mechanisms that increase the likelihood that extra-pair matings will produce offspring compared to monogamous matings. These findings are similar to those found in other apparently monogamous species. About a quarter of offspring in birds and humans (in some hospitals) have been found by blood typing or DNA studies to have different fathers than the monogamous father. Usually, the father in birds exhibits higher social status.

Baker and Bellis ask no questions about the emotional state of their experimental subjects, but I think it is reasonable to argue that in humans, infidelity is more likely to

occur when the bloom has gone off the rose; when a couple is no longer madly in love, do they find fault in each other? Are they quick to do so?

Decisions made by all animals involve information processing. Special modules of the brain are thought to have evolved to make assessments of opportunities. Furthermore, they are good at it. In general, the repeated finding of experimental ethology is that animals are effective in choosing to act in their own best interests to promote inclusive fitness.

We know information processing mechanism are affected by moods and goals. Clinical psychological testing takes for granted the interconnections between motivation and cognition. Parenthetically, recently psychologists have been impressed by the apparently counter-intuitive finding that depressed people are more "realistic" than non-depressed people. They notice more unfavorable features in the environment. This should not be suprising: if cognition is regularly flavored by current goals and affects, then ignoring bad things is, within limits, a good idea—at least if you are out to get the good things in life. The limit of usefulness for a cheerful cognitive show is when the habitat is so depleted that good things simply can't be found. Then it is sensible to shift to noticing how bad everything is.

Putting all this together, it is reasonable to suspect that the ultimate goal of finding new genetic partners may be served by the proximate mechanism of finding fault and diminished sexual interest—after
a; five years of monogamy. About that time, he's just not looking as
good to her as he used to. She mates witn reluctance. He does much the same thing, and they divorce. Does it matter if they have had a child or not? Probably not.

Of course, divorce is not required for maintaining genetic diversity. Take an alternative scenario: He is a good provider and good company. She wants to maintain the marriage, so she deceived him, mating with a man who looks especially good to her. As we have learned from Baker and Bellis, mechanisms intrinsic to reproductive physiology help to insure that she will get a baby by her extra pair mating. Her husband is unlikely to be willing to expend his resources on somebody else's baby; hopefully, the baby will look something like him.

Of course, illegitimacy is not inevitable. The third scenario is that spouses value each other, resist sexual boredom and keep it all together.

If serial monogamy is wired in, why does everyone get so upset. The answer is, of course, that the "whispering within" that prompts genetic diversity is only one voice among many. Attachment occurs regardless of the quality of marital relationship; the loss of an impossible person hurts. In our society, divorce means loss of resources. Both men and women lose big.

Women protest that they are the vulnerable ones; they are the ones who are left. Why? It is a matter of resources; in cultures like ours, where men make most of the money, loss of a husband is likely to be more of a financial disaster for the wife than the husband. In hunting and gathering bands, where resources are gathered daily and women tend to be more effective food gatherers than men, changing partners is upsetting but involves no serious loss of resources; the women do not see themselves as victims. (Another element in this story is women's habit of using submission and weakness as a way of asking for resources from men, and for sympathy from other women.)

Everyone knows that it takes work to make a marriage, and, no doubt, many forces contribute to the breakup

of monogamous relations after about five years, but reproductive biology may also make our lives more difficult. The demonstration that the female reproductive system favors illegitimacy makes it plausible that a similar shift may occur in information processing about the value of the spouse. In time, in marriage, spouses are undervalued; sex wanes.

It is not obvious how these ideas can be put to a test. One possible way would be to compare sexual satisfaction and general valuation of spouses in upwardly mobile families, compared with families that are neither upwardly, nor downwardly mobile. Upwardly mobile families would be more likely to express satisfaction. Furthermore, the partner, husband or wife, who is relatively most upwardly mobile (for example, a working class girl who marries a man who makes a lot of money) would be expected to be the most satisfied. Problem is, even if the results were as expected they could be explained by the importance of resources alone, although it may be the case that no one has demonstrated the power of resources to preserve marriage. In fact, riches can make it easier to divorce.

The therapeutic implications of this biology influenced scenario are benign: after the first couple of years, monogamous couples who do not wish to follow their preferred biological pattern of serial monogamy had better try hard to value each other, please each other, and make the most of their sex life. Nature is not going to make it easy for them.

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Quote: Asfaw B, Beyene Y, Suwa G, Walter RC, White TD, WoldeGabriel G, Yemane T: The earliest Acheulean from Konso-Gardula. Nature 1992;733-735.

Despite repeated claims to the contrary, well dated Eurasian evidence of occupation is unknown before -1.0-1.4 Myr. The preponderance of evidence is that H. erectus made its evolutionary appearance in Africa sometime before 1.7 Myr and dispersed into Eurasia within the next 0.3-0.5 Myr...

The last decade witnessed...attempts to causally link hominid evolutionary events to changes in global climate...Vrba suggests that "...changes to open and arid conditions may have triggered the origin of H. erectus and of his characteristic tool kit...[11], and that "the period around 0.9 Myr may also coinicide with the earliest massive geographic expansion of any hominid species, namely, H. erectus."...The first African records of this taxon and the Acheulean, however, are now set solidly at 1.7 and 1.4 Myr. These appearances substantially post-date the global cooling between 2.4 and 2.8 Myr. The probable dispersal of H. erectus and the Acheulean from Africa into Eurasia substantially predates pronounced changes in global ice budget between 0.9 and 0.7 Myr. The period between 1.8 and 1.4 Myr was unmarked by major global climatic changes, but witnessed profound changes in hominid anatomy and technology.

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Abstract: Pellis SM, McKenna MM: Intrinsic and extrinsic influences on play fighting in rats: effects of dominance, partner's playfulness, temperament and neonatal exposure to testosterone proprionate. Behavioural Brain Research 1992;50:135-145.

Play fighting is a frequent activity of juvenile rats and appears to show marked variability amongst individuals in that some rats play a great deal and others very little. This study attempted to identify some of the factors involved in producing this individual variability. The major influence over an individual's frequency of play as a juvenile was found to be the frequency of play by the partner. That is, play appears to be contagious, in that a high playing animal stimulates its partner to play frequently as well. In male juveniles, but seemingly not in female juveniles, the subsequent adult status of one partner as dominant influences the subordinate-to-be to initiate more playful contacts. Higher players tend to be more susceptible to the stereotypy-inducing effects of the dopamine agonist, apomorphine, and tend to be more dependent upon the playful activity of the partner to maintain their own high levels of play. Both of these characteristics are consistent with other studies comparing bold and timid rats. Boldness, however.

only seems to influence how much play a rat will exhibit, not how much play it is capable of exhibiting. Neonatal testosterone augmentation increases juvenile play fighting but not apomorphine susceptibility, suggesting that a high player need not be a bold animal. The total frequency of play an individual is capable of initiating appears to depend upon perinatal exposure to androgen. Boldness and the playfulness of the partner appear to modulate the expression of this hormonally set value.

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Abstract: Sorensen PW: Hormonally derived sex pheromones in goldfish: a model for understanding the evolution of sex pheromone systems in fish. Biol Bull 1992;183:173-177.

It is now well established that female goldfish release unmodified and metabolized sex hormones to the water and that some of these compounds function as potent sex pheromones detected by the male's olfactory sense. In goldfish, both olfactory pheromonal receptors and their corresponding hormonal receptors coupled with G proteins. Recent studies of teleost fish indicate that fish commonly use 'hormonal-pheromones. ' Taken together, these data suggest that fish pheromone systems may have evolved as a consequence of a chance expression of hormone receptor molecules on olfactory receptor cells. Isolation and identification of olfactory and hormonal receptors may be the next step in resolving this question.

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Abstract: Berthold P, Helbig AJ, Mohr G, Querner U: Rapid microevolution of migratory behaviour in a wild bird species. Nature 1992;360:6688-670.

The Blackcap, Sylvia atricapilla, a widespread Paleartic migratory bird, rarely wintered in Britain until the 1950s. The winter population Has since increased to several thousand birds. Ringing indicates that these are not British blackcaps orestalling migration, but birds breeding in Continental Europe reaching Britain on a novel westerly migration route. The proportion of northwestern migrants among Blackcaps ringed in parts of Germany and Austria has increased from 0% before 1960 to 7-11%. We bred British wintering Blackcaps in captivity and determined the migratory direction of their offspring. Here we report that these birds migrate west-northwest in autumn, a direction genetically distinct from the British breeding population of west-central Europe. The novel route must have developed within the past 30 years with selection favouring birds wintering some 1,500 km further north than most of of their conspecifics. To our knowledge, this is the first case in any vertebrate in which a drastic and recent evolutionary change of behaviour has been documented and its genetic basis established.

(This remarkable finding is discussed in a *News and Views* section of the 17 Dec issue by WJ Sutherland, p.??)

[T]he blackcap has undergone dramatic [behaviour] changes...and, moreover, these changes have a genetic basis...For such a rapid change to take place, selection pressure must have been strong.

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Abstract: Shapiro JA: Natural genetic engineering in evolution. Genetica 1992;86:99-111.

The results of molecular genetics have frequently been difficult to explain by conventional evolutionary theory. New findings about the genetic conservation of protein structure and function across very broad taxonomic boundaries, the mosaic structure of genomes and genetic loci, and the molecular mechanisms of genetic change an old point of a view of evolution as involving the rearrangement of basic genetic motifs. A more detailed examination of how living cells restructure their genomes reveals a wide variety of sophisticated biochemical systems responsive to elaborate regulatory network. In some cases, we know that cells are able to accomplish extensive genome reorganization within one or a few cell generations. The emergence of bacterial antibiotic resistance is a contemporary example of evolutionary change; molecular analysis of this phenomenon has shown that it occurs by the addition and rearrangement of resistance determinants and genetic mobility systems rather than by gradual modification of preexisting cellular genomes. In addition, bacteria and other organisms have intricate repair systems to prevent genetic change by sporadic physicochemical damage or errors of replication machinery. In their ensemble, these results show that living cells have (and use) the biochemical apparatus to evolve by a genetic engineering process. Future research will reveal how well the regulatory systems integrate genomic change into basic life processes during evolution.

1. Nash 0: The Anniversary. In The Private Dining Room And Other New Verses. Boston: Little Brown, 1949, 1950, 1951, 1952, 1953, p.9.

2. c/o R Gardner, 1.200 Graves Building (D29), University of Texas Medical Branch, Galveston, TX 77555-0429 FAX: 409-772-4288. For ASCAP Newsletter Volumes 3 (Jan through Dec, 1990), 4 (same months, 1991), and 5 (same months, 1992), please send $18 (or equivalent) for each 12 issue set. The first two volumes (1988 and 1989) of thirteen and twelve issues respectively are available on request without cost. For subscription to the 1993 set of 12 issues (Volume 6), the cost is $20/year. Make checks or money orders out to "Department of Psychiatry and Behavioral Sciences, UTMB."

3. EXECUTIVE COUNCIL:
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**Past-President: Michael R A Chance**

**At this time this "informal" organization has no official budget.**

**4. Hunter KM: Doctor's Stories: The Narrative Structure of Medical Knowledge. Princeton, N.J.: Princeton U Press, 1991, p.21.**

**5. Sacks 0: The Man Who Mistook His Wife for a Hat and Other Clinical Tales. NY: HarperPerennial, 1987, p.vii.**

**6. Compare to Birtchnell's distance dimension. ASCAP Newsletter 1991;4(9) September issue. Brain mechanisms illustrated by recent findings in the imaging literature in psychiatry were reviewed in last issue (ASCAP Newsletter 1992;5(12) December issue.**

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c. Baker RR, Bellis MA: Human sperm competition: ejaculate manipulation by females and a function for the human orgasm. Animal Behavior 1993;45: in press

b. Baker RR, Bellis MA: Human sperm competition: ejaculate adjustment by males and the function of masturbation. Animal Behavior 1993;45: in press