# ASCAP NEWSLETTER

**Across-Species Comparisons And Psychiatry Newsletter**

Volume 4, No. 3, 15 March 1991

"... [T]op-down strategies (as characteristic of philosophy, cognitive psychology and artificial intelligence research) and bottom-up strategies (as characteristic of the neurosciences) for solving the mysteries of mind-brain function should not be pursued in icy isolation from one another." Churchland, 1986[1]

(c/o Russell Gardner, 1.200 Graves Building (D29), University of Texas Medical Branch, Galveston, TX 77550)[2]

For the philosophy guiding this newsletter, predicated upon combinations of top-down and bottom-up analyses, see footnote on p11[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.



Also in Letters, Michael Chance and Paul Gilbert provide ASCAP a challenge (p2), Grethe Sorensen and Axel Randrup supply help with the sociobiology chapter requested of me (p3), and Glenn Weisfeld offers ASCAP an invitation to contribute to the Human Ethology Newsletter, which submission is here replicated (p4).

Renewed Announcement 1 We reiterate the plans for the two day London-Sussex meeting on July 7 & 8, 1991, of the fourth meeting of the -Basic Plan Group, but the first with John Price. For those planning to come who have not yet touched base, please let me know. This is the meeting in which we make definitive plans in a brainstorming but goal directed manner integrating past meetings with present considerations about hypotheses and data needed to test them.

Call for abstracts for Human Behavior and Evolution Society.

Abstracts for the third annual meeting to held at McMaster University, Hamilton, Ontario, CANADA, hosted by Margo Wilson and Martin Daly , are due April 1, 1991. Send abstracts to the Program Co-Chairs Nancy Thornhill and Randy Thornhill, Dept. Biology, U New Mexico, Albuquerque NM 87131.

Use a format suitable for publication: 4x6 inch area; title on line 1, author(s) on line 2, institutional affiliation and address on lines 3-5. Abstracts are considered for both lecture and poster formats: state your preference.

Comment: Jurgen Brosius's discussion in Science of retroposons as seeds of evolution commands the interest to those of us invested in combining bottom-up and top-down analyses of the biology of behavior (see below (Chance/Gilbert letter) discussion of the meanings of these terms. He notes that so-called non-functional genes in the mammalian genome may arise by a process called retroposition. Retroposons are stretches of DNA that arose from RNA sequences that got reverse transcribed. Usually DNA gets transcribed to RNA and RNA gets translated into proteins (Crick's dogma), but obviously cellular machinery has some exceptions to Crick's dogma. These retroposons have been known for several years and have distinctive hallmarks, including lack of introns (non-coding parts of genes).

Retroposons can act as "insertional mutagens with detrimental or beneficial effects, especially when an ac-

tive gene is targeted. . . .Apart from the ability of retroposons to keep the genome in flux, thus favoring genetic diversity, they can be considered a shotgun approach of nature wherein the majority of these genetic elements are inactive and left to rot in the genomic soil. Nevertheless, some seeds will integrate near a fertile genomic environment, giving rise (usually after mutational alterations) to new genes or gene domains and complementing the conventional gene duplication that is essential to evolution. Retroposition may also match existing genes with new regulatory elements."

Brosius lists molecular evidence for expressed retroposons. He notes that "In higher eukaryotes there are a number of intronless genes, including members of the potassium channel family and the ever-growing list of G protein-linked receptors. It is conceivable that evolutionary diversity in vertebrate nervous systems, for example, was accomplished, in part, by retroposition."

Letter *17.1.91*

*Item 3 on the back page of issue 12 for 1990 states that "Both top-down and bottom-up analyses are needed" so that new ways of viewing psychological and behavioural phenomena are obtained, but nowhere does it direct us towards defining the healthy personality in the present world or toward an awareness that this personality is rapidly evolving itself today towards an expanded awareness of reality. This phenomenon easily visible through the media (at least in the U.K.) is perhaps the one prominent adaptive feature of humanity today. So, much more of our attention should be paid to defining this feature as the top from which to work down.*

*An awareness of reality both personal and of the world outside ourselves is the single feature seen by many as the common feature of a healthy mind. Here is a list of them.*

*1. Paul D MacLean in the Triune Brain in Evolution. London: Plenum Press, 1990.*

*2. G.E. Vaillant in Adaptation to Life. Boston: Little Brown, 1977, who now adds The Joyful Expression of Sex and Anger.*

*3. William Glasser in Reality Therapy. N.Y.: Harper and* Row, *1965.*

*What 2 and 3 lack is setting this awareness reality in an evolutionary context. We do not think it is yet possible to do this fully without finding some intermediate links which are as yet to be found. This only emphasises that we need to concentrate our attention on the top layers of personality and get guidance from those who have already tackled aspects of the problem of defining these top layers of organization: such as those already quoted and Paul Gilbert in Human Nature and Suffering, L.E.A., Hove U.K. and Hillsdale N.J. and soon to be published by L.E.A. Depression: an Evolutionary Synthesis.*

*Put briefly, I think ASCAP is becoming dominated by too undiluted a concern with the pathological which leads to a distorted bottom-up perspective.*

*Finally, we think we should accumulate accepted definitions of top layer features (the bottom layers are already largely established in sociobiology and other long established biological dimensions). These should be listed in a supplement published yearly or half-yearly.*

*As a start, we will be sending a rigourous definition of the two mental systems and corresponding social modes. The Mental Systems defined by Paul Gilbert are 'The Self-defensive protective mental system' which corresponds to the 'Agonic social mode' and the 'Safe mental system' which corresponds to the 'Hedonic social*

*mode.'*

*Please would others offer their contributions so that we can build up an adequate descriptive framework defining the core features of the healthy mind.*

*MRA Chance & P Gilbert, Birmingham and Derby, England.*

This vigorous challenge represents the kind of debate for which ASCAP was designed. Are there opponents and/or sympathetic hearers of this Chance-Gilbert message? Are C&G right about ASCAP being "...dominated by too undiluted a concern with the pathological..."?

We look forward to expansion of the definition of Paul Gilbert's definitions of the self-defensive protection system and safe mental system mentioned! These concepts are, to my thinking, "top-down" analytic approaches that approach research on the organism's behavior from how postulated "systems" affect the whole organism's behavior.

However, "bottom-up" analyses are not be illustrated by sociobiology's "already established" "bottom layers" but rather with Churchland's lead quote for this issue or the Comment section's focus on "retroposons" — a molecular or cellular view of the parts of a whole are the focus of the researcher, rather than from the whole to the parts. Churchland herself goes on to say that she "...aims to bestir a yen for the enrichment and excitement to be had by an interanimation of...top-down and bottom-up research."

To pursue the point a bit further, genetic diseases like Huntington's disease and cystic fibrosis have represented paradigm cases of modern clinical genetics. Top-down analyses start with the disease and proceed down, asking how cellular-mechanisms work to affect the whole. Another approach is to explain these diseases at the molecular level, to go from the bottom up, from the genes to the defective phenotype. Bottom up or reverse genetics involves identifying the stretch of DNA that segregates with the condition through affected families; zeroing in on the gene(s) involved; identifying the protein(s) coded for; and finally elucidating the role of the protein(s) in normal metabolism[6].

This was the procedure used in the inherited eye disease, choroideraemia, in which a "candidate gene" was used via known DNA probes from the affected chromosomal site[7].

Letters (cont.): *91/02/01*

*Thank you for ASCAP January. We are pleased to learn about your chapter in the Comprehensive Textbook. It will surely help to establish ethological psychiatry. Here are some suggestions.*

*Abnormal behavior is only exceptionally reported in wild-living animals, whereas it is abundant in animals confined in cages or stables. Is it possible that with respect to psychiatry, humans are more comparable to caged animals than to wild-living ones? Or that abnormal behaviour does occur in nature but is only exceptionally observed?*

*In ASCAP (1990;May:9, and 1990;3: Sept:5), we have cited evidence that unfavourable living conditions (social and physical) can cause psycho- or neurogenic death in animals and man; there is also evidence indicating that abnormal behaviours, particularly stereotypies, may (at least in some cases) have stress-relieving effects and function as a defense against lethal milieu effects*[9].

*These may be loose ends, but we think that the idea of defense against lethal effects is well founded in general and evolutionary biology as well as in systems science and that it may have important im-*

*plications for psychiatry (etiology, therapy).*

*Extremely unfavourable living conditions and death occur of course frequently in nature as well as in cage life and in human life, eg, in connection with competition, want, and overpopulation .*

*References...enclosed.*

*We wish you good luck with the writing of the chapter.*

*Grethe Sorenson & A Randrup*
*Roskilde, Denmark*

I'm much obliged for your quick and gracious help. I am reading carefully these useful references.

Letters (cont.): *7-2-91*

*...[I've sent an ASCAP letter in response to J. Price].*

*I have been invited to run a 4 day course on evolutionary approaches to psychopathology at the University of Freiburg (Switzerland) in June...it's all exciting.*

*Congratulations on the chapter you have been asked to write....*

*Paul Gilbert, Derby, England*

Your letter goes in next issue. You too are to be congratulated for the honor in Switzerland. I expect to hear about it at the July meeting! Contacts made in Switzerland should help in the research planning.

Letters (cont.): *Feb. 11, 1991*

*...I'll mention the [ASCAP] newsletter in the Human Ethology Newsletter [HEN]; my first issue is March. ...Feel free to send me any other information on ASCAP such as convention news.*

*Glenn Weisfeld, Editor, HEN*

For those of you who do not receive the Human Ethology Newsletter, the following is what I submitted for possible inclusion:

The Across-Species Comparisons and Psychiatry (ASCAP) Newsletter is an informal monthly publication now in its fourth volume issued by Russell Gardner, Jr., M.D., an academic psychiatrist from University of Texas Medical Branch (UTMB), Galveston, TX 77550-2774, Phone:(409) 761-3474, FAX:(409) 761-4288. Gardner was interested in how evolutionary mechanisms might have resulted in body processes that go awry in psychiatric disorders. He corresponded with others similarly curious when a new word processor provided the opening for a newsletter. He sent a trial balloon to these colleagues to see if they desired a newsletter. They did; the project has continued, gaining friends. Funded first by UTMB, subscribers have paid $18 per year for the 12 issues of Volume 3 and 4 (calendar years 1990 and 1991).

ASCAP's philosophy and goals states that high scientific importance rests on comparing animal behaviors across-species to understand better human behavior, knowing as we do so that evolutionary factors must be considered for understanding properly such behaviors. To accomplish these comparisons, different ways of viewing psychological and behavioral phenomena as biological are required. We expect that work in natural history biology combined with cellular-molecular wet lab research will emerge as a comprehensive biologic basic science of psychiatry. Both top-down and bottom-up analyses are needed. Indeed, this must happen if we are to explain psychiatric illnesses as deviations from normal processes, something not done now. Compare this approach to pathogenesis in psychiatry to pathogenetic formulations in diseases of internal medicine.

The aims as stated at the onset are: 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; and 4. Proposals for new initiatives, joint research endeavors, etc.

All of the above four aims have been realized with interest from investigators, clinicians and others from around the world: England, Canada, Denmark, Belgium, South Africa, Germany, Australia, Italy, Crimea, Hungary, Switzerland, Russia, Mexico, some briefly curious, some as continuing subscribers. Book excerpts and summaries have been published, along with citations of new articles,

many essays and responses to others' essays, abstracts from relevant published articles as in Science and Nature. Published reactions to the Human Behavior and Evolution society annual meetings have elicited some spirited exchanges. Disciplines have included psychiatrists, ethologists, psychologists, physiologists, sociologists, engineers, historians, philosophers, psychoanalysts, anthropologists, former patients and still others not formally and specifically educated but interested and knowledgeable. Topics have ranged from sociobiological interpretations of psychoanalytic theory to how spinal fluid from patients with psychiatric illnesses impacts socially interactive rats when injected into them.

Often "basic plans" are at issue: these are biologically driven neuronal programs that emanated from remote ancestors to whole varieties of descendants, such as the yielding response discussed at length by John Price who saw the lowest ranking chicken in the peck order as exhibiting an ancient survival response that is also exhibited by the depressed patient. Gardner himself has been interested in mania as an example of a human-flavored ancient program that has a normal human counterpart in charismatic leadership and an animal counterpart in alpha dominance.

There are connecting links to the Human Ethology Newsletter. Ethologists who have observed behaving patients have integrated their information with those who work clinically or conceptually. An important ethologist and regular contributor, Michael Chance, has been interested in the "atmosphere" of primate groups, such as friendly, playful, cooperative (hedonic) vs competitive, tense, authoritative (agonic).

We hope to implement Dr. Chance's hedonic atmosphere in the ASCAP Newsletter; friendliness, playfulness, not uncritical, but with a long distance give-and-take that is overall fun. "Work is play for mortal stakes" said poet Frost and we hope that our play is groundwork for data collection. Certainly there have been other outcomes: a publisher is interested in the collected materials and Gardner has been asked to write the next chapter on sociobiology as it applies to psychiatry for the sixth edition of The Comprehensive Textbook of Psychiatry.

But collaborative research remains a most important aim: a basic plan working group convened during 1990 in Boston, New York, and Los Angeles with an expectation of a fourth meeting near London on July 7 and 8, 1991, with Dr. Price for the first time able to come. This will be a two day, open-format session in which we hope to work out research data-gathering plans that would involve several nations with an emphasis, at this point, upon collecting data on the hypothesized overlap between the psychiatric disorder of depression on the one hand and the ethologically appreciated concept of defeat on the other. Contact Russell Gardner if interested either in the ASCAP Newsletter or in the London planning meeting.

## Zhdanova-Gardner-Zhdanova exchange

[Previous contributions bearing on this exchange with Irina Zhdanova from Budapest, Hungary & Leningrad, USSR, were in the Nov 1989, May 1990, and Aug 1990 issues of ASCAP (Vol2#ll, Vol 3#5, Vol 3#8). To find the questions that Dr Zhdanova answers, the reader may wish to refer again to the Aug 1990 issue.]

I have just now (11/6/90) received ASCAP #8 (Aug issue) with your discussion of my essay. It was very interesting for me to read your opinion. Now I'll try to answer your questions about method.

First of all, the injections were done to animals that lived constantly in the group. There were no injections into strange rats. The only reason to put the strange rat into the group was to provocate the agonic situation, which then gives additional information about social relations in the group and social ranks. This provocation was done after 4-7 days of observation of rats' relations in the group. A strange rat was in the group during 0.5-1 hour and then it taken out of the cage.

The CSF injections were done to the dominants or subordinates of the constant group.

As for diagnoses, they were made by doctors in the hospital, using Russian standardized interviews, supplemented by hospital records and information from other family members. Unfortunately we have not got the inter

Table 1: Rat behavior in the experiment 'emotional resonance' before and after the injection of experimental fluids.

| Group No. | Behavior | Before injection | After the injection of CSFm | CSFd | CSFh | CSFr |
|---|---|---|---|---|---|---|
| 1 | Time in dark, means/s.d. (seconds) | 0 | 0 | 17.33+4.50 | 0 | 0 |
| | Transfers | 0 | 0 | 1.50+0.41 | 0 | 0 |
| 2 | Time in dark, means/s.d. (seconds) | 24.57+2.66 | 0 | 244.79+77.43 | 0 | 27.32+1.12 |
| | Transfers | 2.24+0.16 | 0 | 2.71±0.16 | 0 | 2.45+0.15 |
| 3 | Time in dark, means/s.d. (seconds) | 251.74+3.18 | 0 | 294.77+1.80 | 29.5+2.72 | 249.72+1.18 |
| | Transfers | 2.91+0.13 | 0 | 1 | 2.0+0.37 | 2.54+1.22 |
| 4 | Time in dark, means/s.d. (seconds) | 295.08+0.24 | 0 | 300+0.16 | 287.50+5.53 | 296.12+1.71 |
| | Transfers | 1 | 0 | 1 | 1 | 1 |

Table 2: Number of animals with different effect of CSF injection.

| | Respected | | Effect of injection Contrary | | No Effect | |
|---|---|---|---|---|---|---|
| Social rank | Domin. | Subm. | Domin. | Subm. | Domin. | Subm. |
| CSF,m (bipolar) | 7 | 4 | | | 1 | — |
| CSF,m (schizoaff) | - | - | 1 | 3 | 2 | - |
| CSF,d (bipolar) | 11 | 4 | - | - | 1 | 2 |
| CSF,d (schizoaff) | 3 | 5 | - | - | - | - |

national standardized interviews with which to compare them because scientific and especially psychiatric information is difficult to obtain in the Soviet Union.

The diagnoses of schizoaffective disorder was made when the patient with affective symptoms had got 1-2 schizophrenic symptoms of first rank according to Schneider.

We also distinguish "mixed" affective states, but we did not take CSF from such patients.

We used CSF of 13 patients with bipolar disorder (6 when manic [CSFm,bp]) and 7 when depressed [CSFd,bp]) and 6 patients with schizoaffective disorder (7 when manic [CSFm, sa] and 2 when depressed [CSFd,sa]).

As for false positives in controls, there were no such pronounced changes in rat behavior from the injection of control CSF as from patients' CSF, but there was a difference between the effect of intact rats' CSF and CSF of healthy humans. The injection of human CSF produced a mild "manic-like" effect--in the "emotional resonance" experiment, rats spent more time in the light compartment after the injection than before it.

Table 1 illustrates these results and answers questions about statistics.

In the socio-hierarchical experiment, the CSF of 11 patients, 6 bipolar and 5 schizoaffective, was used: 4CSFm,bp; 2CSFm,sa; 3 CSFd,bp; and 2 CSFd,sa.

The result of this bioassay is seen in Table 2.

The behavioral effect was not similarly strong in various animals. It depended on their hierarchical status in the group and on patients' CSF. In some cases the effect was considered as positive when the animal became more or less active in its movement, in others, when its sexual activity was changed or the time that it spent in a social contact became bigger or smaller. In more pronounced cases there were changes in the hierarchical structure, but it was only as a result of injection of CSF of depressive patients. CSF of different patients (manic and depressive) had different "strengths" in its influence on rat behavior.

I regret that my first explanation was incomplete and hope that now it will be more understandable.

As for the basic plan of the social interactions and of hedonic and agonic mood of the social contacts, I suppose they had their beginnings on a very low level of evolution. Even in some of the most primitive creatures, for example Dictiostelium Discoideum, we can observe such behavior, which can be understood as social and even hedonic. Such models, though of course, they are very far from the mammals' behavior, can facilitate the understanding of such fundamental ideas as RHP, giving the possibility to investigate level by level its molecular basis. That's why I think that the evolutionary approach to the problem of normal and pathological social communication is very fruitful.

Price-Wylie Exchange by John Wylie

The issue as to whether or not a depressed person is really sick and worthy of all the nurturant benefits which accrue to a medically ill person is one which effects every mentally ill patient. Psychoanalysis has always been suspect because not only does the theory postulate domination by an inner, infantile homunculus, which has never "grown up", but the treatment seems to submit to the infant's most regressive desire: unlimited nurturant attention. Although John Price's theory clearly has the ring of truth, it lends credence to the idea that mental patients "just want attention", and that depression, among other mental illnesses, might be the result of an "arms race" between submissive individuals who want attention and dominant individuals who have evolved sensitivities to separate the medical wheat from the chaff. In fact, just that empathetic "separating" skill is crucial to the modern psychiatrist in trying to ferret out which depression are psychological in nature and which are psychophysiological and, therefore, will respond to antidepressants. This skill could be based on a constitutional ability to "feel" whether the patient is being subtly manipulative, or whether the illness has "broken away" from the object world and exists as a "closed loop" process wholly within the sufferer.

My own theory as to the mechanism of transferral from the manipulative, psychological state to the pathophysiological state is that, at a certain level of intensity in genetically predisposed patients, the interaction shifts from the hedonic real-world object to the patient's own internal dominance agency: the superego. For reasons too complex to include here, the superego can only interact with the patient in the agonic mode so that a pathological "closed loop" feedback circuit is estab-

lished: submissive self asserting depressive need; the superego responding aggressively to this assertion, which stimulates more depressive symptoms and so on. The submissive self is stuck in the hedonic mode, the superego in the agonic, both reinforcing each other resulting in a psychotic vicious cycle of self hatred and depressive symptoms. Observed biological changes could be a RESULT of the biochemical stress from such a massive "revving" of the neurological hardware.

Obviously, John Price's hope that his hypothesis has heuristic value is, for me, fulfilled.

Coursin-Price Exchange by D Coursin

I have wanted to respond with some attention and thoughtfulness, but have had difficulty with Price's paper. It has not been sinking in very easily and it is hard for me to tell if that comes from my relative inexperience in the thinking it expresses, the late evening fatigue that seems to set in just as I have time to devote to it, or the quality of his ideas and presentation.

The paper leaves me curious about a number of things. I like the general notion that a pathological situation like depression may be an understandable consequence of deeper mechanisms that have served well as survival strategies. Is this the "psalic theory" he refers to? Traditional analytic notions of depression as "impacted rage" would fit nicely with the proposal that "the depressive reaction evolved as an involuntary yielding component..." I have certainly seen dogs spar a few rounds with a snarling intensity that seemed surely to qualify as "murderous rage" only to have one of the two suddenly turn belly-up with its jugular exposed. That aggressive intensity went somewhere, was forestalled or impacted in some way.

It also seems that the psychodynamic notion of depression as "learned helplessness" would be consistent with the circumstance he describes when "submission and escape are blocked or do not work, so that a losing individual continues to receive punishment..." As I understand learned helplessness it arises when the animal sees that it is going to suffer and is helpless to stop it as its usual mechanisms of protection do not work.

I disagree with the contention that "rank order between two human beings is seldom determined by ritual agonistic behavior." It seems to me that a great deal of what we engage in daily is organized by just such concerns, however subterranean they may be. It could be argued that the great success of empires such as the Roman and the British arose, in part, out of their ability to justify vicious and cruel behaviors of oppression and dominance under the rubric of "law." By and large, those same laws choreograph the ritual, agonistic dances of the present day court. I have seen serious clinical depressions clearly arising from loss in the agonistic competition of the marketplace: being passed over for promotions, being laid off, losing a contract bid, and the like. Further, *I* wonder about the Japanese ritualization of suicide as a response to a loss of face.

I think Fenichel is right as quoted in recognizing the ways that depressed individuals can dominate their worlds. I think that in certain depressive neuroses, or dysthymias, the depression has become a major transactional device for self-protection and power. However, I do also think that a significant percentage of depressed patients are suffering a condition of hopelessness and despair that is tormenting to the point of outweighing any conceivable secondary gain. I think these in-

dividuals are no longer able to experience human connection and have lost all sense of the impact of their actions on others.

I know that many therapists view suicide as an aggressive act that is directed at someone and I believe that to be true at times, but not at all times. Those who are severely depressed are drawn to suicide because it is the only respite they can see for a situation so isolating as to be terrifying in a way I can barely conceive of. Concern about getting one's own way would be totally irrelevant to someone in this dire strait. The dominating power of such a patient over us comes precisely from the impact on us of his total disconnection from the palpable concerns of relationship and hierarchy. I don't think it is a power that such a patient wants anymore than we want to be in its grip.

As I said above, I am attracted by the idea that a pathological condition like depression arises because valuable sub-routines like yielding become dominant in a dysfunctional way in an individual's repertoire. However, I don't think that the more severe forms of depression can be explained by positing an over-abundance of yielding behaviors. This is where the issue of self-referential thinking comes into play. I think that depressions become severe because they involved a pathological excess of self-referential perception.

Self-referential may well be another example of a valuable cognitive behavior that contributes to survival when functioning normally, but creates pathology when it is over-activated or under-activated. When overactive, the patient loses connection with a normal framework for understanding inter-personal cause-and-effect and becomes increasingly convinced that he or she is the cause, that events have directly to do with his or her behavior. When under-activated the patient loses all concept of any connection between himself and others, nothing references back to the self. The former may be the source of Fenichel's observation that depressed patients are "perpetually taken offense and behaving as if they had been treated with great injustice and of Price's observations about grandiose delusions. The latter may be the source of the nihilistic delusions of severe depression.

I am still unclear about Price's ideas about the sickness metaphor, illness as submission and the overlay of agonistic and hedonic goals. I reconnect with him in the last paragraph and agree with his idea about neurochemical changes seen in monkey ranking behavior.

I don't know if this speaks to your goal, but I enjoyed the opportunity. I think it was Churchill who said, "Pardon the length of this missive. If I'd had more time it would have been shorter."

Price-Erickson Exchange

As time passes and there is more opportunity to think about the "Yielding Hypothesis" I find myself becoming more intrigued. Below are some thoughts regarding Price's July (1990) article in ASCAP and the hypothesis is general.

Regarding Price's article, it seems to me that there may be a somewhat different way to think about the adaptiveness of submissive and demanding depressive behaviors. In natural settings a subdominant (depressed) individual might maximize its own fitness by adapting distinctly different behavioral strategies relative to the genetic relatedness of an interactant. If, for example, submissive or yielding depressive behavior decreases the likelihood of aggression by a conspecific then submission might be expected as the preferred

strategy when non-kin are encountered; this because dominant non-kin could well benefit from further weakening a potential future competitor. In contrast, nonyielding or demanding depressive behavior might be used to elicit guilt induced altruistic responses. This strategy would, theoretically, be expected to work only when encountering close kin who may benefit from helping a subdominant relative survive until more favorable circumstances exist. In contemporary society these strategies might be played out quite differently. I recall working on inpatient units in which depressed patients were often submissive to physicians who were clearly dominant; the same patients were often demanding of nursing staff who had little recourse to this behavior other than to comply to the patients wishes.

So, I would tend to refocus the argument when Price writes, "we would predict that depressed (nonyielding individuals) would...get their way as much...as anyone else...when the group is oriented toward nonagonistic behavior..."(p6). Instead, I would suggest that a depressive strategy would be most powerfully influenced by the relatedness of an interactant (e.g. one's mother vs. a distantly related dominant male) rather than the affective state of the group (agonic vs. hedonic) at a particular time. With one's mother, demanding or nonyielding depression might frequently payoff with food, etc.; and given that mothers are not likely to attack offspring, submissive behavior probably makes little sense in this context. In contrast, in the presence of an unrelated dominant male, attack is more likely, hence submission or yielding depression would be the strategic response whereas demanding depressive behavior would seen to make attack all the more likely.

In a completely different vein I have wondered if anyone has suggested a link between the "learned helplessness" model and the "yielding" hypotheses of depression. It may be that learned helplessness is a specially evolved type of learning which initiates depressive yielding. If, for example, an animal has learned through repeated trial that it is almost certain to be defeated, learned helplessness may trigger depression and the depressive strategy of yielding. Here I think it is interesting to note that depression has, in our species, a natural history of about six months. If the function of depression is to prolong the life of a subdominant individual until reproductive success is more likely, then depression should be of limited duration. In this way of looking at things, a learned helplessness 'program' triggers depressive yielding under hopeless conditions. Depressive yielding persists involuntarily for several months and then lifts. The individual would then begin to reassert itself to the goal of dominance and reproductive success.

As is obvious, Price's hypothesis has me fascinated. Otherwise, I am still working on a draft of my familial bonding hypothesis for a psychiatric journal.

Incidentally, there seems to be an increasing interest in this evolutionary perspective on incest avoidance in San Diego. I've been asked to give a talk on the familial bonding hypothesis at the Psychoanalytic Institute if San Diego and also the Dept. of Anthropology at UCSD, both for later this winter. In October I presented my hypothesis to a group of residents and faculty here at UCSD and response was quite favorable. This surprised me somewhat, but I am beginning to believe that the refined and elegant evolutionary approach to behavior made possible by the work of Hamilton and others is beginning to capture the attention of a much wider audience.

1. Church land PS:Neurophilosophy: Toward a Unified Science of the Mind/Brain Cambridge MA: HIT Press, 1986,p3

2. For ASCAP Newsletter Volume 3 (Jan through Dec, 1990) please send $18 (or equivalent) for the 12 issues. Make checks or money orders out to "Department of Psychiatry and Behavioral Sciences, UTMB." Note on page 11 that subscriptions for ASCAP Newsletter Volume 4 (Jan through Dec, 1991) are now being taken.

3. ASCAP philosophy and goal. High scientific importance rests on comparing animal behaviors across-species to understand better human behavior, knowing as we do so that evolutionary factors must be considered for understanding properly such behaviors. To accomplish these comparisons, very different new ways of viewing psychological and behavioral phenomena are required. This in turn explains why we need new words to define and illustrate new dimensions of comparisons across species. We expect that work in natural history biology combined with cellular-molecular biologic research will emerge as a comprehensive biologic basic science of psychiatry. Both top-down and bottom-up analyses are needed. Indeed, this must happen if we are to explain psychiatric illnesses as deviations from normal processes, something not possible now. Compare to pathogenesis in diseases of internal medicine.

4. The HBES Newletter will include registration forms and further details of the meeting. Contact Drs Wilson or Daly if you are not on that mailing list at the following addresses:

Department of Psychology
McMaster University
Hamilton, Ontario, CANADA L8S 4K1, phone 416-525-9140 ext. 3033; FAX 416-529-6225.

5. Brosius J: Retroposons--seeds of evolution. Science 1991;251:(15 Feb issue)753

6. Lewin R: Research news: molecular biology of Homo sapiens Science 1986;233:157-158

7. a. Dryja TP: Deficiencies in sight with the candidate gene approach. Nature 1990;347:614.
b. Cremers FPM, van de Pol DJR, van Kerkhoff LPM, Wieringa B, Ropers H-H: Cloning of a gene that is rearranged in patients with choroideraemia. Nature 1990;247:674-677.)

8. Goodall J: The Chimpanzees of Gombe: Patterns of Behavior Cambridge, MA: Harvard U Press, 1986.

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