# ASCAP NEWSLETTER

Across-Species Comparisons And Psychopathology Newsletter
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"For reasons that remain unclear neither Cannon nor Selye appears to have been instructed by the writings of C. R. Darwin (1859, 1872)." Herbert Weiner[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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Announcement: Alas, the American Psychiatric Association Program committee did not approve our IASCAP symposium request. Since this means that Dr Price, our president, is unlikely to come to San Francisco in May, 1993, for the APA annual meeting, the plan for our first IASCAP meeting there has collapsed.

The Executive Committee proposes instead to have a meeting somehow in conjunction with the Human Behavior and Evolution Society (HBES) meeting scheduled for 4-7 Aug 1992 in Binghamton, NY. Could we potentially meet before that meeting? John Pearce has suggested that a meeting site might occur in the Boston area and has agreed to to look into possibilities.

*Of course,* members are encouraged to send abstracts for presentations at HBES as well (John Pearce has suggested that he is willing to lend some large cars for the journey there; John argues against having the meeting *after* Binghamton because people would be too tired).

Paul Gilbert who will be president then suggests that we inter-link as much as possible with the other group (remember John Pearce's vivid description of last year's HBES meeting). Leon Sloman is planning to request time on HBES program for a panel on psychotherapy.

But our issues are different, and our task of meeting will have a aim dissimilar from that of HBES which concerns itself little with basic plan issues. With increasing clarity of our own framework, we need to integrate existing data and to collect more of our own (one of the motives for an Institute of Sociophysiological Medicine--presented last issue).

But Paul also points out that we need to publicize the basic plan thesis. This provides added impetus for the material summarized in the IASCAP symposium abstracts to be discussed.

But Paul's point goes beyond. We as a group need to publish beyond this Newletter format, as in books and in the archival literature (he has modeled this for us). Part of the discussions of the meeting should involve how to best accomplish this and how to carry out investigatory aims.

Planning for an book may be in order--see below Roger Pitman's letter (perhaps the abstracts intended for the APA are appropropriate as a guiding format).

Or, as suggested by the communique from Price and Wilson (p 4), the topic organization suggested by the plan last issue for an Institute of Sociophysiological Medicine could guide the organization of the book. There are additional topics highlighted in the communique. Also see John Price's essay p 11 this issue.

Perhaps, as Paul has suggested, a special issue of a receptive journal may be in order. Think about it. We ask membership to indicate your interests, desires and suggestions.

Letters; August *25th, 1992*

*For many years I had been working in the area of violence and antisocial behavior with young people. I am particularly interested in ethological and evolutionary perspectives regarding this problem. In a recent issue of the HB&E Newsletter, your work was mentioned. I wonder if you could send me any ideas that you have on this subject, appropriate references, and pertinent reprints.*

*Wm A Abruzzi, Leicester, NC, OSA*

*I* wonder if this Newsletter itself is the form in which "my work" was meant, because my personal efforts bear only indirectly on the topic, although I'm highly interested. Therefore I send out a clarion call to any of the readership who might help Dr Abruzzi out. The abstract reproduced below of the work done by Mos, Olivier, & Tulp may possibly bear on your interest as well.

Finally, I send you the Oct issue of ASCAP which tells you of a meeting in London that may apply to the problem and therefore may bear on your needs. If you subscribe to ASCAP. you may learn of additional information as it emerges.

Abstract; Mos J, Olivier B, Tulp MTHM: Ethopharmacological studies differentiate the effects of various serotonergic compounds on aggression in the rat. Drug Development Research 1992;26:343-360. [Serotonin = 5-HT]

A series of experiments was performed to investigate the effects of aggression of various drugs

affecting serotonergic transmission in rats. Using ethologically derived behavioural categories, the behaviour of treated animals was described. Drug effects were observed in two aggression modes: resident-intruder aggression (RI) in male rats, and maternal aggression in lactating females during the post-partum period (MA). The 5-$HT_{1A}$ agonists, buspirone, ipsapirone, and 8-OH-DPAT, decreased aggression in RI and MA but simultaneously led to a marked decrease in social interest and activity, indicative of a nonspecific anti-aggressive profile. Nonselective 5-HT agonists, such as RU24969, eltoprazine (DU 28853), and TFMPP, reduced aggression quite specifically and did not decrease social interest or exploration, but sometimes even increased these behaviors. In RI and MA, the behavioural effects of these drugs were quite similar. By contrast MA was more sensitive to treatment with the 5-HT reuptake blocker fluvoxamine, which blocked RI aggression only nonspecifically at the highest dose. DOI, a 5-$HT_2$ and 5-$HT_{1C}$ agonist, decreased aggressive behaviour and increased inactivity, without affecting social interest and exploration in RI as well as MA. This was, however, accompanied by "wet dog shaking," characteristic of 5-$HT_2$ -receptor stimulation. The nonspecific 5-HT agonist (and 5-HT, antagonist) quipazine also induced "wet dog shaking" at doses that suppressed aggression, social interest and exploration but increased inactive behaviors (sitting and lying). The discussion attempts to delineate a role for 5-HT receptor subtype involvement in the modulation of aggression, with the restrictions we clearly face with regard to the lack of specific serotonergic agonists and antagonists for certain receptor subtypes. By and large, male and female rats react similarly to treatment with serotonergic drugs stressing the consistent role of 5-HT in different forms of aggression.

Letters (cont). *October 5, 1992*

*I was excited to read about the proposed APA Symposium and hope that there is room for me as well as Henry [Nasrallah] in the front row. In the event that there might be a publication coming from the symposium, that would be extra special. If such turns out to be the case, and you can include non-presenters, will you consider me for a PTSD-related article? I enclose a couple of publications of mine on this topic.*


1. Pitman RK: Editorial: Post-traumatic stress disorder, hormones, and memory. Biol Psychiat 1989:26:221-223.

2. Pitman RK: Editorial: The black hole of trauma. Biol Psychiat 1990:27:469-471.

3. Pitman RK, Orr SP: Psychophysiology of emotional memory networks in post-traumatic stress disorder. Written as a chapter for proceedings of the Fifth Conference on the Neurobiology of Learning and Memory, UC Irvine, October 22-24, 1992.


*R Pitman, Harvard, Manchester, NH*

Letters (cont). *1-10-92*

*Thanks for the review [September ASCAP]. I have written a couple of things to amplify certain points ...[see below] I also enclose my comments on Beck's paper [next issue].*

*I think you slightly, just a touch, overstated my lack of information on biology. I have explored neurochemistry in other books (in the appendix of Human Nature and Suffering,[4] there are even pictures of pre- and post-synoptic activity), but not here....*

*[Regarding the idea of an Institute of Sociophysiological Medicine], I think you should go for the name of a "Biopsychosocial (BPS) Institute rather than coming up with a new name that few will understand. A biopsychosocial institute would be the first, and if this were underpinned with evolution theory that would be really something. People have a vague understanding of what it is about (eg, see Rutter)[5]...*

*My new book, Counselling for Depression, is now out (Sage, 1992) and is doing ok.*

*Paul Gilbert, Derbyshire, OK*

Letters (cont). *October 16, 1992*

*...The particular organ that you have produced (ASCAP Newsletter) has been probably the major stimulus for my forays into evolution and behavior. I also profited a great deal*

*from reading your chapter in the Kaplan/Sadock book...*
*Aaron T. Beck, U Penn, Philadelphia*

Letters (cont). *23 Oct 1992*

*I am writing to you in something of a panic. If you look again at my letter of the 25 Sept and the copy of the page from the Spectator I sent you, you will see that there was no suggestion on my or Max Hasting's part of fraudulent behaviour. He simply felt that, to put it at its kindest, others were available who could run the bank more successfully.*

*My concern is that as the quoted extract is referenced, the bank chairman can readily be identified and that you and I could find ourselves subject to a libel action. Can you think of an appropriate way of straightening it out in the next edition. It seems a sensible procedure.*
*Mike Waller, Worcestershire, UK*

My turn for *mea culpa*! Many apologies. I believe your letter sets the record straight. I overread the material provided. RG

Letters (cont). 23.10.92

*ASCAP readers will like to know that Adrian Kortlandt, one of the early primate ethologists, gave a communication to the 1992 International Primatological Society Congress on the implications of methods of observation on the results obtained: in one of which (using a hide), he discovered that chimpanzees have a gestural language (using arms and hands) used in the wild, completely missed by other methods of observation. A film is available. Those interested could obtain a free copy of the abstract from Dr. Adrian Kortlandt, 88 Woodstock Road, OXFORD, UK. I can confirm that lions behave differently when viewed from behind a one-way screen, as can be experienced at the Seattle Zoo.*
*Michael RA. Chance, Birmingham, UK*

Abstract: Dillon JE, Raleigh MJ, McGuire MT, Pollack DB: Acute changes in social composition and agonistic behavior in male vervet monkeys. Am J Primatol 1992;27:225-230.

Among nonhuman primates the composition of social groups influences the interactions of group members. We assessed the effects of acute changes in social composition on behavior among 15 adult male vervet monkeys (Cercopithecus aethiops sabaeus). Subjects were observed in their basal social groups which comprised 3 adult males, 2-4 adult females, and offspring; and in two subgroups consisting of either two or three adult males.

Agonism and vigilance increased in smaller groups relative to basal conditions, while subjects in two-male groups displayed more aggression than those in three-male groups. These findings suggest that, among male vervet monkeys, acute disruption of stable social groups increases aggressive behavior and that the amount of agonism is influenced by the composition of the consequent groups.

COMMUNIQUE FROM JOHN PRICE AND DAN WILSON AT ODINTUNE IN SUSSEX

Autumnal greetings from the South Downs! Dan is spending a year in Cambridge, England, attached to the Departments of Psychiatry and Anthropology, pursuing his research in the evolutionary epidemiology of psychiatric disorders. On the side, and on behalf of his sponsors, he is bringing his unique views on the American way of life to English Rotary Clubs. While he is spending a weekend at Odintune, we are taking the opportunity to review the current state of evolutionary psychiatry, and we feel motivated to respond to the Editor's proposal for an Institute of Sociophysiological Medicine which has just appeared in the October ASCAP. This appears to us to be a major policy document for evolutionary psychiatry, and we note in passing

that it is an expansion of the ideas first expressed in the author's chapter in Michael Chance's Social Fabrics of the Mind. The prospect of a centre, however, organised, which might focus on key study areas of evolutionary psychopathology is just what is needed at this stage.

The gist of RG's chapter in Fabrics held that psychopathological states represent exaggerations or distortions of items of normal adaptive behaviour, usually communicative forms of behaviour; and that both normal and equivalent abnormal forms can also be expected to occur in animals. RG identified eight types of behaviour which could underlie forms of psychopathology, and he suggested that these behaviours should be studied, together with their pathological deviations, both in animals and man. This seems to us to underlie his proposal. If we compare the "eight areas of physiological study" in the proposal with the eight "psalics" on p 213 of Fabrics,[7] we find that five are identical, as follows:

1. "Eating and nurturance" are equivalent to the nurturant and nurturant-recipient psalics.
2. "Mating and sexual preoccupations" match the sexual psalic.
3. "Planning and leadership" corresponds with alpha psalic.
4. "Powerlessness and yielding" equals the in-group omega psalic.
5. "Xenophobia and in/out groups" is the out-group omega psalic.

New candidates which the proposal suggests for study not mentioned in Fabrics are "Flight, fight and danger"; "Phonology and communication"; and "Suffering and pain." Psalics in Fabrics that have not made it through to the proposal are Alpha-Reciprocal and Spacing. We think this represents a constructive development in thinking. We must remember that basic to all these ideas of study is the concept of relationships, and also the idea of the life cycle, so that it would not be appropriate to include these as areas of specific study. Four additional areas which we feel should be considered for inclusion are:

1. Territorial behaviour, possibly relevant for fathoming agoraphobia.
2. Reciprocity of exchange, suggested by Kalman Glantz and John Pearce as important for the genesis of anger, guilt, depression, etc.
3. Behaviour related to storage and checking, possibly relevant for obsessive/compulsive symptoms.
4. Leaving home behaviour (cf the patrilocality of apes and the matrilocality of monkeys) as possibly relevant for adolescent behaviour problems.

There are, of course, other possibilities, such as sleep, but one must draw the line somewhere and there are already a number of sleep laboratories.

Overall, we feel the proposal represents a realistic but ambitious goal, one achieved by evolution as well as by creation. It is a proposal which might well be attractive to a host institution because it represents a new approach to the study of psychiatric disorders based on no less than a complete paradigm shift. As such, it would offer the possibility of taking that institution into the forefront of twenty-first century psychiatric research. Because of the essentially interdisciplinary nature of the research, it would be important to find a university or research institute in which relevant allied disciplines such as biology, psychology and anthropology were sympathetic to the approach. It is quite possible that a forward-thinking department could foster the emergence of such an institute, in the first instance, simply by drawing together a critical mass of evolutionary psychopathologists in a medical center and ensuring that they have "pro-

tected" research time. The rest might accrue over time with success in interdisciplinary research, grantsmanship and (critically) teaching.

We do not feel competent to comment on the organisational aspects of the proposal, but we remember that in **IAS-CAP** we have an expert on organisational matters and we would be very interested in Mike Waller's comments on the proposal.

Finally, what about getting eight volunteers to review the present state of knowledge in each of the eight study areas? That would give everyone a good baseline to start from, and an overview of the whole field. It might make an interesting edited volume. (For instance, we do not know of a comprehensive review of the physiology of "powerlessness and yielding", dealing with the mouse work mentioned by Mark Erickson in the Mar 92 ASCAP; Norbert Sachser's work on guinea pigs; Dietrich von Hoist's work on tree shrews; Barry Keverne's work on talapoin monkeys, etc, etc).

So, dear Editor, from Odintune, our congratulations, blessings, & good wishes for your plan for an Institute of Sociophysiological Medicine.

## Response to RG Review on Depression and Powerlessness

by P Gilbert

RG has asked me to respond to his critique of my Depression book and to use this as an opportunity to share some thoughts and go out on a speculative limb about biology and drugs. So I guess my first problem here is: RG critiques the book I didn't write rather than the book I did. Hence one can simply say, "You're right. I did not go into much detail on neurochemistry." But this was not lack of interest--far from it but a recognition of personal limitation. Still, at the risk of being defensive, it is not entirely true that I neglect the biological treatments. There are brief sections on the findings of genetic factors, drugs (which affect symptoms rather than specific illness types, eg, Hudson and Pope, 1990), biological changes associated with helplessness (that section was quite detailed I thought) and rank changes. Further, the reader is referred to reviews on the neurochemistry of depression (eg, Healy and Paykel, 1989)[9] which do a far better job than I could do. Further I stressed on a number of occasions that severe depression and cyclical depression require physical interventions because the brain state has become so encapsulated from external influence.

The book tries to link evolutionary theory with other current theories. Now it is, in fact, rare that those interested in evolutionary processes engage the details of neurochemistry. In Brant Wenegrat's influential book on the sociobiology of mental illness, there is not a neuron or receptor in sight![10] The field of the biology of attachment and separation followed from Bowlby's ethological theories of attachment and did not precedeit.[11]

It looks like our big difference of view is that RG is a more reductionist than I. For example, he states clearly that microsecond by microsecond evolved structures operate with genetically driven metabolic machinery that *causes* mood and reacts to it. Yet in the next sentence he says that pace-making cells are responsive to social information, coordinate other processes and direct the actions of other cells. But this is having one's cake and eating it too (I'm trying to lose weight at the moment, hence the metaphor). And the key question is what information influences these pace-making cells?

And now the major debate is a major one: is depression best viewed as a mood disorder (a basic abnormality in

the neurochemistry of affect regulation) being powered within, or, on the other hand, is mood a symptom--a manifestation of more complex interacting processes that are concerned with information processing, decision making and behavior. As Daniel Freedman recently stated while we look at the brain and can find consequences of decisions we cannot find where decisions are actually made. [12]

This is not a trivial point. Once we say that depression results from decisions that the brain has made then top-down and bottom-up distinctions are meaningless, especially if they are superimposed on biology (bottom) versus psychology (top) distinctions (a common error in my view). Every thought, action, desire, and feeling must have some representation in patterns of neuronal activity. Psychological events do not do *something* to biology; *they are biological.* For example, the startle reflex is clearly biologically mediated but also represents decisions--it can habituate, ie, after a number of presentations of (say) a loud sound, neuronal sensitivity changes; the brain decides not to respond. But also if you tell people that a loud sound is coming they are less likely to startle. Thus, decisions can be taken in different ways in the brain. But there is no separate psychology operating on a biology. Patterns are our keys and patterns can take circular forms rather than linear up-down ones.

However, RG pinpoints the issue when he looks to the future. How does the loss of a sense of personal power and influence translate across our various domains of "being in the world," from genes, neurons to feelings, "that we later experience on the whole organism level as a slumped down non-moving expressionless defeated person"? Now that is Nobel Prize stuff and frankly I don't know. But I suspect that to answer the question, we must change our questions fundamentally.

First, it has yet to be accepted that depression has anything to do with evolved defeat programs in the brain, or that there are inhibitory energy control systems that evolved to inhibit animals in a no-win or lose situations. Until those steps are taken then little else can follow for this approach. So the book was making this argument: Depression results from the activation of some internal control mechanism(s) that evolved to inhibit animals in low rank/status or losing positions; it is a low-risk, low-gain strategy. Mania may be a high-gain high-risk strategy; bipolar illness suggests major racket-like oscillations rather than mild transitions between these two basic strategies. We know that normal mood shows mild cyclic oscillations [13] and recent work by WHO suggests that in 5-10% these oscillations may be short cycles of distressing intensity. Thus RG's pace-maker cells might lie in these oscillators. [4] pp383-391.

But still we need more linkage with evolutionary function. Thus the downturns represent (in our theory) the operation of inhibitory mechanisms of the low-risk low-gain (no-challenge) strategy that puts a powerful break on aspiration. This may account for the biological state of depression, the psychological preoccupations in depression and the social behaviour of depression. It is the mechanism(s) for *involuntary* subordinate/loser behaviour.

In severe depression it is as if much of the brain (especially in the positive reward areas) has habituated to external inputs and is non-responsive. Whether or not habituation or inhibition proves a more valid model is yet to be decided, and in any case, they are not mutually exclusive. But only when these evolutionary ideas or some modification

thereof are shown to have value (and there may yet be powerful argument against them) can the *biology* of defeat and its effects on habituation/inhibition become the next step. We are almost (but not quite) at the same stage of research as that on attachment theory thirty odd years ago. We need a coherent theory of the evolved function of depression (eg, as a no-challenge strategy) before we can search out biological mediators. To do this there are some important first steps:

1. We need to recognise that phenomenology (descriptive psychiatry) is riddled with difficulties. It is doubtful whether evolutionary theory can continue to try to marry their findings with DSM phenomenologies--which arouse from very different forms of thinking. Although, of course, phenomenonogy is a necessary first step, things do need to be identified before they can be studied, there are in fact comparatively few diseases that have been understood by relying on sophisticated phenomenology. Often what happens is that some pathogenic process is intensified and then a host of unrelated symptoms/phenomena make sense. For example, the causes of pus-filled wounds or pneumonia would still puzzle us without a knowledge of bacteria and antibiotics. Similarly Cushing disease would still perplex us if we had to rely on phenomenology alone and had no understanding of endocrines.[14]

2. The search for diseases of depression may well be doomed. Rather we must have better conceptualisations that depression is some part of an evolved social behavioural system. So what is the system? Is it related to attachments or rank or control? What are the salient dimensions of decisions that are at issue in depression? Hence depression can be seen as a symptom of defeat, or attachment problems, or loss of control/helplessness etc. Changing the search away from depression as such to disturbances in the evolved infrastructure could have major payoffs. For example, there is growing evidence of at least two types of depression.[15] One is triggered by attachment losses and separations and the other is triggered by failures in achievements. There is some preliminary evidence that these are associated with different patterns of symptoms. (But where is the psychobiological research to explore such distinctions?) Is it therefore more meaningful to think of different types of depression, or to drop the depression label altogether and think of very different types of disorders that will share symptoms (much like the physical illnesses share high temperature, vomiting etc), but are actually related to different basic plans (eg, disorders of attachment versus disorders of achievement)?

3. Thus, before we can move to a meaningful understanding of neurochemistry that is more than an ever growing list of neuron-complexities, eg, number of receptors, etc, we must make the case for dropping concepts like depression--and instead switch our nosologies to evolved biosocial meaning-creating mechanisms (eg, attachment, rank, etc).

4. In my view we are still trying to do this. There are many, indeed perhaps the majority, who see no reason to recognize that brain design is a consequence of evolution or that the brain is primarily a decision-making organ following evolved stable strategies. For many, depression has no function; it is simply an illness. They prefer to look for illusive substrates for depressed mood (but as mentioned before, moods are consequences of decisions and switch the brain into particular patterns of activity). So even if these substrates are found, they will not tell us that much about depression—

however useful for drug companies.

To what extent are anti-depressants psychic pain-killers that can switch off many negative affects and allow change to take place while the body repairs itself or the depression runs its course, or protected from pain the person is now able to take up new strategies? To what extent does relapse on anti-depressants show habituations effects to the drugs? Serotonin or 5-HT, in the limelight now of current research on depression, has been implicated in just about every psychiatric disorder one cares to think about. So to call a drug an anti-depressant when it is well known that such drugs affect many conditions is grossly misleading. New drugs like fluoxetine are not only good for depression, but obsessional disorders and possibly panic disorders as well. 5-HT seems implicated in depression but I don't think that one can say more than that at the moment. These are all questions I can't answer. So I will always remember a patient who said that while on medication he still felt bad about himself and that he has various negative ideas, but then these don't bother him. But they had lost their emotional impact, and now he gets on with life in spite of them.

5. The ranking and social investment theory of depression is still very new and not accepted and therefore has to be articulated and argued for. In my view John [Price]'s major contribution of the importance of rank in depression (now 25 years old) could not grow and develop because people simply did not consider pathologies of mind from the evolutionary point of view. Until recently most have hoped the phenomenologies will someday map neatly onto pathophysiology. And thus there have been endless efforts to measure symptoms over and over with this or that statistical technique just hoping that some disease will fall out of the computer. Furthermore, as pointed out in the book, many of the symptoms key to most clinicians who get to know their patients, such as shame, envy, rage, inhibition, dichotomous thinking, etc, do not even appear in most classification research. Thus, a double problem here: first to bring these symptoms into the limelight and then to offer a coherent explanation of why they are so often operative when people also experience the draining of their energy in what we call depression.

6. This brings me to my main concern with RG's review: that one gets no feel for what the book is actually about. Basically, I tried to say that if you first make the issue of rank the key question of interest, then two further dimensions open up: a) we can separate the hostile and fearful means of navigating rank (do what I want, or I'll whack you) from the attention-seeking (invest in me; I'm a good, clever, able, talented, attractive person, ie, the need for recognition/admiration) aspects, and b) we might be able to distinguish the very primitive defeat program (possibly located in the R-complex) from the later evolved subordinating programs (possibly located in the limbic system)--though such locations are hopelessly crude right now. But if one goes for depression as a no-challenge, low-risk low-gain strategy (which is not the same as energy conservation—depressives are very poor at conserving their resources) then a whole lot of things about depression start to make sense. Depression becomes part of a challenge-inhibition strategy, often associated with involuntary, subordinate self-perception, idealising, negative social comparisons, shame, rage, envy, loss of energy or motivation, fear of asserting the self. But the biology of all this cannot proceed until we have debated the merits of this approach and either

thrown it out or really started to sort out these ranking systems. Thus RG's complaint about my lack of biology is putting the cart before the horse. Also, as he and I have discussed many times (and I have written about this), I do not think that our current drugs affect specific social domains but operate at a much less differentiated way on defense and safety systems — but that is another point.[4]

Maybe I should just have come out and said that depression is a myth. The DSM approach is leading us further away from understanding and we must consider our subject matter (human mental suffering) as variations of evolved stable strategies; Beck's approach in the April ASCAP Newsletter is a far more sensible approach than the DSM. But I think Beck, like myself, in an effort to map the pathologies of innate strategies with DSM ended up with a compromise that doesn't work too well. The DSM view of there being a thing called depression forces us to put things together that don't fit and leave out things that do.

So I see the future in the following way: a) some group comes together to mount a serious challenge to the DSM (which has no view or model of brain design, function or development) and engages in careful biopsychosocial modeling that is derived from theories about the function of the brain as a decision making organ; b) to start classification in terms of social decision making processes, eg, attachment, in-group/out-group, reciprocation, rank, etc or whatever research suggests are relevant dimensions); c) to develop theories which also enable researchers to be aware of developmental issues (ie, attachment threats and brain responses that vary with age, being different in one vs twenty year olds) and so pathologies represent challenges to functions that have matured in certain ways or failed to mature in certain ways.

We also need to be sensitive to the ecologies and social context of individuals to avoid constantly locating the error in the clockwork of the person. An extreme view to make the point: to say that the depressions and experiences of the hostages in Beirut are caused by brain amine changes is to say little. Or that if someone breaks my leg, my pain is caused by opiate receptors. Pain rests on a bed of biology and the book makes this clear, but as to causes, these are multi-factorial and complex. As the last chapter points out, unless we sort this out, we are going to be lost when it comes to prevention. It would be like living in a violent society where people break each others' legs and responding by making pain-killer medications available, or even giving pain-killers to prevent pain.

So ideas of cause are also serious political ideas, and the limitation of our causal theories of depression must be made clear. I have no doubt that depression is a biological process, that thresholds can vary, that genes are important, and that changing biology can alleviate suffering--I said so throughout the book. But an evolutionary approach must state clearly that the brain is a decision-making organ and that helping people understand the sources of their decisions is also involved (hence various therapies). Also decisions are made under certain ecological and historical conditions. There is growing evidence that borderline systems are related to a history of abuse, for example.

But after all the speculations and half-formed thoughts, let me come clean. It is true that I had a half-chapter on neurochemistry and then was forced to abandon it, for much the same reasons that McKinney notes (Sept ASCAP), and I got lost in the

complexity. I was never happy with it. When I took advice from my biological colleagues, some were confident about the models but others thought the biological models were a mess. The monoamines remain the central focus of interest in depression but they have been involved in just about every psychiatric disorder one cares to think about. While we have learnt much about the details of neuronal transmission, what do all the findings on the depletions, receptor changes, and secondary messengers mean? Are they correlates, symptoms, causes, or amplifying deviation mechanisms? Then the endocrinologists can also argue that their data should be included (some regard endocrines as the real pacemakers of activity), and what about the brain imaging and neuropsychological findings--where should those fit?

In general I agree with RG's critique, but don't know enough about the biology of defeat to say much useful. That may be the next challenge.

Behavior & Genetics; Alternative Strategies by John Price

I drink a toast to the initiative of setting up the symposium on "From genes to behavior: evolution and psychiatry." After the unsuccessful attempt to get us a meeting at the Royal College last year, I know how much effort it takes.

Perhaps we could use some of the space in ASCAP between now and May to calibrate our ideas on evolutionary biology, so that we present a consistent message to an audience which may not know much about the field. There are two points which are rather basic, and which I will attempt to expound here. One concerns the relation between the evolution of behavior and genetics, the other concerns the concept of alternative strategies.

Clearly it is important to know the method of genetic transmission of any inherited behaviour, or lack of it, if at all possible. We want to know what the heritability is, whether a single gene can be identified, and if so, what chromosome it is on, or at least whether it is on a sex chromosome or an autosome; for polygenic traits we want to know whether there is evidence of directional selection, and so on. Your work on the Prader-Willi and Angelman syndromes is a good example of this approach. However, if we are not able to identify genes, we should not feel inhibited from discussing the inheritance of behaviour in terms of function. This is the current view of behavioural ecology. Grafen calls it the organismal approach, and claims that for practical purposes we can ignore problems of genetic transmission if genetic analysis proves impossible.[16] He writes:

> The organismal level of the conclusions reached by inclusive fitness theory is important first because it achieves a radical simplification if genetics can be passed over. It is also important because in social traits, even more than in non-social traits, virtually nothing is known of the genetics of evolutionarily interesting characters. We observe organisms and interesting morphology and behaviour, but we rarely observe interesting genes. Practical applications therefore require a principle at the level of the organism.

Perhaps it is because it has freed itself from the discipline of genetics, and from the feeling that without genetic analysis there is nothing, that behavioural ecology has made such advances in the last few years. The message seems to be: identify the genes if you possibly can, but if you can't, not to worry. Are we agreed on that?

The other point is on alternative strategies. As we know, the early ethologists were not interested in within-species variation. It was a challenging enough task for them to identify species-typical behaviour for analysis and to compare the

phylogeny of these units of behaviour in the way that paleontologists compared the phylogeny of teeth, etc. But when they did start looking at within-species variation, they seem to have gone about it in quite a different way from the way psychologists have approached human variation.

We are familiar with the concepts of mean and variation around the mean, and we have ideas that, on the whole, mean values are more adaptive, and extreme values in any trait are associated with decreased reproductive success. But the behavioural ecologists have approached it differently.

They look at behaviour as strategies, and they see within-species variation in behaviour as a choice between alternative strategies. Sometimes these strategies are incompatible, so that the distribution of behaviour departs markedly from the unimodality, and it makes no sense to speak in terms of means and standard deviations. At other times, the "mean" behaviour is clearly less adaptive than the extremes. What, for instance, is the mean between hibernating and staying awake, or between migrating and staying at home? The physical analogy used is sex. One can choose (or, in the case of some insects, one's mother or the workers can choose for one) whether to be male or female, and as we know this is a case of frequency dependent selection when it pays to be male if most of one's peers are female and vice versa. A choice has to be made, because to be half male and half female is not adaptive, at least in vertebrates. Sex approaches the sort of behavioural strategies we are interested in only in the sense of those fish whose sex is socially mediated; they make a choice, in negotiation with at least one conspecific, to change sex or stay the way they are--but they never choose something in between.

Looking at behavioral variation as alternative strategies has some interesting implications. For one thing, it makes heritability unimportant. If evolution has produced two highly prepared strategies, such as the mating strategies of the silk moth, and the choice between them depends on the food supply, then the variation in mating behaviour of the silk moth will appear entirely environmentally determined and it might be assumed incorrectly that genetic factors are unimportant. One has to apportion the variance into within-strategy and between-strategy components. In the case of the silk moth, the between-strategy variance is almost entirely environmentally determined, but the within-strategy is genetically determined. So even if the overall heritability is near to zero, environment and genetics may still both be crucially important. I think it is possible that something like this is happening in the case of the human capacity for mild episodes of depression.

In a chapter entitled "Alternative strategies," Krebs and Davies discuss theory and give examples.[17] They identify three ways that variation between alternative strategies is maintained in populations. This deals with the problem, "If there are strategies A and B, one must be slightly better than the other, so why is the less satisfactory strategy not eliminated?" One reason is environmental variation, eg, that strategy A does better in environment x and strategy B in environment y. For instance, the varying qualities of different depths of the sea maintain variation in the colouring of the male sticklebacks: in the deep sea where it is dark, stickleback males with bright red throats do best because they attract more females; but in the shallow sea, where there is more light, males with dull throats do best because they are less

susceptible to predation. A human equivalent here might be introversion\extroversion, the former doing better in arid, sparsely populated habitats, the latter in lush, densely populated areas.[18]

The second factor maintaining different strategies is variation in the phenotype of the individual playing the strategy. For instance, finding itself with a phenotype less well endowed than the average, the individual plays what Krebs and Davies call a "making the best of a bad job" strategy, which is more successful than the standard strategy (which is in turn more successful in the better endowed phenotype). Because of variable feeding when young, certain male bees are three times as large as their fellows. They search for females by patrolling, searching for, digging up and mating with females as they emerge from the ground, usually vying to fight other large males in the process. Small males hover above the ground and mate with females who have escaped the large diggers. Krebs and Davies comment:

> These are all types of strategies that are conditional on an individual's phenotype, eg, 'if big, fight; if small, sneak'. The largest and strongest individuals have the greatest success and others are forced by circumstance to adopt less successful, alternative strategies.[19]

It seems to me that the genetic basis of this strategy is very similar to Mike Waller's 'comparator gene'. It includes a decision-making process by which the bee decides if it is big or small relative to its peers, and a prescription for one or more "make the best of a bad job" strategies, which could well include depression and/or anxiety, and in fact this source of variation could account for human variation on John Birtchnell's 'verticaldimension'.[20]

The third source of variation is frequency dependent selection, in which the success of each variant varies inversely with its own frequency, and directly with the frequency of the alternate strategy (as in the case of sex determination). This applies to a lot of examples quoted by the behavioural ecologists, for instance, calling versus intercepting in frogs, and digging versus entering in wasps.

A fourth source of variation might be alternative strategies occurring in the opposite sex. If the male has two strategies, there may be one female strategy which succeeds better with one male variant, and another with the other. Then within each male variant there may be one sub-type which succeeds better than the other; and so on, in infinite regression. But this source of variation could be subsumed under environmental variation, as the other sex is part of each sex's environment.

In fact, these four causes of variation seem to me to be reducible to two. One is that strategy A does better in some circumstances and strategy B in other circumstances, and the variation in circumstances could relate to the environment (including conspecifics of the same and other sex) or to the circumstances of the actor itself (its phenotype). The other cause of variation is frequency dependent selection, which seems quite independent of variation in circumstances.

In olden days, I remember reading a paper on the factors maintaining genetic variation (I think by EB Ford). This list included mutation, recessiveness, balanced polymorphism, environmental variation in selection pressure, and balanced translocations (the release of stored variation during crossing over of chromosomes). But I cannot remember frequency dependent selection being on that list. Does anyone know who 'discovered' it? What causes of genetic variation remain undiscovered?

To view variation as alternative strategies concentrates the attention

on decision-making processes. There are at least three types of decision to be made in relation to a pair of alternative strategies. When to choose the strategy? When to play the strategy? And, which strategy to choose? These decisions are likely to be affected by different kinds of information, of both genetic and environmental kinds. In the choice of strategy, we also have the possibility of randomisation, eg, the phenotype could be "play strategy A with probability p and strategy B with probability 1-p"; this is the sort of possibility one should bear in mind when thinking about manic-depression, in view of the difficulty in predicting whether the next attack will be manic or depressive.

Let me leave aside the timing of strategies and concentrate on the choice between strategies. Clearly, if the relative success of two strategies depends on varying environmental circumstances, any capacity to assess those circumstances and play the most favorable strategy would be selected for. For instance, if the male stickleback could assess the depth of the sea, and play 'bright red' in deep water and 'dull colour' in shallow water, it would outperform either morph which played its strategy regardless. In fact, this does not seem to have evolved in the stickleback. A good example of an environmentally contingent colour strategy is the chameleon. Another species in which the variation occurs but is not contingent is the moth with white and melanic versions in which the latter became common in 'sooty' city environments.

But the environmentally contingent strategies we are really interested in are the ones that depend on the actor's own phenotype, or on a comparison between his own phenotype and that of same-sexed conspecifics. Here we are dealing with variation in RHP\self esteem\vertical dimension — these are the alternatives that make the difference between good and bad mental health, and that is why Mike Waller is right to keep on banging on about his 'comparator gene' , and why we should all be reading current work on social comparison, however boring it might seem.

Abstract; Kinomura K, Yamauchi K: Fighting and mating behaviors of dimorphic males in the ant *Cardiocondyla wroughtoni* J Ethol 1987;5:75-81.

Colony composition in Cardiocondyla wroughtoni and the fighting and mating behaviors of 2 types of males, alates [unaggressive] and ergatoids [aggressive] are described. This species is polygynous, with a mean of 7.0 queens per nest... Within nests, ergatoid males fight with each others, leading to the death of all but one in single nests. [Alate males after two days move so quickly they are rarely caught.] On the other hand, alate males exhibit no aggressive behavior towards any of their colony members. Both types of males conduct intranidal [within nest], matings with their sisters, though the alate males also conduct nuptial flights. Many alate females leave their maternal nest even if they have already been inseminated by intranidal mating.

Abstract: Srere HK, Wang LCH, Martin SL: Central role for differential gene expression in mammalian hibernation. Proc Natl Acad Sci 1992;89:7119-7123.

Mammalian hibernators experience dramatic reductions in temperature, metabolic rate, respiratory rate, and heart rate during hibernation. These changes are precisely controlled and reversible with only internally driven mechanisms, suggesting specific biochemical regulation. We present a model that integrates our observations of differential liver gene expression during preparation for, and maintenance of, the hibernating state, with the known phylogenetic interspersion of hibernating species in several major mammalian lineages. This model predicts a major role for the differential expression of existing mammalian genes in the biochemical regulation of hibernation.

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2. c/o R Gardner, 1.200 Graves Building (D29), University of Texas Medical Branch, Galveston, TX 77550 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 going to $20/year. Make checks or money orders out to "Department of Psychiatry and Behavioral Sciences, UTMB."

3. EXECUTIVE COUNCIL:
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At this time this "informal[11] organization has no official budget.

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7. Ibid, p 212: PSALIC refers to "Propensity States Antedating Language in Communication" and "Programmed Spacings and Linkages in Conspecifics." This noun defines a primitive communicational state, mediated by deeply homologous neural structures, that, when stimulated and activated, causes the organism to demonstrate an unusual readiness to assume distinctive roles relating to functional activities involving one or more of its conspecifics. Psalics can overlap (ie, two or more psalics can be simultaneously active in the same individual at the same time), and can override to varying extents other factors influencing the organism's behaviour and perceptions."

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