ASCAP NEWSLETTER

Across-Species Comparisons And Psychiatry Newsletter
Volume 3, No. 12, 15 December 1990

"A consideration of any evolutionary process must include genetics...even complex behavioral systems such as sexual selection, which sociobiologists refer to as a 'strategy', can have a relatively simple genetic basis." Hoy[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 p12[3]

Newsletter aims; I. A tree 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.

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Request for Information!

. . . Could you ask in ASCAP these questions?

1. Does any researcher have data on sleep patterns in subordinate primates? Do subordinates wake earlier than dominants? Does loss of rank change sleep patterns?

2. Given the work on sleep architecture and depression, has anyone researched this in primates in relation to rank?

3. Is REM latency related to explorative behaviour, ie. the less explorative, the more REM is shifted forward?

... any data would be welcomed.
Paul Gilbert, Derby, England

Letter *Oct 22nd '90*

Did I omit the address of "Prof BA Robertson, Dept of Psychiatry, Groote Schuur, Observatory 7925, Cape Town, RSA" in my last letter? In case I did, here it is. As I mentioned in my letter of a few days ago, it seems likely that either someone in his dept or in the dept of Psychology at UCT will respond positively to the

proposed depression study.

Patrick J Tummon, Cape Town, RSA

I think that the earlier letter may not have arrived, but the message in this one is welcome.

Letters (Cont) *October, 1990*

Thank you for the ASCAP Newsletter and for your warm letter. Under this cover, I enclose my, and my colleague Dr. Egorov's, opinion on Price's essay "Metaphors of submission." ... Simferopol is near Yalta (approximately 70 km) . There is a Medical Institute with 2500 students. Our department of psychiatry is one of the oldest in the country and we have our scientific roots in the ancient Moscow school of psychiatry. Every year in 1-4 hug we organize Crimean meetings on evolutionary psychiatry. Our meetings take a place in a field (natural) condition and forthcoming meeting will be devoted to the different aspects of sociobiology and psychiatry. I would be very glad to see you among us. ...

Now I am continuing an essay about my work for ASCAP and will send it soon.

VP Samohvalov, Simferopol, Crimea

Alas about the Crimean meeting: already our summer plans have become set for the London meeting of a two day brain-storming meeting in July near London of those interested in a basic plan approach to this research. But ASCAP readers with a penchant for sociobiology now know about a meeting of which this westerner had not known. Thank you very much for the invitation! Perhaps another year!

I have sent your response (with Dr Egorov) to Price's July essay to JSP and hope to put your interaction together in 1991. We also look forward to hearing about your work.

Letters (Cont) *12/2/90*

What a pleasure the most recent ASCAP was. Dr Heisel's ideas about the cross-cultural study made great sense, I thought, because many variables get eliminated that way--although you're still going to need to get to the most primitive eventually. And Badcock's contribution was a delight and will be helpful in my lecture material (obviously I think he's right). And the Sloman-Price discussion! I question somewhat Sloman's idea that yielding responses may originate in separation anxiety; yielding is built-in to the animal as a part of the structuring of the entire agonistic display and appropriate responses thereto. The way the impulse to yield is applied to the situation is probably influenced by separation experiences and the toddler's genetically biased reaction to them.

For me one of the very best parts of Dr Price's writing is his interweaving of English literature into his essays. What delight!

About the meeting in England, I have questions about whether abstracts are needed. We would like to come but cannot prepare a formal presentation now.

Carolyn Reichelt, Wadena, MN, USA

I re-reviewed what was written in the November issue and I can see where ambiguity arose. Any abstracts would be for the Royal College of Psychiatrists meeting *before* our contemplated planning meeting. *Our* meeting does *not* depend on formal presentations, rather on what one brings to it by way of knowledge, interests, critical thinking and planning ability. We will work on how to carry out multinational research for testing hypotheses relating basic plan biology to normal and clinical behavior. I am very pleased at your interest; specific details forthcoming.

Quotes from Francis Crick

"Biological replication, so central to the process of natural selection, produces many exact copies of an almost infinite variety of intricate chemical molecules. There is nothing like it in physics or its related disciplines. (p5)

"By the time most scientists have reached age thirty they are trapped by their own expertise. (p15)

"It came to me [before Crick pursued graduate study] that I was not really telling...about science. I was *gossiping* about it. This insight was a revelation to me. I had discovered the gossip test--what you are really interested in is what you gossip about. (p16)

"The double-helical structure of DNA was..finally confirmed only in the early 1980's. It took over twenty-five years for our model of DNA to go from being only rather plausible, to being *very* plausible (as a result of the detailed work on DNA fibers), and from there to being virtually correct. Even then it was correct only in outline, not in precise detail. (p74-5)

"We have..been criticized because we had not perfectly mastered all of the very diverse fields needed to guess the double helix, but at least we were *trying* to master them all, which is more than can be said for some of our critics. (p75)

"In the broad perspective of the exact sciences, we were not thinking very hard, but we were thinking a lot harder than most people in that corner of biology, since in those days..., most of biology was not thought of as having a highly structured logic. (p76)

"..Stent had argued that a scientific discovery is more akin to a work of art than is generally admitted. Style, he argues, is as important as content. (p76)

"..for those wanting to build a bridge between two distinct but obviously related fields..I am not sure that reasoned arguments, however well contructed, do much good. (p107)

"What makes people really appreciate the connection between two fields is some new and striking result that obviously connects them in a dramatic way. One good example is worth a ton of theoretical arguments. (p107)

"What is the use of ..general ideas? Obviously they are speculative and so may turn out to be wrong. Nevertheless, they help to organize more positive and explicit hypotheses. If well formulated, they can act as a guide through a tangled jumble of theories. Without such a guide, any theory seems possible. With it, many hypotheses fall away and one sees more clearly which ones to concentrate on. If such an approach still leaves one lost in the jungle, one tries again with a new dogma, to see if that fares any better. (p109)

"... it is virtually impossible for a theorist, by thought alone, to arrive at the correct solution to a set of biological problems. (p109)

"A good model in biology, then, not only should address the problem in hand, but if at all possible should serve to unite evidence from several different approaches so that various sorts of tests can be made of it. This may not always be possible to do straight away--the theory of natural selection could not immediately at the cellular and the molecular level--but a theory will always command more attention if it is supported by unexpected evidence, particularly evidence of a different *kind.* (p115)

"Just a single wrong assumption (that the ribosomal RNA was the messenger RNA) had completely messed up our thinking, so that it appeared as if we were wandering in a dense fog. I woke up that morning with only a set of confused ideas about the over-

all control of protein synthesis. When I went to bed all our difficulties had resolved and the shining answers stood clearly before us. Of course, it would take months and years of work to establish these new ideas, but we no longer felt lost in the jungle. (p120)

"..[On] a new reason for rapid-eye-movement (REM) sleep [with Greame Mitchison]...memories are likely to be stored in the mammalian brain in a ..different way from the way they are stored in a filing cabinet...It is..believed that, in the brain, memories are both "distributed" and to some extent superimposed. Simulations show that this need not cause a problem unless the system becomes overloaded, in which case it can throw up false memories. Often these are mixtures of stored memories that have something in common.

"Such mixtures..remind me of dreams and of what Freud called condensation. For example, when we dream of someone, the person in the dream is usually a mixture of two or three similar people. ...in REM sleep there is an automatic correction mechanism that acts to reduce this possible confusion of memories." (p161)

Price-Hofer-Price Exchange by JSP

I sympathise with Dr Hofer's preference for research into proximate rather than ultimate causes, but I would like to comment on his statements that "it is the wrong time to try to draw maps" and that "we know very little about the biological nature of mental illnesses, and have not yet established a basis for their cross-species analysis, two serious obstacles for any attempt to reconstruct their possible evolution." One possible bridge is between psychiatry and behavioural ecology, and I would exhort readers who have not already done so the read Krebs and Davies' Introduction to Behavioural Ecology particularly Chap 7 on "Fighting and Assessment". Here the authors discuss the evolution of ritual agonistic behaviour which they call contest behaviour, or pairwise contests. The variables thought to be important in the evolution of pairwise contests are owner/intruder, high/low resource-holding potential (RHP), evaluation of opponent's RHP, resource value (V) and estimate of costs likely to result from fighting. I believe there is a close correspondence between these variables which are thought important in the evolution of vertebrate agonistic behaviour and the cognitive changes which occur in human mood disorders, particularly the switch from normal mood to the depressed state. Let me list the important ecological variables and relate them to the cognitive changes of depression:

1. Resource value (V) (how valuable is the thing which is being fought about?). This may vary between contestants, for instance a female hamadryas baboon which has been impregnated by one of two rival males. The higher the resource value, the more likely is attack *vs* yield.

In depression there is a general loss of interest in the environment. Things seem less significant and lass important. Psychologists talk about a reduction in incentives and loss of reinforcer effectiveness. Even if the depressed patient thought he could fight and had any chance of winning, he would see things as not worth fighting about.

2. Each contestant evaluates his resource-holding potential (RHP). The higher he thinks his RHP, or fighting capacity, the more likely he is to attack.

In depression there is a marked diminution in self-image. We generally talk of low self-esteem, which is a wider concept that RHP or even self-confidence; but those components

of self-esteem which reflect RHP are lowered along with the rest. Paul Gilbert uses the phrase "involuntary subordinate self-perception."

3. Each contestant evaluates his opponent's resource-holding potential (RHP). The higher the estimation, the less likely he is to attack.

In depression other people tend to appear frightening, and their "put-downs" or catathetic signals are more painful. The whole sense of pain is more acute, and there is even spontaneous pain in many cases. Since the power of an adversary is partly judged by the pain (mental and physical) which his insults and blows cause, it follows that the more pain a contestant feels, the sooner he is likely to stop attacking.

4. Likely costs of combat. In the cost benefit analysis of contests, whereas resource value (V) reflects the potential benefit, the possibility of mental and physical damage reflects the potential cost.

This again accounts for the association between depression and pain. Physical pain is an indicator of tissue damage, so that the more pain is experienced, the higher the estimate of likely costs if fighting is continued, and the more likely the contestant is to yield.

5. Ownership. It is a general rule in vertebrate contests, particularly among territorial species, that owners win over intruders. Possession is nine-tenths of the law, it is truly said. Therefore each contestant calculates the degree to which he is the owner of the resource being fought about, or of the territory on which the contest is taking place. The more he feels the owner, the more likely he is to attack.

Depressed patients have a reduced sense of ownership. They sometimes say they have no right to be anywhere at all, or even to exist. A depressed golfer says that he has no "standing" on the course of life. This is in stark contrast to the patient with elevated mood who often feels that he owns the whole world.

If we add to the above a pessimistic view of the future, so that no engagement seems likely to be successful, and a negative view of the world as a hostile place where no allies are to be found, we have identified most of the cognitive changes of depression as likely to result in a decision to yield rather than attack in a pairwise contest. Therefore it does not seem excessively speculative to suggest that mood changes between mania and depression represent some sort of modulating system which influences the overall tendency to attack or yield in pairwise contests.

I think the advantage of this kind of thinking is two-fold. It clarifies our ideas about the core variables of depression; and it identifies contest behaviour in animals as an important subject for research basic to human depressive disorders. Depressed patients are like those Harris sparrows in pale winter plumage who do not attack other birds and are not perceived as a threat by others . The Harris sparrows can be induced to attack by injections of testosterone, and it seems to be generally the case that RHP in birds can be raised by testosterone; testosterone does not affect RHP in mammals, but it may well be that some other hormonal system exerts a similar modulating effect, regulating agoinistic behaviour by altering those evaluative processes listed above which decide between attack and yielding. The pale Harris sparrows do not get their own way *vis-a-vis* other Harris sparrows - they survive by picking up the crumbs under other birds' tables - and I think that depressed patients do not get their own way unless they are in the fortunate position of having other people who love and care for them and treat them as sick or old or infirm.

What little experimental work is being done on animal contests is being financed by heart and kidney charities, because the subordinate animals develop hypertension and renal failure. This ignores the fact that by the time it dies of renal failure the animal has probably undergone a long ordeal of conspecific-induced torture and has developed at least a state of learned helplessness, if not more a recognisable dysphoric state. It needs to be accepted that studies of contest and ranking behaviour in animals may well be as important for psychiatry as for cardiology; existing promising work such as that of Michael McGuire (on the relation of blood serotonin to vervet monkey ranking) needs to be replicated and extended, and new searches need to be made for proximate mechanisms underlying the regulation of RHP. The drawing of maps based on ultimate causation may help us to enter the right areas for research into proximate causation, by establishing a biologically valid basis for cross-species analysis. Unless we can produce a promising map, no-one is going to finance the voyage of exploration.

Price-Parkes-Price Exchange by JSP

I should like to comment on Parkes' interesting observations on his cat, and also on the question of why (in the ultimate, evolutionary sense) people get depressed after the death of a baby.

The cat who withdraws and refuses offers of nurturance after punishment or defeat is a reasonable candidate for the role of animal model of human depression - research on the cat brain has elucidated some of the mechanisms of human sleep, why should it not do the same for depression? Is the reaction of Parkes' cat typical of the species? Is there any literature on this?

Is the cat adopting the metaphor of the sick role to express its depression? Possibly to competing cats (with whom it interacts in the agonic mode) it is giving a message such as "I am out of action, and therefore not likely to invade your territory, but I will defend to the death this corner of my home territory in which I am cowering." This is ritual submission combined with the threat of unritualised "defence of the nest." aggression. To the home team of the Parkes family (with whom it interacts in the hedonic mode) it is clearly not using the sick role to obtain nurturance, which in fact it refuses. But it could be saying, "I am sick, therefore do not force me out into the competitive arena of life to fight on your behalf. In particular, do not take me to a cat show, I am not up to it." With this message the cat may be transmitting the "off games, out of action" (as opposed to the nurturance-eliciting) component of the sick role signal.

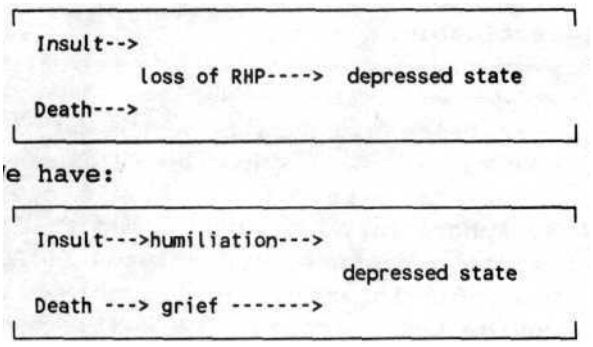
It is interesting that the depressed cat does not give invituations to aggressive play. This may be due to two separate mechanisms. Firstly, all the activities of the hedonic mode are inhibited in depression, so that play is inhibited along with exploration, curiosity, affiliation, etc. Secondly, the threat and attack components of agonic interaction are inhibited in depression, leaving only the escape and submission components active. Aggressive play contains the play element of hedonic interaction and the attack element of agonic interaction and so may be inhibited on both counts.

The death of a baby does not cause a fall of status or RHP, so why (in the ultimate sense) does it sometime cause depression (over and above grief) in the parents? The reality of depressive illness after the death of a baby is beyond doubt. Recently it was brought home to me vividly by a

patient who was so incapacitated by the death of one of her twins that she was quite unable to care for the surviving twin. That is difficult to account for in adaptationist terms. Do we know the incidence of depression following the death of a baby in societies in which death of a baby is a common occurrence? Probably the sequence "death - grief - depression" is not sufficiently fine-grained to distinguish between the death of a powerful ally and the death of a baby. Evolution could have produced two different kinds of grief: one following death of an ally which could trigger depression, and one following death of a baby or other dependant which could not trigger depression; but it has not done so, and all grief-induced depression is probably not adaptive.

In terms of RHP, the yielding hypothesis of depression states that depression is caused by loss of RHP, and the depression itself leads to further loss of RHP, like the devaluation of a currency. Depression following bereavement is a problem for both these propositions. Firstly, as discussed above, the bereavement does not always lead to loss of RHP; secondly, it has been thought since Freud's Mourning and Melancholia that depression following bereavement (mourning) is not associated with lowered self-esteem (including, presumably, the RHP component of self-esteem). Is this still thought to be true? Has there been measurement of self-esteem in depressions following bereavement compared to other depressions? Or can we say that whereas there is no loss of self-esteem during grief, when grief triggers depression loss of self-esteem does occur?

In general, grief is one of the dysphoric emotions which seem to have evolved as part of the causal chain between stimulus and depressed state, others being shame, guilt, anxiety, depressed mood (short of depressed state), disappointment, mental and physical pain, loss of face and the sense of being insulted or put down. These emotions are the subjective correlates of intervening processes between stimulus and mood change. I think what they have in common is the fact that the stimuli or conditions which cause them are sufficiently often associated with loss of RHP that it has been advantageous for them to cause the internal reduction of RHP which seems to be the crucial functional element of depression. Instead of having:



in which humiliation is the subjective correlate of being put down, and grief is the subjective correlate of loss of a loved one. Humiliation and grief share with other dysphoric emotions the capacity for triggering depression. The only difference is that whereas humiliation is always associated with loss of RHP, grief only sometimes is. This second model is less simple and satisfying than the first; but the only alternative that I can see is to postulate that the depressed state following loss is an entirely separate condition not involving changes in RHP.

Finally, Parkes says that nurturance of the sick is unlikely to be an innate human characteristic. There is a lot of variation, but to my knowledge genetic studies have not attempted to analyse it. Are more MZ than DZ twins concordant for nursing? I do not think that other primates nurture the sick. In The Chimpanzees

of Gombe, Jane Goodall describes chimpanzees avoiding and throwing stones at disabled conspecifics, treating the physically ill rather as some humans treat the mentally ill. However, "Cape hunting dogs....will even take care of the sick and injured animals for long periods of time"[6,7]. Perhaps this is because (in the ultimate sense) they share with us hunting, or having a home, or living on the ground; hunting, because injuries in the hunt would be too costly if the injured were not cared for, having a home and living on the ground because otherwise caring for the sick might just not be practicable.

Katie-Price Exchange by Alain Katic

Recently I was asked by Dr Russell Gardner to read John S Price's paper "Metaphors of Submission," and to contribute a response to an issue of ASCAP. The intent of my letter was to provide the reaction of a senior medical student and recent novice to the field of psychiatry. Subsequently I am unsure how helpful, if at all, my contribution will be to Dr Price and other ASCAP readers.

Dr Price's essay is a stimulating and thought provoking manuscript regarding the yielding hypothesis of depression, and specifically an attempt to resolve the problem of how it is that depressed patients get their own way. To this end Dr Price introduced the sick-role metaphor as a solution to why these patients seem to get their way. He goes on to explain how this behavior conveys submission in the agonic mode and at the same time elicits a nurturing response from the receiver who being human tends to interact with such patients in the hedonic mode. Having read the article a number of times and discussed it with Dr. Gardner I felt comfortable in agreeing with the conclusion that "patients getting their own way in the hedonic mode does not negate a hypothesis which states that depression evolved as a losing strategy in agonic interactions." However I was left with the major problem of, 'how does this help me better understand depression, and of what use is it to me in dealing with such patients?'

Having only recently begun my studies in psychiatry I can remember being repeatedly instructed to look for symptoms in a patient which enable a diagnosis, and subsequently a line of therapy. My early studies have also emphasized the intent of the DSM III-R to concentrate specifically on symptomatology and not etiology for depression. So I eagerly read Dr. Price's article as an opportunity for exposure to new ways of viewing psychopathology. I was disappointed to not find any references in Dr. Price's article regarding his actually attempting to put these ideas to use in the psychotherapy of some of his own patients and the outcome of such therapy.

Finally, I'm left feeling that I have ventured into rather new and uncharted territory, not only for me, but for a large number of people already trained in psychiatry as well. Furthermore after having read not only this article, but a number of others on the ethology of psychiatric disorders and particularly the role of RAB and yielding in depression, I find myself less than convinced about just how useful evolutionary biology/psychology is for me at this stage of my education. However, I am grateful for having received exposure to a new and different way of viewing depression and as my training continues and my knowledge and skill in psychotherapy grow I might be able to better understand or even incorporate such theories into my own personal approach to the depressed patient. I regret not being able to make a more helpful contribution.

Price-Katic-Price by J Price

I am grateful to you for giving careful attention to my essay, and I can see from your reply that you have understood it. You are quite right not to get involved with ultimate explanations at this stage of clinical training; observation should come first, then attempted explanations in terms of proximate factors such as physiology, and then (possibly in old age) speculation about ultimate causes (one reason for speculating about adaptive function is to facilitate the search for proximate causes). Moreover, one must bear in mind that the sort of theory we are dealing with here is far from accepted in medical schools and might not be palatable to examiners.

The important thing is to look at behaviour from many perspectives, if not exactly at the same time, at least one after the other. Even within the same category of explanation, such as proximate causes, one should entertain more than one model, even if they appear incompatible, rather as physicists regard light as both waves and particles. Also one should distinguish between the formulations one makes to oneself, and those which one communicates to patients. A diagnosis communicated to a patient is part of the treatment, and is likely to affect how he feels about himself and his compliance with other aspects of treatment. Some depressed patients benefit from being told they are ill; it helps them to slow down and gets their families off their backs. Others benefit from being told they are undergoing involuntary yielding, as it enables them to make an appropriate voluntary yielding response in submitting to the inevitable, and thus escape from the failure to achieve which led to, and may be perpetuating, the depression. Some patients suffer a fall in self-esteem if they are told they have schizophrenia; others are helped because it enables them to join organisations such as the Schizophrenia Fellowship and to obtain certain welfare benefits.

An understanding of how and why a certain behaviour (or illness) evolved is part of its total explanation, along with explanations in terms of physiology, learning and other "proximate" mechanisms. Some people think I put too much emphasis on yielding, but one should remember Darwin's point that in social animals it is a social process which usually decides who succumbs to the forces of natural selection, and until very recently, this social process took the form of agonistic behaviour of which yielding in some form or another is an essential part. In every generation for three hundred million years, our ancestors have won in this social game, while their brothers and first cousins have yielded; and this yielding on the part of close relatives allowed our ancestors to compete successfully against their second and third (etc) cousins; so successfully, in fact, that the inclusive fitness of our yielding and non-reproducing brothers and first-cousins was actually increased with the result that yielding strategies have been consistently selected and have finally descended to us; therefore, it should not be surprising if primitive mechanisms for yielding are built into the human genome. It is these primitive yielding strategies which I think are being manifested in some forms of depressive and anxiety states.

More modern methods of yielding are also very apparent in human life. If yielding were not a crucial issue in evolution, it would be very surprising that most world religions are basically concerned with submission and annihilation of the self. The same is true of much philosophy. And codes of politeness are concerned with submission and with the under-

statement of the self. Could I not legitimately end this reply by signing myself, 'Your obedient servant'? In using these sophisticated cultural forms of yielding, we are avoiding the agonistic confrontations which historically have ended in depressed mood and reduced reproduction in one of the parties to the conflict. To the extent that these cultural strategies pre-empt depressive illness, they are a form of prophylactic psychiatry.

I would like to illustrate to you how this phylogenetic approach can contribute to the clinical management of depressed patients and also to research into the neurophysiology of depression.

First of all the treatment. You will have seen from previous contributions to ASCAP that the yielding hypothesis does have implications for treatment, in that it suggests that the involuntary yielding of depression can be replaced by the voluntary yielding of submission. The first act of submission that the depressed patient makes is to put himself in the hands of the doctor, or at least to agree to relatives taking him to the doctor. In the severely depressed patient, this nascent tendency to submission should be encouraged, and the patient should be advised to abdicate all responsibility for his progress, and to retain only the responsibility to follow the doctor's advice. In particular, he should be encouraged to stop 'fighting the illness' and to stop trying to make himself well by an act of will (thus helping him to get out of a vicious circle of failure and increasing depression). I tell the patient that depression is like a 'sprained brain', that decisions and responsibility are to the brain what movement is to the ankle, and that the patient should consider his brain to be in a plaster cast until further notice. I then allow the patient to demonstrate his submission by carrying out simple set tasks such as completing a daily record of the intensity of depressive symptoms. I then acknowledge this submission and confirm to the patient that since he is following my advice he will get better. Meanwhile in the context of the therapeutic relationship one is exploring his attitudes, goals and lifestyle and trying to identify areas of pathological non-yielding. The skill of therapy lies not so much in identifying these areas of blocked or incomplete yielding but in reframing them in such a way that the patient gets out of the situation of 'irresistable force meets immovable object'. When the patient recovers from the depression, there is seldom any difficulty in getting him to take over responsibility for himself again, to examine options and take decisions; then the role of the therapist switches from director to that of counselor or sounding board, and the previous asymmetry of power in the relationship is much reduced. Many physicians instinctively encourage this state of temporary dependency in the treatment of an episode of depression. All we are doing by supplying the evolutionary perspective is to explain why what they are doing is helpful, and to enable them to do it more insightfully and thus more efficiently. I send a case history to illustrate what I have been saying.[8]

Secondly, research. I have a hunch that the fantastic proliferation of complex forms of life six hundred million years ago was due to the development of sexual selection and the reason that the vertebrates survived was that their method of intrasexual selection (ritual agonistic behaviour) was so efficient. And since there is no reason for this method even to have died out and thus been reevolved from different components, the likelihood is that the mechanisms underlying all forms of

vertebrate agonistic behaviour are homologous. And that includes the mechanisms of underlying yielding behaviour. Therefore the physiological changes underlying the development of depression in a human being may be the same as those underlying retreat in a defeated lizard. It makes more sense to explore the mechanisms in the lizard than in the depressed patient, not only because the lizard is not required to give informed consent, but because many lizards in this state change the colour of their skin to that of the immature form. The central mechanisms of defeat reach out to the periphery, providing a string which the researcher might be able to follow back to the centre. Many fish such as the green sunfish change their skin colour to match their social rank. And in other fish which change sex at a certain stage of their lives, the sex change is inhibited by the behaviour of a dominant fish. Should we not be encouraging research into this sort of phenomenon if we think that the behaviour of dominant human beings influence the health of their subordinates?

Visible physiological changes with rank are not so common in mammals, but there is a monkey which offers itself as an experimental model in this respect. The East African variety of vervet monkey has a bright blue scrotal skin which is probably a signal of dominance, because when an animal falls in rank the scrotum turns white. Colleagues and I were able to show that the colour change was due to hydration of the dermis, thus abolishing the optical conditions necessary for the Tyndall blue[10]. Blue skin is common in monkeys in various parts of the body, but there is only one area of monkey skin which is regularly subjected to hydration, and that is the genital skin. In the female baboon and chimpanzee, the genital skin becomes hydrated at oestrus and *the swelling* (not a color change) acts as a signal to the male. It seems likely that in the vervet monkey two adaptations have joined together to form a very effective signal, on the one hand the blue skin which is a widespread characteristic of the forest-dwelling guenons, and on the other the simian capacity for phasic hydration of the genital skin. Thus whereas in the baboon female the genital swelling signals oestrus to the opposite sex, in the vervet male the switch of scrotal skin colour to white signals yielding to other males. We were able to show that the gential skin hydration of the vervet was not mediated by sex steroids nor by the other common adrenal cortical hormones. It seemed likely that some other hormone was responsible, and that it might also be affecting behaviour in the way that oestrogen causes both the genital swelling and the sexually receptive behaviour. However, the funding for this research was withdrawn on the grounds that it was "out of line with current thinking" and so our search for a 'yielding' hormone was brought to a close. Hopefully the 'across-species' evolutionary approach to psychiatry which is fostered by ASCAP may help to change current thinking.

Where possible, theories should be testable (refutable) and parsimonious. Evolutionary theories have difficulty satisfying these requirements. But there is a third criterion of theory-making, that the theory should be heuristic; that is, it should lead to more effective exploration of the complexity of nature, and thus lead to testable postulates which otherwise explore the yielding hypothesis of depression (and other evolutionary theories) in the sort of informal debate which ASCAP makes possible. *[Editor's note: compare this point to those made by Crick on p3 on 'general ideas' and 'a good model'].*

1. Hoy RR: Evolutionary innovation in behavior and speciation: opportunities for behavioral neuroethology. Brain Behav Evol 1990;36:141-153.

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.

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