Newsletter of the Society for Sociophysiological Integration

ASCAP

Volume 8, No 4, (Cumulative #89) April 1995

"Perhaps the most widespread misconception about the molecular evolutionary studies is that they represent reductionistic attempts to explain evolution in terms of genic-level properties and causes. ... evolutionary patterns and processes [are] revealed at all levels of biological complexity by molecular markers." *Allan Larson*[1]

Newsletter Aims
•**A** free exchange of letters, notes, articles, essays or ideas in brief format.
•Elaboration of others' ideas.
•Keeping up with productions, events, and other news.
•Proposals for new initiatives, joint research endeavors, etc.

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ASCAP Society 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 cellular processes to individuals in groups. The ASCAP Newsletter is a function of the ASCAP society.

Contents

ADDRESSED TO & FROM

The second annual ASCAP Society meeting will take place

Tuesday, June 27,1995
from 9 a.m. to 5 p.m.

at the
Best Western South Coast Inn
in the beautiful Goleta Valley
5620 Calle Real Santa Barbara/Goleta
California 93117

ASCAP SOCIETY MEETING ARRANGEMENT

We have reserved a meeting room for June 27. For those dovetailing this meeting with the Human Evolution and Behavior Society meeting later in the week, a move could take place the next day (June **28** is the beginning of HBES). Or one could stay at the hotel for the next meeting if that is a preference. The South Coast Inn is about two miles from the UC Santa Barbara campus, just inland near the highway to the university.

Make your reservations now! See FAX form included with the newsletter. You are reasonably guaranteed a room if your reservations are made prior to mid-April. **Phone is (800) 350-3614 or (805) 967-3200; FAX (805) 683-4466.**

Rooms are $80.30 per night including tax and is the same for singles or a pair. There is a free continental breakfast along with pool and spa. Specify that you desire the special UCSB rate as you will be attending a conference in conjunction with UCSB (this presumes that you will attend the HBES conference in addition to the ASCAP meeting). An additional room is already reserved for the winner of the Aaron T Beck ASCAP Award competition for June 26 and 27.

The conference times will include registration from 8:30 a.m. to 9:00 a.m. and scientific sessions for six hours in the form of brief presentations by many of us, group discussion, and review of the discussions of the Internal Working Models of Bowlby as elaborated by Dan G. Freedman and the Involuntary Subordinate Strategy of Price, Sloman, Gardner and Gilbert as discussed by John Price. Both stimulus papers were in the February ASCAP Newsletter. The Beck Awardee will also present the winning essay.

These activities will take place from 9 to 10:30 a.m., 10:45 a.m. to 12:15 p.m., 1:15 to 2:45 p.m., and from 3 to 4:30 p.m. A business meeting will go from 4:30 for as long as needed (future meeting sites and dates, meeting formats, guidance for the newsletter, review of the Beck competition, etc.). Thus there will be two coffee breaks (furnished by the hotel) and a luncheon (on your own). Six hours of credit may be earned for the scientific sessions.

For our planning purposes **please - as soon as you know - register your intent to come with Erica Ainsbury TEL: (409) 772-7029; FAX: (409) 772-6771; erica.ainsbury@utmb.edu.**

I am working with UTMB for Category I Continuing Medical Education credits. (Credits for clinical psychologists may also be pursued, if there exists need for them.) This requires a statement of faculty, goals and objectives: faculty whom I know at this time from informal communications to

be coming include Aaron T. Beck, M.D., Daniel G. Freedman, Ph.D., Seymour Itzkoff, Ph.D., John Pearce, M.D., Leon Sloman, M.D., Daniel Wilson, M.D., Kent Bailey, Ph.D., Mark Erickson, M.D., Russell Gardner, M.D., Tim Miller, Ph.D., and Nancy Segal, Ph.D.

The conference goal focuses upon the integration of the science and realities of biology on all levels to normal human relating and communication as these relate to psychopathology and its treatment.

Specific objectives include that the conference participants will, upon conclusion of their attendance at this meeting, be able to:

1. Define internal working model, involuntary subordinate strategy, basic plans, hedonic and agonic modes, sociophysiology, resource holding potential, paleopsychology, paleopsychopathology, social attention holding potential/power, familial bonding, psychiatric epidemiology, triune brain, psalic, era of evolutionary adaptation (EEA), Harveian basic science, the two axes of relating and the interpersonal octagon.
2. Recapitulate Aaron T. Beck's evolutionary model for depression.
3. Formulate the shiver-ATP modification of cognitive-behavioral psychotherapy (ATP = Allies, Thought & Planning) and illustrate how it has worked in patients.
4. Describe the healing mechanism of audienceship (listening to one's patient) via the biology of leadership.
5. Summarize the Itzkoff formulation of evidences of intelligence differences amongst human groups and its implications.
6. Rationalize need for a basic science of psychiatry.
7. Summarize examination of involuntary subordinate strategy from perspectives in psychiatry and clinical psychology, including cognitive-behavioral, psychoanalytic and developmental.
8. Outline an approach to evolutionary psychotherapy that focuses upon the "ordinariness" of experience.
9. In evaluating the role of genetics in thinking and relating, describe the role of twin studies.
10. Outline evolutionary reasons that people are dissatisfied with their status in life, even if well off.
11. Note how the theory of Darwinian medicine has implications for restraint in treatment recommendations.
12. Illustrate the use of mismatch algorithms and quantitative formulae for understanding anorexia nervosa and other illnesses.

Russell Gardner, Jr.
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PLANS FOR HBES MEETING 1995

I would like to see panel discussion organized for HBES on a particular topic and then invite specific people to present on selected aspects. I feel that this would create a nicely cohesive group in the audience. There might be room to include some who want to volunteer to participate. Possible topics could include: (1) integrating the biological, physiological and psychological models of depression (this could include phytogeny and ontogeny); or (2) biological approaches in psychotherapy; or (3) adaptive mechanisms in depression.

We would have to run our proposal by the HBES conference organizers to get their agreement. I would support keeping an ASCAP business meeting as short as possible, but if we have ASCAP members who have travelled a long distance to attend, one might want to give them the opportunity to describe developments in their own geographic area. The idea of going out to a restaurant the evening of our June 27 meeting sounds lovely -- this should provide an excellent additional forum for informal discussion.

Leon Sloman
Ontario, CANADA

THANK YOU

Thank you for your letter asking me to write a 500 word response *[to Freedman and Price, February issue]* for your newsletter.

I am sympathetic to the mission of ASCAP and I appreciate your selecting me to write the response. I am afraid, however, that I am too overcommitted with writing responsibilities to take on the task at this time.

Nancy C Andreasen
Iowa City IA, USA

SUPPORTER

I have become increasingly guilty about not responding to your letter and kindness in sending copies of The ASCAP Newsletter. I had

planned some kind of measured reply. But, as yet, I must confess I've not managed to come to terms with the appeal of the general field and my concern about the relative lack of supporting research. But I'm very much a supporter what you are doing; and at very least I find many of the ideas of considerable heuristic value. I send a current paper *[Editor's Note: See abstract on page 19*] that might interest you where the literature was helpful in getting clear ideas which we had had for some time - ideas that had arisen from our experience with looking at 'life events' and depression. (It is of interest that a recent study of a black sample in Zimbabwe has arrived at very similar results.)

George Brown
London, ENGLAND

VARIOUS ASCAP MATTERS

Your account of the Acapulco meeting made excellent reading. All those alphas in hedonic "companionable interaction"! It emphasises that one of the essential requirements for leadership is the capacity to take a back seat and refrain from "conspicuous participation" when circumstances demand it - one of the things that manic patients have difficulty doing. If only one had Superman's X-ray eyes and could see right through to their red nuclei and see them glowing with the neural substrate of alpha activity.

At a seminar with the registrars the other day I asked them about the attachment behaviour of the Tasmanian seagull. All along the shoreline here are gulls of the usual grey sort, and many of them are in pairs with one clearly adolescent bird and one parent. The young one is frequently giving a sustained "cheep, cheep, cheep" (which must be an attachment signal) and orienting in the direction of the parent, while at the same time adopting a curious posture in which it makes its body horizontal, and fluffs up the feathers on its back so that they look as though someone has stroked them the wrong way. Meanwhile the parent walks around totally ignoring its offspring. This goes on for a long time. The offspring have black beaks and legs, while the adult birds are pink - I suppose they have had time to absorb more red pigment from shrimps.

The registrars did not know about these birds, but we got on to human attachment signals, and wondered why the human baby has such a small repertory of distress cries. Why does it not have a separate cry for being hungry, another for being cold, another for being in pain, another for having wind, and so on. Some of the registrars seemed to think they could interpret the cries better than others, who found the cry most ambiguous. Since the baby has such a large brain at its command, surely evolution could have arranged for different cries to express different types of discomfort, to help the mother to make appropriate ministrations. In answer to this problem, one registrar pointed out that babies should be born at 22 months, and that birth has been brought forward because of the pelvic outlet constraint. Perhaps three million years ago babies were born at 22 months, so before that state of brain maturation there was no call for attachment signals. Since the advanced birth date evolved, there has not been time for evolution to distinguish the signal, probably because the infant death rate from signal misinterpretation is not very high.

I thought this was a good idea, and suggested the alternative idea that the ambiguous signal of the baby's distress had the function of discomfiting the mother and making her feel guilty and inadequate, for which she compensated by devoting more resources to the baby.

Has anyone done any work on the interpretation of babies' crying by mothers? And is there any evidence that at 22 months the quality of signal improves more than would be expected from continuous maturation?

I am glad you are finding the shivering model useful. There is another example in the essay I append on a good new book on self-esteem (though it totally lacks an evolutionary perspective). *[Editor's Note: Look for this in a future issue.]* I will try ATP in Hobart!

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ARTICLE: *Both* Freedman's IWM and Price's ISS: Five principles of hypercomplexity

by R Masters

Ockham's Razor has been worn out by six centuries of use. Nature - and especially human nature - is not rigidly governed by the law of parsimony. In fact, the principles of evolution almost guarantee that living beings will be hypercomplex. These broad generalizations provide a useful basis when thinking about Dan Freedman's Internal Working Model and John Price's Involuntary Subordinate Strategy (see February issue).

We are thoroughly accustomed to the method of exclusion. Even those who have gone beyond "nature versus nurture" habitually seek to dismiss an explanatory variable as soon as it can be reduced to or explained by another level of analysis. I think that contemporary neuroscience, ethology, and evolutionary theory should lead us to take the opposite view. Usually, the answer will be *both ... and.*

From this perspective, I'd like to propose five principles that might explain why it's valuable to look at *both* Freedman's Internal Working Models *and* Price's Involuntary Subordinate Strategies together. Precisely because they seem almost incompatible, these two approaches to social behavior fit together in a remarkably valuable manner.

First, then, five principles and their implications for human and nonhuman social behavior. I'll spare the footnotes.

Modularity

The central nervous system is structured into functionally specialized modules. The Lockean theory of the *tabula rasa,* and its extension to the stimulus-response model of behaviorist psychology, was simply wrong. Because virtually all modern social science was built on this foundation, we have to go back to an older epistemological tradition; diverse features of behavior will typically be modulated by distinct and irreducible structures and processes in the brain. For example, visual and vocal nonverbal social cues are processed by different structures in the brain. Indeed, even in a single sensory pathway like vision, there are dedicated structures for perceiving and processing the facial displays, bodily movement, etc.

Parallel Processing

The diverse modules in the brain function simultaneously and in parallel. In the visual pathway, for example, facial display behavior is processed simultaneously by at least two pathways: ventral (through the inferior temporal lobes) and dorsal (through the neocortex). As a result, an expression of anger and reassurance by a known conspecific elicits *both* an immediate emotional response primarily related to the cue-value of the display *and* a mediated response dependent on the prior relationship between the interacting individuals. Moreover, both pathways are sensitive to the prior state of the organism. In human terms, emotional reactions occur independently of cognition *and* cognition mediates emotional response.

Mapping

Both environmental stimuli and sensorimotor output are mapped in brain structures. These mappings are subject to reshaping and modulation by early experience and training, but once in place, they provide the matrix within which all perception and behavior take place. Obvious in the famous deprivation experiments (e.g., cats unable to see curved lines if reared from birth in environments without this cue), this principle explains imprinting, critical periods, and social learning. In humans, it also accounts for such phenomena as the cultural differences in phonemic and proxemic perception and response. Dan Freedman's IWM obviously reflects mapping at a higher order of social behavior and strategy.

Tuning

The modular, parallel processing systems of the brain are modulated and controlled by complex biochemical systems. Neurotransmitters and their related regulatory molecules -- precursors, enzymes,

receptors, metabolites, blockers, reuptake inhibitors, and the like - function to modulate signal/noise ratios. Any given neurotransmitter system depends, for its function, not only on the specific neuroanatomical structures it subserves, but on the levels of other neurotransmitters *and* on the overall milieu of brain biochemistry. John Price's ISS obviously reflects systems at the neuroanatomical neurotransmitter level that have evolved to control mammalian social behavior.

Chaos Theory

The brain is a nonlinear dynamic system. What seems to be blooming confusion is often simplified by the *Winner Take All* principle, modeled most effectively as a "basin of attraction" or "strange attractor". For example, positive or hedonic emotions are processed independently of and simultaneously with negative, agonic feelings; the stronger response tends to establish the value of the experience for purposes of associative learning, memory, cognitive processing, and memory.

These principles help explain both Freedman's IWM and Price's ISS. They justify the fact that both seem to contain an element of truth, while neither is fully reducible to the other. *Both* cognitive mapping (IWMs) *and* innate submissive routines preprogrammed in neuroanatomy and neurochemistry (ISSs) are evident in my own experimental studies of the way viewers perceive leaders, respond emotionally to their nonverbal and verbal behavior, and form or modify cognitive attitudes.

If this approach is correct, there is a small but crucial change that needs to be made in both Freedman's concept of the IWM and Price's approach to the ISS. The singular nouns should be replaced by plurals. All humans have multiple *Internal Working Models* of ongoing social behavior: when encountering a social situation, we have to figure out which IWM (or "strange attractor") is relevant. Mammals have multiple *Involuntary Submissive Strategies,* not only because there are different responses available in the repertoire (e.g., depending on whether escape is possible or not), but because both genetic and developmental constraints may lead to behavioral polymorphism in the preferred strategies of different individuals.

My undergraduate students have reduced these principles to Masters' Three Laws of Behavior. First, "it's more complicated." Second, "it's even more complicated than that." And third, "the first two laws are overly optimistic." Or, for those who want to go back to Ockham and reduce it all to a single formula: "it's nature *and* nurture, stupid!"

References: page 22 .

ARTICLE:

by D Wilson

Ethological studies of subjectivity in feral vs. domesticated canines: Cognitive implications

Freedman's essay "Ethological studies of subjectivity: The Internal Working Model" in Volume 8, #2, is cogent and persuasive. I have always admired his body of work. Still, I have a few sidebar comments.

First of all, I doubt that anyone else in the world could have read it with as much personal amusement as did I. Like most ASCAPians, I am a dyed-in-the-wool clinician/anthropologist. But our family long has had an affection for the Basenjis which have been esteemed members of our household(s) for nigh-on three human generations. Tempting though it may be, I shall refrain from defending the many Basenji virtues, which, I must say, are insufficiently mentioned in Freedman's otherwise splendid work! Instead let me merely note what I think is factual error and then briefly address secondary issues of methodology and interpretation which this error might raise.

Basenjis are a breed which arose naturally like the Australian dingo. The name is a Swahili conjunction for "monkey like face, curly tail". As a feral type, Basenjis enter heat only once per year; even in suburban America they instinctively tamp down the "African underbrush" before curling up to sleep; they do not bark but rather yodel, especially, in the context of their dominance hierarchies which are robust. There are also three naturally occurring races each with slightly distinct behavioral tendencies: red, black and the (rare) stippled. All are often noted to exhibit cat-like behavior: self-washing, nimble branch-climbing and a truly feline aloofness. They are thought to be faster than Greyhounds. They are also deeply social and proud. So much so that the treatment of choice for an ill-disciplined Basenji is to acquire more of them! They then settle into a calmer mode of authentic canine dominance hierarchy. No pseudo dog (i.e., human) leaders will suffice for these pups.

All curious enough. But the key point being that, unlike most other dogs including the others compared in Freedman's study, Basenjis are the product of *natural* not artificial selection. Of course, Darwin himself emphasized important and distinct consequences of these two modes by which genomic traits differentially accumulate.

None of this necessarily detracts from Freedman's work or his conclusions. However, it may open up other avenues of discussion. My own sense - based on considerable "open-label, non-placebo controlled" empirical work with Basenjis -- is that they retain a more deeply cannulated basic plan as to what a Basenji is and is not. Most humans are, to their eyes, not. Their range of phenotypic reactivity is less able to include humans as dominators of their Internal Working Models. Indeed, the assumption that Basenjis subjectively incline to recognize humans as dominant due to size and control of resources is far less plausible than it seems. It is, after all, a little known fact that Basenjis are one of the few predators of big cats (tigers or lions, I believe). Packs of Basenjis have been reported to chase down the big kitties. Their remarkable fleet-footedness, group morale and cooperation allows them to best the big cats. One of a few dogs nip at the heels of the cat to annoy it in the chase. Eventually, the cat turns to pounce on the nipper but instead the other dogs then lunge at the throat of the cat and, sometimes, kill it.

Here we have the African genesis of kinship-selection, altruism, social carnivory, division of labor and other elements so reminiscent of what we know concerning human evolution. As it happens, Basenjis also evolved a canine anemia quite in parallel to human sickle-cell disease.

Put otherwise, mechanisms of Basenji imprinting and internal working models are perhaps more similar to other dog varieties than it seems. They are just less easily duped into a phylogenetically false position of subordinance to humans. Bailey and Roswell's (ASCAP Vol 7 #12) concepts of mismatch are of considerable relevance, by the way.

Thus one must look more closely at Basenji "in-group" behavior to ascertain their indulgent or disciplinary tendencies. That they do not comparably respond to unauthentic (human) dominants does not fully inform us as to their capacities for infra-species indulgence or discipline. For example, on the "incorporation of punishment test", I suspect the internal Basenji dialogue did not keen upon whether "was the handler present as an internal representation in his physical absence". Rather, "who does this non-Basenji think he is and where is my real pack?". The other dogs, having more readily entered into the cognitive realm of human dominance, behave in ways more consistent with the guiding hypothesis and expected finding of the experiment. They seem to say "what's up, chief?". In contemporary parlance they are clearly more "multicultural" than Basenjis, but it remains unclear whether they are actually more imprintable.

To summarize, Freedman's research has been a benchmark for some decades. His informal extension of the data toward new themes is worthwhile. Canine models support his notions of Internal Working Models. Basenji data is subject to unique interpretation.

ARTICLE: Why there are eight primary emotions[1]

by E A Salzen

There is a widespread assumption in theories of emotion that there exists a small set of basic emotions. Table 1 shows a selection overtime of such categories empirically determined from judgements of emotional behaviour.

Table 1. Categories of Emotion based on Facial Expression

Salzen[2]	Ekman *et al.*[3]	Tomkins[4] Izard[5]	Allport[6]
Happiness	Happiness	Enjoyment/Joy	Pleasure
Surprise	Surprise	Surprise/Startle	
Fear	Fear	Fear/Terror	Surprise/Fear
Sadness	Sadness		
Distress		Distress/Anguish	Pain/Grief
Anger	Anger	Anger/Rage Anger	
Disgust	Disgust/Contempt	Contempt/Disgust	Disgust
Interest/Desire	Interest	Interest/Excitement	Attitude
		Shame/Humiliation	

Table 2. Thwarting Situations

I Absence of indispensible stimuli following intense arousal
II Simple physical obstruction of aroused activity
III Simultaneous arousal of two or more incompatible tendencies

PRIMARY RESPONSES TO THWARTING

SOMATIC

1 *Perseverance* - persistent approach and adjustment
2 *Snap Decision* - capricious choice of response
3 *Threshold Intention Movements* - initial element of response (I, II)
4 *Ambivalent Posturing* - elements of both responses (III)
5 *Alternating Intention Movements* - successive responses (III)

AUTONOMIC

1 *Alimentary* - salivation increase or urination, defecation
2 *Circulatory* - pallor, flushing, genital vasodilation, fainting
3 *Respiratory* - changes in rate and amplitude, panting, gasps, sighs
4 *Thermoregulatory* - sweating, raised/sleeked hair
5 *Lacrimatory* - weeping

SECONDARY RESPONSES TO THWARTING

1 *Displacement Activities* - irrelevant behaviour
2 *Redirection Activities* - response to another stimulus
3 *Regressive Activities* - immature resonses
4 *Neurotic Inactivity* - loss of responsiveness
5 *Aggressive Behaviour* - intense approach and adjustment
6 *Stereotypic Activities* - repetitive movement patterns
7 *Visceral Dysfunction* - chronic autonomic imbalances

Table 1 shows the categories recognised in several major reviews of facial expression studies. Izard is alone in including the category of "shame/humiliation".[5] But this could be explained as a mixed expression of social approach and withdrawal representing a combination of the primary categories of Interest and Fear.[6] Allport is alone in making Surprise and Fear a single category. Allport and Izard recognise Distress but not Sadness. Ekman *et al.* do the converse,[3] while Salzen includes them both but as separate categories.[2] With these provisos, the correspondences and agreement in Table 1 become clear and it seems reasonable to say that there appear to be no more than 8 commonly recognised basic or primary categories of emotion.

Few theories even attempt to explain either the existence or nature of such categories. In a recent article in Psychological Reviews Ortony and Turner examined the evidence for the existence of primary emotions and concluded that the assumption cannot be upheld but that this does not mean that there might not be basic elements out of which different emotions are built.[7] They suggest that the elements may be components of cognitions, feelings, and responses.

For some time I have been trying to promulgate a theory of emotion based on elemental responses and their feelings.[8,9] This theory not only implies that there are no basic categories, but it can explain why, despite Ortony and Turner's conclusion, common experience so often results in the recognition of about the same number (6-8) of the same categories of primary emotion. Thus the full title of this talk should be "Why there *appear to be* about eight categories of primary emotions".

The theory is based on an ethological analysis of animal displays that seem to correspond with human emotional displays (courtship, parenting, agonistic, alarm, etc.) It uses the analysis of the origin and evolution of social signaling in animals given by Desmond Morris.[10]

Table 2 is a modified and extended version of a table from Morris' paper. It defines thwarting and summarises the behavioural consequences of thwarting. The Primary responses to thwarting are the intention movements and the necessary orientation behaviour of the aroused activities, their combinations in conflict, and their accompanying supportive visceral system arousal states. Certain other Secondary Response patterns may result if the Primary responses fail to bring about the end-of-thwarting and thwarting is persistent or chronic.

Thus an oversimplified statement of the theory would say that (c.f. Table 3):

Emotional behaviour can be understood as the responses to conflict, thwarting, and release from thwarting, of aroused motivational action states.

The responses may come to form displays that induce the social partner to behave so as to end the thwarting and facilitate the performance of the aroused consummatory activity.

Emotional expressions, therefore, are the social signals of thwarted action states or of their release from thwarting.

Table 3. Thwarted State Signalling Theory of Emotion

Emotional behaviour can be understood as the responses to conflict, thwarting, and release from thwarting, and release from thwarting, of aroused motivational action states.

These responses may form *displays* that induce the social partner to behave so as to end the thwarting and facilitate the performance of the aroused consummatory activity.

Emotional expressions, therefore, are the social signals of thwarted action states or of their release from thwarting.

Differently motivated behaviours may use the same appetitive or aversive orienting actions so that these actions can signal only the *general category of emotion.*

The *specific quality or nuance of the emotion* is conveyed by the Intention movements of the consummatory acts of the specific aroused motivation.

A few theories of emotion have tried to account for specific categories of emotion by equating them with specific classes of motivated behaviour e.g., sex, parenting.[11,12]

[These earlier theorists regard the emotions as accompaniments of ongoing instinctive behaviours which they group into distinct classes. My own theory is quite different in saying that emotion is the *thwarted behaviour* and that the classes are based on classes of orientation behaviour and their state of activation and not on the class of the aroused consummatory or instinctive behaviour. The analysis I made in 1981 was based on an ethological analysis of human facial expressions and is quite consistent with the theoretical analysis being presented now.]

Yet what is distinctive (and odd) about categories of emotion is that they apply across motivational classes. Thus desire or love are emotional terms that are applied to food, comfort, sex, parenting, safety, curiosity, etc. This can be explained by thwarting theory because: *Differently motivated behaviours may use the same appetitive or aversive orienting actions,* such as seeking or fleeing, orienting to or from, or adjusting the goal stimulus itself, so that these actions can signal only the general category of emotion.

The specific quality or nuance of the emotion (cupboard love, sexual love, motherly love, brotherly love, etc.) *is conveyed by the Intention movements of the consummatory acts of the specific aroused motivation.* Without such specific intention movements the perceiver must rely on the nature of the arousing stimulus or situation to judge the specific goal and hence the specific quality of the emotion.

The possible classes of orientation responses in relation to stimulus objects is quite limited and this accounts for the limited number of general categories of emotion. Table 4 (page 10) shows these possibilities along with the categories of emotion for which they can account. They are as follows:

The arousing stimulus may be at a distance or more or less in contact with the body, and different response patterns must result. If the "Distal" stimulus fails to arouse a specific motivated action state and the stimulus is unidentified or "Novel" it evokes

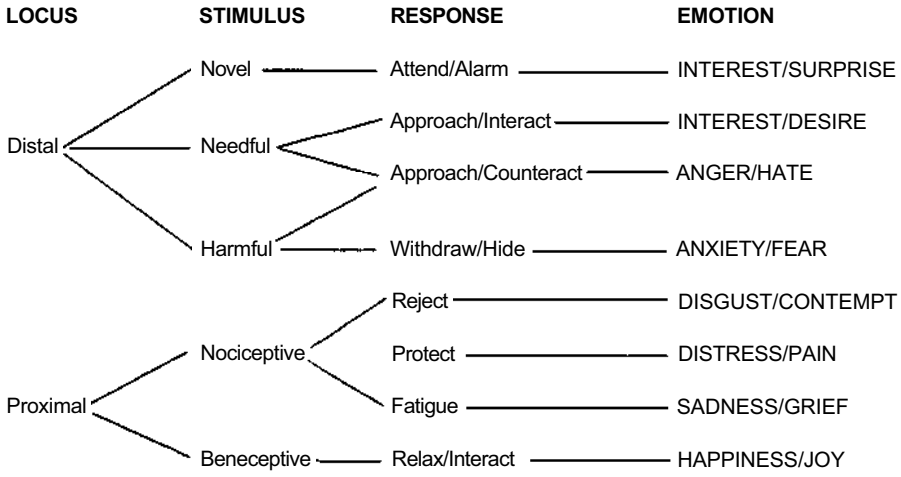
patterns of attention labelled "Attend" in Table 4. If the stimulation is sudden or at high intensity this attention response may be arrested in an "Alarm" display. These responses correspond with the emotion category of Interest/Surprise. (It is arguable whether Interest and Surprise are "really" emotions or simply the precursors to emotions once the source of novelty has been identified. But both can serve as social signals of an interrupted action state and can be included in the present emotion concept.

If the "Distal" stimulus arouses motivated action states that require close proximity or contact for their performance then the stimulus may be said to be "Needful" and the orientation will be approach behaviour. This appetitive approach will be to interact in a positive manner, i.e., to perform consummatory behaviour, and is labelled "Approach to Interact" in Table 4. When thwarted it corresponds with an emotion category that I have labelled in Table 4 "Interest/Desire" (this is a specific interest unlike the general interest in a novel stimulus). However, intense approach behaviour may lead to actions that forcibly alter the arousing but thwarting stimulus - by re-orienting it and changing its behaviour or characteristics. This is called "Approach to Counteract" in Table 4. When thwarted and coupled with intention movements of the forcible counteraction (attacking, striking, biting, shaking, pushing, etc.), it is clearly seen as corresponding with the emotion category of Anger/Hate. This conceives of aggressive behaviour as attempts to re-orient, readjust, and alter the stimulus so as to end thwarting.

If the Distal stimulus arouses actions of defensive or protective (pain) responses then the orientation behaviour will be aversive in the form of withdrawal from the stimulus as unwanted or potentially noxious. The stimulus is accordingly labelled "Harmful" in Table 4 and the aversive response "Withdraw" includes avoidance, flight, escape and hiding. The emotion category of Anxiety/Fear then corresponds with the thwarting or anticipated thwarting of these withdrawal responses. This can turn to defensive aggression when flight is blocked and approach to pass or remove the stimulus is the only orientation behaviour left.

If the arousing stimulus is close or in contact ("Proximal") then it will give either nociceptor or beneceptor responses, i.e., the stimulus may be physiologically "Nociceptive" or "Beneceptive" c.f. Table 4. The nociceptor responses may initially be actions that remove the noxious stimulus from the body surface and vicinity. These actions are labelled "Reject" in Table 4 and, when thwarted, correspond behaviourial and perceptually with the emotion category of Disgust/Contempt. Strong or persistent nociception may lead to intense flexor and protective reflexes characteristic of pain. I have labelled these "Protect" responses, and when thwarted, they correspond

Table 4. Stimulus-Response Classes and Categories of Emotion



behaviourally and perceptually with the emotion category of Distress/Pain. If nociception persists despite the Reject and Protect responses then physical fatigue may become evident and this response, which I have labelled "Fatigue" in Table 4, combines with the residual thwarted nociceptive response patterns to give the appearance and perception that corresponds with the emotion category of Sadness/Grief. (Persistent failure of distant stimulus-response orientation may also lead to fatigue and the resulting pattern corresponds with sadness and despair. However, persistent proximate noxious stimulation, which can be in the form of images and thoughts, is most likely to lead to fatigue and the concomitant patterns of sadness, grief and despair.)

Finally, if the proximal stimulus operates on beneceptors the resulting behaviour will be the cessation of appetitive or aversive orientation responses and the onset of consummatory responses and interactions. I have labelled this pattern "Relax/Interact" in Table 4. It represents the rapid decline of the appetitive/aversive behaviour with high arousal and tension and with a predominance of sympathetic nervous activation and the onset of consummatory actions or quiescent resting states with relaxation or relief from tension and with lower arousal and a predominance of parasympathetic nervous activation. It corresponds behaviourally and perceptually with the emotion category of Happiness/Joy which normally does not outlast this changeover unless re-evoked in memory, but gives place to Feelings of pleasure which are the perceptions of the consummatory actions, stimuli and motivational state, and which give the happiness and joy specific qualities.

These relaxation states can become anticipatory in thwarted approach and withdrawal from distal stimuli, so that relief happiness is not confined to proximal stimulus interactions.

The theory of emotion as the social signalling of thwarting and the end-of-thwarting is not only consistent with categories of emotion but is also a powerful explanatory concept that can account for many other known aspects of and findings on emotion and its development and possible evolution. Some of these have been outlined in previous publications.[8,2,9] A fuller account which includes the implications of the theory for the development and nature of self-awareness and self-control and for the neural systems involved in emotion is to be published soon in the International Journal for Comparative Psychology.

References: page 22

ARTICLE: Mania and winning: A problem in the ranks?

by P Gilbert

Leon Sloman and Russell Gardner have entered a fascinating dialogue to which I would like to add a couple of points (see letters section of January 1995 issue). The ranking theory of affective illness (as developed by John Price) has suggested that mania and depression are related to the same mechanism -- the one that controls challenge behaviour. I would like to quote from a paper I wrote a couple of years ago.[1] I looked at attachment theory (protest-despair) as a mechanism for depression and then suggested:

... sexual selection theory suggests another possibility. This is that animals have to be able to signal their readiness for engaging in conflict and competing for breeding resources. There seem to be two basic strategies.[2] High-risk high-gain (here the animal can be high profile and aggressive or charismatic, in which it might do well early in its career, but poorly later if deposed) versus a low-risk low-gain (have small successes but over a longer time). The low-risk low-gain strategy involves internal inhibition of aspiration and challenge. In this theory, depression

results from the activation of some internal control mechanism(s) that evolved to inhibit animals low in rank/status or in losing (uncontrollable) positions from taking on challenges they would lose.[3,4] Mania may be an amplification of the high-gain high-risk strategy; bipolar illness suggests major ratchet-like oscillations rather than mild transitions between these two basic strategies. We know that normal mood shows mild cyclic oscillation.[5] It is my clinical impression that some bipolar and cyclothymic patients have strong desires to play the high profile high risk strategy (associated with being successful, admired, in leader positions and/or dominating others and winning fights) but are also very sensitive to put-down, shame, guilt and rejection. Thus, they can oscillate between the two and suffer much internal conflict. This can be analysed using catastrophe or chaos theory.[6] To say that this is either a psychological or biologically caused 'illness' is to seek a micro theory, and miss the point that it is both. Further, disorders like bipolar affective disorder often begin at the time of entering sexual maturity and as the person is moving into their biological prime, requiring separation from parents and entering the world as a competitor.

The downturns into depression represent the (involuntary - outside conscious control) operation of inhibitory mechanisms of the low-risk low-gain (no-challenge) strategies that put a powerful brake on aspiration and reduce hope.

... Mood, then, is in part an energy control system that signals to self and others the readiness (confidence) to challenge and seek resources. *(This is not the same as energy conservation since depressed people may be poor at conserving their resources or defending themselves.)*

I could have added that manics are not good at conserving resources either, but tend to use them up quickly in a kind of "fast (easy) come, fast (easy) go" way. Leon and Russ raise the possibility that mania is related to alpha personality. I see ranking theory as suggesting that the internal mechanisms of rank control are unstable in bipolar illness. It seems to me that reproductive strategies are flexible devices that are recruited to fit different breeding environments -- that is they are not rigid but were designed to be ecologically sensitive; bipolar states may reflect much flexibility or amplification. What this means for me is that personality and bipolar depression are different issues -- which may be related but need not be -- any more than there is one personality type that underwrites depression.

Two key questions arise suggesting it is (a) the instability of mood that is interesting -- knowing as we do that even normal mood is a naturally oscillating system, and (b) the degree of amplitude of mood change. Genetic research suggests that it may not be bipolar illness that is inherited as such but the flexibility in the (rank) system, the instability of it. We know that in monozygotic twins reared apart, if one twin gets it, there is a high chance of the illness in the other.

Russ may be correct that in some there is a premorbid alpha state - but this is not always the case. One does not need this to be vulnerable to bipolar illness. In cognitive terms I would say that in bipolar illness the schema that related to the two strategies (high-risk high-gain *versus* low-risk low-gain) are highly developed and that it is fundamentally a narcissistic disorder in the context of a ranking system that is biologically unstable. Again, as noted above, I think catastrophe or chaos theory is the best model for this. Let me give two cases.

Case 1: Sam was a rather introverted man who had taken over the family farm when his father died. His mother was highly controlling of him and though he had many doubts about looking after the farm (he was actually training to be a teacher when his father died) he felt he had to look after her and the farm out of loyalty to his father. However, through much of his twenties he had strong desires to escape this burden, but being a compliant and super-ego driven person, never voiced these inner feelings. He married to please his mother (someone of the same religion) but she turned out to be fairly dominant too. He said he felt his life had always been about pleasing others. After a few years he moved the farm, against his wishes, to another county - to please his wife -

but she was (according to his account) not that pleased and on reflection he felt that she probably didn't like farming at all. Anyway, after a lot of stress from the move (when he wasn't sleeping, and getting depressed, etc.) and with not a lot of help from others, he began to feel increasingly resentful - for some months he'd felt a new anger building in him "wanting to burst out". One day, without warning, he went out and got into debt by some thousands of pounds. He started to have ideas: "sod what others want, I'm going to do it my way". He believed that he was going to make lots of money from his investments - buy in managers to run the business and sail around the world. Unfortunately he was carted off to hospital!

For many years he oscillated in a bipolar state, given loads of drugs and ECT. Eventually he came to see me and we began the slow process of looking at the triggers of his mood swings. These turned out to be frustration and conflict triggered. After two years of therapy he got out of his marriage and met someone who was a much less dominant personality and his moods have settled for a couple of years now with just some lithium. The point about this case is that there was never any evidence of alpha personality but considerable evidence of over control, high subordination and long desires to escape from the control of others.

Case 2: John had always been highly competitive and extravert. However, he was also terrified of envious attacks if he won and thus highly ambivalent about doing his best. He felt others would see him as "too big for his boots". He was caught between a "go it alone" strategy and an "alliance formation" strategy. He was riddled with sexual conflicts and fears, and fear of God as a punitive, dominant male, but also he would engage in secret rebellions, especially in abuse of alcohol. When I saw him he was staying up all night working out new ideas about the origins of the universe! Some years previously he had been diagnosed as bipolar and put on loads of drugs. Luckily for him a new psychiatrist had thought that his problem was as much narcissistic defense and gradually got his medications down to a reasonable level. In therapy I did indeed have to "out rank him". Over one week I saw him on a number of occasions (including dashing to the ward when there was a crisis). I held to a line that he could not sort out his problems himself and he had to settle down (submit?) to therapy, and that I had not much experience in the origins of the universe and therefore it was pointless to try to impress me that way. This, of course, brought a flood of "me not understanding him" and also recognition that he never really trusted anyone - that he had to look to himself for solutions. Time and again we tackled it head on, but I did everything I could to stay hyper-friendly and non-punitive (hedonic authority?) but firm. I empathised a lot and was pleased as he gradually came to need to see me more and more, saying he could relax with me. Later, he told me that my firmness had "done something to him" and he had felt himself feel a growing relief that the "fight might be over".

Both therapies were long, emotionally taxing and difficult, but both have settled down now - for some time. The moral of the story is that for me it is the instability of the rank system that is at issue and personality something of a separate question. John would have seemed alpha but Sam would not. Over the years other observations have struck me: 1) the extent to which there is a conflict (a clash of authority) with either parent or spouse and where the bipolar-to-be at some point is highly submissive only later to rebel. 2) The basic narcissistic vulnerability of bipolar people. 3) Often there are sexual issues and conflicts. 4) There are (like depression) many different forms of mania and we should not at this point see it as one "illness". I think there are various types; some may be purely biological, others related to more psychosocial, developmental factors. I am not sure why depression is seen as a highly complex and heterogeneous disorder but (hypo)mania as relatively homogeneous.

Well, these are just some thoughts. I think the ranking theory is a good model for bipolar illness, and I can't think of any other that really explains the swings between two such different states.

References: page 22

ARTICLE:

by K Glantz

The Adapted Mind: Evolutionary Psychology and the Generation of Culture.[1] A review.

One of the problems that has long plagued psychotherapy is failure to distinguish between what is normal and what is pathological. The problem started with Sigmund Freud, who based most of his theory of human nature on observations of patients, thus elevating some of the problems of 19th century Viennese to the status of universal psychological mechanisms. Many subsequent thinkers about therapy followed these giant footsteps into evocative jungles of jargon. The Adapted Mind, a collection of essays summarizing many years of research by the founders of evolutionary psychology, is a work that helps to establish what is normal. It is primarily concerned with two vast subjects: 1) the shared characteristics of human beings - the universal psychological mechanisms that generate our apparent diversity of behavior and culture; and 2) the difference between the way these mechanisms play themselves out in the genders.

Since knowledge of the universal is essential for any therapist trying to fathom the idiosyncrasies of a stranger's mind, the book ought be required reading for serious students of psychotherapy. In therapy as in music, you can't really understand the variations unless you can hear the themes.

The central tenet of the book -- the meaning of its title - is stated with clarity in the Introduction: The human mind is made up of discrete psychological mechanisms (modules; Darwinian algorithms) each of which is adapted to a specific purpose. Each mechanism was crafted by natural selection to help cope with some recurring feature of the environment at some time in the past (environment of evolutionary adaptedness or EEA). In the jargon of the book, the new science of the mind is "domain-specific," i.e., it is based on the analysis of components of mind and brain; to study the mind, one must ask what its components were designed to do.

Because it enables us to identify the place of the human mind in the natural world, evolutionary psychology is a threat to the so-called sciences that have traditionally studied the mind, and it is to these fields of study -- the academic world of psychology and the other social sciences -- that the book is primarily addressed.

The opening article, "The Evolutionary and Psychological Foundations of the Social Sciences," by Tooby and Cosmides, exposes the loathing that most social scientists have for the idea that humans are animals and that the mind is material. This loathing motivates the sad but maddening declaration by social scientists that their field is "autonomous" (independent of the hard sciences). This allows them to reject any scientific finding that displeases them.

With endless patience, the authors lay bare the elements of what they call the Standard Social Science Model (SSSM). The SSSM includes such notions as equipotentiality, the notion that the mind is a general purpose (domain-general) device that can do anything and solve any type of problem equally well, that it is a content-independent or content-free processing mechanism, that there is no such thing as human nature, that human behavior is infinitely malleable, that culture determines the individual, that any individual can become anything, etc., etc. The astute reader will notice that psychotherapy has not been immune to such ideas.

In the second article, Don Symons goes after another myth, the idea that current adaptive value - how well a behavior now contributes to inclusive fitness -- is a valid test of Darwinian hypotheses. Given that the mind evolved in order to cope with specific features of the EEA, there is no reason to assume, says Symons, that any given mechanism will work as designed in a changed environment.

This evolutionary psychological position is the reason John Pearce and I developed the idea that the mismatch between the current environment and the EEA was responsible for some of the psychological

distress experienced by people who seek therapy. Some people seem to have the flexibility to adjust their behavior to current conditions; those who don't can use the help of therapists who understand the mismatch.

The rest of the book is devoted to specific psychological mechanisms. There is hardly any need to dwell on Cosmides and Tooby's "Cognitive Adaptations for Social Exchange. Among other things, it's a fantastic piece of detective work. The authors were able to deduce a fundamental principles of mental functioning ~ the existence of a psychological mechanism specifically designed to facilitate the calculation of reciprocal altruism - from the characteristic pattern of errors human beings make on a standard psychological test (the Wason Selection Task). For me, this work established the importance of focusing on a client's ability to sustain social relationships based on give and take.

David Buss' classic work on the differences between what men and women seek in mates (the different priorities given to appearance, age, possession of resources, status, and reliability), highlights another mechanism with importance for therapists, especially those who work with couples. Over the years, Buss has painstakingly demonstrated that current human mating behavior reflects the different selection pressures that operated on males and females over time. Note that Buss has tried to show, not that evolved mate preferences are adaptive today, but rather that these preferences continue to influence behavior. This is an entirely different proposition.

Margo Wilson and Martin Daly ("The Man Who Mistook His Wife for a Chattel") discuss the selective pressures that explain why men tend to feel that their mates belong to them. They trace this trait to the combination of uncertainty of paternity and paternal investment that characterizes our species. In my opinion, they might have mentioned that male possessiveness was exacerbated by the invention of agriculture, private property and inheritance, which provide a dramatically-increased incentive to establish paternal certainty. But even if true, this point does not change the fact that every therapist needs to understand just how deep are the roots of this troublesome trait.

Janet Mann's "Nurturance or Negligence: Maternal Psychology and Behavioral Preference Among Preterm Twins" is an intriguing attempt to explain why mothers sometimes neglect and sometimes overinvest in sickly babies. She thinks it's because moderate (normal) investment would fail with these babies. The chapter is of interest to therapists counseling clients with parenting issues, but the basic papers by Robert Trivers, on parent-offspring conflict and parental investment, provide a more direct exposition of issues of general interest to parents and to people having family problems.

There are other important chapters, including Margie Profet's brilliant explanation of why women get nausea and have food aversions during pregnancy (to protect the embryo against toxins during the first 50 days when it is most susceptible), but most of these chapters don't seem to me to have much direct relevance to clinical issues.

One would think that clinicians familiar with evolutionary psychology would proceed to develop a list of psychological mechanisms - normal functions - whose malfunctioning could be expected to cause psychological distress. I mentioned three so far: the calculation of reciprocity; mate-choice; and parental investment and parent-offspring conflict. Other mechanisms derived from the evolutionary literature literally jump to mind: the assessment of danger; the focusing of attention; reproductive strategies; the assessment of status; the assessment of the feelings, motivations and goals of others (empathy); the maintenance of mood stability; the control and direction of aggression; etc.

Unfortunately, to my mind, the one article that explicitly addresses clinical matters, "The Evolution of Psychodynamic Mechanisms," by Randy Nesse and Alan Lloyd, does not take this tack at all. Rather than develop an approach based on the evolutionary mechanisms identified by evolutionary psychologists, Nesse and Lloyd take as their starting point a handful of mechanisms invented by Freud: regression, psychological defenses, intrapsychic conflict, conscience, transference, and childhood sexuality. Their

stated purpose is to show that an integration of psychoanalysis and evolutionary psychology is both possible and valuable.

As some of you probably know, I don't see any reason to do this. Years ago, in Freud: Biologist of the Mind. Frank Sulloway showed that Freud was immersed in an outdated, 19th century version of Darwinism and biology. What is the utility of trying to "merge" all that? Doesn't it make more sense to start with the ideas we now believe are valid, rather than seeking to cobble together a synthesis of the ideas of different historical figures? If we develop a coherent view of pathology based on current scientific thinking, what is valid in the work of past authors will naturally incorporate itself into the new framework. The rest (of their work) is history, or should be.

Nesse and Lloyd would argue, I imagine, that this would be a terrible loss, for they end their chapter with a call to evolutionists to use the "data" collected by psychoanalysts over the years. But as has been pointed out so many times, this so-called data has been skewed by the intellectual biases of the observers. The observations have to be redone, by people who understand The Adapted Mind.

If evolutionary psychology is correct in its contention that ancient psychological mechanisms continue to influence our behavior today, there seems little reason to believe that these mechanisms will operate as designed; the environment has changed too much. Hence one should expect to find some individuals with normal mechanisms that are not working well. These, I surmise, make up a substantial portion of the people who come to therapists for help (the other portion being made of up those whose mechanisms are not intact).

The continued misfiring of psychological mechanisms can reasonably be expected to produce distress. Life presents a series of challenges to every individual. If the challenges are handled well enough at every stage, as they might be if all one's psychological mechanisms were working as designed, some degree of satisfaction with self can reasonably be expected. If life constantly presents challenges that can't be handled - perhaps because they weren't anticipated by natural selection -- one should not be too surprised to find self-doubt, anxiety and depression.

A word of caution, to myself more than anyone else. For clinicians, the distinction between "current behavior" and "evolved psychological mechanisms" is a powerful tool, but it doesn't solve all problems. For example, is "anxiety" a current behavior or a psychological mechanism and hence an evolved strategy? In my opinion, anxiety is a behavior; the mechanism is the fight-or-flight response. One should expect malfunctions on both ends of a continuum ~ too much and too little anxiety. Others (e.g. Marks and Nesse, 1995) have argued that anxiety is the mechanism, for two reasons, if I understand correctly: 1) because normal anxiety is vital for an organism to defend itself against a variety of threats; and 2) because anxiety shows signs of design (e.g. excess anxiety is adaptive because a false positive is less serious than a false negative). And what about depression? In my opinion, depression is a behavior. It is the final common pathway following the failure of one or more of several evolved mechanisms. At least three known candidates exist: 1) mood stabilization; 2) the mechanism that causes the weaker individual to yield to a stronger adversary (the Gardner, Gilbert, Price, Sloman hypothesis); and 3) a mechanism that causes an individual to conserve energy when activity would be useless (a Randy Nesse hypothesis). But Price *et al.* and Nesse all argue that depression is itself the mechanism. So I could very well be wrong.

I would like to conclude with a short meditation on bias. At least four sources of bias can affect a therapist's understanding of another human being: gender, culture, individual genetic differences, and personal history. An understanding of the psychological mechanisms that are common to all individuals and cultures, and of those that are specific to the genders, is one of the greatest possible resources for overcoming bias. To paraphrase AA, we must recognize what is similar, accept what is different, and have the wisdom to know which is which.

References: page 22

ANNOUNCEMENT:

by R Nesse

What is Darwinian Medicine? A book summary

Darwinian medicine is the enterprise of trying to find evolutionary explanations for vulnerabilities to disease. Every trait needs an evolutionary as well as a proximate explanation. Disease, not being a product of selection, would seem to be excluded. This is one reason why doctors have not realized that evolution might be useful. Another reason is that medical research looks for differences between individuals in order to explain why one person gets sick while another stays healthy. But Darwinian medicine does not seek evolutionary explanations for disease itself, and does not usually try to understand why one individual becomes ill when another does not. Instead, it tries to understand why all humans are vulnerable to each disease. It asks how it is possible that natural selection can shape the eye or heart or brain but cannot eliminate our vulnerabilities to nearsightedness, atherosclerosis, depression, or cancer. Darwinian Medicine applies the advances that have revolutionized evolutionary biology to the problems of medicine and tries to provide, for each disease, an explanation for why the body isn't better. These evolutionary explanations for disease fit nicely into just a few categories: defenses, infection, novel environments, genes, design compromises, and evolutionary legacies.

Defenses are often confused with diseases. Knowing the difference is crucial, because interfering with a defense is often unwise. Pain is a defense against tissue damage; people who lack this defense usually die by age thirty. Fever is a defense that protects against infection. The low iron levels associated with infection are the body's way of keeping iron away from invading bacteria. Nausea and vomiting and diarrhea are useful ways to rid the body of infection and toxins. The nausea that accompanies pregnancy discourages the mother from eating toxic substances that may harm her baby. Even anxiety and sadness can be useful. As for the runny nose that accompanies colds, we don't yet know if it benefits us or viruses, but we certainly need to know in order to decide if nose sprays will help or harm us. Much of clinical medicine relieves people's discomfort by blocking defenses like fever, pain, nausea and diarrhea. How can this be safe? Just as smoke detectors are designed to give many annoying but inexpensive false alarms so that they are sure to warn about any actual fire, the mechanisms that regulate the body's defenses have evolved to express defenses whenever they are possibly useful, thus causing much unnecessary suffering.

Infections are neither a divine punishment nor an arbitrary failing, but merely a contest between our bodies and smaller organisms that want to eat us. Because these viruses and bacteria reproduce so rapidly, they evolve faster than we can, so we cannot escape them. We can and do, however, evolve weapons of ever more subtle destruction. They, in turn, evolve ever more sophisticated ways to escape our defenses. Is evolution moving towards some happy accommodation? Not at all. This is war to the death, except, that is, when death is not in the pathogen's interests.

Genes that cause disease usually turn out not to be simple mistakes. Many offer a benefit, like the sickle cell gene that protects against malaria, or a tendency to gout that may delay aging. The gene that causes cystic fibrosis may protect against death from dehydration. Others, like most genes that cause nearsightedness and heart attacks, are harmless quirks for people who live in the natural environment; they cause problems only when they interact with novel aspects of the modern world, like learning to read or a high fat diet. Some "outlaw" genes even manage, by intracellular warfare with other genes, to get themselves transmitted even though they harm the carrier. Even the genes that cause aging are not just accidents; many of them seem to have been selected because they offer benefits early in life. Before we use our new technologies to eliminate genes that cause diseases, we must consider the

possibility that they also offer benefits.

Environmental factors that cause disease are mostly novel, new in the past 10,000 years, that is. Intermittent exposure to sunlight is novel and results in cancer. Suntan creams may, paradoxically, cause even more cancer. We crave fat salt and sugar because they were in short supply in the paleolithic. Now these appetites prove more powerful than our willpower and cause epidemics of obesity and atherosclerosis. We are well protected against plant toxins, but cannot reliably detoxify novel substances. The differences between the setting we evolved in and the setting we live in is vast and often dangerous.

Design compromises account for much disease. Just as there are costs associated with many genes that offer an overall benefit, there are costs associated with every major structural change preserved by natural selection. Walking upright gives us the ability to carry food and babies, but it predisposes us to back problems. Many of the body's apparent design flaws aren't simply mistakes, they are just compromises. To better understand disease, we need to understand the hidden benefits of apparent mistakes in design.

Finally, evolution is an incremental process without the possibility of fresh starts. Our food passes through a tube in front of the windpipe, and must cross it to get to the stomach, thus exposing us to the danger of choking. It would be sensible to relocate the nostrils to somewhere on the neck, but that will never happen.

In summary, Darwinian Medicine proposes that descriptions of disease in current medical textbooks omit a crucial section - an evolutionary explanation for why humans are vulnerable to this disease. Finding these explanations will have immediate practical benefits for medical practice. General physicians still don't think of fever as useful and they still give iron supplements to patients with chronic infections. Infectious disease specialists still think that pathogens evolve to benign co-existence. Psychiatrists still act as if all anxiety, sadness, and jealousy is abnormal and they don't yet look for the selective advantages of genes that predispose to schizophrenia and bipolar disorder. Rheumatologists don't know that the high uric acid levels of gout may have been selected to slow aging and they prescribe anti-inflammatory agents that may hasten hip regeneration. Obstetricians have not considered the possibility that nausea of pregnancy may be a defense against toxins. The foundations for a new field of inquiry are now being laid by many people including Ewald, Profet, Rose, Konner, Eaton, Buss, Cosmides, Tooby, Diamond, Trivers, Durham, Austad, Ames, Daly, Wilson, and many others. It will not be easy to explain all this to doctors. They will, like the reporters, imagine that we are saying that disease is useful, or that we should let nature take her course or any number of other misunderstandings that will no doubt rain on our heads for the next few years.

George and I will be criticized for writing for a general audience, for daring to speculate about the causes of disease before the research is done. But our goal is not to prove any specific hypothesis, but to highlight a set of questions that are, we think, genuinely new. As people take them seriously, the resulting research should prove profoundly useful as well as most interesting.

Why We Get Sick: The New Science of Darwinian Medicine by Randolph Nesse and George Williams.

Publishers: Times Books/Random House
Tel: (800) 733-3000. ISBN #812922247.
Hardback: $23.50.

This is not a dense academic book. The authors have tried to make it interesting and accessible in hopes of reaching a wide audience, but expect that it will also serve as a preliminary but scientifically valid guide for physicians and researchers who are asking evolutionary questions in their own areas of expertise. They hope that it will prove useful for some classes as well.

ABSTRACTS & EXTRACTS...

Brown GW, Harris TO & Hepworth C: Loss, humiliation and entrapment among women developing depression: A patient and non-patient comparison.

Klein DF: Testing the suffocation false alarm theory of panic disorder.

Klein DF: False suffocation alarms, spontaneous panics and related conditions.

Szent-Gyorgyi A, quoted by H Gest: The breath of life: Legacies from virtuosos of biology, chemistry and medicine.

Nobre AC, Allison T & McCarthy G: Word recognition in the human inferior temporal lobe.

Johren O, Flugge G & Fuchs E: Hippocampal glucocorticoid receptor expression in the tree shrew: Regulation by psychosocial conflict.

Mani SK, Allen JMC, Clark JL, Blaustein JD & O'Malley BW: Convergent pathways for steroid hormone -- and neurotransmitter-induced rat sexual behavior.

Brown GW, Harris TO & Hepworth C: Loss, humiliation and entrapment among women developing depression: A patient and non-patient comparison. Psychological Medicine 1995;25:7-21.

Synopsis: This paper is part of a series dealing with the role of life events in the onset of depressive disorders. Women who developed depression in a general population sample in Islington in North London are contrasted with a National Health Service-treated series of depressed patients in the same area. Findings among the latter confirm the importance of a severely threatening provoking event for onset among the majority of depressed women patients. The results for the two series are similar except for a small subgroup of patients characterized by a melancholic/psychotic condition with a prior episode.

The severe events of importance have been recognized for some time by the traditional ratings of the Life Events and Difficulty Schedule (LEDS). However, the full descriptive material collected by the LEDS has been used to develop a new refined measure reflecting the likelihood of feelings of humiliation and being trapped following a severely threatening event, in addition to existing measures of loss or danger. The experience of humiliation and entrapment was important in provoking depression in both the patient and non-patient series. It proved to be associated with a far greater risk of depression than the experience of loss or danger without humiliation or entrapment.

Klein DF: Testing the suffocation false alarm theory of panic disorder. Anxiety 1994:1:1-7.

Abstract: The need for hypotheses concerning the nature of those functions that have been impaired in stereotyped psychiatric syndromes is emphasized. With regard to panic disorder, the key role of the spontaneous panic attack became apparent from several viewpoints. However, panics seem to be a type of misreleased fear, which guided the thinking concerning the nature of possible psychological or physiological malfunctions. We indicate that spontaneous panic cannot be fear, but must represent some other malfunction and suggest that the spontaneous panic is a suffocation false alarm.

The development of this idea is outlined, and attempts to develop tests of this hypothesis are indicated. In particular, studies of children with congenital central hypoventilation syndrome, patients with Chronic Obstructive Pulmonary Disease, dyspnea, field measures of panic, pregnancy, childbirth and the postpartum period, as well as the premenstrual syndrome afford pointed opportunities, new information and potential tests of the theory. A recent challenge to the theory from acetazolamide infusion is discussed. Developing a possible antecedent for the pathologically depressed threshold for the suffocation alarm, in the form of a phasic endorphinergic deficiency, is presented.

Klein DF: False suffocation alarms, spontaneous panics and related conditions. Arch Gen Psych 1993;50:306-317.

Abstract: A carbon dioxide hypersensitivity theory of panic has been posited. We hypothesize more broadly that a physiologic misinterpretation by a suffocation monitor misfires an evolved suffocation alarm system. This produces sudden respiratory distress followed swiftly by a brief hyperventilation, panic, and the urge to flee. Carbon dioxide hypersensitivity is seen as due to the deranged suffocation alarm monitor. If other indicators of potential suffocation provoke panic, this theoretical extension is supported. We broadly pursue this theory by examining Ondine's curse as the physiologic and pharmacologic converse of panic disorder, splitting panic in terms of symptomatology and challenge studies, reevaluating the role of hyperventilation, and reinterpreting the contagiousness of sighing and yawning, as well as mass hysteria. Further, the phenomena of panic during relaxation and sleep, late luteal phase dysphoric disorder, pregnancy, childbirth, pulmonary disease, separation anxiety, and treatment are used to test and illuminate the suffocation false alarm theory.

Szent-Gyorgyi A, quoted by Gest H: The breath of life: Legacies from virtuosos of biology, chemistry and medicine. Perspectives in Biology and Medicine 1994:38(1):2-20.

Abstract: I must ask you to climb with me on the evolutionary ladder up to the cell, the most wonderful product of the biosphere, the cornerstone of life. I will even have to take you one rung higher, to the multicellular state. The lowest rung of the evolutionary ladder, the simplest form of independent life known today is represented by the bacteria. Their existence must have been based originally on the discovery of nature that energy can be released from molecules by twisting or breaking them, preserving the energy thus liberated in the form of what are called high energy bonds of ATP. This process is called "fermentation".

The next important rung on the ladder of evolution was construction of the porphyrin ring, which made oxidation and photosynthesis possible and also introduced beauty in to life, for what is more beautiful than the green of the forest, the pink of the cheek of girls ... In the next step of development order was made and the new functions with their mechanisms were enshrined in little boxes by putting membranes around them. So the genetic material was rounded off as a nucleus, the oxidative apparatus as mitochondria, the instruments of photosynthesis as chromophores or chloroplasts. After this nothing basically new was added, but once order was achieved the way was opened for cells to get together to form more complex organisms which could perform more complex functions.

The principle which emerges here is what I would like to call "vertical organization", by which I mean that if nature discovers a new principle it does not throw the old ones away: It simply builds the new one on top. So fermentation, the most primitive form of energy release, is still the basis of our metabolism. Not only does nature not throw old things away, but the older a process, the deeper it is ingrained, and the less the chance to lose it. Although nothing basically new came into the picture by building multicellular organisms, the cells had to learn to live together, give up certain liberties, and have regard

for their neighbors and the common interest, as individuals or nations have to do when wanting to form bigger viable units.

Nobre AC, Allison T & McCarthy G: Word recognition in the human inferior temporal lobe. Letters to Nature 1994:372:260-263.

Abstract: Studies of primates and of patients with brain lesions have shown that the visual system represents the external world in regions and pathways specialized to compute visual features and attributes. For example, object recognition is performed by a ventral pathway located in the inferior portion of the temporal lobe. We studied visual processing of words and word-like stimuli (letter-strings) by recording field potentials directly from the human inferior temporal lobe. Our results showed that two discrete portions of the fusiform gyms responded preferentially to letter-strings. A region of the posterior fusiform gyrus responded equally to words and non-words, and was unaffected by the semantic context in which words were presented. In contrast, a region of the anterior fusiform gyrus was sensitive to these stimulus dimensions. These regions were distinct from areas that responded to other types of complex visual stimuli, including faces and coloured patterns, and thus form a functionally specialized stream within the ventral visual pathway.

Johren O, Flugge G & Fuchs E: Hippocampal glucocorticoid receptor expression in the tree shrew: Regulation by psychosocial conflict. Cellular and Molecular Neurobiology 1994;14(3):281-296.

Summary:
1. This study was conducted to determine whether chronic psychosocial conflict alters the expression of glucocorticoid receptor (GR) mRNA in the hippocampus of male tree shrews *(Tupaia belangeri)*.
2. To generate probes for the *in situ* hybridization, the tree shrew GR gene was partly cloned. There was a 90% homology between the deduced amino acid sequence of the cloned tree shrew GR and that of the corresponding human GR sequence. S-Labeled riboprobes which had been transcribed from the tree shrew GR clone hybridized to pyramidal neurons in all subregions of the tree shrew hippocampal formation and to granule neurons in the dentate gyrus.
3. After *in situ* hybridization, the expression of GR mRNA was semiquantitatively determined by counting silver grains over single neurons of the hippocampal formation of psychosocially stressed tree shrews and control animals. After 12 days of social conflict, the number of silver grains in the CA1 and CA3 pyramidal neurons of stressed animals was significantly lower than in controls. No statistically significant differences in mRNA expression were observed in the pyramidal neurons of the subiculum and in the granule neurons of the dentate gyrus.
4. The present results suggest that psychosocial stress leads to a site-specific down-regulation of hippocampal GR via modification of mRNA expression.

Mani SK, Allen JMC, Clark JK, Blaustein JD & O'Malley BW: Convergent pathways for steroid hormone - and neurotransmitter-induced rat sexual behavior. Science 1994:265:1246-1250.

Abstract: Estrogen and progesterone modulate gene expression in rodents by activation of intracellular receptors in the hypothalamus, which regulate neuronal networks that control femal sexual behavior. However, the neurotransmitter dopamine has been shown to activate certain steroid receptors in a ligand-independent manner. A dopamine receptor stimulant and a D_1 receptor agonist, but not a D_2 receptor agonist, mimicked the effects of progesterone in facilitating sexual behavior in female rats. The facilitatory effect of the neurotransmitter was blocked by progesterone receptor antagonists, a D_1 receptor antagonist, or antisense oligonucleotides to the progesterone receptor. The results suggest that in rodents neurotransmitters may regulate *in vivo* gene expression and abehavior by means of cross-talk with steroid receptors in the brain.

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