For evolutionary psychiatrists, psychologists and others interested in sociophysiological integration

ASCAP

Volume 11, No. 05 (Cumulative #126) May 1998

"Good intepretations give the patient something he wants to receive."

Joseph Weiss[1]

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ASCAP Society Mission Statement:

The ASCAP 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 psychopathologi-cally related states. We are interested in the integration of various methods of study ranging from cellular processes to individuals in groups.

The ASCAP 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.

The ASCAP Newsletter is a function

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The ASCAP Newsletter* is the official newsletter of the *Psychotherapy Section* of the *World Psychiatric Association.

ADDRESSED TO & FROM ...

Beck Award Winner

Congratulations go to the 1998 Aaron T. Beck ASCAP Award to be awarded in Davis, California, on July 8,1998, during the Annual Meeting of The ASCAP Society. He is:

Bruce J. Ellis, Ph.D.
Department of Psychology and Human Development Vanderbilt University Nashville, Tennessee

For an essay entitled:

"Psychosocial antecedents of pubertal maturation in girls: Parental psychopathology, stepfather presence, and family and marital stress."

Dr. Ellis is a post-doctoral fellow, having achieved his doctorate from the University of Michigan in 1995. He will receive a $1,000 check, will deliver his essay in the form of a talk, and receive a plaque.

Updated Program for the Annual ASCAP Meeting

Updated program for annual ASCAP meeting. Davis, California. July 8,1998 just before the HBES meeting.

The program has changed slightly. Ivor Jones, our 1 st Vice President, is unable to come to the 1998 meeting but Lynn O'Connor from San Francisco is eager to present her data, some of which stems from her collaboration with Paul Gilbert in Derby, England (ASCAP's 3rd president).

8:00-8:30 a.m. **Dan Wilson:**

"Toward empirical research in the clinical application of the human evolutionary sciences." (Presidential address).

8:30-9:15 **Paul J. Watson:**

"A behavioral ecologist's social niche change theory of unipolar depression."

9:15-9:35 **Carolyn Reichelt:**

"Response from a sociophysiol-ogy perspective".

9:35-9:55 **Penelope K. Knapp:**

"Response from a child psychiatrist".

9:55-10:10 ***-Break***

10:10-10:30 **Ed Hagen:** "Case of depression that addresses niche theory"

10:30-10:50 **John S. Price:**

"Response from a pioneer theorist on social rank theory and depression."

10:50-11:10 **John K. Pearce:**

"Response of a psychiatrist clinician also an evolutionary psychologist"

11:10-11:30 **Lynn O'Connor:**

"Control mastery theory: new data.

11:30-11:50 -- **Large group discussion** led by Dr. Wilson

1:15-2 p.m. **Bruce Ellis:**

"Psychosocial antecedents of pubertal maturation in girls: Parental psychopathology, stepfather presence, and family and marital stress."

2:00-2:20 **Andrew Solomon:**

'The experience of depressive illness from a writer's perspective."

2:20-2:40 **David Evans:**

"Writers depict experience juxtaposed to evolutionary theory."

2:40-2:50 - **Group discussion** led by Dr. Wilson

2:50-3:05 ~ *Break*

3:05-3:25 **James Brody:**

"Fabre's Tactics of Scientific Research"

3:25-3:45 **J.Anderson Thomson:**

'The Serotonin Story".

3:45-4:15 MarkErickson:

"ASCAP Theme for 1998-1999."

4:15-5 - **Business meeting**

(Dr. Wilson); at conclusion, the gavel is passed to Dr. Erickson.'

Gorilla Grief

The following dispatch appeared in the Sydney Daily Telegraph as the sole front page story on March 31. It describes gorilla family grief over the death of an infant. Zookeepers are also reported as being in shock, although this response seems to lie a little outside the range of inclusive fitness explanation.

A Death in the Family by Simon Benson

He only lived for 3 weeks, but yesterday the unexpected and unexplained death of Australia's first naturally born gorilla had a devastating impact. As the news emerged it soon became clear that no one was more heartbroken than the baby's mother, 17 year old Frala.

Keepers at Taronga Park Zoo first realised something was wrong about 6.30 a.m., when they noticed Frala holding her baby in a distressed state. Their worst fears were soon confirmed. While the gorilla section was closed to the public, the entire family of 10 western lowland gorillas was hidden away in their sleeping pen grieving. Zoo staff said Frala has clung to her baby in a painfully familiar way as she mourned her loss.

The family patriarch, 200 kg silverback Kibabu, stood guard making threatening gestures to anyone who tried to approach. After one failed attempt to encourage Frala to hand over the baby the keepers decided to let the parents dictate the timetable of grieving. They will not try to remove the body before Frala is ready to let it go. That could take up to three more days. In a moving farewell ritual, the other relatives came and paid their respects by touching the dead infant.

Zoo curator Erin Walrazen said: "We can't really know what they feel, but Frala is depressed, listless, doesn't eat. They do realise the baby is dead but it is difficult to know what a gorilla is thinking … we are only assuming." Behind the scenes, a distressed group of 10 keepers were themselves also mourning the death. The zoo has no idea how the baby died although the mortality rate in captivity is up to 30% for young gorillas. In the wild it is 40%.

" *The emotions they show are not a lot different to the emotions humans show.",* said zoo deputy director Glenn Smith yesterday, ' *They are grieving. We are greatly disappointed and very upset. But we will have to leave it up to them to let the baby go. We were very excited about this birth . . . it is tragic."*

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GABA-A, GABA-B Defined

In response to a post on an E-Mail list, there are two general classes of GABA receptors, GABA-A and GABA-B, which is alt the average practitioner probably needs to know. But since [I was] asked about how many GABA-A receptors have been identified, and since the rest of [the inquiring] post seems to indicate an above-average interest in this sort of thing, I'll try to answer:

The GABA-A receptor is a ligand-gated ion channel made up of different subunits. This distinction is important because it is a fundamentally different structure than "classical" neu-rotransmitter receptors such as those for dopamine and serotonin. Each of the five known dopamine receptors, for example, is a one-piece "chunk" made up of a single polypeptide chain. (It is coupled to G-proteins, but the receptor itself is still a single protein.)

The GABA-A receptor, on the other hand, is made up of five subunits, separate proteins coded for by genes that can be found on different chromosomes. (You can picture the structure by folding the five fingers of one hand around a central "pore" representing the ion channel.)

We know of at least 6 alpha subunits, 3 betas, 3 gammas, a delta and 2 rhos in humans. Since a given GABA-A receptor might be made up of any combination of these subunits (for example: an alphai, an alpha5, two beta2's and a gamma3), there are over two thousand

mathematically possible combinations of subunits which can form distinctly different GABA-A receptors.

What this means is that we really don't know yet how many different GABA-A receptors there are. Probably not all possible combinations actually exist — for example, alpha5 might always associate with a beta3 subunit, or alphas might never associate with alpha6, etc. But if even a fraction of the 2000+ combos exist, this represents heterogeneity far beyond the handful of different dopamine or serotonin receptors possible.

What we do know is that the various subunits have distinct distributions in the brain: for example, the alpha5 subunit is found mostly in the hippocampus, and the alpha6 is found almost entirely in the cerebellum. This hints at possibly unique physiological roles.

We also know that it's the alpha subunits that determine benzodi-azepine binding — alpha 1, alpha2, alpha3, and alpha5 subunits will bind benzodiaz-epines, while alpha4 and alpha6 will not. The binding site for benzodiazepines is an allosteric modulator for GABA binding: benzodiazepines bind at a different site on the receptor complex and act to enhance GABA's effect. Barbiturates and ethanol work at other sites on the GABA-A receptor complex, and are GABA agonists as well.

Bottom line: We know of at least 15 or 16 subunits for the GABA-A receptor. There are probably dozens or hundreds of different receptors made of combinations of these subunits, making the 5 dopamine and 14 serotonin receptors pale in comparison. And we are just beginning to scratch the surface of what we could do with all those different combos. Anti-convulsants that don't impair coordination! Anxiolytics that aren't sedating! Perhaps benzodiazepine-like medications with fewer side effects and less propensity for physical dependence. This is why I've spent a certain proportion of the last six years trying to understand the GABA-A receptor.

Questions or comments? Always happy to talk GABA. The single best reference I know of is a book called: *"GABA-A Receptors and Anxiety: From Neurobiologyto Treatment'* by Biggio, Sanna, and Costa, 1995.

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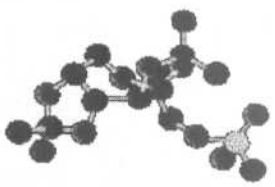
GABA Agonists/Facilitators Home Page

http://tnason.home.mindspring.com/GABA.htm



GABA Research WebSite:

Neuroendocrinology Laboratory

http: //
www.otago.ac.nz/research/Neuro/Grattan/

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Please E-mail any contributions to ascap@utmb.edu, or mail hard copy and 3.5" HD diskette to: Russell Gardner, Jr., c/o Frank Carrel, Department of Psychiatry & Behavioral Sciences, University of Texas Medical Branch, Galveston, Texas 77555-0428, USA. WordPerfect, Microsoft Word or ASCII format preferred. Diskettes will be returned to you. Thank you.

ARTICLE:

by Daniel R. Wilson

Reply to Don Klein

I appreciated the opportunity to share an overview of my method of Evolutionary Epidemiology with the ASCAP readership. It is rewarding to have evoked some considered discussion which may presage the Summer ASCAP Annual Meeting. Having labored in the here-to-fore rather obscure vineyards of evolutionary psychopathology nigh on twenty years (counting undergraduate days...), it especially gratifying to have engaged the talent of so estimable a figure in psychiatric theory and practice as Don Klein not to mention the interesting commentary of Tyge Schelde which brings some international flair!

First, let me say a few words with respect to Dr. Klein's thoughtful critique (I shall reply to Dr. Schelde at a later date). His comments are appreciated as I have learned a great deal in taking up the dialog. Crucially, Dr. Klein has grasped most of the essential points of evolutionary epidemiological analysis. It seems generally agreed manic-depression likely arose as a balanced polymorphism and therefore, the genes associated with contemporary manic-depression were heuristic. We are then, in the words of Professor Ernst Mayr, beyond the "whether" question (ie, is epidemiological prevalence interpretable as genetic frequency and is manic-depression a population polymorphism?). Instead there is interest as to questions of "what, why, how, when and where" things happened. Moreover and again following Mayr among others, we are about the business of delineating proximal from ultimate mechanisms.

Thereafter, it seems Dr. Klein has mistaken my general thrust. In confusing proximal with ultimate explanations, he misconstrued a critical matter of population genetics. Addressing the latter first, genomic polymorphisms arise in only a few essential ways. Many are stochastic events, some are transient and a few are balanced. Balanced polymorphisms are rare but stable. Transient polymorphisms are, by definition, not stable and typically due either to population admixture (migratory flux) or environmental change and are also common. Stochastic accumulation of neutral genes is quite ubiquitous and fairly steady. Stochastic accumulation of deleterious genes is rare. Given that manic-depression constitutes an obvious disease burden with significant genetic load, given that the time frames since the epi-paleolithic are too short to account for (dubiously reliable) observed variance in disease rates as due to selective force and given that non-darwinian mechanisms are likewise insufficient; then how are we to explain the abundance of these genes? It was exactly this point in specific reference to the spectrum of manic-depressive epigenesis that was first brought to my attention by Professor E. O. Wilson some twelve years ago.

It is no "auxiliary hypothesis" that highly prevalent quasi-mendelian gene systems evolved due to advantageous characteristics acquired phylogeneti-cally or that deleterious aspects were not the accumulating force in phylogeny. Quite to the contrary. Absent refutory evidence, standard neo-darwinian science requires such an inference. Admittedly, this is not the point of departure for most bio-medical considerations. Yet Mayr refers to a necessary shift in the burden of proof from the epigeneticist to the sceptic and notes the failure to so shift is a frequent source of error in epigenetic analysis (cf: *Growth of Biological Thought: Populations, Species and Evolution).* Such errors are quite common amongst medics as they attempt to reconcile a clinical Zeitgeist with non-pathological biological reasoning.

It is, therefore, necessary to elucidate how a heuristic gene system — prevalence greater than adjusted mutation rate without evidence of non-darwinian factors — now expresses a disease which, *senso stricto,* could not have been the object of cumulative natural selection. In an ultimate sense, manic-depression *perse* cannot be said to have been selected unless what we regard as the "disease" actually is more salubrious than the non-disease epigenetic state (cf: the sickle-cell carrier trait was adaptive despite the genetic load of sickle-cell disease).

As to the former matter of my general thrust, here Dr. Klein has inadvertently created a straw man contention. Nowhere have I reduced manic-depression to an assumption this is only a reactive abberration of psychosocial modules in the Ho-locene nor otherwise indulged in radical social constructionism! As it happens, in the past my sharpest critics lamented that I was NOT so inclined. Be all that as it may, I posited the:

1. Presumptive case manic-depression arose as heuristic genomic polymorphism with a prelimi nary calculation of its selection index,
2. Need to explain its significant prevalence as a serious disease burden specifically in such a context,
3. Lines of enquiry which may advance such contextualization, **and**
4. Warm welcome to research and discussion of such questions.

My reading of the quite extensive literature on game-theoretical models of phylogenetic neuroscience impresses me as that this is an established domain far beyond "thinly supported speculation." So too the rapidly maturing work, theoretical and empirical, concerning social comparison as a fundamental component of affective illness. Likewise the elucidation of "silver-linings" embedded in the manic-depressive syndrome (as presented at the last ASCAP Meeting not to mention Goodwin and Jamison's textbook review). In any event, I regret if I did not sufficiently emphasize manic-depressive phenotypy *ala* DSM-IV does not constitute the full spectrum required for comprehensive evolutionary interpretation or how relevant is frequency-dependent selection as a factor in manic-depressive phenotypy of the Holocene, among other avenues of discourse. Such themes seemed beyond the scope of what was already a lengthy submission.

Still, one need not assume cultures have departed a similar "distance" from an archetypic past to ennable a fairly modal global prevalence. Both frequency-dependent selection and standard mechanisms of phenotypic reaction are often evident only at thresholds or in step-wise fashion (cf: the peppered moth scenario). Certainly, psychosocial disruptions sometimes seem the fault of entirely different modules; Dr. Klein cited porphyria, panic-agoraphobia and obsessive-compulsive disorder. Even a decade ago I identified evolved aspects of these same syndromes. It is true that the dearth of valid and reliable cross-cultural — especially of "paleolithic" tribes — is a conspicuous problem (and one I am trying to address via field studies which appear imminent). Further, if only *en passant,* I should note porphyria seems not to surpass evolutionary epidemiological thresholds for consideration as a heuristic polymorph (most saliently, its prevalence does not surpass its likely mutation rate). Nevertheless, these are very exciting developments in their early days, indeed. The interest in evolutionary perspectives of such luminaries as Drs. Klein and Rapoport add much substance to our field.

Finally, Dr. Klein mentions the Glantz-Thomson dialog as to how mere human potential is to be disentangled from true pathologies of the same before tossing his keenest javelin my way at the end of his delightful letter. I am happy to catch his spear! Compelling evidence, both theoretical and empirical, is abundantly available in epidemiological studies and via the application of standard neo-darwinian analytic techniques: this quasi-mendelian system sustains high prevalence throughout the species despite its manifestly deleterious disease expression. Based on available evidence manic-depression *perse* cannot have been selected into the human genome although the genes associated with its current expression clearly were. Likewise, the "default" medical assumption that manic-depression is wholly a disease in the narrow sense defies both systematic clinical evidence and numerous central principles of evolution. Moreover this is a genomic polymorphism not a generic trait of human beings. Thus, to square this circle some element of manic-depressive phenotypy must be reactive or "mismatched".

It would be indeed fascinating to encounter a clear and convincing demonstration that the familiar pathophenotypy of manic-depressive disease, per-se, conferred on individuals sufficient ultimate advantage to sustain even a 0.5% prevalence in the face of all manner of liabilities — including excess, early mortality (through suicide, homicide and accidents) and marked psychosocial morbidity. In closing, I must say such a proposition seems beyond the prodigious genius even of Don Klein. *c8*

ARTICLE :

by James F. Brody

Mania Sketches #4, #5, #6, & #7

Mania Sketch # 4: Bipolar Courtship

Both mania and bipolar disorder are defined by highly distressing, disruptive social behaviors. There are statistical links with increased substance abuse, physical abuse, oppositional behavior, and certain types of conduct disorder. None of these things reach the Symons-Buss Top 10. Why marry such a person? Indeed, manics can be so extremely annoying that they should have gone extinct either for lack of partners or through being killed before mating.

Some possibilities include:

1. The Cuckoo Bird Option: Manics are often sexually driven. Thus, they could propagate by making a good first impression (lively and entertaining) and leave a child in a prospective mother who's single or married to someone else. This tactic has similarities to that used by r-selection species that breed freely but offer little parental guidance to their offspring. One manic in 10 might breed sufficiently often to keep the trait going.

2. Clinical mania is often of later onset, after mate selection has occurred: The syndrome thus mimics other disorders that exert no selective pressures because they begin after mating and after children are produced (the Medawar Effect). However, Faedda, et al. documents the prevalence of prepubertal bipolar disorder as well as sharing data that most adult onset cases were symptomatic before age 21.[1] The earlier the onset, the more severe the expression and the greater the incidence of familial bipolar disorder. No surprise except that bipolar teens (who should be easily recognized and avoided in the mating contest) can form destructively intense pairs. It may be that they are more efficient in recruiting enablers (who carry manic traits) or bipolar mates via some linkage attraction ("Everyone else is boring; I'm not sure you're tough enough for me, etc.")

3. Manic traits have survival value: Subclinical mania and alpha status appear to share many features that Miller labels as *"vitality markers".*[2] Domineering behavior, greater sexual interest, and lively personal styles sometimes are related to greater social persistence. Toddler manics often have tantrums; older ones have feuds. Churchill, Teddy Roosevelt, Nietzsche, and other prominent individuals show evidence of mania. Sulloway's "Born to Rebel" is as much a tribute to mania as it is to Darwin and to familial rebellion. We might expect manics not only to consume resources but also to generate them, albeit at other's expense, and to share them with allies and mates.

4. Manics and "enablers" (non-assertive, depen dent personalities) might be co-evolved teams. That is, the determined but periodically offensive manic may benefit from an enabler mother (whether she be timid or manic herself) and be later attracted to an enabler mate (through imprinting or through the more shadowy concept of Linkage Disequilibrium). Betty, for example, was a very shy teen and adult, generally avoid ing confrontations but was an advocate for a string of males who abused her and other people. Being tied to a large, gruff male gave her more assurance. She commented once that she and her tattooed guy were usually moved to the front of lines and his gang often brought her food and assured her safety whenever he was in jail. It's as each member of the partnership had Psych Adaptations that complemented those of the other. Suomi's observations about young rhesus monkeys apply here:[3]

"High-reactive females appear to be at greater risk for neglecting or abusing their first-born offspring, especially when social support is minimal. On the other hand, high-reactive rhesus monkeys are at lower risk for accidental injury or wounding under benign environmental conditions, although relative reactivity can become a significant risk factor for injury under high-stress conditions. Furthermore, high-reactive infants, reared by unusually nurturant attachment figures, are remarkably precocious socially and typically rise to the top of their group's dominance hierarchy."[3], pages 142-143

5. Mania is associated with heightened manipulation and lying. Wright comments that deception is an old tactic in courtship and business.[4] Manics use more of it in order to get their way. This is especially likely since many of them intensely dislike embarrassment levied in public from others, even as small children. Thus, the phenomenon of civility during a social function and tirades in the car going home.

In any event, there ought to be some mechanism for abating the severity of manic traits during courtship so that likely partners are enchanted rather than frightened. One possibility is that the opportunity for reciprocated courtship is an antide-pressant, a mood elevator. Beck compared the similarity of "Being in Love" with elation and euphoria and how the quality of the experience approaches the status of a disorder. The partner is idealized and there is great peace and joy simply by being near them. A happy manic often clowns and laughs, behaviors that might themselves fill the role of vitality markers. Irritability, criticism, and domineering styles abate, at least in the company of the prospective spouse. "He's so different since you came along" could reflect one symptom of a bipolar switch.[(a,b)] Tired mothers are apt to regain hope of escaping the advocacy and defending roles that go with rearing an active, manic child. Courtship is the chance for anxious mom to unload a difficult youngster without feeling guilty. "Betty will care for him" (pay his bills, intercede with the police, sober him, and "make him responsible").

Betty, unfortunately, will miss a warning glimpse of her future unless she interviews his past loves or does a family screen for bipolar disorder. Or, Betty may be a touch grandiose (since we are attracted to people similar to ourselves) and think, "I know I can make him behave when no one else can." Once married, the infatuation fades, irritability recurs, and Betty retorts, "You're acting just like your father!" Betty also blames herself that he's acting as he used to do; because she once changed his behavior, she must be responsible for his relapse.[(c)]

Solutions have evolutionary ties. Enablers often have a manic in their parentage. (In the Linkage Disequilibrium model, the enablers are attracted to mania because of carrying identical traits themselves even if unexpressed phenotypically.) Push and abuse an enabler sufficiently and sometimes break a manic forth ... a nasty, vindictive, spiteful individual (who feels guilty for acting like one of their parents). Use a pinch of sertraline to reduce guilt, do some assertion training, and watch the dragon hatch. When the despot finds his bags on the step, there is some higher chance they will lose weight, stop sleeping but start nightmares, stay out of tap rooms, and go to psychologists. I would also bet that these shaken manics had early, substantial separation anxiety.

Martha had a manic husband and was to the point of leaving him. She started sertraline to cut her guilt. I outlined the last few points with Martha and she responded, "That's absolutely true." She's very bright and very seasoned; it must be true if she believes it.

Notes:

(a) Sports teams, movie stars, and diagnoses have their fans. I periodically encounter a devotee of Borderline Personality Disorder, a label that designates another lively group of people who become distraught when attachments erode. Genetics seems well represented by a "parallel distributed regulatory network" [5, page 10] that recognizes contributions from 2-10 genes to any given trait.[6] Given contemporary models of

genetic function, it's possible that the Borderlines and the Bipolars will share many critical features even in their chromosomes.

(b) There is periodic mention of bipolar sequences that are elicited by environmental changes; most of the literature seems devoted to cyclic changes in behavior.[7] However, there is substantial variability in onset points for most "cyclers" and we might reexamine the close interaction between circumstance and season for these people.

(c) Robert Axelrod's "The Complexity of Coopera tion" arrived today [8], and on page 31 cites a study by Axelrod & Dion (1988) that concluded "noise" calls for forgiveness; too much forgive ness invites exploitation." Axelrod cleverly inserted "noise" into the Prisoner's Dilemma by having a computer randomly change the option taken by one of the participants. The second player had no information whether the overt response was the same as or different from that intended by the first one. Under noise condi tions, Generous Tit for Tat and Contrite Tit for Tat emerged as superior to regular T4T. Gener ous T4T means that several betrayals are allowed before retaliation occurs. Contrite T4T means that the first player, after committing a betrayal, is cooperative with the second until the second shows positive reciprocity.

If mania is conceived as a state of heightened response variability, then enablers make reciprocity more likely to survive episodes of erratic conduct or selfish deceit. "Contrition" overlaps with the verbal "I'm sorry and won't do it again" that softens some mothers, wives, husbands, and religions to continue or to reinstate their forgiving tactics. There is a cost, however, to forgiveness because being forgiving also raises the probability of being cheated. Thus, the adolescent who shows no "remorse" is judged a poorer treatment candidate and less eligible for Generous T4T than one who apologizes. Strings of betrayals imply systematic "exploitation" rather than "noise"" These analogs suggest ways that alliances can be protected while also protecting the interests of individual participants.

Mania Sketch #5: I Won

I've speculated elsewhere that dysthymia may sometimes reflect a manic with no power and no alliances. Rachel called several months ago to make an appointment for her boyfriend whom she suspected of having ADHD. He refused to come. She called again two weeks later but to schedule an appointment for herself because she "was sick of going to support groups for 2 years and getting nowhere."

Rachel's family history had several domineering, critical, feuding characters, ones who expected the world to go their way. She had combative, tearful relationships with her sisters and brother who gave her orders. Her only ally was her mother who led a similar existence with Rachel's bossy father.

Her job was sorting office mail, sitting before a computer monitor for much of the day, and pushing the mouse while pretending to be busy. She described her boss as domineering, rude, and demeaning.

She had dated 3 abusive males, ones who hit or stalked her, who yelled, criticized, or failed to keep dates. Her current tyrant, Chuck, visited but only when his mother approved. His mother treated Chuck in a manner highly similar to the way Chuck treated Rachel.

I offered her some chats about alphas, about being number 1, and about sertraline's effects on self-esteem. She started on 25 mg. and got sick. We waited for her flu to pass and started her on 12.5 mg. and after a month, increased her to 12.5 mg., bid.

Rachel started winning some things, initially with her guy (dropping him, a first for her), then her family. She accepted a holiday dinner from one bossy sister whom Rachel usually avoided and

refused to collaborate in a family gift when her other sister failed to give advance information about costs. Both events were also firsts. She no longer minded staying at home alone, preferring to sew rather than go to tap rooms or to shoot pool with male smokers in dirty T-shirts.

Work was still not satisfying even though she became more assertive, got her highest performance evaluation and the largest raise in the company. Her autocratic boss was leaving; Rachel campaigned for a job change but ignited when the slot went to an office socialite who, had depended on Rachel daily for technical help. Rachel got a reassignment; she was told to work for the promoted ditz. At this point, she denied any intent to leave the firm but publicly moved her belongings out to her car and called 6 temporary agencies to "match my rate." She commented that "I get so frustrated because I think I can win this one and it's not happening fast enough." She was also pleased that she was angry instead of sniffling about these events. Rachel's mother thought that I (and sertraline) were blessings even if mysterious.

My phone rang a week later at 1:05 P.M. A soft voice said, "I won, thanks for being there for me." She was given another assignment, at a higher rung than that occupied by the socialite. She consolidated her role at work, liked her new boss for a spell, and signed up for a college course. However, the new boss corrected nearly every document that she processed for him. She angered but historical events saved her. He missed a lot of work because of illness; Rachel then submitted everything to his boss who never made corrections and appreciated Rachel's accurate and rapid output. She became cheerful again and may earn another advancement.

My cautious interpretation is that sertraline kicked her into a mild hypomania. Some evolutionary talk about winning, about dominance, about alliances and hierarchies with respect to peers and to lovers helped target her energy. Chuck SixPack came back into her life temporarily but without sex. She thought he was funny and, in order to be around her, Chuck tolerated her talking to other guys. She commented, *"I'm using him but I don't feel bad about it."* She treated him just like his mother treated him and he meekly enjoyed it most of the time. She then became annoyed with Chuck because she was more intelligent than him and no longer wanted him around. However, he failed to maintain his posture, demanded his former exclusive access to her while keeping Darwinian privileges for himself. She eventually canned him, not for infidelity but for the hierarchic violation of calling her names.

Another, complementary, interpretation is that sertraline made her less fearful of social rejection, her behavior variability increased, she was reinforced, and "winning" kicked her into the hypomania once she had some victories and found some alliances. I mention this possibility because some dysthymic clients do respond poorly to medications;. Such people may have genetic traits that are different from the responders. They may also be in the wrong niche for their particular talents and still lose the social and vocational contests. Their Psych Adaptations will continue to be accurate and to indicate a low position on the dominance scale, a position that discourages any of us even with an SSRI platform.

Notes:

Rachel and several other women I know may exhibit the relationship that Suomi described for young rhesus and that I've extrapolated to older, male manics.[3]

Katlyn is in her mid-30s and has worked her way upward from clerking in a jewelry store to owning several of them. She's noisily leveraging each of her projects upward every few years. She also has Joe quietly in the background. People who know them both say that Joe is very quiet but firm and acts to stabilize her.

Cora is marketing director for a large national company. She has a long succession of male "connections" - without her husband's knowledge ~

that she often calls long distance late at night in order to share her poetry, gossip, fantasies, jokes, and complaints. She seems to go through one guy at a time (serial connectionism?); each lasts 6-12 months and is terminated when they start to demand a larger part of her life. She promptly labels such demands as "controlling," perhaps another manifestation of the 10 year old who retorts, "Don't tell me what to do."

Of course, I had some fears about Rachel. I had only known her intermittently for 8 months and she might not be at asymptote in her trajectory. While she might eventually challenge Gore for office, she spent only $300 on herself this past Christmas (an increase but money she could afford) and she seemed to be making good decisions about the details of her life. She slept fine and her rate of speech was clearly within normal limits. She sometimes had a voice inside of her whispering that she was doing too well and getting *"too big for her britches".* She used me to argue with it.

Her irritability increased in late February and a small boost in her dose exacerbated her hostility. She stopped sertraline completely and remained a "wise-guy" but liked herself. It could be that her need for sertraline will vary with social opportunity and season.

Things are usually not this easy and her example does not mean comparable results for the next client. There's substantial individual variability in the effects of sertraline or any other medication; however, I no longer believe the variance is entirely within chemistry's domain. Clients need some honest victories, not prattle about their self-esteem.

I sometimes become annoyed with sertraline's being a prescription item; however, Rachel's sensitivity to it as well as her interpretations of its effects as well as the interactions between sertraline and her environmental options reminds me that professional advice is a necessity and that such direction may not always be available from a family doctor. The same considerations hold for St. John's Wort as well as for caffeine.

Rachel, if she's another one of Suomi's kids, needs an anchor to replace me. She will next do some dating but armed with information about Symons-Buss guidelines for mate selection. I think she's going to do fine. I have an eidetic recall of her 1:05 p.m. phone message; I, too, feel that I have won.

Mania Sketch #6: Teen Alpha

Teen Alpha's exuberance declined a bit as the days grew shorter. He increasingly spoke with his parents about private school and obtained their support for "whatever would make him happy."[(a)] He complained more to me about being confined during the day ("The honor students get to leave whenever they don't have a regular class and I need the break more than they do"), about teacher attitudes, and about homework.

I called the healer and raised Alpha's sertraline from 50 mg. up to 75 mg. per day. The irritability left him and his parents didn't have to spend $10K for a local private school for the rest of the year. He was calm but still remembered his thoughts about some of the school staff, cognitions that I knew to be relatively accurate from dealing with one of the same personalities nearly 15 years ago.

According to Alpha: [(b)]

Vice Principal Horton was the chief thorn. "He smiles to your face, nods as if he agrees with you, but still gives a suspension, doing it by mail or by calling your parents. No one likes him and I don't think he likes anyone else."

Alpha felt himself in tighter with his guidance counselor this year. "Mr. Jones even said in a meeting to Horton, *"Alpha's right in this case and you are interrupting and shouting. Let him talk." "I couldn't believe it, I had somebody on my side."* Perhaps the first ally.

"Principal Crabbe told me that he sees a change in me from last year, that I'm trying and he will work

with me." A very important ally, one that could neutralize Horton.

Mr. Ringer (the most senior and the best paid) taught chemistry and things almost didn't work between the two personalities. However, Alpha persevered and earned the top grade on a major examination. Ringer commented, *"Well, now we know that you're really pretty smart."* Bingo! Respect extended, another alliance formed.

Alpha had dominance contests with one teacher who handed him a series of one-liner sarcasms. Alpha talked directly with that teacher, explained his feelings, and the sarcasm disappeared. One more alliance; Alpha sensed that he might win this thing called school.

A little sertraline in a 190 #, no body fat, 17 year old. male with outcomes including no fights with other kids this year, no leaving classes in a rage, the unsanctioned grudge game against the regular football team never occurred, there was no police contact... all different from last year's explosive combatant who would have led the secret scrimmage against the varsity to *"prove a point that the best players aren't on the team"* and who regularly fought dominance battles with teachers, the school administration, and his father. And his parents did not have to go to war with the school to stop unfair treatment of their son.

A little sertraline and Alpha plans to work in his father's business, currently netting $1+ million a year, instead of doing a immediate Sulloway and working for an hourly rate in an auto shop.[9] Of course, it was only halfway through the year but Alpha had two significant allies that he didn't have last year and further had gained some respect and trust from every one of his teachers except for 1 st period. *"She never yells but always has a big smile when she gives you a detention or suspension."* That class was dropped; it was an elective that he did not need.

Alpha was very bright with many topics; the dominance buttons obscured his ability and competed with his willingness to work for teachers that irritated him. The dominance relationships became more of a matrix arrangement in which he was given respect and he reciprocates with cooperation. I think sertraline helped him drop his guard momentarily but a change in environmental circumstance shifted the rest of his mental chemistry.

Alpha is wanting to be assigned to the Resource Room. " / *need somebody looking over my shoulder."* This is the first instance in 20 years that I have heard an oppositional child/teen agree to have someone check on him or her. I challenged him on his sincerity and he explained,"/ *don't mind if I know that they are on my side."*

Certainly, all of us can smell the bipolar soup and another practitioner might well have started Depa-kote or Lithium.[(c)] Alpha came to me, through hiss parents, for difficulties with rage. I had stumbled on the irritability changes with SSRIs several years ago; 25 mg. of sertraline does appear to evaporate "road rage" as well as other things irritable husbands do. (Several of these guys complain of feeling doped, drowsy, or more irritable on 50 mg.; their mates concur.)

Alpha reflects the need for a *"paradigm shift',* for relabeling for fundamental treatment concepts. We are not treating amorphous, impersonal unbalanced mixes of juices. Instead, the ancient motives of dominance, respect, standing, and achievement are manipulated concurrently with serotonin … our 1 billion year tool for self esteem. The SSRIs are not *"antidepressants"* because the more relevant continuum is inferiority-dominance-grandiosity rather than mood. In the future we may use mood shifts as signals to look further for social gains and losses, for changes in the pecking order, rather than allowing moods to be the key variable and attributing mood changes solely to day length, imbalanced juice, or to inevitable cycles, products of defective genes.[(d,e)]

There is also a need for a paradigm shift in certain diagnoses. Alpha qualified for an ADHD label yet was keenly aware of alliances, sensitive to facial

expressions, and managed his personal relationships pretty well. He could be highly persistent in feuds or when overcoming social obstacles between him and a personal goal. Such strengths vary in every person; identifying them ought to help with academic and vocational planning.

Notes:

(a) Suomi comments, *"high reactive infants reared by unusually nurturant attachment figures are relatively precocious socially and typically rise to the top of their group's dominance hierarchy."*[3] Suomi's observations reinforce the importance of adults' being responsive to children's strategies and because they could apply to a number of mother-child pairs that I know. Teen Alpha and his parents are certainly part of that group. Parents really are extended phenotypes of children.

(b) If manics are keen about alliances (at least as a tool for meeting their personal needs) and I am seen as a member of his alliance, then chances are marked that he will lie to keep me where he thinks I need to be. Be clear about confirmation from other sources. "If you want me on your side, I need to hear it from you first. I need to hear ALL of it." Some of these people will become extremely angry if embarrassing comments are made by their parents or mate. "Why did you have to tell them that about me?!"

(c) Alpha also, like other such students, does not do well on a continuous performance test, his developmental history is consistent with many things, including ADHD. He is taking methylphenidate as well as sertraline.

(d) Even the current *Newsweek* has a paragraph about serotonin and controlling aggression[10]. There were hypotheses a few years ago that excess serotonin levels were correlated posi tively with aggression. My hunch is that aggressive behavior can exist for multiple reasons. High serotonin levels may exist independently of the availability of serotonin to receptor sites. It could also be that high serotonin availability supports grandiosity, an attitude of being impervious to any sort of consequence, and indirectly abetting aggression that is elicited and reinforced by economic or territorial gains.

(e) Goodwin & Jamison in 938 pages on *"manic depressive illness"* make no references to evolution or to external triggers.[7] I suspect they are representative of current theorists on bipolar disorder. Kuhn's writings suggest that the BPD (bipolar disorder) establishment will resist changes but such appears to be the nature of scientific communities and other reactive organizations.[11] Nonetheless, the *"disease"* analogy of BPD will fade (unless you want to argue that infectious personalities are truly infectious); a *"fuzzy trait'* characterization will ascend.

We will also likely separate grandiosity from high rate behaviors, dissociating the latter from mania since they occur in mania, ADHD, Borderline Personality Disorder, some anxiety disorders, depressions, and agitated schizophrenic states. There may also be research differentiating familial incidence of varied types of alpha dysfunctions... that mating, grandiosity, political power, and economic power may appear differently in different families and perhaps in relation to different genes in combination with environmental triggers.

Mania Sketch: #7: Mania and Shamans""

We made a substantial marketing error by identifying ourselves with medicine. It occurred sometime after Freud who was more in the shaman tradition than that of plumbing or carpentry. By joining medicine, we subscribed to the notion that weakness is the reason for consulting us. Unfortunately, a lot of stubborn children and mulish, oppositional husbands refuse to admit to an impairment (likewise for wives or children). Art Buchwald put it well when he said that he stopped his lithium because

he "wanted to do it on his own."[(b)] Kay Jamison once made a similar decision that almost killed her.

Psychiatry is perhaps the ultimate degradation because the Alphas place so much value on their being right. Domineering males will accept or boast about injuries to a knee or a fist. It's mechanical and we all understand the need for a mechanic. Such injuries can also be a mark of valor and an elevation of standing.

However, visiting a psychiatrist implies the possibility of weakness, of error, of inferiority in that part of us that plans, evaluates, and competes. "Seeing the shrink" implies taking a lower position on our hierarchic scale, as if we are literally "shrunk" because of our need for psychiatric consultation.

Manics, however, will listen to other alphas, to gods, or to the stars.[(c)] If forced to a doctor, they seek the best doctor so that even if admitting to a weakness, they are superior in their choice of a mechanic. Manics will sometimes believe in non-observable sources of power and influence. One bipolar grandmother chose her orthopedist because his secretary performed astrological readings. The bully husband [(4)]can be attentive, polite, and even receptive to orders from a greater alpha than himself and especially if those directions move toward goals that the bully appreciates such as getting his wife back or escaping trouble while blaming someone else.

There would probably be a lot more of these guys in treatment if we wore fancy robes and consulting us was an affirmation, rather than a negation of power.[(e)]

REFERENCES AND **Notes:**

(a) This series could be extended in many directions. For example, there's a brief study of female manic patterns and another of a manic grandmother's possible role in the life of a manic child whose mom and dad are more average or avoidant in their temperaments.

(b) BuchwaldA. Presentation at the 11th Annual Mood Disorders: Research/Education Sympo sium. Johns Hopkins Affective Disorders Clinic, May 1997.

(c) Lewontin R (1991) Biology as Ideology: The Doctrine of DNA, NY: Harper, has identified similarities between science and religion. The similarities include the use of a specialized language, the implication of truths that have constancy beyond the present moment, and the creation of a specialized group of practitio ners.

(d) Of course, we all know the shallowness of their bluster. If you want to see them crack and crawl, have their wife put the suitcases, packed, on the porch. The Taproom Toms, the Flitting Franks often lose 20 pounds overnight, cannot sleep, and will attend "therapy" and even take medication in order to get their wife back.

Despite several generations of misimpression, women do wield the ultimate power. We guys run around, swell our chests, swill our beer, and shout orders but always orbit back to the woman. She keeps our kids and knows our relative's birthdays; when we marry, we often join her family instead of the reverse.

My stubborn son once met a girl equally stubborn. He announced himself as usually getting whatever he goes after. She responded with similar claims. He capitulated with, *"If it's a contest between what I want and what you want, you will probably win."* Lorraine Rice recently shared a remark by Richard Brinsley Sheridan," *Through all the drama - whether damned or not - Love gilds the scene while women guide the plot."*

(e) "Natural products" have penetrated the mental health market and psychiatry/psychology experience significant competitive pressure from naturopaths, chiropractors, and wellness consultants. GNC opens approximately one

new store each day and perhaps capitalizes on our wishes to save money while avoiding prescriptions and insurance records or asking permission from a "doctor" to do as we wish. There may also be some involvement of our possible Psych Adaptations in the area of food selection; that pills with side effects are easily seen as poisons, a weed with comparable activity is tolerated.

Another company, Equinox International, Inc., sells 3rd party products -- soaps, showerheads, skin creams, water filtration, thermogenic weight management, shark oil -- that are alleged to be environmentally benign. Part of their strategy is to rely heavily on personal introductions in order to pyramid the sales organization. (Each sales person of the 100,000 that they had in 1996 creates a network of others sales people reporting to him or her. Earnings accrue not from selling product but from getting other people to sell product for you.) Companies such as El appear fertile research material for studies of reciprocity, cheater tactics, alliance formation, and hierarchy management.

The mix of environmental concern and our suspicion of "unnatural" medicines generates major commercial success for some of these strategies. Similar organizations in the domains of food distribution, dating services, and child care might be equally lucrative. Some religions are prototypical multilevel marketers. Incidentally, El's founder and CEO, Bill Gouldd, added the second "d" to his name after a spiritual adviser informed him that he was "out of balance." (Greco, S., 1996, "The Buddy System" Inc., 18(15), p 52.)

Author's Note: References to the original articles cited by Suomi were excluded above but are available upon request; send all requests via E-Mail to: jbrody@compuserve.com.[3]

Also, E-Mail me if you have any special interest in these topics at: jbrody@compuserve.com. *c8*

Internet Mental Disorders WebSite

http://www.mentalhealth.com/dis

All disorder Information includes the following:

American description
European description
Treatment information
Research on diagnosis, treatment, & cause
Information booklets
Magazine articles

Here is a sampling of a few of the disorders discussed on this WebSite:

Acute Stress Disorder
Adjustment Disorder
Attention-Deficit Disorder
Autistic Disorder
Bipolar Disorder
Borderline Personality Disorder
Brief Psychotic Disorder
Cyclothymic Disorder
Delusional Disorder
Depression (see Major Depressive Disorder)
Dysthymic Disorder
Generalized Anxiety Disorder
Major Depressive Disorder
Obsessive-Compulsive Disorder
Oppositional Defiant Disorder
Panic Disorder
Post-Traumatic Stress Disorder
Schizoaffective Disorder
Schizophrenia
Schizophreniform Disorder
Separation Anxiety Disorder
Shared Psychotic Disorder
Tourette's Disorder

ARTICLE:

by Paul J. Watson & Paul W. Andrews

Niche Change Model of Depression (Abstract)

Unlike most animal species, the most important determinants of the reproductive success for individual human beings arise from constraints on their socioeconomic pursuits that are imposed by conspecifics. The complex social constraints under which every human must operate are implicitly or explicitly codified in the social contracts that define each of their social exchange relationships and that, collectively, define the individual's "social niche."

The niche change model proposes that primary unipolar depression is an evolutionary adaptation to alleviate a severe *socially imposed mismatch* between an individual's perceived capacities and opportunities for fitness-enhancing activity. The exact origins of such mismatches are as idiosyncratic and diverse as the people whose fitness can be limited by them.

So, under this model, depression is expected to arise in circumstances that, outwardly, appear highly varied. This non-animal model of depression proposes that the core symptoms of minor and major depression were designed by natural selection to help individuals address obstacles to their pursuit of inclusive fitness whose removal demands a relatively comprehensive revision of their social niche that may be difficult to plan and negotiate. Under this model, the niche revisions expected to call the depression adaptation into play usually will require substantive changes in at least one of the niche's defining interpersonal contracts, but usually more than one.

Under the niche change model, all levels of unipolar depression constitute a unitary but contingently escalating phenomenon. We define *minor depression* as any level of depression that can intentionally be hidden from social partners and we think that minor depression generally will be used covertly. The main cognitive changes that appear to occur in minor depression are enhanced social information processing, increased sensitivity to social feedback and loss of the "normal" optimistic bias. Accordingly, we propose that minor depression serves to optimally configure the mind to evaluate the severity of a socially imposed mismatch, formulate mismatch-reducing goals for niche revision and soberly plan an active strategy for their implementation.

If the niche revision strategies developed under minor depression fail to yield the investments or concessions from social partners needed for mismatch-reducing niche revision, then *major depression* may ensue, the crucial features of which are are psychomotor perturbation (usually retardation) and anhedonia. These observable, gradually escalating core symptoms synergistically combine to reduce the sufferer's ability to care for himself. Major depression is expressly designed to prevent the individual from capitalizing on the fitness enhancing and maintaining opportunities commonly available in their current social niche.

The niche change model for the first time uses evolutionary communication theory to perform an analysis of major depression as a costly social display. The model explains major depression as an honest, extortionary display of need. Thus, major depression imposes escalating opportunity costs on the depressed individual - while the display's costliness renders it an honest signal of need, *it simultaneously lowers the fitness of any social partners with whom the depressed person has a strong a priori positive fitness correlation* (i.e., relatives, mates, friends and "business partners").

The depressed person is prevented not only from caring for himself, but also from dispensing normally expected benefits to all their social partners. This ultimately serves to *extort investments from*

social partners that they otherwise would deem too costly to make in spite of their receiving an honest signal of need from the depressed individual.

Fitness extortion often is necessary for obtaining substantive niche revisions even when intimate, loving and genuine positive fitness correlates are involved, with whom it is adaptive or even essential for the subject to nurture long term social exchange relationships. The need for extortion is explicable by making reference to the central premise of the model, namely, that the depressed person seeks investments (usually of indeterminate costliness) to help comprehensively revise his social niche (often in open ended or poorly defined ways). By definition, niche revision necessitates changing the structure of the social contracts the depressed person holds with many of the same social partners from whom he seeks niche-changing investments. Niche revision threatens to alter the overlap of interests with the mismatched individual's potential helpers, making it unusually difficult for them to predict whether:

1. Benefits adequate to compensate them for their assistance will flow to them under the new contracts, **and**

2. The new flow of benefits will be higher or lower than the that under the original contract.

Thus, the costs imposed on positive fitness correlates by a depression display must be great enough to overcome what is expected to be their usual preference to maintain the *status quo* social contract instead of investing in a poorly defined alternative that might entail lower long term benefits or even losses. To be successful in gaining real assistance in niche revision, the costs of depression for the fitness correlates must lead them to assess that their net benefits under a plausible incipient contract are likely to be greater than those they can expect if the displayer's depression continues or worsens. The core symptoms of major depression are inherently self-endangering, especially from a "stone age", EEA perspective. Thus, recipients of a depression display must take into account the distinct possibility that a partner's depression will lead to their death; this can occur as a consequence of their partner's reduced ability to care for him or her self in a harsh, variable evolutionary environment or more directly via depression-related suicidal behavior.

The inherent risk in the major depression strategy is that a social partner who may be persuaded to risk investing in a contract revision with indeterminate benefits may, alternatively, decide to dissolve the relationship altogether. Consequently, we do not expect all depressions to result in positive outcomes. Moreover, reduced positive fitness correlations in modern societies will decrease the extortionary power of major depression, possibly leading to highly escalated depression displays more often than in the human ancestral environment and increasing the risk of wholesale abandonment by partners of the depressed individual. However, we predict that depression may still have adaptive significance in modern environments and suggest that the use of clinical interventions that respect and are in accord with the hypothesized function of depression will be most successful in the long term. *c8*

Author's Note: A manuscript fully explaining the model was submitted to Psychological Review on 14 January 1998. A copy of the submitted manuscript is available on request from Paul Watson.

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ARTICLE:

by James V. Kohl

The Primacy of Olfaction ?

Author's Note:

This explanation parallels a more detailed (and fully referenced to 1996) explanation in my review article at the following URL: http://psych.lmu.edu/ahbe/sexes/kohl96.htm.

Here is the *"short"* version — with added comments on the role of odor in the classical conditioning of behavior.

The hypothalamus bridges experimental and clinical neuroscience via psychiatry, neurology, and endocrinology.[1] Its response, particular as reflected by hypothalamic gonadotropin releasing hormone (GnRH) pulsatility is conditioned by afferents, depending on which are simultaneously active. Hypothalamic GnRH pulsatility has been repeatedly touted as the biological core of mammalian reproductive sexual behavior. Any sensory input that affects hypothalamic GnRH pulsatility can reasonably be expected to influence behavior, regardless of which afferents are involved.

Hypothalamic GnRH pulsatility cannot be directly measured in humans. However, since GnRH pulsatility modulates pituitary gonadotropin release, the measurement of luteinizing hormone (LH) and follicle stimulating hormone (FSH) is considered to accurately assess GnRH pulsatility changes in humans. Additionally, since LH/FSH ratios are directly linked to gonadal steroidogenesis, and indirectly linked to adrenal steroidogenesis, the measurement of steroid hormones like estradiol and testosterone, also appear to reflect the influence of GnRH on LH/FSH ratios, and on the hypothalamic-pituitary-gonadal/adrenal (HPG/A) axes.

Mammalian pheromones appear to activate early gene (c-fos) expression in GnRH neurons. They also influence LH/FSH ratios and steroidogenesis. Stern and McClintock[2] clearly demonstrate that human female pheromones alter the timing of the ovulatory LH surge, which is prompted by increasing levels of estradiol. Other recent studies referenced on my website indicate that human pheromones influence LH, FSH, and testosterone.

Thus, a direct link from human pheromone exposure (in what may include social-environmental circumstances) to GnRH, LH/FSH ratios, and to steroidogenesis, is complete. This link parallels the mammalian link from pheromones to behavior.

In other mammals, steroidogenesis (particularly testosterone and estradiol) alters neuronal firing patterns and the binding of neuronally released transmitters, either prenatally or postnatally.[1] These alterations form a basis for linking steroidogenesis and behavior—both prenatally and postnatally. Human pheromones appear to activate the prenatally established mammalian pathway that modulates steroidogenesis, and the link between sex steroid hormones and behavior is generally accepted. This provides reason to believe that a link between human pheromones and behavior would be generally accepted. (It is not accepted; there's no proof!)

In any case, a human pathway from sensory stimuli in the social environment to gene activation in cells of nerve tissue in the brain (the most important organ of any organ system that affects behavior) has been demonstrated, albeit indirectly, by Stern and McClintock.[2] This pathway does not require the involvement of non-olfactory stimuli. In the absence of data to suggest that visual, tactile, auditory, or gustatory stimuli directly activate this pathway, the olfactory link between the social environment, genes, and behavior could be considered a primary link between social-environmental chemical stimuli and behavior. (It is not considered a primary link; there's no proof!)

If non-olfactory sensory input appeared to directly alter gene expression in GnRH neurons of brain tissue, there might be some minimal support for the primacy of non-olfactory stimuli in the classical conditioning of human behavior. Furthermore, I am completely unaware of any mammalian model that promotes the primacy of non-olfactory stimuli either in sexual attraction or in the development of behaviors required for properly timed reproductive sexual behavior. (In short, there's no proof!) There is no solid neuroendocrine support for the primacy of visual stimuli.

The effect of human pheromones on LH secretion is a primary consideration in establishing the primacy of olfaction over vision, because LH/FSH ratio regulate steroidogenesis, thus linking human pheromones to behavior.[2] References cited in the review article on my website attest that the effect of human pheromones on LH secretion is innately sexually dimorphic, occurs within minutes of birth and continues to occur throughout the human lifespan. However, whether or not this form of communication is accepted as a primary link between social-environmental sensory stimuli and behavior, seems largely to depend on the demonstration that human pheromones can classically condition sex steroid hormone-related behavior.

Odors classically condition infant behavior. Sullivan et al.,[3] documents that day-old infants have the capacity for olfactory classical conditioning. This suggests that the capacity for olfactory recognition and attraction in humans is genetically established before birth. Also, infants at one-two week's old orient more toward a familiar than toward a novel perfume odor.[4] This preferential orientation appears to be extinguished two weeks after the mothers stopped using the perfume, and also suggests that odor conditioning must be ongoing in the neonate.

Experiences with naturally secreted odors (e.g., putative human pheromones) begins at birth. Putative human pheromones influence adult behavior. Kirk-Smith, Van Toller, & Dodd[5], have demonstrated how odors might acquire values through pairing with emotionally laden experiences. Kirk-Smith, Booth, Carrol, & Davies[6], have shown the effect of naturally secreted pure odor on sexual and agnostic relations between humans. This indicates that putative human pheromones may affect adult human judgements. However, to acquire any meaning, these experiences must somehow influence neurotransmission in the brain.

Odors influence neurotransmission. No data on any physiological correlates of the affected adult human judgements was included in early studies. However, animal studies, most notably Rangel & Leon[7], have reported anatomical and functional changes in the region of the olfactory bulb processing an odor. These changes can be expected to alter neurotransmission, and they accompany the development of early olfactory attraction in rodents. Similarly, some human studies support that anatomical and functional changes in neurotransmission in the brain may occur upon exposure to odors. Zald & Pardo[8] support a critical role of the human amygdala in either the processing of aversive olfactory stimuli, or the transduction of neural signals from smells into emotional responses.

It seems obvious that odors influence the brain and emotion, and that mammalian and putative human pheromones may do so by influencing GnRH and LH/FSH ratios (which are linked to sterodogenesis, neurotransmission, and behavior). So, here are some LH and other correlates of olfactory conditioning:

LH release can be conditioned in male rats by repeated exposure to a female in a box containing an arbitrary odor. This box ultimately comes to elicit LH release by itself. Thus, it is likely that social environmental odor cues, which males learn to associate with sexual activity (e.g., pheromones), induce changes in the HPG and HPA axes, and that classical conditioning may be used to evoke functional changes in the neuroendocrine pathways that mediate the release of LH.[9] These are the same neuroendocrine pathways that have repeatedly been linked to steroidogenesis, behavior, and also to the immune system.

Odor paired with stress elicits immune suppression through classical conditioning. After stress-induced immune suppression is established, only the odor need be used to induce immune suppression.[10]

Based on conditioning of an odor-immune system in animals, the pairing of cod liver oil, the strong "*scent*" (i.e., odor) of rose, and intravenous cyclo-phosphamide (CY) therapy were used to classically condition the immune response of an adolescent human female. This allowed for treatment with half the cumulative amount of CY that might otherwise have been administered.[11]

Laurent[12] has observed oscillating olfactory circuits and centrifugal patterns of odor recognition in trained animals that expect an odor after conditioning. This indicates that behavioral conditions like anticipation may be involved in the odor response.

Similarly, Meyer[13] found that sniffing alcohol increases LH. Alcohol was the unconditioned stimulus that causes a reinforcing positive response. Alcoholics in withdrawal report that they can almost "smell a beer." This is an example of behavioral reinforcement through an olfactory component of an environmental stimulus that causes an incentive drug effect.

There may be insufficient data to scientifically prove beyond a shadow of a doubt that human phero-mones are the primary influence on classically conditioned human behavior. For instance, Berliner, et al.,[14] — another human study that suggested human pheromones influenced LH/FSH pulsatility — was quickly dismissed in previous debates.

However, I have seen no animal or human evidence that supports the primacy of visual, auditory, gustatory, or tactile sensory input in the classical conditioning of human behavior. **GnRH** directs the concurrent maturation of the neuroendocrine, central nervous and reproductive systems. And human pheromones influence GnRH, as shown by their effect on LH secretion. (Also, congenially blind, deaf, or dumb, people tend to exhibit a full range of reproductive sexual behaviors.) Congenital anosmia appears to inhibit expression of the full range of reproductive sexual behaviors.[15]

I could continue to debate whether human pheromones attract, repel, or even influence other humans more than other sensory stimuli in any given scenario. But that type of debate is pointless;, unless someone comes up with some facts from developmental biology to support their position on visual or other sensory aspects of attraction.

What I continue to read is that humans are primarily visual beings. I understand why people who observe other people would believe this. Even Arori Weller's review of Stern and McClintock [2] begins by saying: "Human communication is dominated by auditory and visual information." But when you consider people to be animals, the only substantiated neuroendocrine basis for their social and sexual interaction is odor. Thus, olfaction is primary!!!

I am convinced that behavior is genetically predisposed and directly influenced by the rewards/ punishments associated with cues from the social environment.

Sensory cues, signs, or signals evoke genetically predisposed behaviors due to pairing with specific contingencies of reinforcement, which shape the pattern, intensity, and durability of specific behavior. Behavior changes the environment, and creates reciprocity. With sufficient experience, certain cues promote behavior that may appear to occur without reinforcement (i.e., in the absence of reciprocity).

Olfactory and other sensory cues promote behavior that may appear to occur without reinforcement. But the reinforcement begins at birth, and is rarely considered in adult interactions. *c8*

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Editor's Note: An abstract concerning this article appears on page 22 of this issue.

ABSTRACTS & EXTRACTS...

Rugg MD, Mark RE, Walla P, Schloerscheidt AM, Birch CS, & Allan K: Dissociation of the neural correlates of implicit and explicit memory. Wafure,1998;392:595-598.

Abstract: One presentation of a word to a subject is enough to change the way in which the word is processed subsequently, even when there is no conscious (explicit) memory of the original presentation. This phenomenon is known as implicit memory. The neural correlates of implicit memory have been studied previously, but they have never been compared with the correlates of explicit memory while holding task conditions constant or while using a procedure that ensured that the neural correlates were not 'contaminated' by explicit memory. Here the authors use scalp-recorded event-related brain potentials to identify neural activity associated with implicit and explicit memory during the performance of a recognition memory task. Relative to new words, recently studied words produced activity in three neuroana-tomically and functionally dissociable neural populations. One of these populations was activated whether or not the word was consciously recognized, and its activity therefore represents a neural correlate of implicit memory. Thus, when task and memory contamination effects are eliminated, the neural correlates of explicit and implicit memory differ qualitatively.

Epstein R & Kanwisher N: A cortical representation of the local visual environment. A/a-fure,1998;392:598-601.

Abstract: Medial temporal brain regions such as the hippocampal formation and parahippocampal cortex have been generally implicated in navigation and visual memory. However, the specific function of each of these regions is not yet clear. Here the authors present evidence that a particular area within human parahippocampal cortex is involved in a critical component of navigation: perceiving the local visual environment. This region, which they name the 'parahippocampal place area' (PPA), responds selectively and automatically in functional magnetic resonance imaging (fMRI) to passively viewed scenes, but only weakly to single objects and not at all to faces. The critical factor for this activation appears to be the presence in the stimulus of information about the layout of local space. The response in the PPA to scenes with spatial layout but no discrete objects (empty rooms) is as strong as the response to complex meaningful scenes containing multiple objects (the same rooms furnished) and over twice as strong as the response to arrays of multiple objects without three-dimensional spatial context (the furniture from these rooms on a blank background). This response is reduced if the surfaces in the scene are rearranged so that they no longer define a coherent space. The authors propose that the PPA represents places by encoding the geometry of the local environment.

Diamond M, Binstock T, Kohl JV: From fertilization to adult sexual behavior. Hormones & Behavior, 1996;30(4):333-353

Research has established the broad mammalian developmental plan that genes on the sex chromosomes influence gonad development which determines gonadal hormone production (or its absence) leading to modification of the genitalia and simultaneously biasing the nervous system to organize adult sexual behavior. This might be considered the "gonad to hormones to behavior" model. It is clear, however, that although this model generally works well it is incomplete. The model does not account for behavioral influences attributed to the environment or to genetic but non-gonadal or hormonal factors. In this essay we probe those areas of sexual development that are neither differentiated by hormones nor activated by them.

The concept of the environment used for our discussion is very broad; it incorporates considerations of both the molar and the molecular levels. The general sense of the word "environment" as something exterior to the person is retained, even if that something influences intraperson processes. In addition, we focus directly on molecular events themselves. Here the "environment" involved can be that within a DNA segment.

We also expand the notion of "biologically based sex differences." Although many, and perhaps most, important sex differences arise from gonadal and hormonal development, also important are sex differences which are neither gonadal nor hormonal. All these factors affect the internal workings of the individual and intervene in structuring how the social environment might or might not modify sexual behavior. This discourse calls attention to features that are central to the so-called nature-nurture discussion.

Adler NE, Horowitz M, Garcia A, & Moyer A: Additional validation of a scale to assess positive states of mind. *Psychosomatic Medicine,* 1998;60:26-32.

Abstract:

Objective: Numerous studies have linked stress and negative states to adverse health outcomes. However, in addition to engendering negative states, stress may impair capacities to experience positive states. Such failure to experience positive states may represent a risk factor for poor health in and of itself. The research reported here examines a brief, easily repeated measure of positive states of mind (PSOM) including: focused attention, productivity, responsible caretaking, restful repose, sharing, sensuous nonsexual pleasure, and sensuous sexual pleasure.

Method: The PSOM Scale and measures of psychological distress, stress, and physical symptoms were administered in four independent samples: two samples of college students and two samples of pregnant women, one undergoing a potentially stressful medical procedure (amniocente-sis). The relationship between PSOM scores and several conceptually related, but distinct, psychological variables was examined, controlling for the effect of social desirability where necessary.

Results: The range of scores on the items of the PSOM indicated that impairments of ability to have the relevant experiences did occur in the subjects who were experiencing stress. The overall PSOM Scale score consistently showed an inverse relationship with anxiety and with indicators of stress. In addition, lower scores on the PSOM Scale were associated with more somatic symptomatology.

Conclusions: The results provide evidence that positive states of mind are linked to negative psychological states and are responsive to stressful events. The PSOM scale provides a useful, brief measure for research in stress and health.

Liu D, Diorlo J, Tannenbaum B, Caldji C, Francis D, Freedman A, Sharma S, Pearson D Plotsky PM, Meaney MJ: Maternal care, hippocampal glucocorticod receptors, and hypothalamic-pituitary-adrenal responses to stress. *Science,* 1997;277:1559-1662.

Variations in maternal care affect the development of individual differences in neuroendocrine responses to stress in rats. As adults, the offspring of mothers that exhibited more licking and grooming of pups during the first 10 days of life showed reduced plasma adrenocorticotropic hormone and corticos-terone responses to acute stress, increased kippocampal glucocorticoid receptor messenger RNa expression, enhanced glucocorticoid feedback sensitivity, and decreased levels of hypothalmic corticotropin-releasing hormone messenger RNA. Each measure was significantly correlated with the frequency of maternal licking and grooming (all r's > -0.6). These findings suggest that maternal behavior serves to "program" hypothalmic-pituitary-responses to stress in the offspring.

Kandel E: A new intellectual framework for psychiatry. *American Journal of Psychiatry,* 1998;155:457-469

Abstract: In an attempt to place psychiatric thinking and the training of future psychiatrists more centrally into the context of modern biology, the author outlines the beginnings of a new intellectual framework for psychiatry that derives from current biological thinking about the relationship of the mind to the brain. The purpose of this framework is twofold. First, it is designed to emphasize that the professional requirements forfuture psychiatrists will demand a greater knowledge of the structure and functioning of the brain than is currently available in most training programs. Second, it is designed to illustrate that the unique domain which psychiatry occupies within academic medicine, the analysis of the interaction between the social and biological determinants of behavior, can best be studied by also having a full understanding of the biological components of behavior.

Extract:

Principle 1: All mental processes, even the most complex psychological processes, derive from operations of the brain.

Principle 2: Genes and their protein products are important determinants of the patterns of interconnections between neurons of the brain and the details of their functioning.

Principle 3: Altered genes do not, by themselves, explain all the variance of a given major mental illness. Social and developmental factors contribute very importantly.... Learning, including learning that results in dysfunctional behavior, produces alterations in gene expression. Thus, all of "nurture" is ultimately expressed as "nature."

Principle 4: Alterations in gene expression induced by learning give rise to changes in patterns of neuronal connections. These changes not only contribute to the biological basis of individuality but presumably are responsible for intiating and maintaining abnormalities of behavior that are induced by social contingencies.

Principle 5: Insofar as psychotherapy or counseling is effective and produces long-term changes in behavior, it presumably does so through learning, by producing changes in gene expression that alter the strength of synap-tic connections and structural changes that alter the anatomical pattern of interconnections between nerve cells of the brain.

Goodwin J: Child sexual abuse: Controversy, sequelae, treatment. *Current Opinion in Psychiatry,* 1997; 10:432-435.

Controversy surrounds child sexual abuse and recent malpractice decisions may discourage clinicians from exploring such memories. At the same time, new research in neuropsychology and neuroimaging documents significant sequelae, a careful longitudinal study of abused children supports the concept of a cycle of violence, and the first meta-analysis of studies of survivors confirms the clustering of multiple severe symptoms characterized as disorder of extreme stress. Contexts seem to modify impact, and telling the story remains central to treatment.

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