For evolutionary psychiatrists, psychologists and others interested in sociophysiologicai integration

ASCAP

Volume 10, No. 6 (Cumulative #115) June 1997

"'A powerful molecular technique .... called gene knockout, allows the development of genetically engineered animals (mice) that lack the function of a critical gene and, hence, lack its protein product."

Marc G. Caron

Contents

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, Dept. 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.

Concerning paleobiology, sociophysiology, interpersonal and group relations, and psychopathology

ASCAP Society Executive Council:

President: Kent G. Bailey President-Elect: Daniel R. Wilson 1st **Vice President:** Mark Erickson **2nd** Vice **President:** Russell Gardner **Just** Past **President:** Leon Sloman

Previous Past Presidents:

Michael A. Chance
John S. Price
Paul Gilbert
John Pearce

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 psycho-pathologically related states. We are interested in the integration of various methods of study ranging from cellular processes to individuals in groups.

Across Species Comparison and Psychopathology (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 of the ASCAP Society.

Editor-in-Chief: Russell Gardner, Jr. Dept. of Psychiatry & Behavorial Sciences Room 4.450, Marvin Graves Building, D-28 University of Texas Medical Branch Galveston TX 77555-0428 Tel: (409)772-7029 Fax: (409)772-6771 E-Mail: rgardner@utmb.edu

European Editor: John S. Price
Odintune Place
Plumpton East
Sussex BN7 3AN
ENGLAND (01144)27-389-0362 Fax: (01144)27-389-0850 E-Mail: 100042.2766@compuserve.com

Managing Editor: Frank Carrel
Dept. of Psychiatry & Behavorial Sciences
Room 1.103, Marvin Graves Building, D-28
University of Texas Medical Branch
Galveston TX 77555-0428
Tel: (409)772-3475
Fax: (409)772-4288
E-Mail: ascap@utmb.edu

Previous volumes are available. For details, contact Frank Carrel, the Managing Editor of *The ASCAP Newsletter,* at the address above.

The WWW Address for the The ASCAP Home Page is:

http://psy.utmb.edu/ascap

The WWW address for membership & subscription is:

http://psy.utmb.edu/ascap/aform.htm

The WWW address for the European ASCAP Home Page is:

http://evolution.humb.univie.ac.at/ascap/europe/index.html

ADDRESSED TO & FROM ...

Annual ASCAP Society Meeting Agenda

By Russell Gardner, Jr.

To reiterate and remind, our annual meeting is 8:00 a.m. to 5:00 p.m. Wednesday, 4 June 1997, just before the HBES meeting in Tucson, Arizona, USA. Our meeting hotel is the Plaza Hotel where we have reserved a meeting room for about 50 people.

President Bailey has declared that psychotherapy is the primary focus of the meeting. We expect ample discussion. We have the welcome news that John Price, Chairman of the Psychotherapy Section of the World Congress of Psychiatry, plans to be there.

A more final version of the program is as follows:

8:00 a.m.	Kent Bailey Welcome and keynote address
8:45 a.m.	Dan Wilson
9:15am	Ferdo Knobloch
9:45 a.m. 10:00 a.m.	Break
10:00 a.m.	John S. Price
10:30 am	Andy Thompson
11:00 a.m.	Brant Wenegrat
11:30 a.m.	Helen Wood
12:00 noon	Lunch has been ordered in for continuation of informal discussion
1:00 p.m.	Presentation by Beck Award
1:45 p.m.	Lynn O'Connor & Jack Berry
2:10 p.m	The Bakkers
2:30 p.m. to 2:40 p.m	Break
2:40 p.m	Open Discussion
3:10 p.m	Randy Nesse
3:40 p.m	Leon Sloman
4:10 p.m.	Russell Gardner
4:40 p.m	Business meeting

See you-all there! We would also like to put out a call for items for the business meeting.

Genetics & Social Behavior of Dogs

Those interested in the genetics of behavior may be interested in: *The Antisocial Personalities.* Hillsdale, New Jersey: Erlbaum, 1995 by David Lykken. (Lykken is co-author with Bouchard, McGue, Segal, & Tellegen of the *Minnesota Study of Twins Reared Apart.)*

Pages 13-16 of *The Antisocial Personalities* are entitled, 'An Animal Model - The Bull Terrier.'

"Having owned two bull terriers over the years, I have found my acquaintance with this breed more useful to an understanding of criminality than the perusal of most texts on the subject. The bull terrier was created as a fighting dog in the early 19th century by cross-breeding the English bulldog, for its strength and resolution, with the white English terrier, for its agility and grit. The bull terrier's quickness and tenacity, his powerful jaws, his relative fearlessness, and high pain threshold made him a formidable gladiator." **(page 13)**

" *The unsuspecting owner who just wants a dog around the house and chooses a bull terrier just for the breed's distinctive muzzle is in for a shock when he discovers that he has adopted a juvenile delinquent."* **(page 13)**

"But, reared by an authoritative parent, one able to set firm limits while providing affectionate reinforcement for acceptable alternative behavior, that same animal could become a favorite of the neighborhood..." **(page** 15)

Like Lykken, I've also owned 2 bull terriers and found just about everything he says about the breed to be the gospel truth. And while my two dogs were very alike in common bull terrier characteristics, they certainly had very distinct personal identities (even though both were male).

The City of San Francisco, to its credit, has now started a program to rehabilitate confiscated pit bulls (a related, but sometimes mixed breed). Instead of euthanizing all pit bulls seized from irresponsible owners (some of whom actually illegally fought the dogs), those

that show promise are put through socialization training and are given to owners who also go through training and must periodically demonstrate the good behavior of their new pets. The dogs are officially re-named 'St. Francis Terriers' and have indeed become local celebrities of sorts.

I recommend Lykken's -- *The Antisocial Personalities* -- whether you're interested in bull terriers and dogs in general, or not. Anyone contemplating getting a Bull Terrier or similar breed would do well to consult *Pit Bulls and Tenacious Guard Dogs* by Carl Semencic; Neptune City, New Jersey: TFH, 1991.

Frank Miele
Senior Editor, Skeptic Magazine
FMieleX@aol.com

A Comparatively Good Poem

This poem was written as a straightforward subject-revalatory monologue. For those interested, it's essentially Alan Bennett ("Talking Heads") out of Robert Browning ("My Last Duchess"). "In the real world" it will go under the heading "Second Consultation" or, perhaps, "Denial". However for my old friends in ASCAP, I thought I could repackage it as:

"Extract from comparator gene operating manual: Reproductive strategy in the event that your parent(s) seem to think you are of little value".

Of course I loved my mother; don't we all?
She had her faults, slovenly, sometimes cruel,
But we can't help what we are. My Aunt Ruth
Called her a slut who got lucky. There's truth
In that. I guess that she snared my father
With the usual claims of pregnancy. Rather
Sad; but he never knew what he had bought.
Within six months, on his last op., he caught
A hail of flack over Bremen. So I
Never knew him. But my yearning to fly
Was inspired by what he'd achieved. I had
My education paid for by his dad.
Each time I came home there was a new man
With his feet under the table. I can
Still feel the dread as I walked up the path.
Filth in the kitchen and scum in the bath.
We just lost touch. Class must have played a part.
I was Lancing College and Cranwell. Her start
and finish was council school. Such a gap
Is either the cause of pride or it'll sap
The common bond. In our case the latter.
As I say, we just grew apart.

Oh no, not the business about the cash
For the Morgan. I sensed I had been rash
To mention that. I know that I began
To blub talking about it, but I ran
Out of steam. Silly nonsense with the pills
had only just happened. Don't think it fills
My life. It's not important. It's all past.
Go over the facts? Provided it's the last
Time we talk about it. It was thirty-five
Years ago, for Christ's sake. Why do you strive
To make something out of nothing? I bailed
out over jungle when my engine failed.
It took me three weeks to walk back to base.
Reported missing, believed killed; had to face
The fact that my mother now thought me dead.
Telegrams unanswer'd; strange thoughts in my head,
I was given some leave. As I arrive
Back home, I can see, on my mother's drive,
A new car. Mother's off on the wrong foot.
Truculent not sorry; I'm asked to put
Myself in her place. Son's dead, she and Stan
Need a car. They looted my Morgan
Cash the day they heard. Yet she took the line,
If fault there was, the fault was somehow mine.
So after that, we never met again.
I soon forgot; there was no lasting pain.
Why was it I never married? I just
Didn't find a girl I could really trust.
Even you can't blame my mother for that.

Mike Waller mwaller@comparator.win-uk.net

ARTICLE:

by Edward H. Hagen

Delusions: An Evolutionary Hypothesis

Department of Anthropology, University of California at Santa Barbara, Santa Barbara, California

Abstract

Objective: I will explore the hypothesis that non-bizarre delusions are universal psychological adaptations that evolved to mitigate the dangerous consequences of social exclusion and ostracism. When we lived in small, kin-based groups, delusions may have functioned to combat social exclusion by closely mimicking conditions, such as external threats or illness, where fellow group members were likely to cooperate and provide assistance.

Method: If delusions are an adaptation to social exclusion, they should onset when an individual faces a serious social threat, they should function to prevent exclusion in traditional settings — at least in the short term — and they should cease when the social threat ceases, an hypothesis which is examined in the context of numerous published studies of Delusional Disorder (DD).

Results: Studies of the role of life events in DD, prison psychoses, and the association between both sensory impairment and immigrant status with DD all indicate that social deficits play a significant etiological role. Cross-cultural data collected in traditional social settings show that individuals with delusions often receive social benefits. Finally, studies show that positive social variables are the most important predictors of good outcome for DD. Conclusions: A case can be made that delusions are adaptations to social exclusion. Proof will require controlled studies showing that social threats cause these symptoms, that afflicted individuals living in traditional contexts frequently receive social benefits, and that these symptoms subside if and when the social threat subsides.

Introduction:

Several authors have proposed that certain psychiatric symptoms and syndromes may be adaptations to Pleistocene social environments.[1-12] I examine this proposal for the case of non-bizarre delusions. Because delusions occur in a large number of syndromes, usually in concert with other cognitive and affective symptoms, I will focus primarily on Delusional Disorder (DD) as a preliminary test of the hypothesis. Individuals with DD are cognitively, emotionally, and physically unimpaired, and their only symptom is a delusional framework. Although DD is rare, delusional symptomology is not: the prevalence of persecutory delusions with auditory hallucinations alone is approximately 0.7% (ECA study). Delusional systems appear to take a very similar form in many different societies,[13] and common delusional themes like persecution and grandiosity have a striking social significance.

Just as nausea and fever are physiological adaptations to toxins and infections, delusions may be an evolved psychological adaptation designed to mitigate the serious dangers of social isolation and exclusion,[14-16] experiences that appear to play a significant etiological role in this disorder. In the human Environment of Evolutionary Adaptedness (EEA) — the environment in which we evolved and to which we are adapted[17] — our ancestors lived in small, somewhat isolated kin-based groups with occasionally hostile neighbors. In this (as opposed to a modern) social environment, delusional individuals may have been able to unconsciously deceive others[18] in order to receive badly needed social benefits like food and protection. If this hypothesis is correct, delusions are not a disorder at all, but rather an innate and functional aspect of adult psychology.

Theoretical Foundations:

During the EEA, social exclusion was a deadly threat. While we might intuitively expect individuals facing such a threat to respond by attempting to please members of the group and to conform to group values, there are at least three important situations where this strategy is unlikely to succeed. The fjrst is if an individual has already substantially modified their behavior in an attempt to solidify their social position, yet still faces exclusion for whatever reason. Clearly a new strategy is needed. The second is if an individual violates a serious taboo, such as incest. Such a violation might be unforgivable. Third, if an individual angers a powerful person, perhaps by having an affair with their spouse or child, it is possible that no amount of submissive behavior will prevent their exclusion.

Individuals facing shunning and social exclusion have two critical needs: 1. Prevent further losses, and, 2. Increase social ties. I will explore vigilance strategies designed to deal with the first problem, and mimetic parasite strategies designed to deal with the second.

Socially threatened individuals need to dramatically increase their vigilance towards both the social and natural environment. Those at serious risk of exclusion must monitor their social environment very closely for any sign of threat, especially from powerful individuals. In addition, it is better for such individuals to be overly sensitive since the costs of not reacting to a real threat outweigh the costs of reacting to a false threat. These individuals also need to be very vigilant about injury and disease. In ancestral environments those who were injured or sick relied very heavily on fellow group members for care. A socially threatened person may not be able to rely on others to care for them, or they may place themselves in greater "social debt" by needing care, so they should be especially concerned about hurting themselves or falling ill.

Modern biological theory predicts that organisms that rely heavily on communication for the exchange of social benefits are particularly vulnerable to mimetic parasites.[19] A mimetic parasite is an organism that exploits other organisms by mimicking the signals used for eliciting social benefits. Certain species of beetles have evolved to mimic ant feeding signals in order to receive food, for example.[20] Given that mimicry constitutes possibly the largest single class of adaptations found among large categories of organisms[21] and that humans rely very heavily on communication for the receipt of valuable social benefits, it should not be surprising that a small fraction of individuals may be able to exploit others by mimicking cues and signals with important social implications. Deceptive mimicry is to be expected among social animals, humans in particular. A mimetic adaptation to social threats should have the following characteristics:

1. The mechanism is present in all individu als, but only activates in those who are facing the loss of important social relation ships.
2. Individuals with an activated mechanism behave in ways that are difficult to con sciously imitate, like displaying intense fear or excitement, because such displays are more likely to convince others.
3. The displays are designed to facilitate the forming of relationships and the receipt of social benefits. Convincing other group members of the existence of an external threat would be very effective in this regard.
4. Individuals can give reasons for their behavior that are difficult to independently verify, at least immediately. Examples include the claim that one possesses important information or has an intimate relationship with a high status individual.
5. The mechanism de-activates when the social threat subsides.

Many delusions appear to be examples of such exploitative mimicry. In socially dangerous

circumstances, individuals who experienced delusions were able to convincingly mimic uncommon but powerful signals that would have had a significant impact on people's social decisions. In the ancestral social environment in which humans evolved, delusional individuals may have consequently received the social benefits which they had previously been denied. Individuals who experienced delusions of an external threat, for example, would have acted very consistently with this belief, and displayed intense fear. By doing so, they may have been able to convince others of the reality of the threat, at least in the short term. External threats provided an extremely strong incentive for cooperation. Similarly, individuals who displayed genuine fears and/or symptoms of illness became eligible for care and attention,[15,16,18,22] albeit by incurring a social debt to be repaid in the future, if at all.

If delusions are mimetic adaptations, why don't they seem to work in a modern environment? Citizens of industrialized societies live in large communities with powerful militaries and extensive systems of social control, and have access to many sources of information. Delusional fears of persecution are far less likely to deceive others in these circumstances, or motivate them to cooperate in defense against an external threat. These adaptations are only likely to work in the small, somewhat isolated groups that characterize human prehistory.

Delusional Disorder:

The vigilance and mimetic hypotheses appear to account for many of the distinctive aspects of Delusional Disorder (DSM Ill-R, IV), often known as Paranoid Disorder (DSM III). It is also consistent with the hypothesis that self-deception functions to elicit social benefits.[23,26] According to the DSM IV, DD is characterized by the following clinical, etiological, and demographic features:

Clinical: The presence of non-bizarre delusions of at least one month's duration. The absence of: hallucinations, disorganized speech, disorganized or catatonic behavior, flattening of affect, markedly impaired functioning, odd or bizarre behavior, underlying medical condition, or the physiological effects of a substance.

Etiological: The significant role of adverse life events in the genesis of the disorder,[27,28] particularly those that involve isolation, exclusion, and other social deficits.[29,33]

Demographic: The association with immigrant status,[28,34-39] association with low socio-economic status,[28,33] late age-of-onset, and the extreme rarity of DD among pre-adolescents.[28]

The presence of a relatively permanent and unshakable delusional system accompanied by clear and orderly thinking is the essential clinical feature of DD.[40] The system could, in principle, be true, does not violate fundamental cultural tenets, and forms a logical and coherent whole. Oltmanns[41] lists the following defining characteristics of such belief systems, most of which enhance the ability of delusional individuals to deceive others:

1. The belief is held with firm conviction. The person's statements or behaviors are unresponsive to the presentation of evidence contrary to the belief.
2. The individual is preoccupied with (emotionally committed to) the belief, and finds it difficult to avoid thinking or talking about it.
3. The belief involves personal reference, rather than unconventional religious, scientific, or political conviction.
4. The belief is a source of subjective distress or interferes with the person's occupational or social functioning.
5. The individual does not report subjec tive efforts to resist the belief (in contrast to patients with obsessional ideas).

The Vigilance and Mimetic Systems:

If delusions are universal, evolved psychological adaptations, then the delusional system should have a similar structure in all human societies. Although cross-cultural data on DD is sparse, there is a considerable cross-cultural literature on psychotic delusions, which are components of many disorders. Because there is little evidence that delusional content per se is significant diag-nostically,[42,43] the delusional system can be examined somewhat independently of particular diagnoses for certain aspects of the these hypoth-eses. Cross culturally, the vast majority of non-bizarre delusions can be characterized by a relatively few themes: persecutory, grandiose, jealous, somatic, and erotic. The limited number of delusional themes are not random or arbitrary, but rather appear to be distinct adaptations designed to elicit social benefits through unconscious decep-tion, or to prevent future losses:

Persecutory: As noted above, socially threatened individuals need to greatly increase their vigilance of the social environment. Thus, paranoid fears of persecution by powerful individuals may have been adaptive during our evolutionary history. Fears that are highly elaborated and directed towards external threats may be better explained by the mimetic hypothesis, however. Individuals with these delu-sions can give very convincing accounts of the reputed threat, behave consistently with the delusion,[44] and give cues of genuine fear and distress.[45] Belief in an external threat provides a very strong impetus for cooperation among hu-mans, especially those living in small, autonomous bands with real enemies.

Realistic Group Conflict Theory's most recurrent and explicit proposition is that real threat causes ingroup solidarity[46-53] — the internal political divisions that cause certain individuals to be excluded vanish in the face of external dangers. Group members should readily cooperate against a possible threat because the costs of defending against a false threat are low compared to the costs of not defending against a real threat. Not surprisingly, persecutory delusions were the most common in 8 of 9 immigrant groups in one study of a cross-cultural hospital population.[54]

Somatic: Individuals with somatic delusions, which are often difficult to distinguish from Hypochondriasis,[55] are preoccupied with the fear or idea that they have a serious disease based on a misinterpretation of one or more bodily signs or symptoms. Again, socially threatened individuals need to be particularly concerned about falling ill because of the uncertainty that others will care for them. In addition, individuals displaying convincing fears or signs of physical distress or illness may encourage others to provide care as a social investment. According to evolutionary theory, when an individual receives a benefit from another, they are obligated to repay this "debt" with "interest." When a benefit is given to a needy individual, the "investor" is eligible for a greater return than when the same sized benefit is given to a less needy individual.[56,57] Thus, group members may be tricked into providing care under the mistaken assumption that they are going to receive a substantial payback.

Delusions that an individual emits a foul odor from the skin, mouth, rectum, or vagina, or that certain parts of the body are ugly or misshapen may also be indicative of the vigilance of socially threatened individuals, although an adaptive function for these delusions is not obvious.

Grandiose: Individuals are convinced they have some great (but unrecognized) talent or insight, or have a special relationship with a very important person, which indicates that they are highly valuable social partners. Because group members should prefer to socialize with those of high status, individuals who convincingly present themselves as having high status could have a considerable impact on others, at least in the short term. Erotomanic: Individuals believe that another person, usually of high status, is in love with them. Males with erotomanic delusions often attempt to rescue females from some imagined danger,[55] perhaps attempting to obtain both social and

sexual benefits. Females often believe that high status males are in love with them. Individuals who can claim an intimate relationship with a high status individual increase their own status. Jealous: Individuals with jealous delusions falsely believe their mate is unfaithful. In the EEA, socially threatened individuals would have been at great risk for losing their mates, and needed to increase their vigilance accordingly.

Each of the above delusional themes would have had a powerful, and, I argue, positive social impact in the EEA. This positive impact could have been enhanced by experiencing delusions of different types. Individuals who are attempting to gain social benefits based on defense profit by presenting themselves as valuable and important persons, for example.

Relation of Social Deficits to DD:

Social exclusion should evoke a strong psychological response of some sort in the threatened individual. Humans evolved to rely heavily on sociality, and individuals without social relationships must have faced very negative consequences. In virtually all ethnographically known small, kin-based human societies, individuals obtain protection, food, mates, child care, and other resources through their relationships with fellow group members. If shunning and exclusion were recurrent features of ancestral human social environments as they are among all ethnographically known groups,[58,59] as well as among some non-human primates,[60] they would represent a very strong selection pressure on human social psychology.

If delusions are adaptations to social threats, then social threats should cause delusions (social causation theory) rather than be the result of delusions (social selection theory). Because DD is a disorder whose symptoms are delusions and nothing but delusions, I will use DD as a test case for social causation theory whenever possible. However, since DD is a recent diagnostic category that only first appeared in DSM Ill-R, and whose predecessor Paranoid Disorder first appeared in DSM 111(1980), some of the following studies do not deal specifically with DD. Instead, they deal with psychoses that include a delusional system. Any study dealing solely with Schizophrenia was excluded, however.

Kendler[28] argues that the possible etiological role of immigration, low socioeconomic status, and low educational status, along with the low prevalence of psychiatric illness found in the families of patients with DD, support the idea that environmental factors may be more important than genetic-constitutional ones in the genesis of DD. The hypothesis that these "environmental factors" are social threats is supported by several lines of evidence.

Many researchers have favored an important role for social causation, arguably starting with Kraepelin. Kraepelin noted that while in dementia precox it was possible to observe an internal "morbid process" at work, paranoia could usually be understood as the response of certain persons to "the influence of the stimulus of life."[27] Cameron[29] was among the first to explicitly locate the genesis of delusional systems in the social arena. Cameron identifies the importance of social isolation and lack of social communication in the development of a delusional framework, and notes specifically that *"[p]aranoic attitudes and actions...grow out of a breakdown in the machinery of social cooperation."* Interestingly, he recognizes that delusional behavior may occasionally make an individual a "distinguished person and, though rarely, a leader of men," an important phenomenon that may have occurred more frequently in traditional social contexts.

Lemiert[30] retrospectively studied eight cases of persons with "prominent paranoid characteristics." Four cases involved persons admitted to the state hospital at Napa, California, with diagnoses of paranoid schizophrenia. The lack of any history or evidence of hallucinations or intellectual impairment excludes schizophrenia as a likely diagnosis for these cases, however. The others involved

persons admitted to hospitals, involved with the law, or having chronic job difficulties. One case resembled Paranoid Personality Disorder.

Lemert spent as much as 200 hours per case collecting data from anyone who played a significant role in the life of the person involved, attempting to establish the order in which delusions and social exclusion occurred. He concludes that:

> *"[t]he paranoid process begins with persistent interpersonal difficulties between the individual and his family, or his work associates and superiors, or neighbors, or other persons in the community. These frequently or even typically arise out ofbona fide or recognizable issues centering upon some actual or threatened loss of status for the individual. This is related to such things as the death of relatives, loss of a position, loss of professional certification, failure to be promoted, age and physiological life cycle changes, mutilations, and changes in family and marital relationships, (emphasis added).*"

Difficulties with the real community often result in the formation of a coalition which attempts to exclude the individual, and Lemert concludes that it is this process of exclusion and isolation which leads to the development of the delusional framework, and not the converse. Significantly, Lemert notes that paranoia emerges in situations where individuals must reach their goals through cooperation with others, which is precisely the foundation of ancestral human social environments.

Retterstol's retrospective/case-control study[31] of 301 first admission patients to the Psychiatric Clinic at the University of Oslo who presented with paranoid and paranoic symptoms, also supports a social causation theory. Based on an analysis of case notes and follow-up interviews, he found that 100% of paranoid psychosis are caused by an event that "provokes the insecurity of the individual," i.e. those that tend to isolate the individual and make him feel an outsider, either by making him unpopular within his own group, or by transplanting him to new and strange surroundings. This was true of only 54% of cases diagnosed with schizophrenia.

Kay, et. al.,[33] conducted a case-control study of 132 consecutive first admission or re-admission patients to a psychiatric hospital with diagnoses of either paranoid (n=54) or affective (n=57) psychosis. The paranoid patients were specifically diagnosed as belonging to the following ICD 8 categories: Schizophrenia, 15 cases; Paranoid States, including Paraphrenia, 38 cases; and Alcoholic Hallucinosis, 1 case. The affective diagnoses were: Hypomania, Mania, or Manic-depressive Psychosis, 11 cases; Endogenous Depression, 32 cases; Psychotic or Agitated or Involutional Depression, 10 cases; and Depression Unspecified, 4 cases.

At the onset of illness, the following features were significantly associated with the paranoid group: low social class, having few or no surviving children, living alone, and social deafness. Affective illness was significantly associated with the presence of (presumptive) precipitating factors of the illness, and with a family history of affective disorder in first degree relatives. Five variables were significant in discriminating between the two diagnoses: precipitating factors and family history of affective disorder predicted affective illness; low social class, few surviving children, and deafness predicted paranoid illness. Paranoid patients were rated more highly by both themselves, relatives, and friends on items suggesting difficulty in forming and maintaining satisfactory interpersonal relationships before the onset of the illness, and had been more solitary, shy, reserved, and suspicious, and less able to display sympathy or emotion.

Kay, et. al., concluded that their data support "a multifactorial hypothesis, according to which various adverse circumstances, especially when in

combination, such as being unmarried, having few close relatives, belonging to lower social class groups, or becoming deaf, are expected to increase the chances of hardship, insecurity and loneliness in later life. The accumulated sense of deprivation and injustice might be supposed to conduce to paranoid illness." The lack of a significant association with the personality score and either social class, unmarried state, or living alone lead them to disfavor downward social drift as an explanation for the association of the social variables with paranoid illness.

Kaffman,[61] in a retrospective study of a group of 34 individuals with DD (DSM III Paranoid Disorder), found that in every case there was a clear and realistic connection between paranoid premises, and facts and events in the patients' life. From the limited number of case studies presented, these facts and events can reasonably be interpreted as involving isolation and rejection. Kaffman emphasizes that *"authentic past and current interpersonal transactions play a dominant role in generating and activating the paranoid beliefs [emphasis added]."*

Prison Psychoses:

If social deficits cause DD, then isolated individuals should be particularly at risk, especially those who are purposefully removed from society. The association of delusional systems with solitary confinement — an extreme form of social isolation — has long been recognized.[62] Grassian[63] evaluated inmates of California's maximum security prison, Pelican Bay, in particular those in the Security Housing Unit (SHU) who are isolated 22 1/2 hours a day, and are excluded from work and group exercise programs. Of 50 inmates interviewed, 29 suffered from paranoia largely or completely attributable to SHU conditions, and 17 were actively psychotic. Whether these individuals would be diagnosed with DD is unknown.

Grassian[64] also reports on 14 inmates subjected to solitary confinement at Walpole, a maximum security prison for the state of Massachusetts. Six inmates (43%) reported ideas of reference associated with persecutory fears, although these fears did not reach delusional intensity. All denied experiencing them under any other circumstances and all reported a very rapid diminution of symptoms during periods of relief from isolation status.

Solitary confinement produces many psychiatric symptoms of which paranoid ideation is only one. Furthermore, we should not expect all inmates placed in solitary confinement to experience persecutory fears, since not all will conclude that they are being ostracized by those they consider their 'in-group.'

Sensory Impairment:

Auditory or visual impairment is often cited as a causal factor in psychoses, indirectly supporting a causal role for social deficits in the formation of delusional systems. Since human sociality relies heavily on verbal and visual communication, individuals suffering sensory impairment are arguably at higher risk for experiencing social deficits.

That hearing loss is associated with persecutory delusions has been known since Kraepelin.[65] Cooper[66,67] has found significant correlations between certain subcategories of deafness that he terms social deafness, and paranoid illness, for the same patients studied by Kay, et al (above). Rates of deafness among paranoid patients were over three times higher than expected, as compared to only 1.4 times higher for the affective patients. He concludes that prolonged social deafness is probably a factor in the etiology of paranoid psychoses occurring late in life. Other studies[68,69] have found no correlation between delusions and either deafness or blindness. Cooper stresses, however, that only in contexts where deafness leads to social isolation or exclusion does one find delusional symptoms.

In another study of these patients, Cooper and Porter[70] found that impaired visual acuity and ocular pathology are more common among the paranoid group, although the etiological signifi-

cance is less clear than for social deafness. Ziskind[71] argues for an etiological role for sensory deprivation in cataract "post-operative psychoses" (implied to be primarily of the paranoid type). There is a greater incidence of this complication than after other types of surgery where deprivation does not occur. The psychosis disappears in most cases when the unoperated eye is uncovered, and there was a progressive reduction in the incidence of psychosis with advances in operative technique which reduced the period when both eyes were covered.

Immigrants and Refugees:

Numerous studies have found high rates of delusional and paranoid symptoms among immigrant and refugee populations,[28,34-39] in some cases 40-50 times that of the indigenous population,[36,38] compared to only a 31/2 fold increase for schizophrenia.[36] Kendler[28] found rates of DD among the foreign bom to exceed rates of either schizophrenia or affective illness ($p \ll 0.0001$).

Unresolved is whether these results are best explained by social selection theory, social causation theory, or other factors. In a detailed study of paranoid symptoms and disorders among Hmong refugees living in the United States (9 cases out of a total Hmong population of 97), Westermeyer[38] notes that in 3 cases (33%) pre-emigration personality or genetic characteristics may have been significant in subsequent paranoid disorders, but in the 6 other cases (67%), no such pre-emigration factors could be found.

His study indicates that successful acculturation, assessed in several ways, is associated with low paranoid symptoms. Chiu and Rimon[39] report that 56% of the paranoid immigrants in their study had no history of psychiatric treatment prior to immigration (only 22% of these patients had a DSM III Paranoid Disorder while 61 % were classified as Paranoid Schizophrenic). These studies indicate that both social selection and social causation probably contribute to the high prevalence of delusional symptoms among immigrants.

Impoverished and Exploited Underclasses:

DD is associated with poor social and economic standing, as is mental illness in general.[72] This association seems particularly strong in the case of DD, however. Kendler, in his review of the demographics of DD as compared to schizophrenia and affective illness,[28] notes that patients with DD were more likely to come from poor economic backgrounds and to be more poorly educated than either patients with affective illness or (in most cases) schizophrenia. He argues that since schizophrenia is more likely to produce greater psychosocial disability than DD, downward "social drift" (i.e. social selection) is an unlikely explanation.

Kay, et. al.,[33] in their comparative study of paranoid vs. affective illness also found the paranoid patients to be significantly associated with low social class as compared to the affective patients. They, too, disfavor the social selection hypothesis.

Age-Of-Onset Hypothesis:

DD should not be activated in individuals for whom the deceptive guise would be implausible. Although children faced serious social threats, in particular the loss of one or both parents, it would have been unlikely that they could mitigate these threats by experiencing persecutory or grandiose delusions.

Why would enemies be targeting a particular child? Could a child reasonably pass him or herself off as being high status or having valuable social connections? Adults were unlikely to view children as important social partners. DD should therefore be extremely rare among children, a surprising conclusion given that children often fear imaginary dangers.

The demographic data on DD support this hypothesis. Few individuals younger than 20 are diagnosed with DD (Figure 1). Cases of DD in individuals younger than 15 are almost unheard of, though mental illness in children is itself not uncommon.[72]

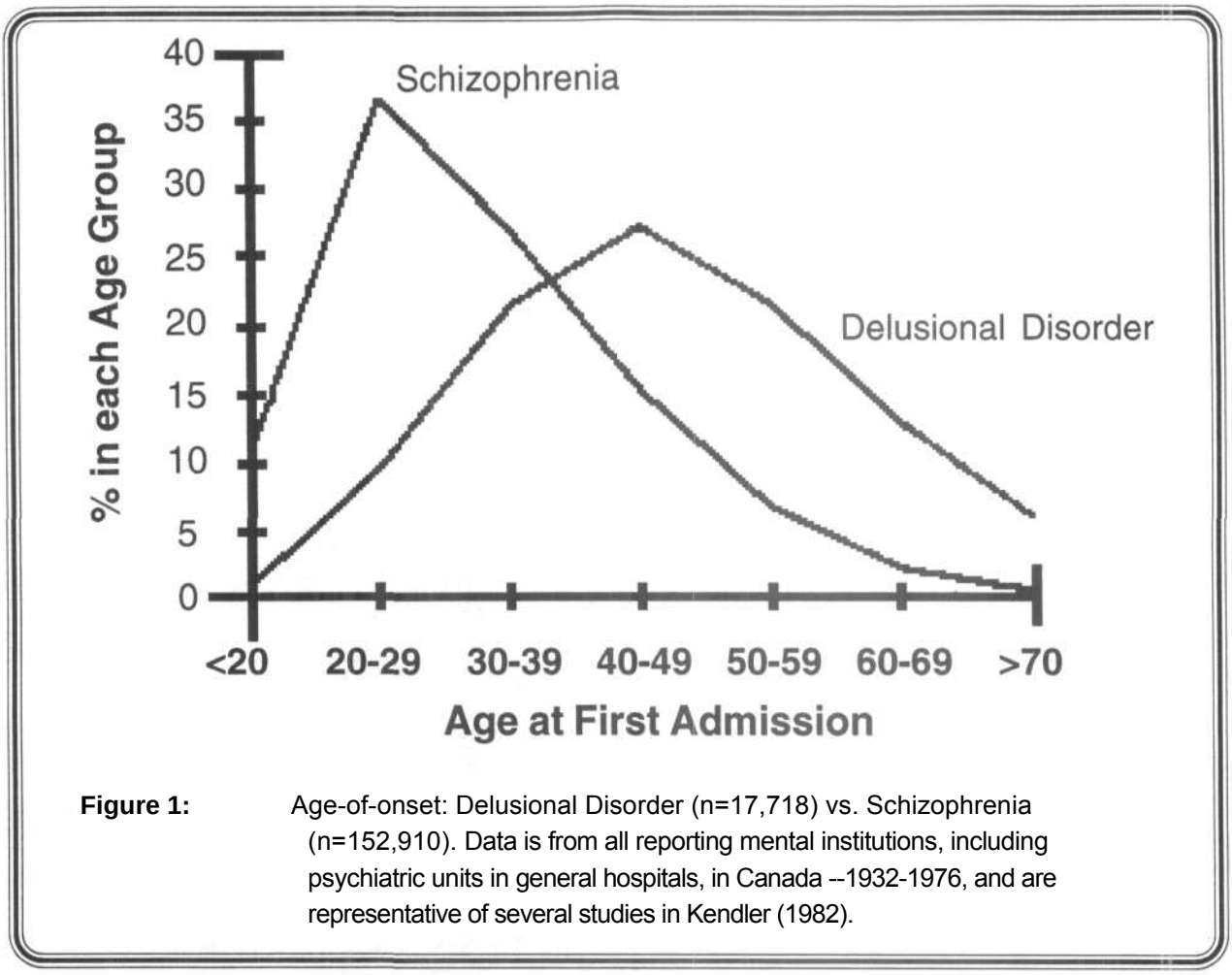


Figure 1: Age-of-onset: Delusional Disorder (n=17,718) vs. Schizophrenia (n=152,910). Data is from all reporting mental institutions, including psychiatric units in general hospitals, in Canada --1932-1976, and are representative of several studies in Kendler (1982).

A diagnosis of DD requires the presence of a delusional system, and such systems appear very rarely in pre-adolescents. Of the scattered case reports on delusional systems in children, almost all are examples of shared psychosis, with a parent as the primary partner. One small study, however, presents convincing evidence of delusional systems with both persecutor/ and grandiose features in 13 pre-adolescents.[73] The subjects ranged in age from 8 yrs, 11 months to 13 years, 9 months (mean = 12 yrs, s.d. = 18 months), and their diagnoses were classified as "borderline psychosis with paranoid features, childhood schizophrenia with paranoid trends, and paranoid schizophrenia." These children may be the exceptions that prove the rule.

Subjects shared two characteristics of relevance to this hypothesis. First, the children experienced social isolation and rejection by their peers. Second, "most had an astonishing facility for engaging in rather appropriate though pseudo-adult and affectless conversations and interactions with adults." It appears that the very rare child who presents persecutory delusions may also present themselves as an adult, a suggestive correlation. These children did not have exceptional IQ's as a group (range: 80-121, mean = 101, s.d. = 10.5), so their notable and convincing adult behavior cannot be interpreted as an artifact of intelligence.

In contrast to persecutory delusions, somatic complaints or fears of illness are a viable means for children to exploit others, especially close kin. Children require social and material investments, and, as discussed above, somatic complaints are designed to elicit such investments. Children are also eligible for these investments from kin. It is

interesting that the demographics of Somatization Disorder supports this hypothesis: 40% of individuals with Somatization Disorder report onset at age 10 or earlier; 55% report an age of onset of 15 years or earlier.[74]

Evidence that DD Provides Social Benefits:

The mimetic hypothesis predicts that individuals experiencing DD in small, kin-based societies will receive social benefits. Only a tiny fraction of the world's population currently lives in such societies, however, making this prediction extremely difficult to test. Although the data are sparse, there are a handful of case studies that appear to illustrate aspects of the proper functioning of delusions not seen in Western contexts: close relatives appear to take delusions seriously and often provide benefits as a result.

Eastwell[75] reports on the Five-Year Epidemiologic Survey of Northern Aboriginal Reserves: 57 individuals out of a total population of 10,500 were diagnosed with reactive psychosis, or 'fear-of-sorcery' syndrome. Characterized as an anxiety state with paranoid features magnified to psychotic proportions, the patient fears imminent death from sorcery, and the severity of the concurrent auto-nomic signs is noteworthy. Paradoxically, the actual practice of sorcery is extremely difficult to document among these groups, and some doubt its existence altogether.[76] Nevertheless, Eastwell observes that fellow clan members do not reject the patient, but rather "close ranks in indignation against the putative sorcerers," exactly the outcome predicted by the mimetic hypothesis.

Waxier[77] presents a case study of a psychotic episode (probably a Brief Psychotic Disorder under DSM IV) that may illustrate both the cause and function of delusions. A young Sinhalese woman was given in marriage to a man from an adjacent village who had chosen her against the wishes of his own family. The woman, fearing that her new sister-in-law was attempting to poison her, fled to a nearby temple where her husband found her crying, incoherent, seeing demons and hearing voices. Her father organized small protective rituals, and then arranged an expensive all-night ceremony to which her in-laws were invited. After the ceremony, her symptoms disappeared, and she went to live with her husband's family with no recurrence. It is possible that the women viewed her marriage into a family that didn't want her as a very socially threatening situation. Her subsequent delusion that her sister-in-law was attempting to poison her appears ultimately to have increased both her and her in-law's connections with her family, and her beliefs about demons seem to have been taken seriously by her family.

El-Islam[78] studied the disappearance of delusions among a group of psychotics from the Arab Gulf states. The patient often attributes the disappearance of his delusions to relatives dealing with the object of delusion through prayer or through traditional healers, or the delusion may become "absorbed" into a cultural belief system and lose its force, illustrating that close kin may take delusions seriously, and by doing so, alleviate the symptoms. The phenomenon where members of the social group accept an individual's delusional framework is also noted by Murphy[79] and Westermeyer.[13]

Cases of Shared Psychosis (Folie a Deux) may provide additional evidence for the ability of individuals with DD to deceive others and acquire benefits. According to DSM IV:

> *"[T]he primary case in Shared Psychotic Disorder is [usually] dominant in the relationship and gradually imposes the delusional system on the more passive and initially healthy second person.... If the relationship with the primary case is interrupted, the delusional beliefs of the other individual usually diminish or disappear."*

Newhill[80] presents a case of shared psychosis that also illustrates the receipt of cooperative benefits from relatives. After being placed under a

voodoo curse, a Jamaican man had immediately become very sick. His wife, with the help of her family, was able to move both her husband and herself to San Francisco to escape the magic. Although the husband initially experienced some relief, he and his wife soon became convinced that they would die from the magic. The couple became so terrified and agitated that both required immediate admission to the local psychiatric inpatient unit. Separated from her husband, the wife's psychotic symptoms soon cleared, while the husband remained acutely paranoid. No information is given on the husband's social circumstances, or the wife's family's motive for helping the couple move.

The following facts argue against this interpretation of Shared Psychotic Disorder: first, the disorder appears to be quite rare, although it is likely that many cases go unnoticed.[81] Second, many of the secondaries have a preexisting psychiatric disorder, personality disorder, dementia, mental retardation, physical disability or a language barrier.[82] Finally, since individuals with Shared Psychosis are often closely related, there may conceivably be genetic factors involved as well.[83] Nevertheless, the receipt of social benefits by deceiving others via a delusional system is recognized by the DSM, and is also supported the very few reports on the social response to delusions in kin-based societies.

Social Benefits & The Remission of DD:

According to the vigilance and mimetic hypothesis, delusions and persecutory fears should remit in individuals who receive sufficient social benefits. Jorgensen and Aagaard[84] studied the relationship of a number of social variables to impairment, remission, and relapse. They found that the variables of living alone, having few social contacts, and not working prior to admission were by far the best predictors of poor outcome for this group of patients. On the other hand, being married, living with others, having frequent social contacts, working full-time, and belonging to high status social groups were important predictors of good outcome. Jo rgensen and Aagaard conclude that social variables like having social contacts and useful work are more valuable than any of the clinical variables, but note that never is even one half of course and outcome variance predicted. Unfortunately, the data presented by these researchers do not favor social causation over social selection theories, but do show the strong and necessary association of positive social variables with the remission of DD.

Conclusion:

The vigilance and mimetic hypotheses are consistent with many of the clinical, epidemiological and demographic aspects of Delusional Disorder: the presence of delusions, specific categories of delusions, emotional involvement with delusions, lack of impaired functioning, distribution of age-of-onset, association with immigrant and low socio-economic status populations, and apparent etiologi-cal role of isolation, exclusion, and other social deficits.

During our evolutionary history, individuals facing the deadly threat of social exclusion would have had to monitor their social and physical environment very carefully, and may have had no choice but to convincingly mimic, albeit unconsciously, important social cues in order to maintain their status as members of the group. Delusional themes appear designed to generate precisely those cues that would have elicited cooperation from the group: fears of external threat, possession of important information, illness, and intimate relations with high status individuals. Each of these situations would have been difficult for other group members to verify, at least in the short term, making them ideal candidates for deceptive, exploitative mimesis. Many lines of evidence indicate that social deficits cause delusions, and the sparse data that are available indicate that delusional individuals in small, kin-based societies receive social benefits, at least in the short term. Proof of the mimetic hypothesis will require controlled studies of the etiological role that social deficits play in delusional symptomology, and convincing evidence that delusional individuals are frequently able to obtain social benefits in small societies.

From a clinical perspective, the mimetic hypothesis has important implications for treatment. First, an individual's assessment of their social environment will need to be obtained. This is non-trivial. Humans may or may not have conscious access to this information, and delusional individuals may have particularly limited access in order to conceal their precarious situation from themselves and others. In addition, current models of human sociality are primitive and untested. The kinds of information that humans use to assess their social environment, and the emotional and other forms in which this information is encoded are largely unknown. Once the social environment is determined, strategies will need to be devised that allow the individual to compete successfully for important social resources. Again, this is non-trivial. It is difficult to regain status after it has been lost. Nevertheless, none of these obstacles is insurmountable, and successful treatment of delusional individuals should be possible in most cases.

Other pathologies may also represent examples of exploitative mimicry in humans. Hypochondriasis and Somatization Disorder may function similarly to somatic delusions. Manic episodes may be the attempts of socially threatened individuals to present themselves as very high value social partners, similar to grandiose delusions. Transvestic fetishism may represent the attempt by ostracized males to sexually exploit other males by presenting themselves as nubile females. These hypotheses will be explored in subsequent papers. O3

The International Society for Human Ethology

http://evolution.humb.univie.ac.at/jump.html

ETHOLOGY AND EVOLUTION ON THE WEB

The Origin of species: The whole text online!

Research and Teaching tools

Studying Human Ethology - Evolutionary Psychology and related fields

Electronic Journals

Societies

Starting points for your search

Ethology and evolution research institutions

Discussion Culture

and Psychology

Neuroscience

General Psychology

Miscellaneous

Usenet

Visit their web site at: http://evolution.humb.univie.ac.at

Announcement

AT AN EUROPEAN INTERNET SITE NEAR YOU!

THE ASCAP EUROPEAN HOME PAGE!

http://evolution.humb.univie.ac.at/ascap/europe/index.html

Dr. Karl Grammar has placed this web site on the server that is at the University of Vienna, in Vienna, Austria. Many thanks to him!

ABSTRACTS a EXTRACTS ...

Knobloch, F: Musical experience as interpersonal process: Revisited. ***Contemporary Music Review,*** **1996;6(19):297-310**

Abstract: In the early sixties, the author formu-lated an interpersonal hypothesis of music, stating that music iconically signifies not only the emotions of the subjects, but also their interpersonal tendencies. He designed several experiments to test the hypothesis, and conducted them with co-workers in 1964 and 1968. Invariably, statistical tests of significance supported the hypothesis.

On the basis of these and other experiments in various cultural settings, the author, a psychiatrist and psychotherapist, conceives musical experience within a conceptual framework of mental life. A small social group is a basic behavioral unit for human beings. Even when alone, we live in a fantasy group, a "group schema". Music is a way to participate in fantasy in group processes, as are dreams and play. It is shown how this fits into a broader context of evolutionary psychology, ethology, and sociobiology.

The potential of the hypothesis of music, interpersonal tendencies, group schema, role schema, self-schema, ethology, and sociobiology of music.

Meerlo, P.; Pragt, B.J.; & Daan S.: Social stress induces high intensity sleep in rats. ***Neuroscience Letters,*** **1997;225:41-44**

Abstract: We studied the effect of social stress on sleep electroencephalogram (EEG) in rats. Animals were subjected to a single social defeat by introducing them in the cage of an aggressive male conspecific for 1 hour. The animals responded to the social conflict by a sharp increase in EEG slow-wave activity (SWA) during non-rapid eye movement sleep (NREM) afterwards. Since SWA has been identified as an indicator of sleep intensity, the results suggest that acute stressors may accelerate the build up of sleep debt. Sleep intensity may, thus, not only depend on the duration of prior wakefulness but also on the nature of the waking experience. The strong increase in SWA after social defeat, indicates that sleep may function to offset the mental loads imposed on the nervous system during wakefuiness

---

Courtney, S.M.; Ungerieldert, L.G.; Kell, K.; & Haxby, J.V.: Transient and sustained activity in a distributed neural system for human working memory. ***Nature,*** **1997;386:608-611**

Abstract: Working memory involves the short-term maintenance of an active representation of information so that it is available for further processing. Visual working memory tasks, in which subjects retain the memory of a stimulus over brief delays, require both the perceptual encoding of the stimulus and the subsequent maintenance of its representation after the stimulus is removed from view. Such tasks activate multiple areas in visual and prefrontal cortices'-'. To delineate the roles these areas play in perception and working memory maintenance, we used functional magnetic resonance imaging (FMRI) to obtain dynamic measures of neural activity related to different components of a face working memory task-non-selective transient responses to visual stimuli, selective transient responses to faces, and sustained responses over memory delays. Three occipitotemporal areas in the ventral object vision pathway had mostly transient responses to stimuli, indicating their predominant role in perceptual processing, whereas three prefrontal areas demonstrated sustained activity over memory delays, indicating their predominant role in working memory. This

distinction, however, was not absolute. Additionally, the visual areas demonstrated different degrees of selectivity, and the prefrontal areas demonstrated different strengths of sustained activity, revealing a continuum of functional specialization, from occipital through multiple prefrontal areas, regarding each area's relative contribution to perceptual and mnemonic processing.

Cabeza, R.; Kapur, S.; Craik, F.I.M., McIntosh, A.R.; Houle, S.; Tulving, E.: Functional neu-**roanatomy of recall and recognition: a PET study of episodic memory.** ***Journal of Cognitive Neuroscience,*** **1997;9(2):254-265**

Abstract: The purpose of this study was to directly compare the brain regions involved in episodic-memory recall and recognition. Changes in regional cerebral blood flow were measured by positron emission tomography while young healthy test persons were either recognizing or recalling previously studied word pairs. Reading of previously nonstudied pairs served as a reference task for subtractive comparisons. Compared to reading, both recall and recognition were associated with higher blood flow (activation) at identical sites in the right prefrontal cortex (areas 47,45, and 10) and the anterior cingulate.

Compared to recognition, recall was associated with higher activation in the anterior cingulate, globus pallidus, thalamus, and cerebellum, suggesting that these components of the cerebello-frontal pathway play a role in recall processes that they do not in recognition.

Compared to recall, recognition was associated with higher activation in the right inferior parietal cortex (areas 39,40, and 19), suggesting a larger perceptual component in recognition than in recall. Contrary to the expectations based on lesion data, the activations of the frontal regions were indistinguishable in recall and recognition. This finding is consistent with the notion that frontal activations in explicit memory tasks are related to the general episodic retrieval mode or retrieval attempt, rather than to specific mechanisms of ecphory (recovery of stored information).

Ullman, M.T.; Corkin, S.: Coppola, M.; Hickok, G.; Growdon, J.H.; Koroshetz, W.J.; & Pinker, S.: A neural dissociation within language: evidence that the mental dictionary Is part of declarative memory, and that grammatical rules are processed by the procedural system. ***Journal of Cognitive Neuroscience,*** **1997;9(2):266-276.**

Abstract: Language comprises a lexicon for storing words and a grammar for generating rule-governed forms. Evidence is presented that the lexicon is part of a temporal-parietal/medial-temporal "declarative memory" system and that grammatical rules are processed by a frontal/ basal-ganglia "procedural" system. Patients produced past tenses of regular and novel verbs (looked and plagged), which require an -ed-suffixation rule, and irregular verbs (dug), which are retrieved from memory.

Word-finding difficulties in posterior aphasia, and the general declarative memory impairment in Alzheimer's disease, led to more errors with irregular than regular and novel verbs. Grammatical difficulties in anterior aphasia, and the general impairment of procedures in Parkinson's disease, led to the opposite pattern. In contrast to the Parkinson's patients, who showed suppressed motor activity and rule use, Huntington's disease patients showed excess motor activity and rule use, underscoring a role for the basal ganglia in grammatical processing.

Paller, K.A.; Acharya, A., Richardson, B.C., Plaisant, O., Shimamura, A.P., Reed, B.R., & Jagust, W.J. Functional neuroimaging on cortical dysfunctioning in alcoholic Korsakoff's Syndrome. ***Journal of Cognitive Neuroscience,*** **1997;9(2):277-293.**

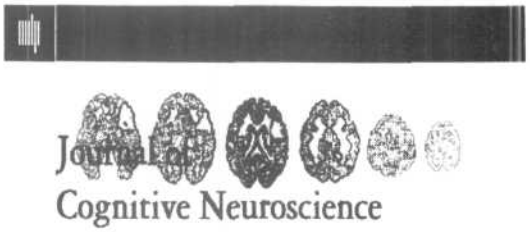
Abstract: Many neuropsychological investigations of human memory have focused on the amnesic deficits of alcoholic Korsakoff's syndrome. Structural neuroimaging suggests that the syndrome results from midline diencephalic damage, but functional neuroimaging has the potential to reveal additional neuropathology that may be responsible for cognitive dysfunction.

Accordingly, high-resolution positron emission tomography (PET) was used to measure regional cerebral metabolic rates for glucose utilization in five alcoholic Korsakoff patients and nine alcoholic control subjects. Results from a continuous recognition test administered during the radiotracer uptake period indicated that all subjects performed normally with respect to immediate memory, whereas Korsakoff patients demonstrated a marked memory impairment in delayed recognition PET results from the Korsakoff group showed a widespread decline in glucose metabolism in front, parietal, and cingulate regions, suggesting that these functional abnormalities in the cerebral cortex contribute to the memory impairment. Hippocampal gluclose metabolism did not differ between the groups.

Thus, the evidence did not support the hypothesis that parallel brain dysfunctions are responsible for the similar amnesic symptomatology after hippocampal and diencephalic damage. We hypothesize that the amnesic dysfunction of Korsakoff's syndrome depends on a disruption of thalamocortical interactions that mediate a function critical for normal memory storage.

Neuroscience WWW Links Page

http://www.neuro.fsu.edu/wwwneuro.htm
Neuroscience Links
Neuroscience Indexes
Neuroscience Related Sites
Books related to Neuroscience
Conferences related to Neuroscience
Institutes related to Neuroscience



http://www-mitpress.mit.edu/jrnls-catalog7cognitive.html

Quarterly, Volume 9 forthcoming
January/March/May/July/September/November
100 pp. per issue, 8 1/2x11, illustrated
Founded: 1989 ISSN 0898-929X

Michael S. Gazzaniga • Editor-in-Chief

Ira B. Black, Steven M. Kosslyn, Gordon M. Shepherd • Senior Editors

Journal of Cognitive Neuroscience investigates brain-behavior interaction and promotes lively interchange among the mind sciences. Contributions address both descriptions of function and underlying brain events and also reflect the interdisciplinary nature of the field covering developments in neuroscience, neuropsychology, cognitive psychology, neurobiology, linguistics, computer science, and philosophy.

Published by The MIT Press and the Cognitive Neuroscience Institute.

This WebSite also has:

Subscription information Abstracting and indexing information Book review information Agents/bookstore terms and policies

It also has abstracts on this site. They can be found at:

http://www-mitpress.mit.edu/jrnls-catalog/cognitive-abstracts/

AS CITED BY.....

Cover page

[1] Caron M.C.: Images in neuroscience: a mouse knockout. *American Journal of Psychiatry,* 1996; 153:1387.

eDelusions: **An Evolutionary Hypothesis... page 21**

[1] McQuire, M., Marks, I., Nesse, R. & Troisi, A.: Evolutionary Biology - A Basic Science For Psychiatry. *Acta Psychiatrics Scandinavica,* 1992;86:89-96

[2] Price, J. S.: Genetic and phylogenetic aspects of mood variation. *International Journal of Mental Health,* 1972;1:124-144,

[3] Gardner, R. J.: Mechanisms in major depressive disorder: an evolutionary model. *Archives of General Psychiatry,* 1982;39:1436-1441

[4] Chance, M. R. A.: *Social Fabrics of the Mind.* London: LEA, 1988.

[5] Gilbert, P.: *Human Nature and Suffering.* London: LEA, 1989.

[6] Wenegrat, B.: *Sociobiology & Mental Disorder: A New View.* Menlo Park: Addison/Wesley, Medical Division, 1984.

[7] Glantz, K. & Pearce, J.: *Exiles From Eden: Psychotherapy From An Evolutionary Perspective.* New York: Norton, 1989.

[8] Badcock, C: *Oedipus in Evolution.* Oxford: Blackwell, 1990.

[9] Thomhill, N. & Thomhill, R.: An Evolutionary Analysis Of Psychological Pain Following Rape. 1. The Effects Of Victims Age And Marital Status. *Ethology And Sociobiology,* 1990;11:155-176

[10] Nesse, R.: What Good Is Feeling Bad - The Evolutionary Benefits Of Psychic Pain. *Sciences,* 1991;31:30-37

[11] Slavin, M. & Kriegman, D.: *The Adaptive Design Of The Human Psyche: Psychoanalysis, Evolutionary Biology, And The Therapeutic Process.* New York: Guilford Press, 1992.

[12] Wilson, D. A.: Evolutionary Epidemiology - Darwinian Theory In The Service Of Medicine And Psychiatry. *Acta Biotheoretica,* 1993;41:205-219

[13] Westermeyer, J.: Some cross-cultural aspects of delusions. In T. F. Oltmanns & B. A. Maher (Eds.), *Delusional beliefs,* New York, John Wiley & Sons, 1988.

[14] Wallace, A. F. C: The Biocultural Theory of Schizphrenia. *International Record of Medicine,* 1960; 173:700-714

[15] Henderson, S.: Care-eliciting behavior in man. *Journal of Nervous Mental Disorders,* 1974;159:172-181

[16] Sullivan, H. S.: *The interpersonal theory of psychiatry.* New York: Norton, 1953.

[17] Tooby, J. & Cosmides, L.: The past explains the present: Emotional adaptations and the structure of ancestral environments. *Ethology & Sociobiology,* 1990; 11:375-424 [18] Szasz, T. S.: *The Myth of Mental Illness.* New York: Dell Publishing Co., 1961.

[19] Markl, H.: Manipulation, modulation, information, cognition: some of the riddles of communication. In B. Holldobler & M. Lindauer (Eds.), *Experimental Behavioral Ecology and Sociobiology,* New York, Gustav Fischer Verlag, 1985.

[20] Holldobler, B.: Communication in social hymenoptera. In A. Sebeok (Eds.), *How animals communicate,* Bloomington, Indiana University Press, 1977.

[21] Starrett, A.: Adaptive resemblance: a unifying concept for mimicry and crypsis. *Biological Journal of the Linnean Society,* 1993;48:299-317

[22] Barsky, A. J. & Klerman, G. L: Overview: Hypochondriasis, Bodily Complaints, and Somatic Styles. *The American Journal of Psychiatry,* 1983; 140:273-283

[23] Alexander, R. D.: *Darwinism and Human Affairs.* Seattle and London: University of Washington Press, 1979.

[24] [24] Nesse, R.: The evolutionary functions of repression and the ego defenses. *Journal of the American Academy of Psychoanalysis,* 1990;18:260-285

[25] Trivers, R. L.: *Social Evolution.* Boston: Addison-Wesley, 1985.

[26] Slavin, M. O.: The origins of psychic conflict and the adaptive function of repression: An evolutionary biological view. *Psychoanalysis and Contemporary Thought,* 1985;8:407-440

[27] Kraepelin, E.: *Praecox and Paraphrenia.* New York: Robert E Krieger Publishing Co Inc, 1971.

[28] Kendler, K. S.: Demography of paranoid psychosis (delusional disorder): A review and comparison with schizophrenia and affective illness. *Archives of General Psychiatry,* 1982;39:890-902

29 Cameron, N.: The Development of Paranoic Thinking. *Psychological Review,* 1943;50:219-234

30 Lemert, E. M.: Paranoia and the Dynamics of Exclusion. *Sociometry,* 1962;25:2-20

31 Retterstol, N.: *Paranoid and Paranoiac Psychoses.* Springfield, Illinois: Charles C. Thomas, 1966.

32 Kaffman, M.: Inflexible belief-constructs in families of paranoid patients. *International Journal of Family Psychiatry,* 1982;3:487-500

33 Kay, D. W., Cooper, A. R, Garside, R. F. & Roth, M.: The differentiation of paranoid from affective psychoses by patients' premorbid characteristics. *British Journal of Psychiatry,* 1976:129:207-215

34 Odegaard, O.: Immigration and insanity. *Acta psychiatrica neurologica Scandinavia,* 1932;supplement 4:

35 Ettinger, L: The incidence of mental disease among refugees in Norway. *Journal of Mental Science,* 1959; 105:326-338

36 Ettinger, L: The symptomology of mental disease among refugees in Norway. *Journal of Mental Science,* 1960:106:947-966

37 Carpenter, L. & Brockington, I. F.: A study of mental illness in Asians, West Indians and Africans living in Manchester. *British Journal of Psychiatry,* 1980:137:201-205

38 Westermeyer, J.: Paranoid symptoms and disorders among 100 Hmong refugees: A longitudinal study. *Acta Psychiatrica Scandinavica,* 1989:80:47-59

39 Chiu, L. P. & Rimon, R.: Relationship of migration to paranoid and somatoform symptoms in Chinese patients. *Psychopathology,* 1987;20:203-212

40 Meissner, W. W.: The diagnosis of paranoid disorders. In F. Flach (Eds.), *Diagnostics and psychopathology. Directions in psychiatry monograph series, No. 1.,* New York, W. W. Norton & Co, Inc, 1987.

41 Oltmanns, T. F.: Approaches to the definition and study of delusions. In T. F. Oltmanns & B. A. Maher (Eds.), *Delusional beliefs. Wiley series on personality processes.,* John Wiley & Sons, New York, NY, US, 1988.

42 [42] Kendler, K. S.: Are there delusions specific for paranoid disorders vs schizophrenia? *Schizophrenia Bulletin,* 1980;6:1-3

43 Retterstol, N.: Course and outcome in paranoid disorders. *Psychopathology,* 1991:24:277-286

44 Wessely, S., Buchanan, A., Reed, A., Cutting, J. & others, a.: Acting on delusions: I. Prevalence. *British Journal of Psychiatry,* 1993:163:69-76

45 Kennedy, H. G., Kemp, L. I. & Dyer, D. E.: Fear and anger in delusional (paranoid) disorder: The association with violence. *British Journal of Psychiatry,* 1992:160:488-492

46 LeVine, R. A. & Campbell, D. T.: *Ethnocentrism: Theories of Conflict, Ethnic Attitudes, and Group Behavior.* New York: John Wiley & Sons, Inc., 1972.

47 Sumner, W. G.: *Folkways.* New York: Ginn, 1906.

48 Sherif, M., Harvey, O. J., White, B. J., Hood, W. R. & Sherif, C. W.: *Intergroup Conflict and Cooperation: The Robbers' Cave Experiment.* Norman, Oklahoma: University of Oklahoma Press, 1961.

49 Coser, L. A.: The Functions of Social Conflict. In L. A. Coser & B. Rosenberg (Eds.), *Sociological theory: a book of readings,* New York, Macmillan, 1957.

50 -- Simmel, G.: *Conflict and the web of group affiliations.* Glencoe, Illinois: The Free Press, 1955.

51 Lewis, W. H.: Feuding and social change in Morocco. *Journal of Conflict Resolution,* 1961;5:43-54

52 Boulding, K. E.: *Conflict and defense: a general theory.* New York: Harper, 1962.

53 Mack, R. W. & Synder, R. C: The analysis of social conflict—toward an overview and synthesis. *Journal of Conflict Resolution,* 1957:1:212-248

54 [54] Ndetei, D. M. & Vadher, A.: Frequency and clinical significance of delusions across cultures. *Acta Psychiatrica Scandinavica,* 1984:70:73-76

55 APA: *Diagnostic and Statistical Manual of Mental Disorders.* Washington, D.C.: American Psychiatric Association, 1994.

56 Gouldner, A.: The norm of reciprocity: a preliminary statement. *American Sociology Review,* 1960:47:73-80

57 Trivers, R. L: The Evolution of Reciprocal Altruism. *The Quarteriy Review of Biology,* 1971;46:35-57

58 Brown, D. E.: *Human Universals.* New York: McGraw-Hill, Inc., 1991.

59 Hoebel, E. A.: *The Law of Primitive Man: A study in Comparative Legal Dynamics.* Cambridge, Massachusetts: Harvard University Press, 1954.

60 -- Goodall, J.: Social rejection, exclusion, and shunning among the Gombe chimpanzees. *Ethology & Sociobiology,* 1986:7:227-236

61 [61] Kaffman, M.: Paranoid disorders: The core of truth behind the delusional system. *International Journal of Family Therapy,* 1981;3:29-41

62 Nitsche, P. & Wilmanns, K.: *The History of the Prison Psychoses.* New York: The Journal of Nervous and Mental Disease Publishing Company, 1912.

63 Henderson, T. E.: Madrid v. Gomez. *Federal Supplement,* 1995;889:1146-1284

64 Grassian, S.: Psychopathological Effects of Solitary Confinement. *American Journal of Psychiatry,* 1983;140:1450-1454

65 Kraepelin, E.: Der Verfolgungswahn der Schwerhorigen. In (Eds.), *Psychiatrie,* Leipzig, Barth, 1915.

66 Cooper, A. F. & Curry, A. R.: The pathology of deafness in the paranoid and affective psychoses of later life. *Journal of Psychosomatic Research,* 1976;20:97-105

67 Cooper, A. F., Garside, R. F. & Kay, D. W.: A comparison of deaf and non-deaf patients with paranoid and affective psychoses. *British Journal of Psychiatry,* 1976;129:532-538

68 Moore, N. C: Is paranoid illness associated with sensory defects in the elderly? *Journal of Psychosomatic Research,* 1981:25:69-74

69 Watt, J. A.: Hearing and premorbid personality in paranoid states. *American Journal of Psychiatry,* 1985;142:1453-1455

70 Cooper, A. F. & Porter, R.: Visual acuity and ocular pathology in the paranoid and affective psychoses of later life. *Journal of Psychosomatic Research,* 1976:20:107-114

71 Ziskind, E.: Isolation Stress in Medical and Mental Illness. *The Journal of the American Medical Association,* 1958;168:1427-1431

72 Robins, L. N., Locke, B. Z. & Regier, D. A.: An Overview of Psychiatric Disorders in America. In L. N. Robins & D. A. Regier (Eds.), *Psychiatric Disorders in America: The Epidemiologic Catchment Area Study,* New York, Macmillan, 1991.

73 Arthur, B. & Schumann, S.: Family and peer relationships in children with paranoid delusions. *Child Psychiatry & Human Development,* 1970:1:83-101

74 Swartz, M., Landerman, R., George, L. K., Blazer, D. G. & Escobar, J.: Somatization Disorder. In L. N. Robins & D. A. Regier (Eds.), *Psychiatric Disorders in America: The Epidemiologic Catchment Area Study,* New York, The Free Press, 1991.

75 Eastwell, H. D.: Psychological Disorders Among the Australian Aboriginals. In C. T. H. Friedmann & R. A. Faguet (Eds.), *Extraordinary Disorders of Human Behavior,* New York, Plenum Press, 1982.

76 Berndt, R. M. & Berndt, C. H.: The concept of abnormality in an Australian society. In G. B. Wilbur & W. Muensterberger (Eds.), *Psychoanalysis and Culture,* New York, International Universities Press, 1951.

77 [77] Waxier, N. E.: Is Mental Illness Cured in Traditional Societies? A Theoretical Analysis. *Culture, Medicine, and Psychiatry,* 1977:1:233-253

78 El-Islam, M. F.: Symptom onset and involution of delusions. *Social Psychiatry,* 1980;15:157-160

79 Murphy, H. B. M.: Cultural aspects of delusion. *Stadium Generate,* 1967;20:684-692

80 [80] Newhill, C. E.: The role of culture in the development of paranoid symptomatology. *American Journal of Orthopsychiatry,* 1990:60:176-185

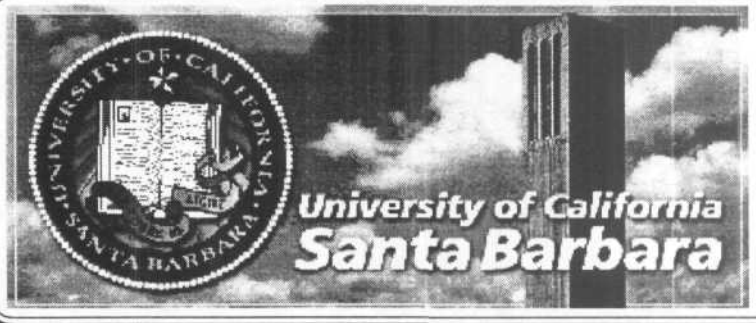
81 Sacks, M. H.: Folie a Deux. *Comprehensive Psychiatry,* 1988;29:270-277

82 Soni, S. D. & Rockley, G. J.: Socio-Clinical Substrates of Folie a Deux. *British Journal of Psychiatry,* 1974:125:230-235

83 Lazarus, A.: Folie a Duex: Psychosis by Association or Genetic Determinism? *Comprehensive Psychiatry,* 1985:26:129-135

84 [84] Jorgensen, P. & Aagaard, J.: A multivariate predictor analysis of course and outcome in delusional psychosis. *Acta Psychiatrica Scandinavica,* 1988;77:543-550

The intuitive framework underlying the hypothesis of this paper was developed during countless conversations with J. Bracker. I thank D. Symons, L. Cosmides, J. Feldman, J. Tooby, P. Walker, and B. Wenegrat for numerous comments and suggestions.



University of California at Santa Barbara

Department of Anthropology Web Site:

http://www.sscf.ucsb.edu:80/anth

The ASCAP Newsletter
Russell Gardner, Jr., M.D., Editor-in-Chief
Frank Carrel, Managing Editor
Department of Psychiatry & Behavioral Sciences
Marvin Graves Building, Room 1.103
University of Texas Medical Branch
Galveston, TX 77555-0428
Tel: (409)772-3475
Fax: (409) 772-4288 or (409) 772-6771
ASCAP E-Mail: ascap®.utmb.edu
rgardner@utmb.edu
f carrel @ psypo.med.utm b.edu

SUBSCRIPTION & MEMBERSHIP FEES

For the twelve 1997 issues (Vol 10, Nos. 110-121), please remit $35.00 with this cover sheet. Checks or money orders are payable to *The University of Texas Medical Branch.* Please mail to Frank Carrel, Managing Editor, at the above-mentioned address.

Reprints of previous volumes are also available:
Indicate below the Volumes that you want. Please send me:

____ Vol. 1,1988 (nos 1-13) $15.00 _____ Vol. 6,1993 (nos 62-73) $15.00
___ Vol. 2,1989 (nos 14-25) $15.00 _____ Vol. 7,1994 (nos 74-85) $15.00
___ Vol. 3,1990 (nos 26-37) $15.00 Vol. 8,1995 (nos 86-97) $15.00
___ Vol. 4,1991 (nos 38-49)$15.00 Vol. 9,1996 (nos 98-109) ___ $25.00
Vol. 5,1992 (nos 50-61)$15.00 Vol. 10,1997 (nos 110-121)

If you prefer to use a credit card, please complete the following:

I authorize The University of Texas Medical Branch to charge to my

Master Card/ Visa/ Discover/ American Express **(circle one)**

The following amount: Card _____

Card _____ Signature: _____

Please fill out the following items for our membership/subscription list.:

Name: _____

Tel: _____ Fax: _____ E-Mail _____

Address: _____

_____

_____