For evolutionary psychiatrists, psychologists and others interested In sociophysiologica! integration

ASCAP

Volume 11, No. 08 (Cumulative #129) August 1998

"What got people out of the trees was something besides thumbs and gadgets. What did it I am convinced, was a warp in the simian brain that made us insatiable for patterns -- patterns of sequence, of behavior, of feeling - connections, reasons, causes: stories."

Kathryn Morton[1]

Contents

Across-Species Comparisons and Psychopathology (ASCAP) Society Executive Council:

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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 of the ASCAP Society.**

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

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

ADDRESSED TO & FROM ...

Report on Annual ASCAP Society Business Meeting July 8, 1998 Hallmark-Ramada Inn Davis, California

by Russell Gardner, Jr., Secretary

The meeting was called to order by President Dan Wilson at 5 p.m. following a full day symposium titled "Toward empirical research in the clinical application of the human evolutionary sciences." The program organized by him and by Russell Gardner was positively experienced and despite a full day of meetings, no-one complained of fatigue.

1. The minutes of the 1997 meeting were distributed as copied from the July, 1997, issue of *The ASCAP Newsletter.* They were approved as written.

2. Appreciations were issued to The Foundation for Cognitive Therapy and Research of Philadelphia for sponsorship of the fourth Aaron T. Beck ASCAP Award. All agreed that the winner this year, Bruce Ellis, made an excellent presentation and was a very good choice for the award. Carolyn Reichelt moved, and Jim Brody seconded, that a letter of appreciation be sent. The motion passed unanimously.

3. Suzanne M. Gardner was issued many thanks for her efforts in arranging the 1998 meeting. Frank Carrel was thanked as well for his efforts as Managing Editor of *The ASCAP Newsletter.*

4. Issues of future institutional home for The ASCAP Society and *The ASCAP Newsletter.* Russell Gardner presented that he is in the process of applying to the state of Wisconsin to establish Neuropsychiatry and Social Brain Institute (NASBI), a nonprofit organization, that would be formed to sponsor the newsletter of the organization.

   This will be based in Wisconsin as the Gardners plan to move from Galveston, TX, to Madison, WI, after RG retires from UTMB in March, 1999. More details on this will be issued as plans and events move forward. John Pearce moved, and Jim Brody seconded, that NASBI formally become the entity to hold the assets of The ASCAP Society after Dr. Gardner's formal disconnection from UTMB in Galveston, Texas. The motion passed unanimously.

5. European Editor report by John Price. Dr. Price acknowledged the recruitment to The ASCAP Society of Michael Davies, his twin brother Henry, and wife, Catherine. They have produced a book on the evolution of modem humans as a result of ice ages and have contributed to the newsletter.

   Dr. Price also reported on the World Psychiatric Association (WPA) Meeting to occur from August 6 through 12,1999 in Hamburg, Germany. He chairs the Psychotherapy sectior which is heavily populated by evolutionary oriented people now and urged that as many as possible from The ASCAP Society attend. Dr. Price supposed that if there were enough there, the current co-chairman, Russell Gardner, might be elected for the next three year term. He disclosed that both of the symposia proposed will be put forth, despite a suggestion by the WPA organizers that they be consolidated. He protested this and they yielded.

6. Dr. Price also commented on the idea of an ASCAP annual meeting to occur in Hamburg, Germany, in

1999, prior to the World Psychiatric Meeting. There was some debate about another location (e.g.,? Derby, England, in view of this being where Paul Gilbert lives), but overall the group agreed that the procedure to maximize attendance would be to have the meeting in Hamburg just before the other meeting.

Mark Erickson agreed to preside there and Ivor Jones had previously agreed to accept the presidency at that time and place. Possibly Paul Gilbert could be enlisted to help with the program. We are eager to provide a venue for our European members to present papers. Mark Erickson moved and Russell Gardner seconded the motion that the meeting be held in Hamburg just before the WPA. The motion passed unanimously.

8. Will there be a U.S.A. located ASCAP meeting next year as well? Dr. Gerald A. Cory, whose piece on Paul MacLean occupies the newsletter's July issue, spoke of on his interest in sponsoring a Paul MacLean celebration or festschrift. He and Russell Gardner have discussed this in a preliminary manner.

Dr. Gardner will determine additional information available from HBES attendees that will help in the planning. Dr. MacLean is 85 years old, travels little, and this may constrain the venue. Dr. Cory moved and John Price seconded that this effort be sponsored in part by The ASCAP Society (but that the official annual meeting of the year — involving the installation of officers —take place in Hamburg, Germany). The motion passed unanimously.

9. Slate for new officers:

Representing the nomination committee, Russell Gardner presented the following slate and the two new additions (Lynn O'Connor and Linda Mealey) agreed to accept the nominations.

New President:
Mark T. Erickson (Alaska)

New President-elect:
Ivor Jones (Tasmania)

New 1st Vice President:
Thomas Joiner (Florida)

New 2nd Vice President:
Lynn O'Connor (California)

New Chair of the Beck ASCAP Award Committee:
Linda Mealey (Australia/Minnesota).

The motion was moved and seconded that the slate be approved as presented. The motion passed unanimously.

10. Dr. Wilson passed the gavel to Dr. Erickson who then adjourned the meeting as there was no additional business.

An E-Mail Thread: Sex Differences in Emotional Expression

Parti: What is hardwired?

by Ray Buck

Mike Waller wrote, "Don't we keep getting driven back to the thesis that females come hardwired with social skills, whilst men have to learn theirs?"

I think that the results of Harlow's research with rhesus isolated for the first year suggest that all complex social animals must learn social skills, but the things that must be learned may differ according to sex (and temperament).[1] It is interesting that Harlow's isolated females were "bad mothers" with their first infants, but were fine with the second.

Regarding the sex difference in decoding nonverbal behavior: the dominance hypothesis was proposed by Nancy Henley,[2] but has been challenged in research by Judith Hall and Amy Halberstadt.[3]

My own view is that decoding abilities are pretty much built in to everyone, given that one attends to the relevant cues.[4] What is important is:

1. Whether one is "educated to attend" to the relevant cues (see J.J.Gibson's view of perception), and

2. Whether one is expressive oneself. The latter, I think, accounts for the gender differences in decoding, empathy, intuition, or whatever. Women tend to be more expressive than men, and therefore carry around with them a "bubble of expressiveness." Expres sive people encourage others to be expressive, and therefore have those expres sive cues to draw on. (Next time you are at a supermar ket, note how much a checker's expressiveness varies with the expressive ness of the customer). In general, nonverbal communi cation is very much a dyadic phenomenon.

<u>Part 2</u>: Infancy data.

by David C. Geary

Research on sex differences in infancy are also inconsistent with the power hypothesis, as differences in social orientation are evident very early in life. As an example, in a very useful review of this literature Haviland and Malatesta noted that:

"There is no doubt that girls and women establish and maintain eye contact more than boys and men. The earliest age for which this is reported is onec/ay."[5 (page 189)]

There are a number of other early sex differences that support the hypothesis that girls and women are more sensitive to social cues than are boys and men. Other differences include more gaze averting in boys (evident by 6 months of age) and more orientation to faces and voices by girls (evident within the first few days after birth).

By 12 months of age there are sex differences in responses to distress to other people, with girls showing more empathy and boys more indifference, although these are not large differences. In contrast to girls' greater orientation to other people, boys show greater orientation to cues in the physical environment (which likely facilitates the development of certain spatial competencies).

Again, this sex difference is evident in the first few months of life.

<u>For more on this see</u>:

McGuinness & Pribram: The origins of sensory bias in the development of gender differences in perception and cognition. In: M. Bortner (editor), *Cognitive Growth and Development.* Brunner/Mazel, 1979, pages 3-56

Zahn-Waxleretal.: Development of concern for others. *Developmental Psychology,* 1992;28:126-136.

Zahn-Waxleretal.: The development of empathy in twins. *Developmental Psychology,* 1992;28:1038-1047.

Want a list of WWW links and References on Sex Differences? Then go to this Home Page:

References on Sex Differences

http://www.home.aone.net.au/think/sdifref.html

The page is divided into the following sections:

World Wide Web (WWW) links

Places to find interesting articles

Recent book recommendations

General references

Other references

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 Tyge** Schelde*

A LETTER TO DANIEL WILSON & A TENTATIVE HYPOTHESIS OF SCHIZOPHRENIA AND MANIC-DEPRESSIVE ILLNESS

Dear Dr. Daniel Wilson.

I just read your reply to Dr. Don Klein in the May 1998 issue of *The ASCAP Newsletter.*

As you mentioned, that you are going to reply to my article in the February 1998 of *The ASCAP Newsletter,* I think I might state my hypothesis as clearly as possible, because after further considerations — I think that I am able to formulate the hypothesis in a simpler way.

Concerning schizophrenia and manic-depressive illness, /am not going to put the question WHY (survival value), but the questions WHAT and HOW (cf. Tinbergen's four whys).

What refers to the etiological cause, i.e., genetics.

How refers to the physiological dynamics, including environmental influences.

The following emphasizes a tentative explanation of manic-depressive illness.

Sincerely,

Tyge Schelde M.Sc,
Research Ethologist
arcirip@cybernet.dk
http://axp.psl.ku.dk/-dhf

Etiology:

Schizophrenia is caused by a deletion of the glutamate receptor-4 site on the long arm of one of the homologous chromosomes no. 11 (11q22-q23).

Manic-depressive illness is caused by a deletion of the dopamine D2 receptor site on the long arm of one of the homologous chromosomes no. 11 (11q22-23).

Physiological dynamics (see Figure 1):

In *schizophrenia* —the cortical glutamatergic and probably also the cholinergic activities are reduced because of the deficit of the glutamate receptor-4. Therefore, the putamen in the basal ganglia is only weakly stimulated. The general consequence is a disinhibition of the dopaminergic activity in the thalamus. This leads to an abnormally high stimulation of the glutamatergic pathways leading to the motor cortex. The consequence —among others —of this hyper-stimulation is stereotyped behavior.

In *manic-depressive illness* there is a 40% deficit of gray matter in the subgenual area of the pref rontal cortex.[1] This deficit might be due to missing D2 receptors and a degeneration of cells in this area. The D2 receptors are supposed —in normal conditions —to be situated on certain glutamate or acetylcholine nerve cells, and their function is supposed to inhibit or modulate glutamatergic/ cholinergic arousal levels.[2]

Because of the dopamine D2 receptor deficit, the glutamatergic/cholinergic resting potential will be constantly a little above the normal resting potential, i.e., -60 versus -65.[3] Hence, an increased excitability and sensitivity. This easily invites *kindling* of both systems by environmental action specific stimuli/social releasers.

By a slightly increased nervous arousal, probably caused by rewarding stimuli, the general behavior will be equivalent to hypomania. By an augmentation, the person in question will suffer from mania. By **a** considerable increase of the arousal level, the manic state will switch over to an agitated state and finally to a deep depression or a psychomotor

inhibition.[4, 5]

The abnormally high arousal level corresponding to the psychomotor inhibition may also be triggered by depression specific stimuli, even very small and trivial ones.

The physiological dynamics of the very high glutamatergic and cholinergic arousal levels are considered to influence the well described *Papez loop or motor loop.* Glutamate and acetylcholine pathways from the cortex stimulate GABA in putamen. GABA inhibits other GABA ganglia in globus pallidus exterior. The last mentioned GABA system disinhibits glutamate in the sub-thalamic nucleus. From here an increased glutamatergic activity goes back to globus pallidus interior where it stimulates another GABA pathway considerably.

This GABA system exerts an abnormally high inhibition of the glutamate ganglia that propagate excitatory stimuli to the motor cortex. Hence the *motor inhibition*—or an almost complete lack of muscle energy.

The subjectively felt *depression (thoughts, motions, anxiety)* may be understood as nervous energy (ruminations) propagated in a nervous circuit from cortex to thalamus to the limbic system (amygdala?) and back to cortex.

Summary:

According to this hypothesis — *Schizophrenia* is characteristic of a decreased glutamatergic activity and a corresponding high dopaminergic activity.

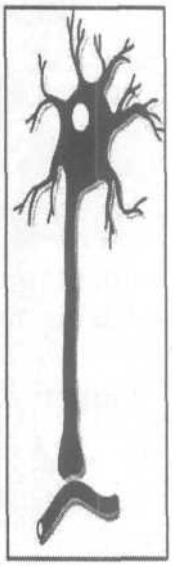
Major Depression—is characterized by an extremely high cholinergic/glutamatergic? activity with a compromised dopaminergic modulation of the cholinergic system. Social releasers of major depression may be even trivial dejecting/frustrating stimuli.

Hypomania/Mania — is a slightly to medium to rather strongly increased glutamatergic?/cholinergic activity. Social releasers of mania may be even feeble rewarding stimuli.

In *Schizoaffective Disorder*—probably both a glutamate receptor-4 site and a dopamine D2 site have been deleted.

Parkinson's Disease is well described and is caused by a low dopaminergic activity and a relatively increased cholinergic activity. Thus, the physiological interactions seem to be similar in Parkinson's Disease and major depression. However, in Parkinson's Disease there is a decay of the whole dopaminergic system, whereas in major depression only the D2 receptors are supposed to be missing. Clinically, the two diseases are related as for depression and partly for motor shaking/ motor inhibition.

The hypothesis predicts that—major depression should be treated by anti-glutamatergic/anti-cholinergic medicine. A psychomotor inhibition induced by a depletion of dopamine or by an inhibition of dopamine receptors can be relieved by reducing the glutamatergic corticostriatal tone.[6a, 6b] (Carlssonetal., 1991). Treatment with cholinergic agonists, e.g. physostigmine, induces both a depressed mood and anergic-inhibitory effects (psychomotor inhibition), whereas the anti-cholin-ergic agent scopolamine relieves the depression.[2]



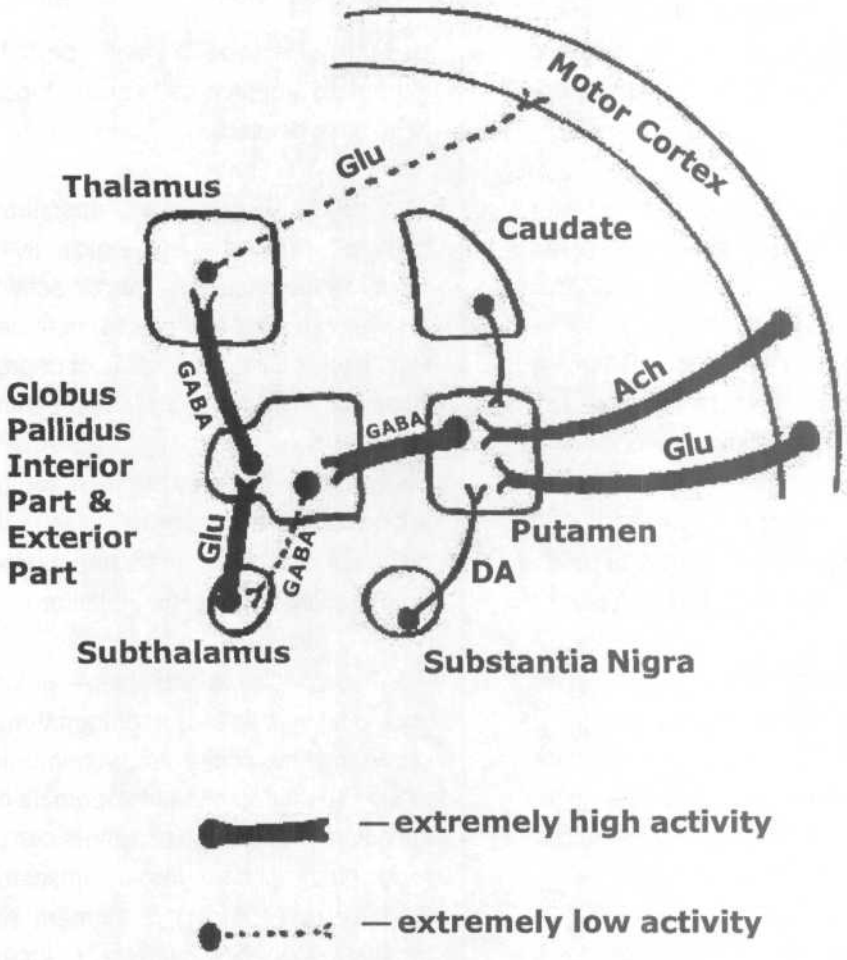
GABA Research WebSite:

Neuroendocrinology Laboratory

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

Dr. David R. Grattan
University of Otago Dunedin, New Zealand Department of Anatomy and
Structural Biology and
Neuroscience Research Centre
david.grattan @ stonebow.otago.acn!

Figure 1 - Major Loop in Major Depression



Tyge Schelde, Chairman
(an ASCAP Society member)

Axel Randrup is the editor of this WebSite. (he also an ASCAP Society member)

Danish Society for Human Ethology

Dansk Human-Etologisk Forening

http://axp.psl.ku.dk/~dhf

Letters to the editor? Send to: arcirip@cybernet.dk

Reference to International Society for Human Ethology with their search page, Ethology and Evolution on the Web at: http://evolution.humb.univie.ac.at

Contents of this WebSite:

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- **Danish Research Letters to the Editor**
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ARTICLE:

by Vitaliy I. Egorov

Schizophrenia and Evolutionary Approach

In this paper, clinical, genetic and ethological data are used to analyze the evolutionary concept of schizophrenia. This review of current literature and clinical experience supported by data from genetics and evolutionary biology stimulate the hypothesis that some behavioral and cognitive impairments observed in schizophrenia may be evolutionarily progressive and also advantageous for the human population more generally.

introduction:

Since the American entomologist Edward O. Wilson introduced his fundamental work, *Sociobi-ology: the New Synthesis,* growing interest arose for applying Darwinian ideas in numerous disciplines.[1] Psychiatry was among the first to make such applications, and contributors have included: Crow,[2] Gardner,[3] McGuire,[4] Marks, Nesse,[5] Troisi,[6] Williams, Samohvalov,[7] Egorov, Slavin and Kriegman,[8] Stevens and Price[9] and Wenegrat[10].

Of course, psychiatrists in the past were interested by Darwinian ideas. We have found some notes on evolutionary theory in the works of such clinicians as E. Kraepelin, E. Bleuler, A. Meyer, H. Jackson, A. Storch, and others. But their notes were more fragmentary than those developed more recently.

It's not easy to explain the growing interest of psychiatry to applying methods and ideas of evolutionary biology and sociobiology to psychia-try. Perhaps, the main reason was that since the time after E. Kraepelin and E. Bleuler first system-atized the concept of schizophrenia, psychiatry was still unable to make any essential progress towards prevention and especially treatment. From these positions schizophrenia was and is one of the most severe mental illnesses whose nature remains a secret.

Despite our obvious advances in psychopharmacol-ogy, molecular biology, biochemistry we are far from understanding the neural mechanisms of schizophrenia. Moreover, the rate of disease remains uniquely stable - approximately 1 per cent and does not differ essentially amongst various cultures. Even such pivotal factors of selection as reproductive success and rate of mortality follow normal human characteristics.

The evolutionary concept of human psychopathol-ogy was recently elegantly outlined in 1995 by TJ Crow.[2] He queries why have the manifest disadvantages associated with the psychosis genes have not caused their elimination from the human population?

Clinical and genetic aspects of schizophrenia:

At the beginning of this century two great psychia-trists, from Germany, Emil Kraepelin, and from Switzerland, Eugen Bleuler, described the common concept of schizophrenia, focusing on four major subtypes of this disease, namely, paranoid, hebe-phrenic, catatonic, and simple. The new classifications of *ICD-10*and *DSM* modified these classifications so that ICD-10 includes nine subtypes of schizophrenia; *DSM-III-R* had 6 subtypes of the illness.

By contrast, the English psychiatrist, T.J. Crow in 1980, suggested two types of schizophrenia which he labeled Type I and Type II respectively.[11] Type I schizophrenia characterizes by prominent positive syndromes (bizarre behaviors, delusions, hallucina-tions, positive formal thought disorders), an acute onset, good premorbid adjustment, a good re-sponse to treatment, intact cognition, intact brain structure and an underlying mechanism that was neurochemical (dopaminergic) and therefore, reversible. In contrast, Type II schizophrenia was characterized by prominent negative symptoms

(alogia, affective flattening, avolition-apathy, anhedonia-asociality, attentional impairment), insidious onset, poor premorbid adjustment, a poor response to treatment, impaired cognition, etc. Other researchers have added third type of schizophrenia, a mixed syndrome that can includes symptoms of both Type I and Type II schizophrenia.[12]

Independently of what hypothesis or theory of schizophrenia we note that we must agree that schizophrenia is a disease with genetic predisposition. The study of heredity, which began less than a century ago, evolved in three major directions:

1. **Biochemical genetics.** Dealing with physiochemical reactions associated with DNA replication and protein synthesis;

2. **Cytogenetics.** Dealing with the chromosomes that carry DNA; **and**

3. **Population genetics.** Dealing with mathematical properties of transmission within families and the genetic compositions of populations.

There are numerous models suggested for schizophrenia but I am briefly discussing the following:

1. **Multifactorial model:** In the multifactorial model, liability is assumed to have a normal (i.e., bell-shaped) distribution in the general population and affection is assumed to correspond to threshold values. The similarity between relatives is given by their correlation in liability. The estimate of the correlation between underlying liability distributors is called the tetrachoric correlation coefficient. The liability distribution and thresholds for males and females may differ and result in different prevalence in the sexes. Sex differences have not been found to be important in schizophrenia.

2. **The generalized single major locus model:** The generalized single major locus (SML) model assumes all relevant genetic variations is due to the presence of two alleles at a single locus.[13] In this case locus refers to the positions of a gene on a chromosome and allele is one of the alternative genes at that locus. With two alleles, A and a, there are three possible genotypes: AA, Aa, aa. The penetrance of a genotype (the proportion of individuals of a given genotype who are affected) is denoted f 1, f2 and f3. The SML model can be applied to large pedigrees, and, moreover, information on chromosomal markers may also be used in analysis to establish linkage of a marker to that major locus.[14]

3. **Single gene models:** Much of the early theorizing about the mode of inheritance of schizophrenia resulted in oversimple models. Heston argued that nonpsychotic relatives of schizophrenics who have personality disorder, neuroses, or who are even in some cases conspicuously successful and creative, might all be carriers of the same dominant gene for "schizoid disease".[15] The most commonly applied type of a single major locus (SML) model assumes that a single gene is the sole cause of diagnostic similarities between relatives. Despite interesting and stimulating ideas connected with single gene models, greatest interest has involved polygenic models.

4. **Polygenic/multifactorial models:** A polygenic multifactorial model is now considered by the majority of psychiatric geneticists at the most plausible model in schizophrenia. The theory was first suggested by Gottesman and Shields in 1969." Without detailing the model, we conclude in summary that a multifactorial model of schizophrenia fits most of the observed facts about the distribution of disorder. Thus, concordance in twins in the first-degree relatives increases with the number of relatives already affected. Furthermore, the disorder persists in the population despite the selective disadvantages conferred by the reduced chances of marrying and producing offspring.

Evolutionary aspects of schizophrenia:

After official scientific coronation of ethology in 1973, when the 3 representatives of this biological discipline — K. von Frisch, K. Lorenz and N.Tinbergen—were awarded the Nobel Prize, the theoretical background and methods have been developed by psychiatrists and clinical psychologists for application clinically.[17] (in Russia), 18, 19 (in Russian)

Ethology can be defined as a discipline which is focused on observation and description of the behavior of living organisms in natural conditions.[20] As to psychiatry, clinicians also pay special attention to the patient's behavior in their diagnostic conclusions. The rapid progress in human ethology was a helpful tool for evaluation from both qualitative and quantitative points of view, of the patient's individual and social behaviors in psychiatric wards.

Particular interest is focusing on a new branch of ethological psychiatry - "ethopharmacology".[21] It may be defined as the application of ethological methods and concepts to the analysis of drug-induced changes in behavior.[22]

Ethopharmacology is a relatively new approach to the study of the behavioral actions of drugs. In contrast to psychopharmacology, ethopharmacology places the behavioral analysis in the forefront and seeks an understanding of a drug's action on behavior in ethological rather than in molecular terms. Unfortunately, many interesting results are obtained during experiments with laboratory animals (mainly rodents) whereas analogous approaches to psychiatric population have not developed yet. [23] (in Russian)

There are many evolutionary hypotheses dealing with schizophrenia. Simplifying them we can mention two general theoretical considerations. The first looks on schizophrenia as rudimentary behavioral patterns which had have some adaptive advantages in our ancestors. Therefore, schizophrenic behavior could be viewed as "the voice" of our evolutionary past. The clear example in support for this point of view is catatonic behavior.

Catatonia displays in the form of two common behaviors - stupor (immobility) and excitement. These patterns of behavior had adaptive advantages in our ancestors in "prey-predator" interactions. Because predators focused more attention on the moving victims, freezing (immobility) could increase chances and therefore, adaptedness for those who were was immobile. Similarly, excitement increases chances to flee from preda-
[24] (In Russian)
tors.

A second group of scholars try to find in schizophrenia markers of adaptation and even selective advantages in some cases. [2, 5, 17] (in Russian), [25] (in Russian) So, V.P. Samohvalov suggests three main evolutionary strategies related to evolution of schizophrenia.[19] (in Russian)

1. "Regressive" strategy. This means that some behavioral patterns observed in schizo phrenic patients (e.g. above mentioned catatonic stupor and excitement) have no behavioral advantages at the present.

2. "Stabilizing" **strategy.** It means that some behavioral patterns are fixed in human popula tion which were earlier probably specific for schizoids or schizophrenics.

3. **"Progressive" strategy.** It means that some behavioral and cognitive patterns which now seem abnormal (e.g., some delusions) would be further saved in humans as adaptive and helpful.

   Thus, according to this hypothesis schizophrenia can sometimes serve as a source of new behavioral and cognitive displays which will be further accepted or rejected by selection. 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. [26, 27] *c8*

ARTICLE:

by Igor V. Ganzin

Using Linguistic Data for Additional Diagnostic Assessment in Psychiatry

Diagnosis in psychiatry may rely overmuch on verbal report. The Crimean psychiatrists, long known for their focus on ethological science, have worked to develop more objective measures. One approach studies nonverbal and verbal components of speech in different forms of mental pathology. The need for this was developed by Samohvalov in his book, *Evolutionary Psychiatry.*[1]

According to this idea, a clinical-linguistic diagnostic method gathers traditional clinical psychopatho-logical and clinical-phenomenological approaches and compares these indices of diagnosis to those provided by linguistic analysis of complex speech and language expression.[2a, 2b, 2c]

The clinical linguistic diagnostic subdivides into five components: psychosemantic, paralinguistic, psycholinguistic, pragmatic and syntactical. When the results of these approaches were compared with clinical characteristics of patients, the following resulted:

Psychosemantic studies the specificity of main psychosemantic spaces. So, we examine the patient's inner picture of disease, describe his/her main intrapsychic conflicts, and inquire how each experiences self-consciousness. Special attention was paid to the study of alexythymia phenomena described by Taylor, because its presence requires a special complex of rehabilitation and psychothera-peutic procedures.[3]

Paralinguistic diagnostic is the analysis of the nonverbal components of speech. During our investigation we have found paralinguistic peculiarities of general psychopathological syndromes that further were reflected in our glossary.[4]

Psycholinguistic aspect of speech analysis is focused on the analysis of the syntactical features of the speech origin[5a, 5b] and determination of the novelty and text's formation.[6] It also includes definition of psycholinguistic indexes [Triger's (Schlissmann)] coefficient, coefficient of directiveness.[7a, 7b]

With pragmatic analysis we study features of the structure and types of speech acts. For this case we have elaborated a special matrix. The syntactical diagnostic aspect indicates the study of the relationships between syntactic connections and relations, and the qualities of syntactic constructions.

For the study of speech features at various psycho-pathological states we have studied 350 patients (180 males and 170 females) using the ICD-10 diagnostic criteria. For a control group, we also described 60 healthy individuals (30 males and 30 females).

General results:

1. We found speech characteristics of healthy individuals to vary with sex, age, personality, and the presence of minimal brain dysfunction (micro-organic background), etc. We found we could describe speech characteristics in the structure of neurotic disorders that provides an avenue for the early diagnosis and manage ment of these disorders.

2. We established linguistic characteristics of different psychopathological states that have diagnostic, differential-diagnostic and prognos tic meanings. These indicators correlate with the leading psychopathological syndrome, its nosology, type of course, stage of disease, state of patient's consciousness, etc.

3. During our work we found three main groups of mental disorders to differ significantly in most aspects of linguistic analysis:

(a) Organic brain disease

(b) Endogenous psychoses, and

(c) Neuroses and reactive states.

4. For organic brain disease we suggest that typically such features include complex linguistic stigmata. These are exemplified by speech regression and regression of con sciousness; the stigmata also include histori cally and ontogenetically earlier forms of speech expression, narrowing of psychosemantic spaces, impoverishment, changes in the psycholinguistic construction and pragmatic characteristics.

5. Neurotic states differ by function (reversible character) of the linguistic indicators. Typical for these states were increasing dynamism, wide spectrum of linguistic characteristics, coordination and harmony of changes in different diagnostic aspects. The phenomenon of alexythymia was expressed.

6. For endogenous psychoses we have found specific disorders characterized by the following signs: different structure of psychosemantic space (principally), as well as semantic-syntactical dissociation, deforma tion of pragmatic characteristics, tendency toward narrowing of characteristic's spectrum, decreasing of dynamic and some organic signs during the course of disease

Future development of linguistic studies in psy-chiatry can be helpful in understanding the nature of mental disorders and can create preconditions for new approaches in the study of speech and consciousness. *c8*

Editor's Note:

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ARTICLE:

by John S. Price

Ten Years After: Alternative Channels for Negotiating Asymmetry in Social Relationships

Adapted from:

Price, J.S.: Alternative channels for negotiating asymmetry in social relationships. In: *Social Fabrics of the Mind (M.R.A.Chance,* editor). Hove: Lawrence Erlbaum, 1988; pages 157-195.

Signalling **of Resource Holding Potential (RHP):**

Behavioural ecologists have for many years been concerned with the mathematics of ritual agonistic behaviour (RAB), or pairwise contests, and the selective forces acting on the strategies used in them.[1] They have introduced an intervening variable which they call Resource Holding Potential (RHP) to assist in the mathematical analysis of such contests.[2-3] RHP is a measure of fighting capacity and on the input side it is determined by such factors as age, size, weapons, previous success and availability of allies. On the output side it determines probability of fighting (rather than submitting or withdrawing) in a contest, and also duration and intensity of fighting once a contest has begun. All the attack components of agonistic behaviour, including dominance display, threat display, challenge, attack and chasing, are looked on as signals of RHP.

There is, to my knowledge, no concept currently in use in psychology which expresses the equivalent of RHP; it is related to the idea of self-confidence, but the latter term is poorly defined and in any case refers to confidence in other areas in addition to fighting ability; it is also related to the "dominance feeling" described by Maslow but this term has not been in use since the last war.[4] I think RHP is a helpful term in the conceptualisation of RAB and I hope that in extending it somewhat in its psychological meaning I am not distorting the meaning it already has in behavioural ecology.

The Calculation of Relative RHP:

In a contest, or ritual agonistic encounter, we are concerned with each contestant's evaluation of his own RHP compared with his assessment of his adversary's RHP ~ what Parker has called relative RHP.[3] This is a somewhat complex evaluation, and it might be useful to recognise the following subdivisions of RHP:

1. **Absolute RHP:** Each individual has some general idea of his own fighting capacity in relation to other individuals, regardless of who may be his specific adversary on any one occasion. This value of RHP determines any undirected dominance display which the individual signals to the world at large, and the directed dominance display (challenge or threat) which he makes to a potential adversary at the beginning of an encounter, before he has had time to assess the other's RHP.

2. **Signal of Absolute RHP:** This is the signal given in the dominance displays mentioned in the preceding paragraph. Such a signal can obviously be faked, but I will assume here that it is an accurate reflection of absolute RHP, in order not to complicate further an already sufficiently complex subject.

3. **Estimate of Adversary's RHP:** I will assume that this is an estimate which reflects the information received from the adversary's "signal of absolute RHP", but, if faking is suspected, the estimate could be revised up or down.

4. **Estimate of Relative RHP:** This is derived from a comparison of (1) and (3) above. We do not know how this comparison is made, but in the simplest case it must give a result which is either favourable or unfavourable, in order to

determine the choice between two possible courses of action: escalation and submission. These actions then become signals in the next round of the conflict.

5. Signal **of** Relative **RHP:** A contest is an iterative process which may last for several "bouts", so that, at each stage, each contestant is estimating his adversary's relative RHP, comparing this estimate with his own absolute RHP to calculate his own relative RHP (which may be either favourable or unfavourable), and signalling this relative RHP by either escalating or submitting. For simplicity I will assume a two-stage contest in which a period of mutual assessment is followed either by the submission of one contestant or by a period of engagement in which there is mutual attack.

   During the assessment stage, either contestant may submit by giving a signal of unfavourable relative RHP, and thus leave the encounter in a subordinate role but without loss of RHP -what Sloman and Price[5] called "voluntary yielding"; or the contestant may enter the engagement stage by giving a signal of favourable relative RHP and thus have a chance of winning, but at the risk of losing and being forced into what we called "agonistic yielding, with associated loss of RHP.

The Two Components of the RHP Signal:

Although it may be convenient to amalgamate them for mathematical purposes,[3] from the psychological point of view "signal of absolute RHP" is very different from "signal of relative RHP". The two differ in the following ways:

1. Stage **of Assessment vs. Stage of Engagement:** Signal of absolute RHP is saying "This is what I am like; examine me and assess my power", and it occurs in the assessment phase of the agonistic encounter, when the adversaries are confronting each other or circling round each other in mutual appraisal. Signal of relative RHP is saying "I am better than you and I will prove it", and it occurs in the engago-ment phase in the encounter, when the adversaries are engaged in some very ritualised and species-specific activity such as repeatedly charging at each other head on.

2. **Semantic vs. Shannon Information:** In signalling absolute RHP, the adversary is presenting himself for examination in all his aspects, having little control over what aspects are attended to; therefore the information offered to the adversary is very extensive, even infinite, and is what Krebs and Dawkins[6] have called Shannon information, and what Lockard[7] has called a composite signal.

   In signalling relative RHP, on the other hand, he is offering only one "bit" of information (namely, whether he is escalating or not) and the nature of the signal insists that the adversary pay attention it and to no other. If the signal varies, it varies in quantity rather than in quality. This is what Krebs and Dawkins call semantic information, and what Lockard calls a graded signal.

   The difference between the signals of absolute and relative RHP is probably due to the fact that in the process of sender/receiver co-evolution the exchange of signals of absolute RHP has been a co-operative matter, in that, if there is a real disparity between the RHP of the contestants, it is in both their interests that the one with lower RHP should rapidly and efficiently identify the disparity and submit. In contrast, the exchange of signals of relative RHP has been a competitive matter during sender/receiver co-evolution, because it occurs only if there is no great difference in absolute RHP and each contestant has a fair chance **of** winning; each is interested not only that the winner should be decided quickly but also that they should be that winner.

3. **Species Similarity vs. Species Specificity:** Signals of absolute RHP tend to be common across species, such as upright posture,

confident gait, display of weapons and large size. Exceptions such as the blue colouring of the rainbow lizard (Harris, 1964) are rare. Signals of relative RHP, on the other hand, tend to be highly species-specific in that each species has its own form of "combat"; some, like the head charging of the bison involve bodily contact whereas others such as the gill erection of the Siamese fighting fish do not, and consist entirely of an exchange of signals at a distance; within these categories the signals are similar in general form but highly specific in detail.

4. **Different effect on allies:** The signalling of absolute and relative RHP can be further differentiated if we postulate the presence of an ally. Display of absolute RHP is received by allies, and it boosts rather than lowers their RHP. However, allies do not signal relative RHP to each other, except in mock fights for practice.

Catathetic Signals:

Because "signal of favourable relative RHP" is a cumbersome phrase, and because there is no exact ethological equivalent, I propose the term catathetic to describe the signals that are exchanged during the engagement phase of the agonistic encounter(and at other times to reinforce dominance). Catathetic comes from the Greek words for "put" and "down", expressing the function of catathetic signals which is to put the other individual down, in the sense of making him yield and/or lowering his RHP. (I am aware that "cathairetic" would be more correct from the etymological point of view, but "catathetic" is easier to use).

Considering the species-specificity and the low information content of catathetic signals (illustrated by the analogy of the remote control TV button) it is likely that some very specific neural structures have co-evolved for the sending and receiving of these signals. Ethologists I have consulted are not happy to accept that catathetic signals are sign stimuli acting on an innate releasing mechanism to release the fixed action pattern of the yielding subroutine; but something similar to this classical ethological process seems likely.

Catathetic Signals in Man:

Human beings are unique in the animal kingdom in being able to verbalise the signal of favourable relative RHP (catathetic signals). The message of the signal is "I am better than you", and whereas other species need to indulge in various pushing and pulling contests in order to get the message across, human beings can simply say it. Of course, if both say it, they are in a contest, and they have to keep on saying it until one gives up or escalates to the next stage which is physical attack.

Therefore, human ritual agonistic encounters take the form of slanging matches in which each contestant continues verbally to assert his superiority over the other, with varying degrees of imagination and sophistication. This verbal interchange is the human species-specific form of catathetic signalling. Since it appears to be generally true that the structures responsible for catathetic signals (such as the stag's antlers) tend to become hypertrophied due to sexual selection, the same argument must be one reason for the development of the richness of human language.

Human catathetic signals may consist of a simple comparative statement (e.g., I am cleverer than you), or, rarely, a statement of the speaker's RHP (boasting) but more usually it is a statement emphasising or implying the other's low RHP, such as criticism, sarcasm, insult, disparagement (of the other and his/her allies) or even silence, implying, "*You are not worth speaking to**. Escalated catathetic signals involve physical contact such as hitting, scratching, biting, caning, flogging, etc.

Raush et al.,[8] were able to reproduce this phenomenon in married couples put in a situation of artificial conflict. When told to choose between a baseball match on TV and a programme on naming

a baby, some couples discussed the matter rationally and came to a decision; some avoided conflict altogether, but a third group generalised the conflict into what was clearly a ritual agonistic encounter. In the latter group the verbal content typically included criticism of the spouse's mother, and complaints about ill-deeds committed many years ago; the content had a stereotyped quality and was reproduced on subsequent occasions.

McLean[9] has used the term "microstressors" for repeated slight stresses such as the receipt of catathetic signals from one's spouse. He thinks these may be more important in causing depression than large events. The sender was often unaware of the catathetic nature of the signals he was sending; for instance, the comment: " *You would feel much better if you didn't cry all the time",* was intended as helpful and supportive but was received as criticism.

The concept of catathetic signals gives us an opportunity to tackle the definition of the term "mental pain" which is often used loosely in connection with depression. We can say that, whereas physical pain is felt on receipt of "contact" catathetic signals (such as hitting), mental pain is felt on receipt of non-contact catathetic signals (such as criticism). Of course, the situation is not so simple, and mental pain may be experienced in other circumstances, such as the receipt of bad news (e.g. loss of an ally); but it may be that such pain-inducing circumstances share with catathetic signals the property of lowering RHP.

Anathetic Signals:

One advantage of the concept of catathetic signalling (whose function is to lower RHP in the recipient) is the facilitation of the opposite concept of anathetic signalling whose function is to raise RHP in the recipient. I think it is true to say that much of social life which is not devoted to justifying our position on various matters,[10] and thus, incidentally, reinforcing our own RHP, is actually devoted to manipulating other people's RHP, either lowering it with catathetic signals or raising it with anathetic signals. As far as the precipitation of depression goes, it may be that reduction in anathetic signals has the same effect as increase in catathetic signals, and this raises the possibility of a bridge between the social competition model and the loss model of depression; if we allow that anathetic signals can operate in the hedonic mode, they would include the "narcissistic supplies" of classical psychoanalysis,[11] whose withdrawal is thought to lead to depression, and also the sociological concept of processual status, whose cessation Kemper[12] has postulated as a cause of depression.

It is possible that anathetic signals evolved as negative catathetic signals, allowing a functional connection between the new mammalian brain subserving affiliation and the old reptilian brain subserving agonism.[13] But it is beyond the scopo of the present discussion to ask whether RHP can be manipulated in the hedonic mode, or how RHF, which could be described as confidence in one's power over people, fits in with the broader concept of self-esteem, which also includes confidence in one's power over things, knowledge of one's popularity and a sense of the integrity of one's honour and virtue.[14]

In the same way that catathetic signals may be, variously, a glorification of the self, a disparagement of the other, or a comparative statement asserting the self's superiority over the other, so also anathetic signals may take different forms. The most common form is praise or glorification of the other; but denigration of the self is also an anathetic signal, a fact which may be useful in conceptualising the self-denigratory speech of depressed patients.

Anathetic signals may also take a comparative form, such as" *You are better than/superior to/ more powerful than me".* This is the message of submission; in other words, the signal of unfavourable relative RHP. Thus we have defined escalation and submission in terms of the signals which act on the adversary's RHP: escalation = signal of favourable relative RHP = catathetic signal =

lowering influence on adversary's RHP; and submission = signal of unfavourable relative RHP = anathetic signal = boosting influence on adversary's RHP.

Down-Hierarchy Catathetic Signals:

The discussion above has been concerned with the exchange of catathetic signals between individuals of equal rank. However, catathetic signals are also exchanged between individuals in asymmetrical relationships. They are usually directed from the dominant to the subordinate, and have the function of confirming and reinforcing the dominance. If they are directed from the subordinate to the dominant, they suggest a rebellion against the existing rank structure.

It is an interesting fact that the quality of catathetic signals is similar whether they are directed to an equal, a subordinate or a dominant. It is the quantity which varies and which differentiates symmetry from asymmetry, and dominance from subordinacy. In fact, it is the relative quantity of catathetic signals which is used to define dominance in many studies; and it is the consistency overtime of this relative quantity, and its correlation with other measures such as supplanting, precedence and advertence which gave rise to the concept of dominance/subordinacy in the first place;[15, 16, 17] a concept which has stood the test of time in spite of a suggestion that it might be an artifact of captivity.[18].

At least in human beings, however, catathetic signals may differ in quality, depending on whether they are directed up or down a hierarchy. To take an extreme example, a pupil may be cheeky to his teacher, and a teacher may cane his pupil; but we cannot imagine the pupil caning the teacher or the teacher being cheeky to the pupil.

The down-hierarchy catathetic signal contains two messages at different logical levels. First of all it is a straightforward catathetic signal which causes mental or physical pain in the usual way. But, secondly, it contains the message "I am in a position to give you a signal which is only given by dominant people to subordinate people". This higher level message also causes mental pain (humiliation). Thus the pupil receives pain from the caning and pain in the form of humiliation from the fact of being caned.

Asymmetrical **Anathetic and Neutral Signals:**

Like catathetic signals, most anathetic signals are similar whether directed up- or down-hierarchy; but, likewise, some are not, such as patronising behaviour (e.g., tipping). Equally, some RHP-neutral signals, such as those related to the task in hand, may be asymmetrical; for instance, to give certain types of order in certain tones of voice may only be appropriate to the one who is dominant (in the agonic mode) even though the pair or group may be operating in the hedonic mode and the subordinate individual may be in the role of leader.

It seems likely that the receipt of such an asymmetrical anathetic or neutral signal has the same effect as a catathetic signal, in that it is a threat to the recipient's RHP and challenges his dominance (or equality). Thus an asymmetrical anathetic signal boosts the recipient's RHP at one logical level and lowers it at another. The net effect may be to lower RHP and/or trigger a yielding subroutine.

Comments in 1998:

By and large I would stand by the above, with the following provisos:

1. While we can say that the function of catathetic signals is to lower RHP in the recipient, they only do so if they are not returned. The function of the RHP signalling system is to break symmetry. A fight in which equal blows are traded does not lower the RHP of either contestant. Only when one contestant fails to return the blows of the other is there a lowering of his RHP and a break in symmetry. This may be important in the training of children not to fight. If the child does not return a blow because of high moral standards, his RHP adjustment

mechanism may not have access to this information, and may assume that the blow has not been returned because of weakness, and so lower his RHP.

2. The fact that one attacks (rather than submits) is a signal of favourable relative RHP. The strength of the attack is a signal of absolute RHP, so that absolute RHP continues to be signalled during a fight, often in different ways, as the fight escalates from, say, roaring, to parallel walking, to locking of horns.

3. Although I still think it is useful to look on the catathetic signal as a signal of favourable relative RHP, it is also influenced by two other variables.

Resource value is a measure of how valuable a resource is to a particular individual, and the higher the resource value, the more likely the individual is to attack. For instance, in certain territorial cichlid fish, a territory becomes more valuable when the fish is in breeding condition; and success in territorial disputes was found to be more related to relative gonad size than to relative body size.[19] Resource value expresses the motivational element in fighting. The message is, "I can and will defeat you!": RHP provides the "can", and resource value provides the "will".

Ownership is another variable that determines the likelihood of attack rather than submit. It is an almost universal convention among vertebrates that a resident or owner attacks an intruder, and is likely to be victorious. When an encounter occurs on neutral territory, and concerns a resource which is of equal value to both contestants, the catathetic signal is a true signal of favourable relative RHP.

We have pointed out that a lowering of RHP, resource value and sense of ownership can account for a lot of the clinical features of depressive states.[20] This is in accord with our view that the capacity for depression evolved as a component of ritual agonistic behaviour. *c8*

Excerpt from *The Triune Brain in Evolution: Role in Paleocerebral Functions,* by Paul D. MacLean.[1]

The Question of Displays

It is one of the intriguing aspects of neurobehavioral evolution that a number of postures and autonomic changes seen in thermoregu-lation acquire symbolic significance in animal communication. For example, the piloerection and ruffling of feathers that serve, respectively, in mammals and birds to insulate against the cold may also serve to enhance the animals' size in aggressive or defensive encounters. Greenberg has cited references to four species of lizards that "use *a similar kind of posture in thermoregulation as in a show of aggression."*[2]

The question arises as to whether or not a detailed study of muscular insertions in therapsids would indicate a capability to engage in communi-cative displays involving, for instance, extension M a gular fold, sagittal expansion, and/or tiptoe extension of the limbs seen in

The Triune Brain - Les Trois Cerveaux

http://www.onf .ca/FMT/E/MSN/1**6/16513.html**

1983,29 minutes, 40 seconds

Abstract: This informative film illustrates American physician Dr. Paul MacLean's theory of human brain structure and evolution~the triune brain. The human brain, he theorizes, functions through the interaction of three cerebral formations: the reptilian complex, the limbic system, and the neocortex. These control instinct, emotions, and intellect, respectively. Using computer animation to explain the development of these formations, the film documents our current knowledge about this most complex of human organs

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ABSTRACTS & EXTRACTS...

Webster C: Computer modeling of adaptive depression. *Behavioral Sciences,* 1995;40:314-330.

Abstract: Mild, delimited, and adaptive depression may be a specific example of a more general class of mechanism by which intelligent systems -individual, social, and artificial - adapt to dynamic, uncertain, and dangerous environments. Computer modeling, based on connectionist and artificial intelligence planning and learning programming techniques, supports this hypothesis by generating both adaptive behavior and analogs for 10 phenomena associated with depression: global, stable, and internal failure explanation, a cognitive loop of failure rumination, decreased motivation, self-esteem, and self-efficacy, and increased realism, negative generalization, and cognitive change. The idea of adaptive depression can be applied to more than one level of living systems. A better understanding of normal and adaptive depression may lead to a better understanding of clinical depression.

Webster C. & Banks, G.: Modeling manic-depression with symbolic logic. *Proceedings of the 13th Symposium on Computer Applications in Medical Care,* November 6-9,1989. Washington, D.C.

Abstract: We characterize manic-depression in terms of symbolic logic and dynamical systems, and describe a computer simulation used to develop our theory. A formal theory of cognitive deficit had 4 parts:

1. For a normal representation we use the concept of sound and complete "self-axioms."
2. Normal processing occurs when changes in our personal environment trigger a search for a new set of sound and complete self-axioms.
3. Deficits can lead to unsound judgment in mania and incomplete judgment in depression.
4. Adaptation may consist of attempts to suppress or use changes in reasoning style.

Since manic-depression involves changes in the temporal organization of mood and judgment, it can be classified as a dynamical disease. Nonlinear dynamical systems exhibit transitions between steady state, periodic, and chaotic behavior. We illustrate our approach with a computer simulation that searches through a small set of "self-axioms" while exhibiting periodic and chaotic behavior. In conclusion, we conjecture that manic-depression may represent a bifurcation from the chaotic dynamics of normal emotional lability to the pathological periodicity of affective illness.

Johnson C.N.: Species extinction and the relationship between distribution and abundance. *Nature,* 1998;394:272-273.

Abstract: Within taxonomic groups, there is almost always a positive relationship between the size of geographic range and the local abundance of species. This pattern has attracted much interest, and several ecological mechanisms have been proposed as causes of it. However, these hypotheses do not consider the effect of the extinction of rare species on range-abundance relationships.

If both range size and local abundance influence the risk of extinction, species with small ranges might avoid extinction if they have high local abundance, whereas species with low local abundance might avoid extinction if they are widespread; species with both small range and low local abundance should be at high risk.

This interaction between range, abundance and extinction should produce negative correlations between range and abundance in groups that have experienced many extinctions. Here the author tests this idea using Australian marsupials, and

shows that although the relationship between range size and local abundance is positive for recently evolved species, it is negative for ancient species. This indicates that positive relationships between range size and abundance may be generated during adaptive radiation, but are then gradually reversed as a result of differential extinction.

Arnsten A.F.T. & Goldman-Rakic P.S.: Noise stress impairs prefrontal cortical cognitive function in monkeys: Evidence for a hyper-dopaminergic **mechanism.** ***Archives of General Psychiatry.*** 1998;55:362-368.

Abstract:

Background: Stress can exacerbate a number of psychiatric disorders, many of which are associated with prefrontal cortical (PFC) cognitive deficits. Biochemical studies demonstrate that mild stress preferentially increases dopamine turnover in the PFC. Our study examined the effects of acute, mild stress exposure on higher cognitive function in monkeys and the role of dopaminergic mechanisms in the stress response.

Methods: The effects of loud (105-dB) noise stress were examined on a spatial working memory task (delayed response) dependent on the PFC, and on a reference memory task with similar motor and motivational demands (visual pattern discrimination) dependent on the inferior temporal cortex. The role of dopamine mechanisms was tested by challenging the stress response with agents that decrease dopamine receptor stimulation.

Results: Exposure to noise stress significantly impaired delayed-response performance. Stress did not impair performance on "0-second" delay control trials and did not alter visual pattern discrimination performance, which is consistent with impaired PFC cognitive function rather than nonspecific changes in performance. Stress-induced deficits in delayed-response performance were ameliorated by pretreatment with drugs that block dopamine receptors (haloperidol, SCH 23390) or reduce stress-induced PFC dopamine turnover in rodents (clonidine, naloxone hydrochloride).

Conclusions: These results indicate that stress impairs PFC cognitive function through a hyper-dopaminergic mechanism. Stress may take the PFC "off-line" to allow more habitual responses mediated by posterior cortical and subcortical structures to regulate behavior. This mechanism may have survival value, but may often be maladap-tive in human society, contributing to the vulnerabi -ity of the PFC in many neuropsychiatric disorders

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Dattani M.T.; Martinez-Barbera **J.P.; Thomas P.Q.;** Brickman J.M.; **Gupta** R.; Martensson **I-L Toresson H.; Fox M., Wales J.K.H.; Hindmarsh P.C.; Krauss S.; Beddington R.S.P.; & Robinson I.C.A.F.: Mutations in the homeoboxgene** HESX1/Hesx1 **associated with** septo-**opticdysplasia in human and mouse.** ***Nature Genetics,*** 1998;19(2):125-133.

Abstract: During early mouse development the homeobox gene Hesxi is expressed in prospective forebrain tissue, but later becomes restricted to Rathke's pouch, the primordium of the anterior pituitary gland. Mice lacking Hesxi exhibit variable anterior CNS defects and pituitary dysplasia. Mutants have a reduced prosencephalon, anopthalmia or micropthalmia, defective olfactory development and bifurcations in Rathke's pouch. Neonates exhibit abnormalities in the corpus callosum, the anterior and hippocampal commis-sures, and the septum pellucidum.

A comparable and equally variable phenotype in humans is septo-optic dysplasia (SOD). We have cloned human HESX1 and screened for mutations in affected individuals. Two siblings with SOD were homozygous for an Arg53Cys missense mutation within the HESX1 homeodomain which destroyed its ability to bind target DNA. These data suggest an important role for Hesx1/HESX1 in forebrain, midline and pituitary development in mouse and human.

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