

# ASCAP

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"Fossilized brains do not exist. Therefore, evolution of the brain is deduced mainly from comparisons of extant species."  
Ivan Divac<sup>1</sup>

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Concerning paleobiology, sociophysiology, interpersonal and group relations, 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 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.**

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# ADDRESSED TO & FROM ...

## ASCAP Memories and Plans

With this issue, nine years of monthly issues of *The ASCAP Newsletter* will have been published. In mid-morning of mid-December, 1987, I drove along Galveston's Seawall to my dentist for a teeth-cleaning and suddenly experienced the solution to a dilemma I faced of communicating with an increasing number of colleagues who like me were interested in the interface of evolutionary biology and psychopathology. I had just acquired a word processing program which included a feature that made double columns. Inspired, I realized that a newsletter was the very thing. I worked on the first one that afternoon. More than one-hundred issues later, we are still going, with many new friends along the way.

Some have encouraged that the newsletter should itself evolve into a peer-reviewed journal and perhaps eventually it should. But from the beginning, I have myself conceived it as a brain-storming operation with comfortable idea-exchange. This doesn't mean that the ideas should be immune from debate and criticism. For example, Don Klein next issue will take us to task for something we have published. Data helps ideas win out and ideas may frequently involve how data

should be best gathered so that idea-competition can be more decisive. But in my years of psychiatry there have often been too few good ideas and I have often felt that we need more brain-storming. This doesn't mean tolerance of stupidity, but openness to fresh thought, not as settled truth but as opening the door a crack to new domains of how we should think of the mysterious phenomena with which the clinicians amongst us deal daily.

The newsletter was not conceived as being a last word but rather a beginning word, sometimes not to go further. The approach of a refereed journal is that too many ideas are bad ones and that they should be winnowed into from the papers accepted. That is as it should be and I approve thoroughly.

But I am proud that articles and books have been fostered by this publication having expressed the ideas in nascent form. Creative authors are always encouraged to write not only for the newsletter but in as many other fora as possible.

I think that has happened and this is what I had envisioned driving down the seawall December 15, 1987. In the Darwin Machine, as William Calvin has described it, there should initially be an overabundance of forms,

whether these are life forms or idea forms. Later they compete for survival and reproductive success. This instrument aims at the overabundance. Refereed journals are the real world tests.

In July, 1991, in a meeting at John Price's home in Odintune Place, Sussex, Leon Sloman proposed the beginnings of what we now call The ASCAP Society. We have small informal yearly meetings, though the formality may be increasing.

Our first president was the extraordinary Michael Chance who originated ethologically oriented psychopharmacological research in Birmingham, UK, after World War II.

Our next president, English psychiatrist John Price, had been stimulated by Dr. Chance and considers depression as a mechanism to signal defeat and yielding as also demonstrated by non-human animals in conflict.

Then prolific English psychologist Paul Gilbert who had rounded out human features of such work ascended to the helm. After him came John Pearce, psychiatrist, family therapist, psychopharmacologist, and co-author of *Exiles from Eden*, who became the first US president.

Canadian child psychiatrist Leon Sloman from the Clarke Institute

in Toronto who has championed the involuntary subordinate strategy came next, preceding psychologist Kent Bailey from Richmond, Virginia, who has just co-authored *Families by Choice*. This refers to our human capacity to use family relationships for salutary family-like purpose though participants may not be genetically connected.

Our small organization clearly has one formal tradition for its annual meeting: for two iterations, psychiatrist Mark Erickson led a committee for the Aaron T. Beck ASCAP Award for the best essay written by a young investigator or thinker on the topic of evolutionary biology and psychopathology. Winners present at the annual gathering. So far these have been Nicholas Allen from Melbourne, Australia (he is now in Oregon) and Souhir Ben Hamida from Northwestern University in Chicago, U.S.A. Dr. Beck, founder of cognitive therapy for depression and anxiety, has been very proud of these winners and eager awaits who next will come forth. As Dr. Erickson is moving to the Menninger Clinic in Topeka, Kansas, he asked if the Beck Award committee this year might be headed by another. President Bailey appointed Thomas Joiner, Ph.D., from UTMB in Galveston, to assume that responsibility. Dr. Joiner, a recent graduate in Psychology from the University of Texas, in Austin, is considered one of the foremost young psychologist researchers in the country and is excited about the responsibility. He focuses on

interpersonal facets of depression and has been very prolific, publishing many papers and doing that in high quality journals. He feels that evolutionary aspects of the condition may be underestimated and has written on this in addition to his data-based research. He is also facile on the Internet and I anticipate that word of the competition will reach many.

Our next annual meeting will take place in Tucson, Arizona, on Wednesday, June 4, 1997, just before the Human Behavior and Evolution Society meeting. Mark your calendars. Our president has been thinking about how the meeting should be organized and has suggested the theme of "Depression, Life Satisfaction, and Happiness." He believes that there should be a keynote speaker along with other invited speakers (in addition, of course, to the Beck Award winner). We are working on those arrangements in the continuing evolution of how we do things.

Russell Gardner, Jr.  
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### *Taking Evolutionary Psychology seriously:*

As a dedicated reader of *The ASCAP Newsletter* the use of notions like "origins of such and such behavior" seems increasingly shortsighted to me. Let me try to explain this uneasiness:

In taking evolutionary psychology seriously, we must not stop

with paleolithic hunter-gatherers as our ancestors. Evolution concerns transition of species, yielding the new by changing the old. Surely arboreal primates anteceded hominids: surely tetrapodal mammals anteceded them. If we go further back, to the origin of the vertebrate/chordate phylum, we can take a kind of fish as an very early ancestors' prototype. Let us suggest: their life, their behavior shaped our brain organisation more than all subsequent changes altogether did.

Surely fish relied on others to keep their life (and line) going. They were mobile, heterotrophic (feeding on others) and sexually active (choosing partners). It is not sure whether they were prey for other animals, but since mammalian times our line was threatened by predators for long periods. Cave-bears and sable-toothed tigers did not substantially change the hominid brain after such a long history of fighting for life.

In short, if paleozoic fish behaved roughly as we might conceive of a recent standard fish, the first categorization of essential objects for them must have been: prey, predator, partner, concurrents (same sex, same species, other species) and all sorts of neutral animals. As all these "objects" were themselves mobile, their discrimination by means of specific movement patterns were the first action tasks for the vertebrate nervous system. Inanimate objects did not matter much, because motion by physical causes is less complex compared to behavior.

In other words:  
Vertebrates are natural observers,  
or  
Vertebrate brains evolved in  
interaction with other brains, **or**  
A vertebrate brain needs another  
brain to show basic functions, **or**

A vertebrate brain cannot be  
explained alone, **or**  
Neurophysiology originated as  
sociophysiology.  
P.S.: An article describing  
interactive behavior as "mimetic"  
in vertebrate evolution is in

preparation (in German). A poster  
presented at the 1996 ISHE  
Meeting in Vienna ("Hunting or  
following each other: Origin of  
mimetic skills?") is available from  
the author.

Anton Furlinger

### ***Announcement of the 3rd Annual Aaron T. Beck ASCAP Award Competition:***

The ASCAP Society is sponsoring the 3rd Annual Aaron T. Beck Award competition. This award is given to the best unpublished paper related to the subjects of evolutionary biology and psychopathology. Single authored papers may be submitted by students of relevant fields (e.g. psychiatry, psychology, biology, anthropology), as well as by recent graduates (within 3 years of terminal degree or residency graduation).

Over the last two years, a number of outstanding papers were submitted. The award winners thus far have been Dr. Nicholas Allen of the University of Melbourne, for "Towards a Computational Theory of Depression: An Evolutionary Perspective, and Souhir Ben Hamida for "Mate Preferences: Implications for the Gender Difference in Unipolar Depression". Dr. Beck has taken a personal interest in the awardees.

Society members feel that an evolutionary perspective can be used to integrate various levels of understanding and thereby generate new or broadened perspectives for solving clinical problems. There are many possible areas for research and theorizing, including, to name a few, the relevance of evolutionary theory for psychotherapy, comparative psychology and psychiatric illness, comparative brain anatomy and pathological behavior, relations of attachment processes or social rank hierarchy to psychopathology. Data-driven papers are acceptable as are literature reviews supporting novel concepts.

The award will be presented in the form of a plaque, at our annual meeting to be held on 4 June 1997, in Tucson, Arizona, one day prior to the annual meetings of the American Psychiatric Association. The Aaron T. Beck ASCAP Award carries with it a cash prize of \$1000.00 to support travel expenses. The essay will be published in *The ASCAP Newsletter*. The winner will also receive a year's free subscription to *The ASCAP Newsletter* as well.

The ASCAP Society is an international group of clinicians and academics who are linked by a common interest in evolutionary biology and how this perspective can inform our work and research (ASCAP refers to Across-Species Comparisons and Psychopathology).

We take this opportunity to ask you to notify residents, graduate students, fellows, and recent graduates of your department about this competition. With this issue, we have included a flyer to post this notice. Please feel free to copy the flyer for any distribution that you might want to make.

#### **All participants should send 3 copies of their paper to:**

Thomas Joiner, Ph.D. - Beck ASCAP Award  
Department of Psychiatry & Behavioral Sciences  
Room 3.104, Marvin Graves Building  
University of Texas Medical Branch  
Galveston TX 77555-0425, USA

The postmark deadline for entries will be March 1, 1997. Do not hesitate to call (409) 772-2419 for further information about the Beck Award or the ASCAP Society or use E-Mail to:  
Thomas.Joiner@utmb.edu.

## **Mismatch Theory 4: The four-fold model and varieties of depression**

In earlier essays in *The ASCAP Newsletter*, the fundamental assumptions of mismatch theory were outlined (February 1996), the distinction between nature/culture conflicts and mismatches was discussed (March 1996), and implications of the four-fold model were outlined (April, 1996). Several *Newsletter readers* have commented on the ongoing series, and Tim Miller provided an in-depth critique in the August 1996 issue.

In this essay, I explore the heuristic possibilities of the four-fold model in reference to the varieties of depression. I was privileged to present a paper on this topic at the annual meeting of the ASCAP Society in New York in May 1996, and I wish to thank members for their helpful suggestions and encouragement.

Depression is one of the most omnipresent features of human existence, and psychology textbooks call it the common cold of psychiatry. As Rosenhan and Seligman say, "Almost everyone reacts to loss with some of the symptoms of depression".<sup>1</sup> Whatever it is, depression is a complex neurophysiological and neuropsychological event that impacts the individual from the deepest sociophysiological levels of experience to the highest cognitive ones.

Debates rage over endogenous/exogenous issues, the evolutionary normality/abnormality of depression, the evolutionary continuity of depression, genetic and biochemical etiology, class and gender issues, cognitive etiologies, preferred treatment procedures, and so on. Much like schizophrenia, depression is a collective term for what is probably a variety of disorders; each variation shares the emotion of sadness and differing degrees of psychomotor retardation, but each varies in the particular patterning of neurophysiological, cognitive, and situational determinants.

Many approaches to depression proceed from clinical induction (e. g., observations in psychotherapy) to theoretical deduction (e. g., Freud's notions of incorporation of the object, aggression turned inward, superego domination of the id, etc.), and, finally, to empirical verification. Others apply pre-existing models, often cognitive-behavioral ones (e. g., Beck, Lewinsohn, Seligman) to the etiology and treatment of depression. Evolutionary models have also been especially fruitful (Price, Sloman, Gardner, and Gilbert).

Mismatch theory and the four-fold model offers a fundamentally deductive approach to depression where particular cells in the model allow inferences about probable conflicts between human nature (*viz.*, the evolved impulses to strive for biological success in the areas of physical health, mating, and, ultimately, reproduction and parenting) and our cultural selves (*viz.*, the acquired needs to incorporate the rules and symbols of culture, and to compete favorably with others for cultural resources and prestige).

I suspect that real or perceived failure in the biological or cultural arenas - or failure at both - is at the root of the paleodynamics of defeat and submission, loss of status, learned helplessness, and neuropsychological inhibition that give rise to the varieties of depression.

More specifically, depressed mood may serve as a signal that the individual is "failing" in the pursuit of proximate species goals and, probabilistically, this will eventually lead to failure at ultimate levels of analysis. Herrnstein theorized that animals experience psychological pleasure when acting in accordance with species imperatives,<sup>2</sup> and we may presume, contrariwise, that failure in being a good species member will induce psychological pain or

unpleasure. Much as anxiety serves as a signal of impending internal or external threat, or physical pain serves as a signal of damage or dysfunction structurally, depressed mood signals one that things are problematic in the domain of biological adaptation.

Thus, when one loses a loved one, loses status or prestige, loses material resources, or is rebuffed by a potential mate, nature has provided a very painful signaling mechanism that motivates the individual to sit back, re-assess the situation and devise alternate strategies.

So viewed, depressed mood is a species-evolved adaptive characteristic that serves "normal" functions in most instances, but clinical depression would occur when the individual is incapable of devising alternative strategies, or circumstances block such alternatives. "Learned helplessness" is the term used by Martin Seligman and his colleagues to describe such circumstances.

### **Inhibition and depression:**

The four-fold model, and paleopsychology in general, assumes that order and integrity of living organisms is premised on powerful and highly conservative processes of inhibition that maintain structural organization of systems with almost perfect fidelity. For example, hemoglobin consists of 576 amino acids in chains and its function is to carry oxygen through the bloodstream to cells throughout the body. Change of only one amino acid in the normal sequence converts a normal red blood cell into the cell responsible for sickle-cell anemia.<sup>3</sup> Such fundamental sociophysiological processes are highly constrained, operate within narrow limits, and are tolerant of little or no error in the system.

With the need for high-perfect functioning at the genetic and biochemical levels to avoid pathology, and with the added constraints of frontal-prefrontal inhibition at the neurological level,<sup>4</sup> conscience, superego, and constraining values at the psychological levels, and rules of law, ethics, and morality

at cultural levels, human beings are not all that "free" in fact. (Given this argument, it follows that it is quite easy for an individual to sense dysfunction or "failure" in the many highly coordinated, tightly constrained adaptive systems that subservise the striving for biological success in the domains of health/attractiveness, mating, parenting, and kinship/social support).

Many forms of physical disease and psychopathology reflect an abnormal release of constraint in pre-existing normal physiological systems. Genetic mutation is a deviation from the species-normal sequencing of amino acids and nucleotides and, with its dark handmaiden, cancer, is capable of inducing great misery and often the ultimate destruction of the organism.

According to Boyd Eaton, various mismatch stresses in the areas of diet, exercise, social relations, and the physical environment may contribute to the pathogenesis of cancer.<sup>5</sup> Nesse and Williams offer similar arguments,<sup>6</sup> and in *Evolutionary Medicine*. Lappe discusses the price the body plays when certain renegade cells come out of inhibition.<sup>7</sup>

Clearly, physiological and psychological normality are tenuous things that must be constantly maintained by genetic, biochemical, psychological, and external/cultural constraints. Given such widespread and powerful constraining influences, some physical and mental diseases based on excess inhibition would be expected.

I suggest that "normal" depression is, to a significant degree, the painful perception of real or perceived failure in the arenas of biology and/or culture, which sets into motion two basic plans of adaptation: (1) a temporary cessation or reduction of motivational and behavioral activity- that is, a time-out period from interpersonal competition, and (2) the activation of cognitive re-assessment and coping processes whose primary function is to alert the individual to problems and to guide the re-assessment and re-adjustment process.

Clinical depression, by contrast, reflects failure in immediate re-assessment and coping, and powerful physiological and psychological processes are set into motion that massively inhibit ongoing processes and essentially remove the helpless individual from the various arenas of biological and cultural competition (as suggested in the Price-Sloman-Gardner ISS approach to depression), perhaps for purposes of rest, energy remobilization, social support seeking and alliance development, and the formulation of new adaptive strategies. Somewhat like the reproduction-suppression theory of anorexia nervosa, immediate response suppression and shut-down of various systems may set the stage for more effective behavioral outputs under more propitious circumstances.

Whatever its true causes and functions, depression is a disorder defined primarily by inhibitory processes, negative symptoms, and avoidance behavior including psychomotor retardation, retardation of sexual interest and behavior, suppression of dominance and aggression (e. g., the ISS), suppression of appetite and eating, suppression of sleep patterns, suppression of warm sociality and affection, immunosuppression, and, in extremis, the total suppression of self— suicide.

#### **Depression and the four-fold model:**

Even in the normal ranges of sadness and blue-ness, hyper-inhibition is evident in high self-control, strong superego development, guilt proneness, cultural conformity, and constrained emotion. In moderation, these characteristics are often conducive to classroom success, vocational success, and cultural success in general. The disinhibited, impulsive individual (e. g., attention-deficit hyperactivity disorder), by contrast, may pursue biological success quite effectively, but he or she may be at a great disadvantage in the classroom, in most high-paying jobs, and certainly in the intellectual meritocracy.

Numerous writers have commented on the role of inhibition in intellectual performance (e. g., David Stenouse's notion of the postponement factor in

creative intelligence),<sup>8</sup> and given the highly touted role of IQ in cultural success (e. g., The Bell Curve we might infer that a certain level of personal constraint is necessary to achieve cultural success. Thus, mild forms of depression just might facilitate striving for cultural success, and certainly many great writers and poets were noted for both their depression and alcoholism.<sup>9</sup>

Whereas, mild-to-moderate depression may be adaptive culturally, it is likely to be generally mal-adaptive in the biological domain. Depressives are often low on libido, unattractive and unsexy as potential mates, low on dominance, and high on emotional neediness and the desire for social support.

Depression may be evolutionary adaptive in limited instances where delay of response and energy remobilization are concerned, but even there the dominant, sexy, non-depressed individual is going to find mates and produce offspring while the depressive is "resting", hoping for better times.

I suggest that mild depression is typically adaptive culturally, but only occasionally adaptive biologically, whereas severe depression is rarely if ever adaptive either culturally or biologically. Once the depressive is inhibited to the point of physical and cognitive immobilization, little biologically or culturally adaptive output is possible, and the system borders on shut-down, somewhat like the effects of shock following a serious accident. In extreme depression, there may be a "giving up" across all systems producing a kind of psychological death or ego-cide.<sup>10</sup>

#### **Cell analysis and depression:**

Table 1 outlines some of the cell expectations for the varieties of depression. In Cell 1, we find biologically and culturally successful people who, nevertheless, occasionally encounter non-catastrophic illnesses, losses, and problems in life that activate the unpleasure signaling mechanism short of clinical depression. Their blues or situational depression is more-or-less adaptively tracked in that



signaling of unpleasure leads to constructive attempts to reinstitute proximal patterns consistent with ultimate biological success. For example, the death of a loved one may reduce his/her inclusive fitness and thereby activate negative signaling, but the healthy, resourceful Cell 1 person will weather the storm and quickly get back into biological competition. Given that depression is an inhibition disorder, it is no surprise that Cell 1 depression is about twice as likely in women as men. Epidemio-

logical surveys<sup>11</sup> have confirmed that women tend to suffer from inhibition disorders or type 2 paleopsychopathology (anxiety, depression, anorexia, dependency, etc.), whereas men tend to suffer from disinhibition disorders or type 1 paleopsychopathology (crime, violence, psychopathy, substance abuse, etc.).

Also, women expend far more energy in the reproductive process than men, seem to be more K-oriented and focused on parenting than men, and physical

		<b>The Four-fold Model and Depressive Disorders CULTURAL/NEOCULTURAL SUCCESS</b>	
		<u>High</u> Cell 1	<u>Low</u> Cell 2
BIOLOGICAL SUCCESS	<u>High</u>	<u>Optimality</u> MILD "BLUES" SITUATIONAL DEPRESSION (females+) (high guilt) (adaptive depression) (mild mismatch)	<u>Social Pathology</u> SERIOUS "BLUES" "DEPRESSED WIFE BEATERS" "DEPRESSED" CRIMINALS SUBSTANCE ABUSE SITUATIONAL DEPRESSION (males+??) (high frustration) (adaptive/maladaptive) (least mismatch)
	<u>Low</u>	Cell 3 <u>Personal Maladjustment</u> MAJOR DEPRESSION DYSTHYMIA DEPRESSION SUICIDE (females+++) (high guilt) (maladaptive) (high mismatch)	Cell 4 <u>Severe Psychopathology</u> MAJOR DEPRESSION (DELUSIONAL) BIPOLAR DISORDER I & II ANXIOUS ILLNESS-RELATED DEPRESSION SUICIDE (females+) (high frustration) (highly maladaptive) (highest mismatch)

attractiveness and mating seem to be more basic to female psychology. This suggests to me that women either have a stronger unpleasure signaling mechanism than men, or that they simply respond more strongly to unpleasure signals (indicating "you are off-base biologically"). If women are, first, more inhibited and, second, more sensitive to biological deficiencies than men, then depression would be much more likely in women.

Both Cell 1 and Cell 3 persons are culturally successful by definition, and we would expect them to typically be very conforming, self-controlled, and prone to both guilt and anxiety. Persons in both cells tend to internalize, objectify, and live in a rich psychological world of self-reflection and epistemological angst. They are highly involved in the social comparison processes Paul Gilbert<sup>12</sup> describes so well, especially in the realm of cultural prestige and social resource holding power. According to the accuracy theory of depression,<sup>13</sup> the more sensitive of these people worry and fret about the imperfections of the world, and just may be more "realistic" about it than the rest of us. For the Cell 1 person, these processes are subclinical, whereas the Cell 3 person is the veritable prototype for moderate-to-severe unipolar depression.

When a person is vigorously pursuing ancient biological goals in contemporary environments, considerable EEA-current environment matching is involved, but failure to pursue such goals involves some degree of mismatch, since we assume that ancestral people were reasonably vigorous, on the average, in their biological goal-seeking. Moreover, mismatching enters in at all points where the pursuit of cultural success interferes with or diminishes the pursuit of biological success.

The Cell 1 person is thus mismatched to a degree due to vigorous pursuit of cultural success, but he or she, nevertheless, is able to reconcile the separate prerogatives of biology and culture better than anyone else. In theory, Cells 1, 3, and 4 contain relatively few people, whereas Cell 2 is the main repository for human beings, even in privileged technological societies. In the richest societies,

there are many more cultural failures than successes, and I personally do not know many people who have reached or exceeded their desires for prestige and SAHP. In technological meritocracies, there is always a deep longing to be more than you actually are, and this goes right to the heart of the Cell 2 dilemma.

Cell 1 and 3 people may suffer angst over their perceived cultural failures and frustrations, but cultural failure is an oppressive reality in Cell 2. In America, the unemployed, the poor, and even the lower middle classes are bombarded with the celebrity, the social power, and wealth of the very few, and, for them, the American Dream can only come through a lucky hit in the lottery or compensatory criminality. The Cell 2 person is biologically successful by definition, and cultural pursuits do not drain away energy as in Cells 1 and 3. Therefore, the Cell 2 person is maximally matched and minimally mismatched relative to EEA-current environment relationships. He or she pursues biological goals with vigor, but is likely to be conflicted due to the disparity between biological and cultural success.

Whereas, the Cell 1 person is mildly mismatched and conflicted due to interference of cultural pursuits with biological goals, and the Cell 3 person neurotically conflicted, the more flexible Cell 2 person may not fret excessively over cultural failure, and may find solace in family, children, and friends. But for many, there is a deep frustration and longing for better times, which may progress to serious levels of anomie and alienation from the mainstream, as in so-called Black Rage. Paleodynamically, the Cell 2 person does not tend toward depression, but his/her sense of frustration and hopelessness may emulate clinical depression. But there is little inhibition, on the one hand, and minimal unpleasant signaling, on the other, since biological goals are being met. Promiscuous young males in the inner city who impregnate numerous young women, and the young mothers with several children by different fathers, are seldom guilty or depressed about behavior that sabotages any hope of cultural success; rather, they are likely to be more pleased with life than the privileged but neurotic Cell 3 person, and certainly more happy than the Cell 4 person. Given the high biological success in Cell 2,

we might suspect that internal signals will be predominantly pleasurable rather than unpleasurable. Unpleasure, frustration, and rage come from "being looked down upon" culturally, not from intrapsychically induced depression.

In Cells 1, 3, and 4, women predominate in rates of depression. Once careful distinctions are made between genuine depression and cultural frustration, I theorize that men suffer the most in Cell 2. The female can remain ensconced in the homemaker role or revert to it when cultural doors are closed. Her natural roles as mother and kinkeeper give her an out, but what escape is there for the male whose natural roles of impregnator, hunter, and warrior constitute criminal acts in most modern contexts?<sup>14</sup> When the uneducated and culturally unsophisticated male cannot attract or keep a mate, when he loses his children in divorce, and when he loses his job, there is nowhere to go. Is it any wonder that lower class black men (rhythm and blues) and white men (blue grass music) can sing the "blues" with such feeling? Nothing gives the Cell 2 man the blues more quickly than for another more sophisticated and culturally successful man to take "his woman" away.

Cell 3 is where the classic dynamics of conformity, dependency, inhibition, and internal signaling of biological failure play themselves out. Anorexia nervosa—which is highly comorbid with depression—is probably the true Cell 3 pathotype, and, of course, suicide is the ultimate renunciation of biological goal-seeking. Psychodynamically, depression also is mainly a Cell 3 disorder, where internal inhibition and displeasure are experienced within a context of high cultural and low biological success.

It is from the Cell 3 ranks that most psychotherapy patients come, and therapy tends to focus on "relationship" issues more than anything else. Most of these conflicted relationships tend to curtail or sabotage biological goals in the area of mateships, marriage, parenting, and extended family relations. Somehow, the depressed Cell 3 person must find a way to reconcile his/her biological and cultural goal-seeking so that internal signals of unpleasure are actually reduced, or simply diminished psychologically by cognitive restructuring ("I should learn to be happy with my social success") and re-labeling (e. g., "a family is any loving relationship...its does not have to be biological family").

Cell 4 persons are mismatched the most because they fail to achieve matches either biologically or culturally— that is, they cannot effectively seek either biological or cultural goals. These are people who often have to be hospitalized for serious physical and/or mental illness, and they come squarely under the purview of the medical and mental health establishments. Their depressions are severe and debilitating, and their extreme levels of learned helplessness require often equally extreme treatments: electroshock, hospital supervision, and powerful anti-depressive medications. The four-fold model does not add a lot to our understanding here, except perhaps in formulating plans for psychotherapy once medical treatments are implemented. It seems to me that both Cell 3 and Cell 4 pathology would be ameliorated somewhat by moving toward the Cell 2 pattern—facilitating biological goal seeking and maximizing biological success. More discussion of these issues later. c8



## ***Announcement***

**COMING SOON TO AN INTERNET SITE NEAR YOU!**

**THE ASCAP EUROPEAN HOME PAGE!**

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

The resultant World Wide Web address will be announced in a future newsletter.

## Personality Traits and the EEA

Specialised roles are common in nature, for example the social insects. The conjecture is that humans are also specialised, notably in their personality traits. This individualism is posited to be the most economical means of ensuring a wide range of inputs to problem-solving. The nature of each person's contribution to problem-solving typifies their personality traits.

If Darwinian theory is true, humankind is the product of evolution by natural selection. Those aspects of humankind which assisted survival were favoured at the expense of those which did not. "Attribute" is the term used for those features of humankind which were shaped by natural selection.

Physical attributes include, among others, bipedal motion, free hands, large brain, relative hairlessness and dependence on water. Social attributes cover the totality of living arrangements, for example the size and nature of the groups. Individual attributes are those features which distinguish one person from another and encompass the whole range of human abilities, including intelligence, aptitudes and personality.

Humankind evolved as a social gatherer-hunter, one who lived in groups. Gathering and hunting was a problem-solving activity. What resources were worth exploiting? When and where? Would the potential gain have been worth the effort? Cosmides, Tooby, and others have shown that humans are adapted to a collective lifestyle. For example, group members are adept at the detection of social cheating, in which individuals try to gain the benefits of cooperation without the costs.

Long-term, climatic instability in Africa demanded from humankind the capacity to create new social rules and structures to match the diverse environ-

mental conditions. The attribute which enabled humankind to cope with abundance or scarcity in the habitat through social innovation is called the "scale".<sup>1</sup> The task was an undertaking of utmost difficulty. Individual attributes, including personality traits, had to be rich enough in content and variety to actualise the scale.

For the preceding reasons, problem-solving and the group nature of many decisions are posited to be the most important evolutionary criteria which shaped human individuality and personality. For gatherers and hunters, problems had four distinct parameters which represented a range between, at each extreme, mutually exclusive alternatives, namely:

1. a. Social (involvement in personal relationships), or  
b. Nonsocial (interest in things, objects, concepts and ideas), **and**
2. a. Complex (many variables), or  
b. Simple (few variables), **and**
3. a. Familiar (a variant of a problem already solved), or  
b. Novel (a new problem), **and**
4. a. Reasoned (a problem that could be solved only by rational thought), or  
b. Imaginative (only guessed at by intuition).

Accordingly, problems could fall into one of sixteen categories ( $2^4$ ). Thus, if the first two categories are viewed in isolation (social or nonsocial and complex or simple), four types of problem can be enumerated (social-complex, social-simple, nonsocial-complex and nonsocial-simple). In like manner, two possibilities from the total of sixteen are: first, a complex dispute between people which had arisen before and

had a reasoned solution, and, second, a simple problem about the manufacture of a tool which had not been met before and had only an imaginative solution.

For the sake of argument, a dichotomy is assumed between simple and complex. In reality, a continuum of ever-harder tasks links the easy to the difficult. A similar spread, which has significant implications for the distribution of personality traits, is found in the other parameters.

The four problem types determine humankind's four personality traits, both of which are summarised and matched with each other in Table 1.

<b>Table 1</b>	
<b>Problem Category:</b>	
<b>From:</b>	<b>To:</b>
1. Social	Nonsocial
2. Complex	Simple
3. Familiar	Novel
4. Reasoned	Imaginative
<b>Personality Traits:</b>	
<b>From:</b>	<b>To:</b>
1. Socializer	Inquirer
2. Planner	Spontaneous
3. Known methods	New ideas
4. Rational	Intuitive

Consequently, the concept is that key features of human personality consist of, at the extremes, mutually exclusive traits which mirror the characteristics of the problems to be solved. Thus, individuals may be interested in personal relationships with others (socializers) or in things, objects, concepts and ideas (inquirers). Planners have the inclination and persistence to tackle more complex questions, while people with a bias towards spontaneity prefer to move quickly from one simple problem to the next. Difficulties analogous to those met previously are solved by individuals who prefer

known methods, while novel situations are resolved by those interested in new ideas. Finally, problems solvable by reason are the province of rational thinkers, while problems with no rational solution are unravelled by intuitive thinkers.

Key personality attributes can now be predicted to consist of four pairs of traits. Moreover, some individuals tend to have a stronger and weaker preference in one or more of the four traits. For example, one personality may be biased towards the traits of inquirer-planner-new ideas-rational, while another could be predisposed towards the traits of socializer-spontaneous-known methods-intuitive.

For the majority (all-purpose individuals), the preference in each trait is modest, if any, and all aspects of each trait can be used to solve problems. For instance, both a rational and intuitive approach can be employed. However, some individuals (specialists) are more highly specialised and only work effectively in the favoured aspect of their trait. For example, with rationals, all actions have to be justified by reasoned argument. These individuals are better at rational thought than their colleagues and can point out the logical or practical flaws in any proposal.

Thus, even among a small group, at least one member with a bias towards one of the eight trait preferences is likely to be found. Human reasoning abilities are, thereby, significantly strengthened and are, therefore, capable of solving the sixteen potential types of problem. Cooperative decision-making by individuals who exhibit modestly or highly specialised personality traits is the technique by which humankind maximises its intellectual resources.

The existence of human traits essentially similar to those described is widely acknowledged and derives from the work of Jung, whose terms of extrovert (socializer-spontaneous) and introvert (inquirer-planner) are in popular usage. One respected personality test (The Myers-Briggs Type Indicator test<sup>2</sup>) specifically measures, although with different nomenclature, the four traits described,

recording, for example, that, at the extremes, a minority of managers are compulsive planners, while others avoid the task. Some researchers never stray far from existing authorities, while others are only excited by original ideas. Some people largely justify their actions by rational arguments, while others, on the contrary, only really trust their intuition.

If personality traits can be shown to have an evolutionary origin, the consequences for understanding ourselves are profound.

Michael Davies is co-author, with Henry Davies and Kathryn Davies, of "*HUMANKIND THE GATHERER-HUNTER: From Earliest Times to Industry*" (ISBN number 0 9519445 0 9), Kent, England, Myddle-Brockton Publishers, 1992, in which the ideas discussed in this article are more fully explored and integrated into a comprehensive, coherent theory of human behaviour.

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***Announcement***

As of 1 December 1996, the new E-Mail address for the Managing Editor is no longer [ascap@beach.utmb.edu](mailto:ascap@beach.utmb.edu).

The new E-Mail address is:  
**[ascap@utmb.edu](mailto:ascap@utmb.edu)**.

The alternate E-Mail address is:  
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## Population Crises and Population Cycles

### 4. The Northern Mediterranean: Greece

(Reproduced with permission from the Galton Institute Newsletter, September 1995.)

**Editor's Note:** This is the 4th installment to this article. Three other 3 installments were in our March, October, and November issues of *The ASCAP Newsletter*. The reference list for this article and the articles before will appear in a next issue.

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In China and the dry belt, the food surplus was ample for developing high civilisation, but high population density and the demands of water control (hydraulic) engineering produced what Karl Wittfogel (1956) called *hydraulic societies* - arbitrary autocracy, bureaucratic elite, and mass labour, with no one really free. The peoples of the ancient Northern Mediterranean lacked sufficient food surplus to build high civilisation. They did so only by exploiting the surplus of the neighbouring dry belt, through piracy, trade, and conquest (**Table 1**).

At first, they were not forced into the hydraulic pattern, and they could develop the rule of law, and real freedom for many people, even democracy, with leisure for fundamental scientific inquiry. But their dependence on imported food meant *two* factors for population crisis: their own population increase, and a failure of food imports. Over-population was enhanced when slavery diffused into their societies from the dry belt (**Table 2**).

They could not possibly afford slaves from their own meagre surplus, and the slaves' grotesquely high proportions: Athens, in 431 B.C., probably had some 80,000 slaves, nearly one *quarter* of the population of some 340,000. So high civilisation in this region was a conditional and precarious affair.

In the 2nd millennium B.C., immigrants from all over the Aegean, formed the brilliant territory of Attica. But, the literate civilisation of Minoan Crete, based on a trading network from Sicily, to Egypt, and the

Levant, suddenly collapsed in 1460-1450 B.C. (dated from Egyptian records), and the island was taken over by Mycenaean Greeks from the Greek mainland. This collapse was hastened by a colossal volcanic eruption on the nearby island of Thera, probably in 1520 B.C. But there were anyway signs of serious population crisis: a settlement density only matched in Roman and modern times, exhaustion of timber (before the eruption), famine, epidemics, war between the Minoan cities, human sacrifice, and cannibalism.

The Mycenaean took over the trade network, but their population grew and trade was disturbed by the population crisis in the dry belt. The combined result was a catastrophic population crisis in the Aegean, leading to a Dark Age from 1100 to 800 B.C. Literacy was lost, and towns disappeared. The important Mycenaean town of Athens shrank to a tiny hamlet of a dozen people.

In the 8th century B.C., thanks to improved arable farming, population increased dramatically (**Figure 1**), and Greek civilisation re-appeared, with literacy (using a new script), towns, sophisticated art and architecture (**Table 3**). The islands and the plains isolated by the mountains gave rise to some 200 city-states, each a city with a surrounding farmland territory, such as Athens and its population soon outstripped resources, leading to land disputes, rural unrest, food shortages, and epidemics.

The problem was eased in the 7th and 6th centuries (B.C.), by the planting of colonies all over the Black Sea and Mediterranean, until the emigration was blocked by the rival colonising movement of the Phoenicians (which had started earlier), especially the Empire of their greatest colony, Carthage, in (modern) Tunisia. Meanwhile, some city-states, notably Aegina, Chios, Corinth, and

Athens, began to export manufactures to and import goods from Egypt.

In the early 5th century B.C., a monopoly of silver enabled Athens to control grain imports from the Ukraine and Egypt, so becoming the *entropot* of Greece. The temporary large surplus of resources and over-population produced the greatest concentration of creative activity in world history. But the increase in the free population and the mass importing of slaves, led to a desperate Food supply problem (**Figure 2**). Competition for grain import sources with Corinth (in a similar position), resulted in 431 B.C., in the first and worst of the major wars between the over-populated Greek city-states that occupied 53 out of the following 85 years, with recurrent inflation, unemployment, food shortages, and epidemics. In the late 4th century B.C., thanks to the development of alluvial gold, the Macedonians dominated Greece, weakened by over-population, and suppressed democracy.

Alexander the Great used a combined Macedonian-Greek army to conquer Egypt and Western Asia as far as India. On his death, his dry belt Empire, broke up into a number of typically hydraulic states, the Hellenistic monarchies, with bureaucracies staffed by Greeks. The Aegean mainland and islands lost their monopoly of manufactures, as industry developed elsewhere, and their populations shrank back to the reduced carrying capacity of their lands, with deforestation and exhausted mines. A survey of Aegean Greek settlement in the 1970's, covering 14.4% of the country, showed ancient population, at ITS peak in 431 B.C., was *higher* than the modern figure of 6 *million* (excluding the swollen modern city of Athens). By the 1st century B.C., it had probably shrunk to less than 2 *million*, and (except for areas of Roman settlement, **Figure 3**), it probably shrank still further in Roman and Byzantine times (cf. **Figure 2**). When visited by Chateaubriand in 1806, Greece was still a desolate land, and as late as 1826 may have been below 1 million.

In the 3rd century B.C., the Hellenistic monarchies in the dry belt enjoyed some relief from population

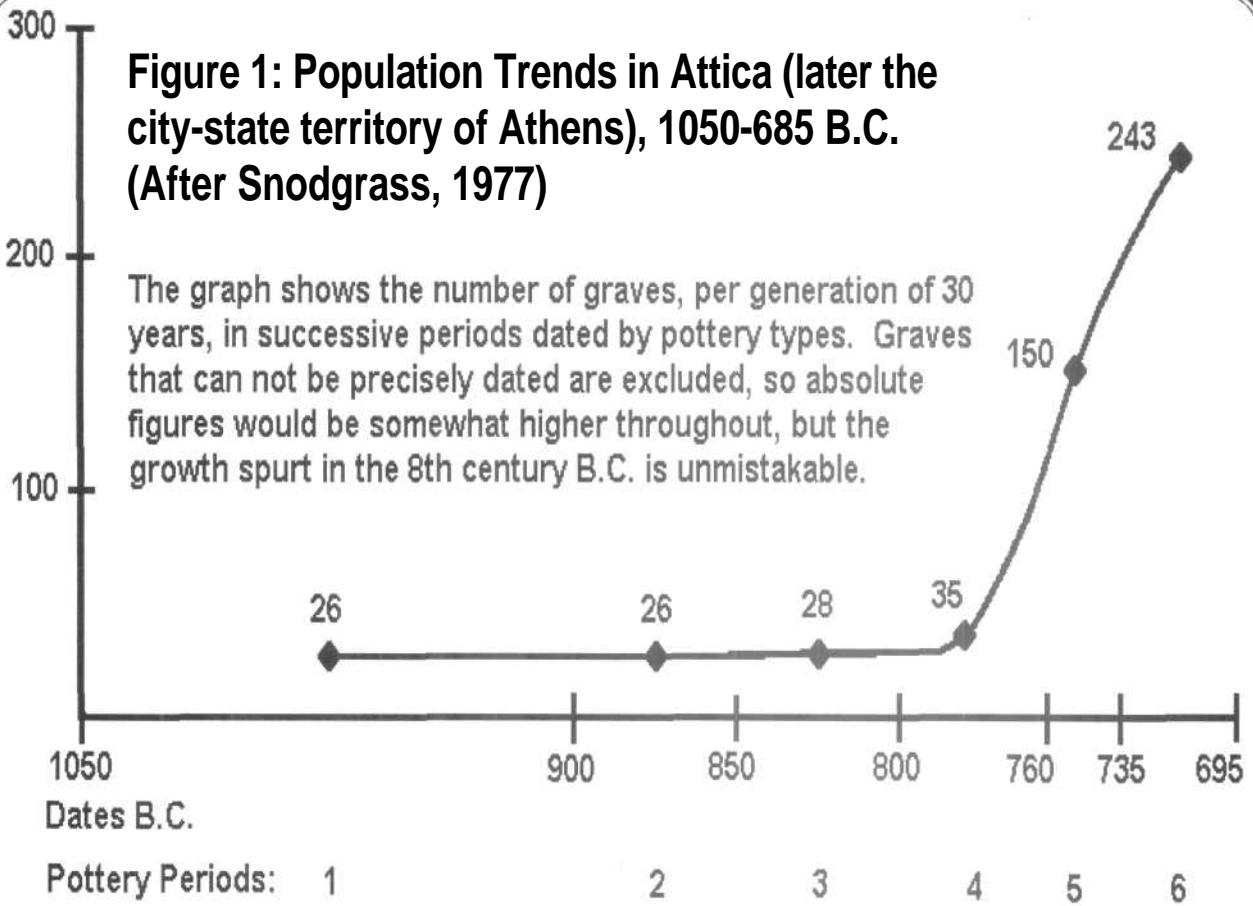
pressure, with scientific advances, brilliant art, and more humane and less destructive warfare. But Carthage suffered from severe stress culture, the heritage of chronic over-population in its Phoenician homeland; it was culturally backward, with a frequency of human sacrifice unparalleled in the Old World. In the 2nd and 1st centuries B.C., rising populations produced a devastating crisis in the Hellenistic kingdoms. Hence Carthage was conquered in the 3rd century B.C., and the Hellenistic kingdoms in the 2nd and 1st centuries, by the new power of Rome. c8

**Figure 2** shows probable changes in the total population of the city-state of Athens in its territory of Attica, that is citizens, resident aliens (including freed slaves), and slaves. There was only one census of adult male citizens, in the late 4th century B.C., but military and naval records and other kinds of evidence are available for these (rounded estimates. As shown in the figure, the territory of Attica could regularly feed no more than 70,000 people.

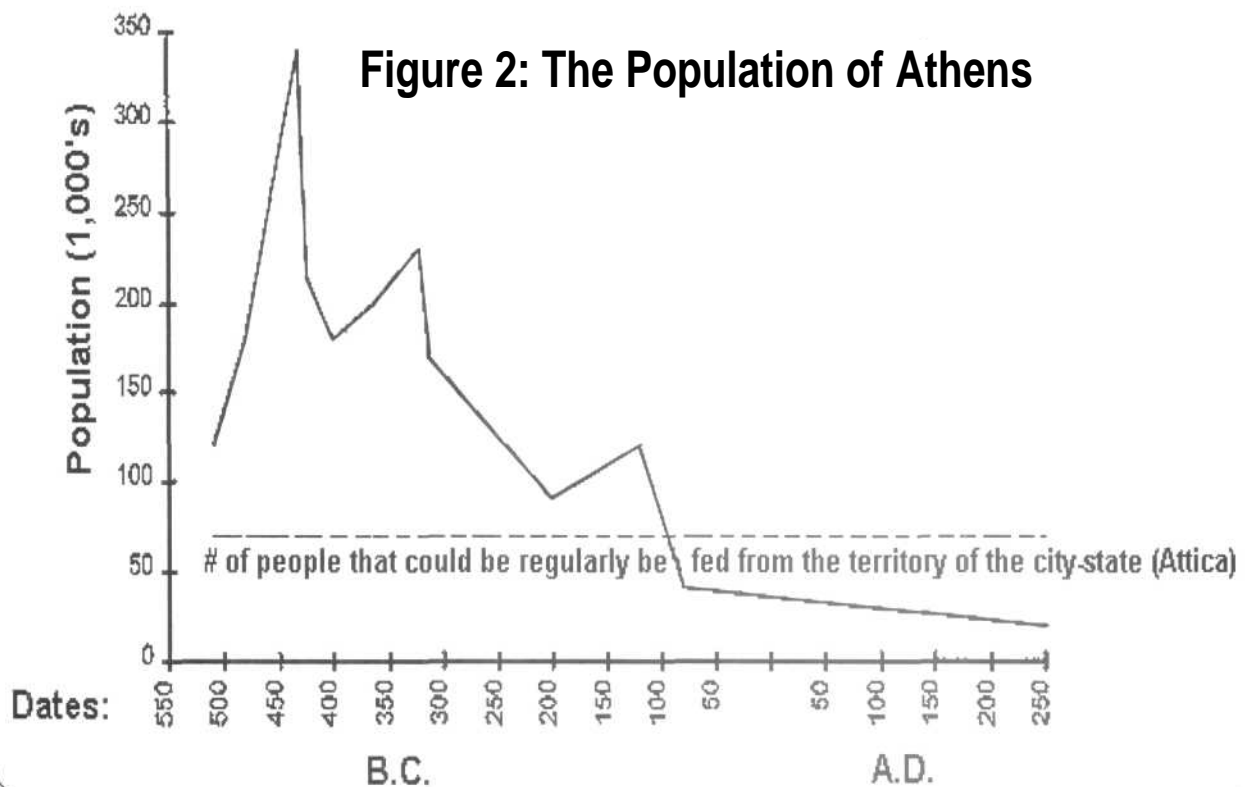
The population crisis in the 5th century B.C., ended with a disastrous defeat for Athens, and a big drop in population which was caused by war casualties and the serious epidemics of 430 B.C. and 427-426 B.C. After a short relief period (while the crisis continued in the other parts of Greece), the population growth resumed, and a further crisis ended in Macedonian domination. After 323 B.C., Athens lost control of the Aegean grain trade and could no longer afford massive grain imports. After 167 B.C., when the Romans overcame the Macedonians, Athens recovered control of the grain trade, but over-population soon caused a final crisis that ended in the sack of the city by the Romans in 86 B.C. Thereafter the population remained very small until modern times; by this time the carrying capacity of Attica had been reduced by deforestation.



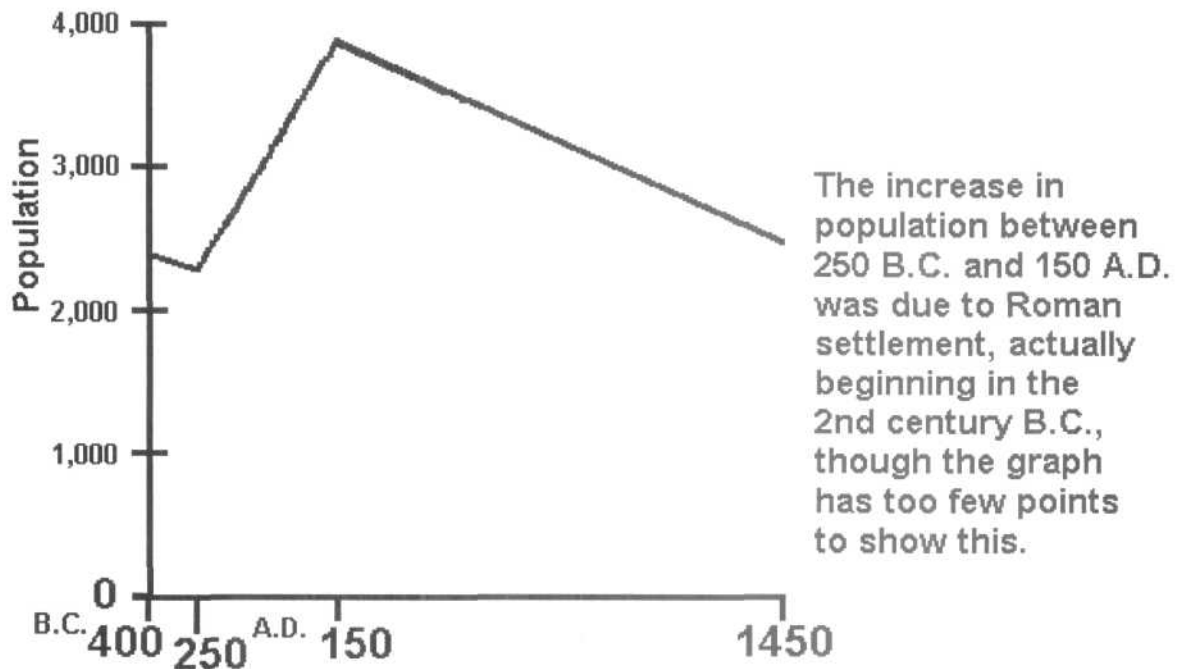
**Figure 1: Population Trends in Attica (later the city-state territory of Athens), 1050-685 B.C. (After Snodgrass, 1977)**



**Figure 2: The Population of Athens**



**Figure 3: Population Trends in the Greek Island of Melos, 400 B.C. - 1450 A.D., derived from the Archaeological Survey of Settlements (After Greene, 1986)**



**Table 1: Grain Imports into the Northern Mediterranean:**

This table shows how utterly the ancient civilisations of the Northern Mediterranean depended on food imports from outside the region, chiefly from North Africa and Western Asia.

The imports of Rome and Constantinople were collected as *taxes* from the provinces of the Empire.

Unless otherwise specified, each table or figure is based on a number of sources.

Importing City	Period or (in one case) Year	Amount of Grain Tons per Year	Region of Origin in (modern names)
Athens	mid-4th century B.C.	40,000	Ukraine, Egypt, Syria, Sicily, Cyprus
41 Greek Cities	328-327 B.C.	48,000	Libya
Rome	1st century B.C. - 1st century A.D.	300,000	Tunisia, Algeria, Egypt
Rome	early 3rd century A.D.	180,000	Tunisia, Algeria,
Egypt Rome	mid-4th century A.D.	67,000	Tunisia, Algeria
Constantinople	mid-6th century A.D.	240,000	Egypt

**Table 2: The Westward Diffusion of Slavery from Western Asia**

(Data from Beloch, 1886)

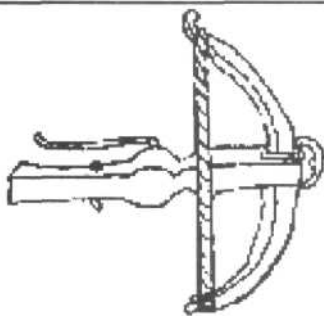
This table shows how slavery diffused into the Northern Mediterranean from Asia Minor (modern Turkey). The centuries are those in which slavery became well established in the regions mentioned, with substantial numbers of slaves.

Centuries B.C.	Regions with Slavery Established
7th	Cities and islands on the coast of Asia Minor, especially Chios
6th 4 <sup>th</sup>	Central Greece, especially Corinth, Aegina, Megara, Athens
2nd	Remainder of Greece  Rome and Roman Italy

**Table 3: The Growth of Greek Civilisation, 700 B.C. to 500 B.C.**  
(Data from Staff, 1977)

This table show the dramatic increase during the period in the production of works of art and architecture. Some of the 7th century temples were very small, and some of the 6th century temples among the largest ever built by the Greeks. The figure of 88 is a minimum.

Centuries B.C.	Stone Temples Built	Large Statues Made
7th	39	66
6th	88	301



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# ARTICLE:

by Russell Gardner, Jr.

## ***Renaissance Times for Evolutionary and Sociophysiological Medicine***

In a 6 week period I traveled to Texas A&M University, College Station, Texas, and the Renaissance Inn of Westchester County, New York (just north of New York City). A first meeting featured "The Evolution of the Psyche" and the second was the twice yearly meeting of the Group for the Advancement of Psychiatry (GAP) where I'm a member of the Committee on Research whose chairman is currently Zebulon Taintor from New York University, a passionate advocate of psychiatric rehabilitation.

GAP which is now 50 years old, emerged post-World War II through the efforts of Will Menninger whose energy and post-war zeal led the initial members to provide a counter to the then staid and conservative APA. Menninger's son and grandson show their continuing involvement with leadership roles. GAP impacts the world often through the enormously read columns of Dear Abby and Ann Landers, the twin columnists whose psychiatric authority often features GAP members; Abby spoke at GAP'S April, 1996, celebration of its 50th birthday as an organization.

ASCAPIan and Jungian analyst David Rosen hosted and mostly arranged the Texas A&M meeting. Notable speakers included David Buss at the University of Texas, newly arrived there from the University of Michigan, Allen Lloyd from The Houston-Galveston Psychoanalytic Institute, and from England, Anthony Stevens and John Price who joined a number of students and faculty from Texas A&M who collectively did a splendid job.

Since then, I have reflected a number of times how David's low key enthusiastic audienceship to his students and peers has splendidly redounded to everyone's advantage. The proceedings will be published by Greenwood Publishers, after editing by David, myself, and Michael Luebbert (one of the

impressive Texas A&M students who contributed the very last paper - on his documentation that the invention of writing has had considerable impact on how we think).

Anthony and John brought along some copies of their just-published book, *Evolutionary Psychiatry*. Lamentably, too few arrived from the publishers to supply the wants of the conference attendees, the book being so hot off the press. I was the lucky recipient of an autographed copy, however, so, gratefully, I can review it below. But to give its timeliness some added background, let me return to GAP where some extraordinary things occurred.

A statement leapt at me from the first page of the pre-meeting materials that the Committee on Child Research was working on a book-length manuscript entitled (would you believe?), *Evolutionary Psychiatry*. Later I learned that David Mrazek of the National Children's Hospital and Chairman of Psychiatry at George Washington Medical Center in Washington, D.C., and Peter Jensen, Director of the Child Psychiatry Research Branch of the NIH, were co-chairpersons of the Committee which has many other prominent child psychiatrists on it. I have known David Mrazek for years but had no idea of his interest on this topic.

David told me later that they spearheaded an effort on which the committee had worked on extensively all day. This stemmed from a sense of pervasive dissatisfaction with nosology and diagnosis in child psychiatry; a solution the group felt, would be to frame five kinds of disorders in an evolutionary framework, suggesting that the behavior patterns seemed to solve adaptive problems. Behavior patterns of interest included depression-anxiety, oppositional-defiant behavior disorders, attention

deficit and the autism-Asperger spectrum (I don't recall the fifth and wish now I'd have taken notes; perhaps it was on learning and memory?!). One of this GAP committee, John McDermott from Hawaii, is the just-former editor of their premier journal, *Journal of the Academy of Child and Adolescent Psychiatry*. David mentioned that in Dr. McDermott's valedictory address to the Academy, he heralded this approach as "The new paradigm in Child Psychiatry."

At the GAP reception, we (myself and Suzie) encountered and greatly enjoyed another senior psychiatrist colleague, the urbane, informed, charmingly skeptical Sidney Werkman also from Washington, D.C., who stated that while he is intensely interested in a basic science of psychiatry, he considered from a review of some 1994 issues of *The ASCAP Newsletter* that our efforts are premature, that too much of an immense gap yawns between what is needed and what we so far know. His fundamental view of behavioral research holds it to be not science, but only disciplined subjectivity. Science seemed in his view to involve only the hard data of cellular-molecular research.

I had known of his high interest and understanding of basic plans because he had previously sent (later it turned out inadvertently) notes and illustrations of the vertebrate movement patterns embedded in the spinal cord. Despite a polite though dim view of our endeavors so far, he then talked of his knowledge of the ventral tegmentum as a dopamine-mediated reward center with great versatility for reinforcing the various things that we like to do, from eating to sex to other things (I nominate leadership behaviors of course).

He mentioned Deborah Tannen's recent talk at the NIH concerning sexual differences. Her book on this topic has been well received and seen as novel, although, of course, on another level, everybody knows her findings already. Videotapes of 5-year old boys revealed them to out-compete each other with vigor whereas counterpart little girls showed prevailing inclinations to relate and support each other, including other little boys. I realized that the views

of Sidney were very familiar from my own education several decades ago; he and my teachers then remain entrenched in a curious and paralyzing mind-set perhaps stemming from a sense of gap where in fact there is none: there is no mind-body split although terms such as psyche and mind ultimately imply lack of body. This was a trouble I had with the very title of "Evolution of the Psyche" but David Rosen and the rest of us at that meeting focused without question on how that body evolved.

Returning to the GAP research committee, the one on which I serve, the work we did which most related to ASCAP concerns featured discussions with Vassilis Kaliatsos, a colleague originally from Greece who was brought into GAP by the Ginsburg fellowship (which pays the way of about 20 outstanding residents-in-training). He has yet to complete his psychiatry residency at Shepherd Pratt in Maryland, because at Johns Hopkins University he mostly does cell-biology bench science of a kind that Sidney Werkman would accede to being indeed worthy of the name. Vassilis studies an animal model of nerve growth factor in Alzheimers Disease and has achieved major grant support. Obviously, he has an interest in integration because he hasn't limited himself to cell biology only, — his work as a physician is highly meaningful to him. He told me he had approvingly read the chapter I had authored in the 6th edition of *The Comprehensive Textbook of Psychiatry* (Eds. H. Kaplan and B. Sadock).

We ate lunch together in the traditional large common room and were there joined by retired psychoanalyst Robert Dorn of Sacramento, California, whose interest in leadership had brought the two of us together several years ago in the same large room. He wished to have answered some questions previously discussed. We three then had an animated conversation in which, I believe, we agreed about the following:

Psychiatry now has no basic science in the medical pattern through which the diseases represent dysfunctional variations of normal body processes;

but such pathophysiological mechanisms should be sought as a primary aim of our work;

cellular-molecular processes are variations at another conceptual level of the activities of the whole organism notably and powerfully including its social arrangements which we agreed are largely mediated in the brain;

there are emergent properties at the higher levels of organization that can't be predicted by full and complete knowledge of the "lower" level although reductionistic attempts to explain phenomena partially in this way is a powerful conceptual and scientific endeavor;

top-down and bottom-up approaches to investigating such pathology refer to integrative approaches that contrast to the top-up avenue that considers only behavior and the bottom-down avenue that considers only cellular-molecular activities;

conceptualizing basic plans that are putatively foundation to both pathology and normality is a highly useful exercise;

that is, pathology is highlighted when the behavioral state in question is deployed at the wrong time and wrong place and normality is highlighted if it works to enhance an individual's adaptation;

this distinctly differs from the often inadvertent "pathologizing" of normal behavior; thus, to talk of a leader as manic or hypomanic when describing his/her elated, animated, energetic and sleepless ways is erroneous in that the basic plan involved is then undercut and under-emphasized; leaders are not pathological unless there is something disadvantageous and maladaptive about the way that the behavior is expressed.

Additionally, Vassilis and I discovered at the lunch a common enthusiasm for the work of English author-scientist, C.U.M. Smith, whose textbook

*Molecular Neurobiology* was the mainstay of a course on neurobiology Vassilis formerly taught at Hopkins. I had recently discovered the second edition published only this year when browsing in our local medical bookstore and was already planning to review it in this essay even before this lunchtime discussion. Vassilis had admired the clearheadedness and readability of the first edition and was pleased to hear of the enhanced evolutionary thrust of the second one.

We returned to our committee meeting and proceeded further on the work already been underway for the previous three years. The current committee chairman and intellectual leader noted his intentions to lead a timely completion of the present project and suggested that Vassilis and I assume core leadership of a subsequent iteration of the committee which could involve a focus on "pathophysiology".

This meeting of GAP represented a dream come true: good debate, sympathetic colleagues, renaissance of interest in a fully integrative approach, and the discovery from a most unexpected angle that evolutionary psychiatry had another duty to serve: rationalizing child psychiatry and rendering it more scientific!

I had told David Mrazek their title had been anticipated. Let me now tell about that anticipation and put it into a context fleshed out by C.U.M. Smith's *Molecular Neurobiology*. Taken together these two very new books splendidly represent the range of data that must be mastered for the next millennium's understanding of scientific psychiatry, as well as for its basic science. They together make 1996 a very good year for this goal.

Fleshing out the lunchtime conclusions above, I mean by a basic science that one can understand pathology of the vascular, gastrointestinal, urinary and neural systems because the consequences of ill health happen when tubes are blocked or broken, or circuits are interrupted. Like a detective, a physician figures out what is happening and possesses a logic in providing a remedy. Physiology,

anatomy and biochemistry are basic sciences that explain disease conditions. Matters are not fully worked out, and some diseases resist the investigative effort, but the framework of understanding is there. Reading both of these books will launch one into the exercise though neither have the foregoing as their primary endeavor.

Stevens and Price describe behavioral and group levels of understanding with knowledge and appreciation of evolutionary history; they may imply but don't actually state their acknowledgment that cellular-molecular understandings underlie implicitly and explicitly all that is presently said and will develop further in the future. They especially focus on evolutionary or ultimate causes of behavior, though in the case of homosexuality, they review hypothalamic and other findings that have captivated scientific and lay attention.

Smith on the other hand discusses evolution on a molecular level almost exclusively — stating in his preface that it "now forms a major and recurring theme." In this second edition of the book, he now has made evolution more clearly his focus. He talks specifically of "molecular evolution" and provides two chapters on the genetics and epigenetics of the brain address how this might work.

Both books, despite intimidating subject matters, are highly readable. Smith's preface states, "Molecular neurobiology is not written in tablets of stone, a fossilized unchanging body of facts. It is a living, developing subject. I have, accordingly, sought to show something of the excitement of the chase." He does. Not in this book but elsewhere, he has also spoken of the integrative requirement of psychiatry's basic science. He has been one of the few who understands the necessity for top-down and bottom-up linkages.

Twenty chapters occupy the six parts of *Evolutionary Psychiatry*. The authors amply convey respect for Carl Jung's awareness that biological history is a core ingredient in the determination of thinking and behavior. "Archetype" repeatedly recurs in their exposition. This is a "dynamic unit of the phyloge-

netic psyche", not, they emphasize, the collective unconscious which they specifically disavow. "Archetypes are conceived as neuropsychic units which evolved through natural selection and which are responsible for determining the behavioural characteristics as well as the affective and cognitive experiences typical of human beings." (p 6)

They also review the triune brain of MacLean and recognize Glantz and Pearce's contribution with respect to power of the ancestral environment in determining present day behavior. But especially do they accord respect to John Bowlby, whose basic principle of psychopathology they adopt and which reads as follows, "Psychopathology results when the environment fails, either partially or totally, to meet one (or more) archetypal need(s) in the developing individual."

The six parts, then, involve an introduction (summarized in part above), disorders of attachment and rank (affective, personality, obsessional, anxiety/ phobic and eating disorders), borderline states, spacing disorders (schizophrenia and the schizoid disorders), reproductive disorders (reproductive issue, homosexuality and sadomasochism), and dreams, treatment and the future. They summarize dreams adequately and interestingly.

Old ground long covered in *The ASCAP Newsletter* takes center stage at times, especially formulations of depression as the exhibition of a communicational state of yielding. Although mania is mentioned, its possible evolutionary origins curiously are not! A distinct contribution of the book, however, and one of which the authors justifiably are proud, involves an explanation of homosexuality and sadomasochism as fusions of social rank and sexual strivings.

Examination of the proximate detail provided by C.U.M. Smith contrasts with the functional overviews of Stevens and Price. But the books resemble each other in that a thorough understanding of the subject matter is conveyed with a minimum of fuss to the reader. For instance in the first chapter providing an introductory orientation, Smith tells us how the 'wiring' of the cortex remains one of the most difficult

areas in neuroscience. A strong tradition of cortical modules (a localization metaphor) has had to contend with the metaphor of hologram, which holds that information is indeed held in localities but is also "smeared throughout."

Smith notes that "Neocortex appears to be a mosaic of... columns which.... vary little in diameter throughout the mammals, from mouse to monkey." This interesting point makes the reader wonder about the essential differences in the genome: the same computing equipment prevails in the various species, but the reason that there is so little difference between the genome of the other large primates and humans is that there may be a simple command made about the number of modules that get turned out. In humans more of the same perhaps makes a major difference in how we conduct our lives.

Smith's book has twenty chapters too, longer and denser ones, as they convey information used by neuroscience students in their courses. I hate to say it to those of us who haven't studied this before, and who may feel phobic about biochemistry, that the future of our field will involve such knowledge. Thus, I believe that one must in the future know of informational macromolecules, information processes in and between cells with the increasing detail that exists on how exactly these work: membranes, channels through these cellular membranes, G-proteins operating on the underside of these membranes, interactions between the cytoplasm and the nucleus, and molecular evolution. And much, much more.

Let me give you a flavor of Smith's teaching. He notes, for instance, that "cells, like brains, are deeply involved in information processing. In contrast to the brain, however, most (though by no means all) of the information available to the cell is hereditary information. It has been accumulated over two or three billion years of trial and error interaction with the environment. By far the greatest amount is stored in the base sequences of DNA" (p 46).

Bearing on the importance of his endeavors for our

work, he suggests, "Francis Bacon said, long ago, that knowledge is power. The recent vast increase of our understanding of molecular biology is beginning to give us the power to manipulate living processes." (p. 91) With respect to depression, which he described in a common-sense manner uninformed by the insights of Stevens and Price, and without an explicitly expressed need for a psychiatric basic science, he notes that "Too exclusive a concentration on upward causation, from molecule to man, leads to the absurdity of extreme 'reductionism': that we are nothing but molecules." (p. 252) His caution is laudable but his straightforward but unsophisticated view of psychiatry means that much integrative work has yet to be done. Fortunately, Stevens and Price have begun that task.

In the last chapter, Smith also alludes briefly to Fragile X syndrome (FraX) (and a number of others, Huntington's, Parkinson's and Alzheimer's diseases are instances). He notes that FraX is common (1/2000 births) and that it not only involves mental retardation, but autism. This provides a linkage to the Stevens-Price book where they stress 'spacing' disorders. FraX seems to stem from a gene that generally produces a protein involved in learning.

Interestingly, Randi Hagermann, a pediatrician from Denver, international expert on FraX is coming in March to UTMB to discuss these psychiatric attributes and to discuss a possible collaboration. It turns out in mild forms of the disorder, social phobia and schizotypy, are the first signs of the genetic alteration so that the survey of a population at risk with an inexpensive measure (a methodological problem in itself) would be of high interest.

In summary then, two meetings and two books signal new beginnings for psychiatry and its basic science. New frameworks are necessary, new data needs gathering, many blanks are yet to be filled in. Evolutionary history and sociophysiological integration stand together at the threshold, tentatively but inevitably, of the exciting things to be learned in the next millennium. c8



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# ABSTRACTS & EXTRACTS...

**Pain responses, anxiety and aggression in mice deficient in pre-proenkephalin.**

**Role of posterior parietal cortex in the recalibration of visually guided reaching.**

**Shared neural control of attentional shifts and eye movements.**

**Differentiation of adult hippocampus-derived progenitors in olfactory neurons *in vivo*.**

**A mechanism for generation of long-range synchronous fast**

**Konig, M., Zimmer A.M., Steiner, H., Holmes, P.V., Crawley, J. N., Browstein, M.J., & Zimmer A.: Pain responses, anxiety and aggression in mice deficient in pre-proenkephalin. *Nature*, 1996;383:535-538**

**Abstract:** Enkephalins are endogenous opioid peptides that are derived from a pre-proenkephalin precursor protein. They are thought to be vital in regulating many physiological functions, including pain perception and analgesia, responses to stress, aggression and dominance. Here we have used a genetic approach to study the role of the mammalian opioid system. We disrupted the pre-proenkephalin gene using homologous recombination in embryonic stem cells to generate enkephalin-deficient mice. Mutant *enk-1* animals are healthy, fertile, and care for their offspring, but display significant behavioural abnormalities. Mice with the *enk-1* genotype are more anxious and

males display increased offensive aggressiveness. Mutant animals show marked differences from controls in supraspinal, but not in spinal, responses to painful stimuli. Unexpectedly, *enk-1* mice exhibit normal stress-induced analgesia. Our results show that enkephalins modulate responses to painful stimuli. Thus, genetic factors may contribute significantly to the experience of pain.

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**Clower, D.M., Hoffman, J.M., Votaw, J.R., Faber, T.L., Woods, R.P., & Alexander, G.E.: Role of posterior parietal cortex in the recalibration of visually guided reaching. *Nature*, 1996;383:618-621**

**Abstract:** Visually guided reaching requires complex neural transformations to link visual and proprioceptive inputs with appropriate motor outputs. Despite the complexity of these transformations, hand-eye coordination in humans is remarkably flexible, as demonstrated by the ease with which reaching can be adapted to distortions in visual feedback. If subjects attempt to reach to visual targets while wearing displacing prisms, they initially misreach in the direction of visual displacement. Given feedback about their reaching errors, however, they quickly adapt to the visual distortion. This is shown by the gradual resumption of accurate reaching while the prisms remain in place, and by the immediate onset of reaching errors in the opposite direction after the prisms have been removed. Despite an abundance of psychophysical data on adaptation to prisms, the functional localization of this form of sensorimotor adaptation is uncertain. Here we use positron tomography (PET) to localize changes in regional cerebral blood flow (rCBF) in subjects who performed a prism-adaptation task as well as a task that controlled for the sensory, motor, and cognitive conditions of the adaptation experiment. Difference images that reflected the net effects of the adaptation process

showed selective activation of posterior parietal cortex contralateral to the reaching limb.

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**Kustov, A., & Robinson, D.L.: Shared neural control of attentional shifts and eye movements. *Nature*, 1996;384:74-77**

**Abstract:** We are able to move visual attention away from the direction of gaze, fixating on one object while attending to something else at a different location, within the region of peripheral vision. It has been widely assumed that the attentional neural systems are separate from the motor systems, but some studies challenge this idea. It has not been suggested that the attentional system is part of the premotor processing in the brain. This model proposes that attentional processes evolved as part of the motor systems, with isolated attentional shifts representing an artificial separation of a natural linkage. Here we test how attentional shifts might be linked to the preparations for making saccadic eye movements. We studied the superior colliculus in monkeys as they shifted their attention during different tasks, and found that each attentional shift is associated with eye-movement preparation.

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**Suhonen, J.O., Peterson, D.A., Ray, J., & Gage, F.H.: Differentiation of adult hippocampus-derived progenitors in olfactory neurons *in vivo*. *Nature*, 1996;383:624-627**

**Abstract:** Neurogenesis continues throughout adulthood in discrete regions. Proliferative zones include the subependymal zone, from where progenitors migrate along the rostral migratory pathway to differentiate into neurons in the olfactory bulb, and the hippocampal subgranular zone, where they migrate and differentiate into granule neurons. Progenitors isolated from the adult subependymal zone exhibit *in vitro* neurogenesis when stimulated with epidermal or fibroblast growth factor. Cultured

adult rat hippocampal progenitors (AHPs) grafted to adult rat hippocampus show site-specific neuronal differentiation. Here we investigate determinants of multipotentiality in the adult central nervous system, by grafting AHPs into homotypic (hippocampus) or heterotypic (the rostral migratory pathway) neuro-genic sites, showed neuronal differentiation. Furthermore, AHPs grafted in the rostral migratory pathway migrated into the olfactory bulb, differentiating into tyrosine-hydroxylase-positive neurons, a non-hippocampus phenotype. These results reveal that AHP populations can respond to persistent neuronal differentiation cues in the adult central nervous system.

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**Traub, R.D., Whittington, M.A., Stanford, I.M., & Jeffreys, J.G.R.: A mechanism for generation of long-range synchronous fast oscillations in the cortex. *Nature*, 1996;383:624-627**

**Abstract:** Synchronous neuronal oscillations in the 30-70 Hz range, known as gamma oscillations, occur in the cortex of many species. This synchronization can occur over large distances, and in some cases over multiple cortical area and in both hemispheres; it has been proposed to underlie the binding of several features into a single perceptual entity. The mechanism by which coherent oscillations are generated remains unclear, because they often show zero or near-zero phase lags over long distances, whereas much greater phase lags would be expected from the slow speed of axonal conduction. We have previously shown that interneuron networks alone can generate gamma oscillations; here we propose a simple model to explain how an interconnected chain of such networks can generate coherent oscillations. The model incorporates known properties of excitatory pyramidal cells and inhibitory interneurons; it predicts that when excitation of interneurons reaches a level sufficient to induce pairs of spikes in rapid succession (spike doublets), the network will generate gamma oscillations that are synchronized on a millisecond timescale from one end of the chain to the other.

We show that in rat hippocampal slices interneurons do indeed fire spike doublets under conditions in which gamma oscillations are synchronized over several millimetres, whereas they fire single spikes

under other conditions. Thus, known properties of neurons and local synaptic circuits can account for tightly synchronized oscillations in large neuronal ensembles.

## AS CITED BY .....

### Cover page

- <sup>1</sup> Divac, I.: Monotremunculi and brain evolution. *TINS* 1995;18:2-4.
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### **Mismatch Theory4: The four-fold model and varieties of depression... page 6**

- <sup>1</sup> Rosenhan, D., Seligman, M.: *Abnormal Psychology*. New York: Norton, 1995, p. 352.
- <sup>2</sup> Herrnstein, R.: Doing what comes naturally. *American Psychologist*. 1977;32;1013-1016.
- <sup>3</sup> Fox, S.: *The Emergence of Life*. New York: Basic Books, 1988.
- <sup>4</sup> Gardner, R.: The Bhagavad Gita and the frontal lobes. *The ASCAP Newsletter*, 1996,9(3):9-16.
- <sup>5</sup> Eaton, B.: *Old genes, new ways, and health: Reproduction*. HBES paper, Santa Barbara, CA, June 1995.
- <sup>6</sup> Nesse, R., Williams, G.: *Why We Get Sick*. New York: Random House, 1994.
- <sup>7</sup> Lappe, M.: *Evolutionary Medicine*. San Francisco: Sierra Club, 1994.
- <sup>8</sup> Stenhouse, D.: *The Evolution of Intelligence*. New York: Harper & Row, 1973.
- <sup>9</sup> Andreason, N.C.: Mania and creativity. In R.H. Belmaker & H.M. van Praag (Eds.), *Mania: An Evolving Concept*. New York: Spectrum, 1980.
- <sup>10</sup> Rosen, D.: Transforming depression. *The ASCAP Newsletter*, 1996, 9(9):3-4.
- <sup>11</sup> Smith, A.L. & Weissman, M.M.: Epidemiology. In E.S. Paykel (Ed.), *Handbook of Affective Disorders*. New York: Guilford, 1991.
- <sup>12</sup> Gilbert, P.: Depression: *The Evolution of Powerlessness*. New York: Guilford, 1992.
- <sup>13</sup> Sackheim, H.A. & Wegner, A.Z.: Attributional patterns in depression and euthymia. *Arch. Gen. Psychiat.* 1986;43;553-560.
- <sup>14</sup> Bailey, K.: The sociopath: Cheater or warrior hawk? *Behav. & Brain Sci.* 1995;18;542-543.
- 

### **Personality Traits and the EEA ... page 12**

- <sup>1</sup> Davies, M., Culture and the Era of Evolutionary Adaptation, *The ASCAP Newsletter*, 1996; 9(9):15-16.
- <sup>2</sup> Briggs Myers, I., *Introduction to Type*, Palo Alto, California, Consulting Psychologists Press, Inc., 1980.
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