

ASCAP

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"What got people out of the trees was something besides thumbs and gadgets. What did 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 Morton¹

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Across-Species Comparisons and
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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 psychopathologically related states. We are interested in the integration of various methods of study ranging from cellular processes to individuals in groups.

The ASCAP Newsletter 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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The WWW Address for the The ASCAP Home Page is:

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World Psychiatric Association, Psychotherapy Section Home Page is:

<http://www.psychiatry.ubc.ca/WPA/psychother.htm>



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

ADDRESSED TO & FROM ...

WPA News

The Scientific Committee of the 11th World Congress of Psychiatry has accepted the two Symposia submitted by the Chairman, and the list of speakers and their abstracts follow below. A Symposium on "Attachment Behavior" has been submitted by the Secretary, and we hope to publish the details of that next month.

We hope that as many ASCAPIANS as possible will join us in Hamburg. Anyone who wants to submit a paper should send in an abstract by 31 December 1998. If you are planning to come, please let us know, as we may be arranging some activities before or after the Congress. Further details from:

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Editor's Note: Abstracts for these WPA Symposia appear on page 19 of this newsletter.

Report on Healing the Moral Animal

"Healing the Moral Animal: Lessons from Evolution," was

taught as part of the 19th Cape Cod Institute, itself sponsored by Albert Einstein Medical College, Bronx, NY. Speakers were:

1. Robert Wright, author of *The Moral Animal: The New Science of Evolutionary Psychology*.
2. Russell Gardner, M.D., Harry K Davis Professor of Psychiatry, University of Texas Medical Branch at Galveston, TX, on "The Moral Animal is a Gregarious, Friendly, Storytelling Animal" and "The Need for a Basic Science of Psychiatry".
3. John Pearce, M.D., on "Our Evolved Natures." Pearce is co-author of *Exiles from Eden*, the first book on evolutionary psychotherapy.
4. Jim Brody, Ph.D., on "Behavior Genetics: Here Comes Granddad Again" and on "Clinical Sociobiology: Evolutionary Psychotherapy".
5. Frank Sulloway, author of *Born to Rebel: Birth Order, Family Dynamics, and Creative Lives*, made a guest appearance with us on Monday, July 20th. Dr. Sulloway is currently with the Institute for Advanced Study in the Behavioral

Sciences at Stanford University.

The students, a mix of psychiatrists, psychologists, social workers, and counselors, were noteworthy for reasons beyond their interest in clinical applications of evolutionary models. The course offered 15 CME units, earned by participation from 9:00 a.m. to 12:15 p.m., but one-third of them remained for extended sessions from a little after noon until 2:00 p.m. on four of the days; also one-third attended evening seminars that ran from 7:00 p.m. until about 9:30 p.m. They attended all of this, despite all the distractions of Cape Cod in the summer!

The evening seminars, unusual for the Institute, were devoted to: "Genes: Conversationalists or Conductors?" led by John Fentress, Ph.D. an ethologist and neuroscientist from Dalhousie University; "Evolutionary Psychology and the DSM" led by Dylan Evans, Ph.D. candidate at the London School of Economics; and "Complexity Theory and Diagnostics: Getting the Client to Maybe" by Jim Brody.

The course manual (375 pages) is now under consideration for publication; notes from the seminars will be available soon and distributed by E-Mail or posted on the Internet.* Please

send me an E-Mail at:
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you wish a copy sent directly to
you.

* At: **www.behavior.net/
mhn/bolforum/message/27**, a
forum that is part of *Behavior
OnLine* and devoted to the topic
of evolutionary psychology and
clinical application.

Note also that *Behavior OnLine*,
a collection of more than a
dozen forums on clinical
psychology and psychiatry was
recently selected "*Website of
the Month*" for March 1998 by
the *APA Monitor*, the first site so
designated. The *APA Monitor* is
an official publication of the
American Psychological
Association. It was also
selected as one of 11 web sites
included in "*Who's Who on the
Web*" by Home Office Comput-
ing magazine, and in addition,
was selected as a "*Recom-
mended Website*" by Encyclo-
pedia Britannica.

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Concerning the Relationship of Dominance, Aggressiveness, and Reproductive Competence

A few years ago research
students and I conducted a
string of Anolis (lizard) experi-
ments:

Background: In nature, the
seasonal resurgence of sex
hormones in males and their
reappearance in the landscape
generally precedes that of
females. Males compete and
possibly fight with each other for
control of prime territories,
exchanging dramatic postures
and displays.

By the time females emerge
from winter sanctuary, male
territories are set up and they
court females with a distinctive
head-nodding display (that
resembles a human unable to
nod "yes" too quickly or too
often). They approach with a
unique "strut," females respond
with one or two head-nods and
then dash away.

Over the days and weeks, the
distance between the approach-
ing male and the "coy" female
lessens and finally there is none
(the "coyness" seems to
correlate with the level of a sex
hormone in the females, the
secretion of which is acceler-
ated by the male courtship
activities). In short — there is
rarely "aggression" against
females.

Observation: In our pilot
experiments, when males with
depressed androgen (social
subordinates) found themselves
one morning in a cage without
their accustomed dominant,
there was an apparent resur-
gence in androgen over the next
couple of days. Although they
would not court females soon
after finding themselves domi-

nants by default, they would
within 36-72 hours. Now an
interesting point: The earliest
responses to females (low
androgen) were aggressive
(fierce displays and biting
attacks), but in the later — true
courtship responses — males
expressed their proper rapid
head-nod courtship display.
Simplistic lesson: real males
(high androgen) treat ladies
properly.

These findings were reported at
the 1990 Annual Meeting of the
American Society of Zoologists;
abstract published with co-
author S. Lumsden in *The
American Zoologist*, volume 30,
number 4.

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faculty/greenberg.html](http://www.bio.utk.edu/ecology/faculty/greenberg.html)

Avoidance Behavior Blossomed in Primates

Murray Sidman designed a
reinforcement schedule on
which there are no overt warn-
ings for an impending shock.
Instead, shocks occur every 5 or
10 seconds if there is no
response; a response delays
the next shock for 20 or 30
seconds. It is logically pos-
sible to avoid all shock by
hitting the key every 19 sec-
onds.

Still, lab rats often receive 10-20
shocks every 30-60 minutes
and in every session that they

perform. Rhesus monkeys get 3; some rhesus get only 3 in a lifetime. Avoidance behavior, particularly when there is no external signal, blossomed in primates.

Barkley and Bronowski likely would see this as an example of "sensing the future" because a successful response tactic relies on internalized cues about the passage of time. The local newspaper is not even aware of Sidman or the rhesus but uses the phenomenon to sell papers; the television station follows monkey wisdom, "If it bleeds, it leads." (And, if it doesn't lead, then make it bleed!)

Threats are on the top of the front page; good news is hidden inside or is listed in the sale papers. Thus, there should be no great surprise about either popular and scientific preoccupation with the seamier parts of nature. Huxley once remarked to Darwin, "*The indecency of the process is to a certain extent in favour of its probability*".

The operative psychological adaptations are likely the same, whether processing gossip about Liz, Leo, or Bill or attending to the spread of a germ in Africa or in a shipment of hamburger. The aspects of evolutionary theory that have impact are the same ones we see on the cover of the *National Enquirer*. Nature made nearly all of us to be great worriers about sex and aggression.

You need to be detached and a bit skeptical in order to notice the powerful, crystalline lattice of altruism that permeates self-interest.

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Niche change theory in action

I harangued Paul Watson & Paul Andrews at HBES and ASCAP with the message that individual differences in life experiences had to be factored into their niche theory. Perhaps the best way is to handle that as variations in the *valuing* of resources. I suppose there must be variations in the way other individual animals value their resources.

Humans, based on their temperament, early rearing experiences, and education, vary enormously. So what people will tolerate, how they value what they have, will vary. Also, we know that genetic factors affect thresholds for depression.

A good book: *Becoming Attached* by Robert Karen (now an Oxford paperback). This is an old-New Yorker magazine type story about the history and peoples who advanced attachment theory. It seems to me that the future will be an integration of attachment theory, temperament theory, and the various evolved mechanisms.

You especially will find *Becoming Attached* useful as a description of the professional turf of developmental psychology. Knowledge about the natural history of attachment is particularly relevant to psychotherapy of difficult relationships.

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WordPerfect, Microsoft Word or ASCII format preferred. Diskettes will be returned to you.

ARTICLE:

by

David A. Evans **"SINCE FEELING IS FIRST": EVOLUTION AND**

LITERATURE

One day when I was 14 or 15 years old, I was looking at some of my father's books and came across Clarence Day's, *This Simian World*, with an epigraph by a man named Barbellion. I was so fascinated by the quote that much of it stayed with me, and when I became interested in Darwin in college, I went to the library and found Day's book. This is the quote:

"How I hate the man who speaks of the 'brute creation,' with an ugly emphasis on 'brute'. As for me, I am proud of my close kinship with other animals. I take a jealous pride in my simian ancestry. I like to think that I was once a magnificent hairy fellow swinging in the trees, and that my body has come down through geologic time via sea slugs and worms and Amphioxus, Fish, Dinosaurs and Apes. Who would exchange these for the pallid couple in the Garden of Eden?"

My college biology professor in my freshman year confirmed for me the strange and wonderful substance of Barbellion's words, not by any direct comments but by his passion for biology. He had a saying on his office desk that read, in bold letters: "DON'T BOTHER ME WITH FACTS; MY MIND IS MADE UP." The facts had to do with the evolution — over millions of years — of all living things, including human beings. Though this quiet, articulate man was teaching in a Lutheran college with a Lutheran name supported by mostly Lutheran alumni, he wasn't about to suffer fools on the evolution question. Not only did he teach biology with passion; he was gifted with the ability to make difficult concepts simple for his students, mainly through metaphor. Once in a lab class he admonished us to be careful with the slip covers we placed over our fragile, one-celled specimens. "How would you like it," he said, "if somebody dropped a 10,000 pound plate glass window on you?" That single utterance was enough to give me

the perspective I needed for handling microscopes.

I might have been a biologist if I had had a background in the hard sciences when I got to college. In any case, since then I've learned a lot about human nature from the evolutionary biologists. My strong conviction as a writer and a teacher of literature is that what I've learned from them is dramatized in the works of the good and great literary artists, past and present.

Here is what Joseph Carroll, in his monumental book, *Evolution and Literary Theory*, says about literature and evolution:

"If literary authors operate within the range of constraints imposed by an evolved human psychology—as they clearly must—evolutionary study can help us to understand both the situations depicted in literature and the personal and social conditions in which literature is produced."²

I agree. I also believe that, as the French novelist Stendahl said: "A novel is a mirror carried along a road." John Price recently sent me a quote from Sigmund Freud:

"..but creative writers are valuable allies and their evidence is to be prized highly, for they are apt to know a whole host of things between heaven and earth of which our philosophy has not yet let us dream. In their knowledge of the mind they are far in advance of us everyday people, for they draw upon sources which we have not yet opened up for science."³

It seems to me that we all need to work toward creating an atmosphere in which biology and literature and art can freely complement each other,

in which we can practice what E. O. Wilson calls consilience.

I make the same assumption that Joseph Carroll makes: that if knowledge is biological, and literature is knowledge, then literature is biological. Carroll thinks of literature as a kind of *"cognitive mapping"*, which, I take it, means that poems and stories are ways in which authors locate themselves in the world and in the cosmos. And so I want to mention a few examples of how literature — mostly poetry — and the findings of evolutionary biology can converge. I will begin with two lines by Robinson Jeffers, an American poet who was born a couple of decades after Darwin's famous book was published, and died in 1962. The lines come from a poem called *The Bloody Sire*, which has to do with Jeffers' notion that violence and human progress cannot be separated:

*"What but the wolf's tooth whittled so fine
the fleet limbs of the antelope."**

In just two lines, the poet captures, for me anyway, something of the essence of natural selection.

The next lines are from a poem by Carl Sandburg, another American poet, who was born in 1878 and died in 1967. The lines are from *Wilderness*, a catalogue poem in which he lists all the animals inside him, his "zoo" as he calls it. Here is the opening stanza and then one more stanza:

*"There is a wolf in me...
fangs pointed for tearing gashes...
a red tongue for raw meat...
and the hot lapping of blood —
I keep this wolf because
the wilderness gave it to me
and the wilderness will not let it go..."*

*"There is a fish in me ...
I know I came from salt-blue watergates...
I scurried with shoals of herring...
I blew waterspouts with porpoises...
before land was...
before the water went down..."*

*before Noah ...
before the first chapter of Genesis.⁵*

In these lines, Sandburg shows his awareness of the evolutionary epic; in this case, a human being is acknowledging his own evolutionary history, and also the notion that the wilderness (nature) cannot cancel out that history.

Now I want to discuss a few lines of *Mending Wall*, a poem by Robert Frost, a contemporary of Jeffers and Sandburg. This is a poem about two New England farmers and how they go about setting the stone wall between them in the spring, a wall that needs to be fixed because the boulders and rocks have been heaved up by the coldness of winter. In this poem, Frost, who read Darwin and thought a lot about his theory, is very much aware of the competition underlying evolution.

There are two points of view in conflict here, represented by each farmer: one man is conservative and prefers to keep fences between himself and his neighbors; the other man — the narrator who initiated the mending — is more open to cooperation, to questioning the notion of why we need walls and fences in the first place. Even though we see competition between two persons here, we see also a sense of cooperation and understanding, which seems to mitigate against the stubbornness of the more conservative farmer. In fact, the cooperation side comes out in just as "natural" a way as the competitive side. The poem's well-known opening dramatizes this powerfully:

*"Something there is that doesn't love a wall,
That sends the frozen groundswell under it
And spills the upper boulders in the sun
And makes gaps even two can pass abreast.."⁶*

After studying chimpanzees and bonobos for many decades, Frans de Waal concluded in a recent book called *Good Natured*, that human life is fundamentally dualistic:

*"We soar somewhere between heaven
and earth on a 'good' wing —*

*an acquired sense of ethics and justice — and a 'bad' wing — a deeply rooted egoism."*⁷

Both de Waal and Frost — scientist and poet — dramatize the same truth in a paradox: human aggression and cooperation have the same source. The wall in the poem is dualistic: on the one hand it separates the two men, and on the other hand it brings them together in a ritual of cooperation that reaches back millions of years to the earliest hominids and even before. Another image in the same poem also shows Frost's interest in the evolutionary struggle: darkness. Near the end of the poem the conventional farmer/neighbor is described as carrying stones to the wall *"like an old stone savage armed"*:

*"He moves in darkness as it seems to me,
Not of woods only and the shade of trees."*

Light/dark is not merely a cultural symbol or motif. It perhaps reaches back to a time when our ancestors were hunted in the dark. This anxiety and fear attached to darkness is still very real; even children who grow up in lighted suburbs fear the dark, so, over time, the image of darkness has come to stand for ignorance/fear/coldness, whereas light has come to stand for knowledge/warmth/understanding.

The next example is a piece of prose I wrote about 15 years ago, in a book about growing up in Sioux City, Iowa in the 1940's and 1950's. In this personal essay I discuss, among other things, the hills and bluffs of Sioux City, and the good feelings I got as a boy and still get, as a man, from being able to stand on a high place and see, as the cliché but true expression goes among prairie dwellers, "as far as the eye can see." Here are two paragraphs from the essay, called *The Place of Your Dream*.

"The desire to get up high and the satisfaction we feel when we can take in so much beneath us, must be part of our biological inheritance. Almost all vertebrates, when challenged, inflate to make themselves look bigger and taller. When they lose a fight or argument they deflate and slink around with

*an averted eye, and appease the winners. When they win they strut, tall and confident."*⁸

"We humans are no different, but we turn the biology into words that parallel our actions. We too get our hackles and our hair up. We put lifts in our shoes to appear more formidable, get pumped up for contests, vie for the tallest trophies and the position of top dog. We look up to our parents. We shoulder responsibilities as well as heroes, dead or living. We elevate our gods to the sky, and heavens to aspire up to. We bow and scrape to superiors and overlords, those over us. Or, as high brows, we raise our supercilious eyebrows and snub low brows, inferiors and underlings. We hold summit conferences, build capitols on hills for our chiefs (heads or higher ups), who step up to power or step down out of it. We feel down or downcast or low when things go badly, high or up when they go well."

And then I quote Shakespeare, who expressed the biology in unforgettable poetry:

*"Why, man, he doth bstride the narrow world
Like a Colossus, and we petty men Walk
under his huge legs and peep about To find
ourselves dishonorable graves."*

The next poem I want to discuss is one by Emily Dickinson, a poem about encountering a snake, which has a famous opening: *"A narrow fellow in the grass/occasionally rides..."* This is a poem that I can't take up in my classes without giving my students some quotes by E. O. Wilson, who invented that wonderful phrase, *"the serpentine gestalt"*. Here are two brief quotes from Wilson, who writes out of personal experiences with snakes, just as the speaker of the poem seems to:

"Science and the humanities, biology and culture, are bridged in a dramatic manner by the phenomenon of the serpent."

"The mind is primed to react emotionally to the sight of snakes, not just to fear them but to be aroused and absorbed in their details, to weave stories [and poems] about them."⁹

With these quotes in mind, I ask my students questions such as: What does this poem tell you about yourself, about all humans everywhere on the earth? What do you feel when you read it? What do you see? What do you think? We discuss the poem's individual lines and images, and especially the last two stanzas, which go:

"Several of nature's people I know, and they know me; I feel for them a transport of cordiality;"

"But never met this fellow, Attended or alone, Without a tighter breathing, And zero at the bone."¹⁰

These lines seem to come out of an experience that has nothing to do with words, an experience of pure feeling and affective response to a danger. ("Since feeling is first," wrote the poet e. e. cummings.) And yet the poet found words to approximate the wordless experience. Reading literature from a strictly cultural and/or historical perspective can be useful and enlightening. But reading literature — especially a poem like Dickinson's — from an evolutionary perspective can get us closer to what Henry James called the felt life. I have experienced *"the serpentine gestalt"* myself, as a boy and as a man. One day about 15 years ago, I was jogging on a highway far from town. Suddenly I glimpsed something out of my peripheral vision — some snake-like shape (actually it was a piece of rubber hose), and experienced that wordless, instinctive veering away in a different direction, the quick fear grabbing my loins and throat. *"Zero at the bone"*, as the poet says. I've felt the same thing Emily Dickinson and E. O. Wilson felt. As a mammal, a primate, a hominid, a human being.

Obviously, sex is a huge theme in literature as it is outside of literature. I mentioned Frans de Waal awhile ago. Last year I heard a talk he gave in Tucson at the Human Behavior and Evolution conference. He mentioned the power that female bonobos can exert over males. What he said reminded me of

some lines by Robert Frost in his poem, *"The Pauper Witch of Grafton,"* about a witch who has enjoyed great power over a number of men in her life. But she is old now, and can no longer command as she once did. At one point in the poem she's referring to one of the men she once controlled with her charm and beauty:

"Up where the trees grow short, the mosses tall, I made him gather me wet snowberries On slippery rocks beside a waterfall. I made him do it for me in the dark. And he liked everything I made him do."

And then, a few lines later the witch speaks a line that I believe is about as accurate and concise a definition of depression as I've ever seen:

" You can come down from everything to nothing."

Finally, I will end with two lines by a great poet born in Swansea, Wales in 1914, who recently joined Shakespeare, Milton, Wordsworth and others with a memorial plaque in Westminster Abbey in London. On a train from Cambridge to Manchester, England recently, I was reading a book by George C. Williams called *Plan and Purpose in Nature*. In his last chapter, called *Philosophical Implications*, Williams discusses the human sense of time — as if the present resides between past and present — and then says that physicists consider time to be extremely deterministic — in other words, the future is, in their calculations, *"already there"*. When I read those words I immediately thought of two of my favorite lines from Dylan Thomas, lines that have haunted me, in good ways, for decades. They come from his long, strange poem called *The Ballad of the Long-Legged Bait*

" The oak is felled in the acorn, and the hawk in the egg kills the wren."¹¹

I don't know of a better way of illustrating what Williams said about time, or a better way of illustrating how the behavior of living things are, at least partly, an expression of genes, c8

COMPUTER MODELING OF THE PSYCHOLOGY AND NEUROLOGY OF ADAPTIVE DEPRESSION*

Introduction:

Adaptive depression is a form of emotional intelligence that forcibly redirects attention away from immediate opportunity and threat, and toward diagnostic failure explanation and off-line behavioral rehearsal. Evolution's survival-of-the-fittest strategy for winnowing defective mental algorithms would have long ago eliminated depression, if it were not adaptive in some circumstances. DEP2 (Depression Emulation Program 2) is a computer model of adaptive depression: mild, time-limited depression in reaction to failure, which facilitates learning from similar failures in memory. DEP2 simulates reaction to events triggering depression, and then behaviors occurring during depression.

To a lesser, but, nonetheless, impressive degree, DEP2 also simulates left versus right cerebral hemisphere dichotomies of processing, representation, function, and interaction. DEP2^{1,2} evolved from DEP^{3,4,5} and DEPlanner,^{5,6} implementing a theory of adaptive depression,^{2a} which applies not only to individual humans, but to artificial and collective intelligences, as well.^{7,8,9}

The depression and depression-related phenomena simulated by DEP2 include: (1) global, (2) stable, and (3) internal explanations for failure; increased (4) failure rumination, (5) depressive realism, (6) negative generalization, and (7) cognitive change; decreased (8) motivation, (9) self-esteem, (10) and self efficacy; and left versus right hemispheric (11) serial versus parallel processing, (12) acting versus monitoring function, (13) vulnerable versus diffuse representation, and (14) left-to-right shift of dominance.

Note, when I refer to DEP2's left and right hemispheres, I am relying on an extremely idealized and abstract account of human left and right

cerebral qualities. I do not mean to suggest that the human left and right hemispheres are as radically opposed as DEP2's subsystems, which are only computational caricatures of human left and right hemispheres. But, I am not simply adopting a single left versus right cerebral hemisphere dichotomy, either. My approach is more similar to the use in linguistic theory of multiple, interacting constraints. All the dichotomies — sequential versus parallel processing, acting versus monitoring function, relatively vulnerable versus relatively distributed representation — apply simultaneously to constrain the design of DEP2's architecture.

In a changing environment, intelligent systems must behave, by turns, both quickly and flexibly. But, quick behavior tends to be rigid, while flexible behavior tends to be slow — similar, by analogy, to the difference between compiled and interpreted computer program translation and execution. When quick, rigid, "compiled" behavior fails, sometimes, slow, flexible, "interpreted" behavior takes its place, during which retreat from an adverse environment is strategic, until new, less failure-prone, compiled behaviors are created.

While DEP2 is not a neural network simulation, it is consistent with popular hypotheses about left and right cerebral hemisphere interaction during perception, problem solving, emotion, and depression. Furthermore, an earlier simulation, DEP,^{3,4,5} relied on rudimentary connectionist simulation techniques, and **DEP2** can be "upgraded" through addition of neural network modules. Another caveat: DEP2 is not a simulation of clinical depression. Rather, it is a simulation of an appropriate, mild depression, through exhibiting behavioral analogies to a large number of more extreme behaviors and symptoms associated with clinical depression. But, even though DEP2 does not

simulate clinical depression, theories of maladaptive and abnormal depression need a theory of adaptive and normal depression in the same sense that theories of heart failure need a theory of normal heart function.

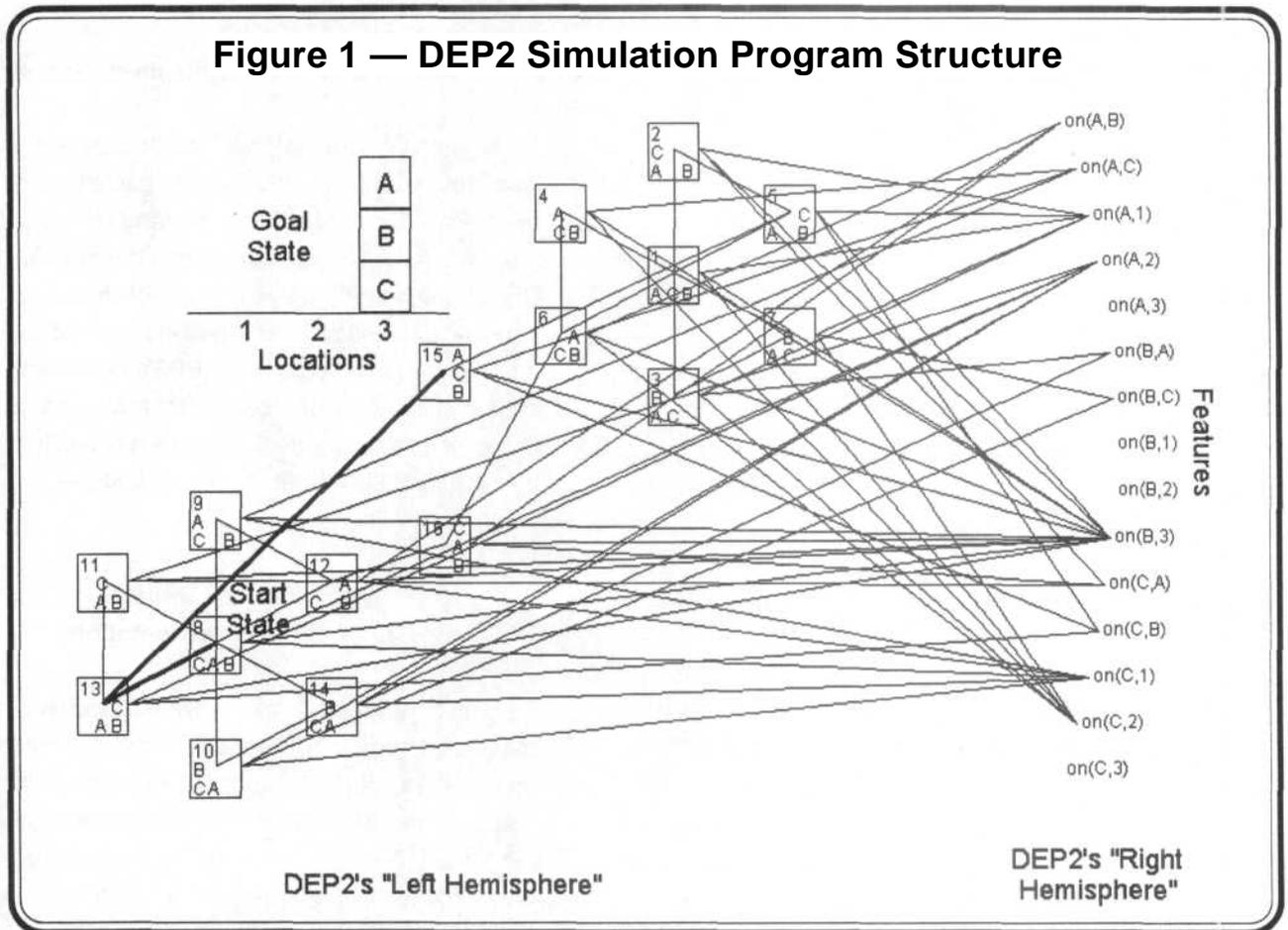
Overview of PEP2 Simulation Program:

DEP2 problem solves, learns, fails, and learns from failure in Blocksworld, a simulated micro-domain popular for prototyping artificial intelligence theories planning and learning. The simulation relies on interactions between two sets of nodes. One set, DEP2's left hemisphere, represents possible states in the Blocksworld micro-domain. For example, state 15 (Figure 1) in DEP2's left hemisphere represents block A on block C on block B located at position 3 on the Blocksworld surface. The other set, DEP2's right hemisphere, represents features associated with each problem state. For example, state 15 has

properties represented by features on (A,C), on (C,B), and on (B,3), which you can see by tracing the lines in Figure 1 from state 15 to its features on the right.

In effect, DEP2 has two different knowledge representations for reasoning about Blocksworld. One representation, corresponding to the left cerebral hemisphere, implements sequential search. The other representation, corresponding to the right cerebral hemisphere, compares problem states to goal states, and failed paths to failed paths, for detection of similarity.

A path through the network of problem states constitutes a sequence of actions, from a potential start state to a potential goal state. When DEP2 is first initialized, it searches between every possible state and goal state, caching the sequences so as to speed subsequent sequential problem solving. Whenever no cached path exists,



the degree of similarity between candidate and state goal state guides search. DEP2's right hemisphere features provide the measure of similarity to guide the search.

Suppose the goal is to reach state 15, but the current world is in state 8. One sequence of actions transforming state 8 into state 15 leads through state 13. State 13 is more similar to goal state 15, sharing two features, on (C,B) and on (B,3), than is state 11, sharing only one feature, on (B,3). During search for a new path from state 8 to goal state 15, state 13 would be favored over state 11, according to a best-first search heuristic. While DEP2's left hemisphere represents possible sequences of action, DEP2's right hemisphere guides search toward goal states.

DEP2 receives a signal from its environment informing it whether a particular sequence of actions succeeds or fails. A path fails whenever it contains a problem state that relies on a disallowed feature. DEP2 is not told which feature is causing failure. It must diagnose the feature, which occasionally changes, from a pattern of failures in memory.

Each time DEP2 fails, it compares recent failures to find which feature or features the failures have in common. Whenever an explanation for past failure is found, all cached paths containing the feature are retrieved and new paths are recreated and re-cached, while avoiding the problematic feature. One version of DEP2,¹ used a spreading activation mechanism to (a) detect similarity, and (b) retrieve implicated paths.

While DEP2 is generating new paths, it withdraws from its environment to avoid penalties for failure. DEP2 accumulates a lifetime performance score in a virtual bank account. Successes deposit; failures withdraw. Some goals have high utility for success, and some have high negative utility for failure. Other goals have smaller utilities and disutilities. By running the DEP2 simulation over and over, while varying independent variables, a user can perform experiments to explore interac-

tion between environment change and depressive reaction in terms of effect on lifetime performance.

Simulating the Neurology of Depression:

Four left-versus-right cerebral hemisphere dichotomies constrain DEP2's construction.

Sequential vs. Parallel Hemispheric Processing

Bradshaw and Nettleton¹⁰ assert that the left hemisphere specializes in sequential behavior while the right operates in a more parallel, pattern-matching mode. DEP2's left hemisphere sequentially searches toward goal states. Its right hemisphere features operate in parallel to compare problem states, guide search, and detect similarity between failures during depressive rumination. By parallel, I mean that, in contrast to DEP2's left hemisphere, successful estimates of similarity among problem states and failed paths do not depend on the order of node processing.

Acting vs. Monitoring Hemispheric Function

Tucker and Williamson¹¹ contend that the left hemisphere tends to act (such as, guide the right hand) while the right hemisphere tends to monitor (such as, recognize faces and their emotions). DEP2's left hemisphere is responsible for sequential behavior directed at its external Blocksworld environment — it acts. In contrast, DEP2's right hemisphere estimates distance, and therefore, progress, toward a goal by calculating degrees of similarity between problem states and goal states — it monitors.

Vulnerable vs. Diffuse Hemispheric Representation

Semmes¹² argues that left hemisphere behaviors are more easily disrupted, because they are more focally represented than in the right hemisphere, and are, therefore, more vulnerable to damage (see also, Springer & Deutsch.^{13, pages 301-303} For example, stroke, more likely, disrupts left rather than right, hemisphere-based cognition.

Each problem state, represented by only one unit in DEP2's left hemisphere, is distributed across three units in DEP2's right hemisphere. Since these right hemisphere units do not participate in online action (only in off-line monitoring and retraining) disabling a right hemisphere unit does not immediately disturb online behavior. In contrast, damage to DEP2's left hemisphere (simulated by randomly disabling a unit) dramatically affects DEP2's ability to generate sequential behaviors — simulating a human-like left side relative susceptibility to damage.

Shifts of Control between Hemispheres

Goldberg, Podell, and Lovell¹⁴ believe that the left hemisphere is responsible for routine behavior, while the right is responsible for novel behavior. Learning a new task activity shifts hemispheric dominance from left to right, and gradually back. During depression, hemispheric dominance shifts right.¹⁵ During DEP2's depression, hemispheric dominance also shifts right, if dominance is construed to be control of processing. Adaptive depression is a kind of learning, and, consistent with this, control of DEP2's processing shifts from left to right as depression deepens, and then back as depression lifts.

Ramachandran et. al.,¹⁶ proposes a theory of communication between left and right hemispheres based on research with brain-damaged patients. Ramachandran argues that the left hemisphere is like a stubborn theorist, while the right monitors inconsistencies, which occasionally build to such a level the left hemisphere is forced to revise its theories (resembling occasional shifts from old to new scientific frameworks in response to accumulating anomalies). DEP2 computer has two subsystems. One subsystem conservatively relies on familiar sequences of behavior. The other subsystem stores failures until they force both systems to work together to find new behaviors that will avoid failure. While Ramachandran did not develop his theory to explain depression, DEP2's subprograms correspond well to Ramachandran's model of left and right hemisphere operation and communication.

Simulating the Psychology of Depression:

Now that I've described DEP2's architecture and its motivations, based on several left versus right hemisphere dichotomies, I will present ten depression phenomena that have analogies in DEP2's behavior.

Global, Stable, and Internal Explanations of Failure

Failure does not trigger depression; its explanation does. Globally important failures, due to stable causes internal to the individual, are more likely to trigger depression.¹⁷

Global Failure explanation: We become more depressed after failure the cause of which also threatens many important goals. Compare losing your job to stubbing your toe. In DEP2's case, some failures affect many high-utility goals while others affect a few goals with only low utility. Only when a sufficient number of high utility goals are threatened might subsequent depression be adaptive.

Stable Failure explanation: We become more depressed after a failure the cause of which is stable and likely to precipitate similar future failures. Compare flunking three tests in a row to flunking a single test. In DEP2's case, the cause of failure must be stable enough to allow DEP2 to find a single, parsimonious explanation for a pattern of recent failures. If the cause of failure changes from failure to failure, DEP2 cannot diagnose it. Therefore, DEP2 does not engage in further failure processing — in effect shrugging it off.

Internal Failure explanation: We become more depressed when the failure is our fault. Compare losing your farm because you forgot to send the mortgage check to losing your farm due to an act of Nature or your ne'er-do-well sibling. For DEP2 to detect an internal failure, the pattern of failure must lead to diagnosis of some assump-

tion about its environment (such as what can be stacked where) that DEP2 can, itself, change.

For example, DEP2 may be able to avoid placing a green block on a blue block (if that causes failure) on the way to achieving the goal of getting a red block on a green block, but it cannot avoid failure if the goal is placing a green block on a blue block, because the goal itself is impossible.

Failure Rumination and the Cognitive Loop

A hallmark of the depression experience is the sequence of failure-related reminders that distract us from usually enjoyable endeavors. Each failure reminds us of the next, in a self-perpetuating and castigating cognitive loop.¹⁸ As noted in Williams, Watts, MacLeod, and Mathews,¹⁹ this may serve the useful purpose of retrieving prior problem solving episodes. Perhaps we reason from them by analogy to our current plight, or compare and contrast past failures to learn. At least, this is the role of DEP2's cognitive loop.

Each time DEP2 detects a failure, it compares the failure to previous failures. In an earlier version of the program,^{3,4,5} a connectionist network was used to store and retrieve similar failures. DEP2 does not use a similar connectionist mechanism, but it does store and retrieve failures, which it compares to detect similarities across failures. For example, if all failure sequences rely on the same feature, such as block A being on block B, then that property is a possible cause for the failures, and reason to avoid placing block A on block B in the future.

Just as in learning from flash cards, where failures are placed in a separate pile until they are gotten right, rumination about failure probably has an important role in focusing attention during learning on those cases which can most accelerate learning. Such strategy is also reminiscent of one method employed to speed up neural network learning: restrict the training set to instances that generate large error signals.

Depressive Realism

During mild depression, people can actually become more accurate processors of information,^{20,21} particularly with regard to task performance and how they are perceived by others. The increase in objectivity seems paradoxical, except when it is considered within a framework that assumes depression can be useful. After all, garbage in, garbage out. Even with the due diligence of rumination's massive outpouring of training cases for diagnosis and rehearsal, unless adequate and unbiased processing capacity is available, it may be all too easy to learn the same wrong lessons again.

DEP2 becomes more realistic during its rumination about failure. When DEP2's world changes and it begins to fail, it does not immediately adjust its assumptions about its world, perpetuating a form of unrealism about its world. But, eventually, accumulating failures finally trigger their own processing, and DEP2 adjusts its assumptions, tracks down threatened goals, and re-achieves them consistent with new assumptions. This is DEP2's analogy to depressive realism or objectivity. Previously ignored errors finally begin to affect knowledge and behavior.

Negative Generalization

To generalize — compare instances and find common properties. If the instances are of failure, which has a negative utility, then this process is negative generalization. True, depressed people negatively generalize.²² But, appropriate negative generalization in the face of real failure is normal and adaptive. DEP2 exhibits negative generalization when it compares failures and isolates the features that failures have in common.

Cognitive Change

Representation of the self changes during depression.²³ Cognitive change is a common element of most forms of psychotherapy.²⁴ In DEP2's non-depressed state, it does not learn, except in the

sense that it adds material to autobiographical memory from which something may be learned later, keyed by the right failure. But, during depression, previously ignored failures begin to drive learning and cause cognitive change that will lead to higher future performance.

DEP2 uses a pattern of failure in autobiographical memory to change its assumptions about what can be stacked where. Then DEP2 uses its new assumptions to retrieve threatened goals and action sequences, to re-achieve threatened goals by generating new action sequences. By changing its assumptions, used to form sequences, as well as the sequences themselves, DEP2 exhibits cognitive change.

Decreased Motivation

Motivation has direction and intensity. Direction is typically a goal to be achieved, such as a full stomach or a deserved promotion. Intensity often reveals itself as speed and persistence. If we repeatedly and quickly achieve the same goal, or, frustrated, persist in attempts to achieve the same goal, then we are said to be highly motivated by that goal.

Depressed people appear to lack motivation.²⁵ They no longer respond with alacrity to external opportunities and threats. The lack of motivation is due to at least two causes within the adaptive depression model. First, the goal of performance is replaced with the goal of learning from failure. Second, the additional overhead of processing inherent to learning, which often relies on repeated attempts to solve the same or similar problems, decreases resources usually available to perform external tasks.

DEP2 simulates this decrease in motivation by simulating a decrease in external goals attended to during normal operation. When DEP2 switches to off-line failure diagnosis and compensatory behavioral rehearsal, it spends less time on achieving external goals, the success of which would normally add to DEP2's lifetime bank account.

Decreased Self-Esteem and Self Efficacy

William James's²⁶ definition of self-esteem is success divided by pretension. Each of DEP2's goals has a utility associated with it. Dividing DEP2's expected utility of achievable goals by expected utility of total goals operationalizes an analog to self-esteem, which declines when DEP2 fails, but re-ascends after successful failure rumination and off-line behavioral rehearsal.

While self-esteem seems sensitive to goal utility (achieving just one really important goal seems to offset failure to achieve many less important goals), self efficacy^{27, 28} seems to abstract away from utility to focus on probability. How likely am I to achieve the task before me (regardless of whether the task is important or not)?

A decline in achievable goals decreases estimated probability that DEP2's next goal will be achieved, operationalizing self efficacy.

Experimental Results: A Qualitative Account:

The DEP2 simulation has four independent variables, based on the presence or absence of four initial conditions:

1. Global versus lack of global explanation for failure,
2. Stable versus lack of stable explanation for failure,
3. Internal versus lack of internal explanation for failure, and
4. Capacity versus lack of capacity for depressive reaction to failure.

Regarding global causes of failure, since some goals are worth more than others, it is easy to prearrange scheduled changes in DEP2's environment that affect or do not affect lots of high utility goals.

Regarding stable causes of failure, this falls out of DEP2's functional design. DEP2 waits until sufficient different failures accumulate so that a single problem state feature explains them. The conservative strategy of waiting until a single, parsimonious explanation for failure is available, confers stability upon resulting explanations. To simulate lack of stability, program the environment to change in ways that defy explanation.

Regarding internal cause of failure, some goals are possible, and some are not. For example, if the goal state, itself, contains a disallowed feature, then failure has an external cause, without possible compensation, because no internally available lever to pull or knob to twist exists, so to speak. To simulate non-internal failure, program DEP2's environment to change in ways for which no such compensation exists.

The first three independent variables — global, stable, and internal failure — together interact with DEP2's capacity for depressive reaction. Only when DEP2's environment changes in global, stable, and internal ways, is depressive reaction useful, in the sense that it leads to greater cumulative lifetime performance. Conversely, depressive reaction to a failure violating one of the global, stable, internal criteria, leads to lesser cumulative lifetime performance.

Discussion:

DEP2 simulates, in effect, an artificial depression. It and its predecessors,^{3,4,5,6} were inspired by Kenneth Colby's computer model of paranoia,²⁹ but, where that model was of a maladaptive cognitive process, DEP2's design is based on the assumption that depression can be a normal and adaptive response to environmental adversity.

Now, the idea of adaptive depression is well represented in the popular literature, such as in Flach's *The Secret Strength of Depression*,³⁰ McGrath's *When Feeling Bad Is Good*,³¹ Nesse and William's *Why We Get Sick*,³² and Goleman's *Emotional Intelligence*.³³ But, this possibly

controversial assessment of depression deserves further context and support.

What I have done is give computational flesh to the idea that "*depression is a normal, adaptive, nonpathological process*",^{34, page 16} in the words of Eric Klinger, who writes, "*Depression is thus a normal part of disengagement that may be either adaptive or maladaptive for the individual but is probably adaptive for the species*".^{34, page 1}

Oatley and Bolton's³⁵ social-cognitive theory of life events postulates that depression is caused by the failure of major social goals, and consequent disruption of important "*well-practiced, complex social plans, the social equivalents of habits* — in James's²⁶ (1892/1961) *sense* — or *well learned skills, whereby a person fulfills major social goals*".^{35, page 377} (In informal simulation, I've addressed social habit-like behaviors, by creating two interacting versions of DEP2, which must search together for new and successful interleaved plans in response to environmental change.) Oatley and Bolton conclude with a hopeful note:

" *The theory presented here identifies depression as the despair of a severe loss or disappointment from which, for a longer or shorter time, there seems to be no escape.... But from this crisis, either by a change in schemata of relating, by a change in circumstance, or by a new role, a renewed sense of self can emerge.*"^{35, page 385}

One way to understand the relationship between adaptive and maladaptive depression is through the lens of evolutionary psychiatry. "Emotions are set to maximize Darwinian fitness, not happiness," Nesse convincingly submits in his "*What Good is Feeling Baa*" article.^{36, page 37} "*Some suffering is part of a vital mechanism shaped by natural selection to help people survive.*"^{36, page 37}

Sloman, Price, Gilbert, and Gardner³⁷ argue, in their aptly titled paper, "*Adaptive Function of Depression: Psychotherapeutic Implications*", that capacity for depressive reaction is necessary for giving up futile social contests, and that under-

standing this evolutionarily-derived purpose can provide clinical therapeutic insight and guidance. They suggest that if patients interpret depression as having evolutionary and adaptive functions, doing so may decrease depression's stigma and lead to better understanding of its triggers in their social environment.

Apply to mild, moderate, and severe depression Bailey's³⁸ three part evolutionary classification of motivational and emotional behavior.

Presently-adaptive behavior, such as mild depression, conserves resources in an adverse environment, enforces reflection, and signals submission. *Once-adaptive* behavior, such as moderate depression, may have benefited the social group by decreasing claims on its resources from incapacitated individuals, but is maladaptive in today's world of medical and retirement insurance. *Never-adaptive* behavior, such as severe depression, may be caused by biochemical defects or brain damage.

How might a computational theory of adaptive depression aid theories of clinical depression? For example: currently, DEP2 is hardwired (though hard coded may be the more apt description) to react to patterns of failure in autobiographical memory. More sophisticated models of adaptive depression should not just learn during depression, they should also learn when and how much to get depressed. Learning when to learn is equivalent to learning when to punish and reward, making relevant to depression the regression-to-the-mean training fallacy.³⁹

Trainers (of pilots in the original analysis) can learn the wrong policies from an observed but misinterpreted pattern: exceptionally bad performance typically improves after punishment, but exceptionally good performance typically decreases after reward. Even though this is due to a natural tendency of a random variable to return toward an average value after an exceptional value, trainers learn a training policy that favors punishment of failure over rewarding of success. Most of the time, people, in effect, must learn to train themselves. Perhaps some fall prey to the regression-to-the-

mean training fallacy, learning a policy of punishing every failure when they should not, and failing to reward success when they should.

DEP2 does not learn when to learn, but if it did have an additional layer of information processing, for learning the rules to allow, or squelch, incipie it depression, then DEP2 might learn the wrong rules, modeling relation of early experience to later vulnerability to maladaptive depression. Such computer modeling will be relevant to beginning 10 model the developmental psychology of depression. While such a layer would not necessarily have to rely on connectionist or neural network learning techniques (there are other potential machine learning algorithms), given the important relationship between artificial and biological neural network mechanisms, such a route may ultimately be the more fruitful.

Conclusion:

Computer models of cognitive disorder do not have to be artificial neural network or connectionist simulations, but, in the long run, I agree they do have to be "upgradable," to more neurally-plausible computational building blocks, if they are to participate in a productive continuum of models, from the symbolic to the subsymbolic, to the neural and biochemical levels of human information processing.

Furthermore, while I find DEP2's simulation of the psychology of adaptive depression to be comprehensive and convincing, its simulation of the neurology of adaptive depression remains only partial and tentative. Not only does it lack neuron-like computing units, but also frontal versus posterior and cortical versus subcortical dimensions as well. However, neither is DEP2 necessarily inconsistent with such units or dimensions.

DEP2 only approximately models depressive behaviors and brain structures. Its design resembles, in philosophy, *animats*, the artificial animals created and studied by artificial life researchers. Storing and releasing change is a

general problem for intelligent adaptive systems, but simpler adaptive systems can sometimes reveal otherwise recondite insights that apply to more complex systems. While DEP2 is a simple system, relative to mind and brain, it coherently accounts for a greater number of disparate phenomena relevant to the psychology and neurology of depression than have been previously explained, within a common information-processing framework. c8

Acknowledgments:

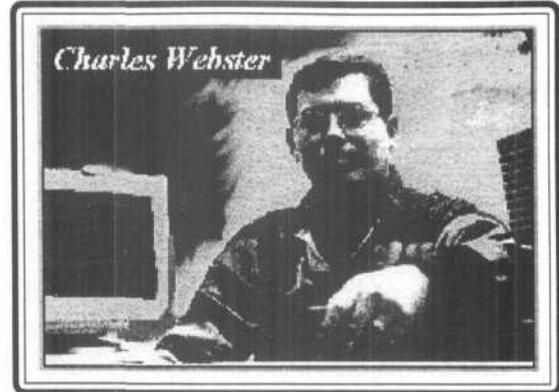
I would like to acknowledge you, the reader, in advance. I hope this paper provokes your comment, which you may direct to:

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Additional abstracts and some full texts relating to adaptive depression may be found at:

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See Dr. Webster's
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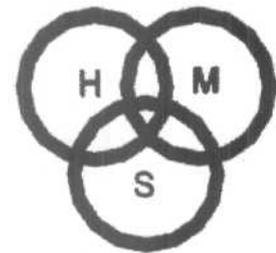
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{The Adaptive Depression Guy}

This WebSite has "many" abstracts by this "author"

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Dr. Webster was formerly a Medical Informatics Fellow at the University of Pittsburgh Medical Center's Department of General Medicine. He has conducted sponsored research at Pitt's Decision Systems Laboratory and at Carnegie Mellon University's Laboratory for Computational Linguistics. Dr. Webster's research interests include cognitive science and the computer-based patient record. Dr. Webster has a B.S. Degree in Accountancy and an M.S. Degree in Industrial Engineering, both from the University Illinois at Urbana/Champaign. He is a graduate of the University of Chicago's Pritzker School of Medicine, and also holds an M.S. Degree in Intelligent Systems from the University of Pittsburgh. At Duquesne University, he teaches Elements of Health Information Science, Health Information Science, Visual Basic and Java, and World Wide Web Programming.

ARTICLE: Accepted Abstracts

by John S. Price

**WORLD PSYCHIATRIC ASSOCIATION
XI World Congress of Psychiatry
Hamburg, Germany - 6-12 August 1999**

Psychotherapy Section Symposium 1:

Threshold to a Biological Psychotherapy

Proposed Participants: John Price (Chair), Piero De Giacomo, (Co-Chair), Marco Bacciagaluppi, Russell Gardner, Jr., & Frank Koerselman.

Symposium Abstract:

Evolutionary biology is a promising basic science for the theory and practice of psychotherapy. Most forms of psychopathology can be seen as adaptive behaviour patterns which may carry costs as well as benefits as a result of evolutionary trade-offs. In some cases, otherwise adaptive behaviours may appear with excessive intensity or duration, or be inappropriately triggered. The manifestation of phylogenetically ancient behaviours represents in some cases the failure of more recently evolved strategies to deal with current problems, in others it represents a mismatch between the ancestral environment in which the behaviours evolved and the conditions of the present day. Analysis along the lines of evolutionary biology can help the psychotherapist to help the patient to replace phylogenetically ancient strategies with rational, conscious, voluntary decisions and behaviours to deal with his or her situation.

References:

Glantz K & Pearce JK: *Exiles from Eden: Psychotherapy from an Evolutionary Perspective*. London, England: W.W. Norton, 1989.

Stevens A & Price J: *Evolutionary Psychiatry: A New Beginning*. London, England: Routledge, 1996.

Clinical Strategies Based on Evolutionary History by Russell Gardner, Jr.

Abstract: Psychiatric practice can be helped by making across-species comparisons (e.g., genomic

programs for behavior in varied species) and contrasts (e.g., genes increasing brain size in humans foster gregariousness and bonding). Treatment strategies and explanations result. Shiver-ATP uses shivering as a model for automatic unthinking reactivity shared with other animals. A = Allies, T = Thought, and P = Planning are uniquely human. Negotiating Until Resentment Abolished (NURA) depicts resentment as a signal that one feels aggressed on by another's defining a situation. Conflict resolution stems from iterative redefining until neither party feels resentment. Euthymia in the Story-Telling Animal In Charge of Itself (EUSTIAC) stems from the observation of heightened serotonin in vervet monkeys and higher status of subordinate vervets treated with an SSRI. Humans contrast to other animals in having metaphorical other people in the form of schedules, possessions, etc. These and other acronyms helpfully formulate illness, therapy and medication.

Reference:

Gardner R: Sociophysiology as the basic science of psychiatry. *Theoretical Medicine*, 1997; 18:335-356.

The Elementary Pragmatic Model: A mental tool to organize psychotherapeutic interventions by Piero De Giacomo

Abstract: The Elementary Pragmatic Model is a tool by which the human mind can be organized to understand human interactions and their changes; it is also used to organize interventions in family and individual psychotherapy, especially short-term and single-session therapies. The model was created over 20 years ago and has been used since then; it has now achieved full maturity of expression and applications in several areas of mental disorders, such as schizophrenia and eating disorders.

Reference:

De Giacomo P: *Finite Systems and Infinite Interactions: The logic of human interaction and its application to psychotherapy*. Connecticut: Bramble Books, 1984.

The Dialectics between Biological and Cultural Evolution by Marco Bacciagaluppi

Abstract: The basic principle of evolutionary psychiatry is that the present environment frustrates innate needs selected in the course of biological evolution. Recent archaeological evidence suggests that in the Upper Palaeolithic and the early Neolithic human communities were peaceful and cooperative, and worshipped a female deity. This gynaeocratic culture was replaced by an androcratic culture through successive invasions by pastoral nomads from marginal areas. It is suggested that when migrations due to population pressure brought the nomads in contact with the food surplus produced by agriculturalists, predation became an advantageous cultural variant, in which superior male strength in turn became advantageous. The predatory androcentric model became dominant. Though apparently similar to animal models, this is really a result of cultural, not biological, evolution. One residue of the original model is the mother-child relationship. The impingement on this relationship of a violent environment is traumatic and produces psychopathology.

Reference:

Fromm E: *The Anatomy of Human Destructive-ness*. New York, New York: Holt, Rinehart, & Winston, 1973.

How to Restore Intentionality by Frank Koerselman

Abstract: A key feature of psychiatric disorder is loss of intentionality. Intentionality refers to the "directedness" of mental functions like thinking, feeling, longing, etc. Patterns of intentionality develop from childhood in a dynamic interchange between individual and real-life experience. This process, however, is guided by biological "values", i.e., by evolutionary selected preferences. The aim of biological psychotherapy is the restoration of disrupted intentionality. Rediscovery of the real goal of feelings and action-tendencies presupposes the identification and integration of motives rooted in "biological" preferences. Similarities and differences

with other psychotherapeutic approaches will be discussed as well as the application to causal treatment and revalidation in psychiatry.

Reference:

Bolton G & Hill J: *Mind, Meaning and Mental Disorder*. Oxford, England: Oxford University Press, 1996.

Psychotherapy Section Symposium 2:

The Psychotherapy of Mood Disorders

Proposed Participants: Russell Gardner, Jr. (Chair), Leon Sloman, Paul Gilbert, Peter Rohde, & John S. Price

Symposium Abstract:

Studies of agonistic interactions by comparative ethologists and the analysis of pairwise contests by behavioural ecologists have recognised a basic strategy set available to competing individuals. The strategies in this set are those of escalation (fight) and de-escalation (flight or submission). These strategies were available to the common human and reptilian ancestor some 300 million years ago, and may be assumed to be still present in man, but overlaid by more recently evolved methods of dealing with social competition and affiliation. We suggest that the remnant of these strategies is manifested in patients with mood disorders, so that the patient with elevated mood has selected an escalating strategy which has been called the Involuntary Dominant Strategy (IDS) and the patient with depressed mood has selected a de-escalating strategy which has been called the Involuntary Subordinate Strategy (ISS). Presenters will discuss the relevance of these ideas for psychotherapy.

References:

Price J, Sloman L, Gardner R, Gilbert P & Rohde P: The social competition hypothesis of depression. *British Journal of Psychiatry*, 1994; 164:309-135.

Sloman L, Price J, Gilbert P & Gardner R: Adaptive function of depression: psychotherapeutic implications. *American Journal of Psychotherapy*, 1994;401-414.

Defeat & Entrapment in Depression by Paul Gilbert

Abstract: There is now much evidence that depressed states are associated with subordinate self-perceptions and unfavourable social comparison (inferior self), submissive behaviour (lack of confident-assertiveness), and social avoidance in conflict situations. These are often referred to as involuntary subordinate strategies. However the original model of Price suggested that it was not so much subordinate strategies that were key to serious depression but defeat. This paper will discuss new data showing that defeat and entrapment are more powerful predictors of depression than subordinate behaviour. Indeed perceptions of being defeated in an environment from which one cannot escape may be a central depressogenic environment (Gilbert and Allan, in press).

Therapy For Prematurely Triggered Subordination by Leon Sloman

Abstract: Whereas Kline and Kramer have put forward formulations of rejection sensitivity in terms of neurotransmitters, rejection sensitivity is also open to psychodynamic interpretation. Here, rejection sensitivity will be attributed to the premature triggering of the Involuntary Subordinate Strategy (ISS), a mechanism that has both psychological and biological dimensions. Premature triggering of the ISS can contribute to being victimised, to rage reactions, to power struggles and to depressive illness. Within this model both medication and psychotherapy can be effective therapeutic tools.

A common goal is to enable individuals to avoid premature triggering of the ISS, which is a useful way to teach self-assertion. Case illustrations will be used to illustrate how this model can be relevant for the purposes of formulation, in pharmacotherapy and in devising strategies of intervention in individual and family psychotherapy.

Reference:

Sloman L; Price JS; Gilbert P; Gardner R: Adaptive function of depression; psychotherapeutic implica-

tions. *American Journal of Psychotherapy*, 1994;148:401-416.

Human Hierarchical and Territorial Behaviour: A Fresh Look by Peter Rohde

Abstract: Hierarchical and territorial behaviour in humans are relatively neglected in the psychiatric literature. In animals it is recognised that competition exists for hierarchical position and territory and that conflicts are resolved by ritualised fighting. The loser signals defeat, usually avoiding serious physical harm. Defeat involves abandoning aspiration and has been proposed as a cause of depression in humans. Some reasons for the neglect of this area of behaviour in humans and the implications for aetiology, diagnosis, and therapy are discussed.

Alternatives to the Involuntary Subordinate Strategy by John S. Price

Abstract: It is one of the functions of evolutionary psychiatry to identify normal, adaptive behaviour patterns which, when distorted, become recognised as psychopathology. When the normal patterns can be identified, therapy may be more informed, and in this way evolutionary psychiatry contributes to the theory and practice of psychotherapy. In this presentation, the normal psychological mechanisms underlying mood changes are discussed, and their function is seen in terms of sexual selection, group homeostasis, relationship formation, agonistic behaviour mechanisms and self-esteem management. In particular, depressive states are seen as intensifications and prolongations of an "Involuntary Subordinate Strategy" which may be replaced by strategies using higher levels of brain function.

Reference:

Stevens A & Price J: *Evolutionary Psychiatry. A New Beginning*. London, England: Routledge, 1996.



ABSTRACTS & EXTRACTS...

Webster C: Medical affective computing: Medical informatics meets affective computing. From a paper to appear in the *Proceedings of the 9th World Congress on Medical Informatics*, August 18-22, 1998, Seoul, South Korea.

Abstract: From advisory systems that understand emotional attitudes toward medical outcomes, to wearable computers that compensate for communication disability, to computer simulations of emotions and their disorders, the research agendas of medical informatics and affective computing — how and why to create computers that detect, convey, and even have emotions — increasingly overlap.

Some psychiatric and neurological researchers state their theories in terms of actual or hypothetical computer programs. Adaptive intelligent systems will increasingly rely on emotions to compensate for their own conflicting goals and limited resources — emotional reactions about which psychiatrists and neurologists have special insights.

DEP2 (go to page 11 of this newsletter to see this diagram) is a computer simulation of adaptive depression — learning from explainable patterns of failure in autobiographical memory — that simulates many depressive behaviors. In the terminology of fault-tolerant computing, adaptive depression involves fault detection (triggered by failure), fault location (strategic retreat and failure diagnosis), and fault recovery (return to on-line operation).

DEP2 relies on subsystems whose structures and behaviors are based on popular hypotheses about left and right brain hemispheric function during depression and emotion. DEP2 and its predecessors DEP and DEPlanner, are relevant to psychiatric and neurological informatics, and to the design of adaptive autonomous robots and software agents.

Perret DI; Lee KJ; Penton-Voak I; Rowland D; Yoshikawa S; Burt DM; Henzi SP; Castles DL & Akamatsu S: Effects of sexual dimorphism on facial attractiveness. *Nature*, 1998;394:884-887

Abstract: Testosterone-dependent secondary sexual characteristics in males may signal immunological competence and are sexually selected for in several species. In humans, oestrogen-dependent characteristics of the female body correlate with health and reproductive fitness and are found attractive. Enhancing the sexual dimorphism of human faces should raise attractiveness by enhancing sex-hormone-related cues to youth and fertility in females, and to dominance and immunocompetence in males.

Here the authors report the results of asking subjects to choose the most attractive faces from continua that enhanced or diminished differences between the average shape of female and male faces. As predicted, subjects preferred feminized to average shapes of a female face. This preference applied across UK and Japanese populations but was stronger for within-population judgements, which indicates that attractiveness cues are learned. Subjects preferred feminized to average or masculinized shapes of a male face. Enhancing masculine facial characteristics increased both perceived dominance and negative attributions (for example, coldness or dishonesty) relevant to relationships and paternal investment. These results indicate a selection pressure that limits sexual dimorphism and encourages neoteny in humans.

Keller L & Ross KG: Selfish genes: A green beard in the red fire ant. *Nature*, 1998;394:573-577.

Abstract: A 'green-beard' gene is defined as a gene that causes a phenotypic effect (such as the

presence of a green beard or any other conspicuous feature), allows the bearer of this feature to recognize it in other individuals, and causes the bearer to behave differently towards other individuals depending on whether or not they possess the feature. Such genes have been proposed on theoretical grounds to be agents mediating both altruism and intragenomic conflicts, but until now few, if any, of these genes have been identified.

Here the authors provide evidence of a green-beard gene in the red imported fire ant, *Solenopsis invicta*. In polygyne (multiple-queen) colonies, all egg-laying queens are Bb heterozygotes at the locus Gp-9. Previous studies suggested that bb females die prematurely from intrinsic causes; the authors now show that BB queens initiating reproduction are killed by workers, and that it is primarily Bb rather than BB workers that are responsible for these executions. This implies that allele Gp-9b is linked to a green-beard allele that preferentially induces workers bearing the allele to kill all queens that do not bear it. Workers appear to distinguish BB from Bb queens on the basis of a transferable odour cue.

Judd LL; Akiskal HS; Maser JD; Zeller PJ; Endicott J; Coryell W; Paulus MP; Kunovac JL; Leon AC; Mueller TI; Rice JA & Keller MB: A prospective 12-year study of subsyndromal and syndromal depressive symptoms in unipolar major depressive disorders. *Archives of General Psychiatry*, 1998;55:694-700

Abstract:

Background: Investigations of unipolar major depressive disorder (MDD) have focused primarily on major depressive episode remission/recovery and relapse/recurrence. This is the first prospective, naturalistic, long-term study of the weekly symptomatic course of MDD.

Methods: The weekly depressive symptoms of 431 patients with MDD seeking treatment at 5 academic centers were divided into 4 levels of severity:

1. Depressive symptoms at the threshold for MDD;
2. Depressive symptoms at the threshold for minor depressive or dysthymic disorder (MinD);
3. Subsyndromal or subthreshold depressive symptoms (SSDs), below the thresholds for MinD and MDD; and
4. No depressive symptoms.

The percentage of weeks at each level, number of changes in symptom level, and medication status were analyzed overall and for 3 subgroups defined by mood disorder history.

Results: Patients were symptomatically ill in 59% of weeks. Symptom levels changed frequently (1.8/y), and 9 of 10 patients spent weeks at 3 or 4 different levels during follow-up. The MinD (27%) and SSD (17%) symptom levels were more common than the MDD (15%) symptom level. Patients with double depression and recurrent depression had more chronic symptoms than patients with their first lifetime major depressive episode (72% and 65%, respectively, vs 46% of follow-up weeks).

Conclusion: The long-term weekly course of unipolar MDD is dominated by prolonged symptomatic chronicity. Combined MinD and SSD level symptoms were about 3 times more common (43%) than MDD level symptoms (15%). The symptomatic course is dynamic and changeable, and MDD, MinD, and SSD symptom levels commonly alternate over time in the same patients as a symptomatic continuum of illness activity of a single clinical disease.



As CITED BY.....

Cover page

- ¹ Morton, Kathryn: The Story-Telling Animal. *The New York Times Book Review*, December 23, 1984, pp 1-2

"Since Feeling is First": Evolution and Literature — page 6

- ¹ Day C: *This Simian World*. New York, New York: Alfred A. Knopf, 1920
- ² Carroll J: *Evolution and Literary Theory*. Columbia, Missouri: University of Missouri Press, 1995.
- ³ Storr A: Psychoanalysis and Creativity. *Freud and the Humanities*, edited by Peregrine Horden. New York, New York: St. Martin's Press, 1985.
- ⁴ Jeffers R: The Bloody Sire. *Robinson Jeffers: Selected Poems*. New York, New York: Alfred A. Knopf, Inc., 1963.
- ⁵ Sandburg, C: Wilderness. *Carl Sandburg: Harvest Poems*. New York, New York: Harcourt, Brace, & World, Inc., 1958.
- ⁶ Frost R: Mending Wall. *The Poetry of Robert Frost*. New York, New York: Henry Holt and Company, 1969.
- ⁷ De Waal F: *Good Natured*. Cambridge, Massachusetts: Harvard University Press, 1996.
- ⁸ Evans D: The Place of Your Dream. *Remembering the Soos*. Marshall, Minnesota: Plains Press, 1986.
- ⁹ Wilson EO: *Biophilia*. Cambridge, Massachusetts: Harvard University Press, 1984.
- ¹⁰ Dickinson E: *The Complete Poems of Emily Dickinson*. New York, New York: Little, Brown and Company, 1957.
- ¹¹ Thomas D: *The Poems of Dylan Thomas*. New York, New York: New Directions Publishing Corporation, 1939.

Computer Modeling of the Psychology and Neurology of Adaptive Depression — page 10

- ¹ Webster C: Computer Modeling of Adaptive Depression and Asymmetric Hemispheric Processing. *Biological Psychiatry*, 1996;39(7):580.
- ^{1a} Second International Workshop on Neural Modeling of Brain & Cognitive Disorders, 4 June 1998 to 6 June 1998, University of Maryland: College Park, Maryland.
- ² Webster C: Medical Affective Computing: Medical informatics meets affective computing. *Proceedings of the 9th World Congress on Medical Informatics*, 1998.
- ^{2a} Webster C: *Adaptive Depression*. Manuscript in preparation, 1998. ³ Webster C; Glass R & Banks G: DEP: A Depression Emulation Program, *Proceedings of the 12th Symposium on Computer Applications in Medical Care*. Los Angeles, California: IEEE Press, 1988a; pages 287-291.
- ⁴ Webster C; Glass R & Banks G: A Computational Model of Reactive Depression. *Proceedings of the 10th Annual Conference of the Cognitive Science Society*. Hillsdale, New Jersey: Lawrence Erlbaum, 1988b; pages 758-764.
- ⁵ Webster C: Computer Modeling of Adaptive Depression, *Behavioral Sciences*, 1995a;40:314-330.
- ⁶ Webster C: Why Intelligent Systems Should Get Depressed Occasionally and Appropriately. *Proceedings of the 14th Annual Conference of the Cognitive Science Society*. Hillsdale, New Jersey: Lawrence Erlbaum, 1992; pages 1-7.
- ⁷ Webster C: Does Organizational Depression Play a Role in Organizational Change? *Proceedings of the 12th Annual International Conference of the Association of Management*. Newport News, Virginia: Maximilian Press, 1994; pages 41-46.
- ⁸ Webster C: Process Cognition and Reengineering Health Care Organizations. *Proceedings of the 1995 Annual Healthcare Information and Management Information Systems Society*. Chicago, Illinois: Healthcare Information and Management Systems Society, 1995b; pages 207-217.
- ⁹ Webster C: Adaptive Depression, Affective Computing, and Intelligent Processing. *Proceedings of the 1997 IEEE International Conference on Intelligent Processing Systems*. Los Angeles, California: IEEE Press, 1997; pages 1181-1184.
- ¹⁰ Bradshaw J & Nettleton N: The Nature of Hemispheric Specialization in Man. *Behavior and Brain Sciences*, 1981:4:51-91.
- ¹¹ Tucker D & Williamson P: Asymmetric Neural Control Systems in Human Self-Regulation. *Psychological Review*, 1984;91(2):185-215.

Computer Modeling of the Psychology and Neurology of Adaptive Depression — page 10
(continued from page 24)

- ¹² Semmes J: Hemispheric Specialization: A Possible Clue to Mechanism. *Neuropsychologia*, 1968;6:11-26.
- ¹³ Springer S & Deutsch G: *Left Brain, Right Brain*. New York, New York: W.H. Freeman, 1993.
- ¹⁴ Goldberg E; Podell K; & Lovell M: Lateralization of Frontal Lobe Functions and Cognitive Novelty. *Journal of Neuropsychiatry*, 6, 4, pages 371-378, 1994.
- ¹⁵ Okada F; Takahashi N & Tokumitsu Y: Dominance of the 'Nondominant' Hemisphere in Depression. *Journal of Affective Disorders*, 37, pages 13-21, 1996.
- ¹⁶ Ramachandran V; Levi L; Stone L; Rogers-Ramachandran D; McKinney R; Stalcup M; Arcilla G; Zweifler R; Schatz A & Flippen A: Illusions of Body Image: What They Reveal about Human Nature. In: R. Llinas and P. Churchland, *The Mind-Brain Continuum*, Cambridge, Massachusetts: MIT Press, 1996.
- ¹⁷ Peterson C & Seligman M: Causal explanations as a Risk Factor for Depression: Theory and evidence. *Psychological Review*, 91, pages 347-374, 1984.
- ¹⁸ Ingram R: Toward an Information-Processing Analysis of Depression. *Cognitive Therapy and Research*, 8, pages 443-478, 1984.
- ¹⁹ Williams J; Watts F; MacLeod C; & Mathews A: *Cognitive Psychology and Emotional Disorders*. New York, New York: John Wiley & Sons, 1988.
- ²⁰ Alloy L & Abramson L: Depressive realism: Four Theoretical Perspectives, in: L. Alloy (editor), *Cognitive Processes in Depression*. New York, New York: Guilford Press, 1988.
- ²¹ Lewinsohn P; Mischel W; Chaplin W; & Barton R: Social competence and depression: The Role of Illusory Self-Perceptions? *Journal of Abnormal Psychology*, 1980;89:203-212.
- ²² Beck A; Rush A; Shaw B; & Emery G: *Cognitive Therapy for Depression: A Treatment Manual*. New York, New York: Guilford Press, 1979.
- ²³ Ross M: Depression, Self-Concept, and Personal Constructs. In: F. Epting & A. Landfield (editors), *Anticipating Personal Construct Psychology*. Lincoln, Nebraska: University of Nebraska Press, 1985.
- ²⁴ Beck A: Cognitive Therapy as the Integrative Therapy: Comments on Alford and Norcross. *Journal of Psychotherapy Integration*, 1991 ;1:191-198.
- ²⁵ Layne C; Merry J; Christian J; & Ginn P: Motivational Deficit in Depression. *Cognitive Therapy and Research*, 1982;6(3):259-274.
- ²⁶ James W; *The Principles of Psychology*. New York, New York: Dover, 1890.
- ²⁷ Bandura A: Self-Efficiency Mechanisms in Human Agency. *American Psychologist*, 37, pages 122-147, 1982.
- ²⁸ Rehm L: Self-Management and Cognitive Processes in Depression. In: L. Alloy (editor), *Cognitive Processes in Depression*. New York, New York: Guilford Press, 1988.
- ²⁹ Colby Kenneth: *Artificial Paranoia*. New York, New York: Pergamon Press, 1975.
- ³⁰ Flach F: *The Secret Strength of Depression*. New York, New York: J.B. Lippincott, 1974.
- ³¹ McGrath E: *When Feeling Bad is Good*. New York, New York: Bantam Books, 1992.
- ³² Nesse R & Williams G: *Why We Get Sick: The New Science of Darwinian Medicine*. Random House, 1994.
- ³³ Goleman D: *Emotional Intelligence*. New York, New York: Bantam Books, 1995.
- ³⁴ Klingler E: Consequences of Commitment to and Disengagement from Incentives. *Psychological Review*, 1975;82(1):1-25.
- ³⁵ Oatley K & Bolton W: A Social-Cognitive Theory of Depression in Reaction of Life Events. *Psychological Review*, 1985;92(3):372-388.
- ³⁶ Nesse R: What Good is Feeling Bad? The Evolutionary Benefits of Psychic Pain. *The Sciences*, November-December 1991.
- ³⁷ Sloman L; Price J; Gilbert P & Gardner R: Adaptive Function of Depression: Psychotherapeutic Implications. *American Journal of Psychotherapy*, 1994;48(3):401-416.
- ³⁸ ³⁸ Bailey K: *Human Paleopsychology: Applications to Aggression and Pathological Processes*, Hillsdale, New Jersey: Lawrence Erlbaum, 1987.
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