For evolutionary psychiatrists, psychologists and others interested in sociophysioiogical integration

ASCAP

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""Evolutionary Theorizing ... requires the utmost stringency in avoiding vague analogies and generalization."

Donald F. Klein [1]

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

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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.

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- ♦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 Annual Meeting Announcement

Don't forget **Annual Meeting on Wednesday, June 4,1997.** Mistakenly in some announcements we have perseverated that our meeting would be prior to the APA meeting as it was in 1996. Wrong, wrong, wrong, we are meeting prior to the HBES meeting in Tucson!! Sorry for any confusion and misunderstandings.

The meeting will take place **8 am to 5 pm in the Marriott University Park Hotel** just next to the university campus (880 East 2nd Street, Tucson Arizona). In the HBES meeting announcements, there were allusions to coming on Wednesday morning but no indications of dorm room availability on Tuesday night. The Marriott number for reservations is: 1-800-228-9290.

The meeting theme will feature "Evolutionary Psychotherapy." More next issue on evolving meeting plans.

Precision needed in Terminology

Re: "Prediction of Antidepressant Activity from Ethological Analysis of Agonistic Behaviour in Rats" by Paul J. Mitchell, *ASCAP Newsletter* 1996; 9(10):9-12.

Mitchell's discussion concerning the rat resident/intruder paradigm highlights problems with drawing ready parallels between behaviors in different species, in this case, rat and man.

The article initially discusses increases in "aggressive behavior" exhibited by vehicle treated resident rats induced by pump implantation. Presumably this "aggressive behavior" was non-social behavior in which the animal was compared with their own baseline while in isolation.

Near the end of the article it is pointed out that the "aggressive" behavior that occurs during the resident/intruder interaction in rats never causes any physical damage and indeed may be heightened social exploration.

There seems to be some slippage here between behavioral changes in isolation labeled aggression and behavior changes in social interaction similarly labeled aggression where neither set of behaviors bears anything but the loosest analogy to the very diverse behaviors labeled aggression in humans.

Similar verbal slippage occurs in the reference to "anti-depressant drugs" since there are many antidepressant drugs and many depressions. It has been clearly shown, for instance, that the monoamine oxidase inhibitors benefit atypical depression far more than the tricyclic antidepres-sants and that neither seem to have much effect upon normal humans.

It also seems inexplicable that the author draws a key distinction between social and aggressive behavior on the basis of which animal initiates the behavior, agrees that this could be handled by slow motion movies, but then casts this crucial distinction aside as slowing down the analysis.

In my double-blind placebo-controlled trial of imipramine on simple phobics, we found no effect on phobia or anxiety, but did find a marked decrease in self reported aggressive behavior. Evolutionary theorizing really requires the utmost stringency in avoiding vague analogies and generalizations.

Donald F. Klein, M.D.
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Lunch with John S. Price

Kathryn, Henry and I had a most enjoyable and constructive lunch with John Price and his wife.

John is keen to publish an extract of our book, in the *ASCAP Newsletter,* on the theory of failsafe. He feels that clinicians will be interested. John will supply a commentary to the extract pointing out similarities between failsafe and the involuntary subordinate strategy (ISS).

We took some persuading by John to agree to a publication of an extract (see page 9 of this issue), largely because the ideas could be taken out of context.

For this reason, I have written an article to introduce it, entitled "The Pathology of Neuroses and the EEA", which broadly covers the same ground but embeds the ideas within general principles (see page 6 of this issue).

As regards the concept of the *scale,* the picture is most encouraging. The scale concept entails that human learned behaviour is, at least in part, an adaptation to climatic variability.

During the past 15 years, Professor Peter Richerson (University of California - Davis) and Professor Robert Boyd (UCLA) (both HBES members) have defined the conditions when learned behaviour would be favoured by evolution by natural selection. The critical aspect is the rate of change of environmental variability, which must be neither too fast nor too slow.

This summer, Dr Richard Potts (Smithsonian Institution, Washington, D.C.), published a short paper in *Science?* summarising his recent book. His key insight is that hominid evolution can be shown, as a matter of fact, to be a response to climatic variability.

Moreover, (a point not made by Potts but picked up by Richerson and Boyd) the rate of change in the climate agrees with the theoretical predictions made by Richerson and Boyd for the evolution of learned behaviour, a truly remarkable result.

If Potts, Richerson and Boyd are correct, the only possible conclusion is that human learned behaviour is, at least in part, a capacity for dealing with climatic variability, a conclusion we had already reached from an entirely different route, namely one based on an analysis of human social change.

Accordingly, climatic variability is the idea which unifies Darwinian theory, hominid evolution and human behaviour. A synthesis of this magnitude cannot, in our view, be a matter of coincidence.

Michael Davies

An Open Letter to ASCAP Members, Re: Comparator Genes

I hate to join in on this discussion about comparator genes because I have not followed it carefully. But it surprised me that Waller and his modelling team are talking so exclusively about the giving up aspect of this system.

If I understand it the hypothesis is that the homo sapiens genome includes a polygenic trait (which I see more on the model of stature or melanin concentration than on the model of sickle cell) which concerns comparisons of the individual with others.

The notion is that other species manifest this comparator capacity largely in terms of dominance-submission systems but that in our species the expression is more complex including aspects of self-image, self-worth, self-esteem and so forth.

Part of my argument would be to urge more thinking about how complex this capacity to compare may become on the human scene. Because we have the capacity to create imaginary individuals—ego ideals, gods, anthropomorphic animals and plants and stars and planets— our self-comparisons with imaginary others may be as important as those with the actual members of our band. It is hard for me to

imagine too that the comparator function is entirely separate from empathy.

If not, one evolutionary effective aspect of its operation would be infant care. When I compare myself with an infant I know it is smaller, more helpless and needs allies. This knowledge can help me render exquisite allyship or alternatively successfully exterminate this other.

Several other questions seem absent in the discussion. Much is said about the giving up aspect of comparisons but it seems also evolutionarily relevant that comparisons may lead us to heroic hanging on at times. (Most people in the band could not make it to the next valley and come back with food but I am faster and stronger and I have a chance.)

Also I am surprised that accuracy of comparisons is seen as such an issue. Given my understanding of the human psyche I don't see 100% accuracy as in the cards for us. Ever.

The variability that I notice in comparison-making is that some humans do it a lot and some do it relatively little (perhaps these last are busy making comparisons with imaginary individuals); it also seems to me that both strategies might have evolutionary advantage in different circumstances, just as shortness wins in some situations and tallness in others.

Jean Goodwin

Evolutionary Psychiatry

What can you be thinking of? In the piece on terminology in Vol 10, number 1, RG suggests that I (and some other evolutionary-minded psychotherapists) might not describe your intervention as evolutionary psychotherapy, on the grounds that it had nothing to do with mismatch or the EEA. Be real, guy.

In *Exiles of Eden,* the notion of mismatch was emphasized because it was new to the field of psychotherapy; to our knowledge, few if any clinicians at the time were attributing any psychological problem to mismatch. But surely you know that John (Pearce) and I emphasized that mismatch was just one idea among many.

Indeed, we have a whole chapter on dominance, which is one of the concepts you use in your intervention. And we made no pretence of covering all the evolutionary concepts that might be helpful in psychotherapy.

Furthermore, I have always maintained that any standard concept that is not incompatible with evolution is *ipso facto* part of evolutionary psychotherapy; if you're not against us, you're with us. It follows that any psychotherapist who tries, however feebly, to see human behavior through the evolutionary lens is an evolutionary psychotherapist.

Let me give you an example. Tim Miller just wrote me about an intervention done by "a famous behaviorist." His client was a woman who complained that although she dated and had sex occasionally, no long-term, satisfying relationships ever came of her adventures. The therapist told her never to drop a man before dating him six times. A year later the client was married. The therapist is probably innocent of evolution, but the intervention is compatible with the female reproductive strategy. In my view, that makes it evolutionary.

So please, be fair. Evolutionary psychotherapy is not limited to one concept or one source of psychopathology. Indeed, if all therapists would study evolution as part of their training, we wouldn't need the evolutionary handle at all. There's enough misunderstanding of our position out there in the profession. *ASCAP* shouldn't add to it.

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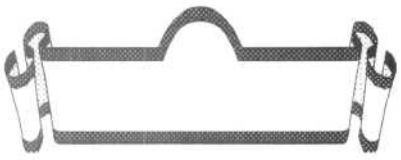

ARTICLE:

by Michael Davies

The Pathology of Neuroses and the EEA

Poor judgement brought about by overwork is dangerous and counterproductive. Among gatherers and hunters, errors and lack of attention from one persistently stressed or tired member could expose the whole group to unacceptable risk. The hypothesis is that involuntary patterns of behaviour act to curb prolonged excessive effort on the part of individuals.

According to geneticists, during the 100,000 years from c. 200,000 years before present (BP) to c. 100,000 years BP, the human population fell from c. 100,000 adults to c. 10.000.[1] During this era, our species was subject to intense environmental constraints, living in times of great hardship as world temperatures fell, at the scarce end of the scale.[2] Humankind is profoundly social. Love of praise and fear of criticism are powerful, sometimes overwhelming motivations. In periods of scarcity, some people might try too hard to fulfil their perceived social obligations.

The mechanism, which evolved by natural selection to protect individuals and groups from the consequences of inordinate pressure, is posited to have two syndromes. A relatively mild form would limit activity without interfering with the capacity to lead a normal life. The advantage of this initial curtailment would be that, while the unsustainable exertions were strongly discouraged, overall output was not seriously diminished. The individuals' anxiety about their physical or mental well-being, on account of worrying physical or mental symptoms, would induce caution and inhibit the assumption of unreasonable burdens.

Nevertheless, the external or self-imposed exigencies might be so compelling that mild symptoms would be insufficient to hold back the zeal of an over-conscientious or over-enthusiastic group member. When the point was reached that survival was threatened, the offender would be incapacitated by a significantly more severe syndrome. The individual would become an invalid and a burden to the group but would cease to be an overt risk.

The first condition broadly corresponds to a mild neurosis. The second syndrome equates to a severe neurosis, a breakdown, which we have called a "failsafe".[3] The term has been taken, by analogy, from engineering where machines are stopped by means of a fail-safe device rather than permitted to exceed their design limits and risk damage. These definitions of neurosis seem to be in fundamental agreement with the observations of Stevens and Price.[4]

If neuroses are adaptive, then, among gatherers and hunters, a gradual recovery could normally be anticipated from each condition. Essentially, self-knowledge would dawn on the sufferer, who would decide that, in the future, only realistic commitments and responsibilities would be pursued. For this reason, in the longer term, individuals would steadily recover and return to normal but with a deeper understanding of their limitations. Any subsequent, over-ambitious effort would bring a temporary recurrence of the neurosis and frighten the subject into abandoning unreasonable objectives.

The hypothesis only demands that mild symptoms restrain while the severe version incapacitates. Their precise character is undetermined. However, both conditions are subject to a critical constraint. The form and content must supply rational grounds for eliciting a meaningful response from both the individual and group. Thus, in the mild version, the sufferwould be worried by an imbalance in their physical or mental well-being. On the other hand,

the failsafe would create an incapacity which would be acceptable to everyone, including the invalid, as sufficient reason for inaction. For this reason, failsafe symptoms would tend to exhibit the known characteristics of a recognized incapacitating illness. Consequently, no moral blame for the passivity could be attributed to those affected, either by themselves or their companions.

Nevertheless, the symptoms of neuroses could be wide ranging. Chance might even play a part, for example some features of a viral illness could be artificially prolonged. In any event, a failsafe would most probably mimic the incapacitating illnesses prevalent within a community, for example recurrent fevers and illnesses inducing lethargy. Since a failsafe would often rescue a person from an intolerable situation, the condition would not always be unwelcome to those afflicted. Their short-term dilemmas would have been resolved.

The adaptive value of these syndromes depends upon individuals making a conscious adjustment to their lifestyle. For this reason, sufferers would remain rational, and personal relationships would be maintained. In a small, closely-knit group, the origin of the disorders would be "obvious" to family, friends and companions. Their over-conscientious colleague had been working too hard, for example, and had, therefore, fallen ill. The consensus would be that the sufferer should relax more and do less. These opinions would be communicated and, with growing self-realisation on the individual's part, be integral to the recovery process.

Two general principles can be proposed as a basis for the pathology of present-day neuroses. First, the further a community diverges from universal gatherer-hunter norms, the greater the stress and, therefore, the higherthe incidence of neuroses. Second, specialized personalities (specialists)[5] would be at greater risk from neurosis, and the more specialized the personality, the greater the risk. The susceptibility of specialists would flow from their onesidedness, especially if their biases were not compensated for by friends and col-leagues.

A key characteristic of specialists is their inflexibility. Thus, if a planner had to cope with a highly variable environment, a mismatch would occur. The level of stress would multiply rapidly, because the most carefully laid plans would soon miscarry. The outcome could be a mild neurosis or even, in extreme circumstances, a failsafe.

Gatherer-hunter and Western industrial societies differ in critical respects. Some of the most significant aspects are specified in **Table 1.**

Table 1

A comparison of gatherer-hunter and Western industrial lifestyles:

Category	Gatherer-hunter	Western Industrial
1. Home life	Extended family	Alone or nuclear family
2. Responsibility	Mainly group	Mostly individual
3. Work	Often enjoyable	Frequently disliked
4. Shared beliefs	Agreed and integrated	Disputed
5. Ceremonies	Regular and collective	Few, sometimes meaningless

Consequently, in view of the discrepancies between Western industrial society and that of gatherers and hunters, the regular occurrence of neuroses in Western industrial societies can be predicted. The social isolation, notably of specialists, commonly met in Western industrial countries, would also exacerbate the condition, since the support and insight of close companions would not contribute to recovery.

Furthermore, in Western industrial society, the voluntary abandonment of excessive undertakings might be unthinkable. The moral, social, financial or legal pressures might impose a wholly unavoidable duty. In these circumstances, the mild neurosis or failsafe might be rendered self-perpetuating, and the sufferer might never fully recover.

A failsafe could be brought about directly and suddenly. Thus, an unanticipated, public humiliation, for example a demotion at work which was an overwhelming threat to feelings of self-worth, could precipitate an almost instant failsafe, possibly, for example, a partial paralysis.

The foregoing analysis illustrates the variety of hypotheses that can be derived if humankind is posited to have the behaviour patterns of our gatherer-hunter forebears. If the relevance of our origins to present-day behaviour were to be more widely recognized, many new vistas would be revealed.

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 derived from first principles. *c8*

ASCAP members who have not received a complimentary copy can apply for their free copy from:

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ARTICLE:

by Michael Davies, Henry Davies, & Kathryn Davies

EXTRACTS from: *"HUMANKIND THE GATHERER-HUNTER'©*

A Failsafe Mechanism:

The obligations assumed by a social animal could be taken too seriously and result in an excessive effort. This endeavour would often be counterproductive and expose companions to needless risks through tiredness. Normally, interference from others would restrain this immoderation, but, should this not succeed, involuntary measures would be invoked to curtail self-inflicted damage. The majority of symptoms would be mild and self-limiting, suggesting and initiating a relaxation in exertions.

However, if these warnings were ignored, eventually a more drastic syndrome would ensue, called a failsafe, whereby the person would become incapacitated, so ending a highly stressful situation. The sufferer would usually begin to reduce commitments and recover spontaneously over a period of time. In like manner, the death of a parent, spouse or friend would lead to a progressive readjustment, so that a normal life could, eventually, be resumed.

Chapter2: Attributes, p.18

... lack of group support, segregation or an absence of social interaction would trigger aberrant behaviour. The precipitating events would be, in the main, an inability to deal with self-imposed or external exigencies in the absence of adequate assistance. These pressures would be even more burdensome without guidance, help and sympathy. Some examples are strains associated with upbringing, personal relationships, work, duty and life's events. In this study, which is no more than a preliminary, theoretical introduction, only specific, highly selective instances, which are in no sense exhaustive, are given to illustrate each of the preceding.

Chapter 20: Troubled Behaviour, p.261

In the preceding illustrations, the symptoms were relatively mild, allowing a superficially normal routine. However, the individual was made aware that something was amiss, perhaps prompting a re-evaluation of priorities and a reduction in the workload. The external pressures, however, might have been so compelling, as in wartime, or the internal motivation so imperative, that the undertakings imposed or accepted would, if unchecked, culminate in utter exhaustion and a risk of permanent damage. The individual might well have already suffered from troubled behaviour which had failed either to restrain or contribute to the dilemma's resolution.

In these circumstances, a failsafe, which was the conclusion to a severe, normative stress, might result. The concept is taken by analogy from engineering, in which a fail-safe device prevented damage to a machine by ensuring the design limits were not exceeded, for example by keeping the rotational speed of a crankshaft within a safe maximum. In a self-aware, social gatherer-hunter, failsafe would not be confined to physical harm but would include the prevention of emotional injury, when self-respect, worldview or dreamtime were under threat. A failsafe would actualize when the adherence to a course of action, by commission or omission, was beyond a person's physical, emotional or intellectual resources.

In view of humankind's limited ability, anyone could be capable of attempting impractical goals. A commitment to an impossible enterprise could naturally be dropped but the endeavour might appear either so momentous or give such joy, that to do so voluntarily would be inconceivable. Again, explicit or implicit coercion, the potential benefits or the urgency to finish might entail that any deliberate choice to stop was unthinkable. On the other hand, when somebody was, for instance, publicly and

irremediably humiliated, the onset of a failsafe might be almost instantaneous.

In order to clarify this hypothesis, an imaginary scenario of gross overpromotion in a noncollective enterprise, in which financial rewards were distributed according to individual performance, is analysed. Seniority, the concomitant authority and higher standard of living would be actively sought. From time to time, the demands made by the higher ranking office on a manager could be too heavy, but rivalry from colleagues might encourage the exploitation of this weakness rather than special counselling or assistance. Thus, as a general rule, since the new job was coveted and the income and prestige welcomed, an excessive effort might be made, which could continue for years; but, imperceptibly, the strains could accumulate, for example disputes with the spouse over the short amount of time spent with the family. When the situation became intolerable, perhaps because of the further complications of a dying parent, a failsafe might occur. The preceding is simply one illustration of many imaginable alternatives. Ordinary, domestic life might also provoke an episode in which faulty personal relationships could precipitate a crisis. The personality of specialists would predispose some to a failsafe.

The syndrome, in effect, would rescue the patient from a hopeless plight, which, if continued, could only end in serious, physical or emotional impairment. The failsafe would be characterized by involuntary incapacitation on socially acceptable grounds. Although the symptoms could take many forms, a pattern would emerge of mimicking known illnesses, the discomfort of which would be irksome, since the infirmity would be partly to condition against future excess.

In most cases, abandonment of the oppressive objectives or immoderate zeal would induce a spontaneous, albeit unhurried remission from the affliction. Indeed, the eventual, voluntary rejection of unrealistic targets would be the function of the failsafe; initially brought about by the involuntary means of disabling symptoms and then, as an awareness of the implications of the misfortune dawned, replaced by conscious choice. Thus, the condition would slowly ameliorate as a readjustment was undertaken, assuming that a more realistic outlook was willingly embraced and morally admissible.

Furthermore, the disagreeable nature of the tribulation would mean that any loss of face was trivial compared with the wish to recover. Though the symptoms would, in the beginning, be a mystery to the sufferer, their origin could be transparent to companions, and, in time, intentionally or otherwise, communicated. Spontaneous remission, however, would not be inevitable, and, undiagnosed, the illness might continue indefinitely, even though the originating state of affairs had disappeared, for instance the dread of compulsory conscription even after the war had finished.

In this case, the patient, who might well have been patriotic, would normally be unwilling to concede that the fear of death was at the root of the illness. A failsafe could permanently alter personality, and, for example, an ambitious executive could become less singleminded, more relaxed and easy-going.

The failsafe hypothesis is a refutable substitute for Freud's theory of the unconscious mind. The aim of both is to explain behaviour which apparently conflicted with a person's sincere desires.

For example, a devoted father, desperate to support his family, might be totally impeded by an almost complete paralysis which had no physical origins. Freud's answer was flawed in the light of later methodological advances in science, because the data could always be reinterpreted to be consistent with his theory. The unconscious mind can never be expunged by research, since this entity is consistent with any factual discovery. The criteria for a scientific theory are elaborated in Part Five. On the other hand, the failsafe solution is specific in the patterns predicted, and their absence would refute this hypothesis.

Chapter 20: Troubled Behaviour, pp. 267 - 270

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Taken from *Humankind the Gatherer-hunter: From earliest times to industry*, published by Myddle-Brockton Publishers, 125 Pinks Hill, Swanley, Kent BR8 8NP, United Kingdom.

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

ANNOUNCEMENT

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Murray Stein, Ph.D. is a Jungian psychoanalyst who practices in the Chicago area. He has a Masters from Yale Divinity School as well as a Ph.D. in Religion and Psychological Studies from the University of Chicago. Dr. Stein is currently the Vice President of the International Association for Analytical (Jungian) Psychology.

In addition to being a well-known lecturer and author, he is also co-editor and publisher of Chiron Publications. Dr. Stein is the author of the following 4 books: *InMidlife* (1984); *Jung's Treatment of Christianity* (1985); *Solar Conscience/Lunar Conscience* (1993), and *Practicing Wholeness* (1995).

ARTICLE:

by ***Claire Russell*** *&* ***W.M.S. Russell***

Population Crises and Population Cycles 6.

North-Westem Europe: The Region and Its Crises

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

Editor's Note: This is the 6th installment to this article. The other 5 installments were in our March 1996, October 1996, November 1996, December 1996, and January 1997 issues of *The ASCAP Newsletter.* The reference list for this article and the article 7 will appear in a future issue.

The region of North-Western Europe **(Table 1**) is of unique interest, for it was here, in the Middle Ages, that an entirely new kind of society evolved, which was eventually to achieve the *technological break-through* (W.M.S. Russell, 1967) that made the modem world. The medieval North-West Europeans were the first to show 'an enthusiasm for the mechanisation of even the simplest activities, and a fascination with technology'(Gille, 1963). They showed 'an enthusiasm never seen before or elsewhere for power technology and the mechanisation of industrial processes' (W.M.S. Russell, 1967). They were 'the first people in history who consistently combined technological expertise with natural energy to create labour-saving devices' (Kealey, 1987). 'By 1250 A.D. or 1300 A.D., foundations had already been laid for the later technological ascendancy of North-Westem Europe' (Pacey, 1974).

This wonderful development was not, of course, due to the North-West Europeans being inherently wiser or more skillful than other peoples. Many of the crucial inventions that made the breakthrough possible came from outside Europe, notably from China. The North-West Europeans succeeded because until the 19th century, they had *far lower population densities* than other civilisations **(Table 2).** They had no mass labour of their own, and they could not afford many slaves: those captured in Charlemagne's wars had to be exported. The Chinese exploited new power sources (notably fossil fuels), but everything was subordinated to the hydraulic mass labour system: water-mills were banned and actually destroyed under the T'ang. Mass slavery in the Northern Mediterranean created a nightmare of unemployment for free workers, which was sometimes realised (e.g., in the 130's B.C. in Rome): the Emperor Vespasian actually paid an engineer to suppress a labour-saving invention. The North-West Europeans, short of docile labour, had no such inhibitions. The Chinese invented the mechanical clock in the 11th century A.D., the North-West Europeans (independently) in the 13th century A.D., but the Chinese lost the prototype and had forgotten all about mechanical clocks (and even sun-dials!), by the 16th century A.D., whereas the North-West Europeans promptly had the hours chimed in their chief cities, creating one of the most wonderful amenities of the modern world.
Hand-made paper was invented by the Chinese in the 2nd century A.D., and diffused to the Islamic world in the 8th century A.D.; in the 13th century A.D. it reached North-Western Europe, and production was promptly mechanised.

Individuals from the Northern Mediterranean (declining after the 16th century A.D., as we saw in the 5th paper of the series), and from North-Eastem Europe, made great contributions to the technological breakthrough. But North-Eastern Europe was vulnerable to massive invasions by nomads from Asia: the Mongol incursion in the 13th century A.D., wrecked the promising development of cities in the region, and without the progressive potential of cities, the region had sunk, by the 16th century, into a plantation system with docile peasant labour, called by Engels, the *second serfdom,* exporting grain at times to North-Western Europe, where progress was surging ahead.

The North-West Europeans practised versatile forms of mixed farming (crops and stock), and two successive sets of improvements enabled them to colonise their clay valleys and increase their yields **(Table 3),**

so the population did rise **(Table** 3, Figure 1), though until the 19th century A.D., densities remained low **(Table** 2). Despite this, Northwestern Europe experienced population crises, producing the classical saw-tooth curve **(Table** 3, **Figure** 1), because, as usual, with every advance in food production, population soon outgrew the new level of resources.

During the crises, they concentrated on arable farming to produce the staple grains, and the resulting shortage of animal proteins and fats rendered the people vulnerable to epidemics: the Dutch, who continued to practise stock-farming and fishery, suffered little from the Black Death. During relief periods, the emphasis was on stock-farming. In Spain, as crisis resumed, that led, as we saw, to the disastrous rise of the Mesta. But in the more tolerant climate of North-Western Europe, the periods of concentration on stock helped to promote new forms of mixed farming **(Table 3).** During crises, arable farming was extended to marginal areas, where yields soon declined: in the Medieval crisis, as a result, hundreds of new villages had to be abandoned. It was the same in the Early Modern crisis: a Swabian chronicler wrote in 1550 that 'there was not a corner, even in the wildest woods and the highest mountains, that was not occupied'.

The crises in this region had all their usual effects. There was severe price inflation and fall in real wages **(Figure 2,** and cf. **Figure 3** of the first paper in the series, the March 1996 *ASCAP Newsletter).* This, together with massive unemployment, created a mass of paupers, comprising up to one quarter of the total population in the region, many of them naturally vagabonds and some brigands. There were massive persecutions - Jews and heretics in the Medieval crisis, Jews, heretics, witches, and supposed werewolves in the Early Modern crisis.

The Medieval crisis included the major part of the 100 Years' War (1339 A.D. to 1453 A.D.), the Early Modern crisis of the 30 Years' War (1618 A.D. TO 1648 A.D.); in terms of proportionate casualties and involvement of civilians, 17th century warfare was the worst in Europe before 1914. In 1641 to 1652 A.D., the English reduced the population of Ireland by about a 1/2 million. Major episodes of famine, epidemics, and civil violence are shown in **Table 4,** but these are only the extreme outbreaks. The threat of famine was virtually continuous during the crises. The city of Bremen (not untypical) had 25 severe epidemics between 1565 and 1657. In South-Westem France there were 282 revolts between 1635 and 1660. In contrast, there is general agreement that famines epidemics and civil conflicts were far fewer during the relief periods of the 12th and 15th centuries. The effect of all of this on the expectations of life at birth was shattering, as shown in **Table 2** of the first paper in the series. Just as in other mammals, the crises greatly reduced the survival of young children, and hence the total population, as vividly shown in **Figure** 3. With this we conclude our brief account of the crises, and shall turn in the next paper to the achievements and cumulative effect of the periods of relief and specially low population density. OS

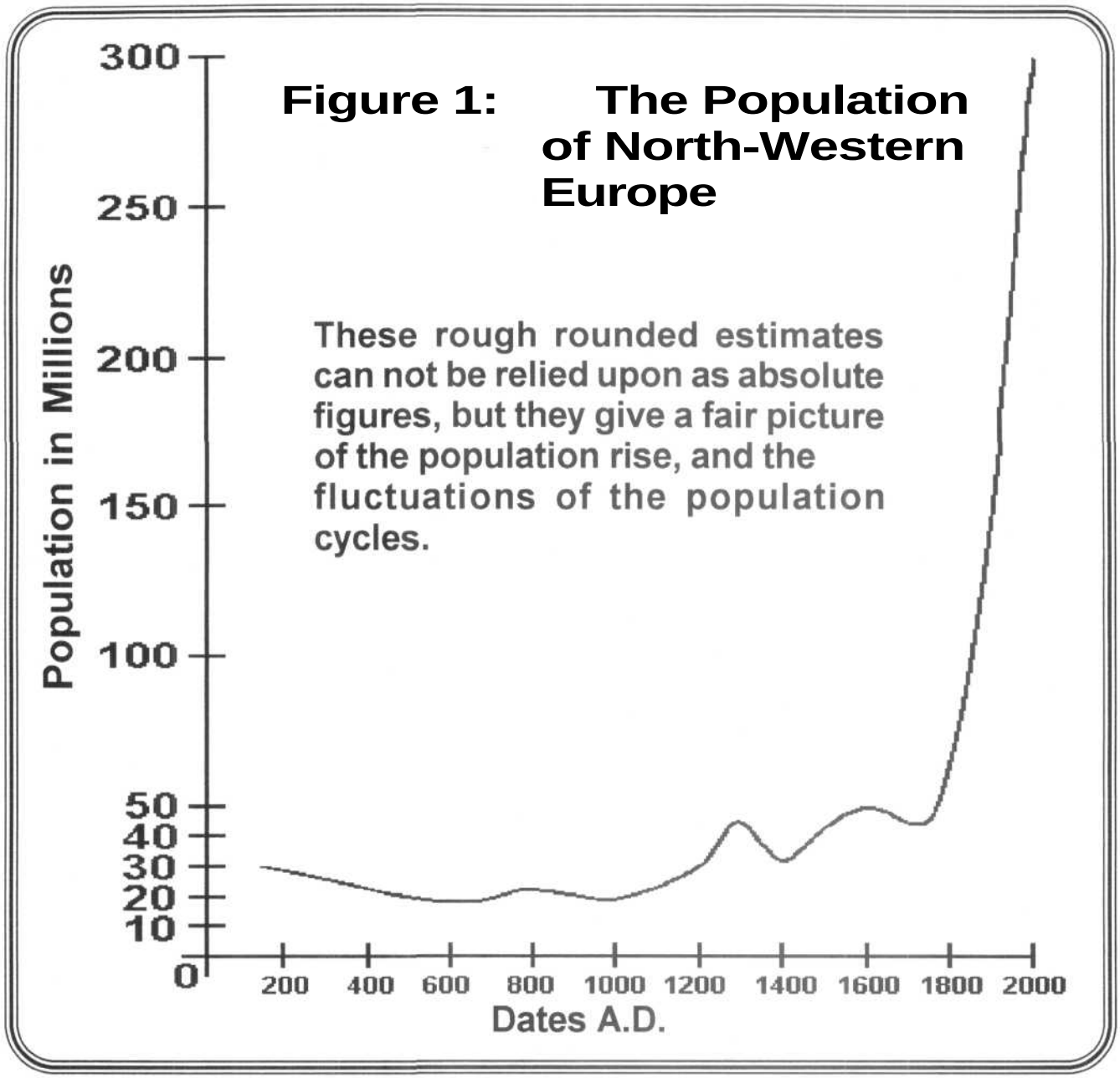


Figure 2 - Shows changes during the 16th century A.D., in two North-West European countries, in the indexes for grain prices and wages (percentages of the average value for 1501-1525). For the price inflation in England and Wales, see **Figure 3** of the first paper in this series, in the March 1996 *ASCAP Newsletter,* p. 18.

Figure 3A - (After Southwick, 1955 - the same ordinate is used for all 3 graphs.) A population of mice in an enclosed space. As the population increases, the fighting becomes more frequent, and the survival of the young drops, until the population begins to decline again.

Figure 3B - (After Kamen, 1976) The town of St. Lambert des Levees in Anjou, Northern France, at the climax of the Early Modern population crisis in this region, during the civil war of the Fronde, which involved fierce fighting.

Figure 2: Price Inflation in Early Modern Population Crisis

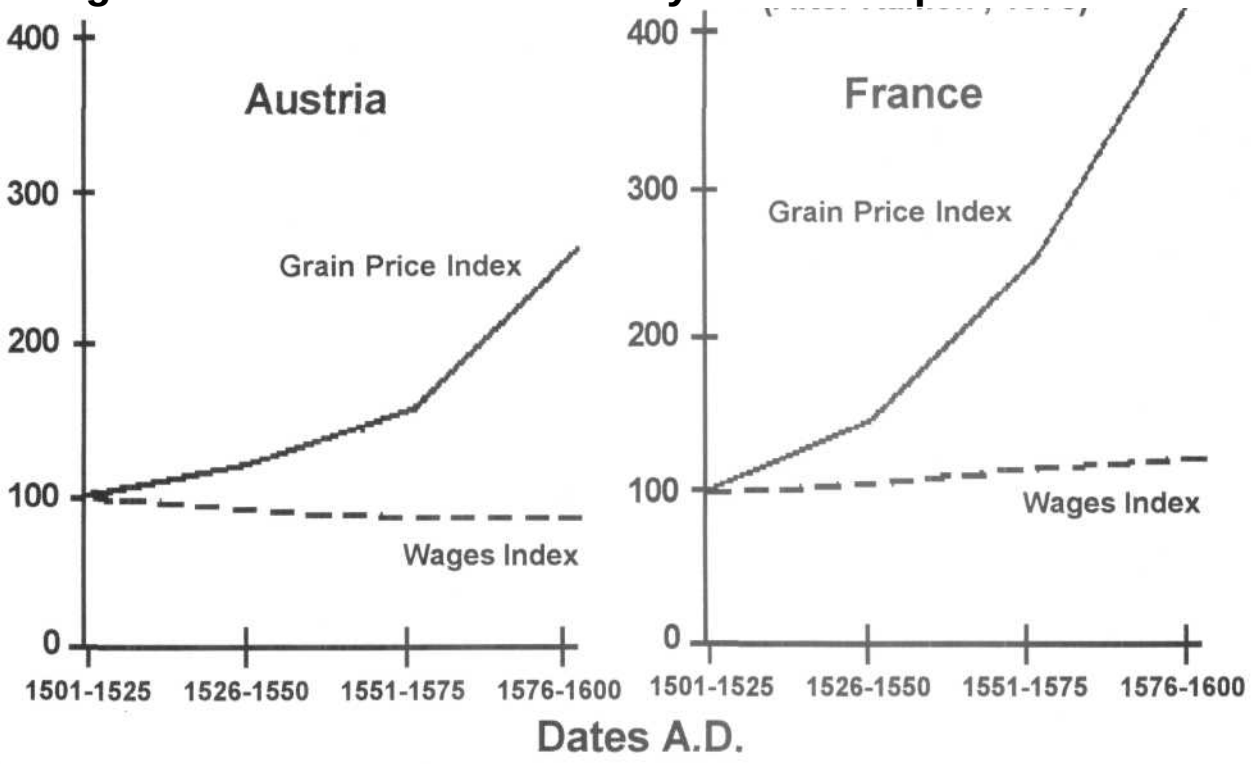


Figure 3: Mice and Men

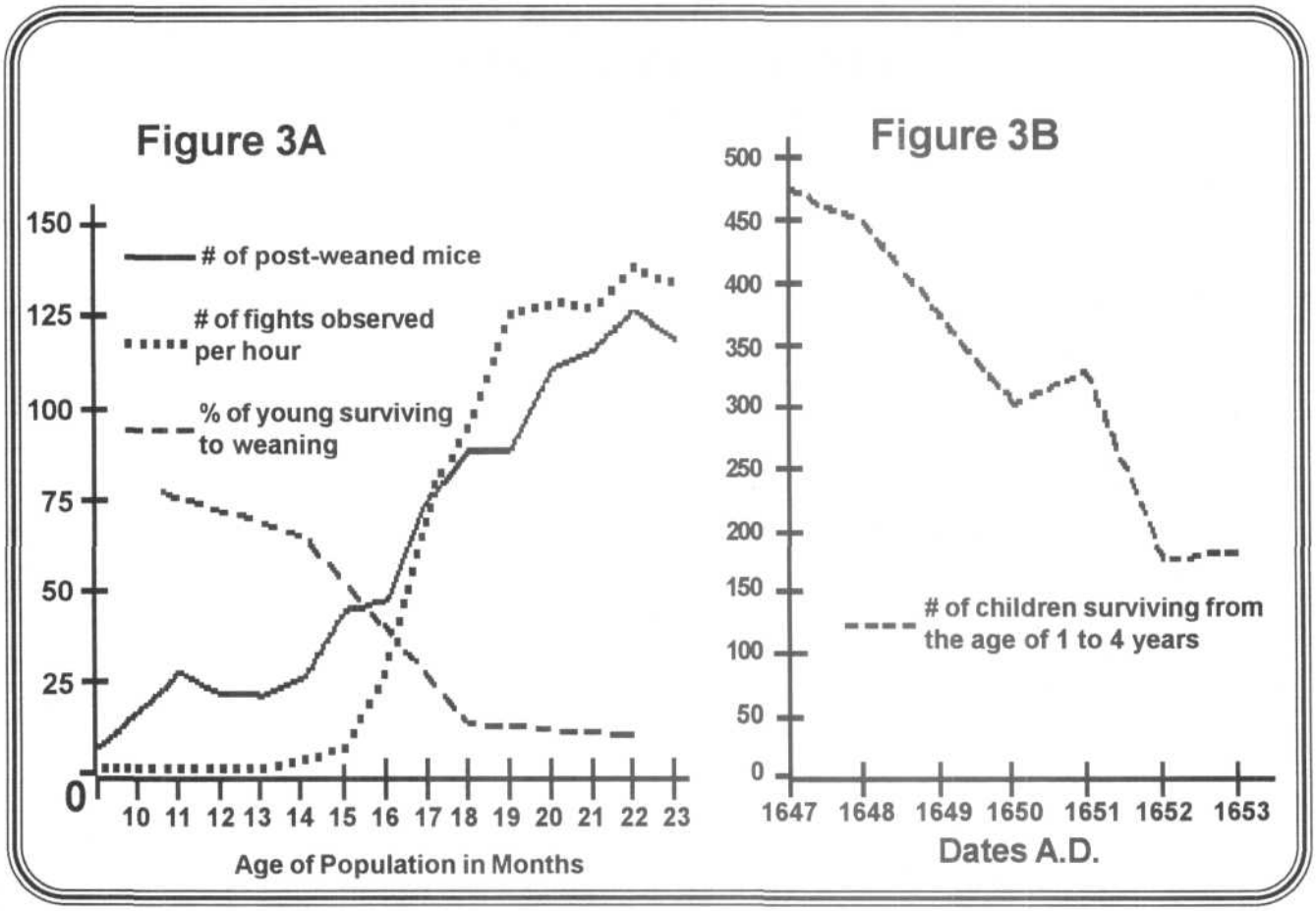


Table 1: North-Western Europe

The table lists (under their modern names) the components of North-Westem Europe. Territorial boundaries, and dynastic unions, or divisions of the modern countries, varied considerably at different times, but the region always maintained a certain cultural identify. It is not perfectly defined by any one ecological boundary, but most of it falls *within* an area where the beech tree grows, and *outside* an area where continual frosts prevail for at least 1 month in the year. The other parts of the continent are the Northern Mediterranean and North-Eastem Europe.

Austria	Italy, north of Naples	Belgium	Luxembourg
Czech Republic	Netherlands	Denmark	Norway
France	Sweden	Germany	Switzerland
Iceland	United Kingdom	Irish Republic	

Table 2: Some Population Densities

These are rough, rounded estimates, but they give a fair picture of the relatively low population density of North-Western Europe, compared with other civilisations, until the late 19th century A.D. Note also the relatively low population density of Britain, the country that launched the Industrial Revolution, until the middle 19th century. By the late 20th century, population densities in the region are quite comparable with those of the hydraulic societies.

Region	Date	Population/km^2
Hydraulic Societies		
Egypt	1st century B.C.	280
Chekiang Province, South China (pre-industrial)	early 20th century A.D.	**214**
Ancient Northern Mediterranean		
Attica	431 B.C.	104

North-Western Europe

Country	Date	Population	Country	Date	Population
England	1086 A.D.	11	United Kingdom	1750	31
France	1300	35		1800	45
Switzerland	1479	15		1850	86
Netherlands	1600	50		1900	155
North Italy	1600	45	France	1992	104
France	1600	34	Germany	1992	225
	1700	35	United Kingdom	1992	236
	1750	43			
	1800	57			
	1850	68			
	1900	76			

Table 3: The Population Cycles of North-Western Europe

Dates (A.D.)	Population Crises	Renaissances	Food Production	Population (in Millions)
400-700	Late Ancient			
600				18
7th Century			Fixed mouldboard plough	
8th Century			3-course rotation	
750-850		Carolingian		
800				22
9th Century			Horse-collar, Horse shoes	
850-950	Dark Age			
1000				19
11 th Century			Combination of above	
1050-1200		Medieval		
1200				30
1200-1400	Medieval			
1300				45
1400				32
1400-1530		Early Modern		
15th Century			New Husbandry, Low Countries	
1500				42
1530-1670	Early Modern			
1600				50
1670-1914		Long		
17th Century			New Husbandry, England	
1700				45
18th Century			New Husbandry, North-West Europe	
1750				46
1790-1850	Incipient			
1800				65
1851-1931		Mass Emigration		
1850-			H.E.I. Agriculture, Imports	
1900				150
1914-	Modem			
1970				280
1990				300

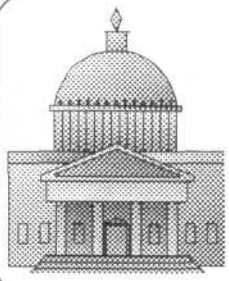
The New Husbandry included root fodder crops and leys (temporary pastures) with clovers and lucerne in rotations, heavy manuring, and marling. H.E.I. Agriculture means agriculture with high energy inputs: from 1850, this applied to agriculture both at home and in the overseas regions from which food was imported. The inputs were of agricultural machinery and agricultural chemicals. In the modern United States, 'intensive systems... may yield as little as one tenth' in food energy 'of the input' (W.M.S. Russell, 1988).

The peak population of the region underthe Roman Empire, in 150 A.D., was probably about 30 million, but, as we saw in the 4th and 5th papers of the series (November & December 1996 *ASCAP Newsletters),* this population level in the Western Empire was dependent on imports from the dry belt provinces.

Between 1851 and 1931, about 18 million people emigrated from the British Isles, and about 17 million from the rest of North-Westem Europe.

Table 4: Some Major Episodes of the Medieval and Early Modern Population Crises

Dates (A.D.)	Civil Violence	Famines	Epidemics
Medieval Crisis			
1302	+ Flanders		
1315-1317		+	
1316			+
1340	+ Denmark		
1346-1350			+ Black Death
1358	+ France		
1360-1370			+
1378	+ Florence		
1378-1385	+ Flanders		
1381	+ England		
Early Modern Crisis			
1525	+ Germany		
1556-1558			+ England
1559-1593	+ France		
1565-1566		+	
1567-1572	+ Austria		
1594-1597	+ Austria	+	
1626-1617	+ Austria		
1630			+ North Italy
1634-1636	+ Austria		
1635-1636			+ Germany
1636-1641	+ France		
1642-1649	+ England		
1647-1649		+	
1648-1653	+ France		
1659-1662		+	
1665			+ England



The Western Civilization Home Page

http://ikra.call. uvic.ca:80/LangCen/Special/westciv.htm

This page provides links to the pages relating to different Western Civilization topics. Each of the linked pages lets you jump to WWW sites all over the world with resources relating to the topic, and provides a brief description of each site. The topics are: Ancient Greece, Ancient Rome, Dark-Age England, Pre-Renaissance Europe, Renaissance Italy, Colonial Expansion, Revolutions, & Industrialization.

ARTICLE:

by Russell Gardner, Jr.

Weight Control Research as Model for Sociophysiological Research

In the 9 January 1997 issue of *Nature,* late-breaking research on the obesity-related, newly described hormone named leptin made the *News and Views* section.[1] I immediately noticed this summary by Jeffrey Friedman of Rockefeller University but put it aside for later review during a next idle moment, which then didn't happen for a month. But director of UTMB's Psychoneuroimmunology Section, Eric Smith, noticed an "open session" in the lab meeting schedule and asked the day before if I wanted to preside.

He and I had earlier discussed the abstracts reproduced last issue of *ASCAP* from *Psychosomatic Medicine* but the journal issue with the original articles was not to be surfaced immediately, so refinding Dr. Friedman's lucid summary on the tabletop was good news, even better for the laboratory group because it included new findings on melanocyte stimulating hormone (MSH) in which I knew Eric and his group has investigatory interest. Also, I was able quite quickly to find two of the source articles Friedman mentions so that laboratory-expert members of the group could inspect detailed methodology, consider the actual data, and help this non-expert understand such issues.

But my excitement on rereading the summary piece upped several notches as I sensed this cellular-molecular research to represent nicely an implicit model for sociophysiological research. As *ASCAP* readers already know, I have proposed sociophysiol-ogy as psychiatry's basic science. When discussing this topic before, I have focused upon clinical, evolutionary and conceptual issues, but I thought I might exemplify some of aims of the research vision in this essay. Its involvement with normal systems contrasts with James Olney and Nuri Farber's striking and important work with glutamate receptors and schizophrenia.[2] Theirs is state-of-the-art psychiatric research using animal models to understand a human condition, but falls short of being sociophysiology, of echoing an improved understanding of the normal system's physiology, an aberrancy of which is core to the understanding of the pathology according to Harveian medicine. They focused on disorder to the exclusion of order. This does not diminish its importance, for clinical findings often predate insights to normal function. Obesity health concerns and consequent research, for instance, gave impetus to research on normal weight control systems.

Which brings us back to Friedman: he focuses on new molecular players that produce what are two behavioral states that operate to restore equilibrium in the body's fat storage mechanism, titled, (1) response to starvation, and (2) response to obesity. If too little fat exists, the animal acts to increase it; if fat increases too much, the animal acts to decrease it.

Ultimately, the most important actions involve eating more or less, but in response to starvation, not only does eating increase (whateverfood is available), but temperature, growth, activity and reproduction decrease, and in the autonomic nervous system, parasympathetics predominate in a conservation strategy for the body. This is what happens in mammals including people in the face of no or very little food for prolonged periods. (Sleep researcher and ethologist Mark Opp at the lab meeting pointed out that in the first stages of food deprivation, food-searching activity is increased, not decreased as in the more extreme condition). The story on starvation is that one does what one can to preserve life for if potential bounty appears later, extended life can occur later. This apparent lesson seems to have been taught the genome and nervous system by the school of

natural selection. On the other hand, over-much fat or obesity has its problems too such as diabetes mellitus, atherosclerosis and other ills of civilization. So natural selection apparently also caused us mammalians to respond to increased fat cells with a behavioral state in which not only does eating decrease, but activity increases and sympathetic discharges predominate in the autonomic nervous system in order to adjust our fat to a best level.

How has Mother Nature done this as she affects the genome using the Darwin machine? What chemical moieties in the brain arrange for one or the other of these states to come about and what is the typical chain of events (or chains, as it happens, at least according to the information I'm about to summarize)?

An extremely obese mutant mouse strain was named *ob* for obese. These mice actuated "starvation mode," eating whatever is available (hence very high obesity because food is always available in the lab setting). But despite adequate nutrients, they demonstrated poor reproduction, less activity, shorter body lengths, lower temperature and heightened parasympathetic activity. They turned out to lack a usual blood constituent, eventually named leptin, now well characterized. Its gene was characterized too; in its *ob/ob* form, the animal doesn't make the hormone usually generated by the fat cells themselves. Leptin replacement slims the animals down and increases activity and reproduction.

That is, less fat —> less leptin —> more starvation response behavior (with consequent obesity if food is ample). Neuropeptide Y (NPY) does the opposite: when injected into the brain, eating increases. Yet, levels of NPY are higher in the *ob* mice and decrease when the mouse is treated with leptin and the starvation response lessens.

So how precisely do the two opposite-direction, weight-control chemicals relate? To find out, Erickson *et al* in a recent *Science* reported on what happens if mice whose *NPY* gene is knocked out are bred to *ob/ob* mice so that their offspring *(NPY+ ob/ob)* have neither.[3] They found out that these next generation mice were obese also, but less so, only twice normal body weight; also they had normal growth and reproduced. They weren't in starvation response mode. These workers concluded that reduced leptin induces NPY to stimulate eating (and the rest of the starvation behavioral state). Moreover, they determined that it did this by stimulating the Y5 receptor in the arcuate nucleus of the hypothalamus. In summary, the chain goes like this: fewer fat cells —> less leptin —> more NPY—> increased activity of Y5 receptor in the arcuate nucleus of the hypothalamus —> behavioral state called response to starvation.

More remained to be solved: not only by weighing them but by using magnetic resonance imaging, the Erickson group determined that the $NPY^{-/-}$ *ob/ ob* mice still had more fat than did normals. Friedman asks why and notes this *"suggests that other downstream factors required for the response to increased weight are not working in these animals. It is intriguing, in this regard, that* $NPY^{-/-}$*ob/ob mice are phenotypically similar to mice that have an induced mutation in the melanocortin-4 (MC-4R) receptor gene."*

Here he refers to a story that first features yellow-agouti mouse mutants (A^{Y}) which are not only bright yellow but obese as well, also weighing about twice normal. Fan *et al* found that the color and the obesity were connected by the secretion of an abnormal peptide.[4] Reduced skin pigment results from the fact that a peptide manufactured by the A^{Y} gene is an amino acid chain with cyclic components that antagonizes the usual receptors of MSH in the skin. This means that they don't fulfil their normal role of darkening the mouse's coat, leaving it instead the brilliant yellow. But this agouti peptide also apparently antagonizes another hypothalamic receptor, MC-4R, which ordinarily receives MSH signals that eating should lessen. With the agouti peptide, the message doesn't get received and the animal eats more if food is available. Another paper to which Friedman's essay refers published in *Cell* shows that when MC-4R is

missing from breeding knockout mice, the coats of these mice are not yellow but the mice are remarkably obese (MC-3R not MC-4R is the receptor in the skin). Yet these animals do not show the starvation response as they are normally fertile, have normal body temperature, and grow normally long. They had very high blood leptin levels.

In conclusion, without normal MC-4R, behavior doesn't respond to leptin but with the receptor, it does. The causal chain involving MC-4R goes like this; more fat —> more leptin —> more MSH —> more activity in MC-4 receptor—> response-to-obesity behavior. The agouti mice were fat because a usual mechanism wasn't in place with the antagonistic peptide. In summary, there are two limbs of weight control systems that stem from adipose tissue producing leptin: absent leptin causes the starvation response syndrome acting through NPY; increased leptin ordinarily causes the obesity response syndrome with less eating and more activity, using MSH and MC-4R as downstream links in the causal chain.

Of course, humans are very interested in the body states that involve eating too much or too little, whether this is NPY induction of the starvation response or MSH failure. Friedman notes that although different parts of the hypothalamus (NPY in the arcuate n.; MC-4R in the dorsal medial area) are involved with the two limbs of the weight control system, there is likely to be considerable crosstalk. Fan *et al* note, for instance, that NPY is elevated in the dorsal medial hypothalamus in A^{Y} and MC-4R knockout mice.

In the offing are possible new drugs such as antagonists to the Y5 receptor. Antagonists to MC-4R like the agouti peptide cause obesity, but Fan and colleagues have also fashioned agonists which limit eating, at least in mice. Clinicians are well familiar with syndromes wherein weight control regulator systems have gone awry in some way and will be grateful for effective medications that intervene in pathological expressions of the weight control mechanisms. Recall, for instance, that the three key members of the Davis family reported in the sociophysiology essay of last issue were obese; this played a pathogenetic part of their many health problems. So if the behavioral syndromes of response to starvation and response to obesity were normal behavioral states, the Davises and innumerable other people exhibit pathological counterparts. We don't yet know just how nor which system goes awry in these people (there may be others of course).

I have met with the Davis family subsequently, and they have provided still another example of how human thinking and planning can overcome lower brain-body responses (in this case, we'll assume hypothalamic involvement). Remember Johnny, the young adult son of the patient and her contentious husband with a crusty exterior/heart of gold whose crust predominated? I learned in a subsequent session that Johnny once weighed 420 lbs, but that he is now 200 aiming at 180. He has three kinds of exercise equipment in his room and puts his mattress up on its side most of the time, as the exercise machines are frequently in use. If the contentious family is a group of entities over which he cannot be in charge, he can be in charge of his body and its activities in best human fashion.

Keeping with the promise of seeing this as a model for sociophysiological research, I now wish to focus upon the fact that Friedman's two behavioral somatic states are parallel to psalics or communicational propensity states. I have postulated the latter to be states fundamental to psychiatric syndromes just as disturbed weight control systems are fundamental to pathological obesity. In the weight control behaviors, we can begin to discern how certain molecules are operative players. In the normal counterparts of psychiatric syndomes we are looking forward to more of this kind of analysis, perhaps springboarded by Olney-Farber kinds of research on pathological states.

Psalics are evident from stereotyped behavior patterns that, when expressed, have the individual signal to others the role that the signaller is

assuming with respect to other conspecifics. I have postulated that we see such states overly expressed in psychiatric illness just as we see obesity and anorexic states as maladaptive variants of the regulation of normal weight control. I like Friedman's formulation for how mechanisms are teased out for opposite behaviors with adaptive significance. We know in psychiatry of other behaviors that have opposite valence (think of bipolar illness) and we might think of parallel mechanisms that might be operative. Not that they will be exactly mirror images. We know that bipolar illness is really a misnomer in that patients frequently present with mixed pictures of both mania and depression simultaneously. Mecha-nisms are probably not best exemplified by an engine in reverse. We know now that this is also the wrong metaphor for the weight control systems: the Y5 receptor is in the hypothalamic arcuate nucleus but MC-4R is mostly in the dorsal medial area of the brain region.

Obesity might be a medical and psychiatric problem so that one doesn't automatically consider it a social communicative attribute. Yet one reacts very differently to a large person than a small. Heavy policemen and bullies are benefitted in their efforts by the extra muscle that carrying more weight stimulates; beauty may involve more or less fat. People like Johnny who lose weight gain our admiration for their will power. So perhaps in addition to being a model to which sociophysiologi-cal research might aspire, weight control research can be directly claimed for the discipline! *c8*

Climate Variability Project Home Page

http://www.cics.uvic.ca:80/canada/research/crn/varia.htm

Principal Investigator: Jacques Derome, McGill University

Project Description:

The purpose of this agreement is to conduct the third year of research described in the proposal entitled "Collaborative Research Program in Climate Variability (VARIABILITY) - dated May 1,1994 submitted to the Scientific Advisory Committee for the Climate Research Network.

The central goal of the Climate Variability program is to improve our understanding of the dynamics of climate variability on time scales ranging from seasons to decades. The research will shed light on the relative importance of the lower boundary conditions and the internal dynamics in generating and maintaining the atmospheric fluctuations on the above time scales. It will also help determine what part of the observed fluctuations might be predictable. The research program will pursue four different types of projects:

(1) those that will gather and validate data sets, both from observations and from the integrations of the Canadian General Circulation Model (GCM) known as GCMII, and to develop the software for climate diagnostics;

(2) those diagnostic studies based again on observations and GCM runs;

(3) those studies dealing with model formulations and sensitivity studies; and

(4) those modeling studies of coupled atmosphere-ocean and ocean-ice systems. The program will be undertaken in close collaboration with the AES Climate Modeling Group located at the University of Victoria and, as appropriate, with the AES Climate Monitoring and Remote Sensing Division in Downsview.

CRUNCHES & ABSTRACTS...

Editor's Note: For his month, the title this section, has changed. The first 3 items are articles that were "crunched", by ASCAP member, BeverlySutton. The othertwo items in this section are abstracts.

Sulloway, F. J.: ***Born to Rebel: Birth Order, Family Dynamics, and Creative Lives,*** **Pantheon Books, New York, 1996, pp. 653.**

Firstborn:

intense
more responsible jealous of their status academically
successful tend to have more offspring
conforming and conventional assertive
and socially dominant identify with power
and authority overrepresented among
political leaders
self-confident
achievement oriented
ambitious & defensive

Laterborn:

altruistic
empathic
peer-oriented
socially successful
support unpopular causes
resist pressure to conform
engage in risky physical activities
explorers
iconoclasts
open to experience
question the status quo

Boys: aggressive and punitive
Girls: verbally antagonistic

A gap of 6 or more years between siblings makes the birth order process start again. Singletons are usually grouped with firstborns; singletons score somewhere between firstborn and laterborn on most measures but closer to firstborns regarding rejection of new experience. Only children are least predictable because they have no siblings. The longer siblings live together, the more they differ from one another. The contrast is greater for those adjacent in birth order.

A firstborn can become a radical if exposed to substantial conflict with a parent. In middle and upper class families, early parent loss decreases sibling differences in radicalism; in lower class families, early parent loss produces extremely conservative firstborn and extremely rebellious laterborn. Firstborn women support social reforms such as health care, welfare, temperance, and social work. They are motivated by high moral conscience not rebellion. Laterborn women tend to advocate lower class reforms such as birth control and anarchy. They are motivated by empathy and not by ambition or high moral conscience. Firstborn tend to think that "biology is destiny" whereas laterborn, women, and minorities do not accept these deterministic ideas, Increased support for radical reform is generally associated with minority status.

Middle children tend to protest in non-violent ways. They are more flexible and willing to compromise.

When parents display favoritism, children feel that a grave social injustice has occurred. Social attitudes of mates are very concordant. Opposing radical change is a firstborn trait and not connected to family wealth. Similarly, the tendency for laterborns to adopt new ideas is true at all socioeconomic levels. There are no gender differences with these tendencies. Political and religious ideas are first learned from parents and are shared by siblings. Laterborns are more open to experience and willing to revise these ideas.

In the Protestant Reformation, firstborn were usually Catholic and laterborn were Protestants. Catholics supported primogeniture and partible inheritance (divide the estate) was supported more by Protestants. Lower class persons rarely become scientists. Among

those persons who do become establishment scientists, firstborn are overrepresented. They are good puzzle solvers and adapt established theory to new and socially accepted fields.

Shyness is about 50% genetic as compared to 30-40% for many personality traits. Shyness is associated with physiological changes especially high stable heart rates (indicator of vigilance) and this higher heart rate is seen before birth in shy persons. Shy people tend to avoid strong opinions. Laterborns are very shy when the age gap is 1-2 years. Laterborns can outgrow shyness when they learn to cope with older siblings. Shyness may increase with early loss of a parent except in lower class families where the loss produces extroversion in the child especially in the firstborn. By assuming the parent role, the firstborn gains power.

Tough-mindedness is associated with the personality traits of extraversion and agreeableness/antago-nism. Tough-minded people tend to be leaders, stubborn, and moralistic. They are usually men, firstborn, and used to getting their own way. In revolutions, they tend to be terrorists. In politics, being tough minded is important and determines how conservative or liberal ideas are put into practice. Being willing to use violence to maintain the status quo is firstborn, conservative, tough minded behavior. Laterborns seek radical political revolutions and are more likely to get killed for their support of freedom (speech, worship, slavery) and equality (race, gender). Among radical innovators, firstborn are the exception. Among radical innovators, firstborns are the exception. A firstborn radical is usually the child of a laterborn radical.

To be labeled a Radical Ideological Revolution, the controversy must be widespread, e.g., Copernican revolution, Hutton's theory of the earth (crust formed by millions of years of weathering), and the Darwinian revolution. Examples of Technical Revolutions are Harvey's blood circulation, Freudian psychoanalysis, and Einstein's special relativity. Examples of Controversial Innovations are preformation theory (e.g., homunculus) and Phrenology. Laterborn are over-represented as supporters of new theories even when the initiator is a firstborn. Examples of Conservative Theories are germ theory, eugenics, and reputation of spontaneous generation. Firstborns are supportive of Conservative Theories and are particularly supportive of orthodox ideas in science. Firstborns and laterborn disagree on most new scientific ideas but over time they tend to think alike. For support of radical innovations, laterbom are about 50 years ahead of firstborn. In their 30's, the firstborn is already supporting the scientific status quo; the laterborn are still open to radical ideas until their early 60's.

Predictions of laterborn behavior regarding scientific controversy: atheistic religious attitude, extensive travel history, liberal political attitude, conflict with parents, young, and supported by peers. Laterborn support occurs early in a controversy and continues for years particularly if the controversy addresses an unpopular theory. Social class, religious denomination and failed theory are nonpredictive.

Marx attributed historical change to differences between families. Freud looked at interpersonal conflict in the family. Sulloway says parent-child conflict is a result of sibling conflict over parental resources. This conflict is not sex linked, associated with a parent of the same or opposite sex, or the result of the sexual drive. A child wants the maximum amount of benefit from both parents. If a favorite child exists in a family, "make sure *that you are the favored one.* " This competition is adaptive as is altruism among siblings.

The nature of historical change predicts the response of the person. Firstborn support technical or conservative innovations and laterborns resist them, seeking instead radical changes of the status quo.

Thomas Henry Huxley said after reading *Origin of Species, "How extremely stupid not to have thought ofthat*!"

Beverly Sutton, M.D.

Brown, J. R.; Ye, H.; Bronson, R. T.; Dikkes, P; & Greenberg, M. E.: A defect in nurturing in mice lacking the immediate early gene *fosB.* *Cell* 1996;86:297-309

For animal survival, learning and remembering where food and danger are found is essential. Young mice require parental nurturing responses for survival. The nervous system must focus on the important environmental stimuli and then adapt behavior.

New gene expression is critical for adaptive neu-ronal responses. Environmental stimuli trigger gene expression in certain brain regions. Immediate early genes (lEGs) are induced quickly without any new protein synthesis. Next are the delayed response genes that require new protein.

The fos family has 4 genes: *c-fos, fosB, fra*-1, and *fra-2.* fosB has two forms, a regular length protein and a shorter protein called ^fosB. These proteins are transcription factors that dimerize with Jun family proteins.

All these heterodimers recognize the AP-1 site (core sequence TGACTCA) of DNA. *Fos* family genes are expressed in the brain at regions that produce nurturing behavior, c-fos mutant mice are small, have osteopetrosis, and show grossly normal behavior. FosB mutants show a non-nurturing behavior - this is seen in postpartum females, young females and males.

FosB mutants are slightly smaller than wild-type mice but have normal organs and normal pregnancies. Their pups die at 1-2 days of age. Mating wild-type and fosS mutant mice show that pup survival correlates with the mother's genotype and not with the father's or the other pups' genotypes.

FosB mutants do not show the change from a presecretory state to a secretary state at parturition because the mutant mothers do not nurse. The reproductive tract, hormonal status and brain regions of the mutant mothers appear to be the same as that of the wild-type mothers.

Nurturing behavior in mice consists of making a nest, cleaning pups, putting pups in the nest and crouching over them for feeding and warmth. FosB mutant mothers have pups all around the cage and ignore them. Exposure to many pregnancies or to a wild-type mother with normal nurturing behavior does not improve the mutant mother's nurturing behavior. Never pregnant wild-type females and wild-type males have a high level of nurturing behaviorwhen exposed to pups. The crouching behavior is the same for fosB mutant and wild-type males. FosB mutants do not have global cognitive problems and are not anosmic. Hypothalamic function (cold adaptation, locomotion, exploration, and aggression towards an intruder) is intact; eating and sexual behavior is normal.

The brain regions necessary for nurturing were identified by lesion studies. Mice were stained with anti-fosB antibodies at 2 and 6 hours after exposure to pups. There was induction of fosB in the preoptic area of the hypothalamus at the 6 hour interval. FosB was also found in the main and accessory olfactory bulbs and pyriform cortex. FosB in this area may modulate the nurturing response. A lesion in the accessory olfactory cortex does not produce any behavior change. The olfactory cues are necessary for pup recognition. FosB (not c-fos) was found in the cortex, striatum, hippocampus, amygdala, and other areas of the hypothalamus and may have other functions besides the nurturing behavior.

Olfactory information goes to the preoptic area of the hypothalamus (especially the medial area) via the amygdala. The preoptic area probably is the main regulator of nurturing behavior. The lateral efferent projections of the preoptic area are also necessary for nurturing and may be responsible for retrieval and crouching behavior by signaling the ventral tegmental area of the midbrain or some area of the brainstem.

Molecular mechanisms that may reinforce the nurturing circuitry of the brain are being studied.

Beverly Sutton, M.D.

Hamer, D.H.: The heritability of **happiness.** ***Nature Genetics*** **1996;(14):2,125-126.**

Lykken & Tellegen (University of Minnesota) write that cheerfulness and contentment are mainly a result of heredity. They studied middle-aged twins bom between 1936 -1955 using the Well Being scale of the Multidimensional Personality Questionnaire.

In 1380 pairs raised together, the correlations were 0.44 for monozygotic twins and 0.08 for dizygotic twins. The same questionnaire was given to a subset of the twins 5-10 years later to determine cross-twin cross-time correlations. The cross-twin cross-time correlation was 0.4 for monozygotic twins and 0.07 for dizygotic twins; the cross-time correlation was about 0.5 for both sets of twins. Therefore, 0.4/0.5 = 80% of the variation in the stable component of Well Being is heritable. How you feel right now is about equally genetic and circumstantial, but how you will feel on average over the next ten years is fully 80% because of your genes.

In looking for the happiness genes, it might be possible to find them by looking at positive and negative affect. Dopamine is a pleasure chemical in that it is released after eating, pleasant sexual interaction, or taking cocaine. (The gene for dopamine D4 receptor has been found and the D4 polymorphism association with the trait of Novelty Seeking has already been reported. Novelty Seeking is correlated with extraversion and positive emotionality.) Serotonin may produce negative affect in that it has been associated with depression, suicide, anxiety and social phobia. Serotonin reuptake inhibitors increase optimism and social confidence.

Beverly Sutton, M.D.

Duan, H. & Dixit, V. M.: RAIDD is a new 'death' adaptor molecule. ***Nature*** **1**997;385:86-89

The effector arm of the cell-death pathway is composed of cysteine proteases belonging to the ICE/CED-3, family. In metazoan cells these exist as inactive polypeptide precursors (zymogens), each composed of a prodomain, which is cleaved to activate the protease, and a large and small catalytic subunit. The coupling of these 'death' proteases to signalling pathways is probably mediated by adaptor molecules that contain protein-protein interaction motifs such as the death domain'. Here we describe such an adaptor molecule, RAIDD, which has an unusual bipartite architecture comprising a carboxy-terminal death domain that binds to the homologous domain in RIP, a serine/threonine kinase component of the death pathway. [RAIDD = RIP-associated ICH-1/CED-3-homolo-gous protein with a death domain] The amino-termi-nal domain is surprisingly homologous with the sequence of the prodomain of two ICE/CED-3 family members, human ICH-1 and *Caenorhabditis elegans* CED-3 . This similar region mediates the binding of RAIDD to ICH-1 and CED-3, serving as a direct link to the death proteases, indicating that the prodomain may, through homophilic interactions, determine the specificity of binding of ICE/CED-3 zymogens to regulatory adaptor molecules. Finally, alternations in the sequence of the N-terminal domain that are equivalent to inactivating mutations in the C *elegans ced-3* gene- prevent homophilic binding, highlighting the potentially primordial nature of this interaction.

Pich, E. M.; Pagliusi, S. R.; Tessari, M.; Tal-abot-Ayer, D.; van Huijsduijnen, R. H.; Chiam-ulera, C: Common neural substrates for the addictive properties of nicotine and cocaine.
Science 1997; 275:83-86

Regional brain activation was assessed by mapping of Fos-related protein expression in rats trained to self-administration of intravenous nicotine and cocaine. Both drugs produced specific overlapping patterns of activation in the shell and the core of the nucleus accumbens, medial prefrontal cortex, but not in the amygdala. Thus, the reinforcing properties of cocaine and nicotine map on selected structures of the terminal fields of the mesocorticolimbic dopamine system, supporting the idea that common substrates for these addictive drugs exist.

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[1] Klein, D.F.: Letter in the To and From section, this issue, page 2.

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[1] Potts, R.: Evolution and Climate Variability. *Science* 1996;273:922-923.

The Pathology of Neuroses and the EEA - page 6

[1] Stringer, C. & McKie, R.: *African Exodus,* London: Jonathan Cape, 1996, p. 150.

[2] Davies, M.: Culture and the Era of Evolutionary Adaptation, *The ASCAP Newsletter,* 1996; 9(9): 15.

[3] Davies, M.; Davies, H.; & Davies, K.: *Humankind the Gatherer-hunter,* Kent, England: Myddle-Brockton Publishers, 1992, pp. 18, 267-70.

[4] Stevens, A. & Price, J.S.: *Evolutionary Psychiatry,* London and New York: Routledge, 1996, p. 118.

[5] Davies, M.: Personality Traits and the EEA, *The ASCAP Newsletter,* 1996; 9(12): 12-14.

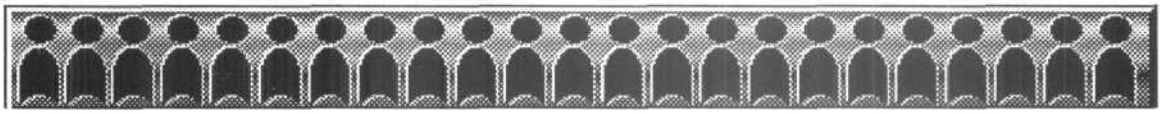
Weight Control Research as Model for Sociophysiological Research - page 19

[1] Friedman JM: Obesity: the alphabet of weight control. *Nature* 1997;385:119-120.

[2] Olney JW, Farber NB: Glutamate receptor dysfunction and schizophrenia. *Arch Gen Psychiat* 1995;52:998-1007.

[3] Erickson JC, Hollopeter G, Palmiter RD: Attentuation of the obesity syndrome of *ob/ob* mice by the loss of neuropeptide Y. *Science* 1996;274:1704-1707.

[4] Fan W, Boston BA, Kesterson RA, Hruby VJ, Cone RD: Role of melanocortinergic neurons in feeding and the *agouti* obesity syndrome. *Nature* 1997;385:165-168.



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