International Journal of Psychology and Counseling Vol. 1(9), pp. 154-186, November, 2009
Available online at http://www.academicjournals.org/ijpc

Review/Research

Risk starvation contributes to dementias and depressions: Whiffs of danger are the antidote

Robin Pope

Bonn University, ZEIb, Walter Flex Str 3, D-53113 Bonn, Germany. Tel: +49-228-731778, Fax: +49-228-3362881. E-mail: robin.pope@uni-bonn.de. Laboratory for Experimental Economics.

Accepted 10 July, 09

This paper's objective is to use SKAT, the author's Stages of Knowledge Ahead Theory of risk, to shed fresh light on the treatment and prevention of mental disorders. SKAT employs a broad definition of risk that allows for nice – not merely nasty – possibilities. SKAT is here shown to solve eight epidemiological puzzles left unexplained by our current theories and associated treatments for the demented and depressed. SKAT does so by enabling a decision model of mental health that puts centre stage why people (and other soft-wired animals) have brains – to make decisions under risk. To make good decisions (be healthy), brains need exercise. Brains get beneficial exercise from what the paper terms "whiffs of danger", namely sets of risks with the characteristics that the risks are 1) tiny, 2) varied, and 3) frequent. Brains deteriorate when there are shortfalls in such risk exercise. The paper terms such shortfalls "risk starvation". Those lacking a history of whiffs find normal mishaps too stressful and frequently become depressed. A lot of time with an inadequate amount of whiffs generates the endemic co-morbidity of becoming demented as well as depressed. Socio-economic cultural changes such as the introduction of unemployment benefits and old age pensions and increasing protection of women and children have had the beneficial effects of removing big challenges and big dangers and thus of prolonging physical longevity. But these changes also removed the tiny challenges and tiny dangers formerly faced by those sub-groups in the population identified as more prone to depressions and dementias. Unintentionally, these sub-groups thus were deprived of whiffs of danger, and suffered from risk starvation. In both drug and psychotherapeutic stress research and treatments of the depressed and demented, there should be injections of whiffs of danger to enhance the likelihood of enduring improvements. It is unkind and dangerous for people's brains to be treated with drugs while maintaining the modern socioeconomic culture of coddling parents and coddling college / university student counsellors, coddling unemployment benefits and coddling old age pensions. These coddles need to be complemented with whiffs of danger, tiny varied chances and challenges. These whiffs of danger need to be introduced in three forms: eliciting social security recipients' whiffs of danger in the form of little obligations to help the community; educating the poor and other sub-groups that believe closeting females at home endangers their mental health; and educating parents on the damage from overprotection. Overprotection prevents children from becoming inoculated against depression with sensible hope developed over a childhood in which they were allowed to experience numerous failures, not merely numerous successes from parents too closely engineering their environment. Research is required on the likely role of risk starvation in mental disorders other than dementias and depressions and in some physical illnesses.

Key words: Stress, whiffs of danger, decision, dementia, depression, risk starvation, risk, learning, hope, fear, risk-based emotions.

INTRODUCTION

For persons suffering from endocrine disorders such as diabetes and mental illnesses, there are three planks to our research and treatment programmes:
(i) Exercise and diet.
(ii) Drugs.
(iii) Coddling the person from fortune's nasty slings and arrows

While drugs and psychotherapies have had some short and medium term successes, they have had limited long term success, suggesting scope for a fresh tack. This paper argues for adding a fourth plank to have long term success in enabling these sufferers to grapple with stress, namely giving sufferers a particular set of risks. For this particular set of risks, the paper coins the name "whiffs of danger". For lack of whiffs of danger, the paper coins the name "risk starvation". The author's whiffs of danger – risk starvation theory of mental health and illness is developed with her umbrella theory of decision making under risk that she terms SKAT, Stages of Knowledge Ahead Theory.

The paper is not claiming that risk starvation is the cause of every mental disorder, or that injecting whiffs of danger can reverse every mental disorder, no matter how severe. That is, the paper does not offer whiffs of danger as a panacea. It does however provide evidence that in a range of circumstances, whiffs of danger have a role to play in the prevention and reversal of a number of mental disorders.

The preventive value of particular sorts of risks has been discerned in the field of immunology. Immune cells termed "T Memory cells" tend to combat pathogens more effectively when they have been exposed to low intense antigens, with different varieties at a certain frequency (Samson and Cairns, 1977). This paper presents evidence that it is not only the body that needs risks, but also the mind. So just as for a better immune system, the message is don't coddle your kid, let him play with "dirty" sand, so also, here the core message is, don't coddle your patient who has mental disorders – entice her to take risks.

In a healthy brain, a small stressor elicits a small risk experience and inoculates against a subsequent big stressor that elicits a big risk experience. But the discovery being reported in this paper is not limited to inoculation benefits against big stressors. Rather, the discovery being reported here is that coddling people from the small stressors, renders their risk-starved brains so morbid that they cannot even make good decisions in the face of *small* stressors.

The discovery is that societal changes over the last century making people's lives safer has not merely had the beneficial effect of increasing longevity. It has also had the detrimental effect of depriving particular groups of people of enough small stressors. In other words it has deprived people of reaping enough whiffs of danger. When deprived, these groups suffer risk starvation. Even small stressors with nasty surprises can yield depression, and an extended period of risk starvation can yield dementia.

To trace the discovery, epidemiological and experimental evidence needs to be seen from a fresh perspective, that of the author's "whiffs of danger – risk starvation theory of mental health and illness that is embedded in the author's umbrella theory of choice under risk and uncertainty that she terms, SKAT, the Stages of Knowledge Ahead Theory.

Part 1 concerns the definition of a stress. It explains why a stress perceptually defined is the same as a risk. It explains the advantages of using a dynamic definition of risk wherein there is no static benchmark. It explains the advantages of using a broad definition of risk wherein chances as well as threats can be considered.

Part 2 begins with a diagrammatic depiction of the overarching decision theoretic model of mental health being used in the remainder of the paper and explains building blocks of the author's SKAT's decision making approach.

Parts 3 and 4 present the author's whiffs of danger / risk starvation theory and delineate affinities to and differences from some other theories/therapies.

Part 5 illustrates how to recode stress studies to identify risk starvation and its reversal via whiffs of danger in experimental data.

Part 6 surveys epidemiological evidence that protection from stress (that is, risk) may have increased the incidence of dementias and depressions in rich countries.

Part 7 canvasses research and treatment agenda.

CONCEPTS DEFINED TO BUILD A FRESH MODEL OF STRESS

A perceptual definition of stress

Stress has been discussed for thousands of years, Cooper and Dewe (2004). It entered our current conceptualisation of mental illnesses via Selye's (1974) analogy with physical material being strained (Hüther, 1996). What then is a useful definition of stress for us taking new fruitful steps in research and treatment of mental disorders?

McEwen and Wingfield (2003) adhere to a Selye-style definition of stress as a threat to homeostasis. A threat is not something that has happened. It is a perception of the animal experiencing the stress about what in the future might– or might not – happen. The animal's perception of what might happen is not necessarily shared by the ("ideal", "objective") researcher or clinician. The researcher or clinician might for instance deem that animal paranoid (in perceiving essentially non-existent threats), or foolhardy (unable to perceive the seriousness of a threat).

The concept of stress can be extended to include all bodily changes arising from the stressor. In this case the concept of stress includes factors like tearing a muscle a little, enabling it to grow bigger and thus constituting good stress; tearing a muscle a lot and thus constituting bad

stress. If we were here to use this extended concept, stress would comprehend and to a degree blur, the distinctions among numerous phenomena including perception and biological stress response to perception. Since for analysing mental disorders, distinctions between these phenomena need to be preserved, it simplifies the exposition not to take this route. It is not taken in this paper, just as it was not taken by Selye. Like Selye, this paper employs a perceptual concept of stress.

Stress, perceptually defined as a possibility of what might or might not happen, distinguishes this perception from the emotional and physical effects that the perception engenders. To illustrate, take an experimental subject forced to view a horror film as in Mian, Shelton-Rayner, Harkin and Williams (2003). The viewing may elicit stress in the form of a perception that a nasty outcome may befall the film's heroes. In turn the threat to the heroes may engender disagreeable fear that the nasty outcome may befall those heroes. The disagreeable fear is distinct from the stress itself. Likewise, under this paper's perceptual definition of stress, the biological stress responses engendered by the fear are distinct from the stress.

A stress equals risk definition

Once we limit stress to being the animal's perception of its future possibilities, the concept of stress is a concept of risk. This is because a set of possibilities constitutes a risk. A threat to homeostasis is a risk of homeostasis.

A dynamic definition of stress

Homeostasis as a benchmark has relevance in considering some effects on the body like temperature – since there is a fairly limited fixed (static) range within which the body can remain healthy. But to use it in research and treatment of mental disorders, is to ignore the entire advantage of an animal being soft-wired, namely to let its decision making evolve in response to its ever changing environment. The static benchmark of homeostasis is a residue from the behaviourist era in which events inside the brain (and thus all of decision making). It was excluded from medical research on the grounds that people's introspective reports about their thinking and deciding were unreliable.

This paper therefore employs a more dynamic definition of stress. It drops reference to any static benchmark. It defines stress as any change in the animal's conscious or more often unconscious, perception of the future, caused by any change in its environment.

A dynamic definition of a stressor

Once we limit stress to being the animal's perception of its future possibilities, a stressor is something likely to elicit in the animal a formulation of a set of possibilities. A stressor is something that is likely to stimulate the soft-wired animal to perceive that its future is uncertain – that its future has more than one possibility.

This means that a stressor is defined in this paper as any change in its environment that the animal perceives. As identified in that landmark review of biological stress, Hüther (1996), societal structure and parental behaviour are crucial ingredients in the animal's external environment. Thus societal changes are a key set of stressors to be researched. In a like spirit, Kagan (1994) underscores the role of society in childhood development.

The uneliminable subjective aspect of stressors, stresses/risks

The stressor's magnitude and other characteristics should be specified with reference to the researcher/clinician's appraisal, not with reference to the person having the stress. This avoids the more severe pathological distortions of their subjects/patients. It allows us to distinguish the stress a stressor elicits in the "average" person from that elicited in a below or above average person in some respect.

But this does not render appraisals of stressors entirely objective. This is because how we wish to objectively classify a stressor is with respect to the stress it elicits in the "average" person. But when a stress is perceptually defined as in this paper, it concerns what may happen, not what has happened. To infer what set of possibilities the average person would formulate, scientists rely on their own empathetic powers (and in the case of investigations of non-human animals, apply anthropomorphism), to introspect and subjectively judge. The stress literature is silent on the subjectivism. Implicitly, having subjective judgments accepted by co-researchers is a matter of shared world views, at times bolstered by only partially objective evidence. The bolstering evidence can approach, but can never reach, objectivity. This is because each stress concerns a set of possibilities, of which in the future at most one will happen. Looking at a single possibility that did happen is insufficient for ascertaining what the other possibilities in the mind of the "average" person were. From large data sets of what happened, with assumptions, we make inferences. Generally, the more complex the situation, the more subjective our assumptions in classifying stressors, and thus also the more subjective our classifications of the stresses/risks experienced by that "average" person.

Non-dichotomous definition of stress

This paper's dynamic definition of stress avoids a false dichotomy that limits research into biological stress responses. This is the false dichotomy of a brain being either healthy or morbid. There is no known upper limit from a beneficial environment to the brain's development, for example, to how good a decision-maker a person can become. It may well be that people can ever improve in overall decision making abilities. The non-dichotomous definition entices us to see brain health as having many levels from very good to very bad. When it is appreciated that there are many levels, some reversals of mental disorders simply amount to reacquiring capacities lost by temporary lack of use, or never gained through a focus on acquiring other capacities. The non-dichotomous definition thereby helps overcome unwarranted resistance to the notion that mental disorders like dementia may be reversible.

This paper's non-dichotomous definition of stress entices biological stress response research on those rated as exceptionally good decision makers. To date in understanding mental disorders, there has been virtually exclusive focus on those rated as inferior to the norm in their decision making capacities. In judging good decision making and identifying whom to select as superior decision makers, a pioneering work is Janis and Mann (1977). Judging what is good decision making involves more than assessing isolated aspects of decision making like memory, computation speed and so forth. It involves attention to the entire set of stages leading to a decision.

A broader definition of stress

In a Selye style definition of stress, the possibilities are of nasty outcomes. This mirrors the narrow negative definition of risk as nasty possible outcomes. Once reference to homeostasis is dropped, the definition of stress can be broadened to the broad definition of risk. The broad definition of risk allows for possibilities to be nice, or neutral, not necessarily exclusively nasty. This paper defines stress as any event stimulating perception of future possibilities. To illustrate, let somebody called Kai see another person entering his office. Then this paper's definition of stress includes all three of the following ways that Kai might formulate his set of possibilities:

1. Either this person will harm me or not – a set of two possibilities comprising one nasty and one neutral possible outcome.
2. Either this person will help me or not – a set of two possibilities comprising one nice and one neutral possible outcome.
3. This person may help me, or harm me, or neither – a set of three possibilities comprising one nice, one nasty and one neutral possible outcome.

If the person sees only one possibility, this is at the certainty end of the possibility continuum, at the opposite end from complete uncertainty. This is a case where the outcome is guaranteed. If that sole possibility is a nasty outcome, it is not what we normally term a threat (something nasty that might not happen), but a dread (something nasty that is sure to happen). Where there are two or more possibilities, the person sees the outcome as uncertain.

In referring to a set of possibilities, this paper will sometimes substitute for the term "threat", which connotes only nasty outcomes, the term "challenge". The term challenge has broader and more positive connotations. The term challenge allows for the fact that sometimes the brain improves in meeting difficulties and averting them or having luck that the nasty possibility do not transpire or else enduring their nasty outcomes.

Using a broad definition of risk does not force researchers and clinicians to ignore the different ramifications of nice and nasty possibilities. Because of these different ramifications, there should, as advocated in Curtis and Cicchetti (2007), continue for instance to be resilience and coping research concentrating on nasty outcomes. But this research can now be cross-fertilised by findings concerning complex stressors that generate the possibilities of nice and neutral as well as nasty outcomes.

Definition of the biological stress response

This paper's dynamic definition of stress allows the biological stress response to be in the spirit of the seminal re-conceptualisation of why soft-wired brains evolved, Hüther (1996, p. 570). The biological role of the stress response … [is] … a trigger for the adaptive modifications of the individual behaviour to the requirements of an ever-changing world.

Thereby Hüther opens the notion of adaptation to one of evolution, not limited to adaptation as reversion to homeostasis. His re-conceptualisation avoids the connotation that all stress is pathological. It opens the way for eustress, for beneficial stresses to be incorporated more consistently than was previously the case.

However in his examples, Hüther focuses on adaptation in the face of small stressors (yielding small threats) serving as conditioning against bigger stressors (yielding bigger threats). Hüther's examples thus limit the concept of stress to external changes that the animal decides have nasty possibilities and limits the concept of eustress to reducing harm from big stressors. Hüther's examples provide no picture of eustress enabling the brain to reach a higher level of performance. There

remains from Hüther's examples an implicit static benchmark of what is a healthy brain.

Hormetic applications

Hormesis concerns the benefits or damage from progressive dosage increases either from injections or hormonal releases, of a single substance. Hormesis occurs if, compared to the control level of no increment above normal of that substance in the body, there are three phases as the dosage rises, first increasing benefits, second decreasing benefits, and third increasing damage. In discerning hormesis for a very wide range of substances, Calabrese and Baldwin (1997) focus on the effect of each dosage level separately, not on temporal sequences of doses in which small doses precede subsequent big doses.

By contrast, in Calabrese et al. (2007) the focus is on sequences of small quantities of hormonal releases followed by big hormonal releases. This is because hormesis is being applied to the biological stress response literature – in the form of a proposal for standardised definitions for hormesis concerning adaptation and conditioning. The examples given of biological stress responses all concern small releases averting pathologies from subsequent bigger releases.

Definition of the biological stress responses in this paper

Hüther (1996) and Calabrese et al. (2007) both make the important step forward of highlighting beneficial biological stress responses, benefits that remained in the background in Selye. But in their examples, the only benefits from biological responses to small stressors are to ward off bigger damage caused by bigger stressors. On the biological stress responses, both the evolutionary re-conceptualisations of Hüther and the Calabrese et al. hormetic re-conceptualisation, thereby retain an implicit stationary benchmark. The best that an animal's experiences with small stressors can do is to prevent long term deterioration from big stressors.

This paper takes the next step. It not merely drops reference to homeostatis, but into the definition of stress itself, introduces possibility that the brain can improve. The paper's new definition refers to nice and neutral possibilities, not merely to nasty, possibilities. Thereby it avoids any implicit new benchmark that small stressors do nothing more than avert pathologies from moderate and big stressors.

This paper's new definition of stress takes the next step of enticing investigation of biological stress responses to eustress and to what are the commonly encountered stresses, namely complex stresses in the sense that each has a range of nice, neutral and nasty possibilities. It takes this next step in the evolutionary spirit that Hüther introduced. Evolution is not a concept of exclusively risks of downside change. Indeed we mostly look at evolution positively. That evolution has connotations of improvements that have a chance of enduring, is a connotation captured in this paper's new definition of stress.

The paper's new definition (re-conceptualisation) of stress and thus of the associated biological stress responses, entices fresh theorising/hypothesising and investigations on biological stress responses. The prior seminal re-conceptualisation of the biological stress response in Hüther (1996) also detailed specifics of the reinterpretation of current findings concerning the neurochemical systems associated with his re-conceptualisation. This paper by contrast provides no details on the associated neurochemical systems likely to be triggered by different sets of stressors eliciting risks. It does however provide details on the prior step to such biological stress response studies. This is the step of recoding situations in past studies with regard to their risk characteristics, and the associated findings on mental disorders and reversals of mental disorders.

Definitional summary

As Calabrese et al. (2007) observe, recognising when a single entity is being called by multiple different names is valuable for avoiding confusion and mistakes and for cross disciplinary fertilisation. Table 1 alerts us to six identities.

Table 2 compares and contrasts the perceptual definition of stress in this paper with that in Selye (1974), Hüther (1996), Lupien et al. (2005, 2006) and Calabrese et al. (2007). Table 3 lists six advantages of going beyond Selye and also of going one step further than Hüther and Calabrese et al. implicitly propose, and shifting to this paper's broad and dynamic definition.

Table 1. Identities.

When stress is *perceptually* defined
1 having a *stress* is experiencing a *risk*
2 the *perception of a threat* is *the formulation of a downside risk*
3 *the perception of a chance* is *the formulation of an upside risk*
4 *having a stress* is *the perception of possibilities*
5 *experiencing a risk* is *the perception of possibilities*

When both a stress and a risk are *broadly* defined as in this paper
6 a *set of possibilities that may contain nice, neutral and nasty possibilities* is a *risk*
7 a *set of possibilities that may contain nice, neutral and nasty possibilities* is a *stress*

Decision-making
8 *well done* is a *healthy brain*
9 *exceedingly well done* is a *superior brain*
10 *badly done* is a *morbid brain*
11 *improvements in decision-making over time are reversals of one or more forms of mental disorder*

Table 2. Contrasts in what is defined as a stress, a stressor and biological stress response

Stress/Risk

1 Homeostatic benchmark in Selye, not in Hüther (1996), nor in Calabrese et al. (2007), nor in this paper

2 Threats (nasty possibilities) or guaranteed future nasty outcomes are almost the sole foci in Selye, Hüther and Calabrese et al. (2007), whereas in this paper there is equal focus on chances as well as threats – ie a focus on stresses/risks that have nice and neutral possibilities, not primarily on those that have nasty possibilities.

Stressor – what elicits Stress that is, risk

3 Stressors that animals process to perceive as having guaranteed nasty outcomes are the investigation focus in Selye and in Calabrese et al. (2007). Whereas in Hüther (1996) and in this paper there is almost no focus on riskless stressors (with guaranteed outcomes). Rather the focus is on stressors that animals process to perceive as yielding threats in Hüther, and in this paper, as yielding either threats or chances or both.

Biological Stress/Risk Response

4 Almost all responses are pathological under the Selye-style definition of stress. The message is virtually that unless we to return to the more relaxed traditional lifestyle, so as to reduce our release of catecholamines down to the level of those in traditional lifestyles, evolutionary selection forces may extinguish us, James and Brown (1977). The message is sometimes only so dire as regards chronic stressors, Lupien et al. (2006). By contrast Hüther (1996), Lupien et al. (2005) and Calabrese et al. (2007) define biological stress responses more broadly such that small stressors can reduce pathological responses to bigger threats from moderate and big stressors. This paper differs from Hüther (1996), Lupien et al. (2005) and Calabrese et al. (2007), in focussing on the scope for biological stress responses to be improvements in their own right, not simply ways of averting pathologies engendered by even worse stressors.

Table 3. Six Advantages of this paper's dynamic, non-dichotomous, broad, definition of stress/risk.

Dynamic – focus on Change Without any Static Benchmark

1	Change opens stress research to focussing on why we have brains, namely to deal with change
2	Change opens stress research to the concept of an evolving, ever-changing animal brain. It does not postulate any upper limit to how good a decision maker can become, and so deters researchers from making a dichotomy of brains being healthy and morbid. It entices them instead to see brain health as having a gradation in each capacity from very bad to very good.

Broad – focus on Chances and Challenges

3	Breadth opens stress research to the fact that in many natural complex situations, those with healthy brains (not unrealistically hopeful and not unrealistically fearful), decide that the possibilities often include nice, not merely nasty, ones. It generates lop-sided overly simple hypothesising and overly simple experimental set-ups if we focus on those events for which animals decide that the possibilities are exclusively nasty.
4	Breadth entices investigation of biological stress responses to eustress, largely neglected to date.
5	Breadth reduces the dangers of a reductionistic-mechanistic view of stress-induced pathology, against which there are warnings in both Hüther (1996, p570) and in Curtis and Cicchetti (2007).
6	Breadth entices focus on the sequel decision making phase whereas in the Selye-style definition, there tends to be a jump from possibilities to mental disorders, not keeping salient the fact that mental disorders are bad decision making. Good decision making involves an analysis of both threats (nasty possibilities) and chances (nice possibilities), not a lop-sided focus on threats alone.

DECISION MAKING, THE BRAIN AND SKAT

The current model of stress-induced mental illness

Research and treatment for stress-induced mental disorders involves an implicit over-arching cause-effect model. That currently underlying much current research and treatment is depicted in Figure 1. It lacks reference to decision making – reference to why we have brains and how we discern if our brain is morbid, namely if it makes bad decisions.

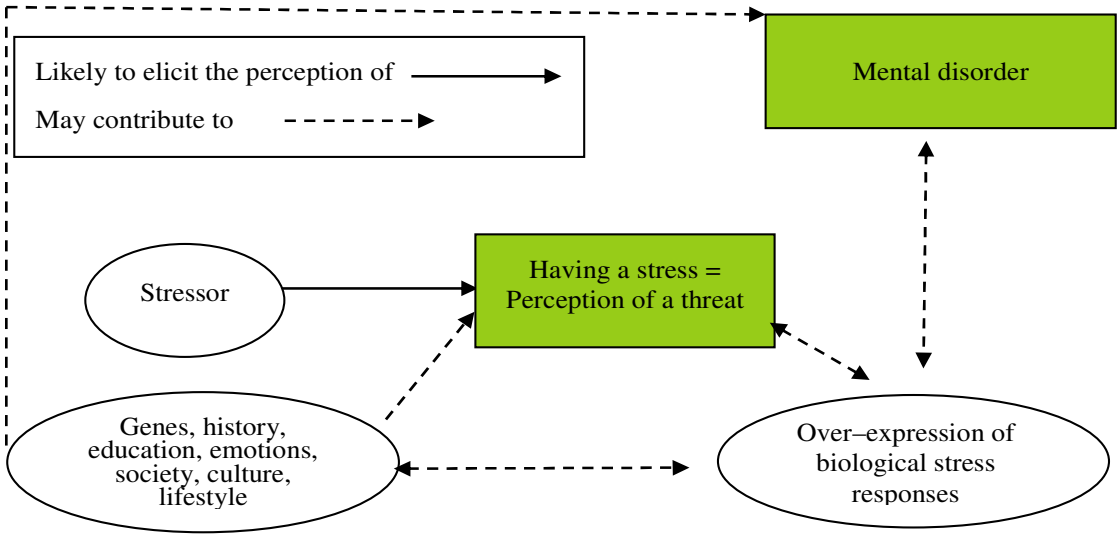

Figure 1. Current model or overarching theory of stress and mental disorders.

A fresh model

Figure 2 is a bird's eye view of the author's model of mental health and illness. It indicates where a broad dynamic definition of stress can take us, to highlighting decision making and when a single entity is being called by multiple different names. It adds fundamental connections missing in Figure 1 – the fact that experiencing a risk (having a stress) is a matter of formulating possibilities – and thus that having a stress is a part of decision making. In Figure 2, note the three new building blocks:

(i) Formulating a *set* of possibilities, that is, a risk – and is part of (ii).

(ii) Making decisions – and this is aided by (iii).

(iii) Perceiving particular sets of risks.

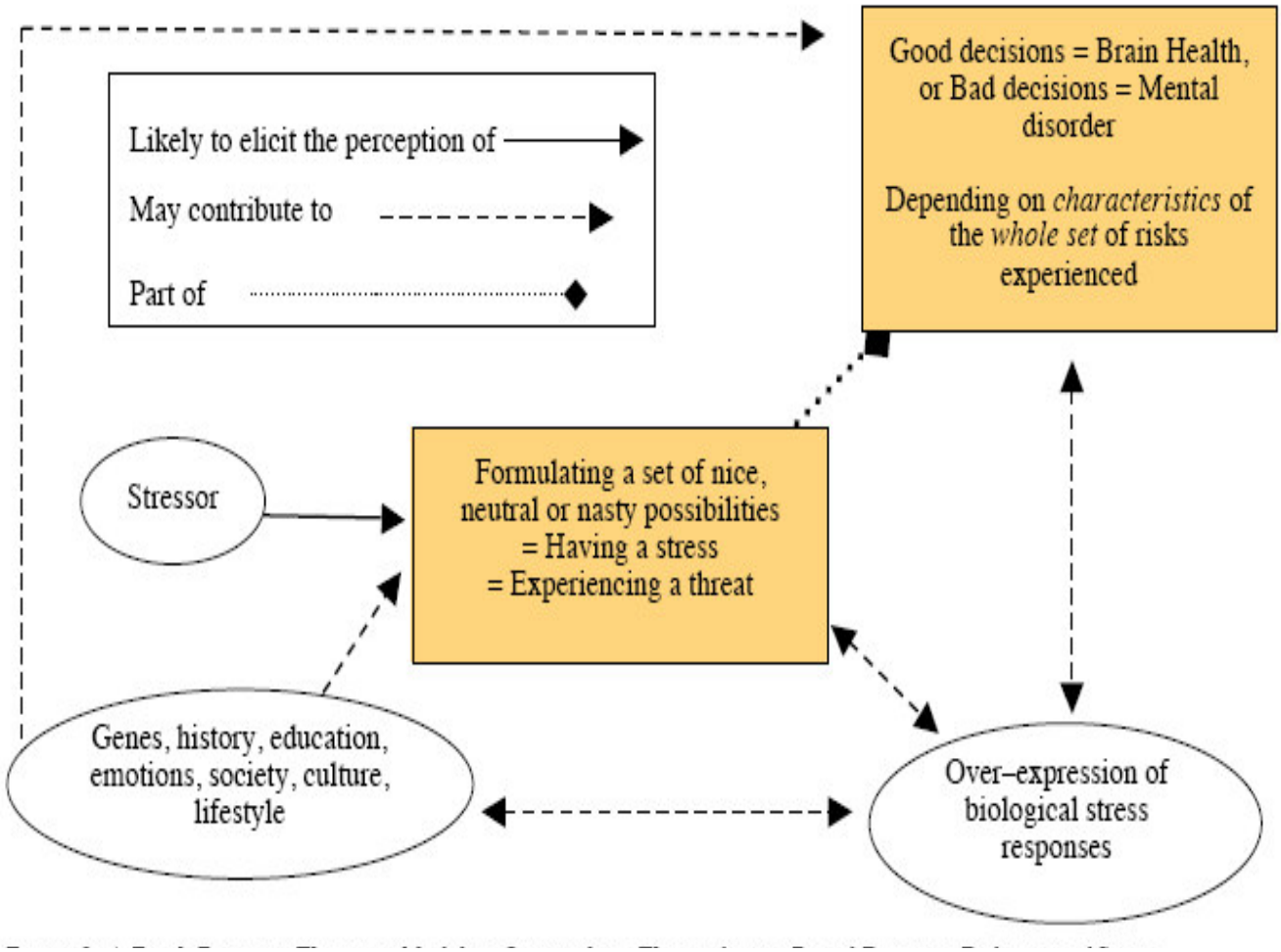

Figure 2. A fresh decision-theoretic model or overarching theory from a broad dynamic definition of stress.

Risk

To help link research on biological stress response with that on decision making, this paper will from here onwards, mainly use the term risk, rather than the term stress. Bear in mind therefore that a (perceptually defined) stress is simply another word for a risk.

The evolving stages of knowledge ahead

Risk refers to the future (Pope, 1983, 1985, 2004, 2005). Decision makers judge some dimensions of how risky their future is from past frequencies of nice, neutral and nasty outcomes (events). Past outcomes are in the

external environment, whereas risks are internal, in the decision maker's brain.

People experiencing a risk recognise that in their current stage they do not know something, but anticipate learning this at a later stage. A risk implies an anticipated change in our knowledge ahead – a new stage when we will in the future have learned something that currently we do not know. Unless we might learn something knew, we face no risk. This reveals that it has been a mistake in economics and finance – and also in psychology and medicine – to analyse a risk as if it were a bad event, as something static (without an anticipated change in knowledge in the future).

In economics the mistake generated a famous puzzle that remained unresolved for nearly forty years of which the following is an example. Many people experience risk-based emotions prior to a job interview. Some of these are enjoyable such as the following: freedom from boredom; challenge in attempting something interesting and difficult and thus maybe not doable as difficulty implies risk, excitement, thrills, hope, faith, trust, wonder and curiosity. Some of these risk-based emotions are disagreeable such as the following: fretful feelings, insecurity, anxiety and crippling fear.

Now risk-based emotions arise from the possible good outcome (being hired) and the bad outcome (not being hired) interacting with (complementing) each other. How, asked von Neumann and Morgenstern, could good and bad outcomes interact. Interaction they declared is a contradiction since these outcomes are mutually exclusive. Unable to find the "higher level" to discern why this is merely a pseudo contradiction, they left this task to future researchers, von Neumann and Morgenstern (1947, 1953, 1972, pp. 628-632).

Pope (1985) shows that a failure to recognise that risk cannot be analysed statically caused the pseudo contradiction – that the higher level needed involves progressive stages of knowledge ahead. To analyse risks stage. These people have decided to be interviewed, but they do not know if the outcome will be success (hired) or failure (not being hired). This means that in the pre-outcome stage, the good outcome of being hired and the bad outcome of not being hired can interact in interviewees' minds, both being possible. But eventually, during what we may term the post-outcome stage, these people will have learned whether or not they succeeded and were hired. Only at this later post-outcome stage are the good and bad outcomes mutually exclusive and no longer both possible. One has occurred rendering the other no longer possible. See Table 4, which includes some risk-based emotions ensuing in the post-outcome stage through memory of what was previously possible.

This paper applies SKAT to building and maintaining a healthy brain. SKAT has been applied in financial and emotional decisions under risk, (Pope, 1983, 1995, 2001, 2004, 2005, 2006a) and Pope, Leitner and Leopold-Wildburger (2006, 2009), Pope, Selten, Kube and von Hagen (2008).

Table 4. The anticipated progression in knowledge ahead from the pre- to the post-outcome stage.

Pre-Outcome Stage	**Post-Outcome Stage**
Both positive *and* negative risk-based emotions from contemplating whether the good or the bad outcome will occur	*Either Or – Mutually Exclusive Outcomes and risk-based emotions* from memory of the pre-outcome risk
Both freedom from boredom, challenge in attempting something interesting/difficult, excitement, hope, faith, trust, wonder, curiosity	*Either* relief, elation, exhilaration at the good outcome occurring, with the positive emotions heightened by memory of the prior danger of a bad outcome
and fretful feelings, insecurity, anxiety, excess tension, crippling fear.	*Or* disappointment, deflation at the bad outcome occurring, with the negative emotions deepened by memory of the prior chance of a good outcome

Risk and the brain

A risk processing brain enables.

(a) Registering signals.
(b) Distilling information from these signals.
(c) Analysing the information.
(d) Deciding.

In Figure 3 a soft-wired animal, a tortoise, faces a succession of “Nows” that yield a succession of risks, decisions and anticipated changes in knowledge ahead from the pre- to the post-outcome. In panel 1 the tortoise has decided that it sees a new thing. In panel 2 it has decided that the new thing is either food (a chance) or not food (maybe either something neutral, or even nasty (that is, a threat), but faces the risk of not knowing which. In panel 3, it has decided that it can go to the new thing or stay, but faces the risk of not knowing whether it will decide to go to it. In panel 4 it has decided to start walking.

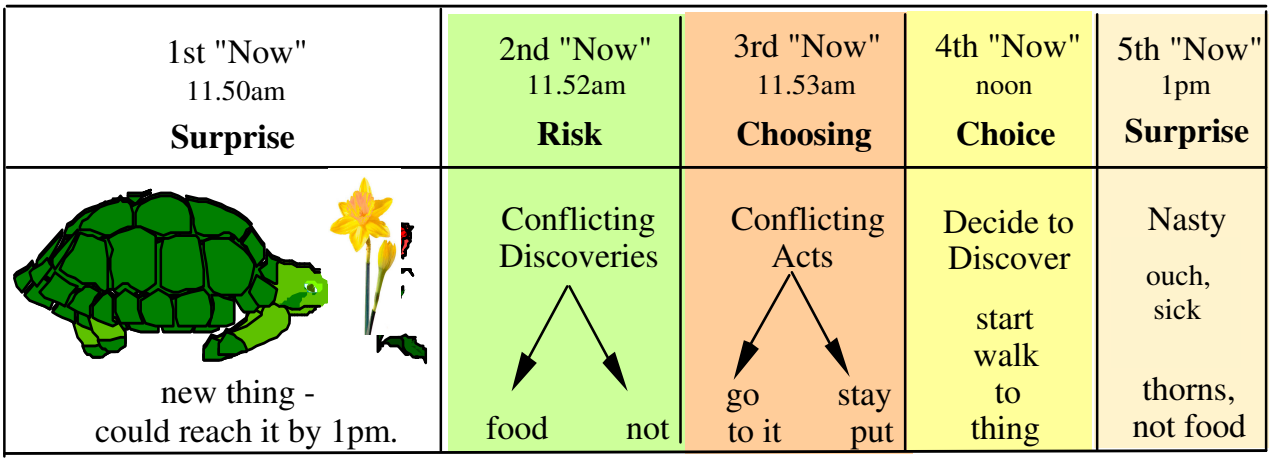

Figure 3. Risk is anticipating, consciously or unconsciously, a change in knowledge — a discovery.

This discovery walk decision may well be influenced by its emotional mood, not merely by “objective” anticipations, Lerner and Keltner (2001), and Loewenstein, Weber, Hsee and Welsch (2001). While on the discovery walk, the tortoise still faces the risk of whether the new thing will turn out to be food. In panel 5 the succession of risks is past: it has discovered that eating the daffodil makes it ill, is not food.

Sensual processing of risks (chances/challenges) in the Tortoise’s 1st and 5th “nows”

In a conscious decision of whether to discover, we understand that we process risks — that as in the middle panel of Figure 3, we may formulate conflicting future projections, analyse these, decide. But we inadequately connect this understanding with the findings from cognitive psychology and neurobiology of the (largely unconscious) analogous risk processing that we (and other animals) do to "feel", “hear”, "see". We need to make the connections and keep them salient. Sensual risk processing is fundamental to brain exercise and health. Higher level risk processing comprises an animal’s analyses and actions undertaken after having decided how to decode the sensory signals. Instances of higher level risk processing include those researched in cognitive psychology under the terminology of “problem solving” (Simon, 1979). Other instances of higher level risk processing are depicted in the middle panels of Figure 3. Sensual risk processing underlies / initiates all higher level risk processing.

How does the tortoise get the information and decide in its 1st “Now” that it sees a rose at a distance reachable by 1 pm? It gets this information by choosing amongst conflicting projections of:

a) What light signals it received.
b) How to decode these signals into informative object-edge distinctions at a given distance.

The tortoise must then decide what it sees. Figure 4 delineates some of the sensual risk processing stages left implicit in that first panel of Figure 3.

Good visual risk processing helps distinguish a more

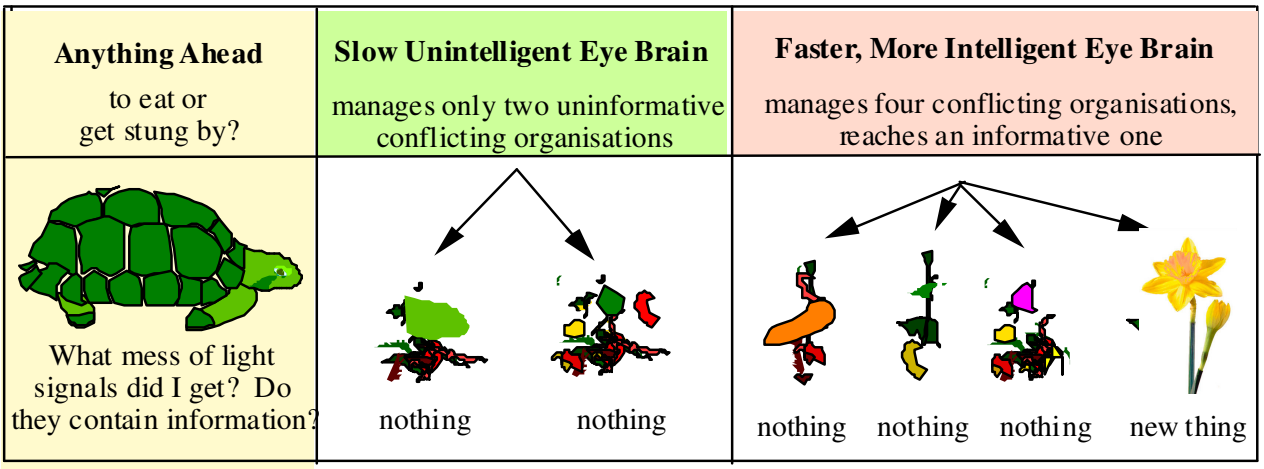

Figure 4. Risk processing in registering and organising light into information — seen objects.

intelligent tortoise able to see the rose, from a less intelligent one unable to see it. Such visual risk analyses involve many parts of the (human and tortoise) brain. They are complex: artificial intelligence is making slow progress mimicking them. We glimpse our own unconscious risk sound processing when we "hear" one word, and then later decide we "heard" another. That is, from what we later "hear" the speaker saying, we decide that we earlier “misheard”. Risk processing of tactile signals (for the tortoise to decide that it was stung by the rose), of olfactory signals, and of depth signals (for sea creatures with a lateral line) is similarly complex (Plachta, Hanke and Bleckmann, 2000).

Seven principal stages of knowledge ahead under risk

In evaluating its decision to explore, the nasty outcome may reduce its readiness to explore in the future. Table 5 itemises as 1 to 7 the stages of knowledge ahead. The first six of these are depicted in Figure 3 with stages 3 and 4 combined in the third “Now”. Figure 4 provides a fuller depiction of stage 1. Soft-wired animals have the brain plasticity to reason / learn from experiences – to become better decision makers. Accordingly Table 5 also includes the sequel stage 7 of evaluating the outcome,

Table 5. Seven stages of knowledge ahead.

Stage 1:	Pre-Discernment if there is a change, if something new has appeared – ends on discerning yes.
Stage 2:	Pre-Formulation of possibilities a chance, neutral or a threat – ends on formulating a set of possibilities.
Stage 3:	Pre-Discovery of choice set – ends on discovering alternative acts for responding to the new thing.
Stage 4:	Pre-Evaluation and Choice among alternative acts – ends on evaluating.
Stage 5:	Pre-Outcome – ends on learning the outcome of the chosen act.
Stage 6:	Post-Outcome but Pre-Evaluation of Choice – ends on evaluating the choice.
Stage 7:	Evaluation of choice.

that is, the learning process for future decisions. Good decision making requires stages 1-4 and 7 to be performed well. Doing a good evaluation in stage 7 involves having registered whether well-being differed from that anticipated when during the pre-outcome and post-outcome stages 5 and 6 compared to what the animal anticipated in choosing compared to what the animal anticipated in choosing that act.

Mental disorders are defects in decision making in one or more of the above seven stages. The defects in decision making can arise from people facing sets of risks with a lack of inappropriate characteristics for good learning

The author's of danger / risk starvation theory

Variety in risks means variety over the spectrum of risks that the brain is devised to process, namely for humans (and many, arguably most, other animals), this spectrum includes sensual, physical, intellectual, psychological, spiritual, ethical and social risks. For a set of risks with the three characteristics of:

(i) great variety;
(ii) high frequency;
(iii) each individual risk tiny, we here coin the name whiffs of danger (of chances/challenges).

For inadequacy in whiffs, we here coin the name risk starvation. Note that whiffs of danger are defined narrowly. The term "whiffs of danger" does not refer to all risk sets, only to sets of risks of risks with all three characteristics. Risk starvation is likewise defined narrowly. An animal does not avoid risk starvation simply by experiencing any set of risks. The animal only avoids risk starvation by having adequate whiffs of danger.

What is a risk set with these three characteristics is subject to individual differences. Further experience, including education and culture, alters which risks are perceivable and of those perceived, which are experienced as tiny, even too tiny to be whiffs, and which are experienced as alarmingly huge, too large to be whiffs.

Most sets of risks lie outside the set of risks defined as whiffs of danger. In particular what do not qualify as whiffs of danger, are sets of big unvaried risks. big unvaried risks beset disproportionately those in the lower socioeconomic strata such as chronic domestic violence, marital conflict, sexual abuse, compulsive gambling, chaotic lives from unrealistic drug impaired parents or spouses. One must be careful not to conflate whiffs of danger with these other sets of big unvaried risks that can result in a damagingly high allostatic load (McEwen, 1997; McEwen and Seeman, 1999; Lupien, King, Meaney and McEwen, 2000, 2001) and perhaps a sub-sized hippocampus (Lupien et al., 2007).

Deficiencies in whiffs

In a modern city in a rich country behind the sensually abstracted walls of offices / homes seated on a chair, we lack the tiny sensory and physical risks faced each nanosecond by the tortoise in Figure 3 and by our ancestors who lived in the wild and so had to check where they put their feet and whether the item ahead might be food or water or a poisonous thorn. The variety and frequency of tiny intellectual, ethical, spiritual and social risks that we rich modernists encounter are also often far below those of earlier environments in which our brains evolved. This is because we have modified our environment to make it safer, to have more "controlled" lives, and in so doing deprived some sub-groups of whiffs of danger.

The whiffs of danger theory / therapy

For where a person lacks whiffs of danger, this paper coins the name risk starvation. Whiffs of danger, according to this theory, enhance brains, while risk starvation damages them. For this, evidence is discussed in part 6. A side theory is that sometimes brain damage can be alleviated / reversed by injecting components of whiffs of danger that an individual lacks.

Emotions and associated cognitive anticipations affect which risks (chances/challenges) get attention, which decisions get considered and which decision is chosen (Simon, 1967; Damasio, 1994; Kagan, 1994; Lerner and Keltner, 2001; Loewenstein et al., 2001; Izard, 2002; Camille et al., 2004; Blair, 2006). Improved performance – good decision making – is aided by an appropriate spectrum of positive and negative emotions and associated cognitive anticipations (Clynes, 1968, 1973, 1978, pp. 107-170, 1988; Moore, 1992).

A single act (eg one involving physical risk) chosen can by luck turn out well or badly, and may have no relevance for a different sort of act (eg one involving a social risk). Learning good decision making thus requires the animal to have experienced more than one risk – that is, requires it to have experienced sets of risks – and also to have experienced more than one sort of risk – requires it to have experienced different sorts of risks. People need to face numerous risks, make numerous choices, experience many good outcomes of their choices and many bad outcomes of their choices, and do numerous evaluations of their choices, in order to become good decision makers, and in order to stay good decision makers.

The experience of good outcomes following bad outcomes (that happens to people who face enough risks), enables people to build up hope as against learned helplessness. It avoids them being unduly fearful and unduly preoccupied about whether any particular risk might turn out badly. The risks cannot mainly be big, or even those who survive are too jostled to learn from the proportions of good and bad outcomes what are good decisions. Nor can the risks be unvaried, or the person is uneducated in the actual variety of risks that life entails. Table 6, whiffs elicit such an appropriate spectrum in that their three defining characteristics aid realistic learning.

Table 6. The three characteristics of whiffs of danger that aid his decision making.

Each risk is tiny	1 Other things equal, he has a high enough survival probability to warrant him learning. 2 He is not too emotionally distracted by a great chance, or a great danger, to act and learn. 3 Among tiny risks there is a sufficient proportion that are of short enough duration for him to get the rapid feedback that facilitates his discerning of actual cause-effect chains and thus his realistic learning.
Great variety	4 He encounters the varieties of decisions for which his brain is designed which in the case of humans include sensual, physical, intellectual, psychological, ethical, spiritual and social risks, each of which needs practice, and each of which, we may anticipate, elicits different stress effects, Oishi et al. (2003) 5 Variety avoids the physiological adverse stress build-ups that occur when his risk taking is concentrated on too few aspects of his life, as when a depressed person gets nearly all his risks from social gambling. 6 Variety increases the likelihood that he finds ways of slaking his appetite for risk and novelty and brain exercise without needing to choose foolhardy acts. 7 There is a degree of independence amongst the external conditions. This aids in generating a mix of nice, neutral and nasty surprises, and such a mix contributes to him having emotional balance. It precludes the “learned helplessness” of Martin Seligman’s dog (Seligman 1975) whose laboratory set-up artificially excluded this mix and thus excluded the dog Seligman from discovering that taking risks sometimes brings success, and thus hope remains.
High frequency	8 The cavalcade of new chances / challenges deters him from being obsessed by any individual past nice or nasty surprise, or on any individual future chance / challenge, and thus aids his overall perspective. 9 With many new tiny risks to attend to all the time, he does not cling to old higher aspirations for too long if encountering bad luck. Take for example someone who anticipated a life-long high paying prestigious post, but then is dismissed after a year. The high frequency of whiffs help prevent him getting stuck with gloom and depression and perpetual unemployment through failure to seek another job, as could happen if his family or the unemployment system is too financially supportive. Instead, if he is absorbed in enough other little risks (chances/challenges), these distract him and help him adapt over time to a feeling of equanimity with his new situation. 10 The many risks recently encountered keep his brain exercised. 11 The many risks encountered entice him to filter and aggregate enough and thus deter him from becoming too pre-occupied emotionally or intellectually with individual risks and as a consequence too focused on the danger side of each risk – or on its chance side 12 The many risks recently encountered furnish him with information from an appropriate spectrum of nice, neutral and nasty surprises with their associated risk-based emotions, and thus assist in realistic learning. 13 Note that what is high frequency is specific to the individual. It is not a higher frequency than, given his risk filtering capacity, enables him to process through to effective action a satisfactory number of risks. Where he has an inadequate risk filtering capacity in a situation, his risks frequency is for him ultra high. He is then aided (eg in getting out of depression) by reducing the frequency of his risks from ultra high to high. One means of bringing the frequency down from ultra high is for him or others to impose more structure on his life. This deletes some tiny risks (attractive chances, challenges) to enable him to analyse other tiny risks through to action.

RELATED AND OPPOSING THEORIES AND THERAPIES

Uncovering synergies with and differences from other theories and therapies is indirect, a matter of tracing the risk implications implicit in these other theories and therapies. This is because essentially none of the other theories /therapies are articulated explicitly with reference to sets of risks, let alone with reference to the three risk properties that characterise a set of risks as constituting whiffs of danger itemised in Table 5. The below is a selection from the vast array of theories and therapies to indicate how to decode and thus compare and contrast them with the whiffs of danger theory.

Resilience, coping, allostatic load contrasted with good stress, whiffs of danger

Most of the focus in research and treatment is on stress as a "bad". If we look, for example, at the valuable set of findings that Pierre Neveu collected and gave us in Stress 2003 vol. 6 (1), it comprises reports on damage from bad stress. It combines this with comforting information on the scope for the brain cum immune system to sometimes alleviate, even eliminate, long term deleterious effects. There is a similar combination of looking at past nasty outcomes and offering some comforting information on avoiding this causing permanent damage in the resilience literature (Curtis and Chicchetti, 2007), in the coping literature (Lazarus, 2000, 2006; Folkman, 2007) and in the allostatic load literature (Lupien et al., 2006).

The whiff of danger theory is more closely connected to celebrations of "good" stress as in (Scitovsky, 1976, 1971, 1999; Roth, 2001/3; Patmore, 2006). It involves in some respects a more precise concept of what aids brains and why brains need this exercise. The thrust of the whiffs of danger theory is that small risks, with their attendant portion of bad outcomes, are not simply an evil with which we must cope, exhibit resilience and avoid allostatic overload. It is rather good decision making is developed and maintained via risks that sometimes yield bad outcomes and associated negative emotions. If the risks previously faced were *un*balanced in the sense of yielding only good outcomes, the person lacks a balanced sense of hope and fear and of other risk-based positive and negative emotions. Without this balance, according to the whiffs of danger theory the person could eg become a foolhardy compulsive gambler, or at the first bad outcome encountered, could lapse into depression with all hope lost. Balanced hope and fear stems from a history of life yielding good outcomes after runs of bad ones.

Further the sorts of bad outcomes on which the coping and resilience literature focus (loss of significant other, job, physical injury, domestic violence and so forth) do not arise from whiffs of danger – from tiny risks/stresses. Rather these are occasioned by big risks/stresses. In addition, in the resilience and coping literature, the focus is on infrequent risks, while whiffs of danger arise from sets of risks that occur often. The allostatic load literature does examine sets of risks that occur often, eg repeated aircraft noise. But these are all the same sort whereas the whiffs of danger theory's set of beneficial tiny high frequency risks, is a set diversified across sorts of risks.

Therapies injecting psycho-social risks

Under a psychoanalytic therapy, the sufferer faces the mental, social and psychological risks (chances/challenges) of analysing her own thinking and behaviour and talking about what she discovers to another, or to a group. Likewise other psychosocial therapies implicitly introduce sufferers to this limited spectrum of risks. An example is the support-challenge theory of Hüther (1996) wherein development is assisted by supportive parents and communities that encourage their children to undertake activities that will yield minor stresses/risks. Without such parental and communal support, what would be tiny psycho-social stresses, too often become moderate or huge ones that can damage, instead of aiding, brain development.

Hüther's support-challenge theory implies that tiny psycho-social risks help mental development. Hüther's theory is a marked advance on most other support/challenge theories in two respects. First, it discriminates between tiny and larger risks. Second it goes beyond parental support to communal support – to the key role of societal structure in keeping psychosocial risks tiny, not massive and damaging.

The whiff of danger theory differs from Hüther's support-challenge theory in that it involves a wider spectrum of risks. To have adequate whiffs, the person must not only have an adequate number of social and emotional tiny risks that fall into the psychosocial category. To have adequate whiffs, the person must also have an adequate number of ethical and spiritual tiny risks. These could be components of psychosocial widely defined. To have adequate whiffs, the person must also have an adequate number of sensual, physical and mental tiny risks. Such sorts of risks unambiguously lie outside the category of psychosocial risks.

Holistic therapies can comprehend the entire range of sorts of risks of whiffs of danger. Rozman (2002), for example, reports treating her compulsive gamblers with what amounts to most of range of tiny risks involved in whiffs.

Treatment involves undertaking sensual risks from

being in nature, physical ones from activities like running marathons, mental, social, psychological ethical ones from preparing and giving public speeches on topics of individual and social concern. The undertaking of the entire gamut of risks she reports that she ensures by participating in all the events with her clients, including the marathons (and she claims a 100% cure rate).

Concentration/emotion free therapies: Interludes of eliminating most sorts of risk

Under relaxation and meditation therapies, the person has the challenge of limiting risks perceived so far as possible to two sorts, one concerns maintenance of a particular bodily stance, and the other concerns concentration on a single entity such as God, or a confined set of entities such as the passage of the person's own breath or the recitation of a mantra (Benson and Proctor, 1985; Benson and Klipper, 2000; Pagnoni and Cekic, 2007).

Relaxation and meditation thus have physical components of whiffs. In the relaxation and meditation techniques the person attempts to hold the body in a particular position, for some relaxation techniques with the body in as full relaxation as the person has mastered. For most meditation techniques, it is with the spine held as vertical and as straight as the person's exercise of his back muscles has so far enabled. These therapies also have a mental component of whiffs, namely a challenge to eliminate all risk perceptions bar those pertaining to the entity (or set of entities) of concentration.

The daily performance of Clyne's Sentic finger therapy involves swift traversal through a range of positive and negative emotions, plus a period also of concentrating on having no emotions, Clynes (1968, 1973, 1978, pp. 107-170, 1988). The finger exercises for positive and negative emotions are a means of experiencing some of the variety of tiny risks in whiffs of danger. The finger exercise of having no emotions has parallels to the relaxation and meditation therapies in eliminating most (but not all) sorts of whiffs.

The decision making improvements attained with relaxation, meditation and Sentic therapies point to the temporal pattern of risks mattering. They point to the brain benefitting from at least one daily interlude in which the varieties of risks processed are sharply curtailed. (A somewhat similar claim is made for the benefits of having enough deep sleep per day when the brain is doing far less risk processing and consequently using far less energy.)

Desirable temporal patterns of stressors and thus risk sets is part of a more general theory of risk sets and mental health, discussed in the section on evolutionary theories below. The whiffs of danger theory says nothing about whether the brain works better if there are oscillations over time in the varieties of risks faced, with interludes of minimal risk variety. The whiffs of danger theory only concerns a particular set of risks that involve great variety being beneficial to receive on a regular basis. It is silent on the issue of whether a particular pattern of oscillation in the varieties of risks experienced is beneficial.

The Sentic exercises by contrast build in such an oscillating pattern of tiny risks. The relaxation and meditation techniques do not mandate oscillation, but they permit it. This is because the relaxation and meditation episodes can be sequelled within each day by periods experiencing a great variety of tiny risks.

A simple hormetic theory of risk sets of different magnitudes

Under hormesis, as noted above, increasing doses of items are increasingly beneficial up to some point, and increasingly harmful beyond a further point. Evidence of hormesis in numerous biological stress responses is why Calabrese et al. (2007) proposed standardisation of the terminology for tracing such effects. An example of a hormetic biological stress response is the following. Tiny risks for a person generate tiny increases in glucocortoids and big risks generate big increases in glucocortoids. Small increments in glucocortoids enhance memory (an aspect of decision making), whereas big increments damage it, Lupien et al. (2005).

The whiffs of danger theory however is not itself a hormetic theory of *sets* of risks of different sizes. It is silent on whether the effects of sets of minute risk doses are less beneficial than sets of small risk doses and silent on whether the effects of sufficiently high doses of risks are generally detrimental to decision making, ie brain health. The whiffs of danger theory concerns exclusively the benefits of sets of small varied high frequency risks.

A general risks set and an associated general hormetic theory?

The thrust of this paper is to suggest that an overarching theory of mental disorders be organised about mechanisms that damage decision making using SKAT, the Stages of Knowledge Ahead Theory of risk, to delineate. In turn one of these mechanisms is the impact on mental disorder of sets of risks with different characteristics. In this regard the whiffs theory is but one set of risks with one set of characteristics, and thus but one step in formulating a general theory of how risk sets impact on the brain.

Research into more complex sets of risks might for

instance reveal that what is even more beneficial than whiffs of danger is a set of risks primarily composed of risks with the three characteristics of whiffs, but not exclusively. The yet more beneficial risk set might turn out to be one that contains also: less frequently encountered medium magnitude risks; infrequently encountered high risks: and, extremely rarely encountered, ultra high risks. In addition there are indications (see section on concentration/emotion free therapies above), that a full risk theory may indicate the value of interludes over the day of close to none apart from risks of a special sort, interspersed with times when the person is open to and so perceives a wide variety of risks.

Moreover, it seems implausible that the full picture of the biology of stress could be delineated by examining each hormonal secretion separately, as in simple hormetic theories. For instance, in whether glucocortoids are too high or too low, it seems plausible to postulate that this depends on what other hormonal secretions are occurring over related time intervals. As regards decision making, these other secretions may at some levels have beneficial interactions with glucocortoids, at other levels detrimental ones. The simple hormetic relations seem more plausible in partial analyses, when (ideally) all these other factors are being held constant.

The whiffs of danger theory rests on the healthy brain having an appropriate mix of hope and fear and of other risk-based positive and negative emotions. In this regard, Lupien et al. (2006) cite research finding that stressors anticipated to elicit positive risk-based emotions do indeed elicit different hormonal responses. Research of situations likely to elicit substantial amounts of positive as well as negative risk-based emotions in each individual and measuring a range of hormonal responses, can be steps toward discerning whether a general theory of risks uncovers a hormetic relation.

It is unclear whether further research will endorse the tri-phasic hormetic shape as regards risk sets of different magnitudes. Indeed it might reveal no systematic relation between risk magnitude alone and brain health. It could turn out for instance that those who develop healthy brains (via whiffs of danger and another factors) avoid mental disorders because of their capacity to devise for themselves a constant flow of whiffs even when huge risks constantly befall them and they have the ill luck of the bad outcome ensuing.

Such is the way we describe some of the great. For example, while held in a concentration camp in the Second World War, the Ukranian Jew Moshé Feldenkrais developed techniques for using inner muscles and maintained a good enough brain to disseminate his findings afterwards such that his techniques are still being taught and found useful today. Again, while interned for organising armed resistance to apartheid in South Africa, Nelson Mandela changed to running on the spot for exercise to keep fit, and to educating his guards in his liberation goals, and after the end of apartheid, had kept a good enough brain to lead his country to a reasonable outcome. In short, it remains to discover whether such people are the exceptions so that on average those facing big risks have on average more mental disorders (that is, are on average inferior decision makers) than those facing mainly tiny or moderate risks – as a general hormetic theory of risk sets requires.

As regards the associated biological stress response to big stressors risks, for general hormesis, what needs to be investigated is the following. To what extent do those rated as being better decision makers, attain their better brain health via
a) lower levels of biological stress responses to these big stressors, or b) different mixes of biological stress responses or c) not being damaged as much by high biological stress responses, or d) not experiencing the big stressors as big risks. We need to add d) since the experienced magnitude of the stress/risk depends critically on the individual, Lupien et al. (2006). In this regard, most major religious and philosophical movements have a strand seeking via attitudes such as faith and trust, to enable people to experience massive stressors as personally minor risks.

Evolutionary theories

The whiffs of danger theory focus on change, and on the brain ever changing (making fresh decisions). It thus differs from that strand of evolutionary theorising about the brain, wherein evolution resulted in set strategies (acts) being selected. These set strategies arise out of models in which the pre-historical environment was itself fixed, eg Allen and Badcock (2003). If the environment were so fixed, the acts could have evolved as automatic responses. There would have been no advantage in having the soft-wired brain that can decide in each new circumstance.

In emphasising a changing environment, the whiffs of danger theory accords with Curtin and Chicchetti (2003) and the seminal work on the biology of stress, Hüther (1996). Hüther however essentially postulates a single sort of risk faced previously (physical attack) and a different single sort of risk faced today (that of a psychosocial difficulty). But consider what transpires in the lives of tribal people, according to the accounts of anthropologists over the previous two centuries. Children tend to belong to and are reared by the entire group. Women gather and men hunt for the entire group. These extensive interactions with members of one's own group and activities in forwarding the interests of the entire group give rise to a vast array of tiny challenges and tiny

chances. The proportion of effort spent on threats of physical attack is minute in these accounts, even if it exceeds that spent by those in rich countries not cursed by being in a vicious violent poverty-stricken ghetto.

In prehistory there was a much higher density of animals that prey on humans. So the proportion of decision effort expended on fight or flight will have been higher. However, it is implausible that it exceeds that of wild animals today in the middle of the food chain and thus preyed upon, and who in addition face conflicts with other groups of the same species and within group conflict. As regards within group conflict, these animals typically employ a fair bit of ritual strength competitions to determine hierarchies and territories and only engage in fight or flight for a tiny fraction of a typical day. Further fight or flight can never have been a very high fraction of decision effort. This is because the capacity to consider fight or flight entails numerous decisions on other matters requisite to the person having enough physical energy to fight or flee. Consider for instance what happens today in a nation described as on a full war footing, that is, organised only for fight and flight. Most of the population will be busy making decisions pertaining to production of food, shelter, munitions, the formation and maintenance of alliances and of attaining cohesion of the group. The proportion of time in actual physical conflict or physical flight is small. Any animal, including any person, who spent even half a day making decisions exclusively on fight or flight, ignoring his other objectives (like water, food, protection from the elements, retention of societal connections), would be unlikely to survive 24 h.

Living has ever been complex for a soft-wired animal: it involves multiple sorts of decisions, not simply one decision making ability of when to physically fight and when to physically flee. Accordingly, the whiff of danger theory disagrees with the assumption that the human brain developed exclusively to make fight or flight decisions. It postulates that soft-wired brains and most especially the human brain, developed to address the range of sensual, physical, intellectual, social, emotional, ethical and spiritual risks (chances and challenges) reported by anthropologists and ethnologists.

The whiff of danger theory differs in two other ways from essentially all mental health theories that appeal to evolution. First, such evolutionarily inspired theories have a conception of optimal evolutionary adaptation, akin to Darwinian language about survival of the fittest. The whiff of danger theory avoids mention of optimisation since in no reasonably complex situation, let alone any so complex as to involve risk, can we specify what would be optimal. (All that is modelled in algebraic so-called optimising evolutionary models is ultra simplified for algebraic tractability). Second, such evolutionary models have a focus on life in pre-history being primarily dangerous, whereas in the whiffs of danger theory the focus is on life in the wild having involved also numerous nice surprises, not merely nasty ones.

Theory that education enhances decision making

Those of higher socio-economic status have superior health, including superior mental health, Lupien et al. (2001). In a rich country like the US, after excluding reverse causal chains (such as that of poor health damaging income and thus social status), the principal intermediate cause is years of education. Each additional year of education, into tertiary education, enhances health. Only a limited extent of how education enhances health can be attributed to factors like healthier behaviours. The prime way in which education enhances health is inferred to be that “increasing levels of education lead to different thinking and decision-making patterns”, (Cutler and Lleras-Muney, 2008). This finding is in accord with the whiffs of danger theory as follows. Education assists people in obtaining attractive jobs, social environs and relationships that yield whiffs of danger rather than either boredom or excessively large risks, or both.

Competencies, attitudinal, philosophical, spiritual and individual-specific theories

The whiffs of danger theory advocates a particular set of risk experience as conducive to learning good decision making, that is, mental health. What whiffs we receive are education and culture dependent. An urbanite on a hike in nature can miss most sensual whiffs potentially there. Accompanied by someone living in a tribe or a remote area or by a biologist, geologist or historian of the area, the person sees and hears much otherwise missed. Hence a person’s scope to a) experience whiffs potentially on offer in the external environment, and b) learn better decision making from whiffs experienced, can both be enhanced.

One means of achieving a) and b) is geared to the specifics of each individual through sessions with a therapist, acquaintance, friend, or tour leader (as in the hike example) who is aware of where, via culture and education, the person will have areas of ignorance. Another means of achieving a) and b) is by people acquiring life skills deemed good for everyone. Such competencies, behaviours, attitudes, philosophies of life, spiritual and toughening up techniques are advocated and promoted by religious and community organisations, self-help books, and scientists (Heylighen, 1992; Fogel, 2000a).

These methods are diverse. Those that seek to shelter people from experiencing any risks whatsoever by

seeking to give them a total security blanket are incompatible with the whiffs of danger theory. Many of these techniques however have close affinities with, and virtually offer a programme for acquiring whiffs of danger, or at least some key components of whiffs, eg that of Vaillant (2003). Thus, Vaillant in effect advises the aged to embark on tiny sensual, physical, mental, social, ethical and spiritual risks that constitute the varieties of whiffs in the whiffs of danger theory.

Control theories

Anything that people can control perfectly, they can predict perfectly and thus face zero risk concerning that thing. If therefore a person could control perfectly their life, their future would be certain; they would cease to need a brain – there would be no future decisions to make. If they could control perfectly their job, their need for a brain would be drastically reduced. Brains with nothing or little to do cannot plausibly be anticipated to remain healthy. When scientists formulate theories that control enhances mental health therefore the theories would be implausible if, in their theories the ideal degree of control denoted perfect control. Even though such control scientists are not particularly explicit concerning the certainty / risk implications of their theories, such is not the case, as can be seen from the below analysis of two influential versions of control theory, those of Csikszentmihalyi (1990) and Marmot et al (1997, 1999, 2001, 2002).

The flow control theory

For over threefold increase in clinical mental health interventions between 1955 and 1975 recorded in US Social Indicators, in his wise and inspirational flow theory, Csikszentmihalyi (1990, pp. 209-210, re-arranged) agrees with Pascal (1670) that existential anxiety is a root problem. He does not propose Pascal's solution of giving oneself to another cause in the form of doing god's will. He proposes a related one for the modern man who, Csikszentmihalyi (1990) notes may not accept all the "baggage" of a traditional religious deity. Some have sought an antidote to the "baggage" problem by reformulating their religious beliefs in a way compatible with current scientific understandings, eg this might be broadly construed as the programme of Jung and Jaffe (1989) and Dozier (1991). Csikszentmihalyi's instead sees an antidote in people acquiring: control of their lives, an autotelic self that is never bored, seldom anxious, easily translates potential threats into enjoyable challenges and its acquisition requires consciously setting goals that are neither unrealistic so that hopes are dashed … or have the safety of being trivial.

Since control in Csikzentmihalyi's denotation cannot be attained if the person chooses trivially safe goals, Csikzentmihalyi unambiguously uses the term control, not to denote perfect control, but merely a limited degree of control. Csikzentmihalyi's theory differs from the whiffs of danger theory in three respects. First, it advocates particular attitudes of mind and developing particular competencies in responding to risks, a matter on which the whiffs theory is silent. Second it sees the risks more as bad outcomes to be overcome via flow aiding in resilience, coping, adaptation, whereas the whiffs theory embraces a particular set of risks as essential for developing and maintaining a healthy brain. Third it concerns conscious choice, whereas the whiffs theory concerns the effects of risks on both conscious and unconscious choice. Most sensual risks, for instance, are decision-processed at the unconscious level for concluding what is seen, heard, felt, smelt.

A capabilities /societal structure control theory

On aiding those of lower status to better health and thereby to better decision making, the findings of Cutler and Llera have been discussed in section on education enhances decision making and of Csikzentmihalyi in the immediately preceding section. Compared to these Marmot puts less focus on personal education and personal conscious decisions. He puts more focus on societal structures that damage the minds of those of lower status. He reports in an editorial and in interviews (Marmot, 1999, 2001, 2002) that he has inferred from his own epidemiological studies (Marmot, Bosma, Hemingway, Brunner and Stansfeld, 1997; Marmot, Shipley and Rose, 1984)), and those of others as follows.

It was not the case that people in high stress jobs had a higher risk of heart attack, rather it went exactly the other way: people at the bottom of the hierarchy had a higher risk of heart attacks. [Good health] depended on how much control they had at work, how fairly they were treated at work, how interesting their work was. We found clear social gradients in people's participation in social networks, … in psychological attributes like hostility. [Poor health springs from lack of] control over your life, lack of opportunity to participate socially in a meaningful way, [lack of] what Amartya Sen calls capabilities.

Since Marmot notes the riskier nature of higher echelon jobs, it is clear that in arguing that society alter to enable those lower echelons more control over their lives, he is not proposing that risk-free lives enhance health. Indeed he is arguing in effect that those in lower echelons be enabled to have more interesting and hence more risky work, and given more social risks via participation in social networks. Marmot's version of the capabilities-freedom theory of Sen (1999) in effect advocates

changes in societal structure to introduce some components of whiffs of danger for those in lower echelons and to thereby end their risk starvation.

Explicitness on risks

In the expositions of control theories and other techniques that have a substantial overlap with the whiffs of danger theory, there is an issue of explicitness that the practices being advocated involve risk-taking. In many of these expositions there is an enticement to risk-taking by referring to the positive risk-based emotions that these elicit, namely hope, faith, trust, excitement, exhilaration, curiosity, getting enough challenge from interesting and difficult activities and avoiding boredom, and so forth. The section on evolving stages of knowledge ahead with its Table 4, explains how these emotions arise in the pre-outcome stage during which the person's risk is unresolved and so the person may contemplate and feel the tension of whether the nice or the nasty outcome will eventuate.

None of the expositions, include particularly explicit dynamics of a pre-outcome stage during which these emotions are experienced, followed by other risk-based emotions once the post-outcome stage is entered and the outcome is learned, namely exhilaration if the nice outcome occurred or disappointment if the nasty outcome occurred. But some expositions are explicit that the person having these risk-based emotions is in a risky situation and thus faces possibilities of inferior, and in some cases nasty, outcomes. Thus in advocating belief in God and doing his will in order to overcome existential anxiety and insanity, Pascal (1670) states explicitly that making this decision has a downside risk – the possibility that the belief is false. Afro-American lay Episcopal theologian Dozier (1991, p. 61) is likewise explicit on ethical risks – that if we take these in faith we may learn later that we have the nasty outcome (that is, learn that we made the wrong ethical decision).

The Christian church succumbs to the temptation to know absolutely when it calls doubt the opposite of faith. Fear is, fear will not risk that even if I am wrong, I will trust that if I move today by the light that is given me, knowing it is only finite and partial, I will know more and different things tomorrow than I know today and I can be open to the new possibility I cannot even imagine today.

But few religious or secular presenters of attitudinal procedures are as explicit as Pascal and Dozier, on the risks in what they advocate. Pascal is the father of probability theory, which renders it natural for him to make such connections explicit. Dozier stands in support of Pascal's tradition of faith, encouraging people to take appropriate risks and to help people through their risks. The opposite tradition is of religious or secular beliefs offering a security blanket of guaranteed outcomes, that is, of a certain, risk-free future. The whiffs of danger theory is that this opposite tradition, if taken literally, is the reverse of being saved if salvation means having a healthy brain. Both traditions date back thousands of years in the religious and philosophical literature and both have energetic advocates also in the self-help and scientific writings of today. A contribution of this paper's risky choice decision theoretic perspective is to point readers to how they themselves can recode therapies and other practices in order to tease out their risk and risk-free implications.

NOTICING RISK STARVATION

Comparing the whiffs of danger / risk starvation theory with other theories has required us to recode these other theories to trace their implicit risk implications. Detecting the benefits of risk and damage from risk starvation is likewise partly a matter of re-coding past studies for the implicit risks. Bear in mind also that risk starvation arises exclusively from deprivation of the sets of tiny varied frequent risks that constitute whiffs of danger. Sets of moderate or big risks or (boringly) repetitious sequences of the same risks, are risks sets that lie outside whiffs of danger, and thus have no bearing on whether or not the animal is suffering risk starvation.

Recoding stressors reported in prior research

In some set-ups, the animal is in effect born into a nastier environment than normal. Such studies are important. But they do not concern stress as that term is defined dynamically in this paper, as involving a change in the external environment.

In other set-ups the animal is subjected to change in its environment, that is, to a stressor. But it is in effect a permanent change in its environment. After permanent changes, the animal may likely only experience a positive degree of risk initially. In due course, it may see its future as riskless, as certain. In such cases, the animal's perceptions of its evolving risks may be inferred from its behaviour.

To illustrate how risks that are inside the animal's brain (and thus not directly observable) may be inferred, take those experiments in which mice or rats are thrown into a well from which they cannot climb out. Such animals may be inferred to have ceased to see themselves in a risky situation (to have lost all hope), if they cease swimming while yet capable of doing so. How long they swim can be taken as a measure of hope and thus of them still experiencing risk. The swim time has been found to be longer (but not typically the maximum possible swimming

time) for those that in the past have been rescued. Typically they are rescued by having items cast into the well by which they can climb out (Crawley, 1999). Such prior research findings thus need to be recoded for the animal's evolving assessment of the degree of risk, as time extends after the change. When recoded, these swimming experiments suggest that animals that previously lived in set-ups with more components of whiffs of danger suffer less depression, have better decision making.

Each stressor reported in a prior research paper needs to be recoded to ascertain whether that stressor caused the animal to experience a tiny risk that would be a component of whiffs of danger. It may instead have caused a moderate or big risk that would lie outside the set of risks constituting whiffs. In recoding, it does not suffice to look at each risk separately. It is essential to look also at the whole set of risks in order to assess the other characteristics concerning whiffs of danger, namely variety and frequency. Was the set of stressors varied, arising from different sorts of risks, and numerous (hence occurring often)? Let us give three more examples from prior research papers of decoding the risks that stressors cause animals to experience.

First, for mice lacking the ApoE gene (ApoE knock out mice) with cognitive deficits, predator stress reverts their cognitive abilities to normal, Grootendorst, De Kloet, Dalm and Oitzl (2001) and Grootendorst, DeKloet, Vossen, Dalm and Oitzl (2001). For example, stressors causing the animal to experience tiny risks of predation are stressors that mend damaged brains with respect to cognition. Tiny risks are components of whiffs of danger, indicating that whiffs may reverse some mental disorders.

Second, mild repeated stress may increase the density of cortical noradrenergic innervation, whereas long-term stress causes retraction or degeneration of noradrenergic axons in the cerebral cortex, Hüther (1996, p. 591). Since mild repeated stresses arise from tiny frequent stressors and give rise to tiny often experienced risks, the beneficial effects here reported concern tiny risks that would be components of whiffs of danger. Since severe long-term stresses arise from big infrequent severe stressors eliciting big infrequent risk experiences, their damaging effects arise from sets of risks that lie outside the set comprising whiffs.

Third, consider the following set of findings as regards allostatic load from Lupien et al. (2006, pp. 577, 575):

Although short-term responses of the brain to novel and potentially threatening situations may be adaptive and result in new learning and acquire behavioral strategies for coping, as may be the case for certain types of fear related memories, repeated stress can cause both cognitive impairments and structural changes in the hippocampus.

The first finding is that brains are aided generally by little stressors (novelty) causing the animal to experience tiny risks that are threatening, that is, might have nasty outcomes. Such tiny risks are components of whiffs of danger. The first finding, when recoded, thus indicates benefits of whiffs to the brain.

The second finding is that if the future contains an excessive number of repetitions of an identical stressor that always yields the same nasty outcome, the brain is damaged. This shows that high frequency uniformly damaging stressors constitute a bad set of risks for the brain. Now as the repetitions continue, the animal's risk perception will evolve, from initially perceiving the events as risks to eventually treating the events as having certainly a bad outcome, that is, as involving a zero degree of risk. The second finding when recoded thus suggests that maintaining a good brain is aided by two of the characteristics of whiffs of danger:

a) Having tiny risks – not tiny certainties.
b) Variety in the risks – not every one the same sort.

Let us now turn to recoding previous research findings on environmental enrichment.

Recoding literature on environmental enrichment

Transgenic mice were created that develop a neurodegenerative syndrome that closely models Huntington's disease. Those given an environment described as 'enriched' with play items changed every few days, enjoyed spectacular delays in the onset and progress of the degenerations. Learning and memory deficits observed in a transgenic mouse model of Alzheimer's disease can be ameliorated by environmental enrichment (Jankowsky et al., 2005).

For rats, environmental enrichment has:

1) Reversed lead poisoning stress that resulted in learning and long-term potential (LTP) impairment, (Cao, Huang and Ruan, 2008).
2) Reversed damage from chronic prenatal stress that caused addictive and depressive tendencies, cognitive deficits and impaired hippocampal synaptic plasticity, (Yang et al., 2006).
3) Reversed mental disorders from their being stressed from being stereotaxically injected with enough ebotenic acid to cause substantial atrophy of dendritic arborization (that is, correlated with Downs syndrome, Alzheimer's disease, senile dementia and schizoprhenia) and significantly reduced spinal density (that is correlated with learning difficulties) (Bindu et al., 2007).

In delaying morbidity onset and severity in some cases and reversing severe brain damage in other cases, the

enriched environments of rats and mice with play items add exercise (Hockly et al., 2002). What has passed unnoticed is that these play item stressors add something else, small varied risks —components of whiffs of danger – without which the mice and rat environments are virtually risk free — a predictable boring mouse or rat lab.

The play items add tiny frequent varied surprises and risks. One second the mouse has the surprise of seeing a tube. The next second, having decided to run through it, the mouse has the chance of encountering a bit of food and a tiny danger of knocking into the side of the tube.

The second after that, the mouse has the surprise of seeing a wheel. ... This succession of surprises and risks attenuate as the effects of the play items get more predictable from exploration. But within days these items are replaced by new ones, raising the general risk level again. Those replacements generate an oscillating but ever positive level of frequently encountered small varied risks (chances/challenges). Recoded then, environmental enrichment injects components of whiffs of danger.

It is reasonable to propose that most mice, rats and other laboratory animals have been reared in environments
abominably lacking in whiffs of danger and that this severely impairs their ability to deal with any physical and psychological stresses. It would for instance be informative, for understanding stress, to redo past stress experiments with mice that beforehand lived in “enriched” environment, ones closer to what would have been their lives in the wild. There life was much more full of little chances and challenges, whiffs of danger, developing their brains with hope and immunological resilience to the blows of life delivered in the stress experiments, aided in finding the risks tiny initially by having mothers. It would be interesting to see what difference this makes to the conclusions drawn on transient and long term effects of the particular stressor being in each case investigated.

Steps in this direction of serious environmental enrichment were performed back in the 1950s (Barnett, 1956, 1957, 1963, 1972). These studies investigated and confirmed the hypothesis in Darwin (1871) of animal need for and love of what are here termed whiffs of danger in order to get excitement and with most exhibiting curiosity in the form of exploring to seek to discover new things. The experiments required more complex set-ups than was the norm, so as to give the animals choices to explore (or not explore) new things. For retrospectives on these early investigations and the obstacles to getting funding in the behavioristic anti-decision-making scientific culture of that era and progress since (Barnett and Cowan, 1976; Barnett, 1977).

People, like mice and rats in boring laboratories, need whiffs and can suffer risk starvation. People can benefit from the environmental enrichment of stressors of having to, in a supportive environment, discuss their life issues for durations in total of 24-47 h, can improve brain plasticity, Hüther and Sachsse (2007). Such environmentally enriching stressors constitute tiny risks including:

1) Failing to articulate (a nasty outcome).
2) Eliciting condemnation (a nasty outcome).
3) Arousing empathy and interest (a nice outcome).
4) Forming friendships (nice outcomes).

That is, recoded, Hüther and Sachsse found that introducing some psycho-social components of risks in the whiffs of danger set can reverse some mental disorders that reduce plasticity.

Let us look now for evidence of risk starvation causing two of the commonest mental disorders, and for indications that whiffs of danger may reverse these.

DEMENTIAS AND DEPRESSIONS

The current research/treatments thrusts

These are frequently described as genetically originating chemical abnormalities in the brain. The focus on a geneticorigin has happened over the last two decades even though research has yet to connect genetic distributions to epidemiological features of these two mental disorders, and in comparisons of normal people and sufferers, less than 50% of each mental disorder is attributed to genetic predispositions, with the unexplained residual of over 50% attributed to environmental factors. This genetic focus has made sufferers readier to admit their mental disorder as nobody can be blamed for their genes (Cutler, 2004) – and fostered research that might enable genetic modifications in the future.

The genetic focus has drawbacks. First, it has deflected attention from discovering the environmental factors, even though environmental factors are on current evidence more important. Second it has fostered “bandaid” emergency treatment of the chemical imbalances themselves. As therapy, drugs are not merely used in emergencies, instead have risen to centre stage. This is despite mixed evidence on whether drugs make a substantial net contribution and despite user groups, on examination of the published medical evidence, frequently advising against drugs (Reynolds et al., 2006; Miyanaga, 2005; Chatterjee, 2004; Mann, 2005; Ebmeier, Donaghey and Steele, 2006; Reid and Stewart, 2001; de Jonghe et al., 2000; Pagnoni and Cekic, 2007).

For dementias, there is an accelerating focus on injecting components of whiffs of danger such as physical exercise, hobbies, learning new skills including meditation, taking up mentally taxing games. For depression, the main non-drug treatments are protection

from big risks such as incest / domestic violence, injection of one component of whiffs, namely psychosocial activities/therapies (Pinquart and Soerensen, 2001; Lupien et al., 2000, 2001; Reinherz et al., 2003; Turner and Lloyd, 2004; Hensley, Deepa and Uhlenbluth, 2004).

Limited success from current research / treatment policies

In rich countries, dementias are expensive (informal carers, lost productivity and health services) (Access Economics, 2003; Huang, Cartwright and Hu, 1977; O'Shea & Reilly, 1999), as are depressions with typically a severe relapse within four years so that the mental disorder is chronic (Marks, 2002; Paykel et al., 1999, Paykel et al., 2005; Teasdale et al., 2000; Hensley et al., 2004). Over the four-year haul, treatment improvement in excess of placebos is modest (Kirsch, 2002a, 2002b; Salamone, 2002), and for those on drugs, any improvement is muted by undesired serious side-effects. Dementias escalate. Despite multiple new generations of drugs, depressions have become the biggest intellectual disorder in many rich countries, and according to one forecast will become by 2020, after heart disease, the leading cause of disability worldwide (Murray and Lopez, 1996; Berto, D'Ilario, Ruffo, Di Virgillio and Rizzo, 2000). Epidemiological data identifies the policy gap.

In discerning epidemiological features of dementias and depressions, problems are that classifications vary and are contentious; preclinical stages are undetectable and reporting is unsystematic (Hickie, Andrews and Davenport, 2002; Greenberg, 2007). Nevertheless the 11 epidemiological features of Table 7 can be discerned (Greenwald et al., 1979; Riedel-Heller, Busse, Aurich, Matschiner and Angermeyer, 2001; Liu et al., 1994; Shaji, Bose, Verghese, 2005; Suh and Shah, 2001; Ganguly, Dodge, Shen, Pandav and DeKosky, 2005; Larson et al., 2004; Jacobi et al., 2004; Sullivan, Neale and Kendler, 2000; Seligman, 1977; Piccinelli and Wilkinson, 2000; Dudas, 2005; Lupien et al., 2000, 2001; Hasin, Goodwin, Stinson and Grant, 2005).

Only three of the features (1, 6 and 7) identified in Table 7 are explainable as in Table 8, by current theories, leaving the other eight unexplained. Can then this dearth of explanatory power revealed in Table 8 be overcome by the author's whiffs of danger / risk starvation theory?

Table 7. Prevalence features of dementias and depressions.

Dementias
1 The high prevalence group is the elderly, with the prevalence increasing for each five year age cohort
2 The age of onset varies by more than 70 years, ranging from the early twenties (primarily uneducated, poor, unemployed), to never (especially for those continuing with hobbies and other cognitive activities)
There is in addition quite a bit of evidence in rich countries of:
3 A decline over time in the age of onset, and
4 The prevalence being more than double that for people in the same five-year age cohort in poorer countries.
Depressions
In rich countries the prevalence is higher:
5 In peace-time
6 For those in lower socio-economic strata, and
7 For females.
There is also quite a bit of evidence that the prevalence in rich countries is:
7 Substantially higher than in poor countries,
9 Rising over the last century, and
10 Occurring at earlier ages Co morbidity of Dementias and Depressions
11 Is between 10 and 50% depending on the definition used for each mental disorder.

Table 8. Current theories / policies.

Feature	How explains
Dementias 1	stress of aging predisposes
Depressions 6,7	Stress in the form of prior dire happenings in the form of incest and domestic violence predisposes and these two groups encounter more dire happenings. Unexplained 2,3,4,5,7,9,10,11

Risk starvation the missing causal link

Consider first the poor and the elderly, identified as particularly prone to both dementia and depression. The poor still have some components of whiffs (taking a diabetic injection, tending a sick child, coping with a difficult co-worker). But, in tandem with the impressive increase in their life expectancy (but not mental health) over the last century, there has been a dramatic decline in their whiffs. That is to say, there has been a dramatic increase in their risk starvation. The increase in life expectancy of the poor has been attained importantly through the provision of five forms of government transfer payments for being:

a) unemployed.
b) with children.
c) a single paren.,
d) poor, in old age.
e) in need of medical or nursing care (Fogel 2000b, 2003, 2004).

Such provision has dramatically improved the lot of the poor by eliminating many major and moderate risks of death and morbidity caused by lack of money. It is a crowning achievement of the rich world.

The ways in which these government transfers have been instituted however, eliminated inadvertently some good things for mental health. It eliminated the whiffs of danger attendant on the poor in the form of offering sufficient services and friendships to relatives, acquaintances and employers to tide them through these hardships and through old age. In many countries, retirement rules make it difficult for those elderly who wish to contribute to society by remaining in the paid workforce, to do so.

The general cultural norms of today deter people from showing gratitude to the state when the state provides now what once people had to strive to provide for themselves. In other words, such norms make it hard for most people to move away from the television and give themselves the whiffs of danger involved in inventing ways of contributing to society and doing it. But the more educated non-poor are better integrated into religious and other organisations that foster whiffs of danger from making societal contributions. This is because, compared to better educated people, the poor, through the factors identified by researchers such as Cutler and Llera (2008), Marmot (1999, 2000, 2001) and Marmot et al. (1984, 1997) have been weak in engineering such whiffs of danger for themselves. The better educated by contrast typically choose life-styles that furnish them a more adequate set of whiffs that is less frequently suffer risk starvation.

The non-government sector, in particular, religious organisations, have contributed. But these have not managed to furnish enough social networks and mentoring services to facilitate the poor in their acquiring their own whiffs of danger from any sources, Fogel (2000a, 204-215). In short there is a need for NGOs to do missionary outreach of injecting whiffs of danger for the poor in the rich world. This is not to suggest that today's poor in rich countries consider themselves coddled. They feel that they face numerous challenges. As demonstrated by the increases in longevity, they face however fewer challenges than did the poor of even a generation back, and far fewer then did the poor two generations back.

Consider now women and children, the other two groups with high and rising depression rates. The principal change reducing their access to whiffs of danger and subjecting them to risk starvation, has been the rise of the modern family, documented in Shorter (1977), and from about 1970 onwards, the rise of the even smaller unit, the post modern family. For women, the process has left them progressively more isolated in the home, educating their offspring, bereft of the variety of tiny risks involved in operating in the tribe, in the village or in the large establishments of the upper class, in each of which they interacted with a wide variety of children and adults.

The increasing focus on mothering, and mothering smaller and smaller broods, has moreover resulted in

unhealthy forms of protection of the children that risk starve the mother as she sacrifices her external world to keep her brood safe from the external world. As with the poor, this is not to say that women cloistered in the house caring for their partner and children recognise themselves as under-challenged. Rather they may perceive themselves as finding helping their family more challenging through modernisation.

Risk starvation however can occur because females set themselves higher family challenges of more strenuously protecting and educating their young. Such narrowing of goals prevents these women from having the range of challenges enabled by interaction in the larger community, disabled by excess focus on one's spouse and offspring. This range of challenges are required to avoid risk starvation. Whiffs of danger are only attained with variety in the challenges, no in the concentration of all challenges in the home.

For children, the move to the modern and now to the post-modern family, has in a parallel manner to that of females, increased their risk starvation, with their varieties of tiny risks further curtailed by the advent of the television and computer games. Children's risk starvation is also caused in part by their increasing reliance for social interaction on parents. This is due to the diminished number of siblings and diminished contact with the extended family and wider community.

Whiffs rather than bigger risks are partly acquired by the fluidity of being able to move out of unsuitable interactions with a parent or one sibling to the company of other siblings, or neighbours, or more distant relatives, or into the fields or forest. Social fluidity tends to keep risks tinier, lack of fluidity to magnify them. To give one example, it does not matter much if a father is angry if the child can escape to laugh about it with his father's brother, his uncle (who may moreover calm down father). But an angry father is more serious, can even be a disaster, in a post modern family with little scope for the child to ever escape and discover that others hold a a different evaluation of how bad that child really had been – or of whether the child was really bad at all. In short, that formerly wider social net for children made it less dangerous to be the offspring of people with poor parenting skills. In a wide social network, the wider society both helps generate the whiffs that children need, and helps put a curb on bad behaviour of parents generating moderate and big risks.

That wider network of the past also put curbs on children being deprived of whiffs of danger through well-meaning parents and grandparents who did not realise that their behaviour was bad. These are relatives who impose too narrow a set of chances and challenges on a child because they seek to have their child come in the top echelon at school without realising that their child lacks the mental ability to attain such grades. Such modern nuclear families prevent the child from being able to healthily diversify into non-academic challenges.

That child can become depressed since it fails to get top grades despite abandoning all non-academic challenges. That child is being deprived of its whiffs of danger – of undertaking sensual, physical, psychological, social, ethical and spiritual challenges in which it would more often succeed and that would allow its brain to develop to become an adequate decision maker in life's varied challenges. The wider social network of former times diluted the scope for parents to subject a less academically gifted child to risk starvation in this manner.

Table 9 summarises the foregoing forms of risk starvation to which the poor, females and the young have been increasingly subjected. It in addition describes briefly other factors causing risk starvation in the 11 groups identified in Table 9 as prone to dementia or depression.

*Less than about a sixth of the lower incidence in poor countries stems from their shorter life after dementia. More may be accounted for by few surviving in those socio-economic strata with higher dementia rates, namely the lower strata.

Table 9 constitutes evidence of the damage of risk starvation, of how injecting whiffs of danger could have prevented mental disorders. As regards reversal, as the part on noticing risk starvation above indicates, numerous animal studies report that components of whiffs of danger in the form of environmental enrichment reverse some mental disorders. People also can have their environments enriched after contacting dementia, and with all the publicity now arriving on the importance of components of whiffs such as doing mental and physical exercises, engaging in hobbies and good causes, there are indications of this beginning to happen.

The British Journal of Psychiatry has a cautiously optimistic editorial reporting on an Australian study finding dementia reversals from whiffs of danger via activities such as exercise (and a healthy diet), Burke, Hickie, Breakspear and Götz (2007). The complementary medicine literature has a longer list of whiffs claimed to reverse dementia, from sensual whiffs including perfumes, to spiritual/intellectual ones, including meditation (Khalsa, 1998; Thompson, 2001; Horrigan, 2007). Thus from not only the viewpoint of prevention, but also that of remediation, there is support for the whiffs of danger / risk starvation theory and for highlighting the brain's raison d'être, to make decisions, to process risks.

Table 9. Whiffs of danger theory.

Feature	Where the stress and risk starvation was absent / occurred
Dementias	
2	Jobs especially the more challenging jobs of the educated, and hobbies and outside activities when retired) provide whiffs of danger
1, 3 and 4	Rich countries over time gave the elderly and poor state welfare plus (often) forced retirement. These measures have saved some from dire risks like starvation and premature death, increased equality and contributed to longevity Fogel (2000b, 2003, 2004). But state welfare has kept alive many uneducated and these typically have difficulty giving themselves whiffs of danger, and others have not stepped in in sufficient numbers to mentor them in how to obtain these and in a desire to obtain these Fogel (2000a, pp. 204-215). Further state welfare/forced retirement measures deprive many in these groups of the *smaller* risks of paid employment or convincing those in their informal network to support them. Further carers often accentuate the elderly person's risk starvation via precautions to reduce falls.
Depressions	
5	In war-time there is enough stimulation for most civilians to obtain their whiffs of danger
6,7	The inferior status of the poor with boring jobs and many females with decisions taken by superior males deprives them of enough variety in their risks (chances/challenges)
7,9	Increases in the incidence of risk starvation for the poor and women are as follows. In rich countries a century ago, the cityscape afforded more social, visual, aural and olfactory risk processing more akin to that found in poor countries today. The deck entrance functional architecture apartment complexes of the 1960s and more recently are deemed unenticing for neighbourhood interaction. They curtail whiffs of danger arising out of social interaction with neighbours. They also curtail neighbourly support that helps keep risks tiny and, thus in the whiffs set. Without such neighbourly support risks readily become moderate or large, outside the beneficial whiffs set of risks. After controlling for other factors, residents of such complexes have a higher chance of being depressed (Weich et al., 2002). Prior to functional architecture, buildings had intricate shapes and surfaces. Public areas were generally used, and involved interactions with people and animals carting goods, spitting, urinating, defecating in and out of open sewers, chucking large garbage items. Many lower echelon adults have today, as a century ago, risk starvation in their boring low challenge jobs, but then they had adequate whiffs of danger from the varied small frequent risks of subsistence, since there was little of today's social welfare. A century back society had yet to be re-organised about the modern family where wives lack the social whiffs of danger of village interaction because each is sequestered off, raising her offspring in isolation in her family home, Shorter (1977). Around 1900, few husbands could afford non-working wives in suburban lab cages minding two children, facing that narrow range of risks of child minding and housekeeping instead of the normal range of risks of adults interacting in the wider world. Few older females lived alone.
10	A century back in rich countries, few children lacked the small varied frequent risks of daily physical games coupled with the small varied frequent risks of either crowded city activities (apartment dwellers) or exploring nature (those in the suburbs and rural areas). Few adolescents had their own bedroom in which to spend long hours bereft of a rich variety of sensual stimuli. But from the 1950s children began less risky activities of watching TV indoors, losing most of the sensual, social and physical interaction chances and challenges that previously gave them a wider variety of tiny risks. Over the last decade children are losing even the little risks of muted social interaction in communal TV watching and the limited amount of sport continued after TV arrived as they spend time in solo computer games. The increase in parents driving their children to educational events operates in the same direction – depriving children of the whiffs of walking bicycling or using public transport, and of wider social interaction.
11	Both conditions arise from risk starvation.

RESEARCH / TREATMENTS

Research

We need research for facilitating the introduction of whiffs, and ascertaining whether whiffs injections would help in either prevention or treatment of bodily stress effects and ailments besides dementia and depression. There is for instance case study material suggesting that this could be so in the case of type 1 diabetes, strokes, epileptic fits, panic attacks and compulsive gambling, (Pope, 2006b).

Our methods should be epidemiological, case histories on whether reductions in particular sorts of risks preceded these mental disorders, and experiments on injecting particular individual and societal packages of whiffs of danger forestall / alleviate conditions. In obtaining case study material, it will be important to obtain information from others besides the sufferer. The type 1 diabetic sufferer might for instance rate his history normal as regards whiffs of danger, and so might his family, on the set of questions used. School friends, more distant relatives, spouse or colleagues, by contrast, may deem it to have been severely lacking in whiffs either because the sufferer was overly protected, or because the sufferer was subjected, not to frequent varied tiny chances and challenges, but to major infrequent unvaried chances and challenges.

This set of information from sufferers then needs to be benchmarked by like histories covering also the history as perceived by the person themselves and the like set of close and not so close family and acquaintances, to assess whether indeed there is a significant difference in the whiffs backgrounds of sufferers and non-sufferers. Once key terms relating to the smallness, frequency and variety of risks are identified, there are software packages that can help by quantifying the frequency of terms used with respect to each person in the sample.

As with any really new theory, the initial evidence of its role as presented here in this paper is qualitative. In the next stages, quantitative relations in relevant dimensions can be estimated / established improving treatment efficacy. With the whiffs of danger / risk starvation theory, the initial evidence has been primarily cross-sectional (epidemiological, prevalence rates). In advancing to longitudinal studies of individuals receiving whiffs to prevent, reverse mental disorders, we need to bear in mind the need for inbuilt continuing whiffs. This is because, as with the enriched environments described for mice and rats in part 5 above, people need a cavalcade of new little chances and challenges. In our research agenda therefore, we need to include checks that we have altered sufferers so that they create these indefinitely for themselves, or altered their environments so as to perpetually provide these. We also need to check on longer term effects – longer than the four year typical cycle to relapse with depressions, and thus far longer than the time perspective of depression treatment reviews currently done in major medical journals, and the stress experiments reported in *Stress*. In turn this means that, in designing studies, we need a proportion that enable follow up at five, ten, even (McCord, 1977), thirty years hence see table 10.

Table 10: Other Research Questions

1	What are the different dimensions of a risk?
2	What are individual differences in when risks in the environment are predominantly too big or too infrequent or too unvaried, for a particular sufferer and thus cause her risk starvation.
3	How do we identify better and more quickly when the depressed suffer risk starvation from the risks in her environment being too small or too numerous or too few through her inability to filter her risks down to a small enough set to process through to effective action?
4	To what extent might the genetic component of diabetes be mitigated by giving the whole family more whiffs even as an enriched environment so dramatically delayed the on set and mitigated the severity of Huntingdon like symptoms in the transgenic mice?
5	Which are the better political / social / psychological ways of altering our society (that we are ever changing) so as to reinstate the once present whiffs for all societal groups, and to discover whichsets of carrots and sticks work better for injecting whiffs into those who are already suffering or in the future likely of suffering risk starvation.
6	Is it better to tackle stress problems primarily through societal changes, not via individual therapies, so as to avoid the sorts of over-riding adverse effects that McCord discovered from social counselling?

Treatment

Society-wide treatment is needed to undo the accidental removal of whiffs from the poor and elderly when governments introduced social security guards against big risks. This needs to be in the form of either enticing or imposing contributions that constitute components of whiffs of danger. There are numerous forms that such communal contributions can take. The following is but one example.

The more educated and socially integrated (and hence productive) children (including those of immigrants) become, the more they can contribute in the future financially and socially to the community. Hence one form of communally contributing is to improve the education of children. Aside from some Scandinavian school systems, education in rich countries is hampered by lack of classroom support personnel for teachers. The lack of support is of a sort that the unemployed and the elderly could provide, and might well enjoy providing.

This is classroom support to help keep order, to teach the local language to their sizable numbers of children from non-native speakers, and to create friendship groups between the native-speakers and the immigrants. Such support could be extended to enhancing the school grounds with plantings that are too labour intensive and too high skill for communities to afford, eg those in the English landscape genre of the most expensive-to-maintain plots of Aston Park Birmingham. Plantings can be done by retired people who belong to the relevant sorts of garden clubs that in most countries maintain such otherwise vanished skills. They can transmit some of these skills to the unemployed and to children, and educate these two groups on this array of sensual whiffs of danger on which the average urbanite misses out, due to his uneducated oblivion.

Let us now consider individual treatments as distinct from those that changes in the social structure can effect. In selecting whiffs for an individual, remember that risks are in the perception and capacity of the sufferer. Begin with injections of sensory, physical, mental, social, ethical risks that are likely to be too small to be whiffs. Only later should we increase the risk dosage, after we gauge that what dosages we initially selected really were too tiny to be whiffs.

In selecting the mental varieties of whiffs, be aware of professionals' tendency to overestimate others' mental skills. Most normal people are incapable of what professionals think of as simple reading and mental arithmetic tasks (Hebb, 1949). Setting non-professionals such tasks is to set major mental challenges coupled with massive social challenges. That is, a medical researcher who gives normal ability people what are to the researcher simple reading and mental arithmetic tasks, is giving those normal people major risks. Their major risks are that they will not avoid the embarrassment and shame of admitting that they are essentially innumerate or illiterate or both. When these normal ability people have pectoral angina and coronary heart disease, they will be prone to silent myocardial ischaemia when confronted with these major risks. This has already happened (Deanfield et al., 1984).

The first steps of injecting the whiffs need involve merely ascertaining the sufferer's typical day, then prescribing a missing whiff – a little physical, social, mental, psychological or ethical challenge. This whiff might be a walk in the forest (sensual components of whiffs) or going to an aerobics class (physical social components of whiffs), or one from the repertoire of tiny challenges entailed in holistic and some cognitive behavioural therapies. As the sufferer's risk processing capacity grows, increase the difficulty (challenge level) of the risks, and their variety and frequency.

For implementing whiffs at the individual and at the societal level, small initial changes in chances and obligations have the like advantage that as yet, we have little knowledge of their precise impact on those in stress from risk starvation, and reforms come with unexpected side effects, some nice, some not. This caution, however is not to suggest that we should only proceed with individual and societal injections of whiffs after more research. We have already the general evidence of the brain's need for risks akin to whiffs. We have already the general evidence of the damage in the case of two

should we increase the risk dosage, after we gauge that what dosages we initially selected really were too tiny to be whiffs.

In selecting the mental varieties of whiffs, be aware of professionals' tendency to overestimate others' mental skills. Most normal people are incapable of what professionals think of as simple reading and mental arithmetic tasks (Hebb, 1949). Setting non-professionals such tasks is to set major mental challenges coupled with massive social challenges. That is, a medical researcher who gives normal ability people what are to the researcher simple reading and mental arithmetic tasks, is giving those normal people major risks. Their major risks are that they will not avoid the embarrassment and shame of admitting that they are essentially innumerate or illiterate or both. When these normal ability people have pectoral angina and coronary heart disease, they will be prone to silent myocardial ischaemia when confronted with these major risks. This has already happened (Deanfield et al., 1984).

The first steps of injecting the whiffs need involve merely ascertaining the sufferer's typical day, then prescribing a missing whiff – a little physical, social, mental, psychological or ethical challenge. This whiff might be a walk in the forest (sensual components of whiffs) or going to an aerobics class (physical social

components of whiffs), or one from the repertoire of tiny challenges entailed in holistic and some cognitive behavioural therapies. As the sufferer's risk processing capacity grows, increase the difficulty (challenge level) of the risks, and their variety and frequency.

For implementing whiffs at the individual and at the societal level, small initial changes in chances and obligations have the like advantage that as yet, we have little knowledge of their precise impact on those in stress from risk starvation, and reforms come with unexpected side effects, some nice, some not. This caution, however is not to suggest that we should only proceed with individual and societal injections of whiffs after more research. We have already the general evidence of the brain's need for risks akin to whiffs. We have already the general evidence of the damage in the case of two common illnesses, dementia and depression, from risk starvation. Failing to start injecting whiffs into our treatments is to choose the less likely path for helping stress sufferers, and especially for attaining enduring cures. It is moreover a case of ignoring the principle of precaution. We should not wish to repeat the mistakes with asbestos and nicotine, of waiting more than 30 to 60 years for "absolute" proof before action. On this historical episode, see the appendix.

ACKNOWLEDGEMENTS

I thank for financial support for the conduct of this research the Centre for Health Economics, Monash University and the Department of Economics, University of Bonn. I thank for comments and background information members of this Monash centre, in particular its foundation director, Jeff Richardson; as also Murray Esler, Baker Heart Research Institute, Monash University, Tony Hannan, the Howard Florey Institute, University of Melbourne; Duncan Foley New School University New York and the Santa Fe Institute; Beverley McGavin, the American Society of Coloscopy and Cervical Pathology; Jerome Kagan, Department of Psychology, Harvard University; Christoph Helmstaedter, Epilepsy Clinic, Bonn University; Kanishka Hiththetiya, Experimental Economics Laboratory, Bonn University; Gerald Hüther, Centre for Psycho-Social Medicine, Neurobiological Research, Department of Psychiatry and Psychotherapy, Gottingen University; Sonia Lupien, Centre for Studies on Human Stress, Montreal University; Alexandra Niessen, Department of Corporate Finance, University of Cologne; Dennis Plachta, Institute for Biomedical Microtechniques University of Freiburg; Howard Rachlin the Psychology Department, State University of New York, Norman Roberts, Old Stone House Community Centre; Gerhard Roth, Department of Behavioural Physiology and Development Neurology, Institute of Brain Research, University of Bremen; Tania E. Schramek Centre for Studies on Human Stress, Montreal University; Reinhard Selten, the Experimental Economics Laboratory, Bonn University; Michael Trimble, Institute of Neurology, National Hospital for Neurology and Neurosurgery, UK; Andrew Whyte, School of Medicine, University of Tasmania; Kerry Whyte, the Austin Repatriation and Medical Centre, Heidelberg; and two exceedingly helpful anonymous referees of this journal. I thank for assistance with the references, proofing and figures Amira Allam (who has donated her time – giving herself whiffs – during her husband's visiting medical research at Bonn University), Andreas Orland of Bochum Univrsity, Deon Marais of the Experimental Economics Laboratory, Bonn University, and Nirosha Paranavitana, Linguistics Department, Bonn University.

REFERENCES

Access Economics. The dementia epidemic: economic impact and positive solutions for Australia. Alzheimer's Australia: Canberra; 2003.

Allen NB, Badcock PBT (2003). The social risk hypothesis of depressed mood: Evolutionary, psychosocial, and neurobiological perspective. Psychol Bull. 129: 887-913.

Barnett SA (1956). Behaviour components in the feeding of wild and laboratory rats. Behaviour. 9: 24-43.

Barnett SA (1957). Experiments on "xenophobia" in wild and laboratory rats'. Br. J. Psychol. 49: 195-201.

Barnett SA (1963). Instinct. Proceedings of the American Academy of the Arts and Sciences. 92: 564-570.

Barnett SA (1972). The ontogeny of behavior and the concept of instinct, in A.G. Karezmar and J.C.

Barnett SA (1977). Biology and freedom. Cambridge University Press, London. Darwin 1770.

Barnett SA, Cowan PE (1976). Activity, exploration, curiosity and fear: An ethological study. Interdisciplinary Science Review 1 (1): 43–61.

Benson H, Klipper M (2000).The Relaxation Response, Updated, expanded edition.Harper Collins, US.

Benson H, Proctor W (1985). Beyond the Relaxation Response. Berkeley Publishing Group, New York

Berto P, D'Ilario D, Ruffo P, Di Virgilio R, Rizzo F (2000). Depression:

Barnett SA (1963). Instinct. Proceedings of the American Academy of the Arts and Sciences. 92: 564-570.

Barnett SA (1972). The ontogeny of behavior and the concept of instinct, in A.G. Karezmar and J.C.

Barnett SA (1977). Biology and freedom. Cambridge University Press, London. Darwin 1770.

Barnett SA, Cowan PE (1976). Activity, exploration, curiosity and fear: An ethological study. Interdisciplinary Science Review 1 (1): 43–61.

Benson H, Klipper M (2000).The Relaxation Response, Updated, expanded edition Harper Collins, US.

Benson H, Proctor W (1985). Beyond the Relaxation Response. Berkeley Publishing Group, New York

Berto P, D'Ilario D, Ruffo P, Di Virgilio R, Rizzo F (2000). Depression: cost-of-illness studies in the international literature, a review. J. Ment. health Policy Econ. 3: 3–10.
Bindu B, Alladi PA, Mansooralikhan BM, Srikumar BN, Raju TR, Kutty BM (2007). Short-term exposure to an enriched environment enhances dendritic branching but not brain-derived neurotrophic factor expression in the hippocampus of rats with ventral subicular lesions. Neurosci. 144 (2).
Blair C (2006). How similar are fluid cognition and general intelligence? A developmental neuroscience perspective on fluid cognition as an aspect of human cognitive ability. Behav Brain Sci. 29: 109–160.
Burke D, Hickie I, Breakspear M, and Götz J (2007). Possibilities for the prevention and treatment of cognitive impairment and dementia. Br J Psychiatry. 190: 371-2.
Calabrese EJ, Bachmann KA, Bailer AJ, Bolgerd PM, Borak J, Cai L, Cedergreen N, Cherianh MG, Chiueh CC, Clarksonj TW, Cook RR, Diamond DM, Doolittle DJ, Dorato MA, Duke SO, Feinendegen L, Gardner DE, Hart RW, Hastings KL, Hayes AW, Hoffmann GR, Ives JA, Jaworowski Z, Johnson TE, Jonas WB, Kaminski NE, Kellery JG, Klaunig JE, Knudsena TB, Kozumboa WJ, Lettieria T, Liuad S-Z, Maisseu A, Maynard KI, Masoro EJ, McClellan RO, Mehendale HM, (2007). Biological stress response terminology: Integrating the concepts of adaptive response and preconditioning stress within a hormetic dose–response framework. Toxicol Appl Pharmacol. 1: 122-128.
Calabrese EJ, Baldwin LA (1997). Hormesis as a Biological Hypothesis: Toxicological Defense Mechanisms and the Shape of Dose-Response Relationships. Environ Health Perspect. 06 (Suppl 1): 357-362.
Camille N, Coricelli G, Sallet J, Pradat-Diehl P, Duhamel JR, Sirugu A (2004). The involvement of the orbitofrontal cortex in the experience of regret. Sci. 304: 1167–70.
Cao X, Huang S, Ruan D (2008). Enriched environment restores impaired hippocampal long-term potentiation and water maze performance induced by developmental lead exposure in rats. Dev. Psychobiol. 50(3):307-13.
Chatterjee A (2004). Cosmetic neurology: The controversy over enhancing movement, mentation, and mood. Neurol. 63: 967–74.
Clynes M (1968). Essentic form-aspects of control, function and measurement Proceedings of the 21st annual Conference of Engineering in Medicine and Biology. Houston, Texas.
Clynes M (1973). Sentics: Biocybernetics of Emotion Communication. Ann N Y Acad Sci. 57-88.
Clynes M (1978). Sentics: The Touch of Emotions. Anchor Press, Garden City.
Clynes M (1988). Generalised emotion, its production, and sentic cycle therapy in Emotions and Psychopathology, M.Clynes and J. Panksepp, eds. Plenum Press, New York.
Cooper CL, Dewe P (2004). Stress – A Brief History. Blackwell Publishing Malden, MA.
Crawley JN (1999). Behavioral phenotyping of transgenic and knockout mice: experimental design and evaluation of general health, sensory
Csíkszentmihályi, M (1990). Flow: The Psychology of Optimal Experience. Harper Collins, New York.
Curtis WJ, Cicchetti D (2007). Moving research on resilience into the 21st century: Theoretical and methodological considerations in examining the biological contributors to resilience. Dev. Psychopathol. 15: 773-810.
Cutler DM (2004). Your money or your life: Strong medicine for America's health care system. Oxford University Press, New York.
Cutler DM, Lleras-Muney A (2008). Education and Health: Evaluating Theories and Evidence, in The Effects of Social and Economic Policy on Health, in House, J., Schoeni, R., Kaplan, G. Pollack, H. Russell Sage Press, New York.
Damasio A (1994). Emotion, reason and the human brain. Avon Books, New York.
Darwin CR (1871). The descent of man, and selection in relation to sex. John Murray, London.
De Jonghe F, Hendricksen M, van Aalst G, Kool S, Peen J Van R, Eijnden E van den, Dekker J (2000). Psychotherapy alone and combined with pharmacotherapy in the treatment of depression. Br. J. Psychiatry. 61: 466–72.
Deanfield JE, Shea M, Kensett M, Horlock P, Wilson RA, De Landsheere CM, Selwyn AP (1984). Silent myocardial ischemia due to mental stress. Lancet. 324: 1001–5.
Denissenko MF, Pao A, Tang M, Pfeifer GP (1996). Preferential formation of benzo[a]pyrene adducts at lung cancer mutational hotspots in P53. Sci. 274(5276): 430-2.
Dudas RB (2005). Anxiety, depression and smoking in schoolchildren - implications for smoking prevention. J. R. Soc. Health. 125: 77–92.
Ebmeier KP, Donaghey C, Steele JD (2006). Recent developments and current controversies in depression. Lancet. 367: 153–167.
Fogel RW (2000a). The Fourth Great Awakening and the Future of Egalitarianism. University of Chicago press, Chicago and London.
Fogel, RW (2000b). The Extension of Life in Developed Countries and Its Implications for Social Policy in the Twenty-First Century. Population and Development Review. Supplement: Population and Economic Change in East Asia. 26: 291–317.
Fogel RW (2003). Secular Trends in Physiological Capital: Implications for Equity in Health Care. NBER Working Paper. 9771, http://www.nber.orgpapers/w9771.
Fogel RW. Changes in the Disparities in Chronic Disease During the Course of the Twentieth Century (2004). NBER Working Paper. 10311, http://www.nber.org/papers/w10311.
Folkman S (2007). The Case for Positive Emotions in the Stress Process. Anxiety Stress Coping. 21(1): 3-14.
functions, motor abilities, and specific behavioral tests. Rev. Brain Res. 835(1): 18-26.
Ganguly M, Dodge HH, Shen C, Pandav RS, DeKosky ST (2005). Alzheimer disease and mortality – A 15-year epidemiological study. Arch Neurol. 62: 779–74.
Greenberg G (2007). Manufacturing Depression: A Journey into the Economy of Depression. Harper's Magazine. May: 35-46.
Greenwald BS, Kramer-Ginsberg E, Marin DB, Laitman LB, Hermann CK, Mohs RC, Davis KL (1979). Dementia with coexistent major depression. Am. J. Psychiatry. 146: 1472–7.
Grootendorst J, DeKloet ER, Dalm S, Oitzl MS (2001). Reversal of cognitive deficit of apolipoprotein E knockout mice after repeated exposure to a common environmental experience. Neurosci. 108: 237-247.
Grootendorst J, DeKloet ER, Vossen C, Dalm S, Oitzl, MS (2001) Repeated exposure to rats has persistent genotype- dependent effects on learning and locomotor activity of apolipoprotein E knockout and C57B1/6 mice. Behav. Brain Res. 125: 249-259.
Grüning T, Gilmore AB, McKee M (2006). Tobacco Industry Influence on Science and Scientists in Germany. Am. J. Public Health. 96(1).
Hasin D, Goodwin R, Stinson F, Grant BF (2005). The epidemiology of major depressive disorder: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. Arch. Gen. Psychiatry. 672: 1097–1106.
Hebb DO (1949). The organisation of behavior: a neuropsychological theory. Wiley: New York.
Hensley PL, Deepa N, Uhlenbuth EH (2004). Long-term effectiveness of cognitive therapy in major depressive disorder. Depress Anxiety. 20: 1–7.
Heylighen F (1992). A Cognitive-Systemic Reconstruction of Maslow's Theory of Self-Actualization", Behav. Sci. 37: 39-58.
Hickie IB, Andrews G, Davenport TA (2002). Measuring outcomes in patients with depression or anxiety: an essential part of clinical practice. Med. J. Aust. 177: 2005–7.
Hockly E, Cordery P, Woodman B, Mahal A, Van Dellen A, Blakemore C, Lewis C, Hannan A, Bates G (2002). Environmental enrichment slows disease progression in R6/2 Huntington's disease mice. Ann Neurol. 51: 235–42.
Horrigan BJ (2007). New studies support the therapeutic value of meditation. Explore. 3(5): 449-52.

Huang L, Cartwright WS, Hu T (1977). The economic cost of senile dementia in the United States. Public Health Rep. 103: 3–7.
Hüther G (1996). The Central Adaptation Syndrome: Psychosocial Stress as a Trigger for Adaptive Modifications of Brain Structure and Brain Function. Neurobiology 47: 569–612.
Hüther G, Sachsse U (2007). Angst- und stressbedingte Störungen: Auf dem Weg zu einer neurobiologisch fundierten Psychotherapie (Damage from Fear and Stress: Towards Neurobiolgically Grounded Psychotherapy). Psychotherapeut. 52: 166–179.
in Mice. Stress. 6: 5-9.
Izard CE (2002). Translating Emotion Theory and Research Into Preventive Interventions. Psychol Bull 127(5): 796-724.
Jacobi F, Wittchen HU, Hoelting C, Hoefler M, Pfister H, Mueller N, Lieb R (2004). Prevalence, co-morbidity and correlates of mental disorders in the general population: results from the German health interview and examination survery (GHS). Psychol. Med. 34: 1–15.
James GD, Brown DE (1977). The Biological Stress Response and Lifestyle: Catecholamines and Blood Pressure, Annu Rev Anthropol. 26: 313-335.
Janis IL, Mann L (1977). Decision making: a psychological analysis of conflict, choice and commitment. Free Press, New York.Jankowsky JL, Melnikova T, Fadale DJ, Xu GM, Slunt HH, Gonzales V, Younkin LH, Younkin SG, Borchelt DR, Savonenko AV (2005). Environmental enrichment mitigates cognitive deficits in a mouse model of Alzheimer's disease. J. Neurosci. 25(21):5217-24.
Jung CG, Jaffe A (1989). Memories, Dreams, Reflections. Random House, US.
Kagan J. (1994). On the nature of emotion. In N. Fox (Ed), The development of emotion regulation: Biological and behavioral considerations. Monogr. Soc. Res. Child. Dev. 59(2-3): 7-24.
Kallistratos G Fasske E (1976). Prevention of 3,4-Benzopyrene carcinogenesis in presence of putrescine, J. Cancer Res. Clin. Oncol. January 77(1).
Khalsa, DS (1998) Integrated Medicine and The Prevention and Reversal of Memory Loss. Alternative Therapies 4:6 (November) 39-40.
Kirsch I (2002a). The emperor's new drugs: an analysis of antidepressant medication data submitted to the U.S. food and drug administration. Prevention and Treatment. (July 15): 5 (23).
Kirsch I (2002b). Yes, there is a placebo effect, but is there a powerful antidepressant drug effect? Prevention and Treatment. (July 15): 5 (22).
Larson EB, Shadlen M-F, Wang L, McCormick WC, Bowen JD, Teri L, Kukull WA (2004). Survival after initial diagnosis of Alzheimer disease. Ann. Intern. Med. 140: 501–9.
Lazarus RS (2000). Toward Better Research on Stress and Coping. Am Psychol. 55(6): 665-673.
Lazarus RS (2006). Emotions and Interpersonal Relationships: Toward a Person-Centered Conceptualization of Emotions and Coping. J Pers. 74(1): 1-46.
Lerner JS, Keltner D (2001). Fear, anger and risk. J. Pers. Soc. Psychol. 81: 146-159.
Liu HC, Chou P, Lin KN, Wang SJ, Fuh JL, Lin HC, Liu CY, Wu GS, Larson EB, White LR, Graves AB, Teng EL (1994). Assessing cognitive abilities and dementia in a predominantly illiterate population of older individuals in Kinmen. Psychol. Med. 24: 763–70.
Loewenstein GF, Weber EU, Hsee CK and Welsch N (2001). Risk as feelings. Psychol. Bull. 127: 267-286
Lupien SJ, Buss C, Schramek TE, Maheu F, and Pruessner J (2005). Hormetic Influence of Glucocorticoids on Human Memory. Nonlinearity Biol. Toxicol. Med. 3: 23-56.
Lupien SJ, Evans A, Lord C, Miles J, Pruessner M, Pike B, and Pruessner JC (2007). Hippocampal volume is as variable in young as in older adults: Implications for the notion of hippocampal atrophy in humans. NeuroImage. 34: 479-75.
Lupien SJ, King S, Meaney MJ, McEwen BS (2000). Child's Stress Hormone Levels Correlate with Mother's Socioeconomic Status and Depressive State. Biol. Psychiatry. 47: 976-970.
Lupien SJ, King S, Meaney MJ, McEwen BS (2001). Can poverty get under your skin? Basal cortisol levels and cognitive function in children from low and high socioeconomic status. Dev. Psychopathol. 13: 653-76.
Lupien SJ, Ouellet-Morin I, Hupbach A, Tu M T, Buss C, Walker D, Pruessner J, and McEwen BS (2006). Beyond the Stress Concept: Allostatic Load-A Developmental Biological and Cognitive Perspective. Developmental Psychology 2 – Developmental Neurscience 2nd edition. Wiley, Hoboken New Jersey.
Mann JJ (2005). The medical management of depression. N. Engl. J. Med. 353: 1719–1734.
Marks IM (2002). The maturing of therapy. Br. J. Psychiatry. 170: 200–4.
Marmot MG (1999). World Health Organisation Regional Office in Europe,
http://www.euro.who.int/socialdeterminants/socmarketing/20050912_1
Marmot MG (2001). Editorial: Inequalities in Health N. Engl. J. Med. 345 (2). Neurol. 51: 235–42.
Marmot MG (2002). Redefining Public Health, Epidemilogy and Social Stratification: Conversations recorded by Harry Kreisler in Conversations with History. Institute of International Studies, UC Berkeley. http://globetrotter.berkeley.edu/people2/Marmot/marmot-con3.html
Marmot MG, Bosma H, Hemingway H, Brunner E, Stansfeld S (1997). Contribution of Job control and other risk factors to social variations in coronary heart disease incidence. Lancet. 26: 350(9073): 231-232.
Marmot MG, Shipley MJ, Rose G (1984). Inequalities in death - specific explanations of a general pattern? Lance. 1:1003-6.
McCord J (1977). A thirty year follow-up of treatment effects. Am Psychol. Pp.274-279.
McEwen BS (1997). Possible mechanisms for atrophy of the human hippocampus. Mol. Psychiatry 2: 255-262.
McEwen BS, Seeman TE (1999). Allostatic Load and Allostasis. John D and Catherine T MacArthur Research Network on Socioeconomic Status and Health.
http://www.macses.ucsf.edu/Research/Allostatic/notebook/allostatic
McEwen BS, Wingfield JC (2003). The concept of allostasis in biology and biomedicine. Horm. Behav. 43(1): 2-15.
Mian R, Shelton-Rayner G, Harkin B, Williams P (2003). Observing a Fictitious Stressful Event: Haematological Changes, Including Circulating Leukocyte Activation. Stress. 6: 41-47
Miyanaga K (2005). Treatment of dementia. Nippon Ronen Igakkai Zasshi. 42: 49–51.
Modelling, Int. J. Syst. Manage. 22 (1): 1-12.
Moore T (1992). Care Of The Soul: How To Add Depth And Meaning To Your Everyday Life, 1st Edition. Piatkus Books, United Kingdom.
Mothersill C, Newlin DB, Niggal HN, Oehme FW, Phalen RF, Philbert MA, Rattan SIS, Riviere JE, Rodricks J, Sapolsky RM, Scott BR, Seymour C, Sinclair DA, Smith-Sonneborn J, Snow ET, Spear L, Stevenson DE, Thomas Y, Tubiana M, Williams GM, Mattson MP Murray CJL, Lopez A (1996). The Global Burden of Disease: A comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. World Health
Neveu P (2003). Cycotine Stress Responses Depend on Lateralization
O'Shea E, Reilly S (1999). The economic and social costs of Alzheimer's disease and related dementias in Ireland: an aggregate analysis. Int. J. Geriatr. Psychiatry. 15: 207–17.
Organisation, World Bank and Harvard University School of Public Health: I, Harvard University Press, Harvard.
Pagnoni G, Cekic M (2007). Age-related changes in grey matter volume and attentional performance associated with the practice of Zen meditation, in Büchel, C, Nichols, T, Mesulam, M., eds., NeuroImage 36 S1–S125 NeuroImage: Special Issue – 13th Annual Meeting of the Organization for Human Brain Mapping, 55-56 TH-AM
Pascal B (1670). Pensées. Reprinted by Hachette CL (1845) Paris
Patmore A (2006). The Truth About Stress. Atlantic Books, London.
Paykel ES, Scott J, Teasdale JD, Johnson AL, Garland A, Moore R, Jenaway A, Cornwall PL, Hayhurst H, Abbott R, Pope M (1999). Prevention of Relapse in Residual Depression by Cognitive Therapy:

A Controlled Trial. Archives of General Psychiatry. 56: 729–35.
Paykel ES, Scott J, Cornwall PL, Abbott R, Crane C, Pope M, Johnson AL (2005). Duration of relapseprevention after cognitive therapy in residual depression: follow-up of controlled trial. Psychol Med. 20: 1–7. Piccinelli M, Wilkinson G (2000). Gender differences in depression. Critical review. Br. J. Med. Psychol. 177: 476–92.
Piccinelli M, Wilkinson G (2000). Gender differences in depression. Critical review. Br. J. Med. Psychol. 177: 476–92.
Pinquart M, Soerensen S (2001). How effective are psychotherapeutic and other psychosocial interventions with older adults? A meta analysis. J. Ment**.** Health Aging. 7: 207–243.
Plachta D, Hanke W, Bleckmann H (2000). The responses of midbrain lateral line units of goldfish (Carassius auratus) to water movements visualized with particle image velocimetry. Zool. Suppl. III: 20-21.
Pope RE (1983). The pre-outcome period and the utility of gambling. In: Stigum BP, Wenstøp F (Eds), Foundations of Utility and Risk Theory with Applications. Reidel, Dordrecht, 1973. pp. 37–177.
Pope RE (1985). Timing contradictions in von Neumann and Morgenstern's axioms and in Savage's sure-thing proof. Theory and Decision. 17: 229–61.
Pope RE (1995). Towards a More Precise Decision Framework, A Separation of the Negative Utility of Chance from Diminishing Marginal Utility and the Preference for Safety", Theory and Decision 39 (3): 241-265.
Pope RE (2004). Biases from omitted risk effects in standard gamble utilities. J. Health Econ. 25: 695–735.
Pope RE (2005). The riskless utility mapping of expected utility and all theories imposing the dominance principle: its inability to include loans, commitments even with fully described decision trees. In: Schmidt U, Traub S (Eds), Advances in Public Economics: Utility, Choice & Welfare. Springer, Dordrecht, pp. 279–327.
Pope RE (2006a). The Illusion of Risk Effects in Stochastic Industrial
Pope RE (2006b). Whiffs of Danger to Reduce Anxiety Attacks, Epileptic Fits and Strokes. Mimeo.
Pope RE, Leitner J, Leopold-Wildburger U (2006). The Knowledge Ahead Approach to Risk: Theory and Experimental Evidence. Springer Lecture Notes.
Pope RE, Selten R, Kube S, von Hagen J (2008). Experimental Evidence on the Benefits of Eliminating Exchange Rate Uncertainties and Why Expected Utility Theory causes Economists to Miss Them. Indian J. Econ. Bus. 7(1), 1-31.
Pope, RE Leitner J, Leopold-Wildburger U (2009). Expected Utility versus Changes in Knowledge Ahead," Eur. J. Oper Res. 199 (3): 892-901.
Reid IC, Stewart CA (2001). How antidepressants work: new perspectives on the pathophysiology of depressive disorder. Br. J. Psychiatry. 177; 299–303.
Reinherz HZ, Paradis AD, Giaconia RM, Stashwick CK, Fitzmaurice G (2003). Childhood and adolescent predictors of major depression in the transition to adulthood. Am. J. Psychiatry. 160: 2141–7.
Reynolds CF, Dew MA, Pollock BG, Mulsant BH, Frank E, Miller MD, Houck PR, Mazumdar S, Butters MA, Stack JA, Schlernitzauer MA, Whyte EM, Gildengers A, Karp J, Lenz E, Szanto K, Bensasi BS, Kupfer, DJ (2006). Maintenance Treatment of Major Depression in Old Age. N. Engl. J. Med. 354: 1130–1137.
Riedel-Heller SG, Busse A, Aurich C, Matschiner H, Angermeyer MC (2001). The prevalence of dementia according to DSM III-R and ICD-10: results of the Leipzig longitudinal study of the aged (LEILA75+) Part 1. Br. J. Psychiatry. 179; 250–254.
Roth G (2001/2003). Fühlen, Denken, Handeln. Wie das Gehirn unser Verhalten steuert. Suhrkamp, Frankfurt.
Rozman S (2002). Treatment of Problem gambling. European association of study of Gambling.Salamone JD (2002). Antidepressants and placebos: conceptual problems and research strategies. Prevention and Treatment 2002 (July): 5 (24).
Samson L, Cairns J (1977). A new pathway for DNA repair in Escherichia coli. Nature. 267(5608): 281-283.
Scitovsky T (1971). The Desire for Excitement in Modern Society. Kyklos.
Scitovsky T (1976). The Joyless Economy: An Inquiry into Human Satisfaction and Consumer dissatisfaction. Oxford University Press, Oxford.
Scitovsky T (1999). Proud Hungarian. The Hungarian Quarterly XL. 155:33–53 and 156:24–43.
Seligman MEP (1975). Helplessness: On Depression, Development and Death. Freeman, New York.
Seligman MEP (1997). Learned optimism. Simon and Schuster, New York.
Selye H (1974). Stress without distress. Free Press, New York.
Sen AK (1999). Development as Freedom. Knopf, New York.
Shaji S, Bose S, Verghese A (2005). Prevalence of dementia in an urban population in Kerala, India. Br. J. Psychiatry. 176: 136–140.
Shorter, E (1977). The Making of the Modern Family. Basic Books, New York.
Simon HA (1967). Motivational and emotional controls of cognition. Psychol. Rev. 74:29–39.
Simon HA (1979). Rational Decision Making in Business Organizations. Am. Econ. Rev. 69: 493-513.
Selye H (1974). Stress without distress. Free Press, New York.
Sen AK (1999). Development as Freedom. Knopf, New York.
Shaji S, Bose S, Verghese A (2005). Prevalence of dementia in an urban population in Kerala, India. Br. J. Psychiatry. 176: 136–140.
Shorter, E (1977). The Making of the Modern Family. Basic Books, New York.
Simon HA (1967). Motivational and emotional controls of cognition. Psychol. Rev. 74:29–39.
Simon HA (1979). Rational Decision Making in Business Organizations. Am. Econ. Rev. 69: 493-513.
Suh GH, Shah A (2001). A review of the epidemiological transition in dementia – cross-national comparisons of the indices related to Alzheimer's disease and vascular dementia. Acta Psychiatr. Scand. 104:4–11.
Sullivan PF, Neale MC, Kendler KS (2000). Genetic epidemiology of major depression: review and meta-analysis. Am. J. Psychiatry. 157:1552–1562.
Teasdale JD, Segal ZV, Williams JMG, Ridgeway VA, Soulsby JM, Lau MA (2000). Prevention of Relapse/Recurrence in Major Depression by Mindfulness-Based Cognitive Therapy. J. Consult. Clin. Psychol. 67:615–623.
Thompson S (2001). Complementary therapies in aged care. In: McCabe, P. (ed.) Complement Ther Nurs Midwifery. Ausmed, Melbourne.
Turner RJ, Lloyd DA (2004). Stress burden and the lifetime incidence of psychiatric disorder in young adults: racial and ethnic contrasts. Arch Gen Psychiatry. 61:471–477.
Von Neumann J, Morgenstern O (1947/1953/1972). Theory of Games and Economic Behavior. Princeton University Press, Princeton, New Jersey.
Wagner JC, Sleggs CA, Marchand P (1960) Diffuse pleural mesothelioma and asbestos exposure in the North Western Cape Province. Brit. J. Indust. Med. 17:260-271.
Weich S, Blanchard M, Prince, M, Burton E, Eren B, Sproston K (2002) Mental health and the built environment: cross-sectional survey of individual and contextual risk factors for depression. Br. J. Psychiatry. 170:427–433.
Yang J, Li W, Liu X, Li Z, Li H, Yang G, Xu L, Li L (2006). Enriched environment treatment counteracts enhanced addictive and depressive-like behavior induced by prenatal chronic stress. Brain Res. 1125:132-137.

Appendix: Medical evidence norms contrasted with the precautionary principle

The principle of precaution is in the Science and Environmental Health Network's Wingspread 1977 Conference Statement.

When an activity raises threats of harm to human health or the environment, precautionary measures should be taken even if some cause and effect relationships are not fully established scientifically.

Since evidence can never be fully established scientifically, the above statement ought to be uncontroversial. Instead, the medical evidence norm is to erroneously believe that there is such a thing as “absolute proof”, and to improperly delay taking action on the basis of robust epidemiological findings, as shown below.

Epidemiological evidence that had been garnered before World War 2 that asbestos causes one form of lung cancer, namely Mesothelioma, and cigarette smoking causes another form, namely bronchogenic carcinoma. By the mid 1930s, some medicos were seeking to deter asbestos mining and cigarette smoking as lung cancer hazards. But as a concerted voice, the medical profession held back. It failed to apply the precautionary principle. It did so on the grounds that epidemiological correlations of asbestos and cigarette smoke with forms of cancer do not *prove* causation.

As a concerted voice, the medical profession only granted that asbestos caused Mesothelioma after Wagner et al. (1960) identified some intermediate links. See Figure A1.

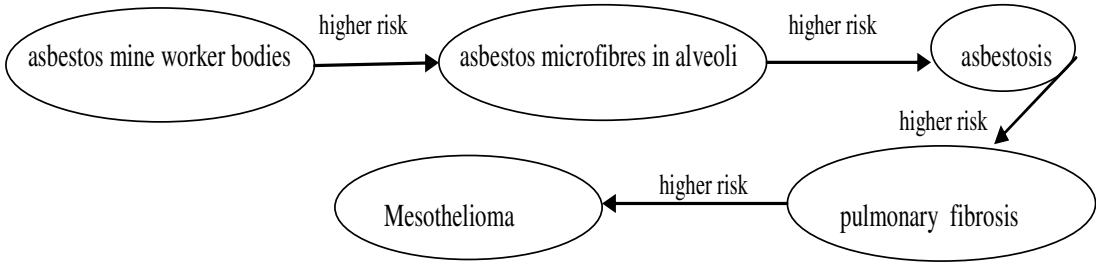

Figure A1. Causal links resulting in Mesothelioma.

It took another 35 plus years, till Denissenko, Pao, Tang and Pfeifer (1996) identified Benzo-Pyrene as an intermediate link between cigarette smoke and bronchogenic carcinoma. See Figure A2.

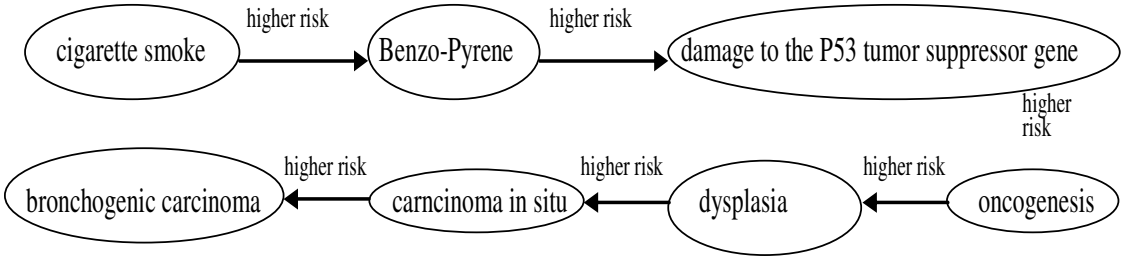

Figure A2. Causal links resulting in bronchogenic carcinoma

The time lapse between the solid epidemiological evidence being supplemented with information on intermediate links was 25 years, in the case of asbestos and 60 years in the case of cigarettes. Over these decades, a sizable body of medical practitioners received salaries from asbestos and cigarette-related firms. As regards medical practitioners employed in the tobacco industry, from subsequent US freedom-of-information releases, a number worked to reinforce the medica profession's readiness to ignore pure epidemiological evidence, Grüning, Gilmore and McKee (2006). The methodological warning that correlation does not prove

causation has aided this ignoring of pure epidemiological evidence, even when the pure epidemiological evidence is very solid.

Indeed correlation does not prove causation. But then we never prove causation. No-one literally sees a cause. All our scientific evidence is derived from direct or indirect correlations. If the medical profession were consistent in its insistence on discovering all the intermediate links, it would deny that we have evidence linking asbestos and cigarette smoke to lung cancers. It would to this day say that evidence is lacking since we have not yet adequately established all these other causal factors and their relative importance. Each link is identified as a cause of the next stage in morbidity partly because of its *direct* correlation with the morbidity. We exclude some other correlations that we could have added because they fail to fit our larger cause-effect model of the world. In turn this larger cause-effect model is built indirectly from a larger body of other correlations.

We accept a correlation as evidence for causation because this correlation does not conflict with other correlations that enter our larger adult cause-effect model – not because we literally see causation. All we literally see in the cause-effect sequence leading to bronchogenic carcinoma, is the correlation of one stage in the process, eg the production of Benzo-Pyrene, followed by sequel stages, eg the stage of carcinoma in situ.

We do an experiment to counter the oncogenetic effects of the Benzo-Pyrene, then observe that the oncogenesis in situ is postponed, Kallistratos and Fasske (1976). We may rashly say that we see causation, have proved causation by our inhibition experiment. But we have not literally seen causation, merely strong evidence for this causal link.

We have strong evidence, not proof, for many reasons. One reason is that we have not seen, and never shall, see every instance and every magnitude of each factor that we infer is a cause. Thus earlier correlations had shown that biological hazards were positively correlated with reduced immunity, and had drawn the conclusion that biological hazards cause the reduced immunity. Sequel researchers examined the correlation for varying amounts of biological hazards. They found that the negative correlation only held for high levels of biological hazards and that for low levels, the reverse correlation obtained.

Nowadays an inverted U-shaped or Beta relation is seen as the correlation between the amount of biological hazards and immunity. Nowadays we infer that a little dirt is good for the immune, that a little dirt causes a better immune system. The evidence is strong, but we do not literally see the little dirt causing the improvement. All we literally see is that the correlation is much better than when we had the crude all or nothing theory that all dirt is bad for the immune system. Consistency requires that we likewise treat findings from epidemiological correlations as evidence.

Using the fact that correlation does not prove causation to exclude epidemiological evidence, leaves medical evidence subject to the impossible evidence requirements of total reductionism. Under reductionism, each level of causation is reduced to a more detailed underlying level. In the case of medicine, these underlying levels concern intermediate linkages in the form of successive bodily changes caused by the environmental factor prior to the morbidity occurring. Getting evidence on such intermediate links however is an endless process. Being consistent in insisting on intermediate links, therefore, precludes ever concluding anything – precludes all evidence-based medicine.

For neither asbestos nor for cigarettes have all the links been discovered. This can be seen on two levels. One level is to note that few of these intermediate causal links are fully described, either as regards timing, or as regards the component atoms, and their subatomic elements and so on. The other level is to note that each each link in the causal chain merely increases the risk of progressing to the next stage of the pathology. It does not ensure that the patient progresses to the next stage, nor even that the patient does not have an entire remission of the pathology. This means that there are in addition other causes. Some of these other causes are solidly established, eg that cigarette smoking massively increases the probability of damage from asbestos.

Others of these multiple causes are less solidly established, eg that physical fitness, mental outlook and genetic predisposition, play a role. If therefore the medical profession were consistent in its insistence on discovering all the intermediate links, it would deny that we have evidence linking asbestos and cigarette smoke to lung cancers. It would to this day say that evidence is lacking since we have not yet adequately established all these other causal factors and the relative importance of each.

In short, the current medical evidence model is inconsistent in its demands for intermediate links as these can never be fully specified, and in its discarding of *all* purely epidemiological evidence. The history of decades of morbidity from asbestos and from cigarettes arose through the current medical model being defective and in disaccord with the principle of precaution. The relevance of pure epidemiological evidence moreover, is not missed by everyone in the medical profession. There has been some unease at the way medicos continued in supposedly objective research papers and treatment of firm clients, to sidestep the epidemiological evidence on the causes of Mesothelioma and bronchogenic carcinoma. This paper has focused on epidemiological evidence to argue that a decision oriented approach can aid in our construction of theories/hypotheses of the biology of stress, and in therapies ensuing.