**Prototype for a *Scientific* Classification of Mental Disorders**

**Public Lecture given by Robert Miller at Department of Psychiatry, Li Ka Shing Faculty of Medicine, The University of Hong Kong, 7.00pm, April 12th 2016.**

**Abstract:** The classification of mental disorders, as used by the psychiatric profession in the last century, has never been based on true scientific reasoning. The one pioneer who attempted this - long neglected even in the German-speaking world - was Carl Wernicke, whose life’s work remained incomplete, due to his premature death in 1905. Recently, in collaboration with John Dennison of Otago University, I was involved in producing the first available edited English translation of Wernicke's *Grundriss der Psychiatrie*from 1906.Inspired by this seminal work, I can now see at least the framework for recasting the description and classification of mental disorders, based on a neuroscience-based concept of human nature in its vast variety. This also draws on some of my own theory of normal brain function, and work of pioneers such as Ernst Kretschmer and Victor Frankl.

It is always good to come back to the University of Hong Kong. I find here an intellectual vigour, and a willingness to tackled the hardest questions which I rarely find elsewhere. . . Butlet me start by explaining my title. I refer to ‘scientific’ classification of mental disorders. The natural science tradition emerged in the seventeenth century, and was first formulated, I believe by Francis Bacon, in his major work – *Novum Organum* - from 1620. Here is a crucial quote:-

 Those who have handled sciences have been either men of experiment or men of dogmas. The men of experiment are like the ant, they only collect and use; the reasoners resemble spiders, who make cobwebs out of their own substance. But the bee takes a middle course: it gathers its material from the flowers of the garden and of the field, but transforms and digests it by a power of its own. Not unlike this is the true business of philosophy: for it neither relies solely or chiefly on the powers of the mind, nor does it take the matter which it gathers from natural history and mechanical experiments and lay it up in the memory whole as it finds it, but lays it up in the understanding altered and digested. Therefore from a closer and purer league between these two faculties, the experimental and the rational (such as has never been made), much may be hoped.

(from Francis Bacon: *Novum Organum,* 1620).

So, in brief, we could define that tradition as ‘a method of combining reasoning and empirical evidence, as a way to approach “truth” about the natural world’. Bacon resolved a debate which had been fought over for the previous 2000 years between empiricism and rationalism as the way to truth. Modern insistence on ‘evidence based’ medicine, or policy, has thus missed one half of the tradition, although it probably thinks itself as being scientific. In any case, in study of mental disorders, a serious question is begged: Is the human mind part of the natural world? . . .or is it better dealt with by theologians, philosophers, or legal minds?

What about the word *classification?* This is a formalised type of description. I ask: ‘Can description ever be neutral with respect to theory? I don’t believe so. Carl Linnaeus – the seventeenth century Swedish scholar who classified plants - he also classified diseases - held that classification should be based on the reproductive organs of plants - a shrewd hunch you might say, which gained some sort of rationality after publication of Darwin’s *Origin of Species.* For Linnaeus himself however it lacked any rational basis – but it was not theory-neutral. Likewise, DSM[[1]](#footnote--1) claims to be theory-neutral, but implicitly assumes that disorders are *imposed* on normal mental function, rather than being an imbalance of normally-present factors – a debate between two models of disease which also goes back to antiquity – but which DSM never addresses.

Often theoretical assumptions are not made explicit, and may be unknown even to those who propose a scheme of classification. Given this, overtly or covertly, classification is often based on authority – of a person, or of an organization; to put it crudely, those with the biggest boots. However, there is no room for authority in the science tradition, other than the authority of reason and evidence, carefully combined. So, most systems of classification, lack scientific rationality.

Linnaeus appears to have been quite a bombastic individual. He had the temerity to name the species of which he was a member *Homo sapiens.* Yeah right! *Homo vana[[2]](#footnote-0)* might have been a bit more accurate. In classifying plants, he thought he was defining a natural order, a set of ‘natural types’. That was transferred in the nineteenth century to classification of diseases, with the same Latin binomial terminology. With hindsight, you could now say that, for plants at least, Linnaeus *was* mainly correct; but in his own day it was not obvious. His contemporary, the Frenchman Georges Buffon, had the aphorism – ‘Natural knows only the individual’. That line has singular application in psychiatry, and I suggest that there the issue is not resolved.

 Let’s move on to the relevant history of classification in psychiatry. We can start with *Wilhelm Griesinger,* and his *Einheitspsychose* concept – unitary psychosis. That idea probably grew from debate in the Germanic world amongst romantic philosophers, rejecting the endless tendency to conceptual splitting of earlier philosophers from the ‘Age of Reason’, so that somehow the mind - whether in health or sickness – was taken to be a unity.

The next person is *Karl Ludwig Kalhbaum* – a fine clinician, I understand. He never held an academic position, and his significance was grasped only after his death. He stressed careful clinical interviewing – and documenting the longitudinal trajectory of each patient’s disorder. He rejected the Unitary Psychosis concept, and thus constructed what was probably the first system of classification in the Germanic world. At the time, his system was held to be too complicated to be used in practice, although some of his concepts survived. The concept I draw on – only a small part of his system – is that there are ‘transitional’ stages of life, when people become specially vulnerable to particular forms of mental disorder. He stressed late adolescence - hence the concept of hebephrenia, from which the concepts of *Dementia praecox* and later schizophrenia arose - and the transition that occurs in the later years of life, when dementia of the elderly becomes more likely.

Then we come to *Emil Kraepelin*. He still dominates classification in psychiatry, so I need to go into detail. He claimed to base his work on Kalhlbaum’s, but was far less a true clinician than Kahlbaum, let alone a clinical scientist. His term *Dementia praecox*, was a Latin version of the French term *Démence précoce –* of Benedict Morel, a designation – as a form of dementia - which most experts today would regard as completely wrong. It is said that he based his classification on evidence about many patients, compiled in a complex ‘diagnostic card system’ (*Zählkarten)*. The most important distinction we owe to Kraepelin is that between manic depressive illness (‘bipolar disorder’), and *Dementia praecox* (which became schizophrenia), first made public in 1898. It is claimed that the distinction was based on a combination of long-term course of an illness, and symptom profile. Some of Kraepelin’s cards from that period still exist. An analysis of 705 cards from 1887 to 1904[[3]](#footnote-1) found they were often incomplete, rarely contained anything but the most rudimentary detail of symptoms, and in 54% no detail on course. German Berrios (editor of the journal in which this paper appeared) comments that these cards cannot be seen as the sole evidential basis for the classification system, without analysis being ‘guided by deep *a priori* views as to how objects in his field should be defined, explained and classified’; yet the distinction still exists, without fundamental change. So much for the modern insistence on ‘evidence based medicine’!

Since Kraepelin is still important, it is worth looking more closely at his credentials. His medical studies began at Leipzig, where he came to know Paul Flechsig (foremost neuroanatomist of the day). He graduated there in 1878 and went to Munich to work under Bernhard von Gudden, an asylum director, who did some research in neuroanatomy, but was never interested in clinical psychology of his patients, nor in careful interviewing. Kraepelin’s work in back wards there was not only unpleasant, but at times very frightening, with the violent behaviour he sometimes witnessed[[4]](#footnote-2). It is unlikely that he received expert guidance in either clinical interviewing or interpreting what he might hear in a clinic; nor, in all probability, did he meet patients in situations where careful interviewing was even possible.

After leaving Gudden, Kraepelin returned to Leipzig and worked briefly under Flechsig (who was also responsible for psychiatric wards). Accounts differ[[5]](#footnote-3), but it appears that he was dismissed by Flechsig, probably because ‘hygienic conditions in his wards were unacceptable and he did not take good care of his patients.’ Whatever the truth, there were hard feelings between him and Flechsig; and at this point, it seems, he had no money, no job, and no possibility of further training. However, in 1886 he applied for and was appointed to the chair of psychiatry at the University of Dorpat (present day Tartu, in Estonia), part of Russia at the time, more remote even than Königsburg, 400 km south-west (in German-speaking East Prussia). The significant point about the Dorpat years was that he could not converse with patients in their own language – Estonian and other languages - only with the help of an interpreter[[6]](#footnote-4). This severely limited his effectiveness as a clinician and in clinical research. In 1891 he returned to Germany, to a prestigious chair at Heidelberg, and the rest (as they say) is history. The key point is that he *still* appears to have had little real clinical experience, and no chance to acquire interviewing skills. His fame grew later not as a clinician-scientist, but as an administrator of asylums, and later of a research institute, and as a wide-ranging mental health political advocate.

This detail may seem to be a diversion. It is actually highly relevant. Kraepelin’s system for separating *Dementia praecox* from Manic depressive disorder appears to have depended more on what he thought the end-state would be than on full information on the course of a disorder, as Kahlbaum would have advised. Thus it is an administrator’s classification, to separate those who would get better from those who could never leave the asylum, not that of a clinician-scientist, to aid personal health care. Systems for diagnosis since then have *never* been based comprehensively on scientific reasoning. Psychiatric diagnosis as it evolved over the last century was not designed to help the practice of conscientious health professionals – of which there have been many - concerned about personal healthcare, but rather to serve the interests of administrators. In Kraepelin’s days this was administration of asylums; today, in DSM, ICD, or whatever, it is administration of health finances, especially in the USA. These are sharp words; but my view is even worse: At present, diagnostic systems are characterise by incoherence, more than by their serving *any* purpose with clear logic. *Any* diagnostic entity I have studied turns out, on closer scrutiny to be an unruly mix of different types of disorder, which might then be regrouped in completely different ways, which could be more useful to practitioners and patients alike.

 Now we come to the real pioneer – *Carl Wernicke.* He is known in the English speaking world, as a pioneer of neurology. In fact, for the last twenty years of his life, he worked in a psychiatric institution. In the last two or three years, with John Dennison – a linguist friend of mine in Otago - we have produced the first available edited English translation of his *Grundriss der Psychiatrie[[7]](#footnote-5).* Sadly, Wernicke died prematurely in a bicycling accident in 1905, his life’s work incomplete; and his work was soon eclipsed by other prominent figures at the time, especially Kraepelin. Wernicke was usually generous to his professional rivals, but for Kraepelin he has nothing but withering scorn.

Without hesitation, I regard the work which we translated and edited (41 lectures, given to his very advanced students), as the best scientific writing on psychiatry I have ever read; and the only person I have read who really tried to develop a system of classification based on scientific reasoning; and he also appears to have been a skilled and compassionate clinician.

Let me give detail here on what Wernicke achieved as a scientist. In his day, the neurone theory was hot news, but no-one had a clue about the nature of signals carried by nerve cells. Nonetheless, in the 1890s, he had worked out (i) that the fundamental basis for learning was modification of groups of synapses converging on cortical nerve cells; (ii) that the cerebral cortex was ‘the organ of association’; (iii) that any percept or concept which aroused subjective awareness was represented not by single nerve cells but by collections of connected nerve cells scattered across the cortex. Here he was 50 years ahead of Donald Hebb, to whom we usually attribute that insight[[8]](#footnote-6); (iv) He saw the trove of memories so acquired – contents of consciousness in Wernicke’s terms – as falling into three parts, *first* our awareness of the continuity over time of our own body – the awareness from which we first gain a notion of ourselves as enduring entities – the ‘primary Ego’ in Wernicke’s terms[[9]](#footnote-7). *Second,* the continuities we learn, in large part from our ‘distance senses’ from the external environment (including our social environment); and *Third*, the unique set of episodic memories we each accumulate about our own life story. (v) His theory of personhood: Here we see Wernicke at his very best, and I think well ahead of most of *today’s* practitioners. With the cortex as the organ of association, we strive through our lives to assimilate the three components of memory, to create for each of us a ‘sense of self’ (using modern terms) – that is, throughout our lives, we construct and continually reconstruct our sense of personhood, to be as fully integrated as possible.

It is Wernicke’s inspiration which emboldens me to think I can produce a system to describe and classify mental disorders, which really *is* suitable for today’s clinical scientists working in mental health, a system based on solid scientific reasoning, which *does* aid clinical practice of physicians dedicated to personal health care, in a way which DSM or ICD, in their various versions, never did.

Today additional forces are at play which make wholesale reconstruction of the ways of describing and classifying mental disorders a high priority.

(i) Getting away from academia, sixteen years ago, and knocking around with community groups, has exposed me to a wide variety of conceptual languages to describe mental disorders. Sometimes these have been carefully thought out and coherent ways of thinking, albeit challenging to orthodoxy, with which I have much sympathy; sometimes they are less coherent, or even just emotional polemics.

(ii) In 2013 there was a major dispute between the American Psychiatric Association and NIMH over DSM 5, released in May of that year. NIMH has let it be known that it is moving away from DSM categories in the research it supports. Three years later, DSM-5 is hardly used at all in NZ.

(iii) The debate is especially sharp over the diagnosis of schizophrenia. My book on the theory of schizophrenia was published in 2008 - and you might expect me to be a staunch supporter of the S-word, and the underlying concept. Not so. The book never made use of official definitions. It started from three preliminary criteria to define the area of interest – [a] occurrence of episodes of active psychosis; [b] a constellation of non-psychotic traits, involving most areas of psychological function; [c] peak age of onset in late adolescence or early adulthood. I intended to refine these as I developed the theory – and I did that, with regard to the first two; but the third – the age-of-onset criterion - was seldom referred to, and never in a crucial way. In addition, the largest part of the book attempted to explain, on the basis of assumptions about the brain, all the non-psychotic traits associated with schizophrenia (as diagnosed). Eight years later, I see no reason to reject the reasoning used to explain those traits; but I realise that they are not specific to schizophrenia. They sometimes occur in the general population, and are probably present at elevated rates in persons given other diagnoses (autism, ADHD, dyslexia etc). So now, I have stopped using the S-word, as though it had any meaning of scientific significance.

 So now, let’s get on to the Basic Principles for a Scientific Classification:

(i) At the heart of a scientific classification there should be *conceptual clarity*. Again, I quote Bacon:-

‘If the notions themselves (which is the root of the matter) are confused and over-hastily abstracted from the facts, there can be no firmness in the suprastructure’.

So, how do we move from the present state, of basic concepts which are ‘confused and over-hastily abstracted from the facts’, to a situation where concepts are robust, and therefore of greater practical use?

(ii) *Cross-level explanations.* There is a maxim which has guided me for many years, which I recently included in a review paper on the subject of mental disorder:-

The *only* way in which scientific concepts can be validated in a way which will stand the test of time, is to find a way of defining them which supports and is supported by strong explanatory arguments[[10]](#footnote-8).

By ‘explanatory arguments’ I refer especially to what I call *‘cross-level explanations.*’ A sound explanation should be based on assumptions *external* to what it is intended to explain, otherwise there is a danger of explanation being circular. In psychiatry, this means that explanations of psychological findings in terms of psychological concepts or assumptions runs this risk. In other areas of the natural sciences, explanations which really made history explained a range of findings at one level, in terms of assumptions at a lower level, themselves grounded in the existing common language of the natural sciences. In psychiatry, that means explaining psychological findings in terms of assumptions at the level of nerve cells, their morphology and biophysics – and possibly also transmitter neurochemistry, although this has been over-emphasised, because of the power of the drugs industry.

(iii) *Normal in relation to abnormal.* In general medicine, understanding an abnormal situation has usually arisen in close relation to understanding the normal one. Sometimes one takes the lead, sometimes the other, but the two usually advance roughly in parallel. So we came to understand diabetes mellitus in parallel with understanding the endocrine role of the pancreas; or heart disease in relation to normal heart function. For psychiatry, that implies that we need to have a scientific theory of human nature in order to understand mental disorders. Hitherto, going back to Plato, human nature has been defined by philosophers or theologians – along the lines that we are fundamentally rational beings – Linnaeus’s *Homo sapiens* again. This is hardly credible. *Some* of us are *slightly* rational, *some* of the time. The scientific revolution of the seventeenth century, and the Age of Reason, in the following century, overlooked this question. Supposedly scientific psychiatry grew by grafting its specialist knowledge onto a model of human nature which is not itself grounded in the common language of the natural sciences. As a result there has been something of a dysjunction between claims the profession makes about its scientific status and the reality. Wernicke’s model of human personhood, which I outlined, is a magnificent start to resolving this; but today we can do much better, based on modern understanding of the brain.

 (iv) *Representation of contexts.* Wernicke’s designation of the cerebral cortex as the ‘organ of association’ is essentially correct, but not the whole story: As an organ of association, the cortex cannot represent percepts or concepts unambiguously: To resolve that ambiguity, representation of percepts and concepts has to locate them in their appropriate ‘context’. For mammalian species generally, many of which are territorial, as they navigate through their environment, the most important such context is a spatial one, a function for which the brain structure called the hippocampus plays a crucial role[[11]](#footnote-9). This insight led to John O’Keefe being awarded the Nobel prize 18 months ago. However, it always struck me that, for humans, we should view the term ‘context’ in a more abstract and general way, for the many varieties of information processing, all needing different contexts, with which we operate; but the basic principle is the same: To have the right context active in the brain permits us to do things that are beyond the capacity of the cerebral cortex just as an organ of association. One faculty so enabled, is to retrieve episodic memories, of incidents in our lives, which happened only once, provided we can also reinstate the context in which we acquired those memories. In 1991 I published a theory of how the cerebral cortex and hippocampus interact so as to acquire the context which best serves particular sorts of information processing[[12]](#footnote-10) – acquired in a difficult and lengthy process - and then uses those contexts for memory retrieval, and other higher cognitive functions. The theory came mainly from research in animals; but recently, I see how it can apply to human psychology, to the notion of personhood, and to defining many forms of mental disorder.

(v) *‘Contexts for living’ in humans:* Infants’ awareness of themselves as enduring entities starts at the same age -2-3 years old - as they acquire both ‘continuity of memory’, and the beginnings of language. By then, they have discovered their ‘primary Ego’ from awareness of the continuity of their own body; but, from another point of view they have, on the same basis, also discovered the context which enables them to retrieve episodic memories. That ability, then, is the defining feature - more-or-less synonymous with - ‘being a person’: Continuity of memory, as a psychological fact is the *sine qua non* for personhood. From such reasoning I now have a concept which may be important in psychiatry: the notion of a ‘context for living’ (closely related to ‘sense of self’). This then, is the modern version of Wernicke’s idea, that each of us is engaged, until the day we die, in constructing and forever reconstructing our sense, of ‘being a person’. It is a concept similar to that of Victor Frankl, explained in his work ‘Man’s search for meaning’, based on experience in concentration camps in WWII[[13]](#footnote-11).

‘Meaning’, quite generally, depends on locating information in its appropriate context. In the dire situation of Frankl’s experiences, for a person to have a context within which his or her life still held meaning, could, quite literally, be a life saver. My concept of ‘context for living’ is essentially the same as Frankl’s, albeit developed initially from brain theory, rather than from experience of the savagery of concentration camp life. ‘Contexts for living’ or ‘sense of meaning to one’s life’ are formulated best, if at all, by putting them into words, which is no doubt why Frankl named his variety of psychotherapy ‘logotherapy’.

There is another facet to the neuroscientific basis of human personhood. Sages from time immemorial wrote in praise of ‘self-knowledge’, a concept important in most forms of psychotherapy; yet, logically, if we assume ‘the self’ to be a unitary indivisible entity, self-knowledge should not be possible. In resolving this, I am indebted to my former student, Kate Ball. In a section of her doctoral thesis[[14]](#footnote-12), she analyses ‘the self’ as having *two* parts continually interacting, corresponding to psychic functions of left and right hemispheres. The right hemisphere is adept at responding moment-by-moment to events of daily living as they occur (the ‘self experiencing now’); while the left hemisphere continually conceptualises these experiences, and the growing notion of ‘the self’. Continual interplay between the two, to create a subjective sense of a unified self, occurs via the corpus callosum. This makes ‘self-knowledge’ possible – actually the ‘conceptualising self’ continually updating its perspective of the ‘self-experiencing now’. It gives a convincing account of the otherwise-strange anomaly of selfhood (in severe schizophrenia): A person prefers to refer to themselves objectively – in the third person – rather than subjectively as the ‘I’ – of everyday speech. This arises by predominance of the left-hemisphere, conceptualising self, with atrophy of the ‘self experiencing now’.

(vi) *Personality theory.* Another key concept is the theory of normal personality. Ways to describe personality go back to antiquity. In the modern era, two pioneers defined *enduring* personality traits, I.P.Pavlov, and C.G.Jung. To designate a personality trait as permanent relies on evidence of life-long continuity of a trait, and its being determined at least in part by genetic factors. There is a large body of research on this. Some traits are determined mainly by environmental factors, others substantially by genetic ones. In so far as personality traits *are* determined partly by genetic factors, one can conclude that they arise from aspects of each individual’s brain. Since they *are* enduring, determinants are likely to be aspects of the brain which are relatively permanent, that is aspects of morphology – especially cellular morphology - not the ever-changing dynamics of neurotransmitters. Aspects of morphology on which I focus are: (a) the relative size of (and therefore number of neurones in) a variety of cortical areas, and other regions, such as the basal ganglia; (b) characteristics of axonal projections between cortical regions (especially the range of axon calibres and degree of myelination in populations of axons, which in turn determine their conduction velocity; (c) the relative number of different types of interneurones - short-axon neurones - which are likely to be important in determining the balance between excitation and inhibition in that region.

(vii) *Emphasis on Unusual Experiences (‘Symptoms’), not Diagnoses.* I believe this follows Wernicke’s own emphasis. He was scornful of many of the diagnostic entities flying around in his day, but sometimes he insisted ‘You have to get the diagnosis right’. Deeper than this, close reading of his *Grundriss,* leads me to conclude that he saw the unusual experiences reported by his patients as the primary realities; but he could incorporate these into his system only in so far as he could envisage how they arose from processes in the brain. Diagnoses, if they were to be used, were of secondary importance compared with those reports of first-hand experience. Here, he may have been guided by that seminal figure in the empiricist philosophy of science in the nineteenth century – Ernst Mach, who was important both in physical sciences and in sensory physiology and psychology. Mach’s aphorism says it all: ‘The world consists of sensation, for the scientist and for the common man’[[15]](#footnote-13). Wernicke, may have adapted that aphorism to understand mental disorders. In the foreword to the 1894 edition of *Grundriss,* Wernicke pays a handsome tribute to an un-named ‘advocate of theoretical natural science’. There is a strong case that this was actually Ernst Mach’.

 *The Framework for the System of Classification:* We can begin by excluding conditions attributed exclusively to *external* causes with a definite neuropathology: In Wernicke’s day these included *Delirium tremens*, GPI (‘General Paralysis of the Insane’, soon to be classed as tertiary syphilis), and the Wernicke/Korsakoff encephalopathy (later found to be due to thiamine deficiency). Today’s equivalents might be disorders due to adverse effects of street drugs, psychiatric sequelae of HIV/AIDS etc. *What remains,* to be incorporated in this prototype scheme of classification, can be called *‘Intrinsic Disorders of Personal Wholeness’*. These are based on what the human brain *is,* its normal variation between individuals, and how it reacts to social or psychological assaults. These are the core ‘mental disorders’ with which the system deals There are then four main subdivisions.

A: *Epochs when a person’s context for living has been undermined, and needs to be rebuilt.*

B: *Extremes of personality*

C: *Complications of either A or B*;

D: *Combinations of A and B.*

A fifth subdivision may need to be added: Although conditions with definite neuropathology (in a strict sense) and probable external physical causes are excluded, some disorders combine these features with ones intrinsic to each individual’s brain and resultant personality, or with life experience. The most obvious example here is dementia in the elderly, although (see below), this can develop with no discernible neuro-pathological signature.

In addition, there are aspects of ‘cognitive lifestyle’ which appear to be *protective factors*, preventing of delaying the onset of some disorders. Major examples of such factors here are the development of language, literacy, and the capacity for reasoning. As detailed in the notes below, symptoms of conversion disorders are more common in illiterate populations, and have declined in Western countries as illiteracy decreased. Likewise, evidence on dementia in the elderly, shows that, if it occurs, it is delayed for persons with life-long habits of vigorous mental activity (by virtue of their level of education[[16]](#footnote-14), or by their being bilingual versus those who are monolingual[[17]](#footnote-15)).

The dividing line between ‘mental disorder’ and normality is arbitrary. So, personality variants *could* be dealt with either as normal variants or as ‘disorders’. It is assumed that there is huge variation between individuals in their brains, part of normal human diversity. Moreover, the psychological and social insults to which humanity is exposed are also endlessly varied. It is the *combination* of characteristics of each individual’s brain – each with its unique strengths and weaknesses – and the particular insults and abuses to which we are all subjected, which produces what we refer to as ‘mental disorders’. Nowhere in this can one find definite ‘pathology’ using the term as used in general medicine. Moreover, we are *all* vulnerable, given our individual characteristics, and the ‘slings and arrows’ which life throws at us all. From this it follows *either* that we are all subject to ‘mental disorders’ (in so far as ‘order’ in our minds is not perfect); *or* that there is no category we can call ‘mental disorder. In either case, concepts of mental disorder no longer fit within wholly medical paradigms of ‘illness’ or ‘disease’, but fit better in a larger frame. This does not however mean that the task of classification is pointless.

--------------------------------------------------------------------------------------------[***ADDED NOTES:*** *The remaining text was not part of this lecture as delivered, but was referred to in discussion. Many proposals for neural basis of disorders are at present conjectures – hypotheses to be followed – rather than definite theories. However, the conjectural nature of these proposals does not affect the basis framework for the classification,*]

**The four classes of disorder mentioned above can be illustrated in more detail, as follows:-**

***A: EPOCHS WHEN A PERSON’S CONTEXT FOR LIVING HAS BEEN UNDERMINED, AND NEEDS TO BE REBUILT.***

There are many circumstances in which a ‘context for living’ - constructed slowly and painstakingly over a number of years so that we can conduct ourselves in a smooth, effortless manner in our environment – is undermined; then, it is ‘back to the drawing board’, and a person has to reconstruct their individual ‘context for living’ again.

These epochs can be subdivided into three subgroups:

***A1: Periods of life transition.*** Following Kahlbaum, there are *periods of* *life transition* which put people at heightened risk of mental disorders. Often these are times when the physical body changes, and a person loses a secure sense of the continuity of their own body. These periods include: *In either gender*:- puberty/adolescence; transition to old age; PLUS (for the sake of consistency), the first few years of life after a baby’s birth, before the sense of an enduring Ego has been developed; and *in females*, in relation to onset of menstruation, pregnancy, childbirth and weeks/months immediately after childbirth; and the menopause.

***A2:* *Life hazards which undermine pre-existing ‘contexts for living’*.** These include: Life-threatening illnesses; bereavement; accidents and natural disasters; major disappointments in love, exams, career, employment or finance; bad experiences with street drugs; religious conversion experiences; brutal legal proceedings; cultural dislocation and disruption of identity in a social group; trauma and abuse; wartime trauma; torture, etc.

*Psychological manifestations:-* In both A1 and A2 the tempo for reconstructing ‘contexts for living’ is roughly the same in most cases – a few years. The intervening period is one of emotional turmoil (most intensely when a person’s sense of bodily continuity is changed), heightened alertness, plus other signs of impaired function in tasks of everyday life, including (reversible) impairment in cognitive functions, including retrieval of details within episodic memory, even uncoordinated motor abilities, including walking and speaking. In addition, during periods when contexts are being reformulated, a person may struggle to make sense of the world, and adopts looser criteria than at other times for interpreting the world. In this situation ideas may emerge which appear to be false interpretations of the world, imposed on personal biases of various sorts – ideas of self-reference, grandiosity, persecution or hypochondria. Although this group of ideas is similar to those emerging in active psychotic states (see below), their fundamental origin is different; and it may be a mistake to treat people holding such ideas with antipsychotic medications.

In principle, the above abnormalities or impairments are all reversible.

***A3:* *Conflicted contexts.***Conflicts here may be of various types – between intrinsic motives, between any of these and social demands put upon someone by the situation in which they live and work, or by the fact they have found themselves having to play roles beyond their capacity. These conflicts have something in common with Freud’s concept of neurosis, and may give rise to contradictory behaviour, with lack of purpose, loss of moral sense, tantrums or other emotional outbursts, muted emotional responses to major events, sometimes manipulative behaviour, or ambivalent behaviour – oscillating between a desire to do something and to refrain from doing it. Some of this may be liked to obsessive-compulsive behaviour; but this is not a unitary condition (see below for another aspect of OCD with superficial similarity). There may be ever-more-energetic attempts to cope with the opposed demands, merging then into an overall passive attitude to an insoluble problem, and then developing into denial of an increasingly obvious problem.

***Resilience:***This is a countervailing protective personal attribute, usually *acquired* by long experience, and therefore not an enduring personality trait in the sense used here, and therefore without a strong genetic basis. It is based on having eventually developed a very versatile all-purpose contextual framework, scarcely shaken by the worst of life events. Those who, in the past, have had direct experience of severe life events, including psychic trauma, but also mental illness in a stricter sense, may have a long and difficult journey in rebuilding their sense of personal wholeness; but in the end they may achieve a sense of wholeness more complete than the average person, simply because they have had to assimilate into their image of themselves and the world they inhabit a wider range of human experience.

***B: EXTREMES OF PERSONALITY VARIABLES:***

The detail here is likely to develop into a long and complex treatise. The emphasis is on enduring traits, rooted in distinctive characteristics of each individual’s brain. Thus, further subdivision is based on such morphological variation.

It may be possible to divide personality extremes into ones whose morphological basis is found in the cerebral cortex, and those where it is sub-cortical (basal ganglia, diencephalon etc). Beyond this, some broad subgroups of disorder can be suggested.

***B1: The size of particular cortical and subcortical regions,*** and therefore the number of nerve cells therein.

*B1a: Size variations in neocortical regions.* This may account for personality variants, such as the preference for input in different modalities: Some persons focus on visual sense, others on auditory images. More serious problems are autism spectrum disorders and ADHD (which overlaps with autism), and possibly also dyslexia. Such disorders include unusual sensory preferences (not only weaknesses, but also sometimes strengths).

*B1b: Vulnerabilities due to reduced size of the hippocampus.* A number of psychological problems appear to be related to a relatively smaller volume of the hippocampus. The best defined is PTSD (see below). Since, according to theory, the hippocampus is critical for enabling representations of contexts (including ‘contexts for living’) to form, such disorders may arise because it is difficult either to form robust contexts in the first place, or to modify them, in the face of life’s challenges and traumata. This may lead to personality traits which cause difficulty (or conversely, to unique strengths, when the hippocampus is larger than normal), even without life stresses such as those applying to PTSD.

*B1c: Vulnerabilities due to differences in size of regions, or of unusual numbers or proportions of neurones, in the basal ganglia or other subcortical structures.*

*B1(c)(i):*  The basal ganglia contain are two independent sets of pathways whose respective role is to permit, or to refrain from or ‘veto’ strategies of active behaviour[[18]](#footnote-16). In Tourette’s disorder, and some forms of obsessive compulsive disorder (which are closely related to Tourette’s), the balance between the ability to initiate and to ‘veto’ behaviour is altered, so that ‘vetoing’ of motor or behavioural acts is impaired. A specific suggestion is that syndromes involving abnormal involuntary movements arise from a deficit, or a loss of specific groups neurones in the basal ganglia involved in vetoing. There is some direct morphological evidence that the number of neurones in the pathways for ‘vetoing’ is reduced in this condition[[19]](#footnote-17).

*B1(c)(ii): Proper hierarchy and balanced connectivity in subcortical brain regions dealing with emotions.* Our various emotional drives are normally arranged in a some sort of hierarchy, albeit a flexible one, which is probably rooted in the size and mutual connectivity between structures in parts of the basal ganglia and the basal forebrain. Numerous variants are possible, some of which are severe enough to constitute recognisable disorders. These might include *anorexia nervosa* (where, probably, the motive of *control* comes prior to others); motives of food and sex; *trait anxiety disorders,* where the motive of fear projected to the future is excessively active; and disorders of control of the emotion of *anger.*

***B2: Disorders due to unusual range of axonal conduction times[[20]](#footnote-18) in specific pathways***

*B2a:* *Disorders due to axonal conduction times in specific pathways being* ***longer*** *than normal.*

*B2a (i) -* *Perceptual* ***sensitivities.*** This section includes a wide variety of sensitivities (e.g. ‘noise sensitivity’), and probably many unexplained pain syndromes (usually dealt with today in specialties other than psychiatry). I believe these can be dealt with generically - in terms of the same underlying cause, but applying to different axonal pathways within the hemispheres. My hypothesis is that these disorders arise, wholly or partly, from conduction velocities in specific axonal pathways close to areas dealing with perception being rather slower than normal (and therefore conduction times being longer than normal for a given distance). Some traits so defined may overlap with ‘Somatisation disorders’ as currently defined.

*B2a (ii):* *Tendency to* ***excessive******association*** The same shift of axonal conduction times in other pathways will have other psychological effects, such that mental associations are made more readily than normal; and, by the same token, people become specially vulnerable to distracting effects of extraneous stimuli. They may be easily overwhelmed by ‘sensory overload’. Making mental distinctions then becomes more difficult.

(Symptoms mentioned in B2a may contribute to more severe disorders dealt with in section C, not least the constellation of non-psychotic traits underlying the diagnosis of ‘schizophrenia’. The theory relating the many perceptual and cognitive aspects of these traits to axonal conduction time is dealt with in another work[[21]](#footnote-19), and this theory is also relevant to the problems mentioned in B2b (i) and (ii).)

*B2b: Disorders due to axonal conduction times in specific pathways being* ***shorter*** *than normal.*

*B2b (i):* *Perceptual* ***insensitivities.*** Just as there is a class of psychological disorders accounted for in terms of reduced conduction velocity of axons in selected pathways, so there may be a converse situation where selected pathways contain axons which, on average, have faster conduction than normal. Conduction times, for a given distance, are then shorter than normal. Psychological manifestation would then be the opposite of those in B2a, namely relative *in*sensitivity in perception (for instance to loud noises). Such persons may be ‘sensation seekers’ because experiences and sensations of normal intensity are boring; but they operate best when other people would be overwhelmed.

*B2b(ii):* *Tendency towards* ***excessive******dissociation.*** In the cognitive domain such shifts in axonal conduction time (but in different pathways) would favour separating sources of information, and focusing on a single source while shutting out distractions. People would then make distinctions easily, might spot associations with difficulty, and can focus their attention well, even in challenging environments.

(The reasoning relating these perceptual and cognitive abnormalities to axonal conduction time is dealt with in another work[[22]](#footnote-20), with the proviso that, that since the abnormalities are the opposite of what is dealt with there, the shift in population properties of axons is also in the opposite direction.)

*B2c: Imbalance between the right and left hemispheres.* The theory relating population distribution of axonal conduction time to various psychological parameters (as traits in schizophrenia, and, by extension, to perceptual sensitivities and insensitivities) grew from an earlier theory accounting for psychological differences between right and left hemispheres (based on a similar assumption). This being so, it would be expected that some extremes of personality other than those already mentioned would arise from an imbalance between the two hemispheres. There are many possibilities here, which need not be spelt out; but such imbalances may contribute to many disorders as currently diagnosed.

***B3: Disorders due to unusual numbers (excesses or deficits) of short-axons neurones (interneurones) in specific brain regions.***

*B3a:* *Primary deficits and excesses of cortical activity (forerunners of definite mood disorders).* Wernicke saw depressive disorder (which he called ‘Affective melancholia’) not as a primary disorder of mood, but as a state where the normal level of cortical activity is severely reduced. Likewise, mania was a state of overall *excess* of cortical neural activity, and therefore of general hyperactivity of association across the cortex. Any change of mood in these conditions was secondary, a response to a person’s awareness of deficit (or enhancement) of their mental processing capacity. This proposal has much merit, since, at times, the deficits in information processing capacity may occur without the lowered mood typical of depression. If deficits and excesses in cortical information processing capacity are primary disturbances, this suggests that the normal balance between excitation and inhibition in the cortex is destabilised. Interneurones (short-axon, or ‘local circuit’ neurones) are almost all inhibitory, and therefore play a key role in determining the balance between excitation and inhibition. A tentative suggestion, yet to be explored in detail, is that depressive disorders arise in part due to unusual numbers of interneurones in cortical regions. Something along the same line, but in the opposite sense, may underlie risk of mania.

*B3b: Potential for neuroleptic refractory psychosis.*  Several syndromes, manifest at both cognitive or motor levels can be accounted for by lack (or loss) of cholinergic inter-neurones in the striatum. Some of these arise in patients who have been treated for extended period with antipsychotic medications (perhaps in excessive doses), and are mentioned below, as ‘complications’. However, sometimes similar syndromes occur without such adverse predisposing factors, and can therefore be included here as personality extremes. The theory linking the cellular deficit to the manifest symptoms is in either case the same[[23]](#footnote-21).

***C:*** ***COMBINATIONS OF A AND B.***

***C1: Combinations of personality extremes.*** Since the neural bases of different personality variations are numerous, and probably mainly independent of each, there are many possible permutations of the extremes discussed in B above. Two suggestions are given here:-

*C1a: Combinations of axonal conduction time distribution and problems due to small hippocampi.* Neurone networks of the normal *right* hemisphere are specialised for recognising visuospatial patterns; context representations of which they are a part, will enhance visuo-spatial pattern recognition (‘I never forget a face’), and related skills. Those of the normal *left* hemisphere and the context of whose representation they are a part, will be likely to serve rational schemes of inference, and related faculties. Similarly, episodic memory comes in two forms (which may be equally combined, or with one or the other taking precedence). In the first the memory detail is of right hemisphere type – such as visuo-spatial detail; while in the second the emphasis is on temporal sequence.

If there is also a shift in the balance between the two hemispheres (B2c), a shift favouring left-hemisphere type function means that any advantage which cortico-hippocampal interplay might give to visuo-spatial pattern recognition may be absent. The syndrome that then develops may include *prosopagnosia*.

More serious effect might arise. Sensations upon which perception - including visual perception - is based, are always subject to potential bias (since the proportion of sensory inputs to principal neurones of the sensory cortex is vastly outnumbered by inputs from other cortical regions). This means that major perceptual distortion may occur if the balance between hemispheres, or of the size of hippocampi on either side favours the left. In these situations, contexts constructed between left hemisphere and left hippocampus activity may impose on right hemisphere perception a vision which completely overwhelms direct sensory input – this being a form of visual hallucination.

*C1b: ‘Sensation seeking’ combined with cognitive rigidity.* In neuro-anatomical terms, this is the combination of perceptual insensitivity (hypothetically because axonal conduction times tend towards shorter-than-normal values: B2b[i]), with relatively small hippocampal size (B1b). This may lead to individuals who like noisy environments, like making noise, and who scarcely recognise that other people have quite different preferences.

*C1c: Disorders due the combination of aberrant emotional predilections: and cognitive inflexibility.* In morphological terms, this would be a combination of aberrant size or connectivity in relevant parts of the basal forebrain (B1c[ii]), and relatively small hippocampi (B1b). Again, such a combination may lead to great difficulty in many social situations,

***C2: Reactive depression.*** It is well recognised that periods of depression may be triggered by adverse life events, and that the psychological characteristics of such depression differ little, if at all, from depressive episodes without such triggering factors. However, stresses which lead to depression in some persons do not do so in many others. Thus such reactive depression appears to be the *combined* effect of adverse life events (‘life hazards’, listed in A2 above) *plus* underlying personality traits (based on proneness to episodes of underactivity of cortex: B3a above).

***C3: Reactive mania.*** This is shown in several studies[[24]](#footnote-22), and may be more common than usually understood. It may arise from the combined effect of proneness to episodes of overactivity of cortex (B above) *plus* any of the list of ‘life hazards’ under A2.

***C4: Conversion symptoms:***Conversion symptoms were first recognised in the late nineteenth century in the work of Jean Martin Charcot and Sigmund Freud, as quasi-neurological symptoms, but unlike syndromes due to lesions of spinal cord, brainstem, or brain. Examples are loss of somatic sensation that is not dermatomal, or loss of motor function without expected change of tendon reflexes. For Freud such syndromes were thought to be part of what was called hysteria, supposedly found only in women. He believed that conversion syndromes were brought about by unresolved emotional conflict (and therefore ‘conversion’ of anxiety into physical symptoms). Physicians dealing with psychic casualties of the First World War saw such syndromes in far larger numbers than did Freud, and in men; and the best descriptions come in the post-War period. Today, such syndromes are still defined by psychiatrists, but are usually seen in other clinics, especially in neurology[[25]](#footnote-23). It is a difficult area, because of the many specialties which deal with conversion disorders, confusion between somatisation, dissociation and malingering, the multiple meanings of the term hysteria, and the large demands on clinicians when they identify the disorder. Psychic trauma is involved, yet trait factors also play a part. Conversion symptoms are more likely in illiterate or uneducated persons (and are studied today in countries with large illiterate population)[[26]](#footnote-24); and in persons who score highly on alexithymia (who find it difficult to identify or describe feelings)[[27]](#footnote-25). The latter are likely to be common amongst unlettered persons, unused to verbal expression of complex experiences. Conversion symptoms may also be common in children and adolescents[[28]](#footnote-26), although rarely diagnosed as such, or subject to epidemiological study.

An hypothesis, not yet examined in depth, is that these syndromes are related to the ‘freezing’ or the ‘death feint’ response, seen in all mammalian species under stress[[29]](#footnote-27). This response appears to originate in the basal ganglia (specifically the striatum), as a short-lived shut-down of all neural activity. In human brains, structures are larger than in other mammals, and there may be *localised* shut-down limited to parts of the striatum. The striatum has prolific, topographically-organised pathways to the whole of the cerebral cortex. Therefore any shut-down in the parts of the striatum is likely to have corresponding effects on cortical function; but the pattern of impairment is then likely to be different from that typically seen after lesions in neurology - with loss of function area-by-area in the cortex, rather than according to patterns well known in classical neurology. Protection from this in literate or educated persons may arise because, when a person is fluent with language, and rational analysis, he or she has another means to represent experience within the cortex, which helps maintain unity of cortical function. This can prevent conversion symptoms, despite major disruptive impacts.

***C5: Post-Traumatic Stress Disorder.***In DSM-III, this is the only entity for which a clear external cause is identified, namely severe psychic trauma (for instance for military personnel who have seen active service in combat situations). However, while this may be necessary, it is not sufficient, since most persons exposed to such stress do not develop PTSD. A fuller account includes occurrence of such trauma combined with some trait vulnerability (see B1b, above). There is much evidence that those whodevelop PTSD, have smaller hippocampi than normal. This might mean that reconstructing new ‘contexts for living’, in the aftermath of severe trauma, is more difficult than normal, resulting in a state of labile emotionality and impaired function, which persists longer than usual afterwards, and perhaps even to become a permanent condition.

***C6: Dissociative disorders.*** As just suggested (B1b), there is a personality type which makes distinctions, and shuts out distractions easily, yet is slow to spot associations. There are also life circumstances, especially traumatic experiences, where this trait leads to dissociative symptoms - difficult aspects of experience being excluded from full awareness. The most serious such disorder is what, in DSM, is called ‘Dissociative Identity Disorder’ (formerly ‘Multiple Personality’). It appears to occur in people whose earliest years are dominated by abuse from primary caregivers, a profoundly contradictory set of experiences. Many would question the validity of the diagnosis, perhaps because it threatens their philosophical preconceptions. However, the diagnosis need not pose such a threat: A person inclined to make distinctions easily, and whose life experience when their first sense of personhood is forming is beset with profound contradictions, may develop opposing views of - and relationship to - their primary caregiver; and their own sense of self becomes not the usual approximation to a unified entity, but a disorder in which personhood is split into different ‘personas’, to be deployed in different circumstances.

***C7:* *Episodes of Active Psychosis, mediated by dopamine:*** This word has come to encompass an excessively wide range of disorders. Here the word is used to denote an episode of mental turmoil in which delusions occur, and any tendency to experience auditory verbal hallucinations becomes manifest for the first time, or, if already present, is enhanced. The delusions in this case are similar in content to those sometimes seen when life contexts are being reformulated (see A1 and A2, above), but the basic mechanism is quite different: In this state the process of formation of delusions (if not the existence of those already laid down), and often the hallucinations, can be brought to an end with the usual range of dopamine-blocking antipsychotic drugs. While the deeper causes are debated, it is likely that such active psychotic states occur mainly in persons with underlying personality traits (B2a), the tendency to psychosis then being revealed in *any* of the stages of life, or following the life hazards dealt with under [A]. The theory behind the relationship between the traits and the state of active psychosis was discussed in another work[[30]](#footnote-28). In principle however, psychotic breakdowns can occur in persons with relevant traits at *any* stage of life transition (giving rise, for instance, to *post-partum* psychosis, psychosis after the menopause, and in the elderly), or after a variety of serious life hazards. However, psychotic states arising in late adolescence or early adulthood are probably usually the most critical ones, since at this stage the adult ‘sense of self’ is in a critical process of formation and most vulnerable to challenge, with the consequence that recovery may be long and difficult.

***D: COMPLICATIONS OF A, OR B, OR OF A-PLUS-B:*** There are likely to be many disorders falling in this category, mostly related to severe disorders in current classification. At present, I merely offer a few examples.

***D1: Dementia in the elderly.*** Dementia, especially of the Alzheimer type, has been seen as paradigmatic of disorders characterised by a combination of mental symptoms (in this case, of cognitive decline) *and* definite neuropathology. However, recent evidence shows that this is far from the whole story. In a community-based study[[31]](#footnote-29) of elderly persons, characterised clinically and with post-mortem evidence on neuropathology, 48% of individuals met clinical criteria for dementia. 22% met neuropathological criteria for definite Alzheimer’s disease, and a further 26% for ‘probable Alzheimer’s disease’, leaving 52% with no (or minimal) such evidence. Amongst those with dementia, 36% showed evidence of definite Alzheimer’s disease, and a further 28% of ‘probable’, leaving 36% with no or minimal Alzheimer’s pathology. Conversely, 33% of individuals without dementia had moderate or severe Alzheimer’s pathology. Taking a wider range of pathological evidence (vascular lesions, Lewy bodies etc) improved the prediction of clinical status; yet in the final model there was still 23.7% of false positive cases (neuro-pathologicaal signs without dementia) and 15% of false negative cases (dementia, without neuro-pathological signs). Clearly there are predisposing or protective factors, beyond the supposed neuro-pathological features characteristic of dementia. These are likely to lie in the domain of factors considered in the present classification, including both risks after stages of life transition, and after life’s hazards, and protective factors such as a cognitive lifestyle (life-long maintenance of an active mind). In this sense, dementia in the elderly can be seen to arise in part as a complication of the stage of life transition in elderly persons (A1), often - but by no means invariably - compounded by neuro-pathological changes.

 ***D2: Misinterpretations of Unusual sensations.*** Some disorders may originate with unusual sensory sensitivity (B2a[i]), which for persons with minimal knowledge of their own biology may be misinterpreted, leading to some varieties of mono-symptomatic delusion (such as delusions of infestation). This identification overlaps with what is currently called ‘delusional disorder’.

***D3: Depression with lowered mood.*** As described in B3a, the primary deficit in what is now called depressive disorder may be a state of deficient neural activity across the cortex, with impairment of information processing, especially in the capacity to take decisions. Lowering of mood, states of despair, matching delusional misinterpretations, suicidal tendencies and other features of depression are then seen as secondary effects, as a person comes to realise the degree of their own impairment. If this analysis is correct, depression *as a disorder of lowered mood* is a complication of the primary disorder. The relation between this proposal and the vegetative abnormalities seen in depressive disorders remains to be examined.

***D4: ‘Confused mania’*** (after Wernicke). Wernicke describes how, in manic states, excessive activity across the cortex (as he inferred) is at first not only perceived subjectively as ‘above normal’ performance, but in some respects may be above normal in an objective way. However, as this state intensifies, fluidity of associations develops to such a degree that it leads to definite impairment, that is an ever-worsening state of mental confusion. Today such states can usually be controlled with medications.

***D5: Mania with features of active psychosis.*** The excesses of activity in the cerebral cortex hypothetically underlying manic states are likely to have effects beyond the cortex. In particular since the midbrain dopamine neurones are under control from cortical regions, excess activity in the cortex is likely eventually to destabilise the dopamine neurones leading to a dopamine-dependent psychosis. The origin of this state would then be different from the endogenous psychosis described above (C8). However, in clinical situations where little evidence is available about how a state characterised by psychotic delusions and hallucination arose, the differential diagnosis may be difficult.

***D6: Psychotic states refractory to usual antipsychotic medication, yet responsive to clozapine.***This hard-to-manage condition is distinct from most psychotic disorders. It may arise *de novo*, or grows gradually in persons with a tendency to psychotic breakdowns, following long periods of receiving antipsychotic drugs in large doses. The condition is related to one considered above (B3b: where there is no such risk factor); and theory for this is based on the absence, or progressive loss of a class of cholinergic interneurones in the striatum (part of the basal ganglia)[[32]](#footnote-30).

***D7: Progression to chronic psychotic state (Wernicke’s ‘paranoid states’).*** Wernicke separated chronic from acute mental disorders on the grounds that the delusional misinterpretations of the acute phases of disorder become so entrenched that they are essentially fixed. Today, this progression may be a result of prolonged inadequacy of drug treatment of states of active psychosis. There may however be personality factors involved, which can be recognised independent of any mental disorder - the relative rigidity or tenacity with which beliefs, once acquired, are held (in the face of mounting contradictory evidence). This trait may in turn have its neurobiological basis (for instance in relatively small hippocampi, so that versatile reformulation of contexts is difficult).

***To Conclude:- What became of ‘schizophrenia’?*** In my book in 2008, the ‘age of onset’ criterion hardly appeared at all, and never in a critical way. In the scheme for classification just outlined, non-psychotic traits are contained in sections B1a: In section C1, it is implied that these may be precursors to episodes of psychosis *at any age*, and in a variety of other challenging life situations. In addition, as I now believe, most of the enduring traits are by no means limited to ‘schizophrenia’: They occur at elevated frequencies in other conditions (as currently diagnosed), and, at lower frequency, in persons with no diagnosis; and in such cases, these traits never lead to episodes of psychosis. Thus, as hinted earlier, the concept of ‘schizophrenia’ is carved up in what seems to me to be a more logical fashion, based on better scientific reasoning, and perhaps permitting a more rational approach to treatment and care of its several entities.

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