Schizophrenia: A Conceptual History

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ABSTRACT

The current concept of schizophrenia is regarded as the consequence of a linear progress from different definitions concluding in the present. According to the "continuity hypothesis" schizophrenia has always existed and 19th and 20th centuries alienists have polished away its blemishes and impurities, culminating in the DSM-IV definition which can therefore be considered as a paragon of a real, recognizable, unitary and stable object of inquiry. However, historical research shows that there is little conceptual continuity between Morel, Kraepelin, Bleuler and Schneider. Two consequences follow from this finding. One is that the idea of a linear progression culminating in the present is a myth. The other that the current view of schizophrenia is not the result of one definition and one object of inquiry successively studied by various psychiatric groups bat a patchwork made out of clinical features plucked from different definitions. The history of schizophrenia can be best described as the history of a set of research programmes running in parallel rather than serialism and each based on a different concept of disease, of mental symptom and of mind. In this paper some of these programmes are discussed.

Key words: schizophrenia, psychosis, dementia praecox, conceptual history.

RESUMEN

Esquizofrenia: una historia conceptual. El concepto actual de esquizofrenia ha sido considerado como resultado de la progresión lineal de una serie de definiciones que han concluido en la definición vigente en los manuales diagnósticos. Según la "hipótesis de la continuidad", la esquizofrenia ha existido siempre como un objeto natural "real", y los alienistas de los siglos XIX y XX han ido puliendo sus aristas e impurezas hasta culminar en la definición del DSM-IV, que se considera como el paradigma de de un objeto de estudio real, reconocible, unitario y estable. Sin embargo la investigación histórica muestra que la continuidad existente entre los planteamientos de Morel, Kraepelin, Bleuler y Schneider es escasa y que, por tanto, los sucesivos abordajes son inconexos y contrapuestos. Esto tiene dos consecuencias: en primer lugar, la idea de la progresión lineal que culmina en la actual definición es un mito. En segundo lugar, el concepto actual de esquizofrenia no procede de un único objeto de investigación sino de un mosaico compuesto de fenómenos clínicos provenientes de distintos enfoques y definiciones. La mejor forma de estudiar la historia de la esquizofrenia sería describiendo la historia de una serie de programas en paralelo, cada uno basado en diferentes conceptos de la enfermedad, el síntoma mental y la mente. En este trabajo se abordan algunos de estos programas.

Palabras clave: esquizofrenia, demencia precoz, psicosis, historia conceptual.

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'When the story of dementia precox is written
it will be the history of psychiatry'.
Nolan Lewis (1936)

Because 'schizophrenia' is regarded as the psychiatric 'illness' *par excellence*, it may be reasonable to assume that it is the paragon of a ***r**eal*, ***r**ecognizable*, ***u**nitary* and ***s**table* object of inquiry ('RRUS') and that on account of this enduring quality its history is straightforward, in the sense that its 'first description' can be considered as tantamount to 'finding' it. In other words, it does not *add* anything substantial to the object itself which for years was simply 'hidden' from view. However, since its construction, various definitions of 'schizophrenia' have been proposed, and it is not yet clear which is to be preferred (Meduna & McCulloch, 1945; Sarbin, 1972; Rieder, 1974; Boyle, 1990; Brockington & Nalpas, 1993). On the assumptions of *recency* and *high usage*, some might wish to adopt the definition promoted by DSM IV, but writing on the 'history' of DSM IV schizophrenia can only tell about the clinical views of a Task Force of the American Psychiatric Association and about its political and social context. It cannot tell anything about how certain words, concepts and behaviours interacted in late 19th century Germany and France to generate the notion of 'dementia praecox'.Before this is accepted, however, a rule of 'evidence-based medicine' that pivotal claims be empirically supported must be applied. In this regard, it would seem that no crucial experiment has ever been carried out to demonstrate that 'latest means truest' or that 'high usage' constitutes adequate evidence for validity. Indeed, the only support for the 'recency' assumption is the view that in this paper will be called the 'continuity hypothesis'. By the same token, peer and medico-legal pressures are a better explanation of 'high usage' than 'truth-value'. In fact, there is no 'objective' or 'empirical' way to decide which of the various definitions (referents) of 'schizophrenia' should be considered as *the* definitive RRUS.

Historiographical Issues

Why the difficulty? One explanation is that the naming function of 'schizophrenia' is defective and generates an unacceptable number of borderline cases; another that the referent of 'schizophrenia' is intrinsically 'vague'. A concept is considered vague for no amount of additional criteria will improve upon its definition or sharpen its boundaries; in other words, there will always be 'uncertain' cases which cannot be definitely considered as falling within the ssaid category (Williamson, 1994). Yet a third explanation is that its object-referent is only a *construct*, i.e., a concept whose boundaries depend more upon interpretation of the historical and social context that upon intrinsic variants controlling the stability of the object of inquiry. Each of these explanations will generate a different historical account; the one to be followed in this chapter makes use of a

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version of the 'vagueness' view (second above), modified to take into account the needs of psychiatry.

The first description of most mental symptoms and diseases can be traced back to specific historical conjuncture or 'convergence'. By the latter it is meant the coming together in the work of (usually) an identified clinical writer of a *term* which may be newly coined or recycled, a *behaviour*, which may be the expression of a brain disturbance, and a *concept* which is the carrier of definitions and explanations, and also rules for the use of the 'convergence'. The 'disease' now called 'schizophrenia' resulted from a convergence reported by an alienist called Eugene Bleuler about 90 years ago which included a neologism (of his own making) (Allen, 1990), a behavioural referent (partially borrowed from an earlier convergence), and a concept (which linked the 'disease' to psychological and psychodynamic theories prevalent during the early 20th century).

In spite of episodic challenges —that started with Kraepelin himself who late in life recanted on his dichotomy— (Hoche, 1912; Kraepelin, 1920; Bumke, 1932; Colodrón, 1999), 'schizophrenia' has survived well. Why so? In general, little is known about why some 'diseases' survive longer than others. To some, the obvious explanation might be 'degree of truth', i.e. the more a description pictures a 'real' object in nature the more enduring it will be; unfortunately, things are not as simply as this and social and financial factors may play a crucial role: for example, in the case of 'schizophrenia', social factors such as academic reputations, power groups, and relevance to other disciplines (e.g. genetics) (Gaupp, 1939) and financial factors such as the investment of the pharmaceutical (and more recently the neuroimaging) industry, have undoubtedly contributed to its survival.

Strictly defined, therefore, the history of 'schizophrenia' should start with the convergence reported by Bleuler. Because the absence of serious research in this field, whether there is a direct link between the latter and earlier convergences (e.g. those reported by others as dementia praecox, paranoia, vesania, melancholia, mania or insanity) remains a historical hypothesis (Ewald, 1930). In this regard, and not to clutter the field, two hypotheses will be touched upon in this paper: according to the continuity hypothesis there is a direct line of progress between the old notion of insanity and DSM IV; according to the discontinuity hypothesis, such a progressive line is mythological, i.e. although associated in some way, the various convergences to be studied below are conceptually independent. In other words, there is little historical evidence that the continuity hypothesis is supported by the historical facts: its survival, therefore, must be explained in other ways.

In order to test these two hypotheses, the notion of 'schizophrenia' will have to be taken to mean a shorthand for a collection of (some defunct) convergences whose relationship *ab initio* is uncertain.

The 'continuity hypothesis'

This hypothesis states the unsubstantiated belief that all definitions of schizophrenia can be arranged in a progressive and continuous series; that this linearity reflects an incremental truth gain, and that hence, 'latest means truest'. The continuity myth is a

powerful way of converting a construct (i.e. schizophrenia) into a RRUS. Its power and plausibility results from the fact that during each historical period fashionable 'standards of evidence' and 'truth' are 'meshed' into the ongoing definition of schizophrenia (or homologous clinical category). Thus, in the same way as current DSM IV definition is closely linked to post Fisherian standards of statistical significance, genetics and neuroimaging, Kraepelin's definition of 'dementia praecox' was closely linked to descriptivism, microscopy and neuropathology. It goes without saying that in his time Kraepelin used this linkage to impose his own definition in the same way as the DSM IV definition is imposed nowadays. The fallacious reasoning that leads to the continuity myth issues from this linkage: 'evidence' for the validity of any earlier definitions always falls short of the standards of the present and hence historians oblige by constructing a progressive line of definitions putatively to get ever closer to the final truth.

Things are even more complicated. Due to the fact that social decisions as to what is true or valid are current taken on the basis of 'science', the latest definition of schizophrenia also becomes the 'official' one, and this has consequences in regards to clinical and academic freedom. In a clinical world controlled by standards of 'good practice' (which originate also in the continuity myth), it is a risky business the challenge the official (i.e. latest) definition in terms of funds, publications and medico-legal implications. In consequence, the latest definition gets the lion's share of everything and earlier definitions never get a chance.

According to this view: (a) 'schizophrenia' has *always* existed (say, as a 'rough diamond'), (b) 19th and 20th centuries alienists (Kraepelin, the Bleulers, the Schneiders, etc.) have polished away its blemishes and impurities, culminating in: (c) the DSM IV definition which can therefore be considered as a paragon (RRUS), and (d) The end of history is nigh for it is only matter of months before the genetics and aetiology of schizophrenia is sorted out for good.

Duly fleshed out, the story is seductive. For centuries, it goes, the terms insanity and madness were used to refer to a melee of diseases which physicians were unable to tease out. Early in the 19th century, and by dint of observation, John Haslam described a typical ICD-10 case of schizophrenia but no one paid attention (Haslam, 1810). In the 1850s, Benedict Morel coined the term *démence précoce* to refer to states of cognitive deficit seen in adolescence or soon after, but his proposals also sunk without trace (Morel, 1860). During the second half of the 19th century things begin to improve: 'catatonia' was described by Kahlbaum (1874) and 'hebephrenia' by Hecker (1871). By the very end of the century, Emil Kraepelin truly 'discovered' the disease by bringing together the two earlier (partial) descriptions and adding one of his own (i.e. dementia paranoides); he decided to call it 'dementia praecox' (a latinized version of Morel's term). The continuity myth is keen to emphasizes that Kraepelin was only able to make his 'discovery' because he based it on pure empirical observation. First, in special cards he painstakingly recorded thousands of case histories, and followed his patients up; then, he analyzed these data reaching the ineluctable conclusion that dementia praecox and manic depressive insanity were separable on symptoms, course and outcome. However, there is evidence that the Kraepelin's cards contained insufficient information to draw any statistical conclusions (Weber & Engstrom, 1997). Further, the two forms of insanity

were separable in terms of their 'course' and 'outcome': dementia praecox was chronic, manic depressive insanity episodic. There was little difference in terms of their cross-sectional symptoms.

The fact that the 'continuity myth' says nothing about the major *rival* taxonomies and descriptions of madness (or the psychoses) going on at the time (for example, Wernicke's) needs explanation. One is that such information is considered as irrelevant to the 'history of schizophrenia'. To the historian, however, this does not sound right: what evidence is there that survival of a view is guarantee of its truth? Could there have been other reason why Kraepelin's view lived on and Wernicke's did not? Should it not be central concern to a proper history of schizophrenia to deal with this issue? To say that a history of schizophrenia should be about what the work names as such is no answer for that will make it into a history of a word, which from the clinical point of view is the less important of issues: the central concern should be the history of the behaviours in question, whatever the names they have travelled under.

From then on, the story continues, all that Kraepelin's RRUS required was re-adjustments. Influenced by Gustav Jung, Eugen Bleuler rebaptised it as 'schizophrenia', regrouped its symptoms, and offered explanations that differed a trifle from Kraepelin's. Then, under the impact of psychoanalysis the newly named RRUS went through some turbulence and its boundaries became indistinct. Worried by this, and by the fact that even some of Bleuler's 'symptoms' were difficult to identify, Kurt Schneider took the practical and 'atheoretical' step of creating an enduring set of 'empirical' diagnostic criteria.

The 'continuity hypothesis' can be embellished further. It is said that whilst European psychiatry remained influenced by Kraepelin, American psychiatry followed Adolph Meyer and Bleuler and that this, together with the impact of psychoanalysis and Adolph Mayer led to the blur definitions of schizophrenia offered by DSM I and II (Raskin, 1975; Fullinwider, 1982). But this could not last: definitional disparities between the USA and Europe are picked up by empirical research (e.g. the UK-USA study; the WHO study, etc), Kraepelin and Schneider are discovered, and the obscurantist period gives way to the new gospel of biological psychiatry. DSM III is the first expression of the new science and DSM IV offers a definition of schizophrenia which, although not *de jure* (the empiricist creed forbids that!), seems *de facto* to be the new RRUS.

This narrative has now the status of a myth and is almost beyond the reach of empirical correction or falsification. It is not difficult to understand why. Firstly, it makes use of a powerful rhetorical device: a linear progress from chaos to order, from subjectivity to objectivity, from confusion to real science. Secondly, it justifies the view that the current definition of schizophrenia is superior to anything that went on before and legitimates the fact that it is socially and financially favoured. Thirdly, it protects researchers from worrying about the *validity* issue, i.e. whether or not DSM IV clinical descriptions are relevant to patients.

Clinical psychiatric history illuminates the present by interrogating the past but it must also explain why recent important research into the history of schizophrenia has been disregarded (Guiraud, 1968; Lanteri-Laura & Gros, 1982; Janzarik, 1986); doing so might, however, cause it to enter into conflict with the continuity hypothesis. However,

this challenge is not about denying the existence of the behaviours captured by a series of defunct convergences or in the present one (namely, schizophrenia). Nor it is about playing down the contribution of Haslam, Morel, Kahlbaum, Hecker, Diem, Stransky, Kraepelin, Bleuler or Schneider and many others. It is about understanding the convergences (whether stable or not) that throughout history have been dedicated to the capture of the purported disease.

The discontinuity hypothesis

Powerful intellectual currents shaped the construction of the 'referent' of schizophrenia: 'associationistic psychology', 'neo-Kantianism', and 'evolutionary theory'. According to associationism, psychological events are bundles of primary 'atomic components' or unit of analysis: for example, perception and thinking result from the association of simple ideas or thoughts, respectively. For example, Bleuler resorted to 'disordered associations' to explain some the 'primary symptoms' of schizophrenia' (Bleuler, 1911) and Jaspers (1963) to account for speech disorders in the psychoses. Neo-Kantianism named a German intellectual movement which, between the 1850s and the first World War, promoted a return to the philosophy of Kant. Kraepelin, Bleuler and Jaspers, were influenced by the neo-Kantian definition of 'cognition' as reflected in their definition of formal thought disorder (which in the event became one of the central features of 'schizophrenia'). Lastly, evolutionary theory, an expression of the historicist beliefs that influenced Europe during the 19th century, encouraged alienists to conceive of some of the symptoms of dementia praecox as reflecting a regression to atavistic behaviours. For example, according to Kraepelin's view of disease (of which dementia praecox was the best example), the disease process activated a set of 'pre-formed reactions' (responsible for the clinical picture) which were biological and evolutionary in origin (Hoff, 1994). Bleuler suggested that the *Schnauzkrampf* (according to Kahlbaum, protrusion of lips resembling a snout seen in patients with catatonia) seen in schizophrenia might be related to protrusion of lips in chimpanzees (to 'express dissatisfaction') (Bleuler, 1911).

History of Words

In psychiatry, terms are just handles for 'convergences'. Whether coined anew (schizophrenia) or recycled (mania, melancholia) they are rarely informative. Their study (historical etymology) remains important, however, for like semantic ghosts, original meanings may linger on and influence later usage.

'Dementia Praecox'

The term dementia has participated in at least three earlier convergences. Up to the 17th century, it was used to referred to states of psychosocial incompetence, whether inborn or acquired; age and irreversibility were not part of its semantics. By the 18th century, the term dementia is set into a new convergence. To the old general meaning

(which remains as a legal term) a clinical one is added to refer to states of acquired intellectual deficit at whatever age and of whatever aetiology. By the end of the 19th century, a third convergence, the one that we have today, is negotiated. Known as the 'cognitive paradigm' this third meaning of dementia defines cognition as 'memory' and sets criteria for age, aetiology and evolution. Thus, children with cognitive deficits or adults who acquire them after head trauma are no longer called 'demented'; nor are those who develop them overnight or improve after few days: i.e. dementia is an irreversible state that affects cognition (particularly memory) in the elderly (Berrios, 1987a). By the turn of century, senile and other forms dementia were gradually described (Berrios, 1990; 1991).

Without knowing about these changes in meaning it would be impossible to understand what did Morel mean by the term *démence précoce*. Firstly, some historical facts need to be established. It is not true, as Alexander & Selesnick (1966) have claimed, that the term was coined by Benedict Morel in 1850. In fact, the term does not appear in his *Études Cliniques* but only ten years later in his *Traité*, and then not as an official clinical category but as a mere description: 'surdi-mutité, faiblesse congénitale des facultés, démence précoce' (p. 516); and 'Une espèce de torpeur voisine de l'hébétude remplaça l'activité première, et lorsque je le revis, je jugeai que la transition fatale à l'état de démence précoce était en voie de s'opérer' (p. 566) (Morel, 1860). Morel's views were still controlled by the second meaning of dementia (see above) and all he wants to do is set apart a group of patients with *stupidité* (stupor), at the time considered as a disorder of motility and the will and related to melancholia (Berrios, 1981a). *Démence précoce* is not a 'precursor' of schizophrenia (Minkowski, 1925,1927; Gruhle, 1932). As Minkowski stated: 'An abyss separates Morel's démence précoce from that of Kraepelin where the streamlet has become a river which, having forgotten its humble origins, threatened to flood everything' (Minkowski, 1925, p. 195).

Because Morel did not propose 'démence précoce' as an official clinical category but as a mere description, it had already sunk into oblivion by the time Kraepelin had decided to use the term 'dementia praecox'. There were other reasons too: dementia had changed meaning (Gross, 1904) and the clinical and classificatory problems of stupor had been more or less sorted out. Indeed, there is no evidence that around the time he first used it, Kraepelin knew of the existence of Morel's descriptive term (Hoff, 1994). The term first appeared in the 5th edition of Kraepelin's textbook (1896) where, under *Verblödungsprocesse*, Kraepelin lists three independent conditions: 'Dementia praecox' (mild and severe forms, and hebephrenia), 'catatonia', and 'dementia paranoides'. No where in this text is Morel's name mentioned; it only appeared three editions later. By the time Kraepelin was writing this edition, the term dementia had already changed meaning hence he saw the need to qualify dementia by the term 'praecox', by which he meant 'early', 'not at the expected age', etc. The very fact that he went on to acknowledge the French man in the last edition of his book (Kraepelin, 1919) suggests that someone in the interim might have drawn his attention to the fact that a French version of the term already existed. Kraepelin might have taken the term from Arnold Pick who used it in its Latinized version as early as 1891 (Pick, 1891). There is little point, therefore, in trying to establish a continuity between Morel and Kraepelin as

some author has done (Wender, 1963). Surprisingly efforts to do so are not the result of bad 20th century historical scholarship but of French chauvinism. As Lanteri-Laura & Gros have noticed, after loosing the Franco-Prussian War and having to accept the terms of the Treaty of Frankfurt, the French were keen to keep the little they had left, and *démence précoce* 'belonged to their national patrimony' (Lanteri-Laura & Gross, 1992); according to these authors, this explains the haste with they claimed that Kraepelin's disease was in fact the same as that first described by Morel (Garrabé, 1992).

'Schizophrenia'

Bleuler

The term schizophrenia seems to have been coined around 1907 and remains a misleading neologism (Bleuler, 1908). The reasons put forward by Bleuler to justify its coining are also unconvincing: 'I call dementia praecox schizophrenia because, as I hope to show, the splitting of the different psychic functions is one of its most important features.' ("*Ich nenne die Dementia praecox Schizophrenie, weil, wie ich zu zeigen hoffe, die Spaltung der verschiedensten psychischen Funktionen eine ihren wichtigsten Eigenschaften ist.*") (Bleuler, 1911, p. 5). This seems straightforward but it is not. The meaning he gave to 'Spaltung' and 'psychischen Funktionen' is ambiguous and needs clarification. Bleuler acknowledged that the idea of a 'splitting of psychic functions' was in the air, and that in schizophrenia such 'splitting' occurred vertically (i.e., it separated mental functions from each other). This entails a 'functional' model of the mind and hence (in spite of Bleuler's reassuring claims that a clear continuity was established between his schizophrenia and Kraepelin's dementia praecox and indeed earlier conceptions including Morel's) there is a marked difference between Kraepelin's dementia praecox and schizophrenia (Bleuler, 1911). In other words, the change was not just one of the name but, as it is suggested in this paper, Bleuler offered a new convergence.

Jung

This is the way that Jung seems to have understood it: 'according to his description [Bleuler's], some kind of disintegration is involved, inasmuch as the associations seem to be peculiarly mutilated and disjointed. He [Bleuler] refuses to accept Wernicke's concept of 'sejunction' because of its anatomical implications. He prefers the term 'schizophrenia' obviously understanding by this a functional disturbance' (Jung, 1972, p. 234). Such view of schizophrenia created a problem of differential diagnosis of hysteria and other states putatively due to dissociative mechanisms (Guraud, 1985). Jung was inclined to thing that 'the dissociation in schizophrenia is not only far more serious, but often it is irreversible. The dissociation is no longer fluid and changeable as it is in the neuroses, it is more like a mirror broken into splinters. The unity of the personality which, in the case of hysteria, lends a humanly understandable character to its own secondary personalities is definitely shattered into fragments' (Jung, 1972, p.

235). And regarding the change of name itself: 'from the psychological point of view, the change of name is unimportant, for it is of less value to know what a thing is called than to know what it is' (Jung, 1972, p. 155).

The 'Splitting' Metaphor

In general, what was the role of the mechanism of 'splitting' (Spaltung) in German psychiatry at the time? Originating in early 19th century Romantic psychology and in the work of Herbart (Scharfetter, 1975), the metaphor of separating, dividing, breaking, dissociating, divorcing or splitting of mental functions became popular to explain unpredictable and strange human behaviour. The metaphor was used in popular literature and folk and scientific psychology. For example it is already present, in the shape of an anatomical model in the two-brains model of Wigan. By the second half of the 19th century it pervaded literature (e.g., Dr Jekyll & Mr. Hyde), philosophy (Hartmann's model of the unconscious), neurophilosophy (Jackson hierarchical model of the brain), psychology (psychological automatism, Azam's dissociation; multiple personalities, etc.) and neurology (e.g. Charcot's notion of hysteria); at the very end of the century went on to inspire both Freud (concept of splitting of the Ego) and Wernicke ('sejunction').

Thus, it is little wonder that Bleuler's usage made sense to many. In its long history, the splitting of 'splitting' was made to occur horizontally and vertically, and both between and within structures and/or functions. A number of alternatives to dementia praecox, based on a version of the splitting mechanism, were available at the time: dementia dessecans, dementia sejuctiva, intrapsychic ataxia, discordance, etc. Its explanatory power was, therefore, almost infinite.

History of Behaviours

On the assumptions that a brain disease underlying the symptomatology of schizophrenia (RRUS) has existed since before it was 'first described', some historians have asked whether descriptions of it be found in earlier documents —even if such descriptions call it something else and do not conceptualised it as a disease?

'Did schizophrenia exist before the eighteenth century?'

This question was asked during the 1980s and was left unresolved. Based on a pseudo-historical epidemiological claim, that the historical record shows no authentic cases before the 19th century (Gruhle, 1932, pp. 1-20), supporters of the 'recency hypothesis' believed that 'some change of a biological kind occurred about 1800 such that a particular type of schizophrenia thereafter became much commoner' (Hare, 1988a). Consequently, potential cases were re-diagnosed (Hare, 1988b; Macalpine & Hunter, 1956), and searches of the literature performed (Ellard, 1987). On the other hand, there were those like Strömgren who believed that schizophrenia had existed as long as mankind' (Strömgren, 1982). After re-reporting Haslam's description of James Tilly

Matthews as 'the earliest clear description of schizophrenia', Carpenter explained that the absence of reported cases resulted from a 'different selection and description of cases for publication' (Carpenter, 1989); and Jeste et al ferreted out from historical documents 'a substantial number of clinical descriptions resembling modern conceptions of schizophrenia.' (Jeste et al., 1985).

A problem without solution

Looking back, it seems clear that, in terms of the conceptual parameters accepted by the participants, this debate could not have been resolved. Firstly there was the issue of what *counted as evidence*: would one historical fact had sufficed to falsify 'the recency hypothesis' (Turner, 1992); what level of diagnostic clarity was required for such case to be accepted as one of schizophrenia? Secondly, there was the unprovable nature of both the claim that schizophrenia 'had always existed' and that 'some biological change' had brought into life during the 19th century.

Unfortunately, there is no neat solution to this conundrum for the bad interaction between the definition (what counts as) and availability of evidence (will crucial documents be found in the future) and the assumptions made concerning the nature of schizophrenia (is it a RRUS, a vague concept or a social construct?). It can be concluded, therefore, that the history of behaviours redolent of schizophrenia is difficult to undertake for there are no reliable sources and all reports can be challenged from the diagnostic viewpoint (Allen, 1995). There is also the more complex problem of description: since the concept of mental symptom as we know it was only developed during the 19th century, it would be extremely uncommon that the cluster that there is schizophrenia would have been seen as we see it nowadays. Likewise, since the concept of mental illness as we have it nowadays (let alone that of chronic disease) was not available before the 19th century, then observation could not be made longitudinally (which is the current sine qua non of the disease) and all that observers would have had was either report cross-sectional acute stages or chronic ones and either would have been classified under a different rubric.

History of Concepts

Important changes in the conceptual frames governing views on mental illness preceded the construction of the two Kraepelinian psychoses. Occurred throughout the 19th century, these changes facilitated the acceptance of the view that there were only two psychoses, that one primarily affected the intellect and the other the emotions, and that they were constituted by different types of symptoms and had a different prognosis. The following background changes will be briefly touched upon: the passing of the old 'monolithic' view of madness, the inception of new units of analysis, and the complex process of transformation of the insanities into psychoses.

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The conceptual backdrop

Madness as a monolithic state

Until the end of the 18th century, insanity was considered by most as an all-or-none, metaphysical state which related to the body in an abstract way (Berthold-Bond, 1995). The mind was fully alienated, and in spite of forensic adumbrations, there was little medical interest in 'partial' insane. This 'holistic' approach was somewhat concealed by the Lockean *intellectualistic* definition of madness. Thus, in real life, the insane were considered as having 'fully lost his reason' and when he showed some improvement there were no conceptual tools to differentiate between 'improvement', 'remission', and 'cure'. Even Haslam's concept of 'lucid interval', created to deal with this very problem, carries with it the implicit assumption that it consists not in absence but in a 'stifling' of the mental disorder by the clever insane' (Haslam, 1809); remnants of this view can be found later in the 19th century (Bigot, 1877).

New units of analysis

As far as 18th century taxonomic theory was concerned, 'signs' (of diseases) and 'features' (of plants) played the same role. Unfortunately, the rules governing their combination were clearer for plants than for diseases. Whilst since Linné, botanical classifications had privileged certain specific features and hence led to an understandable classification of plants (Larson, 1971), medical classifications were mere undifferentiated lists of what nowadays would be called 'symptoms', 'syndromes' and 'diseases'. This is of course understandable for the current differentiation between these three clinical categories was only constructed during the 19th century. Our analytical and hierarchical model for the 'organization' of disease would have been alien to an 18th century physician.

'Mental atomism' (Dijksterhuis, 1961; Kargon, 1966), a view of the mind predominant since the time of John Locke, was one of the conceptual sources for the view that mental functions and (later) disorders were also divisible into stable units of analysis. It did not take long for the view to develop that such units were the mental symptoms, and that they could be combined into new clusters according to certain rules. The combination rules were based on the new ideas that, on the brain and its functions, were to develop during the 19th century. Some of these rules concerned the nature and organization of putative mental functions (e.g. modular versus non modular); others views on how mental functions related to the brain or other organ in the body (e.g. the stomach); and others about the ways in which normal mental function was disrupted by disease (leading to hyper, hypo and para changes of functionality).

In practical terms, this led both to the fragmentation of the old monolithic insanities (melancholia, mania, dementia, etc.), and to the creation out of the fragments (i.e. mental symptoms) of new clusters (i.e. diseases). Writers at the time were aware of these momentous changes, for example, one author distinguished between phenomenon, symptom and sign and believed the latter to carry information on occult changes (lesions)

in the body (Landré-Beauvais, 1813).

The insanities Become Psychoses

19th century alienists were confronted with the task of classifying objects whose definition was in a state of flux. As hinted at above, there were changes at various levels: *symptom-description* (new units of analysis and by the middle of the century new sources of information, e.g. subjective experience); *disease-construction* (new rules to put together symptoms into diseases made their appearance); *causality* (psychological vs. physical or organic, and external versus internal), *lesion type* (anatomical vs. physiological); *localization* (brain sites vs. diffuse location); *temporal context* (acute vs. chronic disease); and *outcome* (reversible versus irreversible). Two additional confounding factors appeared by the division of the 19th century: degeneration theory and accompanying views on the inheritance of mental disorder. The transformation of the old monolithic insanities into the new psychoses was presided over by these factors; only some are touched upon presently.

Clinical content

It would be reassuring and straightforward for the historian to find that what now are called 'psychotic symptoms' resulted from clinical observation alone. This is far from being the case. The inclusion of delusions, hallucinations, etc. as the 'hallmarks' of the new psychoses is the result of a complex process often driven by models and constraints upon symptom-availability rather than clinical inference. In regards to the former, the template for the functional psychoses, particularly schizophrenia, was 'acute delirium' (Berrios, 1981b). In regards to the latter, a variety of mental symptoms, particularly those of hypochondriasis, anxiety, obsessional disease, etc. were not available to alienists in their clinical practice (these patients were during the 19th century seen by general physicians). By the time these conditions became part of the territory of psychiatry, it was to late to incorporate their symptoms into the definition of the conventional psychoses (Berrios, 1999a).

Boundaries

At their inception, one of the fundamental distinction between dementia praecox and manic depressive insanity was the view that the former was a primary disorder of intellect and the latter of emotions or affect. This boundary was not provided by clinical observation for from the start affective symptoms were allowed in the former and delusions in the latter. The distinction concerned in Faculty psychology, the conceptual frame for both disorders. The acceptance by alienists at the beginning of the 19th century that, as suggested by faculty psychology, there were three bundles of mental functions (intellectual, emotional and volitional) prepared the terrain for the question of what diseases would originate if each bundle became diseased independently. The answer was intellectual insanity (by the 1870s the main candidate was the new version

of paranoia and by the turn of the century it became dementia praecox), emotional insanity (which was to become manic depressive illness), and volitional insanity (which became psychopathic and antisocial personality) (Berrios, 1988).

Classification

In regards to classification, the old taxonomic lists of the 18th century (based upon a different view of both symptom and disease) were broken up during the 19th century, including the old Cullean notion of neuroses (López Piñero, 1983). Thus set asunder, the 'vesanias' (which carried some of the old forms of insanity) were included into a variety of new classifications (Berrios, 1999b).

Aetiology

The anatomo-clinical model of disease was imported from medicine into alienism at the beginning of the 19th century and culminated in the work of the French Bayle. This encouraged a shift in the conceptualization of 'cause', namely the traditional Aristotelian externalist and sequential view was replaced the new simultaneous 'internal causal mechanism'. The two types of causality competed for supremacy and the question of which has more explanatory capacity has not been resolved even in our own day. The anatomo-clinical model was linked with two important 'dichotomies': organic/functional and endogenous/exogenous.

Organic/functional

This dichotomy becomes established by the second half the 19th century as the direct result of the redefinition of the neuroses and psychoses. At the beginning of the 19th century, the Cullean neuroses (which under 'vesanias' included all the known forms of madness) were considered as the 'organic' states par excellence (i.e. as conditions resulting from lesions in the nervous system and other parts of the body). At the time, the term 'psychoses' referred to subjective, experiential states and carried no aetiological implications. However, by the middle of the century, 'psychoses' began to be used to refer to the refurbished insanities (Flemming, 1859); and by the end of the century, 'psychosis' had all but replaced insanity as the generic term for both dementia praecox and manic depressive illness (Sauri, 1979; Schmidt, 1979; Berrios, 1987b; Beer, 1992).

By then the concept of neuroses had also been radically transformed, mainly but not exclusively as a result of the Freudian revolution. The 'new' neuroses only included anxiety disorders, hypochondria, hysteria and obsessional disease. Raynaud's phenomenon was separated off at the beginning of the 20th century. By the 1890s, Raynaud's disease was considered by many as occurring in 'hysterical, emotional, and excitable persons, in whom the neurotic element is highly developed. Indeed, there is every reason to believe that the unstable equilibrium of the nervous system is a potent predisposing factor in the pathogenesis of the disease'. (Quain, 1894). By the 1920s, and as a result of the great debate of the 1920s to open up the depressions themselves to psychodynamic

interpretation, 'neurotic depression' (the 'fifth neurosis') was added to the list (Berrios, 1992). The new neuroses were considered as a family of disorders all susceptible to functional explanation in terms of a particular model of the mind. In other words, they were not the result of pathology of the brain (hardware) but constituted special configurations of memories and emotions (i.e. they were a result of distorted learning and concerned the software component of behaviour) which made the person suffer.

During the second half of the 19th century, the organic/functional dichotomy started to be used *within* the field of the psychoses and this caused much confusion. This usage is found as early as 1881 less as an aetiological claim but to differentiate between psychoses with somatic symptoms (i.e. signs) (as in general paralysis of the insane) and with subjective symptoms alone (i.e. 'functional') (Fürstner, 1881). Although accepting that in principle all psychoses were 'organic', alienists nonetheless felt fit to subdivided them into 'organic' and 'functional' where the former term meant 'focal' and 'known' and the second 'unknown'. In his famous 1912 lecture, Hoche identified a weak and strong senses: 'for a time, 'functional' meant merely that we cannot yet prove anatomical changes with our present day equipment -always with the discreet proviso that this was available to the extent it should be. Today, however, the adjective is also used in the sense that by such disorders we mean those that will never have a pathological anatomy because they cannot have one' (Hoche, 1912). Thus, the psychosis seen in the context of head trauma, senile dementia, progressive paralysis of the insane, brain tumours, etc. were considered as 'organic' whilst dementia praecox and manic depressive illness were called 'functional' for their organic cause remained unknown (Bleuler, 1978). One of the earliest references to functional psychoses is Mendel's: 'on the other hand, there is a great difference of opinion amongst authors as to how to divide those mental diseases in which no anatomical findings have hitherto been met and which do not belong under any of the forms named. They are designated as functional psychoses, by which it is not said that anatomical changes do not exist, but only that we have so far been unable to verify them' (Mendel, 1907, p. 160).

This new division was as confusing then as it is now (Wilmanns, 1907): for example, Nissl protested and expressed dissatisfaction with the view that there could be mental disorders without organic basis and proposed that the assumption should be made that all did (Nissl, (1899). Interestingly enough, Alzheimer disagreed, stating that 'Nissl's statement that all mental disorders already have pathological basis has not yet been proven. Indeed, subjects with functional disorders die not from their mental disease but from other causes.' (Alzheimer, 1910). Bleuler was unable to reconcile his own organicist stance with Jung's psychological view that dementia praecox was a functional disorder. Jung was firm in his belief that the organic changes were secondary, rather than primary, to the psychological disorder: 'we must therefore postulate for dementia praecox a specific concomitant of the affect -toxins?- which causes the final fixation of the complex and injures the psychic functions as a whole.' (Jung, 1972). Likewise with Jaspers: in the last edition of his 'General Psychopathology' (published during the Second World War and much aided by Kurt Schneider), he allowed the inclusion under 'functional psychoses' of schizophrenia, manic depressive insanity and genuine epilepsy: 'these three (…) have four points in common. In the first place their study gave rise

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to the concept of disease entity (...) [There is not much historical evidence that this historiographical clain is actually correct], in the second place the cases which belong to this group cannot be subsumed under the disorders of Groups I and III [By Group I and III Jaspers meant 'cerebral illness' and 'personality-disorders', respectively]. One must however assume that many of these psychoses have a somatic basis (…) in the third place [they] are not exogenous but endogenous psychoses. Heredity is an important cause (…) in the fourth place they all lack anatomical cerebral pathology…' (Jaspers, 1963, pp. 607-608). It is informative to compare this section with that of the first edition where Jaspers offered a very different classification: organic or exogenous psychoses; process psychoses and degenerative insanity. The only process psychoses mentioned are paranoia and catatonia, and manic-depressive insanity as the main disease in the degenerative group (Jaspers, 1913, pp. 265-266).

Exogenous/endogenous

Nineteenth century neurobiological beliefs on the causality of the psychoses are no better preserved than in the exogenous/endogenous distinction. Controversial and unclear in meaning at the time of its inception, the terms are now but noble archaisms (Mundt, 1991). Kraepelin intimated that Möbius introduced the term into medicine in 1893 (Kraepelin, 1924) [Beer (1992) has since found that Möbius first explained his usage of 'endogenous' in a paper published a year earlier (Möbius, 1892)]: 'the principal condition [of the disease] must lie in the individual, in a congenital disposition (*Anlage*), other factors being merely contingent and quantitative'.

Before this is interpreted hastily, the reader must be reminded that for Möbius examples of 'endogenous disorders' were neurasthenia, hysteria, epilepsy, migraine, Huntington's chorea and Friedrichs' disease but not melancholia, mania, katatonia or hebephrenia (at the time of the publication of this book, dementia praecox had not yet been constructed by Kraepelin). On the other hand, Möbius considered as exogenous diseases toxic and infections conditions, trigeminal neuralgia, thyroid disease, multiple sclerosis and Parkinson's disease for they were supposed to be 'engendered from without'. In this regard, Gaston & Tatarelli are right when they claim that 'The endogenous has a double definition: negatively in relation to ongoing causes, and positive in relationship to a [putative] disposition' (Gaston & Tatarelli, 1984, p. 570).

It is unclear what did Möbius mean. He was at the cross-roads of a number of traditions: the 'Anlage' view active in 19th century German biology, degeneration theory, and the idea of predisposition or diathesis which concerned the depth of the metaphysical being rather than mind or brain, and the influence of Darwinism and the new idea of atavic drives. Möbius, however, was uncommitted in regards to a particular hereditary theory and his notion of endogenous is compatible both with a Mendelian or Lamarckian view as he was writing at a time when Mendel's ideas had not yet spread out and hence were unknown to him.

One thing is clear: that endogenous did not mean 'organic' or 'genetic' nor 'exogenous' 'environmental' in the current meaning of these terms (Heron, 1965; Lewis, 1971). Of recent French origin, *endogène* was defined in the field of Botany by de

Candolle in 1813 as 'something that comes from the inside, that has an internal cause' (Rey, 1995, p689). By 1845, the term appeared in English in a translation of von Humboldt's *Kosmos*: "I entitled (1832) the plutonic and volcanic eruptive rocks *endogenous* (that which is engendered in the interior,) the sedimentary and flœtz rocks *exogenous*, (externally engendered)" (Oxford English Dictionary, 2nd edition).

Möbius himself did not seem to have meant that the boundary between the two concepts was the skin (hence exogenous did not mean external or environmental) nor the neck (hence exogenous did not mean somatic or symptomatic as opposed to cerebral). Endogenous/exogenous, in fact, would have died a quiet death had not Kraepelin in 1896 used the terms to name his newly described psychoses.

The meaning of 'endogenous' was confused further by Aschaffenburg who in his influential book on the 'Classification of the Psychoses' suggested three classes: 'Endogenous psychoses' (psychasthenia, personality disorders, obsessional disorder and the constitutional psychoses: manic depressive and paranoia); 'exogenous psychoses' (intoxications, cretinism, myxoedema, alcohol- and drug-related); and organic diseases (neurosyphilis, psychoses related to brain disease, dementia praecox, epilepsy and the mental retardation) (Aschaffenburg, 1915, p. 32).

'Convergences' up to the Great War

The concepts of dementia praecox and schizophrenia were formulated at the turn of the 20th century but are based on earlier convergences, i.e. on descriptive events whereby identifiable alienists put together some of the fragments of the old insanities, called it a new name, and offered an concept to anchor them to ongoing medical and psychological theory. According the historiographical taste, these convergences can be called either rehearsals with partial insight or forms of 'dementia praecox' in their own right. Some of these are presently studied.

Kahlbaum

Jaspers (1948) dated the change in Kraepelin's intellectual fortunes to the time when he 'engaged in the fruitful use of Kahlbaum's (1828-1899) ideas' (Leibbrand & Wettley, 1969; Katzenstein, 1963). Kraepelin himself wrote that as a young man he always kept 'Kahlbaum's and Hecker's ideas in mind' (Kraepelin, 1983, p67). Recent historians have also considered Kahlbaum's ideas as essential to the Kraepelinian system and to his notion of dementia praecox (Bercherie, 1980; Pichot, 1984; Hoff, 1995; Berrios & Hauser, 1995). Kahlbaum developed his nosological ideas in '*Die Gruppirung der psychischen Krankheiten*' (Kahlbaum, 1863). In 1874 appeared his monograph on Catatonia (Kahlbaum, 1874), and his thoughtful paper on clinico-diagnostic perspective in psychopathology was published in 1878 (Kahlbaum, 1878).

Influenced by the French nosologists of the first half of the 19th century (Lanczik, 1992), Kahlbaum came to believe that cross-sectional presentations of disease were misleading and that the variable 'time' needed to incorporated into the definition of mental disease. This meant that the 'course' of the disease, as identified in longitudinal

observation of patients' cohorts, was the key to defining its boundaries. Kraepelin (1918) put this well:

> '[Kahlbaum was] the first to remark upon the need to combine the condition of the patient, his temporary symptoms, and the basic pattern of the disease. Disease in one and the same patient may change often and in various ways and confound treatment. Identical or similar symptoms may accompany different diseases. Hence their nature may only be revealed in their course and termination and, in some instances, at post-mortem. On the basis of these, Kahlbaum outlined a *second pattern* of illness similar to that of general paralysis which also included mental and physical features: an example of this was catatonia, where 'muscular tension' provided [Kahlbaum] with a basis to compare it with general paralysis. Although this view is open to criticism, Kahlbaum should be credited for suggesting a new approach. Attention to the progress and termination of mental disorders, information obtained from autopsies, and extra-knowledge into causes have made possible the combination of clinical evidence and diagnosis, on the basis of symptom pattern'.

In his book on Katatonia, Kahlbaum did not mean the 'insanity of tension' to be a 'new disease' but to redraw the clinical boundaries of *melancholia attonita*. Of his 26 cases, only 10 meet current diagnosis of 'catatonia'; the rest are cases of depression, epilepsy, and motility disorders (some due to basal ganglia damage secondary attempted hanging). Indeed, Kraepelin commented upon this: 'Although I must question, for the time being, the homogeneity of all the clinical pictures consolidated by Kahlbaum, many observations nevertheless induced me to recognize that the vast majority of those cases as examples of a special form of the disease' (Kraepelin, 1899a, p. 120). Thus, it was an act of faith that led Kraepelin to combine 'catatonia' with dementia simplex and paranoides and hebephrenia.

Hecker

Ewald Hecker (1843-1909) was a collaborator of Kahlbaum whose paper on hebephrenia (a putative variety of early onset of schizophrenia) was to become one of the classical publications of the late 19th century (Hecker, 1871). Hecker was a distinguished non-academic psychiatrist who in 1907 was awarded a professorial title by the Prussian government.

The word 'hebephrenia' was coined by Kahlbaum in 1863. He defined the group of the 'paraphrenias' as mental disorders occurring in periods of physiological change during vital development (physiologisch begründeten Wechselzustände der gesamten Lebensenwickelung). There were two paraphrenias (Kahlbaum, 1863): 'paraphrenia pubertatis s adolescens s. hebetica' 'for which I would like to coin the word hebephrenia because it can be used as a noun' and 'paraphrenia senilis with its related noun presbyophrenia'.

Hecker used this term to refer to an illness which starts between ages 18 and 22 with feelings of sadness, oppression, delusions, self-confession, and sometimes a tendency

to laugh and tell silly jokes, bizarre behaviour and fury. The melancholic phase is followed by a gradual disintegration of thinking, speech, writing, with repetitiousness, adoption of strange language accents, and obscenities. This is then replaced by a form of silly and irreversible dementia which included mental dullness, deterioration, agitation, and occasional hallucinations. Hecker illustrated his paper with 7 cases and was unable to report any brain changes (Hecker, 1871).

Kahlbaum returned to the topic in 1890 suggesting that the term 'Heboïdophrenie' might be applied to cases with less cognitive impairment and more antisocial behaviour (Kahlbaum, 1890). Another 17 cases and a review of the history of pubertal insanity was provided by Leon Daraszkiewicz in his brilliant doctoral thesis (Daraszkiewicz, 1892). The concept of hebephrenia caught on as attested by the great work of J. Christian (1899). It has been suggested that hebephrenia provided Kraepelin with a clinical model for the development of the notion of dementia praecox (Petho, 1972; Viallard, 1985).

Pick: a convergence manqué?

In 1881, Arnold Pick, the creative alienist from Prague, suggested that cases of primary dementia in young people should be called 'Dementia praecox' (Pick, 1891). He sought to postulated a continuity of his own case (Clara G.) with those reported earlier by Esquirol, Rousseau, Morel, Langdon Down, Clouston, Kahlbaum and Hecker. In fact, there is little difference between what Pick says here and what Kraepelin stated in 1896. Why is it then that historians of medicine keen on the 'who said it first' creed have not acknowledged this paper as the locus classicus for the history of de dementia praecox and considered Kraepelin as a follower of Pick? Those keen on explaining this interesting historical misrepresentation might want to resort two types of explanations: 'general' suggesting that official historical accounts do not necessarily follow the 'facts' but provide narratives to justify the dominant views in the present; and 'topical' concerning the fact that Pick was a minor alienist writing from Prague in a second line medical Journal.

Kraepelin's first convergence (1896)

Repeatedly did Kraepelin state that it was he who in 1896 had described dementia praecox for the first time. In the fifth edition of his *Psychiatrie*, published when he was still Professor at Heidelberg, Kraepelin divided all mental disorders into 'acquired (*Erworbene*) and 'predispositional' (*Veranlagung*). The former group is in turn divided into 5 subgroups: exhaustion states, intoxications, metabolic diseases, insanities related to neurological disease, and involutional diseases. The metabolic diseases (*Stoffwechselerkrankungen*), Kraepelin subdivided into myxedematous madness, cretinism, dementing processes (*Verblödungsprocesse*) and paralytic dementia. Finally, he includes three disorders under 'dementing processes': Dementia praecox, Katatonia and Dementia paranoides.

It is essential to keep in mind this classification. By dementia praecox in 1896, Kraepelin did not mean the omnibus category that it was to become in 1908 but a

specific disorder which he considered as close to Hecker's hebephrenia in that it occurred in the young as was a deteriorating process involving intellectual functions and behaviour but no involvement of consciousness (*'Das Bewusstsein des Kranken ist dauernd vollständig klar'*) (Kraepelin, 1896, p. 428). Indeed, in 1896, Kraepelin still considered katatonia and dementia paranoides as separate diseases. In this classification manic depressive insanity was included under a different order altogether.

Kraepelin's second convergence (1899)

By 1899, in the sixth edition of his *Psychiatrie*, Kraepelin had reached the conclusion that he needed 'to class together under the name dementia praecox a series of clinical pictures whose common characteristic is that they result in peculiar debilities (…) we shall call these forms hebephrenic, catatonic and paranoid. The first one is identical with the dementia praecox which I described earlier [i.e. in 1986 as per Kraepelin's first convergence], the second with catatonia, and the third embraces paranoid dementia, and in addition those cases otherwise counted as paranoia which lead rapidly to a considerable degree of mental weakness' (Kraepelin, 1899a, p. 103; Kraepelin, 1899b).

By 'paranoid dementia' Kraepelin meant a 'group of clinical pictures in which, aside from the symptoms of a rapidly developing mental deficiency in which the presence of mind remains wholly intact, delusions and in most cases also hallucinations constitute the most prominent disturbances for many years…'(Kraepelin, 1899a, p. 137; Ödegard, 1967). It is also noticeable here that 'paranoia', (which Kraepelin always found difficult to classify) could on occasions contribute to the omnibus category 'dementia praecox' if the patient deteriorated sufficiently.

The redefinition of dementia praecox also led to its reclassification; indeed, in volume 2 of the sixth edition Kraepelin abandoned the hierarchical, pyramidal system and simply lists 13 disorders which appear at the same level of organization: infectious insanity, insanity due to exhaustion, intoxication, thyroid-related insanity, dementia praecox, paralytic dementia, insanity in neurological disease, involutional insanities, manic depressive insanity, paranoia, general neuroses, hysterical insanity, psychopathic states, and mental retardation.

German reaction to Kraepelin's second convergence

Kraepelin's ideas on dementia praecox resulted of an interaction between the evolution of his own thought and reactions by contemporary alienists (Kraepelin, 1905; Kolle, 1955). In general, Kraepelin was laconic about the views that influenced him, and on some occasions it is difficult to know whether some ideas he presents are meant to be his or someone else's. For example, the term 'dementia praecox' had been around since 1860 (Morel) and Pick had used it specifically in its latinized version 1891 to name the same type of patient as Kraepelin was to do both in 1896 and 1899. However, in neither of these occasions Kraepelin acknowledged Morel or Pick (he went on to do so only in 1904 and 1913). The same can be said about Kraepelin's attitude towards the

work of authors writing before his final synthesis (e.g., Wernicke, Diem, Stransky, Gross, Weygandt, Zweig, Albrecht and Hoche). There is space here only to deal with some of these writers.

Werkicke

Based on his own speculative physiopathology, Wernicke developed a multiplex classification for the psychoses (Lanczik, 1988). His untimely death in 1905 deprived European psychiatry from an important alternative to Kraepelin's classification. Surprisingly, there was little contact between the two men (Kraepelin, 1987).

Wernicke dealt with the crucial issue of the hebephrenic psychoses in lecture Nº 39 of his *Grundriss*. He accepted the clinical existence of (Wernicke, 1906):

> 'Kahlbaum's hebephrenia or his abbreviation Heboid as a specific psychosis of puberty because the symptoms did fit in well within the psychology of this age group (…) however, Kahlbaum did not notice in this condition the presence of other symptoms such as experiences and images of anguish and hypochondriacal sensations…' (p. 518).

Importantly, Wernicke goes on:

> More frequent are cases hebephrenia in which the original madness is followed by a progressive dementia (*fortschreitenden Demenz*). These cases, which can be called expansive hebephrenic autopsychoses, after half a year progress to a state of atonicity and deep dementia (*tiefsten Blödsinn*). This suggests that motility psychoses of any type, but particularly the akinetic type, may have a predilection for the period of puberty. The tendency of the akinetic psychoses to end up in dementia may be based on some aetiological factor. However, I have seen cases of akinetic psychoses in puberty that have improved and left the mind of these young people intact. Thus, Kraepelin's prediction of bad prognosis does not seem even to be correct in cases occurring during puberty.' (pp. 518-519).

Thus, whilst the clinical state of hebephrenia with motor disorder occurring during puberty, as described by Kahlbaum and Hecker is acknowledged by Wernicke, he proceeds to include it under his own classification. As far as one is able to determine, nowhere in his *Grundriss* did Wernicke veer use the term 'dementia praecox'; by the time that the term 'schizophrenia' appeared, he had already died.

Diem

In 1903, Otto Diem, an assistant of Bleuler at the Burghölzli Clinic, published a paper entitled 'The simple dementing form of dementia praecox (dementia simplex)' (Diem, 1903), reporting 19 cases of a condition characterised by onset during early puberty, with instability, lack of will power, vagrancy, lasciviousness, excitability, quarrelsomeness, criminal activity, and occasional depression. Then they show indifference,

apathy, memory impairment, inability to monitor the social environment, and thought disorder. Diem found that in 15 out of his 19 cases there was family history of mental disorder. He compared his cases with those reported by Kahlbaum, Hecker, Daraszkiewicz and Kraepelin himself emphasizing that the common feature to all these patients (including his own) was that they developed dementia without having shown any major symptoms or episodes; i.e. the disease was insidious and had no warning signs.

There have since been various interpretations of the clinical nature of schizophrenia simplex (Peters, 1988). As late as 1948, Otto Kant, a German emigree to the USA complained that 'the simple type of schizophrenia, wanting the colour and dramatic intensity of other subtypes, has received little scientific attention'. In a sample of 64 patients, he found that simple schizophrenia seemed to have a rate of family history, typical personality, periods of acute psychoses before 'disintegration' and (in keeping with his psychodynamic leanings), Kant believed that these patients 'attempted reality fulfilment of their basis drives on a primitive level' (Kant, 1948).

Twenty years later, Stone et al. (1968) reported that simple schizophrenia was an empty diagnosis and should be abandoned. This proposal went unheeded and in 1989 Black & Boffeli (1989) felt that simple schizophrenia should, after all, be incorporated in DSM IV. This did not happen but in a twin sample, Dworkin (1992) ascertained that cases diagnosed as simple schizophrenia did not differ from other types in respect to negative symptoms, premorbid social adjustment and age of onset. Unfortunately, these authors do not refer to a major German paper on the subject (Klosterkötter, 1983), written from the perspective of Huber's 'basic syndrome' (Huber, 1966).

Stransky

Erwin Stransky (1877-1962) was an Austrian alienist who suggested that central to dementia praecox was a disorders called intrapsychic ataxia which consisted in a rather hesitating relationship (not a full disconnection) between the intellectual (noo-psyche) and emotional functions (thymo-psyche) (Stransky, 1904; Kretzschmar & Petiti, 1994). This caused the patient to show incongruous and bizarre behaviour. Stransky was at paints to differentiate his mechanism from Wernicke's and Gross's 'disjunction'. Stransky's ataxia is conceptually parasitical upon the metaphor of splitting.

Gross

Another Austrian writer, Otto Gross (1877-1920), also made use of the splitting metaphor but preferred the name 'dementia sejunctiva'. By dementia Gross meant a 'process' rather than a static end-state; the accompanying term he admitted to having borrowed from Wernicke but insisted that he used it with a different meaning: i.e. as a synthesis of associations and functions taking place in space and not in time, as Wernicke had thought (Gross, 1904). In his sense, 'sejunction meant a breakdown of consciousness of a particular form, one in which there was a simultaneous collapse of various functionally independent components' (Gross, 1904, p. 1146).

Hoche

In 1906 (Hoche, 1906) and 1912 (Hoche, 1912), Alfred Hoche (1865-1943) contributed to the debate with two important conceptual papers on 'symptom-complexes', the meaning of mental disorder, and the relationship between brain and mental symptoms. Hoche emphasised description, pre-formed and stereotyped brain responses to injury, and suggested that the more stable clinical manifestations of mental disorder were neither diseases nor symptom but symptom-complexes. He dismissed the excessive over-searching for causes (whether organic and psychological) and believed that it was nonsense to try and establish closed correlations between specific sites and diseases as psychiatric syndromes were likely to result from the collective and synthetic activity of multiple brain sites and no one in particular was to be considered as individually responsible: 'perhaps the explanation of the strange inaccessibility of a number of clinical syndromes -as for example the catatonic blockage in stupor- lies in the fact that the primary change occurring is not to be sought in the nearest, most obvious field, i.e. in the instance of catatonia in the psychomotor field' (p. 339). Hence, the search for 'entities', Hoche called 'the hunt after a phantom'. These ideas Hoche applied to dementia praecox:

> 'the broadly anatomically-conditioned disorders are the least subject to any rules in their individual symptomatology. In the same way, this applies to progressive paralysis, to arteriosclerotic and senile mental disorders and to cases arising from the narrower and exact areas of dementia praecox that lead to serious and definitive deficit symptoms. Certain characteristics of the syndrome are common to all cases, though with the disorders mentioned the progressive disintegration of the mental personality is really the only thing that remains… however this process is accompanied by a colourful palette of symptoms in a great variety of combinations.' (p. 340).

Bleuler

Dementia praecox became 'schizophrenia' in a book published by Bleuler in 1911, although both the term and the idea had been around since 1907 and originated from a collective effort at the Burghölzli Hospital (Berrios, 1987c). For example, Jung's *Psychology of Dementia Praecox* (Jung, 1972) contains similar concepts. Bleuler's notion of schizophrenia was shaped by associationistic psychology and psychodynamic theory and underwent changes between 1911 and his paper at the Congress of Geneva (Lanteri-Laura & Gros, 1982; Guiraud, 1968; Mondragon, 1952). Bleuler justified the use of his neologism by saying that dementia praecox had fatalistic connotations and was taken by many to mean dementia affecting the young. The change in name, however, led to a change in metaphor. As Lanteri-Laura and Gros (1992) have suggested, however, 'splitting' (*Spaltung*) turned out to be as misleading a metaphors as earlier ones.

During the early years of this century, the view that mental symptoms were just 'symbols' of events occurring beyond awareness became popular, and not surprisingly,

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there is much of this in the view put forward by Jung and Bleuler. In the event, even signs became 'psychologized': for example, stereotypies and echo phenomena were no longer considered as disorders of the motor system but of the 'will' (Berrios & Gili, 1995). Bleuler's conception was successful for it offered a compromise between the old neuropsychiatry and the new psychodynamic ideas: symptoms were, to certain extent, 'understandable' although they might still be caused by some 'unknown toxin'.

Kraepelin's third convergence (1913)

In the eight and final edition, Kraepelin (1908-1913) dealt with comments on his 1899 views and added a new clinical category to the list: 9. 'Endogenous conditions with evolution towards deterioration' which include dementia praecox and paraphrenia. The separation of paraphrenia was not acceptable to some of Kraepelin's own followers. For example, Wilmans argued that family studies showed that paraphrenia was no different from dementia praecox, and Wilhelm Mayer (1921) found that on follow up patients diagnosed as paraphrenias by Kraepelin went on to develop clinical states indistinguishable from 'dementia praecox'. Dementia praecox is here at its broadest and subsumes the following 'forms': simple, simple-stuporous, hebephrenia, delusional-depressive, circular, agitated, periodic, catatonia (both excited and stuporous), paranoid (mitis and gravis), and confusional speech (schizophasia). 'Endogenous dementias', in turn, are defined as (Kraepelin, 1919):

> 'their clinical relations are not yet clear but they all display two peculiarities, that they are in the first place, so far as can be seen, not occasioned from within but arise from internal causes, and that secondly, at least in the great majority of cases, they lead to a more or less well-marked mental enfeeblement. It appears that this form of mental weakness, in spite of great differences in detail, exhibits many features in common with other forms of dementia, such as are known to us as the result of paralysis, senility or epilepsy' (p. 1).

In the meantime, 'manic depressive psychosis' maintained its separate position but it had become wider (Kraepelin, 1921):

> 'it includes on the one hand the whole domain of so-called periodic and circular insanity, on the other hand simple mania, the greater part of the morbid states termed melancholia and also a not inconsiderable number of cases of amentia. Lastly, we include here certain sight and slightest colourings of mood, some of them periodic, some of them continuously morbid, which on the one hand are to be regarded as the rudiment of more severe disorders, on the other hand pass over without sharp boundary into the domain of personal predisposition. In the course of the years I have become more and more convinced that all the above mentioned states only represent manifestations of a single morbid process' (p. 1).

Kraepelin was careful not to propose a speculative pathophysiology of dementia praecox (Roelcke, 1997), and contented himself with offering clinical description, natural history and prognosis. In his acceptance of a longitudinal definition of disease he had

been influenced by Kahlbaum, and in his concepts of 'species', 'natural kind', and dichotomous taxonomy he received the advise of his elder brother Kurt, the great taxonomist from Hamburg (Berrios & Hauser, 1988; Jablensky et al., 1993; Braem, 1983).

Kraepelin's change of mind (1920)

But all along Kraepelin was aware of the fact that mental disorders often merged into one another (Kraepelin, 1920):

> 'It is becoming increasingly obvious that we cannot satisfactorily distinguish these two diseases'.

In 1920, he abandoned the view that symptoms were pathognomonic:

> 'It is incorrect to attribute signs to specific disease processes ... symptoms are not limited to a distinct disease process but occur in the same form in response to different morbid insults.'

Indeed, in this paper, Kraepelin, did even cast doubt on the distinction between dementia praecox and manic-depressive illness on the basis of symptoms alone:

> 'We shall have to get used to the fact that our much used clinical check-list does not permit us to differentiate reliably between manic-depressive illness and dementia praecox.'

However, Kraepelin claimed that the two diseases are different in terms of their course:

> 'we cannot help but maintain that the two disease processes themselves are distinct. On the one hand we find those patients with irreversible dementia and severe cortical lesions. On the other are those patients whose personality remains intact. Thos distinction is too overwhelming for us to accept much overlap between the two groups, particularly as we can often predict the course of the two from the clinical signs'.

Conclusions

The history of schizophrenia can be best described as the history of a set of research programmes running in parallel rather than seriatim and each based on a different concept of disease, of mental symptom and of mind. *Per force* only few of these programmes have been discussed in this short chapter.

Historical research shows that there is little conceptual continuity between Morel, Kraepelin, Bleuler and Schneider. Two consequences follow from this finding. One is that the idea of a linear progression culminating in the present is a myth. The other that

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the *current* view of schizophrenia is *not* the result of one definition and one object of inquiry successively studied by various psychiatric teams but a patchwork made out of clinical features plucked from different definitions. More research is needed to find out what led to this sorry state of affairs. It might simply be the result of historical ignorance or the application of some pedestrian operationalism.

In a way it does not matter. What matters is that the continuity story be rejected because its main role has been not to illuminate but to flatter and justify the present. Hopefully, the discontinuity history will tell about unsung heroes and their imaginative solutions and will offer uncommitted researchers a trove of psychiatric truths. One of these may be that there is no such a thing as a unitary disease called schizophrenia but only a collection of mental symptoms, some congenital, some relics from evolution, and others acquired. Be that as it may, some of the symptoms listed above are blighting people's lives and need urgent research; but to this work, the current concept of schizophrenia may be irrelevant.

References

Alexander, F.G. & Selesnick, S.T. (1966) *The History of Psychiatry*. New York: Harper & Row.

Allen, D.F. (1990). *Autour du Palais Somptueux. Contribution à l'étude du mot «schizophrénie»*. University of Paris-VII, Thesis de DEA.

Allen, D.F. (1995). *Vers une perspective axiologique de la schizophrénie (éléments pour une histoire critique)*. Thèse de la Université de Paris 7.

Alzheimer, A. (1910). Die diagnostischen Schwierigkeiten in der Psychiatrie. *Zeitschrift für die gesamte Neurologie und Psychiatrie 1*, 1-19.

Aschaffenburg, G. (1915). *Die Einteilung der Psychosen*. Leipzig: Franz Deuticke.

Beer, M.D. (1992). *Psychosis: A History of the Concept*. MD Thesis, University of London.

Bercherie, P. (1980). *Les Fondements de la Clinique*. Paris: La Bibliothèque d'Ornicar?

Berrios, G.E. (1981a). Stupor: A Conceptual History. *Psychological Medicine, 11*, 677-688.

Berrios, G.E. (1981b). Delirium and Confusion in the 19th century. *British Journal of Psychiatry, 139*, 439-449.

Berrios, G.E. (1987a). Dementia during the 17th and 18th Century: A Conceptual History. *Psychological Medicine, 17*, 829-837

Berrios, G.E. (1987b). Historical Aspects of Psychoses: 19th Century Issues. *British Medical Bulletin, 43*, 484-498

Berrios, G.E. (1987c). Introduction to 'The Fundamental Symptoms of dementia Praecox or the Group of Schizophrenias'. In C. Thompson (ed) *The Origins of Modern Psychiatry*. Chichester: Wiley (pp200-209).

Berrios, G.E. (1988). Historical background to abnormal Psychology. In Miller E and Cooper P (eds) *Adult Abnormal Psychology*. London: Churchill Livingstone (pp 26-51).

Berrios, G.E. (1990). Alzheimer's disease: a conceptual history. *International Journal of Geriatric Psychiatry, 5*, 355-365.

Berrios, G.E. (1992). The history of the affective disorders. In E.S. Paykel (ed) *Handbook of Affective*

Disorders, 2nd edition. Churchill Livingstone (pp 43-56).

Berrios, G.E. (1999a). The anxiety Disorders: a conceptual history. *Journal of the Affective Disorders, 56*: 83-94.

Berrios, G.E. (1999). Classification in Psychiatry: a conceptual history. *Australian and New Zealand Journal of Psychiatry, 33*, 145-160.

Berrios, G.E. & Hauser, R. (1988). The early development of Kraepelin's ideas on classification. a conceptual history. *Psychological Medicine, 18*, 813-821.

Berrios, G.E. & Freeman, H. (1991). *Alzheimer and the dementias*. London: Royal Society of Medicine.

Berrios, G.E. & Gili, M. (1995). Will and its disorders. A conceptual history. *History of Psychiatry, 6*, 87-10.

Berrios, G.E. & Hauser, R. (1995). Kraepelin. In G.E. Berrios & R. Porter (eds) *History of Clinical Psychiatry.* London: Athlone Press (pp280-291).

Berthold-Bond, D. (1995). *Hegel's Theory of Madness*. New York: State University of New York Press.

Bigot, V. (1877). *Périodes raisonnantes de l'aliénation mentale*. Paris: Baillière.

Black, D.W. & Boffeli, T.J. (1989). Simple Schizophrenia: past, present and future. *American Journal of Psychiatry, 146*, 1267-1273.

Bleuler, E. (1908). Die Prognose der Dementia Praecox - Schziophreniegruppe. *Allgemeine Zeitschrift für Psychiatrie, 65*, 436-464.

Bleuler, E. (1911). *Dementia Praecox oder Gruppe der Schizophrenien*. Leipzig: Deuticke.

Bleuler, M. (1978). *The Schizophrenic Disorders*. New Haven: Yale University Press.

Boyle, M. (1990). Is schizophrenia what it was? *Journal of the History of the Behavioral Sciences, 26*, 323-333.

Braem, F. (1983). Ein Wort ueber Herrn Prof. Karl Kraepelin und seinen neuesten beitrag zur Bryozoenkunde. *Naturforschenden Gesellschaft, 95*, 3-14.

Brockington, I.F. & Nalpas, A. (1993). L'approche clinique et le concept de schizophrénie. In P. Pichot & W. Rein (eds) *L'Approche Clinique en Psychiatrie* Vol II. Collection Les Empêcheurs de Penser en Rond (pp139-158).

Bumke, O. (1932). Die Auflösung der Dementia Praecox. *Klinische Wochenschrift, 3*, 437-440 (translation available in *History of Psychiatry, 4*, 129-139, 1993).

Carpenter, P.K. (1989). Descriptions of schizophrenia in the Psychiatry of Georgian Britain: John Haslam and James Tilly Matthews. *Comprehensive Psychiatry, 30*, 332-338.

Christian, J. (1899). De la Démence Précoce des jeunes gens. Contribution a l'étude de l'hébéphrénie. *Annales Médico-Psychologiques, 9*, 43-65; 200-216; 421-436; 10, 5-23; 177-188.

Colodrón, A. (1999). *Cinco Conferencias sobre esquizofrenia*. Madrid: Editorial Triacastela.

Daraszkiewicz, L. (1892). *Ueber Hebephrenie insbesondere deren schwere Form*. Dorpat, H. Laakmann.

Diem, O. (1903). Die einfach demente Form der Dementia praecox (Dementia simplex). *Archiv für Psychiatrie und Nervenkrankheiten, 37*, 111-187.

Dijksterhuis, E.J. (1961). *The Mechanization of the World Picture*. Oxford: Oxford University Press.

Dworkin, R.H. (1992). Simple schizophrenia. *Acta Psychiatrica Scandinavica, 85*, 61-64.

Ellard, J. (1987) Did schizophrenia exist before the eighteenth century. *Australian and New Zealand Journal of Psychiatry, 21*, 306-314.

Ewald, G. (1930) Dementia Praecox und Schizophrenie. *Zeitschrift für die gesamte Neurologie und*

Psychiatrie, 123, 465-471.
Flemming, C.F. (1859). *Pathologie und Therapie der Psychosen.* Berlin: August Hirschwald.
Fürstner, C. (1881). Über delirium acutum. *Archiv für Psychiatrie und Nervenkrankheiten, 5*, 505-543
Garrabé, J. (1992). *Histoire de la Schizophrénie*. Paris: Seghers.
Gaston, A. & Tatarelli, R. (1984) Analyse critique de l'évolution du concept d'endogène. *L'Evolution Psychiatrique. 49*, 569-575
Gaupp, R. (1939). Die Lehren Kraepelins in ihrer Bedeutung für die heutigen Psychiatrie, *Zeitschrift für die gesamte Neurologie und Psychiatrie, 165*, 47-75.
Gross, O. (1904). Zur Nomenclatur "Dementia Sejunctiva". *Neurologisches Zentrablatt, 23*, 1144-1146
Gruhle, H.W. (1932). Geschichtliches. In K. Wilmanns (ed) *Die Schizophrenie*. Fifth Part of Bumke's Handbuch der Geisteskrankheiten. Berlin: Springer
Guiraud, P. (1968). Origine et évolution de la notion de la schizophrénie. *Confrontations Psychiatriques* 9-29.
Guraud, J.M. (1985). C.G. Jung et les schizophrénies. Étude chronologique (1903-1957). *L'Information Psychiatrique. 61*, 1077-1085.
Janzarik, W. (1986). Geschichte und Problematik des Schizophreniebegriffes. *Nervenarrzt, 57*, 681-685.
Hare, E. (1988a). Schizophrenia as a recent disease. *British Journal of Psychiatry 153*: 521-531.
Hare, E. (1988b). Schizophrenia before 1800? The case of the Revd. George Trosse. *Psychological Medicine, 18*: 279-285.
Haslam, J. (1809). *Observations on Madness and Melancholy*. 2nd Edition. London: J. Callow.
Haslam, J. (1810). *Illustrations of Madness*. London, G. Hayden.
Hecker, E. (1871). Die Hebephrenie. *Virchows Archiv. 52*, 394-429.
Heron, M.J. (1965). A note on the concept endogenous-exogenous. *British Journal of Medical Psychology, 38*, 241-245.
Hoche, A. (1906).Kritisches Vortrag zur psychiatrischen Formenlehre. *Allgemeine Zeitschrift für Psychiatrie, 63*, 559-563.
Hoche, A. (1912). Die Bedeutung der Symptomenkomplexe in der Psychiatrie. *Zeitschrift für die gesamte Neurologie und Psychiatrie, 12*: 540-551 (English translation available in *History of Psychiatry, 3*: 334-343, 1991).
Hoff, P. (1994). *Emil Kraepelin und die Psychiatrie als klinische Wissenschaft*. Berlin, Springer.
Hoff, P. (1995). Kraepelin. In Berrios G.E. & Porter R. (Eds) *History of Clinical Psychiatry*. London, Athlone Press, pp261-279
Huber, G. (1966). Reine Defektssyndrome und Basisstadien endogener Psychosen. *Fortschritte der Neurologie Psychiatrie, 34*, 409-425.
Jablensky, A., Hugher, H., Von Cranach, M., & Kalinov, K. (1993). Karepelin revisited: a reassessment and statistical analysis of dementia praecox and manic depressive insanity in 1908. *Psychological Medicine, 23*, 843-858.
Jaspers, K. (1913). *Allgemeine Psychopathologie.* 1st Edition. Berlin: Springer.
Jaspers, K. (1948). *Allgemeine Psychopathologie*. 5th Edition. Berlin: Springer.
Jaspers, K. (1963). *General Psychopathology*. 7th Edition. Translated by J. Hoenig & M.W. Hamilton. Manchester: Manchester University Press.
Jeste, D.V., Carmen, R., Lohr, J.B. & Whyatt, R.J. (1985). Did schizophrenia exist before the eighteenth

century. *Comprehensive Psychiatry, 26*, 493-503.
Jung, C.G. (1972). *The Psychogenesis of Mental Disorder*. Volume 3 of Collected Works. London, Routledge. (First published in German in 1914).
Kahlbaum, K. (1863). *Die Gruppirung der psychischen Krankheiten und die Eintheilung der Seelenstörungen. Entwurf einer historische-kritischen Darstellung der bisherigen Eintheilungen und Versuch zur Anbahnung einer empirisch-wissenschaftlichen Grundlage der Psychiatrie als klinischer Disciplin*. Danzig: A.W. Kafemann.
Kahlbaum, K. (1874). *Die Katatonie oder das Spannungsirresein*. Berlin, Kirschwald.
Kahlbaum, K. (1878). Die klinisch-diagnostischen Gesichspunkte der Psychopathologie. *Sammlung klinischer Vorträge, 126*: 1127-1146. This paper needs urgent translation into English.
Kahlbaum, K. (1890). Ueber Heboïdophrenie. *Zeitschrift für Psychiatrie, 46*, 461-474.
Kant, O. (1948). Clinical Investigation of simple schizophrenia. *Psychiatric Quarterly, 17*, 141-151.
Kargon, R.H. (1966). *Atomism in England from Hariot to Newton*. Oxford: Oxford University Press.
Katzenstein, R. (1963). *Karl Ludwig Kahlbaum und sein Beitrag zur Entwicklung der Psychiatrie*. Zürich, Juris.
Klosterkötter, J. (1983). Schizophrenia simplex. Gibt es das? *Nervenarzt, 54*, 340-346.
Kolle, K. (1955). Die endogenen Psychoses. Das delphische Orakel der Psychiatrie. *Münchener medicinischen Wochenschrift, 97*, 135-154.
Kraepelin, E. (1896). *Psychiatrie*. 5th edition. Leipzig: Barth.
Kraepelin, E. (1899a). *Psychiatrie. Ein Lehrbuch*. Sixth Edition. Leipzig: Barth.
Kraepelin, K. (1899b). Diagnose und Prognose der Dementia praecox. *Allgemeine Zeitschrift für Psychiatrie, 56*, 254-263.
Kraepelin, E. (1908-1913). *Psychiatrie*. 4 vols, 8th Edition. Leipzig. Bart
Kraepelin, E. (1905). Fragenstellungen der klinischen Psychiatrie. *Centralblatt für Nervenheilkunden und Psychiatrie, 28*: 573-590.
Kraepelin, K. (1918). Hundert Jahre Psychiatrie. Ein Beitrag zur Geschichte menschlicher Gesittung. *Zeitschrift der Neurologie, 38*, 161-275.
Kraepelin, E. (1919). Dementia Praecox and Paraphrenia. Translated by R.M. Barclay & G.M. Robertson. Edinburgh, Livingstone (extracted from 8th and last edition, published in 1910).
Kraepelin, E. (1920). Die Erscheinungsformen des Irreseins. *Zeitschrift für die gesamte Neurologie und Psychiatrie, 62*, 1-29 (translation available in *History of Psychiatry, 4*, 509-529, 1992).
Kraepelin, E. (1921). *Manic depressive Insanity and Paranoia*. Translation of the 8th edition by R.M. Barclay and G.M. Robertson. Edinburgh, Livingstone.
Kraepelin, E. (1924). Paul Julius Möbius (1853-1907). In T. Kirchhoff (ed) *Deutsche Irrenärzte*. Vol.2, Berlin, Springer (pp274-279).
Kraepelin, E. (1983). *Lebens-Erinnerungen*. Berlin, Springer.
Kraepelin, E. (1987). *Memoirs*. Berlin, Springer.
Kretzschmar, C. & Petiti, M. (1994). Erwin Stransky et l'ataxia intrapsychique'. *L'Encéphale, 20*, 377-383.
Lanczik, M. (1988). *Der Breslauer Psychiater Carl Wernicke*, Sigmaringen, Thorbecke.
Lanczik, M. (1992). Karl Ludwig Kahlbaum (1828-1899) and the emergence of psychopathological and nosological research in German psychiatry. *History of Psychiatry, 3*, 53-58
Landré-Beauvais, A.J. (1813). *Séméiotique ou Traité des signes de maladies*. 2nd Edition. Paris, Brosson).
Lanteri-Laura, G. & Gros, M. (1982). Historique de la schizophrénie. *Encyclopédie Médico-Chirurgicale*

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(Paris) 37281 C[10].
Lanteri Laura, G. & Gros, M. (1992). *Essai sur la discordance dans la psychiatrie contemporaine*. Paris: Epel.
Larson, J.L. (1971). *Reason and Experience. The Representation of Natural Order in the work of Carl Von Linné*. Berkeley: University of California Press.
Leibbrand, W. & Wettley, A. (1969). *Der Wahnsinn. Geschichte der abendländischen Psychopathologie*. Freiburg: Karl Aber.
Lewis, A. (1971). 'Endogenous' and 'exogenous' a useful dichotomy? *Psychological Medicine, 1*, 191-196.
López Piñero, J.M. (1983). *History of the concept of neuroses*. Translated by D. Berrios, Cambridge: Cambridge University Press.
Macalpine I. & Hunter R. (1956). *Schizophrenia 1677*. London, Dawson
Mayer W. (1921). Über paraphrene Psychosen. *Zeitschrift für die gesamte Neurologie und Psychiatrie* 71: 197-206.
Meduna, L.J. & McCulloch, W.S. (1945). The Modern Concept of Schizophrenia. *The Medical Clinics of North America*, January Issue (pp147-164).
Mendel, E. (1907). *Textbook of Psychiatry*. Translated by W.C. Krauss. Philadelphia: F.A. Davies.
Minkowski, E. (1925). La Génése de la notion de schizophrénie et ses caractères essentiels. *L'Évolution Psychiatrique*, 1, 193-236.
Minkowski, E. (1927). La Schizophénie. *Psychopathologie des schizoïdes et des schizophrènes*. Paris: Payot.
Möbius, P.J. (1892). Über die Einteilung der Krankheiten. *Centralblatt für die gesamte Neurologie in Wissenschaft und Praxis mit besonderer Berücksichtigung der Degenerations-Anthropologie, 15*, 289-301.
Mondragon, J. (1952). *Le développement de la notion de schizophrénie*. Paris: Thèse.
Morel, B.A. (1860). *Traité des Maladies Mentales*. Paris: Masson.
Mundt, Ch. (1991). Endogenität von Psychosen: Anachronismus oder aktueller Wegweiser für die Pathogeneseforschung? *Nervenarzt, 62*, 3-15.
Nissl, F. (1899). Über die sogenannten functionellen Geisteskrankheiten. *Münchener medizinischen Wochenschrift, 46*, 1453-1456.
Ödegard, Ö. (1967). Changes in the prognosis of functional psychoses since the days of Kraepelin *British Journal of Psychiatry, 113*, 813-822).
Peters, U.H. (1988). Die deutsche Schizophrenielehre und die psychiatrische Emigration. *Forschritte Neurologie Psychiatrie, 56*, 347-360.
Petho, B. (1972). Hundert Jahre Hebephrenie. *Psychiatrie, Neurologie und medizinische Psychologie 24*, 305-317.
Pichot, P. (1984). *A Century of Psychiatry*. Paris: Dacosta (pp106-112).
Pick, A. (1891) Ueber primäre chronische Demenz (so. Dementia praecox) im jugendlichen Alter. *Prager medicinische Wochenschrift, 16*, 312-315
Quain, R. (1894). *A Dictionary of Medicine*. 2 vols. New Edition. London: Longmans, Green and Co, (p628).
Rey, A. (1995). *Dictionnaire Historique de la Langue Française*. Paris: Le Robert, Vol. 1 (p689).
Rieder, R.O. (1974). The origin of our confusion about schizophrenia. *Psychiatry, 37*, 197-208.
Roelcke, V. (1997). Biologizing social facts: an early 20th century debate on Kraepelin's concepts of

culture, neurasthenia and degeneration. *Culture, Medicine and Psychiatry, 21*, 383-403.
Sarbin, T.R. (1972). Schizophrenia is a myth, born of metaphor, meaningless. *Psychology Today*, June, 18-27.
Scharfetter, C. (1975). The Historical Development of the Concept of Schizophrenia. In M.H. Lader (ed) *Studies of Schizophrenia*. Ashford: Headley Brothers, (pp5-8).
Schmidt, P. (1979). A propos de problèmes posés par la définition de la psychose. *Annales Médico-Psychologiques, 137*: 595-604
Stone, A.A., Hopkins, R., Mahnke, M.W., Shapiro, D.W., & Silvergate, H.A. (1968). Simple schizophrenia-syndrome of shibboleth. *American Journal of Psychiatry, 125*, 305-315.
Stransky, E. (1904). Zur Auffassung gewisser Symptome der Dementia praecox. *Neurologisches Centralblatt, 23*: 1074-1085.
Strömgren, E. (1982). Development of the Concept of Schizophrenia. In J. Wing & L. Wind (eds) *Psychoses of Uncertain Aetiology*. Cambridge: Cambridge University Press.
Turner, T.H. (1992) Schizophrenia as a permanent problem. *History of Psychiatry, 3*, 413-429.
Viallard, A. (1985). Introduction à la traduction du text de Hecker sur l'hébéphrénie. *L'Evolution Psychiatrique, 50*, 317-323.
Weber, M.M. & Engstrom, E.J. (1997). Kraepelin's 'diagnostic cards': the confluence of clinical research and preconceived categories. *History of Psychiatry, 8*, 375-385.
Wender, P.H. (1963). Dementia Praecox: the development of the concept. *American Journal of Psychiatry, 119*, 1143-1151.
Wernicke, C. (1906). *Grundriss der Psychiatrie*. 2nd Edition. Leipzig: Thieme.
Williamson, T. (1994). *Vagueness*. London: Routledge.
Wilmanns, K. (1907). Zur Differentialdiagnostik der "funktionellen" Psychosen. *Centralblatt für Nervenheilkunde und Psychiatrie, 30*,h 569-590.

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