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EDITORIAL

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Late-onset schizophrenia and the delusional disorders in old age

The *European Archives of Psychiatry and Clinical Neuroscience* dedicate special issues to important topics of current interest not yet sufficiently elucidated. With late- and very-late-onset functional psychoses we have chosen one such topic. The authors of all four contributions complain that many questions are still unanswered in their fields. The nosology is inadequate (Burns and Förstl). The European and the U.S. traditions differ and therefore also the main diagnostic systems, the ICD-10 and the DSM-IV (Riecher-Rössler et al.). The epidemiological prevalence data on schizophrenia and delusional disorders of old age show considerable variation (Henderson and Kay), which is understandable in view of the inconsistency in case definitions, the lack of instruments and the difficulty of access to target populations. The therapy of these conditions, although particularly beset with problems, has been accorded far too little interest (Eastham and Jeste).

An annual prevalence rate of approximately 6% for the elderly aged 70 years and over who present at least one symptom of a paranoid delusion or an auditory hallucination probably is a very close estimate, judged globally by rates of similar size in the general population of that age. It is based on a study, exemplary in its methodology, of 1045 persons aged 70 years and over from among the resident population of Australian municipalities and homes (Henderson et al. 1997). Utilization studies usually underestimate prevalence and incidence to a considerable extent and therefore tend to yield markedly lower values. This is especially true for paranoid psychoses and syndromes of old age (Häfner et al. 1997). The exceptionally high rates of administrative incidence for very-late-onset schizophrenia reported from a Dutch–British case-register study (van Os et al. 1995) – at 70/100 000 the annual first-admission rates for DSM-III schizophrenia were at their highest for women over 75 years – are indeed difficult to explain and must be viewed with caution.

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The epidemiology of depressive disorders of later life stands on a more solid basis. The topic is better, though not yet sufficiently researched. The rates vary less than those for schizophrenia and delusional disorders in old age.

Another aspect that has been given only little attention is brain morphology, e.g. the localisation and functional basis of depression, schizophrenic symptoms and hallucinations in general in old age. Burns and Förstl in their contribution have for a good reason refrained from discussing the nosology in terms of organic brain changes. Their analyses at the clinical level, based on their own psychopathologically related neuropathological findings, are consistent in showing what is already known from risk-factor research, namely the comorbidity of functional psychoses in old age with cognitive impairment and dementia. Against the backdrop of the diverging results of the studies on the topic, ranging from 10 to 70%, the authors consider 30% a plausible estimate. This value coincides – of course depending on the cutoff for cognitive impairment – well with a value of 25% obtained at the epidemiological level (Henderson et al. 1997).

Comparative analyses of neuropathological post-mortem findings as well as morphological and functional neuroimaging have started to reveal associations between structural brain changes and psychopathology in late life. Apart from the fact that in Lewy body dementia hallucinations and delusions are more prominent than in Alzheimer's disease, patients with typical late-onset psychotic disorders appear to have larger lateral ventricles than controls. Delusional disorders seem to involve an excess of minor temporal lobe damage, whereas in patients with visual hallucinations and misidentifications enlargement of the frontal horn up to the anterior horn of the right lateral ventricle is a frequent finding. Of course, these early results on the localisation of various schizophrenia-like symptoms in old age need replication. But a more or less intact cortex seems to be required to sustain delusional ideas and the more so, the more complex, systematic and stable the paranoid delusions in question are (Burns et al. 1990a,b). Simple delusions of theft and suspicion are the

commonest, presumably because they may also occur in advanced cognitive impairment. In severe dementia, however, they are not present. Severe dementia is generally inversely related to expressed functional psychopathology (Burns et al. 1990).

Research into the morphological and functional basis of functional late-onset psychoses brings us nearer to the development of plausible aetiological hypotheses, especially when personality and environmental factors are taken into account. There is a great deal that indicates that the conceptualisation of hallucinatory and delusional syndromes and functional psychoses in general with onset in late life as diagnostic categories is justifiable only in terms of clinical practice and consensus definitions. The underlying symptoms and syndromes are dimensional. As assumed by Ernst Kretschmer as early as 1921, abnormal personality traits, subclinical psychopathology and fully elaborated symptoms seem to involve dimensional patterns and transitions without any clearcut discontinuity up to the cases meeting the criteria for schizophrenia and depression in old age.

The same seems to be true for external risk factors: besides, moderately severe cognitive impairment and sensory impairment above all deficits in social relationships, such as subjective loneliness, never married, female without children and immigrant with language problems, are frequently encountered in persons with late paranoid or schizophrenic psychosis (Henderson and Kay). The vulnerability hypothesis, which postulates that functional psychotic syndromes in old age not only involve degenerative brain changes as underlying factors, but may also be triggered insidiously or suddenly by psychological and social precipitants, sounds plausible. But the functional associations between these levels are still largely unknown. Henderson and Kay stress the importance of late-onset psychoses in research directed towards uncovering the origins of psychotic symptoms in any age group. This is a reasonable assumption, since the sequence and nature of the degenerative brain processes and psychopathological changes in old age are better accessible and less masked by rapid development and, hence, easier to study than the pre- and perinatal changes and neurodevelopmental anomalies preceding early-onset psychoses.

In any case, the parallelism between the findings reported in this volume from late-onset research and the incomparable abundance of results from studies on early-onset schizophrenias is highly relevant: minor brain anomalies accentuated in the temporal and prefrontal regions and the accompanying functional processes of auditory hallucinations (McGuire et al. 1995; Frith 1995; Frith et al. 1995) accentuated in the temporal lobe suggest that a common organic basis of non-affective functional psychoses might just be conceivable in terms of localisation. The symptom pattern encountered in both early- and late-onset psychoses, i.e. delusions, hallucinations, thought disorders and negative symptoms, might be triggered primarily by a neurodevelopmental disorder at an early age and primarily by a neurodegenerative disorder involving the same brain regions at a later age. Particular differences in the symptomatology, e.g. the linear increase in paranoid and systematic delusions and the decrease in language disorders and disorders of self with increasing age, might to some extent result from normal personality development (Häfner et al. 1997). It seems that a considerable degree of cortical functioning is required for the production of these symptoms at any age. In more severe structural brain changes involving considerable loss of cortical functioning, they seem to become masked by severe cognitive impairment at any age: by mental handicap in childhood and dementia in old age.

From this perspective it is desirable to analyse comparable stages of illness, ideally first episodes, in functional psychoses and their brain morphological and functional basis across the total age range. This approach was recently called for by Werry et al. (1994) from another pespective, but for the same reason: to study the influences of age, developmental processes and specific risk factors in very-early-onset schizophrenias. The approach would enable the investigation of disease-independent developmental factors, of environmental factors differing by age and of developmental and degenerative processes of the brain in young and old age with respect to their contribution to similarities and differences in symptomatology.

As in the fields of epidemiology, psychopathology and brain research, surprisingly little research has been conducted on the crucial issues concerning the therapy of functional late-onset psychoses. Questions frequently encountered, such as interaction of drugs in multiple medications, the risk of adverse effects (Eastham and Jeste) and pharmacinetics are of a much greater relevance in later than in earlier life. There is a wealth of publications on antipsychotic therapy in young and middle adult life, but an absolute inadequacy of studies focusing on late-life psychosis (Eastham & Jeste), although increasing life expectancy is bound to lead to a growing number of late-onset disorders and to a substantial increase in older patients with psychosis of an earlier onset. These issues reflect the ethical dimension of the urgent need for research.

We hope that the comprehensive and critical discussion of the state of the art of the main aspects of late-onset schizophrenia and the delusional disorders of old age, the identification of the vast gaps in our knowledge and the enormous importance of filling them will stimulate new studies in this central field.

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