Schizophrenia is a severe form of mental illness affecting about 7 per thousand (12-month prevalence rate) of the adult population in the age group 15-35 years (Wittchen et al., 1992). A Dutch survey reported a lifetime prevalence of 4 per thousand and a 12-month prevalence of 2 per thousand in all age groups (Bijl et al., 1998). A recent review (Saha et al., 2005) analysed 188 studies from 46 countries and reported a point prevalence rate of 4.6 per thousand and a lifetime risk of 7.2.

It is estimated that schizophrenia accounts for 2.6% of the global burden of diseases (Murray & Lopez, 1996). The WHO World Health Report (2001) indicated that schizophrenia is the 8th leading cause of disability-adjusted life years worldwide in the age group 15-44. Active psychosis was ranked in the highest class of disability in another study (Harwood et al., 2004) that assessed the degree of dependency, i.e. the need for daily assistance from another person.

Epidemiological studies conducted with comparable methodology in different parts of the world showed that prevalence rates of schizophrenia may vary, being lower in developing countries compared with well developed ones (probably because of less demanding social pressure in the former that facilitates recovery) (Jablensky et al., 1992; Hopper & Wanderling, 2000). On the other hand the incidence of schizophrenia for a long time has been considered to be distributed more uniformly in various regions and settings. New insight in this topic has been provided recently by studies comparing incidence rates of schizophrenia in urban and rural areas.

Despite an enormous amount of published studies, the aetiology of schizophrenia remains unclear, with several models proposed to explain the aetiological mechanisms involved. It is now generally accepted that schizophrenia is a heritable illness, with a complex interplay of genetic and environmental factors. Genetics have been implicated in the development of schizophrenia, with evidence suggesting that certain genes may predispose individuals to the illness. Twin studies have shown that the concordance rate for schizophrenia is higher in monozygotic twins than in dizygotic twins, highlighting the role of genetic factors. Environmental factors, such as prenatal stress and early life trauma, have also been proposed to contribute to the development of schizophrenia. The precise mechanisms by which these factors interact to cause schizophrenia are still not fully understood.
is a complex disorder with a major genetic contribution to its aetiology, probably involving multiple genes and locus heterogeneity (Riley et al., 2003). Environmental factors may interact with genetic factors to produce considerable variation in its phenotypic expression, which includes a proportion of transmitted genotypes that remain unexpressed as clinical disorders (Gottesman & Bertelsen, 1989). Again comparative studies conducted in urban and rural settings may bring a new light to help clarify the role of biological and environmental factors in the incidence of this disorder.

In the current issue of Epidemiologia e Psichiatria Sociale we publish three Editorials that summarise current knowledge of the relationship between urbanicity and the risk of developing schizophrenia.

The first Editorial, written by McGrath and Scott (2006), reports the results of studies and reviews on this topic. The Authors show that robust and consistent evidence from epidemiological studies exists to explain the incidence of schizophrenia, whilst prevalence does not seem to be linked with an urban-rural gradient. Urban birth hides the real risk-factors and McGrath and Scott hope to see in the near future studies focused on candidate exposures instead of replications studies. What the Authors make clear is the urgency to gain a better understanding of the urban birth risk factors. In fact, whereas the relative risk for people who live in urban sites is modest (about two fold compared with rural birth), the attributable risk is high: 30% of the variance in the incidence of schizophrenia is explained by urbanicity. The number of people exposed to this risk is high and it will increase rapidly as urban populations are increasing in both developed and developing countries.

The second Editorial, by Pedersen and Mortensen (2006), discusses the potential pitfall of temporality as, to be considered causal, “urbanicity” has to be measured at birth and during upbringing. The Authors demonstrated in a previous study (Pedersen & Mortensen, 2001) that urbanicity is a risk factor when the exposure is from birth to at least the 15th birthday. In fact, only in these conditions, it is possible to disentangle the finding of a migration toward the city due to the illness from a causal-effect. Finally, the Editorial hypothesises that the urban-rural differences in the risk of schizophrenia are rooted entirely in the family; all the published studies potentially comply with this hypothesis.

In the third Editorial, Spauwen and van Os (2006) explore all the possible mechanisms and pathways that could be hidden beneath the concept of urbanicity. Apart from the well-known, already cited, factors that could be Nonostante l’enorme quantità di studi pubblicati, l’epidemiologia della malattia rimane poco chiara e diversi modelli sono stati proposti per spiegare i meccanismi eziologici. È oggi generalmente accettato che la schizofrenia è un disturbo complesso con una importante componente genetica, che probabilmente coinvolge diversi generi e con un’eterogeneità di loci (Riley et al., 2003). I fattori ambientali possono interagire con quelli genetici per produrre una notevole variazione nella sua espressione fenotipica. Questo fa supporre che una proporzione di genotipi trasmessi rimanga inespressa come disturbo clinico (Gottesman & Bertelsen, 1989). Di nuovo, gli studi comparativi, condotti in setting urbani e rurali, possono portare nuova luce, per chiarire il ruolo dei fattori biologici e sociali sull’incidenza di questo disturbo.

In questo numero di Epidemiologia e Psichiatria Sociale, pubblichiamo tre Editoriali che riassumono le conoscenze attuali sulla relazione tra urbanizzazione e rischio di sviluppare la schizofrenia.

Il primo Editoriale, scritto da McGrath e Scott (2006), riporta i risultati di studi e revisioni della letteratura su questo argomento. Gli Autori mostrano come esistano evidenze forti e replicabili, derivanti da studi epidemiologici, per l’incidenza, mentre per la prevalenza non ci sono dati a favore di un gradiente urbano-rurale. La nascita in ambiente urbano nasconde il reale fattore di rischio e McGrath e Scott si augurano di vedere, nel prossimo futuro, studi che si focalizzino su possibili fattori di rischio, invece che studi di replicazione. Ciò che gli Autori rendono chiaro è l’urgenza di comprendere i fattori legati alla nascita in ambiente urbano. Infatti, anche se il rischio relativo per le persone che vivono in siti urbani è modesto (circa due volte rispetto all’ambiente rurale), il rischio attribuibile è alto: il 30% della varianza nell’incidenza della schizofrenia è spiegato infatti dall’urbanizzazione. Il numero di persone esposte al rischio è alto ed è destinato a crescere molto rapidamente, dato che la popolazione urbana sta crescendo sia nei Paesi sviluppati sia in quelli in via di sviluppo.

Il secondo Editoriale, di Pedersen e Mortensen (2006), discute il potenziale ruolo “tranello” giocato dal fattore tempo, dato che, per essere considerata causale, l’urbanizzazione deve essere misurata alla nascita e durante la crescita. Gli Autori avevano dimostrato, in un loro precedente studio (Pedersen & Mortensen, 2001), che l’urbanizzazione è un fattore di rischio quando l’esposizione avviene nel periodo che intercorre dalla nascita al compimento del 15esimo compleanno. Infatti solo in queste condizioni, è possibile distinguere una migrazione verso la città dovuta alla malattia (la schizofrenia determina la migrazione) da un effetto causale (l’ambiente urbano
linked with urbanicity, this Editorial focuses on aspects of the wider social environment, such as social fragmentation at the community level, social isolation, social inequality and cognitive social capital. An interesting model, called psychosis proneness - psychosis persistence, is well explained in the Editorial; and it could provide an explanation of the interaction between genes and environment. The Authors review the most recent evidence on the role of genes in the expression of psychosis.

Since urbanization is consistently increasing in many areas of the planet, to clarify the role of the urban environment in influencing the number of new cases of schizophrenia that we will see in the future in different areas and regions is a particularly interesting and important topic. New, well focused studies are needed to attempt to identify the mechanisms that may explain the possible causal link between urbanicity and onset of schizophrenia. To be born and brought up in an urban environment is one of the most significant, and at the same time most mysterious, risk factor for developing schizophrenia later in life. We need to do our best to resolve this mystery, hoping that this will improve our understanding of this severe mental disorder.

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