Does urbanicity shift the population expression of psychosis?

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Abstract

Growing up in an urban area has been shown to be associated with an increased risk of psychotic disorder in later life. While it is commonly held that only a tiny fraction of exposed individuals will develop schizophrenia, recent evidence suggests that expression of psychosis in exposed individuals may be much more common, albeit at attenuated levels. Findings are based on a population sample of 2548 adolescents and young adults aged originally 14–24 years, and followed up over almost 5 years up to ages 17–28 years. Trained psychologists assessed all these subjects with the core psychosis sections on delusions and hallucinations of the Munich-Composite International Diagnostic Interview. Growing up in an urban area was associated with an increased risk of expression of psychosis in the adolescents and young adults (adjusted OR 1.31, 95% CI 1.03–1.66). The proxy environmental risk factor that urbanicity represents may shift a relatively large section of the adolescent population along a continuum of expression of psychosis. Other causal influences may be required to make the transition to schizophrenia in adult life.

Keywords: Schizophrenia; Adolescent; Cohort study; Urbanicity

1. Introduction

A very consistent, intriguing and key epidemiological finding concerns the association between exposure to an urban environment early in life and the occurrence of later psychotic illness (Lewis et al., 1992; Marcelis et al., 1998; Marcelis et al., 1999a; Mortensen et al., 1999; Torrey et al., 1997). Uncovering the nature of this relationship is likely to significantly further our knowledge on psychosis and the processes involved in its aetiology.

A plausible, albeit very general, explanation for the association is that environmental factors associated with urban life make individuals more vulnerable to the development of psychotic states (van Os, 2004).

Examples of environmental factors associated with urban life are health related environmental factors (high traffic densities, infections, and pollution), repeated exposure to stress

(neighbourhood noise, family and partner cohesion, neighbourhood cohesion), or health behaviour (smoking, drinking and substance misuse) (Eaton et al., 2000; Marcelis et al., 1999a; Pedersen and Mortensen, 2001b; Verheij et al., 1988). These environmental risk factors may make individuals more susceptible to developing psychosis, possibly by affecting early brain development and/or cognitive schemata. Given the relatively high attributable risks associated with urbanicity (Marcelis et al., 1998; Mortensen et al., 1999) it is crucially important to learn more on how this exposure impacts on population risk for psychosis. In particular, it is important to assess whether the urban exposure remains largely phenotypically silent with regard to the psychosis outcome in exposed individuals, or whether the exposed, as a group, exhibit a broad shift in clinical and non-clinical expression of psychosis (Fig. 1). In model 1A, the effect of urbanicity only impacts on emergence of schizophrenia, but not on the less severe, non-clinical expression of psychosis. This model would suggest that the psychosis phenotype in exposed individuals only emerges if combined with another rare cause for schizophrenia, i.e. urbanicity in itself does not induce expression of psychosis. In model 1B, however, there is an entire shift in the distribution of expression of psychosis. The exposed show a higher population level of psychosis than the non-exposed in a continuous fashion. This model would suggest that something in the urban environment facilitates a shift to higher population expression of psychosis, although other causal influences may be needed to reach a sufficiently severe level to attract a diagnosis of schizophrenia.

A recent study suggested that the increase in risk associated with the proxy environmental risk factor that „urbanicity“ is thought to represent does not only apply to narrowly defined psychotic disorder, but also to the much more prevalent class of non-clinical positive psychotic experiences in the general population (van Os et al., 2001, 2002), compatible with model 1B in the figure. However, this study was carried out in a sample aged up to age 65 years with assessment of lifetime prevalence of psychosis and current urban residence as the exposure. In the current study, we wished to replicate and extend this finding by using a sample of young people who were actually growing up in either an urban or a more rural environment. In this sample, we investigated to what degree the increase in risk for psychotic disorder associated with growing up in an urban environment is reflected in similar increases in psychotic and psychosislike symptoms. In addition, we addressed the issue of specificity and independence with regard to other symptom domains.

2. Subjects and methods

2.1. Sample

The Early Developmental Stages of Psychopathology (EDSP) study (Lieb et al., 2000) collected data on the prevalence, incidence, risk factors, comorbidity and course of mental disorders in a random representative population sample of adolescents and young adults (age range 14–24 years at baseline) in the Munich area (Germany). The overall design of the study is prospective (almost 5 years), consisting of a baseline survey ($n = 3021$), two follow-up surveys and a family supplement. Fourteen to 15-year-olds were sampled at twice the rate of persons 16–21 years of age, and 22- to 24- year-olds were sampled at half this rate. A complete and detailed description of design, sample, instruments, procedures and statistical methods of the EDSP is given elsewhere (Wittchen et al., 1998).

The baseline sample was drawn in 1994 from the government registries in Munich, Germany, of registrants expected to be 14–24 years of age at the time of the baseline interview in 1995. Details about the sampling and representativeness of the whole EDSP sample, along with its

sociodemographic characteristics, have been previously presented (Lieb et al., 2000; Wittchen et al., 1998). A total of 3021 interviews were completed at baseline (T0; response rate, 71%). The first follow-up study (T1) was conducted only for respondents aged 14–17 years at baseline, whereas the second follow-up study was conducted for all respondents. The current results are based on the second follow-up. From the 3021 respondents of the baseline study, a total of 2548 interviews were completed at the second follow-up (T2), which occurred at an average of 42 months after baseline (response rate, 84%).

In the EDSP family supplement at T1, direct diagnostic interviews were conducted with the parents of the younger cohort (the 14- to 17-year-olds at baseline) to obtain information about familial psychopathology (Lieb et al., 2000). Primarily the mothers were interviewed. Fathers were interviewed only if the mother was not available (deceased or not located). The sample of parents thus consisted of 1053 individuals (1026 mothers and 27 fathers). Non-response in parents was predominantly because of refusal to participate (12.9%), failure to contact parents (0.7%), and lack of time (0.5%).

For the current report, the risk set consisted of all subjects who had completed the second follow-up and whose demographic data was documented.

3. Instruments

Subjects were assessed with the computer-assisted version of the Munich-Composite International Diagnostic Interview (M-CIDI) (Wittchen, 1997), an updated version of the World Health Organization's Composite International Diagnostic Interview version 1.2 (WHO, 1990). Diagnostic findings, according to the explicit diagnostic criteria of the Diagnostic and Statistical Manual of Mental Disorders, 4th revision (DSMIV) (American Psychiatric Association, 1994), were obtained by using the M-CIDI diagnostic algorithms. The CIDI is designed for use by trained interviewers who are not clinicians and has high interrater reliability (Cottler et al., 1991; Wittchen et al., 1991) and high test-retest reliability (Wittchen, 1994). The assessment of psychosis with CIDI interviews by lay interviewers is not considered reliable (Anthony et al., 1985). Therefore, in the EDSP trained psychologists who were allowed to probe with follow-up clinical questions conducted the interviews. Most interviews were carried out in the homes of the respondents. At baseline, the lifetime version of the M-CIDI was used. At each of the follow-up assessments, the M-CIDI interval version was applied, which refers to the period of assessment from the last interview until the present. Data on the M-CIDI-G-section about psychosis were only collected at T2 assessment, at which time lifetime ratings of psychosis were made, yielding cumulative incidence data up to the respective age of respondents at T2 (range 17–28). Data on urban or rural residence was collected at baseline (T0).

In the adolescents and young adults, the ratings from the 15 M-CIDI core psychosis items on delusions (11 items) and hallucinations (4 items) were used to assess the presence of possibly psychotic syndromes (items G3-G5, G7-G14, G17, G18, G20, G21). These concern classic psychotic experiences involving, for example, persecution, thought interference and auditory hallucinations. Subjects were first asked to read a list with all the psychotic experiences and then interviewed about it by the psychologist (list and phrasing available on request).

All psychosis items could be rated in two ways: 0, no and 1, yes. The study was not powered for the study of rare psychotic disorders, but instead focussed on the presence of positive psychotic experiences. The psychosis outcome in this study was defined as at least one

positive rating on any of the 15 M-CIDI core psychosis items. More details on the methods have been described elsewhere (Lieb et al., 2000).

Urbanicity was defined as the German city of Munich (Stadt München) versus the surroundings of Munich (Landkreis München). The population density of the surroundings was 553 persons per square mile and that of the city was 4061 persons per square mile.

4. Statistical analysis

All standard errors and test statistics were estimated using the software package STATA, version 7 (Stata- Corp, 2001). Logistic regression analysis was used to examine the association between the cumulative incidence of positive psychotic symptoms in the adolescents and young adults on the one hand and urbanicity on the other.

Associations were expressed as odds ratios (OR) with their 95% confidence intervals (95% CIs). We adjusted, guided by previous literature (van Os et al., 2001, 2002), for the following a priori chosen confounders: gender, SES (Socio-Economic Status: a combination of social status and financial status), any drug use, family history of psychosis and a dichotomous variable indicating any DSM-IV psychiatric diagnosis in the parent (substance abuse or dependence, mood disorder, anxiety disorder, obsessive-compulsive disorder, somatoform/conversion disorder and eating disorder). This last confounder, any DSM-IV psychiatric diagnosis in the parent, could only be used in the younger subsample, because only the parents of the younger respondents had been interviewed. In order to examine whether any association between urbanicity and psychosis was independent from presence of other psychopathology in the young adults, analyses were adjusted for depression and mania, defined as the total score of the core CIDI items on depression and mania (Krabbendam et al., 2004), and any other DSM-IV diagnosis in the young adult.

Finally, in order to see whether the findings were specific for psychosis, the analyses were repeated using depression and mania as dependent variables. For the purpose of these analyses, the depression and mania outcomes were defined exclusively as at least one positive rating on any of the CIDI depression and mania items in the absence of psychotic symptoms.

5. Results

The analysis is based on a total of 2548 adolescents and young adults (50.9% males) with a mean age of 21.7 years (SD, 3.4) who had completed the final follow-up investigation. Four-hundred-and-forty-one (17.3%) adolescents and young adults had ever had at least one psychotic experience. Of the 441 subjects with psychotic experiences, 258 had had one experience, 98 had had two and 85 had had three or more experiences. Of the 2548 adolescents and young adults, almost 1800 (70.5%) were living in an urban area, while 750 (29.5%) were not.

Logistic regression of the unadjusted data indicated that living in an urban area significantly increased the risk for reporting at least one psychotic experience (urban: 18.5%, rural: 14.6%; OR 1.32, 95% CI 1.04–1.67). These effects remained after controlling for gender, SES, any drug use, family history of psychosis and any psychiatric diagnosis in the adolescent (OR 1.31, 95% CI 1.03–1.66). Adjustment for the total scores of depression and mania reduced but not nullified the risk (OR 1.23, 95% CI 0.96–1.58). There was a small and non-significant

effect of urbanicity on the occurrence of depressive symptoms, exclusively defined (OR 1.08, 95% CI 0.89– 1.33). The association between urbanicity and mania, exclusively defined, was stronger and in the same order of what was found for the psychosis outcome, although statistically non-significant (OR 1.24, 95% CI 0.97– 1.58). In the subsample where parental interviews had been conducted, the effect of urbanicity was similar albeit statistically non-significant due to the smaller sample size (OR 1.32, 95% CI: 95% CI 0.92, 1.91) and adjustment for any diagnosis in the parent only marginally changed the parameter (OR 1.29, 95% CI: 0.90, 1.87).

6. Discussion

The results indicated that adolescents and young adults living in an urban are at increased risk for psychotic symptoms. Independent of the association with psychosis, urbanicity also increased the risk for symptoms of mania, although the effect was not statistically significant, suggesting that the results may not be specific for psychosis. This finding is in agreement with the suggestion that there may be a degree of aetiological continuity between psychotic and non-psychotic affective disorder on the one hand, in this case especially mania, and non-affective psychosis on the other (Van Os et al., 1999). The effect size for mania, however, has consistently been shown to be lower than for psychosis (Marcelis et al., 1998; Marcelis et al., 1999b; Pedersen and Mortensen, 2001a).

The psychosis outcome in this study was defined as at least one positive rating on any of the 15 M-CIDI core psychosis items. A post-hoc analysis with a narrower outcome with a lower prevalence (at least two positive ratings) was carried out in order to exclude the possibility of spurious results due to false-positive misclassification. The analysis with this variable showed a similar effect, although statistically non-significant (OR 1.27, 95% CI 0.90–1.80).

Our results agree with Pedersen and Mortensen (2001a) that continuous, or repeated exposures during upbringing that occur more frequently in urbanized areas form the basis for the association between urbanization and schizophrenia risk. We have previously found that the prevalence of abnormal mental states that facilitate development to overt psychotic illness increases with level of urbanization (van Os et al., 2001). This study was also conducted with a normal population sample, showing that community level of psychotic and psychosis-like symptoms may be inextricably linked to the prevalence of psychotic disorder (van Os et al., 2001). Our present study replicates this in a young population growing up in urban and rural environments and supports the suggestion that nonclinical and clinical psychotic experiences in adolescence are expressed along a continuum of severity. In addition, it demonstrates that exposed individuals as a group display higher levels of psychosis, suggesting that urbanicity induces a broad shift along the non-clinical expression of psychosis. This is in agreement conceptually with model 1B of Fig. 1. However, exposure to other causal factors may be required to subsequently make the shift to levels of psychosis that are compatible with a diagnosis of schizophrenia or other psychotic disorders.

The possible etiological factors that might explain our findings remain unknown. Pedersen (2004) suggested that the level of air pollution from traffic at the residence of birth might explain some of the urban-rural differences in schizophrenia risk. Social isolation in urban areas was found to be associated with the incidence of schizophrenia (van Os et al., 2001). A related possible mechanism of risk may reside in differences in neighbourhood social capital. Kawachi and colleagues have summarized the work of Putnam and Coleman and have „defined“ social capital as „those features of social organisations – such as networks of

secondary associations, high levels of interpersonal trust and norms of mutual aid and reciprocity – which act as resources for individuals and facilitate collective action“ (Bourdieu, 1986; Coleman, 1990; Kawachi et al., 1997b; Kawachi et al., 1999; Putnam, 1993).

It has been embraced as a possible explanation for differences in health that are found between places or between groups of people (Baum, 1999; Kawachi et al., 1997a). Exposure to low levels of social capital may play a role in the incidence and prevalence of mental illness, including psychosis (McKenzie et al., 2002). Drukker et al. (2003) studied health related quality of life and neighbourhood social capital in children growing up in the city. Lower levels of social capital were associated with poorer general and mental health in children, independent of possible individual-level confounders (Drukker et al., 2003), suggesting that social capital may play a role in shaping the risk for later mental health outcomes.

In this study, psychotic symptoms rated by CIDI interview were considered continuous with clinical psychotic disorder. This approach is justified by many studies (Johns and van Os, 2001) showing continuity between the clinical and the subclinical domain of psychosis including longitudinal continuity (Poulton et al., 2000; Chapman et al., 1994; Kwapil et al., 1997), neuropsychological continuity (Chen et al., 1988), neuroradiological continuity (Buchsbaum et al., 1997), psychopathological continuity (Hanssen et al., 2003a,b; Gruzelier, 1996), familial continuity (Kendler et al., 1995), and epidemiological continuity (van Os, 2000, 2001; Peters et al., 1999).

A possible limitation of the study is that urban or rural residence assessed at baseline was employed as the exposure variable and that it is not known whether place of residence was the same as place of growing up. However, given the fact that this was a sample of individuals who were either still growing up or who had just reached adulthood, the rate of exposure misclassification is likely to be low and misclassification would have served to reduce rather than to increase the reported association. The fact that the difference between the urban and rural population densities was almost eightfold attests to the validity of the exposure, although it did not allow for examination of dose–response relationships.

Selective migration of predisposed parents or predisposed children has been invoked to explain the higher rates of psychosis in urban areas. Although it may play a role, most of the association between urbanicity and psychosis is not caused by selection. This was demonstrated by the Danish studies (Pedersen 2001) and the Dutch studies (Marcelis et al., 1998; Marcelis et al., 1999a; Marcelis et al., 1999b) where it was shown that children who moved from urban to rural areas mitigated their risk for later psychosis and vice versa, a finding which is incompatible with an effect of selective migration where children’s risk would be similar regardless of the degree of urban exposure (Cederlof et al., 1977).

Another limitation of this work concerns the use of the CIDI to assess psychosis (Anthony et al., 1985). However, the use of face-to-face interviewing by psychologists can be expected to yield a much better result than a self-report questionnaire. Furthermore, the psychologists were allowed to probe with follow-up clinical questions so that the respondents’ answers cannot be taken to represent self-report, as would be the case in the event of lay interviewer assessments.

Acknowledgements

The EDSP is funded by the German Ministry of Research and Education (BMBF) as part of the Addiction Research Initiative (01EB94056).

It is assumed that subtle, non-clinical psychosis-like phenomena in the general population are indicators of an underlying continuously distributed psychosis phenotype. In figure 1(a), the only difference between exposed and non-exposed is that a tiny fraction of the exposed population exceeds, compared to the non-exposed, the threshold of clinical disorder. This suggests that the urban exposure is required to combine with a rare causal factor in order to cause the rare disorder schizophrenia. In figure 1(b), however, the exposed, as a group, exhibit a broad shift in expression of psychosis, suggesting that something in the urban environment induces expression of psychosis, although other causal influences may be needed to reach a sufficiently severe level to attract a diagnosis of schizophrenia.

Fig. 1. Impact of urban exposure on population risk.

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