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ORIGINAL PAPER

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Does the urban environment independently increase the risk for both negative and positive features of psychosis?

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Abstract *Background* Psychotic disorders are more common in urban environments. It is not known whether the increase in risk applies to both the positive and negative dimensions of psychosis. *Methods* In a random general population sample of 7076, measures of positive and negative symptoms of psychosis were constructed using Composite International Diagnostic Interview (CIDI) data. Three CIDI observed items of flat affect, retarded speech and retarded movement served as indicators of a negative symptom variable. *Results* Both negative and positive symptoms of psychosis were, independent of each other, associated with a five-level measure of population density of place of residence (adjusted OR negative symptoms: 1.42, 95 % CI: 1.18, 1.71; adjusted OR positive symptoms: 1.19, 95 % CI: 1.13, 1.24). These associations remained after exclusion of vulnerable individuals with any lifetime psychiatric disorder (n = 2910), any lifetime psychiatric treatment (n = 1352) and history of psychosis in the parents (n = 142). *Conclusions* An environmental risk factor associated with urbanicity may act in early life to non-specifically influence risk for both negative and positive experience of psychosis, regardless of whether a formal psychiatric disorder is diagnosable.

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Introduction

There is a well-established association between urban birth and upbringing on the one hand and psychotic disorder on the other [1–4]. Recent research indicates 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 [5]. As the psychosis phenotype is considered to vary along several symptom dimensions, including positive, negative, depressive, disorganisation and manic dimensions [6–16], the question arises whether the effect of urbanicity on the positive dimension of psychosis can also independently be demonstrated for other symptom dimensions. If the effect of urbanicity is restricted to one symptom dimension, the conclusion would be that the proxy environmental risk factor is dimension-specific and that specific causes contribute to specific psychopathological outcomes in psychosis. If, on the other hand, positive and other symptom dimensions share the same urban risk factor, the possible conclusion would be that a single risk factor gives rise to different psychopathological outcomes in psychosis. In the current investigation, we wished to examine whether the increase in risk brought about by urbanicity could be shown for the negative symptom dimension independently of the positive dimension.

Subjects and methods

Subjects

The Netherlands Mental Health Survey and Incidence Study (NEMESIS) is a prospective study with three measurement points (hereafter: T1, T2 and T3) over a period of 3 years [17–19]. The current report is

based on the lifetime prevalence of psychosis assessed at T1 (n = 7076 interviewed). For some of the analyses, first-degree family history data assessed at T2 (n = 5618 interviewed), and data on CIDI DSM-III-R diagnosis of psychotic disorder at T3 (n = 4848 interviewed) were also used. A multistage, stratified, random sampling procedure was used to first select 90 municipalities, then a sample of private households, and finally a Dutch-speaking individual aged 18–64 years within each household. Selected households were sent an introductory letter by the Minister of Health inviting them to participate. A total of 7076 individuals provided informed consent and were interviewed at baseline, representing a response rate of 69.7 %. Nearly 44 % of non-responders agreed to fill in a postal questionnaire, including a General Health Questionnaire [20], and were found to have the same mean GHQ score (responders: 1.19; non-responders: 1.16). Non-response was not associated with level of urbanicity [17, 18]. The sample was found to be representative of the Dutch population in terms of gender, marital status and level of urbanisation [18], with the exception of a slight underrepresentation of individuals in the age group 18–24 years. As this was a study of relative rather than absolute risk, no post-stratification weightings were applied to the data.

Instruments

Subjects were interviewed at home. The Composite International Diagnostic Interview (CIDI) version 1.1 [21–23] was used, yielding DSM-III-R diagnoses. The CIDI was designed for trained interviewers who are not clinicians and has been found to have high inter-rater reliability [24, 25], and high test-retest reliability [26–28]. Ninety interviewers experienced in systematic data collection collected the data, having received a 3-day training course in recruiting and interviewing, followed by a 4-day course at the WHO-CIDI training centre in Amsterdam. Extensive monitoring and quality checks took place throughout the entire data collection period [18].

Psychosis ratings

Lifetime ratings from the 17 CIDI core psychosis sections on delusions (13 items) and hallucinations (4 items) were used (items G1–G13, G15, G16, G20, G21). These concern classic psychotic symptoms involving, for example, persecution, thought interference, auditory hallucinations and passivity phenomena. All these items can be rated in six ways: "1"– no symptom, "2"– symptom present but not clinically relevant (not bothered by it and not seeking help for it), "3"– symptom result of ingestion of drugs, "4"– symptom result of somatic disease, "5"– true psychiatric symptom, "6"– symptom may not really be a symptom because there appears to be some plausible explanation for it. Because psychotic symptoms are difficult to diagnose in a structured interview [29–31], clinical re-interviews were conducted over the telephone by an experienced trainee psychiatrist for all individuals who had at least one rating of "5" or "6", using questions from the Structured Clinical Interview for DSM-III-R (SCID), an instrument with proven reliability and validity in diagnosing schizophrenia [32]. Out of a total of 479 individuals who were eligible for a clinical re-interview over the telephone, 226 (47.2 %) were actually interviewed. CIDI ratings of symptoms were corrected on the basis of these clinical interviews. The NEMESIS lifetime DSM-III-R diagnoses of psychotic disorder are based on the data from the clinical re-interviews as described above. Psychotic disorder was defined as any DSM-III-R affective or non-affective psychotic diagnosis (n = 107, 1.5 % of the total sample of 7076).

Positive psychotic symptom rating

In the baseline sample of 7076, the prevalences of the different CIDI ratings on the 17 psychosis items were: any CIDI rating of "2": n = 915 (12.9 %); any CIDI rating of "3" or "4": n = 39 (0.6 %); any CIDI rating of "5": n = 295 (4.2 %); and any CIDI rating of "6": n = 285 (4 %). Positive psychotic symptom was broadly defined as any CIDI rating of 2, 3, 4, 5 or 6 on any of the 17 CIDI core psychosis items. In a previous study, it was shown that all these different ratings on the CIDI psychosis items were strongly associated with each other, including the clinical re-interview ratings of psychotic symptom (i. e. a rating of "5" on any of the CIDI psychosis items). In addition, the different ratings independently showed a similar pattern of associations with known risk factors for psychosis [5, 33]. As they, therefore, appear to reflect the same underlying latent dimension of "positive psychosis", they were joined together into a single broad dichotomous rating of "positive psychotic symptoms" for the purpose of the current study (prevalence: n = 1237, 17.5 %).

Negative psychotic symptom rating

Ratings of negative symptoms in the sample of 7076 were based on the three CIDI ratings of observed negative symptoms. These were: i) blunted affect (n = 24, 0.3 %), ii) retardation of movement (n = 13, 0.2 %) and iii) retardation of speech (n = 49, 0.7 %). These ratings were strongly associated with each other (blunted affect and retarded speech: OR = 30.71, 95 % CI: 10.09, 93.45; blunted affect and retarded movement: OR = 99.21, 95 % CI: 25.48, 386.31; retarded speech and retarded movement: OR = 192.33, 95 % CI: 62.01, 596.49). A dichotomous rating of "negative symptoms" was constructed with score "present" if any of these three items had been rated by the trained lay interviewer (sample prevalence: n = 74, 1.1 %). In order to validate this rating, the following analyses were conducted. The rating of negative symptoms was strongly associated with psychotic disorder (any DSM-III-R affective or non-affective psychotic disorder as described above; OR = 7.18, 95 % CI: 3.22, 16.04). This association remained if all individuals who had been eligible for clinical re-interview but who had not been seen were excluded, leaving only the psychotic disorder cases whose diagnosis had been established on the basis of clinical re-interview (OR = 8.88, 95 % CI: 3.45, 22.86). This association was attenuated, but still large and statistically significant after adjustment for presence of positive psychotic symptoms (any CIDI rating of 2, 3, 4, 5 or 6 on any of the 17 CIDI core psychosis items as described above; OR = 3.01, 95 % CI: 1.28, 7.10) and adjustment for presence of lifetime DSM-III-R depressive disorder and bipolar disorder (OR = 4.09, 95 % CI: 1.73, 9.66). It also remained large and significant if all individuals who had a lifetime history of psychiatric treatment (including medications n = 1352, 19.1 %) were excluded (OR = 11.67, 95 % CI: 1.51, 90.46).

At the T2 interview (n = 5618), probands were asked separately for each parent whether he or she had ever had delusions or hallucinations (n = 142, 2.5 %) or a depressive episode (n = 776, 14 %). The negative symptom rating was strongly associated with a parental history of delusions of hallucinations (OR = 3.48, 95 % CI: 1.23, 9.83) but not a parental history of depression (OR = 1.35, 95 % CI: 0.66, 2.80).

At the T3 interview (n = 4848), 11 individuals had a CIDI DSM-III-R diagnosis of affective or non-affective psychosis on the basis of a CIDI interview followed by re-interview by clinicians for those with CIDI-evidence of psychosis. Negative symptoms at T1 were strongly associated with presence of DSM-III-R psychotic disorder at T3 three years later (OR = 14.81, 95 % CI: 1.84, 119.09), also after adjustment for positive psychotic symptoms at T1 (OR = 9.68, 95 % CI: 1.16, 80.87).

Level of urbanicity

Five levels of urbanisation were defined, following the standard classification of urbanisation of place of residence according to the Dutch Central Bureau of Statistics. These are based on the density of addresses per km^2 in an area and are classified as < 500, 500–999, 1000–1499, 1500–2499 and 2500. This density is calculated by assessing, for each address, the density of addresses in a circle of 1 km around that address. The density of addresses in an area is then calculated as the mean address density of all the addresses in that area [34].

Data analyses

The prevalence of negative symptoms and positive symptoms as defined above was examined in relation to level of urbanicity of place of residence, adjusted for the *a priori* selected possible confounding effects of age in years, sex, single marital status, level of education (4 levels), ethnic group (coded "0" if subject and both parents were Dutch-born and "1" for other) and unemployment. Associations were expressed as odds ratios (OR) from the logistic regression procedure in STATA [35]. In order to assess whether any effect of urbanicity on negative symptoms could be explained by confounding by positive psychotic symptoms, lifetime positive psychotic symptoms were adjusted and restricted for. Similarly, in order to assess whether any effect of urbanicity on negative symptoms could be explained by parental drift, i. e. parents with psychosis vulnerability drifting towards urban areas, or personal drift by vulnerable individuals with psychosis or any other psychiatric disorder, additional adjustment and restriction was made for: i) history of delusions or hallucinations in the mother or the father as reported by the subject, ii) lifetime presence of any psychotic disorder (n = 107), iii) lifetime presence of any psychiatric disorder (n = 2910) and lifetime psychiatric treatment (n = 1352). Exclusion of individuals with a lifetime history of psychiatric treatment (including medication) also served to assess whether any effect of urbanicity on negative symptoms could be explained by negative symptoms secondary to (possibly) more frequent use of antipsychotic medication in urban areas. Exclusion of individuals with other psychiatric disorders also served to exclude confounding of negative symptoms by, for example, depressive symptomatology.

Results

The rating of negative symptoms was strongly associated with the rating of positive symptoms (OR = 3.89, 95 % CI: 2.45, 6.18). Negative symptoms were more prevalent in progressively more urbanised areas (OR linear trend: 1.48, 95 % CI: 1.24, 1.78; Table1). This effect could not be explained by the association between negative and positive symptoms, as it remained after adjustment for positive symptoms (OR = 1.42, 95 % CI: 1.18, 1.71). Similarly, the effect of urbanicity on positive symptoms (OR = 1.19, 95 % CI: 1.14, 1.25) remained after adjustment for negative symptoms (OR = 1.19, 95 % CI: 1.13, 1.24). The effect on positive symptoms also remained if individuals who had been eligible for clinical re-interview after the lay-interviewer CIDI interview but who had not been seen were excluded from the analyses (OR = 1.20, 95 % CI: 1.14, 1.26). In addition, the effect of urbanicity on negative symptoms remained when the 107 individuals with any DSM-III-R psychotic disorder were excluded from the analyses (OR = 1.37, 95 % CI: 1.14, 1.65) and when all 1237 individuals with any lifetime evidence of positive psychotic symptoms were excluded from the analysis (OR = 1.54, 95 % CI: 1.20, 1.97). The association also remained after adjustment for age, sex, single marital status, educational level, unemployment and ethnic group, and after additional adjustment for parental history of psychosis, any lifetime psychiatric treatment and any lifetime psychiatric diagnosis (Table 1). Similarly, the association between urbanicity and negative symptoms remained after exclusion of all individuals with a parental history of psychosis, a lifetime history of any psychiatric diagnosis and a lifetime history of any psychiatric treatment (OR = 2.08, 95 % CI: 1.06, 4.08). The same applied to the association between urbanicity and positive symptoms (OR after exclusion of the three groups = 1.17, 95 % CI: 1.07, 1.28).

Discussion

The results indicate that the urban environment increases the risk independently for both positive and negative features of psychosis, regardless of whether a formal psychiatric disorder is diagnosable. Neither drift of vulnerable individuals nor drift of vulnerable parents towards urban areas was a likely explanation for the findings, nor was confounding by demographic factors, psychiatric diagnoses including depression, psychotropic medication or the association between negative and positive dimensions of psychosis.

These findings suggest that the presumed environmental risk factor that is captured by measures of urbanicity non-specifically increases the risk of both positive and negative dimensions of psychosis. It may do so

Table 1 Effects of population density on negative and positive psychotic symptoms

Population density per km^2	N interviewed	Prevalence negative symptoms n (%)	Odds ratio (95 % CI)	Prevalence positive symptoms n (%)	Odds ratio (95 % CI)
< 500	1185	4 (0.3)	1[a]	163 (13.76)	1[a]
500–999	1610	10 (0.6)	1.85 (0.58, 5.91)	223 (13.85)	1.01 (0.81, 1.25)
1000–1499	1541	18 (1.2)	3.50 (1.18, 10.37)	262 (17.00)	1.28 (1.04, 1.59)
1500–2499	1497	16 (1.1)	3.21 (1.07, 9.62)	303 (20.24)	1.59 (1.29, 1.96)
≥ 2500	1242	26 (2.1)	6.30 (2.19, 18.11)	286 (23.03)	1.88 (1.52, 2.32)
OR linear trend[b]			1.48 (1.24, 1.78)		1.19 (1.14, 1.25)
Adjusted for other dimension[c]			1.42 (1.18, 1.71)		1.19 (1.13, 1.24)
+ adjusted for demographics[d]			1.36 (1.13, 1.64)		1.16 (1.10, 1.22)
+ adjusted for family history and any lifetime diagnosis and lifetime psychiatric treatment			1.32 (1.03, 1.68)		1.13 (1.06, 1.19)

[a] Reference category
[b] The summary increase in risk with one unit change in population density
[c] Negative adjusted for positive and positive adjusted for negative
[d] Age, sex, ethnic group, single marital status, unemployment, educational level; "+" indicates additional adjustment on top of confounders in previous model

either alone or in interaction with genetic factors. Recent research suggests that the risk factor associated with urbanicity acts early in life, up to age 15 years [36, 37]. Therefore, one explanation for the findings is that the environmental risk factor acts early in life, giving rise to a general vulnerability that may later in life express itself as positive or negative features of psychosis, depending on the presence of other intervening factors that co-participate in the causation of psychotic symptoms.

An interesting finding, commented on before [5], was that the urban environment increased the risk of negative and positive features of psychosis regardless of the presence of formal psychiatric disorder. Thus, even when all the individuals with any lifetime DSM-III-R psychiatric disorder were excluded from the sample (n = 2910, 41.1 %), the association with both negative and positive features of psychosis remained. These findings, therefore, strongly suggest a dimensional view, both within psychosis as an entity consisting of several symptom dimensions, as across the population, psychosis being a continuum of experiences with a distribution in the general population.

One explanation for the findings is that symptomatic probands or symptomatic parents of probands could have "drifted" to urban areas. However, in a previous study in the Netherlands, we found that there was a high degree of lifetime stability of urban exposure status (around 75 % of individuals living in urbanised areas had also been born there), indicating that current exposure is likely to reflect stable lifetime exposure in the majority of cases [36]. In addition, the association with urbanicity remained after exclusion of individuals with any psychiatric disorder or any psychiatric treatment and probands whose parents displayed evidence of psychosis. These findings, therefore, suggest that it is very unlikely that the findings can be explained solely by a process of urban drift of symptomatic individuals or their symptomatic parents. Therefore, the risk associated with an urban environment may reflect social or biological factors that increase the risk for psychosis. The fact that the increase in risk is not specific for psychosis, but is also seen, albeit to a lesser extent, in affective disorders and anxiety disorders [17, 36], suggests a mechanism common to psychiatric disturbance in general, such as psychosocial stress and/or social isolation. The less parsimonious explanation would be that different risk factors in the urban environment have different effects on specific dimensions of psychopathology.

These findings should be viewed in the context of several limitations. Firstly, the measures of negative symptoms consisted of only three observations by lay interviewers, which arguably can be considered a weak measure of this symptom dimension. Although the interviewers had been trained, a degree of misclassification will have been introduced as well as a degree of incompleteness, given that negative symptoms include a much wider range of features than assessed in the current study. However, there is no reason to suspect that the degree of misclassification or incompleteness would have been different in progressively more urbanised areas. Furthermore, the measures of negative symptoms, limited as they were, nonetheless showed discriminant and predictive validity. Secondly, the measure of negative symptoms had a low prevalence, resulting in low statistical power. However, the lack of statistical power was not an issue, given that the effect size of negative symptoms was large enough for the analyses to be conclusive. Thirdly, as far as the positive symptom dimension is concerned, bias could have been introduced by differential rating of psychosis due to incomplete telephone clinical re-interview rates at baseline. However, excluding the individuals who were eligible for clinical re-interview but who had not been contacted, thus leaving only those who had been rated by clinicians, did not affect the results. A similar potential bias did not apply to negative symptoms, as these solely concerned lay-interviewer observed symptoms that could not be reassessed during telephone interview.

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