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Psychiatry Research

journal homepage: www.elsevier.com/locate/psychres

Association of adoptive child's thought disorders and schizophrenia spectrum disorders with their genetic liability for schizophrenia spectrum disorders, season of birth and parental Communication Deviance



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ARTICLE INFO

Article history:

Received 28 February 2014

Received in revised form

31 July 2014

Accepted 22 December 2014

Available online 31 December 2014

Keywords:

Adoption

CD

Endophenotype

Genetic Predisposition

Gene–environment interaction

TDI

Winter/spring birth

ABSTRACT

Joint effects of genotype and the environment have turned out to be significant in the development of psychotic disorders. The purpose of the present study was to assess the association of an adoptive child's thought and schizophrenia spectrum disorders with genetic and environmental risk indicators and their interactions. A subgroup of the total sample used in the Finnish Adoptive Family Study was considered in the present study. The subjects were 125 adoptees at a high ($n=53$) or low ($n=72$) genetic risk of schizophrenia spectrum disorders and their adoptive parents. The risk factors evaluated were the adoptive child's genetic risk for schizophrenia spectrum disorders, winter or spring birth and parental Communication Deviance (CD). Thought disorders in the adoptees were assessed using the Thought Disorder Index and diagnoses were made according to DSM-III-R criteria. The adoptive child's Thought Disorder Index was only associated with parental Communication Deviance. The adoptive child's heightened genetic risk or winter or spring birth or parental CD or their interactions did not predict the adoptee's schizophrenia spectrum disorder. The results suggest that studies taking several risk indicators and their interactions into account may change views on the mutual significance of well-known risk factors.

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1. Introduction

Despite extensive research for over a century, the origin of schizophrenia has remained an unresolved question. It has long been known that schizophrenia, as well as other severe psychiatric disorders, runs in families, and the heritability of the liability for schizophrenia is estimated to be as high as 80% (Tandon et al., 2008). These estimates of heritability have been criticised as being too high and underestimating the role of the environment by ignoring gene–environment interactions and also the effect of genes that operate by altering the environment (Brown, 2011). The search for the cause of schizophrenia has also been complicated for the heterogeneous phenotype of the disease and that is why major research efforts have been focused on exploring schizophrenia endophenotypes, which could reflect schizophrenia susceptibility better than a diagnosis as such (Allen et al., 2009). Thought disorders (TD) are one of the proposed endophenotypes

for schizophrenia (Gooding et al., 2012; Gooding et al., 2013) or even for a wider spectrum of psychotic disorders (Levy et al., 2010).

Among biological risk indicators of schizophrenia, the season of birth is one of the most intensively studied. In a comprehensive review (Torrey et al., 1997), it was concluded that the rate of the increase of winter-spring births in schizophrenia and also in bipolar and schizoaffective disorder (from November to April, the peak from January to February) is 5–10% compared to the expected number of births during the season. However, the increase in winter/spring births in schizophrenia has only been detected in the Northern Hemisphere (McGrath and Welham, 1999; Davies et al., 2003). Studies on the association between the season of birth and other psychiatric diagnoses, apart from schizophrenia endophenotypes, are rare. They suggest that an excess of winter-spring births similar to that noted in the case of schizophrenia may be present in relation to affective psychoses (Castrogiovanni et al., 1998; Hultman et al., 1999). Several studies also show that schizotypy, an indicator of vulnerability to schizophrenia, is associated with winter or early spring births (Lahti et al., 2009; Hori et al., 2011; Bolinskey et al., 2013), but such an

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association is not observed by all studies (Cohen and Najolia, 2011). Only one study has explored the association between psychotic-like experiences and the season of birth (Tochigi et al., 2013). In the study in question, an association was observed between a significant excess in winter births (from November to March) and the prevalence of psychotic-like experiences.

Even though the absolute increase in risk related to winter–spring birth is small, it is not insignificant, since a considerable proportion of the population is exposed to risk. For this reason the population attributable fraction (PAF) according to season of birth is estimated to be as high as 10% (Torrey et al., 1997; Mortensen et al., 1999), while PAF according to the strongest risk factor known, namely, family history, is 5.5%. It has been suggested that the season of birth effect would be present only in those population groups where there is no genetic liability to psychotic disorders. However, the results of the studies on this issue are controversial and inconclusive (Torrey et al., 1997; Mortensen et al., 1999; Kinney et al., 2000). It is not known how the season of birth effect is mediated, but it is proposed that it represents a proxy for certain meteorological factors, prenatal infections, nutritional deficiencies, the effect of external materials (e.g. heavy metals), factors on the paternal side, maternal hormones and seasonal variation of procreation (Tochigi et al., 2004). Accumulating evidence supports the hypotheses that the season of birth effect is mediated by either prenatal infections or vitamin D deficiency, the latter of which could also explain the variability in the risk of schizophrenia according to place of birth and migrant status (McGrath et al., 2004; Kinney et al., 2009). Results of both epidemiological and animal experimental research support the hypothesis on the association between developmental vitamin D deficiency and increased risk for schizophrenia (McGrath et al., 2010). In Finland vitamin D depletion is common on account of long periods without a sufficient amount of sunshine hours. Vitamin D supplementation for pregnant women was not recommended until 1997 (Hasunen et al., 1997).

One of the known psychosocial risk indicators for thought disorders and schizophrenia spectrum disorders is frequent Communication Deviance (CD) in the rearing parents (Wynne and Singer, 1963a, 1963b; Singer and Wynne, 1965a, 1965b). Communication Deviance is a scale for measuring the degree to which family members are unable to share and maintain the focus of attention during communication (Singer and Wynne, 1966). Frequent CD makes speech difficult to follow and meanings are not consensually or visually validated (Singer et al., 1978). It is hypothesised that frequent CD in the parents' speech may impair the growing child's ability to derive and convey consensually understood meanings as a core cognitive developmental task (Wahlberg et al., 1997; Wahlberg et al., 2000). Frequent parental Communication Deviance has been shown to be connected with thought disorders and schizophrenia spectrum disorders in the offspring (Wynne et al., 1977; Sass et al., 1984; Goldstein, 1985; Wahlberg et al., 1997, 2000; de Sousa et al., 2014; Roisko et al., 2014).

Studies on isolated biological or psychosocial risk factors have not succeeded in identifying any single risk factor, either necessary or sufficient, for the development of schizophrenia. On the other hand, there is evidence for the significance of joint effects of genotype and the environment in psychotic disorders (van Os et al., 2008). The joint effects are supposed to be mainly synergistic coparticipation, where the effect of one factor is conditional to the other. Evidence supports the hypothesis that the mechanism of interaction in most cases is genetic control of sensitivity to the environment (van Os et al., 2008). Also other types of joint effects, like additive effects on genotype and environment, genetic control of exposure to the environment and epigenetic mechanisms, play a role in the aetiology of psychotic disorders (Kendler and Eaves,

1986; van Os et al., 2008). Several adoption studies provide evidence for the interaction of inherited vulnerability and risk factors in the psychosocial environment (parental Communication Deviance, adverse rearing environment, and disadvantaged socioeconomic position) in the manifestation of thought disorders and psychotic and other psychiatric disorders (Wahlberg et al., 1997; 2004; Tienari et al., 2004; Wicks et al., 2010).

The first aim of the present study is to examine whether the proposed endophenotype for psychotic disorders, specifically child's thought disorders, is associated with biological and psychosocial risk indicators and their mutual interactions. The risk indicators include the adoptive child's genetic risk for schizophrenia spectrum disorders, the season of birth of the adoptee and the adoptive parents' Communication Deviance. Secondly, we evaluate the relationship of these risk indicators and their interactions in the adoptive child's schizophrenia spectrum diagnosis. We hypothesised that the risk for thought disorder and diagnosed schizophrenia spectrum disorder in the adoptive child increases not only in relation to the presence of the known risk indicators but also especially when there is an interaction of two risk indicators.

2. Methods

2.1. Subjects

2.1.1. Sample used in the Finnish Adoptive Family Study

A subgroup of the total sample used in the Finnish Adoptive Family Study was considered here. Full details of the selection and procedures have been reported elsewhere (Tienari et al., 2000). Briefly, hospital records for all women ($n = 19\,447$) admitted to Finnish psychiatric hospitals from 1.1.1960 to 31.12.1979 were reviewed to identify those women who had at least once received a diagnosis of a schizophrenia or paranoid psychosis (Tienari et al., 1987). Subjects were excluded if they had only received a diagnosis of a manic-depressive, depressive, reactive or psychogenic psychosis, or any other disorder. Research diagnoses using the DSM-III-R criteria (American Psychiatric Association, 1987) were obtained through a review of initial and subsequent hospital and clinic records and personal research interviews for all the respondent biological parents. Diagnostic reliability was carefully monitored and the inter-rater reliability for a total of 14 interviewees was 0.71 (Tienari et al., 2000). The list of biological index mothers was checked through every national census and parish register in the country to find the children who had been adopted (Tienari et al., 2000). After exclusions for various reasons including diagnostic reassignment, the study group consisted of 190 adoptees at genetic high risk (HR) of schizophrenia spectrum disorders, and of 192 adoptees at genetic low risk (LR) (control group). The high risk group consisted of children who were given up for adoption by biological mothers with schizophrenia spectrum diagnoses (mainly schizophrenia), while the biological mothers of children in the control group had only non-spectrum diagnoses or no psychiatric diagnoses at all. An extensive evaluation of the adoptive children and parents included individual, spouse and family interviews and psychological tests (Tienari et al., 1987), such as the ten-card individual Rorschach tests (Klopfer and Davidson, 1962) and diagnostic evaluation.

The Ethics Committee of Oulu University Hospital approved the Finnish Adoptive Family Study on 2 May 1988. The study design was reviewed by the Ethics Committee of Oulu University Hospital on 15 October 1991. In the Finnish Adoptive Family Study, verbal informed consent has been obtained since 1977; this procedure has proven satisfactory and followed the ethical practices of the time.

2.1.2. The subsample used here

Starting with a total sample of 382 families, a family was included in the present study if the family was intact (i.e., all of its members were alive, the parents were not divorced) and/or, in the case of single-parent families, the parent (in this sample, the father) was permanently absent. This first criterion led to the exclusion of 140 families. The second inclusion criterion was that the individual Rorschach tests on the adoptive parents and the adoptee had been carried out using all ten cards and that the Rorschach records had been tape recorded, which led to the exclusion of another 117 families. These strict inclusion criteria were applied in order to avoid unnecessary substitutions for missing Communication Deviance scores, since not enough information is available on the validity of different substitution methods. The eventual subsample included 125 adoptive families, 53 of which were families of genetically high-risk adoptees and 72 families of low-risk adoptees, with 119 families consisting of both parents and six of the mother alone (the father had never been present in the family). In six single-parent families the mother's CD score was multiplied by two to compensate for that of the missing father.

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