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Susceptibility to infectious diseases of childhood: A behavior genetics perspective

Podatność na choroby infekcyjne wieku dziecięcego: perspektywa genetyki zachowania

INTRODUCTION

A clear majority of recent research on behavior disorders, based on the paradigm of behavior genetics, is concerned with psychopathology, addictions, and, to a smaller extend, psychosomatics (DiLalla, 2004; Los et al., 2001; McGuffin et al., 2002; Plomin et al., 2003, see also Dragan & Polak, 2004). Its fundamental aim is to determine the role of both genetic and environmental factors in the complex mechanism of disorder development. All results of the said research explicitly indicate that genetic factors have a substantial impact on the forming of a human's susceptibility to the development of disorders, which are in themselves shaped also by environmental risk factors.

The general aim of my study was to assess the proportion between genetic and environmental factors in their influence on an individual's susceptibility to going down with various childhood infections. My particular focus, however, was to determine whether the method of behavioral genetic analysis may serve as a useful tool in the estimation of the role of genetic and environmental factors with regard to human susceptibility to infectious diseases of childhood, such as epidemic parotitis, the measles, pertussis, roseola, and varicella. Since these diseases are typically transmitted via droplet infection, it was reasonable to expect that result analysis will reveal a significant input of environmental factors (both shared and nonshared) in the risk of the development of these infectious diseases. Common environment is understood as the whole of conditions specific to a given family (including the interactions between family members). Nonshared environment is, in turn, the store of distinctive, unique environmental experience of an individual, resulting from both inside and outside family influence. The additive genetic factor is a term applied to parents – children genetic transmission. Finally, nonadditive genetic factors involve gene interaction (dominance and epistasis) in the descendant organism (Oniszczenko, 1997).

SUBJECTS

The studied sample comprised two groups of subjects. The first one consisted of children: 57 MZ twin pairs and 69 DZ same-sex twin pairs aged 3 to 10 reared together. The mean age of the MZ twins was 7,54 (SD=2,12), whereas of the DZ twins, it was 7,23 (SD=2,44). We have also retrospectively examined adult persons: 52 MZ twin pairs and 30 DZ same-sex twin pairs reared together aged 28 to 40 (M=33,05; SD=2,83). Twin zygosity was diagnosed with the use of the full version of the Questionnaire of Twins Physical Resemblance QTPR (Oniszczenko & Rogucka, 1996).

METHOD

The QTPR version we have employed in the study included questions regarding past infectious diseases of childhood. With reference to children subjects, their mothers provided the answers, whereas adult subjects personally indicated which of the diseases listed in the questionnaire they had already gone through. The results were analyzed by way of genetic model fitting, with the use of LISREL8 (Jöreskog & Sörbom, 1993). We tested full as well as reduced models taking into consideration the input of the additive (A) and nonadditive (D) genetic factors as well as of shared (C) and nonshared (E) environment into the susceptibility to childhood infectious diseases.

RESULTS

Results obtained in the study have been presented in the Table by specifying what percent of the variation of the variable under study (i.e. susceptibility to the diseases in question) is explained by particular components of the best fitting model.

Table

The input of genetic and environmental factors into the variation of the susceptibility to infectious diseases of childhood (percent of the explained variation) and goodness of fit parameters for the best fitting models

Group \ Model Components	A	C	E	$\chi 2$	df	p
Children	5	89	6	1,78	3	0,62
Adults	0	55	45	1,98	3	0,58

Note. A = additive genetic factor, C = shared environment, E = nonshared environment

DISCUSSION

The results we have obtained are consistent with our prior expectations. As the data presented in the Table indicates, the genetic factor explains merely 5% of the variation in children and is insignificant in the adult group, while the nonshared environment factor accounts for, respectively, 6% (children) and 45% (adults) of the variation of the variable under study. The absence of genetic factors in the data concerning the adult group does not necessarily mean that influence of this kind does not take place at all. Its contribution to the results in the adult sub-sample may be so small and statistically insignificant that in the course of the analysis it came down to zero. In reality, genetic factors may explain less than 5% of the results in the adult group. The largest portion of the variation of the susceptibility to children's diseases is explained by the shared environment factor – 55% in the case of adults and 89% in the case of children. These findings are conceivable once we consider the information regarding the portals of infection for the diseases in question (contact with other ill persons). Remaining in the same environment with a sick person substantially increases the risk of infection. The input of genetic factors into the susceptibility to childhood diseases appeared to be proportionally minute and virtually within the margin of error. These results confirm our expectations concerning the role of the environment in the mechanism of infection. Moreover, they indicate a possibility of using the behavioral genetic paradigm in the assessment of genetic and environmental influence on the risk factors of other diseases, including psychosomatic ones.

CONCLUSIONS

- ⇨ In the susceptibility to infectious diseases of childhood, environmental factors play a substantial role, while genetic ones are relatively insignificant,
- ⇨ The methods used in behavior genetics, model fitting analysis in particular, may prove to be useful tools in the determination of the role of risk factors in the process of infection.

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ABSTRACT

The aim of the study presented in the paper was to determine to what extend genetic and environmental factors participate in the formation of our susceptibility to childhood infectious diseases (epidemic parotitis, the measles, pertussis, roseola, and varicella). We have also endeavored to assess whether methods worked out within the field of behavior genetics may prove useful in estimating these influences. The study sample comprised two groups of same-sex MZ and DZ twins aged 3 to 10 and 28 to 40. The participants or their parents indicated which infectious diseases the subjects had gone through during their childhood. Model fitting analysis demonstrated, in accordance with our prior expectations, that the factors which have the strongest impact on the appearance of an infectious disease are shared environment and, to a lesser extend, nonshared environment (the input of the latter factor is more considerable in the adult group as compared to the children's group). The genetic factor explains merely 5% of the susceptibility to infectious diseases in the children's group and is insignificant in the adult group.

STRESZCZENIE

Celem badań przedstawionych w artykule było określenie, w jakim stopniu czynniki genetyczne i środowiskowe uczestniczą w kształtowaniu podatności ludzi na choroby infekcyjne wieku dziecięcego (koklusz, odra, ospa wietrzna, różyczka i świnka) oraz ocena przydatności metod genetyki zachowania dla szacowania tych wpływów. W badaniu uczestniczyły dwie grupy bliźniąt MZ i DZ tej samej płci w wieku od 3 do 10 lat oraz od 28 do 40 lat. Badane osoby lub ich rodzice wskazywali choroby infekcyjne przebyte przez badanych w dzieciństwie. Analiza dopasowania modeli wykazała – zgodnie z oczekiwaniami - że największą rolę w pojawieniu się choroby odgrywa wspólne środowisko i w mniejszym -specyficzne (udział tego czynnika jest większy w grupie osób dorosłych w porównaniu z dziećmi). Udział czynnika genetycznego wyjaśnia tylko 5 procent podatności na zachorowanie w grupie dzieci i jest nieistotny w grupie dorosłych.