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Dermatoglyphics and abnormal palmar flexion creases as markers of early prenatal stress in children with idiopathic intellectual disability

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Abstract

A number of studies have shown the importance of dermatoglyphics as markers of prenatal disturbance in developmental disorders of unknown origin. Genetic and non-genetic factors are involved in the aetiology of intellectual disability (ID), although the cause remains unknown in up to 50% of cases. The aim of the present study was to analyse dermatoglyphic traits and abnormal palmar flexion creases as markers of environmental prenatal stress in children with idiopathic ID (IID) using a case-control study design. Three dermatoglyphic variables, which have been reported as altered in other congenital disorders, were considered were studied in a sample of 62 children with IID (IQ < 70) and 75 healthy controls (IQ > 70): (1) fingerprint patterns; (2) total a–b ridge count (TABRC); and (3) abnormal palmar flexion creases (APFCs). More arches, the simplest fingerprint pattern, and more radial loops, an unusual pattern, were found in IID cases in comparison to controls ($\chi^2_3 = 9.26$; $P = 0.02$), with especially marked differences in boys ($\chi^2_3 = 6.5$; $P = 0.0008$). A significant increase of APFCs was also found in the affected children ($\chi^2_4 = 28.52$; $P < 0.00$; odds ration = 3.86, 95% confidence interval = 1.77–8.47). For TABRC, the differences between IID cases and controls failed to reach the conventional level of significance. These findings suggest that environmental factors acting early in development, or mechanisms involving an interaction of genotype and environment could be involved in the aetiology of some cases of ID.

Keywords brain development, dermatoglyphics, environment, palmar flexion creases, prenatal risk factors

Introduction

Intellectual disability (ID) is a life-long disability and the most common neurological handicap in childhood. It can be defined in a number of ways; for example, by IQ, neurological functioning, social adaptation or behavioural competence, or by any combination of these (Kiely 1987). Epidemiological studies of ID generally report its prevalence by degree of intellectual impairment. Thus, the prevalence of moderate and severe ID (IQ < 50) has been estimated at 1.3–2 per 1000 and that of mild ID (IQ = 50–70) at 3.7–5.9 per 1000 (Frost 1977; Fishbach *et al.* 1982) in the general population.

The role of many factors in the aetiology of ID (e.g. chromosomal abnormalities, genetic metabolic disorders, and pregnancy complications such as maternal infection, exposure to toxins and radia-

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tion, perinatal hypoxia, and postnatal infections) has been demonstrated. However, the causes in a high percentage of cases remain unknown (McLaren & Bryson 1987; Hou *et al.* 1998). The aetiology is partially dependent on the level of ID. Mild ID is generally idiopathic, but both severe and profound ID are more commonly of genetic origin. Additionally, labour-delivery complications (LDCs) have been suggested as being responsible in a high proportion of cases. Nevertheless, many authors have suggested that LDCs merely represent a secondary consequence of pre-existent abnormality in the foetus (Goodman 1988).

In cases of idiopathic ID, there is substantial evidence for a biological basis to brain abnormalities. Enlarged ventricles, similar to findings in schizophrenia, have been reported in 75% of children with ID of unknown origin (Prassopoulos *et al.* 1996).

Although dermatoglyphic characteristics have high but varying levels of heritability depending on the particular trait (Holt 1968), one part of their morphology is determined by intrauterine environmental influences acting early in the prenatal period and its embryology has been well established (Babler 1991; Kimura 1991). The development of the ridges begins with the formation of pads in the fingers, and the interdigital, hypothenar and thenar areas of the embryo's palm during the second month of intrauterine life. Epidermal ridges appear on the surface of the hands after the regression of the pads by the end of the fourth foetal month, when significant and critical growth of another ectodermal derivative, the brain, is also taking place (Rakik 1988). After this period, dermatoglyphic patterns remain unchanged. The presence of abnormalities in dermatoglyphics constitutes fossilized evidence of a prenatal insult that has occurred in the second trimester of prenatal life or before (Schauman & Alter 1976; Babler 1991).

Several environmental factors such as hypoxia, viral infections and delaying growth factors may modify the symmetry and size of the pad, modifying the future dermatoglyphic patterns and number of ridges (Mulhivill & Smith 1969).

Babler (1978) showed the relationship between embryonic stress and the presence of the simplest digital patterns. In the above study, more arches and, in consequence, lower ridge counts were associated with spontaneous abortion when cases were compared with elective abortions. Likewise, the association between dermatoglyphic abnormalities and maternal exposure to a range of environmental agents such as rubella, cytomegalovirus and alcohol has been well documented (for review, see Schaumann & Alter 1976).

The a–b ridge count (ABRC) has similarly shown a high degree of morphological variability related to environmental factors, and consequently, has been considered particularly suitable in the investigation of developmental disorders of idiopathic origin. The ABRC has been found to differentiate between cases of schizophrenia and normal controls (Turek 1990; Fañanás *et al.* 1990, 1996; Fearon *et al.* 2001), and to be sensitive to exposure to rubella in pregnancy (Schaumann & Alter 1976). In both cases, low ridge count values have been demonstrated in exposed individuals.

The association between Down's syndrome and abnormal palmar flexion creases (APFCs) is probably the best documented (Plato *et al.* 1973; Borbolla *et al.* 1980; Rajangam *et al.* 1995). However, APFC patterns have also been found with increased frequency in individuals with developmental defects caused by intrauterine exposure to adverse environmental factors, mainly infectious agents (Schaumann & Kimura 1991).

Environmental factors such as rubella (Purvis-Smith & Menser 1968), prenatal toxaemia, hypertension (Davies & Smallpeice 1963) and intrauterine methadone exposure (Dar *et al.* 1977) have also been associated with APFCs. A significant increase in Sydney lines and a large number of arches have been found in the fingers of victims of sudden infant death syndrome (Wilber *et al.* 1993). Previous studies have reported an association between unusual creases and schizophrenia in affected individuals (Bracha *et al.* 1991; Van Os *et al.* 1997; Rosa *et al.* 2000).

Although a large number of dermatoglyphic variables can be analysed as indirect markers of prenatal stress, some are particularly relevant to the investigation of congenital disorders, including: (1) simplification of finger patterns, as measured by the relative number of arches; (2) a decrease in the number of lines in the second interdigital area (the ABRC); and (3) abnormalities in the palmar flexion creases.