

Cytogenetic and Neurobiological Advances in Down syndrome

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Título: Avances Citogenéticos y Neurobiológicos en el Síndrome de Down.

Resumen: El síndrome de Down es una alteración autosómica que, tradicionalmente, ha sido estudiada de forma independiente, desde ámbitos como la medicina, la biología o la psicología. En este artículo pretendemos ir más allá e incorporar una perspectiva multidisciplinar que contemple, por una parte, los principales hallazgos de estas disciplinas y por otra, las teorías que intentan explicar las complejas relaciones que se producen entre dichos hallazgos. Con este objetivo, revisamos los avances que se han realizado en el campo de la genética, la neuroanatomía y la neuroquímica en relación a este síndrome, así como las explicaciones que se han desarrollado para intentar entender el perfil neuropsicológico asociado con esta alteración. Creemos que la incorporación de esta perspectiva ayudará a abordar una visión general sobre los correlatos psicobiológicos del síndrome de Down.

Palabras Clave: Síndrome de Down; genética; neuroanatomía; neuroquímica; neuropsicología; psicobiología.

Abstract: Down syndrome is an autosomal trisomy that traditionally has been studied independently from fields such as medicine, biology or psychology. In this article, we intend to go further and incorporate a multidisciplinary approach that includes, on the one hand, the main findings of these disciplines and, the theories that attempt to explain the complex relationships that occur between such findings. With this aim, we review the progress that has been made in the field of genetics, neuroanatomy and neurochemistry in relation to this syndrome, as well as the explanations that have been developed to try to understand the neuropsychological profile associated with this condition. We believe that the incorporation of this perspective will help achieve an overview of the psychological correlates of Down syndrome.

Key words: Down syndrome; genetics; neuroanatomy; neurochemistry; neuropsychology; psychobiology.

Introduction

Down syndrome (DS) is the most common genetic cause of intellectual disability (Nadel, 2003; Patterson, 2007). It was first described by Langdon Down in 1866, in an article published in the London Hospital Reports (Parajuli-Pozo & Casis-Arquero, 2000; Sherman, Freeman, Allen & Lamb, 2005). In this publication was already highlighted the presence of intellectual disability and a range of distinctive facial features, which to the author were similar to those of some eastern populations. The presence of these characteristic facial features allowed Langdon Down to define DS as a specific clinical entity (Carvajal, Iglesias & Loeches, 1994; Mégarbane et al., 2009).

Initially many hypotheses about the origin of DS were raised that alluded that DS was a throwback to more primitive races, or it was due, either diseases or additions of parents, problems during pregnancy, endocrine disorders, age of the mother or even gynecological irregularities (Mardomingo, 1995).

Afterwards, Waardenburg raised its genetic origin in 1932 (Capone, 2001). But it was not until 1959 when Lejeune and his colleagues confirmed the presence of an extra chromosome in pair 21 in nine children with DS (Lejeune, Gautier & Turpin, 1959). Moreover, the advancement of cytogenetic techniques also revealed the existence of three underlying chromosomal abnormalities on the onset of the syndrome: regular trisomy, translocation trisomy and mosaicism (Patterson, 2007; Seres, Cuatrecasas & Catalá, 2005). In the first and second case, the error occurs in the formation of gametes or the first mitotic division so it affects all somat-

ic cells of the organism, the main difference being that in the case of regular trisomy has 47 whole chromosomes whereas in the translocation case has 46 chromosomes and an extra portion of chromosome 21 (HSA21). In contrast, in mosaicism the error occurs from the second mitotic division, which gives rise to two different cell lines, one with 47 chromosomes and one with a normal genetic endowment.

Currently, it is widely known that between 90-95% of cases are due to regular trisomy of HSA21, while the rest are due to HSA21 trisomy by translocation or mosaicism (Bornstein et al., 2010; Patterson, 2007).

When it was compared the phenotype of people with regular trisomy against the phenotype of people with DS due to translocation no significant differences were found. In any case, the varying extension of certain phenotypic traits seems to be related to the amount of additional genetic material, but this does not seem to exert influence on the intellectual level (Bornstein et al., 2010; Devlin & Morrison, 2004a; Loeches, Iglesias & Carvajal, 1991). However, when this comparison has been carried out with cases caused by mosaicism it was observed that the latter present less accurate phenotypic characteristics in relation to the number of cells affected (Devlin & Morrison, 2004b; Drews et al., 2008; Seres et al., 2005). However, as in the previous case, the existence of this relationship has not been demonstrated in the case of intellectual level (Carvajal et al., 1994).

In addition to its relationship with intellectual disability, regular trisomy appears to lead to a specific neuropsychological profile (Kistler, Krinsky-McHale & Devenny, 2006; Menghini, Costanzo & Vicari, 2011; Ruggieri & Arberas, 2003), mainly characterized by : a) deteriorated language skills, affecting this impairment more to the production than to the comprehension, as well as to the phonological and morphological domain than the semantic and pragmatic (Fidler, Philofsky & Hepburn, 2007; Galeote, Soto, Sebastián, Rey & Checa, 2012; Vicari, Caselli, Gagliardi, Tomucci

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