Introduction

Risk factors represent a research priority regarding the cause of ASD. It is necessary to establish their exact specification, especially the interaction of environmental and genetics factors in the most vulnerable periods of development such as pregnancy and neonatal period. Small focused studies are necessary to test hypotheses and to create the opportunity for inventing the risk factors. We observed a statistically significant relationship between ASD group and the exposure to pollutants during pregnancy, infections in the newborn period and parent’s ages. In addition to case control type small studies, a cohort type approach is required in order to distinguish different genetic and environmental factors involved in the pathogenesis of ASD.

Objectives

We pursued the characterization of a series of environmental factors suspected as being risk factors, with an increased risk for developing autism spectrum disorders, as well as the medical conditions, case control studies may be a necessary first line. Some environmental factors, associated in current studies with the risk factor, are necessary to test hypotheses and to create the opportunity for greater replicated studies. As the studies for relatively rare medical conditions, case control studies may be a necessary first line. Some environmental factors, associated in current studies with an increased risk for developing autism spectrum disorders, have a higher discriminative power.

Materials and Methods

We conducted a clinical trial, case control, analytical, observational, retrospective. We included in the study 54 children diagnosed with ASD and 54 healthy children. For inventorying the risk factors, we used a set of questions about the pre/ peri and postnatal periods. We performed statistical analysis for assessing the existing link between the disease and the risk factor. We observed a statistically significant relationship between ASD group and the exposure to pollutants during pregnancy, infections in the newborn period and parent’s ages. In addition to case control type small studies, a cohort type approach is required in order to distinguish different genetic and environmental factors involved in the pathogenesis of ASD.
**RESULTS AND DISCUSSIONS**

Between ASD and control groups there was a balanced distribution with regard to gender ($\chi^2 = 0.52 \ p > 0.05$, $\phi = 0.02 \ p > 0.05$); origin environment ($\chi^2 = 0.20 \ p > 0.05$ $\phi = -0.04 \ p > 0.05$); level of parents education ($\chi^2 = 0.54 \ p > 0.05$, $\phi = 0.07 \ p > 0.05$); drugs consumption during pregnancy ($\chi^2 = 0.98 \ p > 0.05$, $\phi = 0.09 \ p > 0.05$); alcohol consumption during pregnancy ($\chi^2 = 3.08 \ p > 0.05$ respectively $\phi = 0.16 \ p > 0.05$); smoking during pregnancy ($\chi^2 = 0.21 \ p > 0.05$ respectively $\phi = -0.04 \ p > 0.05$); caesarean section ($\chi^2 = 0.07 \ p > 0.05$ and $\phi = -0.02 \ p > 0.05$). The familial histories of different psychiatric disorders were not balanced distributed and their association with ASD group was of borderline statistical significance ($\chi^2 = 3.53 \ p = 0.06$, $\phi = -0.18$, $p = 0.06$). We observed a statistically significant relationship between ASD group and the exposure to pollutants during pregnancy (pesticides, volatile solvents, heavy metals) ($\chi^2 = 6.27 \ p < 0.05$, $\phi = 0.24 \ p < 0.05$), with an average size of the statistical effect for this risk factor. From the 42 subjects with hypoxia at birth, 29 belong to the ASD group and only 13 to the control group, suggesting a relationship between two variables. This is confirmed by both value of hi square test ($\chi^2 = 9.97 \ p < 0.05$) and non-parametric correlation coefficient $\phi$ ($\phi = 0.30 \ p < 0.05$), both being statistically significant.

With regard to the infections in the newborn period, the data showed that there is an association between this factor and ASD ($\chi^2 = 6.35 \ p < 0.05$ respectively $\phi = -0.24 \ p < 0.05$).

**CONCLUSIONS**

We found no statistically significant differences between ASD and control groups in the duration of pregnancy and birth weight ($t = 0.31 \ p > 0.05$ and $t = 0.32 \ p > 0.05$).

In terms of father age, the results showed a significantly higher mean age for ASD group subjects ($M = 31.00$, $SD = 5.24$) than for the control group subjects ($M = 28.09$, $SD = 4.70$), $t (106) = 3.03$, $p < 0.01$. The statistical effect size in this context, measured using Cohen's d coefficient is $d = 0.58$ which means a medium effect. With regard to the maternal age, the results also showed a significantly higher mean age for ASD group subjects ($M = 28.13$, $SD = 4.77$) than the control group subjects ($M = 25.93$, $SD = 5.10$), $t (106) = 2.31$, $p < 0.05$. The statistical effect size, also measured using Cohen’s d coefficient is $d = 0.44$ which means a medium effect. Maternal age discriminate significantly better than a random test, the corresponding area under the ROC curve was 0.64 (from the maximum AUC = 1), statistically significant value ($Z = 2.70 \ p < 0.01$). Father’s age discriminate significantly better than a random test, the area under the corresponding ROC curve was 0.66, statistically significant value ($Z = 3.25 \ p < 0.01$). The curves of the two variables have similar evolutions, the described areas under ROC curves by the variables maternal and father age showed lack of difference in the discriminative value. The difference between the areas described by the two curves (0.02) was not a statistically significant one ($Z = 0.48 \ p > 0.05$), which means that there are no differences in their ability to discriminate between ASD and the control groups (fig. no1 insert about here).

1. We identified no statistically significant relationships between the factors gender, origin environment, parent’s education level, drugs use during pregnancy, alcohol consumption during pregnancy, smoking during pregnancy, caesarean section and ASD group.
2. Association of family history of mental illness with ASD group is borderline statistical significant ($\chi^2 = 3.53 \ p = 0.06$, $\phi = -0.18 \ p = 0.06$).
3. The mean father’s age in the ASD group is significantly higher compared with that of the control subjects ($t (106) = 3.03$, $p < 0.01$). The mean maternal age in ASD group is significantly higher compared with that of the control subjects ($t (106) = 2.31$, $p < 0.05$).
4. Maternal age ($Z = 2.70 \ p < 0.01$) and father’s age ($Z = 3.25 \ p < 0.01$) discriminate significantly better than a random test. We detected no differences in the ability of the variables mother and father’s age to discriminate between
ASD and the control groups (Z = 0.48 p > 0.05).

5. We observed a statistically significant relationship between ASD group and exposure to pollutants during pregnancy (pesticides, volatile solvents, heavy metals) ($\chi^2 = 6.27$ p < 0.05, $\varphi = 0.24$ p < 0.05), with an average size of the statistical effect for this risk factor.

6. We found a statistically significant relationship between ASD group and hypoxia at birth factor compared with the control group ($\chi^2 = 9.97$ p < 0.05, $\varphi = 0.30$ p < 0.05). The size of the statistical effect for this risk factor is an average one. This variable discriminate significantly better between the two groups than a random test (area = 0.64, sensitivity of 0.75, and a specificity of 0.64).

7. Association of neonatal infection, bacterial or viral, which required pharmacotherapy, and ASD group was statistically significant compared with control subjects ($\chi^2 = 6.35$ p < 0.05, respectively $\varphi = 0.24$ p < 0.05), but this variable did not discriminate better between the two groups than a random test.

8. In addition to case control type small studies, a cohort type approach on large samples is required in order to distinguish different genetic and environmental factors that may explain the pathogenesis of ASD and the comorbidities frequently presented. These studies should aim the development of standard methods to collect and storage the biological samples during pregnancy and neonatal period, clinical monitoring and subjects’ exposure to different environmental factors from birth, with concomitant collection of DNA samples. Also, it is important to study the influence of the disorder clinical profile, particularly in ASD subpopulations. These epidemiological studies must be interdisciplinary, made during pregnancy and neonatal period.

BIBLIOGRAPHY


Read about pervasive development disorder (PDD) causes, signs, symptoms, diagnosis facts and treatment of developmental delays. Autism, Asperger's syndrome, Rett's syndrome, childhood disintegrative disorder and PDDNOS are forms of this disorder. Because children with pervasive development disorders have a range of symptoms and abilities, a plan of therapy must be developed with the child's specific needs in mind. The treatment plan -- or more appropriately, a program of intervention -- will address the child's needs at home and at school. Pervasive developmental disorders (PDD) are indicated by more specific deficits in social behaviour, communication and language, together with a narrowed range of activities or interests that are often carried out repetitively. From: Manson's Tropical Infectious Diseases (Twenty-third Edition), 2014. Related terms Autism spectrum disorder (ASD) is a developmental disorder associated with a range of genetic, pathophysiological, and environmental conditions. There has been a growing interest in the immunological aspects of ASD, which encompass familial, maternal, and intraindividual factors. The possibility exists that autism is influenced by autoimmune factors and abnormalities in cytokine production both systemically and in the brain.