PRIMAL HEALTH RESEARCH

A NEW ERA IN HEALTH RESEARCH

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ATTENTION DEFICIT HYPERACTIVITY DISORDER (ADHD) AND OBESITY

TWO FACETS OF THE SAME DISEASE?

Until now, nosology—the naming and classification of diseases—was based on descriptions of symptoms (e.g. scarlet fever), or on altered functions (e.g., hyperthyroidism) or on altered organs (e.g., myocardial infarction) or on the name of a physician who described the pathological entity (e.g. Addison's disease). Today, the Primal Health Research Database suggests new ways to classify diseases. Classification is now possible according to critical periods for

gene-environment interaction. When two conditions seem to be similar with regard to timing for such interactions, we can go beyond the concept of co-morbidity to explore other possible similarities, particularly from clinical and pathophysiological perspectives. This is how, after studying in parallel *anorexia nervosa* and *autism*, two entities with similar critical periods for gene-environment interaction, we suggested that *anorexia nervosa* might be considered a female variant of the autistic spectrum.[1] A plausible interpretation of why *anorexia nervosa* is undoubtedly seen more in females is that prenatal exposure to male hormones might protect against the expression of this disease. A study of twins suggests such an interpretation. Girls who have a twin brother are at low risk for *anorexia nervosa*, compared with girls who have a twin sister, and with controls.[2] This interpretation is reinforced by the negative results of genetic linkage analyses that detected no change on the X chromosome.[3] Not only can Primal Health Research establish links between pathological entities, but it can also dismantle existing entities. For example, the primal health research perspective significantly differentiates subjects with *anorexia nervosa* from those with *bulimia nervosa* and suggests that the broad concept of 'eating disorder' should be reconsidered.

We'll use the same method to explore possible links between ADHD and obesity.

<u>ADHD from a Primal Health Research perspective</u>

Today the keyword ADHD leads to a dozen studies included in the Primal Health Research Database. The results of them all suggest a critical prenatal period for gene-environment interactions. A great diversity of environmental factors may be involved in these interactions: the similarities are in the timing.

An Australian randomised controlled study looked at children who had been exposed to repeated injections of corticosteroids into the pregnant mother before 32 weeks of gestation. The only significant finding was that these children were more likely than the control group to warrant assessment for attention problems.[4] An Italian study looked at children of mothers whose thyroid function had been monitored during early pregnancy. It appeared that children whose mothers became deficient in thyroid hormones in early pregnancy were at high risk of ADHD.[5] A Belgium prospective study evaluated the level of anxiety of

November 1943 and February 1947 in Amsterdam.[16] They compared people exposed to famine in late, mid, or early gestation with those born before, or conceived after, the famine period. It appeared that maternal malnutrition during early gestation was associated with higher Body Mass Index and waist circumference in 50 year-old women but not in men. Another study (published in 1998) looked at the glucose tolerance of adults who had been exposed either to the famine during fetal life, or who were born in the same area the year before the famine, or who had been conceived after the famine.[17] Glucose tolerance—which is closely related to obesity—was significantly decreased among adults who spent their intrauterine life during the period of starvation. The siege of Leningrad also exposed the entire population of a well-defined area to a severe famine. Among those who suffered malnutrition the influence of obesity on blood pressure was stronger.[18]

We must also add important negative findings concerning the difficulty to identify risk factors for obesity after birth. It is usually claimed that breastfeeding has a protective effect. However, the associations between the duration of breastfeeding and the risks of being overweight in childhood, adolescence and adulthood have not been confirmed by large authoritative studies extending to adulthood, such as the 1958 British birth cohort.[19] These results reinforce our conclusion that, in the case of obesity, the most critical period for gene-environment interaction is during fetal life.

Can other perspectives detect links between ADHD and obesity?

Several clinical studies have recently suggested links between ADHD and obesity. There have been reports by parents of sleep/alertness problems and ADHD traits in a clinical sample of obese adolescents. This led to studies of obese adolescents described as excessively sleepy by their parents: it appeared that they were at high risk for ADHD symptoms,[20] Other studies came to the conclusion that obese adolescents with bulimic behaviours may have a high probability of developing ADHD symptoms independently of associated depressive or anxiety symptoms.[21] A systematic review of the literature confirmed that the ADHD-obesity association is more that anecdotal: all reviewed studies indicate that subjects with ADHD are heavier than expected.[22]

pregnant women with the 'State Trait Anxiety Inventory'. The children were assessed when aged 8 or 9 years. It appeared that anxiety at 12 to 22 weeks of gestation was a significant independent predictor of ADHD whereas anxiety at 32 to 40 weeks was not.[6] Two valuable studies identified smoking in pregnancy as a significant risk factor.[7,8,] Furthermore, according to an American study, ADHD might be an additional deleterious outcome associated with prenatal exposure to alcohol independently of exposure to nicotine and other smoked substances.[9] The importance of the prenatal period in the genesis of ADHD is confirmed by the results of several studies indicating that low birth weight[10,11] and prematurity[12] are also independent risk factors.

Obesity from a primal health research perspective.

Today the keyword 'obesity' (and the related keywords 'insulin resistance' and 'diabetes type 2') leads to about 25 entries in our database. From an overview of these studies we can easily draw the conclusion that the critical period for gene-environment interaction is the same as for ADHD.

For example, it appears from one study that *betamethasone* (a corticosteroid) given to a pregnant woman to prevent respiratory distress in her newborn baby might result in insulin resistance of that child 30 years later.[13] Smoking in pregnancy was always found to be a risk factor.[14]

One of the oldest and most valuable studies in our database focused on risk factors for obesity. It was published as early as 1976 in an authoritative medical journal.[15] From October 1944 to May 1945 an acute famine affected the western Netherlands. The authors combined information about prenatal and early postnatal status at the time of the famine with weight and height at the age of 19 when 300,000 men were examined for military service. The main conclusion was that deprivation during the first half of pregnancy was related to significantly higher obesity rates at age 19, while deprivation during the last trimester of pregnancy and the first months after birth, was associated with lower obesity rates.

This historical study opened the way to further research about the long-term prenatal effects during the Dutch famine. In one study, published in 1999, the authors measured the body size of 741 people born at term between

There have been, of course, suggested pathophysiological interpretations of these associations. It makes sense to include binge eating in the framework of hyperactivity-impulsivity.[23] The propensity for binge eating is an obvious risk factor for obesity. A complementary (non- contradictory) interpretation is that excessive daytime sleepiness helps explain the association between obesity and ADHD symptoms.[24]

The clinical and pathophysiological connections have inspired pharmacological treatments of obesity with drugs, such as Adderall® and Concerta®, also commonly used to treat hyperactive children. The ratio of benefits to risks of such empirical treatments has not been published in the medical literature.

Conclusion

The data provided by the Primal Health Research database, complemented by clinical observation and pathophysiological considerations, suggest that ADHD-obesity-bulimia nervosa should be looked at as one multifaceted disease. Furthermore, the study of this broad pathological entity tends to confirm that the concept of "eating disorder" should be reconsidered. *Anorexia nervosa* seems to belong to another pathological entity that includes the autistic spectrum, with critical periods for gene-environment interaction in the perinatal period.

Will Primal Health Research revolutionise nosology?

Michel Odent

UPDATED INFORMATION ABOUT

THE MIDATLANTIC CONFERENCE ON BIRTH AND PRIMAL HEALTH RESEARCH

With the participation of WHO

Las Palmas, Gran Canaria, February 26-28, 2010

Objectives, programme and practical details at www.wombecology.com .

Everybody can actively participate in the conference by presenting a poster (click on 'posters').

Register as early as possible so that your choice of workshops will be guaranteed.

Explore the Canary Islands Convention Centre. From its prestigious symphony Hall (1,656 seats), when the curtain behind the stage is not completely closed, you can see the ocean and grasp the meaning of our logo: it is a unique place to dream of the Rebirth of the Goddess of Love – the one who was born from the foams of the waves.

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