Nature, Nurture, and Human Behavior; an Endless Debate

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Editorial

Abstract

This editorial aims at addressing the endless debate concerned with the extent to which particular aspects of behavior are a product of either nature (inherited i.e. genetic), nurture (acquired i.e. learned), or their interaction. In spite of the philosophical conflict between nativists who adopt an extreme hereditary i.e. aaryale-sMle-áldistp pat]ele¥M(#yMy! Mr ryM

unraveling these different and reciprocal influences on human behavior [1].

A perfect example of nature and nurture interaction is perfect pitch which is the ability to detect the pitch of a musical tone without any reference. Researchers have found that this ability tends to run in families and believed that it might be tied to a single gene. However, they have also discovered that possessing the gene alone is not enough to develop this ability. Instead, musical training during early childhood is necessary to allow this inherited ability to manifest itself [2].

Pediatric mental disorders and genes' influence (nature)

Variance is a measure of how much a trait varies between people in the population being studied while heritability is a term that refers to the proportion of the variance explained by genetic factors. Most psychological traits have been found to have a heritability of around 50%. This means that genetic differences between individuals account for roughly half of the observed variance in a given population. Over recent years, behavioral geneticists have made the dramatic claim that shared family environment has little if any effect on most psychological traits; family resemblances are almost all attributable to shared genes rather than shared environment. Conduct problems are one likely exception to this rule, with most studies showing a relatively small genetic contribution to this sort of behavior and documented that shared environment is the main reason for these problems running in families. At the opposite extreme, liability to autism may have a heritability of over 90% [9].

It is worthy to mention that pediatric mental disorders are often observed in association with other malformations and as a feature of well- defined genetic syndromes. As an example, there are many reported neuropsychiatric and behavioral disorders associated with 22q11.2 micro-deletion syndrome [DiGeorge syndrome (DS), velocardiofacial syndrome (VCFS), and conotruncal anomaly face syndrome] that include elevated rates of shyness, disinhibition, autism spectrum disorders, psychosis, severe attentional difficulties, executive dysfunction, behavioral phenotype reflective of non-verbal learning disabilities, concomitant language deficits, and socio-emotional concerns [10,11]. People with 22q11.2 micro-deletion syndrome are missing a sequence of about 3 million DNA building blocks (base pairs) on one copy of chromosome 22 in each cell. Researchers have determined that the loss of a particular gene on chromosome 22, TBX1, is probably responsible for many of the syndrome's characteristic signs (such as heart defects, a cleft palate, distinctive facial features, hearing loss, and low calcium levels). Some studies suggested that a deletion of this gene may contribute to behavioral problems as well. The loss of another gene, COMT, in the same region of chromosome 22 may also help to explain the increased risk of behavioral problems and mental illness. The loss of additional genes in the deleted region likely contributes to the varied features of 22q11.2 deletion syndrome [12]. This region contains 30 to 40 genes. Zaky et al (2015) a [13] reported Fluorecence In Situ Hybridization (FISH) documented microdeletion 22q11.2 in 2 out of 16 FISH examined cases with neurodevelopmental disorders (12.5%); one was diagnosed at the age of 11 years and the other at the age of 9 years with concomitant congenital heart disease and hypocalcaemia in both cases and mild intellectual disability in one of them and specifc learning disorder (dyslexia, dyscalculia) in the other.

Pediatric mental disorders and environmental influence (nuture)

Family adversities

Many known family factors are associated with an increase in the rate of one or more of pediatric mental disorders. So, it is too easy to fall into the trap of assuming that such association implies causation. On the other hand, many pediatric mental disorders are associated with well documented family adversities but whether this means

poor supervision. The absence of warmth in family relationships is not as relevant as a predictor for poor outcome as the presence of discord as family discord is so often connected with poor discipline. Children may learn that aversive behaviors are a particularly effective way of getting parental attention. By time, children appear to have internalized the parents' mode of interaction "repeating the same pattern in other relationships. After divorce, parents tend to feel

correlated with the total Childhood Autism Rating Scale (CARS) scores i.e. the lower the 25 (OH) vitamin D, the higher the total CARS scores, the severe the autistic manifestations.

Another example of non-familial environmental factors that might compromise pediatric mental health is secondhand smoke (SHS). Studies indicated that prenatal tobacco and childhood SHS exposure are associated with child behavioral problems, including internalizing and externalizing behaviors, ADHD, and conduct disorder [27]. It was reported that primate postnatal tobacco smoke exposure leads to changes in brain cell development similar to prenatal nicotine exposure [28]. Zaky et al (2015)$_d$ [29] investigated the potential association of SHS exposure with children's mental health disorders. T ey found that SHS exposed children had signi f cantly higher mean value of urinary cotinine level (a biological marker of SHS), total Pediatric Symptom Checklist's (PSC), and Strength di f culties Questionnaire's (SDQ) scores compared to controls with signi f cantly

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