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Mental Retardation is Caused by a Combination of Genetic and Environmental Factors

Emmet McManus*

Department of Psychology, University of California, California, USA

Description

Mental Retardation (MR) is a neurodevelopmental condition that causes severe impairment in intellectual and adaptive functioning. It is described as having an IQ of less than 70, as well as deficiencies in two or more adaptive behaviors that affect day-to-day living. Reasoning, problem-solving, planning, abstract thinking, judgment, academic learning, learning through instruction and experience, and practical knowledge proven by both clinical assessment and standardized testing are all listed as intellectual functioning in the DSM-V. Adaptive behavior is characterized as a set of cognitive, social, and practical skills that people use in their daily lives to complete tasks.

The majority of young children show no physical indications of FXS. Physical features of FXS do not begin to develop until adolescence. Aside from intellectual incapacity, elongated face, huge or protruding ears, flat feet, enlarged testes, and low muscular tone are all possible symptoms of the illness. Early childhood is plagued by recurrent otitis media (middle ear infection) and sinusitis. It's possible that your speech is clumsy or nervous. Stereotypical movements (e.g., hand flapping) and abnormal social development, including shyness, restricted eye contact, memory issues, and difficulty with face encoding, are examples of behavioral features.

As a result of having a second, normal X chromosome, males with a full mutation have nearly total penetrance and will almost always show symptoms of FXS, whereas females with a full mutation have around 50% penetrance and will almost always show signs of FXS. Females with FXS might have mild to severe symptoms, however they are often less afflicted than males.

Autism

Fragile X syndrome co-occurs with autism in many cases and is a suspected genetic cause of the autism in these cases. This finding has resulted in screening for FMR1 mutation to be considered mandatory in children diagnosed with autism. The prevalence of concurrent autism spectrum disorder (ASD) in people with fragile X syndrome has been estimated to be between 15 and 60%, with the difference due to differences in diagnostic methods and the high frequency of autistic features in people with fragile X syndrome who do not meet the DSM criteria for an ASD. Individuals with FXS have a hard time making friendships, and those with FXS and ASD typically have a hard time conversing with their peers in a reciprocal manner. In FXS, social withdrawal behaviors such as avoidance and apathy tend to be the best predictors of ASD, with avoidance appearing to be more closely linked to social anxiety and indifference to ASD. When both autism and FXS are present, children have a higher language deficit and a lower IQ than children who only have FXS.

Inheritance

Fragile X syndrome has long been thought to be an X-linked recessive disorder with variable expressivity and potentially low penetrance. Due to genetic anticipation and X-inactivation in females, the inheritance of Fragile X syndrome does not follow the conventional pattern of X-linked dominant inheritance, and some experts have proposed that X-linked illnesses be labeled as recessive rather than dominant. Females with full FMR1 mutations may have a milder phenotype than males due to variability in X-inactivation. Prior to the discovery of the FMR as a gene, pedigree research revealed the presence of male carriers who were asymptomatic, with their grandchildren affected at a higher rate than their siblings, implying that genetic anticipation was at work. ale carriers pass on their permutation to all of their daughters, with the length of the FMR1 CGG repeat often not expanding during meiosis, the cell division necessary to create sperm. Males with a full mutation, by the way, only pass on permutations to their daughters. Females with the entire mutation, on the other hand, can pass it on to their children, thus there is a 50% chance that a child will be impacted. Furthermore, due to instability, the length of the CGG repeat in female permutation carriers frequently increases during meiosis, and depending on the length of their permutation, they may pass on a full mutation to their children, who will subsequently be impacted. Strand slippage during DNA replication or DNA repair synthesis is thought to be the cause of repeat expansion.

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***Address for Correspondence:** Dr. Emmet McManus, Department of Psychology, University of California, California, USA; E-mail: emmetmcmanus@hotmail.com*

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