

Autism Aetiology: The journey of discovery from the “refrigerator mother” to the neurodevelopmental hypothesis

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Leo Kanner a child psychiatrist in the US reported a clinical description in 1943 based on his observation of 11 children who were extremely preferred for loneliness and highly focused on objects with repetitive patterns of behaviour. An Austrian paediatrician Hans Asperger (1944) reported similar symptoms mostly with people of high intelligence (1). Kanner proposed these symptoms as ‘childhood or early-onset schizophrenia’ (1944), later he coined it as a specific condition called ‘infantile autism’. Many theories and hypothesis were emerged to explain this enigmatic condition (2).

Aetiology to autism was first portrayed by Leo Kanner who attributed autism to a lack of maternal warmth and attachment. Bruno Bettelheim’s book - *The Empty Fortress* (1967) widely popularized the theory of “refrigerator mother” in which maternal coldness or emotionless parenting style were explicated for developing autism. Bettelheim’s notion of blaming the parents for their children’s autism was largely criticized and currently it is as an obsolete concept (3).

Several cognitive models attempted to untangle the conundrum in autism aetiology by following a symptomatology or phenotype approach. In this, the theory of mind hypothesis was the pioneer one which suggests that children with autism have a deficit in understanding the feelings of themselves and others which were manifested as ‘self-absorption’ in social situations (4). The theory of executive dysfunctioning hypothesis proposes that children with autism have some restricted patterns of behaviour such as obsessive sameness, rocking, repetitive hand flapping, difficulty in sustaining attention and these problems of executive functions are akin to the deficit in motor tasks associated with certain brain injuries such as frontal lobe damage. The weak central theory hypothesis further expounded the ritualized patterns of behaviour in autism by suggesting that individual with autism have a weak drive for global coherence in which they process information in a detailed manner ranging from pedantry to obsession (5). However, all of these approaches failed to explain both the pathogenesis and phenotypes of ASD.

The biological research identified an excessive brain growth during early developmental life in the child with autism following a normal developmental period. This was explained as “anabolic hypothesis” in which autism was attributed to abnormal enlargement of brain volume that results from neuronal overgrowth. Although macrocephaly is considered as one of the most replicated biological findings, the emerging evidence supports the role of both hyper and hypoactivity of certain functional areas of the brain in autism (6). The empirical data also suggests that autism may be associated with imbalanced levels of certain neurotransmitters such as GABA, serotonin, glutamate and dopamine (7). Autism predominantly affects boys than girls and the twin and family studies advocates the possible link of heritability for autism. However, more research is required to establish autism as a disorder of abnormalities in the genetic pathways such as X linked autosomal recessive or dominant (8).

DSM-5 defines Autism spectrum disorders (ASD) as a neurodevelopmental condition characterized by persistent impairment in reciprocal social communication and social interaction and restricted, repetitive patterns of behaviour, interests or activities (9). Recent epidemiological studies conducted across the world revealed an increasing prevalence rate of ASD as per various categories such as age, gender, socioeconomic status etc. (9-13). In summary, research on autism aetiology is increased over the past two decades in terms of both phenotype and pathogenesis to autism. However, it still remains as an inexplicable condition as cited by Kathrynne (2008) “despite many promising hypotheses, the current literature is filled with conflicting findings and no one hypothesis has appeared to take centre stage” (14).

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