Institutional Deprivation - a risk factor for neuro-developmental disorders.

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Neurodevelopment disorders (NDDs) are complex conditions which arise in childhood but can last throughout life. It includes conditions like attention deficit hyperactive disorder (ADHD), specific learning disorders, intellectual disability, autism spectrum disorder (ASD) and communication disorders (Thapar, et al., 2017). These disorders have long been studied in relation to institutional deprivation and have resulted in deprivation-specific problems (DSPs). This is because institutional deprivation results in reduced social interaction, cognitive stimulation, language development and forming attachments (Nelson, 2007) which lead to NDDs. The age and length of institutionalisation together with environmental features of the institution seem to affect the level of deprivation in children, making this heterogenous concept a risk factor for NDDs.
Moreover, the role of genetics, pre-institutional and prenatal environmental factors can also aid the occurrence of NDDs which challenges the results of literature provided on institutional deprivation as a risk factor. As a risk factor, institutional deprivation accentuates NDDs among children.

NDDs show multidimensional problems which are affected by institutionalisation. The place where the child is brought up and the behaviour of caregivers play a central role in deciding the fate of a child's cognition, social behaviour as well as language (Siegel & Mc Daniel, 2004). Also, early adversity (family violence, physical or sexual abuse and family discord) has been associated with various behavioural and emotional problems (Kendler et al., 2004). As institutionalised kids were deprived of proper care and the ratio of staff to the child was extremely low (Children's Health Care Collaborative Group, 1992); it can be said that these children lived in adversity exposing them to a range of social, motor, cognitive, affective and language deficits (Homberg et al., 2016), suggesting a role of institutional deprivation as a risk factor for developing NDDs. These deprived conditions led to DSPs which is defined as pattern appearing in response to institutional deprivation with long-lasting biological effect and is difficult to be eradicated even by post-adoption experience (Rutter, et al., 2010). Four DSPs evident in institutionalised kids were a) ADHD capturing mainly inactivity, hyperactivity and combined types; b) quasi-autism (QA), reflecting symptoms of autism; c) disinhibited social engagement disorder (DSE), attachment disorder with lack of social specificity; d) cognitive impairment (CI) where the IQ of the individual is less than 80 (Kumsta et al., 2015). As all DSPs are some form of NDDs, it can be said that institutional deprivation is a risk factor leading to NDDs.

The age of the child during institutional deprivation affects NDDs. Longitudinal studies have analysed the effect of deprivation on institutionalised children with those who belonged to the normal population. A study comparing children from the UK of the same ages were divided as a) those who were adopted from institutions between ages 2 and 4; b) children who returned to their biological parents between the ages of 2 and 4 c) group that lived in institution d) children who were never institutionalised. The results indicate that children who were adopted early fared better across all domains of social activity, language development and IQ when compared to late adopters or the normal population (Tizard, 1977; Tizard & Hodges, 1978; Tizard & Reese, 1974,1975). A study of Lebanese orphanages by Dennis (1973) found that children adopted before 2 years of age were able to have the same IQ as non-institutionalised children. But those with more than 2 years of age at the institution were found to have a permanent loss in their IQ. Although this study has not been tested adequately and lacks a sample size which can be used to generalise (Maclean, 2003), similar results were drawn from the studies of Morisson & Ellwood (2000) and Le Mare and Audet (2002). They found that children institutionalised after 2 years of age had a profound effect on all aspects of intelligence. These studies prove that the age at which a child is adopted will influence the ex-

tremity in their behaviour and increase the risk of NDDS. This also indicates that length of stay in institutional deprivation is an important factor.

The length of institutional deprivation increases the risk of NDDs. Ames & Chisholm (2001) found that children who have spent 8 or more months in Romanian institutions had more behaviour problems, attachment disturbances and lower IQs when compared with non-institutionalised or early adopted children in the Canadian population of the same age and sex. These results reflect the deleterious outcomes of institutional deprivation. However, these studies lack randomly assigned samples and the selection bias may have tempered the results. Also, more mentally developed children might have been adopted first (Maclean, 2003), suggesting the presence of pre-institutional impairment in children. Another study which proves the length of institutionalisation affecting NDDs by Sonuga-Barke with colleagues (2017) showed that children who have spent less than 6 months at an institution have a similar result when compared to UK controls while those who spent more than 6 months showed symptoms ADHD, DSE, QA to young adulthood (see fig 1, 2, 3 & 4). Deprivation-related CI showed a remittance and normalisation in the study sample. The improved CI might indicate towards the neuroplasticity of the brain which helped in this normalisation with the change in environment.

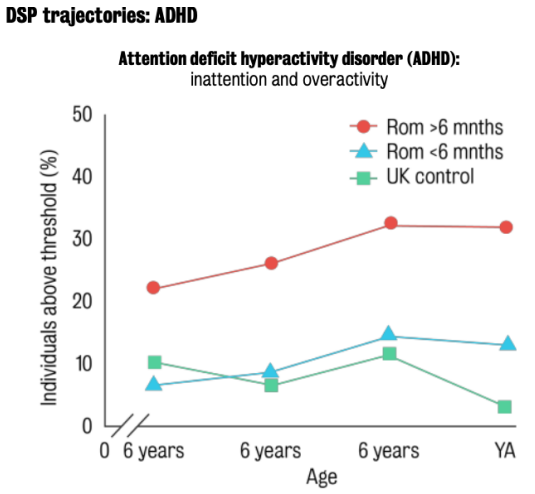

Fig 1- Extended institutional deprivation leads to persistence in ADHD to young adulthood

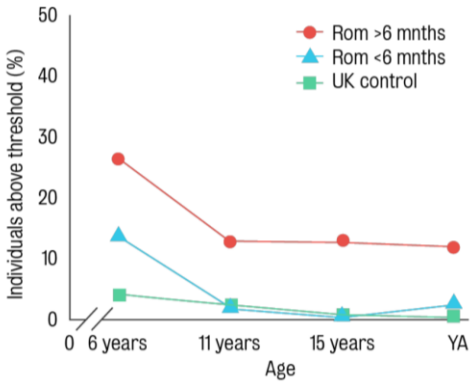

Fig 2-Extended institutional deprivation leads to persistence in DSE to young adulthood

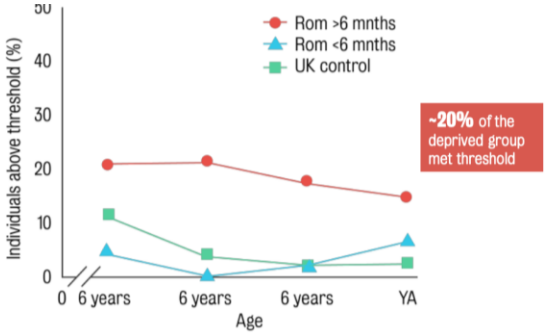

Fig 3- Extended institutional deprivation leads to persistence in QA to young adulthood

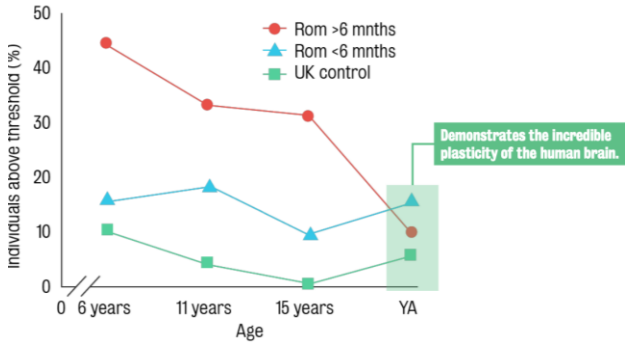

Fig 4- Extended institutional deprivation had no effect on CI when compared to control group

Although, another study showed a marked difference in CI with children who had institutional deprivation for over six months (Kumsta et al., 2015) showing that CI might be accompanied by other DSPs and thus affected the results. The result suggests the length of institutional deprivation is a risk factor affecting

NDDs. The research lacked information on features of the environment during deprivation which might have affected the children under study.

The environmental factors during institutional deprivation not only lead to behavioural problems but also affects brain development (Mez & McCall, 2010). It was seen that institutional deprivation of more than 6 months with psychosocial deprivation (extreme environmental conditions) taking sub-nutrition as a control variable was found to be a major risk factor leading to NDDs. It leads to developmental delays and low IQ scores (around one standard deviation below)in all assessments ages of 6, 11 and 15 years (Kumsta et al., 2015). There is also a decrease in brain size (Rutter et al., 2012), reduced cortical activity (MacLaughlin et al., 2014) and reduced connectivity (Helmeke et al., 2009). Hanson et al. (2013) found that there was low white matter volume in the pre-frontal cortex (PFC), an area associated with cognition. He assumed that neglect during deprivation might impact the development of PFC as it requires a longer time than other brain areas. The results suggest that there is reduced white matter volume and poor scores on neurocognitive performance by environment-deprived children. Therefore, a higher level of psychosocial deprivation leads to more of behaviour, emotional and cognitive problems in children (Landry et al., 2006). But the intervention of enhancing the psychosocial environment has brought developments in the children again indicating the plasticity of the brain to adjust to changing deprived conditions (Research TSPUO, 2008). These studies focus on the risk of psychosocial deprivation in developing NDDs. However, these studies did not consider the role of prenatal, pre-institutional risks and the effect of genes as influential factors for the NDDs.

The prenatal, pre-institutional and genetic heritability might be the cause of NDDs instead of institutional deprivation. The evidence on pre-institutional and pre-natal influence on DSPs has shown a large variance. Prenatal stress has shown to be a major cause of ADHD, ASD, cognitive impairments and different emotional as well as behavioural problems (O'Donnell et al., 2009). Alcohol, drugs or the use of antidepressants have also shown to make the child prone to ADHD, similarly, ASD risks include foetal alcohol syndrome, advanced parents' age, infections and delivery complications (Bailey et al., 1995). Twin studies indicate that three-fourths of variance can be attributed to genes, showing ADHD is not a genetic illness but impulsiveness and inattentiveness are influenced by heritable genes in ADHD (Larsson et al., 2014). Alternatively, ASD has high heritability with a concordance rate of upto 60% in monozygotic twins (Bailey et al., 1995). The presence of the gene DAT1 haplotype has shown to interact with the environment to enhance the risk of ADHD (Stevens et al., 2009). This study showed children possessing gene DAT1 haplotype when institutionally deprived for more than 6 months had more chances of developing ADHD than others who did not possess gene risk (see fig 5). This suggests possessing risk genes (DAT1 haplotype) might make children more prone to developing NDDs when experiencing institutional deprivation. The gene and environment (GxE) interaction provides new dimensions for research in institutional deprivation studies.

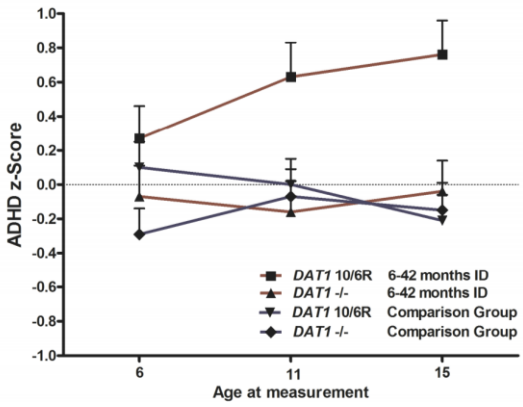

Fig- 5 Children carrying DAT1 haplotype and extended deprivation were at increased risk of developing ADHD.

Therefore, it can be said that institutional deprivation being a heterogenous concept act as a risk factor for NDDs. But as the studies discussed above, differ in the type and severity of adversity in the sample, the results may not be consistent. Also, longitudinal studies often lack the response from the complete sample at different time points which hampers the results. The persistence of NDDs even after adoption points towards neurobiological causes. The prenatal and pre-institutional factors might influence the NDDs and genetic heritability might interact with the deprived environment to increase the risk of NDDs. These new dimensions can be explored further to gain a complete understanding of institutional deprivation as a risk factor for NDDs. The gene-environment interaction will help decipher major risk factors which will provide a piece of evidence for better interventions within the institutions as well for the treatment of these disorders in adult life.

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FIGURES

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