https://doi.org/10.46344/JBINO.2025.v14i03(a).02

CLINICAL NEUROSCIENCE: PHYSICAL AND MENTAL HEALTH COMPLICATIONS IN CHILDREN DEPRIVED OF FATHERHOOD

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ABSTRACT

Introduction: Maternal separation and neonatal paternal deprivation (PPD) have had profound and persistent effects on the physiological and behavioral development of children, with other problems neglected in professional practice, such as adverse childhood emotions and post-traumatic stress disorder (PTSD) in children, which accumulate evidence of the same cumulative effects while a harmful vicious cycle is not identified. ***Objective:*** To update the different areas of professional activity, which exert direct or indirect influence on the mental health of children, such as social workers, lawyers, judges, psychologists, psychiatrists, doctors, and educators, as well as the parents themselves, through concrete evidence from neuroscience and psychosocial neurobiology, exclusive to paternal deprivation. ***Methods:*** Review of the literature and performance of a clinical synthesis with theoretical implications, based on neurobiological and neurogenomic mechanisms, which demonstrates a concrete and harmful factor for evolutionary medicine. ***Results and Discussion:*** There are few studies on paternal deprivation in humans, and many studies in rats and mice were used to explore the mechanisms in this review, as they are equivalent to human brains. In addition to the harmful effects of deprivation, we explored other neurobiological mechanisms that are associated with paternal presence. ***Conclusion:*** This work brings together some consequences for the physical and mental health of children, as well as their future generations, which are being neglected and complicated by several professionals in medicine, law, psychology, social assistance, and politics.

Keywords: mental health, neurological injury, child neglect, paternal deprivation.

1.Introduction

Currently, children have been the focus of family disputes in contexts rich in emotional neglect of the child and an alienating state in dysfunctional families, which are complicated by domestic violence, family crimes, crimes of passion, parental alienation, and family courts that may be aggravating such conditions due to the lack of knowledge and updated medical approaches, in addition to presenting laws that favor women. [1]

The state, together with the parents and the professionals involved, should provide extensive care for children, since the interpersonal relationships of biological parents have pronounced effects on the survival and development of neuroendocrine, immuno-inflammatory, social, cognitive, and affective functions. [1-2] Although fathers do not experience the profound hormonal and physiological changes associated with pregnancy, childbirth, and lactation, males in biparental species often undergo behavioral, endocrine, and physiological changes that allow them to meet the demands of fatherhood. [1-2]

Numerous studies have demonstrated that neuroplasticity in parents can manifest itself in a variety of processes, including changes in neuron production and survival, functional and structural modifications of existing neurons, and large-scale morphological changes in brain regions. [3-4]

Over the past decade, strong empirical evidence from several long-term studies has concluded that physical and sexual abuse, as well as emotional deprivation in childhood, makes individuals significantly more vulnerable to mental and functional disorders later in life. [3-4]

In addition, increased vulnerability to several somatic disorders, cardiovascular disorders, type 2 diabetes, obesity, chronic obstructive pulmonary disease, immunological disorders, pharyngeal and lung cancer, mental disorders such as addiction, and family conflicts such as domestic violence has been demonstrated. [3-4] Several current studies describe the neurobiological and psychological mechanisms underlying development that mediate these long-term effects. [3-4] Although maternal separation and neonatal paternal deprivation (PPD) have been shown to exert profound and persistent effects on the physiological and behavioral development of children, other problems neglected in professional practice, such as adverse childhood emotions and posttraumatic stress disorder (PTSD) in children, accumulate evidence of the same cumulative effects while a harmful vicious cycle is not identified.[3-4]

Feldman (2016) has shown that fathers provide the same level of sensitivity as mothers during interactions with their children and make a unique contribution to the social development and emotional regulation of the child, particularly to the child's later ability to function adaptively within the social environment. [5-6] The absence of a paternal caregiver

influences socialization and increases the likelihood of drug and alcohol abuse, mental illness, poor educational achievement, and criminal activity in affected children. [5-6]

2. Objective

To update the different areas of professional activity that directly or indirectly influence children's mental health, such as social workers, lawyers, judges, psychologists, psychiatrists, doctors, and educators, as well as parents themselves, through concrete evidence from neuroscience and psychosocial neurobiology. In this study, we only reviewed the neurobiological changes in children in relation to paternal deprivation.

3. Methodology

A bibliographic search was conducted in the PubMed and Web of Science databases. The inclusion criteria were studies that demonstrated clinical evidence associated with neurobiological mechanisms through functional neuroimaging exams related to paternal deprivation. Studies that presented data on adverse childhood emotions were also chosen for convenience.

A clinical synthesis with theoretical implications was performed, based on neurobiological and neurogenomic mechanisms, which demonstrates a concrete and harmful factor for evolutionary medicine.

4. Results

Studies on paternal deprivation in humans are scarce, and many studies in rats and mice were used to explore the mechanisms in this review, as they are equivalent to human brains. In addition to the detrimental effects of deprivation, we explored other neurobiological mechanisms that are associated with paternal presence.

Exposure to enriched or impoverished environmental conditions, experience, and learning are factors that influence brain development, and it has been shown that neonatal emotional experience significantly interferes with the synaptic development of higher associative areas of the forebrain and with the underdevelopment of the prefrontal cortex (PFC). [7]

4.1 Paternal deprivation in animals

Pinkernelle J et al. evaluated the impact of paternal deprivation on dendritic and synaptic development in the somatosensory cortex and showed at the behavioral level that paternal care comprises 37% of total parent-offspring interactions and that the somatosensory stimulation provided by parents consists mainly of huddling, licking/cleaning, and playing. [3]

At the morphological level, compared to offspring raised by both parents (mother and father), animals deprived of their father presented significantly reduced numbers of spines in the basal dendrites of pyramidal neurons. [3]

Furthermore, paternal deprivation induces hemispheric asymmetry of dendritic morphology of somatosensory pyramidal neurons. Paternally deprived animals have shorter and less complex basal dendrites in the left somatosensory cortex compared with the right hemisphere.[3]

Ovtscharoff W Jr et al. demonstrated through optical and electron microscopy the impact of paternal care (emotional contribution of the father to his offspring) on the synaptic

development of the anterior cingulate cortex, with findings of significantly reduced densities (-33%) of synapses in layer II of fatherless animals, indicating an imbalance between excitatory and inhibitory synapses in the anterior cingulate cortex of fatherless animals.[4]

Another study using monogamous mandarin rats evaluated the effects of early biparental separation (EBPS) or neonatal paternal deprivation (NPD) on adult paternal behavior and found evidence that EBPS or NPD have long-term consequences and reduce paternal behavior in adult animals and are associated with the oxytocin system in the medial preoptic area, in addition to being able to interact with the dopamine systems of the NAcc and thus regulate paternal behavior. EBPS may affect interactions between the medial preoptic area and the NAcc. [5]

A study in rats found that PPD decreased serum oxytocin levels and increased corticosterone levels in females only, and in both males and females, PPD decreased oxytocin receptor mRNA and protein expression in the medial preoptic area (MPOA), nucleus accumbens (NAcc), and medial prefrontal cortex (mPFC), but increased it in the medial amygdala (MeA) and decreased estrogen receptor mRNA and protein expression in the MPOA. [5–6]

PPD increased dopamine type I receptor expression in the NAcc but decreased it in the mPFC. PPD decreased dopamine type II receptor (D2R) in the NAcc in both males and females, but increased D2R in the mPFC in females and decreased D2R protein expression in males. Furthermore, PPD decreased vasopressin receptor 1A (V1AR) in the MPOA, MeA, and mPFC, but only in males. [6]

Wang B et al. demonstrated in rats that paternity experience can increase the active components of parental care and alter OTR and D2R expression levels in a region- and time-dependent manner. The effects of paternity experience on paternal behavior levels are derived from mechanisms of oxytocin (OT) and dopamine-2 (D2) receptors in the nucleus accumbens (NaCC) and medial nucleus of the amygdala (MeA). They showed that experienced fathers exhibited more active paternal behaviors, such as licking, retrieving, and nest building, than novel fathers; however, novel fathers spent more time in inactive huddling than experienced fathers. [7]

Experienced fathers' OTR levels increased significantly with pup age. The D2R level in the MeA of new fathers did not change significantly, while the D2R levels of experienced fathers increased with the age of the pups. [8]

A study in rats examined the development of juvenile social play and paternal influence on play fighting and found that paternal deprivation influences the development of play fighting in offspring; hypothalamic vasopressin, oxytocin, and serum corticosterone may play a modulatory role in the alteration of play fighting caused by paternal deprivation; the decrease in play fighting may be correlated with depressed levels of vasopressin in the hypothalamus.[8]

4.2 Paternal Deprivation in Humans

Schultz T et al. demonstrated with comparisons of (a) single mother

upbringing, (b) biparental upbringing by both father and mother, and (c) biparental rearing by two female caregivers resulted in:[9]

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Paternal care represents a critical environmental factor for the synaptic and dendritic development of pyramidal neurons in the vmPFC of their offspring; [9]

-a second caregiver ("aunt") does not "buffer" the neuronal consequences of paternal deprivation; [9]

- neuronal development in the vmPFC is affected differently in male and female offspring in response to different family members. [9]

In another study, Schultz T et al. demonstrated that growing up without paternal care affects dendritic and synaptic development in the nucleus accumbens of male and female offspring and that replacing the father with a female caregiver does not "compensate" for the impact of paternal deprivation. [10]

The effects of paternal deprivation indicate a critical impact of paternal caregiving behavior on the development and maturation of neuronal networks in the nucleus accumbens of children. One study has shown that complete deprivation of paternal care permanently delays or retards synaptic connectivity in the brain, particularly in the medial prefrontal cortex (mPFC) of offspring in a sex-specific manner. Replacing the father with a female caregiver ("aunt") may "buffer" the detrimental effects of paternal deprivation on neuronal development. [10]

Helmeke C et al. demonstrate that paternal deprivation delays and partially suppresses the development of orbitofrontal circuits. The delayed dendritic and synaptic development of the apical dendrites of layer II/III pyramidal neurons in the orbitofrontal cortex of fatherless adults may reflect reduced excitatory connectivity of this cortical subregion. [11]

These results demonstrate that early traumatic emotional experience alters the synaptic input to pyramidal neurons. This experience-induced modulation of limbic cortex development may determine psychosocial and cognitive capacities during adulthood. [12]

4.3 Biobehavioral Family Synchrony

Shimon-Raz O et al. showed widespread cross-brain synchrony for attachment cues that was even greater in response to mother-infant social cues, i.e., any cue of mother-infant attachment (self or generic) induces substantial concordance across multiple brains and reduces neural variability. [18-19]

Feldman's group demonstrated that primary attachment is a central survival phenomenon that recruits substantial neural resources and suggests that attachment bonds may function to construct a neural blueprint for the consolidation of humans in social groups through neuronal signaling mechanisms between peers. [20–23]

Between-person correlations in the ACC dynamically tracked moment-to-moment variations in mother-infant behavioral synchrony, suggesting that moments of coordinated mother-infant behavior trigger cross-brain uniformity. [18–19]

When infants and children affiliate with others, signals may direct their social efforts toward "better" or

"worse" social partners. [20–23]

An example of harmful modulation through synchrony is the activation of neuromaladaptive behaviors or family schemas, which can be activated by the simple presence or voice of a family member. An example of a positive modulation effect is when one of the parents gives advice or teaches something more easily to the child, rather than someone else. [20-23]

Clinically, synchrony is observed by the effectiveness of the bond and the ability to identify the child's emotional state through the parents (mentalism). [20-23] Several studies have shown the direct link between poor family functioning and the onset, development, and maintenance of various risk behaviors and those related to different addictions through the indirect effect of affective involvement and the parallel mediation role played by impulsivity and psychopathological symptoms. [20-23]

Evidently, the greater the presence of avoidant, denial, punitive, and aversive behaviors, such as family schemas and other family coping behaviors, the lower the effectiveness of family synchrony between the parent and the child. [20-23]

4.2 Neuropsychodynamics/Neuromirroring

Although early mammalian parent-offspring interactions are primarily maternal, there is growing evidence of the impact of fathers on offspring development, which may include paternal absence or deprivation, paternal presence but ineffective interaction, and paternal presence with excessive interaction (authoritarianism). [24-27] There is now ample evidence that even in the absence of contact with their offspring, fathers can transmit environmentally induced effects (behavioral, neurobiological, and metabolic phenotypes induced by stress, nutrition, and toxins) to their offspring, and it has been speculated that these effects are achieved through epigenetic variation inherited within the paternal lineage. [24-27]

Like mothers, fathers directly influence the neurodevelopment of their offspring through direct care, as observed in biparental studies. The functions of family roles are fundamental to this context, since children's brains are programmed so that the father is the limiter, validator, and teacher of survival and exploration tasks. [24-27]

Through family neuropsychodynamics, the mechanisms of neurological synchronization, the amygdala, limbic systems, and mirror neurons are involved, in which parents produce little or very significant influence that can impact the quality of mother-child interactions. [24-27]

Due to the lack of family synchrony and mainly parental insufficiency and deficit of emotional intelligence (mentalism), parents may present intrusive behaviors, which play a role in cases of marital disputes, exercising parental influence. [24-27] However, these influencing characteristics have an important neuromaladaptive component and can be transmitted through generations, thus leading to a transgenerational transmission of neurobiological and neurodysfunctional behavioral phenotypes. [24-27]

Most studies suggest that rTPJ activity is

associated with the distinction between self and other, so that activations may emphasize the incongruence between self and other or allow switching between their related representations. [24-27]

The mirror mechanism is a basic brain mechanism that transforms sensory representations of the behavior of others into motor or visceromotor representations of that behavior. [24-27]

Depending on its location, it can fulfill a number of cognitive functions, including action and emotional understanding. [24-27]

4.1 Transgenerational epigenetic inheritance

Transgenerational epigenetic inheritance is a mechanism by which environmental exposures and lifestyle decisions of parents alter their own epigenetic states and transmit these changes to their offspring through gametes. It is this pattern of inheritance that has shed light on the fact that preconception lifestyle decisions that parents make are significant, as they significantly impact their offspring.[13-14]

Considering epigenetic changes in gametes, together with neurological changes that occur in interpersonal relationships between family members, is essential for the physical and mental health of future generations. [13-14]

Over the past decade, a growing body of evidence has shown that male exposure to toxins and pharmaceutical drugs, chemical weapons, and ionizing radiation; undernutrition/overnutrition; lifestyle factors such as exercise, alcohol, and tobacco consumption; trauma; and stressful situations impact fetal development and the health and disease of adult offspring through direct (genetic/epigenetic) and indirect (maternal uterine environment) effects. [13-14]

Beginning preconception and during in utero and early postnatal life, cells acquire an epigenetic memory of early exposure that can be influential throughout life and program the health of the child. [15]

According to Dolinoy and Jirtle (2008), paternally expressed genes such as Peg3 and Gnasxl also regulate postnatal mother-offspring interactions and promote nursing behavior in offspring, and there is emerging evidence of the susceptibility of these imprinted genes to modification in expression in response to paternal environmental exposures. [15-17]

Thus, inheritance by offspring of paternal genetic/epigenetic variation may have direct effects on offspring and/or indirect effects on offspring through maternal investment, subsequently leading to complex interactions between mothers and offspring with developmental consequences. [15-17]

Several clinical studies have observed intergenerational transmission of stress effects, with an impact on neuroendocrine, cognitive, and psychological traits, involving epigenetic changes in sperm manifested by the organism's body in response to the environment. [15-17]

Offspring of Holocaust survivors, both male and female, have shown increased symptoms of posttraumatic stress disorder, elevated risk of anxiety and depression, impaired cortisol levels, and altered epigenetic regulation of the

glucocorticoid receptor gene, Nr3c1, suggesting a possible role for the HPA axis in this form of transgenerational inheritance. [15-17]

The impact on Nr3c1 is of particular interest, as increased levels of Nr3c1 DNA methylation have been found in women reporting childhood abuse. [15–17]

Additional intergenerational influence of paternal trauma has been observed in children of men exposed to the Kosovo war and in children of Vietnamese refugees in Norway. [15–17]

A recent study evaluated a four-generation family and found that DNA methylation patterns are transmitted to offspring during spermatogenesis, suggesting that modification of DNA methylation profiles across generations may result in an altered phenotype of their progeny. [15–17]

Furthermore, analysis of DNA methylation in human germ cells has identified several loci that resist reprogramming, including loci that are associated with neurological disorders, making them potential candidates for transgenerational epigenetic inheritance. [15-17]

In addition to impacts on the HPA axis of children, paternal experiences also affect offspring cognitive abilities through cellular and molecular processes. It has been shown that exposing males to stress had an impact on their progeny over two generations. [18-19]

Offspring showed impaired long-term memory associated with reduced long-term potentiation (LTP) and elevated long-term depression (LTD) in the hippocampus, as well as reduced brain-specific gamma subunit of protein kinase C (Prkcc), which is implicated in synaptic plasticity and memory performance. [18-19]

Furthermore, DNA methylation of the Prkcc promoter was decreased in paternal sperm as well as in the hippocampus of offspring. We have recently shown that paternal exposure to the stress hormone corticosterone (CORT) can influence offspring cognitive performance.

Only female offspring of CORT-treated fathers showed decreased memory retention compared to controls, suggesting that the influence on offspring is sex-specific. Paternal CORT exposure was associated with reduced body weight in both female and male offspring. [18–19]

Interestingly, the same behavioral traits were transmitted to female offspring in the following two generations, but only through the paternal lineage and without changes in CORT levels. [18–19]

Furthermore, all three female generations evaluated showed increased mRNA levels of Rcan 1 and Rcan 2, which are related to synaptic plasticity in the CA1 region of the hippocampus. [18–19]

A model combining maternal separation with unpredictable maternal stress (MSUS) provided several important insights into epigenetic inheritance, which are translatable to childhood trauma in humans. [18–19]

MSUS exposure of male mice before

weaning induces affective behavioral changes in their offspring for up to three generations in a sex-dependent manner. [18-19] Paternal exposure to MSUS also alters the DNA methylation profile in both the fathers' sperm and the offspring's cortex, including genes related to transcriptional regulation (such as methyl CpG-binding protein 2, MeCP2) and affective and stress responses (such as cannabinoid receptor 1, CB1, and corticotropin-releasing factor receptor 2, CRFR2, respectively). [18-19]

The same group further demonstrated that exposing males to the MSUS paradigm reduced the sociability of their male progeny and reduced the social memory of their female progeny by modifying their serotonergic signaling. [18-19]

Furthermore, offspring of prenatally stressed parents are likely to be more sensitive to stress, which may be explained by the observed alterations in gene expression. [18–19]

Furthermore, a mouse model of chronic social defeat stress demonstrated that female and male offspring of stressed adult male mice exhibited increased affective behavioral responses, as well as increased levels of plasma CORT and vascular endothelial growth factor (VEGF) in male offspring. [18–19]

In rats, offspring of socially isolated parents exhibited elevated anxiety responses, as well as increased plasma CORT and ACTH levels, increased hippocampal Nr3c1 expression, and decreased hypothalamic and pituitary corticotropin-releasing hormone (CRF) receptor (CRFR1), respectively, demonstrating HPA axis dysregulation. [18-19]

Another study examined the effects of HPA axis activation through the administration of synthetic glucocorticoids in male mice and found that HPA axis activation produced modifications in the sperm DNA methylation profile, as well as altered DNA methylation and gene expression in the hippocampus of the offspring. [18-19]

Similarly, in rats, paternal stress resulted in modified DNA methylation profiles in the hippocampus of their offspring. 18-19

Altogether, the aforementioned studies indicate that paternal stressful experiences can induce changes in sperm content and subsequent elevations in HPA axis activity in the offspring. [18-19]

Role of sperm-borne microRNAs in the epigenetic inheritance of stress. The results of the aforementioned studies, and others, imply that the mechanism of intergenerational transmission from the brain to gametes (and via the HPA axis when related to anxiety) is epigenetic, including changes in the sperm DNA methylation profile. [18–19]

Furthermore, small noncoding RNAs (sncRNAs) have also been suggested to play an important role in transgenerational epigenetic inheritance. [18–19] Indeed, both microRNAs (miRNAs) and PIWI-interacting RNAs (piRNAs) have been shown to be detected in mature human sperm and regulate key genes in the zygote that are important for normal development. [18–19]

Following paternal stress, HPA axis dysregulation has been found in both

female and male offspring, and these intergenerational effects have been proposed to have been transmitted by changes in sperm miRNA content. [18-19]

Furthermore, male grandsons of CORT-treated mice exhibited increased depression-like behavior and, surprisingly, reduced anxiety-related behavior. [18-19]

Although CORT treatment had no apparent effect on parental behavior, it did result in altered sperm miRNA content, with over 100 miRNAs showing differential expression compared with controls. [18-19]

These studies demonstrate that stressful experiences and elevated stress hormone levels (CORT administration) can modify sperm miRNA content and that these changes have downstream effects on offspring behavior for at least two generations. [18-19]

5. Conclusion

This work brings together some consequences for the physical and mental health of children, as well as their future generations, which are being neglected and complicated by several professionals in medicine, law, psychology, social assistance, and politics.

The result of this review corroborates the historical social and political denial or other forms of child abuse in many parts of the world.

According to Harman JJ et al., reframing parental alienating behaviors as a form of family violence also serves as a desperate call for social scientists to focus more theoretical and empirical attention on this topic. With the same thought, we highlight the harmful and concrete effects related to neurology, endocrinology, genetics, epigenetics, psychology, and social and intellectual aspects of the simple and harmful paternal exclusion. 1

6. Declaration of conflict of interests

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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