

Antagonistic New-born Child Care Dulls the Mother's Neural Handling

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Editorial

The obligatory attachment of young mammals to their primary caregiver is a distinguishing feature. Despite this obligation, there are individual differences in the quality of this attachment, which reflect the nature of early caregiving and how the infant psychologically represents routinized interactions with the parent. Individual differences are best identified by observing brief separations followed by parent-infant reunions. Separations from parents are stressful at young ages, so how the infant uses the parent for comfort when they reunite has proven diagnostic for classifying infants' attachment quality within the range of typically developing children (i.e., secure or insecure) vs. attachment quality associated with later-life pathology (i.e., disorganized)

The stress of the separation–reunion procedure is thought to be critical for identifying these abnormal attachment styles as the source of later-life socio-emotional difficulties such as poor stress management, reactive attachment disorder, and future psychopathology. However, the causal and mechanistic pathways that link poor caregiving, attachment quality, and altered socio-emotional development have not been identified.

Because the existing literature relied on humans, current knowledge is based on correlational designs and investigative approaches that do not involve invasive biological procedures. The current study aimed to overcome this methodological barrier by assessing human and rodent reunion behaviours concurrently, and then investigating the neuron hormonal mechanisms of the observed attachment behaviours within the causal rodent model. Because the desire to reunite after separation is conserved across mammals, reunion behaviours can easily be transferred from humans to rodents to study neuron hormonal mechanisms.

We capitalise on the power of using animal models to understand human behaviour in this study by measuring localised brain activity and behaviour during experimental treatment/testing, randomising assignment, and assessing causation with a clinically-informed question. Although attachment pathways are complex, we focus here on the causal pathways between stress physiology and cortical function induced by the parent as a function of attachment quality. We concentrate on cortical oscillations, which are rhythmic neural activity that synchronises brain activity to coordinate functions within and across neuronal networks. Cortical oscillations are a biological cornerstone of brain development that are heavily influenced by corticosterone fluctuations and associated environmental stressors. While social stimuli are important modulators of neural oscillations throughout human development, the attachment figure (biological or adoptive caregiver) is an especially effective stimulus [1-5].

We begin with a translational framework to investigate a causal link between attachment quality, infant experience with maternal care quality, and

maternal regulation of the infant brain during separation–reunion. The Strange Situation Procedure is the framework within which we test the infant's reaction to reunion with the mother (SSP). This procedure, which employs separation-induced distress followed by reunion with the parent, has diagnostic value for classifying infant attachment quality. We present data from high-risk human infants to serve as a template for a rodent SSP (rSSP), in which rat pups are randomly assigned to adversity-rearing with a maltreating mother or control rearing to assess attachment behaviours. Using the rodent model again, we directly assess causality by inhibiting pup stress hormone synthesis (metyrapone; MET) for rescue during the rSSP. Using the rodent model once more, we investigate the causes of rSSP deficits by measuring infant pups' neurobehavioral responses during adversity-rearing, thereby determining which processes of the infant were disrupted by which behaviours of the mother in the maltreating context. Again, we test causality in the rodent by inhibiting pup stress hormone synthesis (metyrapone) in order to prevent the behavioural and neurobiological aberrations identified in the rSSP. We show that during adversity, the dynamic range of pup cortical local field potentials (LFPs) decreases during nurturing maternal behaviours but is unaffected by rough handling. During reunion, adversity-experiencing pups exhibit abnormal interactions with their mothers as well as blunted cortical LFP. During adversity or reunion, blocking pup stress hormone restores normal behaviour, LFP power, and cross-frequency coupling. These findings suggest that adversity-rearing cause's stress-induced aberrant neurobehavioral processing in the mother, which can be used as an early biomarker of later-life pathology across species.

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