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# Enduring Consequences of Maternal Separation on the Volume of Parvalbumin Interneurons in the Hippocampus

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**Abstract**

Parvalbumin interneurons (PV-INs) are a subset of GABAergic inhibitory neurons that play a crucial role in regulating cortical and hippocampal circuits. Dysfunction of PV-INs is implicated in neurodevelopmental and psychiatric disorders. Maternal separation (MS) is a well-established rodent paradigm used to study adverse early-life experience and its long-term behavioral effects. MS has been shown to reduce PV-INs expression in different brain regions leading to disrupted cognitive behaviors. This research investigated the long-term effect of the MS on the volume of the PV-INs in the CA1 subregion of the hippocampus in rats. Male rat pups were separated from their mothers for 3 hours daily from postnatal day (PND) 1 to PND 14. After weaning, the animals were maintained in a standard manner until adolescence. At adolescence, brain samples were extracted and the volume of the PV-INs in the CA1 area of the hippocampus was measured using the 3D stereological technique with nucleator method. The results showed that the volume of the PV-INs in the CA1 subregion of the hippocampus in maternally separated rats was significantly smaller ($p < 0.05$) than the intact rats in adolescence. This study showed that early life adverse experiences can have a persistent effect on the volume of PV-INs in the hippocampus into adolescence and suggests that MS may lead to a disruption of synaptic excitatory inhibitory balance and may be associated with symptoms related to autistic behaviors resulting from MS that have been reported in previous studies.

**Keywords***:* Parvalbumin interneurons volume, maternal separation, animal model, neurodevelopmental disorders

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## 1. Introduction

PV-INs play a vital role in maintaining the balance between excitation and inhibition in the brain (Nahar et al., 2021). Owing to their role as strong modulators of activity of pyramidal neurons, they can be a potential target to intervene against glutamate imbalances as well as against excitotoxicity in diverse psychiatric and neurological conditions such as autism spectrum disorders, schizophrenia, Alzheimer's disease, and drug abuse (Marín, 2012). There is evidence that these interneurons are significantly vulnerable under conditions with cognitive impairment (Deng et al., 2019). The functional complexities of PV-INs highlight the need to further investigate their roles, particularly considering the mounting evidence of their morphological and synaptic diversity. Interneurons are intrinsic to the hippocampus, playing a critical role in processing stimuli from the environment and encoding memories (Zou et al., 2016). The ventral hippocampus was identified as a regulator of social memory, with parvalbumin interneuron having a role in discriminating between new and familiar experiences (Zou et al., 2016). Within CA1, where a large percentage of GABAergic cells are interneurons of parvalbumin type, these interneurons are significantly active in the retrieval phase of social memory and not in its encoding phase (Aery Jones et al., 2021). Hyperactivation of PV-INs was found to disrupt mice's recognition of familiar mice, demonstrating their role in social discrimination (Bezaire & Soltesz, 2013). Additionally, PV-INs play a role in producing gamma oscillations needed for brain function and cognitive processing, especially in cortical information processing (Engel & Singer, 2001). Optogenetic stimulation of these interneurons can elicit gamma oscillations in cortical networks, whereas parvalbumin deficiencies are associated with elevated gamma oscillations (Cardin et al., 2009). With PV-INs having the ability to stabilize CA1 communication networks over long timescales, investigation of these interneurons in the highly dense CA1 region of the hippocampus represents a fruitful avenue through which to understand pathophysiology and mechanisms of disease development in neurological conditions.

Neural development is very sensitive to environmental stimuli, especially in early postnatal stages. Any adverse experience, such as maternal separation (MS) in critical periods of development, has far-reaching effects on neuronal maturation of circuits, including PV-INs (Ueno et al., 2017). Enriched or impoverished conditions have been demonstrated to impact parvalbumin expression and structural evolution, possibly with long-term behavioral effects (Feng et al., 2021). One of the strongest early environmental influences is maternal care quality. Disruptions in infant-mother attachment—like extended maternal separation—have been associated with deficits in emotional and cognitive growth (Li et al., 2013). They are also capable of simulating early-life stress in rodents and underlie typical symptoms of autism spectrum disorder, including diminished social interaction and heightened repetitive behaviors (Reisi-Vanani et al., 2024), (Lippmann et al., 2007). Previously, we showed that maternal separation not only causes autistic-like behaviors, but also results in changes in hippocampal structure, specifically an increase in the CA1.SR volume, mediated by changes in the oxytocin system (Mansouri et al., 2020). Recent neuroanatomical and molecular evidence has implicated PV-INs in the CA1 region as particularly susceptible to early adversity and as possibly crucial to determine the hippocampal involvement in neurodevelopmental disorders (Riga et al., 2014). Following earlier accounts of increased CA1 volumetric changes in animals separated from their mothers, this study examines if these separations also impact PV-INs volume in this region. With a

particular focus towards structural plasticity in the hippocampus, this work sets out to continue to shed light on the possible cellular mechanisms by which early adverse experiences may contribute to neurodevelopmental disorders such as autism.

## 2. Method

### Animals and Maternal Separation Procedure

In this study, Wistar male and female rats weighing 250 grams were purchased from the animal house of the Faculty of Biology of the Shahid Beheshti University. They were randomly kept in cages for mating. After ensuring pregnancy, female rats were kept in individual cages until the day of delivery. The born rats were randomly divided into two groups: control and MS (n=5 per group). The animals were kept in standard conditions with controlled temperature (21°C), controlled humidity, and a 12-hour light-dark cycle, and they had free access to food and water. We considered the day of offspring birth as postnatal day 0 (PND 0). The maternal separation procedure started at PND 1 in which offspring were separated from the mothers for 3 hours daily (9 am- 12 am) during PND 1–14, each pup in a separate compartment. Control littermates were kept with their mothers until they were weaned (PND 21). After weaning all animals were kept at standard condition without any intervention until adolescent (PND 42). All stages of this research have been conducted in accordance with Ethics Committee standards of Shahid Beheshti University (IR.SBU.ICBS.97.1045).

### Tissue processing

At PND 42 rats were deeply anesthetized with ketamine (Ratiopharm, Germany; 150 mg/kg,i.p) and were then transcardially perfused with ice-cold heparinized saline for 10 min, and subsequently with 4 % paraformaldehyde (PFA) solution in 0.1 M phosphate buffer (PB, pH 7.4) for 10 min. The brains were removed from the skull of the sacrificed animals and kept in 4 % PFA until tissue processing. Right or left hemisphere of brain was randomly selected and kept in a 30 % sucrose solution for 72 h. Subsequently, the brains were frozen using liquid nitrogen, and were cut coronally at 50 µm thickness on a cryostat (SCILab, Cool-Cut, SCI85683, England). The first section of each series was chosen randomly using a random table. The sections from hippocampus, were selected based on a systematic sampling principle, with a section sampling fraction of 1/12. Brain sections were then immune-stained with anti-parvalbumin antibody for quantifying the volume of the PV positive interneurons in the CA1 area of the hippocampus.

### 3D quantification of the volume of the PV positive interneurons

To measure the volume of PV-INs, a light microscope (Olympus BX56) and NewCAST software ((Visiopharm, Hørsholm, Denmark)) were used. All slices of each brain were image captured with a 4x objective lens. Then, the area of interest (CA1) was delineated with 100x objective lens. Using the nucleator method with 6 lines while the mode was vertical uniform random (VUR) based on the assumption of rotational symmetry of the cells the volume of the soma of interneurons (Figure 1B) was measured in each brain for at least 50 interneurons at a height of -5 to -30 $\mu m$. The criterion for selecting cells was that the cell body was completely clear.

### Statistical analysis of data

GraphPad Prism software version 10 (GraphPad Software, San Diego, CA, USA) was used to analyze the data and generating graphs. The normality of the data distribution was examined

using Q-Q plot. The difference between groups (Control and MS) was analyzed using independent t-test. The significance level was set at $p < 0.05$. The results are presented as mean ± standard deviation (SD).

## 3. Result

As shown in figure 1A, independent t-test analysis demonstrated a significant difference between the volume of the PV-INs in the CA1 area of the control and MS groups ($p = 0.009$). Which means that maternal separation had led to enduring morphological change in the size of the PV-INs of this area.

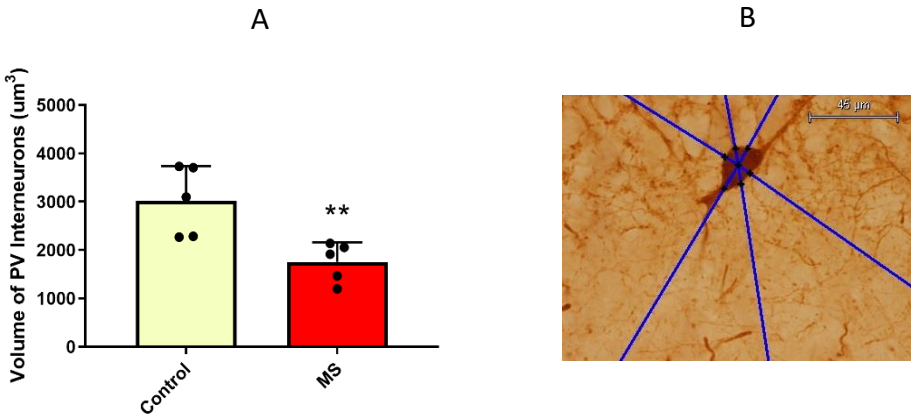


Figure 1. (A) Effect of the maternal separation on the volume of the PV-INs in CA1 area of the hippocampus, $^{**}P < 0.01$; (B) Estimation of the volume of the PV-INs using objective lens 100x applying nucleator method in NewCAST software. Scale bar=45 µm

## 4. Discussion

This study showed that early life adverse experiences, such as MS, can have a persistent effect on the volume of PV-INs in the hippocampus CA1 area. This region was chosen because we had seen structural changes of it in our previous study in relation to the emergence of autistic behaviors in adolescence due to MS (Mansouri et al., 2020). Considering the role of PV-INs in autism spectrum disorders (Yao & Li, 2024), it is important to investigate possible changes in them due to environmental factors . Although the effect of MS on changes in the volume of PV-INs has not been reported so far, our result is consistent with previous studies that examined the effect of MS on the number of PV-INs and showed that MS leads to lower PV-INs cell counts in the hippocampus and PFC (Brenhouse & Andersen, 2011). Studies report decreased PV immunoreactivity in the prefrontal cortex (PFC), hippocampus, and amygdala following maternal separation (Perlman et al., 2021). In these studies, (Czéh et al., 2015; Filipović et al., 2018) the effect of stress on PV-INs using a maternal or social deprivation model was investigated, in which PV-INs were detected using immunohistochemical staining and their number or density was counted. They also measured the expression of PV-INs mRNA or protein. The results showed that the number of PV-INs and PV protein expression were reduced in the hippocampus of stressed

animals. It has been debated whether these findings were due to PV-INs death or reduced maturation. It seems that this reduction occurs more due to cell immaturity than cell death because PV-INs reductions in the hippocampus was not associated with increased apoptotic markers. Anyway, this reduction may impair GABAergic inhibition, leading to hyperexcitability and disrupted neural synchrony. MS can lead to dendritic atrophy and reduced perineuronal nets (PNNs) extracellular matrix structures that stabilize PV-INs synapses (Gildawie et al., 2020) which may contribute to long-term deficits in synaptic plasticity and cognitive flexibility. PV-INs dysfunction is linked to impaired working memory and attention, seen in MS models (Abraham et al., 2023). Reduced PV activity in the amygdala and PFC is associated with increased anxiety and depression-like behaviors (Page et al., 2019). PV-INs abnormalities are implicated in schizophrenia, autism, and mood disorders (Juarez & Martínez Cerdeño, 2022), which are more prevalent in individuals with early-life stress. Early-life adversities increases oxidative damage, which PV-INs (high metabolic demand) are particularly sensitive to (Horn et al., 2019). Elevated glucocorticoids from maternal separation may suppress PV-INs development (Soares et al., 2020).

The CA1 region of the hippocampus plays a crucial role in memory formation, spatial navigation, and social cognition (Geiller et al., 2023), and emerging research suggests it may be involved in autism spectrum disorder (Tao et al., 2022). Studies in ASD mouse models show hyperexcitability in CA1 pyramidal neurons, leading to altered synaptic plasticity (LTP/LTD) (De Introna et al., 2025). CA1 may have disrupted excitatory (glutamatergic) and inhibitory (GABAergic) signaling, contributing to ASD-like behaviors (e.g., repetitive behaviors, social deficits). CA1 helps distinguish similar memories and its dysfunction may contribute to cognitive inflexibility in ASD (Banker et al., 2021). Some individuals with ASD show difficulties in context-dependent learning (Skoyles, 2011), which relies on hippocampal processing. The hippocampus modulates stress via the HPA axis (Cole et al., 2022) so CA1 abnormalities may contribute to comorbid anxiety in ASD. Altered hippocampus connectivity with prefrontal cortex and amygdala may affect social-emotional integration (Ghasemi et al., 2022). We suggest that drugs targeting NMDA/AMPA receptors or GABAergic transmission in CA1 may help restore balance. The CA1 area's role in ASD likely involves synaptic dysfunction, memory processing deficits, and social-cognitive impairments. Further research is needed to clarify whether CA1 abnormalities are a cause or consequence of ASD and whether targeting this region could improve symptoms.

MS in rodents has significant effects on hippocampal structure and function, particularly in the CA1 region. For example, MS leads to reduced dendritic branching and spine density in CA1 pyramidal neurons, impairing synaptic connectivity(Nakhal et al., 2025) and can impair Long-term potentiation (LTP) (Hao et al., 2025), enhance long term depression (LTD), and reduce the frequency of mEPSCs in pyramidal neurons in the CA1 region (Hu et al., 2024) disrupting memory encoding and flexibility. Maternal separation induces lasting structural, functional, and molecular changes in the CA1 hippocampus, contributing to cognitive and emotional deficits (Albadawi, 2025). These effects highlight the importance of early-life environment in shaping hippocampal health and our result, by showing changes in the volume of interneurons in this area due to MS, is consistent with the above studies, indicating the importance of early life experiences on the formation of the hippocampus and its relationship with neurodevelopmental disorders as soma size is a key determinant of **metabolic capacity, firing properties, and connectivity**, influencing

how the cells shape circuit dynamics (Beaulieu-Laroche et al., 2021). Accordingly, it seems that reduced soma size of PV-INs may affect their specialized roles in inhibition, oscillation generation (e.g., gamma rhythms), and plasticity which finally can have behavioral consequences.

## 5. Conclusion

Taken together, this study showed that MS, which can also be associated with the emergence of autistic-like behaviors in rat according to the previous studies, induces long-lasting impairments in PV-INs in the CA1 area of the hippocampus that is in addition to affecting the number of PV-INs, affects their size and leads to a significant reduction in the soma volume. This reduction in volume may affect the synaptic connections and ultimately the necessary inhibitory effects. This can be considered by researchers as a therapeutic target in autism spectrum disorders and other psychiatric disorders in which the role of interneurons is discussed.

## Data Availability and Supplementary Data

The data presented in this study are available on request from the corresponding author.

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