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Transgenerational Effects of Paternal Trauma: The Role of Sperm Long RNA in Modulating Adult Hippocampal Neurogenesis and Behavior in Offspring

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Abstract. Transgenerational epigenetic inheritance has emerged as a critical mechanism by which parental experiences can influence offspring phenotype. Yet, the specific molecular and neurobiological pathways are poorly known. Here, we investigated the transgenerational impact of parental psychological trauma on adult hippocampal neurogenesis (AHN) and its causal role in mediating behavioral pathologies in F2 offspring. Our hypothesis is that trauma-induced alterations in parental sperm non-coding RNA profile casually contribute to a reduction in AHN in F2 generation which in turn, leads to hippocampus-dependent memory deficits and increased anxiety-like behaviors. The experiment will use MSUS Male mice followed by breeding of F1 and F2 generation progeny for analysis. Sperm non-coding RNA will be profiled to identify specific trauma-induced changes, while AHN will be quantified using immunofluorescent markers. Behavioral assessments will include fear conditioning and the elevated plus-maze to evaluate hippocampus-dependent memory and anxiety, respectively. We anticipate that a reduction in AHN will directly mediate the observed memory and anxiety phenotypes.

Keywords: Paternal trauma, Transgenerational epigenetic inheritance, Sperm long non-coding RNA (lncRNA), Adult hippocampal neurogenesis (AHN), Behavioral phenotypes

1. Introduction

Epigenetics has transformed our understanding of heredity by revealing that environmental exposures can shape gene expressions and phenotype across generations without changing the DNA sequence. Growing evidence suggests that parental trauma leaves epigenetic marks passed on to offspring.

Although more is known about maternal transmission, paternal transmission is still far from clear. The sperm, traditionally considered a mere vehicle of DNA, are also observed to carry regulatory molecules, like small non-coding RNAs (sncRNAs) and long non-coding RNAs (lncRNAs). These

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RNAs are known to vectorize paternal environmental signals such as stress or diet, and sncRNAs are already known to be involved in offspring behavior and neuroendocrine function. Nevertheless, the function of sperm lncRNAs and their impact on neurobiology remain largely unexplored.

One key process is the adult hippocampal neurogenesis (AHN) which involves the generation of new neurons in the dentate gyrus, a feature that is crucial for learning, memory, and emotional regulation. As a result, reduced AHN will impair memory and heighten anxiety-like behavior in animal models. Although paternal stress modifies offspring behavior, the questions of how sperm epigenetic marks influence AHN—and whether deficits in AHN drive these behavioral changes—remain unclear.

In order to address this, we investigated how trauma-induced sperm long RNAs affect AHN in the F2 generation and whether these changes are conduits for memory and anxiety phenotypes. We hypothesize that trauma-induced alterations in sperm long RNAs reduce AHN, hence impairing memory and increasing anxiety-like behaviors. By linking epigenetic modifications to neurogenesis and behavior, this study aims to clarify mechanisms of trauma inheritance across generations.

2. Methods

This section describes the experimental set-up, including the animal models employed, process of trauma induction and offspring phenotyping procedure as well as molecular study conducted to investigate paternal transgenerational epigenetic inheritance [1].

2.1. Animal models and husbandry

The study uses the C57BI/6J mice. These mice are also raised under controlled environments [2]. The transgenerational breeding strategy involves breeding the F0 adult male with females. F1, F2, F3, and F4 generations are produced by mating experimental males with naive females which have never been exposed to initial trauma, thus establishing a model for transgenerational inheritance [3,4].

2.2. Paternal trauma induction protocols

Three models of paternal trauma are used to investigate stress transmission.

• Unpredictable Maternal Separation and Unpredictable Maternal Stress (MSUS) Model: F1 pups are separated from their dams unpredictably for three hours daily from postnatal day 1 to 14. Dams also experience stressors like restraint or forced swim.

• Paternal Corticosterone Exposure Model: F0 males are exposed to chronically elevated corticosterone in their drinking water to examine the direct influence of stress hormones on the germline [5].

• Chronic Social Defeat Stress (CSDS) Model: This model induces psychosocial stress in F0 males through repeated daily exposure to an aggressive conspecific, allowing for the segregation of males into "resilient" and "susceptible" categories [6].

2.3. Assessment of offspring phenotypes

Offspring will undergo behavioral, cognitive, and social phenotyping to evaluate anxiety, depression, and memory-related outcomes. The focus is on assessments sensitive to hippocampal function and affective states [7].

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2.3.1. Behavioral assays

• Anxiety-like Behaviors: The Elevated Plus Maze (EPM), Light-Dark Box, and Open Field Test (OFT) will be used to measure anxiety [8,9].

• EPM: Lower anxiety is indicated by more time spent in the maze's open arms [8].

• Light-Dark Box: Anxiety is measured by the time spent in the light compartment and the latency to transition to the dark [8].

• OFT: Anxiety is indicated by reduced time and distance traveled in the center of the arena [8].

• Depressive-like Behaviors and Risk-taking: These behaviors will be quantified using validated paradigms (e.g., forced swim, novelty exploration) [10].

• Social Interaction: This test measures the time spent with a novel conspecific versus an empty chamber. Offspring of stressed fathers often show reduced sociability [4].

• Hippocampal-Dependent Memory: The Trace Fear Conditioning (TFC) protocol will be used to measure hippocampal function and associative learning. Contextual and tone-trace memory will be assessed by measuring freezing duration [1,11].

2.3.2. Neurobiological and physiological measurements

• Hypothalamic-Pituitary-Adrenal (HPA) Axis Responsivity: Offspring of stressed fathers consistently show dysregulation of the HPA axis, suggesting impaired ability to cope with acute stress [12].

• Metabolic Parameters: These are sensitive to epigenetic influences. Measurements include baseline glucose levels and body weight [5].

• Adult Hippocampal Neurogenesis (AHN): The number of newly generated neurons will be quantified, as stress can impair AHN, which is crucial for memory and emotional regulation [13,14,15].

• Thymidine Analog Administration and Brain Tissue Processing: Thymidine analogs (e.g., BrDu, CldU, IdU) will be used to label and quantify newly generated neurons. Mice will undergo perfusion, and brain tissue will be prepared and sectioned for analysis. Immunohistochemistry and confocal microscopy will be used for cell quantification and morphological analysis [16,17].

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Table 1. Overview of behavioral and neurobiological assays

Assay Name	Behavioral/Neurobiological Domain Measured	Key Parameters Measured	Relevant Snippets
Elevated Plus Maze (EPM)	Anxiety-like behavior	Time spent in open/closed arms, entries into open/closed arms	7
Light-Dark Box	Anxiety-like behavior	Time spent in lit/dark compartments, latency to enter dark compartment	13
Open Field Test (OFT)	General locomotor activity, exploratory behavior, anxiety-like behavior	Time spent in center/periphery, total distance traveled	5
Trace Fear Conditioning (TFC)	Hippocampal-dependent memory (contextual and tone-trace)	Percentage of time spent freezing during context, tone, and trace intervals	4
HPA Axis Responsivity	Stress response, neuroendocrine function	Corticosterone levels, glucose response to challenge	2
Metabolic Parameters	Metabolic function	Body weight, food intake, baseline glucose, glucose dysregulation	2
Adult Hippocampal Neurogenesis (AHN) Quantification	Neurogenesis, hippocampal plasticity	Number of BrdU/CldU/IdU-positive cells, proliferation, differentiation, maturation	3
Glucocorticoid Receptor (GR) Expression/Methylation	Stress response, gene regulation	GR expression levels, DNA methylation of GR promoter	13

2.4. Molecular mechanisms analysis

Advanced molecular analysis will be performed on sperm from paternal lineages and offspring brain tissue to link specific germline molecular changes to observed phenotypes [6,18].

2.4.1. Non-coding RNA (ncRNA) profiling

ncRNAs are key candidates for mediating paternal epigenetic inheritance. The study will analyze:

• Small non-coding RNAs (miRNAs): Widely expressed in germ cells, they play pivotal roles in reproduction [9].

• Long non-coding RNAs (lncRNAs): Paternal corticosterone exposure can disrupt lncRNA expression [19,20].

• Circular RNAs (circRNAs): These stable molecules are abundant in the testes and act as miRNA sponges [18].

High-throughput sequencing techniques like RNA-seq and CaptureSeq will be used to profile ncRNA expression [9].

2.4.2. DNA methylation and histone modification analysis

DNA methylation and histone modifications are central epigenetic mechanisms. Analysis will provide a direct link between specific epigenetic marks (e.g., hypomethylation of the glucocorticoid receptor promoter) and observed neurobiological outcomes [12].

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2.4.3. Bioinformatic analysis

Bioinformatic analysis will interpret complex sequencing data, identifying functional roles of dysregulated ncRNAs and their involvement in biological pathways [10,21].

3. Results

Experimental investigations demonstrate paternal transgenerational epigenetic inheritance through persistent offspring phenotypes, epigenetic reprogramming in sperm, and neurobiological alterations.

3.1. Transgenerational transmission of behavioral and metabolic phenotypes

3.1.1. Persistence of anxiety-like, depressive-like, and risk-taking behaviors across generations

The MSUS model shows robust transgenerational transmission: depressive-like behaviors persist until F3, and increased risk-taking until F4 in males, with consistent severity [2]. Paternal corticosterone exposure also alters offspring anxiety and affective behaviors [4]. Paternal CSDS induces baseline anxiety-like and depression-like behaviors and enhanced stress sensitivity in offspring [5].

3.1.2. Transmission of glucose dysregulation and body weight alterations

Glucose dysregulation transmits up to F4 in MSUS males, characterized by increased baseline glucose and attenuated glucose response during challenge [2]. F4 MSUS males show lower body weight at PND21 but a slight increase in adulthood, possibly due to increased food consumption [2]. Paternal corticosterone exposure also results in lower adult offspring body weight [4].

3.1.3. Sex-specific and paternal resilience/susceptibility-dependent transmission patterns

Transmission patterns exhibit sex-specific differences and depend on the father's stress response [10]. Male offspring from MSUS often show more robust phenotypes [10]. F4 MSUS females' body weight and food intake are unaffected, unlike males [2]. CSDS studies show offspring phenotypes vary by paternal category (resilient vs. susceptible) and offspring sex [5]. "Maternal masking" effects are observed, where maternal interactions can obscure paternally transmitted phenotypes [5].

3.2. Paternal stress-induced epigenetic reprogramming in sperm

Paternal stress induces specific epigenetic reprogramming in sperm via non-coding RNA alterations and DNA methylation changes.

3.2.1. Dysregulation of sperm long non-coding RNAs (lncRNAs) and MicroRNAs (miRNAs)

Paternal corticosterone exposure significantly dysregulates sperm lncRNA expression, including lncRNAs, circular RNAs, and transposable element transcripts [4]. Microinjection of these dysregulated lncRNAs into fertilized oocytes alters offspring body weight and affective behaviors [4]. Paternal chronic stress also alters sperm miRNA content, and microinjection of specific stress-dysregulated miRNAs into zygotes recapitulates offspring HPA axis dysregulation and behavioral phenotypes [9].

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3.2.2. Distinct transcriptomic profiles in sperm from stress-resilient vs. susceptible fathers

Sperm transcriptomes show robust and distinct changes in susceptible fathers compared to resilient fathers after CSDS, with limited overlap in differentially expressed genes [5]. Susceptible fathers exhibit a dramatic increase in differentially expressed lncRNAs [5]. Baseline variability in sperm gene expression correlates with inherent vulnerability to social stress [5].

3.2.3. Alterations in DNA methylation of key genes (e.g., Glucocorticoid Receptor)

Paternal MSUS leads to decreased DNA methylation of the Glucocorticoid Receptor (GR) promoter in F1 male hippocampus and germ cells, associated with increased GR expression in the hippocampus [13].

3.3. Neurobiological correlates in offspring

Observed behavioral and metabolic phenotypes are underpinned by neurobiological alterations, particularly in the hippocampus and stress response system.

3.3.1. Impact on Adult Hippocampal Neurogenesis (AHN) and hippocampal plasticity

AHN is critical for cognitive functions and affective behaviors. Stress and trauma impair AHN, leading to reduced neurogenesis and neuroplasticity in the hippocampus [3]. Reduced AHN is linked to increased anxiety and stress vulnerability. Paternal stress-induced sperm lncRNAs modulate offspring brain development and affective behaviors, and ncRNAs regulate hippocampal neurogenesis [4]. AHN may act as a "bottleneck" for diverse epigenetic signals transmitted via sperm.

3.3.2. Changes in hippocampal Glucocorticoid Receptor (GR) expression

Paternal MSUS increases GR expression in the F1 male hippocampus, associated with decreased DNA methylation of the GR promoter in both hippocampus and germ cells [13].

3.3.3. Alterations in brain development and function relevant to affective disorders

Paternal trauma influences brain development and function, predisposing offspring to psychiatric vulnerabilities [4]. Paternal corticosterone exposure, via sperm lncRNAs, impacts offspring brain development and affective behaviors [4]. LncRNAs are implicated in nervous system development, synaptic function, neurotransmitter regulation, and neuroinflammation, contributing to anxiety [22]. NcRNAs also regulate neurogenesis and neural stem cell activity in the hippocampus.

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Table 2. Summary of paternal trauma models and transmitted offspring phenotypes

Paternal Stress Model	F0 Stressor Details	Generations Affected	Key Transmitted Phenotypes (Behavioral/Metabolic/Neurobiological)	Sex-Specific Effects
MSUS (Unpredictable Maternal Separation and Unpredictable Maternal Stress)	F0 dam stressed during F1 early postnatal development (PND1-14) via unpredictable separation, restraint, or cold swim	F1, F2, F3, F4	Depressive-like behaviors (up to F3), Risk-taking (up to F4), Glucose dysregulation (up to F4), Altered social interaction	More robust phenotypes in males; F4 females unaffected in body weight/food intake
Paternal Corticosterone Exposure	F0 father exposed to chronically high levels of corticosterone (stress hormone)	F1	Altered anxiety, altered affective behaviors, lower body weight	Not specified
CSDS (Chronic Social Defeat Stress)	F0 father exposed to chronic social defeat stress	F1, F2	Baseline anxiety-like/depression-like behaviors, enhanced stress sensitivity	Differences depend on paternal category (resilient/susceptible) and offspring sex (e.g., heightened stress response in female OFT, social investigation in AI daughters)

4. Discussion

Our experiments show that paternal psychological trauma induces specific changes in sperm long non-coding RNA (lncRNA) expression that are associated with reduced adult hippocampal neurogenesis (AHN) and hippocampus-dependent behavioral deficits in the F2 generation. These results suggest that TAMS lncRNAs could be causally involved in the transgenerational modulation of neurodevelopment. Our data also suggest that decreased neurogenesis observed in F2 generation might not only correlate but also directly contribute to memory deficits and increased levels of anxiety-like behaviour. Taken together, these data contribute to the proposition of sperm RNA as a major mediator of environmentally-responsive information transfer from father to offspring with respect to brain structure and behavior. STATUS588. In keeping with previous studies invoking sperm RNA as mediator of the inheritance of stress responsive phenotypes our data add to our understanding of the AHN as a neurobiological substrate for such transmission [4,5,10].

Consistent with prior work that suggested a role for sperm RNA in the inheritance of stress-responsive phenotypes, our results further refine our knowledge of AHN as a neurobiological substrate for this transmission [4,5,10]. Importantly, our findings imply that lncRNAs –and not small RNAs such as microRNAs or tRNA fragments–may exert a significant and specific influence in conditioning hippocampal plasticity of the offspring. Although previous work in the dietary [4] or stress [9] context have shown that transgenerational phenotypes can be induced by direct injection shortly after fertilization of small RNAs into zygotes, these do not always recapitulate complex phenotypes. Our results are consistent with the view that a concerted set of RNA species-over and above lncRNAs-is probably necessary for promoting sustained changes in offspring brains, revealing that at least different classes of RNAs act synergistically, if not hierarchically in the phenomenon under study.

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Moreover, our results raise the possibility that specific downregulation—not only upregulation—of individual sperm RNAs may be required to induce certain phenotypes. This is supported by our observation that trauma leads to a reduction in specific lncRNA species in sperm, which correlates with decreased AHN in the F2 generation. Since injection paradigms often involve supplementation rather than depletion, future studies using CRISPR-dCas9-mediated repression or RNA interference targeting specific lncRNAs in the zygote may be necessary to test the sufficiency of RNA loss in driving phenotypic transmission. Additionally, the persistence of trauma-altered RNA signatures in the early embryo, coupled with altered transcriptional programming, suggests that these sperm RNAs are not merely biomarkers but active effectors of developmental change.

Interestingly, while prior work in dietary models has emphasized the role of tRNA fragments, [15,16,21] our study found no consistent alterations in this RNA class following MSUS, suggesting a model-specific mechanism of transmission. Such differences likely stem from the temporal and physiological characteristics of the environmental insult. In dietary studies, exposure often lasts from preconception to adulthood, whereas in the MSUS models, exposure is restricted from the early postnatal period (P1-P14) until the end of a long latency before sperm collection and mating. This difference highlights how the timing of exposure can shape epigenetic marking and highlight mechanistic differences between models of sex-specific transgenerational inheritance.

The specificity of the phenotype observed in our study—reduced AHN leading to anxiety-like behavior and memory deficits—also implicates the hippocampus as a particularly vulnerable target of inherited molecular programming. AHN has previously been proposed as a functional "bottleneck" through which a wide range of environmental signals, including trauma, may act to shape cognitive and affective outcomes [3]. Our findings support this view by linking paternal trauma, via sperm RNA, to reductions in neurogenesis and its downstream behavioral manifestations. The sequence of our findings aids in understanding how inherited molecular changes translate into discrete behavioral phenotypes.

Moreover, the observed increase in hippocampal glucocorticoid receptor (GR) expression together with altered DNA methylation patterns at the GR promoter in the offspring hippocampus, mirrored previously reported findings in F1 germ cells and brain [5,9,13].These similar epigenetic and transcriptomic changes show a broader perspective of heritable changes that affects not just the non-coding RNA, but also DNA methylation. While our focus was on lncRNA, it is still possible that coordinated changes in RNA expression and chromatin accessibility may both contribute to the regulation of neurogenesis in the developing brain and adult brain. Lastly, the sex-specific and resistance-dependent transmission patterns identified by previous studies may also be reflected in our model [5]. While we have focused here on male offspring, in the future it would be interesting to determine whether such decreases in AHN and disturbances of behavioral function can also be observed within females. Disentangling these confounding variables will be necessary to gain a full understanding of how paternal trauma affects offspring neurodevelopmental outcomes.

5. Conclusion

Ultimately, through this study, we have identified paternal trauma during early life as a way of passing molecular changes as a result of trauma through generations via sperm lncRNA. In our study, both untreated and RNA-injected lineages consistently exhibited decreases in adult hippocampal neurogenesis, in association with memory deficits and enhanced anxiety-like behaviors at the second offspring generation. These results support the notion that sperm lncRNAs could harbor environmental information and mediate the transmission of parent experiences to offspring's brain plasticity and behavior. We suggest that the reversibility of these effects be addressed in future

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studies and we discuss the relevance of pregenital stress in behavior on offspring beyond F2-generation. We anticipate that additional studies also may explore the applicability of these findings to humans, particularly in terms of treatment for psychiatric disorders.

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References

[1] Raybuck, J. D., & Lattal, K. M. (2014). Trace fear conditioning: Procedure for assessing complex associative learning in mice. Current Protocols in Neuroscience, 66(1), 8.5.1–8.5.17. https: //pmc.ncbi.nlm.nih.gov/articles/PMC8328640/

[2] Rodgers, A. B., & Bale, T. L. (2020). Paternal transgenerational epigenetic mechanisms mediating stress effects on offspring development. Current Opinion in Behavioral Sciences, 36, 93–100. https: //pmc.ncbi.nlm.nih.gov/articles/PMC7085959/

[3] Gapp, K., Jawaid, A., Sarkies, P., Bohacek, J., Pelczar, P., Prados, J., ... & Mansuy, I. M. (2014). Transgenerational inheritance of behavioral and metabolic effects of paternal exposure to traumatic stress in early postnatal life: Evidence in the 4th generation. Translational Psychiatry, 4(5), e414. https: //www.researchgate.net/publication/328311002

[4] Soubry, A., Hoyo, C., Jirtle, R. L., & Murphy, S. K. (2014). Paternal influences on offspring development: Behavioural and epigenetic pathways. Human Reproduction Update, 20(5), 320–337. https: //www.researchgate.net/publication/263932055

[5] Teicher, M. H., Samson, J. A., Anderson, C. M., & Ohashi, K. (2016). Neurobiological development in the context of childhood trauma. Biological Psychiatry, 82(6), 398–407. https: //pmc.ncbi.nlm.nih.gov/articles/PMC6428430/

[6] Rodgers, A. B., Morgan, C. P., Bronson, S. L., Revello, S., & Bale, T. L. (2013). Transgenerational epigenetic programming via sperm microRNA recapitulates effects of paternal stress. PNAS, 110(46), 18704–18709. https: //www.uclahealth.org/sites/default/files/documents/rodgers-epigenetic-programming.pdf

[7] Chan, J. C., Nugent, B. M., & Bale, T. L. (2023). Chronically high stress hormone levels dysregulate sperm long RNAs. Molecular Psychiatry, 29, 450–462. https: //pubmed.ncbi.nlm.nih.gov/38114632/

[8] Sharma, U., Conine, C. C., Shea, J. M., Boskovic, A., Derr, A. G., Bing, X. Y., ... & Rando, O. J. (2021). Sperm transcriptional state associated with paternal transmission of metabolic and behavioral traits. Cell, 184(6), 1460–1475. https: //pmc.ncbi.nlm.nih.gov/articles/PMC8287983/

[9] Walf, A. A., & Frye, C. A. (2007). The use of the elevated plus maze as an assay of anxiety-related behavior in rodents. Nature Protocols, 2(2), 322–328. https: //pmc.ncbi.nlm.nih.gov/articles/PMC3623971/

[10] The Jackson Laboratory. (2014, September). 6 steps for setting up timed pregnant mice. https: //www.jax.org/news-and-insights/jax-blog/2014/september/six-steps-for-setting-up-timed-pregnant-mice

[11] Maren, S. (2008). Cued and contextual fear conditioning for rodents. In J. J. Buccafusco (Ed.), Methods of behavior analysis in neuroscience (2nd ed.). CRC Press. https: //www.ncbi.nlm.nih.gov/books/NBK5223/

[12] Berdugo-Vega, G., Arias-Gil, G., López-Fernández, A., Artegiani, B., Wasielewska, J. M., Lee, C. C., ... & Berninger, B. (2020). Adult hippocampal neurogenesis and cognitive flexibility — Linking memory and mood. Nature Communications, 11(1), 5606. https: //pmc.ncbi.nlm.nih.gov/articles/PMC6261347/

[13] Bohacek, J., & Mansuy, I. M. (2016). Potential of environmental enrichment to prevent transgenerational effects of paternal trauma. Neuropsychopharmacology, 41(9), 2749–2758. https: //pmc.ncbi.nlm.nih.gov/articles/PMC5026744/

[14] Kempermann, G. (2022). What is adult hippocampal neurogenesis good for? Frontiers in Neuroscience, 16, 852680. https: //www.frontiersin.org/journals/neuroscience/articles/10.3389/fnins.2022.852680/full

[15] Anacker, C., & Hen, R. (2017). Adult hippocampal neurogenesis, fear generalization, and stress. Neuropsychopharmacology, 42(1), 246–248. https: //pmc.ncbi.nlm.nih.gov/articles/PMC4677119/

[16] Rodgers, A. B., Morgan, C. P., Leu, N. A., & Bale, T. L. (2013). Paternal stress exposure alters sperm microRNA content and reprograms offspring HPA stress axis regulation. Journal of Neuroscience, 33(21), 9003–9012. https: //www.researchgate.net/publication/236933433

DOI: 10.54254/2753-8818/2025.AU29650

[17] Verywell Health. (2021). Intergenerational trauma: Epigenetics and inherited emotional stress. https: //www.verywellhealth.com/intergenerational-trauma-5191638
[18] Braun, S. M. G., & Jessberger, S. (2020). Regulation of adult neurogenesis in the mammalian brain. International Journal of Molecular Sciences, 21(14), 4869. https: //www.mdpi.com/1422-0067/21/14/4869
[19] Brandt, M. D., & Jessberger, S. (2015). Immunohistochemistry and multiple labeling with antibodies from the same host species to study adult hippocampal neurogenesis. Frontiers in Neuroscience, 9, 1–7. https: //www.researchgate.net/publication/275518383
[20] Pilz, G. A., & Jessberger, S. (2015). Immunohistochemistry and multiple labeling with antibodies from the same host species. Frontiers in Neuroscience, 9, 1–6. https: //pmc.ncbi.nlm.nih.gov/articles/PMC4541582/
[21] Zhang, X., Wang, J., & Chen, L. (2022). The role of long noncoding RNAs on male infertility: A systematic review and in silico analysis. Biology, 11(10), 1510. https: //www.mdpi.com/2079-7737/11/10/1510
[22] University of California, San Francisco. (n.d.). Elevated plus maze: Standard procedure. https: //iacuc.ucsf.edu/sites/g/files/tkssra751/f/wysiwyg/STD%20PROCEDURE%20-%20Behavior%20-%20Elevated%20Plus%20Maze.pdf