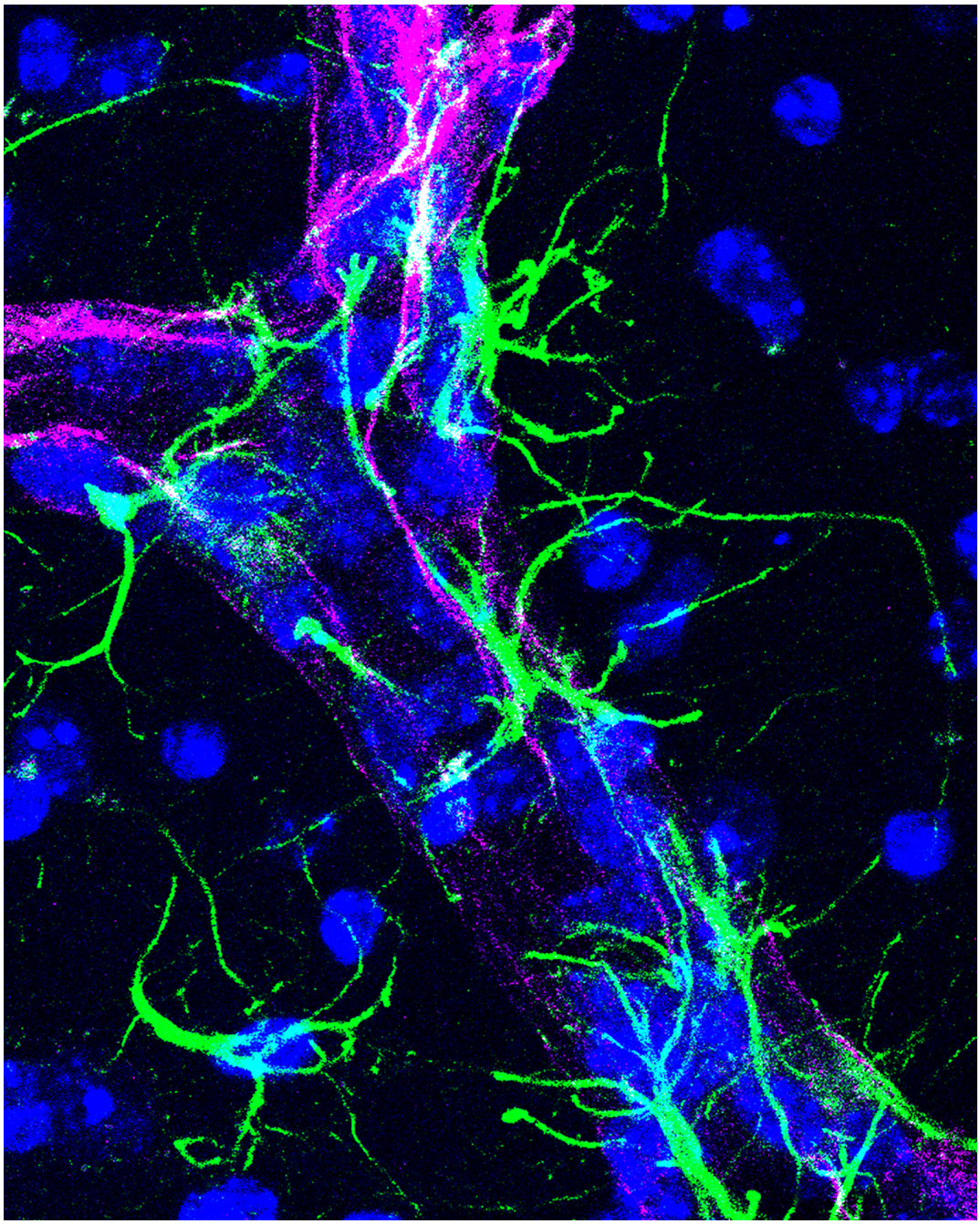
SNE Impacts 2025



Blood–brain barrier in the preoptic area of adult male mouse. Astrocytic endfeet (GFAP, green) interact closely with the pericapillary basement membrane (laminin, magenta) ; nuclei are highlighted in blue (Hoechst).
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A summary of breakthroughs in Neuroendocrinology in 2025
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Elodie Desroziers (LinkedIn), Giuseppe Gangarossa (Bluesky),
Marialetizia Rastelli, Clara Sanchez (Facebook/Instagram), Pieter Vancamps

List of selected papers

Barelle PY, Schaller F, Park S, Caron E, Klucznik J, Ciofi P, Muscatelli F, and Bouret SG. Oxytocin neurons drive melanocortin circuit maturation via vesicle release during a neonatal critical period. ***PLoS Biol.*** *2025 Nov 12;23(11):e3003158. doi: 10.1371/journal.pbio.3003158. eCollection 2025.*

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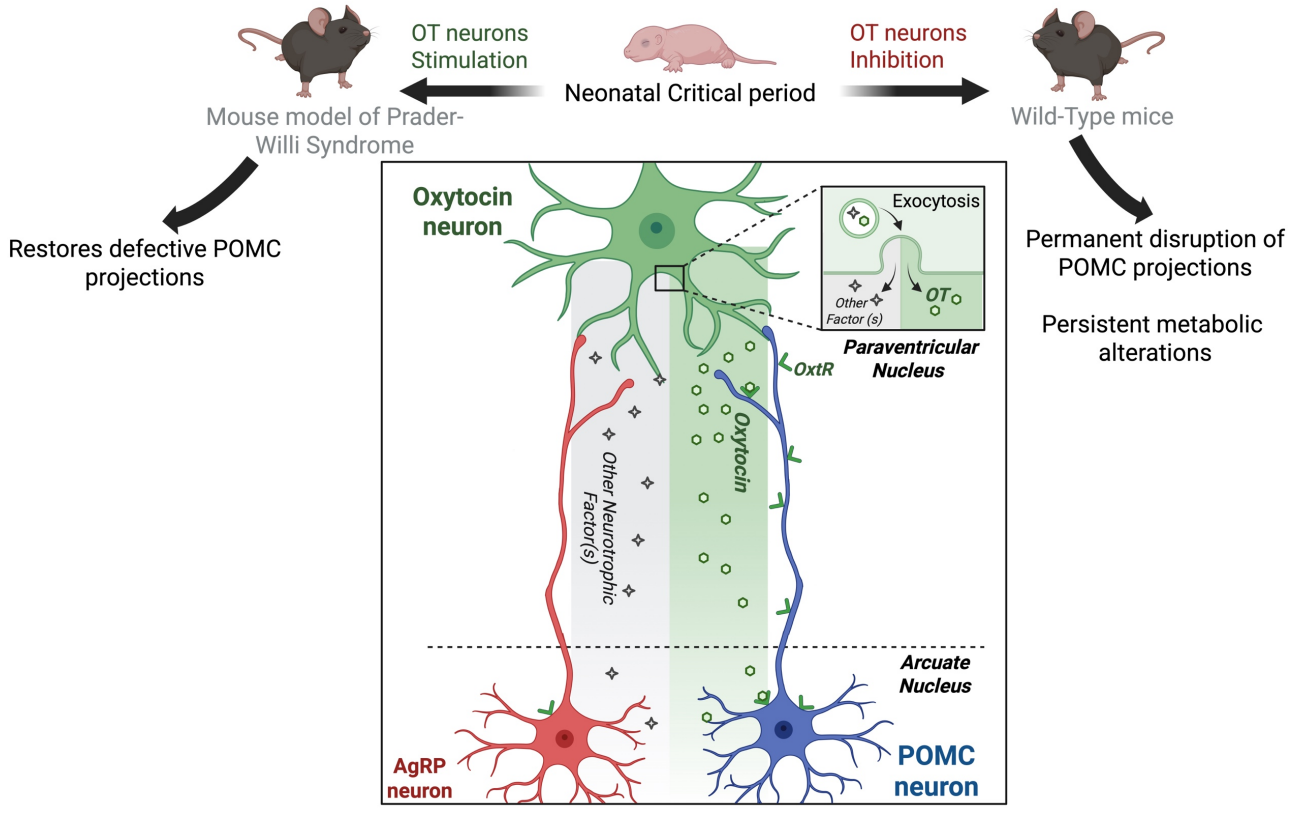
Oxytocin neurons shape hypothalamic feeding circuits during a neonatal critical period

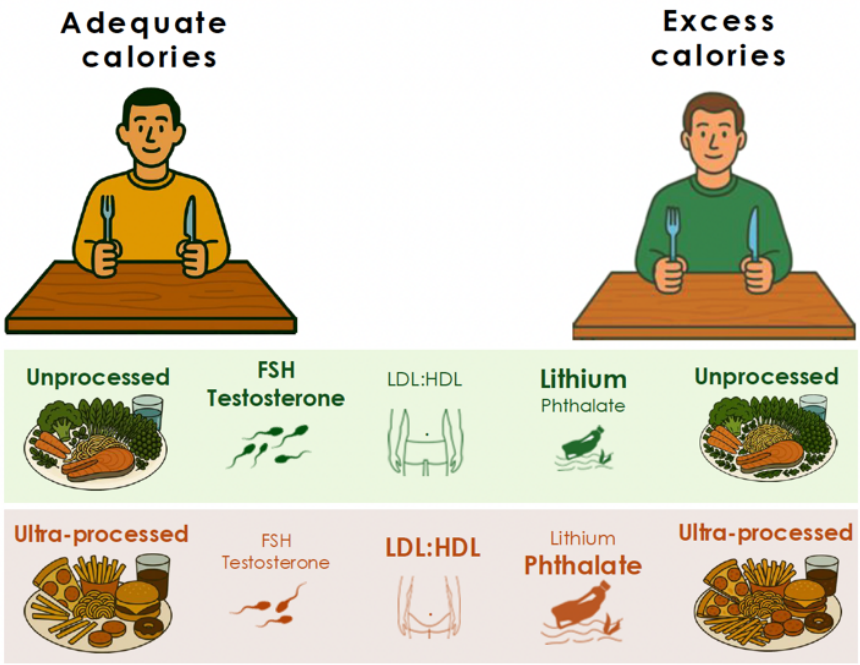
The hypothalamic melanocortin system is a central regulator of energy balance and feeding behavior, yet the mechanisms governing its developmental wiring remain incompletely understood. Oxytocin (OT), classically known for its roles in parturition and social behavior, has recently emerged as a key developmental signal with long-lasting metabolic consequences. In this study, Barelle and colleagues demonstrate that OT neurons actively orchestrate the maturation of melanocortin circuits during a restricted neonatal critical period. Using chemogenetic and genetic approaches in mice, they showed that transient inhibition of OT neurons during the first postnatal week selectively disrupted pro-opiomelanocortin (POMC) and agouti-related peptide projections to the paraventricular nucleus of the hypothalamus, without affecting other target regions. These developmental effects were age-dependent, as similar manipulations during juvenile or adult life had no impact on melanocortin connectivity. Mechanistically, they revealed that OT neurons influence melanocortin circuits' formation through SNARE-dependent vesicular release and OT receptor signaling. Consistent with these findings, pharmacological blockade of OT receptors during neonatal life reproduced the structural defects and led to persistent metabolic alterations in adulthood. Notably, neonatal chemogenetic activation of OT neurons restored defective POMC projections in a *Magel2* KO mouse model of Prader-Willi Syndrome, highlighting the pathological relevance of this mechanism. Together, these findings identify OT as a paracrine neurotrophic signal that programs hypothalamic feeding circuits during early life, providing new insights into the developmental origins of metabolic disorders and opening perspectives for time-specific therapeutic interventions.



Barelle PY, Schaller F, Park S, Caron E, Klucznik J, Ciofi P, Muscatelli F, and Bouret SG. Oxytocin neurons drive melanocortin circuit maturation via vesicle release during a neonatal critical period. ***PLoS Biol.*** *2025 Nov 12;23(11):e3003158. doi: 10.1371/journal.pbio.3003158. eCollection 2025.*

Effect of ultra-processed food consumption on male reproductive and metabolic health

It's not just the calories – ultra-processed foods affects our health. The consumption of Ultra-processed foods is associated with poor health. In this carefully controlled nutrition trial, researchers explored whether these foods directly harm metabolism and reproductive health, and test if these effect happen even if people don't eat more calories. Volunteers participated in a study where they alternated between eating unprocessed foods (like fruits, vegetables, and minimally prepared meals) and ultra-processed ones, under a tightly monitored diet plan. The results were revealing: participants gained weight and showed a higher LDL-to-HDL cholesterol ratio (a marker linked to heart disease) when eating ultra-processed foods—regardless of how many calories they consumed. Several key hormones related to energy balance and sperm production changed too. Levels of growth/differentiation factor 15, which helps regulate metabolism, and follicle-stimulating hormone (important for sperm development) decreased. Sperm motility—the ability of sperm to swim properly—tended to worsen after the ultra-processed diet. The study also found that chemical pollutants accumulated differently depending on diet type. For example, blood levels of lithium dropped, while certain plastic-related compounds (phthalates) tended to rise after the ultra-processed phase. Interestingly, simply changing how many calories participants ate produced distinct metabolic and hormonal effects, showing that calorie intake and food processing affect the body in different ways. In summary, this trial provides strong evidence that ultra-processed foods can harm both metabolic and reproductive health—even without overeating—highlighting the importance of food quality, not just quantity, in maintaining wellbeing.



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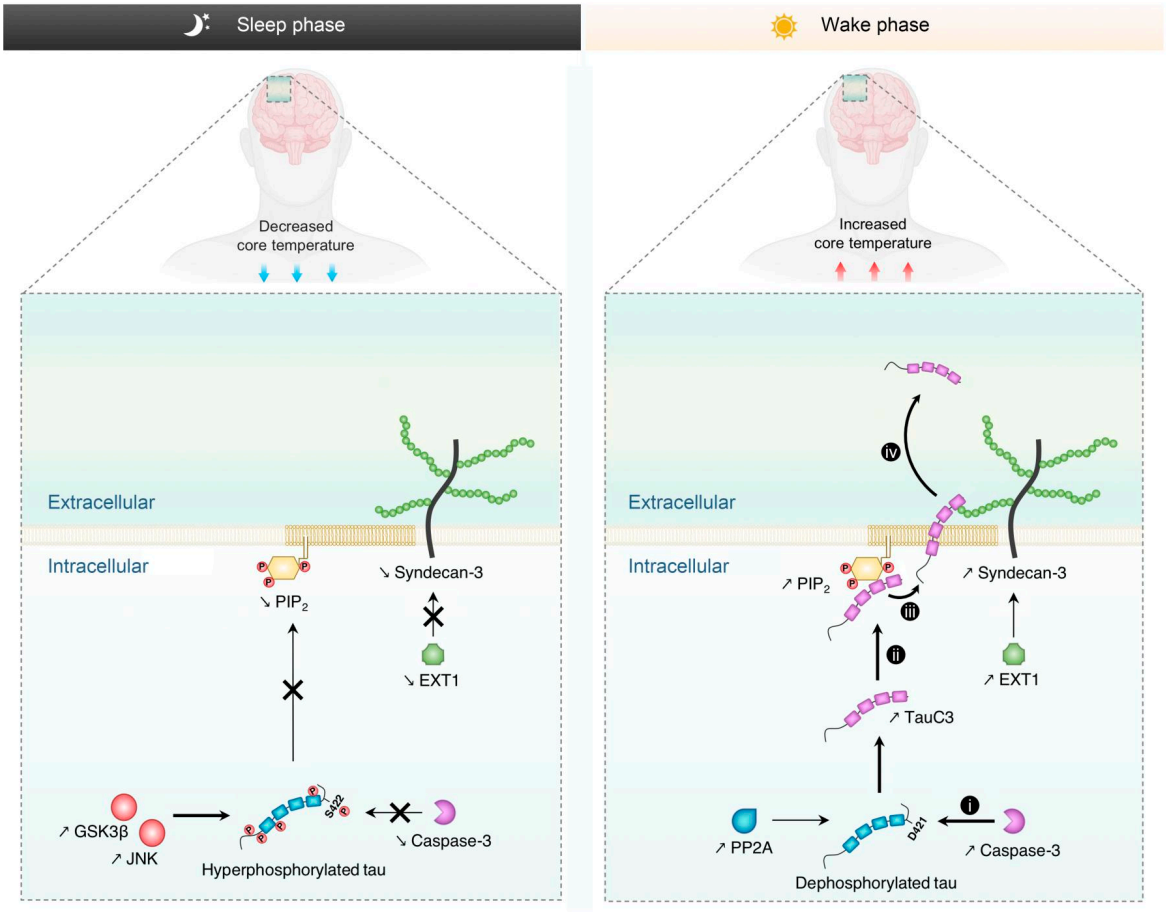
Sleep-wake variation in body temperature regulates tau secretion and correlates with CSF and plasma tau

Sleep disturbance is bidirectionally associated with an increased risk of Alzheimer's disease and other tauopathies. While the sleep-wake cycle regulates interstitial and cerebrospinal fluid (CSF) tau levels, the underlying mechanisms remain unknown. Understanding these mechanisms is crucial, given the evidence that tau pathology spreads through neuron-to-neuron transfer, involving the secretion and internalization of pathological tau forms. Here, we combined in vitro, in vivo, and clinical methods to reveal a pathway by which changes in body temperature (BT) over the sleep-wake cycle modulate extracellular tau levels. In mice, a higher BT during wakefulness and sleep deprivation increased CSF and plasma tau levels, while also upregulating unconventional protein secretion pathway I (UPS-I) events including (a) intracellular tau dephosphorylation, (b) caspase 3–mediated cleavage of tau (TauC3), and (c) membrane translocation of tau through binding to phosphatidylinositol 4,5-bisphosphate (PIP_2) and syndecan 3. In humans, the increase in CSF and plasma tau levels observed after wakefulness correlated with BT increases during wakefulness. By demonstrating that sleep-wake variation in BT regulates extracellular tau levels, our findings highlight the importance of thermoregulation in linking sleep disturbances to tau-mediated neurodegeneration and the preventative potential of thermal interventions.



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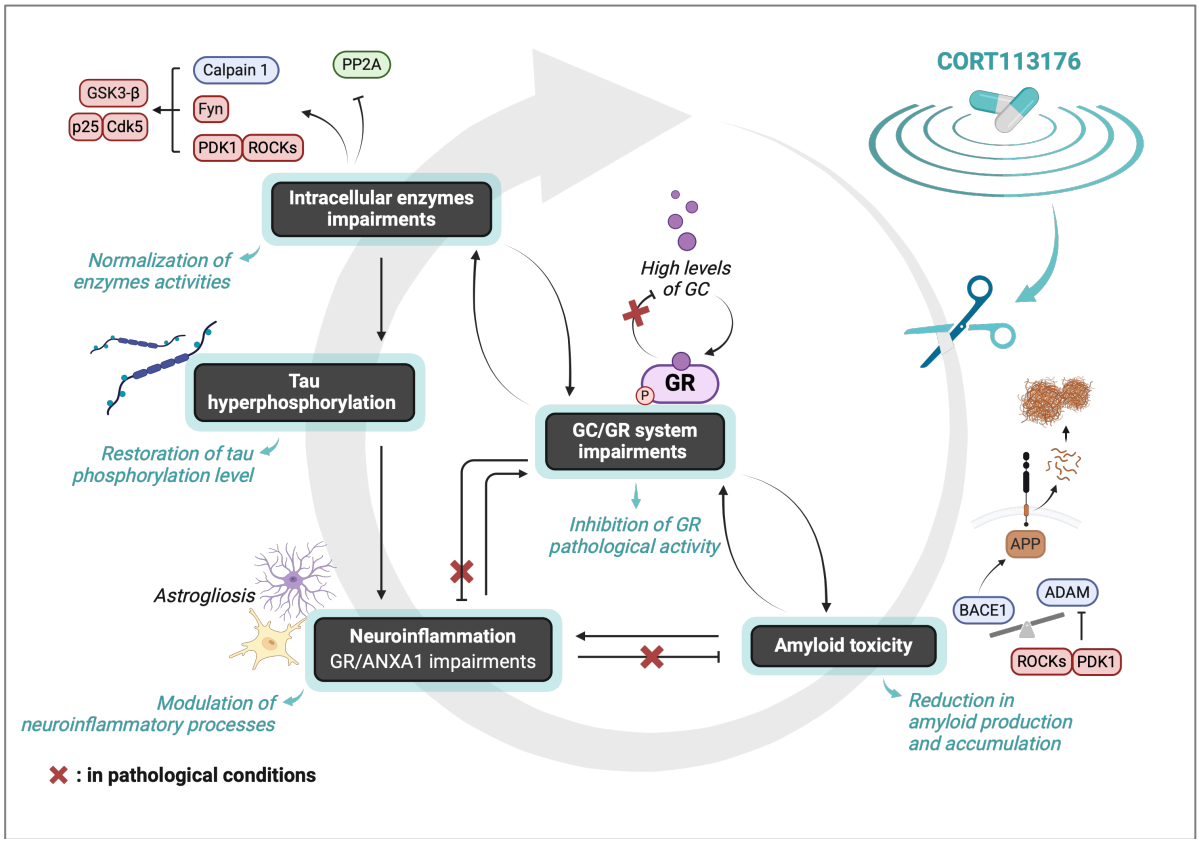
Advancing Alzheimer's pharmacotherapy: Efficacy of glucocorticoid modulation with CORT113176 (Dazucorilant®) in preclinical mouse models

Exposure to chronic stress and high levels of glucocorticoid (GC) hormones in adulthood has been associated with cognitive deficits and an increased risk of Alzheimer's disease (AD). CORT113176 has recently emerged as a selective glucocorticoid receptor (GR) modulator, exhibiting efficacy in counteracting amyloid-β toxicity in an acute model of AD. This study aims to assess the therapeutic potential of CORT113176 in reversing amyloid and tau pathologies through the inhibition of GR pathological activity, and providing additional evidence for its consideration in AD treatment. The efficacy of CORT11376 was evaluated in two transgenic mouse models of amyloid pathology: the slowly progressing J20 and the aggressively pathological 5xFAD mice. Behavioral analysis was conducted to evaluate welfare, cognitive performances and anxiety levels. The activity of the GR system, neuroinflammation, amyloid burden, and tau phosphorylation were examined in hippocampi. In both AD mouse models, chronic treatment with CORT113176 improved working and long-term spatial memories along with the inhibition of GR-dependent pathogenic processes and the normalization of plasma GC levels. CORT113176 treatment also resulted in a reduction in tau hyperphosphorylation and amyloid production and aggregation. Additionally, CORT113176 seemed to mediate a specific re-localization of activated glial cells onto amyloid plaques in J20 mice, suggesting a restoration of physiological neuroinflammatory processes. CORT113176 exhibited sustained disease-modifying effects in two AD models. Given that this compound has already demonstrated safety and tolerability in human subjects, our results provide pre-clinical support for conducting clinical trials to evaluate its potential in AD.



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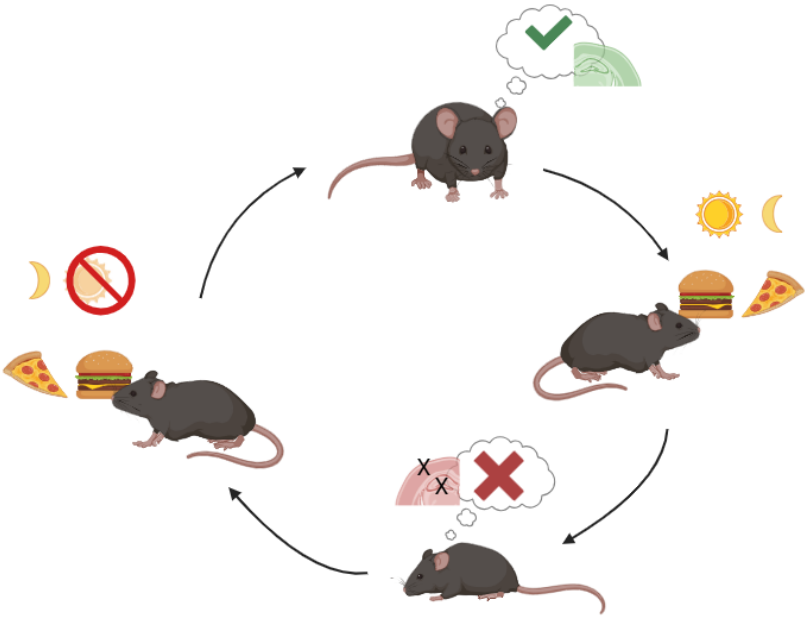
Mind it – meal scheduling also matters

An emerging hypothesis is challenging a central tenet of nutritional science—which constantly told us the contents of our diet are all that matter. New research suggests that the timing of our meals may be a powerful, untapped lever for cognitive health. By simply restricting access to high-fat, high-sugar foods during the body's active period, it may be possible to mitigate—and even reverse—some of the most damaging effects of an unhealthy diet on brain functioning. This study focused on the transition between adolescence to adulthood in mice fed for 8-weeks with unlimited unhealthy food since weaning, and then either continued with the same regimen for another 4-weeks or solely during the active period of the day. The 14-hours fasting was associated with better activity coupling between the sensory cortex and dorsal hippocampus, both of which are necessary to provide the attention necessary for remembering. Engram size and connectivity abnormally grew in the hippocampus and shrunk in the sensory cortex on unlimited junk foods, an effect that reverted to normal thanks to the 14-hours fasting during the inactive period of the day despite similar calorie intake. Aligning our meals "even unhealthy" with the body's clock respects circadian secretions of hormones like the stress hormone corticosterone that we find necessary for neuronal connectivity and functioning. The ramifications could be profound if replicated in humans, reshaping medical advice for metabolic disorders and guiding new treatments for obesity-related cognitive decline because when calories go "in-versus-out" also matters beyond just their quantity.



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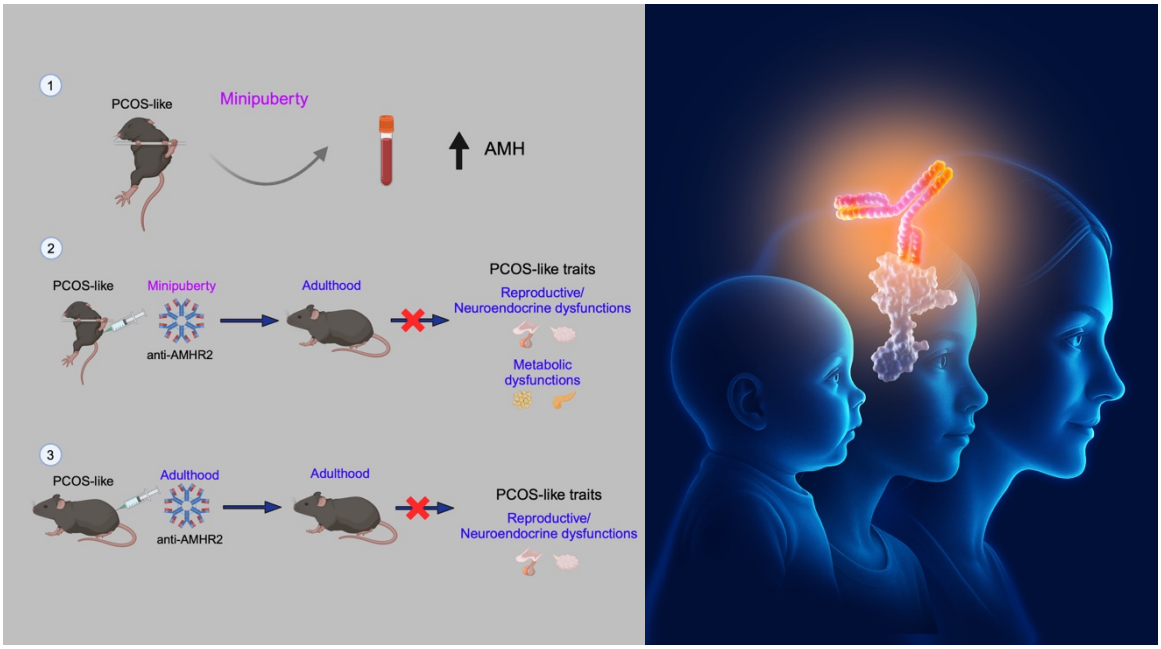
Stopping PCOS before it starts: A new clue from early life

Polycystic ovary syndrome (PCOS) is a common hormonal disorder affecting millions of women worldwide, often leading to irregular menstrual cycles, infertility, weight gain, and increased risk for type 2 diabetes and heart disease. Despite its prevalence, there are currently no treatments that target the root causes of PCOS, only therapies that help manage symptoms. This new study used mouse models to explore how a hormone called anti-Müllerian hormone (AMH) influences the development of PCOS. AMH is best known for its role in early reproductive development. Researchers found that exposure to high levels of AMH shortly after birth (during “minipuberty”) caused animals to develop reproductive and metabolic problems that resemble PCOS in humans. Importantly, the team showed that these effects could be prevented or reversed by blocking the AMH signaling pathway using a specific antibody. When this treatment was given during minipuberty, the animals developed normally; when given in adulthood, many PCOS-like symptoms were reduced. These findings suggest that elevated AMH plays a direct role in triggering PCOS traits and that targeting AMH signaling could be a promising new strategy for preventing and treating PCOS, rather than just addressing symptoms. If similar mechanisms exist in humans, this research could point toward future therapies that fundamentally change how PCOS is treated.



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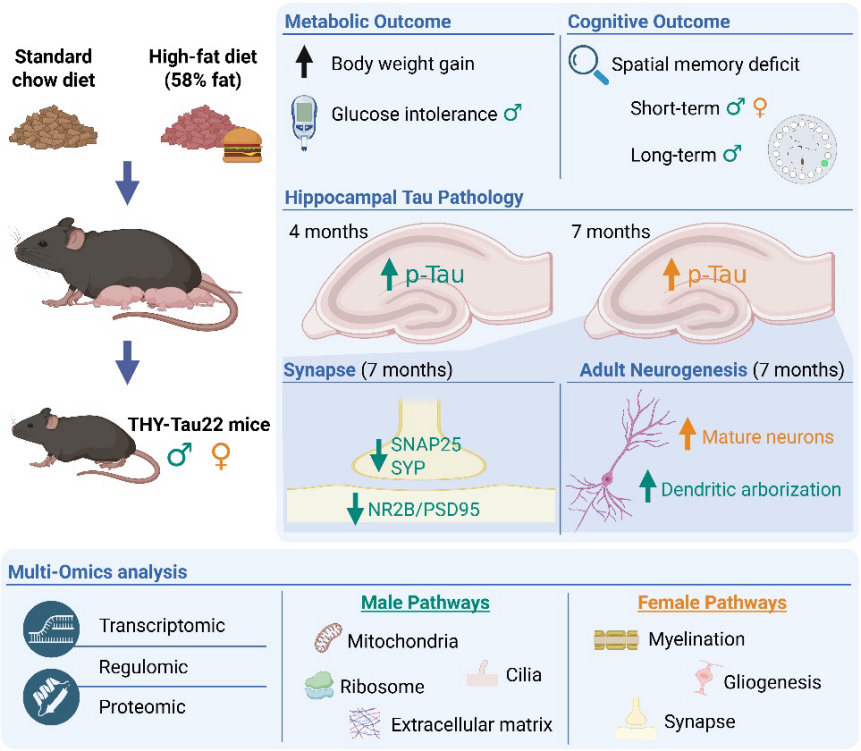
Sex-dependent effects of maternal high-fat diet during lactation in the offspring of adult THY-Tau22 mice

This study investigates how a maternal high-fat diet (HFD) during lactation influences the development of tauopathy in THY-Tau22 mice, a model of progressive tau pathology and cognitive decline. Dams were fed either a standard chow diet or a high-fat diet (58% fat) throughout lactation. After weaning, all offspring received a standard diet and were examined at 4 months (onset of tau pathology) or 7 months (onset of cognitive impairment). Maternal HFD increased body-weight gain in offspring at weaning (post-natal day 21) and induced mild glucose intolerance in males at 3 months. At 6 months, it impaired spatial memory in both sexes, with males showing stronger deficits. These cognitive impairments were associated with increased hippocampal tau phosphorylation, appearing earlier in males (4 months) than in females (7 months), indicating a sex-specific temporal shift. Maternal HFD also altered adult hippocampal neurogenesis: females showed increased numbers of mature neurons, whereas males exhibited enhanced dendritic arborization. Synaptic analyses revealed synaptic loss exclusively in males. Multi-omics profiling demonstrated long-lasting, sex-dependent effects on transcriptomic, proteomic, and regulomic landscapes. Males showed predominant alterations in mitochondrial, ribosomal, ciliary, and extracellular-matrix pathways, while females displayed changes in gliogenesis, myelination, and synaptic plasticity. Overall, maternal HFD accelerates tauopathy development in offspring, with males being affected earlier than females, highlighting the perinatal period as a critical window of vulner ability—and potential intervention.



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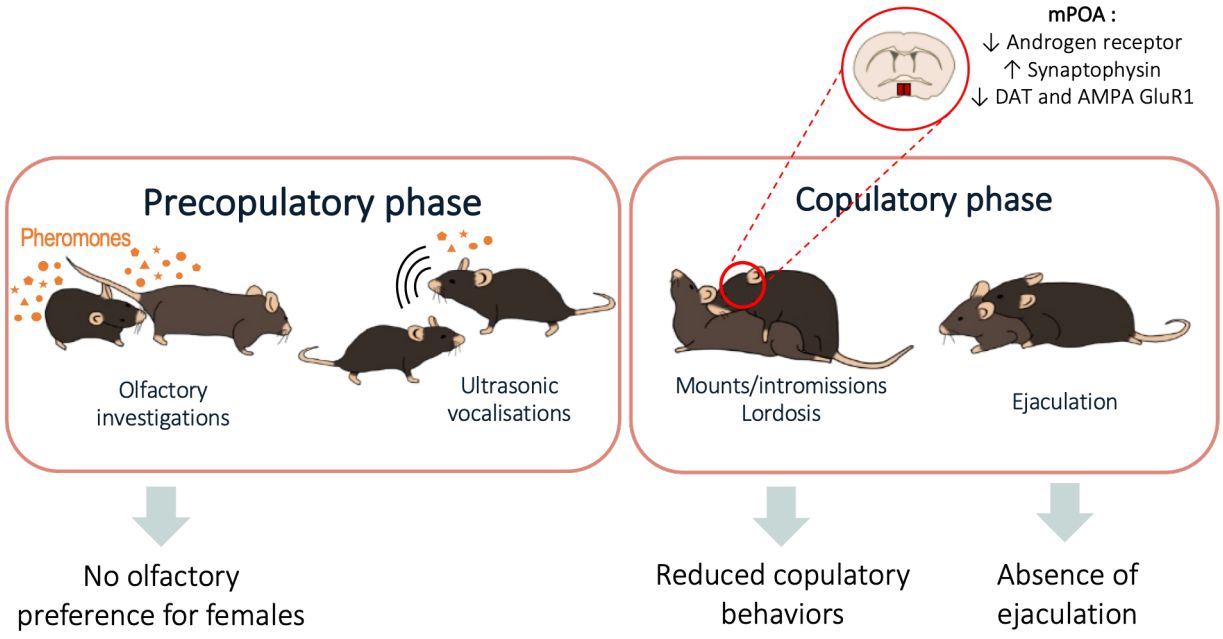
Sexual behavior is worsened by phthalate exposure in male mice fed with an obesogenic diet

The decline in male fertility reported in Western countries since the early 20th century is a major health public concern. The incriminated factors include genetic causes, but also dietary changes and exposure to industrial chemicals, notably endocrine disruptors such as phthalates. This study investigated the combined effects of a high-fat/high-fructose (HFHF) diet and environmentally relevant phthalate exposure on male sexual behavior in adult C57Bl/6J mice. For this purpose, 4 groups of animals were fed with either a standard or HFHF diet in the absence or presence of a mixture (Mix) of phthalates (DEHP 5 µg/kg/d, DBP 0.5 µg/kg/d, BBP 0.5 µg/kg/d, DiBP 0.5 µg/kg/d, DEP 0.25 µg/kg/d).
The obtained data confirmed metabolic alterations and weight gain in HFHF and HFHF/Mix animals. Interestingly, the combined HFHF diet and phthalate exposure highly impaired sexual behavior, as evidenced by the hugely increased mating latency or absence of mating together with impaired olfactory discrimination toward females, without any effect on general activity. These behavioral alterations were associated with changes in the levels of pre- and post-synaptic proteins (synaptophysin, dopamine transporter, and AMPA GluR1) in the medial preoptic area of the hypothalamus, a key region controlling male sexual behavior. Together, these findings reveal a high vulnerability of the neural circuitry underlying male sexual behavior to combined HFHF diet and phthalate exposure at doses relevant for the human exposure, highlighting important implications for public health.



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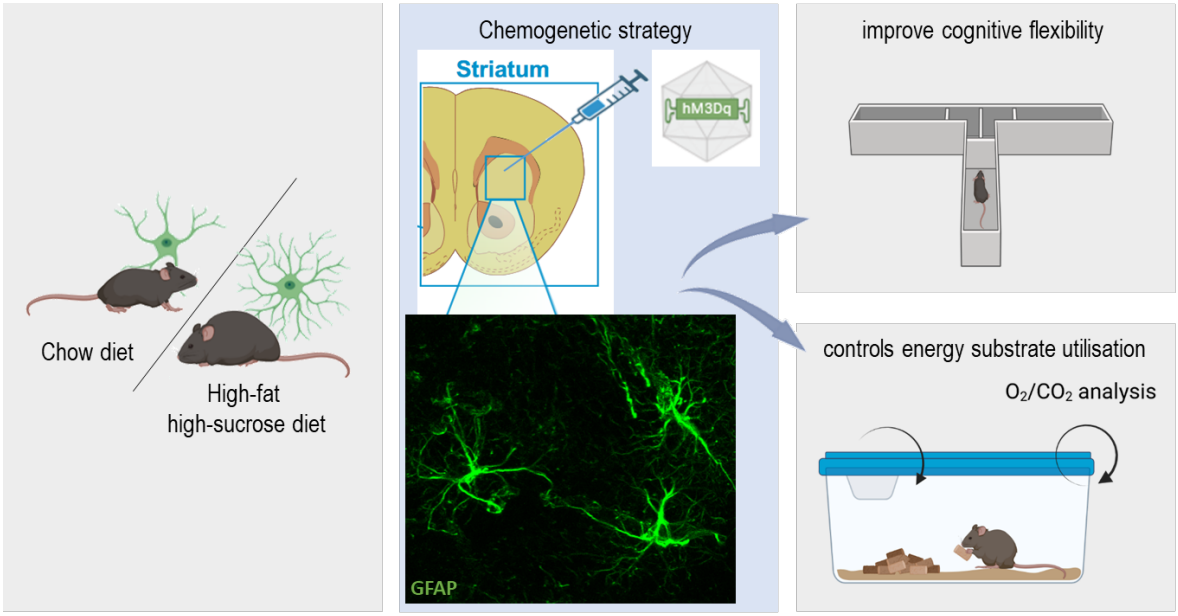
Striatal astrocytes modulate behavioral flexibility and whole-body metabolism in mice

Obesity and the consumption of high caloric food affect brain cells among which neurons and astrocyte. While the impact of enriched food on the physiology of astrocytes has been studied in the hypothalamus, the role of these cells in other neural networks affected by obesity remains poorly understood. In this study, we investigated the role of astrocytes in the striatum, which is part of the dopaminergic reward circuit, in the context of diet-induced obesity. We observed that a high-fat, high-sugar diet leads to astrocyte reactivity, characterized by morphological changes and altered calcium flow dynamics. This may affect communication between astrocytes and neurons within the structure. Using chemogenetics approaches to manipulate striatal astrocytes we observed that acute activation of calcium signaling in dorso-striatal astrocytes is sufficient to restore obesity-associated impairment independently of body weight loss. Furthermore, we demonstrate that manipulation of striatal astrocytes exerts a direct control over animal energy metabolism by influencing the metabolic substrate (lipids or carbohydrates) utilized by the mouse to maintain its metabolism. Given their potential as therapeutic targets, however, astrocytes may offer promising avenues for treating metabolic disorders. Future work will focus on identifying the exact role of the brain regions associated with reward and motivation in energy metabolism, as well as the role of astrocytes in these processes.



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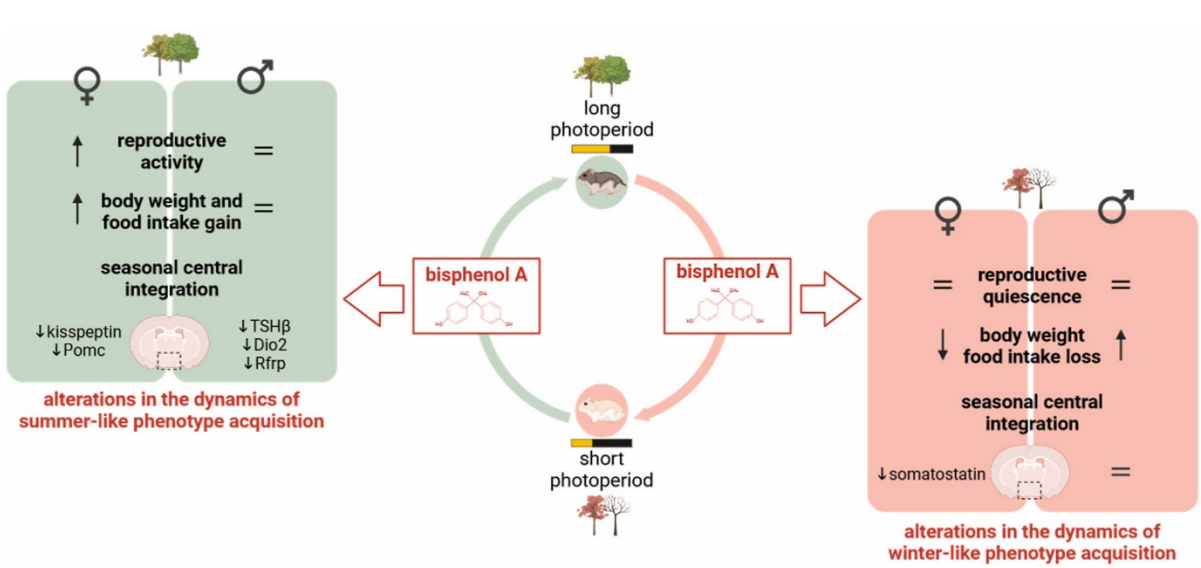
When plastic chemicals disrupt seasonal biological rhythms

Chemical pollution has risen dramatically in recent decades, releasing thousands of substances into the environment. Among them, endocrine-disrupting chemicals (EDCs) can interfere with hormonal systems and contribute to reproductive and metabolic disorders. While most studies on EDCs relies on traditional rodents, much less is known about how these pollutants affect wildlife species whose physiology is tightly linked to natural seasonal cycles. In temperate regions, most mammals adjust their reproductive and metabolic timing to seasonal changes so that offspring are born under favourable conditions, a process govern by a central photoneuroendocrine pathway. In this study, the Djungarian hamster, a species that adapts its physiology in response to day-length (photoperiod) variations, was used to assess how exposure to Bisphenol A (BPA), a widespread EDC found in plastics and resins, affects the neuroendocrine regulation of seasonal biology. The results show that adult BPA exposure alters the dynamics of reproductive and metabolic adaptations triggered by photoperiodic variations, with effects depending on sex. During a long- to short-day transition (winter-like shift), BPA-exposed females exhibited a delayed onset of the winter metabolic phenotype, associated with decreased hypothalamic expression of somatostatin, a neuropeptide involved in seasonal body-weight regulation. In contrast, BPA-exposed males developed winter metabolic features more rapidly. During the reverse transition (mimicking summer), BPA-exposed females adapted more quickly to long-day reproductive and metabolic states, accompanied by altered expression dynamics of hypothalamic kisspeptin and proopiomelanocortin. In males, BPA reduced the expression of key photoperiodic signalling molecules without impairing acquisition of the long-day phenotype. These pioneer findings reveal that mammalian seasonal physiology and its neuroendocrine mechanisms represent sensitive targets of EDCs.



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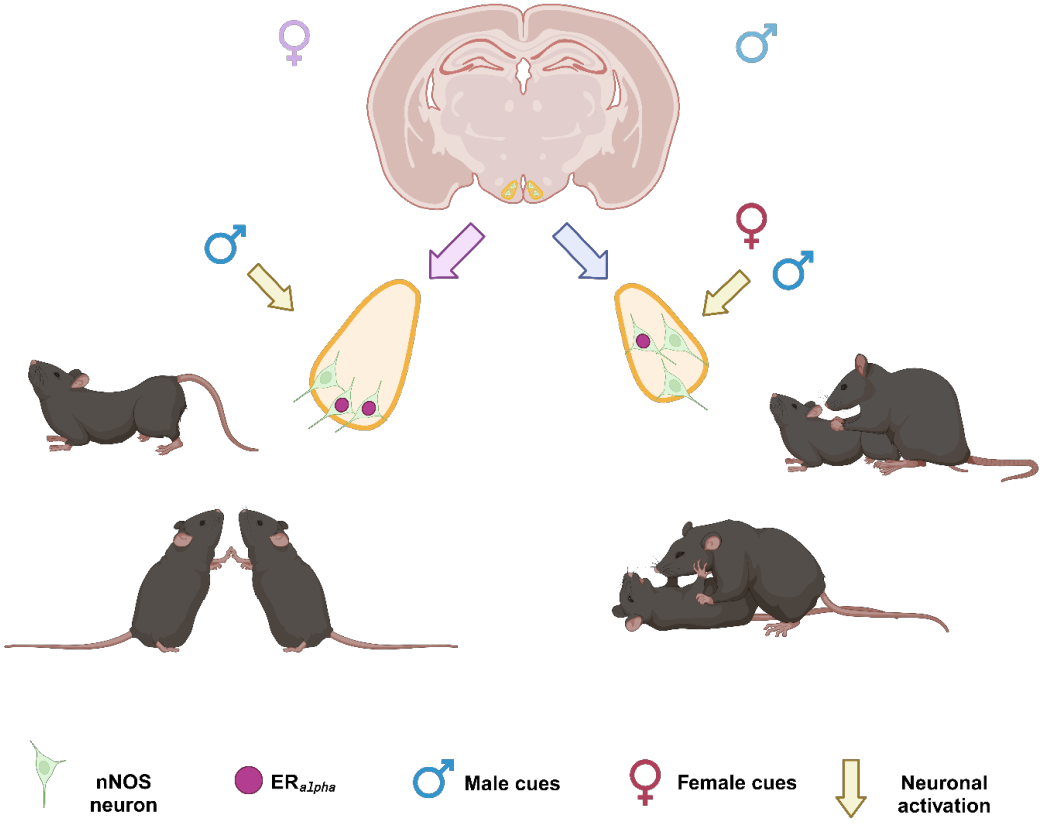
Ventromedial hypothalamus (VMHvl) nNOS neurons regulate social behaviors in a sex-specific manner

Neuronal nitric oxide synthase (nNOS) neurons are ubiquitously spread in the mouse brain. Data using knockouts and pharmacology have revealed that nNOS is essential for the display of sexual and aggressive behavior. Yet, the specific neuronal populations regulating those behaviors remain elusive. Here, we aim to study the role of the ventromedial hypothalamus (VMHvl)-nNOS neurons in social behaviors in both sexes. First, we evaluate whether the expression of nNOS overlaps with the well-characterized estrogen receptor alpha (ERα+)-VMHvl population. Next, we assess how different social stimuli affected VMHvl-nNOS neurons' activity. Lastly, we use transgenic mice and viral approaches to ablate VMHvl-nNOS neurons and evaluate their impact on behavior. Our findings suggest that nNOS neurons constitute a small cluster within the VMHvl-ERα+ population that regulates social behaviors in a sex-specific manner. In males, those neurons seem to be essential for aggression, whereas in females for sexual behavior and social motivation.



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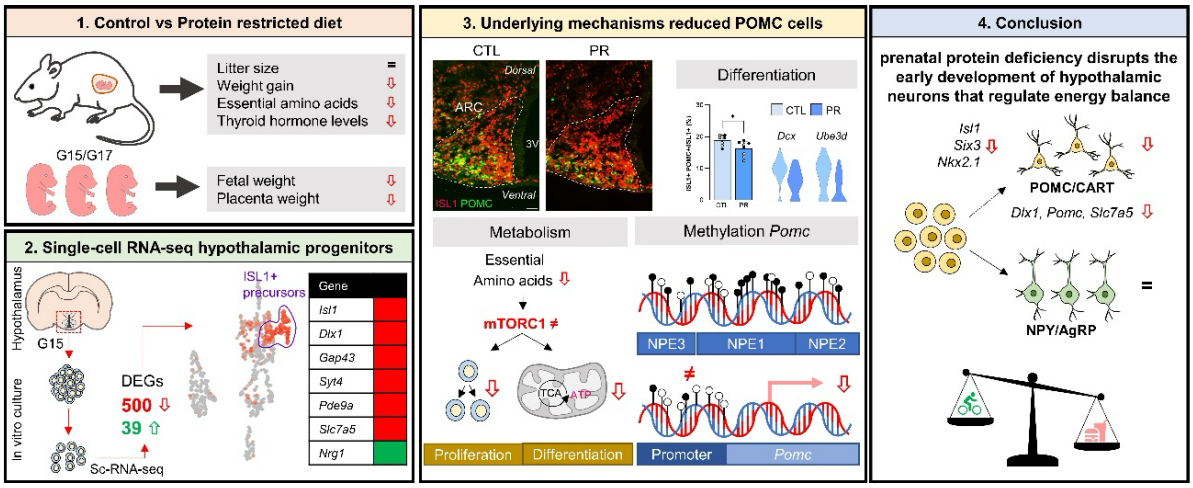
Reduced availability of essential amino acids disrupts differentiation of anorexigenic POMC neurons in the fetal rat hypothalamus

Intrauterine growth restriction (IUGR) is linked to an increased risk of long-term metabolic disorders, such as obesity and type 2 diabetes, consistent with the Developmental Origins of Health and Disease theory. However, the mechanisms driving metabolic programming remain poorly understood. In this study, we investigated the effects of gestational protein restriction (PR) on fetal hypothalamic development, specifically focusing on the formation of neuronal populations that regulate appetite and energy balance. Using a rat isocaloric PR model (8% protein during preconception, 4% during gestation), we analyzed hypothalamic development at gestational days 15 and 17—critical periods for cell fate determination and differentiation. We monitored maternal, fetal, and placental weight gain, as well as maternal plasma amino acid concentrations. To assess the impact on neuronal differentiation, we performed single-cell RNA-sequencing, EdU-labeling, immunohistochemistry, and RNAscope. Our findings revealed that gestational PR reduced maternal concentrations of essential amino acids, impaired fetal growth, and selectively disrupted the differentiation of ISL1 precursors into POMC neurons, while NPY precursor differentiation remained unaffected. These group differences correlated with downregulation of neuronal differentiation genes and disrupted mTOR signaling, potentially linked to decreased maternal branched-chain amino acids and altered expression of the amino acid transporter Lat1. Epigenetic changes in the *Pomc* promoter, but not in its enhancers, may also contribute to the observed phenotype. These results underscore the importance of adequate protein intake for POMC neuron differentiation in the fetal arcuate nucleus. Future research should explore additional developmental windows to refine dietary recommendations for at-risk pregnancies.



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