converting it to CO_2 for the Calvin cycle during the day. CAM is a matter of timing, not the enzymes at play, and is therefore challenging to infer from genome analysis, but methods to do so are improving.

Thus, plants have undergone something of an evolutionary seesaw, adapting to life on land, then, influenced by factors that remain to be fully explored, returning to the water, and perhaps to the land and back several more times. Each time the organism adapted to its new environment, but also carried along a bit of the old, mixing it with a bit of the new. Guo *et al.*[3] present analyses that hint at the patterns of selective pressures involved. They identify a complex pattern of relationships between the aquatic habit and latitude, annual mean temperature, elevation, and rainfall (to name a few). They also find further support for the long standing but controversial hypothesis that the distinctive traits of monocots reflect derivation from an aquatic ancestor. Their approach is powerful, and the analysis provides plenty of food for thought. It left me with more questions than answers, as every great scientific work should.

DECLARATION OF INTERESTS

The author declares no competing interests.

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Social neuroscience: Nosh or nurture?

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Mothers exhibit an increased appetite to cope with the energetic demands of lactation. A new study has identified a neural circuit that interfaces between food seeking and pup caring.

As the old adage goes, during pregnancy you are eating for two. Energy demands during pregnancy increase[1] and this continues during lactation — production of breastmilk is energetically expensive[2]. To adapt to these energetic costs, rat mothers eat more frequent meals[3] and pursue protein-rich foods during lactation[4]. When confronted with this need to feed, animals must make decisions to balance food consumption with caring for their offspring. A new study published in *Nature* by Alcantara *et al.*[5] identifies a neural circuit to prioritize feeding over maternal care (Figure 1).

Alcantara *et al.*[5] first characterized the food intake of female mice following mating and parturition, finding that postpartum mothers consume up to five times as much food as they did prior to mating. This increased caloric consumption positively correlated with the number of pups the females birthed, suggesting that the postpartum surge in food intake is likely driven by the energetic demands of nursing and offspring care.

To understand how the increased feeding during lactation is orchestrated by the brain, the authors investigated the arcuate nucleus of the hypothalamus (ARC), a region with a well-established role in controlling feeding behavior and metabolism[6]. Single-cell RNA sequencing of the ARC cells revealed hundreds of genes that are commonly modulated by fasting and lactation[5]. In particular, both fasted and lactating animals showed elevated expression of Agouti-related peptide (Agrp) and neuropeptide Y (NPY), which are highly co-expressed and have previously been shown to drive food intake[7]. The increase of Agrp and NPY levels is constant in lactating mothers, irrespective of food availability, suggesting a tonically increased food-needing state[5]. Beyond changes in the neuropeptide level, *in vitro* slice electrophysiology revealed increased $ARC^{Agrp/NPY}$ cell excitability during lactation, which will further facilitate Agrp and NPY release. The heightened

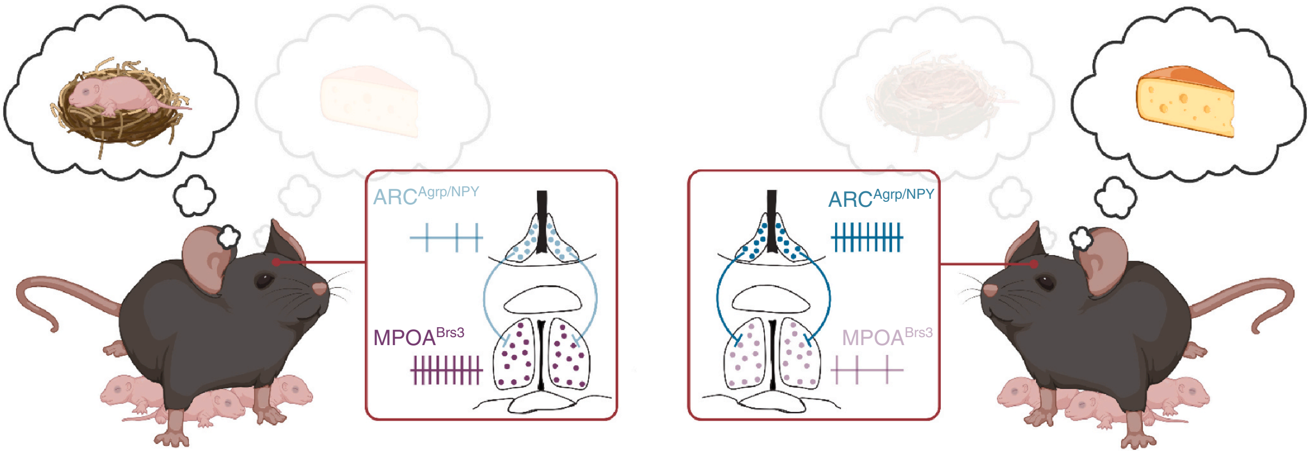

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Figure 1. A neural circuit that balances feeding drive and maternal care.
An inhibitory connection from $\text{ARC}^{\text{Agrp/NPY}}$ neurons to $\text{MPOA}^{\text{Brs3}}$ neurons biases behavior toward food over maternal care.

excitability is also reflected in the $\text{ARC}^{\text{Agrp/NPY}}$ cell responses *in vivo*. In fasted mice, $\text{ARC}^{\text{Agrp/NPY}}$ neural activity decreases upon detecting the food[8], while in fed mice, food presence does not suppress ARC^{Agrp} neural activity further, presumably due to low spontaneous activity of the cells. However, in fed lactating females, food presence remains effective in suppressing ARC^{Agrp} cell activity, suggesting elevated basal activity of the cells[5]. That perhaps also explains the constant appetite of mothers. This series of experiments suggests that lactating mothers possess a neural signature of increased hunger.

Hunger can strongly influence pup-directed behaviors. In extreme cases, fasted virgin females could attack pups[9]. During infanticide, ARC^{Agrp} neurons were significantly suppressed, mimicking the activity of these cells during feeding. To quantify the relationship between feeding and maternal care drives, Alcantara *et al.*[5] developed a behavioral assay in which animals chose to seek food or to care for pups. Animals were placed in an arena with three chambers: one containing a food dispenser, one containing a shelter with scattered pups, and a neutral chamber in between the two. Fed virgin females and lactating mothers always gathered the pups first, built nests around them, and spent the majority of their time in the pup-containing chamber[5]. In contrast, although fasted virgins were more likely to consume food before attending to pups, the majority of fasted mothers brought the pup back to the nest before having a meal, although some mothers took a snack in the food chamber before bringing all the pups back to the nest[5]. Both fasted virgins and lactating mothers spent less time in the pup-containing chamber and more time in the food chamber, suggesting feeding competes with the drive for maternal care[5].

To understand how the brain orchestrates the conflict between feeding and nurturing, the authors zeroed in on the connection from the arcuate nucleus (ARC) to the medial preoptic area (MPOA), a hypothalamic nucleus controlling parenting (Figure 1)[10]. Alcantara *et al.*[5] drove expression of the light-activated cation channel channelrhodopsin 2 in ARC^{Agrp} neurons and implanted optical fibers in the MPOA to selectively stimulate the $\text{ARC}^{\text{Agrp}} \rightarrow \text{MPOA}$ pathway. Upon stimulation, both virgin and lactating females increased food consumption and reduced maternal behaviors even when animals were fed, suggesting that this pathway biases females' drive toward feeding over pup care. Consistent with prior work[11], this manipulation also impaired nest-building in addition to pup-directed behaviors, perhaps signaling an overall decrease in maternal motivation.

Alcantara *et al.*[5] next wanted to get a grasp on parenting-relevant neuronal populations in the MPOA. A number of genetically defined MPOA cell types critical for parenting have been previously explored[12]. Opting instead to take an unbiased approach, Alcantara *et al.*[5] used Fos-targeted recombination of active populations[13]: upon transcription of the immediate early gene c-Fos, which serves as a surrogate of neural activity, Cre recombinase will be expressed, permitting genetic access to activated cells. The authors then labeled parenting-activated neurons and used a chemogenetic approach to inhibit their neural activity[5]. Indeed, inhibiting parenting-activated neurons reduced parental behaviors and transiently prioritized feeding when lactating females fasted. Critically, when pups were not present, animals did not consume more food, suggesting that this manipulation primarily decreased maternal motivation rather than drove feeding.

Following up on this result, the authors next sought to identify the MPOA cells that are relevant for maternal care and could be modulated by the hunger state and decided to focus on bombesin receptor subtype 3 (Brs3) expressing cells. Brs3 is a gene previously found enriched in the MPOA parental care-activating cells based on multiplexed robust fluorescent *in situ* hybridization[12]. Alcantara *et al.*[5] further demonstrated that Brs3 is in the MPOA cell cluster that shows extensive transcriptomic changes during lactation and fasting. Indeed, $\text{MPOA}^{\text{Brs3}}$ neurons bidirectionally controlled the animals' propensity for food or maternal care; inhibiting these neurons suppressed maternal behaviors and upregulated feeding, whereas activation of this neural population accelerated maternal behaviors in fasted virgin females[5]. In line with these manipulations, MPOA^{Brs3} neurons respond strongly during pup interactions. When fasted, the cells also respond to food, but to a lesser extent than to pups. Thus, MPOA^{Brs3} neurons appear to primarily regulate maternal behaviors, but may additionally sense the hunger state of the animal.

Alcantara *et al.*[5] next tested how ARC^{Agrp} and MPOA^{Brs3} neurons interact. Channelrhodopsin-assisted circuit mapping revealed that a subset of the MPOA^{Brs3} neurons is directly inhibited by ARC^{Agrp} cells. The ARC^{Agrp}-driven inhibition was mediated by GABA neurotransmission — administration of picrotoxin, a GABA$_A$ receptor antagonist, blocked this inhibition. To probe the function of this connection *in vivo*, the authors applied an intersectional genetic approach to chemogenetically activate ARC$^{Agrp/NPY}$ neurons while recording calcium signals from MPOA^{Brs3} neurons. Consistent with inhibition seen *in vitro*, ARC$^{Agrp/NPY}$ activation suppressed MPOA^{Brs3} activity and blunted the response to pup interactions[5]. This result provides a neural basis for how the hunger-activated ARC cells can inhibit the parenting-driving MPOA neurons.

Notably, a relatively modest subset of MPOA^{Brs3} neurons was inhibited by ARC^{Agrp} photostimulation; approximately 9% of MPOA^{Brs3} neurons receive direct inhibitory input from ARC^{Agrp} cells, raising the possibility that either a small subset of MPOA^{Brs3} neurons is responsible for resolving the conflict between feeding and parenting, or that ARC^{Agrp} neurons could recruit other inhibitory inputs to dampen the MPOA activity.

Motherhood involves a suite of physiological and behavioral adaptations that ensure offspring and species survival. This study revealed remodeling of ARC^{Agrp} cells during lactation to signal a high energy-demanding state for milk production. It also showed a circuit motif that allows the feeding drive to suppress maternal motivation. The existence of this motif is crucial, as ultimately, mothers can only provide quality care and produce enough milk when taking in sufficient nutrients. During motherhood, maternal motivation is extremely high, but sometimes, mothers have to leave the pups to seek food not only for their own survival but also for their young.

DECLARATION OF INTERESTS

The authors declare no competing interests.

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Ecology: The role of flowers as microclimatic chambers

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The important role of pollinators in floral evolution has been well established through centuries of research. A new study combining micrometeorological and behavioral experiments demonstrates that abiotic conditions also play a key role in shaping floral morphology and function in relation to insect visitors.

Flowers are a key evolutionary innovation that has supported the diversification of angiosperms and the many organisms that interact with them. Primarily, flowers are considered structures that offer rewards (e.g., nectar, pollen, resins) to floral visitors to facilitate plant reproduction[1]. When floral visitors inadvertently contact the