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Review

Sensory thalamus function, plasticity and neuromodulation in health and disease

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ABSTRACT

For each of the many sensory channels through which animals perceive the world, sensory thalamus is an important processing station whose role lies between faithful stimulus encoding and cognitive interpretation. Located deep in the brain, sensory thalamus neurons must receive and transmit peripheral information reliably, while modulating it based on valence, internal states and memory from previous experience. It has to speak to the neocortex with the appropriate volume, and in an orderly way, to prioritize attention to what matters most in each circumstance. In this review, we recapitulate classic and recent findings on the sensory thalamus, and how its plasticity and modulation allow it to provide a basis not only for perception, but also memory and cognition. Finally, we discuss how alterations in sensory thalamus may underlie pathogenesis or contribute to specific symptoms of cognitive and neuropsychiatric disorders.

1. Introduction

First named by Galen (129 – 216 AD), the thalamus is believed to owe its name to the ancient Greek word “thalamos” (transliterated to English from θάλαμος), which refers to the innermost part of Greek houses usually the bed or bridal room (García-Cabezas et al., 2021; Serra et al., 2019; Cassel and Pereira de Vasconcelos, 2021). The thalamus is a grey matter structure located deep in the brain as part of the diencephalon. It is highly connected with many cortical and subcortical areas (Hwang et al., 2017). Due to its location and extensive connectivity, it was classically understood as a relay center that communicates with many different areas - the cortex connection being thought of as the one with the most sophisticated computations (Jones, 1991). However, over the last decades many studies expanded this role and support that the thalamus may act as an integrative hub, combining inputs from multiple sources and participating in brain-wide information processing and cognitive control (Halassa and Kastner, 2017; Shine et al., 2023).

2. Two-stage signal transmission in sensory thalamus

All sensory pathways, with the exception of the olfactory system (Courtiol and Wilson, 2015), transmit their information to cerebral cortex via dedicated sensory nuclei in the thalamus. The so-called first-order (or lemniscal) (Ahissar et al., 2000) sensory thalamic nuclei are the first to receive these sensory inputs, which are excitatory and also inhibitory (Winer et al., 1996a); Peruzzi et al., 1997; Mellott et al., 2014; Beebe et al., 2018; Whyland et al., 2020) and send it to sensory cortices (Fig. 1a). These nuclei respond to sensory stimuli (Sumser et al., 2025; Taylor et al., 2021; El-Boustani et al., 2020), are necessary for perception (Hasegawa et al., 2024; Leva et al., 2024) and include the lateral geniculate nucleus (LGN, for vision) (Le Gros Clark and Penman, 1934; Bishop et al., 1962), the ventral portion of the medial geniculate body (MGBv, for audition) (Adrian et al., 1966; Rouiller et al., 1979), the parvocellular portion of the ventral posterior medial nucleus (VPMpc, gustatory) (Ogawa and Nomura, 1988), the ventral posterior medial (VPM, somatosensory) (El-Boustani et al., 2020; Pierret et al., 2000; Wimmer et al., 2010; Diamond et al., 2008), the ventral posterior lateral (VPL, somatosensory) (Zhang et al., 2006; Vázquez et al., 2013), the posterior ventral medial (VMpo, in primates, somatosensory) (Craig et al., 1994) and its rodent homolog posterior triangular (PoT, in rodents, somatosensory) (Leva et al., 2024; Gauriau and Bernard, 2004; Bokiniec et al., 2018). Occasionally, VPM and VPL are investigated together and named ventrobasal complex (VB) (Koyama et al., 1998). First-order sensory thalamic nuclei receive modulatory feedback

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from deep cortical layers (typically L6), forming the so-called cortico-thalamic loops (Sherman and Guillery, 1996).

The so called higher-order thalamic (or paralemniscal) (Ahissar et al., 2000) nuclei also respond to sensory stimuli (Sumser et al., 2025; Taylor et al., 2021; Petty and Bruno, 2024; Odegaard et al., 2025) and play a role in perception (Taylor et al., 2021; La Terra et al., 2022), yet a large fraction of their input arrives as feedback from sensory cortical areas – primarily layer 5/6 pyramidal neurons – as well as directly from the periphery (El-Boustani et al., 2020). These nuclei are also part of the cortico-thalamic loops (Diamond et al., 1992), but they receive more contextual information from layer 6 neurons than first-order nuclei (Kirchgessner et al., 2020) and exclusive cortical input from layer 5 (Sherman, 2016). They receive more processed inputs including global, multisensory information. These nuclei include the pulvinar (LP, visual) (Petty and Bruno, 2024; Petty et al., 2021; Kurzawski et al., 2022), the dorsal and medial portions of MGB (MGBd and MGBm, auditory) (Wepsic, 1966; Love and Scott, 1969; Edeline and Weinberger, 1991a; Anderson and Linden, 2011), as well as posterior medial (POm, somatosensory) (El-Boustani et al., 2020; Wimmer et al., 2010; Petty and Bruno, 2024; Diamond et al., 1992; Petty et al., 2021) and mediodorsal thalamus (MD or MDT, mainly linked to non-sensory, higher-order functions but also encodes olfactory and gustatory stimuli) (Courtiol and Wilson, 2015; Fredericksen and Samuelsen, 2022).

Both first and higher-order sensory thalamic nuclei are mainly composed of excitatory neurons. The presence of local interneurons is limited and nucleus-specific in rodent thalamus, particularly mouse (Seabrook et al., 2013; Jager et al., 2021; Simko and Markram, 2021a; Gorin et al., 2023), yet interneurons in sensory thalamus are more frequent in primates (Butler, 2008). Ferrets (Sanchez-Vives et al., 1996), cats (Huang et al., 1999) and guinea pigs (Spreafico et al., 1994) also exhibit local interneurons in sensory thalamic nuclei.

As expected, based on their connectivity, first-order and higher-order sensory thalamus display functional differences. Higher-order thalamus responses to sensory stimuli are strongly driven by sensory cortex, whereas those in first-order do not, and are present upon cortical inactivation (Diamond et al., 1992). First and higher-order thalamus also show different mechanisms to encode stimulus features. For example, first-order auditory thalamic cells vary their response amplitudes to sounds of different frequencies, whereas higher-order auditory thalamus change instead their response latencies (Ahissar et al., 2000). Higher-order sensory thalamus has been shown to respond to multimodal stimulation (Wepsic, 1966; Love and Scott, 1969), behavioral state (e.g., active sensing) (Petty et al., 2021) and arousal (Wang et al., 2023). Furthermore, higher order neurons show greater bursting, and less spontaneous activity, than first-order cells (Ramcharan et al., 2005), and project to both sensory and motor cortical areas (Casas-Torremocha et al., 2017, 2019).

Both first and higher-order thalamus are under tight inhibitory control by GABAergic projections from the thalamic reticular nucleus (TRN) (Wimmer et al., 2015; Liu et al., 1995; Pinault, 2004; Li et al., 2020). However, higher-order nuclei appear to receive a greater GABAergic inhibition from other sources, such as the anterior pretectal nucleus (Bokor et al., 2005), and the zona incerta (Barthó et al., 2002) (Fig. 1b). Moreover, higher-order nuclei project to areas beyond the neocortex, like the high-order auditory thalamus projections to amygdala (Taylor et al., 2021; Li et al., 1995), or the high-order visual thalamus terminals in the brainstem (Vega-Zuniga et al., 2025).

In summary, both first and higher-order thalamic nuclei show robust, replicable and time-locked neuronal responses to sensory stimuli, and specific inactivation of either first and higher-order sensory thalamus leads to strong deficits in perceptual acuity. First-order nuclei receive sensory information at a pre-cortical stage and are specialized in stimulus feature encoding, whereas higher-order thalamus receive extensive cortical input and is linked to broader stimulus modalities and cognitive context (Sherman, 2016; Wolff et al., 2021).

3. Beyond the relay

Despite this classical separation of sensory thalamic functions in first and higher-order nuclei, first-order thalamus can carry higher-order information. For instance, first-order visual thalamus has been shown to respond to visual stimulation in either eye, performing binocular integration, a process that was believed to arise only in cortex (Howarth et al., 2014). LGN and MGB also encode sensory features kept in working

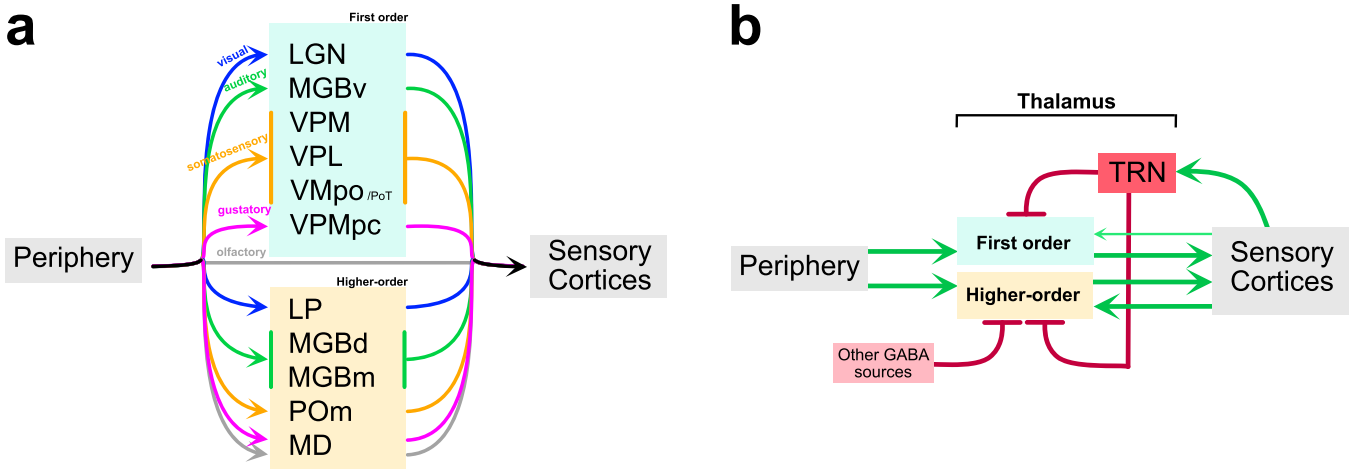

Fig. 1. Sensory nuclei of the thalamus in a first and higher-order classification. **a.** Diagram of the functional division between first order and higher-order nuclei of sensory thalamus. LGN, MGBv, VPM, VPL, VMpo (or PoT, in mice) and VPMpc are first order thalamic nuclei, whereas LP, MGBd, MGBm, POm and MD are higher-order thalamic nuclei. Both types get sensory input from the periphery, segregated by sensory modality, and both send projections to sensory cortices (and to other regions, not depicted here). Olfactory information reaches cortex without a first order thalamic nucleus. MD is typically not regarded as a sensory nucleus, but rather a high-order site involved in cognitive functions. However, it is included here due to recently identified olfactory and gustatory sensory information encoding. **b.** Summary of the major excitatory inputs and outputs of sensory thalamic nuclei. Peripheral information, mainly excitatory, travels to first and higher-order sensory thalamic nuclei, which in turn send projections to sensory cortices. Cortex sends feedback to thalamus, in the so-called thalamo-cortico-thalamic loops. Higher-order nuclei get particularly rich excitatory input from cortex. On the other hand, sensory cortices activate TRN, which in turn inhibits both kinds of sensory thalamic nuclei. Finally, higher-order nuclei also receive inhibitory input from other GABAergic structures (anterior pretectal nucleus and zona incerta). Overall, first order sensory thalamus is mainly driven by the peripheral input, whereas higher-order thalamic nuclei receive more innervation from either cortex or other sources, making them richer in multimodal and cognitive information.

memory (Hasegawa et al., 2024; Rahmati et al., 2023), just like the higher-order MD (Peräkylä et al., 2017). In somatosensory thalamus, both first and higher-order nuclei integrate highly complex whisker and head kinematics (Oram et al., 2024). Moreover, first-order thalamic nuclei exhibit response plasticity to stimuli upon learning (Edeline and Weinberger, 1991b), which was classically attributed to higher-order nuclei (Edeline and Weinberger, 1991a). In sensory goal-oriented tasks in mice, sensory thalamus additionally encodes choice (Gilad et al., 2020). Furthermore, cochlear lesions trigger tonotopic reorganization in first-order auditory thalamus that is very similar to that observed in primary auditory cortex, suggesting its participation in cortical plasticity (Kamke et al., 2003). Conversely, subsets of cells in higher-order thalamus also encode stimulus features reliably (Anderson and Linden, 2011).

Recently, the presence of neural ensembles has been reported in sensory thalamus, similar to cortex (Hu et al., 2024). Cells that fire synchronously are physically closer to each other and have similar tuning properties. Synchrony in these thalamic neural ensembles is seen under stimulus-driven firing but also spontaneously (Hu et al., 2024). It is believed that ensembles refine the encoding of information, such as stimulus features, which would be more poorly transmitted by individual neurons. Coordinated neural activity is more likely to trigger neural responses in downstream targets (Zandvakili and Kohn, 2015).

Finally, communication between sensory thalamus and cortex in thalamo-cortical loops plays an important role in perception. A recent study inhibited feedback projections from sensory cortex back to higher-order thalamus, which suppressed perception in mice as well as sensory stimulus feature encoding in primary sensory cortex (Mo et al., 2024). These findings support the notion that sensory systems do not rely solely on bottom-up feature encoding to generate percepts. Instead, perception may arise from an integration of feature encoding with top-down contextual influence. This framework is commonly described as Bayesian or predictive coding (Knill and Pouget, 2004; Kanai et al., 2015; Furutachi et al., 2024; Keller and Mrsic-Flogel, 2018). This view is supported by animal studies demonstrating that internal states shape stimulus encoding in sensory thalamus (Taylor et al., 2021; Hasegawa et al., 2024; Petty and Bruno, 2024) and sensory cortex (Furutachi et al., 2024; English et al., 2023). In line with this, human studies have shown that expectations can modulate sensory responses both in the auditory cortex (SanMiguel et al., 2013) and in the auditory thalamus (Tabas et al., 2020; Cacciaglia et al., 2015).

Together, these findings suggest that the roles of first and higher-order thalamic nuclei are more diffuse and diverse than traditionally suggested. Sensory thalamus performs high-level sensory processing and is involved in cognitive processing and behavioral adaptation and is referred to as an integrative hub for brain networks (Hwang et al., 2017).

4. Sensing under the watch of the thalamic reticular nucleus

TRN projections are the main form of inhibitory control over sensory thalamus, both first and higher-order nuclei. Because TRN neurons are, in turn, driven by prefrontal (Cornwall et al., 1990; Nakajima et al., 2019) and primary sensory cortices (Pinault, 2004), as well as other structures like basolateral amygdala (BLA) (Aizenberg et al., 2019), thalamic responses can be modulated quickly on a context and attentional basis. This is important because the relevance of a sensory modality depends on the specific contexts (e.g., an animal may first rely on audition to assess predator sounds outside a safe location or may attend to vision to find a specific food source). Studies have shown that sensory thalamic nuclei modulate their activity by suppression or enhancing the encoding of a given modality according to task demands (Wimmer et al., 2015; Williamson et al., 2015). TRN mediates this control by specifically modulating thalamic nuclei that are less relevant in a given context (Wimmer et al., 2015; McAlonan et al., 2006) and thus TRN has been referred to as a "searchlight" (Crick, 1984) that provides an additional dimension to sensory thalamus coding during cognition.

5. Sensory thalamus plasticity

Activity in the thalamic circuit has the ability to change with time. Plasticity in the sensory thalamus has been identified in two forms: First, lesions or sensory deprivation were shown to trigger changes in thalamic receptive fields or responsiveness. Such modifications may allow sensory systems to be adaptable and optimize the neural encoding of stimuli in every circumstance, similar to synapses being refined or brain areas taking over functions upon sensory loss (Bedny et al., 2011; von Melchner et al., 2000; Diniz CRAF, 2023). Second, plastic changes in sensory thalamus can also occur in a cognitive context, such as identifying a previously known stimulus (Disterhoft and Olds, 1972; O'Connor et al., 1997; Halverson et al., 2010), assigning a valence (Taylor et al., 2021; Buchwald et al., 1966), or responding according to internal state (Gilad et al., 2020; Peelman and Haider, 2024).

Several lines of research studied thalamic plasticity upon sensory deprivation. For example, skin lidocaine anesthesia reversibly rearranges receptive fields in somatosensory VPM (Nicolelis et al., 1993). This fast thalamic plasticity depends, at least in part, on primary sensory cortex (Krupa et al., 1999). In the visual system, monocular deprivation triggers synaptic boutons to shift their responsiveness between monocular and binocular tuning, and vice versa. This effect is reversible and independent of cortico-thalamic feedback (Jaepel et al., 2017). Synaptic inhibition is necessary for ocular dominance thalamic plasticity (Sommeijer et al., 2017; Qin et al., 2023). In humans, short-term visual deprivation with an eye patch elicits plasticity in the higher-order visual thalamus, as measured by monocular stimulation responses in the ventral pulvinar nucleus, with the deprived eye responses being enhanced (Kurzawski et al., 2022).This form of plasticity has also been observed in the first-order auditory thalamus, MGBv (Kamke et al., 2003). Altogether, these data show that sensory thalamic nuclei adapt to injury or sensory deprivation to optimally harvest the neural resources available for perception. This sort of plasticity can also occur at pre-thalamic stages, and first-order sensory thalamus itself can receive new connections upon injury (Takeuchi et al., 2012).

On the other hand, sensory thalamus plastically modulates its responses upon associative learning. Studies on fear conditioning have shown that auditory thalamus has enhanced responses to a tone that is coupled to an aversive outcome (Disterhoft and Olds, 1972; O'Connor et al., 1997; Halverson et al., 2010; Buchwald et al., 1966). This has been shown in cats (Buchwald et al., 1966; Ryugo and Weinberger, 1978), rabbits (O'Connor et al., 1997), rats (Disterhoft and Olds, 1972; Halverson et al., 2010) and mice (Barsy et al., 2020; Pardi et al., 2020; Taylor et al., 2021). Enhanced thalamic responses to conditioned stimuli also weaken upon extinction learning of the association (Taylor et al., 2021; Buchwald et al., 1966), and reversal training experiments show that neurons can re-tune to stimuli if they become more relevant (Hasegawa et al., 2024; Gabriel et al., 1975). Mechanistic experiments show that inactivating sensory thalamus projections to amygdala impairs fear learning (Barsy et al., 2020; Pardi et al., 2020; Taylor et al., 2021). Furthermore, thalamic plasticity is complex, with cells being able to up- or downregulate their responses to many elements, such as conditioned stimuli, safe stimuli, and expected or unexpected outcomes (Taylor et al., 2021; Hasegawa et al., 2024; Ryugo and Weinberger, 1978; Supple and Kapp, 1989). While it has been shown that plasticity occurs in higher-order thalamic nuclei (e.g., during fear learning, Barsy et al., 2020; Pardi et al., 2020), first-order sensory thalamus neurons are also plastic upon learning (Fig. 2a).

Recently, behavioral studies demonstrated that sensory thalamus also shows plasticity upon goal-oriented, appetitive learning. Upon learning an association between a stimulus and reward, sensory thalamic cells re-tune, while the coding of the reward-predicting stimulus gets enhanced (Hasegawa et al., 2024; Petty and Bruno, 2024; Gilad et al., 2020). Moreover, internal states, motor features and behavioral

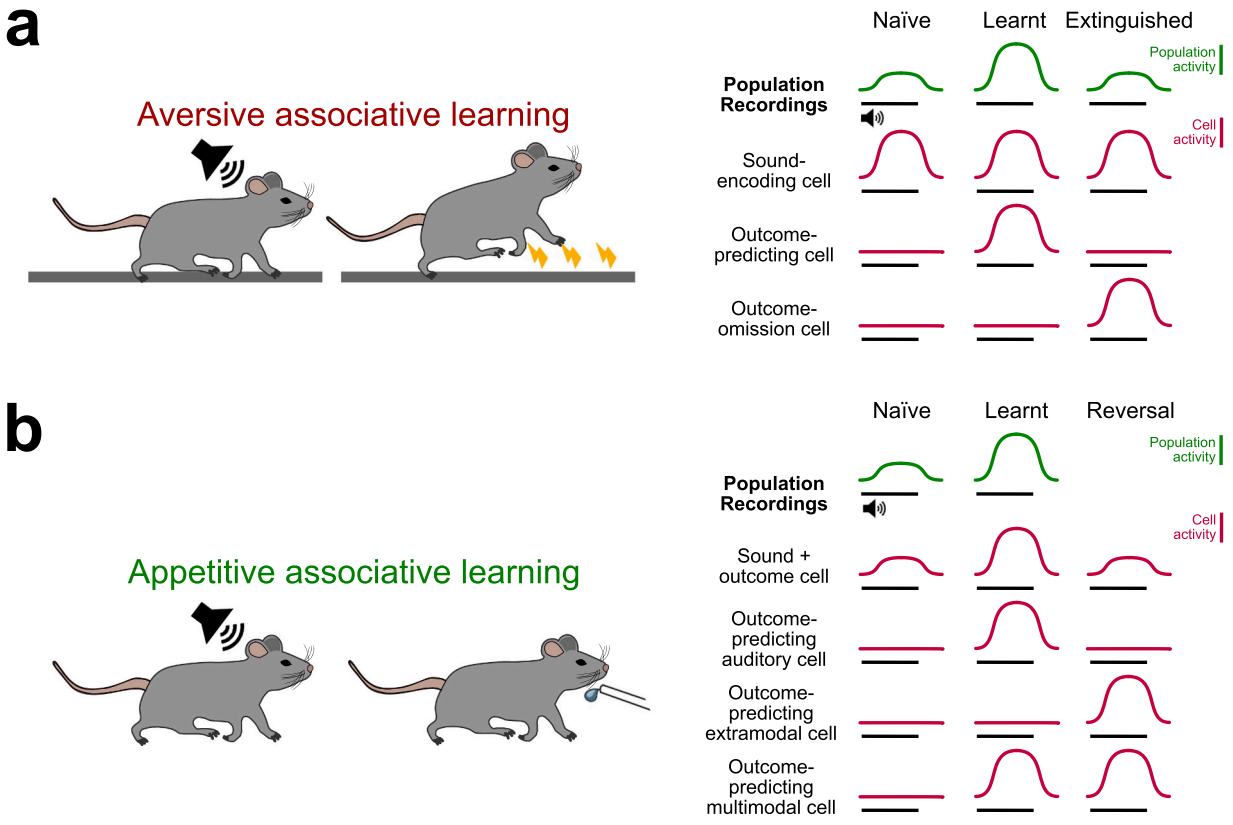

Fig. 2. Sensory thalamus shows plastic responses upon learning and cognitive variables. **A.** Left: Example of an aversive associative learning task in mice. In fear conditioning, mice learn to associate an auditory stimulus with an unpleasant outcome, in this case a mild electrical foot shock on the floor. Right: Sensory thalamus response examples over learning stages reveal that some cells stably encode the stimulus ("sound-encoding cells"), but others show plastic changes over learning ("outcome-predicting cells", which respond to the tone for as long as it is predictive of the aversive outcome, and "outcome-omission cells", which respond to the tone only after the animal has learnt that it no longer predicts the shock). Traces in red represent illustrative cell activity, and the stimulus presentation window is depicted with a black line. These plots summarize some findings of Ryugo and Weinberger, 1978, Halverson et al., (2010) (population data) and Taylor et al., (2021) (single cell data). **B.** Left: Example of an appetitive associative learning task. In Go/No-Go sensory tasks, animals associate a sensory stimulus to a positive outcome, such as the delivery of a soymilk reward droplet, and they need to report the stimulus prior to obtaining the reward. In some tasks, experimenters use reversal training, where a new cue predicts the reward, and the original stimulus is no longer predictive of it. Right: Example responses of sensory thalamus cells during these behaviors. Some cells that respond to the sensory cue prior to the learning change their responses once the cue is predictive of the reward ("sound+outcome cells"). On the other hand, there are cells in sensory thalamus that respond to the stimulus only when it is predictive of reward ("outcome-predicting cells"), and even when the cue is of a different sensory modality ("outcome-predicting extramodal cell"). Stable cells are also present in these datasets, but are only shown in A. These data summarize some of the findings of Gilad et al., (2020) (population data), Hasegawa et al., (2024), Petty and Bruno, 2024 and other unpublished findings (single cell data, Paricio-Montesinos & Gründemann, in preparation). Some cells respond by enhancing their firing rate, and some by lowering it. Here we show all changes in activity as increased activity for clarity purposes.

choice to respond to a stimulus are encoded in sensory thalamic cells (Gilad et al., 2020; Peelman and Haider, 2024), and sensory nuclei from one sensory modality can become tuned to respond to stimuli of a different modality, if it is predictive of reward (Hasegawa et al., 2024; Petty and Bruno, 2024) (Fig. 2b).

6. Sources of plasticity in thalamus

Sensory thalamus has minimal connectivity between neighboring neurons (Halassa and Sherman, 2019) as well as a very small number of local interneurons (Halassa and Acsády, 2016; Simko and Markram, 2021b), which makes it structurally distinct from cortical areas. As such, plasticity in sensory thalamus is most likely mediated by changes in intrinsic excitability and plastic synaptic inputs or changes in presynaptic activity from external sources, rather than through local circuit dynamics.

Non-synaptic plasticity, particularly changes in intrinsic excitability, have been suggested to occur in sensory thalamus (Apergis-Schoute et al., 2005). These changes may occur in the context of sensory exposure and learning, and are supported by evidence of transcriptional regulation in auditory thalamus upon sensory exposure and learning (Ran et al., 2003; Han et al., 2008; Brauth et al., 2007). Such modifications can influence the responsiveness of thalamic neurons to incoming signals without altering synaptic strength.

On the other hand, synaptic plasticity in sensory thalamus may arise from several input sources: i) pre-thalamic sensory nuclei (such as the superior and inferior colliculi, dorsal column nuclei or the trigemino-thalamic tract) (Lee and Sherman, 2011; Winer et al., 1996b; Xue et al., 1994; Lund and Webster, 1967), ii) sensory cortex feedback from layer 6 neurons (Sherman and Guillery, 1996), iii) primary and higher-order sensory cortex layer 5 neurons driving predominantly higher-order nuclei (Lee and Sherman, 2010; Miller-Hansen and Sherman, 2022), and iv) TRN (Halassa and Acsády, 2016; Zikopoulos and Barbas, 2012). Additionally, sensory thalamus receives neuromodulatory input from other sources, which will be discussed in the next section.

While the pre-thalamic nuclei that feed sensory information to thalamus (found at the midbrain and brainstem) have been observed to exhibit plasticity, it is limited to adaptations following functional loss. For example, alterations in the peripheral sensory pathways can lead to adaptations in the superior and inferior colliculi (Gold and Knudsen, 2000, 1999; Rauschecker and Harris, 1983). In contrast, sensory thalamic nuclei display a broader repertoire of plasticity, encompassing

not only responses to injury or sensory deprivation but also processes such as encoding associative learning, with individual neurons finely tuned to cognitively-relevant features (Taylor et al., 2021; Hasegawa et al., 2024). This processing is likely mediated instead by projections from the cerebral cortex and the inhibitory control exerted by TRN. For instance, layer 6 corticothalamic projections modulate the gain and temporal dynamics of sensory thalamic responses to sensory stimuli (Mease et al., 2014). These projections can directly excite sensory thalamic cells or inhibit them indirectly by driving TRN activity, depending on the context (Crandall et al., 2015). Additionally, higher-order sensory thalamic nuclei receive "driver" inputs from multiple cortical areas, allowing them to integrate information from diverse sources (Sampathkumar et al., 2021), and some of these nuclei have shown plasticity across sensory learning (Audette et al., 2019). Furthermore, the amygdala and prefrontal cortex also target TRN, positioning it as a hub for emotional and cognitive convergence that subsequently regulates sensory thalamus activity (Zikopoulos and Barbas, 2012). Together, these cortical and TRN inputs are likely candidates to mediate plasticity in sensory thalamic nuclei.

Regarding the cellular mechanisms of sensory thalamic plasticity, there are various identified sources. In retinogeniculate synapses, trains of stimulation cause short-term depression of AMPA receptor-mediated currents (Chen et al., 2002), which requires and is mediated by the protein CKAMP44 (Chen et al., 2018). Cortico-thalamic feedback synapses onto sensory thalamus show reversible, NMDA-independent long-term potentiation (Castro-Alamancos and Calcagnotto, 1999). In TRN cells, repetitive stimulation of glutamatergic cortico-reticular synapses results in an increase in excitability via GluN2C-NMDA receptors (Fernandez et al., 2017). Together, these mechanisms of plasticity influence the responsiveness and synaptic transmission in sensory thalamus.

Altogether, the studies above show that thalamic nuclei are able to modify their connectivity and responsiveness upon sensory deprivation and learning. The fact that sensory thalamic nuclei can quickly and reversibly update their tuning according to behavioral demands postulates sensory thalamus as a key cognitive structure in the brain.

7. Neuromodulation of sensory thalamus

Neural networks change their function and plasticity through neuromodulation (Bazzari and Parri, 2019; Brzosko et al., 2019; Varela, 2014). Here, we will briefly discuss the main neuromodulators of sensory thalamus: acetylcholine (ACh), serotonin (5-HT), histamine, noradrenaline and dopamine.

Cholinergic input to the sensory thalamus originates mostly from the Pontomesencephalic tegmentum (PMT) in the brainstem (Schofield et al., 2011), while inputs from basal forebrain are less prominent (Heckers et al., 1992; Hallanger et al., 1987). The PMT is subdivided in two nuclei: the Pedunculopontine nucleus (PPN) and the Laterodorsal Tegmental nucleus (LDT) (Schofield et al., 2011). First and higher-order thalamic nuclei exhibit distinct cholinergic modulation. Electrophysiological data in auditory thalamus indicate that, in the first-order MGBv, ACh muscarinic receptor activation triggers depolarization and tonic firing, whereas cholinergic modulation in higher-order MGBd is more diverse, and many cells exhibit hyperpolarizion by ACh (Mooney et al., 2004). This suggests that ACh may elicit specific modulation in first and higher-order auditory thalamus. VPM increases its spontaneous firing upon activation of ACh receptors, possibly thereby increasing noise levels (Hirata et al., 2006). Cholinergic modulation is most likely brain state-dependent, as cells in the PMT, the main source of cholinergic input to auditory thalamus (Schofield et al., 2011), show sleep-waking cycle-dependent activity, with peaks during wakefulness (Boucetta et al., 2014). PMT also sends extensive cholinergic modulation to visual thalamus (Billet et al., 1999) and TRN (Jourdain et al., 1989; Beierlein, 2014). In TRN, ACh triggers both direct action potentials via activation of nicotinic acetylcholine receptors (nAChRs), but also changes the ability of TRN neurons to respond to presynaptic stimuli via muscarinic receptors (mAChRs), both expressed in TRN (Sun et al., 2013). Local administration of carbachol, which activates both receptor types, results in a decrease in spontaneous firing of TRN cells (Hirata et al., 2006). However, recent *in vivo* experiments found that optogenetic activation of cholinergic inputs onto TRN enhanced the activity of TRN cells and even promoted sleep (Ni et al., 2016). Finally, cholinergic cells from the PMT also target sensory areas beyond sensory thalamus, such as the inferior colliculus or the cochlear nucleus, underlining global cholinergic effects on sensory pathways (Schofield et al., 2011).

Therefore, cholinergic input appears to exert complex control over sensory thalamus. Both directly, possibly eliciting different effects in first and higher-order sensory nuclei, as well as by modulating its inhibition indirectly through TRN neuron control and other stages of the sensory pathway. Overall, cholinergic modulation in thalamus plays a role in sleep and arousal, attention and state-dependent sensory processing.

Serotoninergic afferents to the sensory thalamus arise from the median (Gonzalo-Ruiz et al., 1995; Vertes et al., 1999) and dorsal (Vertes, 1991; Kirifides et al., 2001) raphe nuclei in the brainstem. Direct application of 5-HT elicits depolarization in most neurons of first-order sensory thalamic nuclei (Varela and Sherman, 2009) and, in higher-order nuclei, it depolarizes many but not all: it also hyperpolarizes a significant subset (~15 %) (Varela and Sherman, 2009; Monckton and McCormick, 2002). On the other hand, 5-HT has been reported to act on the retinal, presynaptic terminals that target visual thalamus, decreasing calcium activity and vesicle release onto thalamic cells (Reggiani et al., 2023). In TRN, there is expression of serotonin receptors with opposing net effects on GABAergic release (Goitia et al., 2016). The dorsal raphe nucleus has also been identified as an important modulator of pain perception (Wang and Nakai, 1994). Overall, serotonin appears to exert a nuanced control over sensory thalamus, supporting sensory gating. Moreover, 5-HT may be of particular importance during development, where it plays a role in the plasticity of thalamocortical axons (Sinclair-Wilson et al., 2023).

Histamine signaling in the central nervous system arises exclusively from the tuberomammillary nucleus of the posterior hypothalamus and it innervates, among other brain regions, sensory thalamus (Yoshikawa et al., 2021; Panula and Nuutinen, 2013; Scammell et al., 2019). Sensory thalamic nuclei, but not TRN, express receptors for histamine (Jin et al., 2002). This suggests that histaminergic influence over sensory thalamus is local. Histaminergic activation of first-order visual thalamus has been shown to depolarize cells via suppression of K^+ currents (McCormick and Williamson, 1991), and has been suggested to mediate sensory arousal (Uhlrich et al., 1993). The expression of histaminergic receptors appears similar between first and higher-order sensory thalamic nuclei (Jin et al., 2005), suggesting that this neuromodulator may affect both in similar ways. Histamine signaling in general has also been extensively linked to wakefulness and arousal (Yoshikawa et al., 2021; Panula and Nuutinen, 2013).

Noradrenaline is an additional modulator of sensory thalamus. Noradrenergic projections that target sensory thalamus originate in locus coeruleus of the brainstem (Rogawski and Aghajanian, 1980a; Pérez-Santos et al., 2021; Rico and Cavada, 1998; Simpson et al., 1997). Neurons in sensory thalamic nuclei can be activated by noradrenaline *in vitro*, through alpha-adrenergic receptors (Rogawski and Aghajanian, 1980a, 1980b). However, the picture is more complex *in vivo*, where both enhancement and suppression of thalamic firing were found upon locus coeruleus stimulation (Moxon et al., 2007; Devilbiss and Waterhouse, 2011). The direct effects of noradrenaline on higher-order thalamus are unknown. Because the receptor alpha1/alpha2 ratio of expression differs between some first and higher-order nuclei, there may be specific outcomes for noradrenergic release depending on the region (Pérez-Santos et al., 2021). In a neural circuit context, cortical loop input to sensory thalamus is suppressed in the presence of noradrenaline (Nersisyan et al., 2021). Moreover, norepinephrine activates TRN

neurons and, therefore, indirectly reduces spontaneous firing of sensory thalamus, enhancing the signal-to-noise ratio in sensory thalamic cells (Hirata et al., 2006; Castro-Alamancos and Calcagnotto, 2001). Together, evidence suggests that noradrenaline modulates sensory thalamus in sensory processing and arousal.

Dopamine in the primate thalamus has various sources. Retrograde tracing of dopaminergic axons from the thalamus shows that these arise from the hypothalamus, ventral mesencephalon, periaqueductal gray (PAG) and the lateral parabrachial nucleus (LPbN) (Sánchez-González et al., 2005). While primate thalamus is densely innervated with dopamine, especially higher-order nuclei, its dopaminergic input in rodents is much scarcer (Sánchez-González et al., 2005). However, some dopaminergic axons can still be found in rodent sensory thalamus (Papadopoulos and Parnavelas, 1990). In slices, the direct effect of dopamine on sensory thalamus neurons depends on D1 and D2 receptors and it seems to be overall excitatory in both first and higher-order nuclei (Govindaiah and Cox, 2005; Govindaiah et al., 2010a; Lavin and Grace, 1998). However, dopamine may also inhibit the presynaptic terminals that target sensory thalamus (Govindaiah and Cox, 2006). The neuromodulating effects of dopamine in sensory thalamus *in vivo* have been reported to be dose-dependent: while small amounts of dopamine result in sensory facilitation, larger amounts appear to reduce sensory responses (Zhao et al., 2002, 2001). The high dose inhibition, however, is most likely an indirect effect mediated by the action of dopamine on GABAergic inhibition (Zhao et al., 2002; Albrecht et al., 1996). In TRN, dopamine signaling appears to increase activity. On one hand, local dopamine application elicits an increase in spontaneous firing in reticular cells (Barrientos et al., 2019). On the other hand, local dopamine release acts on presynaptic GABAergic terminals from globus pallidus that target TRN, inhibiting their GABA release. This results in a disinhibition of TRN cells and a tighter silencing of sensory nuclei (Govindaiah et al., 2010b; Gasca-Martinez et al., 2010). Altogether, dopaminergic input to sensory thalamus and TRN modulates sensory processing and gating.

Overall, research has shown that sensory thalamus receives extensive neuromodulation (Table 1). Acetylcholine and serotonin are mainly activating but also inactivate a subset of higher-order cells. Histamine and noradrenaline are excitatory too; however, noradrenaline also activates TRN – which may result in a net silencing effect in sensory thalamus. Finally, dopamine appears to exert opposing, complex modulation in sensory thalamus, and an increase in TRN activity.

Table 1
Summary of neuromodulatory input onto sensory thalamus. Acetylcholine, serotonin, histamine, noradrenaline and dopamine innervate first and higher-order sensory thalamic nuclei in different ways, triggering different outcomes in the cells they target. The source, innervation, effects and main references are summarized here.

	Source	Innervation & effect	References
Acetylcholine (ACh)	Pontomesencephalic tegmentum & Basal forebrain	1st order: depolarization Higher-order: depolarization & hyperpolarization (subset) TRN: depolarization	Mooney et al., (2004) Mooney et al., (2004) Sun et al., (2013), Ni et al., (2016)
Serotonin	Median & Dorsal Raphe nuclei	1st order: depolarization Higher-order: depolarization & hyperpolarization (subset) TRN: depolarization & hyperpolarization	Varela and Sherman, (2009) Monckton and McCormick, (2002), Varela and Sherman, (2009) Goitia et al., (2016)
Histamine	Tuberomammillary nucleus of Posterior Hypothalamus	1st order: depolarization Higher-order: unknown (similar receptors to 1st order) TRN: no local receptors	McCormick and Williamson, (1991) Jin et al., (2005) Jin et al., (2002)
Noradrenaline (NA)	Locus coeruleus	1st order: *in vitro* depolarization, *in vivo* mixed Higher-order: unknown (nuclei-specific α1/α2 expression) TRN: depolarization	Rogawski and Aghajanian, (1980a), (1980b), Moxon et al., (2007) Pérez-Santos et al., (2021) Hirata et al., (2006)
Dopamine	Hypothalamus, Ventral Mesencephalon, Periaqueductal Gray, Lateral Parabrachial nucleus	1st order: *in vitro* activity increase, *in vivo* mixed Higher-order: hyperpolarization but increase in excitability TRN: activity increase	Papadopoulos and Parnavelas, (1990), Govindaiah et al., 2010 Lavin and Grace, (1998) Barrientos et al., (2019), Gasca-Martínez et al., 2010

8. The sensory thalamus in disease states

Sensory thalamic function has been linked to either the pathogenesis or the symptomatology of some neuropsychiatric disorders. It appears to be a key structure involved in schizophrenia, autism, and may also underlie some of the symptoms of Alzheimer's disease and other illnesses (Fig. 3).

8.1. Schizophrenia

Schizophrenia is a neuropsychiatric disorder characterized by impairments in reality testing, with delusions, hallucinations, formal thought disorder and disorganized behavior (World Health Organization, 2024). Schizophrenic patients may also show anhedonia, deficits in attention, problem solving and speech (among other symptoms) (Orsolini et al., 2022). In schizophrenia, the size of the thalamus is reduced and its function is impaired (Buchsbaum et al., 1996). Despite differing findings (Selemon and Begovic, 2007; Dorph-Petersen et al., 2009), evidence suggests that both first (*i.e.,* LGN, MGB) and higher-order (*i.e.,* MD, LP) sensory thalamic nuclei – as well as other non-sensory nuclei – are of smaller volume in schizophrenia patients than in healthy individuals (Adriano et al., 2010; Perez-Rando et al., 2022; Mørch-Johnsen et al., 2023). In addition, the thalamus of schizophrenic patients has abnormally high levels of dopamine (Oke et al., 1988), which has classically been linked to schizophrenia pathogenesis (Lau et al., 2013; Brisch et al., 2014). Lower thalamic D2/D3 dopamine receptor binding has also been reported (Talvik et al., 2003; Yasuno et al., 2004).

Schizophrenia patients suffer from sensory hallucinations (*i.e.,* experience of percepts in absence of external stimuli, therefore self-generated), which are linked to activity in thalamic nuclei, striatum, hippocampus, cingulate gyrus and orbitofrontal cortex (Silbersweig et al., 1995). Alterations in thalamocortical connectivity are also reported in schizophrenia patients (Marenco et al., 2012).

The link between sensory thalamic function and schizophrenia may partially have genetic roots. In humans, a specific deletion of the chromosome 22 (22q11.2) causes a number of signs and symptoms, one of them being a proneness to develop auditory hallucinations and schizophrenia (Mancini et al., 2020). Patients with the 22q11.2 deletion syndrome also have a smaller sensory thalamus and abnormal hyperconnectivity between MGB and auditory cortex (Mancini et al., 2020). A mouse model lacking

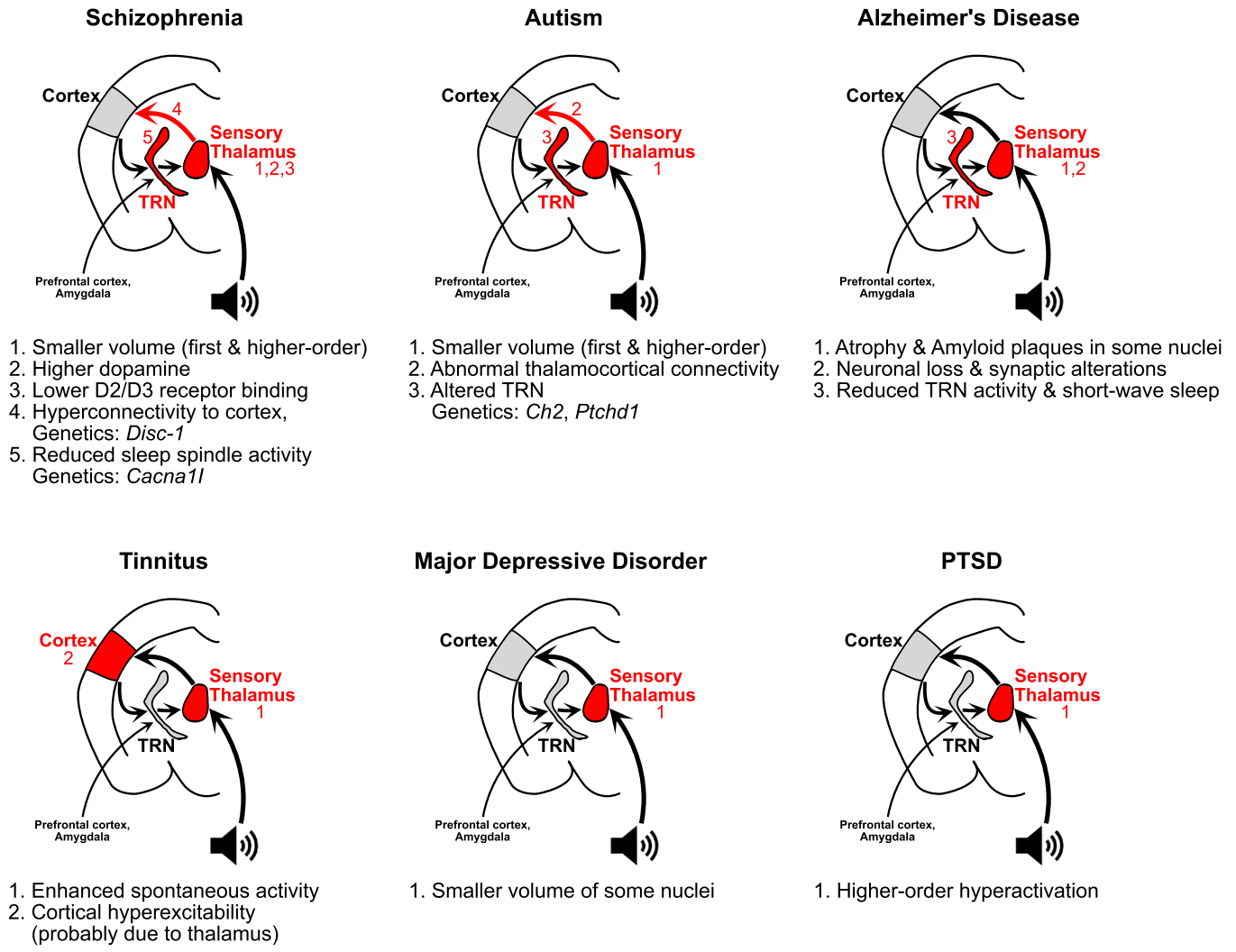

Fig. 3. Summary of identified disease alterations in sensory thalamus. Schizophrenia and autism are linked to genetic mutations that trigger morphological and functional changes in sensory thalamus, TRN and thalamocortical projections. In Alzheimer's disease, amyloid plaques and neuronal loss are found in sensory thalamus, and TRN activity and sleep are also affected. In tinnitus, auditory thalamus and cortex are hyperexcitable. Finally, patients with major depressive disorder and PTSD also show sensory thalamus alterations. The schemes depict the mouse brain anatomy but recapitulate both human and mouse findings of altered regions (red) and unaffected ones (grey).

Dgcr8 was proposed as a model of the 22q11.2 syndrome, and these mice have abnormal numbers of thalamic D2 receptors and synaptic transmission between MGB and cortex (Chun et al., 2014). The latter was reversed by the treatment with antipsychotic drugs and dopamine receptor antagonists (Chun et al., 2014). Later, it was found that *Dgcr8* deficiency triggers a microRNA loss (miR-338–3p), which appears to be the root of the thalamocortical disruption and can be replenished in adult mice to rescue it (Chun et al., 2017). Finally, the genes *Disc-1* and *Cacna1l* are also associated to schizophrenia (Schizophrenia Working Group of the Psychiatric Genomics Consortium, 2014; Krol et al., 2018). *Disc-1* is prominently expressed during brain development and may be involved in the establishment of thalamocortical connections (Austin et al., 2004), and *Cacna1l* has allelic variants that alter TRN neuron excitability (Baez-Nieto et al., 2022), which is the main inhibitory controller of sensory thalamus. Furthermore, TRN participates in the generation of sleep spindle activity, which is reduced in schizophrenic patients (Ferrarelli et al., 2007). For these reasons, TRN has been proposed to be involved in schizophrenia pathogenesis (Krol et al., 2018; Ferrarelli and Tononi, 2011; Steullet et al., 2018).

Overall, abnormalities in the structure and function of sensory thalamus may underlie some of the symptoms arising in schizophrenia. Thalamic volume loss, alterations in sensory responsiveness and impairment of thalamocortical connectivity and transmission appear to be common hallmarks in schizophrenic patients (Jiang et al., 2021). Recent studies suggest that these deficits may arise from genetic deficiencies, which may be compensated exogenously to alleviate some of the symptoms (Chun et al., 2017).

8.2. *Autism*

Autism spectrum disorder (ASD) is a set of lifelong neurodevelopmental conditions, which manifest during early childhood, and cause impaired social interactions, executive dysfunction (Cook et al., 2013) with repetitive and inflexible patterns of behavior and altered sensory and information processing (Lai et al., 2014; Ayub et al., 2021). Individuals diagnosed with ASD show hypo- and hyperreactivity to sensory stimuli (World Health Organization, 2024; Balasco et al., 2020). One of the suggested causes of this altered sensory perception is that ASD patients have altered thalamocortical connectivity, in sensory but also many other thalamic and cortical regions (Ayub et al., 2021; Woodward et al., 2017; Linke et al., 2023; Karavallil Achuthan et al., 2023). Furthermore, studies in animal models of ASD have reported fewer and smaller neurons in first and higher-order auditory thalamus, with respect to controls (Mansour et al., 2021). Patients with ASD also

exhibit smaller thalamic volume (Tsatsanis et al., 2003; Tamura et al., 2010), and the magnocellular portion of LGN shows reduced activity in adults with autism (Schelinski et al., 2024).

Another thalamic link to ASD is its inhibitory control via TRN. The genes *Chd2* and *Ptchd1* are highly expressed in TRN (Krol et al., 2018), and their mutations have been linked to autism (Iossifov et al., 2012; Pinto et al., 2010). In line with this, it has been proposed that decreased thalamic inhibition triggers sensory thalamus to transmit to cortex excessive, unfiltered sensory information that disrupts attention (Baran et al., 2023).

Overall, research suggests that thalamic function is affected in ASD, with part of the symptoms (*i.e.* altered stimulus sensitivity, abnormal sensory gating) possibly being linked to changes in sensory thalamus nuclei and inhibitory control. However, thalamocortical connectivity in more executive areas is likely key as well and may underlie social and executive functions.

8.3. Alzheimer's disease

Alzheimer's disease (AD) is a form of dementia that generally impacts memory at first and progressively results in loss of most cognitive functions (Knopman et al., 2021). It is associated to encephalic grey matter loss and cell death caused by the accumulation of the so-called amyloid beta plaques, oligomers and neurofibrillary tangles, initially in the temporal lobes, but also affecting thalamus and other structures (Braak and Braak, 1991).

Among thalamic structures, the anterior nuclei are more prominently affected in AD (Biesbroek et al., 2024), but sensory thalamus also shows changes. Beta-amyloid senile plaques and neurofibrillary tangles can be found in auditory thalamus (Sinha et al., 1993). Moreover, VPL, MD and LP thalamic nuclei show atrophy in AD (Forno et al., 2023; van de Mortel et al., 2021). Pulvinar atrophy appears to occur especially in the earlier cognitive decline stage of AD, referred to as mild cognitive impairment (van de Mortel et al., 2021). In addition, neuronal loss and synaptic alterations are found in MGB in AD patients (Baloyannis et al., 2009).

In mouse models of AD, amyloid plaques can be found in amygdala and sensory thalamus, impairing the learning of fear memories (Knafo et al., 2009). This is similar to human AD patients, who have deficits in fear conditioning (Hamann et al., 2002). Because associative learning of sensory stimuli with negative outcomes heavily depends on amygdala and its direct inputs from sensory thalamus (Barsy et al., 2020; Taylor et al., 2021), it is possible that thalamic deficiencies contribute to the sensory, attentional or memory deficits observed in AD patients (van de Mortel et al., 2021).

Moreover, thalamic inhibitory control may also be affected in AD. In mouse models of the disease, TRN activity is reduced (Hazra et al., 2016), and restoring short wave sleep by chemogenetic TRN activation ameliorates amyloid plaque deposition (Jagirdar et al., 2021).

Altogether, sensory thalamus and TRN show alterations during Alzheimer's disease and may contribute to the course and progression of the disease in addition to other structures such as the hippocampus (Braak and Braak, 1991) or anterior thalamic nuclei (Aggleton et al., 2016).

8.4. Other diseases

Auditory thalamus has been linked to tinnitus, the phantom perception of sound in absence of sound stimulation that is linked to hearing loss (Almasabi et al., 2022). In animal models of tinnitus, MGB shows enhanced spontaneous activity with respect to healthy animals (Cook et al., 2021). This could underlie the cortical hyperexcitability observed in tinnitus patients (Leaver et al., 2011).

Furthermore, sensory thalamus may also play a role in mood and mood disorders. In major depressive disorder (MDD), patients have smaller volume of several thalamic regions, including sensory nuclei (Chibaatar et al., 2023). Also, one way to alleviate symptoms is via light therapy (Tao et al., 2020), which appears to act via direct projections to lateral habenula from visual thalamus (Huang et al., 2019). Moreover, sensory thalamus has been reported to drive a specific population of parvalbumin-expressing cells in the sensory cortex, which plays a role in stress resilience. Enhancing this pathway had antidepressant-like effects in mice (Li et al., 2023).

In post-traumatic stress disorder (PTSD), the higher order somatosensory POm shows hyperactivation in mouse models. Inhibiting its activity results in a decrease of defensive responses in mice (Xi et al., 2023).

Finally, studies in developmental dyslexia, a learning disorder with impairment in reading and spelling, revealed dysfunctions of LGN (Galaburda and Livingstone, 1993; Müller-Axt et al., 2017) and MGB (Díaz et al., 2012).

9. Concluding remarks

In this review, we aimed to recapitulate the most recent understanding of sensory thalamus with a focus on functions and disease states. While first-order nuclei are more specialized in reliable encoding of sensory stimuli and higher-order thalamus generally contains more processed information, both participate in perception, associative learning, choice and other cognitive functions. Sensory thalamus is plastic and receives top-down as well as inhibitory (mainly from TRN) and neuromodulatory control, with higher-order nuclei being particularly targeted. Moreover, the impairment of sensory thalamus likely underlies some of the symptoms associated with neuropsychiatric disorders such as schizophrenia or autism as well as Alzheimer's disease. Therefore, expanding our understanding of sensory thalamus is key to pinpoint the mechanisms behind highly complex percepts, emotions and memory formation, as well as to identify more specific and effective therapeutic targets for debilitating diseases of mental health and cognitive decline.

Novel, cutting-edge technological advances such as intravital single cell deep brain imaging of sensory thalamus (Taylor et al., 2021; Hasegawa et al., 2024), high-density electrophysiological recordings (Jun et al., 2017), neuromodulator sensors (Zhuo et al., 2024; Dong et al., 2023; Feng et al., 2019; Jing et al., 2020; Deng et al., 2024) and selective neuron manipulation approaches like chemogenetics (Roth, 2016) and optogenetics (Madisen et al., 2012), combined with behavioral testing, will together enable us to advance our understanding of sensory thalamic function, plasticity and neuromodulation in health and disease.

CRediT authorship contribution statement

Ricardo Paricio-Montesinos: Writing – review & editing, Writing – original draft, Resources, Project administration, Funding acquisition, Conceptualization. **Jan Gründemann:** Writing – review & editing, Supervision, Resources, Project administration, Funding acquisition, Conceptualization.

Declaration of Competing Interest

The authors declare no conflicts of interest in relation to this work.

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Data Availability

No data was used for the research described in the article.

References

Adrian, H.O., Lifschitz, W.M., Tavitas, R.J., Galli, F.P., 1966. Activity of neural units in medial geniculate body of cat and rabbit. J. Neurophysiol. 29, 1046–1060. https://doi.org/10.1152/jn.1966.29.6.1046.

Adriano, F., Spoletini, I., Caltagirone, C., Spalletta, G., 2010. Updated meta-analyses reveal thalamus volume reduction in patients with first-episode and chronic schizophrenia. Schizophr. Res 123, 1–14. https://doi.org/10.1016/j.schres.2010.07.007.

Aggleton, J.P., Pralus, A., Nelson, A.J.D., Hornberger, M., 2016. Thalamic pathology and memory loss in early Alzheimer's disease: moving the focus from the medial temporal lobe to papez circuit. Brain 139, 1877–1890. https://doi.org/10.1093/brain/aww083.

Ahissar, E., Sosnik, R., Haidarliu, S., 2000. Transformation from temporal to rate coding in a somatosensory thalamocortical pathway. Nature 406, 302–306. https://doi.org/10.1038/35018568.

Aizenberg, M., Rolón-Martínez, S., Pham, T., Rao, W., Haas, J.S., Geffen, M.N., 2019. Projection from the amygdala to the thalamic reticular nucleus amplifies cortical sound responses. e4 Cell Rep. 28, 605–615. https://doi.org/10.1016/j.celrep.2019.06.050.

Albrecht, D., Quäschling, U., Zippel, U., Davidowa, H., 1996. Effects of dopamine on neurons of the lateral geniculate nucleus: an iontophoretic study. Synapse 23, 70–78. https://doi.org/10.1002/(SICI)1098-2396(199606)23:2<70::AID-SYN2>3.0.CO;2-D.

Almasabi, F., Janssen, M.L.F., Devos, J., Moerel, M., Schwartze, M., Kotz, S.A., et al., 2022. The role of the medial geniculate body of the thalamus in the pathophysiology of tinnitus and implications for treatment. Brain Res. 1779, 147797. https://doi.org/10.1016/j.brainres.2022.147797.

Anderson, L.A., Linden, J.F., 2011. Physiological differences between histologically defined subdivisions in the mouse auditory thalamus. Hear Res 274, 48–60. https://doi.org/10.1016/j.heares.2010.12.016.

Apergis-Schoute, A.M., Debiec, J., Doyère, V., LeDoux, J.E., Schafe, G.E., 2005. Auditory fear conditioning and long-term potentiation in the lateral amygdala require ERK/MAP kinase signaling in the auditory thalamus: a role for presynaptic plasticity in the fear system. J. Neurosci. 25, 5730–5739. https://doi.org/10.1523/JNEUROSCI.0096-05.2005.

Audette, N.J., Bernhard, S.M., Ray, A., Stewart, L.T., Barth, A.L., 2019. Rapid plasticity of Higher-Order thalamocortical inputs during sensory learning. Neuron 103 (277-291), e4. https://doi.org/10.1016/j.neuron.2019.04.037.

Austin, C.P., Ky, B., Ma, L., Morris, J.A., Shughrue, P.J., 2004. Expression of Disrupted-In-Schizophrenia-1, a schizophrenia-associated gene, is prominent in the mouse hippocampus throughout brain development. Neuroscience 124, 3–10. https://doi.org/10.1016/j.neuroscience.2003.11.010.

Ayub, R., Sun, K.L., Flores, R.E., Lam, V.T., Jo, B., Saggar, M., et al., 2021. Thalamocortical connectivity is associated with autism symptoms in high-functioning adults with autism and typically developing adults. Transl. Psychiatry 11, 93. https://doi.org/10.1038/s41398-021-01221-0.

Baez-Nieto, D., Allen, A., Akers-Campbell, S., Yang, L., Budnik, N., Pupo, A., et al., 2022. Analysing an allelic series of rare missense variants of CACNA1I in a Swedish schizophrenia cohort. Brain 145, 1839–1853. https://doi.org/10.1093/brain/awab443.

Balasco, L., Provenzano, G., Bozzi, Y., 2020. Sensory abnormalities in autism spectrum disorders: a focus on the tactile domain, from genetic mouse models to the clinic. Front Psychiatry 10. https://doi.org/10.3389/fpsyt.2019.01016.

Baloyannis, S.J., Mauroudis, I., Manolides, S.L., Manolides, L.S., 2009. Synaptic alterations in the medial geniculate bodies and the inferior colliculi in Alzheimer's disease: a Golgi and electron microscope study. Acta Otolaryngol. 129, 416–418. https://doi.org/10.1080/00016480802579074.

Baran, B., Nguyen, Q.T.H., Mylonas, D., Santangelo, S.L., Manoach, D.S., 2023. Increased resting-state thalamocortical functional connectivity in children and young adults with autism spectrum disorder. Autism Res. 16, 271–279. https://doi.org/10.1002/aur.2875.

Barrientos, R., Alatorre, A., Martínez-Escudero, J., García-Ramírez, M., Oviedo-Chávez, A., Delgado, A., et al., 2019. Effects of local activation and blockade of dopamine D4 receptors in the spiking activity of the reticular thalamic nucleus in normal and in ipsilateral dopamine-depleted rats. Brain Res. 1712, 34–46. https://doi.org/10.1016/j.brainres.2019.01.042.

Barsy, B., Kocsis, K., Magyar, A., Babiczky, Á., Szabó, M., Veres, J.M., Hillier, D., Ulbert, I., Yizhar, O., Mátyás, F., 2020. Associative and plastic thalamic signaling to the lateral amygdala controls fear behavior. Nat. Neurosci. 23 (5), 625–637. https://doi.org/10.1038/s41593-020-0620-z.

Barthó, P., Freund, T.F., Acsády, L., 2002. Selective GABAergic innervation of thalamic nuclei from zona incerta. Eur. J. Neurosci. 16, 999–1014. https://doi.org/10.1046/j.1460-9568.2002.02157.x.

Bazzari, A., Parri, H., 2019. Neuromodulators and Long-Term synaptic plasticity in learning and memory: a Steered-Glutamatergic perspective. Brain Sci. 9, 300. https://doi.org/10.3390/brainsci9110300.

Bedny, M., Pascual-Leone, A., Dodell-Feder, D., Fedorenko, E., Saxe, R., 2011. Language processing in the occipital cortex of congenitally blind adults. Proc. Natl. Acad. Sci. 108, 4429–4434. https://doi.org/10.1073/pnas.1014818108.

Beebe, N.L., Mellott, J.G., Schofield, B.R., 2018. Inhibitory projections from the inferior colliculus to the medial geniculate body originate from four subtypes of GABAergic cells. ENeuro 5. https://doi.org/10.1523/ENEURO.0406-18.2018.

Beierlein, M., 2014. Synaptic mechanisms underlying cholinergic control of thalamic reticular nucleus neurons. J. Physiol. 592, 4137–4145. https://doi.org/10.1113/jphysiol.2014.277376.

Biesbroek, J.M., Verhagen, M.G., van der Stigchel, S., Biessels, G.J., 2024. When the central integrator disintegrates: a review of the role of the thalamus in cognition and dementia. Alzheimer'S. Dement. 20, 2209–2222. https://doi.org/10.1002/alz.13563.

Billet, S., Cant, N.B., Hall, W.C., 1999. Cholinergic projections to the visual thalamus and superior colliculus. Brain Res. 847, 121–123. https://doi.org/10.1016/S0006-8993(99)01900-9.

Bishop, P.O., Kozak, W., Levick, W.R., Vakkur, G.J., 1962. The determination of the projection of the visual field on to the lateral geniculate nucleus in the cat. J. Physiol. 163, 503–539. https://doi.org/10.1113/jphysiol.1962.sp006991.

Bokiniec, P., Zampieri, N., Lewin, G.R., Poulet, J.F., 2018. The neural circuits of thermal perception. Curr. Opin. Neurobiol. 52, 98–106. https://doi.org/10.1016/j.conb.2018.04.006.

Bokor, H., Frère, S.G.A., Eyre, M.D., Slézia, A., Ulbert, I., Lüthi, A., et al., 2005. Selective GABAergic control of higher-order thalamic relays. Neuron 45, 929–940. https://doi.org/10.1016/j.neuron.2005.01.048.

Boucetta, S., Cissé, Y., Mainville, L., Morales, M., Jones, B.E., 2014. Discharge profiles across the sleep-waking cycle of identified cholinergic, GABAergic, and glutamatergic neurons in the pontomesencephalic tegmentum of the rat. J. Neurosci. 34, 4708–4727. https://doi.org/10.1523/JNEUROSCI.2617-13.2014.

Braak, H., Braak, E., 1991. Neuropathological stageing of Alzheimer-related changes. Acta Neuropathol. 82, 239–259. https://doi.org/10.1007/BF00308809.

Brauth, S.E., Liang, W., Tang, Y., Galdzicka, E., Hall, W.S., 2007. Rapid contact call-driven induction of NR2A and NR2B NMDA subunit mRNAs in the auditory thalamus of the budgerigar (Melopsittacus undulatus). Neurobiol. Learn Mem. 88, 33–39. https://doi.org/10.1016/j.nlm.2007.03.006.

Brisch, R., Saniotis, A., Wolf, R., Bielau, H., Bernstein, H.-G., Steiner, J., et al., 2014. The role of dopamine in schizophrenia from a neurobiological and evolutionary perspective: old fashioned, but still in vogue. Front. Psychiatry 5, 47. https://doi.org/10.3389/fpsyt.2014.00047.

Brzosko, Z., Mierau, S.B., Paulsen, O., 2019. Neuromodulation of Spike-Timing-Dependent plasticity: past, present, and future. Neuron 103, 563–581. https://doi.org/10.1016/j.neuron.2019.05.041.

Buchsbaum, M.S., Someya, T., Teng, C.Y., Abel, L., Chin, S., Najafi, A., et al., 1996. PET and MRI of the thalamus in never-medicated patients with schizophrenia. Am. J. Psychiatry 153, 191–199. https://doi.org/10.1176/ajp.153.2.191.

Buchwald, J.S., Halas, E.S., Schramm, S., 1966. Changes in cortical and subcortical unit activity during behavioral conditioning, 11-IN7 Physiol. Behav. 1. https://doi.org/10.1016/0031-9384(66)90037-0.

Butler, A.B., 2008. Evolution of the thalamus: a morphological and functional review. Thalamus Relat. Syst. 4. https://doi.org/10.1017/S1472928808000356.

Cacciaglia, R., Escera, C., Slabu, L., Grimm, S., Sanjuán, A., Ventura-Campos, N., et al., 2015. Involvement of the human midbrain and thalamus in auditory deviance detection. Neuropsychologia 68, 51–58. https://doi.org/10.1016/j.neuropsychologia.2015.01.001.

Casas-Torremocha, D., Clascá, F., Núñez, Á., 2017. Posterior thalamic nucleus modulation of tactile stimuli processing in rat motor and primary somatosensory cortices. Front. Neural Circuits 11, 69. https://doi.org/10.3389/fncir.2017.00069.

Casas-Torremocha, D., Porrero, C., Rodriguez-Moreno, J., García-Amado, M., Lübke, J.H.R., Núñez, Á., et al., 2019. Posterior thalamic nucleus axon terminals have different structure and functional impact in the motor and somatosensory vibrissal cortices. Brain Struct. Funct. 224, 1627–1645. https://doi.org/10.1007/s00429-019-01862-4.

Cassel, J.-C., Pereira de Vasconcelos, A., 2021. Routes of the thalamus through the history of neuroanatomy. Neurosci. Biobehav Rev. 125, 442–465. https://doi.org/10.1016/j.neubiorev.2021.03.001.

Castro-Alamancos, M.A., Calcagnotto, M.E., 1999. Presynaptic Long-Term potentiation in corticothalamic synapses. J. Neurosci. 19, 9090–9097. https://doi.org/10.1523/JNEUROSCI.19-20-09090.1999.

Castro-Alamancos, M.A., Calcagnotto, M.E., 2001. High-Pass filtering of corticothalamic activity by neuromodulators released in the thalamus during arousal: in vitro and in vivo. J. Neurophysiol. 85, 1489–1497. https://doi.org/10.1152/jn.2001.85.4.1489.

Chen, X., Aslam, M., Gollisch, T., Allen, K., von Engelhardt, J., 2018. CKAMP44 modulates integration of visual inputs in the lateral geniculate nucleus. Nat. Commun. 9, 261. https://doi.org/10.1038/s41467-017-02415-1.

Chen, C., Blitz, D.M., Regehr, W.G., 2002. Contributions of receptor desensitization and saturation to plasticity at the retinogeniculate synapse. Neuron 33, 779–788. https://doi.org/10.1016/S0896-6273(02)00611-6.

Chibaatar, E., Watanabe, K., Okamoto, N., Orkhonselenge, N., Natsuyama, T., Hayakawa, G., et al., 2023. Volumetric assessment of individual thalamic nuclei in patients with drug-naïve, first-episode major depressive disorder. Front Psychiatry 14. https://doi.org/10.3389/fpsyt.2023.1151551.

Chun, S., Du, F., Westmoreland, J.J., Han, S.B., Wang, Y.-D., Eddins, D., et al., 2017. Thalamic miR-338-3p mediates auditory thalamocortical disruption and its late onset in models of 22q11.2 microdeletion. Nat. Med. 23, 39–48. https://doi.org/10.1038/nm.4240.

Chun, S., Westmoreland, J.J., Bayazitov, I.T., Eddins, D., Pani, A.K., Smeyne, R.J., et al., 2014. Specific disruption of thalamic inputs to the auditory cortex in schizophrenia models. Science 344 (1979), 1178–1182. https://doi.org/10.1126/science.1253895.

Cook, J.A., Barry, K.M., Zimdahl, J.W., Leggett, K., Mulders, W.H.A.M., 2021. Spontaneous firing patterns in the medial geniculate nucleus in a Guinea pig model of tinnitus. Hear Res. 403, 108190. https://doi.org/10.1016/j.heares.2021.108190.
Cook, J.L., Blakemore, S.-J., Press, C., 2013. Atypical basic movement kinematics in autism spectrum conditions. Brain 136, 2816–2824. https://doi.org/10.1093/brain/awt208.
Cornwall, J., Cooper, J.D., Phillipson, O.T., 1990. Projections to the rostral reticular thalamic nucleus in the rat. Exp. Brain Res. 80. https://doi.org/10.1007/BF00228857.
Courtiol, E., Wilson, D.A., 2015. The olfactory thalamus: unanswered questions about the role of the mediodorsal thalamic nucleus in olfaction. Front. Neural Circuits 9. https://doi.org/10.3389/fncir.2015.00049.
Craig, A.D., Bushnell, M.C., Zhang, E.-T., Blomqvist, A., 1994. A thalamic nucleus specific for pain and temperature sensation. Nature 372, 770–773. https://doi.org/10.1038/372770a0.
Crandall, S.R., Cruikshank, S.J., Connors, B.W., 2015. A corticothalamic switch: controlling the thalamus with dynamic synapses. Neuron 86, 768–782. https://doi.org/10.1016/j.neuron.2015.03.040.
Crick, F., 1984. Function of the thalamic reticular complex: the searchlight hypothesis. Proc. Natl. Acad. Sci. 81, 4586–4590. https://doi.org/10.1073/pnas.81.14.4586.
Deng, F., Wan, J., Li, G., Dong, H., Xia, X., Wang, Y., et al., 2024. Improved Green and red GRAB sensors for monitoring spatiotemporal serotonin release in vivo. Nat. Methods 21, 692–702. https://doi.org/10.1038/s41592-024-02188-8.
Devilbiss, D.M., Waterhouse, B.D., 2011. Phasic and tonic patterns of locus coeruleus output differentially modulate sensory network function in the awake rat. J. Neurophysiol. 105, 69–87. https://doi.org/10.1152/jn.00445.2010.
Diamond, M.E., Armstrong-James, M., Budway, M.J., Ebner, F.F., 1992. Somatic sensory responses in the rostral sector of the posterior group (POm) and in the ventral posterior medial nucleus (VPM) of the rat thalamus: dependence on the barrel field cortex. J. Comp. Neurol. 319, 66–84. https://doi.org/10.1002/cne.903190108.
Diamond, M.E., von Heimendahl, M., Knutsen, P.M., Kleinfeld, D., Ahissar, E., 2008. "Where" and "what" in the whisker sensorimotor system. Nat. Rev. Neurosci. 9, 601–612. https://doi.org/10.1038/nrn2411.
Díaz, B., Hintz, F., Kiebel, S.J., von Kriegstein, K., 2012. Dysfunction of the auditory thalamus in developmental dyslexia. Proc. Natl. Acad. Sci. 109, 13841–13846. https://doi.org/10.1073/pnas.1119828109.
Diniz CRAF, Crestani A.P., 2023. The Times they are a-changin': a proposal on how brain flexibility goes beyond the obvious to include the concepts of "upward" and "downward" to neuroplasticity. Mol. Psychiatry 28, 977–992. https://doi.org/10.1038/s41380-022-01931-x.
Disterhoft, J.F., Olds, J., 1972. Differential development of conditioned unit changes in thalamus and cortex of rat. J. Neurophysiol. 35, 665–679. https://doi.org/10.1152/jn.1972.35.5.665.
Dong, H., Li, M., Yan, Y., Qian, T., Lin, Y., Ma, X., et al., 2023. Genetically encoded sensors for measuring histamine release both in vitro and in vivo. e6 Neuron 111, 1564–1576. https://doi.org/10.1016/j.neuron.2023.02.024.
Dorph-Petersen, K.-A., Caric, D., Saghafi, R., Zhang, W., Sampson, A.R., Lewis, D.A., 2009. Volume and neuron number of the lateral geniculate nucleus in schizophrenia and mood disorders. Acta Neuropathol. 117, 369–384. https://doi.org/10.1007/s00401-008-0410-2.
Edeline, J.-M., Weinberger, N.M., 1991a. Subcortical adaptive filtering in the auditory system: associative receptive field plasticity in the dorsal medial geniculate body. Behav. Neurosci. 105, 154–175. https://doi.org/10.1037/0735-7044.105.1.154.
Edeline, J.M., Weinberger, N.M., 1991b. Thalamic short-term plasticity in the auditory system: associative returning of receptive fields in the ventral medial geniculate body. Behav. Neurosci. 105, 618–639. https://doi.org/10.1037//0735-7044.105.5.618.
El-Boustani, S., Sermet, B.S., Foustoukos, G., Oram, T.B., Yizhar, O., Petersen, C.C.H., 2020. Anatomically and functionally distinct thalamocortical inputs to primary and secondary mouse whisker somatosensory cortices. Nat. Commun. 11, 3342. https://doi.org/10.1038/s41467-020-17087-7.
English, G., Ghasemi Nejad, N., Sommerfelt, M., Yanik, M.F., von der Behrens, W., 2023. Bayesian surprise shapes neural responses in somatosensory cortical circuits. Cell Rep. 42, 112009. https://doi.org/10.1016/j.celrep.2023.112009.
Feng, J., Zhang, C., Lischinsky, J.E., Jing, M., Zhou, J., Wang, H., et al., 2019. A genetically encoded fluorescent sensor for rapid and specific in vivo detection of norepinephrine. e8 Neuron 102, 745–761. https://doi.org/10.1016/j.neuron.2019.02.037.
Fernandez, L.M.J., Pellegrini, C., Vantomme, G., Béard, E., Lüthi, A., Astori, S., 2017. Cortical afferents onto the nucleus reticularis thalami promote plasticity of low-threshold excitability through GluN2C-NMDARs. Sci. Rep. 7, 12271. https://doi.org/10.1038/s41598-017-12552-8.
Ferrarelli, F., Huber, R., Peterson, M.J., Massimini, M., Murphy, M., Riedner, B.A., et al., 2007. Reduced sleep spindle activity in schizophrenia patients. Am. J. Psychiatry 164, 483–492. https://doi.org/10.1176/ajp.2007.164.3.483.
Ferrarelli, F., Tononi, G., 2011. The thalamic reticular nucleus and schizophrenia. Schizophr. Bull. 37, 306–315. https://doi.org/10.1093/schbul/sbq142.
Forno, G., Saranathan, M., Contador, J., Guillen, N., Falgàs, N., Tort-Merino, A., et al., 2023. Thalamic nuclei changes in early and late onset Alzheimer's disease. Curr. Res. Neurobiol. 4, 100084. https://doi.org/10.1016/j.crneur.2023.100084.
Fredericksen, K.E., Samuelsen, C.L., 2022. Neural representation of intraoral olfactory and gustatory signals by the mediodorsal thalamus in alert rats. J. Neurosci. 42, 8136–8153. https://doi.org/10.1523/JNEUROSCI.0674-22.2022.
Furutachi, S., Franklin, A.D., Aldea, A.M., Mrsic-Flogel, T.D., Hofer, S.B., 2024. Cooperative thalamocortical circuit mechanism for sensory prediction errors. Nature 633, 398–406. https://doi.org/10.1038/s41586-024-07851-w.
Gabriel, M., Saltwick, S.E., Miller, J.D., 1975. Conditioning and reversal of short-latency multiple-unit responses in the rabbit medial geniculate nucleus. Science 189, 1108–1109. https://doi.org/10.1126/science.1162365.
Galaburda, A., Livingstone, M., 1993. Evidence for a magnocellular defect in developmental dyslexia. Ann. N. Y Acad. Sci. 682, 70–82. https://doi.org/10.1111/j.1749-6632.1993.tb22960.x.
García-Cabezas, M.Á., Pérez-Santos, I., Cavada, C., 2021. The epic of the thalamus in anatomical language. Front. Neuroanat. 15, 744095. https://doi.org/10.3389/fnana.2021.744095.
Gasca-Martinez, D., Hernandez, A., Sierra, A., Valdiosera, R., Anaya-Martinez, V., Floran, B., et al., 2010. Dopamine inhibits GABA transmission from the globus pallidus to the thalamic reticular nucleus via presynaptic D4 receptors. Neuroscience 169, 1672–1681. https://doi.org/10.1016/j.neuroscience.2010.05.048.
Gauriau, C., Bernard, J.-F., 2004. Posterior triangular thalamic neurons convey nociceptive messages to the secondary somatosensory and insular cortices in the rat. J. Neurosci. 24, 752–761. https://doi.org/10.1523/JNEUROSCI.3272-03.2004.
Gilad, A., Maor, I., Mizrahi, A., 2020. Learning-related population dynamics in the auditory thalamus. Elife 9. https://doi.org/10.7554/eLife.56307.
Goitia, B., Rivero-Echeto, M.C., Weisstaub, N.V., Gingrich, J.A., Garcia-Rill, E., Bisagno, V., et al., 2016. Modulation of GABA release from the thalamic reticular nucleus by cocaine and caffeine: role of serotonin receptors. J. Neurochem. 136, 526–535. https://doi.org/10.1111/jnc.13398.
Gold, J.I., Knudsen, E.I., 1999. Hearing impairment induces Frequency-Specific adjustments in auditory spatial tuning in the optic tectum of young owls. J. Neurophysiol. 82, 2197–2209. https://doi.org/10.1152/jn.1999.82.5.2197.
Gold, J.I., Knudsen, E.I., 2000. A site of auditory Experience-Dependent plasticity in the neural representation of auditory space in the barn Owl's inferior colliculus. J. Neurosci. 20, 3469–3486. https://doi.org/10.1523/JNEUROSCI.20-09-03469.2000.
Gonzalo-Ruiz, A., Lieberman, A.R., Sanz-Anquela, J.M., 1995. Organization of serotoninergic projections from the raphé nuclei to the anterior thalamic nuclei in the rat: a combined retrograde tracing and 5-HT immunohistochemical study. J. Chem. Neuroanat. 8, 103–115. https://doi.org/10.1016/0891-0618(94)00039-V.
Gorin, A.S., Miao, Y., Ahn, S., Suresh, V., Su, Y., Ciftcioglu, U.M., et al., 2023. Local interneurons in the murine visual thalamus have diverse receptive fields and can provide feature selective inhibition to relay cells. BioRxiv. https://doi.org/10.1101/2023.08.10.549394.
Govindaiah, G., Cox, C.L., 2005. Excitatory actions of dopamine via D1-Like receptors in the rat lateral geniculate nucleus. J. Neurophysiol. 94, 3708–3718. https://doi.org/10.1152/jn.00583.2005.
Govindaiah, G., Cox, C.L., 2006. Depression of retinogeniculate synaptic transmission by presynaptic D2-like dopamine receptors in rat lateral geniculate nucleus. Eur. J. Neurosci. 23, 423–434. https://doi.org/10.1111/j.1460-9568.2005.04575.x.
Govindaiah, G., Wang, Y., Cox, C.L., 2010a. Dopamine enhances the excitability of somatosensory thalamocortical neurons. Neuroscience 170, 981–991. https://doi.org/10.1016/j.neuroscience.2010.08.043.
Govindaiah, G., Wang, T., Gillette, M.U., Crandall, S.R., Cox, C.L., 2010b. Regulation of inhibitory synapses by presynaptic D_4 dopamine receptors in thalamus. J. Neurophysiol. 104, 2757–2765. https://doi.org/10.1152/jn.00361.2010.
Halassa, M.M., Acsády, L., 2016. Thalamic inhibition: diverse sources, diverse scales. Trends Neurosci. 39, 680–693. https://doi.org/10.1016/j.tins.2016.08.001.
Halassa, M.M., Sherman, S.M., 2019. Thalamocortical circuit motifs: a general framework. Neuron 103, 762–770. https://doi.org/10.1016/j.neuron.2019.06.005.
Halassa, M.H., Kastner, S., 2017. Thalamic functions in distributed cognitive control. Nat Neurosci 20 (12), 1669–1679. https://doi.org/10.1038/s41593-017-0020-1.
Hallanger, A.E., Levey, A.I., Lee, H.J., Rye, D.B., Wainer, B.H., 1987. The origins of cholinergic and other subcortical afferents to the thalamus in the rat. J. Comp. Neurol. 262, 105–124. https://doi.org/10.1002/cne.902620109.
Halverson, H.E., Lee, I., Freeman, J.H., 2010. Associative plasticity in the medial auditory thalamus and cerebellar interpositus nucleus during eyeblink conditioning. J. Neurosci. 30, 8787–8796. https://doi.org/10.1523/JNEUROSCI.0208-10.2010.
Hamann, S., Monarch, E.S., Goldstein, F.C., 2002. Impaired fear conditioning in Alzheimer's disease. Neuropsychologia 40, 1187–1195. https://doi.org/10.1016/S0028-3932(01)00223-8.
Han, J.-H., Yiu, A.P., Cole, C.J., Hsiang, H.-L., Neve, R.L., Josselyn, S.A., 2008. Increasing CREB in the auditory thalamus enhances memory and generalization of auditory conditioned fear. Learn Mem. 15, 443–453. https://doi.org/10.1101/lm.993608.
Hasegawa, M., Huang, Z., Paricio-Montesinos, R., Gründemann, J., 2024. Network state changes in sensory thalamus represent learned outcomes. Nat. Commun. 15, 7830. https://doi.org/10.1038/s41467-024-51868-8.
Hazra, A., Corbett, B.F., You, J.C., Aschmies, S., Zhao, L., Li, K., et al., 2016. Corticothalamic network dysfunction and behavioral deficits in a mouse model of Alzheimer's disease. Neurobiol. Aging 44, 96–107. https://doi.org/10.1016/j.neurobiolaging.2016.04.016.
Heckers, S., Geula, C., Mesulam, M.M., 1992. Cholinergic innervation of the human thalamus: dual origin and differential nuclear distribution. J. Comp. Neurol. 325, 68–82. https://doi.org/10.1002/cne.903250107.
Hirata, A., Aguilar, J., Castro-Alamancos, M.A., 2006. Noradrenergic activation amplifies Bottom-Up and Top-Down Signal-to-Noise ratios in sensory thalamus. J. Neurosci. 26, 4426–4436. https://doi.org/10.1523/JNEUROSCI.5298-05.2006.
Howarth, M., Walmsley, L., Brown, T.M., 2014. Binocular integration in the mouse lateral geniculate nuclei. Curr. Biol. 24, 1241–1247. https://doi.org/10.1016/j.cub.2014.04.014.
Hu, C., Hasenstaub, A.R., Schreiner, C.E., 2024. Basic properties of coordinated neuronal ensembles in the auditory thalamus. J. Neurosci. 44. https://doi.org/10.1523/JNEUROSCI.1729-23.2024.

Huang, C.L., Larue, D.T., Winer, J.A., 1999. GABAergic organization of the cat medial geniculate body. J. Comp. Neurol. 415, 368–392.
Huang, L., Xi, Y., Peng, Y., Yang, Y., Huang, X., Fu, Y., et al., 2019. A visual circuit related to habenula underlies the antidepressive effects of light therapy. e8 Neuron 102, 128–142. https://doi.org/10.1016/j.neuron.2019.01.037.
Hwang, K., Bertolero, M.A., Liu, W.B., D'Esposito, M., 2017. The human thalamus is an integrative hub for functional brain networks. J. Neurosci. 37, 5594–5607. https://doi.org/10.1523/JNEUROSCI.0067-17.2017.
Iossifov, I., Ronemus, M., Levy, D., Wang, Z., Hakker, I., Rosenbaum, J., et al., 2012. De novo gene disruptions in children on the autistic spectrum. Neuron 74, 285–299. https://doi.org/10.1016/j.neuron.2012.04.009.
Jaepel, J., Hübener, M., Bonhoeffer, T., Rose, T., 2017. Lateral geniculate neurons projecting to primary visual cortex show ocular dominance plasticity in adult mice. Nat. Neurosci. 20, 1708–1714. https://doi.org/10.1038/s41593-017-0021-0.
Jager, P., Moore, G., Calpin, P., Durmishi, X., Salgarella, I., Menage, L., et al., 2021. Dual midbrain and forebrain origins of thalamic inhibitory interneurons. Elife 10. https://doi.org/10.7554/eLife.59272.
Jagirdar, R., Fu, C.-H., Park, J., Corbett, B.F., Seibt, F.M., Beierlein, M., et al., 2021. Restoring activity in the thalamic reticular nucleus improves sleep architecture and reduces Aβ accumulation in mice. eabh4284 Sci. Transl. Med 13. https://doi.org/10.1126/scitranslmed.abh4284.
Jiang, Y., Patton, M.H., Zakharenko, S.S., 2021. A case for thalamic mechanisms of schizophrenia: perspective from modeling 22q11.2 deletion syndrome. Front Neural Circuits 15. https://doi.org/10.3389/fncir.2021.769969.
Jin, C.Y., Kalimo, H., Panula, P., 2002. The histaminergic system in human thalamus: correlation of innervation to receptor expression. Eur. J. Neurosci. 15, 1125–1138. https://doi.org/10.1046/j.1460-9568.2002.01951.x.
Jin, C., Lintunen, M., Panula, P., 2005. Histamine H1 and H3 receptors in the rat thalamus and their modulation after systemic kainic acid administration. Exp. Neurol. 194, 43–56. https://doi.org/10.1016/j.expneurol.2005.01.012.
Jing, M., Li, Y., Zeng, J., Huang, P., Skirzewski, M., Kljakic, O., et al., 2020. An optimized acetylcholine sensor for monitoring in vivo cholinergic activity. Nat. Methods 17, 1139–1146. https://doi.org/10.1038/s41592-020-0953-2.
Jones, E.G., 1991. The anatomy of sensory relay functions in the thalamus, 1991, pp. 29–52. https://doi.org/10.1016/S0079-6123(08)63046-0.
Jourdain, A., Semba, K., Fibiger, H.C., 1989. Basal forebrain and mesopontine tegmental projections to the reticular thalamic nucleus: an axonal collateralization and immunohistochemical study in the rat. Brain Res 505, 55–65. https://doi.org/10.1016/0006-8993(89)90115-7.
Jun, J.J., Steinmetz, N.A., Siegle, J.H., Denman, D.J., Bauza, M., Barbarits, B., et al., 2017. Fully integrated silicon probes for high-density recording of neural activity. Nature 551, 232–236. https://doi.org/10.1038/nature24636.
Kamke, M.R., Brown, M., Irvine, D.R.F., 2003. Plasticity in the tonotopic organization of the medial geniculate body in adult cats following restricted unilateral cochlear lesions. J. Comp. Neurol. 459, 355–367. https://doi.org/10.1002/cne.10586.
Kanai, R., Komura, Y., Shipp, S., Friston, K., 2015. Cerebral hierarchies: predictive processing, precision and the pulvinar. Philos. Trans. R. Soc. Lond. B Biol. Sci. 370. https://doi.org/10.1098/rstb.2014.0169.
Karavallil Achuthan, S., Stavrinos, D., Argueta, P., Vanderburgh, C., Holm, H.B., Kana, R. K., 2023. Thalamic functional connectivity and sensorimotor processing in neurodevelopmental disorders. Front Neurosci. 17. https://doi.org/10.3389/fnins.2023.1279909.
Keller, G.B., Mrsic-Flogel, T.D., 2018. Predictive processing: a canonical cortical computation. Neuron 100, 424–435. https://doi.org/10.1016/j.neuron.2018.10.003.
Kirchgessner, M.A., Franklin, A.D., Callaway, E.M., 2020. Context-dependent and dynamic functional influence of corticothalamic pathways to first- and higher-order visual thalamus. Proc. Natl. Acad. Sci. USA 117, 13066–13077. https://doi.org/10.1073/pnas.2002080117.
Kirifides, M.L., Simpson, K.L., Lin, R.C. -S., Waterhouse, B.D., 2001. Topographic organization and neurochemical identity of dorsal raphe neurons that project to the trigeminal somatosensory pathway in the rat. J. Comp. Neurol. 435, 325–340. https://doi.org/10.1002/cne.1033.
Knafo, S., Venero, C., Merino-Serrais, P., Fernaud-Espinosa, I., Gonzalez-Soriano, J., Ferrer, I., et al., 2009. Morphological alterations to neurons of the amygdala and impaired fear conditioning in a transgenic mouse model of Alzheimer's disease. J. Pathol. 219, 41–51. https://doi.org/10.1002/path.2565.
Knill, D.C., Pouget, A., 2004. The Bayesian brain: the role of uncertainty in neural coding and computation. Trends Neurosci. 27, 712–719. https://doi.org/10.1016/j.tins.2004.10.007.
Knopman, D.S., Amieva, H., Petersen, R.C., Chételat, G., Holtzman, D.M., Hyman, B.T., et al., 2021. Alzheimer disease. Nat. Rev. Dis. Prim. 7, 33. https://doi.org/10.1038/s41572-021-00269-y.
Koyama, N., Nishikawa, Y., Yokota, T., 1998. Distribution of nociceptive neurons in the ventrobasal complex of macaque thalamus. Neurosci. Res 31, 39–51. https://doi.org/10.1016/S0168-0102(98)00021-2.
Krol, A., Wimmer, R.D., Halassa, M.M., Feng, G., 2018. Thalamic reticular dysfunction as a circuit endophenotype in neurodevelopmental disorders. Neuron 98, 282–295. https://doi.org/10.1016/j.neuron.2018.03.021.
Krupa, D.J., Ghazanfar, A.A., Nicolelis, M.A., 1999. Immediate thalamic sensory plasticity depends on corticothalamic feedback. Proc. Natl. Acad. Sci. USA 96, 8200–8205. https://doi.org/10.1073/pnas.96.14.8200.
Kurzawski, J.W., Lunghi, C., Biagi, L., Tosetti, M., Morrone, M.C., Binda, P., 2022. Short-term plasticity in the human visual thalamus. Elife 11. https://doi.org/10.7554/eLife.74565.
La Terra, D., Bjerre, A.-S., Rosier, M., Masuda, R., Ryan, T.J., Palmer, L.M., 2022. The role of higher-order thalamus during learning and correct performance in goal-directed behavior. Elife 11. https://doi.org/10.7554/eLife.77177.
Lai, M.-C., Lombardo, M.V., Baron-Cohen, S., 2014. Autism. Lancet 383, 896–910. https://doi.org/10.1016/S0140-6736(13)61539-1.
Lau, C.-I., Wang, H.-C., Hsu, J.-L., Liu, M.-E., 2013. Does the dopamine hypothesis explain schizophrenia. Rev. Neurosci. 24, 389–400. https://doi.org/10.1515/revneuro-2013-0011.
Lavin, A., Grace, A.A., 1998. Dopamine modulates the responsivity of mediodorsal thalamic cells recorded in vitro. J. Neurosci. 18, 10566–10578. https://doi.org/10.1523/JNEUROSCI.18-24-10566.1998.
Le Gros Clark, W.E., Penman, G.G., 1934. The projection of the retina in the lateral geniculate body. Proceedings of the royal society of London series b. Contain. Pap. a Biol. Character 114, 291–313. https://doi.org/10.1098/rspb.1934.0008.
Leaver, A.M., Renier, L., Chevillet, M.A., Morgan, S., Kim, H.J., Rauschecker, J.P., 2011. Dysregulation of limbic and auditory networks in tinnitus. Neuron 69, 33–43. https://doi.org/10.1016/j.neuron.2010.12.002.
Lee, C.C., Sherman, S.M., 2010. Drivers and modulators in the central auditory pathways. Front. Neurosci. https://doi.org/10.3389/neuro.01.014.2010.
Lee, C.C., Sherman, S.M., 2011. On the classification of pathways in the auditory midbrain, thalamus, and cortex. Hear Res. 276, 79–87. https://doi.org/10.1016/j.heares.2010.12.012.
Leva, T., Whitmire, C., Sauve, I., Bokiniec, P., Memler, C., Horn, B., et al., 2024. The cellular representation of temperature across the somatosensory thalamus. BioRxiv. https://doi.org/10.1101/2024.02.13.580167.
Li, Y., Lopez-Huerta, V.G., Adiconis, X., Levandowski, K., Choi, S., Simmons, S.K., et al., 2020. Distinct subnetworks of the thalamic reticular nucleus. Nature 583, 819–824. https://doi.org/10.1038/s41586-020-2504-5.
Li, X., Phillips, R., LeDoux, JosephE., 1995. NMDA and non-NMDA receptors contribute to synaptic transmission between the medial geniculate body and the lateral nucleus of the amygdala. Exp. Brain Res. 105. https://doi.org/10.1007/BF00242185.
Li, H.-Y., Zhu, M.-Z., Yuan, X.-R., Guo, Z.-X., Pan, Y.-D., Li, Y.-Q., et al., 2023. A thalamic-primary auditory cortex circuit mediates resilience to stress. e18 Cell 186, 1352–1368. https://doi.org/10.1016/j.cell.2023.02.036.
Linke, A.C., Chen, B., Olson, L., Ibarra, C., Fong, C., Reynolds, S., et al., 2023. Sleep problems in preschoolers with autism spectrum disorder are associated with sensory sensitivities and thalamocortical overconnectivity. Biol. Psychiatry Cogn. Neurosci. Neuroimaging 8, 21–31. https://doi.org/10.1016/j.bpsc.2021.07.008.
Liu, X., Warren, R.A., Jones, E.G., 1995. Synaptic distribution of afferents from reticular nucleus in ventroposterior nucleus of cat thalamus. J. Comp. Neurol. 352, 187–202. https://doi.org/10.1002/cne.903520203.
Love, J.A., Scott, J.W., 1969. Some response characteristics of cells of the magnocellular division of the medial geniculate body of the cat. Can. J. Physiol. Pharm. 47, 881–888. https://doi.org/10.1139/y69-145.
Lund, R.D., Webster, K.E., 1967. Thalamic afferents from the dorsal column nuclei. An experimental anatomical study in the rat. J. Comp. Neurol. 130, 301–311. https://doi.org/10.1002/cne.901300403.
Madisen, L., Mao, T., Koch, H., Zhuo, J., Berenyi, A., Fujisawa, S., et al., 2012. A toolbox of Cre-dependent optogenetic transgenic mice for light-induced activation and silencing. Nat. Neurosci. 15, 793–802. https://doi.org/10.1038/nn.3078.
Mancini, V., Zöller, D., Schneider, M., Schaer, M., Eliez, S., 2020. Abnormal development and dysconnectivity of distinct thalamic nuclei in patients with 22q11.2 deletion syndrome experiencing auditory hallucinations. Biol. Psychiatry Cogn. Neurosci. Neuroimaging 5, 875–890. https://doi.org/10.1016/j.bpsc.2020.04.015.
Mansour, Y., Ahmed, S.N., Kulesza, R., 2021. Abnormal morphology and subcortical projections to the medial geniculate in an animal model of autism. Exp. Brain Res. 239, 381–400. https://doi.org/10.1007/s00221-020-05982-w.
Marenco, S., Stein, J.L., Savostyanova, A.A., Sambataro, F., Tan, H.-Y., Goldman, A.L., et al., 2012. Investigation of anatomical Thalamo-Cortical connectivity and fMRI activation in schizophrenia. Neuropsychopharmacology 37, 499–507. https://doi.org/10.1038/npp.2011.215.
McAlonan, K., Cavanaugh, J., Wurtz, R.H., 2006. Attentional modulation of thalamic reticular neurons. J. Neurosci. 26, 4444–4450. https://doi.org/10.1523/JNEUROSCI.5602-05.2006.
McCormick, D., Williamson, A., 1991. Modulation of neuronal firing mode in cat and Guinea pig LGNd by histamine: possible cellular mechanisms of histaminergic control of arousal. J. Neurosci. 11, 3188–3199. https://doi.org/10.1523/JNEUROSCI.11-10-03188.1991.
Mease, R.A., Krieger, P., Groh, A., 2014. Cortical control of adaptation and sensory relay mode in the thalamus. Proc. Natl. Acad. Sci. 111, 6798–6803. https://doi.org/10.1073/pnas.1318665111.
Mellott, J.G., Foster, N.L., Ohl, A.P., Schofield, B.R., 2014. Excitatory and inhibitory projections in parallel pathways from the inferior colliculus to the auditory thalamus. Front. Neuroanat. 8. https://doi.org/10.3389/fnana.2014.00124.
Miller-Hansen, A.J., Sherman, S.M., 2022. Conserved patterns of functional organization between cortex and thalamus in mice. Proc. Natl. Acad. Sci. 119. https://doi.org/10.1073/pnas.2201481119.
Mo, C., McKinnon, C., Murray Sherman, S., 2024. A transthalamic pathway crucial for perception. Nat. Commun. 15, 6300. https://doi.org/10.1038/s41467-024-50163-w.
Monckton, J.E., McCormick, D.A., 2002. Neuromodulatory role of serotonin in the ferret thalamus. J. Neurophysiol. 87, 2124–2136. https://doi.org/10.1152/jn.00650.2001.
Mooney, D.M., Zhang, L., Basile, C., Senatorov, V.V., Ngsee, J., Omar, A., et al., 2004. Distinct forms of cholinergic modulation in parallel thalamic sensory pathways. Proc. Natl. Acad. Sci. 101, 320–324. https://doi.org/10.1073/pnas.0304445101.

Mørch-Johnsen, L., Jørgensen, K.N., Barth, C., Nerland, S., Bringslid, I.K., Wortinger, L. A., et al., 2023. Thalamic nuclei volumes in schizophrenia and bipolar spectrum disorders – associations with diagnosis and clinical characteristics. Schizophr. Res 256, 26–35. https://doi.org/10.1016/j.schres.2023.04.008.
Moxon, K.A., Devilbiss, D.M., Chapin, J.K., Waterhouse, B.D., 2007. Influence of norepinephrine on somatosensory neuronal responses in the rat thalamus: a combined modeling and in vivo multi-channel, multi-neuron recording study. Brain Res. 1147, 105–123. https://doi.org/10.1016/j.brainres.2007.02.006.
Müller-Axt, C., Anwander, A., von Kriegstein, K., 2017. Altered structural connectivity of the left visual thalamus in developmental dyslexia. e4 Curr. Biol. 27, 3692–3698. https://doi.org/10.1016/j.cub.2017.10.034.
Nakajima, M., Schmitt, L.I., Halassa, M.M., 2019. Prefrontal cortex regulates sensory filtering through a basal Ganglia-to-Thalamus pathway. e10 Neuron 103, 445–458. https://doi.org/10.1016/j.neuron.2019.05.026.
Nersisyan, S., Bekisz, M., Kublik, E., Granseth, B., Wróbel, A., 2021. Cholinergic and noradrenergic modulation of corticothalamic synaptic input from layer 6 to the posteromedial thalamic nucleus in the rat. Front Neural Circuits 15. https://doi.org/10.3389/fncir.2021.624381.
Ni, K.-M., Hou, X.-J., Yang, C.-H., Dong, P., Li, Y., Zhang, Y., et al., 2016. Selectively driving cholinergic fibers optically in the thalamic reticular nucleus promotes sleep. Elife 5. https://doi.org/10.7554/eLife.10382.
Nicolelis, M.A., Lin, R.C., Woodward, D.J., Chapin, J.K., 1993. Induction of immediate spatiotemporal changes in thalamic networks by peripheral block of ascending cutaneous information. Nature 361, 533–536. https://doi.org/10.1038/361533a0.
O'Connor, K.N., Allison, T.L., Rosenfield, M.E., Moore, J.W., 1997. Neural activity in the medial geniculate nucleus during auditory trace conditioning. Exp. Brain Res. 113, 534–556. https://doi.org/10.1007/pl00005605.
Odegaard, K.E., Bouaichi, C.G., Owanga, G., Vincis, R., 2025. Neural processing of Taste-Related signals in the mediodorsal thalamus of mice. e1500242025 J. Neurosci. 45. https://doi.org/10.1523/JNEUROSCI.1500-24.2025.
Ogawa, H., Nomura, T., 1988. Receptive field properties of thalamo-cortical taste relay neurons in the parvicellular part of the posteromedial ventral nucleus in rats. Exp. Brain Res 73, 364–370. https://doi.org/10.1007/BF00248229.
Oke, A.F., Adams, R.N., Winblad, B., von Knorring, L., 1988. Elevated dopamine/norepinephrine ratios in thalami of schizophrenic brains. Biol. Psychiatry 24, 79–82. https://doi.org/10.1016/0006-3223(88)90123-0.
Oram, T.B., Tenzer, A., Saraf-Sinik, I., Yizhar, O., Ahissar, E., 2024. Co-coding of head and whisker movements by both VPM and POm thalamic neurons. Nat. Commun. 15, 5883. https://doi.org/10.1038/s41467-024-50039-z.
Orsolini, L., Pompili, S., Volpe, U., 2022. Schizophrenia: a narrative review of etiopathogenetic, diagnostic and treatment aspects. J. Clin. Med. 11. https://doi.org/10.3390/jcm11175040.
Panula, P., Nuutinen, S., 2013. The histaminergic network in the brain: basic organization and role in disease. Nat. Rev. Neurosci. 14, 472–487. https://doi.org/10.1038/nrn3526.
Papadopoulos, G.C., Parnavelas, J.G., 1990. Distribution and synaptic organization of dopaminergic axons in the lateral geniculate nucleus of the rat. J. Comp. Neurol. 294, 356–361. https://doi.org/10.1002/cne.902940305.
Pardi, M.B., Vogenstahl, J., Dalmay, T., Spanò, T., Pu, D.-L., Naumann, L.B., Kretschmer, F., Sprekeler, H., Letzkus, J.J., 2020. A thalamocortical top-down circuit for associative memory. Science 370 (6518), 844–848. https://doi.org/10.1126/science.abc2399.
Peelman, K., Haider, B., 2024. Environmental context sculpts spatial and temporal visual processing in thalamus. BioRxiv. https://doi.org/10.1101/2024.07.26.605345.
Peräkylä, J., Sun, L., Lehtimäki, K., Peltola, J., Öhman, J., Möttönen, T., et al., 2017. Causal evidence from humans for the role of mediodorsal nucleus of the thalamus in working memory. J. Cogn. Neurosci. 29, 2090–2102. https://doi.org/10.1162/jocn_a_01176.
Perez-Rando, M., Elvira, U.K.A., García-Martí, G., Gadea, M., Aguilar, E.J., Escarti, M.J., et al., 2022. Alterations in the volume of thalamic nuclei in patients with schizophrenia and persistent auditory hallucinations. Neuroimage Clin. 35, 103070. https://doi.org/10.1016/j.nicl.2022.103070.
Pérez-Santos, I., Palomero-Gallagher, N., Zilles, K., Cavada, C., 2021. Distribution of the noradrenaline innervation and adrenoceptors in the macaque monkey thalamus. Cereb. Cortex 31, 4115–4139. https://doi.org/10.1093/cercor/bhab073.
Peruzzi, D., Bartlett, E., Smith, P.H., Oliver, D.L., 1997. A monosynaptic GABAergic input from the inferior colliculus to the medial geniculate body in rat. J. Neurosci. 17, 3766–3777. https://doi.org/10.1523/JNEUROSCI.17-10-03766.1997.
Petty, G.H., Bruno, R.M., 2024. Attentional modulation of secondary somatosensory and visual thalamus of mice. Elife 13. https://doi.org/10.7554/eLife.97188.3.
Petty, G.H., Kinnischtzke, A.K., Hong, Y.K., Bruno, R.M., 2021. Effects of arousal and movement on secondary somatosensory and visual thalamus. Elife 10. https://doi.org/10.7554/eLife.67611.
Pierret, T., Lavallée, P., Deschênes, M., 2000. Parallel streams for the relay of vibrissal information through thalamic barreloids. J. Neurosci. 20, 7455–7462. https://doi.org/10.1523/JNEUROSCI.20-19-07455.2000.
Pinault, D., 2004. The thalamic reticular nucleus: structure, function and concept. Brain Res. Rev. 46, 1–31. https://doi.org/10.1016/j.brainresrev.2004.04.008.
Pinto, D., Pagnamenta, A.T., Klei, L., Anney, R., Merico, D., Regan, R., et al., 2010. Functional impact of global rare copy number variation in autism spectrum disorders. Nature 466, 368–372. https://doi.org/10.1038/nature09146.
Qin, Y., Ahmadlou, M., Suhai, S., Neering, P., de Kraker, L., Heimel, J.A., et al., 2023. Thalamic regulation of ocular dominance plasticity in adult visual cortex. Elife 12. https://doi.org/10.7554/eLife.88124.3.
Rahmati, M., Curtis, C.E., Sreenivasan, K.K., 2023. Mnemonic representations in human lateral geniculate nucleus. Front. Behav. Neurosci. 17. https://doi.org/10.3389/fnbeh.2023.1094226.
Ramcharan, E.J., Gnadt, J.W., Sherman, S.M., 2005. Higher-order thalamic relays burst more than first-order relays. Proc. Natl. Acad. Sci. 102, 12236–12241. https://doi.org/10.1073/pnas.0502843102.
Ran, I., Miura, R.M., Puil, E., 2003. Spermine modulates neuronal excitability and NMDA receptors in juvenile gerbil auditory thalamus. Hear Res. 176, 65–79. https://doi.org/10.1016/S0378-5955(02)00746-3.
Rauschecker, J.P., Harris, L.R., 1983. Auditory compensation of the effects of visual deprivation in the cat's superior colliculus. Exp. Brain Res 50, 69–83. https://doi.org/10.1007/BF00238233.
Reggiani, J.D.S., Jiang, Q., Barbini, M., Lutas, A., Liang, L., Fernando, J., et al., 2023. Brainstem serotonin neurons selectively gate retinal information flow to thalamus. Neuron 111 (711-726), e11. https://doi.org/10.1016/j.neuron.2022.12.006.
Rico, B., Cavada, C., 1998. Adrenergic innervation of the monkey thalamus: an immunohistochemical study. Neuroscience 84, 839–847. https://doi.org/10.1016/S0306-4522(97)00549-6.
Rogawski, M.A., Aghajanian, G.K., 1980a. Activation of lateral geniculate neurons by norepinephrine: mediation by an α-adrenergic receptor. Brain Res. 182, 345–359. https://doi.org/10.1016/0006-8993(80)91193-2.
Rogawski, M.A., Aghajanian, G.K., 1980b. Modulation of lateral geniculate neurone excitability by noradrenaline microiontophoresis or locus coeruleus stimulation. Nature 287, 731–734. https://doi.org/10.1038/287731a0.
Roth, B.L., 2016. DREADDs for neuroscientists. Neuron 89, 683–694. https://doi.org/10.1016/j.neuron.2016.01.040.
Rouiller, E., de Ribaupierre, Y., de Ribaupierre, F., 1979. Phase-locked responses to low frequency tones in the medial geniculate body. Hear Res. 1, 213–226. https://doi.org/10.1016/0378-5955(79)90015-7.
Ryugo, D.K., Weinberger, N.M., 1978. Differential plasticity of morphologically distinct neuron populations in the medical geniculate body of the cat during classical conditioning. Behav. Biol. 22, 275–301. https://doi.org/10.1016/s0091-6773(78)92351-9.
Sampathkumar, V., Miller-Hansen, A., Sherman, S.M., Kasthuri, N., 2021. Integration of signals from different cortical areas in higher order thalamic neurons. Proc. Natl. Acad. Sci. 118. https://doi.org/10.1073/pnas.2104137118.
Sánchez-González, M.Á., García-Cabezas, M.Á., Rico, B., Cavada, C., 2005. The primate thalamus is a key target for brain dopamine. J. Neurosci. 25, 6076–6083. https://doi.org/10.1523/JNEUROSCI.0968-05.2005.
Sanchez-Vives, M.V., Bal, T., Kim, U., von Krosigk, M., McCormick, D.A., 1996. Are the interlaminar zones of the ferret dorsal lateral geniculate nucleus actually part of the perigeniculate nucleus. J. Neurosci. 16, 5923–5941. https://doi.org/10.1523/JNEUROSCI.16-19-05923.1996.
SanMiguel, I., Widmann, A., Bendixen, A., Trujillo-Barreto, N., Schröger, E., 2013. Hearing silences: human auditory processing relies on preactivation of sound-specific brain activity patterns. J. Neurosci. 33, 8633–8639. https://doi.org/10.1523/JNEUROSCI.5821-12.2013.
Scammell, T.E., Jackson, A.C., Franks, N.P., Wisden, W., Dauvilliers, Y., 2019. Histamine: neural circuits and new medications. Sleep 42. https://doi.org/10.1093/sleep/zsy183.
Schelinski, S., Kauffmann, L., Tabas, A., Müller-Axt, C., von Kriegstein, K., 2024. Functional alterations of the magnocellular subdivision of the visual sensory thalamus in autism. Proc. Natl. Acad. Sci. 121. https://doi.org/10.1073/pnas.2413409121.
Schizophrenia Working Group of the Psychiatric Genomics Consortium, 2014. Biological insights from 108 schizophrenia-associated genetic loci. Nature 511, 421–427. https://doi.org/10.1038/nature13595.
Schofield, B.R., Motts, S.D., Mellott, J.G., 2011. Cholinergic cells of the pontomesencephalic tegmentum: connections with auditory structures from cochlear nucleus to cortex. Hear Res 279, 85–95. https://doi.org/10.1016/j.heares.2010.12.019.
Seabrook, T.A., Krahe, T.E., Govindaiah, G., Guido, W., 2013. Interneurons in the mouse visual thalamus maintain a high degree of retinal convergence throughout postnatal development. Neural Dev. 8, 24. https://doi.org/10.1186/1749-8104-8-24.
Selemon, L.D., Begović, A., 2007. Stereologic analysis of the lateral geniculate nucleus of the thalamus in normal and schizophrenic subjects. Psychiatry Res 151, 1–10. https://doi.org/10.1016/j.psychres.2006.11.003.
Serra, C., Guida, L., Staartjes, V.E., Krayenbühl, N., Türe, U., 2019. Historical controversies about the thalamus: from etymology to function. Neurosurg. Focus 47, E13. https://doi.org/10.3171/2019.6.FOCUS19331.
Sherman, S.M., 2016. Thalamus plays a central role in ongoing cortical functioning. Nat. Neurosci. 19, 533–541. https://doi.org/10.1038/nn.4269.
Sherman, S.M., Guillery, R.W., 1996. Functional organization of thalamocortical relays. J. Neurophysiol. 76, 1367–1395. https://doi.org/10.1152/jn.1996.76.3.1367.
Shine, J.M., Lewis, L.D., Garrett, D.D., Hwang, K., 2023. The impact of the human thalamus on brain-wide information processing. Nat. Rev. Neurosci. 24, 416–430. https://doi.org/10.1038/s41583-023-00701-0.
Silbersweig, D.A., Stern, E., Frith, C., Cahill, C., Holmes, A., Grootoonk, S., et al., 1995. A functional neuroanatomy of hallucinations in schizophrenia. Nature 378, 176–179. https://doi.org/10.1038/378176a0.
Simko, J., Markram, H., 2021a. Morphology, physiology and synaptic connectivity of local interneurons in the mouse somatosensory thalamus. J. Physiol. 599, 5085–5101. https://doi.org/10.1113/JP281711.
Simko, J., Markram, H., 2021b. Morphology, physiology and synaptic connectivity of local interneurons in the mouse somatosensory thalamus. J. Physiol. 599, 5085–5101. https://doi.org/10.1113/JP281711.

Simpson, K.L., Altman, D.W., Wang, L., Kirifides, M.L., Lin, R.C., Waterhouse, B.D., 1997. Lateralization and functional organization of the locus coeruleus projection to the trigeminal somatosensory pathway in rat. J. Comp. Neurol. 385, 135–147.
Sinclair-Wilson, A., Lawrence, A., Ferezou, I., Cartonnet, H., Mailhes, C., Garel, S., et al., 2023. Plasticity of thalamocortical axons is regulated by serotonin levels modulated by preterm birth. Proc. Natl. Acad. Sci. 120. https://doi.org/10.1073/pnas.2301644120.
Sinha, U.K., Hollen, K.M., Rodriguez, R., Miller, C.A., 1993. Auditory system degeneration in Alzheimer's disease, 779–779 Neurology 43. https://doi.org/10.1212/WNL.43.4.779.
Sommeijer, J.-P., Ahmadlou, M., Saiepour, M.H., Seignette, K., Min, R., Heimel, J.A., et al., 2017. Thalamic inhibition regulates critical-period plasticity in visual cortex and thalamus. Nat. Neurosci. 20, 1715–1721. https://doi.org/10.1038/s41593-017-0002-3.
Spreafico, R., Frassoni, C., Arcelli, P., De Biasi, S., 1994. GABAergic interneurons in the somatosensory thalamus of the Guinea-pig: a light and ultrastructural immunocytochemical investigation. Neuroscience 59, 961–973. https://doi.org/10.1016/0306-4522(94)90299-2.
Steullet, P., Cabungcal, J.-H., Bukhari, S.A., Ardelt, M.I., Pantazopoulos, H., Hamati, F., et al., 2018. The thalamic reticular nucleus in schizophrenia and bipolar disorder: role of parvalbumin-expressing neuron networks and oxidative stress. Mol. Psychiatry 23, 2057–2065. https://doi.org/10.1038/mp.2017.230.
Sumser, A., Isaías-Camacho, E.U., Mease, R.A., Groh, A., 2025. Active and passive touch are differentially represented in the mouse somatosensory thalamus. PLoS Biol. 23, e3003108. https://doi.org/10.1371/journal.pbio.3003108.
Sun, Y.-G., Pita-Almenar, J.D., Wu, C.-S., Renger, J.J., Uebele, V.N., Lu, H.-C., et al., 2013. Biphasic cholinergic synaptic transmission controls action potential activity in thalamic reticular nucleus neurons. J. Neurosci. 33, 2048–2059. https://doi.org/10.1523/JNEUROSCI.3177-12.2013.
Supple, W.F., Kapp, B.S., 1989. Response characteristics of neurons in the medial component of the medial geniculate nucleus during pavlovian differential fear conditioning in rabbits. Behav. Neurosci. 103, 1276–1286. https://doi.org/10.1037/0735-7044.103.6.1276.
Tabas, A., Mihai, G., Kiebel, S., Trampel, R., von Kriegstein, K., 2020. Abstract rules drive adaptation in the subcortical sensory pathway. Elife 9. https://doi.org/10.7554/eLife.64501.
Takeuchi, Y., Yamasaki, M., Nagumo, Y., Imoto, K., Watanabe, M., Miyata, M., 2012. Rewiring of afferent fibers in the somatosensory thalamus of mice caused by peripheral sensory nerve transection. J. Neurosci. 32, 6917–6930. https://doi.org/10.1523/JNEUROSCI.5008-11.2012.
Talvik, M., Nordström, A.-L., Olsson, H., Halldin, C., Farde, L., 2003. Decreased thalamic D2/D3 receptor binding in drug-naive patients with schizophrenia: a PET study with [11C]FLB 457. S1461145703003699 Int. J. Neuropsychopharmacol. 6. https://doi.org/10.1017/S1461145703003699.
Tamura, R., Kitamura, H., Endo, T., Hasegawa, N., Someya, T., 2010. Reduced thalamic volume observed across different subgroups of autism spectrum disorders. Psychiatry Res Neuroimaging 184, 186–188. https://doi.org/10.1016/j.pscychresns.2010.07.001.
Tao, L., Jiang, R., Zhang, K., Qian, Z., Chen, P., Lv, Y., et al., 2020. Light therapy in non-seasonal depression: an update meta-analysis. Psychiatry Res 291, 113247. https://doi.org/10.1016/j.psychres.2020.113247.
Taylor, J.A., Hasegawa, M., Benoit, C.M., Freire, J.A., Theodore, M., Ganea, D.A., et al., 2021. Single cell plasticity and population coding stability in auditory thalamus upon associative learning. Nat. Commun. 12, 2438. https://doi.org/10.1038/s41467-021-22421-8.
Tsatsanis, K.D., Rourke, B.P., Klin, A., Volkmar, F.R., Cicchetti, D., Schultz, R.T., 2003. Reduced thalamic volume in high-functioning individuals with autism. Biol. Psychiatry 53, 121–129. https://doi.org/10.1016/S0006-3223(02)01530-5.
Uhlrich, D.J., Manning, K.A., Pienkowski, T.P., 1993. The histaminergic innervation of the lateral geniculate complex in the cat. Vis. Neurosci. 10, 225–235. https://doi.org/10.1017/S0952523800003631.
van de Mortel, L.A., Thomas, R.M., van Wingen, G.A., 2021. Grey matter loss at different stages of cognitive decline: a role for the thalamus in developing Alzheimer's disease. J. Alzheimer'S. Dis. 83, 705–720. https://doi.org/10.3233/JAD-210173.
Varela, C., 2014. Thalamic neuromodulation and its implications for executive networks. Front Neural Circuits 8. https://doi.org/10.3389/fncir.2014.00069.
Varela, C., Sherman, S.M., 2009. Differences in response to serotonergic activation between first and higher order thalamic nuclei. Cereb. Cortex 19, 1776–1786. https://doi.org/10.1093/cercor/bhn208.
Vázquez, Y., Salinas, E., Romo, R., 2013. Transformation of the neural code for tactile detection from thalamus to cortex. Proc. Natl. Acad. Sci. 110. https://doi.org/10.1073/pnas.1309728110.
Vega-Zuniga, T., Sumser, A., Symonova, O., Koppensteiner, P., Schmidt, F.H., Joesch, M., 2025. A thalamic hub-and-spoke network enables visual perception during action by coordinating visuomotor dynamics. Nat. Neurosci. 28, 627–639. https://doi.org/10.1038/s41593-025-01874-w.
Vertes, R.P., 1991. A PHA-L analysis of ascending projections of the dorsal raphe nucleus in the rat. J. Comp. Neurol. 313, 643–668. https://doi.org/10.1002/cne.903130409.
Vertes, R.P., Fortin, W.J., Crane, A.M., 1999. Projections of the median raphe nucleus in the rat. J. Comp. Neurol. 407, 555–582.
von Melchner, L., Pallas, S.L., Sur, M., 2000. Visual behaviour mediated by retinal projections directed to the auditory pathway. Nature 404, 871–876. https://doi.org/10.1038/35009102.
Wang, Q.P., Nakai, Y., 1994. The dorsal raphe: an important nucleus in pain modulation. Brain Res. Bull. 34, 575–585. https://doi.org/10.1016/0361-9230(94)90143-0.
Wang, Y., You, L., Tan, K., Li, M., Zou, J., Zhao, Z., et al., 2023. A common thalamic hub for general and defensive arousal control. e8 Neuron 111, 3270–3287. https://doi.org/10.1016/j.neuron.2023.07.007.
Wepsic, J.G., 1966. Multimodal sensory activation of cells in the magnocellular medial geniculate nucleus. Exp. Neurol. 15, 299–318. https://doi.org/10.1016/0014-4886(66)90053-7.
Whyland, K.L., Slusarczyk, A.S., Bickford, M.E., 2020. GABAergic cell types in the superficial layers of the mouse superior colliculus. J. Comp. Neurol. 528, 308–320. https://doi.org/10.1002/cne.24754.
Williamson, R.S., Hancock, K.E., Shinn-Cunningham, B.G., Polley, D.B., 2015. Locomotion and task demands differentially modulate thalamic audiovisual processing during active search. Curr. Biol. 25, 1885–1891. https://doi.org/10.1016/j.cub.2015.05.045.
Wimmer, V.C., Bruno, R.M., de Kock, C.P.J., Kuner, T., Sakmann, B., 2010. Dimensions of a projection column and architecture of VPM and POm axons in rat vibrissal cortex. Cereb. Cortex 20, 2265–2276. https://doi.org/10.1093/cercor/bhq068.
Wimmer, R.D., Schmitt, L.I., Davidson, T.J., Nakajima, M., Deisseroth, K., Halassa, M.M., 2015. Thalamic control of sensory selection in divided attention. Nature 526, 705–709. https://doi.org/10.1038/nature15398.
Winer, J.A., Saint Marie, R.L., Larue, D.T., Oliver, D.L., 1996a. GABAergic feedforward projections from the inferior colliculus to the medial geniculate body. Proc. Natl. Acad. Sci. USA 93, 8005–8010. https://doi.org/10.1073/pnas.93.15.8005.
Winer, J.A., Saint Marie, R.L., Larue, D.T., Oliver, D.L., 1996b. GABAergic feedforward projections from the inferior colliculus to the medial geniculate body. Proc. Natl. Acad. Sci. USA 93, 8005–8010. https://doi.org/10.1073/pnas.93.15.8005.
Wolff, M., Morceau, S., Folkard, R., Martin-Cortecero, J., Groh, A., 2021. A thalamic bridge from sensory perception to cognition. Neurosci. Biobehav Rev. 120, 222–235. https://doi.org/10.1016/j.neubiorev.2020.11.013.
Woodward, N.D., Giraldo-Chica, M., Rogers, B., Cascio, C.J., 2017. Thalamocortical dysconnectivity in autism spectrum disorder: an analysis of the autism brain imaging data exchange. Biol. Psychiatry Cogn. Neurosci. Neuroimaging 2, 76–84. https://doi.org/10.1016/j.bpsc.2016.09.002.
World Health Organization, 2024. Clinical descriptions and diagnostic requirements for ICD-11 mental. behavioural and Neurodevelopmental Disorders. World Health Organization.
Xi, K., Xiao, H., Huang, X., Yuan, Z., Liu, M., Mao, H., et al., 2023. Reversal of hyperactive higher-order thalamus attenuates defensiveness in a mouse model of PTSD. Sci. Adv. 9. https://doi.org/10.1126/sciadv.ade5987.
Xue, J.T., Kim, C.B., Moore, R.J., Spear, P.D., 1994. Influence of the superior colliculus on responses of lateral geniculate neurons in the cat. Vis. Neurosci. 11, 1059–1076. https://doi.org/10.1017/s095252380000688x.
Yasuno, F., Suhara, T., Okubo, Y., Sudo, Y., Inoue, M., Ichimiya, T., et al., 2004. Low dopamine D2 receptor binding in subregions of the thalamus in schizophrenia. Am. J. Psychiatry 161, 1016–1022. https://doi.org/10.1176/appi.ajp.161.6.1016.
Yoshikawa, T., Nakamura, T., Yanai, K., 2021. Histaminergic neurons in the tuberomammillary nucleus as a control centre for wakefulness. Br. J. Pharm. 178, 750–769. https://doi.org/10.1111/bph.15220.
Zandvakili, A., Kohn, A., 2015. Coordinated neuronal activity enhances corticocortical communication. Neuron 87, 827–839. https://doi.org/10.1016/j.neuron.2015.07.026.
Zhang, X., Davidson, S., Giesler Jr, G.J., 2006. Thermally identified subgroups of marginal zone neurons project to distinct regions of the ventral posterior lateral nucleus in rats. J. Neurosci. 26, 5215–5223. https://doi.org/10.1523/JNEUROSCI.0701-06.2006.
Zhao, Y., Kerscher, N., Eysel, U., Funke, K., 2001. Changes of contrast gain in cat dorsal lateral geniculate nucleus by dopamine receptor agonists. Neuroreport 12, 2939–2945. https://doi.org/10.1097/00001756-200109170-00037.
Zhao, Y., Kerscher, N., Eysel, U., Funke, K., 2002. D1 and D2 receptor-mediated dopaminergic modulation of visual responses in cat dorsal lateral geniculate nucleus. J. Physiol. 539, 223–238. https://doi.org/10.1113/jphysiol.2001.012721.
Zhuo, Y., Luo, B., Yi, X., Dong, H., Miao, X., Wan, J., et al., 2024. Improved Green and red GRAB sensors for monitoring dopaminergic activity in vivo. Nat. Methods 21, 680–691. https://doi.org/10.1038/s41592-023-02100-w.
Zikopoulos, B., Barbas, H., 2012. Pathways for emotions and attention converge on the thalamic reticular nucleus in primates. J. Neurosci. 32, 5338–5350. https://doi.org/10.1523/JNEUROSCI.4793-11.2012.