

Interneurons and triadic circuitry of the thalamus

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The thalamus is strategically placed to control the flow of information to cortex and thus conscious perception. A key player in this control is a local GABAergic interneuron that inhibits relay cells. This interneuron is especially interesting because, in addition to a conventional axonal output, most of its output is via distal dendrites. The latter seem to be electrotonically and thus functionally isolated from the soma and axon, and they enter into complex synaptic arrangements. It is proposed that, because of special synaptic properties of its dendritic outputs, this local GABAergic interneuron of the thalamus provides gain control for the relay cell and thereby keeps relay of information to cortex within a fairly linear regime.

Because all information reaching cortex, and thus consciousness, is relayed dynamically by the thalamus [1], it is clearly important to understand thalamic relay functions. A major player in this is the local GABAergic interneuron, which provides strong inhibitory input to thalamic relay cells and thereby helps control the flow of information to cortex. In this review, 'interneuron' refers specifically to such GABAergic cells found within thalamic relay nuclei,

not to GABAergic neurons of the nearby thalamic reticular nucleus, which also inhibit relay cells. Throughout the thalamus, with occasional variation depending on relay nucleus or species, the local GABAergic interneurons comprise ~20–25% of the cells present, the remainder being relay cells [1–3]. Similar to relay cells, there is more than one type of local GABAergic interneuron [4,5], but this account focuses on that found in the lateral geniculate nucleus of the cat. This interneuron is particularly interesting because of its synaptic outputs, which are both axonal and dendritic, and the type of synaptic circuits entered into by the dendritic outputs. It is emphasized here partly because it has been intensively studied but mainly because it is widespread in the mammalian thalamus, meaning that the following description is likely to apply generally to thalamus.

Outputs of the interneuron

Figure 1 shows the local GABAergic interneuron in question plus the two relay cell types, X and Y, that it innervates [6,7]. The interneuron has both a conventional synaptic output via an axon, which arborizes within the dendritic arbor of the parent interneuron, and clusters of

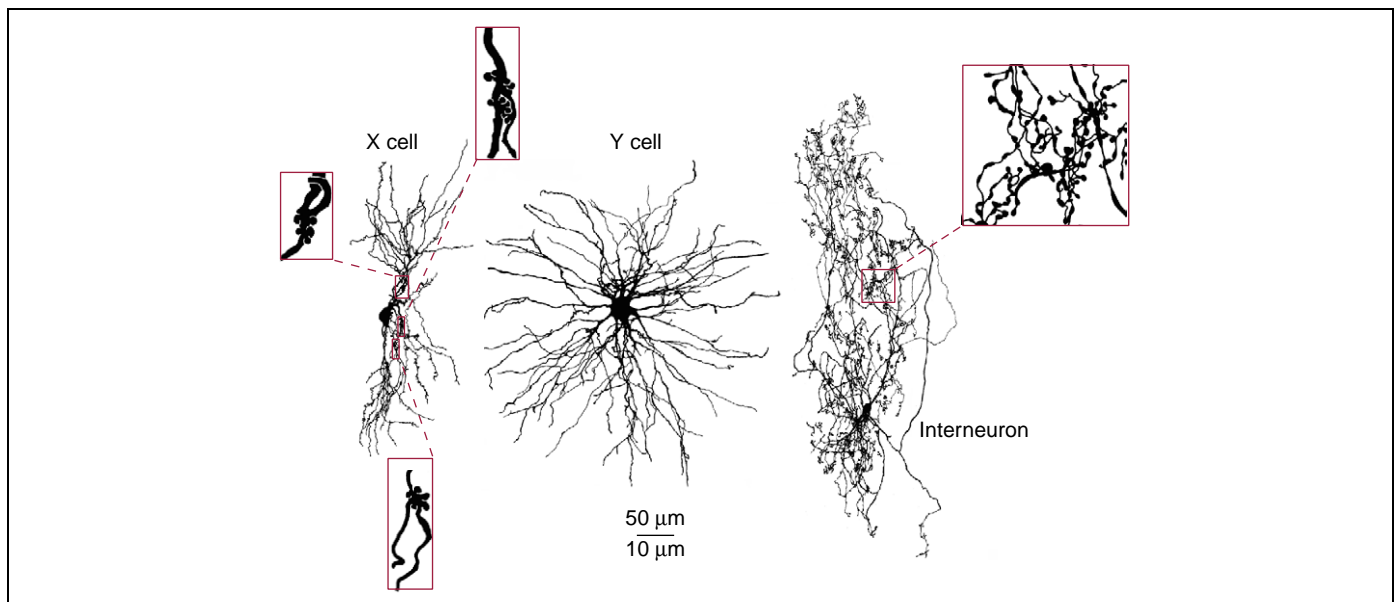


Figure 1. Representative cells of the cat lateral geniculate nucleus. Cells were labeled intracellularly with horseradish peroxidase [6,7]; shown are relay X and Y cells and an interneuron. Insets for the X cell show appendages near proximal branch points, and that for the interneuron shows the numerous swellings that are the presynaptic terminals. Scale bar: 50 μm for main figure; 10 μm for insets.

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presynaptic terminals that emanate from distal dendrites [8–12]. Both outputs are inhibitory, but there are differences: the axonal terminal, called F1 ('F' for flattened vesicle), forms a simple contact onto dendrites of both X and Y cells, although it is unclear whether a given axon innervates both types; the dendritic terminal, called F2, is both postsynaptic to various terminals and presynaptic to X cells (but rarely to Y cells). F2 outputs outnumber F1 outputs; other differences will be mentioned later. There is evidence that some of these interneurons have no axon, and thus have their only output via dendrites [13,14].

The Y cell, which receives predominantly F1 (axonal) inputs from the interneuron onto proximal dendrites, has a fairly simple, radiate dendritic arbor with few appendages. The X cell has a bipolar dendritic tree, oriented perpendicular to laminar borders, with clusters of grape-like appendages near primary branch points (Figure 1). These are not spines, lacking the apparatus found in true spines in cortex and hippocampus, but are simple appendages that represent a local bulging of the dendrite. They mark the postsynaptic targets of both F2 (dendritic) interneuron terminals and retinal terminals. F1 terminals can contact these appendages but also frequently contact nearby dendritic shafts; in addition, they contact Y cells on proximal dendrites.

Triads and glomeruli

F2 terminals commonly enter into complex synaptic arrangements known as triads (Figure 2). In the more common form, a single retinal terminal contacts both an

F2 terminal and a relay X-cell appendage, and the F2 terminal contacts the same appendage; thus, three synapses are involved. In other thalamic relays, the main input (e.g. medial lemniscal input to the ventral posterior nucleus or inferior collicular input to the medial geniculate nucleus) replaces the retinal input in this circuit. Less commonly, an F2 terminal is postsynaptic to a cholinergic terminal from the brainstem parabrachial region, and the same parabrachial axon (but not the same terminal) contacts the same relay cell (Figure 2). Because three synapses are involved, this forms another sort of triadic arrangement. Although not illustrated, occasionally F1 terminals are found presynaptic to F2 terminals [8–12].

This intricate circuitry is found within a glomerulus, a complex synaptic zone [15] (Figure 2). Individual synapses here are not juxtaposed to glial processes, but instead the entire glomerulus is enclosed in a glial sheath. The function of this arrangement is not known but, because glial processes have been implicated in the uptake and regulation of neurotransmitters and other neuroactive substances [16,17], their lack within a glomerulus might affect the extent to which neurotransmitters and other substances remain active and spill over to affect more distant receptors.

Every glomerulus contains at least one retinal terminal and one F2 terminal in a triad. F1 and parabrachial terminals are also commonly present in glomeruli, but terminals from cortical layer 6 are not [18] (although see Ref. [19]). The other main GABAergic innervation of relay cells, besides that from the interneurons, arises from the nearby thalamic reticular nucleus. However, reticular

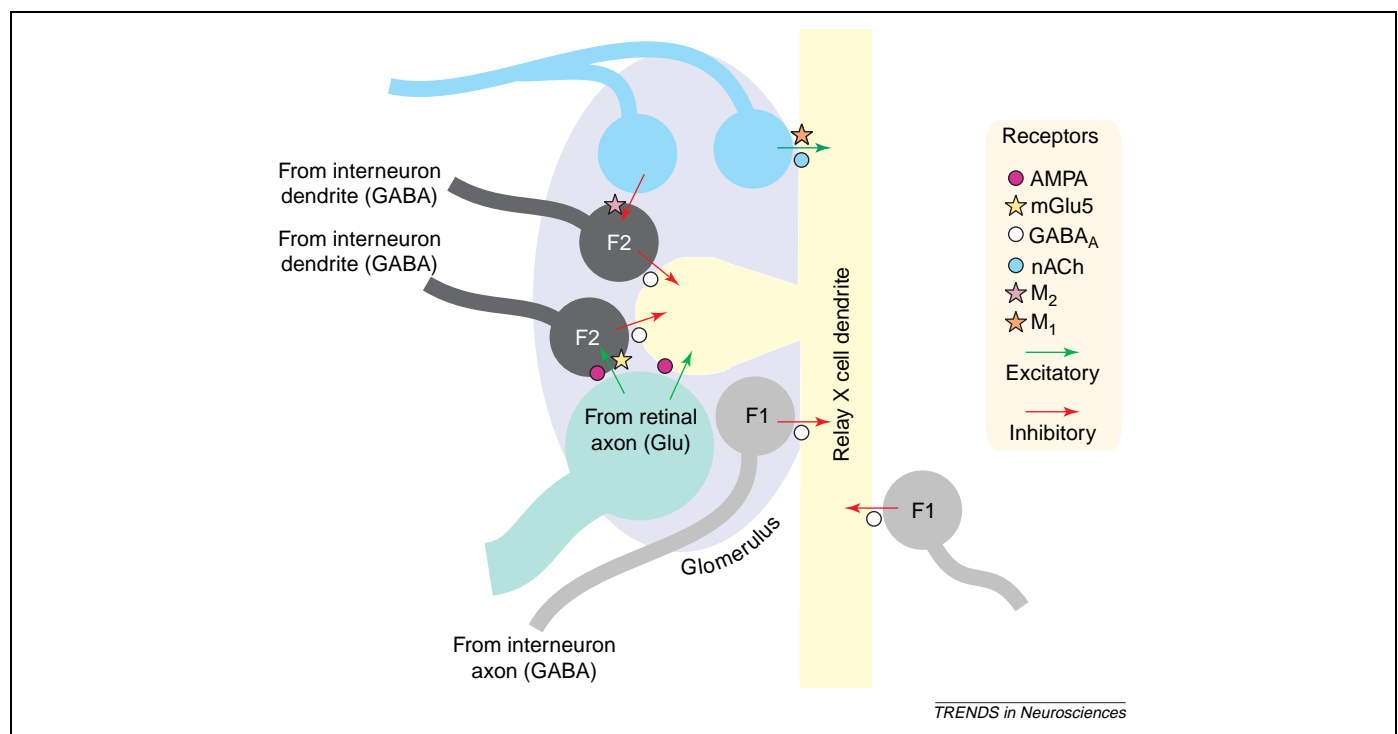


Figure 2. A triad and glomerulus in cat lateral geniculate nucleus. Shown are the various inhibitory and excitatory synaptic contacts (arrows) and related postsynaptic receptors. Here, the lower F2 terminal is part of a conventional triad, involving three synapses (from the retinal terminal to the F2 terminal, from the retinal terminal to an appendage of the X cell dendrite, and from the F2 terminal to the same appendage). The upper F2 terminal forms part of another type of triad, also involving three synapses (from one parabrachial terminal to the F2 terminal, from the F2 terminal to the relay X cell dendrite, and from another parabrachial terminal to the same dendrite). For simplicity, the NMDA receptor on the relay cell postsynaptic to the retinal input has been omitted. Abbreviations: F1 and F2, 'flattened vesicle' synaptic terminals; GABA_A receptor, type A receptor for GABA; Glu; glutamate; M₁ and M₂, muscarinic ACh receptors; mGlu5, type 5 metabotropic glutamate receptor; nACh receptor, nicotinic ACh receptor.

terminals mostly contact distal dendrites of relay cells and rarely are found in glomeruli [20,21]. Although direct data are lacking, a process of elimination indicates that most F1 terminals onto proximal dendrites, including those in glomeruli, emanate from interneuron axons.

Cellular properties of the interneuron

Given the axonal and dendritic interneuron output routes, obvious questions concern how these are controlled and how they might relate to one another. Modeling of cable properties suggests, among other things, how current flows through the dendrites and thus how synaptic inputs at one site affect membrane voltage at others, including the axon hillock or spike-generating region. For practical purposes, the soma and axon hillock can be considered as isopotential, so the problem reduces to a consideration of how synapses at various sites affect the soma.

Cable modeling indicates a striking difference between interneurons and relay cells [22,23]. Relay cells are electrotonically compact, suggesting that even postsynaptic potentials (PSPs) generated at distal dendritic locations are attenuated by half or less at the soma. By contrast, interneurons are electrotonically extensive, so that PSPs generated distally are attenuated by >90% at the soma. The F2 terminals themselves are usually appended to the distal sites by long ($\geq 10 \mu\text{m}$), thin ($\sim 0.1 \mu\text{m}$ diameter) processes [8], which implies that inputs to these F2 terminals are even more isolated from the soma.

Cable modeling is limited for several reasons [1,24]. For example, many of the parameters on which it is based (e.g. membrane capacitance) must be assumed; it assumes that the membrane is passive, whereas interneurons, like all neurons, possess many dynamically gated ion channels [1] that will affect cable properties. It nonetheless provides a useful, if limited, approach to understanding how inputs affect the postsynaptic cell. The picture that emerges for the interneuron (Figure 3) leads to two interesting conclusions. First, because of the attenuation of PSPs from distal inputs, the main control of the axonal (F1) output rests with relatively proximal inputs. Second, although PSPs onto the dendritic (F2) terminals will affect their release of GABA, these PSPs will have little or no influence on the axonal output; also, local clusters of F2 terminals will be functionally isolated from others. There is evidence to support these conclusions [25]. The interneuron thus seems to be multiplex, with one input–output route that involves proximal synaptic inputs that conventionally control the axonal output with an independent route onto the F2 terminals.

If this cable modeling is correct, several important implications follow. One is that recordings from the interneuron, which have all been from the soma, reveal synaptic processing involving the axonal output but tell us nothing about that involving dendritic output. Likewise, recorded spike activity relates to the axonal, but not to the dendritic, output. Receptive fields of all interneurons recorded *in vivo* from the cat lateral geniculate nucleus are those of X cells [6], indicating that retinal X cells send inputs to proximal dendrites. Other evidence also suggests that retinal inputs to the dendritic terminals are from X cells [9,26] (but see Ref. [27]).

Control of F2 terminals and triad function

It is important to understand how dendritic F2 output from the interneuron is controlled and how the triad functions. The postsynaptic receptors on the F2 terminal have been identified *in vitro* using pharmacological tools and immunocytochemical electron microscopy (Figure 2). Type 5 metabotropic glutamate (mGlu5) receptors dominate postsynaptic to the retinal input, probably along with ionotropic (AMPA and perhaps NMDA) glutamate receptors [25,28]. Activation of either the metabotropic or the ionotropic receptors increases GABA release from F2 terminal [25]. Type 2 muscarinic ACh receptors (M_2 receptors), but not ionotropic nicotinic ACh (nACh) receptors, are also found postsynaptic to the parabrachial input [29,30]; activation of the M_2 receptors decreases GABA release [25]. Note that the dominant receptors on the F2 terminal, mGluR5 and the M_2 receptor, are both metabotropic. Retinal inputs onto the relay cell activate AMPA receptors and NMDA receptors only, and parabrachial inputs activate both nACh receptors and type 1 muscarinic (M_1) receptors; activation of these various receptors on the relay cell produces excitatory PSPs (EPSPs) [1,31].

Although there is no direct evidence for the functional properties of the mGlu5 and M_2 receptors on the F2 terminal, plausible assumptions can be made, based on properties of these and other metabotropic receptors elsewhere [32–37]:

- (i) mGlu5 and M_2 receptors operate by opening or closing a 'leak' K^+ channel. mGlu5 receptor activation closes the channel, depolarizing the terminal and increasing GABA release, whereas M_2 receptor activation opens the channel, hyperpolarizing the terminal and decreasing GABA release.
- (ii) Compared with ionotropic equivalents (e.g. AMPA and nACh receptors), higher rates of firing in the afferent input are required to activate the mGlu5 and M_2 receptors.
- (iii) Although ionotropic receptor activation produces PSPs with a brief latency (~ 1 ms) and duration (≥ 10 ms), mGlu5 and M_2 receptor activation is much slower (~ 10 ms latency; > 100 s of ms duration).

Given these assumptions, plus evidence that release of GABA from the F2 terminal has a background rate that retinal or parabrachial afferents can upregulate or downregulate [25,38], the following can be suggested (Figure 2).

Activation of the parabrachial inputs

Activity of parabrachial inputs corresponds to behavioral state: when the animal is in slow wave sleep, these brainstem inputs are silent; when the animal is drowsy, they are moderately active; and when the animal is fully aroused and alert, they are highly active [39,40]. It follows that, during sleep, the direct excitatory ACh input to the relay cell is absent, and this is exacerbated by the absence of inhibition of F2 terminal output. Together this depresses the relay. During drowsiness, the moderate level of activity among parabrachial afferents would activate nACh receptors on the relay cell, producing some excitation, and the activity could be sufficient to enhance the relay further by activating the M_1 receptors on the

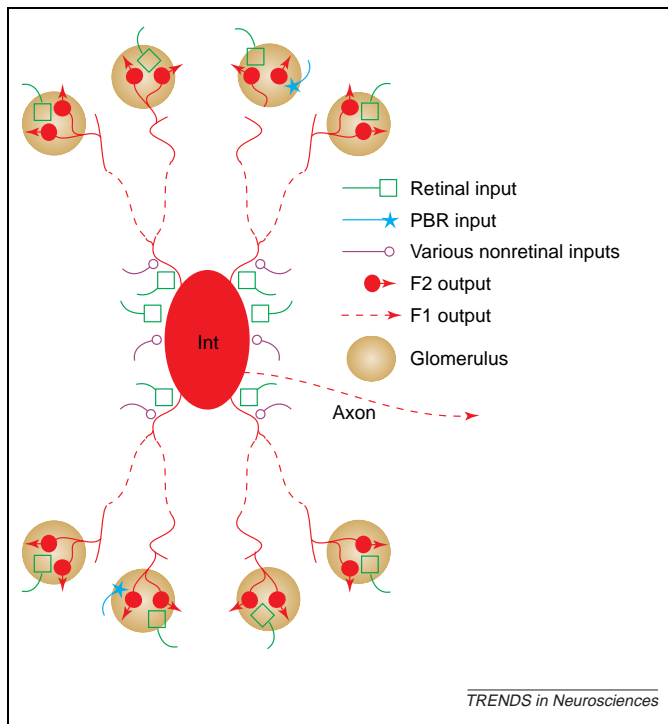


Figure 3. Model for functioning of the interneuron (Int). The axon outputs (F1) are controlled by inputs onto the cell body and proximal dendrites. These interneurons receive many synaptic inputs onto their cell bodies, although relay cells rarely have synapses there [8]. The F1 inputs are mostly from other interneurons, although some can be from the thalamic reticular nucleus. The peripheral dendritic outputs (F2) are controlled locally by direct inputs, mostly from either the retina or parabrachial region (PBR), and these F2 circuits are within glomeruli. Dashed lines connecting proximal dendrites to the F2 circuits represent 5–10 levels of dendritic branching.

relay cell (producing prolonged depolarization) and the M_2 receptors on the F2 terminal (producing prolonged disinhibition). The relay would be more enhanced as the animal became more awake and aroused, because the higher activity among parabrachial afferents would produce more direct nACh and M_1 receptor excitation of the relay cell and more disinhibition via M_2 receptor inhibition of the F2 terminal. Thus, the correlated levels of arousal and activity among parabrachial afferents also correlate with facilitation of the relay, and the circuitry involving F2 terminals plays a significant role in this facilitation.

Activation of the retinal inputs

The predicted result is more complicated in the case of retinal inputs because the triadic circuit provides a basis for direct excitation (retinal cell to relay cell) and indirect inhibition (retinal cell to F2-terminal to relay cell). Low levels of retinal firing activate only ionotropic receptors, generating EPSPs in the relay cell; if there were sufficient AMPA receptors on the F2 terminal, the EPSPs would be opposed and curtailed by disynaptic inhibitory PSPs. Unless there is a major difference between sensitivity of the AMPA receptors on the relay cell versus the F2 terminal, the only role of those on the F2 terminal at any retinal firing level would be to oppose the EPSP in the relay cell in a fairly linear fashion, and this would not change appreciably with firing level in the retinal afferent. However, as retinal firing increases to activate mGlu5

receptors on the F2 terminal, an extra dose of GABA-mediated inhibition appears in the relay cell. This mGlu5-receptor-based inhibition appears and grows only after retinal firing exceeds a threshold but, once activated, it outlasts the retinal activity by several hundreds of milliseconds or even a few seconds, because this is the duration of EPSPs activated by metabotropic glutamate receptors.

Contrast gain control

The firing level of the retinal axon is more-or-less monotonically related to contrast in the visual stimulus. As contrast increases to a certain level, the retinal afferent fires sufficiently to activate the mGlu5 receptors on the F2 terminal, and this increases inhibition in the relay cell. This will reduce the responsiveness, or gain, of the relay cell to retinal inputs and, because of the temporal properties of mGlu5 receptors, this reduced contrast gain will last for a second or so even after the retinal afferent firing returns to normal or prior levels. By this process, the contrast gain of the relay cell adjusts to overall contrast: as contrast increases enough to activate the triad circuit via mGlu5 receptors, gain in the relay cell reduces, and vice versa.

Contrast gain control is an important property of the visual system that, like other forms of adaptation (e.g. to brightness or motion), helps to adjust the sensitivity of visual neurons to ambient levels of stimulation. Such adaptation to contrast is seen at all levels, from the retina to the lateral geniculate nucleus to the cortex [41,42]. However, little attention has been devoted to contrast gain control or adaptation at thalamic levels, especially with regard to the sort of differences between X and Y cells suggested in the context of triadic properties. This hypothesis for triadic function implies contrast effects in the geniculate X cell but not the Y cell, because the latter has few if any triads. Data to test this prediction are generally lacking.

Effects on voltage-sensitive properties

Because activation of the triadic circuit via activation of mGlu5 receptors has a long-lasting effect on membrane potential, it can affect the 'play' of the voltage-sensitive properties of a cell. There are many voltage-gated conductances that can be so affected [1,43]. For one example, consider voltage-gated T-type Ca^{2+} channels: when activated, they lead to an inward current, I_T , and to an all-or-none, low-threshold Ca^{2+} spike that propagates through the dendritic tree and produces a burst of action potentials. When the cell is relatively depolarized, I_T is inactivated, no low threshold spike occurs, and the cell responds to suprathreshold inputs with a stream of unitary action potentials: this is 'tonic firing'. When the cell is relatively hyperpolarized, inactivation of I_T is removed, and now an excitatory input produces a low-threshold spike and 'burst firing'. The firing mode of the relay cell has important implications for functioning of the relay [44]. The point here is that triadic function can affect I_T and other such voltage-sensitive properties in interesting ways. This clearly needs more study.

Concluding remarks

Because the GABAergic interneuron described here, along with its F2 terminals and participation in triadic circuitry, is widespread throughout mammalian thalamus [1,3], the hypothesis specified here for the cat lateral geniculate nucleus can plausibly be extended to mammalian thalamus more generally. This interneuron has several interesting features that stimulate several speculative hypotheses about its function. One is that its dendritic outputs function independently of its conventional axonal output, with each dendritic output or small cluster under local control of its specific inputs. This allows each interneuron to multiplex and provides many independent computational routes to affect relay cells, thereby influencing how information is transmitted to cortex. This input–output mechanism of local dendritic processing closely resembles that of many retinal amacrine cells [45–47]. Another hypothesis involves the triadic circuits entered into by the dendritic outputs, and the predominantly metabotropic receptors found on these outputs. This suggests that the level of activity of inputs to be relayed to cortex act through the triadic circuitry to have a relatively long-lasting influence on membrane voltage of the relay cell. This can serve multiple purposes, including controlling overall responsiveness of the relay, regulating voltage-gated conductances in the relay cell, and providing a gain control mechanism for the relay. The gain control ensures that sensitivity of the relay cell is centered within the dynamic range of its main input, thereby promoting a more linear relay, and this could function in the same way for any thalamic relay having triadic circuitry. These suggested functions for the interneuron make it anything but humble, and the obvious next step is to apply rigorous experimental tests of these hypotheses.

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