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Inhibitory circuits for visual processing in thalamus

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Synapses made by local interneurons dominate the intrinsic circuitry of the mammalian visual thalamus and influence all signals traveling from the eye to cortex. Here we draw on physiological and computational analyses of receptive fields in the cat's lateral geniculate nucleus to describe how inhibition helps to enhance selectivity for stimulus features in space and time and to improve the efficiency of the neural code. Further, we explore specialized synaptic attributes of relay cells and interneurons and discuss how these might be adapted to preserve the temporal precision of retinal spike trains and thereby maximize the rate of information transmitted downstream.

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Current Opinion in Neurobiology 2011, 21:726–733

This review comes from a themed issue on
Networks, Circuits and Computation
Edited by Peter Dayan, Dan Feldman, Marla Feller

Available online 13th July 2011

0959-4388/\$ – see front matter

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DOI 10.1016/j.conb.2011.06.004

Introduction

Spikes traveling from retina to the thalamus excite both relay cells, which project to cortex, and local inhibitory interneurons at the same time. Even though relay cells in the lateral geniculate nucleus outnumber interneurons three to one [1], they are only sparsely interconnected [2]. By contrast, interneurons densely innervate relay cells and each other [3–7]. Further, relay cells send collaterals to the overlying thalamic reticular nucleus, a sheet of inhibitory cells that project to the geniculate in return [2,8–10]. Hence all retinal information transmitted to cortex is influenced by a feedforward and a feedback inhibitory pathway [11]. Here we focus on the role of thalamic inhibition in different aspects of visual processing as well as the structure and synaptic physiology of local inhibitory circuits. For accounts of thalamic inhibition in sleep see [12] and in development [13,14].

Spatial and temporal arrangement of excitation and inhibition within the receptive field: push–pull

The receptive fields of relay cells and interneurons have the same qualitative shape, comprising two concentrically arranged subregions (a center and a surround) that have the opposite preference for luminance contrast, On or Off [15,16]. Further, there is a push–pull relationship (see Glossary) between stimuli of the reverse sign within each subfield [15,16,17] (Figure 1a). For example, in On subregions, bright light excites whereas dark stimuli inhibit, and vice versa for Off subregions (Figure 1b, top). Moreover, simple computational models made using the spatiotemporal receptive fields of interneurons or relay cells suggest that responses of both types of neurons are roughly linear [15].

We do not wish to imply that push and pull are mirror images of one another; they are not. The centers of the excitatory and inhibitory receptive fields are displaced by distances comparable to the spacing between presynaptic On and Off center ganglion cells [16,17,18]. Also, interneurons probably have larger receptive fields than relay cells since they pool more retinal input [19,20] and may be fewer in number than ganglion cells [21].

Push–pull operates in time as well as space; excitation and inhibition are recruited sequentially when luminance contrast reverses as the image over the receptive field changes [15,16,17,22] (Figure 1b, middle). There is a variation of the standard temporal pattern of push–pull; a population of ‘lagged’ cells whose responses involve an inhibitory dip before excitation [23,24] (Figure 1b, bottom); lagged cells might be involved in establishing direction selectivity in cortex [25].

A universal role for push–pull in neural circuits is to extend the dynamic range of response (Figure 1c), and to restore linearity of response following rectification through the synapse [26]. In the geniculate, these actions are supplemented by push–pull in interneurons, which are thus able to inhibit and to disinhibit their targets [15].

Sensitivity to visual features is enhanced by inhibition

Ideas for the value of push–pull in vision arose from studies of retina [27], but also apply to thalamus. When Kuffler [27] and Wiesel [28] mapped retinal receptive fields they found that the center and surround had an antagonistic affect on each other when illuminated together — cells responded vigorously to discs confined to the center but poorly to a larger stimulus that spilled into the surround. This subfield antagonism reflects the influence of the pull in one subregion on the push in the other [28]. Thus, by virtue of the

Glossary

Push–pull excitation and inhibition: This term refers to an arrangement, within an On or an Off subregion of the receptive field, in which stimuli of the preferred contrast elicit excitation and stimuli of the opposite contrast evoke inhibition. For example, a bright stimulus confined to an On subregion excites whereas a dark stimulus shown in the same place inhibits. If the bright stimulus in the On subregion is enlarged to cover the adjacent Off subregion as well, it recruits push and pull at the same time to produce a lesser response. This interaction between push and pull leads to a mutually antagonist relationship between subregions in the receptive field.

Same-sign excitation and inhibition: This term refers to an arrangement in which a stimulus of the optimal sign and size evokes both excitation and inhibition within a single subregion. For example, a bright stimulus shown within an On subregion evokes both excitation and inhibition. This term does not speak to the relative timing of the excitation or inhibition. In lagged cells, same-sign inhibition precedes excitation whereas it is presumably delayed relative to excitation at the triad. Same-sign inhibition differs from subfield antagonism because it arises within a single subregion.

geometry of the receptive field and interactions between push–pull across subregions, ganglion cells become sensitive to local contrast. These features of the receptive field have been formalized mathematically as a difference of Gaussians [29]. Filtering the image through this function essentially forms the second (spatial) derivative, or Laplacian, of the image, a transformation that enhances contrast borders.

Studies using the framework of generalized linear models to analyze retinal and thalamic spike trains are consistent with a role for push–pull inhibition generated *de novo* in

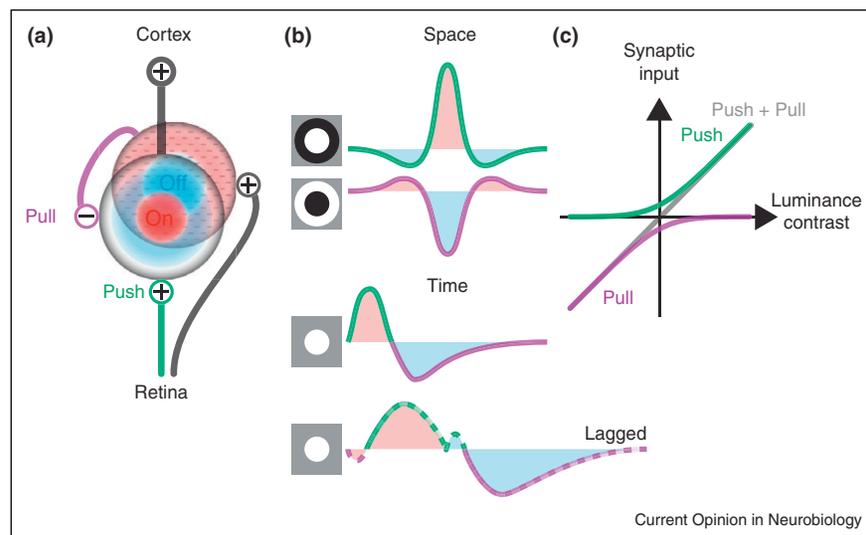
thalamus. [30*] (Figure 2a). The models suggest that excitatory stimuli that extend beyond the center into the surround recruit inhibition. This suppression can be explained by subfield antagonism mediated through the surrounds of (opposite-sign) local interneurons that supply the pull (Figure 2b) and/or input from the reticular nucleus [31]. Last, potential contributions of inhibition to various types of gain control and adaptation [32,33] have yet to be investigated.

Inhibition contributes to efficient coding in thalamus

Barlow considered the retinal receptive field from an alternative vantage point, information theory [34]. From this perspective, processes like subfield antagonism that weaken responses to spatially uniform patterns reduce redundant information about the stimulus and improve the efficiency of the neural code. Barlow’s ideas have gained increasing attention because of observations about the statistics of natural images and about differences in the amount of information encoded by single retinal versus thalamic spikes.

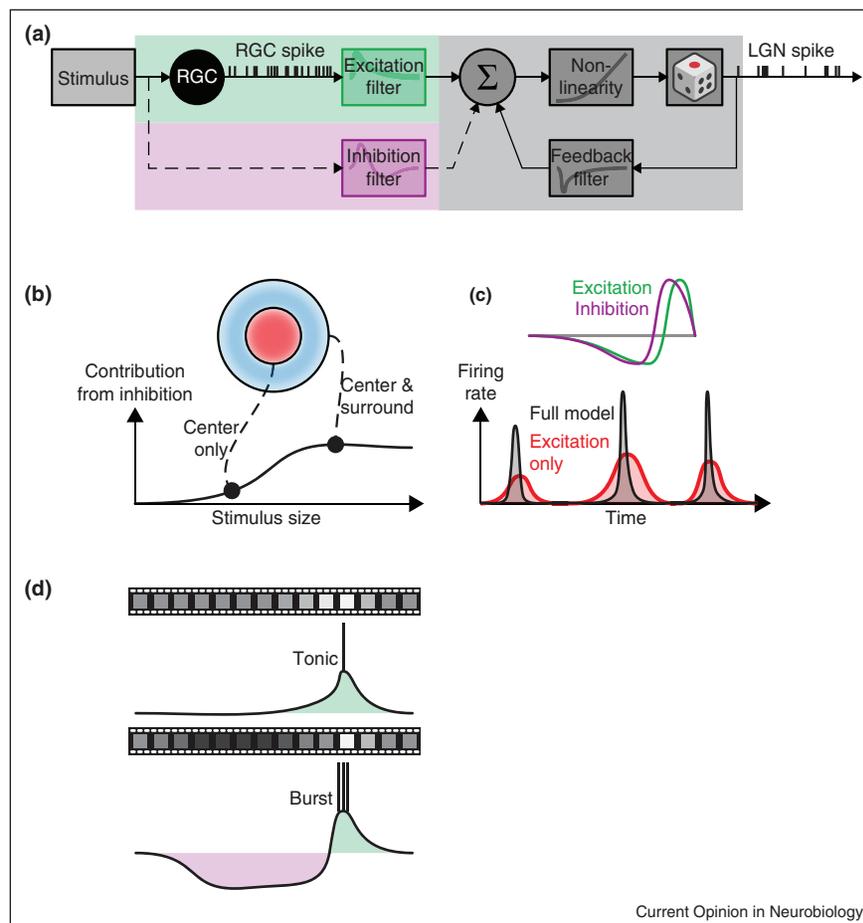
Mathematical analyses show that visual patterns in the environment are, themselves, highly redundant [35]. This can be described intuitively; neighboring points in an image often have similar intensities, forming large regions of homogeneous contrast and, thus, extensive spatial correlations. More formally, the structure of the visual world has $1/f$ statistics, which means that the power spectrum of a given natural image is skewed toward

Figure 1



Synaptic organization of spatiotemporal receptive fields in the visual thalamus. **(a)** Wiring diagram of connections between retinal ganglion cells and thalamic neurons. Neurons are represented by their receptive fields. An On-center (red) thalamic relay cell receives excitatory input (push, green) from an On-center retinal ganglion cell and inhibitory input (pull, purple) from an Off-center (blue) local interneuron which is driven by Off-center retinal afferents. **(b)** Diagrams that illustrate spatial (top) and temporal (middle) receptive fields of push and pull for a typical (On-center) relay cell and the temporal profile for a lagged (On-center) cell (bottom); the dashed lines indicate same-sign inhibition. Icons at left illustrate the stimuli that evoke the schematized patterns of push and pull. For the spatial profiles, the stimulus is presented to the center and surround. For the temporal profiles, a bright stimulus is shown in the center and then removed. **(c)** Illustration of how push–pull circuits extend the dynamic range of stimulus encoding.

Figure 2



How feedforward inhibition influences thalamic output. **(a)** Schematic of a generalized linear model used to describe retinothalamic visual processing [30]. Here a general linear model with excitatory inputs is expanded to include an inhibitory pathway (dashed line), RGC (retinal ganglion cell) LGN (lateral geniculate nucleus). **(b)** The inhibitory component improves the predictive power of the model when the stimulus covers both the center and surround of the receptive field [30]. **(c)** A temporal delay between excitation and inhibition increases the temporal precision of thalamic spike trains [39]. **(d)** Feedforward inhibition evoked by natural movies is able to drive relay cells between tonic (top) and burst (bottom) modes [16] via a push-pull mechanism.

low spatial frequencies [35,36]. Since, as above, the receptive field essentially computes the second derivative of the image, higher frequencies (e.g. edges) are enhanced and redundancy in the stimulus is reduced.

As a consequence of spatial correlations and smooth movement within the environment, there are also temporal correlations in natural stimuli; luminance values at any given point remain similar for long times [35]. For neurons with push-pull, changes in stimulus polarity over time elicit commensurate reversals in the sign of the synaptic response [15,22,26] (Figure 1b, middle). Stimuli with pronounced transitions from the nonpreferred to the preferred sign drive relay cells best [16,22], presumably because the pull readies the membrane to fire, analogous to anode break excitation first described by Hodgkin and Huxley [37]. This selection for biphasic stimuli

corresponds to forming the first (temporal) derivative of the image, thus reducing redundancy of signals with $1/f$ temporal structure such as natural stimuli [38].

Recent computational analyses of thalamic spike trains provide evidence for a second type of inhibition different from push-pull that is evoked by optimal stimuli but is delayed relative to excitation [39] — delayed same-sign inhibition (see Glossary). Since the authors did not compare presynaptic and postsynaptic spike trains, it is not clear whether this form of suppression originates in retina [40], thalamus or both. Like sequential push-pull, delayed same-sign-inhibition can mediate preference for the temporal derivative of the stimulus (Figure 2c). Further, delayed inhibition of any sort increases response precision by shortening the time window in which spikes can fire [39].

A separate line of support for efficient coding in thalamus comes from analyses of simultaneous recordings from ganglion and relay cells. These experiments show that only a fraction of presynaptic impulses trigger the postsynaptic cell, such that each thalamic action potential contains more information than a retinal one [15[•],41,42]. This process of down-sampling across the synapse reduces redundancy in information transmitted about slowly changing signals such as natural stimuli. In addition to inhibition [30[•],39[•],43] processes such as temporal summation [44–46] help increase the economy of the neural code.

Natural stimuli recruit inhibition that drives the membrane between tonic and burst firing

So far, we have only discussed synaptic contributions to sensory integration. However, inhibition also influences neural responses by interacting with membrane dynamics. Relay cells fire either tonic trains or rapid bursts of spikes. Cells fire in tonic mode when the membrane is depolarized, as during waking. Bursts are produced when the membrane voltage is low, as during sleep; these events are generated by T-type calcium channels that cannot respond to fresh excitatory input unless de-inactivated by strong hyperpolarization [47]. Mounting evidence suggests that bursts play an active role in vision [47,48], not only sleep, and are predictably driven by natural stimuli [16[•],49,50]. This matters because bursts preferentially excite cortex [45,51] and also because they can convey information separate from that encoded by single impulses [49,52].

Intracellular studies have illustrated how moving natural scenes drive the membrane from tonic to burst mode [16[•]]. Namely, spatiotemporal correlations in the visual environment lead to instances in which a stimulus of the nonpreferred sign lingers over the receptive field, evoking prolonged inhibition that de-inactivates T-channels. When the stimulus shifts to the preferred sign, retinal input triggers a burst (Figure 2d). Reconstruction of the receptive fields for the inhibition and excitation evoked by moving scenes suggests that the transitions between firing modes rely on push–pull interactions in large part. Specifically, the receptive fields for both excitation and inhibition overlap and are circular, consistent with excitation from retina and inhibition supplied by local interneurons, for example Figure 1a.

Connections of interneurons and of relay cells

The inhibitory cells in the geniculate are unusual in many regards; for example both their axons and dendrites form contacts with postsynaptic targets. The axonal synapses are made with all types of relay cells (X, Y and W) and with interneurons [3–5]. The axonal connections can, in principle, serve multiple purposes. They might contact targets with the opposite selectivity for luminance contrast to mediate push–pull. Alternatively they might link

to cells with the same stimulus preference to supply same-sign inhibition, such as seen in lagged cells.

Dendrodendritic contacts allow cells to communicate even in the absence of action potentials [53[•]]. They often involve triads [53[•]], structures in which a retinal bouton synapses with the apposed dendrites of a relay cell and of an interneuron that synapses onto the relay cell in turn [53[•]]. Triads typically involve X cells, although dendrodendritic synapses onto interneurons or other types of relay cells are present but less frequent [7,20,54]. Further, there is evidence that collaterals of relay cells synapse in the geniculate [55,56], sometimes through triadic synapses [2[•]] where the terminal of a relay cell substitutes for a retinal bouton; these profiles concentrate at interlaminar zones [2[•]] and might promote synchrony or oscillations [2[•],56].

Triads are unlikely to mediate push–pull since common input yokes inhibition to excitation, but are suited for same-sign inhibition. One might hope that comparing X with Y or W cells would give insight into the role of triads; however, differences among relay cells seem inherited from retina. Also the time courses of retinogeniculate EPSPs seem similar across relay cells *in vivo* [16[•],44,46]. Work *in vitro*, however, shows that activation of a single retinal input produces an EPSP immediately followed by an IPSP in some cells [57], consistent with triadic or other mechanisms of same-sign inhibition. Thus, the purpose of triads remains unresolved, with a leading hypothesis suggesting a role in gain control [53[•]]. Notably the rodent somatosensory thalamus lacks interneurons [58]. Perhaps this species difference can be exploited to form new hypotheses about roles of interneurons in general and triads in particular.

Neurons in the reticular nucleus connect with each other via electrical [59] or conventional GABAergic synapses [60–62] and project selectively to relay cells in a topographic fashion [8,10]. Since reticular cells have varied preferences for the stimulus, ranging from On, On–Off to Off, their contributions to visual processing are probably diverse [10], but remain poorly understood. There is, however, compelling evidence that the reticular nucleus modulates relay cells as a function of spatial attention [63], presumably through descending cortical pathways. Whether attention regulates local interneurons, which also receive cortical feedback [64,65] and other sources of modulatory input [11], is unknown.

Patterns of synaptic inputs to relay cells and interneurons and the transmission of information

Even though relay cells and interneurons have similar receptive fields, recent evidence suggests that they use different but complementary forms of synaptic integration to process retinal input during vision [15[•]]. The push in relay cells is generated by series of large, peaked

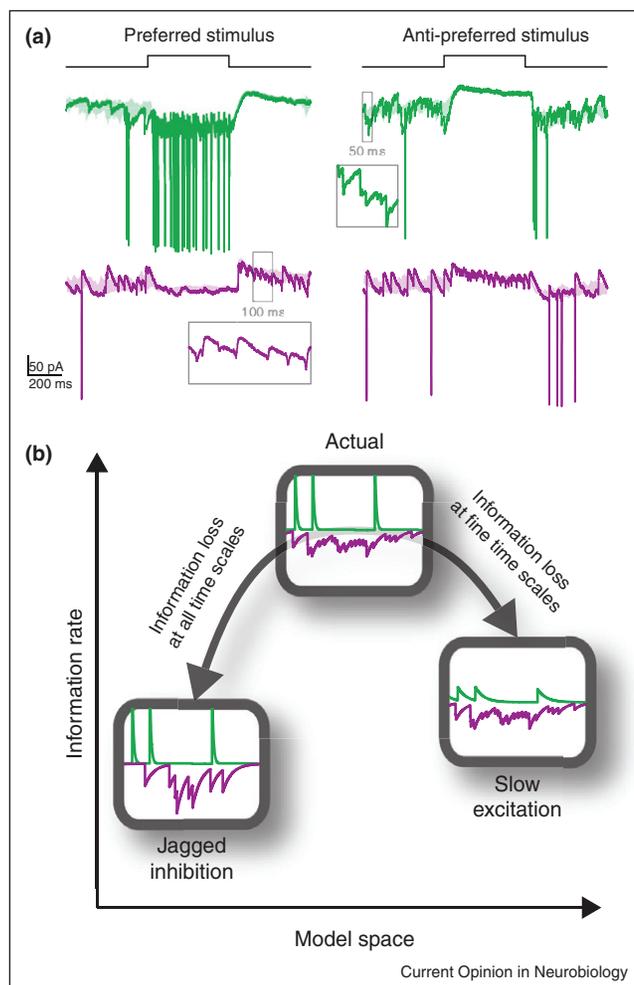
EPSPs while the pull is seamlessly graded — under standard conditions single IPSPs are not visible. On the contrary, the push in interneurons is smooth and the pull is jagged, formed by trains of unitary IPSPs (Figure 3a).

The large, sharp EPSPs originate in retina and evoke spikes from relay cells with millisecond precision [66,67]. Simulations with conductance based models of relay cells indicate that the shape of these EPSPs promotes the rapid and reliable generation of thalamic spikes and thus the ability to transmit information at fine temporal resolution [15]. The simulations further suggest that smooth inhibition does not interfere with this tight coupling between ganglion and relay cells. By contrast, replacing smooth with jagged inhibition reduces the amount of

information relayed to cortex across all time scales (Figure 3b).

The graded inhibition that relay cells receive, that seems optimal for transmitting information downstream, is likely formed by several mechanisms. For example, retinal input to interneurons often targets distal, electronically remote dendrites [53]. Studies *in vitro* show that retinal drive activates L-type calcium currents [68,69] that transmit depolarization to the soma and evoke spikes at variable delay [69]. Also, synapses between retina and interneurons include metabotropic glutamate receptors that might contribute lasting excitation [70]. Thus, EPSPs from ganglion cells might be variously blurred by slow synaptic potentials, passive attenuation and/or intrinsic regenerative currents, thereby decoupling the

Figure 3



Shapes of synaptic inputs and relay of information. **(a)** Membrane current of a relay cell (green) and an interneuron (purple) in response to preferred (left) and anti-preferred (right) stimuli in the center of the receptive field. Insets show magnified synaptic events in time windows marked on the traces. Excitatory (green) and inhibitory (purple) inputs to a relay cell have distinctive shapes: excitation is composed of fast, jagged changes in conductance whereas inhibition is smooth in time. **(b)** The relayed information about the stimulus is optimized with this synaptic configuration: if excitation is made slower, information at fine temporal resolution will be lost; if inhibition is made jagged, transmitted information will suffer loss at all temporal resolutions [15].

timing of presynaptic (retinal) and postsynaptic (interneuron) spikes. Remarkably, work completed over 40 years ago supports this idea of jittered transmission. Cross-correlations made from spike trains of ganglion cells and putative interneurons are broader than those for relay cells [31]. Thus, convergent inhibitory inputs to a relay cell [14,71,72] could arrive asynchronously and average to form smooth pull. It is unclear how the jagged pull in interneurons is formed. One clue is that the frequency of the individual IPSPs matches retinal firing rates; it is possible that they are generated by dendrodendritic synapses between On and Off center interneurons [15]. Why should the pull in interneurons retain the high-frequency structure of retinal spike trains; perhaps to disinhibit relay cells on the timescale of single EPSPs.

Comparison between the lateral geniculate nucleus and cortical layer 4

In cat, thalamic afferents project to simple cells in cortex. Like relay cells, simple cells have receptive fields built of segregated On and Off subregions [73] with push–pull [74,75]. Unlike the concentric arrangement in retina and thalamus, however, cortical subfields are elongated and lie side by side, a change in geometry that confers selectivity to oriented contours. The circuits that construct push–pull in relay cells and cortical simple cells share commonalities. For example, the push can be explained by feedforward drive and the pull by interneurons with receptive fields opposite in sign but similar in shape to those of their postsynaptic partners [76]. There is a salient difference between the thalamus and cortex, however. In the cat's cortex, the shapes of synaptic responses recorded from excitatory and inhibitory cells are similar, formed by graded excitation and inhibition [76–78] (see [79,80] for review of cortical inhibition in various species and modalities and [81,82] for studies of rodent cortex, which seems to lack true simple cells). While there are many subtle variations in the synaptic physiology of excitatory and inhibitory cortical neurons (e.g. [83]), the dramatic differences between the responses of relay cells and neighboring interneurons seem unique to the demands of thalamic processing.

Conclusions

This review focused on inhibition in the visual thalamus, with the aim of integrating perspectives from computation to synaptic physiology. The center-surround structure of receptive fields in the lateral geniculate, coupled with the push–pull arrangement of excitation and inhibition within subregions extends the dynamic range of response, enhances selectivity to luminance contrast in space and time, and serves to reduce redundant information about the stimulus, thereby improving the efficiency of the neural code. Further, the synaptic physiology of interneurons and relay cells is highly specialized and operates in concert to preserve the amount of information encoded at fine time scales and

improves the overall rate of information transmitted downstream.

Acknowledgements

We thank Kilian Koepsell, Luis M. Martinez, Cristina Soto Sanchez, Vishal Vaingankar, and Qingbo Wang for contributions to experiments and for discussion. The work was supported by NIH Grant EY09593 to JAH and NSF Grant (IIS-0713657) to FTS.

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