REVIEW

Deviance detection in auditory subcortical structures: what can we learn from neurochemistry and neural connectivity?

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Abstract A remarkable ability of animals that is critical for survival is to detect and respond to to unexpected stimuli in an ever-changing world. Auditory neurons that show stimulus-specific adaptation (SSA), i.e., a decrease in their response to frequently occurring stimuli while maintaining responsiveness when different stimuli are presented, might participate in the coding of deviance occurrence. Traditionally, deviance detection is measured by the mismatch negativity (MMN) potential in studies of evoked local field potentials. We present a review of the state-of-the-art of SSA in auditory subcortical nuclei, i.e., the inferior colliculus and medial geniculate body of the thalamus, and link the differential receptor distribution and neural connectivity of those regions in which extreme SSA has been found. Furthermore, we review both SSA and MMN-like responses in auditory and non-auditory areas that exhibit multimodal sensitivities that we suggest conform to a distributed network encoding for deviance detection. The un-derstanding of the neurochemistry and response similarities across these different regions will contribute to a better under-standing of the neural mechanism underlying deviance detection.

Keywords Auditory · Stimulus-specific adaptation · Mismatch negativity · Non-lemniscal · Inhibition · Deviance detection

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Introduction

In everyday life, animals are immersed in a continuous flow of sounds arriving from multiple sources, and the auditory sys-tem has the challenge of selecting those acoustic elements relevant for the creation of perceptual constructs (Fishman and Steinschneider 2010). One way of organizing the acoustic scene is as follows: (1) retaining it in a form of sound objects (Winkler et al. 2009), (2) storing the regularity of the objects in a sensory memory trace, (3) generating predictions about forthcoming events, and (4) comparing the subsequent incom-ing sounds with these predictions (Friston 2005; Bendixen et al. 2012). This is the current theoretical processing that occurs in deviance detection (Näätänen et al. 1978, 2001) in which biological systems identify new or deviant contextual events in an otherwise monotonous auditory scene.

The occurrence of auditory deviance detection has been classically associated with the human mismatch negativity (MMN) potential of the event-related potentials (Näätänen et al. 1978). MMN is classically measured by using an oddball paradigm in which a low probability of appearance (deviant) sound is randomly embedded within sequences of common (standard) sounds. MMN is defined as the difference between the event-related potentials evoked by the deviant (larger) and the standard (smaller) sounds. Human MMN peaks between the N1 (100 ms) and the P2 (180 ms) waves and can be

interpreted as an enhancement of the N1 wave or as an independent phenomenon. This differentiation is not trivial, because the N1 wave is attributed to basic auditory processing occurring in the auditory cortex (AC; Hari et al. 1984; Maess et al. 2007), and one of the current interpretations of MMN is that adaptation is related to MMN generation (Fishman and Steinschneider 2012; Fishman 2013). Two conceptually different hypotheses have been proposed to explain the neuronal mechanisms that generate MMN. The first hypothesis is based on the "predictive coding" theory (Friston 2005) that postulates that the brain performs Bayes-optimal sensory learning under uncertainty (Friston 2009, 2012; Moran et al. 2013), whereby the brain creates a prediction concerning the identity of the next sound within an ongoing acoustic sequence based on a memory trace generated by previous stimulation. The violation of the memory-based regularity by a deviant sound would allow the generation of error signals and, therefore, MMN. On the other hand, some other authors (Jaaskelainen et al. 2004; May and Tiitinen 2010) defend a second explanation, namely the so-called "neural adaptation" hypothesis, whereby MMN reflects the release from adaptation elicited by the standard stimulus by the occurrence of a deviant sound. As neurons activated by deviant sounds are stimulated much less than neurons activated by standard sounds, such neurons are consequently less adapted and would elicit a stronger response. Thus, deviant sounds are going to elicit larger responses because adaptation for the standard sound would reduce the N1 wave. To establish the occurrence of "deviance detection", i.e., the degree of enhancement in neuronal activity evoked by the occurrence of a deviant sound that is adaptation-independent, Schröger and colleagues (Schröger and Wolff 1996; Jacobsen and Schröger 2003) have designed a paradigm to control for the probability of occurrence and therefore for the adaptation elicited by the standard tone. Control paradigms have been implemented in subsequent human (Opitz et al. 2005; Maess et al. 2007; Cacciaglia et al. 2015) and animal (rat: Taaseh et al. 2011; Harms et al. 2014) studies. This approach has allowed researchers to separate the signals contributing to MMN attributable to neuronal refractoriness from those evoked by deviance occurrence.

MMN-like phenomena are well known to occur in several different animals, including cats (Csepe et al. 1987a, 1987b; Pincze et al. 2001, 2002), monkeys (Javitt et al. 1992, 1994; Fishman and Steinschneider 2012), guinea pigs (Kraus et al. 1994a, 1994b; Christianson et al. 2014), rabbits (Ruusuvirta et al. 1995a, 2010), rats (Ruusuvirta et al. 1998, 2013; Astikainen et al. 2006, 2011; Nakamura et al. 2011; Jung et al. 2013; Shiramatsu et al. 2013; Harms et al. 2014), and mice (Siegel et al. 2003; Umbricht et al. 2005). Recently, it has been reported that the rat brain is capable of generating human-MMN-like responses, and moreover, that part of the MMN signal is independent of adaptation driven by memory-like processing (Astikainen et al. 2006; Harms et al. 2014). In

agreement with this study, true-deviance detection is also reflected at the single neuron level in the rat AC (Taaseh et al. 2011). Although deviance encoding has been largely thought to involve pure cortical processing, an elegant and recent study by Cacciaglia and colleagues (2015) has demonstrated that the human inferior colliculus (IC) and medial geniculate body (MGB) of the thalamus exhibit genuine deviance detection. This study confirms previous data indicating deviance detection at very short latency responses (~30–40 ms) in auditory-evoked potentials (Slabu et al. 2010, 2012).

Here, in an attempt to gain a better understanding of deviance detection, we review the anatomy and immunocytochemistry of the IC and MGB, since they might reveal the general organizational principles of the subcortical network for deviance detection. Likewise, we present data concerning auditory mismatch responses in non-auditory subcortical structures that might be part of the same network.

Stimulus-specific adaptation

Using the same oddball paradigm as in human MMN studies, Ulanovsky et al. (2003) have found that AC neurons of the cat reduce their response to frequently occurring stimuli (standard) but resume their firing when different rare stimuli are presented (deviant). This neuronal response has been referred to as stimulus-specific adaptation (SSA; Movshon and Lennie 1979). Later studies have demonstrated that SSA also occurs in the IC (rat: Perez-Gonzalez et al. 2005; Malmierca et al. 2009; Duque et al. 2012; Ayala et al. 2013) and the MGB (mouse: Anderson et al. 2009; rat: Antunes et al. 2010). By contrast, neurons in the cochlear nucleus lack SSA (rat: Ayala et al. 2013), suggesting that the IC is the first auditory relay station in which SSA occurs.

Throughout the auditory collicular, thalamic, and cortical areas, SSA has several common characteristics that are similar to the human MMN. SSA is a rapid phenomenon, with a time scale of a few seconds (Ulanovsky et al. 2004; Malmierca et al. 2009; Ayala and Malmierca 2013), and is highly sensitive to stimulus statistics: the smaller the probability of occurrence for the deviant, the larger the SSA (Ulanovsky et al. 2003; Malmierca et al. 2009; Antunes et al. 2010; Zhao et al. 2011; Ayala et al. 2013). These same studies have also revealed that increasing the frequency contrast (the physical separation between the two tones) or the presentation rate (the speed to which the tones are repeated) evokes stronger SSA responses, although SSA can be observed at interstimulus intervals as long as 2000 ms (Ulanovsky et al. 2003; Antunes et al. 2010; Ayala and Malmierca 2013, Fig. 1). As SSA in the AC was first observed in the core region of the AC (Ulanovsky et al. 2003), some authors proposed SSA emerged in the AC as a high order feature that can be "inherited" by the

Fig. 1 Grand-average responses of neurons in the inferior colliculus (*IC*), the medial geniculate body (*MGB*), and the auditory cortex (*AC*). Averaged peristimulus time histograms (PSTH) for the entire population of neurons recorded at various frequency contrasts (Δf) for a standard/deviant ratio of 90/10 %. The mean firing rate elicited by both stimuli (*blue lines* standard, *red lines* deviant) increased at the different Δf (Δf=0.04, 0.1, and 0.37; from *left to right*, respectively). *Black horizontal lines* under the PSTHs indicate the duration of the stimulus: 230 ms for the AC study and 75 ms for the IC and MGB studies (modified from Ulanovsky et al. 2003; Malmierca et al. 2009; Antunes et al. 2010)
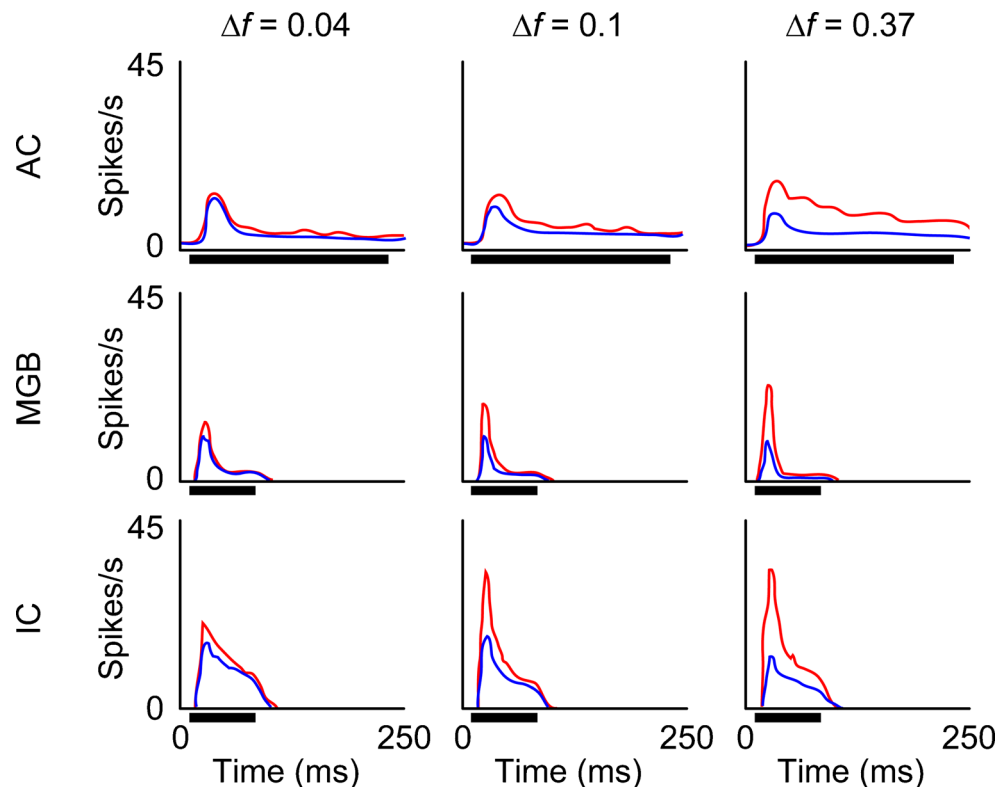
IC and the MGB via the corticofugal pathway (Nelken and Ulanovsky 2007; Bäuerle et al. 2011). However, recent studies with the powerful technique of cortical cooling (Antunes and Malmierca 2011; Anderson and Malmierca 2013) have demonstrated that the corticofugal projections arising from the core AC regions modulate, but do not generate, subcortical SSA. Currently, SSA is thought to be created de novo at each auditory station or to be transmitted in a cascade process from low to higher order nuclei. In this review, we will describe possible mechanisms involved in SSA, with special emphasis on the anatomical substrate that might underlie the connectivity of SSA neurons and on the receptor distributions in the subcortical nuclei of the IC and the MGB.

Throughout the following, SSA will be used to describe a specific reduction of the responses at a neuronal level, whereas MMN (or MMN-like) will be used for differential responses observed at local field potentials. Conceptually, this is different from deviance detection, which refers to an enhancement in the neuronal response evoked by the occurrence of a rare sound and which is independent of adaptation.

SSA is not homogenously distributed in IC and MGB; connectivity of lemniscal and non-lemniscal subdivisions

Auditory processing between the midbrain and the cortex is carried by two parallel streams: the lemniscal (or primary) and non-lemniscal (or secondary) pathways (Fig. 2; Andersen et al.

1980; Lee and Sherman 2010, 2011). The non-lemniscal system comprises the lateral (LCIC), the rostral (RCIC), and the dorsal cortex (DCIC; Loftus et al. 2008, 2010) of the IC and the dorsal (MGBd) and medial subdivisions (MGBm) of the MGB in the thalamus. On the other hand, the lemniscal system comprises the central nucleus of the IC (CNIC) and the ventral division of the MGB (MGBv). Across these subcortical nuclei, SSA responses are not homogenously distributed. Neurons in the cortices of the IC (rat: Malmierca et al. 2009; Duque et al. 2012; Ayala et al. 2013) and in the MGBd and MGBm (mouse: Anderson et al. 2009; rat: Antunes et al. 2010) exhibit the strongest SSA responses. In agreement with SSA data, a pioneering study by Kraus et al. (1994b) has shown that auditory stimuli containing different frequencies or intensities consistently produce a mismatched field potential in the non-lemniscal divisions of the thalamus but not in the MGBv of the guinea pig (Fig. 3). Although such correlation has not been confirmed in the rat AC (Fig. 3, Nakamura et al. 2011; Jung et al. 2013; Shiramatsu et al. 2013), all these data suggest that subcortical acoustic deviance detection is primarily computed by neurons of the non-lemniscal pathway.

Lemniscal and non-lemniscal neurons differ in (1) their response properties (cat: Aitkin et al. 1975; Calford 1983; Calford and Aitkin 1983; Aitkin and Prain 1974; mouse: Anderson and Linden 2011; rat: Lumani and Zhang 2010), (2) their fine morphology, i.e., terminal size and arborization pattern (Oliver 2005; rat: Malmierca et al. 1995; 2011; Stebbings et al. 2014), and in (3) their set of afferent and
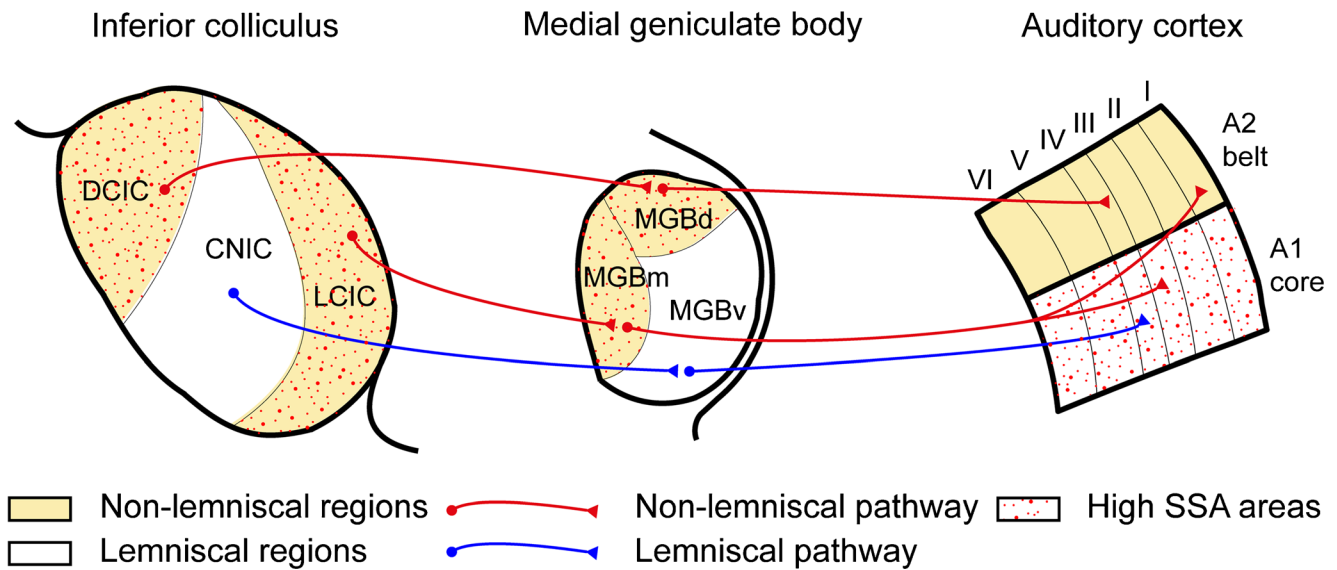
Fig. 2 Topographic segregation of the lemniscal and non-lemniscal projections between the inferior colliculus (IC), medial geniculate body (MGB) and auditory cortex (AC). The strongest SSA responses are distributed in the non-lemniscal subdivisions (*shaded areas*) of the IC (*DCIC* dorsal cortex, *LCIC* lateral cortex, *RCIC* rostral cortex) and MGB (*MGBd* dorsal subdivision, *MGBm* medial subdivision). On the contrary, poor SSA responses are found in the lemniscal areas of the IC (*CNIC* central nucleus) and MGB (*MGBv* ventral subdivision). At the cortical level, the primary auditory cortex (*A1*) exhibits SSA responses that remain to be addressed in areas beyond A1. The major bottom-up projections between non-lemniscal (*red*) and lemniscal (*blue*) collicular, thalamic, and cortical areas are illustrated by *lines* (modified from Escera and Malmierca 2014)
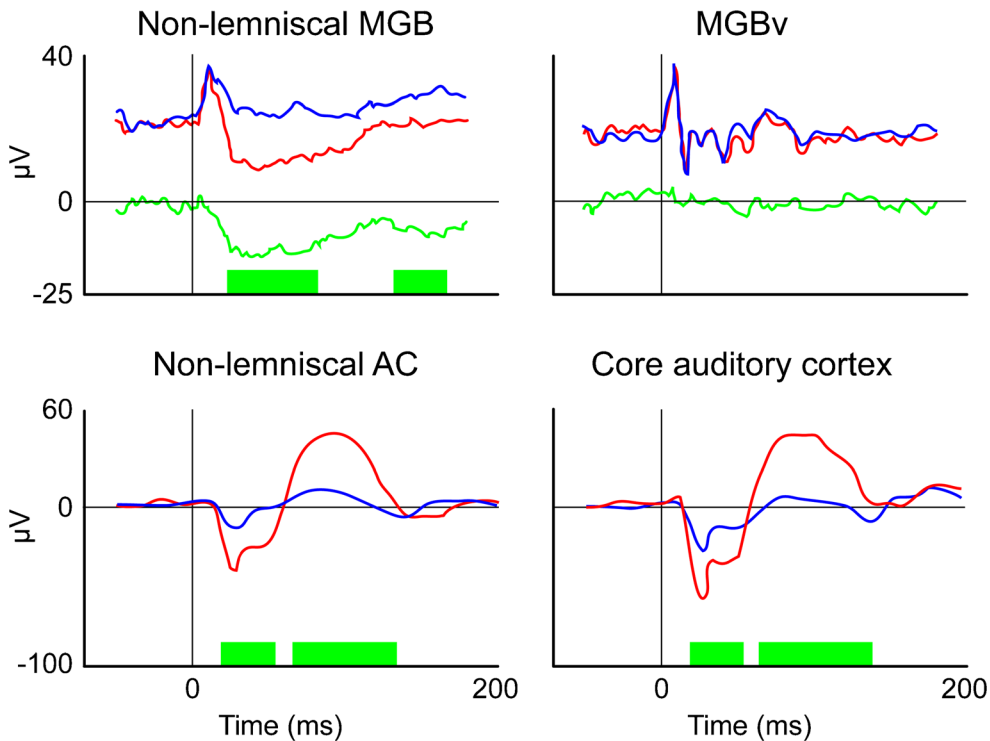
Fig. 3 Comparison of MMN-like responses through the lemniscal and non-lemniscal regions of the medial geniculate body (*MGB*) and the auditory cortex (*AC*). *Top* Grand average responses to standard (*blue lines*) and deviant (*red lines*) stimuli recorded from the non-lemniscal MGB (*left* caudomedial portion of the guinea pig equivalent to the cat MGBm and MGBd) and the MGBv (*right*). Significant differences between the responses to standard and deviant stimuli are indicated by the *green boxes* that appear *under* the various waves (*green lines*). *Bottom* Auditory evoked potentials to deviant (*red lines*) and standard stimuli (*blue lines*) elicited in a non-lemniscal region of the AC (*left* posterior auditory field) and in the core region of the AC of the rat (*right*). Significant differences between the responses to standard and deviant stimuli are indicated by the *green boxes*. Unlike the MGB, MMN-like waves in the AC have not been shown to be spatially distributed (modified from Kraus et al. 1994a; Jung et al. 2013)

efferent projections (rat: Coleman and Clerici 1987; Malmierca and Hackett 2010; for reviews, see Hu 2003; Lee and Sherman 2011). Lemniscal neurons are tonotopically organized exhibiting fast and high-fidelity responses (cat: Miller et al. 2001; rat: Malmierca et al. 2008), whereas non-lemniscal neurons are not tonotopically organized and show longer latencies and habituating responses to unvarying stimuli (cat: Calford 1983; Calford and Aitkin 1983; rat: Bordi and LeDoux 1994a, 1994b; Malmierca et al. 2009; guinea pig: He and Hu 2002). Furthermore, non-lemniscal neurons show broader frequency response areas, i.e., the combination of frequencies and intensities capable of evoking a response (Lennartz and Weinberger 1992; Perez-Gonzalez et al. 2005; Malmierca et al. 2009; Duque et al. 2012; Ayala et al. 2013) and respond preferentially to complex acoustic stimuli (cat: Aitkin et al. 1975). Moreover, the basal discharge and response strength are lower in the non-lemniscal than lemniscal neurons of the IC (rat: Duque et al. 2012) and MGB (cat: Aitkin and Prain 1974; Calford and Aitkin 1983). Interestingly, both the bandwidth of the frequency responses areas (rat: Duque et al. 2012; Ayala et al. 2013) and the onset firing pattern (rat: Duque et al. 2012) correlates with the strength of the SSA responses of IC neurons. Regarding their connectivity, the CNIC primarily sends excitatory and inhibitory projections to the MGBv (for reviews, see Oliver and Huerta 1992; Wenstrup 2005) with a high degree of topographic convergence (Lee and Sherman 2011), whereas the cortices of the IC innervate primarily the MGBd and MGBm (mouse: Romand and Ehret 1990; for reviews, see Hu 2003; Wenstrup 2005). At the level of the MGB, two major afferent streams arise en route to the auditory cortices (cat: Huang and Winer 2000; for a review, see Hu 2003). Generally speaking, secondary auditory cortices are targeted principally by nontonotopic subcortical areas that mainly terminate in layer 1, whereas primary auditory cortices principally receive inputs from tonotopic lemniscal areas that target mainly the middle layers (cat: Kudo and Niimi 1980; Huang and Winer 2000; Lee and Winer 2011; for a review, see Winer 1992).

Traditionally, the early sensory processing neuronal stages are assumed to be unimodal and to operate independently of each other. However, a growing body of evidence has changed this classical view of the sensory hierarchical processing by demonstrating the convergence of multimodal inputs in subcortical nuclei (Stein and Meredith 1993; Stein and Stanford 2008) and early primary sensory cortices (Ghazanfar et al. 2005; Kayser and Logothetis 2007; Lakatos et al. 2007; King and Walker 2012; for a review, see Kayser et al. 2012). In agreement with the above-mentioned, another feature of the non-lemniscal tectal and thalamic areas includes their dense innervation by non-auditory afferents. For instance, the shell area of the IC receives visual (rat and monkey: Itaya and Van Hoesen 1982; rat: Yamauchi and Yamadori 1982; cat: Mascetti and Strozzi 1988) and somatosensory (cat: Aitkin

et al. 1978, 1981; Coleman and Clerici 1987; for a review, see Wu et al. 2014) inputs and projections from the substantia nigra pars compacta lateralis (cat: Coleman and Clerici 1987), globus pallidus (rat: Yasui et al. 1991; cat: Shinonaga et al. 1992), and the ventral tegmental area (rat: Herbert et al. 1997). Likewise, the MGBd and MGBm are innervated by afferents from the tegmental nuclei, spinothalamic tract, and superior colliculus (cat: Graybiel 1972; rat: Ledoux et al. 1987; Iwata et al. 1992). The efferent connections of the non-lemniscal areas of the IC (cat: Aitkin et al. 1978; ferret: King et al. 1998) and MGB (cat: Shinonaga et al. 1994; rat: Doron and Ledoux 1999; 2000) also display divergent projections to non-auditory nuclei.

Together, these structural differences are associated with the different functions exerted by the lemniscal and non-lemniscal systems. The lemniscal pathway is associated primarily with the relay of purely auditory information, whereas the non-lemniscal pathway is part of an integrative system primarily involved in multisensory integration, temporal pattern recognition (cat: Kelly 1973; Layton et al. 1979), change detection (guinea pig: Kraus et al. 1994a; rat: Malmierca et al. 2009), and certain forms of learning (guinea pig: Edeline and Weinberger 1991; rat: Komura et al. 2001). Furthermore, the multimodal afferent and efferent projections characterizing the non-lemniscal pathway are potentially important for sound localization, attending to salient stimuli, and mediating the audio-visual integration of speech stimuli (human: Champoux et al. 2006; for reviews, see Gruters and Groh 2012; Wu et al. 2014).

Major excitatory and inhibitory neurotransmitter receptors

The distribution and expression of receptor subtypes for neurotransmitters and neuromodulators show subtle differences in the IC and MGB subdivisions (summarized in Table 1).

Excitatory neurotransmission is mediated by glutamate in both the IC (cat: Adams and Wenthold 1979) and the MGB (rat: Hu et al. 1994). Both ionotropic AMPA (α-amino-3-hydroxy-5-methylisoxazole-4-propionic acid) and NMDA (N-methyl-D-aspartate) receptors intervene in the synaptic transmission of the IC (rat: Ma et al. 2002; for a review, see Kelly and Zhang 2002) and the MGB (rat: Hu et al. 1994), but recordings from the non-lemniscal regions of the IC do not provide evidence for pharmacological differences from the CNIC (rat: Li et al. 1998, 1999). Immunostaining for AMPA receptors is homogeneous throughout the IC (rat: Petralia and Wenthold 1992; Gaza and Ribak 1997), although the presence of the GluR2-3 AMPA subunit is more common in the non-lemniscal regions of the IC (rat: Caicedo and Eybalin 1999). Additionally, NMDA receptors are more common in the LCIC and the DCIC (rat: Petralia et al. 1994). In accordance with the

Table 1 Main receptor distribution in the human inferior colliculus (*IC*) and medial geniculate body (*MGB*) shown with density (− absent, + to +++ increasing density levels, *SSA* stimulus-specific adaptation, *CNIC* central nucleus of IC, *LCIC* lateral cortex, *RCIC* rostral cortex, *DCIC* dorsal cortex, *MGBv* ventral division of MGB, *MGBd* dorsal subdivision of MGB, *MGBm* medial subdivision of MGB, *AMPA* α-amino-3-hydroxy-5-methylisoxazole-4-propionic acid, *NMDA* N-methyl-D-aspartate, *GABA* Gamma aminobutryic acid, *nNOS* Neural nitric oxide synthase)

Transmitter type	receptor	IC				MGB			SSA implication
		Lemniscal	Non-lemniscal			Lemniscal	Non-lemniscal		
		CNIC	DCIC	LCIC	RCIC	MGBv	MGBd	MGBm	
Glutamate	AMPA	Homogeneous				Homogeneous			Unlikely
	NMDA	+	+++			+	+++		Likely
	mGluR	−	+++			−	+++		Likely
GABA	A	Dorsal	Clusters			Homogeneous			Gain control
	B	+++	+	+++	+	+++	+++	−	Low
Glycine		Ventral	+			−	−	−	Unlikely, IC only
Acetylcholine	Muscarinic	−	+	+	−	Homogeneous			Attention?
	Nicotinic	−	+++	+++	−	Homogeneous			Attention?
Serotonin		+	+++	+++	+++	+	+++	Unknown	Attention?
Norepinephrine		+	+++	+++	+++	Unknown			Learning?
Dopamine		+	+++	+++	+++	Very low			Learning? IC only
Endocannabinoids		Unknown	Unknown	Present		Unknown			Speculative
nNOS		+	+++	+++	+++	Unknown			Speculative
Dynorphin		+	+	+++	+++	Unknown			Unknown
Enkephalin		Homogeneous				Unknown			Unlikely
Substance P		+	+++	+++	+++	Unknown			Speculative

IC data, the presence of AMPA receptors in the MGB has been proved to be homogeneous and moderate in quantity (rat: Sato et al. 1993; Caicedo et al. 1998; for a review, see Parks 2000), whereas the presence of NMDA receptors seems more prominent in the non-lemniscal regions of the MGB (rat: Monaghan and Cotman 1985; Hu et al. 1994). Immunohistochemical studies have also demonstrated the presence of the metabotropic glutamate receptor mGluR$_5$ in the cortical regions of the IC (rat: Abe et al. 1992; Shigemoto et al. 1993). Group I mGluRs, including mGluR$_1$ and mGluR$_5$, are located mostly on postsynaptic parts of cells and act by increasing NMDA activity. Projections from the DCIC and LCIC to the non-lemniscal regions of the MGB recruit mGluR (rat: Bartlett and Smith 2002; mice: Lee and Sherman 2010), as opposed to the mainly ionotropic component of the postsynaptic receptors of the MGBv (for a review, see Lee and Sherman 2011). As NMDA receptors are classically linked to synaptic plasticity, learning, and memory (Morris 2013), the testing of the role, if any, that the NMDA receptors play on shaping SSA would be of great interest in future studies.

Inhibitory neurotransmission in the IC is mediated by both gamma-aminobutryric acid (GABA) and glycine (for a review, see Cant and Benson 2003). GABA$_A$ receptors are located in more dorsal regions of the IC (gerbil: Sanes et al. 1987; bat: Fubara et al. 1996; Lu and Jen 2001), whereas glycine puncta

are more prominent in the ventral region of the IC (cat: Adams and Wenthold 1979; Saint Marie et al. 1989; rat: Merchan et al. 2005). Nevertheless, this is not the only pattern of distribution that emerges with inhibition in the IC, as GABAergic neurons are also organized in cluster modules in the DCIC and the LCIC (rat: Chernock et al. 2004), with an unknown function. Additionally, metabotropic GABA$_B$ receptors are present to a greater extent in the CNIC and the LCIC (rat: Bowery et al. 1983; bat: Fubara et al. 1996; rat: Jamal et al. 2011, 2012). In the thalamus, inhibition in the MGB is primarily mediated by GABA acting at both GABA$_A$ and GABA$_B$ receptors (rat: Bartlett and Smith 1999; Richardson et al. 2011), because MGB lacks glycinergic receptors (rat: Aoki et al. 1988; Friauf et al. 1997). GABAergic interneurons are virtually absent in the rat MGB (only ~1 %: Winer and Larue 1996; Bartlett and Smith 1999), but in this case, the nucleus receives significant GABAergic projections from the IC (bat, rat, cat, and monkey: Winer et al. 1996; rat: Peruzzi et al. 1997; mice and rat: Ito et al. 2011) and the thalamic reticular nucleus (cat: Rouiller et al. 1985).

Recent studies have addressed the effect of neuronal inhibition on SSA by local manipulation of GABA neurotransmission (Fig. 4). The blockade of the GABA$_A$ receptor by using the specific antagonist gabazine elicits an overall increase in the evoked and spontaneous activity in IC (rat: Perez-Gonzalez et al. 2012) and MGB (rat: Duque et al.

2014) neurons. This increase in neuronal discharge is independent of the SSA sensitivity and their discharge pattern. Together, these studies demonstrate that the blockade of GABA$_A$ receptors of IC and MGB neurons with high SSA sensitivity (typically located in non-lemniscal regions) does not abolish SSA (rat: Perez-Gonzalez et al. 2012; Duque et al. 2014), suggesting that the GABAergic system exerts a gain control function on the neuronal responses. Furthermore, the experiments performed in the MGB by Duque et al. (2014) suggest that the ability of GABA to modulate SSA in the MGB is likely to be mediated by the GABA$_A$ rather than by the GABA$_B$ receptors. The effects of gaboxadol, a GABA$_A$-selective agonist that does not bind to GABA$_B$ receptors (rat: Bowery et al. 1983), are no different from the effects elicited by GABA on SSA in MGB. Moreover, as the highest levels of SSA are found in the MGBm (rat: Antunes et al. 2010), an area that lacks GABA$_B$ receptors (rat: Smith et al. 2007), these metabotropic receptors do not seem to be involved in SSA generation. However, any possible synergic effect as a result of the co-application of antagonist or agonists of other subunit or subtype receptors cannot be excluded.

Indeed, as metabotropic receptors (mainly GABA$_B$) are linked to calcium (Ca^{2+}) signaling, Ca^{2+}-binding proteins (namely parvalbumin, calbindin, and calretinin) largely define the lemniscal and non-lemniscal pathways (monkey: Jones 2003; human: Tardif et al. 2003; bats and birds: Covey and Carr 2005; rat: Ouda and Syka 2012). In the IC, parvalbumin is distributed throughout the subdivisions but with a larger presence in the CNIC (bat: Vater and Braun 1994; rat: Lohmann and Friauf 1996; Ouda and Syka 2012). On the other hand, calretinin- and calbindin-positive neurons are usually located in the non-lemniscal regions of the IC (bat: Zettel et al. 1991; rat: Ouda and Syka 2012). In the MGB, parvalbumin is also associated with the lemniscal regions, whereas calbindin is associated with the non-lemniscal regions (rabbit: de Venecia et al. 1995; monkey: Molinari et al. 1995; mice: Cruikshank et al. 2001). The parvalbumin (lemniscal) and calbindin/calretinin (non-lemniscal) distributions are also maintained in the AC (Banks and Smith 2011). The presence of these Ca^{2+}-related proteins might play a critical role in second messenger systems (see: Neuronal mechanisms that might underlie SSA: synaptic depression and retrograde signaling).

Finally, although the presence of glycine in the non-lemniscal regions of the IC is small (Adams and Wenthold 1979; Merchan et al. 2005), the effect of glycinergic receptors in IC neurons on SSA remains unknown. This is an important point, because IC neurons show higher SSA sensitivity at high frequencies (Duque et al. 2012), and glycine is more abundant in the ventral region of the IC (Malmierca and Hackett 2010), a region whose neurons are high-frequency tuned (Malmierca et al. 2008). Thus, in order to have a global picture of the mechanisms that might operate at subcortical SSA, several open questions remain to be answered. Among others, these

include: what is the effect on SSA mediated by the combined activation of the GABA$_A$- and GABA$_B$-receptors and glycinergic receptors, and what is the role, if any, of the NMDA-mediated excitation. Lastly, as will be explained in the following sections, the interactions of any of these systems with specific retrograde messengers could also be crucial to an understanding of the mechanisms that generate SSA.

Neuromodulators in IC and MGB

Recent studies have envisaged a pivotal role of neuromodulators on orienting attention and enhancing the memory for novel stimuli (for reviews, see Ranganath and Rainer 2003; Edeline 2012). Indeed, the four neuromodulatory systems, namely cholinergic (Metherate et al. 2012), serotoninergic (Hurley and Sullivan 2012), dopaminergic (Gittelman et al. 2013), and noradrenergic (Manunta and Edeline 2004), have been implicated in short-term plastic auditory phenomena suggesting that they might also mediate SSA.

Acetylcholine has been implicated in predictive coding and learning under uncertainty in humans (Moran et al. 2013). Moreover, acetylcholine is known to exert frequency-specific plasticity in both cortical (cat: Metherate and Weinberger 1989) and subcortical (bat: Ji et al. 2001) neurons. Cholinergic terminals are abundant in the DCIC and the LCIC (guinea pig: Jain and Shore 2006; for a review, see Shore 2008), and the receptors in these regions are mainly nicotinic. In the MGB, almost all the neurons present either muscarinic and/or nicotinic receptors and are excited by acetylcholine (cat: Tebecis 1970, 1972). Acetylcholine depolarizes most of the neurons in the lemniscal MGBv through the muscarinic receptors, whereas a large amount of the neurons in the MGBd remain unaffected (rat: Varela and Sherman 2007; for a review, see Varela 2014).

Serotoninergic projections to all the regions of the IC arise from the raphe nuclei (bat: Hurley and Pollak 2005), although fibers are denser in non-lemniscal regions such as the DCIC and the LCIC (for reviews, see Hurley et al. 2002; Hurley and Sullivan 2012). Raphe nuclei also present projections to the MGB (monkey: Lavoie and Parent 1991; rat: Vertes et al. 2010), modulating the responses of many of the neurons in the lemniscal MGBv and most of the neurons in the non-lemniscal MGBd (guinea pig: Pape and McCormick 1989; cat: McCormick and Pape 1990; ferret: Monckton and McCormick 2002; rat: Varela and Sherman 2009; Varela 2014).

Attention during task engagement is known to modulate neuronal responses (ferrets: Fritz et al. 2003, 2007), and thus, attention mediated by either acetylcholine or serotonin might produce this modulation, hence affecting the levels of SSA. Indeed, recent work concerning SSA in the IC of the awake mouse (Duque and Malmierca 2014) has demonstrated that

the lower the spontaneous firing rates, the higher the SSA sensitivity. As spontaneous activity can be modulated during attention and can enhance signal detection (gerbils: Buran et al. 2014), both neuromodulators might be involved in SSA sensitivity. In view of the involvement acetylcholine (Jones 2005) and serotonin (Kelly and Caspary 2005) in arousal and attention, the effect of such neurotransmitters on SSA might be more important than previously estimated, and we need further studies in this field.

The response of the neurons of the IC and the MGB are also affected by catecholamines, mainly through noradrenergic- (rat: Swanson and Hartman 1975; Foote et al. 1983; Klepper and Herbert 1991) and dopaminergic (rat, cat and bat: Olazabal and Moore 1989; cat: Paloff and Usunoff 2000) fibers. The locus coeruleus is the main source of noradrenaline for both the IC and the MGB (Aston-Jones 2004). The dopaminergic innervation for the IC arises from neurons in the substantia nigra (Moore and Bloom 1979; Olazabal and Moore 1989), whereas the dopamine sources for the MGB are highly diverse (macaque: Sanchez-Gonzalez et al. 2005). Recent findings suggest that the noradrenergic direct projection from the locus coeruleus to the IC is mainly ipsilateral and just reaches the non-lemniscal regions of the IC (rat: Klepper and Herbert 1991; Hormigo et al. 2012). The dopaminergic neurons identified in the IC also have a distinct distribution, the largest number of dopaminergic neurons being located in the LCIC and the DCIC (rat: Tong et al. 2005).

In summary, considering that (1) noradrenaline and dopamine act as learning signals, and both neuromodulators are released at times of novelty and uncertainty (Harley 2004), and that (2) acetylcholine and serotonin mediate attention, their involvement in SSA sensitivity might be critical. Since SSA is a pre-attentive feature observed under anesthesia (rat: Malmierca et al. 2009), in awake (mouse: Duque and Malmierca 2014), and sleep-like states (rat: Nir et al. 2013), SSA is probably affected by the variations in the levels of neuromodulators occurring during these states. Thus, future studies of the various modulatory substances will undoubtedly shed light on the way that the brain learns to discriminate potentially relevant sounds (Edeline 2012).

Neuronal mechanisms that might underlie SSA: synaptic depression and retrograde signaling

Since GABAergic inhibition plays a key role in modulating SSA rather than in its generation (Perez-Gonzalez et al. 2012; Duque et al. 2014), a synaptic depression model (Grill-Spector et al. 2006; Briley and Krumbholz 2013) might be a more likely explanation for SSA. In this model, the continuous activation of the same set of neurons will eventually produce a specific decrease of the neurotransmitter release at the presynaptic level, and consequently, the postsynaptic neuron will not generate a response.

The frequency-specific adaptation channel theory supports this model (Eytan et al. 2003; Mill et al. 2011a, 2011b; Taaseh et al. 2011; Nelken 2014) in which all the frequencies that are within the same frequency channel will not evoke SSA, because they are encoded by the same neurons. Although recent data from the rat AC (Hershenhoren et al. 2014) suggest other, as yet unknown, more complex mechanisms to explain SSA at the cortical level, in the IC, SSA is not constant within the neuronal receptive field of the neuron, and SSA sensitivity is stronger on the high-frequency edge of the receptive field (Duque et al. 2012). This phenomenon presents interesting correlates in the auditory nerve fibers (Westerman and Smith 1985) and the IC (Dean et al. 2008) in which adaptation is also more prominent at high frequencies. Together, these studies suggest that synaptic depression (Chung et al. 2002; Rothman et al. 2009) might explain subcortical SSA, as it is an input-specific mechanism, and neural responses depend upon the previous history of afferent firing (Abbott et al. 1997; Rothman et al. 2009).

Moreover, as various retrograde signaling pathways can act at the synaptic level (Regehr et al. 2009), synaptic depression does not necessarily have to be a passive phenomenon (i.e., fatigue). Retrograde signaling mediates short-term synaptic plasticity (Regehr 2012), a likely explanation for synaptic depression (Abbott and Regehr 2004): lipids (endocannabinoids), gases (nitric oxide), peptides (i.e., met-enkephalin and/or dynorphin), and conventional amino acid transmitters (i.e., glutamate and/or GABA) can be released in an activation-dependent manner by postsynaptic neurons and then act on the axon terminals of presynaptic neurons (Regehr et al. 2009). The production and release of these messengers is regulated by the activation of postsynaptic glutamatergic metabotropic receptors (Regehr et al. 2009) and/or by postsynaptic Ca^{2+} (de Jong and Verhage 2009), two components mainly located in the non-lemniscal regions of the IC and the MGB (see above). In the mammalian brain, the endocannabinoid signaling system is the best-characterized retrograde signaling pathway and enables neurons specifically to regulate the strength of their inputs in a retrograde manner (Wilson and Nicoll 2002; Freund and Hajos 2003; Kano et al. 2009). Endocannabinoid type-1 (CB1) receptors are thought to be one of the most widely expressed G-protein-coupled receptors in the brain (Herkenham et al. 1990). The presynaptic localization of CB1 receptors and their inhibitory effect on neurotransmitter release has been shown to be a general feature of most axon terminals in the central nervous system (Kano et al. 2009), suppressing synaptic strength for tens of seconds (Wilson et al. 2001). The firing evoked by the standard stimuli in an oddball paradigm can activate group I mGluR, thus inducing endocannabinoid release (Chevaleyre et al. 2006) and contributing to short-term synaptic plasticity (Castillo et al. 2012), as in the fast adaptation occurring in the neuronal firing to the standard tone. Endocannabinoid signaling has been demonstrated to act in the auditory pathway at the level of the cochlear nucleus (Zhao et al. 2009; Zhao and Tzounopoulos 2011),

the superior olivary complex (Trattner et al. 2013), and the external cortex of the IC of the barn owl (Penzo and Pena 2009), but its affect on auditory SSA remains unknown.

Much less is known about other retrograde neurotransmitters. For example, the highest levels of nNOS (the neuronal enzyme that synthesizes nitric oxide) are evident in the DCIC and the LCIC, with much lower levels in the CNIC (Druga and Syka 1993; Paloff and Hinova-Palova 1998; Coote and Rees 2008). Few data are available for understanding the involvement of nNOS in the MGB. As another example, immunostaining for kappa (κ) opioid receptors (dynorphin) is significantly higher in the LCIC, but delta (δ) opioid receptors (met-enkephalins) are similarly distributed within the IC (Aguilar et al. 2004; Tongjaroenbuangam et al. 2006). Thus, the distribution of the opioid receptors cannot be correlated with specific regions of the IC. The data regarding opioid receptor distribution in the MGB are also sparse and difficult to interpret. Lastly, we should mention the existence of extensive labeling for fibers with both somatostatin and substance-P in the DCIC and the LCIC, with somatostatin showing the most extensive encircling of the CNIC (Wynne and Robertson 1997). Interestingly, no significant fibers labeling for either peptide have been observed within the CNIC (Wynne and Robertson 1997). Unfortunately, the involvement of retrograde signaling in SSA remains unknown, and much work is needed to gain an understanding of the functions of these systems in sound discrimination.

Detection of acoustic deviance in non-auditory nuclei

Beyond the auditory pathway, stronger neuronal responses to deviant sounds have also been found in some non-auditory


Fig. 4 Effect of neuronal inhibition on the SSA responses in IC and MGB neurons. **a** *Dot raster plots* obtained after an oddball paradigm for a pair of frequencies in an IC neuron. The *dot rasters* refer to the control (*left*), gabazine (*GBZN, middle*), and recovery (*right*) conditions. Frequencies are shown as *insets over* the control condition. The *first row* shows f1/f2 as a standard/deviant, whereas the *second row* shows the reverse condition (f2/f1 as standard/deviant). The *black line* lying *under* the *dot rasters* represents the stimulus duration. Common SSA index (*CSI*) values are shown over each condition. The mean peristimulus time histograms from both *dot rasters* are shown in the *third row* (*Spk* spike). **b** *Dot raster plots* obtained after an oddball paradigm for a pair of frequencies in an MGB neuron. Same format as in **a**. **c** *Box plot* of the CSI values for the population of neurons of the IC before, during, and after the application of GBZN. **d** *Box plot* of the CSI values for the population of neurons of the MGB before, during, and after the application of GBZN and GABA. In both cases, *asterisks* indicate significant differences (Friedman test, *P*<0.01). **e** In the absence of inhibition, neurons respond to deviants (*orange*) and standards (*light blue*) with high firing rates, and thus the deviant to standard ratio is small. **f** Inhibition reduces the responses to both deviants (*red*) and standards (*dark blue*) increasing the deviant to standard ratio and thus enhancing SSA. **a, c, e, f**: modified from Perez-Gonzalez et al. (2012). **b, d**: modified from Duque et al. (2014)
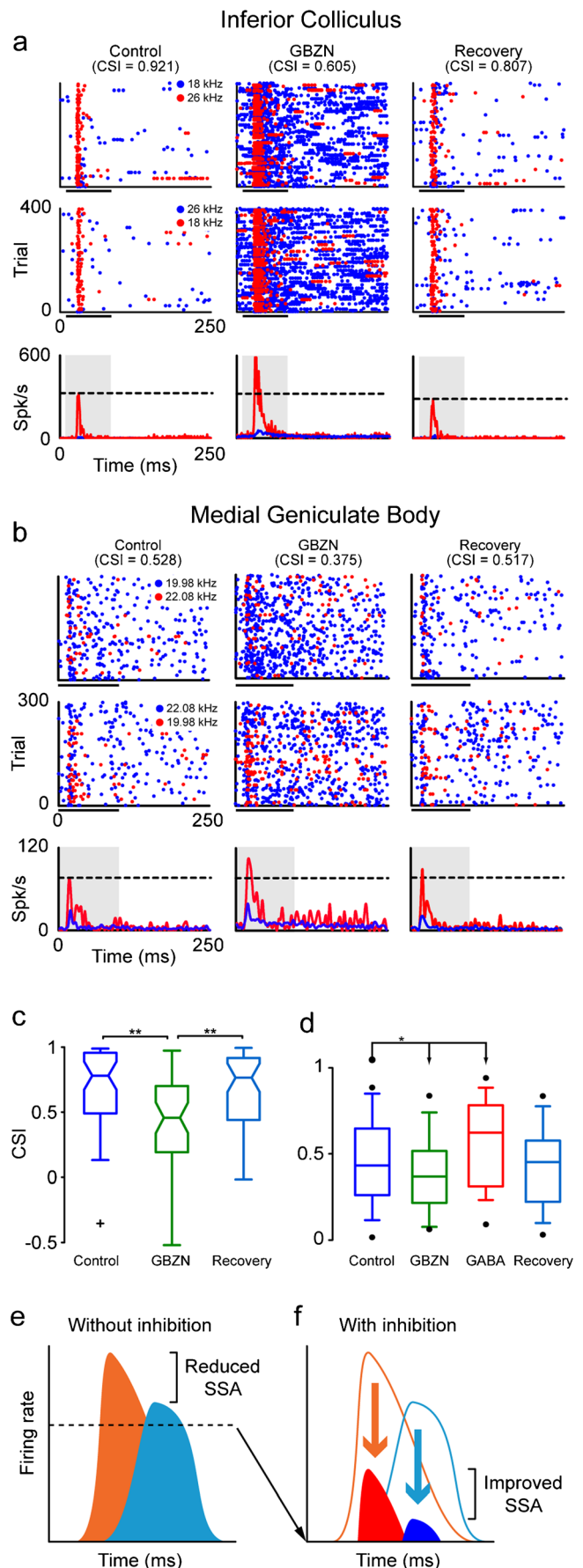
nuclei, including the substantia nigra (Minks et al. 2014), hippocampus (Ruusuvirta et al. 1995b, 1995c), basal ganglia (Mikell et al. 2014), optic tectum (Reches and Gutfreund 2008), and thalamic reticular nucleus (Yu et al. 2009). As mentioned in the previous section, some of these nuclei have anatomical connections with the non-lemniscal auditory subdivisions.

The substantia nigra is typically involved in sensorimotor coordination. However, recently, a study in humans by Mikell et al. (2014) has demonstrated that novel sounds evoke a greater firing rate compared with the activity following standard tones in substantia nigra neurons. The response following the deviant presentations displays a biphasic temporal pattern with peaks at approximately 300 and 500 ms (Fig. 5a). Interestingly, an inverse correlation occurs between the firing rate and the strength of the novelty response across all recorded neurons, i.e., neurons sensitive to deviant stimulus exhibit slow firing, whereas neurons with the highest firing rates are suppressed by the novel stimuli. Mikell and colleagues (2014) speculate that the substantia nigra neurons that discriminate deviant and standard tones correspond to dopaminergic neurons, since previous studies indicate dopaminergic neurons are sensitive to novelty (Bunzeck and Düzel 2006). Moreover, the former authors further suggest that the other group of substantia nigra neurons with high and tonic firing rates suppressed by the deviant tone correspond to GABAergic interneurons that are known to inhibit the local dopaminergic neurons (Tepper et al. 1995). Interestingly, the response in the substantia nigra, i.e., the first peak of the increased activity following novel sounds (250–350 ms after stimulus onset), occurs concurrently with the onset of the hippocampally-dependent potential P300, a scalp-recorded potential linked to novelty detection. Notably, MMN-like responses to pitch (Ruusuvirta et al. 1995b, 1995c; 1996), duration (Rosburg et al. 2007; Ruusuvirta et al. 2013), and frequency deviants (Ruusuvirta et al. 2010) have been recorded across the hippocampus in animal studies. The hippocampus is associated with involuntary attention switches toward auditory changes of high magnitude. Ruusuvirta et al. (2013) have found that MMN responses to duration deviants are elicited across the three hippocampal areas, i.e., CA1, dentate gyrus, and subiculum in anesthetized rats. In a previous report from the same group (Ruusuvirta et al. 2010), higher amplitude local field potentials in response to frequency deviants have been recorded in the CA1 of awake animals. The MMN response to frequency and duration deviants occurs within a similar time window, at 36–80 and 51.5–89 ms post-stimulus, respectively.

Recently, other non-auditory nuclei have been implicated in acoustic deviance detection. The subthalamic nucleus is integrated into the basal ganglia system, and traditionally, it is considered to play a role in cortical motor control regulation
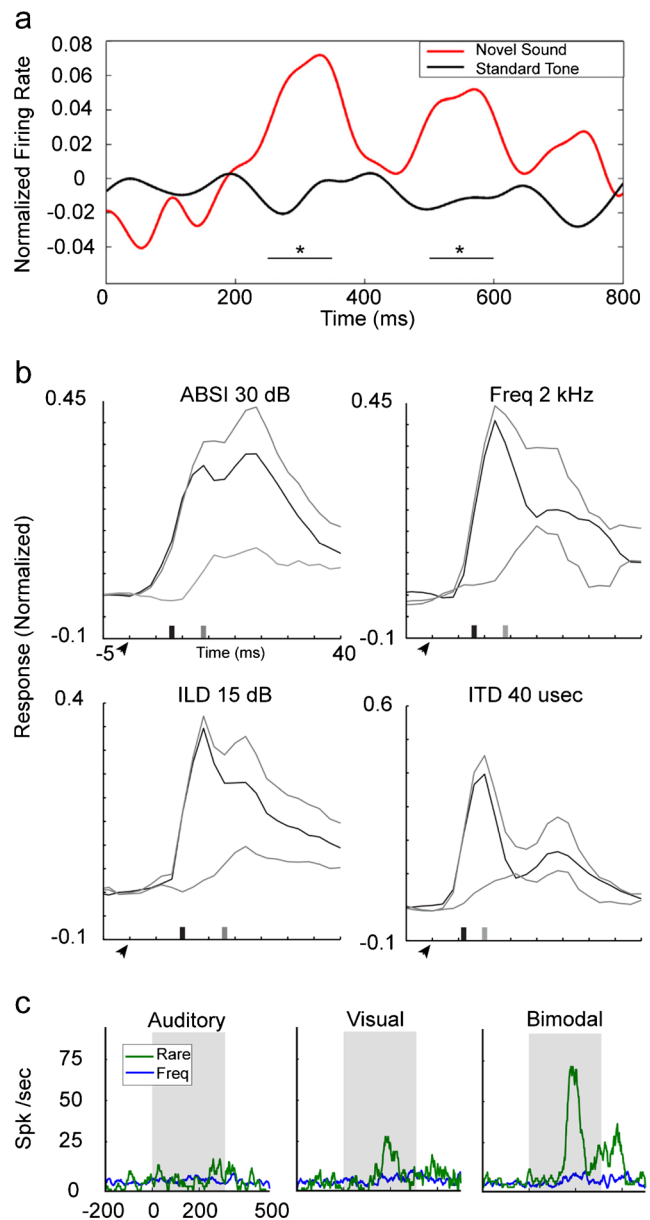
Fig. 5 Response to acoustic deviants in non-auditory subcortical nuclei. **a** Normalized firing response of neurons of the substantia nigra. Novel sound evoked a greater response (*red line*) compared with standard tones (*blue line*) over the 250–350 ms interval and over 500–600 ms (*black lines* regions of *$P<0.05$; modified from Mikell et al. 2014). **b** Optic tectum neuronal SSA responses to average binaural sound intensity (*ABSI*, 30 dB gap), frequency (*Freq*, 2 kHz gap), interaural level difference (*ILD*, 15 dB gap), and interaural time difference (*ITD*, 40 μs gap). *Top gray line* is the response to a rarely presented stimulus, *black line* the response to a frequently presented stimulus, *bottom gray line* the difference between the two, *arrowheads* stimulus onset, *black ticks* latency of the population response to the frequent stimulus, *gray ticks* latency of the difference signal (taken from Reches and Gutfreund 2008). **c** Entopallium neuronal responses to deviant occurrences (*green lines*) together with the corresponding responses to frequent occurrences (*blue lines*) of auditory, visual, and bimodal stimulus (modified from Reches et al. 2010)

(Rektor et al. 2001). Minks et al. (2014) have demonstrated that deviant pure tones elicit MMN-like responses in the

subthalamic nuclei by using intracranial electroencephalograph recordings in humans. The responses are evident in the far-field potentials (intracerebral electrodes referenced to the extracranial electrode) and near-field potentials (intracerebral contacts referenced to one another within the subthalamic nuclei) with a similar average time-to-peak, i.e., 202 and 214 ms, respectively.

SSA responses have been also found in the optic tectum, which is the avian homolog of the superior colliculus of mammals. The optic tectum is involved in orienting gaze toward salient stimuli and receives auditory inputs primarily from the cortices of the IC (Gutfreund 2012). In an elegant study performed by Reches and Gutfreund (2008), SSA responses to more than one acoustic feature, i.e., sound frequencies, amplitudes, and interaural and interlevel time difference, were found in the single- and multi-unit activity of the optic tectum neurons (Fig. 5b). All acoustic features elicited a similar pattern of adaptation developed several milliseconds after the onset of the response. Interestingly, neurons of the IC exhibited SSA only to sound frequency but showed a higher neuronal hyperacuity. These results led the authors to suggest that neurons in higher-level centers of the ascending gaze control system, such as the optic tectum where the IC neurons project to, encompass SSA to multiple acoustic features and not only to one single acoustic dimension as occurs in the IC of the barn own.

The studies highlighted above clearly demonstrate that deviance detection is a widely distributed neuronal network that goes beyond the auditory pathway (Ranganath and Rainer 2003). At the same time, these studies lead to questions regarding the possibility of whether these neuronal structures play an active role in the genesis of deviance detection or simply reflect adaptive neuronal processes taking place at their projecting sources. To gain information in order to permit a direct comparison with the SSA studies, an interesting project would be to record the responses of a single neuron to various acoustic stimuli in the hippocampus, subthalamic nuclei, or superior colliculus under the same paradigms as those used in the auditory SSA studies (Ulanovsky et al. 2003; Malmierca et al. 2009; Yaron et al. 2012).

Multisensory responses and deviance detection

Neurons showing SSA also participate in multisensory processing since information integrated across various sensory modalities can greatly enhance our ability to detect, discriminate, or respond to relevant sensory events (for reviews, see Stein and Meredith 1993; Gleiss and Kayser 2012). As described previously, the non-lemniscal subdivisions of the IC and MGB in which the strongest SSA responses are found receive afferent projections not only from auditory nuclei, but also from visual and somatosensory nuclei (Wu et al. 2014). Indeed, several studies indicate that other subcortical

nuclei involved in deviance detection also engage multimodal sensitivities. Neurons with multimodal sensitivity exhibit suprathreshold responses to stimuli from more than one sensory modality, and often the response of these neurons is stronger to combined stimuli than to the most effective single-modality stimulus (Meredith et al. 2012). Examples of auditory neurons whose sensitivity differs significantly when they are stimulated by bimodal (e.g., audiovisual) stimuli are summarized below. Yu et al. (2009) have found SSA responses in the thalamic reticular nucleus, a multimodal area mainly conformed by GABAergic neurons and have shown that MGB acoustic responses are modulated in a cross-modal manner by a preceding light stimulus. This modulation of the auditory responses is abolished by the inactivation of the thalamic reticular nucleus. Moreover, eye position has been demonstrated to modulate auditory responses as early as in the shell area of the IC of primates (Groh et al. 2001; for reviews, see Gruters and Groh 2012; Wu et al. 2014).

Likewise, other subcortical non-auditory structures that are particularly sensitive to deviant sound also exhibit multimodal sensitivities. Neurons in the substantia nigra are responsive to visual, auditory, and somatosensory stimulation (Nagy et al. 2005; Chudler et al. 1995) exhibiting multisensory response enhancements (Nagy et al. 2006). In the barn owl, optic tectum neurons show multisensory enhancement (Zahar et al. 2009) and SSA for visual stimuli, in addition to SSA for sound frequency deviants (Reches and Gutfreund 2008). Moreover, Reches et al. (2010) have performed a pioneering study demonstrating that a bimodal stimulus enhances the SSA responses of entopallium neurons, a forebrain structure of the barn owl (Fig. 5c). This enhancement occurs only when the auditory and visual stimuli are congruent in space and time (Reches et al. 2010). Whether the same multimodal enhancement of the auditory SSA responses occurs at early subcortical processing stages remains to be determined in future studies.

The superior colliculus is also of special interest for the exploration of SSA, because (1) the optic tectum (which is its avian homolog) encompasses SSA for multiple acoustic parameters and for visual stimulus (Reches and Gutfreund 2008) and (2) the basic principles of multisensory integration have been revealed in single-cell studies in the superior colliculus (Stein and Wallace 1996; Stein and Stanford 2008; Meredith et al. 2012). Interestingly, neurons of the superior colliculus of the anesthetized cat with little or no spontaneous activity and weak sensory responses have the ability to exhibit large multisensory response enhancements in comparison with the poor response enhancements exhibited by superior colliculus neurons with modest spontaneous activity and robust sensory responses (Perrault et al. 2003). The response features of the ongoing multisensory enhancement of the superior colliculus resemble the poor response of the IC neurons showing strong SSA (Duque et al. 2012) and those of substantia nigra neurons sensitive to deviant stimulation

(Mikell et al. 2014). Finally, the tegmental nucleus is another multisensory structure (Koyama et al. 1994; Reese et al. 1995) in which acoustic SSA is likely to occur (Schofield et al. 2011), since its neurons attenuate or even abolish their response to repetitive auditory stimulation (Koyama et al. 1994). Furthermore, tegmental nucleus neurons project to the IC and receive projections from the AC (Schofield 2010).

All the above-mentioned studies support the notion that the subcortical nuclei engaged in auditory deviance detection constitute a neuronal microcircuitry based on the convergence of inputs from various sensory modalities that might act as early integration centers to enhance deviance detection. This wealth of data sets forth a promising field in which to explore and determine whether subcortical neurons allow better deviance coding under multisensory compared with unimodal conditions.

Final remarks

We have reviewed evidence of MMN-like and SSA responses occurring in the shell area of the IC and MGB and in non-auditory nuclei and suggest that they conform to a distributed subcortical network for deviance detection. Likewise, the topographic distribution of SSA in the shell areas of the IC and MGB suggests that the microcircuitry and neurochemistry of the non-lemniscal areas exert a critical role in the generation and modulation of SSA in these nuclei. Further studies combining physiological, anatomical, and molecular approaches will broaden our understanding of the microcircuits of SSA in the auditory and non-auditory nuclei. Moreover, these studies will contribute to the determination of the similar or different contribution of each nucleus to the processing of deviant sounds and to the triggering of the cascade of neuronal processes that allow animals to adapt rapidly to environmental changes.

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