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Review

Development of Auditory Cortex Circuits

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ABSTRACT

The ability to process and perceive sensory stimuli is an essential function for animals. Among the sensory modalities, audition is crucial for communication, pleasure, care for the young, and perceiving threats. The auditory cortex (ACtx) is a key sound processing region that combines ascending signals from the auditory periphery and inputs from other sensory and non-sensory regions. The development of ACtx is a protracted process starting prenatally and requires the complex interplay of molecular programs, spontaneous activity, and sensory experience. Here, we review the development of thalamic and cortical auditory circuits during pre- and early post-natal periods.

Keywords: cortex, activity, subplate, thalamus, development

INTRODUCTION

The auditory cortex (ACtx) is a key central sound processing area. Sound is transmitted in the form of vibration through the ear canal, passing through the tympanic membrane, ossicles, and transduced into electrical activity in the cochlea. This activity propagates through a series of different brainstem and midbrain nuclei before reaching the auditory thalamus (medial geniculate body, MGB) and finally the ACtx (Budinger and Kanold 2018; Goodrich and Kanold 2020). In mammals, the thalamus is subdivided into two major divisions, the first-order (lemniscal) ventral division of the medial geniculate body (MGBv) and higher-order (non-lemniscal) nuclei such as dorsal and medial division of the medial geniculate body (MGBd and MGBm) (Mesulam and Pandya 1973; Ryugo and Killackey 1974; Redies et al. 1989; Budinger et al. 2000; de la Mothe et al. 2006; Horie et al. 2013). These subdivisions project to distinct areas of ACtx. Accordingly, ACtx can be generally divided into (i) lemniscal areas, composed of primary auditory cortex (A1) and anterior auditory field (AAF), which receive inputs from the MGBv, and (ii) non-lemniscal or secondary auditory cortical areas, e.g., dorsal and ventral field of the auditory cortex (Hackett et al. 2011; Anderson and Linden 2011). Primary and secondary cortical areas are also distinguished by their feedback projections to the first-order and higher-order thalamus (Sherman and Guillery 2002; Bartlett 2013). In general, developmental studies have focused on the development of the lemniscal pathway.

Considering that various animal models have been used in studying the auditory system in the past, we first compare the definition and timing of the onset of hearing. We next discuss some of the key developmental events (i.e., neurogenesis, neuronal migration, development of dendrite and synapse) that take place during pre- and early post-natal periods in the ACtx. At times, we include developmental studies from the somatosensory (SSCtx) and visual cortices (VCtx) to highlight general principles for sensory cortical development. In the following subsections, the developmental sequences for excitatory and inhibitory neurons populating the ACtx and their respective role in circuit formation are also discussed. We conclude the review with a list of open questions to inspire further exploration of crucial topics in the field.

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SPECIES CONSIDERATIONS AND THE ONSET OF HEARING

Auditory processing, and thus hearing, starts with the maturation of the cochlea and transmission of sound-evoked neural activity to central structures such as the ACtx. However, the "onset of hearing" has been difficult to define. For decades, the understanding of the development of auditory regions from the molecular to the cellular and systems level has mainly been based on studies performed in different animal species. While auditory processing in humans starts in utero, with physiological or neural responses to sound emerging around the end of the second trimester (Birnholz and Benacerraf 1983; DeCasper and Spence 1986; Hepper and Shahidullah 1994; DeCasper et al. 1994; Kisilevsky et al. 2003; Draganova et al. 2005; Porcaro et al. 2006; Voegtline et al. 2013), most animal models have altricial auditory development. The onset of hearing is somewhat ill-defined as the maturation of neural and functional responses in utero is obscured by attenuation of external sounds in the womb and difficulty in detecting sparse or weak responses (Fig. 1). The ear canal in humans is opened around the 21st gestational week (GW) (Nishimura and Kumoi 1992; Anthwal and Thompson 2016). Sound-evoked fetal movements in response to low sound frequencies (e.g., 100–500 Hz) can be detected in the 19th GW, while responses to higher frequencies (1000 Hz and 3000 Hz) emerge after the 27th GW (Hepper and Shahidullah 1994). Similarly, in altricial animals, closed ear canals and immature transmission through the middle ear can potentially mask functioning neural connections.

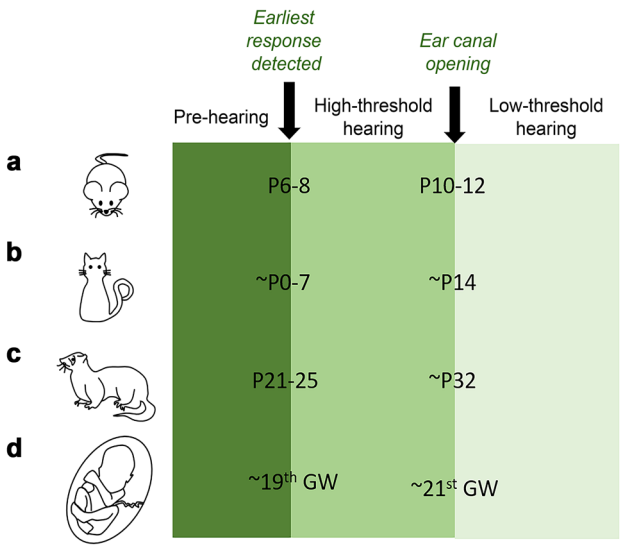

Fig. 1 Development in hearing across species. The auditory system can respond to sound (high-threshold) even before the ear canal open. The estimated timing for pre-hearing (no responses were recorded during sound-stimuli), high-threshold hearing, and low-threshold hearing is described for the mouse **a**, cat **b**, ferret **c**, and human **d**. P: postnatal day. GW, gestational week

Developmental studies measuring the auditory brainstem responses (ABR) or single-unit recordings in the ACtx suggest timing differences of the onset of hearing in several species (Mair et al. 1978; Shipley et al. 1980; Moore 1982; Walsh et al. 1986; Geal-Dor et al. 1993; Wess et al. 2017). Moreover, the presence or absence of sound-evoked responses need to be carefully evaluated as animals respond to different frequencies at distinct development stages due to filtering of sound stimuli and maturational differences across the cochlea and central structures. Several animal studies show that sound evoked neural responses are present in multiple stations along the auditory pathway recorded intracranially or with ABR before or around the opening of the ear canals (Fig. 1). For example, in cats, ABR or evoked potential responses can be recorded in the first postnatal week (ear opening ~ P14) (Pujol and Marty 1970; Konig et al. 1972; Villablanca and Olmstead 1979; Walsh et al. 1986); in ferrets, single units can be recorded in the ACtx at postnatal day (P) 21–25 (ear opening ~ P32) (Wess et al. 2017); in rat, ABR responses can be detected around P7 (Geal-Dor et al. 1993) and local field potential in ACtx can be detected around P8-10 (de Villers-Sidani et al. 2007) (ear opening P10-P12; Pujol et al. 1998; Anthwal and Thompson 2016); and in mouse, widefield and 2 photon Ca^{2+} imaging have shown ACtx responses at ~ P7 (Meng et al. 2020a, 2021). However, there are several caveats with examining sensory responses during development, and as such, these studies are likely rough estimates of when peripheral sounds drive neural activity in the auditory system. First of all, bulk measurements such as ABR recordings that are performed outside the skull depend on the synchronized firing of many neurons. Therefore, neural responses that are present but not synchronized will not be detected. Second, developing synapses can undergo synaptic depression due to limited availability of neurotransmitters or limited Ca^{2+} clearing capacity (Zucker and Regehr 2002; Oswald and Reyes 2008). Thus, sound repetition rates that are common in adult studies (e.g., 1 Hz) might lead to depression in developing synapses and will likely underestimate the responses (Wess et al. 2017). Therefore, what has typically been considered the onset of hearing in altricial animals is in fact the onset of low-threshold hearing after the opening of the ear canals while the period of high-threshold hearing with closed ear canals is reminiscent of the situation in the fetus (Fig. 1). This subtle difference is important when considering manipulations of sensory experience in altricial animals that typically start around ear opening (Zhang et al. 2001, 2002).

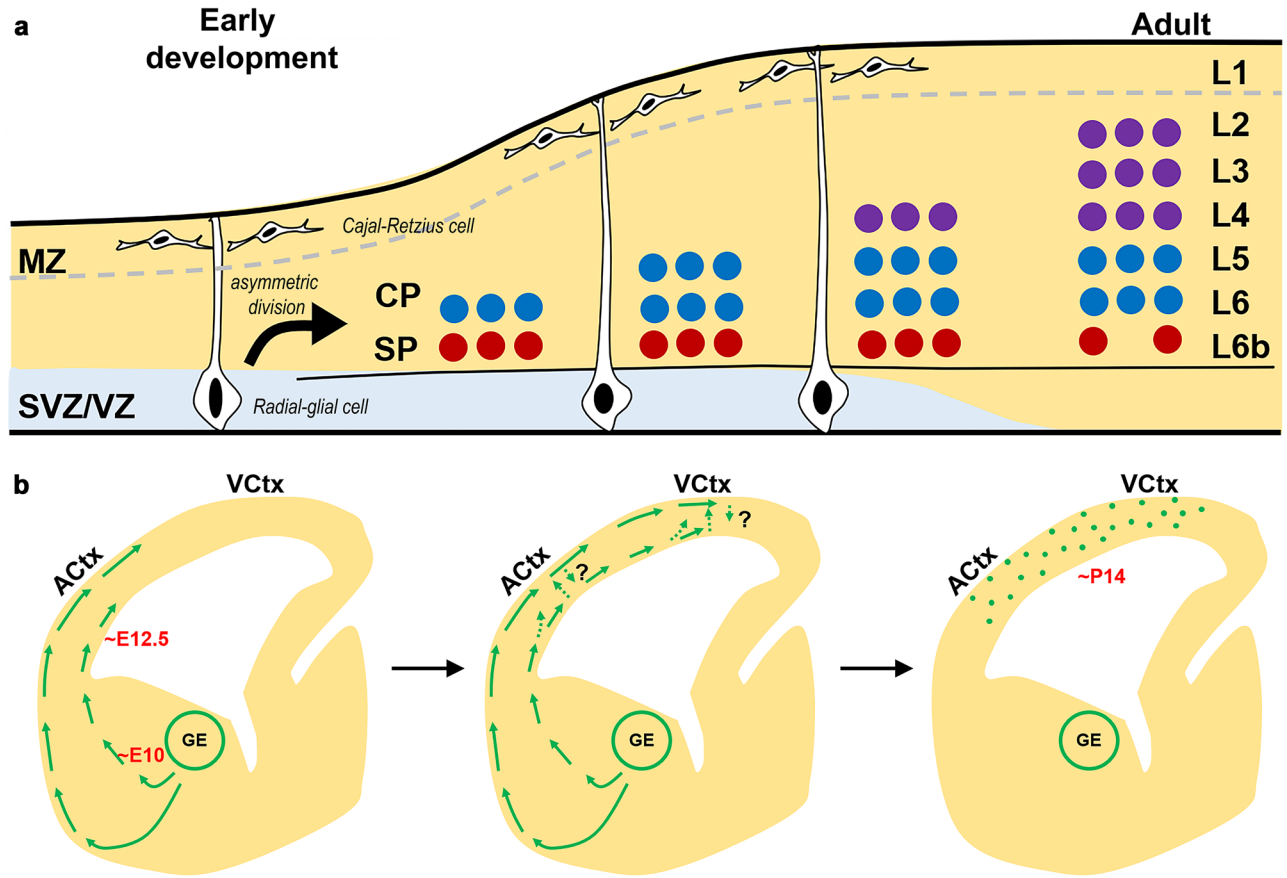

Fig. 2 Development of excitatory and inhibitory neurons during the embryonic period. **a** Cortical excitatory neurons were generated from the radial glial cells and migrate towards their final location within cortical plate (CP) guided by Cajal-Retzius cells. The first generated neurons are the subplate (SP) neurons, followed by deeper layer neurons and upper layer neurons that sequentially migrate into the CP. Cajal-Retzius cells and some subplate neurons largely disappear over development. **b** Inhibitory neurons are generated from the ganglionic eminence (GE) starting around embryonic days (E)10 and migrate tangentially to the cortex (left). The presence of inhibitory neurons in the intermediate/ventricular zone and marginal zone can be detected at the lateral region of the murine cortex as early as E12.5 (left). Some of these inhibitory neurons will continue to migrate towards the dorsomedial region of the cortex, however, whether the timing of these neurons invading the cortical plate happens concurrently is unclear (middle). Around P14, the inhibitory neurons evenly distributed within the cortex (right). ACtx, auditory cortex; L, layer; MZ, marginal zone; SVZ/VZ, subventricular zone/ventricular zone; VCtx, visual cortex

EARLY FORMATION OF AUDITORY CORTEX COMPARED WITH OTHER SENSORY CORTICAL AREAS

ACtx, like other cortical areas, consists of six laminae. Excitatory neurons are formed by sequential neurogenesis during the embryonic period, layering the cerebral mantle in an inside-out manner (Bayer et al. 1991; Polleux et al. 1997; Takahashi et al. 1999; Levers et al. 2001; Bayatti et al. 2008; Duque et al. 2016) (Fig. 2a). Although the period for neurogenesis and migration is completed at different times among different species (Table 1) (mice: Polleux et al. 1997; Takahashi et al. 1999; rat: Bayer and Altman 1991; possum: Sanderson and Aitkin 1990; ferret: Noctor et al. 1997; cat: Luskin and Shatz 1985; monkey: Rakic 1974; human: Rakic 1988; Krmpotić-nemanić et al. 1979; Kostovic and Rakic 1990), ACtx seems to achieve this earlier than the SSCtx and VCtx. In marsupials, the generation of all auditory cortical neurons is complete by P46, about three weeks earlier than neurons in the VCtx (Sanderson and Aitkin 1990). Studies in rodents during the early postnatal period using birth-dating and laminar specific gene expression indicate an earlier arrival of postmitotic neurons to their final position, and earlier maturation of the respective lamina to the adult pattern in ACtx compared with other sensory cortical areas (Ignacio et al. 1995; Chang et al. 2018). The temporal pattern differences observed from the perspective of neurogenesis and cytoarchitectural maturation may indicate that other biological events, such as dendritic and axonal

growth, synaptic formation and maturation, and circuit connectivity between cortical and subcortical regions, might also depend on specific cortical areas.

Cortical inhibitory neurons have a different ancestry from excitatory neurons. Cortical interneurons are generated in the ganglionic eminence (GE) around embryonic day (E)10 (Lavdas et al. 1999; Anderson et al. 2001; Jiménez et al. 2002; Nery et al. 2002; Bandler et al. 2017) (Fig. 2b). During the embryonic period, inhibitory neurons migrate tangentially, in a ventrolateral-to-dorsomedial manner from the GE to the cortical intermediate/ventricular zone and marginal zone (Wichterle et al. 2001; Tanaka et al. 2003; Miyoshi and Fishell 2011; Lim et al. 2018). Cortical interneurons reach the lateral part of the murine cortex by E12.5 (Fig. 2b) and migrate radially or at different angles into the cortex (Tanaka et al. 2003; Gierdalski et al. 2011). The densities of cortical interneurons continue to increase and peak around the second postnatal week, gradually decline, and reach a steady state in adulthood (Micheva and Beaulieu 1995; Schierle et al. 1997; Lee et al. 1998; Gao et al. 1999). The origin and timing of migration for different subtypes of cortical interneurons may contribute to differential distribution patterns among cortical regions (Schierle et al. 1997; Gao et al. 1999; Rudy et al. 2011; Fazzari et al. 2020). In the ACtx, different subtypes of cortical interneurons are likely to have different roles in auditory processing (further discussed below, see also Blackwell and Geffen 2017).

Circuit Development in Auditory Cortex in Comparison with Visual Cortex

A direct comparison of the developmental milestones between sensory cortices is further complicated by the fact that the time of opening of the eyes and ears differs. For example, the ear canal opening is around P9–10 (for mice), P32 (for ferret), and P14 (for cat), while eye opening is around P12–14 (mice), P30 (ferret), and P6–10 (for cat) (Pujol and Marty 1970; Bonds and Freeman 1978; Blake 1979; Moore 1982; Shen and Colonnese 2016; Anthwal and Thompson 2016; Danka Mohammed and Khalil 2020). Considering the time difference of the onset of sensory functions in different species, we shall explore the timing of cortical maturation in ACtx from a circuit perspective.

Early axonal growth from the cortical postmitotic neurons happens almost concurrently with their maturation while further cortical neurogenesis is still ongoing. During development, the continuous cortical expansion, after neurogenesis and neuronal migration, is likely correlated with synaptogenesis (Huttenlocher and Dabholkar 1997). Early studies in primates suggested that the development of proximal synapses on apical dendrites happens concurrently across layers and cortical regions (Rakic et al. 1986) while later studies that included distal synapses showed that changes of branching and spine density varied between cortical areas (Elston et al. 2009). This suggests that the initial growth and morphological maturation occur simultaneously across regions while the development of more distal inputs might vary. In humans, the thickness of the gray matter in each cortical regions increases after birth due to the expansion of layers 2–6, and then slowly thins to adult level, but the timing differs among cortical regions (Sowell et al. 2003, 2004; Raznahan et al. 2011). The temporal cortex in humans, where ACtx is located, is among those regions that continue to grow when other regions experience thinning, with the temporal cortex gray matter reaching a maximum volume at around adolescence (~ age 16–17 years) (Giedd et al. 1999). The difference in changes of cortical thickness may relate to the functional role played by the respective cortical regions (Lu et al. 2007; Squeglia et al. 2013; Kalamangalam and Ellmore 2014).

Despite the regional differences, dendritic and synaptic development among sensory cortices seems to proceed in a laminar gradient similar to most cortical regions. Studies in rat VCtx and SSCtx showed that the complexity of neuronal morphology was achieved earlier for the earlier-born deeper layer neurons than for the later-born superficial layer neurons (Juraska and Fifková 1979; Wise et al. 1979; Miller 1981). It is important to keep in mind that the dynamics of spine development differ based on their distance from the soma since both dendrites and spines that are located at the terminal remain plastic into adulthood (Petit et al. 1988). Additionally, the presynaptic inputs originating from the thalamus may affect the timing of dendritic spines maturation between cortices. Two independent studies in the rat ACtx and VCtx showed a stepwise fashion in dendritic spine development during the early postnatal period, but in a different temporal manner (Miller 1981; Schachtele et al. 2011). Initially, the spine densities at the proximal portion of apical dendrites for pyramidal neurons increase at a slow rate in the ACtx (P4–P9) and VCtx (P6–P9). The slow growth may be contributed by the cochlear spontaneous activity relayed by the thalamus (Babola et al. 2018). This initial slow increase is followed by a period of a rapid increase in both ACtx (i.e., P9–P19) and VCtx (i.e., P12–P15). The differences in the onset of the slow and rapid growth periods are likely due to the difference in the onset of low threshold sensory inputs e.g., the timing of ear (~ P9/10) and eye-opening (~ P12).

In conclusion, compared with the VCtx, ACtx shows an earlier formation of its cytoarchitectural features and formation of dendrites and synapses. However, many neurodevelopmental events in the ACtx take a longer period to complete, for instance, the continuous thickening of the gray matter in the ACtx when other cortical areas are experiencing thinning (Giedd et al. 1999). Such protracted development could make the auditory system more susceptible to insults and neuronal disorders. Indeed, there is an association of ACtx malformation and

multiple disorders (Thompson et al. 2001; Sowell et al. 2003; Holinger et al. 2005; Marco et al. 2011) (see also review: Hugdahl et al. 2008; O'Connor 2012).

DEVELOPMENT OF THALAMOCORTICAL INNERVATION AND TRANSIENT CIRCUITS

The ascending pathway from the thalamus to the cortex is pivotal for relaying sound-evoked activity from the periphery to the ACtx. Generally, there are two pathways for the thalamocortical circuits in the auditory system: the first-order and higher-order thalamocortical circuits. In the first-order thalamocortical circuit, most of the neurons in the MGBv project to A1 and AAF in a tonotopic manner (Aitkin and Webster 1972; Morel et al. 1993; Anderson et al. 2007; Hackett 2011). Higher-order thalamocortical circuits originating from MGBd and MGBm have projections to distinct laminae in all auditory cortical areas and process tonotopic, non-tonotopic, and multisensory information (Aitkin 1973; Lee 2015; Smith et al. 2019).

Development of Thalamocortical Projections into Auditory Cortex

Similar to the neurogenetic gradient between regions, the development of thalamocortical and corticothalamic projections between the thalamus and its respective cortical regions occurs in a temporal gradient (McConnell et al.

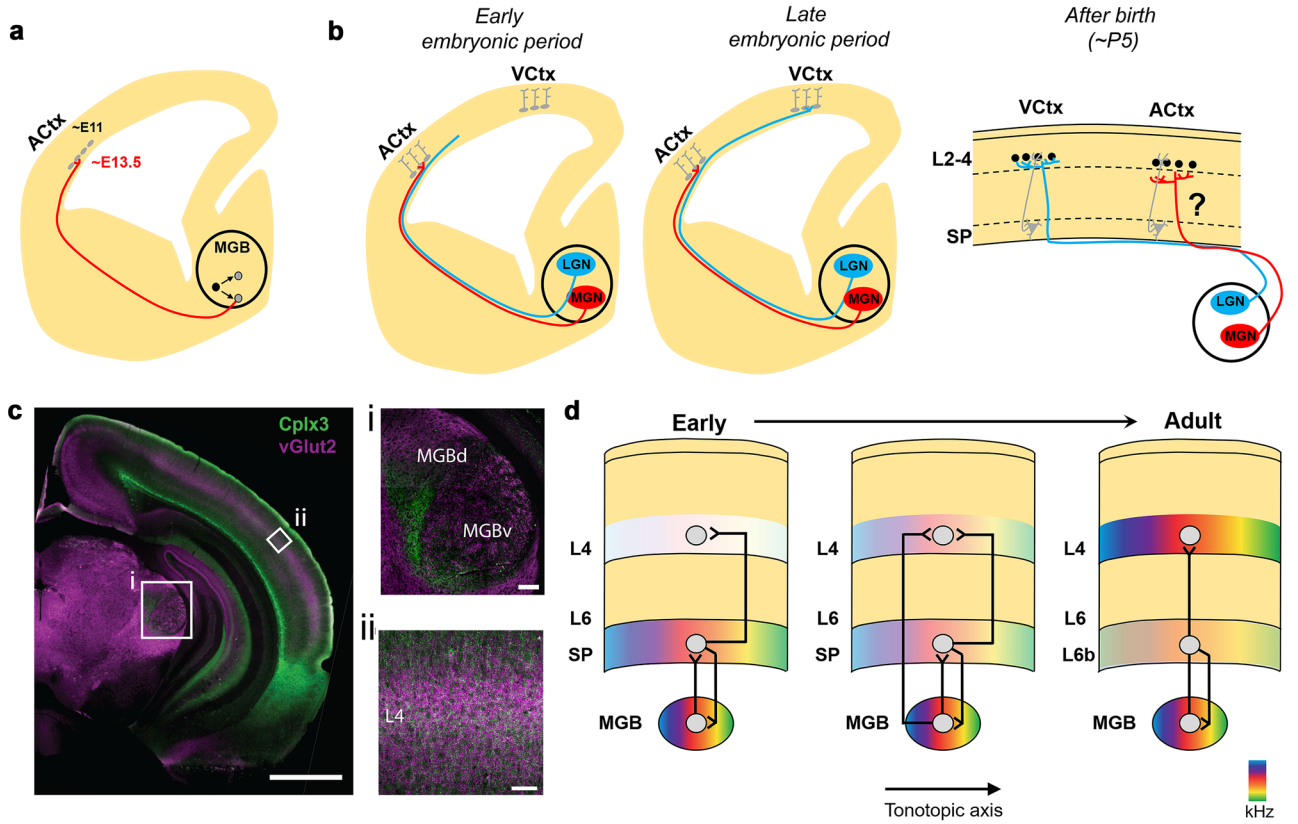

Fig. 3 Transient circuits between subplate neurons and thalamocortical axons in auditory cortex. **a** The first generated neurons are the subplate neurons (SP, gray). These neurons can be detected as early as E11 in the auditory cortex (ACtx), almost similar timing as the thalamic nuclei that are generated in the medial geniculate body (MGB) around E10. The thalamocortical axons from the thalamus contact subplate neurons in ACtx around E13.5. **b** Thalamocortical axons from the medial geniculate nucleus (MGN) arrive in the SP of ACtx (red) earlier than those from the lateral geniculate nucleus (LGN) in the SP of visual cortex (VCtx, blue). Around postnatal days (P) 5, the thalamocortical fibers arrived in the VCtx layer (L) 4, earlier than those in ACtx. **c** SP neurons project to thalamorecipient L4 and L1 as well as to MGB during early postnatal ages. Complexin 3 (Cplx3, green) is expressed in SP neurons and strong puncta immunolabeling can be detected at (i) the thalamus surrounding the ventral division of MGN (MGBv), and in (ii) the L4 and L1. Vesicular glutamate transporter 2 (vGlut2, magenta) labelling thalamocortical fibers and thalamorecipient L4. **d** A transient circuit is formed between the SP and MGB during the early embryonic period, and the SP neurons were projecting to the future L4 neurons (left). During the development, the TCAs from MGB will penetrate the cortex and move towards the L4 neurons (middle). In the adult, when the connections between MGB and L4 are established, the subplate network diminished (right). During this process, SP might be considered a proto-organizational structure, ensuring that L4 is organized in a tonotopic manner. Pseudo-colored represents different frequencies in the tonotopic map. MGBd, dorsal division of MGN; scale bar for **c** is 1 mm; scale bar for (i, ii) is 50 μm

1989, 1994; Ghosh and Shatz 1992b). Subplate neurons are among the first population of postmitotic cortical neurons and are the first cortical target of thalamocortical projections (Kostovic and Rakic 1990; Kanold and Luhmann 2010). Axons from MGB arrive in the subplate region of the ACtx earlier than the lateral geniculate nucleus (LGN) axons arrive in the VCtx subplate (Fig. 3). While the cause of these differences in timing is unknown, differences in peripheral activity or genetic programs or both might play a role in that. In rats, neurogenesis in the MGB begins around E10–E15 (Altman and Bayer 1979, 1989; Gurung and Fritzsch 2004), slightly earlier than the generation of subplate neurons (Valverde et al. 1995)(Fig. 3a). Thalamocortical axons (TCAs) exit the thalamus guided by a cocktail of axon guidance molecules (Slit1, Netrin1, Ephrin-A5, Sema3A/3F) to follow a path towards the cortex, avoiding the hypothalamus, arrive at their respective subplate zones, and wait before they penetrate into the cortex (Leyva-Díaz and López-Bendito 2013; López-Bendito 2018). The presence of TCAs in the ACtx in mice can be detected around E13.5 (Fig. 3a) when projections from the inferior colliculus have not fully innervated the MGBv (Gurung and Fritzsch 2004), suggesting that the topographic axonal pathfinding from the MGBv to ACtx may not require ascending inputs but is regulated by intrinsic programs within the thalamus and/or the cortex. Spontaneous activity in the thalamic neurons regulates the expression of transcription factors, Robo1 and DCC, that switch the rate of extension of TCAs from fast to slow when they enter the neocortex, possibly interacting with the local environment before growing into the future layer (L) 4 (Mire et al. 2012; Castillo-Paterna et al. 2015).

Transient Circuit Between Thalamocortical Projections and Subplate Layer

When thalamic axons enter the cortex, they encounter the earliest generated cortical neurons, the subplate neurons, which in rodents are born at ~ E11 (Kostovic and Molliver 1974; Al-Ghoul and Miller 1989; Wood et al. 1992; Price et al. 1997; Zeng et al. 2009; Kanold and Luhmann 2010). Together with Cajal-Retzius neurons of the marginal zone, subplate neurons form transient circuits that are present mostly during development (Kostovic and Rakic 1984, 1990; Luskin and Shatz 1985; Kanold and Luhmann 2010; Molnár et al. 2020). Thalamocortical inputs accumulate in the subplate (Fig. 3b), forming their first synaptic contacts with subplate neurons (Friauf et al. 1990; Zhao et al. 2009). Consistent with this early ingrowth of thalamocortical fibers, in vivo recordings have shown that subplate neurons respond to sound (Wess et al. 2017). In rodents, the subplate neurons already receive thalamocortical inputs at birth and are capable of firing action potentials, with repetitive firing developing within the first postnatal week (Zhao et al. 2009). Furthermore, these subplate neurons also receive inputs from the developing cortical plate (cortical layers 2 to 6 that is still undergoing maturation), especially from future L4 (Viswanathan et al. 2012; Meng et al. 2014). Subplate axons target L4 as well as L1 (Viswanathan et al. 2017; Meng et al. 2020b), thus forming a feedforward and feedback circuit within the subplate and neurons located within the developing cortical plate.

The subplate consists of a heterogeneous group of neurons expressing specific molecular markers, e.g., complexin 3 (Cplx3), connective tissue growth factor (CTGF), dopamine receptor D1 (Drd1), orphan nuclear receptor Nr4a2 (Nurr1), and G-protein-coupled lysophosphatidic acid receptor 1 (Lpar1/Edg2) (Hoerder-Suabedissen and Molnár 2013; Viswanathan et al. 2012, 2017). Because of its presynaptic localization and specificity, Cplx3 allows the identification of subplate axon terminals (Viswanathan et al. 2017). The puncta-like immunoreactive staining is denser at the thalamorecipient layer and L1 of ACtx (Fig. 3c). Also, the puncta-like staining of Cplx3 is mainly detected in the MGBd and MGBm (Fig. 3c), suggesting that subplate neurons in the ACtx send their axons to higher-order thalamus, in agreement with studies in the somatosensory system (Viswanathan et al. 2017; Hoerder-Suabedissen et al. 2018). As the cortex matures to adult, a fraction of subplate neurons survive as L6b neurons (Kostovic and Rakic 1980; Valverde and Facal-Valverde 1988; Clancy and Cauller 1999; Marx and Feldmeyer 2013; Marx et al. 2017). While previously it has been thought that thalamic axons "wait" in the subplate (Kostovic and Rakic 1990), recent work has shown that this period is highly dynamic and involves the organization of thalamic afferents into topographic maps (Wess et al. 2017). Thus, this period might be considered a proto-organizational period (Fig. 3d). As detailed below, manipulating cochlear function or auditory experience during this early period can change circuits to subplate neurons indicating the dynamic nature of this period (Meng et al. 2021). In short, subplate neurons are in a central position to orchestrate early cortical activity, guide thalamic afferents, and play an early key role in establishing topographic maps and functional circuits.

Thalamocortical Development into Layer 4 of the Primary Auditory Cortex

In early development (before ~ P11 in mouse ACtx and ~ E24 in cat VCtx), TCAs project to subplate neurons, which in turn target the L4 neurons, acting as a "relay hub" (Friauf et al. 1990; Zhao et al. 2009; Kanold and Luhmann 2010)(Fig. 3d). Thus, thalamic activation results in short-latency subplate responses while L4 responds with longer latencies (Friauf et al. 1990; Higashi 2005; Barkat et al. 2011; Wess et al. 2017). When TCAs start innervating the L4 neurons, the functional connection between subplate neurons and the L4 neurons disappears, and L4 responds with short latency to thalamic activation (Friauf et al. 1990; Barkat et al. 2011; Wess et al. 2017).

The presence of subplate neurons is crucial for TCAs to find their appropriate target and also for establishing the topographic and functional properties in L4 (Ghosh et al. 1990; Ghosh and Shatz 1992a; Kanold et al. 2003; Tolner et al. 2012). Ablation studies in the VCtx and SSCtx have shown that subplate neurons are required for the formation of ocular dominance columns in VCtx and the barrels in SSCtx (Ghosh and Shatz 1992a; Kanold et al. 2003; Tolner et al. 2012). Subplate neurons also play a role in the functional maturation of thalamocortical and inhibitory intracortical circuits (Kanold et al. 2003; Kanold and Shatz 2006; Tolner et al. 2012).

When TCAs arrive at the subplate zone, they accumulate for a period (depending on the species) before penetrating into the cortical plate (Table 1). While TCAs from the MGB arrive in the ACtx subplate earlier than those from the LGN in VCtx subplate (McConnell et al. 1989, 1994; Ghosh and Shatz 1992b), Cux1-expressing L4 neurons in VCtx and SSCtx are innervated by TCAs earlier than those in ACtx (Chang et al. 2018). This suggests that TCAs remain in the subplate for a longer duration in ACtx than in the SSCtx and VCtx (Fig. 3b). The reason for this difference and the processes occurring during this time remain to be elucidated.

Thalamocortical Circuit in the Secondary Auditory Cortex

While much is known in the thalamocortical projections from MGBv, little is known about the potential differences between the developments of first- and higher-order thalamocortical circuits during early development. In adult animals, the thalamocortical afferents in MGBd project to L4 of the secondary auditory cortex (A2), while those in MGBm project broadly across many tonotopic, non-tonotopic, multimodal, amygdala, and limbic cortical areas (LeDoux et al. 1991; Huang and Winer 2000; Lee and Winer 2008; Smith et al. 2012; Bartlett 2013; Lee 2015). A study in thalamocortical slices suggested that the first- and higher-order thalamic nuclei share similar synaptic properties (Lee and Sherman 2008). In addition to receiving corticothalamic feedback from A2, MGBd also receives feedback from A1 L5 neurons and the convergent information is passed to L4 in A2 (Huang and Winer 2000; Lee and Sherman 2008; Smith et al. 2012; Petrof et al. 2012). Based on these anatomical studies, it was proposed that there are two distinct pathways conveying information from A1 to A2, the transthalamic cortical pathway (also known as corticothalamocortical pathway, A1 to MGBd to A2), and the direct corticocortical pathway (A1 to A2) (Guillery 1995; Sherman and Guillery 2002; Sherman 2016). The timing of transthalamic circuit establishment during the early stage of development is unknown.

Anatomical studies in gerbils revealed that circuits from higher-order thalamic nuclei tend to develop at earlier ages (Henschke et al. 2018). Because the proportion of labeled fibers from MGBd to A1 is larger than those from MGBv in early development, it is possible that at this stage of development, more connections between MGBd TCAs and subplate neurons are formed (Fig. 4a). In both the auditory and the somatosensory systems, higher-order thalamic systems have been associated with more dynamic stimuli, while static stimuli drive first-order thalamic circuits (Yu et al. 2006; Liu et al. 2019). Such circuits would only show transient activation, which is compatible with transmission via immature synapses that show synaptic depression and thus compatible with earlier functional development. Given the early cortical ingrowth of MGBd fibers in A1, the subplate neurons in A1 might also receive information from MGBd earlier. However, future investigation is needed to detail the development and relative functional connectivity of lemniscal and higher-order thalamic projections.

DEVELOPMENT OF L4 MORPHOLOGY AND INTRACORTICAL CONNECTIVITY

ACtx cell populations within the cortex can be classified by the laminar position as well as by molecular identity and connectivity patterns (Budinger and Kanold 2018). Adult ACtx shows morphological differences from other cortical areas, possibly due to a distinct developmental trajectory. In rodents, L4 neurons in ACtx show larger soma size compared with those in SSCtx and VCtx (Chang and Kawai 2018), suggesting that most of the ACtx L4 neurons are pyramidal neurons. Also, unlike SSCtx and VCtx (Lund 1973; Jones 1975; Braak 1976; Parnavelas and Uylings 1980; Meyer and Ferres-Torres 1984; Simons and Woolsey 1984; Callaway and Borrell 2011), spiny stellate neurons are rare or almost absent in ACtx (Meyer et al. 1989; Fitzpatrick and Henson 1994; Smith and Populin 2001). In contrast to pyramidal cells, spiny stellate neurons have a short apical dendrite, receive thalamocortical inputs, and are involved only in very local networks (Schubert et al. 2003; Staiger et al. 2004; Callaway and Borrell 2011). Since almost all neurons in L4 of ACtx are pyramidal-like, they not only are involved in local networking within the lamina but also receive direct feedback from the upper layers (Barbour and Callaway 2008; Kratz and Manis 2015).

Why are there no spiny stellate neurons in ACtx? The expression of laminar-specific genes (e.g., ROR beta) can be detected in L4 of both primary VCtx (V1) and A1 (Hirokawa et al. 2008), suggesting that L4 of V1 and A1 shares genetic similarities. Intriguingly, most V1 L4 neurons initially show pyramidal morphology transitioning to spiny stellate morphology with the onset of visual activity (Callaway and Borrell 2011). This suggests that the lack of spiny stellate cells in ACtx might be due to different activity patterns during development. Similar to other sensory cortical areas, the main ascending

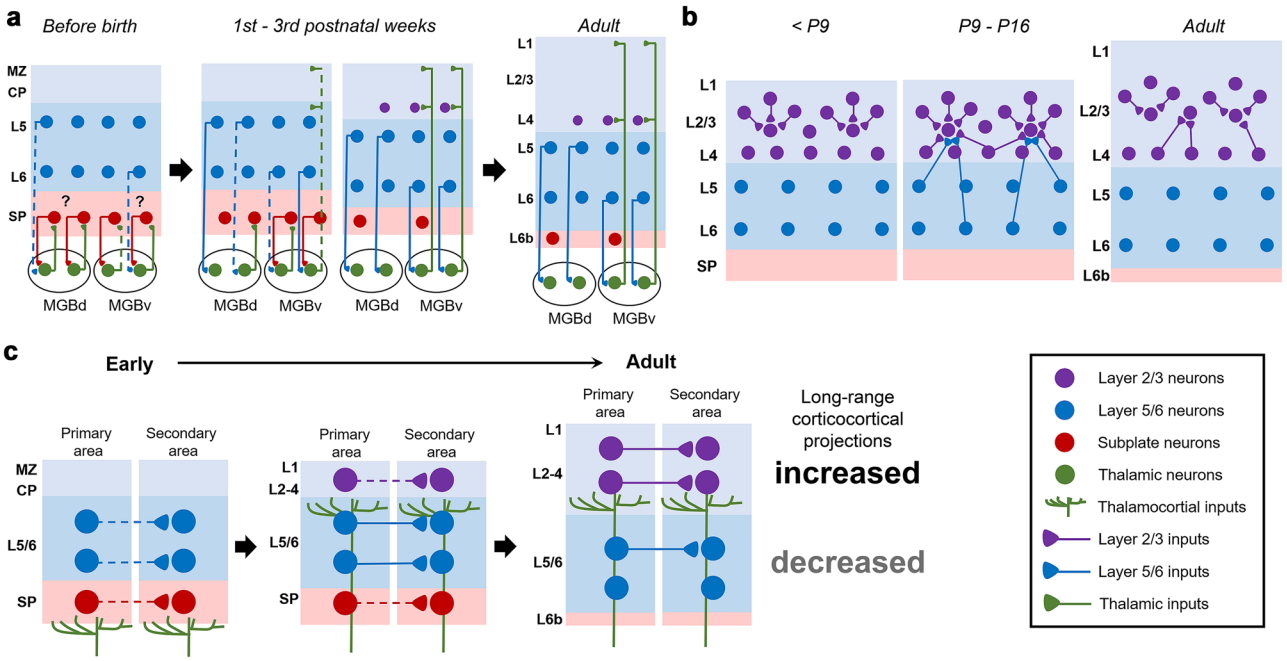

Fig. 4 Schematic figure of connections in primary auditory cortical development. Ages refer to mice. **a** Projections from the medial geniculate body (MGB) arrive in the subplate layers of primary auditory cortex (A1) during embryonic development. After birth, the thalamocortical axons from both MGBd and MGBv refine and terminate into their appropriate target and some SP neurons start to disappear. **b** In the first postnatal week, L2/3 neurons (purple) establish intracortical networks with their surrounding neurons. Besides ascending L4 inputs, between P9 and P16, L2/3 neurons also receive extensive inputs from L5/6 neurons. Such connections disappear in adulthood. **c** During early development, long-range corticocortical connections between primary and secondary areas are established mainly by the lower layer and possibly subplate neurons (left). As the cortex matures, the upper layer neurons form long-range corticocortical connections between primary and secondary cortical areas (middle). As the thalamocortical inputs innervate layer 4 and during maturation of upper layer neurons, the corticocortical connections in the lower layers decrease. MZ, marginal zone; CP, cortical plate

auditory input to ACtx is provided by TCAs (Fig. 4a) which target most layers but most strongly innervates the lower portion of L3b and L4 (Hackett et al. 2001, 2016; Ji et al. 2016; Vasquez-Lopez et al. 2017; Chang and Kawai 2018). Studies in VCtx and SSCtx demonstrated that lemniscal TCAs, and possibly the activity patterns relayed by these axons, are important for the specification of these primary sensory regions (Vue et al. 2013; Chou et al. 2013; Pouchelon et al. 2014). Elimination of the lemniscal TCAs from the ventrobasal thalamus to the primary SSCtx prevented the formation of cortical barrels, leaving the cortex morphologically similar to the secondary SSCtx (Li et al. 2013; Pouchelon et al. 2014). While spiny stellate neurons are usually located near the border of a barrel (Egger et al. 2008), elimination of the TCAs caused retention of the apical dendrite by L4 neurons and lack of spiny stellate morphology (Li et al. 2013). Thus, the absence of spiny stellate cells in A1 may indicate that there are differences in the developmental activity patterns between A1 and other primary sensory cortical areas leading to different morphologies of L4 cells. Alternatively, the late innervation of A1 by TCAs might occur after the period for the possible transformation from pyramids to spiny stellate morphology, thus preventing the transformation of these neurons.

INTRACORTICAL CONNECTIVITY IN THE UPPER LAYERS

Intracortical circuits are highly dynamic during the early developmental period (Fig. 4b). Functionally, adult A1 L2/3 cells can be subdivided into classes based on their local connectivity patterns within A1 (Meng et al. 2017b). In rodents, during the first postnatal week and before the arrival of TCAs, L2/3 neurons receive strong intracortical inputs from within L2/3 (Meng et al. 2020a) (Fig. 4b). During the second postnatal week, these neurons also receive extensive inputs from lower layer neurons including L5/6 (Fig. 4b). Refinement of these intracortical inputs occurs within the second and third postnatal weeks combined with an increase in functional

circuit heterogeneity (Meng et al. 2017b, 2020a). Besides different inputs, some of these L2/3 neurons have long-range axonal projections targeting higher or contralateral cortical areas (Winguth and Winer 1986; Petrof et al. 2012). At least in V1, the development of long-range corticocortical projections follows the neurogenesis gradient, where the deeper layers' neurons project towards the secondary visual cortex before the upper layers' neurons establish their connectivity (Coogan and Burkhalter 1988; Burkhalter 1993; Price et al. 1994). However, this long-range corticocortical circuitry formed by the deeper layers is transient and declines slowly as the connectivity in the upper layer neurons is established (Fig. 4c). In the visual system, sensory deprivation prevents the normal pruning of the corticocortical connections in the deeper layers between primary and secondary VCtx that is usually observed in adulthood (Price et al. 1994). The existence of the transient intracortical or deep intercortical corticocortical circuits might be activity-dependent, where the elimination is followed by the maturation in the upper layers' corticocortical circuitry and innervation of thalamocortical fibers to L4.

CORTICAL AND SUBCORTICAL CONNECTIVITY IN THE LOWER LAYERS

In adults, neurons located in the deeper layers, e.g., L5/6, project mainly to subcortical structures and thus contribute to feedback circuits (Budinger and Kanold 2018). During the critical period, these deep neurons also project across the cortical column to cells in L1 and L2/3 (Meng et al. 2020a, b). The laminae across different cortical regions share similar gene expression, and some of these genes regulate the fate of lower layer neurons for appropriate projections (Molyneaux et al. 2007; Kwan et al. 2012). Transcription factors such as Tbr1, Foxp2, and Ntsr1 are expressed in corticothalamic neurons, and a large population of L6 neurons expresses one or more of these transcription factors (Hisaoka et al. 2010; Sundberg et al. 2018; Chang et al. 2018). In L6, the presence of Tbr1 expression is crucial to maintain the corticothalamic identity of these neurons and for their axons to further extend to subcortical targets, while L5 neurons require the expression of Fezf2 to suppress Tbr1 to maintain their identity (McKenna et al. 2011; Han et al. 2011; Canovas et al. 2015; Darbandi et al. 2018). Foxp2 is a unique transcription factor. Since its discovery in a British family with hereditary speech and language disorders, it is the first identified gene related to speech and language (Vargha-Khadem et al. 1995; Hurst et al. 2010). Multiple studies indicated that Foxp2 plays a role in cortical neurogenesis (Tsui et al. 2013), thalamic patterning (Ebisu et al. 2017), neurite growth, and axon guidance (Vernes et al. 2011). However, Foxp2 mutant mice do not exhibit ectopic projection in L6 neurons and the postnatal cytoarchitecture remains unaffected possibly due to compensatory effects (Kast et al. 2019).

In rats, L6 corticothalamic neurons of VCtx and SSCtx extend their axons towards the internal capsule at around E12 before arriving at the thalamus (Miller et al. 1993; Deck et al. 2013; Briggs 2010; Grant et al. 2012). Studies of early postnatal development of corticothalamic projections from ACtx are limited. Generally, corticothalamic fibers have already innervated the thalamus around birth. However, the completion of these innervations takes place at different time points, with innervation of the ventrobasal complex first, followed by innervation of the LGN, and finally the innervation of the MGB (Jacobs et al. 2007). The innervation of corticothalamic fibers is complete in MGB about 4 days earlier than in the LGN (i.e., P14.5), and this timing is consistent with the developmental pattern observed in the L6 labeled by Foxp2-expressing neurons where ACtx reaches mature cell densities earlier than the VCtx (Chang et al. 2018). This indicates a delayed but more rapid development of corticothalamic innervation in the auditory system. Additionally, during early postnatal development, the connectivity between MGBv, L6, and L4 is mainly coordinated by the subplate, before the establishment of connectivity from MGBv to L4, L6 to L4, and L6 to MGBv (Kanold et al. 2019) (Fig. 4a). How these developmental changes in circuits take place and are controlled during development is yet to be carefully examined.

L5 is formed by a heterogeneous group of cells which can be categorized by distinct neuronal morphology, electrophysiology properties, the target of their axonal projections, and gene expression (Molnár and Cheung 2006). In general, L5 in primary sensory cortical areas comprises corticocortical and subcortical projection neurons in 2 sublaminae (Games and Winer 1988; Petrof et al. 2012). L5 in A1 can be separated in 3 sublaminae based on their target projection towards the inferior colliculus, superior olivary complex, and cochlear nucleus (Doucet et al. 2003) or into 3 sublayers based on genetic identity (Chang and Kawai 2018). In the latter case, corticocollicular, Ctip2-expressing neurons are mainly populated in the middle layer, with corticocortical neurons sparsely distributed in L5 between Cux1-labeled L2-4 and Foxp2-labeled L6 (Chang and Kawai 2018).

Although the connectivity of L5 neurons with different target areas has been well-studied in the adult ACtx, processes during early postnatal development remain to be elucidated. In the somatosensory system of mice, the axonal fibers of L5 corticofugal neurons can be found in their subcortical targets at birth (McKenna et al. 2011), while those from corticostriatal neurons can be detected in the ipsilateral and contralateral striata and the contralateral cortex around P3 (Sohur et al. 2014). L5 projections continue to refine by regulating specific epigenetic modifications during early postnatal stages (Harb et al. 2016). In the ACtx, injection of retrograde tracers into

inferior colliculus at P5 was able to label corticocollicular L5 neurons (Chang et al. 2018), suggesting that the connectivity was established before that period of time. L5 in A1 also contains corticocortical neurons with their axons projecting to higher-order areas of the auditory (Fig. 4c), V1, S1, and frontal cortical regions (Covic and Sherman 2011; Petrof et al. 2012; Kim et al. 2015; Zhang et al. 2016; Masse et al. 2019). But when these projections mature or if there is a critical period for their development is unclear. In the VCtx, corticocortical neurons in the lower layers initially project to secondary areas, but this connectivity between cortical areas is replaced by the upper layer neurons during postnatal development (Price et al. 1994). Does a similar process happen in ACtx and does this process require auditory experience? Understanding these processes will help further elucidate how larger-scale areal connections are established during the early postnatal stage.

DEVELOPMENT OF INHIBITORY CIRCUITS

Inhibition is crucial in auditory processing, and there are many subtypes of cortical GABAergic interneurons (Rudy et al. 2011). In the ACtx, the distribution of different subtypes of cortical interneurons follows a distinct pattern from early postnatal period to adulthood (Ouellet and de Villers-Sidani 2014). We here focus on three major groups of GABAergic neurons: the parvalbumin (PV), somatostatin (SST), and ionotropic serotonin receptor 5HT3a (5HT3aR) neurons.

Developmental Changes of GABAergic Neurons in Layers 2–4

Around the first postnatal week in the ACtx of rats, the proportion of PV-immunopositive (PV+) neurons is higher than SST+ neurons (Rudy et al. 2011; Ouellet and de Villers-Sidani 2014). Both subtypes have a higher proportion localized in L4 compared with other laminae of ACtx (Ouellet and de Villers-Sidani 2014). The maturation of PV and SST neurons in L4 is crucial for maintaining the normal circuitry between cortical excitatory and inhibitory neurons. In the SSCtx, the early-generated SST interneurons are mainly located in the infragranular layers and are transiently innervated by TCAs during the first postnatal weeks (Marques-Smith et al. 2016; Tuncdemir et al. 2016; Anastasiades et al. 2016). The transient innervation of TCAs on SST plays a crucial role for the maturation of cortical inhibitory circuits and affects the maturation of PV interneurons and their inputs on pyramidal neurons (Tuncdemir et al. 2016). In addition, the early-generated SST interneurons are found to be crucial for synchronizing spontaneous networking during early postnatal stages, as they form a higher synaptic connection with their surrounding pyramidal neurons (Wang et al. 2019). In the SSCtx, the presence of SST mRNA expression can be detected as early as P0, while PV mRNA expression was present much later (Mukhopadhyay et al. 2009). Unlike the SSCtx, the PV+ neurons are present much earlier than SST+ neurons in the ACtx (Mukhopadhyay et al. 2009; Ouellet and de Villers-Sidani 2014; Liguz-Lecznar et al. 2016), suggesting that SST and PV play different developmental roles in auditory circuit formation.

PV neurons have fast-spiking (FS) properties, while most of the SST neurons have low-threshold spiking (LTS) properties (Kawaguchi 1993; Galarreta and Hestrin 2002; Cardin et al. 2009; Rudy et al. 2011; Hu et al. 2014; Liguz-Lecznar et al. 2016). Studies in the ACtx of gerbil suggested that the development of FS and spiking LTS neurons in the upper layers are sensory-dependent (Kotak et al. 2008, 2013; Takesian et al. 2012, 2013). Following ear opening, FS neurons receive increased thalamic inputs and therefore relay stronger inhibition onto the pyramidal neurons in the upper layers. In contrast, the amplitude of LTS-evoked inhibitory postsynaptic spontaneous currents (LTS-IPSCs) experienced a developmental decrease, and the inhibitory short-term plasticity of LTS showed a switch from depression to facilitation (Takesian et al. 2010, 2013). GABAergic interneurons also receive intracortical inputs, and L4 GABAergic neurons mainly receive local inputs before ear opening (i.e., during the first postnatal week), with little input coming from lower layers (Deng et al. 2017). However, after ear opening, the inputs from lower layers on these GABAergic neurons increase; hence, these GABAergic cells integrate information from almost all layers. Neonatal deafening altered the pattern of intracortical inputs (Deng et al. 2017). Thus, the maturation of GABAergic neurons in all layers seems to depend on sensory-experience and centrally as well as peripherally generated spontaneous activity.

GABAergic Neurons in Layer 1

Layer 1 (L1) in development (also known as marginal zone) contains mainly early generated Cajal-Retzius cells and sparsely distributed GABAergic neurons (Winer and Larue 1989; Prieto et al. 1994). The Cajal-Retzius cells slowly disappear in early postnatal period, leaving the GABAergic neurons (Gesuita and Karayannis 2021). The majority of the L1 GABAergic neurons can be grouped based on the neurochemical marker reelin, GABA-A receptor subunit delta, neuropeptide Y (NPY), vasoactive intestinal peptide (VIP), calretinin, and somatostatin (Ma et al. 2014). L1 neurons can also be classified into four unique groups based on their molecular profiles, morphologies, and electrophysiological properties: neurogliaform cells, canopy cells, α7-nicotinic acetylcholine receptors (α7-nAChR) expressing cells, and VIP cells (Schuman et al. 2019). Both neurogliaform and canopy cells express neuron-derived neurotrophic factor (NDNF) (Schuman et al. 2019).

During the early postnatal period, L1 GABAergic neurons integrate interlaminar inputs from L4 and subgranular layers (Meng et al. 2020b). This period of integration spans from ~ P5 to ~ P16, peaks at P10–P16, and encompasses the critical period, a period where the developing cortex can be altered profoundly or permanently by deprivation of auditory inputs. After this period, most L1 inputs originate from superficial layers (Meng et al. 2020b). L1 5HT3aR neurons, but not VIP neurons, are thought to be involved in the development of the tonotopic organization during the critical period (Takesian et al. 2018). Only 5HT3aR non-LTS neurons are connected to L4 PV cells, and such events involve nicotinic receptors recruitment. Thus, these neurons likely are non-LTS canopy cells, α7-nAChR-expressing neurons, or reelin-expressing neurons (Lee et al. 2010; Rudy et al. 2011; Schuman et al. 2019). Overall, the developmental changes in different subtypes of inhibitory interneurons may represent their distinct functional roles in circuit maturation during early postnatal development.

THE ROLE OF SENSORY EXPERIENCE AND DEAFNESS ON THALAMIC AND CORTICAL DEVELOPMENT

Neural activity within and among auditory fields is pivotal for shaping brain circuits, and much work has been focused to elucidate the effect of early sensory experience, or the lack thereof, on cortical processing. However, neural activity is present in the auditory system even before the onset of sensory transduction in the cochlea as the cochlea itself produces spontaneous activity patterns that propagate to ACtx (Tritsch et al. 2007; Babola et al. 2018). The role of early spontaneous activity on ACtx is unknown, but altering this activity disrupts the proper establishment of topographic projections in the auditory brainstem (Kandler et al. 2009), suggesting that it may play a key role in sculpting nascent auditory cortical circuits.

Timing Matters: The Effect of Sensory Deprivations on Thalamic Inputs to A1

Congenitally deaf cats have been a useful model to study the effects of auditory deprivation (Ryugo and Menotti-Raymond 2012). Experiments in congenitally deaf cats have shown that experience is important for the development of normal feedforward and feedback circuitry among different auditory cortical areas (Fig. 5). Cat ACtx can be divided into A1, A2, AAF, posterior auditory field (PAF), and dorsal zone (DZ) (different species may have different subregions; see Hackett (2011, 2015)). In congenitally deaf cats, a decrease in cortical thickness due to the thinning of layers 4–6 is observed in different auditory cortical regions (Berger et al. 2017). Meanwhile, in early- and late-onset deafness (induced by ototoxicity), the lamina cytoarchitecture and the total volume for auditory cortical regions are no different than those in hearing cats. The fractional volume, however, shows a decrease in A1 but an increase in A2 in both deafness models, indicating remodeling of auditory cortical areas (Wong et al. 2014). The differences between the studies are likely due to the different initiation of the auditory sensory deprivation and potential effects on spontaneous cochlear activity (Fig. 5). Anatomical studies with tracers in the different deafness models support this idea (Barone et al. 2013; Chabot et al. 2015; Butler et al. 2016, 2017; Kok and

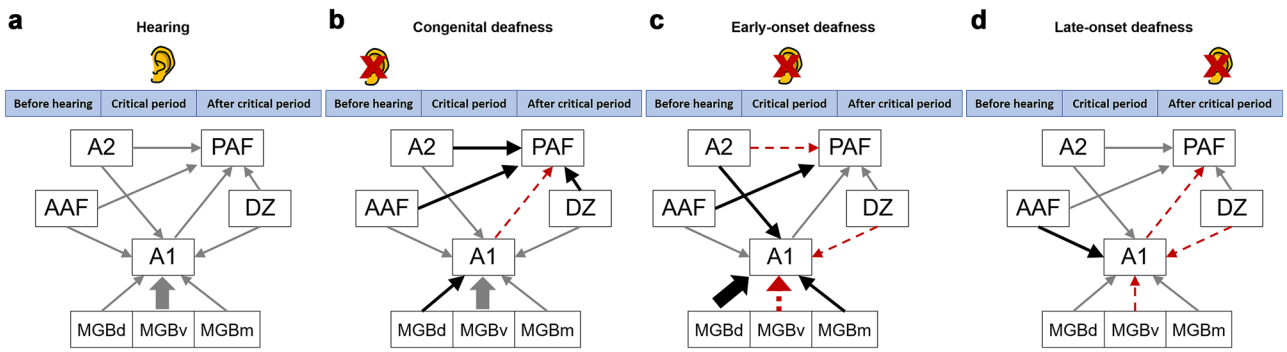

Fig. 5 The connection among thalamus, primary and secondary auditory areas in cat. **a** In normal hearing, the primary auditory cortex (A1) received inputs from all three divisions of the medial geniculate body (MGB), as well as from other secondary auditory fields. **b** In congenital deafness, an increase of ectopic connections can be detected (black arrow), while a decline of intra-areal connectivity is present (dotted red line). **c, d** Early-onset deafness, defined as when the hearing was eliminated near ear canal opening during the critical period, and late-onset deafness defined as the elimination of auditory stimuli after critical period showed different changes in the connections among the thalamus, A1 and other higher-order of auditory fields. **a–d** Gray: normal connection; Black arrow: increased connection; Red dotted arrow: decreased connection; Bolder arrow: larger changes compared with other conditions. MGBv, ventral division of MGB; MGBd, dorsal division of MGB; MGBm, medial division of MGB; A2, secondary auditory cortex; AAF, anterior auditory field; PAF, posterior auditory field; DZ, dorsal zone

TABLE 1

Summary of early cortical development and sensory stimulation among different species

Species	*Mouse*	*Rat*	*Ferret*	*Cat*	*Monkey*	*Human gestation*
Gestation	19 d	21 d	41 d	65 d	167 d	40 GW
SP generation						
General	E11 (Price et al. 1997)	E12 (Al-Ghoul and Miller 1989)				5–6 GW (Kostovic and Rakic 1980)
Auditory cortex	E12 (Zeng et al. 2009)	E14–E15 (Bayer and Altman 1991)				12 GW ~ (Krmpotić-nemanić et al. 1979)
Visual cortex	E12 (Wood et al. 1992)	E14–E15 (Bayer and Altman 1991)	E20–E26 (Noctor et al. 1997)	E24–E30 (Luskin and Shatz 1985)	E43–E45 (Kostovic and Rakic 1980)	13 GW ~ (Kostovic and Rakic 1990)
Somatosensory cortex		E14–E15 (Bayer and Altman 1991)	E22–E26 (Noctor et al. 1997)		E38–E43 (Kostovic and Rakic 1980)	12 GW ~ (Kostovic and Rakic 1990)
Neurogenesis						
General	E11 (Levers et al. 2001)	E12 (Valverde et al. 1995)			E40 ~ (Duque et al. 2016)	8–9 GW (Kostovic and Rakic 1990; Bayatti et al. 2008
Auditory cortex		E13–E20 (Bayer and Altman 1991)				
Visual cortex	E13.5–E18.5 (Polleux et al. 1997)	E14–E21 (Bayer and Altman 1991)	E28–P10 (Noctor et al. 1997)	E31–E57 (Luskin and Shatz 1985)	E45–E102 (Rakic 1974)	
Somatosensory cortex	E12.5–E18.5 (Polleux et al. 1997)	E13–E18 (Bayer and Altman 1991)	E28–P2 (Noctor et al. 1997)			

The timing of subplate (SP) generation and neurogenesis in different sensory cortices across different species, *d* days, *E* embryonic days, *GW* gestational week

Lomber 2017). In cat models of congenital deafness, the predominant thalamocortical projections from the MGBv to A1 remain unaltered (Fig. 5b). In contrast, in animals with both early-onset (around ear opening) and late-onset (>4 postnatal months), deafness experiences a decrease in connectivity between the MGBv and A1, with more severe deficits in early-onset deafness (Barone et al. 2013; Chabot et al. 2015). Early- and late-onset deafness also differentially alters the projections from MGBd to A1 (Fig. 5c, d). After early-onset deafness, there are more projections, while no changes occur in late-onset deafness when compared with normal cats (Chabot et al. 2015). A study in gerbil showed that with auditory deprivation starting near ear opening, the normal pruning in thalamic projection from MGBd to A1 during normal development does not take place (Henschke et al. 2018). Do these TCAs from MGBd project to the thalamorecipient layers to replace the normal MGBv and L4 thalamocortical circuit? Similarly, do corticothalamic neurons in L6 project to MGBv to maintain the normal feedback connections or project ectopically to another target?

These studies suggest that there are different phases in the development of the thalamocortical pathway in the auditory system. In the first phase, the lemniscal connectivity between the thalamus and cortex can develop even without a functional cochlea, possibly via spontaneous activity. When sensory deprivation occurs near ear opening, MGBv inputs decline while MGBm and MGBd inputs to A1 increase (Fig. 5). This supports the idea that during a very early period, sensory input is crucial in shaping

the development of both lemniscal and non-lemniscal thalamocortical projections to A1. Because MGBd innervates higher-order ACtx, these results suggest that early-onset deafness leaves A1 more similar to secondary areas and that hearing (or at least peripheral function) might be necessary for A1 to acquire the hallmarks of a primary sensory area. These profound changes are important to consider when designing restorative therapies. Besides altering thalamocortical projections, deafness alters connections between higher-order areas to A1 (Barone et al. 2013; Chabot et al. 2015; Butler et al. 2016, 2017) (Fig. 5) and connections from non-auditory areas to auditory fields.

Sensory experience continues to maintain and shape the thalamocortical circuit, even after the axonal projections are formed and are mature, as shown in the late-onset deafness animals where a shift of connectivity from MGBv to MGBm (to A1) occurs (Chabot et al. 2015). Since MGBm processes polysensory information (Ryugo and Weinberger 1978; Winer and Morest 1983), the shift may indicate that other sensory information is being sent to A1.

Studies from temporary ear plugging or cochlear ablations near ear opening in rodents revealed that hearing loss alters inhibitory synaptic strength and leads to an increase in excitatory synaptic response (Kotak et al. 2005, 2008; Caras and Sanes 2015; Mowery et al. 2015, 2019). Studies in gerbils have demonstrated that auditory deprivation near ear opening led to a reduction of thalamic input on the FS neurons and disinhibition of pyramidal neurons (Takesian et al. 2010, 2013). Meanwhile, the developmental decrease of inhibition by LTS neurons was prevented by the loss of auditory input (Takesian et al. 2013). Hearing experience is also crucial in maintaining the local circuitry to A1 L2/3 neurons (Levy et al. 2019).

All manipulations discussed above were timed according to ear opening and the onset of thalamic transmission to L4. However, as detailed above, thalamic afferents innervate subplate neurons before L4 (Zhao et al. 2009). Abolishing cochlear mechanotransduction or raising mice with sounds during the early postnatal period before ear opening can change subplate circuits (Meng et al. 2021). This indicates that the auditory environment, even at the earliest ages, can shape auditory cortical circuits. Thus, manipulating cochlear function or auditory experience before ear opening or before the onset of the "classic" critical period can alter the development of cortical auditory circuits (Meng et al. 2021).

Effects of the Auditory Environment During Early Development on Auditory Processing

The studies in deaf cats demonstrated that hearing experience is crucial in shaping the axonal trajectories to their proper targets. However, these models of deafness do not identify when experience influences specific processes and also do not allow examination of how an altered circuit would process sounds. For such studies, sound exposure paradigms or temporary ear plugging in the postnatal period have been used. For example, early studies showed that rearing rodents with clicks or 2-tone sound stimuli from P8 could alter frequency tuning in the inferior colliculus (Sanes and Constantine-Paton 1985). Similarly, raising rodents in the presence of noise from just before ear opening distorts the tonotopic map of A1 and results in decreased frequency selectivity (Zhang et al. 2002). Moreover, exposure to single tones before ear opening enhances the representation of such a tone later (Zhang et al. 2001). Such exposures are effective during an early critical period lasting less than a week following ear opening.

The anatomical and functional circuit changes after these types of manipulations are unknown but presumably involve changes in thalamocortical and intracortical circuits. The current challenge is to link the experience of specific sensory stimuli with the resulting circuit changes. Although this is somewhat a chicken and egg problem, one effect of at least very early sound experience is sensory evoked decorrelation of cortical activity since abolishment of mechanotransduction results in increased correlations of large-scale cortical spontaneous activity and increased intracortical connectivity (Meng et al. 2021). This is consistent with the observed developmental decorrelation observed in vivo and on the circuit level (Liang et al. 2019; Meng et al. 2020a).

Changes in neural responses and sensory representation should lead to perceptual effects. While tone exposure increases tone representation, behavioral performance at the exposure frequency is impaired (Han et al. 2007). Conversely, temporary ear plugging during the developmental critical period results in disruption of the neuronal properties, impairment in perceptual learning skill (i.e., amplitude modulation detection task), which could be partially/fully recovered if the exposure of sound happens before the closure of the critical period (Sanes and Bao 2009; Sanes and Kotak 2011; Mowery et al. 2015). Inducing inhibition through pharmacological manipulations during the critical period manages to preserve thalamic properties and rescuing the behavioral impairments (Kotak et al. 2013; Mowery et al. 2019).

CROSS-MODAL PLASTICITY

Sensory systems do not work in isolation and sensory loss in one system is accompanied by changes in the remaining sensory systems. Multisensory integration in higher-order areas is well established (Schroeder et al. 2003; Cappe et al. 2009; Meijer et al. 2019). Cross-modal interactions can also be found among unimodal sensory areas. Animal studies have demonstrated interconnectivity among the primary sensory cortices; S1 has reciprocal

connectivity with A1 and V1, respectively, and A1 has projections to V1 but not vice versa (Budinger et al. 2006; Henschke et al. 2015; Massé et al. 2017). These interactions among the sensory cortical areas are sensitive to early experience. Early sensory loss can lead to cross-modal plasticity. In blind individuals, the visual cortex was active during somatosensory and auditory processing (Sadato et al. 1996; Cohen et al. 1997; Weeks et al. 2000; Burton et al. 2002a, b; Renier et al. 2010). In addition, subjects/people with early-onset blindness show stronger responses and enhanced perception of auditory stimuli, higher sensitivity to pitch discrimination, and refined frequency tuning (Gougoux et al. 2004; Wan et al. 2010; Huber et al. 2019). Similarly, in deaf humans, activation of the auditory area can be detected with functional MRI (fMRI) when provided with visual stimuli (Finney et al. 2001) and animal studies show that enucleation in early development alters auditory responses of neurons in V1 and A1 (Korte and Rauschecker 1993; Rauschecker and Korte 1993; Izraeli et al. 2002; Piché et al. 2007; Chabot et al. 2007).

Early loss of sensory inputs leads not only to changes in both thalamocortical and intracortical circuits to the deprived but also to the spared sensory areas (Henschke et al. 2018). In congenitally deaf cats, the cross-modal reorganization from visual and somatosensory cortices to A1 is absent, but ectopic projections could be detected to DZ and PAF (Kral et al. 2003; Barone et al. 2013; Butler et al. 2017). Meanwhile, the thalamic inputs from the posterior nucleus to A1 and from the suprageniculate nucleus to A1 and DZ are increased. The presence of ectopic projections from the visual area to DZ and PAF is crucial in processing visual stimuli in deaf animals, where the former is sensitive to motion detection and the latter processes visual peripheral localization (Lomber et al. 2010; Barone et al. 2013; Butler et al. 2017). This is consistent with observation in humans, as deaf people appear to have a more sensitive vision, especially in visual motion detection and attention (see review: Bavelier et al. 2006). Moreover, inputs from V1 (receiving visual stimuli) to A1 have been suggested to influence the auditory critical period (Mowery et al. 2016). These sensory deprivation studies demonstrated a period during early development when the corticocortical connectivity between different sensory modalities is sensitive to modification by sensory experience. Moreover, the differences in timing in the development of the different cortical regions raise a question of how cross-modal integration is achieved at young ages and how signaling from more mature areas possibly influences the developmental trajectory of less mature areas.

However, cross-modal changes are not restricted to the early developmental period. Temporary visual deprivations after the critical period can lead to enhanced auditory responses in A1 (Petrus et al. 2014; Solarana et al. 2019), increased thalamocortical (Petrus et al. 2014), and refined intracortical circuits (Petrus et al. 2015; Meng et al. 2015, 2017a). These suggest that the capacity of ACtx to be plastic is not abolished after the critical period but that the conditions to engage intrinsic plasticity mechanism might have changed.

CLINICAL CONSIDERATIONS BEYOND DEAFNESS

The developing ACtx is highly plastic because of its longer developmental period, leading to a higher risk for re-organizing abnormally due to intrinsic or extrinsic causes. Premature birth is associated with auditory processing disorders (Amin et al. 2015; Durante et al. 2018; Jones and Weaver 2020) including deficits in speech discrimination and language learning (Therien et al. 2004; Durante et al. 2018; Bartha-Doering et al. 2019) suggesting an altered developmental trajectory of the auditory system. Moreover, neurological disorders such as autism spectrum disorders (ASD) or schizophrenia is accompanied by deficits of cortical processing in multiple cortical regions including ACtx (Javitt 2009; Marco et al. 2011). Individuals with either ASD or schizophrenia demonstrate a higher risk of having auditory abnormalities (Toga et al. 2006; Hugdahl et al. 2008; O'Connor 2012). Postmortem schizophrenic brains exhibit a reduction in L3 pyramidal somal volume and dendritic spine density in ACtx, indicating possible abnormalities in feedforward circuitry from primary to higher-order of the auditory system (Sweet et al. 2003, 2004, 2009). The brains of ASD individuals show wider minicolumns in A1 and higher-order association areas, suggesting a possible alteration in local networking (McKavanagh et al. 2015).

One of the key players during development is the subplate and the transient circuits they form (Molnár et al. 2020). In the preterm infants, auditory stimuli evoke particular EEG signals ("delta-brushes") which are unique to fetal development (Chipaux et al. 2013) and might reflect subplate contributions (Molnár et al. 2020). Functional studies on subplate integrity in human development are needed as these circuits are vulnerable and disrupted in disorders, e.g., in ASD models or following hypoxia (Pogledic et al. 2014; Hoerder-Suabedissen and Molnár 2015; McClendon et al. 2017; Nagode et al. 2017; Hadders-Algra 2018; Sheikh et al. 2019; Molnár et al. 2020).

SUMMARY

In the past few decades, multiple studies in embryonic and postnatal period have established the basic understanding of neurogenesis, axonal growth, synapse formation in the neocortex, and how such processes are affected by intrinsic and extrinsic factors at different timing during development. In the ACtx, the neurogenesis for cortical excitatory and inhibitory neurons

(~E10.5 in rodents), the arrival of TCAs in the subplate (~E13.5 in rodents), and the completion of migration of excitatory neurons into respective lamina (~P5 in rodents) happen earlier than the VCtx (Fig. 2). However, TCAs arrive in L4 later in the ACtx than the VCtx, thus the transient period for SP connection with TCAs might be longer in the ACtx than the VCtx (Fig. 3). Moreover, the refinement process of dendrites and spines in ACtx is longer than in other cortical areas. Thus, the basic understanding contributed by studies in other sensory systems has to be carefully interpreted. Furthermore, the primary ACtx shows some distinct morphological features (i.e., L3/4 as thalamocortical recipient layers, lack of spiny stellate cells in L4); hence, some of the functional properties as well as the circuitry and developmental progression in the auditory system may differ from other sensory areas. Many anatomical and functional studies have established a basic understanding of the connectivity between the thalamus, primary and secondary auditory cortical areas in the adult, and how these are altered by changing sensory experience. However, many open questions remain as we do not have a complete picture of the changing circuits across and within ACtx regions. The increasing availability of tools to track and manipulate specific cell classes should aid progress in this direction. Below, we have listed a few important questions worth exploring to elucidate the development of auditory processing and to provide insights into auditory-related developmental disorders.

OUTLOOK/OPEN QUESTIONS

1) How do multiple sensory cortical areas synchronize during development when the temporal patterns of their cortical maturation and critical periods are different?
2) What is the mechanism controlling the timing of the TCAs innervation from SP to L4 in ACtx?
3) What are the developmental differences between first-order and higher-order auditory areas and their respective thalamocortical circuits?
4) What is the underlying mechanism behind the small number/absence of spiny stellate neurons in L4 of A1?
5) How is experience of specific sensory stimuli linked to circuit changes?
6) How can circuit changes after early hearing loss be reversed?
7) How is cross-modal integration achieved at young ages and do more mature areas influence the developmental trajectory of less mature areas?

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