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The effect of noise exposure during the developmental period on the function of the auditory system

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ABSTRACT

Recently, there has been growing evidence that development and maturation of the auditory system depends substantially on the afferent activity supplying inputs to the developing centers. In cases when this activity is altered during early ontogeny as a consequence of, e.g., an unnatural acoustic environment or acoustic trauma, the structure and function of the auditory system may be severely affected. Pathological alterations may be found in populations of ribbon synapses of the inner hair cells, in the structure and function of neuronal circuits, or in auditory driven behavioral and psychophysical performance. Three characteristics of the developmental impairment are of key importance: first, they often persist to adulthood, permanently influencing the quality of life of the subject; second, their manifestations are different and sometimes even contradictory to the impairments induced by noise trauma in adulthood; third, they may be 'hidden' and difficult to diagnose by standard audiometric procedures used in clinical practice. This paper reviews the effects of early interventions to the auditory system, in particular, of sound exposure during ontogeny. We summarize the results of recent morphological, electrophysiological, and behavioral experiments, discuss the putative mechanisms and hypotheses, and draw possible consequences for human neonatal medicine and noise health.

Contents

Abbreviations: ABR, auditory brainstem response; AC, auditory cortex; ASR, acoustic startle response; CAP, compound action potential; CF, characteristic frequency; CIC, central nucleus of the inferior colliculus; DIC, dorsal cortex of the inferior colliculus; DPOAE, distortion-product otoacoustic emissions; EIC, external cortex of the inferior colliculus; FTC, frequency tuning curve; IC, inferior colliculus; IHC, inner hair cells; MGB, medial geniculate body; NICU, Neonatal Intensive Care Unit; OHC, outer hair cells; Pn, postnatal day n; PPI, prepulse inhibition; RIF, rate-intensity function

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1. Introduction

Over the past few decades, the development and maturation of the auditory system has been the subject of numerous studies. However, despite several pioneering works that showed the effects of developmental auditory deprivation (Silverman and Clopton, 1977; Clopton and Silverman, 1978; Coleman and O'Connor, 1979) and suggested that an experience-driven activity during ontogeny is necessary for normal development (Sanes and Constantine-Paton, 1983, 1985; Sanes and Takács, 1993), it was not until recently that the importance of proper stimulation and patterned neuronal activity was fully appreciated (e.g., Zhang et al., 2001, 2002; Bao et al., 2003; Chang and Merzenich, 2003; Kandler, 2004; Nakahara et al., 2004; Chang et al., 2005; Kandler and Gillespie, 2005; Zhang et al., 2008; Zhou and Merzenich, 2008; Zhou et al., 2008; Grécová et al., 2009; Bureš et al., 2010, 2014; Bao et al., 2013). It appears that even a short-time detachment of the developing auditory system from natural and rich auditory experience, resulting from an impaired periphery or anomalous stimulation such as noise or clicks, will often result in altered structural and functional characteristics at various levels of the auditory system. These changes may comprise, for example, a decrease in the number of hair cells and their ribbon synapses (Rybalko et al., 2015; Shi et al., 2015), abnormal dendritic trees and cell sizes of neurons in the central auditory system (Gabriele et al., 2000; Ouda et al., 2014; Lu et al., 2014), altered neuronal responsiveness and representation of stimulus frequency and intensity (e.g., Zhang et al., 2001; Grécová et al., 2009; Bureš et al., 2010; Insanally et al., 2010), or deteriorated psychophysical, behavioral and cognitive functions (Rybalko et al., 2011; Sun et al., 2011; Pan et al., 2011; Rybalko et al., 2015; Šuta et al., 2015; Ruvalcaba-Delgadillo et al., 2015). Despite this volume of work, there still exists great uncertainty as to what effect a specific intervention will have: different interventions may have different consequences at various levels of the auditory system, depending also on the type of intervention, stimulus type, exposure levels, or age of exposure (e.g., Sanes and Constantine-Paton, 1985; Zhang et al., 2001; Chang et al., 2005; Zhou et al., 2008; Grécová et al., 2009; Insanally et al., 2009; Miyakawa et al., 2013). The duration of the insulting exposure plays an important role: for example, a brief intense exposure has often no influence on the hearing thresholds and neuronal excitatory thresholds (Grécová et al., 2009; Kujawa and Liberman, 2009; Bureš et al., 2010; Rybalko et al., 2011; Sanz et al., 2015; Shi et al., 2015), while long-term exposure to moderate-level sounds results in elevated sound thresholds (Gao et al., 2009; Insanally et al., 2010; Lauer and May, 2011).

The plasticity and the influence of somatic and environmental conditions on the hearing system is to a certain extent preserved during the whole life span. However, the developing organism is profoundly more susceptible to anomalous conditions than a mature subject (Rybalko and Syka, 2001; Syka, 2002). Furthermore, the insulting interventions during ontogeny operate via different mechanisms than in adulthood and although the developmental impairments give rise to anomalies that may also persist to adulthood, they may have different manifestations than the consequences of an intervention on a mature and fully developed system (Bureš et al., 2010). In particular, noise trauma in adulthood is assumed to result in a decrease of inhibition and an increased excitability of the auditory centers (Salvi, 1996; Wang et al., 1996; Salvi et al., 2000; Noreña et al., 2003; Sun et al., 2012), while a developmental noise exposure leads to lower maximum response magnitudes and presumably higher overall inhibitory strength (Grécová et al., 2009; Bureš et al., 2010; Rybalko et al., 2011, 2015). It was thus suggested that an intervention during ontogeny disrupts normal development of, particularly, inhibitory connectivity, and the undeveloped circuits partly or completely retain their immature characteristics (Sanes and Takács, 1993; Gabriele et al., 2000; Rybalko and Syka, 2001; Kandler, 2004; Grécová et al., 2009). Moreover, the developmental impairments may affect only certain tonotopical regions: Pierson and Snyder-Keller (1994) showed that adult rats which were transiently deafened on postnatal day 14 (P14) using an intense 8-min noise exposure present signs of improper development of the inferior colliculus (IC) only in the high-frequency regions. In our previous studies, we used similar exposure paradigms to Pierson and Snyder-Keller (1994) to further explore the effects of a brief noise exposure on the development of structure and function of the auditory system (Grécová et al., 2009; Bureš et al., 2010; Rybalko et al., 2011; Ouda et al., 2014; Lu et al., 2014; Rybalko et al., 2015; Šuta et al., 2015). Early noise exposure was used to induce a temporary hearing threshold shift of 10–20 dB during the critical period. Such threshold elevation was shown to be sufficient to interrupt or suppress the flow of signals from the cochlea to auditory centers with consequent preservation of their immature character up to adulthood.

Recently, an important aspect of hearing impairments has been pointed out: despite a seemingly unaffected periphery documented by normal hearing thresholds and oto-acoustic emissions, the subjects may exhibit deficits in higher-order stimulus processing and psychophysical performance (Zhang et al., 2008; Rybalko et al., 2011; Zhou and Merzenich, 2012; Šuta et al., 2015). In particular, such 'hidden' deficits are the result of a temporary preclusion of the developing auditory centers from proper stimulation, and also of long-term exposures to unnatural and non-structured stimuli such as white noise or tones, even when they are presented at a moderate intensity level and/or with a simple amplitude modulation (e.g., Zhang et al., 2002; Kujawa and Liberman, 2009; Oliver et al., 2011; Shi et al., 2015; Liberman et al., 2015; Rybalko et al., 2015).

This paper reviews and discusses the current knowledge of the topic, including the results of recent morphological, electrophysiological, and behavioral experiments. Despite the fact that the reported findings mostly originate from animal experiments, they have important direct implications for human medicine and noise health, showing, for example, that noise exposure of underweighted premature babies in Intensive Care Units may lead to central audio-processing deficits that can be hardly detected by routine audiological measurements (Fortes et al., 2007; Isotani et al., 2009; Gallo et al., 2011).

2. Hearing thresholds and auditory periphery

The effect of sound exposure on the cochlea and auditory nerve largely depends on the stimulus intensity and duration of the exposure. In our experiments, the average thresholds of 14-day-old rats measured using auditory brainstem responses (ABR) ranged before exposure from 62 dB SPL at 8 kHz to 88 dB SPL at 40 kHz. An 8-min noise exposure to broad band noise at 125 dB SPL produced a

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