



Research paper

Effects of passive, moderate-level sound exposure on the mature auditory cortex: Spectral edges, spectrotemporal density, and real-world noise

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ABSTRACT

Persistent, passive exposure of adult cats to bandlimited tone pip ensembles or sharply-filtered white noise at moderate levels (~ 70 dB SPL) leads to a long-term suppression of spontaneous and sound-evoked activity in the region(s) of primary auditory cortex (AI) normally tuned to the exposure spectrum, and to an enhancement of activity in one or more neighboring regions of AI, all in the apparent absence of hearing loss. Here, we first examined the effects of passive exposure to a more structured, real-world noise, consisting of a mix of power tool and construction sounds. This “factory noise” had less pronounced effects on adult cat AI than our previous random tone pip ensembles and white noise, and these effects appeared limited to the region of AI tuned to frequencies near the sharp factory noise cutoff at 16 kHz. To further investigate the role of sharp spectral edges in passive exposure-induced cortical plasticity, a second group of adult cats was exposed to a tone pip ensemble with a flat spectrum between 2 and 4 kHz and shallow cutoff slopes (12 dB/oct) on either side. Compared to our previous ensemble with the same power in the 2–4 kHz band but very steep slopes, exposure to the overall more intense, sloped stimulus had much weaker effects on AI. Finally, we explored the issue of exposure stimulus spectrotemporal density and found that low aggregate tone pip presentation rates of about one per second sufficed to induce changes in the adult AI similar to those characteristic of our previous, much denser exposures. These results are discussed in light of the putative mechanisms underlying exposure-induced auditory cortical plasticity, and the potential adverse consequences of working or living in moderately noisy environments.

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1. Introduction

In previous studies, we have passively exposed adult cats to a number of moderate-level experimental acoustic environments (EAEs) for several days to months at a time (Noreña et al., 2006; Pienkowski and Eggermont, 2009, 2010a; 2010b; Pienkowski et al., 2011). The EAEs consisted either of spectrotemporally-dense random tone pip ensembles, or of white noise, which were varied in bandwidth and center frequency but which were always sharply bandlimited. The cochlea and lower brainstem appeared unaffected by these EAEs, as judged by auditory brainstem responses (ABRs)

and distortion product otoacoustic emissions (DPOAEs). Nevertheless, spike activity and local field potentials (LFPs) in primary (AI) and non-primary auditory cortex were suppressed in response to sound frequencies falling roughly within the exposure bands, and enhanced at frequencies above and/or below those bands. In the course of sufficiently long exposures, the suppression gave way to a reorganization of the AI tonotopic map, such that regions of AI normally tuned to the exposure frequencies became tuned to neighboring frequencies with little or no loss of sensitivity, reminiscent of reorganization induced by partial hearing loss (Robertson and Irvine, 1989; Rajan et al., 1993; Schwaber et al., 1993; Willott et al., 1993; Noreña and Eggermont, 2005). These exposure-induced changes appeared to be reversible (with several caveats), but the reversal required a long time period in a quiet laboratory environment (e.g., >12 weeks of quiet following a 6-week exposure; for a review, see Pienkowski and Eggermont, 2011).

In the present paper, we continue to characterize the effects of passive moderate-level (~ 70 dB SPL) sound exposure on the

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mature auditory brain. In the first experiment, we were interested in the effects of a less random, more “real-world” noise, and exposed adult cats for at least 8 weeks to a mix of power tool and construction sounds that we dubbed the “factory noise EAE” (Fig. 1). The effects on AI of exposure to factory noise were less pronounced than to our previous random tone pip and white noise EAEs, and seemed limited to the frequency region tuned to the sharp 16 kHz factory noise cutoff. To determine whether sharp EAE spectral edges are necessary to drive exposure-induced plasticity, as suggested by our results with factory noise, we put a second group of cats in an EAE consisting of a random tone pip ensemble with a flat spectrum between 2 and 4 kHz and shallow cutoff slopes (12 dB/oct) on either side (between 0.1 and 20 kHz) (Fig. 1: “2–4 kHz sloped EAE”). Compared to a tone pip EAE with the same power in the 2–4 kHz band but no power outside (Fig. 1: “2–4 kHz bandpass EAE”), exposure to the overall more intense sloped EAE

had a much smaller though still significant effect on neural activity in AI.

Having established that sharp spectral edges in the exposure stimulus are largely if not entirely responsible for the observed cortical plasticity, we next investigated the issue of EAE spectrotemporal density. Our previous tone pip EAEs ranged in bandwidth from one-third to two octaves, with individual frequencies generated independently at the average rate of 3 per s and multiplied by 16 frequencies per octave for an average aggregate rate of 48 pips per s per octave. In developing rats, tone pips presented at the much lower rate of 5 per s during a brief sensitive period from postnatal day 11 (P11) to P13 were reported to produce enhanced AI responses around the exposure frequency (e.g., Zhang et al., 2001; de Villiers-Sidani et al., 2007). We were interested if these seemingly opposing results – enhanced activity in developing animals, suppressed activity in adults – could be explained by variations in

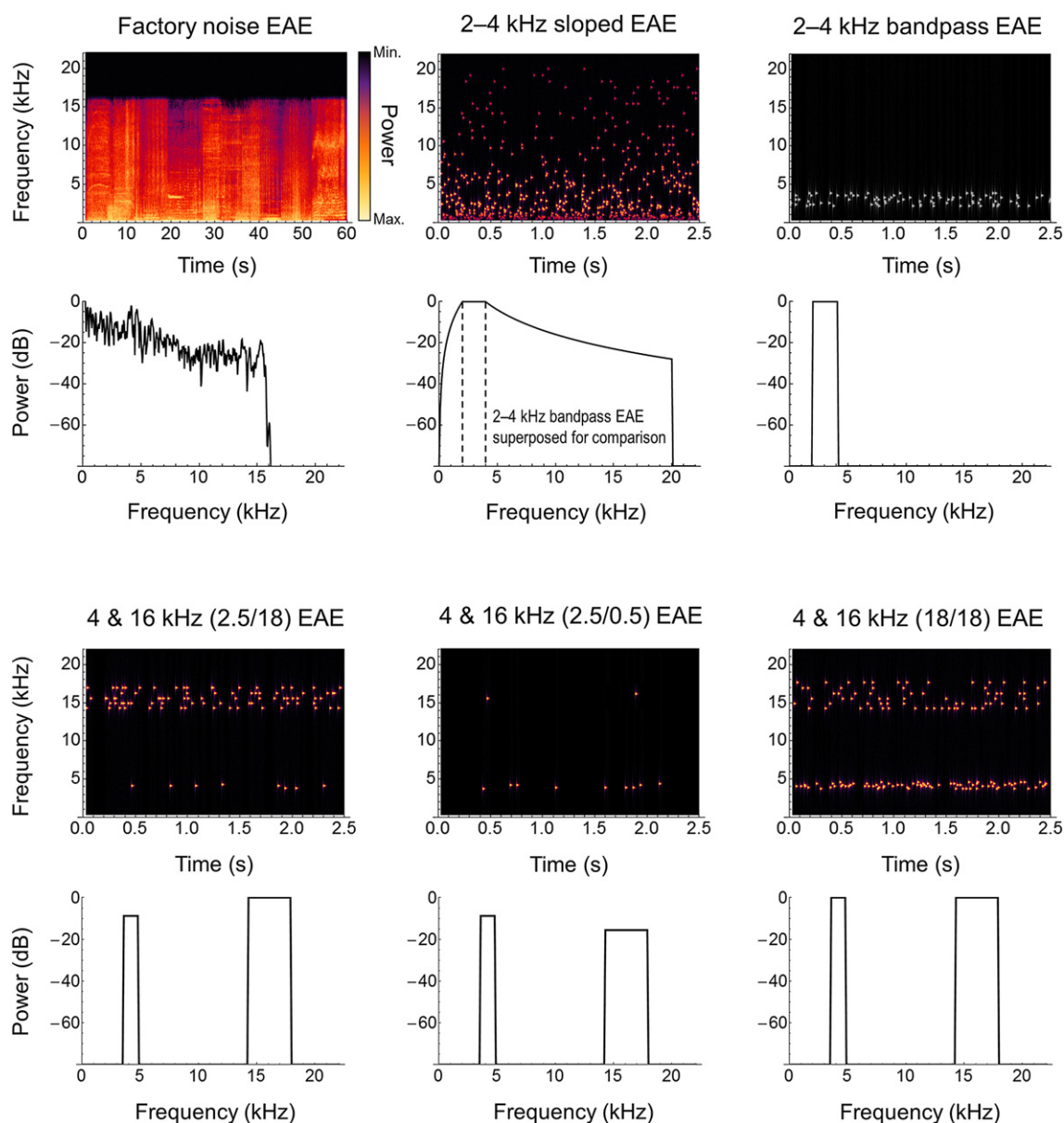


Fig. 1. Spectrograms and long-term power spectra for the six experimental acoustic environments (EAEs) most relevant to the present study. Note that for the 4 & 16 kHz EAEs (bottom row), all pips had the same peak amplitude; differences in the long-term power spectra of these EAEs reflect differences in their temporal densities, not individual pip amplitudes.

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