Amplitude modulation reduces loudness adaptation to high-frequency tones

Dwight P. Wynne,[a] Sahara E. George, and Fan-Gang Zeng
Center for Hearing Research, University of California, Irvine, 110 Medical Science E, Irvine, California 92697, USA

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Long-term loudness perception of a sound has been presumed to depend on the spatial distribution of activated auditory nerve fibers as well as their temporal firing pattern. The relative contributions of those two factors were investigated by measuring loudness adaptation to sinusoidally amplitude-modulated 12-kHz tones. The tones had a total duration of 180 s and were either unmodulated or 100%-modulated at one of three frequencies (4, 20, or 100 Hz), and additionally varied in modulation depth from 0% to 100% at the 4-Hz frequency only. Every 30 s, normal-hearing subjects estimated the loudness of one of the stimuli played at 15 dB above threshold in random order. Without any amplitude modulation, the loudness of the unmodulated tone after 180 s was only 20% of the loudness at the onset of the stimulus. Amplitude modulation systematically reduced the amount of loudness adaptation, with the 100%-modulated stimuli, regardless of modulation frequency, maintaining on average 55%–80% of the loudness at onset after 180 s. Because the present low-frequency amplitude modulation produced minimal changes in long-term spectral cues affecting the spatial distribution of excitation produced by a 12-kHz pure tone, the present result indicates that neural synchronization is critical to maintaining loudness perception over time. © *2015 Acoustical Society of America*.
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I. INTRODUCTION

Everyday listening environments often contain sounds that last for seconds or minutes. Under most conditions, the apparent loudness of these continuous sounds remains stable (Scharf, 1983; Tang *et al.*, 2006). However, normal-hearing listeners perceive pure tones of extremely high frequency or low intensity to quickly decrease in loudness, often decaying to inaudibility within minutes (Hellman *et al.*, 1997). Abnormal adaptation to comfortable-intensity sounds is known to occur in some pathological conditions such as auditory nerve tumors (Carhart, 1957; Johnson, 1979). Previous studies of long-term loudness adaptation have suggested that its physiological correlates are the number and distribution of activated auditory nerve fibers as well as the timing of neural spikes in those fibers (Scharf, 1983; Moore and Peters, 1997; Huss and Moore, 2003). However, the relative contributions of these two cues have been difficult to separate. Compared to low-frequency or high-intensity tones, high-frequency or low-intensity tones both activate many fewer nerve fibers (Kim *et al.*, 1990; Kim and Parham, 1991) and elicit degraded phase locking of neural impulses (Kiang, 1965; Johnson, 1980).

We recently showed that individuals with neural dyssynchrony due to ribbon synapse disorder have abnormal loudness adaptation, even to low-frequency, comfortable-intensity pure tones (Wynne *et al.*, 2013). A hallmark of these individuals is impaired processing of both temporal envelope and fine structure information, including abnormal temporal modulation detection threshold, poor frequency discrimination at low frequencies, and reduced ability to use interaural timing cues (Zeng *et al.*, 2005). In the present study, we asked an opposite question: will introduction of neural synchrony to the envelope reduce loudness adaptation in normal-hearing listeners? We used low-frequency amplitude modulation of high-frequency tones to answer this question.

It is well established that high-frequency tones produce a narrower spread of excitation than low-frequency tones (Kim and Molnar, 1979; Moore and Glasberg, 1983). It is also well established that slow (<10% carrier frequency) amplitude modulation, although it dynamically changes the width of the short-term spread of excitation pattern (e.g., Burns and Viemeister, 1981), does not significantly affect the long-term spread of the excitation pattern of a high frequency carrier (Kohlrausch *et al.*, 2000; Moore and Glasberg, 2001). In particular, at low intensities, neurons whose characteristic frequency is equal to the modulation frequency are not activated by the high-frequency carrier (Javel, 1980). However, amplitude modulation induces auditory nerve fibers to phase lock to the modulation frequency, creating neural synchrony in its response to high-frequency carriers (Javel, 1980; Smith and Brachman, 1980; Joris and Yin, 1992; Dreyer and Delgutte, 2006). Neural synchrony increases with amplitude modulation depth but the firing rate is independent of modulation depth (Joris and Yin, 1992).

[a]Electronic mail: dwynne@uci.edu

According to Scharf (1983), "…fluctuations in the level of stimulation reduce or eliminate adaptation. Fluctuations may be in the stimulus or in the sensory organ." However, to our knowledge, no systematic measurement is available on the effect of short-term fluctuations in stimulus level on loudness adaptation. Here we used a high-frequency carrier to restrict the population of activated nerve fibers, and systemically varied the frequency and depth of modulation to investigate the influence of neural synchrony on loudness adaptation.

II. EFFECT OF MODULATION FREQUENCY ON ADAPTATION

A. Methods

Adaptation was quantified by measuring changes in loudness of a stimulus over a 180-s period (Scharf, 1983). Ten young, normal-hearing subjects listened to 12 kHz tones at 15 dB above their pure-tone threshold at 12 kHz. The tones were sinusoidally amplitude modulated at a depth of 100% and a frequency of 0 (unmodulated), 4, 20, or 100 Hz. All stimuli were generated digitally at a sampling rate of 44.1 kHz and presented to the right ear through circumaural headphones (Sennheiser HDA200). Starting immediately after onset of the tone, subjects estimated the loudness of the tone every thirty seconds until they had listened for 180 s continuously. Subjects could use any number, including decimals and fractions, to represent the loudness of the stimulus. The only restriction on estimates was that a value of zero indicated loss of audibility. The four tones were presented in random order. Each trial was followed by a rest break no shorter than five minutes, in order to minimize effects of residual adaptation (Hellman *et al.*, 1997).

To allow comparison between results for different subjects with different initial loudness judgments, loudness estimates were normalized according to the equation

$$\%\ adaptation = 100 * \frac{(L_t - L_0)}{L_0}. \quad (1)$$

In Eq. (1), L_t is the estimated loudness at time t and L_0 is the estimated initial loudness (Tang *et al.*, 2006; Wynne *et al.*, 2013). Using this normalization, a value of −100% corresponded to an inaudible sound, 0% corresponded to no change in loudness, and positive numbers indicated that the sound became louder over time. In addition, the time course of loudness adaptation was fitted by an exponential equation

$$y(t) = s(1 - e^{-t/\tau}). \quad (2)$$

In Eq. (2), $y(t)$ is the amount of adaptation at time t, s is an asymptotic saturation value representing the amount of adaptation to an infinitely long stimulus, and τ is a "time constant" representing the amount of time required to reach 63% of the asymptotic saturation value (Tang *et al.*, 2006; Wynne *et al.*, 2013). The saturation value was constrained to be between −100% and 0%, and the time constant was constrained to be between 0 and 180 s. Analysis of variance was used to investigate the effects of time and modulation frequency on the normalized loudness estimates.

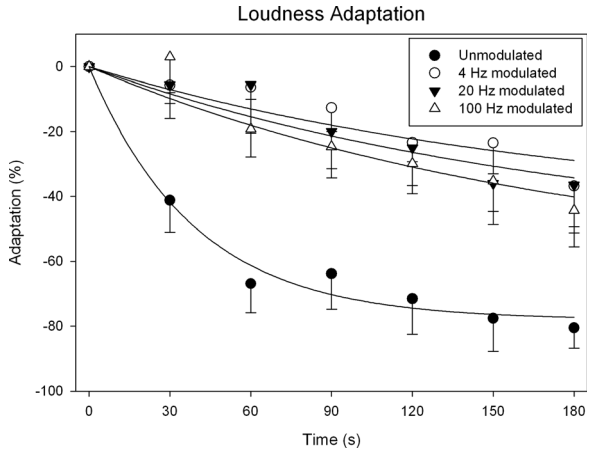

FIG. 1. Loudness adaptation to a 12-kHz tone, amplitude modulated at 100% depth, as a function of time. Each symbol represents a different modulation frequency. Error bars represent plus or minus one standard error of the mean. The solid lines represent fits to an exponential decay [see Eq. (2) in the text].

B. Results

Less adaptation was seen to the 100% amplitude-modulated tones than to the unmodulated pure tone, regardless of modulation frequency. After 180 s, the loudness of the unmodulated pure tone had decreased to approximately 20% of its initial loudness, while the loudness of the three modulated tones had only decreased to approximately 65% of their initial loudness (Fig. 1). Consistent with previous models of loudness adaptation to pure tones, Eq. (2) was a good fit to the loudness estimates for the unmodulated tone (Table I). Equation (2) also fit the loudness estimates well for the modulated tones, but for all three tones the fit was achieved with the time constant set to 180 s.

There was a significant main effect of modulation frequency on the normalized loudness estimates after 180 s [$F(3,27) = 7.849$, $p = 0.001$], but this main effect was between the unmodulated tone and the three modulated tones, with the latter not differing significantly ($p > 0.23$ for each *post hoc* t-test, uncorrected).

Because loudness estimates to the 4 Hz, 20 Hz, and 100 Hz modulated tones were not significantly different, we held modulation frequency constant at 4 Hz in the second experiment exploring the effect of modulation depth on loudness adaptation.

III. EFFECT OF MODULATION DEPTH ON ADAPTATION

A. Methods

Twenty-one young, normal hearing subjects, including the ten subjects who participated in the previous experiment, listened to 12 kHz tones at 15 dB above their pure-tone threshold at 12 kHz. The tones were amplitude modulated at a frequency of 4 Hz with modulation depths of 0%, 5%,

TABLE I. Fitted parameters and goodness-of-fit for Eq. (2).

Frequency (Hz)	s (% adaptation)	τ (s)	Adjusted R^2
0	−78	39	0.95
4	−46	180	0.79
20	−54	180	0.84
100	−64	180	0.80

TABLE II. Fitted parameters and goodness-of-fit for Eq. (2).

Depth (% modulation)	s (% adaptation)	τ (s)	Adjusted R^2
0	−78	39	0.97
5	−70	43	0.97
10	−73	68	0.99
25	−49	75	0.99
50	−32	180	0.87
100	−33	180	0.82

10%, 25%, 50%, or 100%. Stimulus generation, experimental procedure, and data analysis were the same as for the previous experiment.

B. Results

Normalized loudness estimates (Fig. 2) significantly decreased over time [$F(6,120) = 73.477$, $p < 0.001$] and significantly increased with increasing modulation depth [$F(5,100) = 30.362$, $p < 0.001$]. There was a significant interaction between time and depth [$F(30,600) = 8.692$, $p < 0.001$]. Equation (2) was a good fit for the loudness estimates for each stimulus (Table II). For the 50% and 100% modulation depth conditions, the fit was achieved with the time constant set to 180 s.

After 180 s, the loudness of the unmodulated tone had decreased to about 20% of its loudness at onset. This was not significantly different from the loudness ratings in the 5% and 10% depth conditions. All three of these tones had significantly lower final loudness ratings than for the other three conditions ($p < 0.01$ for all pairwise comparisons, *post hoc* t-tests with Bonferroni correction). The loudness of the 100% modulated tone was approximately 80% as loud as at onset after 180 s. It was not significantly louder than the 50% modulated tone, but both high-modulation-depth tones were significantly louder after 180 s than for the other four conditions ($p < 0.05$ for all pairwise comparisons, *post hoc* t-tests with Bonferroni correction).

Over half of the subjects (11/21) reported that the unmodulated tone became inaudible within 180 s. For nine of those subjects, 25% amplitude modulation depth was sufficient to ensure audibility for the entire 180-s duration of the tone. The other two subjects maintained complete perception of the tone only in the 50% and 100% conditions.

There was significantly less loudness adaptation to stimuli in which the modulation was easily detected than to stimuli in which the modulation was difficult to detect. In Fig. 3, the same data as in Fig. 2 are displayed as a function of modulation depth at each of six measurement times. The solid lines represent fits to the equation

$$y(d) = y_0 + a(1 - e^{-d/\Delta}), \tag{3}$$

where d is modulation depth and Δ is a "depth constant" analogous to the time constant. The 70.7% amplitude modulation detection threshold in a subset of seven of these subjects ranged from 23% to 43% (vertical dashed lines, Fig. 3).

Equation (3) fit the data at each of the six measurement times after onset of the stimulus well. The values of the fitted parameters, and the adjusted R^2 of each fit, are shown in Table III. The fitted depth constant remained relatively constant at each time point and was always near the lower bound of the range of 70.7% amplitude modulation detection thresholds.

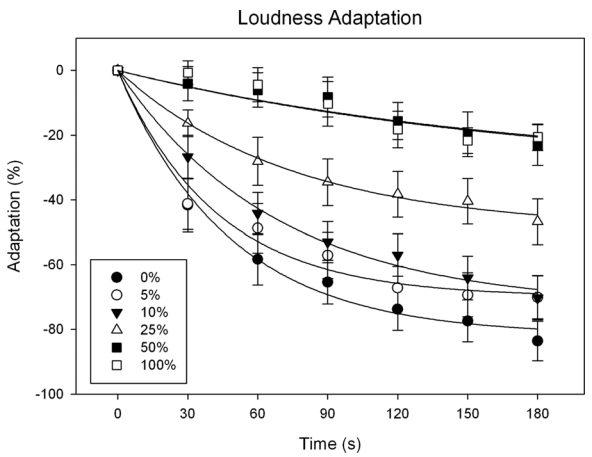

FIG. 2. Loudness adaptation to a 12-kHz tone, amplitude modulated at 4 Hz, as a function of time. Symbols, error bars, and solid lines as in Fig. 1.

IV. DISCUSSION

A. Peripheral mechanisms of loudness adaptation

Previously, we showed that loss of neural synchronization can result in significant long-term loudness adaptation,

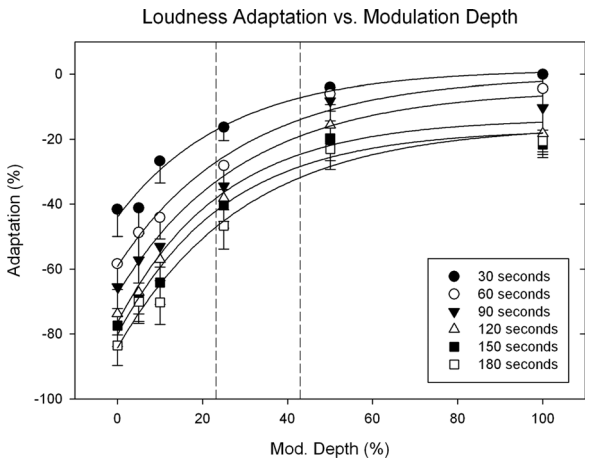

FIG. 3. Loudness adaptation to a 12-kHz tone, amplitude modulated at 4 Hz, as a function of modulation depth. Error bars as in Fig. 1. Each symbol represents a different measurement time after tone onset. The dashed lines represent the boundaries of the range of thresholds for amplitude modulation detection. The solid lines represent fits to an exponential decay [see Eq. (3) in the text].

TABLE III. Fitted parameters and goodness-of-fit for Eq. (3).

Time (s)	y_0 (% adaptation)	a (% adaptation)	Δ (% modulation)	Adjusted R^2
30	−44	45	25	0.93
60	−59	59	29	0.96
90	−68	63	29	0.92
120	−77	63	25	0.94
150	−80	63	25	0.94
180	−84	69	30	0.94

even for combinations of frequency and intensity that would not elicit loudness adaptation in normal hearing (Wynne *et al.*, 2013). Here we show the converse: introducing neural synchronization through amplitude modulation provides "release" from adaptation for combinations of frequency and intensity that would otherwise elicit complete loudness adaptation to the pure tone. Thus, a restricted excitation pattern is not, by itself, sufficient to elicit loudness adaptation; rather, synchronous firing of auditory nerve fibers is both necessary and sufficient for preventing loudness adaptation.

While the firing rate of auditory nerve fibers decreases with constant stimulation lasting seconds or minutes (Kiang, 1965; Javel, 1996), the synchronization of neural spikes remains constant throughout the response (Johnson, 1980). Thus, synchronization does not appear to be linked to adaptation of firing rate in the auditory nerve.

At low intensities, the vast majority of auditory nerve fibers that respond to a stimulus have low thresholds and high spontaneous discharge rates (Liberman, 1978; Joris and Yin, 1992). Thus, greater loudness adaptation at low intensities could result in part from greater adaptation of firing rate in high-spontaneous rate auditory nerve fibers compared to low-spontaneous rate fibers. We find this alternative explanation unlikely as high-spontaneous rate fibers show less long-term adaptation than low-spontaneous rate fibers (Javel, 1996).

At low modulation frequencies and high modulation depths, peaks in the amplitude-modulated signal are clearly detectable but the valleys may not be, meaning that the stimulus is effectively perceived as an interrupted tone. Studies of persons with retrocochlear hearing loss showed significant adaptation for sustained tones but not for interrupted tones, e.g., a type III Bekesy audiogram (Jerger, 1960). This lack of adaptation may be explained by onset and offset cues at the periphery, or by recovery from adaptation during the silent interval. These explanations cannot fully account for our results for two reasons. First, Bekesy audiometry is typically performed with a tone being turned repeatedly on for 200 ms then off for 200 ms, producing effectively 2.5-Hz square wave modulation. The 2.5-Hz gating frequency is similar to our lowest modulation frequency of 4 Hz, but much lower than our highest modulation frequency of 100 Hz. The 100-Hz, 100%-modulated tone, which produces a continuously buzzing percept, was as effective in reducing loudness adaptation as the 4-Hz, 100%-modulated tone, which sounds like an interrupted tone (Fig. 1). Second, the interrupted tone hypothesis cannot account for the results for the 4-Hz modulation depth (Figs. 2 and 3), which showed that 50% amplitude modulation was as effective as 100% modulation in reducing loudness adaptation. Unlike 100% modulation, the 50% modulated tone had no silent interval at all, with the lowest amplitude envelope level being half of the carrier amplitude.

B. Central mechanisms of loudness adaptation

Loudness perception of brief sounds is likely governed by a multi-stage process that involves logarithmic intensity compression by peripheral auditory structures and exponential expansion by central auditory structures (Zeng and Shannon, 1994; Zeng, 2013). Loudness adaptation to longer tones may be best explained as a change in the entire loudness growth curve, with soft tones getting even softer and loud tones getting slightly louder over time (Tang *et al.*, 2006). The active participation of central structures in intensity-to-loudness conversion thus suggests that adaptation of loudness may result from changes in the responses of these central structures to continued stimulation.

A number of psychophysical studies have provided evidence that central mechanisms are involved in loudness adaptation. Shannon (1992) found that cochlear implant listeners could perceive binaural "beats" resulting from pulse trains of different frequencies, even when the listeners had completely adapted to one of the pulse trains. Electrical stimulation of the inferior colliculus has elicited adaptation of "loudness" similar to that in acoustic stimulation (Lim *et al.*, 2008). Furthermore, Reavis and colleagues (2012) found that loudness adaptation to high-frequency, 100% amplitude modulated tones by tinnitus patients was often accompanied by temporary suppression of their tinnitus. Since tinnitus is largely believed to be a disorder of central loudness perception (Hébert *et al.*, 2013), we believe that this induced "adaptation" of tinnitus may result from the effect of amplitude modulation cues on central mechanisms governing loudness adaptation.

There are at least two plausible ways in which the introduction of amplitude modulation cues could affect central mechanisms governing loudness adaptation. The first is by activating a central "modulation filter bank" (Dau *et al.*, 1997; McDermott and Simoncelli, 2011). This filter bank may result from the interplay between excitatory and inhibitory inputs into the ventral cochlear nucleus and/or inferior colliculus (Nelson and Carney, 2004). Our finding that loudness adaptation was significantly less when the amplitude modulation was easily detected suggests that increased output from neurons in this modulation filter bank could provide additional excitation that partially overcomes adaptation mechanisms.

The second plausible way is related to differential neural responses to dynamic changes in the auditory cortex (Lu *et al.*, 2001; Liang *et al.*, 2002). A steady-state sound will likely elicit only onset and offset responses, whereas a modulated sound will likely elicit continuously ongoing cortical response. At present, it remains unclear whether these differential cortical responses are the cause or the result of the dynamic gain control mechanism.

C. Concluding remarks

Loudness adaptation is hypothesized to depend on three properties of the auditory nerve response: the number, distribution, and timing of auditory nerve spikes. Long-term adaptation of auditory nerve fibers would decrease the total amount of input to more central neurons, but sufficiently high synchrony of spikes produced by responding fibers or sufficiently broad distribution of those fibers could overcome this decrease, thus preventing loudness adaptation. Loudness would decrease over time, however, if the number or synchrony of responding auditory nerve fibers were low at onset, or if the number or synchrony of responding auditory nerve fibers abnormally decreased over time due to, e.g., conduction block or dyssynchronous release of neurotransmitter by the inner hair cells. The present result shows that amplitude modulation can reduce loudness adaptation to tones that, when unmodulated, activate neither a high enough number nor a broad enough distribution of nerve fibers to maintain a loudness percept. This suggests that neural synchrony in the auditory nerve fibers and in the auditory cortex plays a significant role in loudness adaptation.

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