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Review Article

Stress across timescales: differential effects of acute and chronic stress on auditory processing and perception

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ABSTRACT

Stress has long been linked to auditory dysfunction and altered sound perception, but its effects are often discussed without fully distinguishing between acute and chronic stress timescales. Here, we integrate evidence that acute and chronic stress engage partly overlapping but temporally distinct mechanisms and produce different auditory outcomes. Acute stress triggers rapid neuromodulatory and hormonal responses that can transiently alter auditory filtering, deviance detection, attention, and sound sensitivity, often biasing the system toward heightened salience and vigilance. In contrast, chronic stress is associated with more persistent changes in auditory attention, loudness perception, sound tolerance, and central auditory coding, reflecting longer-term alterations in circuit function and baseline regulation. In this review, we examine how acute and chronic stress influence auditory processing and perception in adulthood across human and animal studies, drawing together behavioral, physiological, and circuit-level findings. We highlight candidate mechanisms including glucocorticoid and catecholamine signaling, shifts in excitation–inhibition balance, altered thalamocortical and amygdala-related interactions, and stress-driven plasticity within mature auditory networks. We argue that chronic stress should not be viewed simply as a prolonged version of acute stress, but rather as a distinct state that may involve longer-lasting changes in auditory coding, baseline regulation, and perceptual weighting. Organizing stress effects by timescale may provide a framework for interpreting stress-related listening difficulties and for informing clinical conditions such as tinnitus, hyperacusis, and impaired hearing in noisy environments.

1. Introduction

Stress alters brain function across distinct timescales, producing markedly different behavioral outcomes. A looming work deadline, for example, can boost motivation and sharpen efficiency in the short term, whereas prolonged overload gradually erodes productivity and flexibility. These everyday experiences mirror underlying biological processes: acute stress mobilizes rapid neuromodulatory and hormonal signals that bias circuits toward vigilance and rapid responding, often at the expense of top-down control (Hermans et al., 2014; Joëls and Baram, 2009; Qi and Gao, 2020). By contrast, chronic or repeated stress gradually remodels synapses across multiple brain regions, weakening cognitive control and flexibility (Koolhaas et al., 2011; Marin et al., 2011; McGirr et al., 2020; McKlveen et al., 2016). These divergent effects reflect a shift from short-lived, challenge-evoked changes to a slower, cumulative state of dysfunction (Godoy et al., 2018). While stress is typically studied in the context of cognitive domains such as memory and learning (Dias-Ferreira et al., 2009; Gilabert-Juan et al., 2013). However, stress may also have important consequences for auditory perception, where ongoing neuromodulatory states and top-down control continuously tune sensory gain, salience, and noise filtering. As a result, stress could alter auditory coding and the perceptual weighting of sounds, changing how information is filtered, prioritized, and linked to affective meaning. These influences could differ between acute and chronic stress, reflecting distinct underlying mechanisms and timescales.

Converging evidence indicates that stress can alter both sensory perception and the cognitive operations that govern perception (attention, salience assignment, and learning), with effects spanning central auditory processing and perceptual experience. Acute stress can produce transient shifts consistent with a hypervigilant sensory mode—for example, brief increases in sound sensitivity or distractibility that

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typically normalize after stress recovery (Elling et al., 2011; Hasson et al., 2013; Hermans et al., 2014; Ma et al., 2015). Chronic stress, by contrast, often yields cumulative behavioral consequences, including sustained difficulty with auditory attention and listening in noise, or, in some cases, a blunted responsiveness consistent with exhaustion of adaptive capacity (Hasson et al., 2013; Joëls et al., 2007; Kaganovski and Resnik, 2025; Pérez et al., 2013; Pérez-Valenzuela et al., 2016).

A timescale-based framework could help clarify why stress-related auditory changes range from rapid, reversible shifts in sensory filtering and vigilance to persistent alterations in loudness perception, sound tolerance, and central auditory coding. In this review, we focus on how acute and chronic stress shape central auditory processing and perception in adulthood, well beyond the critical period. For a complementary perspective, Rosen and Huyck recently reviewed the effects of developmental and early-life stress on auditory processing (Rosen and Huyck, 2026). Here, we examine behavioral and perceptual outcomes (e.g., auditory attention, discrimination, and loudness perception) together with neural measures of central auditory processing across species, drawing on human studies (psychophysics, EEG/MEG, fMRI, and related approaches) and animal work (systems-level recordings and circuit-level manipulations). Our goal is to organize prior work on stress and auditory processing around timescale, linking acute versus chronic stress effects to candidate mechanisms—such as neuromodulatory and glucocorticoid signaling, shifts in excitation–inhibition balance, and stress-driven plasticity—and considering how this framework may inform clinical phenomena such as tinnitus, hyperacusis, and stress-related listening difficulties.

To examine how stress-related changes in auditory processing vary with stress duration, we identified relevant literature through PubMed and Google Scholar searches using combinations of terms related to acute and chronic stress, auditory processing and perception, auditory cortex, inferior colliculus, auditory thalamus, and central auditory plasticity. Empirical studies were included if they were peer-reviewed, quantitative in nature, published in English, and examined the effects of acute or chronic stress in adults. We focused on central auditory processing beyond the cochlea and auditory nerve, with particular emphasis on higher auditory pathways. Studies were prioritized when they directly assessed auditory neural processing or auditory-related perceptual outcomes in humans or animal models. Representative studies were selected to cover major stress paradigms, timescales, species, and levels of analysis relevant to the mature auditory system. See Table 1 for a complete list of all included studies.

2. Acute and chronic stress: timescales and measurement

Operationally, acute and chronic stress are distinguished less by subjective intensity and more by the temporal structure of the stress response and the measurements that are valid at each timescale. Acute stress is anchored to a time-locked stressor with a clear onset, enabling phase-specific sampling of fast autonomic responses (e.g., heart rate, pupil dilation, blood pressure, locomotor arousal) that rise within seconds to minutes, alongside delayed HPA output (cortisol/corticosterone), which typically peaks tens of minutes after onset and resolves during recovery (Herman et al., 2016; Joëls and Baram, 2009); accordingly, interpretation depends strongly on whether measurements are taken in the immediate, delayed, or recovery window. Chronic or repeated stress is defined by recurrent exposure over days to weeks, where the critical readouts shift from a single peak response to changes in baseline regulation, including altered diurnal glucocorticoid rhythms, modified reactivity to a standardized probe stressor (habituation vs sensitization), and cumulative allostatic-load indices such as weight trajectory and sleep disruption (Joëls et al., 2007; Koolhaas et al., 2011; McEwen, 2017, 2007). Experimentally, these differences matter: acute paradigms must carefully control timing relative to stress induction, as the immediate, delayed, and recovery phases can diverge, whereas chronic stress studies require validation of stress-marker profiles, as habituation or sensitization may emerge over repeated exposures.

An important caveat in comparing stress effects across studies is that “stress” is not a single physiological state. Physical stressors such as cold pressor stress, psychosocial stressors such as mental arithmetic or socially evaluative tasks, anticipatory threat, restraint stress, social defeat, and direct corticosterone or hydrocortisone manipulation differ in the relative engagement of sympathetic arousal, HPA-axis activity, glucocorticoid signaling, pain, threat appraisal, controllability, and inflammatory or plasticity-related processes (Joëls and Baram, 2009; Koolhaas et al., 2011; McEwen, 2017). Therefore, auditory outcomes across paradigms should not be interpreted as directly interchangeable. Rather, we compare findings across these paradigms to identify convergent and divergent principles, while considering the timescale of exposure, the physiological systems engaged, and the level of the auditory pathway examined.

3. Acute stress: transient and heterogeneous effects on auditory processing

3.1. Acute stress effects on auditory processing in humans

Acute stress has heterogeneous effects on auditory perception in humans, and the direction of these effects appears to depend on the stressor type, the physiological response engaged, the timing of measurement, and the auditory process being tested. Physical pain stressors that strongly recruit sympathetic arousal often show rapid, transient changes in early auditory filtering (Elling et al., 2011; Ermutlu et al., 2005; Johnson and Adler, 1993), whereas psychosocial and anticipatory stressors more often implicate cortisol-related variability and effects that extend into deviance detection and attentional control (Rojas-Thomas et al., 2023; Simoens et al., 2007; White and Yee, 1997).

Physical stressors, such as the Cold Pressor Test (CPT), have been suggested to transiently disrupt auditory sensory gating (Johnson and Adler, 1993). Auditory sensory gating refers to the reduced neural response to repeated or redundant sensory input, and is thought to limit the transmission of irrelevant information to higher cortical centers (Boutros and Belger, 1999; Cromwell et al., 2008). Auditory sensory gating is commonly assessed using a paired-click P50 paradigm. In this paradigm, two identical clicks are typically presented 500 ms apart, and the P50 auditory evoked potential, a positive deflection occurring approximately 50 ms after sound onset, is measured in response to each click. Under intact gating, the P50 response to the second stimulus is attenuated relative to the response to the first stimulus, reflecting suppression of redundant auditory input (Adler et al., 1998; Freedman et al., 1991; White and Yee, 1997). CPT has been reported to impair this gating response, reflected in reduced P50 suppression—that is, less attenuation of the response to the second click—consistent with reduced pre-attentive inhibition and less efficient filtering of redundant input (Johnson and Adler, 1993). This effect was short-lived, showing partial resolution by 30 min.

Ermutlu et al. similarly found diminished P50 gating alongside increased N100 and P3a amplitudes, with no significant effect on mismatch negativity (MMN), during cold stress. MMN is an auditory event-related potential elicited when an infrequent “deviant” sound violates a regular pattern of repeated “standard” sounds (Näätänen et al., 1978). Because MMN can be observed even when participants are not actively attending to the sounds, it is commonly interpreted as an index of automatic or pre-attentive auditory change detection (Garrido et al., 2009; Näätänen et al., 2007). Thus, the absence of a significant MMN effect suggests that cold stress altered early sensory gating and later involuntary orienting responses, without necessarily disrupting the automatic detection of auditory deviance (Ermutlu et al., 2005). Consistent with effects on early attentional processing, Elling et al. reported that acute CPT transiently attenuated the auditory negative difference, a component associated with selective attention, indicating increased distractibility or reduced selective filtering shortly after stress

Table 1
List of all auditory-related studies included in the review, detailing stress methods and key information. Pyr, pyramidal cells; PV, parvalbumin-positive interneuron; SOM, somatostatin-positive interneuron.

Study	Species	Stress paradigm	Auditory measure	Stress type	Main finding	Caveat
Johnson and Adler, 1993	Humans	Cold Pressor Test	P50 sensory gating	Acute	Transient reduced P50 suppression	Variance among subjects; no clear control.
Ermutlu et al., 2005	Humans	Adapted Cold Pressor Test	P50, N100, P3a, MMN	Acute	Diminished P50 gating, increased N100 and P3a, no significant MMN effect	Milder stress than the conventional test.
Elling et al., 2011	Humans	Cold Pressor Test	Auditory negative difference and MMN	Acute	Attenuated auditory negative difference	Effects appear HPA independent.
White and Yee, 1997	Humans	Oral arithmetic stress	P50 sensory gating	Acute	Attenuated P50 response to the conditioning stimulus	No measure of cortisol or NE; no effect on test stimulus.
Simoens et al., 2007	Humans	Trier Social Stress	MMN and N1/P2	Acute	Reduced MMN to duration deviants	Effects don't generalize; only male subjects.
Rojas-Thomas et al., 2023	Humans	Montreal Imaging Stress Task	MMN, P3a/P3b, pupil response, target detection	Acute	Increased MMN, decreased P3b	Only male subjects.
Cornwell et al., 2007	Humans	Anticipatory threat of shock	MMN	Acute threat state	Enhanced deviance-related responses in regions including amygdala and auditory cortex	Threat-related, not stress-specific.
Fehm-Wolfsdorf et al., 1993	Humans	Five-min speech	Auditory reflex thresholds	Acute	Elevated auditory reflex thresholds	The effect depends on cortisol elevation
Beckwith et al., 1983	Humans	Hydrocortisol administration	Tone detection	Acute	Reduced auditory sensitivity for higher frequencies	Exogenous cortisol, not a full stress response.
Lei et al., 2014	Rats	Dexamethasone topical administration	Single unit activity in the auditory cortex	Acute	Temporarily increased neural responses to pure-tone stimuli. Effects on spontaneous activity were heterogeneous.	Exogenous cortisol, not a full stress response.
Ma et al., 2015	Rats	Restraint stress	Cortical response to pure tones and click trains	Acute	Responses enhanced in ~33% of neurons, suppressed in ~11%, unchanged in the remainder	Only male animals; mixed findings.
Mazurek et al., 2012	Rats	Sound/vibration repellent + additional handling	Inferior colliculus gene expression	24-h	Post-stress transcriptional changes in the IC, including immediate gene upregulation followed by later cFos downregulation	Auditory/perceptual outcomes not directly measured; females only.
Mazurek et al., 2009	Rats	Rodent repellent that produces sound/vibration + other stressors	ABR and DPOAEs. Expression of the HPA-axis-associated genes in the inferior colliculus.	24-h	Relative numbers of GR transcripts were unchanged in the auditory periphery but up-regulated 3 h after stress in the IC.	Peripheral changes may confound central effects; females only.
Wang and Liberman, 2002	Mice	Restraint stress	Noise-induced hearing loss	Two 12-h restraint stress bouts	Acute restraint stress reduced noise-induced hearing loss;	Males only; auditory/perceptual outcomes not measured.
Henkin and Daly, 1968	Humans	Adrenal cortical insufficiency	Auditory detection sensitivity	Endocrine condition	Low cortisol levels were associated with above-average auditory sensitivity, which normalized after cortisol restoration	Endocrine disorder, not conventional stress.
Hasson and Canlon, 2013	Human	Stroop test and cold pressor exposure	Uncomfortable loudness levels	Acute	Women with high levels of emotional exhaustion become more sensitive to sound after an acute stress task.	Causality is difficult to establish.
Bisharat et al., 2025	Mice	Repeated restraint stress	Loudness categorization; auditory cortical cell-type responses	Chronic	Reduced loudness perception; suppressed sound-evoked activity in pyramidal and PV neurons, enhanced responses in SOM interneurons	Mouse-level stress physiology not correlated with cortical effects.
Pérez et al., 2013	Rats	Restraint stress	Auditory attention, cortical synaptic transmission	Chronic	Stress reduced the frequency of spontaneous inhibitory postsynaptic currents and miniature IPSC in the auditory cortex	Male rats only; behavior and recordings in separate groups; non-standard behavioral scoring.
Li et al., 2023	Mice	Social defeat	A1 CaMKII+ activity, PV activity, thalamocortical input to the auditory cortex	Chronic	Reduced activation of CaMKII+ neurons; resilient mice showed higher activated PV fraction, susceptible mice showed reduced PV firing; linked to increased MGB excitatory drive	Depression-focused mechanism, not stress-specific; males only.
Manohar et al., 2023	Rats	Exogenous corticosterone was administered in water bottles	Startle, sound avoidance, auditory cortex activity	Chronic endocrine manipulation	Increased avoidance of loud sound-paired contexts; upregulation of glucocorticoid receptor expression in the auditory cortex.	Glucocorticoid-dominant manipulation; not physiological multisystem stress; male rats only.
Kaganovski and Resnik, 2025	Mice	Restraint stress	Changes in NE during sound presentation	Chronic	Repetitive stress strongly attenuates NE responses to high-intensity sounds in the auditory cortex	Cortical processing and perception not measured
Pérez-Valenzuela et al., 2016	Rats	Restraint stress	Monoamines levels in the auditory cortex.	Chronic	Lower norepinephrine levels in the auditory cortex	Postmortem monoamine measures; males only.
Bangel et al., 2017	Humans	Post-traumatic stress disorder (PTSD) patients	Mismatch negativity	Chronic	Enhanced mismatch negativity	PTSD-focused, not stress-specific; males only.

exposure and before any measurable rise in salivary cortisol (Elling et al., 2011). These findings are consistent with evidence that the CPT reliably activates the sympathetic–adrenal–medullary axis, whereas activation of the HPA axis is more variable (Buchanan et al., 2006). Indeed, several studies using the CPT have reported only modest cortisol increases (Duncko et al., 2007; McRae et al., 2006; Schwabe et al., 2008), suggesting weaker or less consistent HPA-axis engagement. This makes the CPT a useful contrast to psychosocial stress paradigms, which more consistently recruit the HPA axis.

In contrast, psychosocial stressors have more complex and prolonged effects, which may depend in part on an individual's cortisol-responder status. White and Yee found that mental arithmetic stress disrupted normal P50 suppression (White and Yee, 1997), indicating that early sensory gating can also be stress-sensitive in socially evaluative or performance contexts. Extending beyond gating, Simoens et al. reported that psychosocial stress selectively reduced MMN to duration deviants, most clearly in participants who showed a cortisol increase (Simoens et al., 2007), consistent with stress hormones weakening the sensory-memory/prediction processes needed for automatic detection of timing changes. Rojas-Thomas et al. observed a different profile following a socially evaluative arithmetic stressor: MMN increased while later attention-related components (P3a/P3b) and target-evoked pupil responses decreased, accompanied by worse target detection (Rojas-Thomas et al., 2023). Taken together, these findings suggest that acute psychosocial stress can bias processing toward enhanced bottom-up monitoring while compromising controlled attentional selection, with the direction of MMN effects likely depending on what feature is probed (e.g., duration), task demands (passive vs active oddball), and the timing of measurement relative to stress induction.

Other types of psychosocial stressors, such as anticipatory anxiety, can also shape auditory deviance processing. For example, the threat of electric shock enhances neural responses to stimulus deviance in regions including the amygdala and auditory cortex, consistent with a hypervigilant state in which salience signals exert stronger influence over auditory processing (Cornwell et al., 2007).

Acute stress may additionally shift auditory sensitivity endpoints in a cortisol-dependent manner. Fehm-Wolfsdorf et al. reported elevated auditory reflex thresholds in individuals with high cortisol responses, requiring louder stimuli to elicit a reflex (Fehm-Wolfsdorf et al., 1993). Consistent with this direction, Beckwith, et al. found reduced auditory sensitivity for certain frequencies in healthy men after cortisol administration (Beckwith et al., 1983). Although this approach isolates glucocorticoid signaling rather than modeling the full physiological stress response, it suggests that elevated glucocorticoid tone can dampen auditory sensitivity (Simoens et al., 2007).

Taken together, the heterogeneous acute-stress findings likely reflect differences in stress physiology, auditory endpoint, task demands, stimulus feature, and measurement timing. Cold pressor paradigms often probe the immediate sympathetic-dominant phase, sometimes before measurable cortisol increases, whereas psychosocial stress effects may depend more strongly on cortisol responder status. In addition, P50 gating, MMN, P3 responses, reflex thresholds, and tone-detection measures index different stages of auditory processing, from early filtering and deviance detection to attentional control and auditory sensitivity. Thus, these measures need not change in the same direction. Acute-stress effects should therefore be interpreted in relation to the specific physiological response engaged, the auditory feature tested, the task context, and the time point measured after stress induction.

3.2. *Acute stress effects on auditory processing in animal models*

Acute stress has multifaceted effects on auditory perception in animal models, mirroring the diversity of effects observed in humans, while also allowing these changes to be traced mechanistically from peripheral sensitivity to central encoding and molecular programs across the auditory pathway.

Work at the level of the auditory cortex (AC) shows that stress hormones can rapidly reshape central activity. Lei et al. demonstrated that topical application of the glucocorticoid receptor agonist dexamethasone (DEX) to AC increased tone-evoked firing and broadened tuning curves, with peak effects 20–30 min post-application and a return toward baseline by ~90 min. Effects on spontaneous activity were heterogeneous: units with low pre-application firing tended to increase their firing rates, whereas units with high pre-application firing tended to decrease (Lei et al., 2014), such that the mean spontaneous firing rate averaged across the population did not differ significantly pre- versus post- DEX. Complementing these pharmacological manipulations, Ma et al. showed that acute restraint stress also alters AC responses in a heterogeneous manner: tone- and click-evoked responses were transiently enhanced in ~33% of neurons, suppressed in ~11%, and unchanged in the remainder (Ma et al., 2015). Together, these findings suggest that acute stress can rapidly reweight auditory cortical encoding, but not in a uniform “gain-up” manner across neurons.

Such cortical shifts in the auditory cortex may reflect, at least in part, compensation to stress-driven adaptations that arise earlier in the central auditory pathway and propagate upward. Consistent with this idea, Mazurek et al. reported post-stress transcriptional changes in the inferior colliculus (IC), including immediate upregulation of multiple genes (iNos, Sod2, Ngfb, Hsf1, Tnfa, Tnfar, Sp) followed by a later downregulation of cFos mRNA at six hours post-stress (Mazurek et al., 2012). This evolving molecular response in a key midbrain hub provides a plausible substrate for altered input statistics reaching the cortex during stress.

Mazurek et al. reported that a single 24-h episode of emotional stress in Wistar rats produced transient auditory hypersensitivity, reflected by lower ABR thresholds and increased ABR amplitudes (Mazurek et al., 2009). Because ABR reflects integrated activity from cochlear and early brainstem relays, these results align with the idea that stress can bias auditory processing across multiple nodes, not only within the cortex.

Finally, acute restraint stress can paradoxically protect against peripheral damage: Wang & Liberman, found that two 12-h bouts of restraint stress reduced noise-induced hearing loss if the acoustic trauma occurred within 2 h of stress exposure (Wang and Liberman, 2002). This protective window coincided with peak corticosterone levels, underscoring that acute glucocorticoid signaling can have dual, context-dependent consequences—reshaping central responsiveness while, under specific temporal conditions, engaging short-lived protective mechanisms against peripheral damage.

Summary: Across humans and animal models, acute stress produces rapid, time-dependent shifts in auditory processing that can emerge at multiple levels, from early sensory gating and deviance detection to reflex thresholds and cortical encoding. In most paradigms, these changes are transient and track the acute stress response, typically returning toward baseline over minutes to hours as autonomic and endocrine signals resolve. Importantly, the apparent heterogeneity across P50 gating, MMN, P3 responses, auditory sensitivity, and attention likely reflects differences in cortisol responder status, task demands, auditory feature tested, and timing of measurement after stress induction. Sympathetic-dominant paradigms tend to yield rapid changes in early filtering and selective attention, whereas cortisol-linked responses are more variable and may extend into predictive processing, auditory sensitivity, and controlled attentional selection (Fig. 1**,** middle).

4. Chronic stress: sustained shifts in auditory processing and perception

4.1. *Chronic stress effects on auditory processing in humans*

Compared with acute stress, chronic stress is harder to model experimentally in humans because it unfolds over weeks to months and is shaped by ongoing context, coping, and sleep disruption (Koolhaas

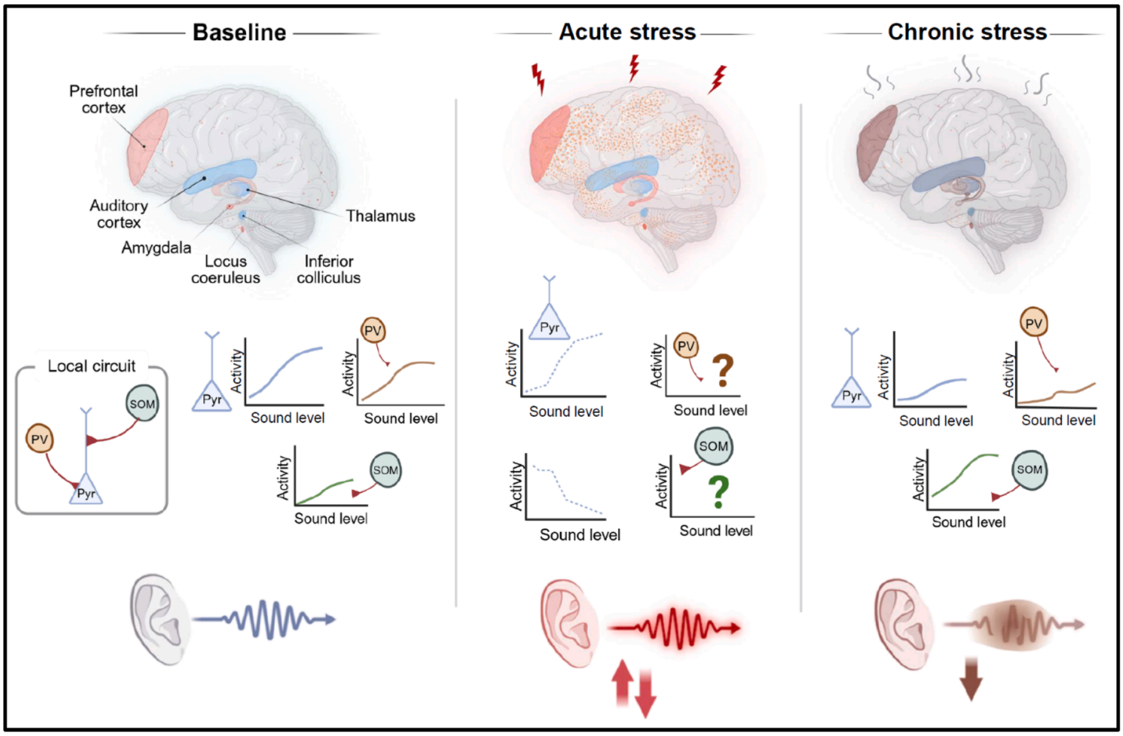

Fig. 1. Effects of acute and chronic stress on auditory circuits and perception. Schematic summary of stress-dependent changes across auditory and stress-related networks, local auditory cortical microcircuits, and perceptual outcome. **Left, baseline:** under non-stress conditions, coordinated activity across the auditory cortex, prefrontal cortex, thalamus, amygdala, and locus coeruleus supports stable sound coding, with balanced interactions between pyramidal neurons and PV and SOM interneurons. **Middle, acute stress:** acute stress rapidly recruits brain-wide stress systems and transiently reconfigures auditory cortical processing, altering pyramidal sound-evoked responses (Ma et al., 2015); Depending on the paradigm and stressor, it can bias the system toward enhanced or reduced sound-evoked activity. Effects on specific interneuron populations remain unknown. **Right, chronic stress:** prolonged stress is proposed to be associated with longer-lasting changes in network and microcircuit function. In restraint-stress models, this includes increased sound-evoked activity in SOM cells and reduced sound-evoked activity in Pyr and PV cells (Bisharat et al., 2025). These changes may contribute to altered population-level sound representations and perception.
Pyr, pyramidal cells; PV, parvalbumin-positive interneuron; SOM, somatostatin-positive interneuron.

et al., 2011). As a result, much of the human evidence comes from naturalistic cohorts (e.g., occupational burnout and emotional exhaustion) or from studies that manipulate or index glucocorticoid tone rather than "stress" per se.

Early endocrine work supports a link between sustained glucocorticoid levels and auditory sensitivity. Henkin and Daly reported that patients with adrenal cortical insufficiency and extremely low cortisol showed above-average auditory detection sensitivity, which returned toward typical levels when cortisol was restored (Henkin and Daly, 1968).

More recent studies of prolonged psychological strain point to changes not only in perceptual experience but also in auditory filtering. Hasson and Canlon found that women reporting high emotional exhaustion, a state closely associated with chronic stress, exhibited elevated loudness discomfort levels and hyperacusis-like symptoms, consistent with reduced tolerance to everyday sounds (Canlon et al., 2013; Hasson et al., 2013).

4.2. *Chronic stress effects on auditory processing in animal models*

Animal models are essential for chronic stress research because they allow controlled, repeated stress exposure and direct measurement of circuit mechanisms that cannot be practically accessed in humans. Across paradigms, chronic stress produces measurable behavioral phenotypes in auditory processing, including changes in loudness perception and auditory attention (Bisharat et al., 2025; Pérez et al., 2013). In mice, repeated restraint stress over several days impairs perceptual performance, with chronically stressed animals showing reduced loudness perception reflected in shifted psychometric thresholds during loudness categorization (Bisharat et al., 2025). In rats, chronic stress also disrupts auditory attention in a two-alternative choice task, consistent with impaired allocation of attention, altered salience, or motivational processing of a behaviorally relevant sound (Pérez et al., 2013).

These behavioral effects are accompanied by robust changes in auditory-cortical inhibitory and excitatory activity. Chronic restraint stress reduces inhibitory synaptic activity in the primary auditory cortex, including decreases in miniature inhibitory postsynaptic currents (Pérez et al., 2013). Bisharat et al. reported that chronic restraint stress suppresses sound-evoked activity in pyramidal neurons and PV interneurons while enhancing responses in SOM interneurons, a pattern that could reshape gain control and timing precision in auditory cortical representations (Bisharat et al., 2025).

A study of social defeat stress further points to thalamocortical–interneuron pathways as a candidate mechanism, with outcome differences that map onto stress susceptibility and the mode of endocrine manipulation. Following social defeat, Li et al. found reduced activation of CaMKII+ neurons in the primary cortex layers 2/3 and 4, alongside stress-phenotype-dependent PV effects: resilient mice showed a higher proportion of activated PV cells, whereas susceptible mice showed reduced PV firing (Li et al., 2023). They further linked these inhibitory changes to increased excitatory drive from the medial geniculate body, highlighting a pathway by which thalamic input may contribute to stress-related changes in cortical inhibition.

Finally, when corticosterone levels were elevated exogenously through pharmacological manipulation, rather than through natural stress responses, and glucocorticoid receptor expression was upregulated in the auditory cortex, animals developed behavioral signs consistent with loudness hyperacusis, including exaggerated startle responses to moderate sounds and increased avoidance of sound-paired contexts (Manohar et al., 2023). Together, these findings suggest that "chronic stress effects" are not unitary: broad multisystem stress paradigms, such as restraint or social defeat, may produce different auditory outcomes than glucocorticoid-dominant manipulations, which unnaturally isolate a single component of the stress response. Depending on the paradigm and physiological systems engaged, chronic exposure may bias auditory processing toward blunted loudness perception in some contexts or heightened sound reactivity in others, with interneuron- and thalamocortical-dependent mechanisms emerging as plausible candidate substrates.

Summary: Across humans and animal models, chronic stress is associated with sustained changes in both auditory processing and perception. In people, evidence largely comes from long-term real-world strain and endocrine-linked measures, pointing to altered auditory sensitivity, weakened sensory gating, and reduced tolerance to everyday sounds. In animals, controlled chronic stress paradigms show longer-lasting deficits in loudness-related behavior and auditory attention, accompanied by changes in auditory cortical and thalamocortical processing. Together, these findings are consistent with the possibility that chronic stress alters how sound is encoded and selected for perception (Fig. 1 right).

5. Candidate mechanisms: neuromodulation, glucocorticoids, and plasticity

Acute and chronic stress are likely to influence auditory processing through partly overlapping neuroendocrine, neuromodulatory, and neuroplastic pathways, though the timescales and consequences of these changes differ substantially. Acute stress rapidly activates the sympathetic nervous system and the hypothalamic–pituitary–adrenal axis, leading to the release of catecholamines (e.g., norepinephrine, dopamine) and glucocorticoids (cortisol in humans, corticosterone in rodents) (Joëls and Baram, 2009). In the auditory cortex, norepinephrine release during acute stress can modulate gain and signal-to-noise by shaping both excitatory and inhibitory synaptic transmission, supporting rapid sensory adaptation and salience-driven processing (Joëls et al., 2007; Joëls and Baram, 2009; Martins and Froemke, 2015). Acute stress also alters amygdala function, including increased amygdala engagement, stress-evoked changes in dendritic structure, and elevated norepinephrine release (Galvez et al., 1996; Grossman et al., 2020; Hegde et al., 2017). Through the amygdala's interactions with auditory thalamocortical circuits (LeDoux et al., 1991; Tovote et al., 2015; Yang et al., 2016), acute stress could transiently modulate auditory processing and the salience of sound.

By contrast, chronic stress recruits the same hormonal systems but over prolonged periods, with a greater emphasis on altered baseline tone and sensory responses (Joëls and Baram, 2009). Chronically stressed rats or mice show reduced NE levels in A1, or lower NE-sensor signals, compared with controls (Kaganovski and Resnik, 2025; Pérez-Valenzuela et al., 2016). Prolonged stress exposure is also associated in some models, with altered inhibitory activity and synaptic remodeling, including reductions in GABAergic interneuron activity and inhibitory synaptic transmission (Bisharat et al., 2025; Li et al., 2023; Pérez et al., 2013). Such changes could reduce the fidelity of auditory signal encoding and destabilize gain regulation, contributing to persistent distortions in loudness-related perception and auditory scene processing. Chronic stress also dysregulates amygdalar activity and output, particularly in the basolateral amygdala (BLA) (Gründemann et al., 2019; Lowery-Gionta et al., 2018; Munshi et al., 2020), which regulates prefrontal control. Repeated stress can disrupt BLA–prefrontal cortex communication by altering presynaptic glutamate release from BLA projections (Lowery-Gionta et al., 2018), while also promoting a pro-inflammatory milieu marked by increased amygdala neuronal and microglial activation and heightened anxiety-like behavior in adult rodents (Munshi et al., 2020). Because the amygdala interacts with the auditory cortex and thalamic pathways to assign affective salience and gate attention (Ciocchi et al., 2010; LeDoux et al., 1991; Tovote et al., 2015; Yang et al., 2016), these chronic changes may bias sound processing toward threat-weighted interpretations, reduce top-down filtering in noisy environments, and contribute to persistent shifts in sound tolerance and perceptual decisions.

Although acute and chronic stress engage overlapping modulatory pathways (catecholamines, glucocorticoids, and amygdala-centered salience circuits (Joëls and Baram, 2009)) that could reweight how sounds are encoded and selected for perception, chronic stress should not be viewed as simply a continuation of more acute stress. Acute stress primarily imposes a phasic, state-dependent reconfiguration that is often reversible as neuromodulatory and endocrine signals resolve, whereas repeated or prolonged stress may drive a shift in baseline regulation—altering receptor function and neuromodulatory tone, recruiting inflammatory processes, and triggering durable synaptic and inhibitory remodeling that could persistently bias sound processing and perception. This distinction matters conceptually and practically: acute effects may be best understood (and targeted) as transient changes in gain and salience, while chronic effects may reflect longer-lasting changes in circuit regulation and control that are less likely to normalize without restoring network balance and plasticity.

6. Clinical implications and future directions

Stress has previously been linked to auditory dysfunction, particularly in clinical contexts such as tinnitus, hyperacusis, and difficulties understanding speech in noisy environments (Canlon et al., 2013; Elarbed et al., 2021; Mazurek et al., 2012; Szczepek and Mazurek, 2021). Consistent with this, women with high emotional exhaustion show increased sound sensitivity following an acute stress task (Hasson et al., 2013), and patients with PTSD exhibit exaggerated neural responses to auditory deviance, including enhanced MMN and altered theta and upper-alpha activity, consistent with a hypervigilant sensory state (Bangel et al., 2017). These observations motivated a broader audiological approach that considers stress assessment and management alongside traditional hearing measures, particularly for patients who report stress-related symptom fluctuations (Baigi et al., 2011). However, further mechanistic and clinical studies are needed to determine whether stress causally contributes to the triggering or worsening of sensory disorders and, if so, which forms or timescales of stress are most relevant. It will also be important to establish whether stress acts as a driver or amplifier of auditory symptoms or instead emerges as a consequence of them.

Several important confounding and moderating factors should also be considered when interpreting stress–audition studies. Baseline hearing status and prior noise exposure can influence auditory thresholds, evoked potentials, ABR measures, and sound tolerance independent of stress. In animal studies, baseline auditory function is often easier to assess or control experimentally, for example, in studies measuring ABR thresholds, sound-evoked cortical responses, or noise-induced hearing loss, although this is not uniformly reported across all paradigms. In human studies, some experiments explicitly define the participant group or health status, such as healthy men in cortisol-administration studies (Beckwith et al., 1983), patients with adrenal cortical insufficiency (Henkin and Daly, 1968), women with high emotional exhaustion (Hasson et al., 2013), or patients with PTSD (Bangel et al., 2017), but baseline hearing status, prior noise exposure, medication use, and affective state are not always consistently controlled or reported. Sleep disruption, anxiety, depression, medication use, and general arousal level may further alter auditory attention, sensory gating, and physiological stress responses, making it difficult to isolate stress-specific effects. Sex differences are also important because stress responsivity, glucocorticoid dynamics, and vulnerability to auditory symptoms can differ between males and females (Bale and

Epperson, 2015; Furman et al., 2022). Future studies should therefore assess and report these variables whenever possible.

Key open questions include the need for longitudinal studies that jointly track stress markers, auditory outcomes, and major confounding variables such as baseline hearing status, prior noise exposure, sleep, affective state, medication use, sex, and general arousal level. Additional mechanistic work is also needed to determine where stress signals act first (cochlea/brainstem vs. cortex) and how timing shapes their effects. Another major question is which baseline physiological and neural signatures predict whether acute stress will transiently sharpen or destabilize listening, and which profiles forecast resilience versus susceptibility to chronic stress-related auditory dysfunction. Finally, future translational studies could test whether targeting specific stress pathways (e.g., beta-adrenergic blockade or modulation of the glucocorticoid receptor) can prevent or reverse stress-related auditory deficits without compromising adaptive arousal responses.

7. Conclusion: toward prediction and resilience

Taken together, the literature supports a view of audition as a state-dependent system in which stress-related neuromodulatory and endocrine signals dynamically reshape gain, filtering, and salience assignment. Importantly, chronic stress is not simply an amplified version of acute stress: repeated exposure can shift baseline regulation and plasticity, potentially leading to reconfiguration of inhibitory control and perceptual calibration. The field now needs principled, time-resolved models that explain the transition from acute, reversible state shifts to chronic, cumulative dysfunction—linking autonomic and endocrine markers to circuit state, and perceptual outcomes. Clinically, defining this transition could identify intervention windows and inform treatments for tinnitus, hyperacusis, and stress-linked listening difficulties. Building these bridges will clarify when stress modulates the auditory system for rapid responding and when it pushes auditory processing toward maladaptive instability.

CRediT authorship contribution statement

Lior Dor: Writing – original draft, Visualization. **Jennifer Resnik:** Writing – review & editing, Writing – original draft, Conceptualization.

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