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Opinion

# Neural adaptation to changes in self-voice during puberty

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**The human voice is a potent social signal and a distinctive marker of individual identity. As individuals go through puberty, their voices undergo acoustic changes, setting them apart from others. In this article, we propose that hormonal fluctuations in conjunction with morphological vocal tract changes during puberty establish a sensitive developmental phase that affects the monitoring of the adolescent voice and, specifically, self–other distinction. Furthermore, the protracted maturation of brain regions responsible for voice processing, coupled with the dynamically evolving social environment of adolescents, likely disrupts a clear differentiation of the self-voice from others' voices. This socioneuroendocrine framework offers a holistic understanding of voice monitoring during adolescence.**

## Highlights

In adolescence, one's own voice changes significantly due to a surge of pubertal hormones, resulting in a distinctive voice signature. A person's unique voice signature signals one's own individuality and becomes increasingly relevant as social networks expand.

While these puberty-related changes contribute to the development of a unique voice signature, they also initiate a sensitive period of voice monitoring.

We propose that, together with hormonal changes, the protracted development of brain regions engaged in voice monitoring and a dynamically changing social environment might affect how the self-voice and others' voices are discriminated. A socioneuroendocrine framework is needed to comprehensively examine how we perceive and differentiate ourselves through our voice as well as how alterations in these capacities can lead to pathologies related to self–other distinction.

## A person's journey toward developing a unique voice: a balance of distinctiveness and vulnerability

There are approximately eight billion speaking humans in the world, and yet there are no two voices alike. Just like a fingerprint, every voice is acoustically unique and encodes a myriad of personal characteristics, including physical and personality cues. As an exceptional means to express oneself [1], the unique voice is linked to bodily self-consciousness [2,3] and a validated marker of altered self-processing [4,5].

Before **puberty** (see Glossary), child voices are acoustically quite similar [6] and more difficult to differentiate from one another than adult voices [7]. During puberty, an individual's own voice undergoes the most significant acoustic changes compared with other developmental stages [8], which lead to a unique adult voice signature. As a window into one's own body (e.g., perceived age, body size [9,10]; masculinity or femininity [10]), the voice also mirrors the dramatic pubertal changes in physical appearance [11]. Despite the profound impact of puberty on vocal development, research has nevertheless mostly focused on the adult voice.

The transition from a child voice to a unique adult voice during puberty raises compelling questions about the underlying sensorimotor adaptations throughout this period of heightened plasticity and rapid neural, behavioral, and biological change [12]. In the following sections, we aim to elucidate the components and roles of **voice monitoring**. We then review the existing evidence that highlights the inherent instability of the voice monitoring system in puberty, particularly in light of a surge in **gonadal hormones**, associated with fluctuations in voice control and nonverbal voice decoding that gradually improve toward the end of puberty. We then explore how this evidence aligns with studies indicating that phenomena such as voice hearing often manifest during early adolescence, coinciding temporally with pubertal changes of one's voice. Finally, we outline potential venues for future research, emphasizing the importance of implementing and testing predictions derived from a socioneuroendocrine framework of voice monitoring (see Outstanding questions). This framework accounts for the natural

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variability in developmental trajectories leading to a unique voice signature and can be applied to study both typical and atypical voice development.

## The building blocks of voice monitoring

Voice monitoring encompasses the intricate processes involved in perceiving and controlling one's own voice feedback in vocal production [13]. It serves multiple and partly dissociable functions [14]. It plays a crucial role in regulating and adjusting ongoing vocal production [15]. Voice monitoring also contributes to distinguishing between self-generated and externally generated sound leading to motor-induced sensory attenuation [13]. Voice monitoring further enables individuals to infer or confirm self-relevant information through vocal cues (e.g., personality traits [16]) and thereby contributes to a more abstract self-representation [16].

### Sensory prediction and adaptation

The perception and control of one's own voice feedback during vocal production rely on an internal **forward model**, which enables the brain to rapidly anticipate the sensory consequences of an action (Box 1). This forward model operates through a feedforward corticopontocerebellar loop that predicts the sensory consequences of vocalizations and a feedback cerebellothalamocortical loop, which compares expected feedback with actual input [17–19]. A distinction between self-generated and externally generated auditory stimuli hinges on the extent to which sensory feedback deviates from the expected input. When there is a match, self-generated voice feedback is perceived as less salient and associated with attenuated sensory cortical responses compared with unexpected sensory feedback or externally produced voices [20]. The ability to differentiate between afferent sensory input resulting from one's own actions (reafferent) and input from external sources (exafferent) lays the foundation for **self–other distinction** in both perception and action [21].

### Internal self-voice representation

Expected sensory consequences during speaking are influenced by the **internal representation** of how one's voice sounds compared with other voices. This expectation is shaped by long-term

### Box 1. Sensorimotor foundations of self–other voice distinction

Voice production is subserved by sensorimotor loops that scaffold one's sense of agency and ownership [102], as well as higher-order levels of the self [103]. It takes the whole brain to monitor the voice. Voice monitoring in volitional vocalization relies on a widespread system of functionally connected brain regions, including not only the right temporal voice areas but also cortical motor and speech planning areas, as well as subcortical regions such as the cerebellum (for a review, see [13]).

During vocalization, an efference copy of the motor command is projected from motor planning to auditory cortical regions via the cerebellum to cancel out impending activity to the expected stimulus. The forward model relies on two key processes: the prediction of the sensory consequences of one's own action and the detection of mismatches between predicted and actual sensory input. When actual sensory feedback matches a prediction, self-voice input is attenuated, as seen in N1 amplitude suppression in the electroencephalogram (EEG) [95], reduced activation of the auditory cortex in functional magnetic resonance imaging (fMRI) [20], and reduced ratings of perceived intensity [104] relative to other voices.

Such indices of motor-induced suppression are considered an implicit proxy of self–other voice distinction [13]. Specifically, the comparison between predicted and incoming sensory signals allows self-attribution when there is a match and external attribution in the case of a considerable mismatch. When expected and actual sensory consequences mismatch (e.g., when sensory feedback is acoustically altered, delayed, replaced with the voice of another speaker, or physically perturbed during vocal production), an error signal is generated. Although sensory attenuation in the auditory cortex decreases, activity in the right inferior frontal gyrus increases, signaling the detection of unexpected self-voice changes [20].

Notwithstanding, these sensorimotor mechanisms are not immune to illusion. For example, auditory feedback that is temporally and phonetically congruent with motor and somatosensory feedback from the articulators can lead to illusory ownership over a stranger's voice [14]. Sound briefly travels outside of the body when it leaves the vocal apparatus before reentering the auditory system. This feature can make the voice more vulnerable to environmental influences and external misattributions.

### Glossary

**Auditory verbal hallucinations:** colloquially referred to as 'hearing voices,' are externally attributed voice percepts in the absence of corresponding sensory input, with perceptual qualities that are indistinguishable from real voices. They are understood to exist along a continuum ranging from benign and transitory to those requiring medical care.
**Foreign accent syndrome:** a rare medical condition where patients show altered speech patterns that perceptually resemble a non-native accent.
**Forward model:** a computational process that allows predicting the sensory consequences of an action and comparing predicted and actual sensory feedback.
**Gonadal steroid hormones:** a set of hormones produced by the gonads – including androgens, estrogens, and progestogens – that play a key role in the reproductive system. When secreted at puberty, they drive the development of secondary sex characteristics and the emergence of sexual dimorphism.
**Hypothalamic–pituitary–gonadal axis:** a coordinated system comprising the hypothalamus, pituitary gland, and gonadal glands that is critical for sexual development and reproduction. It is active during prenatal and early postnatal life, becomes quiescent throughout childhood until approximately the age of 10, and is responsible for the onset and regulation of puberty.
**Internal self-voice representation:** an internal template of how one's own voice should sound, which relies on stored knowledge as a function of one's personal experience of speaking. Partly distinct voice representations for self and others are relevant to preserve a sense of self even in acoustically challenging situations.
**Puberty:** a developmental process in adolescence, characterized by rapid neural, behavioral, and biological change. Its onset is marked by the reactivation of the hypothalamic–pituitary–gonadal axis, on average at 10.85 years in girls and 11.79 years in boys, despite large interindividual variability.
**Self–other voice distinction:** the successful tagging of voice input as self- (versus externally) generated. Self–other voice distinction is considered to be facilitated by motor-related sensory predictions and/or by a robust internal representation of the self-voice as distinct from other voices.

stored knowledge derived from personal experiences with both self and other voices, accumulated in the context of vocal communication [20]. Evidence suggests that the self-voice is distinctly represented from other voices, as shown by different patterns of neural activation [22,23]. This distinct representation enables the detection of differences between self and other voices even in the absence of efferent information from motor regions while passively listening to them.

Acoustic voice changes during puberty likely necessitate a recalibration of the internal sensory prediction and representation of one's voice. Given the substantial variations in pubertal timing, tempo, and hormone levels among male and female adolescents, as well as within individuals of the same pubertal stage and sex [24], it is anticipated that individual differences will emerge in the neural adaptations to changes in the self-voice during puberty.

**Sensitive period:** a developmental window characterized by heightened plasticity, when the function of neural regions or circuits is tuned in an experience-expectant manner. Its onset and offset timing are malleable. During a sensitive period, experiences shape an organism's phenotypic development to a greater extent than during other stages.
**Voice monitoring:** the perception, control, and adaptation of one's own voice feedback during vocal production that relies on comparing what one expects to hear and what one actually hears.

## Sensitive periods in voice monitoring

A **sensitive period** denotes a developmental window marked by heightened plasticity, during which the function of brain regions or neural circuits is tuned in an experience-expectant manner [25]. The initiation of such plasticity is complex and relies on a combination of biological, genetic, and experiential factors [26], thus leading to variability in the specific timing of the onset and offset of each sensitive period among individuals [26].

In infancy and early childhood, crucial anatomical and hormonal changes shape the organization and maturation of the voice-processing circuitry. The early postnatal surge of sex steroids, also known as minipuberty (Box 2), profoundly influences vocal and early language development, contributing to significant sex differences in parameters such as fundamental frequency (F0) and melody of spontaneous crying [27,28], baby babbling [29], and expressive vocabulary [30]. These effects are comparable with the impact of pubertal changes on brain development [31].

Puberty onset marks the reactivation of the **hypothalamic–pituitary–gonadal axis** [32], leading to a surge in **gonadal steroid hormones** such as testosterone and estradiol. This hormone surge triggers a period of heightened vocal plasticity and sets in motion sexually dimorphic trajectories in voice acoustics that could arguably result in sex differences in self–other voice discrimination [33]. Although both male and female adolescents experience increased vocal tract length and volume, acoustic voice changes are more pronounced in males [34]. Differentiation of male and female F0 and formant frequency patterns typically begins around age 11 [6,35] and becomes fully

### Box 2. Socioneuroendocrine interactions in prenatal and early postnatal human life: a first sensitive period?

The hypothalamic–pituitary–gonadal axis is active not only in adulthood but also during prenatal (between 8 and 24 weeks of gestation) and early postnatal life, becoming largely quiescent throughout childhood [105]. Around 1 week of age, there is an increase in the gonadotropins luteinizing hormone (LH) and follicle-stimulating hormone (FSH), which typically peak between week 1 and 3 months and then decline by 6 months of age [106]. As a result, gonadal hormone production is activated in a sex-specific manner. Whereas LH levels are higher in boys, FSH levels increase in girls and remain elevated up to 3–4 years of life [106]. Moreover, estradiol levels fluctuate in girls and decline in the second year of life [106]. The prenatal and postnatal hormone surge seems to significantly impact early vocal production in the evolution of spoken language. Gonadal hormones affect the development and function of the auditory-vocal system in human infants similarly to other vocal learners such as songbirds [107]. For example, peripheral estradiol concentration at 4 weeks of life predicts the ability to produce complex melody patterns in a baby's crying [108], as well as articulatory skills at 20 weeks [29], whereas serum testosterone levels have negative effects [29].

Brain areas that play a key role in voice production and perception, such as the right superior temporal sulcus, also show major maturational changes during this early period of life (e.g., [109]). Several genes are already asymmetrically expressed toward the right side in the human temporal region between 12 and 14 weeks of gestation [110], when the hypothalamic–pituitary–gonadal axis is also active. These rapid structural changes may underlie the acquisition of specialization for voice processing in the right superior temporal cortex found in 7-month-old infants [111].

established around age 15 [35]. After a secondary descent of the larynx, male adolescents undergo a significant lowering of pitch, resulting in deeper and more resonant voices than female adolescents [36] or male children [37]. On average, pubertal pitch change in males is completed within 0.5 to 4.0 years [38]. Some male adolescents experience a sudden breakdown in the modulation of the voice (i.e., an involuntary change in pitch and quality) through a surge in testosterone [39]. Conversely, voice changes in females are more gradual [40], with pitch becoming about one-third lower than that of female children [37].

The hormone-triggered physiological changes culminate in the development of a unique and acoustically distinctive voice, along with an idiosyncratic vocal identity by the end of puberty. However, voice change also brings about increased temporary instability and within-subject variability in temporospectral voice parameters, including F0 [41] and jitter [42]. Adolescents undergoing voice changes may experience transient feelings of being out of synch with their voice, reporting difficulties in controlling vocal loudness, pitch, and vocal range [43] during this period of transition. For some, this could resemble the experience of hearing another person talk when speaking [43]. The puberty-related changes that contribute to the formation of a unique voice signature may arguably start a sensitive period of voice monitoring. This heightened period of plasticity may come at the cost of disrupting self–other distinction, which is an established marker of hallucination proneness [13]. In the subsequent sections, we delve into how hormonal, neural, and social changes may influence the processing of self and other voices, potentially opening or extending a sensitive period of voice monitoring (Figure 1).

### Hormonal changes

There is compelling evidence indicating that sex steroids dynamically modulate vocal learning and communication across various species, including research on birdsong [44], human infants [29], and human adults [45] (Box 2). Adolescence, characterized by heightened sensitivity to steroid-dependent organization [46], represents a particularly significant period for these effects. As the brain is a target organ for steroid hormones [46], their influence extends to the modulation of the neural substrates involved in feedback-based vocal control [45].

Specific and enduring effects of pubertal hormones have been observed in brain regions supporting voice monitoring, including areas in the temporal lobe [47,48] and the cerebellum [49], as well as white matter tracts connecting these regions [50]. Moreover, sex steroids can impact the development of beta-band oscillations in the primary motor cortex independent of chronological age [51]. These oscillations are crucial for mediating top-down motor–auditory interactions during sensory attenuation of self-generated sensory input [52], serving as an implicit proxy for self-other distinction.

The effects of gonadal hormones on the adolescent brain appear to be independent of chronological age [53] but vary by sex [47,48,54] and differ quantitatively and qualitatively from effects observed in prepubertal or postpubertal stages [46]. Additionally, sex steroids influence plasticity mechanisms, affecting the opening and closure of sensitive periods [55–57]. Axonal myelination, a key regulator of sensitive period timing in neurocognitive development, is subject to modulation by pubertal hormones [57]. For instance, synaptic pruning accelerates around puberty onset in temporal brain areas engaged in auditory processing [58]. Moreover, adolescent increases in brain-derived neurotrophic factor, a trigger for plasticity relevant to sensitive periods [26], may also be regulated by sex steroids [59–61]. Puberty thus plays a pivotal role in the neural organization and behavioral maturation of voice monitoring by influencing facilitative factors of plasticity (e.g., GABAergic inhibition [55]) and inhibitory ones (e.g., myelination [57]) that drive the maturation of sensitive periods.

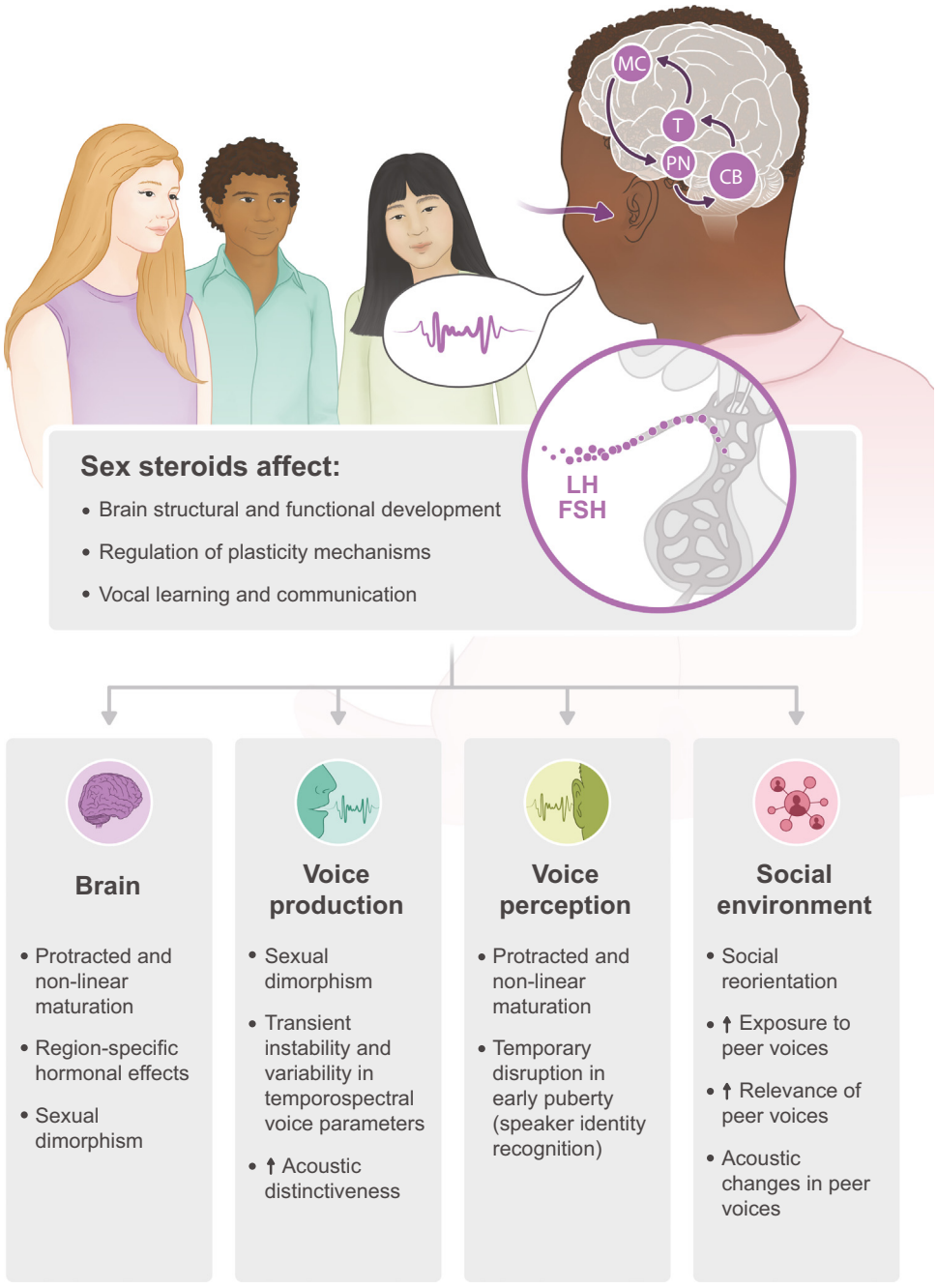


Figure 1. Puberty as a sensitive period for voice monitoring in the context of extensive hormonal, neural, and social changes. A chain of maturational events unfolding in puberty affect voice monitoring, triggered by the reactivation of the hypothalamic–pituitary–gonadal axis. These include changes related to brain maturation, voice production and perception, and social reorientation. Together, these changes can temporarily affect self–other voice distinction. In the brain circuit depicted for the male adolescent, among the thalamocortical projections, only transmission to the motor cortex is highlighted for illustrative purposes (see also [112]). Region-specific hormonal effects on brain development were documented in relation to both pubertal development and circulating levels of sex hormones [47,48,50]. Sexual dimorphism reflects the influence of both organization and activation effects of gonadal hormones [47,48,54]. Notation: ↑, increased; Abbreviations: CB, cerebellum; MC, motor cortex; PN, pontine nuclei; T, thalamus.

### Protracted maturation of brain regions engaged in voice monitoring

Distinct neural systems undergo development at varying rates across childhood and adolescence. Brain regions involved in voice monitoring follow a protracted developmental trajectory extending into late adolescence, rendering them susceptible to extended periods of environmental influences [62]. For example, the cerebellar lobes reach their peak volume later than the cerebrum [54,63] and later in males (15.6 years) than in females (11.8 years) [54], whereas the auditory temporal cortex continues to mature beyond other association areas throughout pubertal development [48,64–66]. In a sample of ten children (aged 8–9 years), ten adolescents (aged 14–15 years), and ten young adults of mostly right-handed participants (90% per age group), the right (but not left) superior temporal cortex exhibited increased anatomical and functional intersubject variability from childhood to young adulthood [67]. This variability might be associated with individual differences in exposure to voices [67] because of social reorientation in adolescence [68]. Adolescence also marks a critical period for the formation of new functional networks, with the developing brain functional connectome following a puberty-dependent nonlinear trajectory [69,70]. Functional connectivity between sensory and motor regions strengthens into early adulthood, indicative of enhanced neural efficiency [71,72]. Furthermore, corticosubcorticocortical interfaces, such as those between the inferior frontal gyrus and right cerebellar regions, undergo maturation in vocal production tasks [73].

However, the maturation of neural circuitry engaged in voice monitoring may follow a nonlinear trajectory, with sensorimotor mechanisms experiencing periods of regression in early puberty [74], potentially influenced by sex hormones [51,75]. For example, this could be manifested in a performance dip in adolescents compared with younger children and adults [76]. During this phase, there may be a notable discrepancy between motor and sensory signals, disrupting sensorimotor loops established before puberty. This manifests as greater variability in the activation patterns of oral articulators [77] and longer compensatory response latencies to F0 perturbations in auditory feedback during early compared with late puberty [78]. Moreover, sensory attenuation of self-voice feedback is reduced in early puberty [79], likely affecting the accuracy of predicting the sensory consequences of speaking, particularly during periods of dramatic vocal change [80]. This discrepancy between motor and sensory signals can impact sensorimotor control, adaptation, and the perceived sense of agency in voice production, leading to reports of limited control over vocal loudness, pitch, and vocal range among adolescents undergoing voice changes [43]. Furthermore, the temporary disruption in **self–other voice distinction** may occur as labeling self-generated voice feedback becomes more challenging when feedback is altered [81].

### Social reorientation

Dramatic vocal changes leading to a unique voice signature during puberty coincide with significant shifts in social environments. While parents and caregivers play primary roles in children's social worlds, adolescents increasingly orient themselves toward peers, resulting in an expansion of their social network size [68]. This shift in social orientation brings about notable changes in the vocal environment. More than in earlier developmental stages, adolescents experience exposure to and learning from a diverse array of voices within their own age group, making peer voices more relevant [82].

However, adolescents also encounter substantial within-subject acoustic variability in peer voices because these typically experience pubertal changes themselves. This increased acoustic voice instability could influence how adolescents form and update individual and averaged representations of others' voices. Research suggests that voices of familiar and unfamiliar speakers are encoded and compared with an internal template or stored representation. This template represents the acoustic average of all voices of the same gender encountered previously and is shaped

by both long-term and recent perceptual experience [83]. Nevertheless, introducing within-speaker variability in voice identity tasks has been found to reduce recognition accuracy in adult listeners [84]. In adolescence, the acoustic instability of peer voice representations is expected to impact speaker discrimination and recognition, particularly in early puberty [76]. This instability may also hinder self–other voice discrimination, which is influenced by the acoustic distance between voices [33]. A less distinct representation of one's own voice from others should result in a less efficient self–other voice discrimination. Due to the role of identity processing and speaker knowledge in speech comprehension [85,86] and social trait inference [87], altered self–other voice discrimination might affect social interactions.

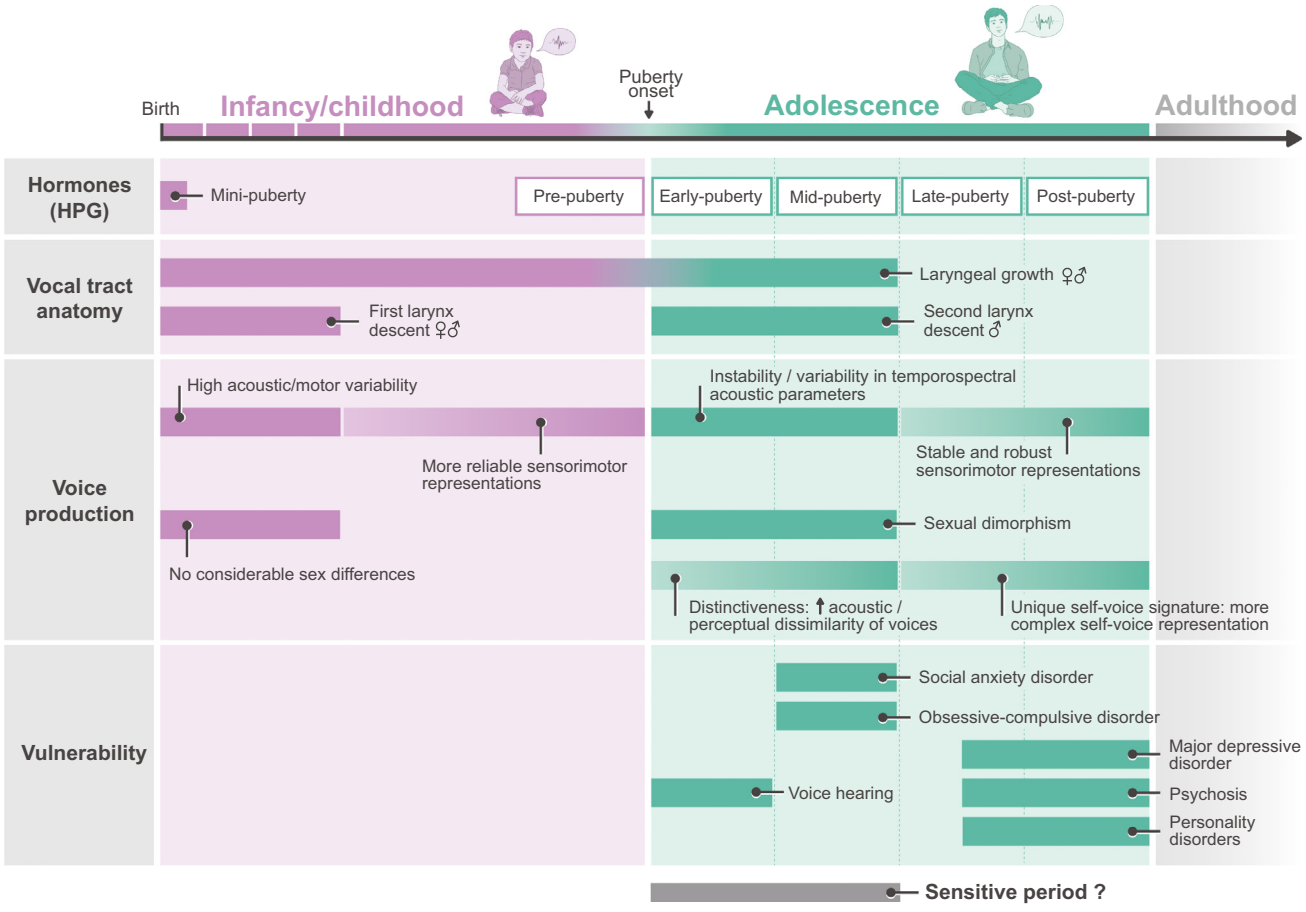


Figure 2. Pre- and postpubertal voice changes in relation to hormonal and anatomic changes and vulnerability for psychopathology. Hormones: the hypothalamic–pituitary–gonadal axis is active during prenatal and early postnatal life and is reactivated in puberty. Five pubertal stages can be identified using the Tanner stages or the self-assessed Pubertal Development Scale: pre-, early, mid-, late, and postpuberty, each lasting approximately 2 years [113]. Girls generally reach puberty earlier than boys, but there is large interindividual variability [113]. Vocal tract anatomy: the primary descent of the larynx occurs in humans in both sexes at approximately 3 months of age and continues until 3–4 years. In males, there is a secondary descent of the larynx in puberty [36]. Voice production: there is large acoustic and motor variability in vocal production in early childhood, but sensorimotor representations become more reliable over multiple productions. When voices change in puberty, there is increased instability and variability in temporal and spectral acoustic parameters in voice production. At the end of puberty, adolescents show a sexually dimorphic and unique voice signature, as well as a more complex self-voice representation. Vulnerability: adolescence is a period of enhanced vulnerability for sex-biased psychopathologies. In particular, voice hearing is most prevalent in the transition from prepubertal to pubertal stages, temporally coinciding with pubertal changes in one's voice. Note: in the lifespan development timeline (top), the first 4 years of life are demarcated. Abbreviation: HPG, hypothalamic–pituitary–gonadal axis.

## Translational aspects of a sensitive period for voice monitoring in puberty

The evidence presented earlier suggests that the natural disruption triggered by changes in hormone levels, brain maturation, and interpersonal environments prompt a recalibration of sensorimotor loops driving adaptation to acoustic changes in the self-voice. A transient alteration of voice abilities in early puberty precedes significant improvements in complex skills such as speaker recognition [76] or the ability to express one's identity and social traits through volitional voice control [88]. The maturation of these abilities extends throughout adolescence into adulthood at a more protracted rate than face processing [89]. This stabilization during the vocal transition from childhood to adulthood is underpinned by an increase in the reliability and effectiveness of neural signaling at the circuit and network levels, which is a functional outcome of a sensitive period in development [26].

However, this developmental phase also presents a specific time window for an atypical reorganization to emerge and interfere with adaptive functioning (Figure 2). Adolescence is a period of enhanced vulnerability for psychopathology, with altered self–other distinction central to numerous psychiatric disorders whose prevalence increases in adolescence or in the transition to young adulthood, such as obsessive-compulsive disorder [90], psychosis [91], or personality disorders [92]. Adolescence appears to be a unique period of vulnerability for developing **auditory hallucinations**, which may occur as a normal variation in development [93]. Voice hearing is most prevalent during the transition from prepubertal to pubertal stages compared with adulthood [94], temporally coinciding with pubertal changes in one's voice. Previous studies in adults suggest a link between hallucination proneness and hyperprocessing of self-voice feedback [95] and blurred self–other voice distinction [96], similar to patterns observed in early puberty.

Atypical social experience, brain maturation, and pubertal timing may enhance vulnerability for auditory hallucinations by altering the way adolescents experience the sensory consequences of speaking and their ability to distinguish these sensations from those expressed by other agents. For example, social isolation, often associated with voice hearing in adolescence [97], could imply reduced exposure to one's own voice feedback and to peer voices, disrupting vocal learning as well as the robustness and stability of voice representations. Studies in rats have shown that social isolation leads to reductions in myelin thickness and myelin-associated glycoprotein [98], known as plasticity braking factors. Moreover, hallucination proneness has been linked to perturbations in circuit function and aberrant functional connectivity between brain regions such as the cerebellum and temporal cortex [13] and the right temporal and frontal brain regions [99]. Gonadal hormones have also been associated with an increased risk for psychosis [100]. These perturbations could lead to long-lasting outcomes that may not occur if they were experienced later in life.

It is important to note that the timing of both pubertal and neural changes differs for male and female adolescents. Differences in the timing of interactions between pubertal hormones and brain maturation could lead to sex differences in the risk for disorders or symptoms associated with altered self–other distinction, such as the higher prevalence of auditory hallucinations in male than female adolescents [101].

## Concluding remarks

The voice serves as a unique expression of individual identity. Puberty instigates the most dramatic changes in the voice across the lifespan, affecting every developing individual. These voice changes can also have enduring effects on one's sense of self. We propose a sensitive developmental voice model that delineates a cascade of maturational events unfolding in puberty,

### Outstanding questions

How does the perceptual representation of the self-voice change from puberty onset to the end of adolescence? How does this new representation align with the acoustic changes in one's own voice at any given time?

Do other-voice representations change as a function of size and composition (peers versus caregivers) of one's social network?

How do dynamic changes in the self-voice representation impact sensory suppression to voice feedback throughout pubertal development?

Voices may change slowly and gradually in some adolescents and quickly in others. Voice change may also be precocious (e.g., in case of central precocious puberty) or delayed for organic reasons (e.g., functional hypogonadotropic hypogonadism). How do these differences impact adaptation to dynamic acoustic changes in one's own voice?

How do changes in voice monitoring and the functional reorganization of the underlying neural circuitry in puberty relate to levels of circulating sex hormones?

How does the self-voice representation differ from representations of familiar and unfamiliar peer voices throughout adolescence? Are differences constant over time, or do they change dynamically as a function of levels of circulating sex hormones?

If the voice is a key defining feature of our individuality, what happens when the voice connected to the self radically changes? Do dynamic changes in voice acoustics and in self-voice representation predict changes in one's self-concept? What are the underlying neural mechanisms?

The self-voice is a multimodal percept. How does the changing body affect self-voice perception and vice-versa?

Can differences in self-voice perception explain why auditory hallucinations remit in most adolescents and persist in others?

influencing voice monitoring potentially at the expense of disrupting self–other voice distinction and heightening vulnerability to phenomena such as auditory hallucinations.

The socioneuroendocrine framework we propose points to several directions for further research on voice monitoring in adolescence (see Outstanding questions). Investigating these processes will enable the identification of optimal timing and targets for intervention and prevention strategies aimed at altering the trajectories of interactions between pubertal, brain, and behavioral development, thereby mitigating impaired self–other voice monitoring. Elucidating how changes in one's voice can impact one's sense of self may offer a basis for investigating a broader spectrum of conditions characterized by persistent alterations in the sound of one's voice, such as those following total laryngectomy, unilateral vocal fold paralysis, expressive aphasia, **foreign accent syndrome**, or cross-sex hormone therapy.

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## Declaration of interests

The authors declare no competing interests.

## References

1. Sidtis, D. and Kreiman, J. (2012) In the beginning was the familiar voice: personally familiar voices in the evolutionary and contemporary biology of communication. *Integr. Psychol. Behav. Sci.* 46, 146–159
2. Orepic, P. *et al.* (2022) Breathing affects self-other voice discrimination in a bodily state associated with somatic passivity. *Psychophysiology* 59, e14016
3. Roel Lesur, M. *et al.* (2021) The monologue of the double: allocentric reduplication of the own voice alters bodily self-perception. *Conscious. Cogn.* 95, 103223
4. Candini, M. *et al.* (2018) The lost ability to distinguish between self and other voice following a brain lesion. *NeuroImage Clin.* 18, 903–911
5. Orepic, P. *et al.* (2023) Experimentally-evidenced personality alterations following meningioma resection: a case report. *Cortex* 168, 157–166
6. Perry, T.L. *et al.* (2001) The acoustic bases for gender identification from children's voices. *J. Acoust. Soc. Am.* 109, 2988–2998
7. Cooper, A. *et al.* (2020) Identifying children's voices. *J. Acoust. Soc. Am.* 148, 324–333
8. Markova, D. *et al.* (2016) Age-and sex-related variations in vocal-tract morphology and voice acoustics during adolescence. *Horm. Behav.* 81, 84–96
9. Sorokowski, P. *et al.* (2024) Voice-based judgments of sex, height, weight, attractiveness, health, and psychological traits based on free speech versus scripted speech. *Psychon. Bull. Rev.*, Published online January 18, 2024. https://doi.org/10.3758/s13423-023-02445-5
10. Pisanski, K. and Rendall, D. (2011) The prioritization of voice fundamental frequency or formants in listeners' assessments of speaker size, masculinity, and attractiveness. *J. Acoust. Soc. Am.* 129, 2201–2212
11. Berger, T. *et al.* (2019) Speaking voice in children and adolescents: normative data and associations with BMI, Tanner stage, and singing activity. *J. Voice* 33, 580.e21–580.e30
12. Dorn, L.D. *et al.* (2019) Conceptualizing puberty as a window of opportunity for impacting health and well-being across the life span. *J. Res. Adolesc.* 29, 155–176
13. Pinheiro, A.P. *et al.* (2020) Cerebellar circuitry and auditory verbal hallucinations: an integrative synthesis and perspective. *Neurosci. Biobehav. Rev.* 118, 485–503
14. Zheng, Z.Z. *et al.* (2011) Perceiving a stranger's voice as being one's own: a 'rubber voice' illusion? *PLoS One* 6, e18655
15. Behroozmand, R. *et al.* (2012) Opposing and following vocal responses to pitch-shifted auditory feedback: evidence for different mechanisms of voice pitch control. *J. Acoust. Soc. Am.* 132, 2468–2477
16. Stern, J. *et al.* (2021) Do voices carry valid information about a speaker's personality? *J. Res. Pers.* 92, 104092
17. Todorović, S. *et al.* (2024) Cortico-cerebellar monitoring of speech sequence production. *Neurobiol. Lang.* 5, 701–721
18. Knolle, F. *et al.* (2012) The cerebellum generates motor-to-auditory predictions: ERP lesion evidence. *J. Cogn. Neurosci.* 24, 698–706
19. Runnqvist, E. *et al.* (2021) Cerebellar and cortical correlates of internal and external speech error monitoring. *Cereb. Cortex Commun.* 2, tgab038
20. Johnson, J. *et al.* (2021) Expectancy changes the self-monitoring of voice identity. *Eur. J. Neurosci.* 53, 2681–2695
21. Christoff, K. *et al.* (2011) Specifying the self for cognitive neuroscience. *Trends Cogn. Sci.* 15, 104–112
22. Kaplan, J.T. *et al.* (2008) The self across the senses: an fMRI study of self-face and self-voice recognition. *Soc. Cogn. Affect. Neurosci.* 3, 218–223
23. Iannotti, G.R. *et al.* (2022) EEG spatiotemporal patterns underlying self-other voice discrimination. *Cereb. Cortex* 32, 1978–1992
24. Dorn, L.D. (2006) Measuring puberty. *J. Adolesc. Health* 39, 625–626
25. Sisk, L.M. and Gee, D.G. (2022) Stress and adolescence: vulnerability and opportunity during a sensitive window of development. *Curr. Opin. Psychol.* 44, 286–292
26. Larsen, B. and Luna, B. (2018) Adolescence as a neurobiological critical period for the development of higher-order cognition. *Neurosci. Biobehav. Rev.* 94, 179–195
27. Wermke, K. *et al.* (2014) Sex hormone influence on human infants' sound characteristics: melody in spontaneous crying. *Biol. Lett.* 10, 20140095
28. Borysiak, A. *et al.* (2017) Fundamental frequency of crying in two-month-old boys and girls: do sex hormones during mini-puberty mediate differences? *J. Voice* 31, 128.e21–128.e28

29. Quast, A. *et al.* (2016) Baby babbling at five months linked to sex hormone levels in early infancy. *Infant Behav. Dev.* 44, 1–10
30. Kung, K.T.F. *et al.* (2016) Early postnatal testosterone predicts sex-related differences in early expressive vocabulary. *Psychoneuroendocrinology* 68, 111–116
31. Quast, A. (2021) The role of sex hormones in human language development. In *Neuroendocrine Regulation of Animal Vocalization: Mechanisms and Anthropogenic Factors in Animal Communication* (Rosenfeld, C.S. and Hoffmann, F., eds), pp. 121–132, Academic Press
32. Blakemore, S.-J. *et al.* (2010) The role of puberty in the developing adolescent brain. *Hum. Brain Mapp.* 31, 926–933
33. Orepic, P. *et al.* (2023) Bone conduction facilitates self-other voice discrimination. *R. Soc. Open Sci.* 10, 221561
34. Xue, S.A. *et al.* (2010) Vocal tract dimensional development of adolescents: an acoustic reflection study. *Int. J. Pediatr. Otorhinolaryngol.* 74, 907–912
35. Lee, S. *et al.* (1999) Acoustics of children's speech: developmental changes of temporal and spectral parameters. *J. Acoust. Soc. Am.* 105, 1455–1468
36. Fitch, W.T. and Giedd, J. (1999) Morphology and development of the human vocal tract: a study using magnetic resonance imaging. *J. Acoust. Soc. Am.* 106, 1511–1522
37. Abitbol, J. *et al.* (1999) Sex hormones and the female voice. *J. Voice* 13, 424–446
38. Hollien, H. *et al.* (1994) Longitudinal research on adolescent voice change in males. *J. Acoust. Soc. Am.* 96, 2646–2654
39. Busch, A.S. *et al.* (2019) Voice break in boys – temporal relations with other pubertal milestones and likely causal effects of BMI. *Hum. Reprod.* 34, 1514–1522
40. Zamponi, V. *et al.* (2021) Effect of sex hormones on human voice physiology: from childhood to senescence. *Hormones* 20, 691–696
41. Harries, M.L.L. *et al.* (1997) Changes in the male voice at puberty. *Arch. Dis. Child.* 77, 445–447
42. Sussman, J.E. and Sapienza, C. (1994) Articulatory, developmental, and gender effects on measures of fundamental frequency and jitter. *J. Voice* 8, 145–156
43. Killian, J.N. (1997) Perceptions of the voice-change process: male adult versus adolescent musicians and nonmusicians. *J. Res. Music. Educ.* 45, 521–535
44. Vahaba, D.M. and Remage-Healey, L. (2018) Neuroestrogens rapidly shape auditory circuits to support communication learning and perception: evidence from songbirds. *Horm. Behav.* 104, 77–87
45. Zhu, X. *et al.* (2016) Menstrual cycle phase modulates auditory-motor integration for vocal pitch regulation. *Front. Neurosci.* 10, 600
46. Sisk, C.L. and Zehr, J.L. (2005) Pubertal hormones organize the adolescent brain and behavior. *Front. Neuroendocrinol.* 26, 163–174
47. Herting, M.M. *et al.* (2012) The impact of sex, puberty, and hormones on white matter microstructure in adolescents. *Cereb. Cortex* 22, 1979–1992
48. Herting, M.M. *et al.* (2015) A longitudinal study: changes in cortical thickness and surface area during pubertal maturation. *PLoS One* 10, e0119774
49. Wierenga, L.M. *et al.* (2018) Unraveling age, puberty and testosterone effects on subcortical brain development across adolescence. *Psychoneuroendocrinology* 91, 105–114
50. Peper, J.S. *et al.* (2015) Short fused? Associations between white matter connections, sex steroids, and aggression across adolescence. *Hum. Brain Mapp.* 36, 1043–1052
51. Fung, M.H. *et al.* (2022) The development of sensorimotor cortical oscillations is mediated by pubertal testosterone. *Neuroimage* 264, 119745
52. Abbasi, O. and Gross, J. (2020) Beta-band oscillations play an essential role in motor–auditory interactions. *Hum. Brain Mapp.* 41, 656–665
53. Vijayakumar, N. *et al.* (2018) Puberty and the human brain: Insights into adolescent development. *Neurosci. Biobehav. Rev.* 92, 417–436
54. Tiemeier, H. *et al.* (2010) Cerebellum development during childhood and adolescence: a longitudinal morphometric MRI study. *Neuroimage* 49, 63–70
55. Piekarski, D.J. *et al.* (2017) Does puberty mark a transition in sensitive periods for plasticity in the associative neocortex? *Brain Res.* 1654, 123–144
56. Peper, J.S. *et al.* (2011) Sex steroids and connectivity in the human brain: a review of neuroimaging studies. *Psychoneuroendocrinology* 36, 1101–1113
57. Gabard-Durnam, L. and McLaughlin, K.A. (2020) Sensitive periods in human development: charting a course for the future. *Curr. Opin. Behav. Sci.* 36, 120–128
58. Vijayakumar, N. *et al.* (2021) The development of structural covariance networks during the transition from childhood to adolescence. *Sci. Rep.* 11, 9451
59. Hill, R.A. *et al.* (2012) Modulatory effects of sex steroid hormones on brain-derived neurotrophic factor-tyrosine kinase B expression during adolescent development in C57BL/6 mice. *J. Neuroendocrinol.* 24, 774–788
60. Purves-Tyson, T.D. *et al.* (2015) Adolescent testosterone influences BDNF and TrkB mRNA and neurotrophin–interneuron marker relationships in mammalian frontal cortex. *Schizophr. Res.* 168, 661–670
61. Sohrabji, F. and Lewis, D.K. (2006) Estrogen–BDNF interactions: implications for neurodegenerative diseases. *Front. Neuroendocrinol.* 27, 404–414
62. Sathyanesan, A. *et al.* (2019) Emerging connections between cerebellar development, behaviour and complex brain disorders. *Nat. Rev. Neurosci.* 20, 298–313
63. Giedd, J.N. *et al.* (2015) Child psychiatry branch of the National Institute of Mental Health longitudinal structural magnetic resonance imaging study of human brain development. *Neuropsychopharmacology* 40, 43–49
64. Gogtay, N. *et al.* (2004) Dynamic mapping of human cortical development during childhood through early adulthood. *Proc. Natl. Acad. Sci. U. S. A.* 101, 8174–8179
65. Bishop, D.V.M. *et al.* (2007) Maturation of the long-latency auditory ERP: step function changes at start and end of adolescence. *Dev. Sci.* 10, 565–575
66. Giedd, J.N. *et al.* (1999) Brain development during childhood and adolescence: a longitudinal MRI study. *Nat. Neurosci.* 2, 861–863
67. Bonte, M. *et al.* (2013) Development from childhood to adulthood increases morphological and functional inter-individual variability in the right superior temporal cortex. *Neuroimage* 83, 739–750
68. Wrzus, C. *et al.* (2013) Social network changes and life events across the life span: a meta-analysis. *Psychol. Bull.* 139, 53–80
69. Gracia-Tabuenca, Z. *et al.* (2021) Development of the brain functional connectome follows puberty-dependent nonlinear trajectories. *Neuroimage* 229, 117769
70. Vijayakumar, N. *et al.* (2021) A longitudinal analysis of puberty-related cortical development. *Neuroimage* 228, 117684
71. Váša, F. *et al.* (2020) Conservative and disruptive modes of adolescent change in human brain functional connectivity. *Proc. Natl. Acad. Sci. U. S. A.* 117, 3248–3253
72. Sanders, A.F.P. *et al.* (2023) Age-related differences in resting-state functional connectivity from childhood to adolescence. *Cereb. Cortex* 33, 6928–6942
73. Youssofzadeh, V. *et al.* (2018) fMRI connectivity of expressive language in young children and adolescents. *Hum. Brain Mapp.* 39, 3586–3596
74. Quatman-Yates, C.C. *et al.* (2012) A systematic review of sensorimotor function during adolescence: a developmental stage of increased motor awkwardness? *Br. J. Sports Med.* 46, 649–655
75. Killanin, A.D. *et al.* (2023) Testosterone levels mediate the dynamics of motor oscillatory coding and behavior in developing youth. *Dev. Cogn. Neurosci.* 61, 101257
76. Mann, V.A. *et al.* (1979) Development of voice recognition: parallels with face recognition. *J. Exp. Child Psychol.* 27, 153–165
77. Smith, A. and Zelaznik, H.N. (2004) Development of functional synergies for speech motor coordination in childhood and adolescence. *Dev. Psychobiol.* 45, 22–33
78. Scheerer, N.E. *et al.* (2013) The developmental trajectory of vocal and event-related potential responses to frequency-altered auditory feedback. *Eur. J. Neurosci.* 38, 3189–3200

79. Mathalon, D.H. *et al.* (2019) Deficient auditory predictive coding during vocalization in the psychosis risk syndrome and in early illness schizophrenia: the final expanded sample. *Psychol. Med.* 49, 1897–1904
80. Johnson, J.F. *et al.* (2022) Hypersensitivity to passive voice hearing in hallucination proneness. *Front. Hum. Neurosci.* 16, 859731
81. Fu, C.H.Y. *et al.* (2006) An fMRI study of verbal self-monitoring: neural correlates of auditory verbal feedback. *Cereb. Cortex* 16, 969–977
82. Abrams, D.A. *et al.* (2022) A neurodevelopmental shift in reward circuitry from mother's to nonfamilial voices in adolescence. *J. Neurosci.* 42, 4164–4173
83. Lavner, Y. *et al.* (2001) The prototype model in speaker identification by human listeners. *Int. J. Speech Technol.* 4, 63–74
84. Lavan, N. *et al.* (2019) How many voices did you hear? Natural variability disrupts identity perception from unfamiliar voices. *Br. J. Psychol.* 110, 576–593
85. Cheung, S. and Babel, M. (2022) The own-voice benefit for word recognition in early bilinguals. *Front. Psychol.* 13, 901326
86. Pinheiro, A.P. *et al.* (2016) Is this my voice or yours? The role of emotion and acoustic quality in self-other voice discrimination in schizophrenia. *Cogn. Neuropsychiatry* 21, 335–353
87. Jiang, X. *et al.* (2020) To believe or not to believe? How voice and accent information in speech alter listener impressions of trust. *Q. J. Exp. Psychol.* 73, 55–79
88. Zhang, Y.S. *et al.* (2019) Vocal state change through laryngeal development. *Nat. Commun.* 10, 4592
89. Morningstar, M. *et al.* (2020) Children and adolescents' neural response to emotional faces and voices: age-related changes in common regions of activation. *Soc. Neurosci.* 15, 613–629
90. Eddy, C.M. (2022) The transdiagnostic relevance of self-other distinction to psychiatry spans emotional, cognitive and motor domains. *Front. Psychiatry* 13, 797952
91. Assaf, R. *et al.* (2022) A functional neuroimaging study of self-other processing alterations in atypical developmental trajectories of psychotic-like experiences. *Sci. Rep.* 12, 16324
92. Sharp, C. *et al.* (2018) Adolescence as a sensitive period for the development of personality disorder. *Psychiatr. Clin.* 41, 669–683
93. Rubio, J.M. *et al.* (2012) Examining the course of hallucinatory experiences in children and adolescents: a systematic review. *Schizophr. Res.* 138, 248–254
94. Maijer, K. *et al.* (2018) Auditory hallucinations across the lifespan: a systematic review and meta-analysis. *Psychol. Med.* 48, 879–888
95. Pinheiro, A.P. *et al.* (2020) Changes in motor preparation affect the sensory consequences of voice production in voice hearers. *Neuropsychologia* 146, 107531
96. Pinheiro, A.P. *et al.* (2019) Self-voice perception and its relationship with hallucination predisposition. *Cogn. Neuropsychiatry* 24, 237–255
97. Parry, S. and Varese, F. (2021) Whispers, echoes, friends and fears: forms and functions of voice-hearing in adolescence. *Child Adolesc. Mental Health* 26, 195–203
98. Makinodan, M. *et al.* (2012) A critical period for social experience–dependent oligodendrocyte maturation and myelination. *Science* 337, 1357–1360
99. Fusar-Poli, P. *et al.* (2012) Predicting psychosis: meta-analysis of transition outcomes in individuals at high clinical risk. *Arch. Gen. Psychiatry* 69, 220–229
100. Huber, T.J. *et al.* (2005) Sex hormones in psychotic men. *Psychoneuroendocrinology* 30, 111–114
101. Spauwen, J. *et al.* (2003) Sex differences in psychosis: normal or pathological? *Schizophr. Res.* 62, 45–49
102. Salomon, R. (2017) The assembly of the self from sensory and motor foundations. *Soc. Cogn.* 35, 87–106
103. Krol, S.A. *et al.* (2019) Self-concept clarity and the bodily self: malleability across modalities. *Personal. Soc. Psychol. Bull.* 46, 808–820
104. Paraskevoudi, N. and SanMiguel, I. (2021) Self-generation and sound intensity interactively modulate perceptual bias, but not perceptual sensitivity. *Sci. Rep.* 11, 17103
105. Peper, J.S. and Dahl, R.E. (2013) Surging hormones: brain-behavior interactions during puberty. *Curr. Dir. Psychol. Sci.* 22, 134–139
106. Kuiri-Hänninen, T. *et al.* (2014) Activation of the hypothalamic-pituitary-gonadal axis in infancy: minipuberty. *Horm. Res. Paediatr.* 82, 73–80
107. Scarpa, G.B. *et al.* (2023) Estrogens rapidly shape synaptic and intrinsic properties to regulate the temporal precision of songbird auditory neurons. *Cereb. Cortex* 33, 3401–3420
108. Wermke, K. *et al.* (2018) From melody to words: the role of sex hormones in early language development. *Horm. Behav.* 104, 206–215
109. Glasel, H. *et al.* (2011) A robust cerebral asymmetry in the infant brain: the rightward superior temporal sulcus. *Neuroimage* 58, 716–723
110. Sun, T. *et al.* (2005) Early asymmetry of gene transcription in embryonic human left and right cerebral cortex. *Science* 308, 1794–1798
111. Grossmann, T. *et al.* (2010) The developmental origins of voice processing in the human brain. *Neuron* 65, 852–858
112. Abram, S.V. *et al.* (2022) Consider the pons: bridging the gap on sensory prediction abnormalities in schizophrenia. *Trends Neurosci.* 45, 798–808
113. Koopman-Verhoeff, M.E. *et al.* (2020) Classifying pubertal development using child and parent report: comparing the Pubertal Development Scales to Tanner staging. *J. Adolesc. Health* 66, 597–602