

1 **Hearing voices as a feature of typical and psychopathological experience**

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9 In press, *Nature Reviews Psychology*

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12 **Acknowledgements**

13 W.L.T was supported by an Australian National Health and Medical Research Council (NHMRC) New Investigator project grant (GNT1161609). C.F. was  
14 supported by Wellcome Trust grant WT108720.

15 **Author contributions**

16 W.L.T and P.M. are joint lead authors. All authors contributed to researching data for article, discussing the content, and writing, reviewing and editing the  
17 manuscript before submission.

18 **Competing interests**

19 The authors declare no competing interests.

20 **Peer review information**

21 *Nature Reviews XXX* thanks [Referee#1 name], [Referee#2 name] and the other, anonymous, reviewer(s) for their contribution to the peer review of this  
22 work.

23 **Publisher's note**

24 Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

25

26 **Abstract**

27 Hearing a voice in the absence of any speaker can be a significant feature of  
28 psychiatric illness, but is also increasingly acknowledged as an important aspect of  
29 everyday, non-pathological experience. This recognition has led to a growth of  
30 interest in voice-hearing in individuals without any psychiatric diagnosis, coupled with  
31 greater attention to the subjective experience of voice-hearing across diagnostic  
32 groups. Research has also focused on the overlap between some aspects of voice-  
33 hearing phenomenology and everyday experiences such as 'hearing' the voices of  
34 fictional characters and spiritual experience. In this Review, we synthesize research  
35 on the range of cognitive, neural, personal, and sociocultural processes that  
36 contribute to voice-hearing as it occurs in clinical, non-clinical, and everyday  
37 experience, with a particular emphasis on linking mechanism to phenomenology.  
38 Heterogeneous forms of voice-hearing can be understood in terms of differing  
39 patterns of association among underlying mechanisms. We suggest an approach to  
40 hallucinatory experience that sees it as partly continuous with everyday inner  
41 experience, but which is critical about whether continuity of phenomenology across  
42 the clinical/non-clinical divide should be taken to entail continuity of mechanism.

## 43 [H1] Introduction

44 Hearing a voice in the absence of any speaker has long been associated with severe  
45 mental illness. Voice-hearing has been proposed as a cardinal symptom of the  
46 schizophrenia spectrum disorders, with up to 80% of those with a diagnosis of  
47 schizophrenia reporting hearing voices at some point over their lifetime<sup>1</sup>. Voice-  
48 hearing is increasingly recognized as a feature of other psychiatric conditions,  
49 including bipolar and depressive disorders<sup>2</sup>, post-traumatic stress disorder<sup>3</sup>, and  
50 borderline personality disorder<sup>4</sup>. Syndromes with neurological origins, such as  
51 Parkinson's disease<sup>5</sup>, temporal lobe epilepsy<sup>6</sup>, and migraine<sup>7</sup>, also involve  
52 hallucinatory experiences (although not always in the auditory realm).

53 Notably, voice-hearing also occurs in a small but significant portion of the  
54 general population without a mental health diagnosis<sup>8</sup>. In the absence of distress,  
55 voice-hearing can be a significant feature of life for these individuals. Many features  
56 of voice-hearing also bear important commonalities with aspects of typical  
57 consciousness that would not be considered signs of mental disorder, such as  
58 engaging with fictional entities and imaginary companions<sup>9</sup>. This move towards  
59 recognizing that hearing voices is not necessarily pathological has led to the  
60 emergence of the term 'voice-hearing' as a replacement for the more medically-  
61 oriented 'auditory verbal hallucination', which has potential pejorative connotations.

62 Historically, there has been a focus on voices as primarily auditory  
63 experiences, likely due to the strong association between auditory hallucinations and  
64 the (controversial) diagnostic category of schizophrenia. However, the experience of  
65 voice-hearing is rarely confined exclusively to the auditory modality. A significant  
66 proportion of voices are described as 'soundless' voices, or as difficult to distinguish  
67 from regular thoughts<sup>10,11</sup>. Voice-hearing is also often accompanied by sensed  
68 presences (that is, feelings that someone is present in the immediate environment,  
69 often without clear sensory content) and experiences in other sensory modalities,  
70 including visual, somatic-tactile, olfactory, **autoscopie [G]**, and kinesthetic<sup>12</sup>. The

71 term voice-hearing thus encompasses a heterogeneous group of experiences that  
72 can vary widely in their auditory phenomenology. Although we favor this term, we will  
73 use the more conventional term ‘hallucination’ when discussing clinically significant  
74 experiences that are specific to non-auditory modalities.

75 In parallel with the growing recognition of the phenomenological complexity of  
76 voice-hearing, there has been an increase in research activity examining the etiology  
77 and maintenance of the voice-hearing experience. In contrast to the ‘deficit model’ of  
78 traditional biomedical psychiatry, which sees voice-hearing as resulting from a  
79 dysfunction in normal brain processes, voice-hearing is increasingly understood as a  
80 varied phenomenon which can emerge from distinct causal pathways. This variability  
81 has important implications for treatment in cases where voice-hearing experiences  
82 are distressing<sup>13</sup>. Research has accordingly begun to focus on voices as an  
83 interesting and important aspect of the diversity of typical human inner experience,  
84 rather than as necessarily the product of a ‘broken brain’.

85 In this Review, we examine the state of research on voice-hearing that  
86 incorporates experiences ranging from the distressing, disabling hallucinations of  
87 severe mental illness to everyday experiences. We begin by describing the varied  
88 kinds of voice-hearing experience, why they are sometimes distressing, and how  
89 their phenomenological features might differ across clinical and non-clinical  
90 categories. The full variety of voice-hearing experiences is then explored in light of  
91 the cognitive, neural, personal, and sociocultural mechanisms that have been  
92 implicated in its etiology, maintenance, phenomenological variability, and clinical  
93 significance, including some mechanisms likely to operate across sensory modalities.  
94 We examine the present state of the ‘continuum’ hypothesis, whereby such  
95 experiences exist on a spectrum from mild, transient experiences to frank clinical  
96 disorder, with significant variability in between. Finally, we illustrate how considering  
97 these three categories of clinical, non-clinical and everyday voice-hearing can help  
98 researchers understand the multiple possible pathways to voice-hearing, and

99 recommend an agenda for future research on this complex, varied, and significant  
100 feature of human experience.

101

## 102 **[H1] Phenomenology of voice-hearing**

103 The characteristics of voice-hearing in clinical and non-clinical cohorts have been  
104 well-documented<sup>8,11,14–26</sup>. Phenomenological features associated with voice-hearing  
105 can differ somewhat across these categories, but there are actually more shared  
106 commonalities than differences<sup>27</sup>. These phenomenological similarities and  
107 differences, organized into perceptual-sensory, cognitive, and affective domains, are  
108 summarized in Table 1. Prevalence estimates for voice-hearing in the general  
109 population vary, with lifetime figures between 0.6% ('Have you ever heard sounds,  
110 music, or voices which other people can't hear?'<sup>28</sup>) and 84% (at least one lifetime  
111 experience of 'brief, auditory hallucinations of the voice type', including sleep-related  
112 experiences)<sup>29</sup>. These estimates depend on what the participant believes is being  
113 asked of them, signalling a need for clear and consistent terminology.

114       Throughout this Review, we broadly categorize voices into three groups:  
115 clinical voices associated with distress or need for clinical care or treatment; non-  
116 clinical voices that are frequent and often recurring, but not associated with distress  
117 or mental health diagnoses; and everyday voices encompassing experiences such  
118 as inner speech or erroneously hearing one's name being called. We do not claim  
119 that these are 'true' categories or natural kinds; rather, we suggest that they provide  
120 a useful way to think about variability in these experiences. Also note that prevalence  
121 statistics cited here do not always clearly map onto one of these categories. For  
122 example, voice-hearing prevalence in the general population typically includes  
123 experiences described in both our non-clinical and everyday categories.

124

### 125 *[H3] Clinical voices*

126 In the clinical group (often, but not always, involving a diagnosis of  
127 schizophrenia), psychosis patients tend to report hearing speech, often with some  
128 non-verbal sounds, with voice onset typically in adolescence or early adulthood<sup>27</sup>.  
129 These experiences occur on a continuum ranging from fully to 'mixed' auditory  
130 percepts (comprising features common both to speech percepts and regular  
131 thoughts), to soundless or thought-like voices<sup>11</sup>. Perceptual-sensory characteristics of  
132 clinical voices tend to exhibit high variability from person to person as well as within a  
133 single person. Frequency and duration can fluctuate from constant, daily voices to  
134 those that only arise during acute psychosis; volume can range from a whisper to  
135 shouting, although conversational levels are common; clarity can vary from  
136 incoherent to sharp; a single voice can predominate, but multiple voices (up to a  
137 crowd) are possible; localization can comprise internal or external voices or both;  
138 linguistic complexity can differ, although repetition of phrases is typical; and second-  
139 or third-person address is most likely<sup>20,24,30,31</sup>.

140 Cognitive characteristics of clinical voices seemingly exhibit less variation  
141 relative to perceptual-sensory characteristics. Levels of conviction as to the veracity  
142 of voices tend to be high; there is limited perceived controllability; voices often have  
143 an autonomous, non-self quality; and beliefs regarding voice origin may incorporate a  
144 mixture of psychological and environmental factors. There are is some variability in  
145 when and how patients interact and comply with their voices, but vivid personification  
146 of known (or familiar) identities is common<sup>21,24,25</sup>.

147 Affective characteristics of clinical voices primarily relate to facets of content,  
148 distress, and functional interference, for which there appears to be broad consistency  
149 in patient experience. Voice content can involve a rich array of themes (including  
150 neutral or positive voices), but critical, derogatory, or threatening voices are most  
151 widespread<sup>32</sup>, and typically have substantial negative life impacts<sup>20,21,24</sup>. Somatic or  
152 bodily sensations, such as tingling or burning on the skin, might also accompany  
153 these experiences<sup>11</sup>.

154

155 *[H3] Non-clinical voices*

156 Non-clinical voice-hearing (often used interchangeably with 'healthy voice-  
157 hearing' in the literature) refers to frequent, recurring experiences, of comparable  
158 complexity and/or persistence to that in clinical groups, but associated with little  
159 distress and no mental health diagnosis. The lifetime prevalence of non-clinical and  
160 everyday voices has been estimated as somewhere between 5% and 15% of the  
161 general population having, at minimum, experienced one-off or fleeting voices<sup>29</sup>.  
162 However, prevalence estimates can vary depending on the methodology employed  
163 and specific questions asked, or even the discipline conducting the study (for  
164 instance, psychology versus anthropology). Of note, non-clinical (relative to clinical)  
165 voice-hearing typically has an earlier age of onset; occurs less frequently; is  
166 associated with greater perceived control; involves less negative content; and causes  
167 negligible emotional and functional disturbance<sup>8,19,22,23,33,34</sup>. Variability in other  
168 phenomenological features implies significant overlaps in these experiential facets  
169 across cohorts, with little utility for predicting mental health status<sup>27</sup>.

170 There have been calls for research to investigate how non-clinical voice-  
171 hearers can temper negative emotions and sustain functional behaviors in the face of  
172 what many would perceive as aberrant, frightful events, and whether voices may  
173 serve some adaptive function that negates the need to seek psychological care<sup>19,33</sup>.  
174 A minority of individuals in this category do transition to psychotic illness, but this is  
175 often underpinned by secondary precipitants, such as pre-existing anxiety or  
176 depression, or ongoing life stressors<sup>14,18,20,35</sup>. Many non-clinical voice-hearers report  
177 spiritual beliefs, and voices are often interpreted as communication from deceased  
178 individuals<sup>36-38</sup>.

179 Other research has focused on the extent to which spiritual practice, and, in  
180 particular, control over the experience, influence affective aspects of voice-hearing<sup>39</sup>.  
181 In particular, it was found that increased control was associated with reduced

182 distress and improved functioning, although clearer delineation between discrete  
183 types of control is needed. Some spiritual practices involve cultivating and controlling  
184 voice-hearing experiences that might have initiated spontaneously<sup>40</sup>; in other cases,  
185 voice-hearing experiences may have started as a result of such practices<sup>38</sup>.  
186 Examining non-clinical voice-hearing, unfettered from the confounds of broader  
187 psychopathology and medication effects, offers an opportunity to discern the  
188 fundamentals of this distinctive phenomenon, as well as its transdiagnostic operation  
189 across the psychosis continuum.

190

### 191 *[H3] Everyday voices*

192         What we refer to here as ‘everyday’ voices typically encompasses common  
193 and benign misperception of auditory events, such as the occasional fleeting,  
194 unexplained voice, or experiences involving imaginary companions. These  
195 occurrences can be sporadic, with their incidence heightened (as in psychosis) by  
196 certain internal factors such as sleep disruption<sup>41</sup> and stress<sup>42</sup>, as well as external  
197 conditions, including noisy settings or even silence<sup>43</sup>. Associated content is typically  
198 mundane or negligible, and there is little emotional significance attached. A possible  
199 exception lies in the case of bereavement hallucinations (perceptual experiences of a  
200 person, usually a loved one, who has passed away, potentially involving multiple  
201 senses, such as hearing their voice, seeing their image, and so on), which can be  
202 associated with psychological distress<sup>44</sup>.

203         Many everyday voice-hearing experiences include elements that appear alien  
204 to the self. For example, inner speech (covert, self-directed speech or verbal  
205 thinking), particularly its misattribution to an external source, is considered to have a  
206 significant mechanistic role in cognitive and neural models of voice-hearing. There is  
207 heterogeneity in inner speech<sup>45</sup>, but one replicated finding is that it is not uncommon  
208 for individuals to report hearing the voices of other people in their inner speech (for



209 example, by endorsing an item such as ‘I hear other people’s actual voices in my  
210 head, saying things that they have never said to me before.’)<sup>46</sup>.

211 Another example of everyday voice-hearing is ‘hearing’ the voices of fictional  
212 characters when reading. A study of more than 1,500 respondents predominantly  
213 from English-speaking countries (such as the UK, US, Australia, Canada, and  
214 Ireland) found that around one in seven experienced such voices with as much  
215 vividness as hearing an actual person<sup>47</sup>. More than half of a sample of professional  
216 writers reported ‘hearing’ the voices of their characters<sup>48</sup>. A further example is the  
217 experience of engaging with imaginary companions. Although imaginary companions  
218 are usually associated with childhood, one study found that 7% of the large sample  
219 of English-speaking adults mentioned above reported currently having an imaginary  
220 companion (versus 41% having had such a companion in childhood). For almost half  
221 of participants with any experience of having an imaginary companion, engaging with  
222 such a companion involved regularly hearing their voice<sup>49</sup>. Hearing the voices of  
223 others in the absence of any speaker thus appears unexceptional as an aspect of  
224 human inner experience.

225 Taken together, these phenomena are consistent with the dominant view that  
226 regular sensory perception involves a process of ‘controlled hallucination’<sup>50</sup>: the  
227 nervous system actively predicts what is in the environment rather than passively  
228 receiving information about it. Reports of such everyday voice-hearing phenomena  
229 also align with growing evidence that hallucinations can be readily elicited in healthy  
230 individuals in experimental situations<sup>51</sup>.

231

### 232 **[H1] Mechanisms of voice-hearing**

233 Research has investigated the cognitive (see Table 2), neural, personal, and  
234 sociocultural mechanisms that might generate voice-hearing experiences across the  
235 three categories of voice-hearing experiences described above. Although these  
236 mechanisms are presented here as categorically distinct, they are not entirely

237 conceptually separable, and in fact typically exert multifarious influences on one  
238 another.

239

240 *[H3] Cognitive mechanisms*

241 Although individuals with a diagnosis of schizophrenia typically show wide-ranging  
242 cognitive impairments<sup>52</sup>, it is difficult to establish which, if any, are specific to voice-  
243 hearing because of comorbid symptoms and the confounding effects of antipsychotic  
244 medication<sup>53</sup>. One study found some impairments in executive function, working  
245 memory, and verbal intelligence, but not in other cognitive measures, for non-clinical  
246 voice-hearers compared to non-voice-hearing controls<sup>17</sup>.

247 Other studies have taken a narrower, theoretically-driven approach. For  
248 example, early cognitive models of voice-hearing suggested that clinical voice-  
249 hearing might result from misattributing internal mental events (such as inner  
250 speech) to an external source<sup>54</sup>. This externalizing bias in source monitoring was  
251 supported by evidence from a source memory task, in which participants were asked  
252 to recall the origin (self or non-self) of previously presented verbal stimuli;  
253 hallucinating psychosis patients were more likely to externally misattribute a self-  
254 generated word as non-self-generated compared to non-hallucinating psychosis  
255 patients or non-clinical controls<sup>54,55</sup>. Numerous studies have reported similar findings,  
256 with meta-analytic evidence suggesting that such external misattributions are  
257 associated with hallucinations in both clinical and non-clinical samples<sup>55</sup>. However,  
258 several studies have failed to replicate these findings<sup>56-58</sup>, with the discrepancy  
259 possibly caused by variability between studies, such as in the specific attributes of  
260 the tasks used.

261 Source-monitoring (or self-monitoring) theories of voice-hearing have been  
262 linked to 'comparator model' accounts. These accounts posit that dysfunctional  
263 efference copies (internal copies of motor signals, sent between motor and sensory  
264 brain regions to predict and typically suppress sensory cortical activity) could lead to

265 feelings of alienness or that one's own actions are not self-generated<sup>52,59,60</sup>. Source-  
266 monitoring theories are also consistent with accounts of hallucination proneness  
267 linked to population-wide variability in the vividness of mental imagery<sup>61</sup>, predicting  
268 that vivid mental imagery would be more likely to be externally misattributed.

269 Externalizing bias accounts of hallucinations are also supported by evidence  
270 from auditory signal detection tasks in which participants must detect speech  
271 embedded in noise (Figure 1a). Clinical and non-clinical voice-hearers have a lower  
272 threshold for responding that there was a voice in the noise compared to non-voice  
273 hearers<sup>62</sup>, and therefore are more likely to respond that a voice was present on  
274 noise-only trials (Figure 1b). These results have been interpreted to suggest that  
275 biases towards responding that signals are external are associated with  
276 hallucinations. However, evidence is mixed regarding whether this response bias is  
277 accompanied by a decrease in detection sensitivity (the perceptual ability to  
278 distinguish speech signals from noise)<sup>63</sup>.

279 Although some researchers assume that biases in source monitoring and  
280 auditory signal detection tasks reflect a common underlying cognitive mechanism<sup>55</sup>,  
281 there is little evidence for this interpretation. Indeed, source monitoring and signal-  
282 detection explanations of voice-hearing operate, to some extent, at different  
283 explanatory levels that are not mutually exclusive<sup>64</sup>. Whereas source-monitoring  
284 tasks assess memory-related decision-making in relation to relevant mnemonic  
285 cues<sup>65</sup>, biases in auditory signal detection might reflect over-weighting of top-down  
286 processes. For example, according to predictive processing accounts the nervous  
287 system makes predictions about the external world and updates these predictions on  
288 the basis of discrepancies between these predictions and observation (prediction  
289 error). In a predictive processing framework, biases in auditory signal detection tasks  
290 might be due to overweighting expectation relative to observation<sup>66</sup>.

291 The role of top-down processes in voice-hearing is also suggested by  
292 evidence that voice-hearing participants, regardless of clinical status, are more

293 susceptible to 'conditioned hallucinations' than non-voice-hearing participants. That  
294 is, voice-hearing participants are more likely to report hearing tones that are not  
295 present in response to a visual stimulus after an auditory and visual stimulus are  
296 repeatedly paired<sup>67</sup>. Other studies have shown that prior expectations for speech, as  
297 assessed by responses to ambiguous speech stimuli, might be present in voice-  
298 hearers, even without experimental induction via conditioning<sup>53</sup>. These prior  
299 expectations are associated with performance in the conditioned hallucinations  
300 paradigm, such that clinical voice-hearers who were more likely to detect speech in  
301 ambiguous stimuli were also more susceptible to conditional hallucinations<sup>68</sup>.

302 Bottom-up perceptual processes might also contribute to voice-hearing. For  
303 example, people with peripheral sensory deficits such as hearing impairment are  
304 predisposed to hallucinatory experiences<sup>69</sup>. Promising computational models of  
305 voice-hearing therefore take into account the integration of top-down and bottom-up  
306 signals<sup>70</sup>.

307 Finally, voices may result from failures to inhibit and place into context  
308 intrusions from episodic memory due to failures in intentional inhibition<sup>71,72</sup>. In typical  
309 assessments of intentional inhibition, participants complete a variant of a continuous  
310 recognition task in which, during later task blocks, participants are asked to inhibit  
311 recognition responses based on earlier stimuli. Both voice-hearing psychosis  
312 patients<sup>71,72</sup> and participants in the general population reporting more hallucinatory  
313 experiences<sup>73,74</sup> exhibit impairments in intentional inhibition, suggesting that voice-  
314 hearing might reflect uncontrolled intrusive memories. However, one study found no  
315 impairment in intentional inhibition in a group of non-clinical voice-hearers<sup>58</sup>. This  
316 raises the possibility that impaired performance on intentional inhibition tasks might  
317 only be associated with voices in psychosis.

318 As outlined in this section, a number of cognitive mechanisms have been  
319 linked to voice-hearing in clinical and non-clinical groups. Further research is needed  
320 to clarify which cognitive mechanisms are specific to clinical (as opposed to non-

321 clinical) voice-hearing, as well as how different mechanisms may be associated with  
322 phenomenologically different kinds of voices.

323

### 324 *[H3] Neural mechanisms*

325 A variety of neural mechanisms have been put forward to account for voice-hearing  
326 in clinical and non-clinical groups. Neuroimaging studies that record brain activity  
327 while clinical and non-clinical participants hear voices (known as symptom-capture  
328 studies) have shown activation in regions including the inferior frontal gyrus, superior  
329 temporal gyrus, insula, and (para)hippocampus<sup>75</sup> during voice-hearing. These  
330 regions overlap with brain areas involved in inner speech<sup>76</sup>, auditory signal  
331 detection<sup>77</sup>, and auditory imagery<sup>78,79</sup>.

332       Studies examining **functional connectivity [G]** point to aberrant fronto-  
333 temporal connectivity in both clinical and non-clinical voice-hearing individuals  
334 compared to non-voice-hearing controls. These findings suggest a functional model  
335 of atypical efference copy signalling between the inferior frontal speech production  
336 region and speech perception areas in the temporal lobe<sup>80,81</sup>. This model is  
337 supported by atypical N1 event-related potentials in response to self-generated (but  
338 not non-self-generated) actions in people with a diagnosis of schizophrenia<sup>82</sup>. The N1  
339 component of the auditory event-related potential is taken as a marker of  
340 suppression of activity in cortical speech perception areas in response to receiving  
341 an efference copy signal during speech production. However, most auditory event-  
342 related potential findings show an association with psychosis, but do not show  
343 specific associations with voice-hearing<sup>83</sup>.

344       Studies of **structural connectivity [G]** confirm atypical white matter  
345 connectivity linking frontal and temporal areas, particularly in relation to the arcuate  
346 fasciculus<sup>84</sup>, in voice-hearers with a diagnosis of schizophrenia. However, findings  
347 regarding this white matter tract have been inconsistent<sup>85</sup>. Other structural  
348 neuroimaging studies find variation in patterns of brain folding in individuals with

349 schizophrenia who experience hallucinations in different modalities<sup>86</sup>, and there is  
350 evidence that decreased length of the paracingulate sulcus (known to be significant  
351 in source monitoring) is associated with a greater susceptibility to hallucinations in  
352 patients with schizophrenia<sup>87</sup>. Taken together, these findings suggest that brain  
353 structure may be altered in clinical populations in ways that are specifically linked to  
354 hallucinations.

355         Voice-hearing is associated with atypicalities in brain networks activated  
356 when participants are not engaged in an explicit task (resting-state networks).  
357 Specifically, there are differences in patterns of interaction among the **default mode**  
358 **network [G]** and networks linked to cognitive control and salience, as well as those  
359 relevant to specific modalities of perceptual processing<sup>88,88,89</sup>. Resting-state auditory  
360 cortex hyperactivation has been implicated in voice-hearing in both clinical<sup>90</sup> and  
361 non-clinical<sup>91</sup> groups. These findings suggest that mechanistic explanations of voice-  
362 hearing may be sought in patterns of brain activation observable even when  
363 participants are not reporting on their own hallucinations or engaged in a specific  
364 task.

365         Evidence for atypical fronto-temporal signalling in clinical and non-clinical  
366 voice-hearers is consistent with the idea that voices result from a failure to predict the  
367 sensory consequences of one's actions<sup>92</sup>. Within the general predictive processing  
368 framework, such models have been understood as congruent with models of  
369 hallucination associated with an overreliance on strong priors<sup>93</sup>, combined with  
370 compromised processing of prediction error, leading to non-veridical acceptance of  
371 perceptual hypotheses<sup>94</sup> (the brain's 'best guesses' about what is out there in the  
372 environment). Predictive-processing approaches to hallucinations also gain support  
373 from research on the role of striatal dopamine in perceptual inference<sup>95</sup>. For example,  
374 perceptual and reward expectations and striatal dopamine have been linked to  
375 behavior consistent with hallucination-like experiences in mice<sup>96</sup>, consistent with

376 models suggesting a role for prior expectations in hallucinations, and with a large  
377 literature suggesting elevated dopamine levels in schizophrenia<sup>97</sup>.

378 Finally, data showing activation in right-hemisphere homologues of speech-  
379 production regions during voice-hearing<sup>98</sup>, combined with behavioural findings that  
380 psychosis patients with hallucinations do not show a typical right-ear advantage<sup>99</sup>  
381 (more accurate reporting of stimuli presented to the right ear than the left ear when  
382 different speech stimuli are simultaneously presented to both ears), implicate atypical  
383 patterns of brain lateralization, particularly relating to language functions<sup>100</sup>, in  
384 schizophrenia, consistent with explanations of voice-hearing as instances of auditory  
385 cortex hyperactivation and decreased cognitive control.

386 Research into the neural mechanisms underlying voice-hearing has  
387 employed a variety of methods to show altered patterns of brain processing and  
388 structure in clinical voice-hearers. In some cases, these patterns have also been  
389 shown to extend to non-clinical voice-hearers. Particularly where such findings  
390 support cognitive models, they offer hope for mechanistic explanations of the  
391 phenomenon, although there remain many gaps in current knowledge, and reasons  
392 to be cautious about assuming continuity of mechanism across the clinical/non-  
393 clinical divide.

394

### 395 *[H3] Personal and sociocultural mechanisms*

396 Certain personality traits, for instance involving heightened absorption (propensity for  
397 total immersion within one's thoughts or experiences), schizotypy (see Box 1), or  
398 openness to experience, are known to increase the likelihood of voice-hearing in  
399 certain cohorts<sup>40,101</sup>. When voice-hearing involves the experience of often personified  
400 voices in interaction with the self, it can be a highly social process<sup>102,103</sup>. Some  
401 accounts of the social nature of voice-hearing relate to its potential origin in the  
402 misattribution of inner speech to an external source. Inner speech is recognized to  
403 have interpersonal qualities, particularly a conversational or dialogic nature,

404 proposed to result from the internalization of linguistically-mediated social  
405 interactions, such as dialogues with caregivers, during development<sup>104</sup>. Other  
406 accounts suggest that voice-hearing is the result of atypical activation of social agent  
407 representations<sup>10</sup>, such that voice-hearing experiences reflect hallucinations of  
408 communicative agents rather than simply auditory experiences. Agent representation  
409 may be particularly important for the role of social threat in distressing voice-hearing  
410 experiences<sup>148</sup>. Relatedly, loneliness and social isolation have been linked to voice-  
411 hearing, with voices and other psychosis-like experiences in the absence of social  
412 interaction playing a role analogous to phantom limbs in the absence of the  
413 amputated limb<sup>105</sup>. However, there is some evidence that only negatively appraised  
414 social exclusion ('social defeat') triggers hallucinations<sup>106</sup>.

415         The social nature of voice-hearing is also apparent in its connections to  
416 trauma and dissociation (discontinuity or separation between one's thoughts and  
417 emotions, or even facets of self-identity). Dissociation is hypothesized to exert a two-  
418 fold influence between voice-hearing and trauma, via fragmentation of self  
419 components<sup>107,108</sup> (resulting from traumatic life events, including interpersonal losses  
420 and stressors) and as a mediator (where depersonalization was able to explain a  
421 specific portion of the variance in childhood abuse)<sup>109,110</sup>. The involvement of  
422 dissociation is underscored by the interpersonal nature of specific types of trauma,  
423 as well as the social relationships that some people share with their voices<sup>103,111</sup>.  
424 Moreover, early trauma has been shown to be a risk factor for psychosis<sup>112-115</sup>, and in  
425 particular hallucination severity<sup>116</sup>. However, other reviews have been more critical  
426 about drawing firm etiological links between trauma and voice-hearing due to existing  
427 conceptual and methodological limitations, such as diagnostic ambiguities,  
428 consensus regarding the definition and measurement of childhood trauma, under-  
429 powered studies, and the lack of longitudinal research<sup>117,118</sup>. Nevertheless, trauma is  
430 often reported in conjunction with voice-hearing in clinical groups<sup>119,120</sup> and  
431 population-based studies<sup>121,122</sup>.



432           However, closer inspection of mechanistic factors potentially related to  
433 trauma elicits more questions than answers. One model outlining possible pathways  
434 from trauma to psychosis has highlighted interactions among personal vulnerabilities  
435 and various kinds of intrusion<sup>123</sup>, but the empirical evidence for memory intrusions is  
436 currently limited<sup>124</sup>. Although certain risk factors for voice-hearing have been  
437 documented (for example, childhood sexual abuse<sup>121</sup>), less is known about individual  
438 differences and protective factors (such as attachment or resilience) that may  
439 determine why some people with similar life and trauma experiences do not go on to  
440 develop voices (or vice versa).

441           Socio-demographic factors have also been linked to the propensity to hear  
442 voices, although it is often difficult to establish specificity to voices (as opposed to  
443 psychotic experiences more broadly). Some evidence suggests that, within  
444 psychosis, biological females are more likely to report voices<sup>125,126</sup>. Other socio-  
445 demographic factors such as ethnicity and migration status are well-established risk  
446 factors for psychosis<sup>127</sup>, but their specific contribution to voice-hearing is less clear.  
447 Several studies have linked ethnic minority status to hallucinations<sup>128,129</sup>; one study  
448 indicated that migration was linked only to delusional ideation, not hallucinations<sup>128</sup>.  
449 Longitudinal research has found that cannabis use, childhood trauma, and ethnic  
450 minority status are linked to persistence of psychotic experiences over a period of six  
451 years<sup>129</sup>, but these findings are not specific to voice-hearing.

452           A final set of mechanisms relate to cultural or subcultural influences. For  
453 example, the Fang ethnic group in Equatorial Guinea describe *mibili* as possession  
454 by evil spirits that manifests as auditory and visual hallucinations<sup>130</sup>. Cultural  
455 influences are complex and multifaceted, involving myriad interfaces along  
456 geographical, ethnic, and religious lines. Cultural expectations profoundly shape the  
457 definition, prevalence, experiential facets, and significance of voice-hearing, as well  
458 as levels of psychopathology and concomitant outcomes<sup>131,132</sup>. For example, one  
459 study found distinct differences in the phenomenological experiences of voice-

460 hearing in the context of schizophrenia across the United States, Ghana, and  
461 India<sup>133</sup>. The American sample mostly had an adversarial relationship with their  
462 voices, which involved themes of violence, and this was readily pathologized as  
463 indicative of mental illness; the Ghanaian sample often linked their voices to a  
464 benevolent God, with whom they shared a social and positive relationship; and the  
465 Indian sample personified their voices, who were often kin, and these voices  
466 provided an adaptive function by dispensing advice or instruction<sup>133</sup>.

467 Most existing studies examining cultural influences on voice-hearing have  
468 relied on rudimentary divides across national borders or between Western and non-  
469 Western cultures<sup>133–138</sup>. The lack of nuanced considerations inherent in such divisions  
470 is problematic for the field because globalization and the mass movement of people  
471 across borders over time means that homogeneous cultural settings are becoming  
472 increasingly rare. Outside of a Western medical disease model, voice-hearing may  
473 be less negatively regarded or pathologized, possibly due to fluid delineations  
474 between reality and fantasy as well as self and other<sup>19,131</sup>, referred to as the concept  
475 of 'porosity'<sup>38</sup>.

476 From a subcultural perspective, understanding voice-hearing as a personally  
477 significant aspect of human experience has taken on political force with the growth of  
478 the international Hearing Voices Movement<sup>139</sup>. This grassroots advocacy initiative  
479 focuses on personal empowerment and the de-pathologization of voices<sup>140</sup>. Its  
480 emphasis on positive acceptance and individual meaning-making denotes significant  
481 departures from, but also some overlaps with, conventional views espoused in  
482 psychiatry and psychology. Other examples of subcultural or religious influences on  
483 voice-hearing include the repositioning of the hallucinatory experiences of historical  
484 figures<sup>141,142</sup>, or studying their attribution to *jinn* (invisible spirits) within Islam<sup>143</sup>.

485 Religious affinity might serve as both a risk and protective factor in shaping  
486 voice-hearing<sup>144,145</sup>. For example, tentative evidence suggests that religious activity  
487 might serve as a coping mechanism for voice-hearing in Brazil<sup>146</sup>, whereas in the

488 Netherlands religious affiliation has been theorized to add to the specificity and  
489 burden of these experiences<sup>147</sup>. Relatedly, unusual sensory experiences can be  
490 deliberately sought out in certain spiritual settings, such as indigenous use of  
491 psychotropic agents to invoke communication with the spirit realm<sup>131,132</sup> or  
492 contemporary rituals performed by psychics and mediums<sup>40,148,149</sup>. These practices  
493 stand apart from clinical voice-hearing in that they are highly valued, often replete  
494 with vivid detail, and accepted within shared cultural norms. Different pathways might  
495 therefore underlie these different voice-hearing experiences<sup>150</sup>. What cultural or  
496 spiritual beliefs like *mibili* and *jinn* may have in common is a collective pursuit of  
497 meaning by adapting interpretations of anomalous perceptual events to fit the  
498 broader cultural milieu. More research is needed to understand the mechanisms  
499 underlying such experiences.

500           It is thus evident that voice-hearing experiences do not occur in isolation, but  
501 are shaped by factors including early life trauma, sociodemographic and personality  
502 variables, and cultural and religious influences. The endeavor to understand voice-  
503 hearing experiences in their full complexity will benefit from situating them within the  
504 broad personal, social, and cultural contexts in which they occur.

505

## 506 **[H1] Continuity and discontinuity**

507 In the previous section we outlined a range of cognitive, neural, personal, and  
508 sociocultural mechanisms that might underlie voice-hearing experiences across  
509 clinical and non-clinical categories. A key question is the extent to which the transient  
510 and non-distressing voice-hearing experiences of individuals without need for  
511 psychiatric care lie along a continuum with recurring and distressing experiences in  
512 psychosis. According to diagnostic discontinuous models, non-clinical voice-hearing  
513 is only explicable in terms of phenomenology and processes also occurring in non-  
514 voice-hearers, and these are distinct from those underlying distressing experiences  
515 that characterize clinical voice-hearing (Figure 2a). By contrast, the continuum

516 hypothesis proposes that non-clinical voice-hearing is simply an attenuated version  
517 of psychotic experience, fundamentally of the same kind and perhaps reflecting an  
518 underlying continuous mechanism, phenotype or ‘proneness’<sup>1</sup>. In continuous models,  
519 psychotic experiences might be directly related to distress and the need for treatment  
520 (Figure 2b) or could occur independently of distress and the need for treatment  
521 (Figure 2c).

522 Here, we do not aim to debate whether psychotic disorders represent an  
523 extreme manifestation of a continuous trait or latent factor in the population (such as  
524 schizotypy<sup>151</sup>), but focus instead on which specific aspects of voice-hearing might be  
525 continuous across the population in phenomenology or mechanism. A related  
526 question is the extent to which aspects of phenomenology and mechanism are  
527 linked, and via which facets of phenomenology and mechanism such linkages occur.  
528 A crucial question is whether any continuity of experience reflects common  
529 underlying cognitive and neural processes; in other words, whether continuity of  
530 frequency and phenomenology translates to continuity of mechanism. The answer to  
531 this question will be informative about whether voice-hearing in different groups  
532 should be considered as representing the same kind of experience.

533

### 534 [H3] *Continuity of experience*

535 At its most basic level, voice-hearing could be continuous in frequency across  
536 the population, with some people reporting regular voices, compared to others who  
537 report only occasional experiences. Epidemiological data provides evidence for  
538 occasional psychotic-like experiences in the general population, with one meta-  
539 analysis suggesting a median lifetime prevalence of 7.2% (and an annual incidence  
540 of 2.5%)<sup>151</sup>, involving similar risk factors (such as cannabis use and stress<sup>127</sup>) as in  
541 psychosis. Numerous standardized self-report assessments show variability in the  
542 frequency of hallucinations in the general population at lower rates than in  
543 psychosis<sup>152</sup>. Although it has been argued that such scales may ask about

544 fundamentally different experiences<sup>153</sup>, it is not contentious to state that some forms  
545 of voice-hearing are present in the general population, with varying frequencies.

546         There may also be continuous variation in aspects of the voice-hearing  
547 experience itself, such as associated distress (Figure 2b). Recent research with non-  
548 clinical voice-hearers shows that, as in psychosis, distress can vary independently of  
549 voice-hearing frequency<sup>22,36,37</sup>, and scales administered in the general population  
550 show some variability in ratings of distress associated with hallucinations<sup>154</sup>. Others  
551 have argued that distress might be discontinuous between clinical and non-clinical  
552 populations<sup>19,153</sup> (Figure 2c), appearing predominantly in those with a diagnosis of (or  
553 at risk for) psychosis.

554         Other aspects of the phenomenology of voices might also vary across the  
555 population. While volitional control is typically low or non-existent in psychosis, non-  
556 clinical voice-hearers often report at least some level of control<sup>39</sup>. Additionally, recent  
557 studies have shown that personification varies within clinical and non-clinical  
558 categories<sup>155,156</sup>, with individuals within both groups reporting different levels of  
559 'person-like' voices. Although no research has assessed personification of infrequent  
560 hallucinatory experiences in the general population, proneness to such experiences  
561 has been associated with the tendency to assign personality characteristics to  
562 neutral auditory-verbal stimuli<sup>157</sup>. These findings suggest that attributions of social  
563 agency might vary within and between groups who report voices.

564

### 565 [H3] *Continuity of mechanism*

566         Evidence regarding continuity of mechanism across different forms of voice-  
567 hearing is mixed. To take an oft-cited example, voice-hearing might result from  
568 biases in source monitoring or auditory signal detection<sup>54,55</sup>. Evidence of such biases  
569 in voice-hearers with psychosis is often reported<sup>62,158,159</sup>. Signal detection biases  
570 have been found in non-clinical populations<sup>55,57</sup>; however, evidence for source-  
571 monitoring biases is more mixed<sup>55-57</sup>. Evidence is also mixed regarding the roles of

572 attention<sup>160,161</sup> and intentional inhibition<sup>58,71,73,74</sup> across clinical and non-clinical voice-  
573 hearing, with some studies reporting differences between voice-hearing and non-  
574 voice-hearing groups, and some not. One interpretation of these discrepancies is that  
575 only some cognitive mechanisms associated with voice-hearing vary continuously,  
576 whereas others are discontinuous, and contribute only to some forms of voice-  
577 hearing.

578         The validity of the continuum hypothesis as applied to voice-hearing therefore  
579 seems to depend on the answer to the question: a continuum of what? A related  
580 question concerns whether any such continuum has an explanatory function. Similar  
581 to debates surrounding the utility of psychiatric diagnostic practices<sup>162</sup>, it is important  
582 to consider whether investigating continuity is useful clinically or valid scientifically.  
583 One solution has been to suggest multiple continua across which voice-hearing  
584 might vary, such as frequency, distress, or vividness, allowing for an exploration of  
585 variance and covariance within and between these continua<sup>163</sup>. This approach could  
586 also be clinically helpful for developing treatments for people with distressing voices  
587 (for example, improving detection of people at risk for developing psychosis, or more  
588 precise targeting of therapeutic options for voices). Another option is to focus less on  
589 putative continua and more on underlying commonalities: that is, factors that might  
590 not vary continuously between individuals, but rather play a common causal role in  
591 voice-hearing across different kinds of experiences.

592

### 593 **[H1] Multiple pathways to voice-hearing**

594 In this section, we illustrate how multifactorial consideration of the varying  
595 phenomenology of voice-hearing in everyday, non-clinical, and clinical categories  
596 can generate testable hypotheses about how various building blocks underlying  
597 voice-hearing might interrelate across the range of experiences. We do not set out to  
598 present a comprehensive model of voice-hearing<sup>164</sup>; there are too many gaps in  
599 current knowledge to propose a feasible integrated model linking cognitive and

600 neural mechanisms to phenomenological features. Rather, we hope to offer a  
601 functional approach that can be used to set a research agenda to advance the field.

602 To this end, Box 2 describes three putative instances of voice-hearing, one  
603 from each of our main categories: an individual with relatively frequent experiences  
604 of the voice of a deceased loved one in the absence of distress (everyday voice-  
605 hearing); an individual with particular spiritual beliefs within a specific cultural context  
606 (spiritualism/mediumship; non-clinical voice-hearer); and an individual hearing voices  
607 in the context of psychosis (clinical voice-hearer). These examples have been  
608 chosen to illustrate how common and distinct processes interact in generating the  
609 varied phenomenology of voice-hearing, including in complex ways that remain to be  
610 fully elucidated. Another important point to reiterate is that the overlap of  
611 phenomenology across groups is considerable, but should not be taken as  
612 necessarily entailing continuity of mechanism<sup>163</sup>. Indeed, we propose considerable  
613 variation in mechanism even within our groups, with differing patterns of association  
614 among top-down and bottom-up mechanisms potentially underlying different  
615 phenomenological clusters. The examples should therefore be read as indicative of  
616 how constituent processes might interrelate in line with select phenomenological  
617 facets of voice-hearing.

618 Comparisons across the three examples can help illustrate our arguments  
619 about continuity and discontinuity across mechanisms and phenomenological  
620 features. The subjective experience of voice-hearing in our everyday example is as  
621 vivid, clear, and complex as the distressing hallucinations experienced in our clinical  
622 case. In our non-clinical example, voices are shaped by underlying cultural beliefs in  
623 a way that is less apparent in both the everyday and clinical examples. Deficits in  
624 peripheral sensory systems, such as hearing loss, play a role in the everyday  
625 example but not in the non-clinical or clinical cases. To be clear, the argument is not  
626 that hearing loss always features in everyday voices and never in clinical ones.  
627 Rather, it is that causal factors underlying voice-hearing can dip in and out across

628 groups in ways not easily described as continuous or discontinuous with  
629 phenomenology.

630 Another interesting point is that the predictive processing framework is  
631 implicated in all three examples but takes different forms in each. In the everyday  
632 example, there is a high expectation of hearing a particular voice. In the non-clinical  
633 example, there are stronger priors for hearing speech stimuli in all auditory stimuli<sup>53</sup>  
634 which are enhanced by learning and cultivation processes relevant to the subcultural  
635 context. In the clinical example, atypical processing of prediction error, in addition to  
636 strong speech priors, is implicated. Although the same general mechanism is thus  
637 involved in each example (predictive processing), the biases involved take different  
638 forms and shape phenomenology in distinct ways.

639 Comparison of phenomenological and mechanistic features across voice-  
640 hearing groups is instructive in other ways. Our example of non-clinical voice-hearing  
641 is particularly dependent on processes that manifest at the personal-sociocultural  
642 level of explanation, rather than at the neural or cognitive levels. This example  
643 highlights how paying attention to neural and cognitive processes at the expense of  
644 personal and sociocultural ones—as research on voice-hearing has tended to do—  
645 might make it more challenging to account for the range of voice-hearing  
646 experiences.

647 Our three examples in Box 2 also shed light on ongoing debates about  
648 continuity in voice-hearing experiences. Constituent processes might be involved  
649 across the three groups with no simple gradation of mechanism, and some degree of  
650 commonality in phenomenology. That said, one pattern that emerges is the increase  
651 in the number of relevant features as we progress from everyday to clinical voices.  
652 This increase in relevant features could suggest that clinical voice-hearing is indeed  
653 characterized by a greater number of underlying mechanisms (and hence, perhaps,  
654 phenomenological features). Alternatively, it could be that there has been less  
655 research on everyday and non-clinical voice-hearing, in which case the absence of



656 evidence should not be taken as evidence of absence. The present state of research  
657 is not sufficient to distinguish between these two possibilities.

658         Owing to a lack of relevant evidence, our proposed links between mechanism  
659 and phenomenology in this section are largely speculative, and highlight areas where  
660 intensive research will be particularly valuable. Indeed, an overarching question for  
661 future research concerns the extent to which researchers might ever expect to be  
662 able to map phenomenology onto mechanism. At a minimum, researchers will need  
663 to address the complexity of interactions among processes at different levels of  
664 explanation, and how this might lead to different kinds of voice-hearing.

665

#### 666 **[H1] Summary and future directions**

667 Voice-hearing is a diverse phenomenon experienced in some form by a significant  
668 proportion of the population (with or without mental health diagnoses), with multiple  
669 cognitive, neural, personal, and sociocultural mechanisms influencing its etiology,  
670 dynamic development, and phenomenology. Our approach to understanding how  
671 multiple mechanisms can constellate in different patterns between and within our  
672 three groups of voice-hearers highlights that we cannot assume any simple continuity  
673 of mechanism or experience across the spectrum from everyday, through non-  
674 clinical, to clinical voice-hearing, even when many aspects of subjective experience  
675 are shared. Importantly, although our focus here has been on voice-hearing, many of  
676 the processes described are modality-general<sup>89</sup> and thus likely to feature in  
677 hallucinations across other modalities (see Box 3). Determining which processes  
678 generalize across modalities remains an empirical question that should guide future  
679 research.

680         The predominant factor that determines diagnostic group membership is the  
681 occurrence of distress. Voice-hearing occurs in a variety of forms that do not  
682 significantly influence functioning, and which can even have beneficent, if not merely  
683 neutral, effects on the individual. These offer hope for advancing understanding of

684 many aspects of typical, non-pathological psychological functioning, such as the  
685 nature of inner speech<sup>165</sup> and cognitive processes such as source monitoring<sup>57</sup>.  
686 Pinning down exactly why some voices are distressing is a complex and challenging  
687 task, and one that lies beyond the scope of our article (see ref<sup>166</sup> for a recent  
688 interdisciplinary attempt).

689         Our Review did not cover therapeutic options for distressing voices<sup>167,168</sup>.  
690 However, the fact that many voice-hearing experiences are intensely distressing  
691 makes the search for further knowledge about links between phenomenology and  
692 mechanism even more pressing. If voice-hearing is as varied an experience as the  
693 research reviewed here suggests, psychological therapies will arguably be best  
694 targeted at particular subtypes of phenomenology to the extent that they can be tied  
695 to underlying mechanisms<sup>169</sup>. These patterns of association are likely to be highly  
696 complex, and 'one size fits all' approaches that do not consider specificity of  
697 phenomenology and mechanism might fail to deliver the therapeutic benefits that  
698 those troubled by distressing voices desperately deserve. In particular, there is an  
699 urgent need to improve understanding of which early-developing manifestations of  
700 voice-hearing relate to future distress, and which remain largely benign, so that  
701 effective therapeutic interventions for distressing voices can be developed and  
702 deployed.

703         The co-occurrence of trauma in some voice-hearing individuals leaves  
704 several questions unanswered. Although clear associations with childhood sexual  
705 abuse exist<sup>116,121</sup>, explicit causal links are unverified<sup>170</sup>, and contradictory findings  
706 remain in relation to other forms of trauma<sup>119,120</sup>. Preliminary evidence supports  
707 distinct voice-hearing subtypes possibly underpinned by a trauma-related  
708 phenotype<sup>31</sup>. Future studies could ask how the precise nature of traumatic events  
709 aligns with voice-hearing and its phenomenological correlates<sup>114</sup>. Complex  
710 interactions among dissociation and other etiological factors, such as memory and  
711 cognitive inhibition, also need further study.

712 In non-clinical voice-hearing, more research is needed on cognitive  
713 processes, with a particular focus on attentional, inhibitory, and source-monitoring  
714 abilities. Inconsistencies in the literature relating to this group, for example, around  
715 paracingulate sulcus morphology<sup>91,171</sup> in different non-clinical samples (possibly  
716 reflecting different recruitment methods and/or inclusion criteria), might be resolved  
717 by new evidence for previously unseen phenomenological heterogeneity in such  
718 groups<sup>163</sup>. More research is also needed on how sociocultural factors such as cultural  
719 beliefs interact with cognitive and neural mechanisms to shape experiential facets of  
720 non-clinical voice-hearing, and on what factors, including age of onset and distress,  
721 distinguish clinical and non-clinical voice-hearing groups. For all three of our voice-  
722 hearing categories, the distinct contributions of predictive processing models to  
723 mechanistic accounts remain to be specified. Such models may be particularly  
724 valuable in determining the mechanistic roles of peripheral sensory deficits (such as  
725 hearing loss) and social agent representation in everyday voice-hearing.

726 Another question concerns whether one would expect to see linear  
727 associations between relevant cognitive mechanisms and phenomenological  
728 features. Such linear associations might only be observed for particular mechanisms.  
729 For example, the degree of bias in auditory signal detection could correlate in a linear  
730 fashion with certain phenomenological variables, such as frequency or intensity,  
731 whereas inhibition might covary non-linearly with those same phenomenological  
732 variables.

733 An approach based on ‘functional systems’<sup>172</sup> of dynamically interacting  
734 cognitive, neural, and personal-sociocultural mechanisms will likely prove useful for  
735 understanding complex patterns of interaction among a large number of constituent  
736 processes and implicated mechanisms. Such an approach has, for example, been  
737 proposed as a way of explaining how modality-general processes (such as source  
738 monitoring) interact with modality-specific processes (such as inner speech) in  
739 ‘networks of networks’ that are differentially sensitive to disruption at different stages

740 of development<sup>89</sup>. Graph-theoretical and other approaches to understanding linkages  
741 among symptoms in psychopathology<sup>173</sup> might be valuable as research aims to  
742 elucidate the complex picture of voice-hearing across diagnostic groups. Although  
743 we have proposed our three-way categorization as a useful heuristic, future research  
744 should explore whether other taxonomies of voice-hearing may be more valuable in  
745 understanding phenomenology, mechanism, and targeting of treatment.

746         Progress in understanding voice-hearing will depend on the willingness of the  
747 research community to embrace principles of open, reproducible, replicable  
748 research. Voice-hearing research has previously relied on small sample sizes and  
749 non-standardized measures<sup>174</sup>. These factors likely account for inconsistencies in  
750 research findings that are apparent across the field. Initiatives that support large, pre-  
751 registered, multisite studies, such as the International Consortium on Hallucination  
752 Research<sup>57</sup>, have the potential to counter some of the methodological problems that  
753 have plagued previous endeavors. As we look ahead to the next decade of research  
754 on this complex, heterogeneous, and profoundly personally significant experience,  
755 there are reasons to be hopeful that the rich and varied phenomenon of voice-  
756 hearing will continue to attract the attention of researchers, with the prospect of real  
757 benefits for those distressed by their experiences.

758

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**Table 1. Perceptual-sensory, cognitive, and affective phenomenological features of clinical versus non-clinical voice-hearing**

	<i>Perceptual-sensory</i>	<i>Cognitive</i>	<i>Affective</i>
<b>Clinical</b>	Frequency ↑	Perceived control ↓ Threat appraisals ↑	Content: Negative (critical/threatening) Emotional distress ↑ Functional interference ↑
<b>Non-clinical</b>	Frequency ↓	Perceived control ↑ Threat appraisals ↓	Content: Neutral/mundane Emotional distress ↓ Functional interference ↓
<b>Shared features</b>	Volume Clarity Number of voices Localization Linguistic complexity Form of address	Levels of conviction Beliefs regarding origin Interaction /compliance Personification of identity	

Note: Although affective features shared between clinical and non-clinical voice-hearing are ostensibly absent from the table, each individual's relationship with their voices tends to be complex and multi-faceted (for example, positive interactions in the context of seemingly hostile, pejorative voices), necessitating a nuanced and individualized approach.

**Table 2. Summary of cognitive mechanisms underlying voice-hearing and associated experimental tasks.**

Cognitive mechanism	Associated tasks	Hypothesized association with voice-hearing	Key findings
Source monitoring (also self-monitoring or reality monitoring).	Source memory: participants speak or hear a series of words, and must later recall which words were self-generated versus non-self-generated.	Voice-hearing individuals make more external misattributions, recalling self-generated words as non-self-generated.	Early studies showed an association between external misattributions and voice-hearing <sup>54,55,158</sup> ; however, more recent studies have failed to replicate this finding <sup>56,58</sup> .
Expectation/strong priors for speech	Auditory signal detection: participants listen to bursts of white noise, and are asked to respond whether speech was present or absent (see Figure 1).	Voice-hearing individuals make more false alarms (respond that they heard a voice when none was present), reflecting a bias towards detection of speech.	Early studies showed an association between an increase in false alarms and voice-hearing in psychosis and in the general population <sup>62</sup> . More recent work has replicated this finding, and shown a similar effect in non-clinical voice-hearers <sup>58,175</sup> .
	Conditioned hallucinations: participants learn an association between a visual and an auditory stimulus, and later have to detect the auditory stimulus.	Voice-hearing individuals make more false alarms to the auditory stimulus when cued by the associated visual stimulus.	There is an association between conditioned hallucinations and voice-hearing in clinical and non-clinical groups, with no effect in non-voice-hearing psychosis patients <sup>67</sup> .
	Sine-wave speech: participants listen to manipulated unrecognizable speech, and are asked to indicate whether speech is present or absent.	Voice-hearing individuals are better able to discriminate speech from non-speech.	There is some evidence that clinical and non-clinical voice-hearers detect more speech compared to healthy non-voice-hearing groups even before training via exposure to speech samples <sup>53,68</sup> .
Memory inhibition	Intentional inhibition of currently irrelevant memories: in a continuous recognition paradigm participants are instructed to inhibit a response to a previously presented stimuli.	Voice-hearing individuals are less able to inhibit previously presented stimuli, and therefore make more errors in later task blocks.	There is evidence for an association between lower memory inhibition and voice-hearing in clinical groups <sup>71</sup> and in the general population <sup>74</sup> . However, another study did not observe this effect in a group of non-clinical voice-hearers <sup>58</sup> .

<p>Language lateralization, attentional control</p>	<p>Dichotic listening: participants are presented with conflicting auditory-verbal stimuli in each ear, and must report which stimuli they heard most clearly. In subsequent trials participants are instructed to attend to one ear at a time only.</p>	<p>Voice-hearing individuals show reduced right-ear correct responses compared to controls, and are less able to orient their attention when instructed.</p>	<p>There is evidence for reduced right-ear responses in clinical voice-hearers<sup>162</sup>, but little evidence for this effect in non-clinical or everyday voices<sup>58,176</sup>.</p>
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### **Box 1. Schizotypy and hallucination-proneness**

Voice-hearing has typically been associated with a diagnosis of schizophrenia, which involves multiple positive symptoms (such as hallucinations, delusions, or disorganized speech) and negative symptoms (such as social anhedonia, that is, a disinterest in and a lack of pleasure from social situations) combined with a decreased level of functioning<sup>177</sup>. The constructs of schizotypy and hallucination-proneness were formulated to explore the distribution of traits that may underlie psychotic-like experiences in the general population, with the assumption that they share at least some components (phenomenological, etiological, or cognitive) with experiences reported by people with schizophrenia.

Schizotypy is generally posited as a set of personality characteristics or latent traits that vary across the population, are assumed to be relatively stable over time, and are possibly associated with an individual's liability to develop schizophrenia (or psychosis more broadly<sup>151</sup>). As such, schizotypy includes the propensity to experience perceptual aberrations, magical ideation, and social anhedonia, amongst other symptoms<sup>178,179</sup>. The construct of schizotypy encompasses, but is not specific to, voice-hearing. Although assessments of schizotypy include items relating to perceptual aberrations, these typically form their own factor in principal components analyses, indicating that this aspect of schizotypy is separately measurable and conceptually distinct from other facets, such as social anhedonia and cognitive disorganization<sup>178,179</sup>.

The construct of hallucination-proneness, although strongly associated with schizotypy, attempts to assess trait-like variation in susceptibility to a broader range of perceptual experiences. The construct is more tightly focused on variation in hallucinatory experiences, and often breaks down into factors associated with specific sensory modalities (for example, auditory, visual, and tactile)<sup>180–182</sup>.

The term 'hallucination' has itself been the subject of debate, and typically includes perceptual experiences that occur in the absence of external stimulation; have a compelling sense of reality; are not amenable to voluntary control; and occur in the awake state<sup>183,184</sup>. However, some experiences typically labelled as hallucinations do not meet all of these criteria, such as experiences triggered by external sensory stimulation (for example, hearing a voice directed at oneself in a noisy crowded room<sup>185</sup>), and hallucinations that occur as a person is on the borders of sleep<sup>28</sup>. In this Review our focus is on voice-hearing experiences that, whether or not meeting the full definition of hallucination, are crucial to understanding the phenomenology and mechanisms involved in such experiences.

## **Box 2. Case examples of everyday, non-clinical and clinical voice-hearing**

Here we provide illustrative case examples of everyday, non-clinical, and clinical voice-hearing. These specific examples should not be taken to suggest that all voice-hearing within these groups can be explained in the same way, or that specific individuals only ever have experiences associated with one of these groups.

### *[H1] Everyday voice-hearing*

Our case of everyday voice-hearing involves a recently bereaved older person. They expect to hear the voice of the deceased partner (strong prior), which lowers the threshold for non-veridical perceptions. Voice-hearing is intensified by vivid mental imagery, personality traits such as absorption, and sensory deprivation due to social isolation. Active social representations arise from the manifestation of a known, personified agent. Phenomenologically, the single voice has a specified gender and identity, is experienced with a high degree of clarity, and is localized externally within familiar environs. The origins of the voice are well understood, and its content is mostly positive or neutral.

### *[H1] Non-clinical voice-hearing*

Our case of non-clinical voice-hearing involves to a spiritualist who holds particular religious beliefs and who regularly engages in the cultivated practice of seeking out voices. They expect to hear one or more voices, with increased susceptibility conferred by spontaneous hyperactivity within the auditory cortex<sup>91</sup>. They have personality traits related to elevated openness to experiences, and active social agent representations take the form of known spiritual identities recognized within the specific subculture. The active pursuit of voices transpires via an altered dissociative state, which at times is aided by the deliberate consumption of psychoactive stimulants. Phenomenologically, these voices take place with high frequency,



typically when they are sought out. Beliefs regarding the voices' origins predominantly involve cultural and spiritual interpretations, with personification aligned to culturally sanctioned figures or spirits. Controllability is desired, and is variable, but typically improves with practice.

*[H1] Clinical voice-hearing*

Our case of clinical voice-hearing involves an individual experiencing psychosis. In addition to mechanisms that also underlie non-clinical voices (dominance of speech priors, auditory cortex hyperactivity, dissociative experiences, social agent representation, and personality traits, in this case heightened schizotypy), further mechanisms significantly alter the nature of voice-hearing experiences. Cognitive processes encompass diminished inhibition of irrelevant stimuli, alongside source-monitoring difficulties. Neural mechanisms include fronto-temporal dysconnectivity and elevated dopamine. These influences are further exacerbated by a history of childhood trauma and existing sleep disruptions. Phenomenologically, they frequently experience voices that cannot be controlled, with a prolonged duration per episode, and typically in third-person address. They have a strong conviction that voices are real and tend to comply with command hallucinations. Voices are generally experienced as intrusive, and are associated with a designated purpose. Valence of content is mixed, but is often negative, involving critical or derogatory themes. Emotional distress and functional interference are significant, and are accompanied by bodily sensations, such as temperature changes or tingling.

### **Box 3. Hallucinations beyond the auditory**

The fact that many voice-hearing experiences do not carry a strong auditory component has triggered growing interest in experiences in modalities beyond the auditory. Up to 14 sensory modes have been proposed, each with their own corresponding type of hallucination<sup>12</sup>. Psychosis studies have disproportionately focused on voices, with a limited subset of research exploring visual, olfactory, and somatic-tactile domains. One study noted that lifetime prevalence of hallucinations in two or more senses was double the prevalence of hallucinations in only a single modality<sup>186</sup>. Emerging evidence has also suggested that specific patterns of clustering by sensory domain may occur<sup>187–189</sup>. Hallucinations in other modalities were twice as likely in people hearing voices, whereas this likelihood was more than 11 times higher in those who experience visual hallucinations<sup>189</sup>. Voice-hearing co-occurs with visual hallucinations, while the latter tend instead to cluster with olfactory and somatic-tactile experiences<sup>187</sup>. In general, experiencing hallucinations in more than one modality is associated with more severe psychopathology and less favorable outcomes<sup>190</sup>. General population studies have also reported multisensory hallucinations, involving hallucinatory experiences across two or more sensory modalities, with heightened variability in sensory involvement that tends to stabilize with age<sup>191</sup>. Multiple modalities of hallucination might be associated with more severe voice-hearing and poorer mental health in non-clinical groups<sup>192</sup>.

Additional consideration needs to be given to the special case of multimodal hallucinations (as distinct from multisensory hallucinations), where there are known overlaps in time order and/or thematic content of hallucinatory experiences<sup>193,194</sup>. The auditory-visual-tactile combination might be most prevalent in psychosis<sup>195</sup>, with serial, rather than simultaneous, experiences more common<sup>196</sup>. Auditory-visual hallucinations are additionally associated with elevated delusionality and negative affect<sup>197</sup>. Some authors have speculated that multimodal hallucinations engender

higher levels of conviction and distress due to greater veracity afforded by simultaneous multisensory involvement<sup>9,195</sup>. However, much remains unknown, including whether multisensory hallucinations are widely shared with non-clinical and everyday voice-hearers, and more importantly, possible therapeutic implications, given the hypothesized elevation in distress.

Emerging evidence suggests that multisensory hallucinations might have links to sociocultural precipitants: more traumatic events correlate with more modalities involved in subsequent hallucinations<sup>198–200</sup>. This growth of interest in modalities beyond the auditory has led to an approach to understanding hallucinations in any particular modality as representing combinations of modality-general mechanisms, such as source-monitoring processes or social agent representations, and modality-specific mechanisms, such as processes relating to peripheral sensory systems<sup>89</sup>.

Experiences in other sensory modes (including gustatory, sensed presence, kinesthetic-vestibular, and autoscopic) and hypnagogic-hypnopompic hallucinations<sup>201</sup> have been less studied, although we do know they are experienced by the general population<sup>191,202</sup>.

## Figure legends

### Figure 1. Auditory signal detection tasks.

(A) In an auditory signal detection task<sup>62</sup> participants listen to short bursts of white noise through headphones (panels 1–3). In some trials, a speech stimulus is presented at a threshold level (panel 2). The participant is instructed to respond, with a button press, as to whether they think a voice was present in the noise or not (panel 4). (B) Everyday, non-clinical, and clinical voices are associated with biased performance towards endorsing that a voice was present (that is, a reduced response criterion; red line) compared to non-voice hearers (gray line). This results in more hits (correctly responding that a voice was present) and more false alarms (responding that a voice was present when it was not). Evidence is mixed regarding whether disproportionately high false alarm rates in voice-hearers are also caused by reduced sensitivity (ability to perceptually differentiate voice signals (blue curve) from noise (yellow curve)).

### Figure 2. Varieties of continuum models.

Intensity of experience in individuals who do not hear voices, clinical voice-hearing, and clinical voice hearing (y-axis represents variation in level of need for care, distress, or psychotic experiences). (A) Diagnostic discontinuous model in which non-clinical voice-hearing is only explicable in terms of benign experiences and processes that also occur in non-voice-hearers and do not necessitate clinical treatment (blue). Distressing experiences (yellow) uniquely characterize clinical voice-hearing. (B) Continuous model in which non-clinical voice-hearing represents an attenuated version of psychotic experience (pink), fundamentally of the same kind as in clinical voice-hearing, and in which the frequency or intensity of psychotic experience is directly related to distress and the need for clinical treatment. (C)

Continuous model in which psychotic experiences occur independently of the distress and the need for treatment that characterizes clinical versus non-clinical voice-hearing. Adapted from ref<sup>14</sup>, CC BY 4.0.

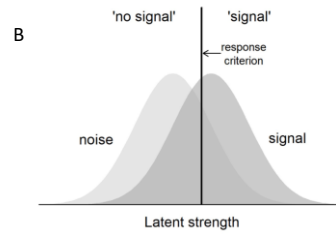
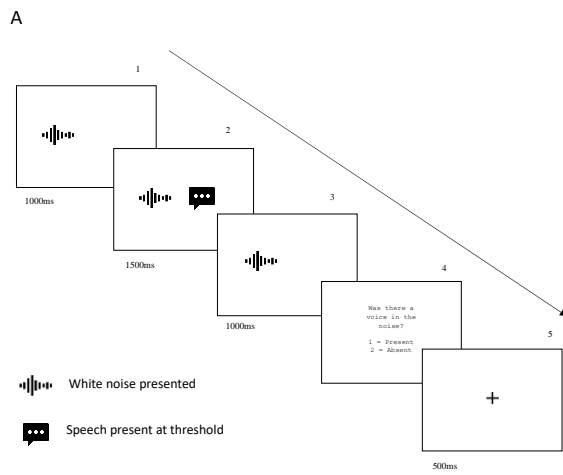
## **Glossary**

Autoscopic: The psychic duplication of a part or whole of one's body in external space, or in other words, an out-of-body experience.

Default mode network: A network of brain regions showing significant activation when the participant is not engaged in any task, thought to be related to self-referential processing.

Functional connectivity: A measure of the temporal correlation of activity in different brain regions.

Structural connectivity: A group of measures of how much different parts of the brain are anatomically connected.



**C**

Voice stimulus

	Present	Absent
Yes	Hit	False Alarm
No	Miss	Correct Rejection

Response

