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LETTERS

Neural substrates of vocalization feedback monitoring in primate auditory cortex

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Vocal communication involves both speaking and hearing, often taking place concurrently. Vocal production, including human speech and animal vocalization, poses a number of unique challenges for the auditory system. It is important for the auditory system to monitor external sounds continuously from the acoustic environment during speaking despite the potential for sensory masking by self-generated sounds[1]. It is also essential for the auditory system to monitor feedback of one's own voice. This self-monitoring may play a part in distinguishing between self-generated or externally generated[2,3] auditory inputs and in detecting errors in our vocal production[4]. Previous work in humans[5–10] and other animals[11–13] has demonstrated that the auditory cortex is largely suppressed during speaking or vocalizing. Despite the importance of self-monitoring, the underlying neural mechanisms in the mammalian brain, in particular the role of vocalization-induced suppression, remain virtually unknown. Here we show that neurons in the auditory cortex of marmoset monkeys (*Callithrix jacchus*) are sensitive to auditory feedback during vocal production, and that changes in the feedback alter the coding properties of these neurons. Furthermore, we found that the previously described cortical suppression during vocalization actually increased the sensitivity of these neurons to vocal feedback. This heightened sensitivity to vocal feedback suggests that these neurons may have an important role in auditory self-monitoring.

Vocal communication has an important role in the everyday lives of humans and many other animal species. When we speak, the sound of our voice is both delivered to an intended listener and conducted back to our own ear. Such feedback is a major input to our auditory system during vocal production[1], and is subjected to continuous self-monitoring[4], which requires sensitive detection of vocal feedback changes by neurons in the auditory system. The neural mechanisms underlying vocal feedback monitory are poorly understood.

A small number of previous studies have attempted to investigate the function of the auditory cortex during vocal production. Imaging and neurophysiological studies in humans have shown reduced activity in the auditory cortex during speech production relative to passive listening[5–10]. Similarly, investigations in non-human primates have demonstrated that most auditory cortex neurons exhibit vocalization-induced suppression of neural firing (spontaneous or sound-evoked) during vocal production[11–13]. Previous studies in primates have also shown a smaller subpopulation of auditory cortex neurons that are excited during self-initiated vocalizations. In addition, attenuation of neural signals is also present in the auditory brainstem during vocalization[14], but differs from the vocalization-induced suppression in cortex in that the latter begins several hundred milliseconds before vocal onset[12]. Suppression of auditory cortex neural activity during vocal production contrasts sharply with the typical excitatory responses of cortical neurons in response to the playback of recorded vocalizations that fall into a neuron's receptive field. This suppression is thought to originate from brain regions that initiate and control vocal production. How vocal feedback is encoded during vocal production and the contribution of vocalization-induced suppression to auditory self-monitoring, however, is unclear.

In this study, we examined whether neurons in the auditory cortex were sensitive to auditory feedback during vocal production. Using chronically implanted multi-electrode arrays (Supplementary Fig. 1a, b), we recorded 240 single neurons from the auditory cortices of marmoset monkeys (*Callithrix jacchus*), a highly vocal primate species, while the animals made voluntary, self-initiated vocalizations. By altering the animal's perceived vocal feedback with custom headphones and real-time frequency shifts of ± 2 semitones we found that many auditory cortex neurons were highly sensitive to feedback during vocalization.

Figure 1 illustrates two representative examples of neural responses during vocalization under normal (baseline) or altered feedback conditions. The first neuron (Fig. 1a–c) was suppressed by the animal's own vocalizations under baseline feedback conditions, with a mean response modulation index (RMI) of -0.39. The RMI measures the relative change in firing rate during vocalization as compared with the firing rate before vocalization. This same neuron became strongly excited when the animal vocalized in the presence of $+2$ semitone frequency-shifted feedback (RMI $= 0.70$), as can be seen for multiple vocalizations in the raster (Fig. 1b) and peri-stimulus time histogram (PSTH) (Fig. 1c). As a control, we also tested amplified ($+10$ dB), but not frequency-shifted, feedback and found that it did not change the response from the baseline condition (that is, unaltered feedback). A second neuron (Fig. 1d–f) was excited during normal vocalizations (RMI $= 0.22$) and showed an increase in firing rate under frequency-shifted feedback conditions (RMI $= 0.55$), but not under amplified feedback conditions (RMI $= 0.20$). The firing rate increased significantly in both neurons during frequency-shifted feedback conditions when compared to both unaltered (baseline) and amplified feedback conditions ($P < 0.001$, Kruskal–Wallis analysis of variance (ANOVA)). These examples demonstrate that neurons in the auditory cortex, despite being highly modulated by vocal production, are sensitive to auditory feedback of self-produced vocalizations. This is surprising, particularly for suppressed neurons (for example, Fig. 1a–c) where the vocalization-induced inhibition might be expected to reduce feedback sensitivity.

Overall, neurons suppressed during vocalization, which account for approximately three-quarters of the neurons studied in the auditory cortex[12], exhibited increased activity during frequency-shifted feedback compared with the baseline condition. The average activity of this population of neurons was strongly inhibited during normal vocal production (Fig. 2a). During altered feedback, the firing rate of these neurons increased, but remained suppressed as compared with

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the pre-vocal activity. The second, smaller, population of neurons excited during normal vocalization seemed to be less sensitive to altered feedback (Fig. 2b). The average firing rate of these neurons was slightly reduced in the presence of altered feedback, although the change was much smaller than that observed for suppressed neurons (Fig. 2a). The effect of altered feedback can still be seen when activities of all neurons (suppressed or excited) are averaged together and when different call types are separately analysed (Supplementary Fig. 2).

We analysed the effect of altered feedback on individual neurons within suppressed and excited populations (Fig. 2c and Supplementary Fig. 3). Within these populations, both increases and decreases occurred in neural firing during altered feedback compared to baseline. When plotted against the baseline vocal modulation (unaltered feedback), the changes in RMI due to altered feedback in each neuron confirm the trends shown by population averages (Fig. 2a, b). Altered feedback effects were prominent in neurons with negative baseline RMI values, but not in neurons with positive or near-zero baseline RMIs ($P < 0.001$, Kruskal–Wallis ANOVA). These data indicate that, as a population, suppressed neurons were more sensitive to auditory feedback during vocalization than excited neurons, suggesting that they may have a greater role in vocal self-monitoring.

The presence of feedback-related changes in auditory cortex activity during vocal production raises an important question as to relative contributions of feedback and internal modulations to the observed neural responses. The persistence of reduced firing in suppressed neurons during altered feedback suggests the continued presence of inhibition. A direct comparison of neural responses during vocalization under baseline and frequency-shifted feedback conditions revealed a correlation (Fig. 3a), indicating that feedback combines with, rather than replaces, the underlying vocalization-induced modulation. Across the sampled neurons, both increased and decreased RMIs were observed during altered feedback as compared to the baseline (unaltered) condition (Fig. 3b), but there was an overall bias towards increased neural activity. The directions of frequency shift (+2 versus −2 semitones; Supplementary Fig. 4a) did not change the population responses, and responses were also not different between the two animals (Supplementary Fig. 5).

An alternative explanation for these results is that the differences could have been due to altered vocal production rather than auditory feedback. Auditory cortex neurons are sensitive to natural fluctuations in vocal production[13]. We analysed further the difference in RMI between altered and baseline feedback in a subset of the data

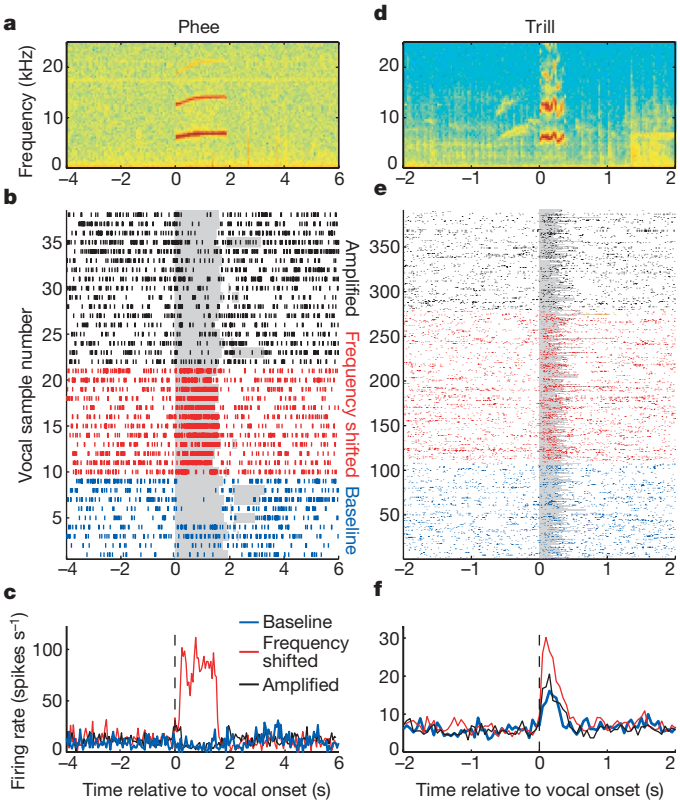

Figure 1 | Examples of vocal suppression and excitation during altered feedback. **a**, Spectrogram of a marmoset phee vocalization. **b**, Raster plot of action potentials before, during and after phees recorded from an auditory cortex neuron that was suppressed during normal vocal production. Shaded areas indicate duration of phees. Neural responses are shown during normal, baseline vocalizations (blue), +2 semitone frequency-shifted feedback (red), and amplified but unshifted feedback (black). Multiple vocalizations and corresponding cortical responses were recorded in each condition. **c**, Peristimulus time histogram (PSTH) illustrating the large increase in firing rate compared to baseline (blue) during frequency-shifted (red), but not amplified (black), feedback. **d**, Spectrogram of a sample trill vocalization. **e**, **f**, Raster plot (**e**) and PSTH (**f**) of an excited neuron whose firing also increased during a +2 semi-tone frequency shift, but not during feedback amplification.

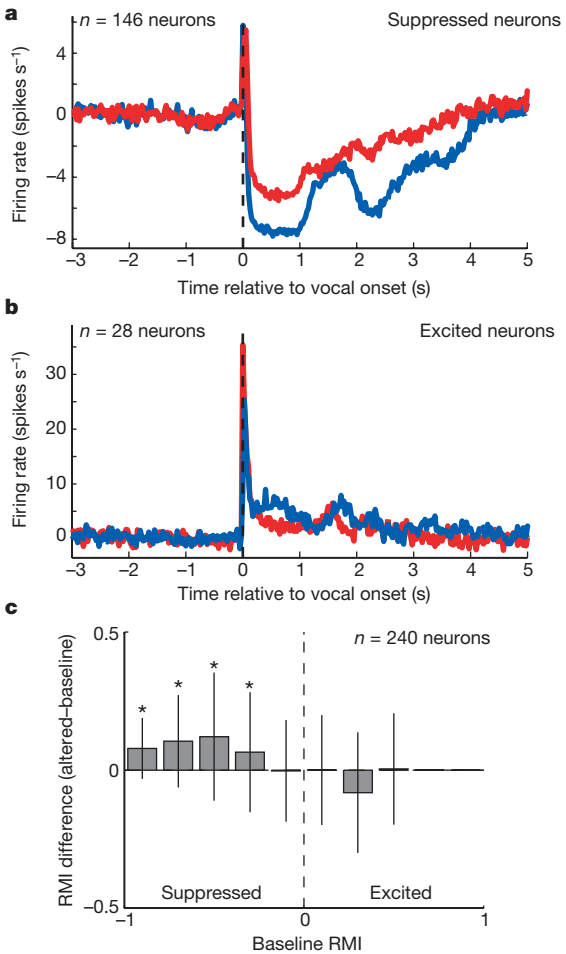

Figure 2 | Feedback effects in suppressed and excited neural populations. **a**, **b**, PSTHs showing average population responses to phee vocalizations in baseline (blue) and frequency-shifted altered feedback (red) conditions. Firing rates during altered feedback were increased in suppressed neurons (**a**; RMI < −0.2), but slightly decreased in excited neurons (**b**; RMI > 0.2). The transient increase in baseline activity (**a**) corresponds to the end of the first phrase of multi-phrased phees. **c**, Relationship between altered feedback effects and baseline vocalization-induced modulation. Differences in the RMI between altered feedback and baseline conditions were calculated for individual neurons ($n = 240$) and averaged for different ranges of baseline RMI. Data are presented as mean values and error bars indicate the s.d. Significant feedback effects are indicated (asterisk, $P < 0.001$, Wilcoxon signed-rank test). Individual data points are shown in Supplementary Fig. 3.

that contained acoustically matched vocalizations in both conditions (Fig. 3c). For each neuron, the average responses for matched and unmatched (acoustically different) vocalizations were calculated during altered feedback conditions. The RMI difference distributions for these three groups were not significantly different ($P > 0.05$, Kruskal–Wallis ANOVA), indicating that changes in neural activity were due to altered acoustic feedback, rather than altered vocal production. Furthermore, this suggests that a previously observed relationship between vocal acoustics and neural modulation[13] was probably due to the auditory feedback rather than variations in the suppressive internal modulations. The existence of auditory feedback-dependent neural responses in auditory cortex is an important observation because, up to this point, it has not been possible to separate out the roles of modulation and feedback. It implies a more complex mechanism, combining both internal modulation and feedback responses, rather than one purely reflecting internal signals.

A question remains as to the origin of the observed sensitivity to feedback during vocalization. A possible explanation is that that the effects of feedback alteration on vocal responses are due to the shifting of vocal acoustic energy into or out of the auditory receptive fields of auditory cortex neurons. This would suggest that the vocal effects of altered feedback could be predicted from the auditory effects of a similar alteration. We therefore examined the relationship between passive auditory (sensory) responses of auditory cortex neurons and the effects of altered feedback during vocalization. Neurons studied during vocal production were examined further with the playback of vocalizations previously recorded from the same animal. These vocalization stimuli were presented both with and without the same frequency shifts used during vocal feedback manipulations (Supplementary Fig. 6a, b). The difference in neural response was compared to that during vocalization. As seen from the population analysis in Fig. 4a, the auditory and vocal frequency shift effects were uncorrelated, indicating that the neural modulations observed

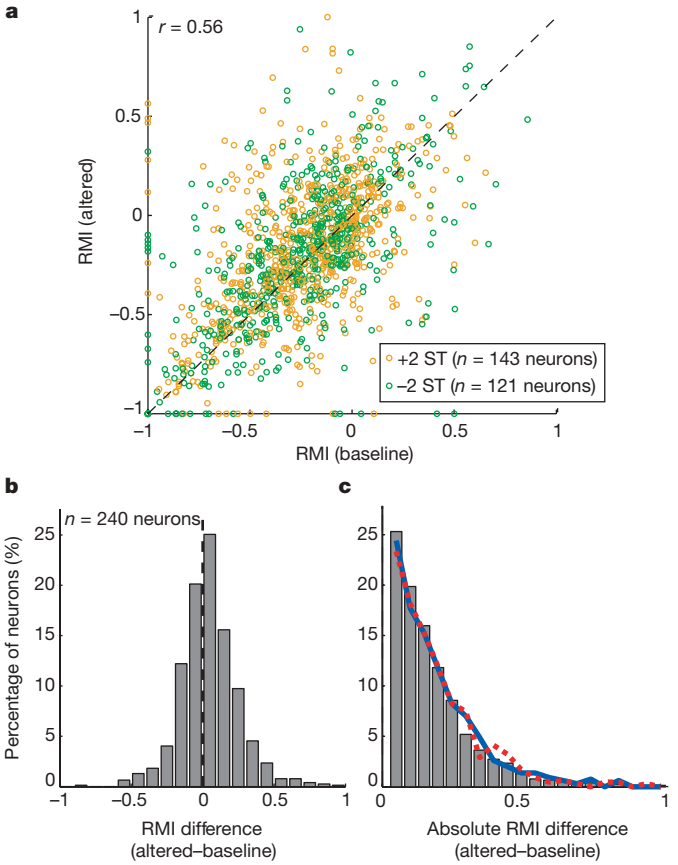

Figure 3 | Population responses to altered feedback. **a**, Scatter plot comparing frequency-shifted RMI with baseline RMI for all neurons and vocalization types. Responses were weakly, but significantly correlated (Spearman rank correlation $r = 0.56$, $P < 0.001$). Positive (orange circles) and negative frequency shifts (green circles) are shown. All three vocalization types studied are included. Points are shown for each vocalization type in each neuron (phee, $n = 197$; trilphee, $n = 162$; trill, $n = 107$). A further breakdown by frequency shift direction (+2 versus −2) and vocalization type is shown as a Supplementary Figure (Supplementary Fig. 4). ST, semitone. **b**, The distribution of the RMI difference between altered and baseline conditions illustrates the presence of both increased and decreased neural activities due to frequency-shifted feedback. The distribution was significantly shifted towards positive RMI differences (mean ± s.d. = 0.05 ± 0.21; $P < 0.001$, Wilcoxon signed-rank test). **c**, The distribution of absolute RMI difference values for all vocalizations (filled bars) is compared with those for which vocalization acoustics during altered feedback either matched (blue line) or did not match (dashed red line) the acoustics of baseline vocalizations. The feedback activity in these three conditions was not significantly affected by acoustic matching ($P > 0.05$, Kruskal–Wallis ANOVA).

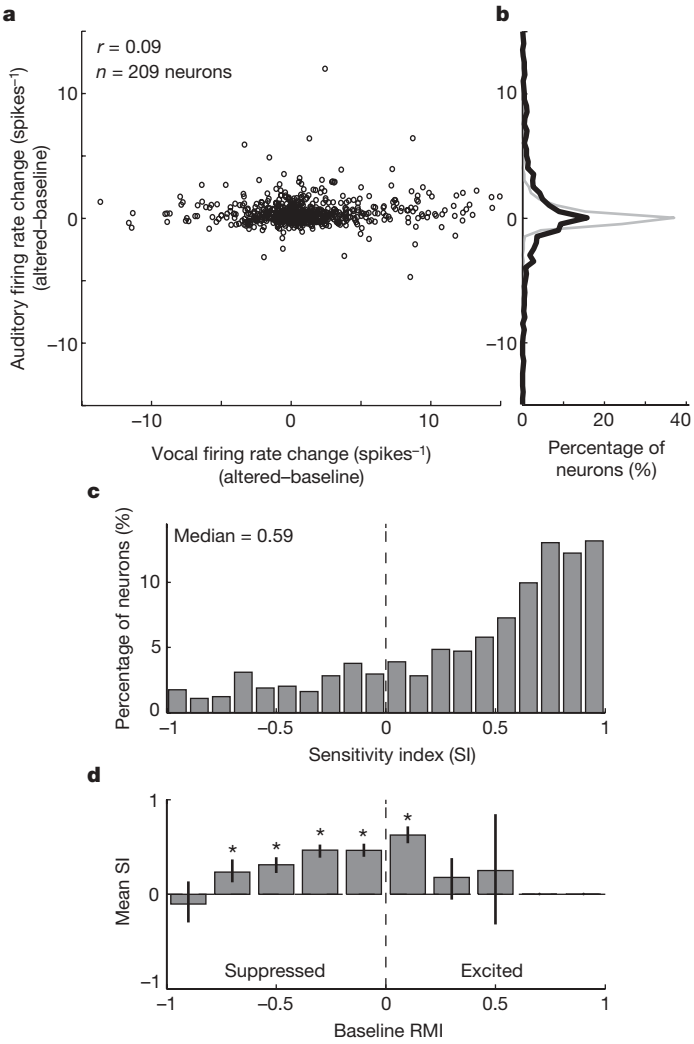

Figure 4 | Auditory responses and feedback sensitivity. **a**, Population scatter plot comparing frequency-shift effects on neural responses during vocal production and auditory playback. The vocal and auditory responses to feedback alterations were not correlated ($r = 0.09$; $P > 0.05$). Distributions of the vocal (black) and auditory (grey) data sets are shown in **b**. **c**, Distribution of the sensitivity index (SI), comparing feedback effects between vocal and auditory for the neurons in **a**, is shown (see Methods). **d**, The relationship between SI and baseline vocalization-induced modulation (measured by RMI) is shown. Most units showed an increase in sensitivity to feedback alteration during vocalization, particularly for suppressed neurons. Error bars represent bootstrapped 95% confidence intervals (asterisk, $P < 0.001$ Wilcoxon signed-rank test).

during altered vocal feedback were not simply due to the auditory (playback) responses of the auditory cortex neurons. Vocal feedback responses were similarly unrelated to the frequency tuning of the neurons (Supplementary Figs 7 and 8). The frequency shift effects were larger (greater firing rate changes) during vocalization than during playback (Fig. 4b). These findings confirm that auditory tuning cannot account for the responses observed during frequency-shifted feedback.

The absence of a clear relationship between auditory and feedback responses suggests that the underlying vocalization-induced modulation may be responsible for changing neural sensitivity during vocal production. We calculated a feedback sensitivity index (SI) for each neuron, which relates the frequency shift effects during vocalization to those during auditory (playback) stimuli. An SI of +1 indicates that a neuron is sensitive to the frequency-shift alteration during vocalization, but not during playback, and an SI of −1 indicates the opposite. The distribution of the SI for the auditory cortical population (Fig. 4c) showed a large concentration towards +1 (median 0.59; $P < 0.001$, Wilcoxon signed-rank test). This indicates that most neurons are more sensitive to frequency shifts during vocalization than predicted from their auditory responses.

To understand this increased feedback sensitivity, we compared the SI with the baseline vocal modulation measured in each neuron (Fig. 4d). The analysis revealed that the largest increases in feedback sensitivity were found in the suppressed and weakly excited neurons. This statistically significant trend ($P < 0.001$, Kruskal–Wallis ANOVA) suggests that vocalization-induced suppression acts to increase, rather than decrease, the sensitivity of auditory cortex neurons to auditory feedback during vocalization. Weakly excited neurons may have been similarly affected because their responses combine elements of both excitation and suppression. The most strongly suppressed neurons failed to show this feedback sensitivity, probably because of the lack of ongoing neural activity during vocalization. Additional analysis of the sensitivity to altered feedback based on a d' (discriminability) measure (Supplementary Fig. 9) demonstrated similar properties to those revealed by the SI analysis (Fig. 4). Population-averaged PSTHs for auditory playback (Supplementary Fig.10) showed, unlike their vocalization counterparts (Fig. 2), larger differences for excited than for suppressed neurons, supporting the notion that the increase in sensitivity to altered feedback is specifically related to vocalization-induced suppression. The observed feedback responses show, for the first time, that one function of vocal suppression is to increase auditory feedback sensitivity during vocalization. Previous work in the cricket cercal (auditory) system has also shown inhibition of auditory responses[15]. However, in contrast to the sensitization during vocalization we have demonstrated in primates, inhibition transiently desensitizes the cricket auditory system to increase sensitivity immediately after stridulation[15].

The effects of vocal feedback alteration or distortion during speaking have been studied previously in the human auditory cortex, with results showing the suppression of the auditory cortex during natural vocalization[5–10] and a small increase in activity during feedback alterations[5,16–18]. The similarity between these results and our current findings suggests that common mechanisms may be shared in the sensory components of both human and non-human primate vocal production. We have previously suggested that the reduced activation of the auditory cortex during speaking seen in human imaging studies may result from a combination of the underlying activity of suppressed and excited neurons[12,13]. It is therefore possible that the altered feedback effects observed in humans may also be due to combinations of increased and decreased activity in individual neurons. Our findings also suggest that the human auditory cortex may exhibit sensitization to auditory feedback during speaking.

How vocalization-induced suppression contributes to the apparent increase in feedback sensitivity remains to be elucidated. One possibility is that the suppression acts to modulate auditory sensitivity non-selectively by scaling the gain of neural responses, thereby magnifying the effects of feedback perturbations. Another possibility is that the modulatory signals (termed corollary discharges or efference copies[19]) contain specific predictions of the expected auditory input (a forward model[20]) that are compared to the actual vocal feedback; the resulting auditory cortex activity represents the deviation from expectancy (error signal). This idea is consistent with self-monitoring for error detection, but conflicts with previously observed changes in AC neural responses that correlate with fluctuations in vocal acoustics in the absence of altered feedback[13]. A final possibility is that modulatory signals induce a transient change in auditory receptive fields during vocalization to better predict acoustic feedback. This might explain why feedback sensitivity exists in neurons whose playback auditory receptive fields do not overlap vocal acoustics. Such changes in receptive field have been observed peri-saccadically in the visuo-motor lateral intraparietal area[21].

Because of the intrinsic variability of marmoset vocal behaviour and the necessity of performing the reported vocal experiments in the marmoset colony, there are several factors that could have affected the apparent increase in feedback sensitivity but cannot be completely controlled for in the present study. For example, differences in background noise between auditory playback (conducted in the sound-proof chamber) and vocalization experiments (conducted in the marmoset colony) were present. Fluctuations in the animal's behavioural state between different experimental conditions may have also been present. Finally, there were potential differences between vocal production and playback because playback stimuli lacked the full diversity of the produced vocalizations. These factors, although unlikely to account completely for the reported observations, need to be kept in mind when interpreting our findings.

Self-monitoring of vocal feedback may have several important functions. In non-human primates, discrimination between self-generated and external sounds may play a part in behaviours where assigning an auditory input as self is important. These include antiphonal calling, an interactive vocal exchange behaviour seen in marmosets and other monkeys[22], and vocal convergence, the tendency of monkeys to match their vocal acoustics to that of their cage-mate[23]. Sensitive monitoring of auditory feedback to detect vocal production errors is also an essential step in feedback-mediated vocal control. Humans constantly monitor their speech and quickly compensate for perceived changes in feedback[24,25], including the frequency-shifted feedback used in these experiments[26]. Feedback-dependant vocal control in non-human primates is less well understood. Monkeys can change the amplitude of their vocalizations when their feedback is disrupted by masking noise[27]. However, there is no published data showing them to exhibit vocal compensation during frequency-shifted feedback. This lack of a direct behavioural correlate for our feedback alterations limits the conclusions that can be drawn from the observed feedback sensitivity in auditory cortex. Nonetheless, a possible role in feedback-mediated vocal control remains an intriguing possibility for future studies, especially as defects in feedback monitoring have been suggested to underlie human communication disorders such as stuttering[28].

METHODS SUMMARY

Two marmoset monkeys were implanted bilaterally with two multi-electrode arrays, one in each auditory cortex. The 16-channel microelectrode arrays (Warp-16, Neuralynx) were adapted from larger multi-electrode designs[29]. Only well-isolated single units were analysed. Neural recordings included both primary auditory cortex and lateral fields and all cortical layers.

Vocalizations were recorded synchronously with neural signals using directional microphones. Experiments were mainly performed with the animals in the setting of the marmoset colony, allowing vocal exchanges between the subject and other animals in the colony. Additional experiments were performed in the laboratory with the animals vocalizing in response to the playback of vocalizations from a loudspeaker. Experiments were conducted with the animal either seated in a primate chair or moving around freely within a cage.

Vocalization feedback was modified in real-time using a digital effects processor and frequency shifts of ±2 semitones. Shifted signals were presented to the

animal through customized headphones at a level ~10 dB SPL (sound pressure level) louder than vocalizations produced by the animal. Auditory control experiments were performed by playing an animal's recorded vocalizations through a loudspeaker at similar amplitudes while the animal sat quietly in a sound-proof chamber.

Neural responses to self-produced vocalizations were quantified using the vocal response modulation index (RMI; see Methods). Sensitivity to feedback alteration was calculated using a sensitivity index (SI; see Methods).

Full Methods and any associated references are available in the online version of the paper at www.nature.com/nature.

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Supplementary Information is linked to the online version of the paper at www.nature.com/nature.

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Author Contributions S.J.E. and X.W. designed the experiments and co-wrote the paper. S.J.E. carried out the experimental recordings and data analysis.

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METHODS

Animal preparation and neural recording. Two marmoset monkeys were implanted bilaterally with two multi-electrode arrays (Supplementary Fig. 1a, b), one in each auditory cortex. The 16-channel microelectrode arrays (Warp-16, Neuralynx Inc.) were scaled-down versions of the larger multi-electrode arrays developed for use in studies of rodents and macaque monkeys[29]. Before array placement, animals were implanted with a headcap using procedures previously developed for marmosets[30]. The microelectrodes used were either tungsten or platinum-iridium (impedances 2–4 MΩ). Electrodes were individually moveable using a removable pushing device (Neuralynx).

Neural signals were recorded via a headstage on the animal end of a wire tether, amplified and band-pass filtered, and then digitized onto a personal computer. Neural signals were monitored online to optimize signal quality by electrode movements, and to guide auditory stimulus selection. Action potentials (spikes) were sorted offline using custom software and a principle component-based clustering method. Spikes were classified as either from a single- or multi-unit based on a minimum SNR (signal-to-noise ratio) $>$ 13 dB ($>$4.5:1) and presence of a refractory period. A total of 501 units were recorded during these experiments, of which 240 were later classified as single units. Only single units were included in the data reported here. Neurons were sampled in both hemispheres of the two animals, including primary auditory cortex and lateral fields (lateral belt and parabelt areas) and all cortical layers.

Vocal recording. Acoustic signals were recorded using directional microphones, placed ~20 cm in front of the animals, and digitized synchronously with neural signals. Vocalizations were extracted offline from the recordings and manually classified into established marmoset call types based on spectrograms. Only three of the major vocalization types were included for analysis: phees, trilphees and trills. Vocal experiments were primarily performed with the animals in the setting of the marmoset colony, allowing vocal exchanges between the subject animal and other animals in the colony. Multiple microphones were used to monitor vocalizations produced by the subject and the rest of the colony. Additional experiments were performed in the laboratory with the animal vocally interacting to the playback of vocalizations from a speaker, a behaviour known as antiphonal calling[22]. Animals made a wide variety of vocalizations in the marmoset colony, but only made isolation calls (phee) during antiphonal experiments. Experiments were conducted either with the animal seated in a primate chair but with head restraint removed, or when moving around freely within a small custom-made cage. Wire tethers were used when recording neural signals from free-roaming animals.

Feedback alteration. Vocalization feedback was modified in real-time using a digital effects processor (Yamaha SPX 2000). Frequency shifts of ± 2 semitones were used. This shift magnitude fell within the normal range of marmoset vocal variation. Shifted signals were presented to the animal through customized headphones (Supplementary Fig. 1c, d), modified to attach to the animal's headcap, at a level ~10 dB SPL louder than direct (air-conducted) feedback. Feedback experiments were conducted in a blocked fashion with: (1) an hour of recording baseline (unaltered) vocalizations; (2) an hour of recording with frequency-shifted feedback; and (3) half an hour of recording with amplified, but not frequency-shifted, feedback as a control. More than one frequency shift per session was generally not possible because of time limitations to obtain sufficient vocalizations.

Auditory stimuli. Before vocal recordings, neurons' auditory responses were characterized with the animal seated in a primate chair within a sound-proof chamber. Auditory stimuli were presented free-field through a speaker located 1 m in front of the animal. Centre frequencies of neurons were determined by pure tone or band-pass noise stimuli. Animals were also presented with multiple samples of its own, previously recorded, vocalizations. Frequency-shifted playback stimuli, created from recorded vocalizations using the vocal effects processor, were added back to the original vocal stimuli with an appropriate relative amplitude (+10 dB) and delay (10 ms) to match acoustically the conditions heard during vocal production with altered feedback. Both normal and frequency-shifted stimuli were presented at multiple sound levels, but only those overlapping the produced vocalizations were used for analysis.

Data analysis. Neural responses to self-produced vocalizations were quantified using the vocal response modulation index (RMI). $\mathrm{RMI} = (R_{\mathrm{vocal}} - R_{\mathrm{prevocal}})/(R_{\mathrm{vocal}} + R_{\mathrm{prevocal}})$, where R_{vocal} is the firing rate during vocalization and R_{prevocal} is the firing rate before vocalization. An RMI of −1 indicated complete suppression of neural activity and +1 indicated strongly driven vocalization responses, a low pre-vocal firing rate, or both. The effect of altered feedback on neurons was determined by calculating RMIs for individual vocalizations samples under both baseline (unaltered) and altered-feedback conditions and comparing the average RMI from both conditions. The effects of amplified feedback alone were examined in a subset of data, but found to be negligible in most neurons and were not subjected to further analyses. Population comparisons of feedback effects on suppressed ($\mathrm{RMI} \leq -0.2$) and excited ($\mathrm{RMI} \geq 0.2$) neural populations were made by calculating PSTHs aligned by vocalization onset. Additional analyses compared responses to acoustically matched and unmatched vocalizations. "Matched" vocalizations were those produced during frequency-shifted feedback whose SPL and mean fundamental frequency fell within the 25th–75th percentile range of the vocal acoustics measured for the baseline vocalizations.

Auditory playback effects of feedback alterations were measured by comparing responses to normal and frequency-shifted vocal samples and then compared to feedback effects during vocalization. Frequency-shifted stimuli were combined with normal stimuli to match the conditions during vocal production and altered feedback. A measure of neural sensitivity to auditory feedback alteration, the feedback Sensitivity Index (SI), was calculated as $\mathrm{SI} = (|\Delta \mathrm{FR}_{\mathrm{voc}}| - |\Delta \mathrm{FR}_{\mathrm{aud}}|)/(|\Delta \mathrm{FR}_{\mathrm{voc}}| + |\Delta \mathrm{FR}_{\mathrm{aud}}|)$, where $\Delta \mathrm{FR}_{\mathrm{voc}}$ was the change in firing rate during vocalization between normal and altered feedback, and $\Delta \mathrm{FR}_{\mathrm{aud}}$ was the change in firing rate between normal and frequency-shifted vocal sounds during auditory stimulus playback.

Statistical tests were performed using non-parametric methods, including Wilcoxon rank-sum and signed-rank tests to test differences between distribution medians. Multiple comparisons were performed using Kruskal–Wallis ANOVAs with Bonferroni corrections. Correlation coefficients were carried out using Spearman rank correlations. Confidence intervals were calculated using 200 repetition bootstrapping.

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