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Title: Specific Brain Regions Involved in Decoding of the Anger Acoustic Parameters

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Abstract

Background. The purpose of the present study was to identify brain regions sensitive to emotion-specific acoustic parameters in healthy individuals.

Method. Three pseudo-words consecutively in the form of one stimulus were spoken with neutral and angry prosody. Then, we changed the acoustic parameters (mean fundamental frequency, intensity, and speech tempo) in angry prosody. The stimuli were presented in a functional magnetic resonance imaging (fMRI) experiment to detect anger or neutrality.

Results. Stronger activation in the left superior temporal gyrus (STG) and Heschl's gyrus (HG) when the mean f0 converted from 300 Hz to 250 Hz was observed. Increased activity in the right posterior STG and posterior middle temporal gyrus (MTG) was revealed in more intensity anger prosody. Moreover, we found stronger activity in the right mid-STG, MTG, and the left STG in a faster speech tempo.

Conclusion. According to the increased activity in the STG and MTG of both hemispheres following the more intense anger (lower fundamental frequency, more intensity, and faster speech tempo), it can probably be concluded that a more intense comprehension of anger is resulted from the sending different information from these regions to the inferior frontal gyrus (IFG) and orbital frontal cortex (OFC).

Keywords: Acoustic parameters; Emotional prosody; Functional MRI; Intensity; Mean F0; Speech tempo

Introduction

Speech prosody refers to fluctuations in pitch (fundamental frequency), variations in loudness (intensity), durational features (e.g., phone, syllable, word, and phrase length; pausing, rhythm, and speech tempo), and voice quality [1-4]. Prosody can serve a range of functions such as linguistic, pragmatic, and emotional functions [5]. These parameters can be influenced by affective states and play an important role in emotion perception [6]. An incorrect interpretation of emotional prosody can cause failure in social interactions and increased risk of social isolation [7-9]. Finding the neural mechanism underlying the emotion-specific acoustic parameters can improve our knowledge of prosodic impairments (dysprosodia).

A large number of neurological studies have found that the right cerebral cortex is responsible for emotional prosody processing [10-38]. Although most lesions and neurological studies have suggested the right hemisphere (RH) dominance for emotional prosody, others have found no difference between the left hemisphere (LH) and RH lesion effects [27, 32, 39-42].

Functional MRI studies showed that widespread brain networks are involved in decoding emotional prosody [43-45]. In detail, auditory temporal regions such as the primary/secondary auditory cortex (AC), superior temporal cortex (STC) [11, 43, 45-61], and other temporal areas such as supramarginal gyrus, right parahippocampal (BA 28) gyrus and subcallosal (BA 34) gyrus [62], frontal areas such as the inferior frontal cortex (IFC) and orbital frontal cortex (OFC) [43, 51-55, 63, 64], insula [56], cerebellum [62] and subcortical structures such as thalamus, basal ganglia and amygdala [53, 54, 56, 59, 63-65] have been found to be involved in the perception of emotional prosody.

Furthermore, according to a hierarchical model that has been proposed for emotional prosody processing [43, 49, 66], voice-sensitive structures of the AC and mid-STC contribute to the extraction of acoustic parameters; the posterior part of the right STC contributes to the identification of emotional prosody, and eventually, the semantic comprehension of affective prosody is concerned with the bilateral IFG and OFC [45, 58].

As discussed above, most studies have focused on emotional prosody processing, and there are few reports on how emotion-specific acoustic parameters are processed. In the following, we

refer to studies related to the processing of acoustic parameters including pitch, intensity and duration separately.

1. pitch

The lesion studies addressed the role of the hemispheres in pitch processing. It has been demonstrated that the RH is related to pitch processing in speech [67]. Patients with the RH lesions use duration cues rather than F0-variability to assess affective prosody. In other words, right temporoparietal lesions could disrupt the discrimination of tones [67].

Several structural and functional neuroimaging studies have shown that inferior frontal regions in RH [68, 69], left STG, left Heschl's gyrus (HG), and the right temporal pole [59], pars triangularis of Broca's area [70], the right posterior STG and left STG subregions [44], and Heschl's gyrus (HG) and adjacent cortical areas in STG [71] were the main structures for pitch processing. In addition, it has been suggested that the lateral HG functions as a general "pitch center" [72], and the processing of pitch patterns such as melodies, involves much more distributed processing in the superior temporal lobes and frontal lobes [73]. However, another study demonstrated that parts of the planum temporal are more relevant for pitch processing than lateral HG [74]. In addition, the anterior temporal cortex is more sensitive to female voices with high F0 than to male voices with low F0 [75].

2. Intensity and duration

Previous reports found widespread activation clusters in the bilateral AC to stimuli that are deviant in intensity or duration [76-78], in the STG and MTG of both hemispheres to intensity and duration, in the right IFG and superior frontal gyrus (SFG) to duration [59], and in the right posterior STG and the left STG to intensity variations in emotional prosody [44, 79].

In sum, it seems that different brain regions may be involved in emotion-specific acoustic parameters processing. Finding the neural representation of each emotion-specific acoustic parameter may improve our knowledge about the neural mechanism of emotional prosody processing. Difficulties in emotional prosody comprehension, including anger in neurological disorders such as Alzheimer's, Parkinson's, Traumatic brain injury (TBI), or psychological disorders such as depression, autism, and alexithymia cause failure in social relationships and increase the risk of isolation [7-9]. Recent studies have demonstrated the improvement of some

neurological, psychological, and motor disorders using non-invasive protocols such as tDCS and rTMS. Previous studies showed that the stimulation of a region such as the dorsal and lateral part of the prefrontal cortex leads to the strengthening of the functional connectivity of the brain networks [80, 81]. Rare studies have been conducted in the treatment of emotional prosody disorders. Findings the brain regions and functional connectivity sensitive to changing the acoustic parameters of the emotional prosody of anger can probably help in providing therapeutic solutions.

The emotional prosody used in the present study was anger. It was demonstrated in the previous reports [82] that lower mean f_0 , louder voice, and faster speech tempo constitute acoustic parameters of anger. However, it was revealed in one study [83] that “hot” anger seemed to be characterized by an increased in mean F_0 and decreased in mean F_0 was probably due to ‘cold’ anger. Thus, it is expected that with increasing intensity and speech tempo and changing the mean f_0 , the anger becomes more intense and the brain regions sensitive to these parameters show different activity. In this study, we tried to use a novel experimental fMRI design in which a range of different anger stimuli with difference in only one acoustic parameter every time were presented to participants. The goals of the present study were two-fold. In the first place, our aim was to identify brain regions sensitive to emotion-specific acoustic parameters. In the second place, we wanted to investigate the difference in brain activity related to emotion-specific acoustic parameters variations. We hypothesized that increasing anger intensity following to lower frequency, louder (more intensity) and faster speech tempo leads to stronger activity in the brain regions sensitive to emotion-specific acoustic parameters. To ensure the relevance of the results with the difference in only one emotion-specific acoustic parameter, we changed one of the acoustic parameters each time while keeping the other two constant, and the stimuli were spoken only by one speech and language pathologist.

Materials and methods

Participants

Twenty healthy young male adults participated in the study (ages ranging from 18 to 35, mean age: 23.51 years, standard deviation [SD] = 5.08 years). All participants were native Persian speakers, right-handed, and had normal or corrected-to-normal vision. No participant had a history of neurological or psychiatric problems, substance abuse, or impaired hearing. Furthermore, the Toronto alexithymia scale was used to identify individuals who have trouble understanding emotions. All participants provided informed and written consent for participation in the fMRI study. MRI data had to be excluded from four participants because of incorrect responses. Thus, the results reported are based on the analysis of the remaining 16 participants. This study was approved by the ethical committee of the Iran University of Medical Sciences. The ethical code was IR.IUMS.REC.1399.1284

Stimuli

Three pseudo-words (“çârs”, “mâruk”, and “nirâpat”) were selected from a Persian language study [84] that was administrated a Persian non-word repetition (NWR) test. These pseudo-words were spoken in either a neutral or an angry tone by a male speech and language pathologist. After that, the three pseudo-words were used consecutively in a form of one stimulus. Then, the acoustic parameters (mean f_0 , intensity, and speech tempo) of the anger stimulus was changed using Audacity software. Every time, one of the parameters was changed while keeping the other two constant. In detail, the mean f_0 was changed between 200 and 400 Hz while keeping the other two parameters constant. Once again, by keeping the mean f_0 and tempo constant, the mean intensity was changed between 50 and 90 dB. Finally, the speech tempo was changed between 1 to 5 seconds without changing the mean f_0 and intensity. After applying the changes, to judge if they were anger or not, they were piloted on a group of healthy adults ($N = 10$) before the fMRI study. These healthy individuals were selected among the students of the Iran University of Medical Sciences. Finally, two changes were selected in the highest and lowest range of anger in each acoustic parameter. The highest and lowest mean f_0 detectable as anger prosody was about 350 and 250 Hz, respectively. The highest and lowest rate of change in speech tempo that could be recognized as anger prosody was about 4.2 and 2.1

seconds, respectively. The highest and lowest audible intensity in the fMRI were about 90 and 70 dBs, respectively. Therefore, six new changes (two changes in each parameter) were administrated (see Table 1).

Experimental design

During the fMRI-scan, auditory stimuli were presented binaurally using magnetic resonance imaging compatible headphones. Participants were lying in the scanner with their eyes open staring at the screen. Auditory stimuli were preceded by a visual fixation cross ($1 \times 1^\circ$) for 2s. The participants were asked to pay attention to the emotion of the auditory stimuli in fMRI experiment to detect anger or neutral stimulus and distinguish the correct option by pressing the button (right index, left index) after displaying the options on the screen for 2s. Auditory stimuli were presented during three blocks of prosody discrimination on the stimuli (angry or neutral; right index and left index) in two runs. Each anger/neutral discrimination block contained 12 conditions with 3 parameters (anger_neutral, stimulus_time, jitter), including 2 silent events with no auditory stimulation, 3 neutral events, and 7 anger events (see Figure 1). Every block of prosody discrimination on the stimuli lasted 132 seconds.

Image Acquisition

Structural and functional imaging data were obtained using a 3T PRISMA scanner in the National Brain Mapping Laboratory (NBML). A magnetization prepared rapid acquisition gradient echo (MPRAGE) m3 sequence was employed to acquire high-resolution ($1 \times 1 \times 1$ mm³) T1-weighted structural images (TR=1600 ms, TE=3.47 ms, TI=800 ms). Functional images were obtained using a multislice echo planar imaging (EPI) sequence (36 axial slices, slice thickness: 3.0 mm, TR=2000 ms, TE=30 ms, field of view (FOV)=195 mm, flip angle=90°).

Image analysis

The fMRI data analysis was performed using Statistical Parametric Mapping SPM (version 12; Wellcome Department of Cognitive Neurology, London, UK). Preprocessing was performed using default settings in SPM 12. Functional images were realigned and coregistered to the

anatomical image. A segmentation of the anatomical image revealed warping parameters that were used to normalize the functional images to the Montreal Neurological Institute (MNI) stereotactic template brain. Normalized images were spatially smoothed with a nonisotropic Gaussian kernel of full-width at half-maximum $3 \times 3 \times 4$ mm.

A general linear model [85] was used for the first-level statistical analysis, in which separate regressors were defined for each trial using a stick function convolved with the hemodynamic response function. Events were time-locked to stimulus onset. Separate regressors were created for each experimental condition. Linear contrasts for the conditions for each participant were taken to a second-level random effects group analysis of variance.

Factorial subtraction analysis was employed to evaluate which brain regions respond more strongly to changes in emotion-specific acoustic parameters of anger prosody. To examine whether hemodynamic responses in the temporal lobe, especially STG, are subject to repetition suppression effects, parameter estimates of the most significantly activated voxel in this area were submitted to a two-factorial analysis of variance (ANOVA) with emotion-specific acoustic parameters variations and repetition (first fMRI session, second fMRI session) as within-subject factors. Activations are reported at a height threshold of $p < 0.001$ uncorrected and an extent threshold of 0 voxels. Significance was examined at the cluster level with an extent threshold of $p < 0.05$ (corresponding to a minimal cluster size of 50 voxels) corrected for multiple comparisons across the whole brain.

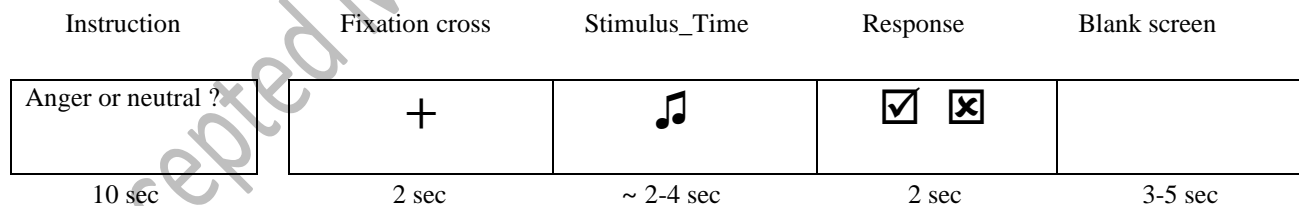


Figure 1: illustration of an example trial of the fMRI task. The instruction was displayed for 10 s before every block. A fixation cross was displayed after a blank screen for a total of 5-7 s. After that, audio stimulus was played for anger/neutral discrimination. The duration of each stimulus was between about 2 and 4 seconds. The time required to respond was 2 seconds.

Table 1: Acoustic parameters description of all stimuli

	Mf0 (Hz)	Intensity (dB)	Speech tempo (s)
Main stimulus (anger)	~ 300	~ 80	~ 3
Mean f0 variations	~ 250	~ 80	~ 3
	~350	~80	~ 3
Intensity variations	~ 300	~ 70	~ 3
	~ 300	~ 90	~ 3
Speech tempo variations	~300	~ 80	~ 2
	~300	~80	~ 4

Results

The hemodynamic responses of the various acoustic parameters of anger prosody will be described in detail.

1. Mean F0

To examine brain activation in response to mean F0 variations in anger prosody, a one-sample t-test was run for the contrasts: anger (300 Hz) > anger (250 Hz) prosody, anger (250 Hz) > anger (300 Hz) prosody, anger (350 Hz) > anger (300 Hz) prosody, and anger (300 Hz) > anger (350 Hz) prosody. Comparison of these contrasts with each other showed increased activity in the left STG (MNI Coordinates: x= -60, y= -20, z= 8; T= 5.33; cluster size= 17 voxels) and in HG (MNI Coordinates: x= -55, y= -22, z= 9; T= 5.33; cluster size= 17 voxels) in response to anger (250 Hz) prosody compared with anger (300 Hz) at $p < 0.001$ (uncorrected). (Figure 2). No significant clusters were found in other contrast comparisons.

2. Mean Intensity

When we compared brain activity among intensity variations (70, 80, and 90 dBs) while keeping the other two acoustic parameters constant, the STG and MTG in the RH showed greater activation to anger prosody with more intensity. This increased activation was greater for anger (90 dB) compared with anger (70 dB) [90 dB > 70 dB] than for anger (90 dB) compared with anger (80dB) [90 dB > 80 dB] (Figure). In detail, when we compared brain activation between anger (90 dB) and anger (70dB) (90 dB > 70 dB), our analysis revealed stronger activity in the right posterior STG (MNI coordinates: $x = 42, y = -38, z = 6$; $T = 7.4$; cluster size = 22 voxels) and a significant cluster in the right posterior MTG (MNI Coordinates: $x = 49, y = -38, z = 4$; $T = 7.4$; cluster size = 22 voxels) at the threshold of $p < 0.001$ uncorrected. In addition, when we compared brain activation between anger (90 dB) and anger (80 dB) (90 dB > 80 dB), we found increased activity in the right posterior STG (MNI Coordinates: $x = 38, y = -38, z = 8$; $T = 6.52$; cluster size = 21 voxels). Furthermore, the difference in activity extent in anger 90 dB compared with anger 70 dB is greater than anger 90 dB compared with anger 80 dB (Figure 2).

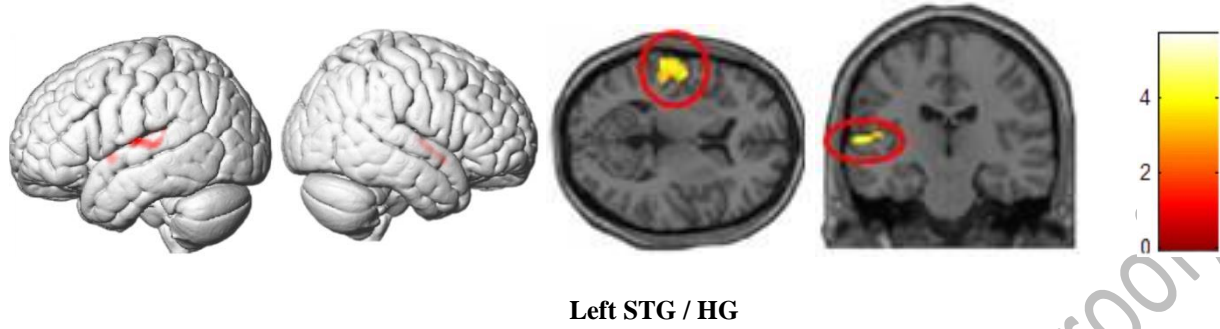
3. Duration (speech tempo)

Comparison of hemodynamic responses to anger (2s) compared with anger (3s) (anger (2s) > anger (3s)) revealed two significant clusters in the temporal lobe of both hemispheres, which were located within the mid-STG in the RH (MNI coordinates: $x=54; y=-20; z=0$; BA:22; $T = 9.3$; cluster size = 27 voxels), and the posterior STG in the LH (MNI coordinates: $x=-64; y=-36; z=16$; BA:22; $T = 6.6$; cluster size = 27 voxels) at the threshold of $P < 0.001$ (uncorrected) with a minimum cluster extent of $k = 0$ voxels. This activation was stronger in the RH (Figure). On the other hand, when we compared brain activity between anger (4s) and anger (3s) (anger (4s) > anger (3s)), we found increased activity in the right MTG (MNI coordinates: $x=50; y=-26; z=-8$; $T = 8.2$; cluster size = 21 voxels), and in the posterior STG in LH (MNI coordinates: $x=-66; y=-40; z=6$; $T = 7.8$; cluster size = 21 voxels) at the level of $P < 0.001$ (uncorrected) with a minimum cluster extent of $k = 0$ voxels. This activation was stronger in LH (Figure). Eventually, the comparison between anger (2s) and anger (4s) (anger (2s) > anger (4s)) revealed stronger activity

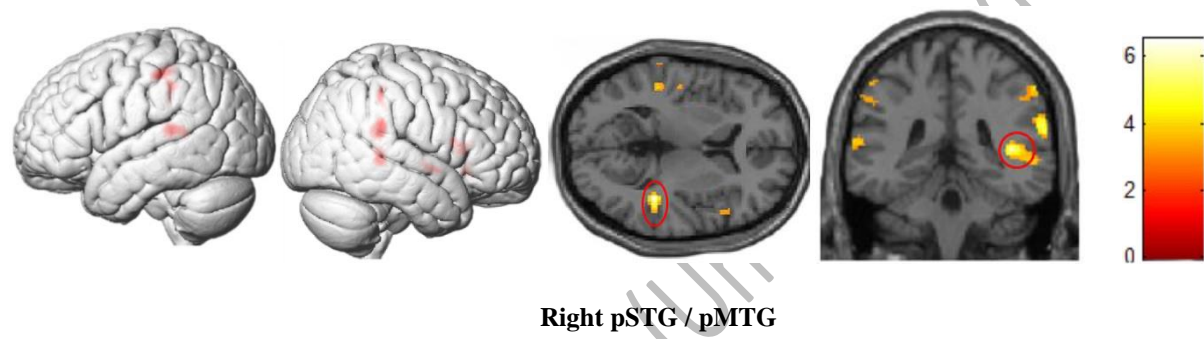
in the right mid-STG (MNI coordinates: $x=54$; $y=-18$; $z=0$; $T = 11.8$; cluster size = 27 voxels) and in the left STG (MNI coordinates: $x=-54$; $y=-2$; $z=0$; $T = 5.9$; cluster size = 27 voxels) at the level of $P < 0.001$ (uncorrected) (Figure 2).

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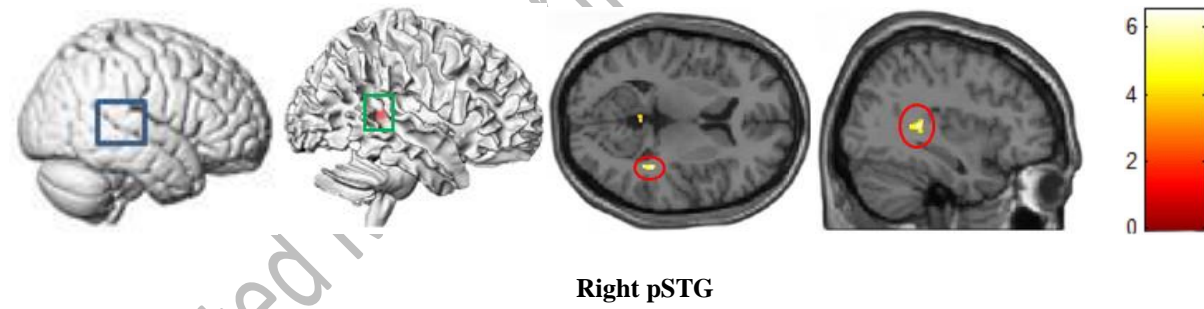
A 250 Hz > 300 Hz



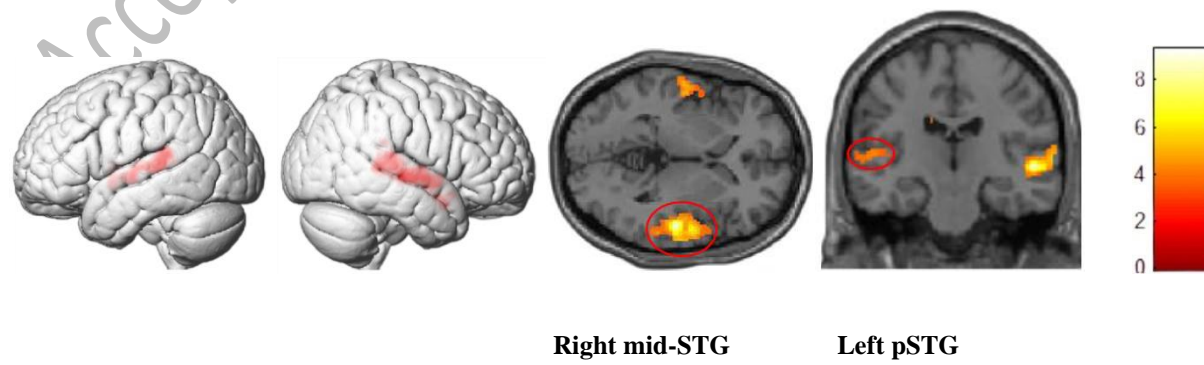
B1 90 dB > 70 dB



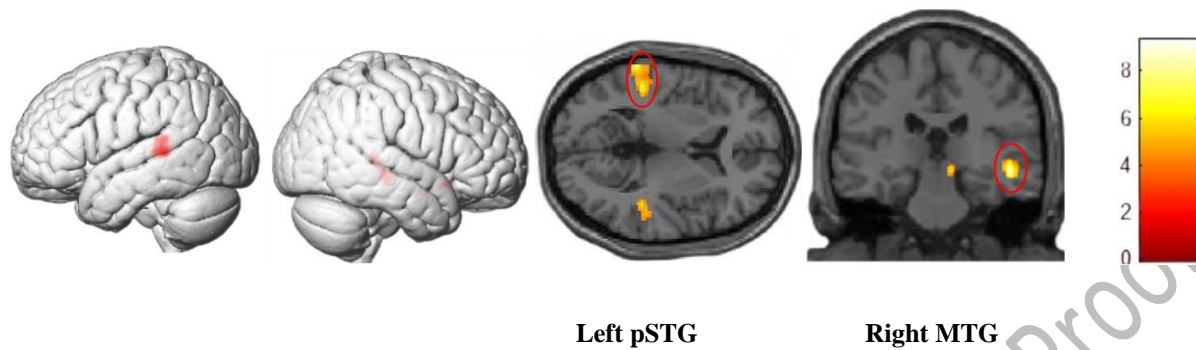
B2 90 dB > 80 dB



C1 2 sec > 3 sec



C2 4 sec > 3 sec



C3 2 sec > 4 sec

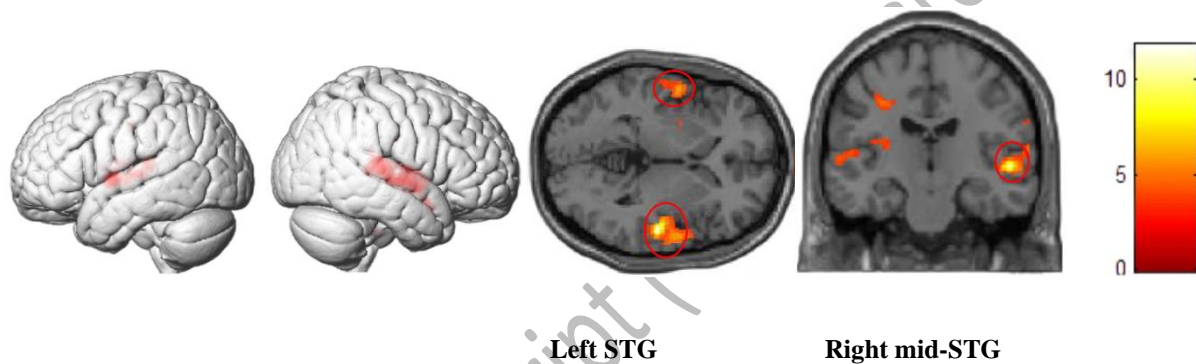


Figure 2. (A) Increased activity in the left STG and the left HG across all individuals for anger (250 Hz) compared with anger (300 Hz) prosody. (B1) Increased activity in the posterior STG and posterior MTG in the RH for anger at 90dB in comparison with anger at 70dB. (B2) Increased activity in the right posterior STG for anger at 90 dB compared with anger at 80 dB. (C1) Stronger activity in the right mid-STG and the left posterior STG for anger (2s) compared with anger (3s). (C2) Stronger activity in the right MTG and the left posterior STG for anger (4s) compared with anger (3s). (C3) Stronger activity in the mid-STG in the RH and the left STG for anger (2s) compared with anger (4s). All contrasts were thresholded at $p < 0.001$ (uncorrected) with a minimum cluster extent of $k = 0$ voxels. The data images were extracted using xjview.

Table 2. Main effects of anger with different acoustic parameters, $P < 0.001$ uncorr.

Anatomical definitions	MNI coordinates				
	X	Y	Z	K	T
<i>Anger (250Hz) > anger (300Hz):</i>					
L-STG	-60	-20	8	328	5.33
L-HG	-55	-22	9	328	5.33
<i>Anger (90dB) > anger (70dB):</i>					
R-posterior STG	42	-38	6	192	7.4
R-posterior MTG	49	-38	4	192	7.4
<i>Anger (90dB) > anger (80dB):</i>					
R-posterior STG	38	-38	8	32	6.52
<i>Anger (2s) > anger (3s):</i>					
R-mid STG	54	-20	0	1771	9.33
L-posterior STG	-64	-36	16	955	6.64
<i>Anger (4s) > anger (3s):</i>					
R-MTG	50	-26	-8	271	8.21
L-posterior STG	-66	-40	6	378	7.84
<i>Anger (2s) > anger (4s):</i>					
R-mid STG	54	-18	0	1330	11.8
L-STG	-54	-2	0	931	5.92

Peak coordinates from significant clusters. (FWE $p < 0.05$, extent threshold= 0 voxels). K = cluster size

Discussion

The present study was conducted to investigate the brain activity underlying the decoding of emotion-specific acoustic parameters in anger prosody. In fact, we wanted to identify brain regions that are sensitive to changing acoustic parameters. According to our assumption, changing any of the emotion-specific acoustic parameters can result in different hemodynamic responses within the STG and MTG of both hemispheres. Our findings showed stronger activation in the STG, HG, and MTG of both hemispheres when the acoustic parameters of anger

were changed. Our results confirm previous reports on regions showing increased responsiveness to acoustic parameters [46, 57, 59]. In previous studies, it has been suggested that the decoding of acoustic parameters occurs in the temporal lobe, especially in STG subregions, and there is no study related to how brain activity differs in the temporal lobe following changes in any of these acoustic parameters. Therefore, we tried to change any of the acoustic parameters separately, keep the other two parameters constant, and examine how the brain activity differs.

When we changed the mean f_0 from 300 Hz to 250 Hz and kept the intensity and duration constant, our analysis revealed stronger activity in HG and adjacent areas in STG in LH. The activity in the left STG in our findings is consistent with the results of previous studies [44, 59].

We also showed the role of HG in pitch processing, which is in agreement with the studies suggesting HG as ‘pitch center’ [72]. Previous studies [83] demonstrated that one of the characteristics of ‘hot’ anger was increased in mean F_0 . Studies in which this feature was not found may have been measuring ‘cold’ anger. Our finding about mean F_0 variations was probably due to the use of ‘cold’ anger. In fact, according to the previous study [82], low fundamental frequency is one of the characteristics of anger. With a further decrease in the fundamental frequency, anger is perceived more intense. Therefore, we expected to see increased activity in the regions related to fundamental frequency decoding by reducing the mean F_0 . Thus, according to the use of ‘cold’ anger in the present study, the increase in the mean F_0 did not lead to an increase in the intensity of anger, and as expected, the brain regions related to mean F_0 decoding should not show increased activity.

In the present study, no activity was observed in the anterior temporal cortex. In fact, due to the use of male voices for the stimuli, we did not expect that the anterior temporal cortex would be active because according to previous studies [75], the anterior temporal cortex is more sensitive to female voices with high F_0 than to male voices with low F_0 . Thus, the inactivity of the anterior temporal cortex was predictable.

Our findings on the speech tempo revealed stronger activity in different parts of the STG and MTG of both hemispheres when we only changed the speech tempo. These findings are pretty in line with the results of previous studies [59, 76, 77] demonstrating activation clusters in the bilateral auditory cortex, including STG, to stimuli that are deviant in duration. Because the

temporal lobe, especially STG, is mentioned in all studies on duration processing, we can address the importance of these regions in the decoding of duration. The slight difference in our results with those of other previous studies probably goes back to the difference in the method and the type of acoustic parameter investigation. We examined the speech tempo as a type of durational feature, whereas others used duration-deviant tone in noise or stimuli duration in emotional prosody.

The results of our study about the effect of mean intensity variety on brain activity showed that increasing the mean intensity of anger prosody leads to increased activity in the posterior part of STG and MTG in the right hemisphere. The activity of right posterior part of STG in our study is in agreement with previous findings [44, 79]. Inspection of responses in the right posterior MTG revealed a fairly linear relationship within the investigated intensity range. These findings converge with previous findings demonstrating a linear relationship of BOLD response and sound intensity for a range of intensity [59, 78]. The type of stimulus used in our study and previous studies led to different results. In our study, only one type of emotional prosody (anger) was used while keeping other parameters (mean F0, duration, voice gender) constant to investigate the effect of mean intensity variations on brain regions activity. Other studies used frequency-modulated tones [78] or types of emotional prosody [59]. In the latter study, they found widespread activity in different brain regions including STG and MTG of both hemispheres due to the use of different types of emotional prosody without considering the fundamental frequency and duration of stimuli, while our results were limited to posterior part of STG and MTG in the right hemisphere which probably was due to the use of only anger prosody and keeping other parameters constant.

In total, the STG and MTG subregions were significantly correlated with changes in mean intensity, mean fundamental frequency, and duration (speech tempo). This sensitivity to a variety of acoustic parameters is probably due to the role of STG subregions in the detailed analysis of voice information [79, 86].

Limitation and strength

The current study aimed at investigating automatic processing of anger acoustic parameters. Thus, the experimental design did not include any behavioral task. Therefore, our study

limitation is the lack of behavioral control indicating that subjects comprehended the auditory information in the presence of scanner noise. However none of the subjects reported difficulties in comprehending the presented stimuli and all subjects reported after scanning that all of the stimuli were spoken in an emotional tone of voice. On the other hand, in order to avoid the influence of various factors on the results of the study, including the use of various emotional prosody, different length of stimuli, presenting stimuli with the voices of different individuals, and the long duration of the task, we decided to use only one emotional prosody (anger) stimulus, change its acoustic parameters, and be spoken by only one male speech and language pathologist. Considering the different acoustic characteristics of emotional prosody and the different role of each acoustic parameter in the semantic comprehension of emotional prosody, we suggest that other emotional prosody should be investigated to improve our knowledge about the decoding of emotion-specific acoustic parameters.

Conclusion

As discussed in the introduction, according to the role of the AC and mid-STC [43, 49, 66] in the extraction of acoustic parameters, and different parts of STG and MTG of both hemispheres in our findings in decoding of the anger acoustic parameters, we can conclude that more intense anger following the lower fundamental frequency, increased intensity, and faster speech tempo leads to increased activity in the specific brain regions related to the decoding of the anger acoustic parameters, and eventually different information is sent to IFG and OFC [45, 58] for more intense comprehension of anger.

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Reference

1. Aziz-Zadeh, L., T. Sheng, and A. Gheyntanchi, *Common premotor regions for the perception and production of prosody and correlations with empathy and prosodic ability*. PLoS One, 2010. **5**(1): p. e8759. doi:10.1371/journal.pone.0008759
2. Mitchell, R.L. and E.D. Ross, *fMRI evidence for the effect of verbal complexity on lateralisation of the neural response associated with decoding prosodic emotion*. Neuropsychologia, 2008. **46**(12): p. 2880-7. doi:10.1016/j.neuropsychologia.2008.05.024
3. Rood, L., et al., *The influence of emotion-focused rumination and distraction on depressive symptoms in non-clinical youth: a meta-analytic review*. Clin Psychol Rev, 2009. **29**(7): p. 607-16. doi:10.1016/j.cpr.2009.07.001
4. Sidtis, J.J. and D. Van Lancker Sidtis, *A neurobehavioral approach to dysprosody*. Semin Speech Lang, 2003. **24**(2): p. 93-105. doi:10.1055/s-2003-38901
5. Lucarini, V., et al., *Speech Prosody as a Bridge Between Psychopathology and Linguistics: The Case of the Schizophrenia Spectrum*. Front Psychiatry, 2020. **11**: p. 531863. doi:10.3389/fpsyt.2020.531863
6. Truong, K. and D. Van Leeuwen, *Automatic discrimination between laughter and speech*. Speech Communication, 2007. **49**: p. 144-158. doi:10.1016/j.specom.2007.01.001
7. Durfee, A.Z., et al., *Lesion loci of impaired affective prosody: A systematic review of evidence from stroke*. Brain Cogn, 2021. **152**: p. 105759. doi:10.1016/j.bandc.2021.105759
8. Koch, K., et al., *Neural correlates of processing emotional prosody in unipolar depression*. Hum Brain Mapp, 2018. **39**(8): p. 3419-3427. doi:10.1002/hbm.24185
9. Struchen, M.A., et al., *Examining the contribution of social communication abilities and affective/behavioral functioning to social integration outcomes for adults with traumatic brain injury*. J Head Trauma Rehabil, 2011. **26**(1): p. 30-42. doi:10.1097/HTR.0b013e3182048f7c
10. Elizalde Acevedo, B., et al., *Brain mapping of emotional prosody in patients with drug-resistant temporal epilepsy: An indicator of plasticity*. Cortex, 2022. **153**: p. 97-109. doi:10.1016/j.cortex.2022.04.014
11. Alba-Ferrara, L., A. Ellison, and R.L.C. Mitchell, *Decoding emotional prosody: resolving differences in functional neuroanatomy from fMRI and lesion studies using TMS*. Brain Stimul, 2012. **5**(3): p. 347-353. doi:10.1016/j.brs.2011.06.004
12. Behrens, S.J., *Characterizing sentence intonation in a right hemisphere-damaged population*. Brain Lang, 1989. **37**(2): p. 181-200. doi:10.1016/0093-934x(89)90014-x
13. Blonder, L.X., D. Bowers, and K.M. Heilman, *The role of the right hemisphere in emotional communication*. Brain, 1991. **114** (Pt 3): p. 1115-27. doi:10.1093/brain/114.3.1115
14. Blonder, L.X., et al., *Prosodic characteristics of speech pre- and post-right hemisphere stroke*. Brain Lang, 1995. **51**(2): p. 318-35. doi:10.1006/brln.1995.1063
15. Brådvik, B., et al., *Disturbances of speech prosody following right hemisphere infarcts*. Acta Neurol Scand, 1991. **84**(2): p. 114-26. doi:10.1111/j.1600-0404.1991.tb04919.x
16. Dara, C., et al., *Right hemisphere dysfunction is better predicted by emotional prosody impairments as compared to neglect*. J Neurol Transl Neurosci, 2014. **2**(1): p. 1037.
17. Demenescu, L.R., Y. Kato, and K. Mathiak, *Neural Processing of Emotional Prosody across the Adult Lifespan*. Biomed Res Int, 2015. **2015**: p. 590216. doi:10.1155/2015/590216
18. Geigenberger, A. and W. Ziegler, *The processing of prosodic patterns in patients with unilateral brain lesions*. Clin Linguist Phon, 2001. **15**(1-2): p. 85-9. doi:10.3109/02699200109167636
19. Gibson, L., et al., *Detection of sarcastic speech: The role of the right hemisphere in ambiguity resolution*. Laterality, 2016. **21**(4-6): p. 549-567. doi:10.1080/1357650x.2015.1105246

20. Grandjean, D., *Brain Networks of Emotional Prosody Processing*. *Emotion Review*, 2021. **13**(1): p. 34-43. doi:10.1177/1754073919898522
21. Grandjean, D., et al., *Effects of emotional prosody on auditory extinction for voices in patients with spatial neglect*. *Neuropsychologia*, 2008. **46**(2): p. 487-96. doi:10.1016/j.neuropsychologia.2007.08.025
22. Heilman, K.M., et al., *Comprehension of affective and nonaffective prosody*. *Neurology*, 1984. **34**(7): p. 917-21. doi:10.1212/wnl.34.7.917
23. Hoekert, M., G. Vingerhoets, and A. Aleman, *Results of a pilot study on the involvement of bilateral inferior frontal gyri in emotional prosody perception: an rTMS study*. *BMC Neurosci*, 2010. **11**: p. 93. doi:10.1186/1471-2202-11-93
24. Jacob, H., et al., *Cerebral processing of prosodic emotional signals: evaluation of a network model using rTMS*. *PLoS One*, 2014. **9**(8): p. e105509. doi:10.1371/journal.pone.0105509
25. Kucharska-Pietura, K., et al., *Perception of emotions from faces and voices following unilateral brain damage*. *Neuropsychologia*, 2003. **41**(8): p. 1082-90. doi:10.1016/s0028-3932(02)00294-4
26. Pell, M.D., *Fundamental frequency encoding of linguistic and emotional prosody by right hemisphere-damaged speakers*. *Brain Lang*, 1999. **69**(2): p. 161-92. doi:10.1006/brln.1999.2065
27. Pell, M.D. and S.R. Baum, *Unilateral brain damage, prosodic comprehension deficits, and the acoustic cues to prosody*. *Brain Lang*, 1997. **57**(2): p. 195-214. doi:10.1006/brln.1997.1736
28. Ross, E.D. and M. Monnot, *Neurology of affective prosody and its functional-anatomic organization in right hemisphere*. *Brain Lang*, 2008. **104**(1): p. 51-74. doi:10.1016/j.bandl.2007.04.007
29. Ross, E.D. and M. Monnot, *Affective prosody: what do comprehension errors tell us about hemispheric lateralization of emotions, sex and aging effects, and the role of cognitive appraisal*. *Neuropsychologia*, 2011. **49**(5): p. 866-877. doi:10.1016/j.neuropsychologia.2010.12.024
30. Ryalls, J., Y. Joannette, and L. Feldman, *An acoustic comparison of normal and right-hemisphere-damaged speech prosody*. *Cortex*, 1987. **23**(4): p. 685-94. doi:10.1016/s0010-9452(87)80059-x
31. Schmidt, J.M., *Emotional prosody production in brain-damaged populations: Human judgments and acoustical analysis*. 2003: City University of New York.
32. Seydell-Greenwald, A., et al., *What you say versus how you say it: Comparing sentence comprehension and emotional prosody processing using fMRI*. *Neuroimage*, 2020. **209**: p. 116509. doi:10.1016/j.neuroimage.2019.116509
33. Shapiro, B.E. and M. Danly, *The role of the right hemisphere in the control of speech prosody in propositional and affective contexts*. *Brain Lang*, 1985. **25**(1): p. 19-36. doi:10.1016/0093-934x(85)90118-x
34. Sherratt, S., *Right brain damage and the verbal expression of emotion: A preliminary investigation*. *Aphasiology*, 2007. **21**: p. 320-339. doi:10.1080/02687030600911401
35. Tompkins, C.A. and C.R. Flowers, *Perception of emotional intonation by brain-damaged adults: the influence of task processing levels*. *J Speech Hear Res*, 1985. **28**(4): p. 527-38. doi:10.1044/jshr.2804.527
36. Witteman, J., et al., *The nature of hemispheric specialization for linguistic and emotional prosodic perception: a meta-analysis of the lesion literature*. *Neuropsychologia*, 2011. **49**(13): p. 3722-38. doi:10.1016/j.neuropsychologia.2011.09.028
37. Wright, A., et al., *Selective impairments in components of affective prosody in neurologically impaired individuals*. *Brain Cogn*, 2018. **124**: p. 29-36. doi:10.1016/j.bandc.2018.04.001
38. Wright, A.E., et al., *Acute Ischemic Lesions Associated with Impairments in Expression and Recognition of Affective Prosody*. *Perspect ASHA Spec Interest Groups*, 2016. **1**(2): p. 82-95. doi:10.1044/persp1.SIG2.82

39. House, A., D. Rowe, and P.J. Standen, *Affective prosody in the reading voice of stroke patients*. J Neurol Neurosurg Psychiatry, 1987. **50**(7): p. 910-2. doi:10.1136/jnnp.50.7.910
40. Pell, M.D., *Cerebral mechanisms for understanding emotional prosody in speech*. Brain Lang, 2006. **96**(2): p. 221-34. doi:10.1016/j.bandl.2005.04.007
41. Schlanger, B.B., P. Schlanger, and L.J. Gerstman, *The perception of emotionally toned sentences by right hemisphere-damaged and aphasic subjects*. Brain Lang, 1976. **3**(3): p. 396-403. doi:10.1016/0093-934x(76)90035-3
42. Twist, D., et al., *Event-Related Potentials in Disorders of Prosodic and Semantic Linguistic Processing 1*. Cognitive and Behavioral Neurology, 1991. **4**.
43. Brück, C., B. Kreifelts, and D. Wildgruber, *Emotional voices in context: a neurobiological model of multimodal affective information processing*. Phys Life Rev, 2011. **8**(4): p. 383-403. doi:10.1016/j.plrev.2011.10.002
44. Frühholz, S., M. Gschwind, and D. Grandjean, *Bilateral dorsal and ventral fiber pathways for the processing of affective prosody identified by probabilistic fiber tracking*. Neuroimage, 2015. **109**: p. 27-34. doi:10.1016/j.neuroimage.2015.01.016
45. Witteman, J., V.J.P. Van Heuven, and N.O. Schiller, *Hearing feelings: a quantitative meta-analysis on the neuroimaging literature of emotional prosody perception*. Neuropsychologia, 2012. **50**(12): p. 2752-2763. doi:10.1016/j.neuropsychologia.2012.07.026
46. Beaucousin, V., et al., *fMRI study of emotional speech comprehension*. Cereb Cortex, 2007. **17**(2): p. 339-52. doi:10.1093/cercor/bhj151
47. Buchanan, T.W., et al., *Recognition of emotional prosody and verbal components of spoken language: an fMRI study*. Brain Res Cogn Brain Res, 2000. **9**(3): p. 227-38. doi:10.1016/s0926-6410(99)00060-9
48. Dietrich, S., et al., *Understanding the emotional expression of verbal interjections: a functional MRI study*. Neuroreport, 2008. **19**(18): p. 1751-5. doi:10.1097/WNR.0b013e3283193e9e
49. Ethofer, T., et al., *Cerebral pathways in processing of affective prosody: a dynamic causal modeling study*. Neuroimage, 2006. **30**(2): p. 580-7. doi:10.1016/j.neuroimage.2005.09.059
50. Ethofer, T., et al., *Emotional voice areas: anatomic location, functional properties, and structural connections revealed by combined fMRI/DTI*. Cereb Cortex, 2012. **22**(1): p. 191-200. doi:10.1093/cercor/bhr113
51. Frühholz, S., L. Ceravolo, and D. Grandjean, *Specific brain networks during explicit and implicit decoding of emotional prosody*. Cereb Cortex, 2012. **22**(5): p. 1107-17. doi:10.1093/cercor/bhr184
52. Kotz, S.A., et al., *Predicting vocal emotion expressions from the human brain*. Hum Brain Mapp, 2013. **34**(8): p. 1971-81. doi:10.1002/hbm.22041
53. Kotz, S.A., et al., *On the lateralization of emotional prosody: an event-related functional MR investigation*. Brain Lang, 2003. **86**(3): p. 366-76. doi:10.1016/s0093-934x(02)00532-1
54. Leitman, D.I., et al., *"It's Not What You Say, But How You Say it": A Reciprocal Temporo-frontal Network for Affective Prosody*. Front Hum Neurosci, 2010. **4**: p. 19. doi:10.3389/fnhum.2010.00019
55. Mitchell, R.L., et al., *The neural response to emotional prosody, as revealed by functional magnetic resonance imaging*. Neuropsychologia, 2003. **41**(10): p. 1410-21. doi:10.1016/s0028-3932(03)00017-4
56. Mothes-Lasch, M., et al., *Visual attention modulates brain activation to angry voices*. J Neurosci, 2011. **31**(26): p. 9594-8. doi:10.1523/jneurosci.6665-10.2011
57. Sander, D., et al., *Emotion and attention interactions in social cognition: brain regions involved in processing anger prosody*. Neuroimage, 2005. **28**(4): p. 848-58. doi:10.1016/j.neuroimage.2005.06.023

58. Schirmer, A. and S.A. Kotz, *Beyond the right hemisphere: brain mechanisms mediating vocal emotional processing*. Trends Cogn Sci, 2006. **10**(1): p. 24-30. doi:10.1016/j.tics.2005.11.009
59. Wiethoff, et al., *Cerebral processing of emotional prosody--influence of acoustic parameters and arousal*. Neuroimage, 2008. **39**(2): p. 885-93. doi:10.1016/j.neuroimage.2007.09.028
60. Wittfoth, M., et al., *On emotional conflict: interference resolution of happy and angry prosody reveals valence-specific effects*. Cereb Cortex, 2010. **20**(2): p. 383-92. doi:10.1093/cercor/bhp106
61. Grandjean, D., et al., *The voices of wrath: brain responses to angry prosody in meaningless speech*. Nat Neurosci, 2005. **8**(2): p. 145-6. doi:10.1038/nn1392
62. Belyk, M. and S. Brown, *Perception of affective and linguistic prosody: an ALE meta-analysis of neuroimaging studies*. Soc Cogn Affect Neurosci, 2014. **9**(9): p. 1395-403. doi:10.1093/scan/nst124
63. Ethofer, T., et al., *Decoding of emotional information in voice-sensitive cortices*. Curr Biol, 2009. **19**(12): p. 1028-33. doi:10.1016/j.cub.2009.04.054
64. Morris, J.S., S.K. Scott, and R.J. Dolan, *Saying it with feeling: neural responses to emotional vocalizations*. Neuropsychologia, 1999. **37**(10): p. 1155-63. doi:10.1016/s0028-3932(99)00015-9
65. Fecteau, S., et al., *Amygdala responses to nonlinguistic emotional vocalizations*. Neuroimage, 2007. **36**(2): p. 480-7. doi:10.1016/j.neuroimage.2007.02.043
66. Bach, D.R., et al., *The effect of appraisal level on processing of emotional prosody in meaningless speech*. Neuroimage, 2008. **42**(2): p. 919-27. doi:10.1016/j.neuroimage.2008.05.034
67. Robin, D.A., D. Tranel, and H. Damasio, *Auditory perception of temporal and spectral events in patients with focal left and right cerebral lesions*. Brain Lang, 1990. **39**(4): p. 539-55. doi:10.1016/0093-934x(90)90161-9
68. Gandour, J., *Frontiers of brain mapping of speech prosody*. Brain Lang, 2000. **71**(1): p. 75-7. doi:10.1006/brln.1999.2217
69. Gandour, J., D. Wong, and G. Hutchins, *Pitch processing in the human brain is influenced by language experience*. Neuroreport, 1998. **9**(9): p. 2115-2119.
70. Nan, Y. and A.D. Friederici, *Differential roles of right temporal cortex and Broca's area in pitch processing: evidence from music and Mandarin*. Hum Brain Mapp, 2013. **34**(9): p. 2045-54. doi:10.1002/hbm.22046
71. Warren, J.D., A.R. Jennings, and T.D. Griffiths, *Analysis of the spectral envelope of sounds by the human brain*. Neuroimage, 2005. **24**(4): p. 1052-7. doi:10.1016/j.neuroimage.2004.10.031
72. Bendor, D. and X. Wang, *Cortical representations of pitch in monkeys and humans*. Curr Opin Neurobiol, 2006. **16**(4): p. 391-9. doi:10.1016/j.conb.2006.07.001
73. Griffiths, T.D., *Functional imaging of pitch analysis*. Ann N Y Acad Sci, 2003. **999**: p. 40-9. doi:10.1196/annals.1284.004
74. Hall, D.A. and C.J. Plack, *Pitch processing sites in the human auditory brain*. Cereb Cortex, 2009. **19**(3): p. 576-85. doi:10.1093/cercor/bhn108
75. Sokhi, D.S., et al., *Male and female voices activate distinct regions in the male brain*. Neuroimage, 2005. **27**(3): p. 572-8. doi:10.1016/j.neuroimage.2005.04.023
76. Mathiak, K., et al., *Mismatch responses to randomized gradient switching noise as reflected by fMRI and whole-head magnetoencephalography*. Hum Brain Mapp, 2002. **16**(3): p. 190-5. doi:10.1002/hbm.10041
77. Schall, U., et al., *Functional neuroanatomy of auditory mismatch processing: an event-related fMRI study of duration-deviant oddballs*. Neuroimage, 2003. **20**(2): p. 729-36. doi:10.1016/s1053-8119(03)00398-7
78. Langers, D.R., et al., *fMRI activation in relation to sound intensity and loudness*. Neuroimage, 2007. **35**(2): p. 709-18. doi:10.1016/j.neuroimage.2006.12.013

79. Warren, J.D., et al., *Human brain mechanisms for the early analysis of voices*. Neuroimage, 2006. **31**(3): p. 1389-97. doi:10.1016/j.neuroimage.2006.01.034
80. Clarke, P.J., et al., *The effects of left DLPFC tDCS on emotion regulation, biased attention, and emotional reactivity to negative content*. Cognitive, Affective, & Behavioral Neuroscience, 2020. **20**: p. 1323-1335.
81. Nitsche, M.A., et al., *Effects of frontal transcranial direct current stimulation on emotional state and processing in healthy humans*. Frontiers in psychiatry, 2012. **3**: p. 58.
82. Sobin, C. and M. Alpert, *Emotion in speech: the acoustic attributes of fear, anger, sadness, and joy*. J Psycholinguist Res, 1999. **28**(4): p. 347-65. doi:10.1023/a:1023237014909
83. Banse, R. and K.R. Scherer, *Acoustic profiles in vocal emotion expression*. J Pers Soc Psychol, 1996. **70**(3): p. 614-36. doi:10.1037//0022-3514.70.3.614
84. Kazemi, Y. and S. Saeednia, *The clinical examination of non-word repetition tasks in identifying Persian-speaking children with primary language impairment*. Int J Pediatr Otorhinolaryngol, 2017. **93**: p. 7-12. doi:10.1016/j.ijporl.2016.11.028
85. Friston, K.J., et al., *Statistical parametric maps in functional imaging: a general linear approach*. Human brain mapping, 1994. **2**(4): p. 189-210.
86. Warren, J.D., A. Jennings, and T.D. Griffiths, *Analysis of the spectral envelope of sounds by the human brain*. Neuroimage, 2005. **24**(4): p. 1052-1057.