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BRIEF COMMUNICATION

Interpersonal prosodic correlation in frontotemporal dementia

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Abstract

Communication accommodation describes how individuals adjust their communicative style to that of their conversational partner. We predicted that interpersonal prosodic correlation related to pitch and timing would be decreased in behavioral variant frontotemporal dementia (bvFTD). We predicted that the interpersonal correlation in a timing measure and a pitch measure would be increased in right temporal FTD (rtFTD) due to sparing of the neural substrate for speech timing and pitch modulation but loss of social semantics. We found no significant effects in bvFTD, but conversations including rtFTD demonstrated higher interpersonal correlations in speech rate than healthy controls.

Introduction

Communication accommodation theory describes how individuals adjust their communicative style to that of their partner in conversation. Communication accommodation can include linguistic (word choices), paralinguistic (pitch, tempo), and nonverbal (smiling, mutual gaze) features of communication. Accommodation establishes a paralinguistic conversational baseline, which may be broken to introduce new socio-emotional information into the conversation[1]. Lack of appropriate accommodation can have important social implications, as accommodation correlates with several measures of communication quality[2].

Neurodegenerative disorders such as frontotemporal dementias (FTD) can cause deficits in communication and social interaction. Aberrant behavior often correlates with regions of neurodegeneration, thereby charting the neural underpinnings of social interactions. Assuming prosodic accommodation is a largely unconscious process, the phenomenon requires that the neural substrate for modulating the prosodic variable is intact, and that there is no recognized reason to break from the accommodative impulse. Paralinguistic pitch variation predominantly relies on a right hemispheric dorsal processing stream that ends in the orbitofrontal cortex. Timing depends on basal ganglia and cerebellar circuitry with some possible left predominance[3]. The neuroanatomy of communication

accommodation has been mapped onto the orbitofrontal cortex[4], a region typically damaged in the behavioral variant of frontotemporal dementia (bvFTD). The right temporal variant of frontotemporal dementia relatively spares these areas, but has been associated with loss of social semantic knowledge and recognition of paralinguistic social signals [5]. Other types of FTD, such as the semantic and nonfluent variants of primary progressive aphasia (svPPA and nfvPPA), primarily affect language while relatively sparing social and emotional functioning[6].

We hypothesized that some types of FTD differ from healthy controls (i.e., conversational partners) in the extent to which prosody correlates between conversational partners. Specifically, we predicted that conversations including individuals with bvFTD would demonstrate less interpersonal correlation in pitch, intensity, and speech rate versus healthy controls due to a convergence of prosodic pathways in the orbitofrontal cortex. We predicted that correlation in speech rate and fundamental frequency modulation might be increased in the right temporal variant of frontotemporal dementia (rtFTD), because spared neural substrates for modulating speech rate and timing would permit unconscious accommodation, while loss of social semantics would diminish any recognized reason to break from that conversational baseline. For example, someone with rsvPPA would not recognize any need to slow or pause speech for emphasis, but would instead reflexively and inflexibly mimic his or her partner. We did not predict that interpersonal speech correlation would be altered in Alzheimer's disease (AD), svPPA, or nfvPPA.

Methods

Participants

About 74 patients with FTD (29 with bvFTD, 14 with rtFTD, 14 nfvPPA, and 19 with svPPA), and 15 patients with Alzheimer's disease (AD) were compared to 170 healthy controls (patients' friends or family) in a recorded conversation lasting 10–15 min with a companion. Demographics and neuropsychological attributes for this population are listed in Table 1.

The Institutional Review Boards of the University of California, San Francisco, and the University of California, Berkeley, approved the study. All participants provided informed consent prior to participation. Prior to being assessed at the University of California, Berkeley, participants with a neurodegenerative illness underwent a detailed clinical evaluation, including a physical examination and neuropsychological testing at the University of California, San Francisco. Alzheimer's disease (AD) was established by National Institute on Aging – Alzheimer's Association criteria[7], primary progressive aphasias (PPA), and bvFTD using consensus criteria[6,8]. Participants were diagnosed clinically with rtFTD based on clinical judgment and structural magnetic resonance imaging (MRI) data[9,10].

Table 1. Participant demographics and neuropsychological characteristics

	HC	AD	bvFTD	nfvPPA	rtFTD	svPPA
CDR	NA	1.1 ± 0.3	1.2 ± 0.6	0.6 ± 0.3*	0.9 ± 0.5	0.4 ± 0.3*
CDR Box	NA	6.1 ± 1.7	6.7 ± 2.9	2.6 ± 1.3*	5.5 ± 2.8	2.3 ± 1.7*
Education (years)	18.2 ± 1.3	15.9 ± 3.1	16.1 ± 2.9	17.4 ± 2.3	16.6 ± 2.3	17.4 ± 2.5
Age (years)	60.4 ± 10.8	60.8 ± 8.0	60.9 ± 8.8	67.4 ± 11.3	65.5 ± 5.7	63.7 ± 7.1
Handed (% right)	NA	86.7	93.1	92.3	92.3	94.7
Sex(%female)	61.7	40	34.5	50	42.9	21*
MMSE	NA	20.4 ± 5.2	25.1 ± 4.8	26.9 ± 3.2	25.6 ± 3.7	24.2 ± 3.9
BNT	NA	11.7 ± 4.3	11.6 ± 4.6	8.2 ± 2.7	8.0 ± 5.0	4.0 ± 2.9*
CVLT (30 sec)	NA	2.7 ± 2.9	4.8 ± 2.0	5.9 ± .6.6	4.0 ± 2.5	1.8 ± 2.3
CVLT (10 min)	NA	1.1 ± 2.5	4.1 ± 2.7	5.8 ± 2.8	2.7 ± 2.5	1.1 ± 1.9
Lexical fluency	NA	8.2 ± 6.0	5.9 ± 4.1	4.4 ± 3.3	6.1 ± 3.3	6.9 ± 3.8
Semantic fluency	NA	7.9 ± 4.7	9.6 ± 6.0	8.0 ± 6.5	9.5 ± 4.7	5.0 ± 3.0
Digits backwards	NA	2.5 ± 1.3	3.4 ± 1.5	3.6 ± 1.9	4.3 ± 1.7	4.4 ± 1.2
Benson copy	NA	9.4 ± 5.1	12.7 ± 5.0	10.4 ± 7.1	14.4 ± 2.9	15.2 ± 3.0
Benson recall (10 min)	NA	2.3 ± 2.8	6.4 ± 5.0	10.1 ± 3.6	5.7 ± 4.9	7.8 ± 4.2
Calculations	NA	2.9 ± 1.2	2.9 ± 1.7	3.5 ± 2.1	3.2 ± 2.4	4.4 ± 1.3
GDS	NA	8.0 ± 4.6	6.4 ± 6.7	4.4 ± 6.7	7.6 ± 9.8	8.2 ± 5.5

CD, clinical dementia rating scale; CDR box, CDR sum of box scores; MMSE, Mini Mental State Exam; BNT, Boston naming test abridged (15 items); CVLT, California Verbal Learning Test – II, GDS, geriatric depression scale.
*Indicates $P < 0.05$ difference. $R^2 < 0.10$.

Task description

Procedures for obtaining samples of conversations were derived from those originally developed by Levenson and Gottman (1983)[11]. Couples were instructed to discuss a mutually selected area of continuing disagreement in their relationship for 10–15 min. Audio recordings of the conversations were obtained using unidirectional Shure lavalier microphones attached to each participant, recorded onto a single audio channel.

Measures

Audio recordings were transformed into .wav files for further analysis. A spectral noise-gaiting algorithm was used to remove background noise[12]. Trained research assistants manually labeled all speech and nonspeech sounds for each speaker in Praat, an acoustic analysis program[13]. Environmental noises and nonspeech sounds were labeled for exclusion. Each labeled conversation was checked for quality before use. Using Praat, the following measures were extracted for each speaker: speech rate (syllables/second)[14], coefficient of variation of fundamental frequency (i.e., pitch in Hz), and standard deviation of intensity (i.e., loudness in decibels).

Statistical methods

A QQ plot was used to visually inspect data distributions of each measure. A cubic transformation was applied to achieve normative distributions for speech rate.

To compare interpersonal prosodic correlation slopes between diagnostic groups, regression between patient and partner speech with an interaction term for patient diagnosis was performed, adjusting for patient age and sex. Plots of residuals versus fitted values were inspected for heteroscedasticity. Regression was then repeated with robust standard errors as needed. All regressions with statistically significant results were assessed for influential outliers. If potential outliers were identified, the regression was repeated without those outliers included. A P value of 0.05 and R^2 of 0.10 was set as being statistically and behaviorally significant, respectively.

Results

Means and standard deviations for prosodic measurements in each group are listed in Table 2. An ANOVA revealed no differences between any diagnostic group in how either speakers or partners varied fundamental frequency or intensity (all $P > 0.15$). Differences were found in speech rate, but these did not meet the effect size criterion.

When comparing the extent to which patient's speech traits correlated with that of their partners across each group (i.e., the interaction term in the regression), conversations between patients with rtFTD and their partners had a markedly higher degree of positive speech rate correlation ($\widehat{\beta} = 0.8$, $P = 0.004$, 95%CI [0.26; 1.39], $R^2 = 0.35$, Fig. 1). Initial analysis suggested possible diminished interpersonal correlation of fundamental frequency variation in conversations including bvFTD ($\widehat{\beta} = -0.45$, $P = 0.014$, 95%CI [−0.8, −0.1], $R^2 = 0.10$). Further regression diagnostics suggested substantial heteroscedasticity in the residuals, as well as the possibility of results being driven by an extreme case. No differences in the interpersonal prosodic slopes were found between groups when using a more robust regression or after removing the potential outlier. We also did not find differing interpersonal prosodic correlations in conversations including nfvPPA or svPPA.

Discussion

We predicted that compared with healthy controls, the extent to which prosodic measures correlated between speakers would be lower for fundamental frequency, intensity, and rate in bvFTD, and increased for rate and

Table 2. Acoustic speech measures

	HC	AD	bvFTD	nfvPPA	rtFTD	svPPA
CoVar of F_0	20.0 ± 6.7	19.8 ± 4.5	21.3 ± 11.0	20.4 ± 7.7	18.6 ± 6.9	18.6 ± 6.3
Partner CoVar of F_0	20.6 ± 10.1	19.0 ± 7.1	22.1 ± 7.7	18.4 ± 6.3	16.7 ± 4.1	18.6 ± 6.3
Speech rate (syl/sec)	3.3 ± 0.7	3.2 ± 0.5	3.2 ± 0.9*	2.75 ± 0.8*	3.7 ± 0.8	3.3 ± 0.6
Partner speech rate	3.3 ± 0.7	3.1 ± 0.5	3.4 ± 0.9	3.4 ± 0.7	3.8 ± 0.4	3.7 ± 0.5
Speech rate (cubed)	48.3 ± 21.6	34.3 ± 14.3	38.2 ± 20.8*	25.8 ± 19.3*	55.2 ± 31.0	38.3 ± 23.5
Partner speech rate (cubed)	39.4 ± 21.7	42.8 ± 16.4	47.3 ± 27.2	42.3 ± 22.3	58.9 ± 18.9*	54.0 ± 20.0*
Intensity SD (dB)	14.1 ± 2.6	15.1 ± 2.3	14.2 ± 2.9	13.8 ± 2.5	14.1 ± 2.4	14.9 ± 2.2
Partner intensity SD	14.4 ± 2.6	14.7 ± 1.4	14.0 ± 3.3	13.3 ± 3.4	13.7 ± 2.0	14.1 ± 2.1

CoVar of F_0, coefficient of variation of fundamental frequency in Hertz. Speech rate is in syllables per second. Intensity as measured in decibels.
*Values in which P values < 0.05.

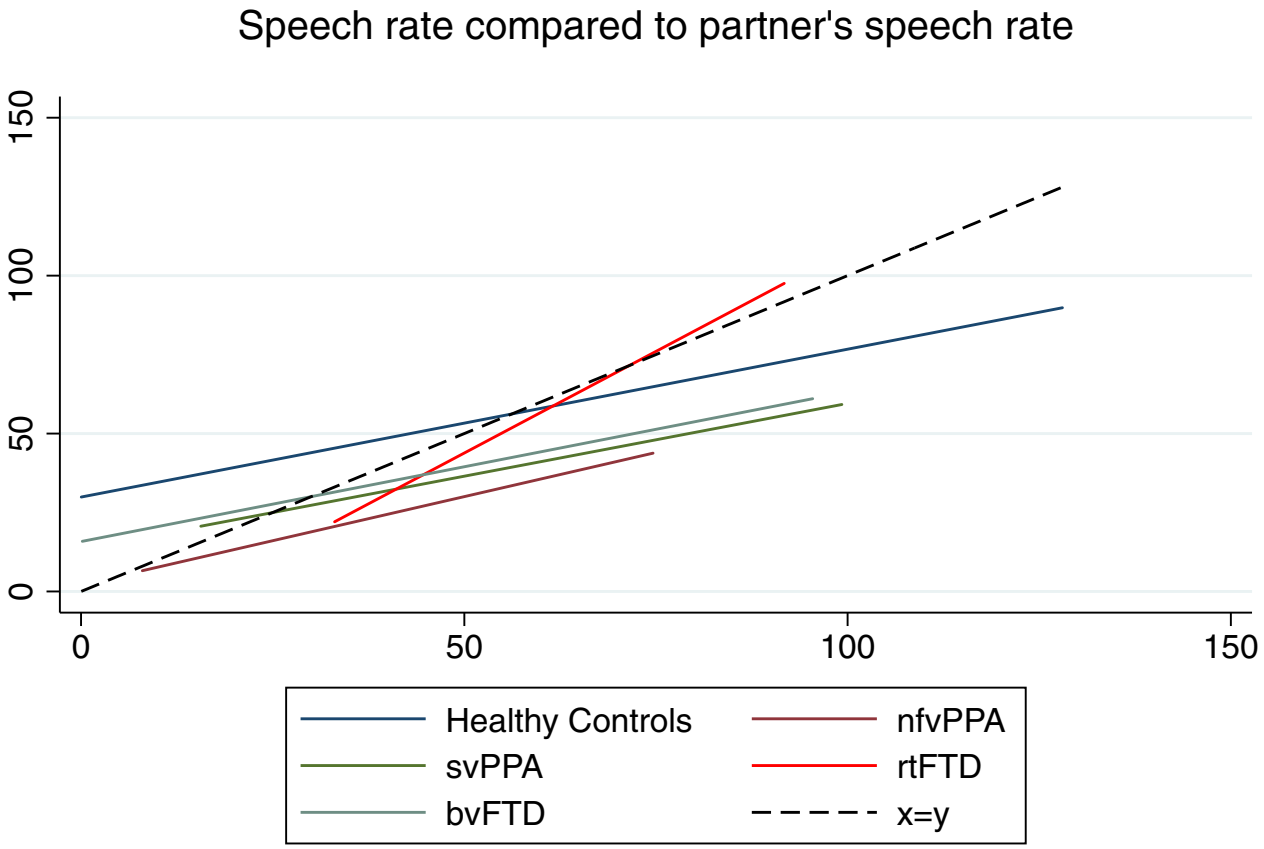

Figure 1. Correlation between participant and partner speech rate as measured in syllables per second of speech cubed. Although most forms of FTD as well as healthy controls show a consistent slope, conversations including rtFTD are associated with a significantly increased slope of correlation between speakers.

fundamental frequency in rtFTD. Contrary to what might be predicted based on reports of orbitofrontal involvement in accommodation, we found no robust evidence to support diminished prosodic correlation in bvFTD. As hypothesized, the slope of interpersonal correlation of speech rate was greater in conversations with patients with rtFTD than healthy controls.

Right frontotemporal dementia is thought to be associated with a loss of semantic knowledge for socially relevant concepts, such as reduced knowledge of social appropriateness, deficits in recognition of facial identity, and expression and reduced empathy[5]. Nevertheless, neural substrates for prosodic expression, particularly for timing, are relatively spared in rtFTD[3]. Therefore, our findings of increased interpersonal prosodic correlation for speech rate in conversations involving rtFTD likely relates to reduced recognition of the need to break from a reflexive imitation of the partner's prosodic style, with spared ability to imitate that partner's timing.

As expected, we did not find altered interpersonal prosodic correlation in svPPA or nfvPPA. This result may seem surprising in nfvPPA, which is partially defined by nonfluency. The sustained ability of nonfluent patients with left frontal damage to mimic fluent speech stimuli, however[15], is sufficiently well established to be the basis of therapeutic trials in nfvPPA[16]. In addition, contrary to some prior research on bvFTD[17], we found no diagnostic group differences in standard deviation of fundamental frequency. This negative finding aligns with other research, however[18], and may represent differences in statistical technique, the speech elicitation task, and/or in the sample selection (e.g., bvFTD is a heterogeneous disorder, and may be prone to unrecognized differences in prosodic accommodation between subtypes)[19].

Our analytic approach was limited to the level of the entire conversation – we did not specify whether patients adjust their speech more to caregivers or vice versa. A different statistical approach is necessary to distinguish the extent to which each individual matches their speech to that of their partner over the course of conversations. This more detailed approach would provide individual (rather than conversational) correlation values, thereby

permitting correlations with other neuropsychological tests and neuroimaging studies. Further limitations of this study include the use of patients' friends and family members as healthy controls, with limited information available about that population to ensure generalizability. The successful prediction of an aspect of interpersonal prosodic correlation nevertheless suggests that speech accommodation may be a quantifiable behavioral marker of neurodegenerative disease.

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Conflict of Interest

Peter S. Pressman reports no disclosures.

Kuan-Hua Chen reports no disclosures.

Elliott Ross reports no disclosures.

Bruce L. Miller has served as director on the medical advisory board of the Larry H. Hillblom Foundation, medical director of the John Douglas French Foundation, and scientific director of the Tau Consortium.

Elliott Ross reports no disclosures.

Kevin B. Cohen reports no disclosures.

Lawrence Hunter reports serves as a member of the Scientific Advisory Board of Medibio, Inc. and is a consultant to Somalogic, Inc.

Maria Luisa Gorno-Tempini reports no disclosures.

Robert W. Levenson reports no disclosures.

Author Contributions

Peter S. Pressman – Study concept and design, analysis, and interpretation.

Kevin B. Cohen – Critical revision of the manuscript for important intellectual content.

Elliott Ross – Analysis and interpretation, critical revision of the manuscript for important intellectual content.

Bruce L. Miller – Study supervision, critical revision of the manuscript for important intellectual content.

Kuan-Hua Chen – Study concept and analysis, critical revision of the manuscript for important intellectual content.

Lawrence E. Hunter – Critical revision of the manuscript for important intellectual content.

Maria Luisa Gorno-Tempini – Study supervision, critical revision of the manuscript for important intellectual content.

Robert W. Levenson – Study supervision, critical revision of the manuscript for important intellectual content.

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