Emotional reactivity and emotion recognition in frontotemporal lobar degeneration

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ABSTRACT Background: Frontotemporal lobar degeneration (FTLD) is associated with a profound decline in social and emotional behavior; however, current understanding regarding the specific aspects of emotional functioning that are preserved and disrupted is limited. Objective: To assess preservation of function and deficits in two aspects of emotional processing (emotional reactivity and emotion recognition) in FTLD. Methods: Twenty-eight FTLD patients were compared with 16 controls in emotional reactivity (self-reported emotional experience, emotional facial behavior, and autonomic nervous system response to film stimuli) and emotion recognition (ability to identify a target emotion of fear, happy, or sad experienced by film characters). Additionally, the neural correlates of emotional reactivity and emotion recognition were investigated. Results: FTLD patients were comparable to controls in 1) emotional reactivity to the fear, happy, and sad film clips and 2) emotion recognition for the happy film clip. However, FTLD patients were significantly impaired compared with controls in emotion recognition for the fear and sad film clips. Volumetric analyses revealed that deficits in emotion recognition were associated with decreased lobar volumes in the frontal and temporal lobes. Conclusions: The socioemotional decline typically seen in frontotemporal lobar degeneration patients may result more from an inability to process certain emotions in other people than from deficits in emotional reactivity. NEUROLOGY 2007;69:148–155

Deficits in emotional functioning are important early symptoms of frontotemporal lobar degeneration (FTLD), especially the semantic dementia (SD) and frontotemporal dementia (FTD) subtypes.1 Most previous research on these symptoms has relied on clinical interviews,2 caregiver reports,3 and having patients identify the emotion portrayed in photographs of faces.4 Studies using clinical and informant interviews have found affective flattening and emotional distance to be hallmark features of FTLD.1,5-8 Studies of patients’ ability to identify emotion in others have consistently found deficits,4,10-12 which are confirmed by caregiver reports.3,13 Research on the neural substrates of emotion recognition using neurologic patients4,14 and using fMRI with normal participants15,16 underscores the important role played by frontal and temporal structures.4,17

Although it is generally thought that the emotional declines in FTLD are quite pervasive, laboratory methods that enable evaluation of specific aspects of emotional functioning18 have rarely been used with these patients. These methods can help to evaluate emotional reactivity (subjective, behavioral, and autonomic responses to emotional stimuli) and emotion recognition (ability to identify emotions in others) separately. The present study extends the existing literature by 1) assessing emotional reactivity using standardized stimuli and measuring subjective, behavioral, and autonomic aspects of emotional responding; 2) assessing emotion recognition using more dynamic, ecologically valid19 stimuli (i.e., emotional films); and 3) using volumetric analyses of structural MRIs to explore brain regions related to these aspects of emotional functioning.
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The Berkeley Psychophysiology Laboratory at the University of California, Berkeley, Committee for the Protection of Human Research. Informed consent was obtained from the participants (as well as each patient’s spouse or other caregiver). Participants were scheduled for a day-long session at the Berkeley Psychophysiology Laboratory at the University of California, Berkeley.

Twenty-eight FTLD patients (19 FTD and 9 SD subtypes) and 16 control participants participated in the study. We did not include FTLD patients diagnosed with progressive nonfluent aphasia because the Neary criteria to diagnose the patients. All patients were studied early in their illness; most had been diagnosed within the 2 years before testing. Controls were recruited through advertisements and word of mouth, were not taking medications that would affect their autonomic nervous system responses, and did not suffer from neurologic or psychiatric conditions. The study was approved by the University of California, Berkeley, Committee for the Protection of Human Subjects and the University of California, San Francisco, Committee on Human Research. Informed consent was obtained from the participants (as well as each patient’s spouse or other caregiver). Participants were scheduled for a day-long session at the Berkeley Psychophysiology Laboratory at the University of California, Berkeley.

Participants and controls were recruited through the Memory and Aging Clinic at the University of California, San Francisco. A clinical team used structural MRIs along with the Neary criteria to diagnose the patients. All patients were studied early in their illness; most had been diagnosed within the 2 years before testing. Controls were recruited through advertisements and word of mouth, were not taking medications that would affect their autonomic nervous system responses, and did not suffer from neurologic or psychiatric conditions. The study was approved by the University of California, Berkeley, Committee for the Protection of Human Subjects and the University of California, San Francisco, Committee on Human Research. Informed consent was obtained from the participants (as well as each patient’s spouse or other caregiver). Participants were scheduled for a day-long session at the Berkeley Psychophysiology Laboratory at the University of California, Berkeley.

METHODS Participants. Twenty-eight FTLD patients (19 FTD and 9 SD subtypes) and 16 control participants participated in the study. We did not include FTLD patients diagnosed with progressive nonfluent aphasia because the Neary criteria to diagnose the patients. All patients were studied early in their illness; most had been diagnosed within the 2 years before testing. Controls were recruited through advertisements and word of mouth, were not taking medications that would affect their autonomic nervous system responses, and did not suffer from neurologic or psychiatric conditions. The study was approved by the University of California, Berkeley, Committee for the Protection of Human Subjects and the University of California, San Francisco, Committee on Human Research. Informed consent was obtained from the participants (as well as each patient’s spouse or other caregiver). Participants were scheduled for a day-long session at the Berkeley Psychophysiology Laboratory at the University of California, Berkeley.

Table 1 Neuropsychological test data by group

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<th>FTLD patients, mean (standard deviation)</th>
<th>Controls, mean (standard deviation)</th>
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<tbody>
<tr>
<td>MMSE, total score out of 30 possible points</td>
<td>24.8 (0.9)*</td>
<td>29.6 (1.1)*</td>
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<tr>
<td>Trails A, time in seconds</td>
<td>59.0 (6.7)</td>
<td>38.1 (10.1)</td>
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<tr>
<td>Trails B, time in seconds</td>
<td>152.2 (17.8)</td>
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<td>Trails B, number of correct switches</td>
<td>9.7 (0.7)</td>
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<td>Digits Backward, raw score</td>
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<td>Boston Naming Test, raw score</td>
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<td>14.7 (1.1)*</td>
</tr>
<tr>
<td>CVLT Trials 1 to 5, number of words remembered</td>
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<td>28.6 (2.0)*</td>
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<tr>
<td>CVLT Recognition, number of words recognized</td>
<td>7.2 (0.4)</td>
<td>8.5 (0.7)</td>
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* Means that do not share a footnote symbol are different from one another (p < 0.05).

MMSE = Mini-Mental State Examination; Trails = Trail Making Test; CVLT = California Verbal Learning Test.

Experimental materials and apparatus. Stimulus films. Participants watched three emotion-eliciting film clips in which the primary emotion being experienced by the main character was fear, happiness, or sadness and which are known to produce the same emotion in most viewers. The fear film was 3 minutes and 12 seconds long and showed a plane crash (from the film Cast Away). The happy film was 3 minutes and 55 seconds long and displayed an Olympic skater winning a gold medal (Sarah Hughes in the 2002 Olympics). The sad film clip (from the film The Champ) was 3 minutes and 42 seconds long and showed a boy crying as he watched his father die. These films were chosen to be thematically simple and to convey prototypical themes for these emotions.

Emotional experience and emotion recognition. After each film, participants completed an inventory that assessed their subjective emotional experience while watching the film. Participants chose between three options (“no,” “a little,” or “a lot”) to rate how strongly they felt each of eight specific emotions (afraid, angry, disgusted, embarrassed, happy, sad, sexually aroused, surprised). Pilot testing with FTLD patients had revealed that they could make these kinds of judgments. To
assess emotion recognition, participants were shown the same list of eight emotions and asked to choose the emotion that the main character was feeling most strongly and second most strongly.

Film comprehension. After each film, participants were asked two multiple-choice questions, one about the general plot and one about a specific incident in the film. These data were used to ensure that any differences between patients and controls in reports of emotional experience or emotion recognition were not due to differences in film comprehension.

Emotion word knowledge. To ensure that any differences between patients and controls in emotional experience or emotion recognition were not due to differences in their knowledge of emotion terms, participants completed an emotion word knowledge questionnaire. Participants were asked “How would you feel if . . . ?”; followed by a situation targeted for each of eight emotions: 1) something unexpected happens (surprise), 2) your good friend dies (sadness), 3) you want to make love to your lover (sexual desire), 4) you smell dog poo (disgust), 5) someone steals your wallet (anger), 6) a man points a gun at your head (fear), 7) you find that your pants zipper is down at a party (embarrassment), and 8) you see some old friends (happiness). For each question, participants had to choose an answer from the list of eight emotion names.

Emotional behavior. A remotely controlled high-resolution video camera, partially concealed behind darkened glass embedded in a bookshelf, was used to obtain a frontal view of each participant’s face and upper torso unobtrusively. The participants were videotaped continuously while they were in the experiment room. A team of assistants coded participants’ videotaped facial behavior during the most intense 30 seconds of each film (as determined by a group of independent raters). Behavioral codes were based on a modified version of the Emotional Expressive Behavior coding system, in which coders determined whether participants displayed specific emotional expressions (e.g., fear, happiness, sadness). Coders were trained by coding videotapes of patients and controls from another study until they reached 85% intercoder agreement. Coding was done without sound, without information as to which film was being watched, and without knowing whether participants were patients or controls.

Physiologic responses. Physiologic responses were monitored continuously using an online data acquisition software package developed by one of the authors (R.W.L.). This software computes second-by-second averages for each measure. Continuous recordings were made using a polygraph and microcomputer system. Measures were selected to provide a broad index of the activity of the physiologic systems important to emotional reactivity, including cardiovascular, electrodermal, respiratory, and striate muscle: 1) Heart rate: Small electrodes attached to the palmar surface of the distal phalanges of the nondominant hand. 2) Skin conductance level: A constant-voltage device was used to pass a small voltage between successive R waves. 3) Finger pulse amplitude: A photoplethysmograph recorded the amplitude of blood volume in the finger using a photocell taped to the distal phalange of the second finger of the nondominant hand. 5) Finger pulse transmission time: The time interval in milliseconds was measured between the R wave of the EKG and the upstroke of the peripheral pulse at the finger site. 6) Ear pulse transmission time: A photoplethysmograph attached to the right earlobe recorded the volume of blood in the ear. The time interval in milliseconds was measured between the R wave of the EKG and the upstroke of peripheral pulse at the ear site. 7) Respiration period: A pneumotachygraph was placed around the thoracic region, and the intercycle interval was measured in milliseconds between successive inspirations. 8) Respiration depth: The point of the maximum inspiration minus the point of maximum expiration was determined from the respiratory signal. 9) General somatic activity: An electromechanical transducer attached to the platform under the participant’s chair generated an electrical signal proportional to the amount of movement in any direction. 10) Systolic and diastolic blood pressure: A blood pressure cuff placed on the third phalange of the nondominant hand continuously recorded the systolic and diastolic blood pressure on each heart beat using an Ohmeda Finapres 2300.

Structural MRI. Structural MRIs were obtained to determine the amount of gray matter in participants’ brain regions of interest. The scans were obtained at the San Francisco Veterans Administration Department of Radiology. A 1.5-T Magnetom VISION system (Siemens Inc., Iselin, NJ) equipped with a standard head coil was used to image the study participants. Images of brain tissue were obtained at different orientations (e.g., axial orientation for the T2 image and coronal orientation for the T1 image) to account more accurately for the correct composition of brain matter in every voxel of space. Three structural MRI sequences were run to obtain these images: 1) a two-dimensional fast low-angle shot (FLASH) MRI of 15 slices at 3 mm thick in three orthogonal directions to obtain scout views of the brain to position it for subsequent slices, 2) protein density and T2-weighted MRIs from a double spin echo sequence with 51 contiguous axial slices at 3 mm thick extending across the entire brain at a 10° angle from the AC-PC line, and 3) T1-weighted images of the entire brain at a 15° angle in the coronal orientation perpendicular to the double spin echo sequence with volumetric magnetization prepared rapid gradient echo MRI at 1.5 mm slab thickness.

Data reduction. Self-reported emotional experience. Participants’ responses indicating how much of the target emotion they felt (i.e., “afraid” for the fear film, “happy,” for the happy film, and “sad” for the sad film) were converted to scores on a 1 to 3 scale (1 = none, 2 = a little, and 3 = a lot).

Emotional facial behavior. The present analyses focus on displays of the target emotion. Target happiness and target sadness facial behavior were quantified by summing intensity ratings on a 0 to 3 scale (0 = none, 1 = mild, 2 = moderate, and 3 = strong) across the most intense 30 seconds of the film clip. Target fear facial behavior to the fear film was excluded because the rate of fear expressions was too low to allow for meaningful group comparisons.

Physiology. Autonomic arousal scores were computed by subtracting averaged prefilm autonomic levels from averaged levels during the most intense sequential 30 seconds of the films (as judged by four independent raters) for each participant. This time window overlapped with the facial coding data, but was longer in duration reflecting the greater latency and typically longer duration for autonomic as opposed to facial responses. Because this study involved a relatively small sample and because these kinds of physiologic systems are often
“noisy” (i.e., they reflect many ongoing bodily and psychological processes in addition to emotion), individual measures were aggregated to increase reliability. An aggregate score was calculated by averaging the standardized reactivity (i.e., activation level during the film clip minus activation level during the baseline) scores for all of the physiologic measures (scores for interbeat interval, finger pulse transit time, finger pulse amplitude, ear pulse transit time, and respiratory period were all multiplied by −1 before averaging so that larger values of all measures indicated greater physiologic arousal). This procedure yielded a single physiologic arousal score for each film.

**Emotion recognition.** For the fear, happy, and sad film clips, correct responses for the initial emotion recognition question (“What did the main character feel the most strongly in the film clip?”) were coded as 2, incorrect responses for the first question and correct responses for the second question (“What did the main character feel the second most strongly?”) were coded as 1, and incorrect responses for both questions were coded as 0.

**Film comprehension.** The responses to the general plot and specific detail multiple choice comprehension questions were coded as 2 for answering both questions correctly, 1 for one correct response, and 0 for zero correct responses for the 3 films.

**Emotion word knowledge.** The items were coded as 1 for correct responses and 0 for incorrect responses and summed for the eight emotions (thus, scores ranged from 0 to 8).

**Lobar volumes obtained from structural MRI scans.** Region of interest volumes were obtained for 35 participants (22 patients and 13 controls). The remaining 9 participants (6 patients [3 FTD, 3 SD] and 3 controls) did not have usable scans available at the time the data were analyzed. Volumes were acquired for four bilateral brain regions: frontal lobes, temporal lobes, parietal lobes, and occipital lobes. To obtain these volumes, magnetic resonance images were processed on Linux workstations using the BRAINS2 software package. Lobar volumes for each study participant were computed by mapping a reference brain onto each individual's brain and then automatically deriving regional classifications to designate lobar regions. To fit each brain onto the Talairach grid, the T1-weighted images were spatially normalized and resampled to 1.0-mm³ voxels so that the anterior–posterior axis of the brain was realigned parallel to the anterior commissure–posterior commissure line and the interhemispheric fissure was aligned on the other two axes. Next, the outermost boundaries of the cortex, as well as the anterior commissure and posterior commissure, were identified to warp the Talairach grid onto each brain. Then, two other structural images (the T2- and proton density [PD]–weighted images) were realigned to the spatially normalized T1-weighted image using an automated image registration program. The BRAINS2 program makes use of all three images (the T1, T2, and PD) in computing lobar volumes, and therefore, all three are needed to be mapped on the Talairach grid.

A brain mask was generated for each of brain images using a previously trained artificial neural network, which is one of the features of the BRAINS2 software package. Then, lobar volumes for the frontal, temporal, parietal, and occipital lobes were calculated using an automated Talairach-based method of regional classification for each brain. Finally, these lobar volumes were normalized to correct for differences in overall head size. To perform this correction, the absolute lobar volume was multiplied by the average total intracranial volume (TIV) of our patient sample and then divided by the individual's TIV. This analysis yielded one number for each of the eight brain regions of interest (i.e., right and left frontal, temporal, parietal, and occipital regions) that represented the amount of gray and white matter volume in the specified region.

**Data analyses.** Analyses were conducted to determine whether 1) FTLD patients differ from controls in emotional reactivity (subjective experience, facial behavior, physiologic response) and emotion recognition; 2) FTLD patients’ emotion recognition ability is correlated with their emotional reactivity; and 3) emotional reactivity and emotion recognition is correlated with frontal and temporal neuronal loss.

**RESULTS**

**Film comprehension.** To rule out possible confounds, before conducting our primary analyses of interest, we compared patients’ and controls’ performance on the film comprehension inventory and on the emotion word knowledge test. Patients and controls did not differ significantly in their comprehension of the film clips: fear film, \( \chi^2(2, n = 44) = 0.61, \) not significant (NS); happy film, \( \chi^2(2, n = 44) = 3.12, \) NS; sad film, \( \chi^2(2, n = 44) = 1.49, \) NS. Patients and controls also did not differ significantly in their knowledge of emotion words: F(1,43) = 2.19, NS.

**Emotional reactivity.** There was no evidence of differences between FTLD patients and controls in emotional reactivity as measured by self-reported subjective experience of the target emotion, facial expression of the target emotion, or autonomic responses. Patients and controls did not differ significantly in their reports of the target emotion for the fear film, \( \chi^2(2, n = 44) = 0.33, \) NS; happy film, \( \chi^2(2, n = 44) = 0.47, \) NS; or sad film, \( \chi^2(2, n = 44) = 4.50, \) NS. Patients and controls did not differ significantly in their displays of target emotional behavior for the happy film, \( t(1,43) = 0.10, \) NS; or the sad film, \( t(1,43) = -0.60, \) NS (as noted above, analyses of facial behavior were not possible for the fear film due to low rates of fear expressions). Furthermore, patients and controls did not differ significantly in their aggregated physiologic response to the fear film \( t(1,43) = 0.02, \) NS; happy film, \( t(1,43) = 0.21, \) NS; or sad film, \( t(1,43) = -0.67, \) NS. Means and standard deviations for emotional reactivity are presented in table 2.

An additional set of analyses was conducted to determine whether FTLD patients differed from controls in their report of nontargeted emotions (e.g., reports of happiness and sadness to the fear film). These revealed no differences between the groups, with the exception of happiness reported to the sad film, where FTLD patients reported more happiness than controls: \( \chi^2(2, n = 49) = 5.82, \) \( p < 0.05. \) Means and standard deviations for reports of nontargeted emotions are presented in table 2. Thus, we found no evidence of reduced emotional
reactivity in FTLD patients in response to these simple emotional films.

**Emotion recognition.** FTLD patients were significantly less likely than control participants to recognize that the main character was feeling fear in the fear film, $\chi^2(1, n = 44) = 9.75, p < 0.04$, and sadness in the sad film, $\chi^2(1, n = 44) = 4.03, p < 0.05$. All incorrect responses by patients were of the correct valence (i.e., a different negative emotion than the correct one). FTLD patients and controls did not differ in recognizing that the main character was feeling happiness in the happy film (100% of participants in both groups chose the correct response). Percentages of patients and controls reporting accurate responses are presented in table 2.

Because patterns of neuronal degeneration associated with FTD and SD subtypes (i.e., more frontal involvement in FTD and more temporal involvement in SD\(^2\)) might differentially affect emotional processing, we compared emotional recognition in the two subtypes. Our ability to consider these groups separately was limited by sample sizes, but exploratory analyses provided hints that the FTD patients might be more impaired in emotion recognition than the SD patients. Analysis of the subgroups revealed that FTD patients were less likely to recognize the target emotion in the fear film compared with controls, $\chi^2(2, n = 19) = 8.02, p < 0.01$; SD patients, in contrast, did not differ from controls, $\chi^2(2, n = 9) = 1.99, \text{NS.}$

**Emotion recognition: Correlations with emotional reactivity.** Individuals who have small emotional responses to the films may not have the kinds of information (e.g., visceral, somatic) available that are useful in making emotional judgments. Thus, we examined whether deficits in emotion recognition were associated with deficits in emotional reactivity. However, for the fear, happy, and sad films, ability to identify the emotion being experienced by the main character was not significantly correlated with any of the indicators of emotional reactivity (self-report, facial behavior, physiologic response), with the exception of a significant positive correlation between fear recognition and autonomic response. These results are presented in table 3.

**Emotional reactivity and emotion recognition: Correlations with lobar volumes.** For emotional reactivity, greater happy facial behavior during the happy film was associated with greater lobar volumes in the right temporal ($r = 0.43; \ p < 0.01$) and right frontal lobes ($r = 0.36; \ p < 0.03$). Additionally, greater sad facial behavior during the sad film was associated with greater neuronal volume in the right frontal lobe ($r = 0.46; \ p < 0.01$). There were no significant correlations between self-report or physiologic response and lobar volumes for any of the films. These results are presented in table 4.

For emotion recognition, greater accuracy for the fear film was associated with greater lobar volumes in the left frontal ($r = 0.35; \ p < 0.04$), right frontal ($r = 0.35; \ p < 0.04$), and right temporal lobes ($r = 0.39; \ p < 0.02$). For the sad film, greater accuracy was associated with greater lobar volumes in the left frontal ($r = 0.43; \ p < 0.01$), left temporal ($r = 0.36; \ p < 0.04$), and right temporal lobes ($r = 0.44; \ p = 0.03$). These results and nonsignificant trends ($p < .10$) are presented in table 4.
A hallmark feature of FTLD is a general deficit in emotional functioning. In this study, we applied laboratory methods derived from basic emotion research to derive a more differentiated view of areas of preserved and diminished emotional functioning, focusing on two aspects of emotional functioning: emotional reactivity and emotion recognition.

Given clinical descriptions of blunted affect in FTLD patients, we expected that they would show deficits in emotional reactivity. However, we found no evidence for this in response to three different emotional films (fear, happy, sad) sampling from three primary emotion response systems (self-report, facial behavior, physiologic response). Thus, these laboratory-based methods revealed something unexpected about FTLD. FTLD patients may have preserved capacity to feel, show, and recruit physiologic activation for basic positive (happy) and negative (sad, fear) emotions in response to stimuli that are thematically simple, are nonambiguous, and do not require extensive higher-level cognitive processing. Of course, these are the kinds of emotional stimuli that likely activate evolved, hardwired reactions to species-typical challenges and opportunities, which are subserved by subcortical brain structures that are relatively preserved in the early stages of FTLD. We do not interpret these findings as indicating that all aspects of emotional reactivity are preserved in FTLD. For example, in studies of emotional reactivity in embarrassing social situations that require more complex self-monitoring and appraisal, we have found clear-cut deficits in emotional reactivity in FTLD. These higher-order processes likely involve frontal circuits that are highly vulnerable in FTLD. Furthermore, our FTLD patients were relatively early in the course of their disease. As the disease progresses and affects increasingly widespread brain areas, we expect that even the simple kinds of emotional reactivity that were found to be preserved in the present study will be diminished. Our neuroanatomical analyses support this view, indicating that lower right frontal volumes were associated with diminished facial displays of the target emotion in response to the happy and sad films.

Less surprising was our finding of clear-cut deficits in emotion recognition in FTLD. These deficits could not be explained in terms of problems with film comprehension or diminished emotional reactivity and are particularly striking given the highly transparent emotional themes in the films (e.g., in the sad film, the main character was crying). This finding is consistent with previous research showing that FTLD patients have difficulties recognizing emotions and with clinical and caregiver reports that patients often show a lack of empathy. Deficits in the ability to detect emotion may be particularly problematic at home and work, because patients may not recognize pain, distress, and suffering.

**DISCUSSION**

| Table 4 Correlations between lobar volumes and emotional reactivity and emotion recognition
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<td></td>
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Values are Pearson correlation coefficients. Correlations with emotion recognition reflect participants’ (patients and controls) response accuracy combining the first and second emotion recognition questions (i.e., the emotion the character felt most strongly and second most strongly). Correlations between lobar volumes and fear expressions to the fear film were not calculated because of the low occurrence of fear expressions. Correlations between lobar volumes and emotion recognition for the happy film were not calculated because all participants were accurate in detecting happiness.

*p < 0.01, †p < 0.05, ‡p < 0.10.
ferring in others. Although we and others have found relative preservation of the ability to detect positive, but not negative, emotions in FTLD,\textsuperscript{10,12} it seems premature to leap to the conclusion that different circuitry subserves the detection of positive and negative emotions. Methodologic limitations in the present study and other studies (e.g., use of multiple negative emotions, but only one positive emotion) and the fact that happiness and its facial signature, the smile, are generally easier to detect than other emotions\textsuperscript{13} raise caveats as to the specificity of this deficit to negative emotions.

Researchers have proposed the idea that accurate emotion recognition may be aided by participants feeling the target emotion.\textsuperscript{34,35} The current study found only limited support for this notion. Emotion recognition did not correlate with emotional reactivity when considering self-report, facial behavior, and autonomic responding for the fear, sad, and happy films. The one exception was a correlation between emotion recognition and autonomic reactivity for the fear film. This finding for fear is reminiscent of previous findings of overlapping neural networks for perceiving and feeling pain or disgust.\textsuperscript{36}

Our exploratory analysis of FTLD subtypes suggested that emotion recognition deficits may be more profound in FTD than in SD patients. Given our small sample sizes, this finding cannot be considered definitive. However, it is consistent with the general view of FTD as more profoundly affecting behavior and SD as more profoundly affecting language.\textsuperscript{1}

Our neuroanatomical analyses revealed that lower volumes in both frontal and temporal regions were associated with poorer emotion recognition for both fear (bilateral frontal, right temporal) and sadness (left frontal, bilateral temporal). Previous research using static photographs has implicated the important role that temporal structures (e.g., the amygdala) play in detecting negative emotions.\textsuperscript{12,37} However, there is considerable evidence that emotion recognition, especially when dynamic scenes are involved,\textsuperscript{14} involves more distributed neural networks.\textsuperscript{1,12}

**CONCLUSIONS** The present study applied laboratory methods derived from basic emotion research to study preservation and loss of emotional functioning in the early stages of FTLD. Results indicated that emotional reactivity (spanning subjective, behavioral, and physiologic responses) to simply themed happy, fear, and sad emotional stimuli, and emotional recognition for happiness experienced by another person are preserved in FTLD. In contrast, emotional recognition for the negative emotions of sadness and fear are clearly impaired. Correlations with regional volume loss indicate that diminished recognition is associated with loss in both frontal and temporal areas. These findings provide a more differentiated view of the emotional deficits in FTLD that may be useful in understanding the clinical presentation of the disease, delineating emotional situations that will be most problematic for patients and their families, and identifying areas of preserved functioning that could be targeted in behavioral interventions (at least in the early stages of the disease). The different patterns of results for emotional reactivity and emotion recognition point to the complexities of human emotion, underscoring the likely differences in neural circuits that underlie different aspects of emotional functioning and the differential vulnerability of these circuits to disease processes such as those found in FTLD and other dementias.

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**REFERENCES**