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Airway Somatosensory Deficits and Dysphagia in Parkinson's Disease

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Abstract

Background—Individuals with Parkinson's disease (PD) often experience substantial impairment of swallow control, and are typically unaware of the presence or severity of their impairments suggesting that these individuals may also experience airway sensory deficits. However, the degree to which impaired swallow function in PD may relate to airway sensory deficits has yet to be formally tested.

Objective—The purpose of this study was to examine whether airway sensory function is associated with swallow impairment in PD.

Methods—Eighteen PD participants and 18 healthy controls participated in this study and underwent endoscopic assessment of airway somatosensory function, endoscopic assessment of swallow function, and clinical ratings of swallow and disease severity.

Results—PD participants exhibited abnormal airway somatosensory function and greater swallow impairment compared with healthy controls. Swallow and sensory deficits in PD were correlated with disease severity. Moreover, PD participants reported similar self-rated swallow function as healthy controls, and swallow deficits were correlated with sensory function suggesting an association between impaired sensory function and poor self-awareness of swallow deficits in PD.

Conclusions—These results suggest that control of swallow is influenced by airway somatosensory function, that swallow-related deficits in PD are related to abnormal somatosensation, and that swallow and airway sensory function may degrade as a function of disease severity. Therefore, the basal ganglia and related neural networks may play an important role to integrate airway sensory input for swallow-related motor control. Furthermore, the airway deficits observed in PD suggest a disintegration of swallow-related sensory and motor control.

Keywords

Swallow; aspiration; penetration; residue; protection; larynx; non-motor

INTRODUCTION

Individuals with Parkinson's Disease (PD) exhibit evidence of impoverished sensorimotor integration and degradation of airway control [1, 2]. In particular, individuals with PD

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CONFLICT OF INTEREST STATEMENT
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frequently exhibit oropharyngeal dysphagia. However, individuals with PD are often unaware of the presence or severity of their dysphagia [3]. Such deficits of swallow and impaired awareness of these deficits may contribute to a primary source of morbidity and mortality in PD, including respiratory infection and airway obstruction [4, 5]. The degree to which dysphagia and its associated comorbidities including aspiration pneumonia and airway obstruction in PD may be related to airway somatosensory deficits is unknown.

Understanding airway somatosensory deficits in PD and the association between sensory and swallow function in PD may improve our ability to gage the presence, progression, severity, and treatment response of PD to various interventions. In addition, if somatosensory deficits are associated with airway function in PD, as demonstrated by our previous work, [1, 6] then it may be reasonable to examine the effects of intervention on airway sensory function, and even focus intervention to improve airway sensory function in PD. This line of research may provide important advances in our ability to improve overall function and quality of life for individuals with PD. Previous investigations suggested that airway somatosensory deficits were associated with deglutition, and that concomitant airway deficits may be present with advanced age and in neurological diseases including stroke or amyotrophic lateral sclerosis [7, 8]. However, previous studies have yet to directly examine whether there is an association between swallow function and airway somatosensory deficits.

Therefore, the purpose of this study was to examine the association between airway somatosensory function and swallow severity in individuals with PD and in healthy controls. We employed an endoscopic technique to assess airway somatosensory function, and compared these results with self-reported and endoscopic measures of swallow function. We hypothesized that PD participants would exhibit higher sensory thresholds, greater swallow severity, and less awareness of swallow deficits. In addition, we hypothesized that sensory thresholds would be associated with PD severity and swallow severity.

MATERIALS AND METHODS

Participants

The institutional ethics committee for the safety of human subjects approved this protocol, and written informed consent was obtained from each participant. We collected data from 36 participants, including 18 with idiopathic PD (9 men, 9 women), and 18 healthy controls (8 men, 10 women). Mean age was 73 (59–82) for PD and 76 (64–84) for control participants. PD participants were tested a mean of 6 years since PD diagnosis, and disease severity included Hoehn & Yahr Stages II, III, and IV [9]. We tested PD participants a minimum of 12 hours after their most recent dose of PD medication. PD participants were in otherwise good general health, with no history of other neurological or psychiatric disorder. Control participants were in good general health, with normal breathing, speech, swallow, and voice, and no history of neurological or psychiatric disease. Participants in each group were non-smokers and were free of pulmonary disease.

Somatosensory assessment

We employed an endoscopic stimulus delivery paradigm to present a pressure-calibrated burst of air to the laryngeal musoca to determine the threshold pressure at which each participant could perceive the stimulus. We previously described the device design and experimental approach, [1, 6] and will briefly review the procedures below. Each participant sat comfortably in an exam chair, and a transnasal laryngoscope (Pentax FNL-13RAP) was placed into the most patent naris. A topical decongestant was administered prior to scope placement, and the exam was completed without anesthesia. The air burst port of the

laryngoscope was coupled to a three-foot long rigid polyethylene tube. The opposite end of the tube was coupled to the output port of the somatosensory stimulus delivery device. The device design included a sound attenuating enclosure to prevent potential acoustic cues of the device from imposing a bias on participant responses.

As described previously, [1, 6] stimulus presentation was triggered during the initiation of the expiratory phase of respiration, and the air burst stimulus was directed to the laryngeal mucosa overlying the superior surface of the arytenoid cartilage. Each participant was instructed to press a hand-held switch as soon as they detected the stimulus, and each found the hand-switch easy to use. Stimulus delivery continued until we determined the pressure (mm Hg) of the air burst stimulus at which the participant reported to feel the stimulus 50% of the time following six crossings of the same stimulus level. We defined this stimulus level as the laryngeal somatosensory detection threshold.

Swallow assessment

To assess a self-rating of swallow function, each participant completed the Sydney Swallow Questionnaire (SSQ) [10]. The SSQ includes 17 questions; 16 items employ a 100 mm visual analog scale (Table 1). To directly assess swallow function, we completed endoscopic swallow assessments [11] of each participant as they ingested three 5 ml sips of water from a cup, three 5 ml bites of pudding by spoon, and one shortbread cookie. Water and pudding were mixed with green food coloring to increase visibility. We examined penetration (into the larynx above the vocal folds) and aspiration (below the vocal folds) of the ingested material into the airway using the Penetration-Aspiration Scale, [12] and assessed residue of food remaining in the oropharyngeal regions using a three point residue scale (0 = no residue, 1 = coating of residue, 2 = pooling of residue) [13].

Statistical analysis

We utilized two-sample *t*-tests to examine group differences in laryngeal sensory thresholds, and to test for group differences in self-rated swallow function, penetration/aspiration, and residue ($\alpha = 0.05$). To examine the potential associations between airway sensory function, PD severity, and swallow function, we computed Pearson product-moment correlation coefficients ($\alpha = 0.05$). We hypothesized that PD participants would exhibit higher laryngeal sensory detection thresholds, greater swallow severity, and less awareness of swallow deficits. In addition, we hypothesized that sensory thresholds would be associated with PD severity and swallow severity.

RESULTS

Laryngeal somatosensory detection thresholds and self-reported swallow severity scores for PD and control participants are displayed in Fig. 1. PD participants exhibited significantly higher thresholds than controls, but significant differences in self-reported swallow severity were only observed for SSQ Item 11 (cough or choke when swallowing liquids). Otherwise, PD and control participants reported similar levels of swallow severity. On average, these levels of swallow severity were comparable to those previously reported by healthy controls [10]. Results for endoscopic assessment of swallow are in Fig. 2. Direct assessment of swallow function revealed an absence of penetration and aspiration for both groups. However, PD participants exhibited more oropharyngeal residue for water, pudding, and cookie boluses than healthy controls.

We also examined the association between sensory function, swallow severity, and PD disease severity [14]. For PD participants, we observed medium to strong positive correlations ($r = 0.30$ to 0.55, Table 2, *p*-values are in table) between sensory and swallow

severity. PD participants also exhibited a medium correlation ($r = 0.40$, $p < 0.02$) between PD severity and sensory thresholds, with medium correlations ($r = 0.32$ to 0.44, Table 2, p-values are in table) between PD severity and swallow severity. For controls, we observed medium negative correlations ($r = -0.29$ to -0.45, Table 2, p-values are in table) between sensory and swallowing severity.

DISCUSSION

The association between airway somatosensory detection and swallow function in PD was previously unknown. To examine this association, we employed endoscopic measures of airway somatosensory and swallow function, and self-reported measures of swallow function in participants with PD and healthy controls. As hypothesized, we found that PD participants exhibited higher airway somatosensory detection thresholds and more oropharyngeal residue, but self-reported swallow severity in PD participants was essentially the same as healthy controls. For controls, interestingly, sensory detection thresholds and self-reported swallow deficits were negatively correlated. However, this finding makes sense because better airway sensory detection would logically be associated with a greater awareness of swallow difficulty.

It may seem surprising that differences in penetration and aspiration were generally unobserved between the control and PD groups. This null finding may be due to our selection of relatively healthy control and PD participants who were free of pulmonary disease. Therefore, it is possible that we have selected individuals who experience minimal penetration and aspiration. However, it is also possible that penetration and aspiration went unobserved in this study due to the use of an endoscopic instead of fluoroscopic examination. An endoscopic examination is able to detect penetration and aspiration, but may be unable to detect these events during the swallow when the view is obstructed by compression of the pharynx around the endoscope [11]. Given the health status of our PD participants, a more plausible alternate explanation may be that these participants had yet to consistently experience these specific deficits. If this is accurate, our findings would suggest that sensory abnormalities may precede penetration and aspiration deficits in the clinical progression of PD. This type of time course analysis would be quite interesting to consider in future investigations to follow the progression of somatosensory changes as PD severity progresses to advanced stages.

The primary swallow deficit observed in PD participants was oropharyngeal residue. This deficit is consistent with reduced movement of the oropharyngeal structures including reduced posterior tongue base movement, reduced pharyngeal wall contraction, and reduced elevation of the hyolaryngeal complex [15]. Although direct observation of these structures and their associated movements was excluded based on our choice of endoscopic assessment, the oropharyngeal residue may be due to reduced range of movement and reduced ability to propel the bolus from the oropharynx into the esophagus.

Our finding that PD participants exhibited swallow and airway somatosensory deficits compared to healthy controls is consistent with other PD motor and nonmotor problems [16–19]. The present work has important implications for understanding the somatosensory mechanisms of swallow control, and the influence of PD on these mechanisms. Swallow-related movements typically occur without auditory or visual guidance. Therefore, other afferent mechanisms guide swallow movements, including rapidly adapting mechanoreceptors within the airway mucosa that are positioned to respond to changes in pressure, and to directly encode mucosal tissue strains associated with swallow movement [20, 21]. Our findings are particularly helpful to elucidate proposed mechanisms of movement abnormalities in PD, including sensory gating deficits.

Clinical symptoms of PD, including dysphagia, may be due in part to abnormally excessive sensory gating, or reduced input of somatosensory information required for normal movement execution [22–29]. Excessive sensory gating in PD may reflect impaired integration of sensory inputs with the planning and execution of movement, resulting in impaired goal-related movements. For example, PD participants in the present report exhibited oropharyngeal residue. The inability of the sensorimotor regions of the basal ganglia, cerebral cortex, and other associated regions to receive accurate afferent input may account for errors in the initiation, timing, and range of swallow movements. The fact that individuals with PD often exhibit abnormally increased muscle tone and rigidity may reflect a tendency of the central nervous system to remain in a state of movement preparation, while awaiting the arrival of the afferent signal, resulting in a delayed or aberrant transition to movement execution [22]. In later stages of PD, this tendency may increase as the ability to initiate a swallow becomes more impaired.

Results from our study are consistent with the fact that individuals with PD are generally unaware of the severity of their swallow deficits, and often report in clinical settings that they feel as though they have ingested the entire food/drink bolus even in the presence of substantial oropharyngeal residue. The present results and this clinical observation suggest that swallow impairments in PD may be related to impaired somatosensory function and that swallow and airway sensory function may degrade as a function of disease severity. Therefore, the basal ganglia and related neural networks may play an important role to integrate airway sensory input for swallow-related motor control. Furthermore, the airway deficits observed in PD suggest a disintegration of swallow-related sensory and motor control.

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References

1. Hammer MJ, Barlow SM. Laryngeal somatosensory deficits in Parkinson's Disease: Implications for speech respiratory and phonatory control. Exp Brain Res. 2010; 201:401–409. [PubMed: 20012947]
2. Tzelepis GE, McCool DF, Friedman JH, Hoppin FG. Respiratory muscle dysfunction in Parkinson's disease. Am Rev Resp Disord. 1988; 138:266–271.
3. Robbins JA, Logemann JA, Kirshner HS. Swallowing and speech production in Parkinson's disease. Ann Neurol. 1986; 19:283–287. [PubMed: 3963773]
4. Beyer M, Herlofson K, Arsland D, Larsen J. Causes of death in a community-based study of Parkinson's disease. Acta Neurologica Scandinavica. 2001; 103:7–11. [PubMed: 11153892]
5. Fall P, Saleh A, Fredrickson M, Olsson J, Granerus A. Survival time, mortality, and cause of death in elderly patients with Parkinson's disease: A 9-year follow-up. Movement Disorders. 2003; 18:1312–1316. [PubMed: 14639673]
6. Hammer MJ. Design of a new somatosensory stimulus delivery device for measuring laryngeal mechanosensory detection thresholds in humans. IEEE Trans Biomed Eng. 2009; 56:1154–1159. [PubMed: 19272888]
7. Aviv JE, Martin JH, Keen MS, Debell M, Blitzer A. Air pulse quantification of supraglottic and pharyngeal sensation: A new technique. Ann Otol Rhinol Laryngol. 1993; 102:777–780. [PubMed: 8215097]
8. Amin MR, Harris D, Cassel SG, Grimes E, Heiman-Patterson T. Sensory testing in the assessment of laryngeal sensation in patients with amyotrophic lateral sclerosis. Ann Otol Rhinol Laryngol. 2006; 115:528–534. [PubMed: 16900807]

9. Hoehn MM, Yahr MD. Parkinsonism: Onset, progression and mortality. Neurology. 1967; 17:427–442. [PubMed: 6067254]
10. Wallace KL, Middleton S, Cook IJ. Development and validation of a self-report symptom inventory to assess the severity of oral-pharyngeal dysphagia. Gastroeneterology. 2000; 118:678–687.
11. Langmore, SE. Endoscopic evaluation and treatment of swallowing disorders. New York: Thieme; 2001.
12. Rosenbek JC, Robbins JA, Roecker EB, Coyle JL, Wood JL. A penetration-aspiration scale. Dysphagia. 1996; 11:93–98. [PubMed: 8721066]
13. Hind JA, Nicosia MA, Gangnon R, Robbins J. The effects of intraoral pressure sensors on normal young and old swallowing patterns. Dysphagia. 1995; 20:249–253. [PubMed: 16633867]
14. Cohen, J. Statistical power analysis for the behavioral sciences. 2. Hillsdale, Lawrence Erlbaum Associates; NJ: 1988.
15. Logemann, JA. Evaluation and treatment of swallowing disorders. Austin: Pro-Ed; 1998.
16. Lee MS, Kim HS, Lyoo CH. “Off” gait freezing and temporal discrimination threshold in patients with Parkinson disease. Neurology. 2005; 64:670–674. [PubMed: 15728290]
17. Liberini P, Parola S, Spano PF, Antonini L. Olfaction in Parkinson’s disease: Methods of assessment and clinical relevance. J Neurol. 2000; 247:88–96. [PubMed: 10751109]
18. Rickards C, Cody FWJ. Proprioceptive control of wrist movements in Parkinson’s disease: Reduced muscle vibration induced errors. Brain. 1997; 120:977–990. [PubMed: 9217682]
19. Snider SR, Fahn S, Isgreen WP, Cote LJ. Primary sensory symptoms in Parkinsonism. Neurology. 1976; 26:423–429. [PubMed: 944393]
20. Andreatta RD, Mann EA, Poletto CJ, Ludlow CL. Mucosal afferents mediate laryngeal adductor responses in the cat. J Appl Physiol. 2002; 93:1622–1629. [PubMed: 12381746]
21. Yoshida Y, Tanaka Y, Hirano M, Nakashima T. Sensory innervation of the pharynx and larynx. Am J Med. 2000; 108:51S–61S. [PubMed: 10718453]
22. Bischoff-Grethe, A.; Crowley, MG.; Arbib, MA. Movement inhibition and next sensory state prediction in the basal ganglia. In: Graybiel, AM.; DeLong, MR.; Kitai, ST., editors. The Basal Ganglia VI. Kluwer Academic/Plenum Publishers; New York: 2002.
23. Kaji R. Basal ganglia as a sensory gating devise for motor control. J Med Invest. 2001; 47:42–46.
24. Kaji R, Urushihara R, Murase N, Shimazu H, Goto S. Abnormal sensory gating in basal ganglia disorders. J Neurol. 2005; 252:IV/13–IV/16.
25. Lee RG. Pathophysiology of rigidity and akinesia in Parkinson’s disease. Eur Neurol. 1989; 29(Suppl 1):13–18. [PubMed: 2653833]
26. Lee RG, Tatton WG. Motor responses to sudden limb displacements in primates with specific CNS lesions and in human patients with motor system disorders. Can J Neurol Sci. 1975; 2:285–293. [PubMed: 809129]
27. Schneider, JS. Basal ganglia-motor influences: Role of sensory gating. In: Schneider, JS.; Lidsky, TI., editors. Basal ganglia and behavior: Sensory aspects of motor functioning. Hans Huber Publishers; Toronto: 1987.
28. Schneider JS, Diamond SG, Markham CH. Deficits in orofacial sensorimotor function in Parkinson’s disease. Ann Neurol. 1986; 19:275–282. [PubMed: 3963772]
29. Schneider JS, Diamond SG, Markham CH. Parkinson’s disease: Sensory and motor problems in arms and hands. Neurology. 1987; 37:951–956. [PubMed: 3587646]

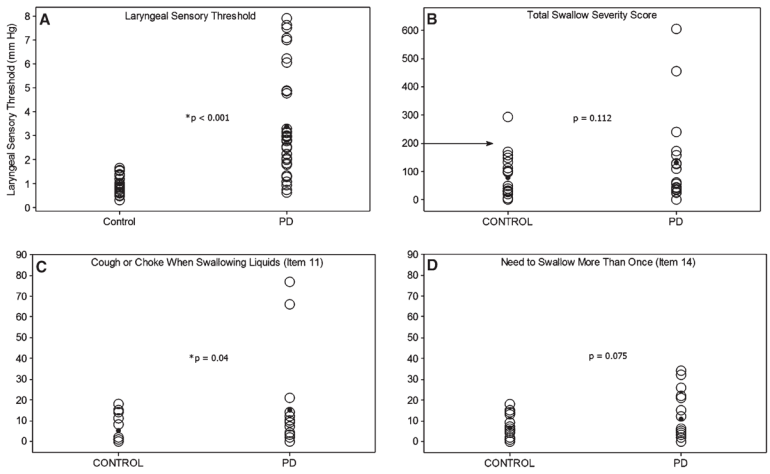

Fig. 1.

(a) Laryngeal somatosensory function for control and PD participants. The small black filled circles represent the group means and the open circles represent individual values for each participant in mm Hg (1 mm Hg = 133.32 Pa). (b through d) Self-reported swallow severity (Sydney Swallow Questionnaire) for control and PD participants. The small black filled circles represent the group means and the open circles represent individual values for each participant in mm. (b) Total swallow severity - arrow identifies upper range of normal, (c) Item 11 (cough or choke when swallowing liquids), (d) Item 14 (need to swallow more than once). Asterisk indicates statistical significance.

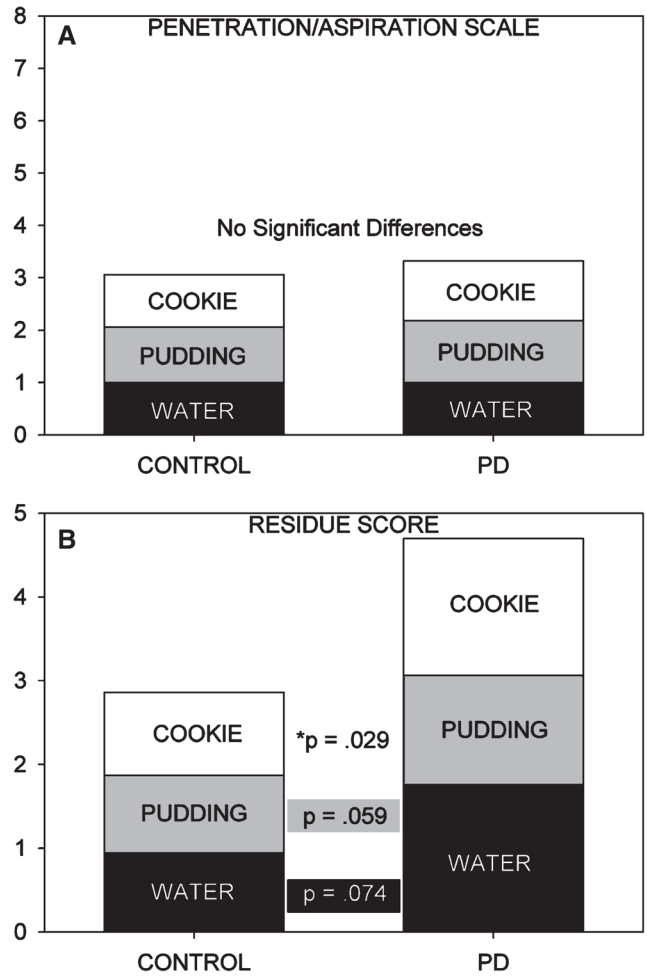

Fig. 2.

Endoscopic assessment of swallow function for control and PD participants. For the stacked bar graphs, individual rectangle height represents mean value for each food item. (a) Penetration/Aspiration (1 = none, 8 = silent aspiration). (b) Residue (0 = none, 1 = coating, 2 = pooling). Asterisk indicates statistical significance.

Table 1

SYDNEY SWALLOW QUESTIONNAIRE
Wallace KL, Middleton S and Cook IJ, Gastroeneterology 2000; 118 : 678–687

1	How much difficulty do you have swallowing at present?
2	How much difficulty do you have swallowing thin liquids?
3	How much difficulty do you have swallowing thick liquids?
4	How much difficulty do you have swallowing soft foods?
5	How much difficulty do you have swallowing hard foods?
6	How much difficulty do you have swallowing dry foods?
7	Do you have any difficulty swallowing your saliva?
8	Do you have any difficulty starting a swallow?
9	Do you ever have a feeling of food getting stuck in your throat when you swallow?
10	Do you ever cough or choke when swallowing solid foods?
11	Do you ever cough or choke when swallowing liquids?
12	How long does it take you to eat an average meal? *
13	When you swallow does food or liquid go up behind your nose or come out of your nose?
14	Do you ever need to swallow more than once for your food to go down?
15	Do you ever cough up or spit out food or liquids during a meal?
16	How do you rate the severity of your swallowing problem today?
17	How much does your swallowing problem interfere with your enjoyment or quality of life?

* Possible responses for Item 12: <15 min, 15–30 min, 30–45 min, 45–60 min, >60 min, unable to swallow at all. Responses to all other items use a 100 mm visual analog scale.

Table 2

PD: Sensory Score vs. Swallow Severity	
ITEM	***r*-value (*p*-value)**
1.	0.352 (0.035)
3.	0.545 (0.001)
4.	0.459 (0.005)
5.	0.379 (0.023)
6.	0.392 (0.018)
7.	0.438 (0.008)
8.	0.336 (0.045)
10.	0.413 (0.012)
11.	0.313 (0.063)
13.	0.316 (0.060)
14.	0.400 (0.016)
15.	0.319 (0.058)
16.	0.403 (0.015)
17.	0.297 (0.078)
TOTAL	0.426 (0.010)
Cookie Residue	0.394 (0.038)
PD: PD Severity vs. Swallow Severity	
ITEM	*r*-value (*p*-value)
1.	0.316 (0.060)
2.	0.386 (0.020)
8.	0.374 (0.025)
9.	0.443 (0.007)
11.	0.319 (0.058)
13.	0.412 (0.012)
14.	0.358 (0.032)
15.	0.363 (0.029)
TOTAL	0.333 (0.047)
CONTROLS: Sensory Score vs. Swallow Severity	
ITEM	*r*-value (*p*-value)
7.	−0.354 (0.034)
8.	−0.327 (0.051)
12.	−0.323 (0.055)
13.	−0.289 (0.087)
15.	−0.453 (0.006)
16.	−0.377 (0.024)
17.	−0.392 (0.018)

(For other items, $p > 0.1$.)