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Neural synchrony indexes impaired motor slowing after errors and novelty following white matter damage

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ABSTRACT

In humans, action errors and perceptual novelty elicit activity in a shared frontostriatal brain network, allowing them to adapt their ongoing behavior to such unexpected action outcomes. Healthy and pathologic aging reduces the integrity of white matter pathways that connect individual hubs of such networks and can impair the associated cognitive functions. Here, we investigated whether structural disconnection within this network because of small-vessel disease impairs the neural processes that subserve motor slowing after errors and novelty (post-error slowing, PES; post-novel slowing, PNS). Participants with intact frontostriatal circuitry showed increased right-lateralized beta-band (12–24 Hz) synchrony between frontocentral and frontolateral electrode sites in the electroencephalogram after errors and novelty, indexing increased neural communication. Importantly, this synchrony correlated with PES and PNS across participants. Furthermore, such synchrony was reduced in participants with frontostriatal white matter damage, in line with reduced PES and PNS. The results demonstrate that behavioral change after errors and novelty result from coordinated neural activity across a frontostriatal brain network and that such cognitive control is impaired by reduced white matter integrity.
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1. Introduction

Controlled behavior requires the ability to adapt ongoing actions to unexpected outcomes. For example, when driving a car, both one's own actions (e.g., accidentally switching on the windshield wiper, "error") and outside factors (e.g., a failing power steering, "novel") can lead to unexpected action outcomes, which require immediate adaptation of behavior. Over the life span, the capacity to implement such cognitive control processes progressively diminishes (Braver and Barch, 2002; Harty et al., 2013; Hedden and Gabrieli, 2004; Verhaeghen and Cerella, 2002), which is further escalated by pathologic age-related processes, such as small-vessel disease (SVD) (Prins et al., 2005).

In healthy individuals, cognitive control after unexpected action outcomes is well characterized. Both errors and novels lead to slower reaction times on subsequent trials (post-novelty slowing, PNS, Barcelo, et al., 2006; Notebaert et al., 2009; Parmentier et al., 2011, post-error slowing, PES, Rabbitt and Rodgers, 1977) and engage a number of prefrontal, cingulate, and basal ganglia brain regions (Hester et al., 2004; Kerns et al., 2004; Polich, 2007; Ridderinkhof et al., 2004; Soltani and Knight, 2000). In fact, errors and novels engage a common distributed brain network (Wessel et al., 2012), which could explain why both types of events evoke similar behavioral changes (PES and PNS). However, it is not clear how exactly PES and PNS are implemented within this network and

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how healthy or pathologic aging affects these cognitive control abilities.

Recent studies suggest that a right-lateralized frontostriatal motor inhibition mechanism could underlie PES and PNS (Danielmeier et al., 2011; Marco-Pallares et al., 2008; Wessel and Aron, 2013). This motor inhibition mechanism is engaged when actions have to be cancelled (Aron et al., 2007b, 2014) or slowed down (Chikazoe et al., 2009; Jahfari et al., 2010; Wessel and Aron, 2014; Wessel et al., 2013). Importantly, the anatomic regions that subserve this motor inhibition mechanism (right inferior gyrus, presupplemetary motor area, and subthalamic nucleus, Aron et al., 2007a; Rae et al., 2015) are in fact part of the wider frontostriatal network that is active after both errors and novelty (Wessel et al., 2012). Activity of this mechanism can be measured using electroencephalography (EEG). When this mechanism is engaged, neural synchrony between frontocentral and right frontolateral brain areas is increased (specifically within the beta frequency band, i.e., 12–24 Hz, Swann et al., 2011, 2012), which ostensibly reflects communication between distant brain areas (Fries, 2005). Hence, the implementation of PES and PNS could crucially depend on the neural communication between the distant nodes of the brain network underlying this mechanism, which is indexed by right-lateralized beta-band synchrony.

However, testing the causal importance of neural synchrony in humans is difficult. Whereas gray matter damage allows testing the causal role of focal brain areas, testing the causal role of the integrity of a distributed brain network necessitates the investigation of changes in white matter pathways. From this perspective, the aging process allows unique insights. Both healthy (e.g., Andrews-Hanna et al., 2007; Barrick et al., 2010; Davis et al., 2009; Westlye et al., 2010) and pathologic aging (e.g., Damoiseaux, et al., 2009; Prins et al., 2005) are characterized by reductions in white matter integrity. Here, we used small-vessel disease (SVD) as a model of structural white matter disconnection to study the causal role of right lateralized beta-band synchrony in PES and PNS. SVD is an aging-related disorder (Pugh and Lipsitz, 2002) characterized by vascular white matter lesions (Cummings, 1995) and lacunar infarcts (Okroglic et al., 2013), which lead to impaired long-range neural communication (Schaefer et al., 2014).

In our present study, EEG was recorded from participants with and without frontostriatal microlesions because of SVD (rated using the age-related white matter changes rating scale, ARWMC, Wahlund et al., 2001) during a hybrid error-novelty task (Wessel et al., 2012, 2014). We predicted that beta-band synchrony between frontocentral and right frontolateral brain regions would be increased after errors and novelty in participants with intact frontostriatal white matter. We furthermore predicted that such synchrony would be decreased in participants with frontostriatal structural disconnections and that this decrease would be correlated with impaired PES and PNS.

2. Materials and methods

2.1. Participants

Twenty-eight native German speakers with corrected or corrected-to-normal vision were recruited from the participant databases of the Day Clinic for Cognitive Neurology in Leipzig and the participant database of the Max Planck Institute for Human Cognitive and Brain Sciences. We specifically recruited participants for whom we had existing ratings of white matter integrity and neuropsychological testing data (these subjects had been recruited as part of another study, Quinque et al., 2012, for which they underwent neuropsychological assessment and magnetic resonance imaging (MRI) scanning, but not all of the current participants ended up participating in Quinque et al., 2012). Participants were excluded based on history of neuropsychiatric disorders (e.g., stroke, craniocerebral injury, neurodegenerative diseases, and dementia) or substance abuse. Furthermore, 4 subjects had to be excluded after recruitment (2 because of technical problems, 1 because of excessive error rate [38%, $z > 0.37$, significant outlier using Grubbs test at $p < 0.0001$], and 1 because of difficulties following the task instructions), resulting in a final sample size of 24. Participants were then grouped based on whether they had focal white matter damage in frontal or striatal regions (see subsequently), leading to an even split of the 24 participants into 2 groups: a frontostriatal lesion group (for ease of reading, we will from here on refer to this group simply as the "lesion group") and a control group without frontostriatal lesions (Table 1).

2.2. Procedure

T2-weighted and fast fluid-attenuated inversion recovery (FLAIR) images were independently rated by 2 experienced clinicians (MLS and ER) according to Wahlund et al. (2001). This assessment took place on average 10.5 months (standard error of the mean = 0.76) before study participation, as part of the recruitment for a prior study (Quinque et al., 2012) (we believe that this delay period is nonproblematic because longitudinal studies using the ARWMC show that the change in white matter that is to be expected in such a period is minor at best [Goos et al., 2010; Kapeller et al., 2003]. This was in line with the fact that our neuropsychological assessment showed no differences in any of the scales between the 2 time points [see Section 3]. Furthermore, any change in ARWMC would have worked against our results pattern because white matter damage can only reasonably progress in positive direction [i.e., toward more damage]. Hence, if there were indeed significant changes in ARWMC in our sample between the MRI and testing, they could have only led to a case in which some of the participants of our control group actually did have WM damage, which would reduce the group effect that we hypothesized). For further analyses, we used the mean of both raters' scores. A white matter lesion was defined as a hyperintensity of >5 mm diameter in the T2-weighted and FLAIR images. Hypointense signal changes on both T2 and FLAIR images with a minimal diameter of 2 mm were rated as lacunes. White matter ratings consisted of global score (range 0–30) and regional subscores for frontal, parietooccipital,

Table 1
Group characteristics

Characteristics	Unit	Lesion group		Control group	
		Mean	SEM	Mean	SEM
Age	y	62.42	1.80	62.00	1.91
Education	y	13.96	0.64	14.10	0.56
Gender	F/M	8/4		2/10	
WML					
Total	#	7.10	1.21	0.38	0.18
Frontal	#	2.75	0.42	0.13	0.07
Basal G.	#	0.96	0.30	0.04	0.04
Par.-occ.	#	2.50	0.51	0.21	0.11
Temporal	#	0.67	0.25	0.00	0.00
Infratentorial	#	0.21	0.17	0.00	0.00
Lacunes					
Total	#	1.83	0.66	0.21	0.10
Frontal	#	0.08	0.08	0.00	0.00
Basal G.	#	1.25	0.47	0.21	0.10
Par.-occ.	#	0.04	0.04	0.00	0.00
Temporal	#	0.00	0.00	0.00	0.00
Infratentorial	#	0.46	0.22	0.00	0.00

Key: Basal G., basal ganglia; F, female; M, male; par.-occ., parietooccipital; SEM, standard error of the mean; WML, white matter lesion; #, number.

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