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Letter to the Editor

Structural connectivity changes within the basal ganglia after 8 weeks of sensory-motor training in individuals with chronic stroke

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Dear Editor,

After a stroke, individuals can present impaired postural control due to deficits in the different domains and systems responsible for postural stability. Furthermore, they struggle with sensory integration of the visual, vestibular and somatosensory systems, that is, the ability to mobilize available sensory systems when one or more of the other sensory inputs are missing or insufficient [1,2]. The basal ganglia consists of subcortical nuclei — caudate, lentiform (putamen and globus pallidus), substantia nigra and subthalamic nucleus [3] — which are key structures, especially the caudate and lentiform nuclei, in motor learning and postural control [3,4]. A few studies have shown that balance training focused on sensory manipulation may improve balance ability, functional mobility and muscle activity in individuals with chronic stroke [1,2,5,6]. However, neuroimaging studies examining the effect of balance training on structural brain changes in chronic stroke individuals are very limited.

This pilot trial aimed to investigate whether an 8-week sensory-motor training (SMT) program focused on balance exercises with sensory system manipulation could change the structural connectivity between the 2 subcortical basal ganglia nuclei, the caudate and lentiform nuclei, with other regions of interest (ROI) in individuals with chronic stroke. We hypothesized that 8 weeks of SMT may improve postural control in individuals with chronic stroke because of improved efficiency of the basal ganglia network.

Nine individuals with chronic stroke were randomly allocated to an SMT or attention-matched control (CON) group by using sealed envelopes. Researchers were blinded to randomization, participant enrolment and data collection. The participants knew they would be allocated to a group after a pre-test but were unaware of the differences between the groups. Eligibility criteria included age 18 years and older, clinical diagnosis of stroke ≥ 6 months ago, and no other diagnosed neurological conditions. Both interventions occurred in a group setting, 3 times a week, in 45 to 60-min sessions, over 8 weeks. All participants underwent MRI within 1 week before and after the intervention. Written informed consents were received by all participants and the study was approved by the institutional Health Research Ethics Committee (S16/07/128).

The SMT program was built on the principles of Janda's sensory-motor training guides [7] as well as Horak and Nashner's movement strategies [8]. Overall, participants progressed through 8 weeks of balance training while their visual, vestibular and somatosensory systems were manipulated. Sessions 1 to 3 focused on posture and alignment, specifically providing input to the sensory-motor system from the ground up. According to Janda [7], sensory information being integrated by the central nervous system should be optimal at the foot, sacroiliac joint and cervical spine because of the large number of proprioceptors in these areas. By increasing the somatosensory (proprioceptive and tactile) input, subcortical pathways can be stimulated to facilitate coordinated movements. Sessions 4 to 9 focused on static balance, therefore maintaining postural stability while progressing to eyes-closed conditions, with head movements as well as on unstable surfaces. Sessions 10 to 15 progressed to dynamic balance, which added arm and leg movements while maintaining postural stability and also manipulating the 3 sensory systems as above. Sessions 16 to 24 focused on functional balancing movements, which included activities of everyday life under sensory manipulation and dual tasking conditions.

Participants in the CON group attended educational talks on the importance of living a healthy lifestyle. The use of an attention-matched control group aimed to control for non-specific intervention effects (i.e., attention, intervention contact, social support, etc.).

Diffusion tensor imaging data were obtained by using an echo planar imaging whole-brain sequence with echo time = 83 ms; repetition time = 10000 ms; bandwidth = 1776 Hz/Px; field of view read 256 mm^2; 69 axial slices, at 2.0-mm slice thickness, for isotropic voxel size $2.0 \times 2.0 \times 2.0$ mm^3; and gradient pulses along 64 different directions with b-value 1500 s/mm^2. Non-diffusion-weighted images ($b = 0$ s/mm^2) were acquired afterward to guide registration of individual diffusion. FSL 5.1 (https://fsl.fmrib.ox.ac.uk/fsl/fslwiki) was used to analyze diffusion-weighted images. A standard Freesurfer 5.3.0 (https://surfer.nmr.mgh.harvard.edu/) parcellation scheme and probabilistic whole-brain tractography was used for structural connectome reconstruction (Table 1). By using the Desikan–Killiany Atlas for automated anatomical segmentation and labelling, 72 ROI (36 in each hemisphere) were created (Fig. 1) [9]. Data were assessed by normal probability plots, and in some cases with outliers, the variables were winsorized, which reduced the effects of outliers without having to remove them. The connectivity strength between both the caudate and lentiform nuclei with the other ROI were analyzed between groups (SMT vs CON) and within

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Table 1
Parcellation of cortical and subcortical structures by using Freesurfer 5.3.0.

Banks superior temporal sulcus	Parahippocampal gyrus
Caudal anterior cingulate cortex	Pars opercularis
Caudal middle frontal gyrus	Pars orbitalis
Caudate nucleus	Pars triangularis
Cuneus cortex	Pericalcarine cortex
Frontal pole	Postcentral gyrus
Fusiform gyrus	Posterior cingulate cortex
Inferior parietal cortex	Precentral gyrus
Inferior temporal gyrus	Precuneus cortex
Insula	Rostral anterior-cingulate cortex
Isthmus-cingulate cortex	Rostral middle frontal gyrus
Lateral occipital cortex	Superior frontal gyrus
Lateral orbital frontal cortex	Superior parietal cortex
Lentiform nucleus	Superior temporal gyrus
Lingual gyrus	Supramarginal gyrus
Medial orbital frontal cortex	Temporal pole
Middle temporal gyrus	Thalamus
Paracentral lobule	Transverse temporal cortex

groups (pre- vs. post-treatment) by using a mixed model repeated-measures Anova with a Fisher least significant difference post-hoc test. Additionally, Cohen's *d* effect size (ES) and magnitude-based inference (Table 2) statistics were calculated to supplement the traditional inferential statistics. $P \leq 0.05$ was considered statistically significant. All statistical tests involved use of STATISTICA v13.

Among 15 individuals who met the inclusion criteria, only 9 completed the study, 5 in the SMT group and 4 in the CON group (Table 3). The 2 groups did not differ in demographic characteristics ($P > 0.05$).

After the intervention, the SMT group showed increased structural connectivity between the left caudate and contralateral and ipsilateral caudal anterior cingulate cortex; between the right caudate and contralateral paracentral lobule and rostral middle frontal gyrus as well as ipsilateral fusiform gyrus, superior frontal gyrus and parahippocampal gyrus; between the left lentiform and contralateral and ipsilateral isthmus cingulate cortex; and between the right lentiform and contralateral temporal pole, inferior parietal cortex and precuneus cortex (Table 2).

For the CON group, structural connectivity was reduced between the left caudate and ipsilateral parsopercularis and superior frontal gyrus but was increased between the left caudate and ipsilateral lingual gyrus, the right caudate and contralateral inferior temporal gyrus and cuneus, and the left lentiform and ipsilateral rostral anterior cingulate cortex (Table 2).

This study is the first to assess the effects of 8 weeks of SMT on structural brain changes between the basal ganglia nuclei and

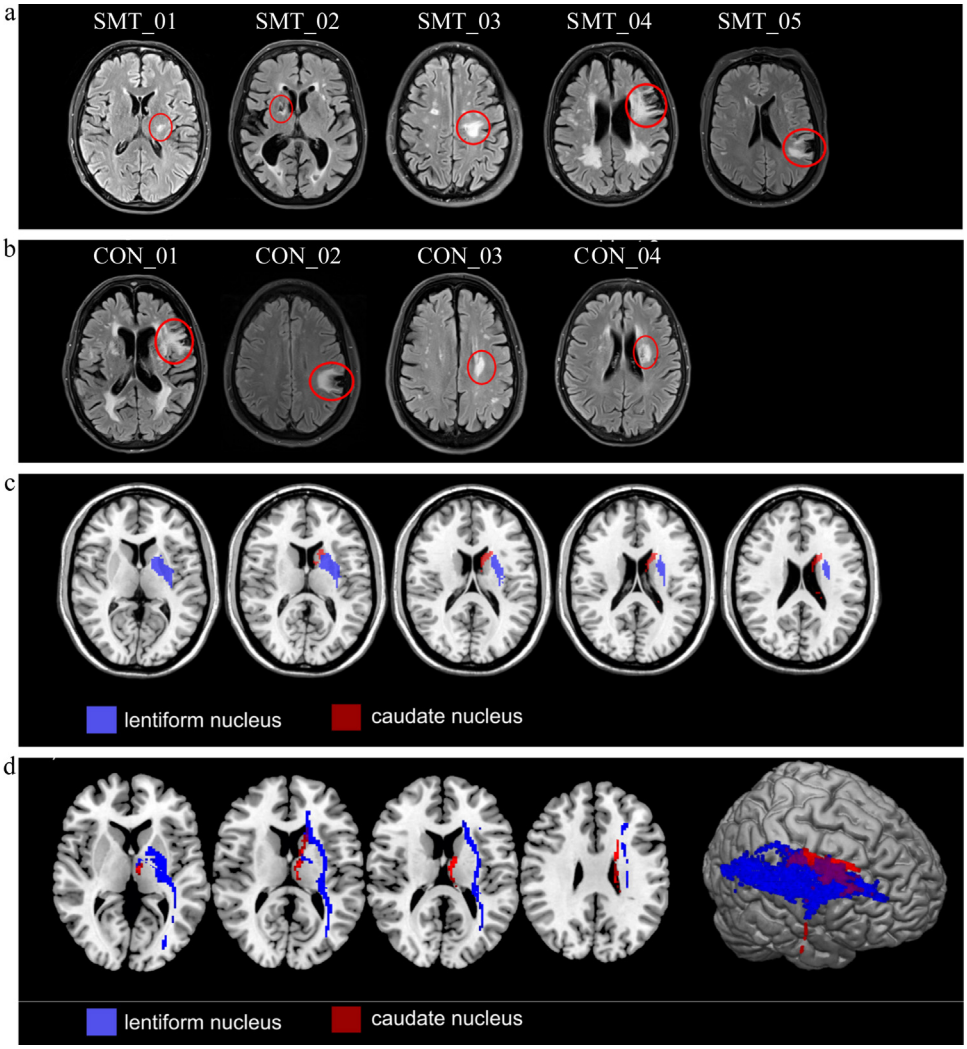

Fig. 1. Stroke lesions of (a): sensory-motor training (SMT) group and; (b): attention-matched control (CON) group; (c) Lentiform nucleus and caudate nucleus region of interest; (d): Fiber tracks derived from lentiform nucleus and caudate nucleus region of interest.

Table 2
Effect size (ES) and magnitude-based inference (MBI) data for caudate and lentiform nuclei for sensory-motor training (SMT) and attention-matched control (CON) groups.

		Within-group difference over time: SMT group		Within-group difference over time: CON group		Between-group difference: After intervention	
Lobes	Region of interest	*P* value (ES) {ES 95% CI}	MBI	*P* value (ES) {ES 95% CI}	MBI	*P* value (ES) {ES 95% CI}	MBI
Left caudate							
Frontal	Left caudal cingulate cortex, anterior	0.05 (1.7^{H}) {−0.1–2.75}	Positive: 96.3%, VL Negligible: 1.9%, VU Negative: 1.7%, VU	0.60 (0.39^{S}) {−1.09–1.70}	Positive: 21.2%, U Negligible: 19.9%, U Negative: 58.9%, P	0.02 (2.11^{H}) {0.14–3.19}	Positive: 98.4%, VL Negligible: 1.0%, VU Negative: 0.6%, VU
	Right caudal cingulate cortex, anterior	0.01 (1.92^{H}) {0.13–2.95}	Positive: 99.2%, VL Negligible: 0.4%, MU Negative: 0.3% MU	0.74 (0.16^{S}) {−1.25–1.52}	Positive: 23.4%, U Negligible: 30.1%, P Negative: 46.4%, P	0.02 (2.3^{H}) {0.24–3.34}	Positive: 98.4%, VL Negligible: 0.9%, VU Negative: 0.6%, VU
	Left pars opercularis	0.47 (0.33^{S}) {−0.99–1.50}	Positive: 58.3%, P Negligible: 29.6%, P Negative: 12.1%, U	0.03 (2.04^{H}) {−0.09–3.04}	Positive: 1.1%, VU Negligible: 0.9%, VU Negative: 98%, VL	0.70 (0.27^{S}) {−1.12–1.52}	Positive: 51.5%, P Negligible: 25.0%, U Negative: 23.5%, U
	Left superior frontal gyrus	0.32 (0.37^{S}) {−0.98–1.51}	Positive: 63.0%, P Negligible: 30.6%, P Negative: 6.4%, U	0.05 (0.81^{L}) {−0.81–2.03}	Positive: 1.3%, VU Negligible: 4.0%, VU Negative: 94.6%, VL	0.30 (0.83^{L}) {−0.73–1.96}	Positive: 77.3%, Li Negligible: 13.1%, U Negative: 9.6%, U
Occipital	Left lingual gyrus	0.41 (0.50^{M}) {−0.90–1.60}	Positive: 50.0%, P Negligible: 43.0%, P Negative: 7.0%, U	0.05 (0.82^{L}) {−0.74–2.11}	Positive: 95.8%, VL Negligible: 2.6%, VU Negative: 1.6%, VU	0.19 (0.94^{L}) {−0.58–2.15}	Positive: 5.6%, U Negligible: 10.1%, U Negative: 84.3%, Li
Right caudate							
Frontal	Left paracentral lobule	0.008 (1.05^{L}) {−0.44–2.15}	Positive: 98.9%, VL Negligible: 0.8%, VU Negative: 0.3%, VU	0.68 (0.59^{M}) {−0.93–1.88}	Positive: 60.7%, P Negligible: 10.2%, U Negative: 29.1%, P	0.06 (1.36^{VL}) {−0.32–2.50}	Positive: 95.0%, VL Negligible: 3.2%, VU Negative: 1.8%, VU
	Left rostral middle frontal gyrus	0.01 (1.51^{H}) {−0.18–2.50}	Positive: 98.9%, VL Negligible: 0.8%, VU Negative: 0.3%, VU	0.92 (0.07^{N}) {−1.24–1.54}	Positive: 49.2%, P Negligible: 9.5%, U Negative: 41.3%, P	0.05 (1.53^{X}) {−0.29–2.53}	Positive: 94.9%, VL Negligible: 3.9%, VU Negative: 1.2%, VU
	Right superior frontal gyrus	0.02 (0.75^{L}) {−0.76–1.87}	Positive: 97.1%, VL Negligible: 2.5%, VU Negative: 0.4%, VU	0.48 (0.67^{M}) {−0.82–2.01}	Positive: 18.6%, U Negligible: 12.0%, U Negative: 69.4%, P	0.12 (1.34^{VL}) {−0.25–2.59}	Positive: 91.0%, VL Negligible: 5.0%, VU Negative: 4.0%, VU
Temporal	Left inferior temporal gyrus	0.46 (0.15^{S}) {−1.11–1.37}	Positive: 35.2%, P Negligible: 59.3%, P Negative: 5.5%, U	0.05 (0.96^{L}) {−0.86–1.96}	Positive: 92.2%, VL Negligible: 6.6%, U Negative: 1.1%, VU	0.74 (0.28^{S}) {−0.93–1.73}	Positive: 57.9%, P Negligible: 9.9%, U Negative: 32.2%, P
	Right fusiform gyrus	0.05 (1.1^{L}) {−0.32–2.31}	Positive: 96.7%, VL Negligible: 1.4%, VU Negative: 1.9%, VU	0.45 (0.79^{L}) {−0.66–2.21}	Positive: 73.1%, P Negligible: 8.2%, U Negative: 18.7%, U	0.06 (1.4^{VL}) {−0.18–2.69}	Positive: 95.3%, VL Negligible: 2.8%, VU Negative: 1.9%, VU
	Right parahippocampal gyrus	0.03 (1.37^{VL}) {−0.76–1.75}	Positive: 94.6%, VL Negligible: 4.9%, VU Negative: 0.5%, VU	0.50 (1.21^{VL}) {−0.56–2.35}	Positive: 70.8%, P Negligible: 7.9%, U Negative: 21.3%, U	0.07 (1.2^{VL}) {0.14–3.19}	Positive: 95.2%, VL Negligible: 2.3%, VU Negative: 2.5%, VU
Occipital	Left cuneus cortex	0.94 (0.04^{N}) {−1.11–1.37}	Positive: 47.0%, P Negligible: 11.9%, U Negative: 41.1%, P	0.03 (1.54^{H}) {−0.39–2.58}	Positive: 97.5%, VL Negligible: 1.5%, VU Negative: 1.0%, VU	0.23 (0.81^{L}) {−0.64–2.07}	Positive: 7.9%, U Negligible: 8.6%, U Negative: 83.6%, Li
Left lentiform							
Frontal	Left rostral cingulate cortex, anterior	0.17 (0.58^{M}) {−0.76–1.76}	Positive: 82.7%, Li Negligible: 11.9%, U Negative: 41.1%, P	0.03 (1.4^{VL}) {−0.43–2.53}	Positive: 97.6%, VL Negligible: 1.4%, VU Negative: 1.0%, VU	0.29 (0.86^{L}) {−0.66–2.05}	Positive: 9.6%, U Negligible: 11.7%, U Negative: 78.7%, Li
Parietal	Right isthmus cingulate cortex	0.04 (0.83^{L}) {−0.64–1.90}	Positive: 94.8%, VL Negligible: 4.3%, VU Negative: 0.9%, VU	0.20 (0.68^{M}) {−1.01–1.79}	Positive: 77.7%, Li Negligible: 17.5%, U Negative: 4.8%, VU	0.30 (0.68^{M}) {−0.80–1.88}	Positive: 75.9%, Li Negligible: 15.2%, U Negative: 8.9%, U
Right lentiform							
Temporal	Left temporal pole	0.02 (1.31^{VL}) {−0.25–2.4}	Positive: 97.6%, VL Negligible: 1.9%, VU Negative: 0.5%, VU	0.48 (0.32^{S}) {−1.16–1.62}	Positive: 55.9%, P Negligible: 31.9%, P Negative: 12.2%, U	0.25 (0.86^{L}) {−0.68–2.02}	Positive: 1.0%, VU Negligible: 99.0%, VL Negative: 0.0%, MU
Parietal	Left inferior parietal cortex	0.02 (1.43^{VL}) {−0.20–2.47}	Positive: 98.0%, VL Negligible: 1.5%, VU Negative: 0.5%, VU	0.56 (0.38^{S}) {−1.07–1.72}	Positive: 61.0%, P Negligible: 19.5%, U Negative: 19.5%, U	0.26 (0.75^{L}) {−0.75–1.94}	Positive: 79.4%, Li Negligible: 12.7%, U Negative: 7.9%, U
	Left precuneus cortex	0.03 (0.92^{L}) {−0.51–2.06}	Positive: 96.8%, VL Negligible: 2.5%, VU Negative: 0.8%, VU	0.47 (0.50^{M}) {−1.18–1.60}	Positive: 56.0%, P Negligible: 32.3%, P Negative: 11.7%, U	0.27 (0.71^{M}) {−0.75–1.93}	Positive: 77.8%, Li Negligible: 14.4%, U Negative: 7.8%, U
	Right isthmus cingulate cortex	0.01 (1.00^{L}) {−0.50–2.01}	Positive: 98.8%, VL Negligible: 1.0%, VU Negative: 0.2%, VU	0.51 (0.24^{S}) {−1.56–1.22}	Positive: 11.2%, U Negligible: 40.2%, P Negative: 48.6%, P	0.13 (1.11^{VL}) {−0.52–2.23}	Positive: 89.1%, Li Negligible: 7.1%, U Negative: 3.8%, VU

CI: confidence interval; ES: effect size; ^{N}Negligible; ^{S}Small; ^{M}Medium; ^{L}Large; ^{VL}Very large; ^{H}Huge; MBI: magnitude-based inference; Li: likely; MU: most unlikely; P: possibly; VL: very likely; VU: very unlikely; U: unlikely

other ROI in individuals with chronic stroke. SMT increased structural connectivity strength between the basal ganglia network and fronto-parietal areas (i.e., caudal anterior cingulate cortex, rostral middle frontal gyrus, superior frontal gyrus and isthmus cingulate). The anterior cingulate cortex forms part of the basal ganglia network, and according to previous results [4,10], activity in the caudal anterior cingulate cortex is associated with sensory-motor regions and plays an important role in motor control. The frontal lobe can be subdivided into the superior frontal gyrus, middle frontal gyrus and inferior frontal gyrus. The superior frontal gyrus is considered to consist of the supplementary motor area (SMA) and connected with the middle frontal gyrus [11]. According to MRI studies, the SMA plays an important role in postural control [12,13]. These areas are involved in various brain functions, such as sequencing and initiation of actions, motor learning and motor control [11,14]. Also, the isthmus cingulate

Table 3
Clinical information for each participant in the SMT and CON groups.

ID	Hemisphere	Location	Type of lesion	Size
CON_01	Left	Frontal partial MCA infarction	Insular cortical lesion	Middle
CON_02	Left	Dorsal partial MCA infarction	Parietal cortical lesion	Middle
CON_03	Left	PLIC and basal ganglia (lentiform nucleus)	Subcortical lesion in the corticospinal tract and lentiform nucleus	Tiny
CON_04	Left	Internal capsule and basal ganglia (lentiform nucleus)	Subcortical lesion in the lentiform nucleus and the corticospinal tract	Small
SMT_01	Left	PLIC	Subcortical lesion	Tiny
SMT_02	Right	PLIC and basal ganglia (lentiform nucleus)	Subcortical lesion	Small
SMT_03	Left	PLIC and its connection to cortex	Subcortical lesion	Small
SMT_04	Left	Frontal partial MCA infarction	Insular cortical lesion	Middle
SMT_05	Left	Dorsal partial MCA infarction	Parietal cortical lesion	Middle

MCA: middle cerebral artery; PLIC: posterior limb of internal capsule.

includes involvement of the medial and inferior lateral parietal areas and has been used to study the default mode network (DMN) because it shows characteristics of the DMN [15,16]. Connectivity within the DMN is associated with various functions such as monitoring the world around us and can be modulated by the basal ganglia via the dopamine system [4].

In contrast, the CON group showed increased connectivity strength between the basal ganglia network and the orbito-temporal and frontal lobe areas (i.e., lingual gyrus, inferior temporal gyrus and rostral anterior cingulate cortex) after the educational talks. The lingual gyrus contains the primary visual cortex, which plays an important role in visual processing [17]. Additionally, the inferior temporal gyrus is connected behind the inferior occipital gyrus and plays a role in higher levels of visual processing [18]. Therefore, increases within these areas in the CON group could be due to the nature of the intervention, consisting solely of educational talks using presentations. The basal ganglia and visual processing are related, in that the output of the basal ganglia targets the occipitotemporal processing pathways within the inferior temporal cortex [19]. Also, the rostral anterior cingulate cortex is related to prefrontal regions, which are responsible for higher mental functions [20]. Consequently, the educational talks could have contributed to improved cognitive functioning in the CON group.

SMT may increase connectivity strength between the basal ganglia nuclei and fronto-parietal areas, and listening to educational talks may increase structural connectivity in visual processing and higher cognitive orbito-temporal and frontal lobe areas. The results for both groups are representative of the type of intervention executed. SMT could have postural control-related restorative effects on structural connectivity and support causal changes in activity-dependent neuroplasticity in individuals with chronic stroke. Nevertheless, this was a preliminary study regarding this topic and results should be interpreted with caution. The clinical implications of the changes in structural connectivity have across-the-board applications and show that it is possible to produce postural control improvements long after stroke by means of SMT. The topic should be researched further.

Disclosure of interest

The authors declare that they have no competing interest.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at https://doi.org/10.1016/j.rehab.2019.02.002.

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Tania Zastron[a,b,*], Simon S. Kessner[c], Karsten Hollander[b,d], Götz Thomalla[c], Karen Estelle Welman[a]
[a]*Department of Sport Science, Movement Laboratory, Stellenbosch University, South Africa*
[b]*Department of Sports and Exercise Medicine, Institute of Human Movement Science, University of Hamburg, Germany*
[c]*Department of Neurology, University Medical Center Hamburg-Eppendorf, Hamburg, Germany*
[d]*Department of Sports and Rehabilitation Medicine, BG Trauma Hospital of Hamburg, Germany*

*Corresponding author at: Department of Sport Science, Movement Laboratory, Stellenbosch University, South Africa
E-mail address: taniazastron@gmail.com (T. Zastron).

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