



Research Report

Arithmetic fact retrieval deficits in chronic stroke — A deficit of relearning?



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ABSTRACT

In acute stroke patients, arithmetic fact retrieval deficits have been observed due to disrupted white matter connections within a left-hemispheric network centered around the angular gyrus and middle temporal gyrus (Smaczny et al., 2023). However, it remains unclear which specific structural disconnections also hinder successful remediation in the chronic stage of stroke. In this study, 92 patients were examined to determine which impairments continue to affect multiplication performance even in the chronic phase after a first-time unilateral left-hemispheric stroke. Our results revealed a strong association between impaired multiplication performance and the disconnection of left long-term memory (para)hippocampal areas from left frontal and right parietal regions. Thus, unlike previous findings in the acute stroke phase, our results in the chronic phase emphasize the importance of (para)hippocampal regions for successful multiplication performance. We suggest that the affected areas and connections in chronic patients with persistent multiplication problems not only indicate areas that are crucial for the relearning of arithmetic facts, but also those crucial for the learning of arithmetic facts in general. More generally, we suggest that the acquisition of arithmetic facts depends on structural integrity of a network centered around the left (para)hippocampus, while the retrieval of consolidated arithmetic facts from memory relies on the integrity of a left-hemispheric network involving angular gyrus and middle temporal gyrus.

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1. Introduction

Multiplication within the small multiplication table (i.e., up to 9×9) is considered to be largely solved using arithmetic fact retrieval and can be explained by the process of retrieving results of highly overlearned arithmetic prompts from memory. This way, the correct result can be retrieved from memorized multiplication tables, supposedly in a verbal format (“Three times seven equals twenty-one”) instead of having to calculate it laboriously (Dehaene, Piazza, Pinel, & Cohen, 2003; Delazer, Domahs, et al., 2003; Grabner, Ansari, et al., 2009; Grabner, Ischebeck, et al., 2009) or grossly approximating the result (Dehaene, Spelke, Pinel, Stanescu, & Tsivkin, 1999). Fact retrieval in simple multiplication processing is, thus, in stark contrast to calculation as present in more complex subtraction processing. The latter tends to be primarily solved using procedure-based quantity manipulation (see Suárez-Pellicioni, Prado, & Booth, 2022 for a review).

The most influential model in numerical cognition, the triple code model, assumes a verbal representation (subserving arithmetic fact retrieval) and a magnitude representation (subserving quantity manipulations) associated with specific grey matter brain regions to explain these different types of processing (Dehaene & Cohen, 1995, 1997). More recent proposals have highlighted the importance of network structures (Arsalidou, Pawliw-Levac, Sadeghi, & Pascual-Leone, 2018; Arsalidou & Taylor, 2011) and white matter connections underlying these two representations (Klein et al., 2016; Smaczny et al., 2023). For instance, the fronto-parietal math-responsive network (Amalric & Dehaene, 2018, 2019) emphasizes the importance of large-scale neocortical network structures, while other proposals have added additional cortico-subcortical memory and control circuits (Menon, 2016). The two-network framework (TNF) was proposed as the first model for numerical cognition that considers the white matter pathways connecting the grey matter networks (Klein & Knops, 2023).

The TNF suggests that number processing relies on distinct and distributed networks for magnitude processing and arithmetic fact retrieval, which interact in an integrated manner dependent on the task's context. Compared to the triple code model, the TNF expands the concept of a verbal representation to an entire network for arithmetic fact retrieval with specific connectivity. The network supports highly overlearned numerical tasks such as retrieving arithmetic facts, naming numbers, and counting. It is located in left-hemispheric temporal, inferior parietal, and prefrontal areas, and is interconnected through dorsal (callosal bundle) and ventral pathways (external/extreme capsule system). Therefore, even unilateral lesions should have the potential to impair fact retrieval, provided they affect the left hemisphere (Klein & Knops, 2023). In contrast, the magnitude processing network is supposed to underly the mental manipulation of numerical magnitude (e.g., during calculation or estimation procedures; Klein et al., 2016). It is situated in both hemispheres, covering widespread and homologue networks that connect parietal areas like the intraparietal sulcus (IPS) and the posterior superior parietal lobule via dorsal (superior longitudinal fasciculus) and ventral pathways (external/

extreme capsule system) with frontal areas. This means that the ability for basic magnitude processing is supposed to be redundantly available to both hemispheres. In other words, an impairment of the number magnitude representation should require bilateral parietal impairment. If a lesion impairs IPS function only unilaterally in either the left or right hemisphere, the other hemisphere should be able to largely compensate for this function (Duffau, 2009; Klein & Knops, 2023).

The TNF assumes that the interrelation between the two systems of magnitude processing and fact retrieval is reciprocal and influenced by task demands (such as problem size, operation, involved carry operations), and individual competences (such as individual training, development, age; Klein et al., 2016; Klein & Knops, 2023): the more a task requires fact retrieval (e.g., in simple multiplication) to be solved, the less it requires magnitude processing and vice versa. Yet, it is essential to note that it is neither assumed that the fact retrieval network is capable of fully compensating for a bilateral IPS impairment, nor that, conversely, the magnitude processing network can entirely compensate for a persistent fact retrieval deficit. This is because, although both networks are assumed to interact closely, a small but stable component of processing from the other network is thought to be required for all task—regardless of task demands or individual competences (Klein et al., 2016; Klein & Knops, 2023).

For learning arithmetic facts, it follows that in the initial phase of learning new arithmetic facts, the processing of numerical magnitudes must still be relied upon heavily. However, once these facts are well established, they can be largely retrieved from long-term memory (Delazer et al., 2019). This dynamic transition from magnitude-based calculation to memory-based fact retrieval in response to the same stimulus material is reflected in additional activation in the medial temporal lobe, particularly in the hippocampus (Bloechle et al., 2016; Menon, 2016), stronger hippocampal structural connectivity (Klein, Willmes, Bieck, Bloechle, & Moeller, 2019) and a shift of activation from the magnitude processing network into the fact retrieval network (Bloechle et al., 2016; Delazer, Domahs, et al., 2003). Notably, the hippocampus does not seem to be required to retrieve facts after they are consolidated in memory (Delazer et al., 2019).

Crucially, both the triple code model and the TNF were based on either data from neuropsychological single-case patient studies (Dehaene & Cohen, 1995, 1997) or correlative data only, such as fMRI and/or DTI data (e.g., Arsalidou & Taylor, 2011; Göbel, Terry, Klein, Hymers, & Kaufmann, 2022; Klein et al., 2016). Yet, causal statements about brain-behavior associations can only be made when a brain area is actively damaged or disrupted (Rorden & Karnath, 2004). With some exceptions (Delazer et al., 2019; Semenza, Salillas, De Palleggrin, & Della Puppa, 2017), few group studies were devoted to arithmetic fact retrieval. Therefore, we recently examined a group of acute stroke patients on addition, subtraction, and multiplication (Smaczny et al., 2023) using a standardized test, the numerical processing and calculation battery (NPC; Delazer, Girelli, Granà, & Domahs, 2003). While there were hardly any patients with deficits in addition and subtraction, multiplication tended to be impaired in several patients with left-hemisphere lesions. Using indirect metrics

of disconnection (Griffis, Metcalf, Corbetta, & Shulman, 2021), we found that multiplication deficits were associated with disruptions within a mostly left-hemisphere network, which largely resembled the arithmetic fact retrieval network as proposed by Klein and Knops (2023). This included left angular gyrus (Areas 39PGp and 39 PGa) and left-hemispheric language areas such as superior temporal cortex (Areas 22 and 38), supramarginal gyrus (Area 40), inferior frontal gyrus (Areas 44 and 45), middle frontal gyrus (Area 10) as well as thalamus, retrosplenial cortex, IPS (Area hIP3), and premotor cortex (Area 4). While these findings showed how disconnection affects multiplication in patients shortly after a stroke, they cannot provide any information on which structures, when disconnected, prevent successful remediation or the use of alternative processing pathways, even in the chronic stage. Thus, it is unclear how network disruptions might affect restructuring processes in the long term.

Therefore, the current study aimed to examine disconnections detrimental to the success of remediation processes in patients in the chronic stage of their stroke, including aphasia patients who may exhibit co-occurring arithmetic fact retrieval and language impairments. Crucially, at the time of testing, all patients had completed at least one previous neuropsychological rehabilitation stage, which included numerical and arithmetic training in addition to other tasks such as language and speech therapy. For arithmetic fact retrieval, the numerical training in the rehabilitation program consisted of a standard fact retrieval drill training (Delazer, Domahs, et al., 2003), which patients had to complete using a computer. We hypothesized that lesions in chronic patients with persistent multiplication deficits despite unsuccessful numerical rehabilitation attempts would predominantly point to areas that are crucial for the (re)learning of arithmetic facts and, vice versa, can explain the disability to (re)learn in the case of a critical lesion. Therefore, a group of 92 chronic, first-time unilateral left-sided stroke patients in rehabilitation carried out the numerical processing and calculation battery (NPC; Delazer, Girelli, et al., 2003) as well as further control tasks, such as the Aachen Aphasia Test (AAT; Huber, Poeck, Weniger, & Willmes, 1983), of which results for this sample had been reported elsewhere (Ochtrup et al., 2013; Rath et al., 2015). We restricted the sample to left-hemispheric patients because - in line with theoretical models of numerical cognition - we had previously observed no deficits in fact retrieval among right-hemispheric patients with acute stroke (Smaczny et al., 2023).

2. Methods

2.1. Participants

Patients were retrospectively analyzed and taken from a larger study on aphasia and acalculia at the RWTH Aachen University Hospital in Aachen, Germany and the Neurological Rehabilitation Unit of the Kliniken Schmieder Konstanz (see Ochtrup et al., 2013; Rath et al., 2015). This larger study prospectively included all patients during a period of two years who were treated in the aphasia ward at RWTH Aachen University or in the rehabilitation clinic in Constance, who had

consented to the study, and who met the inclusion criteria. The inclusion criteria were established prior to testing and specified that only patients were included who had suffered their stroke more than six months before testing. This led to a sample of $n = 92$ patients with a left-hemisphere stroke. Patients or their relatives gave their informed consent to participate in the study. Clinical and demographic data for the whole group are given in Table 1; an illustration of the lesion overlaps can be seen in Fig. 1. The majority of lesions (i.e., voxels that were damaged in at least 50% of the patients) were localized around a gravity center in the insula and included - apart from the insula - thalamus, claustrum, putamen, pallidum, caudate nucleus, inferior frontal gyrus (Areas 44, 45 and 47), middle frontal gyrus (Area 9), superior temporal gyrus (Areas 22, 41 and 42), middle temporal gyrus (Area 21), temporal pole (Area 38), precentral gyrus (Areas 4 and 6), post-central gyrus (Area 2 and Area 43), supramarginal gyrus (Area 40), angular gyrus (Area 39), and amygdala.

2.2. Procedure

The assessment was carried out in a sitting position in a silent room in 3–4 1-h sessions per patient. The session structure was kept as consistent as possible across participants by administering the same tasks in the same order and the same room over a maximum of 1 h. However, since the patients' performance was affected to varying degrees and not necessarily in the same tasks, the testing lasted only 3 sessions for some patients and up to 4 sessions for others. This means that although all tasks were completed in the same order, the test sessions did not necessarily start with the same tasks for all patients. Patients were examined once or twice per week. We ensured that all patients were able to follow the task instructions. Patients were not only administered the NPC (Delazer, Girelli, et al., 2003), but also the AAT (Huber et al., 1983), of which detailed results for this sample had been reported elsewhere (Ochtrup et al., 2013).

2.3. Stimuli

For the current analysis, we were interested in patients' arithmetic fact retrieval as measured by the single-digit addition, subtraction and multiplication tasks from the NPC. The multiplication task comprises 42 items, the addition task 24, and the subtraction task 20 items. Following the procedure

Table 1 – Demographic and clinical data of all patients.

n		92
Sex		29f, 63m
Age (years)	Mean (SD)	52 (11.2)
Interval lesion onset to examination (months)	Mean (SD)	38.0 (34.9)
Interval lesion onset to imaging (months)	Mean (SD)	38.5 (35.5)
Education (qualification for university admission)	# Yes/no	40/52
Aphasia present at the time of testing ^a	# Yes/no	45/19

^a Based on the probabilistic classification of the AAT (Huber et al., 1983).

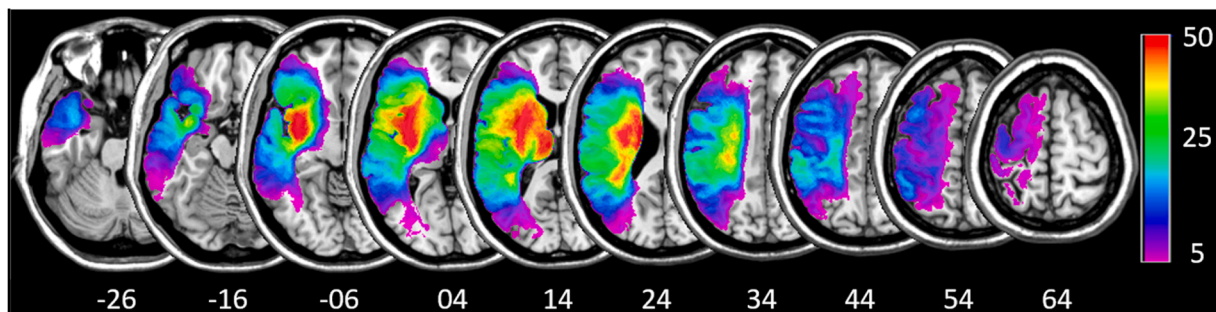


Fig. 1 – Lesion overlap of all patients included in the study ($n = 92$). The color bar indicates the frequency of lesion overlap. The vertical z-coordinate of standardized MNI space is provided below each slice.

from Smaczny et al. (2023), items that are typically solved via rule-based processing (multiplying by ‘0’ or ‘1’, subtracting by ‘0’ or down to ‘0’, and adding ‘0’; (McCloskey, Aliminosa, & Sokol, 1991) were excluded from the analysis, resulting in 36 multiplication facts, 20 addition facts, and 18 subtraction facts. For each item, a single arithmetic problem (“3 + 7”) was printed individually in the center of an individual sheet of A4 paper (black digits on white paper, digit height: 7 mm).

According to the standardized NPC procedure, testing was stopped after five consecutive incorrect or missing responses, and no time limit was imposed. However, responses lasting longer than 10 sec were rated incorrect, implying that the retrieval of the arithmetic facts had failed. Self-corrections were allowed.

In subtraction, 81 (addition: 77) out of 92 patients made a maximum of 2 errors, which is in the normal range. This lack

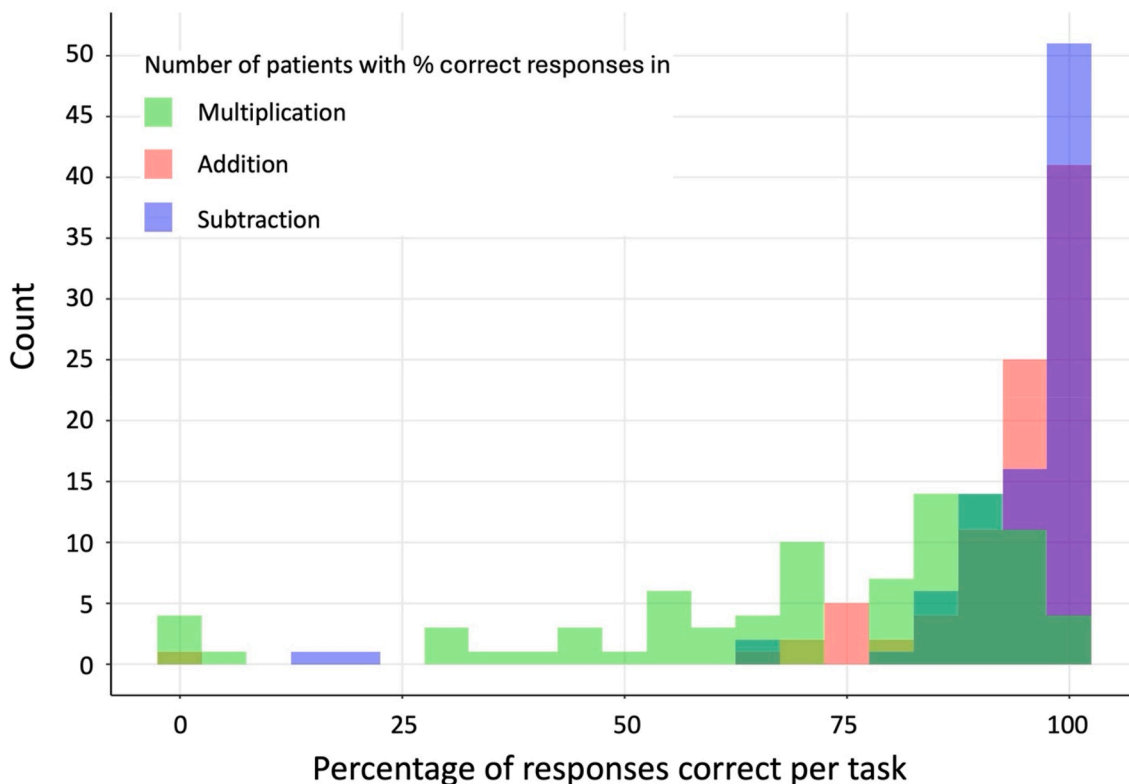


Fig. 2 – An overview of behavioral data. The x-axis depicts the percentage of correct responses. Note that while patients showed remarkable behavioral variance for multiplication (light green), they tended to show ceiling effects with low behavioural variance for addition (red) and subtraction (blue). Note that the violet color indicates an overlay of addition (red) and subtraction (blue) performance, while the dark green color indicates an overlay of all three operations. Petrol green indicates an overlay of multiplication (light green) and subtraction (blue). Similar to the findings in acute stroke (Smaczny et al., 2023), the low variance in addition and subtraction was not sufficient for conducting a reliable lesion analysis.

of behavioural variance (see also Fig. 2) precluded lesion analysis or made it highly unreliable specifically for subtraction and addition.

Notably, 64 out of 92 patients were diagnosed initially with aphasia. Therefore, it was crucial to control for a more general effect of aphasia so that a deficit present only secondarily due to a primary deficit in reading Arabic numerals is not attributed to a deficit in a numerical task. Therefore, we added the performance of the number reading task from the NPC as a covariate to the analyses. The number reading task consists of 18 numbers in Arabic notation that the patient is supposed to read aloud. The numbers range from single-to multi-digit numbers (i.e., 1 single-digit number, 4 two-digit numbers, 6 three-digit numbers, 4 four-digit numbers, 2 five-digit numbers, and 1 six-digit number). This way, it was possible to control for the more general effect that aphasic (number) reading deficits might have secondarily on arithmetic.

2.4. Lesion analysis

Anatomical images of patients tested in Aachen were available from a 1.5T Philips Gyroscan Achieva scanner. In Konstanz, a 3T Siemens Skyra scanner was used in addition to a 1.5T Philips Gyroscan Achieva. Images were acquired within three weeks of all testing sessions for the study, including a T2-weighted fluid-attenuated inversion-recovery (FLAIR) scan with 60 transversal slices of 2 mm thickness (voxel size = $1 \times 1 \times 2 \text{ mm}^3$), TR = 6.0 s, TE = 120 msec and a T1-weighted fast field echo (FFE) 3D sequence with 160 transversal slices of 1 mm thickness (voxel size = $1 \times 1 \times 1 \text{ mm}^3$), TR = 8.3 msec, TE = 120 msec. Raw DICOM data was converted into NIFTI format using Matlab 7.10 (MathWorks Inc., Natick, MA, USA).

Lesion boundaries, including gliotic parts and pseudocysts, were manually delineated directly on every transverse slice of the individual FLAIR image by an experienced experimenter blind to the patients' behavioral performance using MRIcron software. The T1 image was considered to obtain additional information for assessing necrotic tissue.

Then, both the anatomical scan and the lesion map were normalized into stereotaxic space using the 'Clinical Toolbox' (Rorden, Bonilha, Fridriksson, Bender, & Karnath, 2012; www.mccauslandcenter.sc.edu/CRNL/clinical-toolbox) implemented in SPM12 (www.fil.ion.ucl.ac.uk/spm). Since images of chronic patients are usually characterized by structural distortions, sulcal widening and widening of the ventricles, especially in case of a large lesion, the normalization process can lead to unrealistic estimation of lesion location and extent, especially for structures that are located close to the ventricles (e.g., basal ganglia, thalamus; Karnath & Rorden, 2012)). Therefore, if necessary, the normalized lesion image had to be manually adjusted to the standard template by validating specific anatomical landmarks such as basal ganglia and extending the lesion image to these structures.

2.5. Whole-brain disconnectivity mapping

Analogous to the analysis of acute patients in Smaczny et al. (2023), we created individual white matter disconnectivity topographies for each patient. These topographies indicate

the proportion of disconnected fibers for each white matter voxel in the brain imaging space running through this voxel. It allows a topographical assessment of a lesion's impact on brain connectivity. To this end, we applied the Lesion Quantification Toolkit (LQT; Griffis et al., 2021). The LQT utilizes a tract-wise connectome atlas and embeds the patient's lesion map. The toolkit identifies all fiber streamlines that intersect with the lesion and maps connectome-wide disconnection induced by the lesion. In other words, each voxel value corresponds to the percentage of all streamlines within this voxel that are expected to be disconnected by the lesion. We used the LQT's standard HCP-842 atlas (Yeh et al., 2018) for atlas-based tractography, keeping the default parameter setting as suggested in the LQT manual.

Subsequently, we examined multiplication performance by analyzing continuous disconnectivity maps using mass-univariate General Linear Models (GLMs) in 'NiiStat' (<https://github.com/neurolabusc/NiiStat>). In this analysis, the predictor variable was the previously mentioned percentage value of disconnected streamlines of a voxel, while the outcome variable was the multiplication score. We added lesion size and performance in the number reading task from the NPC as covariates to rule out the risk of larger lesions leading to more extended disconnection maps and misclassifications of more general effects of number reading deficits on arithmetic as primary arithmetic deficit. Only voxels with a disconnection in at least five patients were considered in the analysis. Tests were performed one-sided at $p < .05$ and corrected for family-wise errors. The family-wise error rate was determined using 5000 permutations with the Freedman-Lane algorithm for Permutation (Winkler, Ridgway, Webster, Smith, & Nichols, 2014). This disconnectivity topography mapping thus identified voxels for which disconnection is associated with low performance in the task at hand.

2.6. Region-to-region disconnectivity

We additionally analyzed parcel-wise disconnectivity as provided by the LQT. This procedure allowed us to identify which direct disconnections between two grey matter regions are significantly associated with low performance in the multiplication task. We created a structural connectivity matrix by combining the provided tractography atlas and the Brainnetome Atlas (BN-246, Fan et al., 2016) as our grey matter parcellation atlas. The BN-246 is multi-modally derived, contains 210 cortical and 36 subcortical subregions, and was developed specifically for connectivity analyses. The number of streamlines disconnected by the lesion map between each pair of parcels was converted to a percentage of disconnected streamlines, resulting in symmetric 246-by-246 disconnectivity matrices. Each value in this matrix denotes the percentage of disconnected streamlines between two given grey matter areas.

For ROI-to-ROI disconnections, a mass-univariate GLM was run in NiiStat with a family-wise error correction by the Freedman-Lane algorithm (Winkler et al., 2014). Many ROI-to-ROI disconnections were rarely or never present in the data, likely either because the sample's lesion anatomy did not include damage to the connection or because the connection was physiologically non-existent. Therefore, we identified the

Table 2 – Raw scores (number of items solved correctly) observed for the different tasks.

	Items	N of patients	Mean	Median	SD	Range
Multiplication	36	86 ^a	26.36	29.5	8.71	0–36
Addition	20	92	18.55	19	2.64	0–20
Subtraction	18	92	16.84	18	2.37	3–18

^a = For six patients, no multiplication data was provided.

total number of all patients with a disconnection present (i.e., a disconnection score >0) for each ROI-to-ROI connection. We removed all connections affected in less than 15 patients from the analysis (see Sperber, Griffis, & Kasties, 2022). We then computed a general linear model for each ROI-to-ROI connection with the independent variable disconnectivity score, the dependent variable multiplication performance, and the covariates lesion size and number reading performance. Afterwards, the Freedman-Lane permutation routine with 50,000 permutations was employed to assess the distribution of maximum statistics under the null hypothesis. We obtained a one-sided, corrected threshold for statistical significance at $p < .05$ by identifying the 95th percentile of permutation-derived maximum statistics.

3. Results

64 out of 92 patients presented with some form of aphasia at the time of testing based on the probabilistic classification of the Aachen Aphasia Test (AAT; Huber et al., 1983). Table 2 summarizes the behavioral results of all three tasks regarding multiplication, addition and subtraction fact retrieval. As can be seen from this table and from Fig. 2 (green bars), patients

showed a remarkable variation of behavioral scores in multiplication while the variance in the other two tasks (red and blue bars) was not sufficient for lesion analysis. Notably, the few patients who showed deficits in addition and/or subtraction also showed a deficit in multiplication, which was even more pronounced.

Fig. 3 presents the voxel-wise whole-brain analysis carried out in NiiStat. Results indicate voxels from the left parahippocampal cingulum and potentially retrosplenial cingulum (Jones, Christiansen, Chapman, & Aggleton, 2013; sagittal illustration), as well as disconnection of the posterior corpus callosum (axial illustration). To provide more details about clusters, we identified all clusters with at least 5 voxels (standard setting in xjview) and present these findings in supplementary material S1 and in Fig. 4.

Fig. 5 visualizes grey-matter-to-grey-matter disconnections that were significantly associated with lower performance in multiplication. These mainly were disconnections between the left superior frontal gyrus (SFG) and left temporal gyrus, left hippocampal and parahippocampal areas to left SFG and right parietal areas, and interhemispheric occipital disconnections. For an overview of all significant disconnections between two parcels, see Table 3.

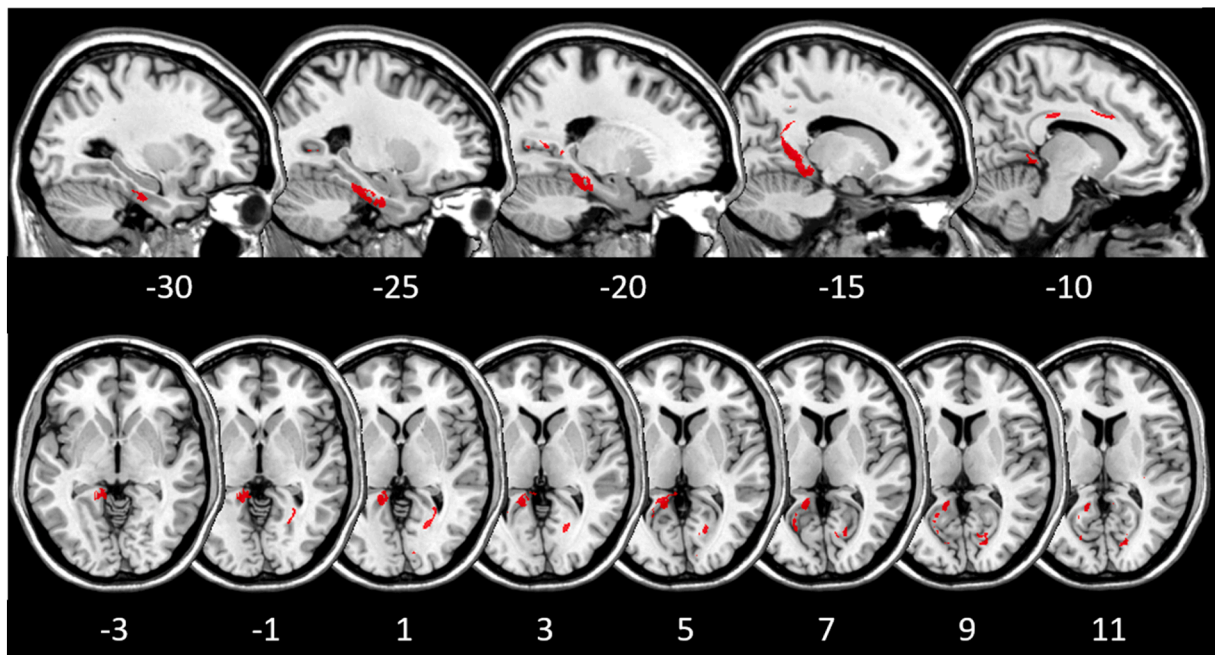


Fig. 3 – Disconnectivity mapping of multiplication. Sagittal and axial view of the brain. Statistical voxel-wise lesion-behavior mapping (VLBM) analyses using mass-univariate GLMs in multiplication. The voxels that survived permutation-based FWE correction at $p < .05$ are plotted. The respective x or z coordinates of standardized MNI space are given below each slice.

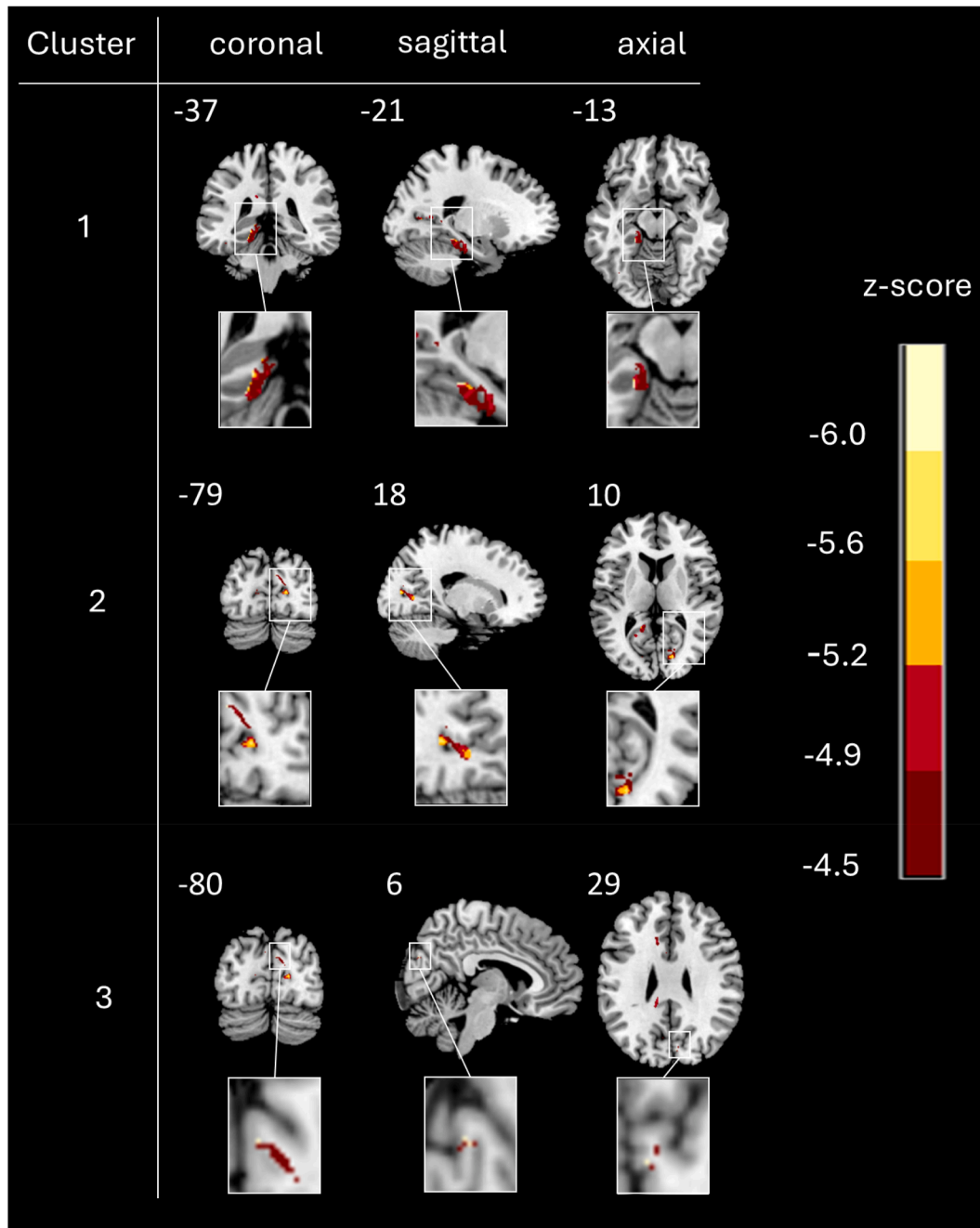


Fig. 4 – Coronal, sagittal, and axial displays of the 3 largest clusters at their peak MNI-coordinates.

4. Discussion

Complementing previous work in acute stroke patients (Smaczny et al., 2023), we examined arithmetic fact retrieval deficits in chronic stroke patients to identify the structures whose disconnection prevents sufficient reorganization/remediation processes in the long term. More precisely, we examined how indirect measures of white matter disconnection derived from chronic stroke imaging were associated with multiplication deficits. While our main research question

was concerned with differences in acute and chronic disconnection related to multiplication, we also aimed to integrate the findings within the framework of the TNF.

4.1. Multiplication fact learning

The voxelwise analysis implied that disconnection of white matter within the left parahippocampal cingulum and the posterior area of the corpus callosum is associated with lower performance in multiplication in chronic stroke patients.

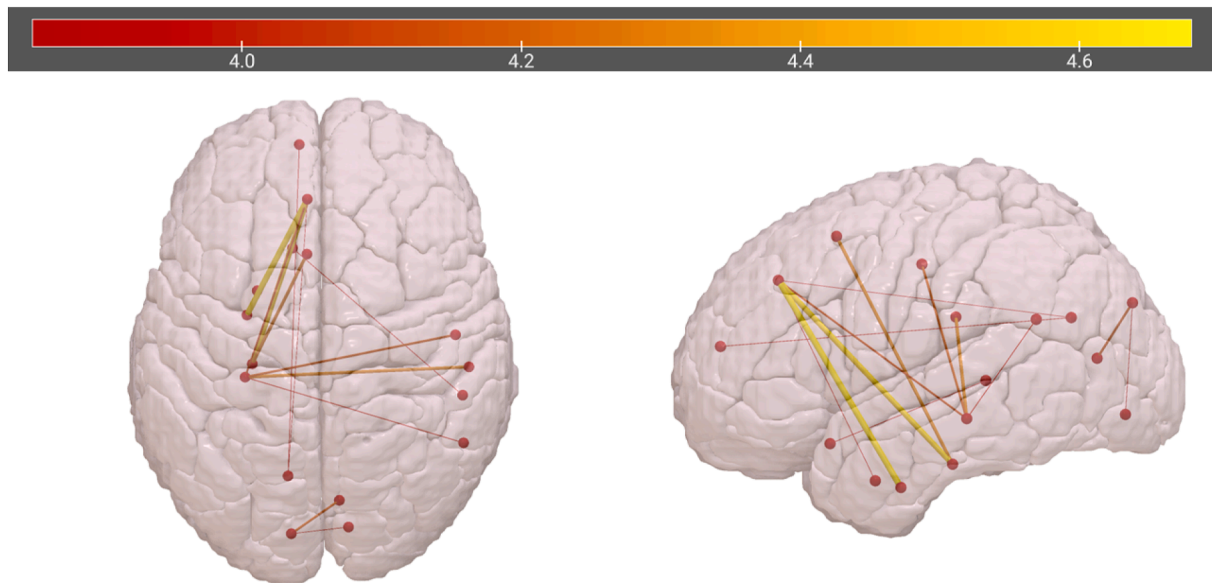


Fig. 5 – Region-to-region disconnectivity mapping of multiplication. Axial and sagittal view of the brain. Statistical region-to-region disconnectivity mapping analyses using mass-univariate linear models with multiplication as the criterion variable. Direct grey-matter-to-grey-matter disconnections that survived permutation-based FWE correction at $p < .05$ are plotted. Z-scores of the disconnection are reflected by the respective strength and colour of the connection.

Table 3 – All significant disconnections identified in the region-to-region analysis of multiplication. The disconnections are located between the parcel on the left part and the parcel on the right part of the table, each specified with the superordinate region and the respective hemisphere. The strength of the disconnection is given with its z-score.

Side	Region	Parcel	z-score	Parcel	Region	Side		
L	SFG	A8m	4.264	A35/36c	PhG	L		
		A9m	4.589	A35/36c				
		A9m	3.863	Area TI				
		A9m	4.679	A35/36r				
		A9m	4.071	Caudal segment			Hippocampus	L
		A9m	3.852	DmpoccS			Prc	L
		A10m	3.871	DmpoccS				
L	Hippocampus	Caudal segment	3.900	PGa	IPL	R		
		Caudal segment	4.265	Pfop				
		Caudal segment	4.125	A1/2/3ulhf			PcG	
L	LoccC	MsoccG	3.862	cLinG	MvoccC	R		
		MsoccG	4.128	rCunG				
L	Orbital gyrus	Area 13	3.870	rpSTS	pSTS	R		

L = Left, R = Right, SFG = Superior Frontal Gyrus, LoccC = Lateral Occipital Cortex, PhG = Parahippocampal Gyrus, Prc = Precuneus, IPL = Inferior Parietal Lobule, PcG = Postcentral Gyrus, MvoccC = Medioventral Occipital Cortex, pSTS = Posterior Superior Temporal Sulcus, A8m = Medial Area 8, A9m = Medial Area 9, A10m = Medial Area 10, MsoccG = Medial Superior occipital Gyrus, area TI = Temporal Agranular Insular Cortex, DmpoccS = Dorsomedial Parietooccipital Sulcus, PGa = Rostroventral Area 39, Pfop = Rostroventral Area 40, A1/2/3ulhf = Area 1/2/3 (upper limb head and face region), cLinG = Caudal Lingual Gyrus, rCunG = Rostral Cuneus Gyrus, rpSTS = Rostroposterior Superior Temporal Sulcus.

Additionally, the parcel-to-parcel analysis implied that disconnections between left (para-)hippocampal and frontal areas, left hippocampus with right parietal areas, and inter-hemispheric occipital disconnections impair multiplication.

These findings contrast with results from acute stroke patients, which show in the same task that a left temporo-parietal disconnection between AG and STG/MTG, often (but not necessarily) occurring together with aphasia, can lead to multiplication errors (Smaczny et al., 2023). The current sample was in the chronic stage of stroke, and 64 of the 92

patients had initially presented with some aphasia. All patients had completed at least one previous neuropsychological rehabilitation program focusing on numerical and arithmetic training in addition to language and speech therapy. Additionally, all patients reported that they had attempted to re-learn arithmetic fact retrieval outside of the rehabilitation program. Therefore, the difference between previous results and the current ones can be explained by a deficit in the ability to (re)-learn arithmetic facts in chronic stroke: The left hippocampus and its connection to parietal

areas is crucial for the encoding of arithmetic facts (Delazer et al., 2019; Klein et al., 2019; Qin et al., 2014). If this area is damaged, relearning is not possible.

In the unaffected brain, arithmetic fact learning involves a dynamic transition from procedure-based calculation processes to memory-based retrieval processes in response to the same stimulus material (Bloechle et al., 2016; Delazer, Domahs, et al., 2003; Qin et al., 2014). New arithmetic problems that are not yet encoded as facts are most likely acquired using effortful procedures or segmentation of the problem into smaller ones (e.g. $4 \times 3 = 3 + 3 + 3 + 3$; De Smedt, 2016; Siegler, 1988), using the neocortical fronto-parietal math-responsive network (Amalric & Dehaene, 2018, 2019; Bloechle et al., 2016; Delazer, Domahs, et al., 2003). With intensive drill training, these processes then become more automated (Zaunmuller et al., 2009), which leads to memory-based encoding of problem–answer associations within the hippocampal archicortex (here with lateralization preference for the left hippocampus; Bloechle et al., 2016; Klein et al., 2019), while less and less neocortical procedure-based calculation is required. Thus, recently learned arithmetic facts are supposed to rely on the hippocampus, which reinstates the cortical ensembles that were active during encoding, whereas neocortical memory develops more slowly through frequent reactivation (Frankland & Bontempi, 2005; McGaugh, 2000). Only after arithmetic facts are well consolidated are they likely to be retrieved via neocortical retrieval processes (e.g., in a network of AG and/or MTG; Delazer, Domahs, et al., 2003; Prado, Mutreja, & Booth, 2014; Smaczny et al., 2023). This is in line with the fact that the involvement of the hippocampus has been described as temporary/time-limited memory-based retrieval (Delazer et al., 2019).

Yet, if the hippocampus is disconnected from the number magnitude network and/or the arithmetic fact retrieval network, this mechanism cannot be employed, impairing the re-learning of arithmetic facts. This is, however, precisely the case for our data. Our results imply disconnections between hippocampal structures, the superior frontal gyrus, and right parietal areas. Both the superior frontal gyrus (which is often referred to as the dorsolateral prefrontal cortex) and the IPS support procedure-based calculation (Göbel et al., 2022; Kong et al., 2005; for meta-analyses see Arsalidou et al., 2018; Arsalidou & Taylor, 2011). Rosenberg-Lee et al. (2018) found that arithmetic training was associated with increased functional connectivity between the left hippocampus and right parietal areas. Given the fact that the problem–answer association for multiplication facts needs to be transiently established within memory regions such as the hippocampus (Bloechle et al., 2016; Delazer et al., 2019) before it is consolidated as verbally mediated memory-based arithmetic facts, this pattern of disconnection may lead to specific difficulties in the re-learning of verbalized facts, including arithmetic facts.

Right parietal areas have also been implied in the “magnitude fact-checking” of results derived via arithmetic fact retrieval (Arcara et al., 2021; Della Puppa et al., 2013; Semenza et al., 2017; Smaczny et al., 2023). Thus, dysfunctional checking of overall result magnitude when establishing new arithmetic facts using calculation-based procedures might add to persisting difficulties in re-learning arithmetic facts.

Furthermore, Fig. 3 suggests that disconnection in the posterior corpus callosum, i.e., the splenium, is detrimental to multiplication, which connects temporal and parietal areas interhemispherically. A disconnection of this interhemispheric connection might hinder contralateral right-hemispheric language areas from becoming more involved in fact retrieval functions compared to the respective contralateral homologue areas, the left temporal gyrus, as it has been described for single-case patient studies with successful remediation in the literature (Zaunmuller et al., 2009; Zipse, Norton, Marchina, & Schlaug, 2012, for the case of aphasia; see also Duffau, 2009, for an overview on brain reorganization after injury). It has also been described that the posterior parietal cortex (PPC) can acquire a memory representation rapidly during learning (Brodt et al., 2016, 2018; Sestieri, Shulman, & Corbetta, 2017). While it is still debated whether these early contributions go beyond an online reinstatement of previous activity or whether they originate from an actual neocortical engram, it is certainly not beneficial if the formation of these temporary or longer-term engrams is restricted.

Finally, there was one disconnection from the left orbital gyrus to the right superior temporal sulcus. This particular disconnection has not been described yet in the literature but may be related to partial injury (Rorden, Fridriksson, & Karnath, 2009), meaning that the right superior temporal sulcus might share some white matter pathways with left hemisphere areas that were disconnected from the orbital gyrus. This might hinder the right superior temporal sulcus from becoming more involved in fact retrieval functions compared to the contralateral homologue area, the left temporal gyrus (Zaunmuller et al., 2009; Zipse et al., 2012).

To conclude, our findings on multiplication strongly point to a deficit in arithmetic fact re-learning and show further areas that may be interesting to examine in future studies.

4.2. Implications for the two-networks-model framework

Some of the current findings fit well with the propositions made by the TNF, while others do not. The fact that white matter disconnections of the hippocampus in chronic stroke lead to significant deficits in multiplication (which was not the case in acute stroke [Smaczny et al., 2023]), reflects that, in acute stroke, the limited retrieval of arithmetic facts was dominating the symptomatology, while in chronic stroke a deficit is only observed if there is an inability to relearn arithmetic facts (fact encoding). While the hippocampus was generally considered in the latest version of the TNF (Klein & Knops, 2023), its inclusion was based on data from the literature (Delazer et al., 2019) and correlative neuroimaging data (Qin et al., 2014). A first confirmation of a more causal role for the entire left hippocampus emerged through interventional fMRI (Bloechle et al., 2016) and interventional DTI data (Klein et al., 2019). However, identifying specific parcel-to-parcel disconnections from the left hippocampus, especially to the right parietal lobe, has been missing. Further, the current results imply several disconnections between the left SFG and hippocampal structures. Therefore, further studies may be necessary to examine a potential interplay between the SFG and the hippocampus in arithmetic fact encoding and retrieval. This

particular connection probably has a specific role in encoding arithmetic facts (Delazer et al., 2019) and needs to be added to the TNF for arithmetic fact encoding.

Finally, the findings from acute patients (Smaczny et al., 2023) were replicated for chronic patients in this study: Participants did not commit enough errors to conduct a meaningful analysis in addition and subtraction retrieval. This indirectly supports the assumption of the TNF that magnitude can be processed bilaterally. Since the magnitude network is supposed to be redundantly available in both hemispheres of the human brain, the contralesional hemisphere might be able to overtake processing during regular processing (Duffau, 2009). In contrast, our data align with the idea that facts are necessarily retrieved in a left-hemispheric network since a unilateral left-hemispheric lesion was sufficient to cause an arithmetic fact retrieval deficit. Yet, the TNF indicates that the arithmetic fact retrieval and the magnitude processing network are constantly competing. It is preferable to solve an arithmetic task with overlearned fact retrieval; only if this is not possible and cannot be compensated by nearby or homologue brain areas, we do switch to effortful procedure-based calculation. It can be assumed that procedure-based multiplication (e.g., $3 \times 4 = 4 + 4 + 4$) is far more difficult for patients than the procedure-based calculation of just $4 + 4$ or $4 - 3$, as multiplication additionally requires keeping intermediate results in working memory.

5. Conclusion

The current results provide evidence that a multiplication deficit in the chronic stage of stroke could reflect a (re-) learning deficit of arithmetic facts following hippocampal disconnection. Our data suggest that a disconnection to the hippocampus via the cingulum might prevent possible compensation processes for the case of multiplication facts. Therefore, a multiplication deficit in the chronic stage of stroke is most likely due to the inability to re-learn arithmetic facts when other compensatory mechanisms cannot be employed. Crucially, when comparing the stroke patients in the chronic phase presented here with those patients in the acute phases reported previously (Smaczny et al., 2023), different networks appear to be relevant to solve multiplication tasks. The acquisition and encoding of arithmetic facts (as indexed by persistent multiplication deficits in chronic stroke) seems to require the structural integrity of a network centered around the left (para)hippocampus. In contrast, retrieving consolidated arithmetic facts from memory (as indexed by multiplication deficits in acute stroke) seems to require the integrity of a left-hemispheric network involving the angular gyrus and middle temporal gyrus.

CRedit authorship contribution statement

S. Smaczny: Writing – review & editing, Writing – original draft, Visualization, Validation, Methodology, Investigation, Formal analysis, Data curation. **S. Jung:** Writing – review & editing, Writing – original draft, Methodology, Funding acquisition, Conceptualization. **K. Willmes:** Writing – review & editing, Writing – original draft, Resources,

Conceptualization. **H.-O. Karnath:** Writing – review & editing, Writing – original draft, Supervision, Resources, Methodology, Funding acquisition, Conceptualization. **E. Klein:** Writing – review & editing, Writing – original draft, Resources, Methodology, Funding acquisition, Conceptualization.

Ethics statement

The study was approved by the local ethics committee of the Medical Faculty of the RWTH Aachen University (Vote EK 094/07) and the ethics committee of the Kliniken Schmierer in Konstanz and followed the ethical standards in the Declaration of Helsinki (Version 2013).

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Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.cortex.2025.08.011>.

Scientific transparency statement

DATA: Some raw and processed data supporting this research are publicly available, while some are subject to restrictions: <https://doi.org/10.17632/g9fd55k94c.2>.

CODE: This research did not make use of any analysis code.
MATERIALS: No study materials supporting this research are publicly available.

DESIGN: This article reports, for all studies, how the author(s) determined all sample sizes, all data exclusions, all data inclusion and exclusion criteria, and whether inclusion and exclusion criteria were established prior to data analysis.

PRE-REGISTRATION: No part of the study procedures was pre-registered in a time-stamped, institutional registry prior to the research being conducted. No part of the analysis plans was pre-registered in a time-stamped, institutional registry prior to the research being conducted.

For full details, see the *Scientific Transparency Report* in the supplementary data to the online version of this article.

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