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The neuropsychology and neuroanatomy of reduplicative paramnesia

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ABSTRACT

Reduplicative paramnesia refers to the delusional belief that there are identical places in different locations. In this case–control study we investigated the clinical, phenomenological, neuropsychological and neuroanatomical data of eleven patients with reduplicative paramnesia and compared them against a control group of eleven patients with severe spatial disorientation without signs of reduplicative paramnesia.

We show that most patients with reduplicative paramnesia report that a current place is reduplicated and/or relocated to an other familiar place. Patients with reduplicative paramnesia show a higher prevalence of deficits in the executive functions compared to the control patients, while mnestic and visuo-spatial deficits were both frequent in patients with reduplicative paramnesia and the control group. Patients with reduplicative paramnesia mostly suffer from right hemispheric lesions with a maximal overlap in the dorsolateral prefrontal cortex. Using lesion network mapping we show that lesions causing reduplicative paramnesia are connected to bilateral anterior insula and the right cingulate cortex.

We argue that patients with reduplicative paramnesia fail to integrate the actual context with visuo-spatial memories and personal relevant emotional information due to a disruption of the neural network within the anterior temporal lobe, the cingulate cortex and the anterior insula. Also patients with reduplicative paramnesia are not able to resolve this conflict due to the lesion of the dorsolateral prefrontal cortex and executive dysfunction.

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1. Introduction

Reduplicative paramnesia (RP) is a delusional misidentification syndrome first described by Arnold Pick (1903). Three main forms of RP have been described. The majority of patients duplicate the hospital and its surroundings (i.e. their current location) and relocate it to another location (e.g. their hometown or another location where they have lived for a significant part of their life) (Darby & Prasad, 2016). Second, some patients relocate without duplication, reporting that a given place (usually the hospital) is not at its current location, but at another familiar location (e.g. the actual location of a hospital in Geneva is experienced to be located not in Geneva, but in the patient's hometown). Finally, some patients virtually merge the current place with another, personally more relevant one (e.g. that the hospital is contiguous with the patients home), implicitly requiring relocation as well (Politis & Loane, 2012; Alves et al., 2021). A striking aspect of RP is the patient's reluctance to admit the impossibility of their claim that the same place exists at two locations. This is often encountered despite the omnipresent physical evidence to the contrary, indicating that the patient is situated at the current location.

Whereas Pick associated RP with disorders of memory, and reported mild to moderate memory impairments in RP patients (Pick, 1903), others have questioned whether a memory disorder was either sufficient or necessary to induce RP (Benson et al., 1979; Luzzatti & Verga, 1996). Feinberg has highlighted additional cognitive deficits, such as impairments in visuo-spatial perception and spatial orientation (Feinberg & Roane, 2005), although the latter deficits were usually mild and general geographical knowledge and topographical representation were intact in most RP patients. Benson and colleagues (Benson et al., 1979) reported three cases of RP after head trauma and prolonged loss of consciousness. Besides retrograde amnesia for the period prior to the head trauma, neither patient suffered from mnestic deficits, despite their persistent RP. Accordingly, Benson (Benson & Stuss, 1990) proposed an influential cognitive model, suggesting that RP is related to the combination of a posterior visuo-spatial deficit leading to perceptual errors that cannot be resolved due to a co-existing frontal executive deficit (i.e. error-monitoring). Finally, based on results from lesion network mapping, it has been recently shown that delusional misidentification syndromes including RP arise from a functional network involved in both familiarity processing and expectation violation (Darby et al., 2017).

Although RP is frequent in neurodegenerative disease (Pick, 1903; Gerace & Blundo, 2013; Förstl et al., 1991; von Gunten et al., 2005) detailed investigations of the behavioral and neural mechanisms are challenging in advanced dementias. RP due to focal brain damage on the other hand is believed to be a rare condition, although it might be more frequent than previously thought (Alves et al., 2021). RP has been reported after traumatic brain injury (Benson et al., 1979; Hakim et al., 1988; Michel Pignat et al., 2013; Paterson & Zangwill, 1944), brain tumor (Nelson, 2017), or stroke (Alves et al., 2021; Hakim et al., 1988; Moser et al., 1998; Peckins et al., 2016), and the authors noted predominant involvement of the right hemisphere (Benson et al., 1979; Borghesani et al., 2019; Hakim et al., 1988), with damage affecting the frontal and posterior cortex (temporal, parietal lobe) (Benson & Stuss, 1990; Kapur et al., 1988; Moser et al., 1998). Recently, Alves and colleagues (Alves et al., 2021) performed a lesion analysis study in 64 patients with RP due to focal brain damage, describing a pattern of thalamo-frontal and right occipito-temporal structural disconnection and demonstrated that the duration of the symptoms is correlated to the degree of the structural decoupling of belief-associated functional networks (Alves et al., 2023). Conversely, Darby et al. (2017) showed in a smaller sample that the lesions causing delusional misidentification syndromes are functionally connected to the left retrosplenial cortex, a region involved in familiarity processing, and the right ventral prefrontal cortex, which has been linked to expectation violation. However, a detailed neuropsychological profile of their patient sample and the information on the exact nature of the phenomenology of RP is missing in both studies.

Here, we aimed to investigate the neuropsychological characteristics as well as the neuroanatomical correlates of patients with RP. We report data from eleven patients with RP following focal brain damage and investigated in detail the neurological, neuropsychological and neuroimaging correlates of RP, including lesion overlap analysis and lesion network mapping as compared to a control group with spatial disorientation without RP (Blondiaux et al., 2021; Heydrich & Blanke, 2013; Ionta et al., 2011). Our data shows that RP is linked to lesions in the right dorsolateral prefrontal cortex, and that lesions causing RP are connected to the bilateral anterior insula and right middle cingulate cortex. We further report a distinct neuropsychological profile in patients with RP characterized by severe executive deficits, while mnestic and visuo-spatial deficits were equally frequent in patients with RP and the control group.

2. Materials and methods

2.1. Patients

In this case–control study we selected patients presenting with RP between 2001 and 2019 seen at the inpatient and outpatient clinics of both the University Hospital of Geneva and the University Hospital of Bern. We report how we determined our sample size, all data exclusions, all inclusion/exclusion criteria, whether inclusion/exclusion criteria were established prior to data analysis, all manipulations, and all

measures in the study. RP was defined as the delusional belief that a given place (usually the hospital) has been relocated (with or without reduplication) or eventually virtually merged with another place (Politis & Loane, 2012). As a control group, we selected patients presenting with spatial disorientation without RP. Based on previous work showing that a sample size of already nine subjects per group can yield significant results in terms of lesion analysis (Heydrich & Blanke, 2013; Ionta et al., 2011) and that RP is a rare condition, we determined a sample size of a 10 patients per group to be sufficient. Patients were only included if they could be unequivocally assigned to one of the above-mentioned two groups and suffered from a well-defined structural brain lesion (e.g. cerebrovascular stroke, tumor, intracerebral bleeding, abscess). Patients with dementia and psychiatric illness were excluded from the analysis. Inclusion and exclusion criteria were established prior to data analysis. Selection of patients was based on the detailed clinical records taken at the time of evaluation. Two of the patients have previously also been included in separate case reports (Michel Pignat et al., 2013; Nyffeler et al., 2005). Ethical approval was obtained from the Ethics Committee at the University Hospital of Geneva and the Cantonal Ethics Committee of Bern. The study procedures were not pre-registered prior to the research being conducted.

Patient characteristics (age, sex, handedness, neurological examination, neuropsychological examination) were compared between the two groups.

2.2. Neuropsychological examination

Most of the patients underwent detailed neuropsychological examinations, including assessment of verbal and visuo-spatial long-term memory, attention, executive functions visuo-construction and visuo-spatial perception by means of standardised tests. Most commonly used was a battery with norms for the Swiss population (Balzer et al., 2011) including tests of memory, executive functions and visuo-construction. In some patients, the CERAD (Aebi & Bern, 2002) or the Hopkins Verbal Learning Test (Benedict et al., 2010) were administered. Attention was commonly assessed by use of the computerised Test for Attentional Performance (TAP) (Zimmermann & Fimm, 1995). Legal copyright restrictions prevent public archiving of these tests which can be obtained from the copyright holders in the cited references. Based on these assessments and on other clinical records and reports, performance levels were collated and scored for each patient and domain separately (see Table 2).

Results of the neuropsychological evaluation were analyzed using a chi-square test for independent samples, or the Fisher's exact test, respectively, if the expected frequencies were <1. The *p*-value was adjusted for multiple comparisons using the Holmes-Bonferroni method. The conditions of our ethics approval do not permit public archiving of study data, that can not be fully anonymized (e.g. neuropsychological tests). Readers seeking access to the data should contact the corresponding author or the local ethics committee at the University of Bern. Access will be granted to named individuals in accordance with ethical procedures governing the reuse of sensitive data.

2.3. Lesion analysis

All imaging data was reviewed for the purpose of this study by two of the authors (LH and AD) in order to determine the lesion location. For lesion overlap and statistical analysis, we used MRIcron and Non Parametric Mapping (NPM), which is part of the MRIcron software package (Rorden et al., 2007). Anatomical structures were labelled according to the AAL atlas implemented in MRIcron (http://www.mccauslandcenter.sc.edu/mricro/mricron).

In order to illustrate the neural correlates underlying RP, we subsequently traced the lesion for each patient on the T1 template using MRIcron. Structural lesions were identified by MRI or CT. MRI brain scans were normalized to the smoothed T1 template using SPM5 (http://www.fil.ion.ucl.ac.uk/spm/software/spm5) (Ashburner & Friston, 2005). Since unified segmentation models give the most precise registration of lesioned structural images (Crinion et al., 2007), no cost-function masking was necessary. If only a CT scan was available, lesions were traced manually slice by slice on the T1 weighted images using MRIcron. The later manual tracing on the template brain was only done when confidence could be achieved for matching corresponding slices between the lesioned brain and the template brain. No patients with unclear lesion boundaries or metallic artefacts were included in the analysis. Lesion volumes (volume of interest, VOI) were determined as the sum of all voxels compromising the traced lesion in all slices and were spatially smoothed using a 3 mm full width at half maximum (FWHM) Gaussian Kernel and a threshold of .5. The same procedure was applied to the control group.

We also carried out statistical lesion overlap comparison by contrasting the lesions of the RP patients with those from the control group using voxel-based lesion symptom mapping (VLSM) (Bates et al., 2003). We used the Liebermeister test and corrected the results for multiple comparisons using a 5% false discovery rate (FDR). The Liebermeister test is a nonparametric implementation of a two-group comparison on a binary variable. It is more appropriate than the chi square test (Rorden et al., 2007). We only included voxels affected in at least 30% of the patients for all subsequent analyses.

2.4. Lesion network mapping analysis

In addition to the lesion analysis, we applied lesion network mapping analysis, a method developed by Boes and colleagues (Boes et al., 2015) that enabled us to investigate the network associated with RP without the need of functional imaging data from patients. This method is very useful when the symptom is thought to arise from a network rather than from a single region (Fox, 2018). To this aim, the RP lesions were used as seed region of interest (ROI) in a resting state analysis with data from 126 healthy subjects of the Enhanced Nathan Kline Institute Rockland Sample (Nooner et al., 2012) (http://fcon_1000.projects.nitrc.org/indi/enhanced/download.html) as described in Blondiaux et al. (2021)

The lesions masks were used as seed ROIs, and their mean time course was extracted and correlated to all other brain voxels using the Functional Connectivity (CONN) toolbox

(v.18. a, http://www.nitrc.org/projects/conn) (Whitfield-Gabrieli & Conn, 2012). The whole brain analysis was restricted to the grey matter voxels. For this analysis, we excluded the lesion of patient 1 since it was covering most of the brain. Each remaining lesion-seed yielded a brain network thresholded at $t > \pm 4.25$ with $p < .00005$ peak-level uncorrected (Boes et al., 2015; Darby et al., 2017). The ten networks were then binarized and overlapped to determine the regions of shared positive and negative correlations. The network overlap was thresholded at 90% (at least 9 cases out of 10) with a minimal cluster extent of 10 voxels. This procedure was repeated with the eleven lesions of the control group.

Next, in order to determine the regions specific to RP, we compared the RP-derived network with the control-derived network using VLSM and the Liebermeister test. These steps of the analysis were performed using the GUI of the SPM toolbox and the NPM toolbox.

The analysis was performed within the 90% RP- network overlap in order to assess its specificity compared to the control group with spatial disorientation. Only voxels showing 30% of overlap across the RP patients (at least six patients) were considered and voxels were considered significant according to FWE correction (with 4000 permutations, only clusters larger than 10 voxels and in the grey matter are reported). Since the Liebermeister test is a binary test indicating whether or not the voxel is connected to the network, the positive and negative maps were analyzed separately.

The study analyses were not pre-registered prior to the research being conducted.

3. Data availability

All data is available under https://osf.io/xuyjw/?view_only=2b08c41da0954cdd9a84f319369894ce (lesion data set) and https://osf.io/ng82u/?view_only=85501d5fd3a34e7180f2409d661a18d2 (program code).

4. Results

4.1. Description of the patient sample

4.1.1. Reduplicative paramnesia

For illustration, characteristic phenomenological, neurological, neuropsychological and neuroradiological findings are described for five RP patients (for the remaining seven patients, please refer to the online Supplementary Material).

4.2. Case reports

4.2.1. Patient 1

Patient 1 was a 35-year-old carpenter who suffered a severe traumatic brain injury with prolonged loss of consciousness after a motorbike accident. The CT scan showed a dislocated skull fracture with bilateral frontal hemorrhagic contusions, a right subdural hematoma and a right frontal subarachnoid hemorrhage (SAH). Additionally, there were multiple facial and limb fractures. After multiple surgical interventions, including a bifrontal external decompression, the patient was admitted to the intensive care unit (ICU) and afterwards to the neurological rehabilitation unit. The neuropsychological examination revealed deficits in the executive functions (impulse control and perseveration), attention and mild deficits in the mnestic functions. The neurological exam was normal.

RP: During testing, Patient 1 claimed that certain units of the Bern University hospital had been relocated (but not duplicated) from Bern to the city of Visp, near his hometown (approx. 100 km; Fig. 1). Patient 1 stated that this relocation was due to construction work at the site of the hospital (during hospitalization, major renovations were carried out at the Bern rehabilitation unit). However, he also claimed he was being transferred back to Bern for each of his surgical interventions, after which he was brought back to the hospital in Visp for rehabilitation. He said that being in Visp was quite helpful because it was close to his workplace (only a few

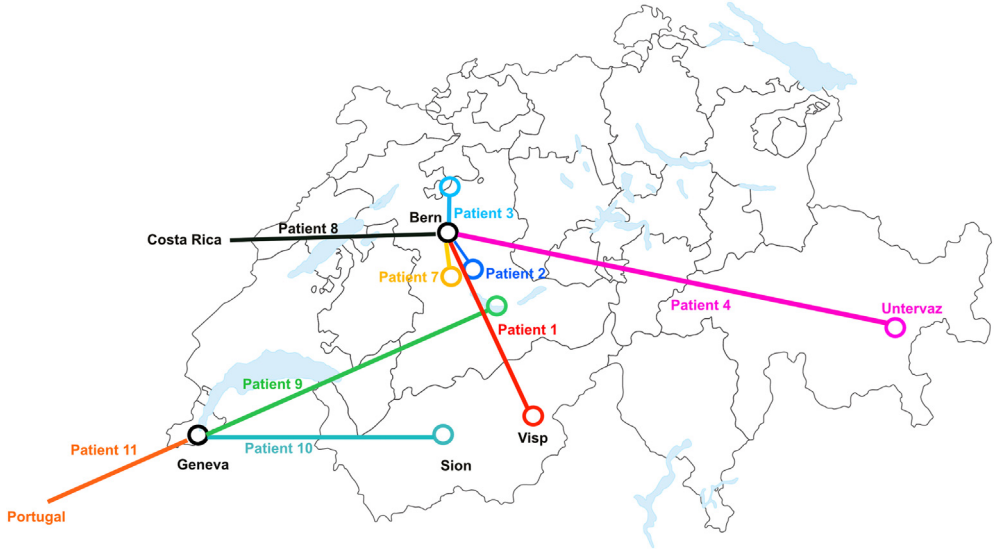

Fig. 1 – *Map*. Map of Switzerland indicating the relocation/duplication of a current place (Bern and Geneva, respectively) to another, usually more familiar place for Patients 1–4 and 7–11.

minutes walking distance). RP persisted for his entire 6-week stay at the rehabilitation unit.

4.2.2. *Patient 4*

Patient 4 was a 77-year-old retired precision mechanic who suffered from a right frontal tumor. The CT scan demonstrated an infiltration of the corpus callosum and the frontal horn of the right lateral ventricle and a perifocal vasogenic edema. A metastasis was postulated as the patient had a history of prostate cancer and melanoma of the lower extremity. Palliative care was implemented. The neuropsychological examination showed mild deficits in attention, executive deficits (perseveration, intrusions) and visuo-constructive deficits. The neurological examination showed a discrete left-sided sensorimotor deficit.

RP: On the day prior to his admission to the Bern University Hospital, the patient called a friend in Untervaz where he used to live years ago and asked them to visit him. After the friend pointed out the long distance (Untervaz is approx. 237 km away from Bern, see Fig. 1), it turned out that Patient 4 was convinced that he himself was currently in Untervaz, too. The friend told the patient to go to the emergency department. Only then did he realize that he was in Bern and the University Hospital was only a short drive away. However, he insisted there must be another Untervaz in Bern, and stated that there are many places with the name Untervaz. He also claimed that a branch of the University Hospital's emergency unit was operating in the area near Untervaz. RP persisted during his whole 2-2-week stay at the hospital.

4.2.3. *Patient 6*

Patient 6 was a 89-year-old retired architect who was referred to the outpatient clinic by his treating physician for neurological evaluation. According to his daughter, he was disorientated and showed mild mnestic (verbal and non-verbal) deficits. MRI demonstrated a subacute ischemia in the territory of the right posterior cerebral artery (occipito-temporal lobe, posterior mesial temporal lobe). At the time of consultation, he was fully oriented and knew he was in Bern. The neurological exam showed a left homonymous temporal quadrantanopia and was otherwise normal. Neuropsychological testing showed marked executive deficits and mild mnestic deficits.

RP: The patient had the strong feeling that his house and also the village where he lived (Worb) had been duplicated and that his house existedat several places at the same time. As a result, he wanted to sue his architect for having sold the original plans of his home. This belief was first noted four months earlier and has persisted ever since.

4.2.4. *Patient 9*

Patient 9 was a 66-year-old retired printer who suffered from an ischemic stroke in the territory of the right posterior and bilateral middle cerebral artery. The neuropsychological examination demonstrated severe executive deficits (impulse control, cognitive flexibility), anosognosia, aphasia and verbal as well as non-verbal memory deficits. The neurological examination showed dysarthria, dysphagia and right facial weakness. Due to aspiration, the patient had to be intubated temporarily and was transferred to the ICU.

RP: The patient was hospitalized at Geneva University Hospital. After transfer to the rehabilitation unit, the patient addressed the physician as "train conductor". Asked why he believed the physician was a "train conductor," he stated that he was in an overnight sleeper cabin that was going back and forth between Geneva (where he currently lived with his family) and his hometown of Spiez at the Lake of Thun (where he grew up, 188 km from Geneva). He indicated that in Spiez, a replica of the University Hospital of Geneva had been built. RP persisted during his 6-week stay at the rehabilitation unit.

4.2.5. *Patient 10*

Patient 10 was a 53-year-old teacher who suffered from a dissection and occlusion of the distal right internal carotid artery and right middle cerebral artery. Despite immediate mechanic thrombectomy and stenting of the right internal carotid artery, re-occlusion occurred. Imaging (CT, MRI) showed infarction of the territory of the right middle and anterior cerebral artery with secondary hemorrhagic transformation. Neuropsychological examination demonstrated severe left-sided visuo-spatial neglect, anosognosia as well as attentional, mnestic and executive deficits. The neurological exam showed dysarthria, left-sided facio-brachio-crural hemiparesis and left homonymous hemianopia.

RP: Patient 10 was hospitalized in the neurology unit of Geneva University Hospital, but claimed that he was in the Hospital of Sion (about 150 km from Geneva). On other occasions, he claimed that he was in his holiday apartment in Vex (a smaller village close to Sion). Patient 10 could not be convinced he was at a hospital in Geneva, even when looking out of the hospital window with his wife, who showed him several landmarks of Geneva (for example the mountain "le Salève"). He also specially thanked his friends from Geneva, who had come to visit him, because he believed they travelled all the way from Geneva to Sion. He sometimes asked his wife to get their daughter to join them because he believed she was just in the next room (of his holiday apartment, which he experienced as contiguous with the hospital room). He also asked his wife if she had recently seen a certain neighbor from Vex. After being transferred from the neurology to the neurorehabilitation unit, a building right next to the University Hospital that he knew well from before the accident, his RP stopped (duration 1 week). However, the patient remained puzzled that the transfer from Sion to Geneva had only taken a few minutes.

4.3. *Summary of the phenomenology*

In the present study, RP involved the hospital in eight patients (72%) and the home or holiday apartment in three patients.

Concerning duplication-relocation, five patients (45%) claimed that the current place (mostly hospital) was duplicated and relocated to another place (e.g. claiming there must be a branch of the University Hospital, e.g. patients 2,3,4), and five patients (45%) stated that the current place was relocated without duplication (e.g. patients 10,11). One patient felt his home was duplicated multiple times (patient 6). Two patients claimed the current place was contiguous with another place (patients 5, 10). Importantly, the place of relocation or the object of reduplication (patient 6) respectively was always of

Table 1 – Demographic characteristics and clinical details.

	Reduplicative Paramnesia ($n = 11$)	Control group ($n = 11$)	*P*-value
Demographics			
Gender (Male/Female)	10/1	11/0	n.s.
Age at evaluation (mean years ± SD)	63.5 ± 17.3	59.0 ± 9.3	n.s.
Type of Lesion (n)			n.s.
Ischemic	4	7	n.s.
Traumatic Brain Injury/ ruptured aneurysm	5	2	n.s.
Malignancy	2	0	n.s.
Encephalitis	0	2	n.s.
Location of the lesion (n)			
Right/Left/both hemisphere(s)	8/0/3	7/0/4	
Neurological Examination			
Aphasia	2	2	n.s.
Neglect	2	3	n.s.
Hemianopsia	2	7	n.s.
Sensorimotor hemi syndrome	6	4	n.s.
Loss of consciousness	8	2	$P = .01$

personal relevance to the patient (e.g. a place where they lived for a long period, place of origin, holiday apartment).

None of our patients showed other misidentification syndromes like Capgras or Fregioli syndrome. The average duration of RP was 40 days (± 35 days).

4.3.1. *Demographics and clinical characteristics*

Eleven patients fulfilled the criteria for RP and were selected for this study. Eleven patients with spatial and topographical disorientation constituted the control group. Prolonged loss of consciousness (>1 day) preceding RP was reported in eight patients, while only two patients from the control group had a prolonged loss of consciousness (72% vs. 18%; p = .01). Results from the clinical neurological exam (aphasia, sensorimotor deficits, neglect and visual field deficits), as well as patient characteristics and between-group comparisons are detailed in Tables 1 and 2. Demographic parameters did not significantly differ between the two groups (all p > .05, see Table 1).

4.3.1.1. Neuropsychology. Standardized neuropsychological testing was available in 21 out of the 22 patients (Ten patients with RP and eleven patients from the control group). Executive functions including impulse control, mental flexibility, error detection and correction as well as planning were moderately to severely impaired in ten patients reporting RP (100%), while only five patients of the control group showed moderate to severe executive deficits (45%; p = .01). Deficits regarding verbal and non-verbal memory, attention and visuo-constriction were frequent both in patients with RP and the control group (no significant difference between the two groups). A detailed description of the neuropsychological profile of both groups can be found in Table 2).

4.3.1.2. Lesion overlap analysis. All patients with RP either suffered from a right hemispheric (N = 8) or bilateral (N = 3) lesion. Figs. 2 and 3 show the results of the voxel-based overlap analysis. In the RP group, the right dorsolateral prefrontal cortex (DLPC) was found to be the region of maximal overlap (MNI coordinates x = 37 y = 35 z = 27; Brodman area 46, right dorsolateral prefrontal cortex) (Fig. 2). This region was found to be lesioned in seven out of the eleven RP patients. The second overlap region (in 6 out of 11 RP patients) was found in the right anterior temporal lobe (MNI coordinates x = 46 y = 9 z = −22; Brodman area 38, right anterior temporal lobe; see Fig. 2).

Four patients from the control group had a bilateral lesion, while one patient had a left hemispheric and six patients had a right hemispheric lesion. Lesion overlap analysis of the control group highlighted the right fusiform gyrus and adjacent structures (maximal overlap in the right hippocampus/posterior MTL) in 6 out of 11 control patients (MNI coordinates x = 30, y = −61, z = −4, Brodmann 37; fusiform gyrus, extending towards parahippocampal, posterior hippocampal and lingual gyrus) (Fig. 3).

VLSM, statistically contrasting the lesions of the RP patients with those from the control group, showed a specific involvement of the right dorsolateral prefrontal cortex in patients with RP (MNI coordinates x = 37 y = 35 z = 27; Brodman area 46; Z-score = 3.20; $p < .01$, FDR-corrected, see Fig. 4).

4.3.1.3. Lesion network mapping analysis. Next, we applied lesion network mapping analysis to identify the brain regions functionally connected to most of the RP lesion locations. We found that most of the lesions (90%) causing RP were functionally connected to bilateral insula, bilateral anterior/middle cingulate cortex, right precuneus, supplemental motor area and left precentral gyrus, superior temporal pole and parahippocampal gyrus (Supplementary Table 1; Fig. 5). All the lesions causing RP were functionally connected to the bilateral inferior/middle frontal gyrus (IFG) (mostly on the right), right middle/superior temporal cortex and right parahippocampal gyrus (Supplementary Table 1). No negative correlations were found with lesions causing RP.

When comparing the RP network maps with the network maps of the control group, the functional connections to bilateral insula and to the right anterior/middle cingulate cortex were found to be specific to lesions causing RP compared to the lesions of the control patients (Supplementary Table 2; Fig. 6).

5. Discussion

Here we demonstrate the clinical, neuropsychological and neuroanatomical correlates of eleven patients with reduplicative paramnesia (RP). The majority of patients reduplicated and/or relocated the current place to another familiar place closer to their home or where they used to live for a significant period of time. The location of the duplicated place was always of emotional value for the patient. Neuropsychological examination showed moderate to severe executive deficits in the majority of patients with RP, while mnestic and attentional deficits were frequent in both patients with RP and patients from the control group. Lesion analysis showed an involvement of the right hemisphere in all eleven patients

Table 2 – Neuropsychological profile of patients with RP and the control group.

		Age	Sex	Hand	Lesion Type	Lesion Side	Confabulations	Memory verbal	Memory visuo-spatial	Attention	Executive functions	Visuo-construction	Visual perception/ visual field	Neglect	Language	Sensorimotor
RP	1	35	m	r	TBI	bilateral	–	–	–	+	+++	–	–	–	–	–
RP	2	43	m	r	Tumour	right	–	++	+	–	+++	–	–	–	–	–
RP	3	70	m	r	TBI	right	–	++	+++	+++	+++	++	–	–	–	–
RP	4	77	m	r	Tumour	right	+	+	n/a	++	+++	+	–	–	–	+
RP	5	84	f	r	Stroke (isch.)	right	–	n/a	n/a	n/a	n/a	n/a	left	–	–	–
RP	6	92	m	r	Stroke (isch.)	bilateral	–	++	++	n/a	+++	n/a	n/a	–	–	–
RP	7	68	m	r	Stroke (RA)	right	+	+++	n/a	+++	+++	n/a	hall.	left	–	+
RP	8	56	m	r	TBI	right	+	++	+++	+	++	++	–	–	–	+
RP	9	67	m	r	Stroke (isch.)	bilateral	–	+++	n/a	+++	+++	++	–	–	+	+
RP	10	53	m	r	Stroke (isch.)	right	–	++	n/a	+++	+++	+++	left	left	–	+
RP	11	54	m	r	TBI	right	+	+++	+++	+++	+++	–	–	–	+	+
TA	1	36	m	r	TBI	right	+	+++	+++	+++	+++	–	–	–	–	+
TA	2	64	m	r	Stroke	right	–	+	+++	++	+	++	left	left	–	+
TA	3	62	m	r	Encephalitis	bilateral	+ (prov)	+++	+++	+	+	–	–	–	–	–
TA	4	59	m	r	Stroke (isch.)	right	–	–	++	–	–	–	left (UQ)	–	–	–
TA	5	57	m	r	Stroke (isch.)	bilateral	–	–	+++	+++	–	+	left	left	–	–
TA	6	65	m	r	Stroke (isch.)	bilateral	+ (prov)	+++	–	++	++	++	–	–	++	+
TA	7	54	m	r	Encephalitis	right	–	+	+++	++	+	++	left (LQ)	–	–	–
TA	8	65	m	r	Stroke (isch.)	right	–	+	+++	++	++	+++	left	left	–	+
TA	9	68	m	l	Stroke (isch.)	left	–	–	+++	–	++	+	right	–	–	–
TA	10	52	m	r	Stroke (isch.)	bilateral	–	++	++	+++	++	+	–	–	++	–
TA	11	67	m	r	Stroke (isch.)	right	–	–	+++	–	–	–	left (UQ)	–	–	–

Scoring: – = no impairment described, + = mild impairment (up to 1.5 standard deviations (SD) below norm), ++ = moderate impairment (between 1.5 and 2.5 SD below norm), +++ = severe impairment (more than 2.5 SD below norm), n/a = no description available.
Abbreviations: hall. = hallucinations, isch. = ischemic, LQ/UQ = lower/upper quadrant, prov. = provoked, RA = ruptured aneurysm, RP = reduplicative paramnesia, TA = topographagnosia, TBI = traumatic brain injury.

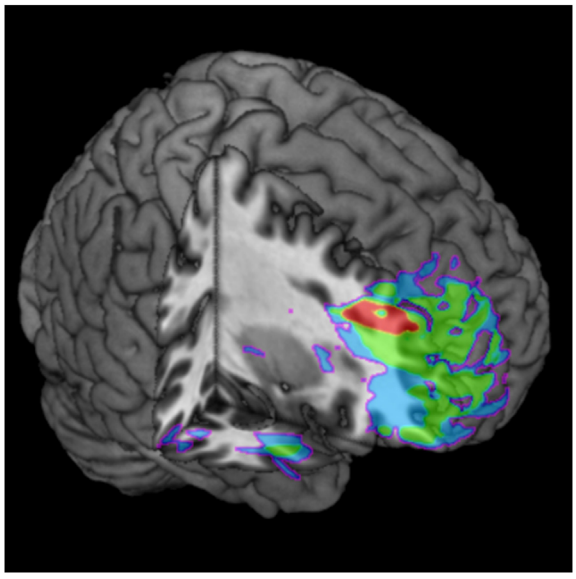

Fig. 2 – *Lesion overlap reduplicative paramnesia*. Maximal overlap in patients with reduplicative paramnesia in the right dorsolateral prefrontal cortex and the right anterior temporal lobe. The degree of overlap is indicated by change of color from blue ($n = 5$) to red (maximal overlap, $n = 7$).

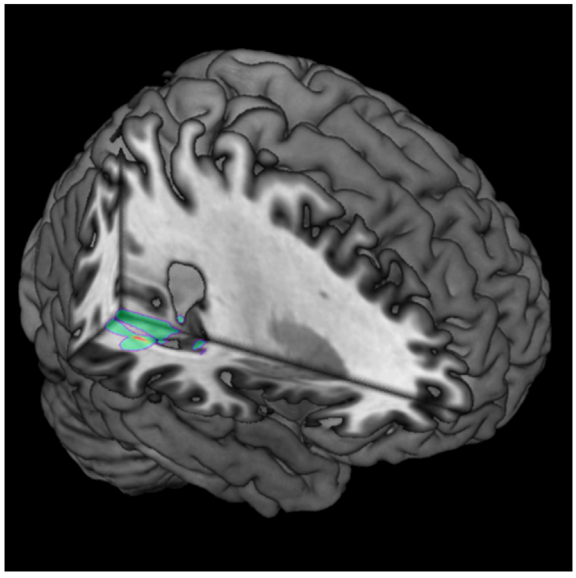

Fig. 3 – *Lesion overlap control group*. Maximal overlap in patients with spatial and topographical disorientation (control group) in the fusiform gyrus in 6 out of 11 patients (MNI coordinates $x = 30$, $y = -61$, $z = -4$, Brodmann 37; extending towards parahippocampal, posterior hippocampal and lingual gyrus).

with RP and two regions with maximal lesion overlap in frontal (DLPC) and in temporal cortex (anterior temporal lobe), of which only the right DLPC was specific to RP (compared to control patients). Lesion network mapping showed that all of the RP lesions were connected with the bilateral inferior/middle frontal gyrus (IFG) (mostly on the right), right middle/

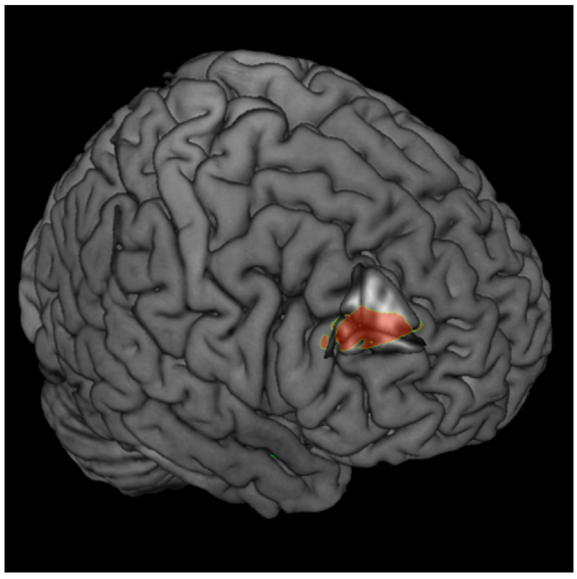

Fig. 4 – *Voxel-based lesion symptom mapping*. Voxel-based lesion symptom mapping in reduplicative paramnesia yielded maximal involvement of the right dorsolateral prefrontal cortex in patients with reduplicative paramnesia (MNI coordinates $x = 37$ $y = 35$ $z = 27$; Brodman area 46; Z-score = 3.20; $p < .01$, FDR-corrected) as compared with the control group. Only significant voxels are displayed.

superior temporal cortex and right parahippocampal gyrus. Most of the lesions associated with RP were functionally connected to bilateral insula, bilateral anterior/middle cingulate cortex, right precuneus, supplemental motor area and left precentral gyrus, superior temporal pole and parahippocampal gyrus. Importantly, connectivity to bilateral insula and right middle cingulate cortex was specific to lesions associated with RP compared to the lesions of the control group.

5.1. *Phenomenological characteristics of RP*

Most patients reduplicated and/or relocated the current place to a place familiar (e.g. place of residence, hometown), usually claiming there must be another branch of the hospital. Thus, reduplicative paramnesia was never just random, but always involved a place familiar and relevant to the patient. Importantly, all patients adapted their behavior according to their belief system (e.g. speaking Spanish to Swiss German hospital staff, judging the distance between the actual position and another place incorrectly, suing the architect for having sold the plans of their home, leaving the hospital bed to go to the kitchen), underlining the delusional aspect of RP. The choice of a familiar location outside the hospital is also in line with the hypothesis by Turnbull et al. (Turnbull et al., 2004; Turnbull & Salas, 2017) who suggested a systematic affective bias towards more positive and pleasant emotions in patients with confabulations. Moreover, despite being presented with facts contradicting the delusional belief (e.g. being shown famous landmarks of the current place, confronted with the

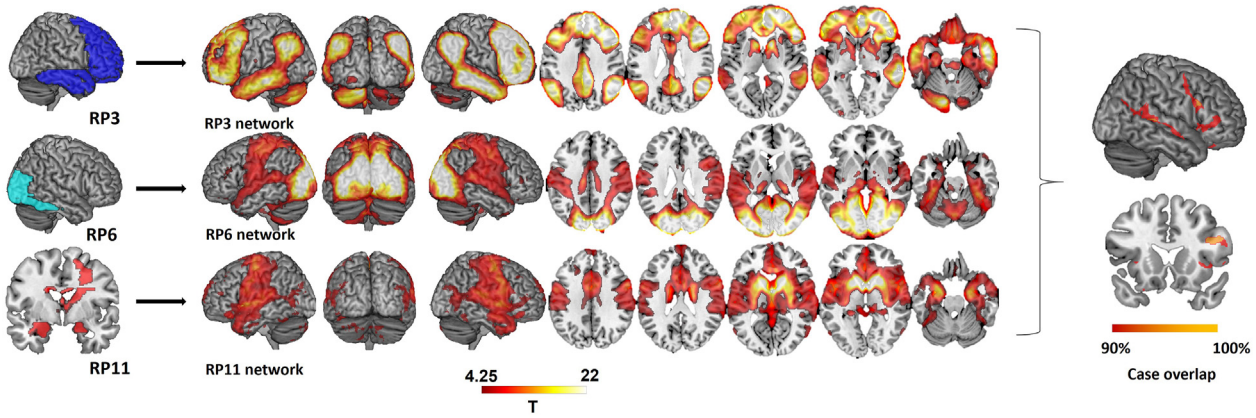

Fig. 5 – Reduplicative paramnesia network mapping analysis. For each brain lesion, the resting state connectivity network was computed in a healthy subject database. Those networks were thresholded, binarized and then overlapped together to localize the common brain regions associated with RP (on the right).

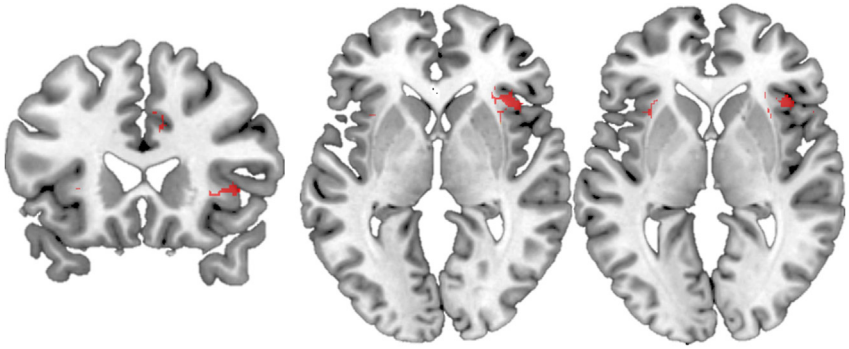

Fig. 6 – Results from the Liebermeister test. RP was specifically connected with bilateral insula and the right anterior/middle cingulate cortex compared to the control group.

fact of being in hospital), patients were not able to adapt their belief system. This behavior confirms previous reports, e.g. the patient described by Paterson & Zangwill (1944), who not only claimed that the English Grimsby and Scotland were the same, but even tried to get out of his room in order to walk home ("only a 5 min walk"). Another approach to delusional misidentification syndromes suggests it is loss of the sense of uniqueness, which allows the existence of various doubles, like in the case of Capgras syndrome (Margariti & Kontaxakis, 2006). Similarly to RP, patients with Capgras reject counter-evidence with confabulations.

5.2. *The neuropsychology of reduplicative paramnesia*

In line with this inability to correctly integrate information of the external world with their own belief system, we found that patients with RP suffered significantly more often from severe executive deficits affecting impulse control, mental flexibility, error detection and correction as well as planning compared to patients who showed spatial disorientation without RP. It is widely postulated that impairments in executive functions are at the core of confabulations but are not however sufficient to elicit them (Turnbull et al., 2004). This also supports the notion of Benson et al. (Benson & Stuss, 1990) that delusions such as RP are linked to executive deficits, making it difficult to correctly monitor and adapt to reality. A recent review by Darby & Prasad (2016) on delusional misidentification syndromes (including RP) also found that predominantly executive functions, but also memory and visuospatial processing were affected in these patients. Borghesani et al. (2019) analysed 51 patients with RP in a systematic review. They found attentional skills, especially attentional shifting, but also abstraction, planning, working memory and problem solving to be most frequently affected. Moreover, they report common deficits in mnestic functions, especially in the non-verbal domain.

In our study, all the patients with RP who were systematically tested showed some sort of memory disorder. Mild memory deficits were already described by Pick in his original description, leading to the name of the condition and further confirmed by Luzzatti and Verga (1996). Paterson's patient was also discharged with a slight deficit in short-term memory, and recently Nelson reported a case of RP with visuo-spatial memory disorder (Nelson, 2017). Others have questioned whether a memory disorder was either sufficient or necessary to induce RP. This is in line with our data, showing that mild memory deficits were present both in the group with RP and the control group. Thus, memory impairment alone cannot account forRP.

Benson and colleagues (Benson et al., 1979) have highlighted the importance of an interruption of awake

consciousness in combination with a memory disorder. In our sample, 8 out of 11 patients with RP had a history of prolonged loss of consciousness (e.g. severe traumatic brain injury, anaesthesia/intubation) while loss of consciousness was reported in only 2 patients from the control group. Although disorientation and confabulation are frequently seen in patients waking from coma, the crucial difference is that patients with RP, unlike the vast majority of patients with disorientation, reject the explanation given to them by their relatives or the hospital staff. On the contrary, they maintain their delusional belief system, trying to rationalize their behavior by claiming the actual place has either changed names or been relocated.

Visuo-spatial disorders are believed to be one of the most prominent features of RP (Benson et al., 1979; Luzzatti & Verga, 1996; Paterson & Zangwill, 1944). Thus, signs of impaired visuo-spatial perception and (usually mild) spatial memory deficits are often associated with RP (Feinberg & Roane, 2005). Although visuo-spatial disorders (visuo-spatial memory, visuo-construction) were frequent in patients with RP, we found this not to be specific for RP, as patients with spatial disorientation without RP were equally showing visuo-spatial disorders.

Thus, we argue that RP results from an incorrect integration of the patient's own belief (where I think I am) with the actual surroundings (what the world tells me about where I am), due to severe executive deficits such as faulty belief evaluation (Benson & Stuss, 1990) and cannot be simply considered a disorder of memory or visuo-spatial processing.

5.3. *The neuroanatomical correlates of reduplicative paramnesia*

Lesion overlap highlights the right DLPFC and the anterior temporal lobe as the regions most frequently affected in RP.

The right DLPFC has been repeatedly linked to executive functioning (Benson & Stuss, 1990; Funahashi & Andreau, 2013). Importantly, it has been shown to play an important role in conflict-induced behavioral adjustment, e.g. when a person has to decide what to do when confronted with conflicting facts (Boschin, Brkic, Simons, & Buckley, 2017; Mansouri et al., 2007). The right dorsal frontal cortex has also been linked to belief evaluation and prediction error (Corlett, Aitken, & Dickinson, 2004) as well as context-dependent retrieval of episodic memory (Henson, Shallice, & Dolan, 1999). Thus, the DLPFC is involved in the processes of reconstructing past events and autobiographical memory (Cabeza & St Jacques, 2007) in order to form a coherent and stable sense of self and identity (Schnell et al., 2007).

The temporal pole is a paralimbic region involved in high-level semantic representation and processing (Olson, Plotzker, & Ezzyat, 2007; Lambon Ralph, Jefferies, Patterson, & Rogers,

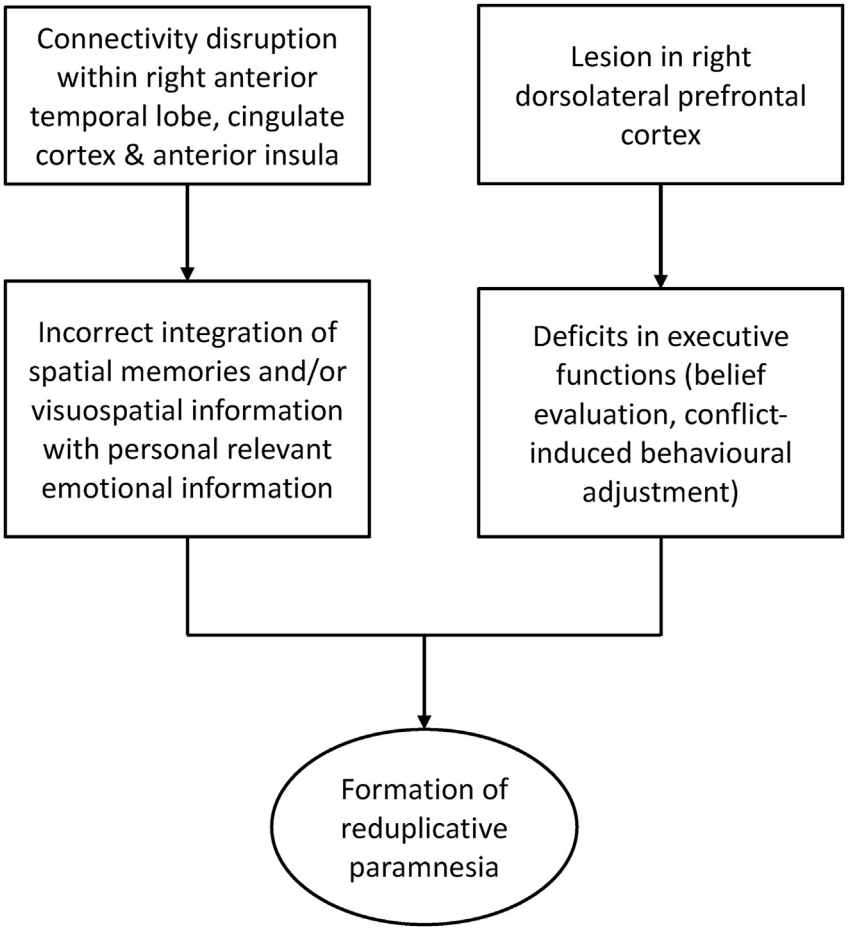

Fig. 7 – Two-hit hypothesis for the formation of delusional beliefs in RP.

2016). The uncinate fasciculus provides a connection to the (orbito-) frontal cortex, allowing mnemonic representations stored in the temporal pole to influence decision making in the frontal lobe. It has also been suggested that the temporal pole is a convergence zone where memories and concepts that are stored in the ventral anterior temporal lobe are imbued with personal meaning and significance (Olson, Plotzker, & Ezzyat, 2007). Interestingly, Pick already suggested that a disturbance of the so-called "Bekanntsheitsgefühl" (sense of familiarity) causes RP (Pick, 1903).

Moreover, lesion network analysis demonstrated that connectivity to bilateral insula and right cingulate cortex was specific to lesions associated with RP compared to the lesions of the control group. This is in line with findings from Darby et al. (2017), showing that lesions causing delusional misidentification syndromes (including reduplicative paramnesia, but also Capgras syndrome, Fregioli syndrome), are connected to the bilateral insula and the right ventral frontal cortex. The right anterior/middle cingulate cortex is a region implicated in attentional and emotional processing (Devinsky et al., 1995). Like the insula, it is believed to be a multisensory area integrating visceral, attentional and emotional information concerning self-regulation and error detection (Bush et al., 2000; Klein et al., 2013), as well as self-awareness (Lou et al., 2017). Interestingly, both the anterior cingulate cortex and the insula have been linked to schizophrenia (Benes, 1993; Costain et al., 2010), a clinical condition characterized by poor error-monitoring (Alain et al., 2002).

Thus, the implication of the anterior cingulate cortex and the insula in patients with RP could reflect the inability to correctly integrate personally relevant emotional information during the process of error-monitoring.

A limitation of the study is the relatively small sample size due to the rare occurrence of RP, making generalization of the results difficult. Moreover, it would be preferable to use a prospective design in order to assess the patients even more systematically (neuropsychological evaluation, taking of history of the phenomenology, standard imaging protocol). This would allow an increase in the number of patients investigated (e.g. Alves et al. identified 64 patients with RP out of 400 patients screened (Alves et al., 2021)) and a better understanding of other factors contributing to RP, given that many patients presenting with a similar lesion pattern do not show symptoms of RP.

In conclusion, we argue that patients with RP fail to update their belief system about their current location with visuo-spatial memories and with personally relevant emotional information due to a disruption of the network within the anterior temporal lobe, the cingulate cortex and the anterior insula. Moreover, they are no longer able to solve this conflict due to the lesion of the DLPC and executive dysfunction, respectively (e.g. conflict-induced behavioral adjustment (Boschin, Brkic, Simons, & Buckley, 2017; Mansouri et al., 2007), belief evaluation (Corlett, Aitken, & Dickinson, 2004), see Fig. 7. This is in line with the model that delusions can be conceptualized as a 'two-hit' process, with (a) an abnormal perception leading to the specific content for a given delusion (e.g. incorrect integration of spatial memories with personal significance); and (b) impaired belief evaluation, allowing the abnormal delusional belief to form (Benson & Stuss, 1990; Coltheart, 2010; Corlett et al., 2010).

Contribution (CRediT author statement)

A. D. Formal analysis, Investigation, Data Curation, Writing Original draft, Writing Review & Editing; E. B. Formal analysis, Software, Data Curation, Writing Original draft; R. S. Formal analysis, Investigation, Writing Original draft; R. M. Writing Original draft, Project administration; O. B. Conceptualization, Investigation, Writing Original draft, Writing Review & Editing; L. H. Conceptualization, Methodology, Formal analysis, Investigation, Writing Original draft, Writing Review & Editing, Supervision, Project administration, Funding acquisition.

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Declaration of competing interest

The authors report no conflicts of interest and no financial relationships relevant to the manuscript.

Supplementary data

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