Revista Mexicana de Neurociencia

PERMANYER

Check for updates

ORIGINAL ARTICLE

A study of fMRI BOLD signals in verbal fluency tasks in young patients with type 1 diabetes mellitus

Miriam E. Jiménez-Maldonado[1,2], Andres A. González-Garrido[2], Esteban Gudayol-Ferré[3], Joan Guàrdia-Olmos[4], Rubén Bañuelos-Acosta[5], and Teresita J. Villaseñor-Cabrera[2,6]*

[1]Instituto de Neurociencias, Universidad de Guadalajara, Guadalajara, Jalisco, Mexico; [2]Departamento de Neurociencias, Centro Universitario de Ciencias de la Salud, Universidad de Guadalajara, Guadalajara, Jalisco, Mexico; [3]Facultad de Psicología, Universidad Michoacana San Nicolás de Hidalgo, Morelia, Michoacán, Mexico; [4]Facultad de Psicología, Instituto de Neurociencias, UB Institute of Complex Systems, Universitat de Barcelona, Barcelona, Spain; [5]Bañuelos Radiólogos, Guadalajara, Jalisco, Mexico; [6]Servicio de Neuropsicología, Hospital Civil Fray Antonio Alcalde, Guadalajara, Jalisco, Mexico

Abstract

Objective: *Our study aimed to evaluate verbal fluency (VF) using the blood oxygen level-dependent (BOLD) signal on functional magnetic resonance imaging (fMRI) in subjects with type 1 diabetes mellitus (T1DM) versus healthy controls. This was done during the execution of several tasks with high cognitive demand.* **Methods:** *A total of 15 right-handed subjects with type 1 diabetes (nine males, six females, with 20.80 average age) and 15 healthy right-handed volunteers (20.93 average age) matched by sex, age, and years of education performed a VF task while undergoing fMRI.* **Results***: Both groups obtained similar cognitive performance, with no significant differences in intelligence (p = 0.424), cognitive flexibility (p = 0.258), semantic VF (p = 0.620), and only phonological VF was close to significance (p = 0.063), which implies that both groups behaved similarly. Although the VF task activated the expected brain areas, such as the prefrontal cortex, in both groups, a difference was detected in young people with T1DM, who required the activation of larger brain areas, including some subcortical regions, such as the basal ganglia, to perform the tasks.* **Conclusions***: Our findings indicate that even among young adults with T1DM, there may be early signs of changes in metabolic brain function. This highlights the crucial role of the health sector in neuroscience to identify and address potential early cognitive changes in T1DM patients.*

Keywords: *Type-1 diabetes mellitus. Verbal fluency. Blood oxygen level-dependent signal.*

Un estudio de las señales fMRI BOLD en la tarea de fluidez verbal en pacientes jóvenes con DM1

Resumen

Objetivo: *Nuestro estudio tuvo como objetivo evaluar la fluidez verbal utilizando la señal dependiente del nivel de oxígeno en sangre (BOLD) en imágenes por resonancia magnética funcional (MRf) en sujetos con DM1 frente a controles sanos. Esto se llevó a cabo durante la ejecución de varias tareas con alta demanda cognitiva. Nuestra hipótesis era que ambos grupos responderían adecuadamente a nivel cognitivo, pero que habría diferencias en la actividad metabólica, según lo evaluado por los métodos de MRf.* **Métodos:** *quince sujetos diestros con diabetes tipo 1 (nueve hombres y seis mujeres, con una edad media de 20,80 años) y quince voluntarios diestros sanos (con una edad media de 20,93 años), emparejados*

***Correspondence:**
Miriam E. Jiménez-Maldonado
E-mail: elizabeth.jimenez@academicos.udg.mx

Date of reception: 30-09-2024
Date of acceptance: 29-08-2025
DOI: 10.24875/RMN.24000053

Available online: 24-11-2025
Rev Mex Neuroci. 2025;26(6):181-189
www.revmexneurociencia.com

por sexo, edad y años de educación, realizaron una tarea de fluidez verbal mientras se sometían a una resonancia magnética funcional. **Resultados:** *Ambos grupos obtuvieron un rendimiento cognitivo similar, sin diferencias significativas en inteligencia (p = 0,424), flexibilidad cognitiva (p = 0,258) y fluidez verbal semántica (p = 0,620), y solo la fluidez verbal fonológica se acercó a la significación (p = 0,063), lo que implica que ambos grupos se comportaron de manera similar. Aunque la tarea de fluidez verbal activó las áreas cerebrales esperadas, como la corteza prefrontal, en ambos grupos, se detectó una diferencia en los jóvenes con DM1, que necesitaron la activación de áreas cerebrales más amplias, incluidas algunas regiones subcorticales, como los ganglios basales, para realizar las tareas.* **Conclusiones:** *Nuestros hallazgos indican que, incluso entre los adultos jóvenes con DM1, pueden aparecer signos tempranos de cambios en la función metabólica del cerebro. Esto pone de relieve el papel crucial del sector sanitario en el campo de la neurociencia para identificar y abordar los posibles cambios cognitivos tempranos en los pacientes con DM1.*

Palabras clave: *Diabetes tipo 1. Fluencia verbal. Señal BOLD.*

Introduction

There is still no consensus on a definition of executive function (EF)[1,2]. However, it is widely accepted that EF includes a range of processes involved in complex behaviors such as decision-making, planning, abstract reasoning, self-monitoring, cognitive flexibility, behavioral inhibition, verbal fluency (VF), and working memory[1,3,4]. In summary, EFs help organize goal-directed actions[5], where the self plays a key role in modulating socially appropriate behaviors, all of which are complex skills.

Although there is still some debate about whether specific skills are related to EF, it is widely accepted that these skills are not uniform and involve moderately interconnected constructs[6]. In general, regardless of the type of ability, these skills are controlled by different regions of the frontal cortex, especially the prefrontal cortex[7]. However, several studies[8,9] have shown that some EF tasks, such as VF and switching tasks, also engage other areas such as the superior left parietal cortex[8,9]. In addition, the frontal cortex is closely connected to various parts of the central nervous system, such as the limbic system, indicating that emotional behavior can also be considered part of EF[1]. It is also important to note that VF depends on neurodevelopment[10] and may be linked to a perspective of functional interconnectivity[11].

The VF task, which is one of the tasks used to assess executive functioning, involves generating words that belong to a specific category or start with a certain phoneme within a limited time (usually 60 s)[1,12]. VF also requires inhibiting words that do not belong to the requested group and involves the ability to determine, update information, and follow specific procedures, making VF a key component of neuropsychological assessment[1,12].

VF tasks consist of two parts: (1) clustering and (2) switching[13]. In the first part, the subject uses a category until it is finished; the second part requires switching from one category (or cluster) to another. Both parts require inhibiting words that do not belong to the chosen category[12-14].

Functional magnetic resonance imaging (fMRI) studies of VF tasks in healthy subjects have shown activation in the medial prefrontal cortex[8], left inferior temporal lobe[14,15], left inferior frontal gyrus (LIFG)[15,16], and posterior parietal cortex[8]. Although the variety in the activation regions reported by different authors may result from task heterogeneity, another explanation highlights the specific verbal task used; the semantic fluency task is linked to temporal brain regions because it involves semantic memory, whereas the supramodal EF within the semantic or phonologic task depends on the frontal lobe[16].

VF, as a subdomain of executive functioning, is usually affected in other diseases such as schizophrenia[17], temporal lobe epilepsy[18], or frontal lesions[13], with less evidence in metabolic disorders.

In addition, some diseases exhibit patterns of changes in cognition and executive functioning at various levels. Severe or repeated hypoglycemia has been shown to cause cognitive impairment and neuronal death, likely because neither neurons nor glial cells can store glucose; therefore, over time, brain structure or function deteriorates, with the hippocampus being particularly vulnerable to these changes[19]. Diabetes mellitus, especially type 1, is a chronic disease that begins in childhood and adolescence, disrupting the balance of glucose and insulin, which affects different organs and systems, including the brain. Some cognitive deficits have been observed in individuals with type 1 diabetes mellitus (T1DM), notably in working memory, attention, EFs, visuospatial skills, and processing speed[19-22]. These alterations vary depending on

comorbidities, hospitalizations, hypo- or hyperglycemia episodes, or disease severity[19,23]; even social support can influence cognitive performance[24].

According to the World Health Organization, more than 463 million people have diabetes; in America, the number of diabetic patients is estimated at 62 million[25]. In addition, it is projected that 578 million people will have diabetes by 2030 and 700 million by 2040[25]. Approximately 3-5% of people with diabetes have type 1, which is one of the most common chronic diseases in childhood and adolescence[20,26]. In Mexico, the Ministry of Health reported that there are 542,000 children with T1DM, and nearly 78,000 develop it each year[27].

Although the severity of cognitive difficulties may be relatively mild, failures in cognitive functioning can be linked to a learning disorder[28,29] and may become more noticeable with poor glycemic control[30]. In addition, even a slight level of cognitive processing difficulty could interfere with or impair daily activities in adolescents and adults, especially when they need to solve complex cognitive problems, which could ultimately impact their quality of life[26]. Furthermore, fluctuations in long-term blood glucose levels have been associated with the development of stress, alexithymia, depression, and anxiety, among other clinical symptoms, and these changes also indicate a deficiency in the cognitive processing and regulation of emotional states[30,31].

Given that the intensity, chronicity, and impact of cognitive abilities in patients with T1DM vary due to multiple comorbidities and differences in glycemic control, and considering that T1DM begins early in life during a critical period for brain development, the adaptive form of metabolic balance requires more resources; therefore, identifying potential differences in activation patterns in brain regions during cognitively demanding tasks such as VF (semantic fluency) is crucial.

Therefore, there is an interest in understanding the differences between the neural substrates involved in solving high-demand cognitive tasks in cognitively normal individuals with T1DM and healthy controls.

Materials and methods

Participants

The study involved 15 right-handed individuals with T1DM (nine males and six females) and 15 healthy right-handed volunteers matched for age, sex, and years of education. All participants had normal or corrected-to-normal vision, average intelligence, and no history of neurological illness, psychiatric disorders, depression, addiction, or related conditions. Diabetic patients, diagnosed according to the American Diabetes Association criteria[32], had fewer than two hospitalizations in the past 2 years, maintained regular glycemic control, and had an average disease duration of 10 years. They did not have nephropathy, retinopathy, or any other clinical diabetes-related complications to be eligible for the study. This study received approval from the Ethics Committee of the "Hospital Civil Fray Antonio Alcalde" and the Neuroscience Institute at Guadalajara University, and informed consent was obtained from both patients and controls.

Stimuli and procedure

Our study used a unique pair of tasks (A/B): A, which assessed semantic VF by asking participants to produce words from a specific category, and B, a control task requiring subjects to recite the months of the year. This sequence was designed to activate brain regions involved in semantic VF.

We used a block design, with eight blocks per condition (A and B) and eight rest blocks between tasks; before each task, participants read a sign with an instruction reminder (Fig. 1). Each block included a beep or brief sound at 60 Hz for 225 ms, followed by 1500 ms allowed for responses, totaling 12 stimuli per block. The auditory stimulation was delivered through headphones to reduce scanner noise, and the session lasted 6 min and 12 s (Fig. 2). We used a modified paradigm of Gurd et al.[8]

All participants completed similar training 1 week before the scanner session to ensure familiarity and comfort with the tasks. Before the fMRI scan, plasma glucose levels were measured using an Accu-Check active glucometer. The task stimuli were presented using E-prime studio v.2.0 (Psychology Software Tools, Inc., 2013), and the images were projected through a Google system.

Image Acquisition: A 1.5 Tesla MR scanner (general electric healthcare system, Milwaukee, Ill) with a functional imaging T2∗-weighted gradient was used to obtain 32 contiguous 4 mm thick axial slices with the following parameters: repetition time (TR) = 3 s, echo time (TE) = 60 ms, field of view = 256 mm, matrix size = 64 × 64, and voxel dimensions = 4 × 4 × 4 mm, covering the whole brain. The experiment was conducted in a single session with two runs, each lasting 6 min and 12 s. The instructions and sounds for each response were presented using E-Prime software with a license.

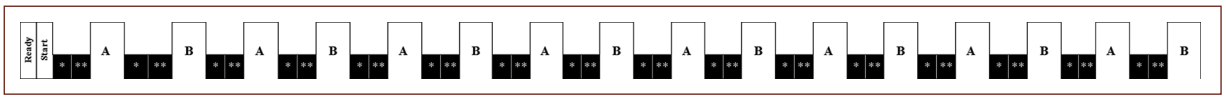

Figure 1. Experimental design.

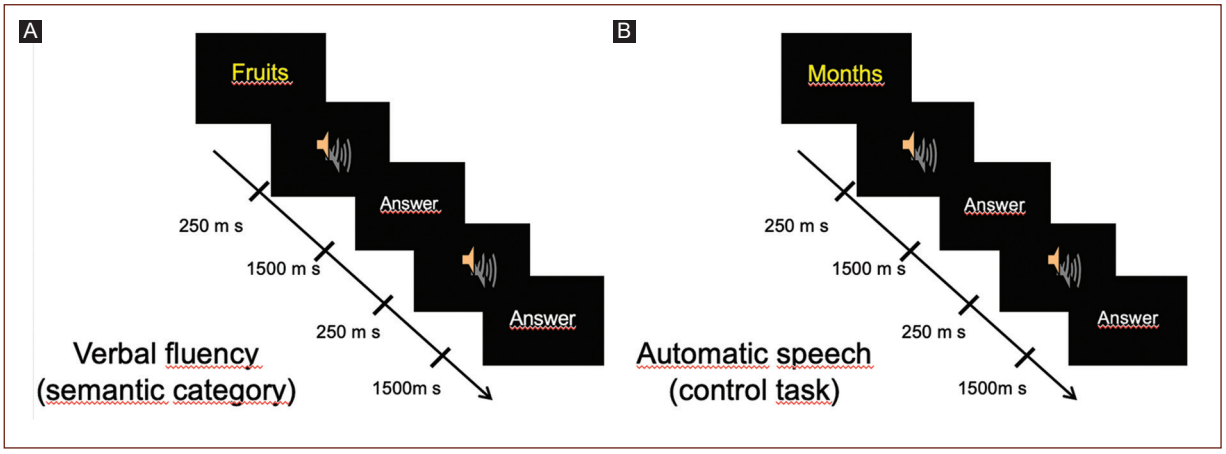

Figure 2. A and **B:** specific task.

Data analysis

Each experimental task involved 124 brain volumes; due to the experimental design, 12 brain volumes per task were discarded, leaving 112 for subsequent analysis. The images were preprocessed and statistically analyzed with the SPM8 software package (http://www.fil.ion.ucl.ac.uk/spm/software/spm8/). The images were spatially realigned, readjusted to the voxel size, and normalized according to the Montreal Neurological Institute and Talairach coordinates. For smoothing, a Gaussian kernel filter was applied three times the voxel size on the x-, y-, and z-axes.

The behavioral data were analyzed using the Statistical Package for the Social Sciences version 20, and statistical significance was set at 0.05.

Table 1. Participants baseline

	T1DM (n = 15)	Controls (n = 15)
Years with the disease	10 ± 3.47	-
Latest glycated hemoglobin	9.2 ± 2,87	-
Age	20.80 ± 4.47	20.93 ± 4.52
Education	12.20 ± 2.78	12.80 ± 3.00
Intelligence quotient	103.80 ± 9.88	109.20 ± 28.19
Verbal fluency (verbs)	18.93 ± 5.74	22.86 ± 4.91
Verbal fluency (semantic)	23.21 ± 5.96	24.14 ± 3.46
Verbal fluency (phonological)	11.86 ± 4.16	14.21 ± 3.40
Glucose level before fMRI (mL/dL)	217.67 ± 92.74	97.00 ± 10.06

T1DM: type 1 diabetes mellitus; fMRI: functional magnetic resonance imaging.

Results

Behavioral and cognitive results

The total sample included 15 individuals with T1DM and 15 healthy controls, with characteristics shown in table 1. Both groups exhibited similar cognitive performance, with no significant differences; they all had normal IQ, semantic, phonological, and VF, as well as cognitive flexibility and planning skills, indicating comparable cognitive abilities.

Since the behavioral data showed similar ratings between groups as expected, there was no difference between the groups in terms of age ($t[28] = -0.81$, $p = 0.936$; $d = 0.015$) or education level ($t[28] = -0.567$, $p = 0.575$; $d=0.10$), but a significant difference in glucose level was observed ($t[28] = 5.01$, $p = 0.000$; $d = 0.68$). We studied T1DM without cognitive alterations. Therefore, the behavioral results aligned with our expectations.

Table 2. Percentage, mean, and standard deviation of correct/incorrect responses for type 1 diabetes and controls

Type of tasks	T1D		Controls	
	Percentage	Mean (SD)	Percentage	Mean (SD)
Task A. Fluency Correct response Incorrect response	 87.08 0.83	 41.80 ± 4.91 0.40 ± 0.699	 93.75 0.45	 45.00 ± 5.24 0.22 ± 667
Task B. Automatic speech Correct response Incorrect response	 100	 48.00 ± 0.00	 99.08 0.91	 47.56 ± 1.33 0.44 ± 1.33

SD: standard deviation.

The average number of correct and incorrect responses per task was entered into a mixed-measures analysis of variance (ANOVA) with task (semantic fluency and automatic language) as a within-subject factor and group (type 1 diabetes vs. controls) as a between-subject factor, as shown in table 2.

As expected, there was no difference between the two groups (intragroup factor) in correct or incorrect responses. However, task complexity (VF vs. automatic speech) mainly influenced correct responses, and both groups responded similarly to the two tasks (Table 3).

Table 3. Mixed measurements ANOVA

Behavioral response	gl	F	Sig.	η^2	1-β
Correct answers (tasks)	1	17.748	0.001	0.511	0.978
Correct answers (groups)	1	3.075	0.098	0.153	0.380
Incorrect answers (tasks)	1	0.106	0.748	0.006	0.061
Incorrect answers (groups)	1	1.301	0.270	0.071	0.190

ANOVA: analysis of variance.

Imaging results

All the neuroimaging analysis results were statistically significant at the 0.01 level. The first step was analyzed separately for activations in each of the tasks: A (verbal semantic fluency) and B (automatic fluency). Based on the averages of activation in each group by experimental condition (Table 4), the main activation cluster in T1DM patients during the VF task was located in the cerebellum, followed by the medial and superior frontal gyri in the left and right hemispheres, respectively, as well as the hippocampal areas in both hemispheres. These findings highlight the importance of this research. In comparison, healthy controls showed greater activation in the frontal-precentral gyrus and cerebellum than T1DM patients, in whom cortical activations were more prominent (Fig. 3).

The second step involved analyzing the interactions using full factorial ANOVA, which showed the main effects of two between-group factors – task (semantic vs. automatic fluency) and group (type 1 diabetes vs. controls) – as well as their interaction. The inferential statistics are presented in table 5.

In the group factor analysis, three significant activation clusters were identified: two in the left hemisphere, one in the medial precentral frontal gyrus (BA 6), and the other in the culmen of the cerebellum (part of the superior vermis), with the third in the right medial temporal gyrus (Brodmann's area [BA] 22) (Fig. 4).

The interaction between tasks showed greater activation in T1DM patients than in controls, mainly in the prefrontal, precentral, and anterior cingulate areas (Fig. 5).

Discussion

The present T1DM sample and healthy controls did not show significant differences in their cognitive performance or behavioral task scores. This was expected due to the characteristics of our T1DM group, which had adequate glycemic control, no comorbidities, and good socio-economic functioning. This result aligns with other studies[33]. However, it has also been reported that T1DM patients performed slightly worse in inhibitory control[34] or working memory tasks despite maintaining reasonable diabetes control[35]. T1DM patients experience significant glycemic fluctuations throughout the day. In this study, although T1DM individuals did not show cognitive differences compared to a group of healthy controls before the fMRI scan, their blood glucose levels were above average. Since glucose level is an inherent factor (but not necessarily the cause) of metabolic changes, the variation in this variable between the groups does not appear to impact behavioral performance.

Table 4. Main activations, task A, semantic fluency

Groups	Z max	Cluster	Talaraich coordinates			H	BA	Area
			x	y	z			
T1DM	3.752	110	10	–72	–26	R	-	Cerebellum (pyramid)
	3.239	54	2	8	66	R	6	Superior frontal gyrus
	3.234	17	–26	–44	10	L	-	Hippocampus
	2.360	1	30	-44	10	R	-	Hippocampus
Controls	3.744	20	–26	–48	6	L	30	Parahippocampal gyrus
	2.746	8	18	–68	–26	R	4	Cerebellum (uvula)
	2.523	1	62	–4	18	R	6	Precentral frontal gyrus
	2.437	1	50	–8	34	R	-	Precentral frontal gyrus
	2.430	3	6	–76	–30	R	-	Cerebellum (pyramid)

T1DM: type 1 diabetes mellitus; Z max: maximum score; x, z, and y: spatial axes; H: hemisphere; BA: Brodmann area probabilistic citoarchitectonic mapping of activations with respect to BA.

Table 5. Effects of activations

Factors	F value	p	Cluster	Talaraich coordinates			H	BA	Area
				x	y	z			
Groups (T1DM vs. controls)	8.845	0.004	4	–62	0	22	L	6	Precentral frontal gyrus
	8.603	0.004	8	–26	–28	–22	L	-	Cerebellum-culmen
	7.822	0.007	5	70	–40	2	R	22	Medial temporal gyrus
Task (semantic vs. automatical fluency)	9.065	0.003	14	42	16	22	R	46	Medial frontal gyrus

T1DM: type 1 diabetes mellitus; F: F of Snedecor; p: P value; x, z, and y: spatial axes; H: hemisphere; BA: Brodmann area probabilistic cytoarchitectonic mapping of activations with respect to BA.

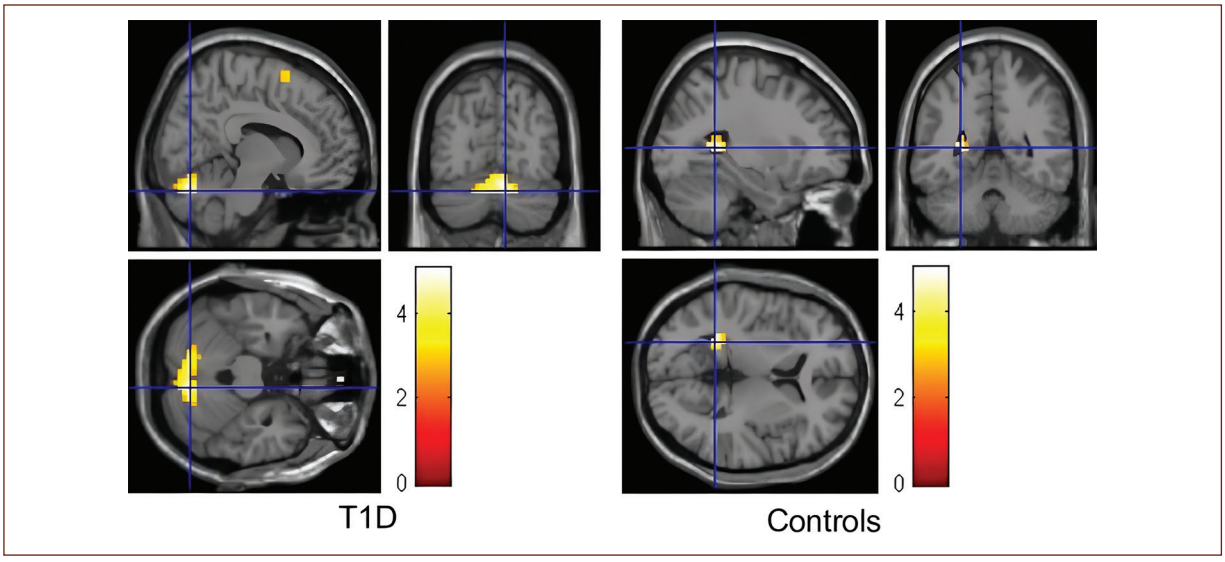

Figure 3. Image of sagittal, coronal, and temporal slices showing the average number of activations for each group of the verbal fluency task; the intersection indicates the main activation cluster.

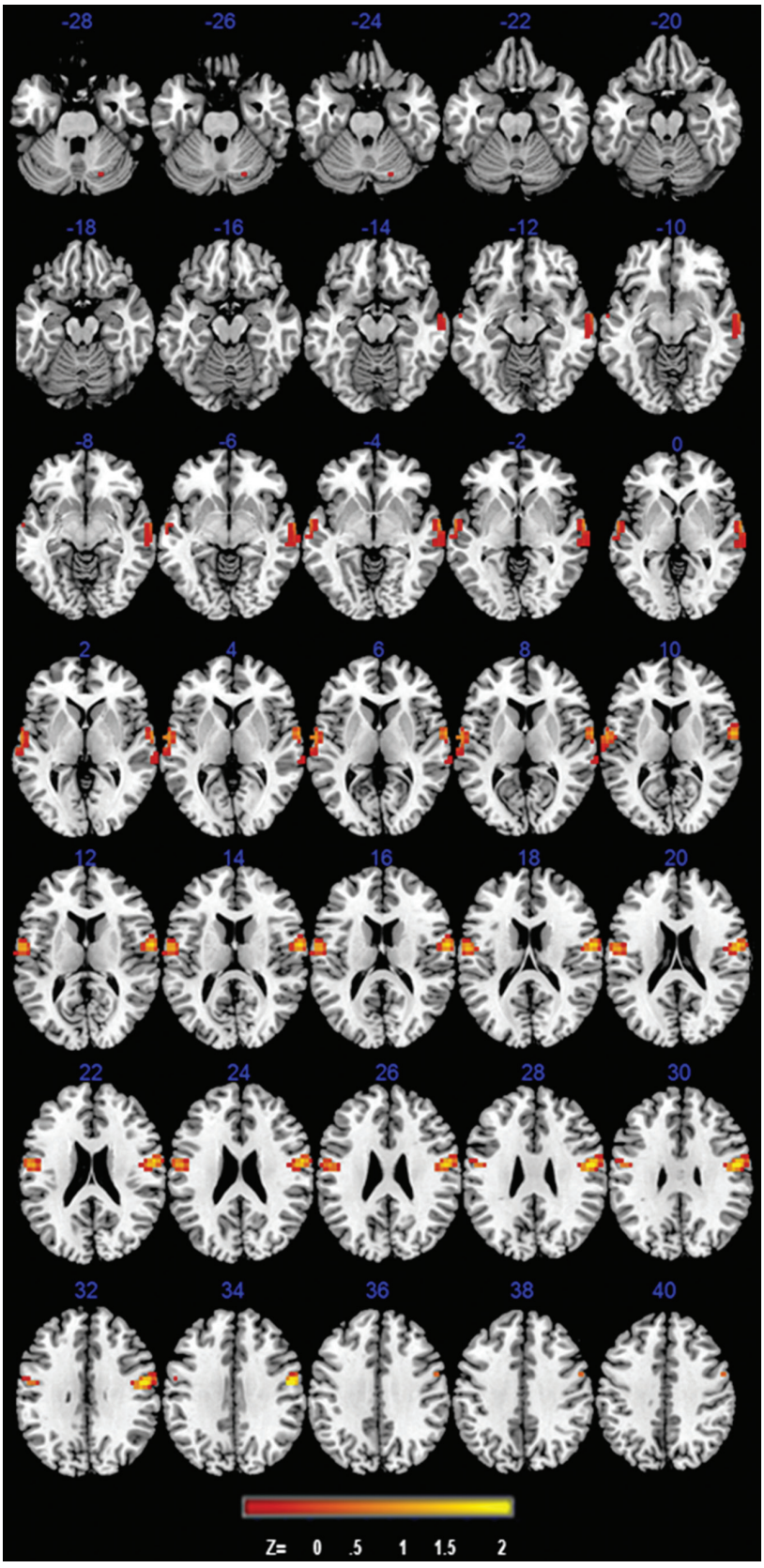

Figure 4. Brain activations, group factor, neurologic view (left-right).

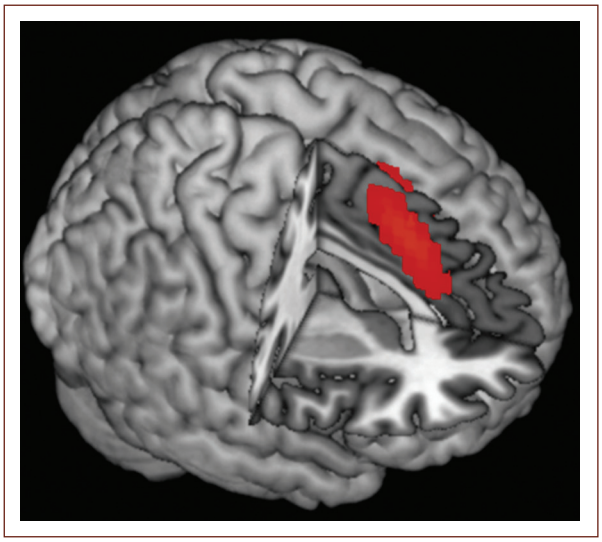

Figure 5. Reconstruction average image of the interaction between verbal fluency versus automatic speech tasks. Shades in red correspond to subjects with type 1 diabetes and shades in blue to control subjects.

The cognitive tasks of interest in VF have been described as activating several related brain areas, including prefrontal regions, especially the dorsolateral area, LIFG, anterior cingulate cortex, and cerebellum[8,14,15,36]. We observed these activations in both groups, particularly in the right hemisphere in the T1DM group; however, such activations were not evident in the overall group average.

Although the LIFG has been strongly associated with VF tasks, including both semantic and phonological ones[8,16,37], its absence from the group averages does not mean that this area is not involved. This could be explained by several factors: (a) the task was straightforward, such as a common category (fruits)[37], and (b) when averaging across all subjects, the activation was not visible, probably because the effect did not last long enough to generate a significant blood oxygen level-dependent signal.

In addition to activations in regions of the dorsolateral prefrontal cortex, both in terms of the average across all groups and within each group, we observed abundant activations in motor and premotor areas, mainly in the premotor cortex corresponding to BA[6]. This can be explained by the verbalization required to complete the task.

By analyzing frequencies in the VF tasks, activations were observed in both groups in the temporal regions, especially in the fusiform and temporopolar gyri (BA 38), which have been previously seen in both hemispheres[36]. In healthy control participants, the highest activations in the temporal lobe corresponded to Wernicke's area BA 22 (also bilaterally), which could be due to the strategy used or the task's low difficulty, as these factors can affect performance[38].

Our results emphasize that the cerebellum is predominantly activated, as reported in most tasks and interactions in other studies[38]. In this work, we observed activations in several regions of the cerebellum, and this structure has infrequently been linked to VF tasks[39].

Since both groups showed similar behavioral responses, it was difficult to differentiate T1DM patients from controls at the cognitive response level. However, differences can be identified in the brain structures involved in producing these responses; although T1DM patients need regulation of areas linked to the VF task, the intensity or clusters of activation are much higher than those of controls.

Conclusion

We must address several limitations in our study, one related to the fMRI scan performed on a 1.5 Tesla scanner, and the other concerning the sample size. However, the results can still be considered valid from both a clinical perspective and based on the findings, given that it is uncommon to evaluate individuals with this metabolic condition who do not have cognitive dysfunction but show different brain organization compared to those without any alterations.

We strongly advocate for a more comprehensive understanding of brain function in patients with type 1 diabetes during cognitively demanding tasks. This knowledge will enable us to provide early attention to the potential development of cognitive impairment, a crucial aspect that is often overlooked in current health-service follow-up. The focus is currently on controlling metabolic status, ensuring adherence to medical treatment, and early detection of common organ complications targeted by illness, such as renal issues, retinal problems, and peripheral nerve damage. However, the prevention of cognitive decline is equally important, as it can significantly impact the quality of life for T1DM patients.

Based on the above, although we are hypothesizing, it would be advisable to conduct longitudinal studies to confirm possible changes in brain organization.

Funding

The authors declare that this work was carried out with the authors' own resources.

Conflicts of interest

The study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics

Committee of Neuroscience Institute of Guadalajara University (ET112014-183).

Acknowledgments

The authors would like to thank Bañuelos Radiólogos group with the Neurosciences Department of the University of Guadalajara.

Ethical considerations

Protection of humans and animals. The authors declare that the procedures followed complied with the ethical standards of the responsible human experimentation committee and adhered to the World Medical Association and the Declaration of Helsinki. The procedures were approved by the institutional Ethics Committee.

Confidentiality, informed consent, and ethical approval. The authors have followed their institution's confidentiality protocols, obtained informed consent from patients, and received approval from the Ethics Committee. The SAGER guidelines were followed according to the nature of the study.

Declaration on the use of artificial intelligence. The authors declare that no generative artificial intelligence was used in the writing of this manuscript.

References

1. Lezak M, Howieson DB, Loring DW. Neuropsychological Assessment. 4th ed. Oxford: Oxford University Press; 2004.
2. Rushworth MF, Hadland KA, Paus T, Sipila SK. Role of the human medial frontal cortex in task switching: a combined fMRI and TMS study. J Neuropsychol. 2002;87:2577-92.
3. Gómez-Beldarrain M. Síndromes disejecutivos y lóbulos frontales. In: Peña-Casanova J, editor. Neurología de la Conducta y Neuropsicología. México: Panamericana; 2007. p. 327-49.
4. Rosselli M, Jurado MJ, Matute M. Las funciones ejecutivas a través de la vida. Rev Neuropsicol Neuropsiquiatr Neuroci 2008;8:23-46.
5. Fuster JM. The prefrontal cortex. 4th ed. Amsterdam: Elsevier; 2008. p. 192-219, 285-330.
6. Miyake A, Friedman N, Emerson M, Witzki A, Howerter A. The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: a latent variable analysis. Cogn Psychol 2000;41:49-100.
7. Tirapu-Ustárroz J, García-Molina A, Luna-Lario P, Verdejo-García A, Ríos-Lago M. Corteza prefrontal, funciones ejecutivas y regulación de la conducta. In: Tirapu-Ustárroz J, García-Molina A, Ríos-Lago M, Ardila-Ardila A, editors. Neuropsicología de la Corteza Prefrontal y Las funciones Ejecutivas. España: Viguera. 2000. p. 89-120.
8. Gurd JM, Amunts K, Weiss PH, Zafiris K, Marshall JC, Fink GR. Posterior parietal cortex is implicated in continuous switching between verbal fluency task: an fMRI study with clinical implications. Brain. 2002;125:1024-38.
9. Sohn MH, Ursu S, Anderson JR, Stenger VA, Carter CS. The role of prefrontal cortex and posterior parietal cortex in task switching. Proc Natl Acad Sci U S A. 2000;97:13448-53.
10. Doebel S. Rethinking executive function and its development. Perspect Psychol Sci. 2020;15:942-56.
11. Perone S, Simmering VR, Buss AT. A dynamical reconceptualization of executive-function development. Perspect Psychol Sci. 2021;16:1198-208.
12. Goñi J, Arrondo G, Sepulcre J, Martincorena I, Vélez de Mendizábal N, Villoslada P, et al. The semantic organization of the animal category: evidence from semantic verbal fluency and network theory. Cogn Process. 2011;12:183-96.
13. Troyer AK, Moscovitch M, Winocur G, Alexander MP, Stuss D. Clustering and switching on verbal fluency: the effects of focal frontal and temporal lobe lesions. Neuropsychologia. 1998;36:499-504.
14. Sheldon S, Moscovith M. The nature and time-course of medial temporal lobe contributions to semantic retrieval: an fMRI study on verbal fluency. Hippocampus. 2012;22:1451-66.
15. Catani M, Allin MP, Husain M, Pugliese L, Mesulam MM, Murray RM. Symmetries in human brain language pathways correlate with verbal recall. Proc Natl Acad Sci U S A. 2007;104:17163-8.
16. Costafreda SG, Fu CH, Lee L, Everitt B, Brammer MJ, David AS. A systematic review and quantitative appraisal of fMRI studies of verbal fluency: role of the left inferior frontal gyrus. Hum Brain Mapp. 2006;27:799-810.
17. Ragland JD, Moelter ST, Bhati MT, Valdez JN, Siegel SJ, Gur RC, et al. Effect of retrieval effort and switching demand on fMRI activation during semantic word generation in schizophrenia. Schizophr Res. 2008;99:312-23.
18. Kucukboyaci NE, Girard HM, Hagler DJ, Kuperman J, Tecoma ES, Iragui VJ, et al. Role of frontotemporal fiber tract integrity in task-switching performance of healthy controls and patients with temporal lobe epilepsy. J Int Neuropsychol Soc. 2012;18:57-67.
19. Wrighten SA, Piroli GG, Grillo CA, Reagan LP. A look inside the diabetic brain: contributors to diabetes-induced brain aging. Biochim Biophys Acta. 2008;444-53.
20. Ferguson SC, Blane A, Wardlaw J, Frier BM, Perros P, McCrimmon RJ, et al. Influence of an early-onset age de type 1 diabetes on cerebral structure and cognitive function. Diabetes Care. 2005;28:1431-7.
21. Kodl CT, Seaquist ER. Cognitive dysfunction and diabetes mellitus. Endocr Rev. 2008;29:494-511.
22. Lin A, Northam EA, Rankins D, Werther GA, Cameron FJ. Neuropsychological profiles of young people with type 1 diabetes 12 yr after disease onset. Pediatr Diabetes. 2010;11:235-43.
23. Li W, Huang E, Gao S. Type 1 diabetes mellitus and cognitive impairments: a systematic review. J Alzheimers Dis. 2017;1:29-36.
24. Ocura T, Heisler M, Langa KM. The association of cognitive and social support with glycemic control in adults with diabetes. J Am Geriatric Soc. 2009;57:1816-24.
25. World Health Organization, Pan American Health Organization. Type 1 diabetes [Internet]. [consulted 2023 Oct 20]. Available from: https://www.paho.org/en/topics/diabetes
26. Brands AM, Kessels RP, Hoogma RP, Henselmans JM, Van der Beek Boter JW, Kappelle LJ, et al. Cognitive Performance, psychological well-being, and brain magnetic resonance imaging in older patients with type 1 diabetes. Diabetes. 2006;55:1800-6.
27. Secretaría de Salud Jalisco. [Internet]. [consulted 2023 Oct 20]. Available from: https://ssj.jalisco.gob.mx/search?query=diabetes%20tipo%201%20en%202023
28. Tatsiopoulou P, Porfyri GN, Bonti E, Diakogiannis I. Priorities in the interdisciplinary approach of specific learning disorders (SLD) in children with type i diabetes mellitus (T1DM). From theory to practice. Brain Sci. 2020;11:4.
29. Tupola S, Salonen I, Hannonen R, Verho S, Saar P, Riikonen R. Comparison of regional cerebral perfusion, EEG and cognitive functions in type 1 diabetic children with and without severe hypoglycaemia. Eur J Pediatri. 2004;163:335-6.
30. Semenkovich K, Patel PP, Pollock AB, Beach KA, Nelson S, Masterson JJ, et al. Academic abilities and glycaemic control in children and young people with Type 1 diabetes mellitus. Diabet Med. 2016;33:668-73.
31. Henríquez-Tejo R, Cartes-Velásquez R. Psychosocial impact of type 1 diabetes mellitus in children, adolescents and their families. Literature review. Rev Chil Pediatr. 2018;89:391-8.
32. American Diabetes Association. Diabetic Statistics. Virginia: American Diabetes Association; 2023.
33. Ly TT, Anderson M, McNamara KA, Davis EA, Jones TW. Neurocognitive outcomes in young adults with early-onset type 1 diabetes trang T Ly 1, mike anderson. Diabetes Care. 2011;34:2192-7.
34. Shinosaki JM, Rossini JC, Jorge ML, Macedo LB, Tannús PJ. Type 1 diabetes, cognition and the neurologist: exploring Go/No-Go and Maze tasks in the search for a practical screening tool. Int J Dev Neurosci. 2017;60:86-93.
35. Gallardo-Moreno GB, González-Garrido AA, Gudayol-Ferré E, Guàrdia-Olmos J. Type 1 diabetes modifies brain activation in young patients while performing visuospatial working memory tasks. J Diabetes Res. 2015;2015:703512.
36. Scaccianoce E, Lagana MM, Baglio F, Preti MG, Bergsalnd N, Cecconi P, et al. Combined DTI-fMRI analysis for a quantitative assessment of connections between WM bundles and their peripheral cortical fields in verbal fluency. Brain Topogr. 2016;29:814-23.
37. Katzev M, Tûscher O, Henning J, Weiller C, Keller CP. Revisiting the functional specialitation of left interior frontal girus in phonological and semantic fluency: the crucial role of task demands and individual ability. J Neurosci. 2013;33:7837-45.
38. Ledoux K, Vannorsdall TD, Pickett EJ, Bosley LV, Gordon B, Schretlen DJ. Capturing additional information about the organization of entries in the lexicon from verbal fluency productions. J Clin Exp Neuropsychol. 2014;36:205-20.
39. Ackermann H. Cerebellar contributions to speech production and speech perception: psycholinguistic and neurobiological perspectives. Trends Neurosci. 2008;31:265-72.