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The role of executive control in bilingual language production: A study with Parkinson's disease individuals

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ABSTRACT

The basal ganglia are critically involved in language control (LC) processes, allowing a bilingual to utter correctly in one language without interference from the non-requested language. It has been hypothesized that the neural mechanism of LC closely resembles domain-general executive control (EC). The purpose of the present study is to investigate the integrity of bilingual LC and its overlap with domain-general EC in a clinical population such as individuals with Parkinson's disease (PD), notoriously associated with structural damage in the basal ganglia.

We approach these issues in two ways. First, we employed a language switching task to investigate the integrity of LC in a group of Catalan–Spanish bilingual individuals with PD, as compared to a group of matched healthy controls. Second, to test the relationship between domain-general EC and LC we compared the performances of individuals with PD and healthy controls also in a non-linguistic switching task. We highlight that, compared to controls, individuals with PD report decreased processing speed, less accuracy and larger switching costs in terms of RT and errors in the language switching task, whereas in the non-linguistic switching task PD patients showed only increased switching cost in terms of errors. However, we report a positive correlation between the magnitudes of linguistic and non-linguistic mixing costs in individuals with PD. Taken together, these results support the notion of a critical role of the basal ganglia and connected structures in LC, and suggest a possible link between LC and domain-general EC.

1. Introduction

For successful communication bilingual speakers need to restrict lexicalization to one language in order to avoid cross-language interferences from the unintended language. The mechanisms that guarantee the success of communication are known as bilingual language control (LC) (Abutalebi and Green, 2007). Despite the fact that the underlying neurocognitive mechanisms are not fully understood, there is a general agreement that LC shares some of the processes involved in domain-general executive control (EC) such as working memory, task-monitoring, task execution, response selection and inhibition.

These control processes are thought to interact at different levels of the language production pipeline: from selection of a concept to be expressed, to the retrieval of its lexical counterpart and its phonological form, and to the planning and monitoring of the articulatory aspects of speech output (e.g., Roelofs and Piai, 2011; Ye and Zhou, 2009). It has been proposed that impairments of domain-general EC processes may have also a role in causing language production deficits (Roelofs and Piai, 2011). For instance, some recent theories have attributed impaired language performance in elderly adults to an overall slowing of mental processing, to a lack of inhibitory control, and to working memory deficits. Similarly, in individuals with Parkinson disease (PD), it has been proposed that deficits related to EC may be responsible for language production deficits (see Dirnberger and Jahanshahi (2013) for a review). Among the linguistic deficits reported in PD, recent studies reported deficits for word-finding (object/verb naming: Cotelli et al., 2007; word fluency: Henry and Crawford, 2004), grammatical rule-based transformations (Ullman et al., 1997), and comprehension of syntactical complex sentences (Grossman et al., 1992; Lieberman et al., 1992).

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PD is considered a neurodegenerative disease characterized by decreased Dopamine production in the midbrain, in particular in substatia nigra pars compacta, affecting mesocortical and mainly nigro-striatal connections. PET studies have revealed that the metabolic dysfunctions in PD also extend to the frontal cortex through its dopaminergic connections (Narayanan et al., 2013). For instance, it has been shown that the dysfunctions in the circuitry connecting the frontal cortex and the basal ganglia, because of striatal dopamine deficiency, are at least in part responsible for the executive control deficits in PD (Brück et al., 2001; Marié et al., 1999; Owen et al., 1998; Polito et al., 2012). The caudate, being part of the striatum, is heavily afflicted by PD. Interestingly, the left caudate is both part of the domain general EC network and the LC network (Abutalebi and Green, 2007). Its role in LC in bilinguals has been well described in healthy individuals through functional and structural neuroimaging (see for review: Abutalebi and Green (2008) and Luk et al. (2012)) and in clinical populations such as bilingual aphasics (see for review: Abutalebi and Green (2008)), individuals with multiple sclerosis (Calabria et al., 2014), and with neurodegenerative diseases such as PD (Zanini et al., 2004, 2010).

In the present study we aim to investigate the functioning of bilingual LC, and its relation with EC, in a group of Catalan–Spanish bilingual individuals with PD. PD provides us with an unique opportunity to shed light into the interesting discussion whether LC in bilinguals and domain-general control are based on a common system.

1.1. Bilingual LC and frontal–subcortical circuits

Neuroimaging and single-case studies of bilingual aphasics have shown that basal ganglia are involved in those processes which allow bilinguals to control two languages (Abutalebi and Green, 2007). Luk et al. (2012) conducted a meta-analysis and found that the caudate, among other brain structures in frontal and temporal areas, is consistently activated during language production tasks in which the alternation between languages or translation across-languages is required, i.e., where LC is most required. Abutalebi et al. (2013) also report that this caudate activity is specific to bilinguals as opposed to monolinguals, and bilinguals were also reported to have increased gray matter densities in the left caudate (Zou et al., 2012). It was proposed that the caudate plays, functionally, a crucial role in bilingual language processing through a subcortical–frontal loop involved in language planning and control (Abutalebi and Green, 2008). Interestingly, selective lesions to the caudate may disrupt this control resulting in specific deficits such as pathological switching and mixing of languages (Abutalebi et al., 2000; Ansaldo et al., 2010; Mariën et al., 2005) or the inability to switch among languages (Aglioti et al., 1996). Therefore, bilingual individuals with PD may offer a good opportunity to further elucidate the role of the basal ganglia and connected structures for LC functions in bilinguals. This interesting field has not received much attention despite the fact that recent studies have shown that bilingual individuals with PD may have language production deficits. Indeed, in a first study by Zanini et al. (2004) the authors reported that Friulian–Italian bilingual speakers with PD were more impaired than controls in syntactic comprehension, and a further study by Zanini et al. (2010) also showed that spontaneous language production was impaired. Interestingly, in both studies it was found that bilingual individuals with PD were more impaired (compared to healthy controls) only when performing tasks in their L1 (Friulian) (for similar results see also Johari et al. (2013)). Similarly, a recent study by Adrover-Roig et al. (2011) suggested the implication of the basal ganglia in the lexicalization of the L1. The authors described a Basque–Spanish bilingual (J.Z.) who was characterized by more impaired language processing in his L1 (Basque) than L2 (Spanish) due to a brain lesion in the left basal ganglia. Interestingly, the fact that J.Z. also showed deficits in the domain of EC functions led the authors to conclude that the patient's impairment in his L1 was probably due to LC deficits. These data suggest that deficits in the EC following damage to the basal ganglia may affect language production, and sometimes in a relatively different manner for the two languages (see also Green et al. (2010), for the role of the EC deficits in bilingual aphasics).

In the present study we focus on dysfunctions of basal ganglia, and connected structures, and their impact on the efficiency of LC and domain general EC in bilinguals. We employed a language switching task in a group of bilingual individuals with PD and compared their performance to a matched control group of healthy individuals. In the task employed participants were required to name a series of pictures in different language conditions, with a cue indicating in which language to name the picture. In such a paradigm two kinds of trials result: switch trials (if the preceding picture was to be named in a different language) and repeat trials (if the preceding picture was to be named in the same language). It is commonly known that participants are slower and less accurate on switch trials than repeat trials, and the difference in reaction times (RTs) between these two types of trials is referred to as the language switching cost (e.g., Costa and Santesteban, 2004, 2006; Meuter and Allport, 1999). Language switching studies on low proficient bilinguals showed that participant have larger switch costs when they switch from their L2 into L1 than vice versa (Costa and Santesteban, 2004; Meuter and Allport, 1999). Conversely, it has been observed that high proficient bilinguals do not show this asymmetrical pattern of switch costs, that is they have the same magnitude of costs for L1 and L2 (Calabria et al., 2011; Costa and Santesteban, 2004).

Moreover, it has also been reported that naming in a “blocked” language condition with one language only, RTs for these trials (single) are usually faster than repeat trials in the mixed condition (Weissberger et al., 2012). The difference of RTs between repeat and single trials is called the language mixing cost. Interestingly, these two types of cost seem to be related to relatively different mechanisms of control. Whereas switching costs would be associated with transient mechanisms of control such as conflict resolution between competing tasks and response selection, mixing costs would rather reflect sustained mechanisms of control such as conflict and response monitoring, and the cost of keeping two task-sets available (Braver et al., 2003; Kray and Lindenberger, 2000; Los, 1996; Rubin and Meiran, 2005; for a similar account in the domain of language see Guo et al. (2011)).

Interestingly, recent evidence points also to an age-related decline of bilingual LC efficiency. For instances, for language switching tasks, elderly bilinguals as compared to younger bilinguals are slower overall, more error-prone and have an increased language switch cost (Gollan and Ferreira, 2009; Kohnert et al., 1999; Weissberger et al., 2012). Given that aging is usually associated to a decline of EC functions, these results suggest a possible link between non-linguistic mechanisms and those of LC (Gollan et al., 2011; but see also Calabria et al. (2013), for a different pattern of results).

Furthermore, there is evidence that patients who experience pathological language switching (pLS) in this type of task produce more cross-language intrusions when required to switch languages (switch trials). For instance, Calabria et al. (2014) described a Catalan–Spanish bilingual speaker (RRT) who produced more cross-language intrusions in a language switching task when she switched into her dominant language (Catalan) from her non-dominant one (Spanish) than the opposite (in contrast with the typical symmetrical switching pattern in healthy high proficient bilinguals; Calabria et al., 2011; Costa and Santesteban, 2004).