


THE UNDERLYING MECHANISM OF SELECTIVE AND DIFFERENTIAL RECOVERY IN BILINGUAL APHASIA

Nele Verreyt

Promotor: Prof. Dr. Wouter Duyck
Copromotoren: Prof. Dr. Patrick Santens & Prof. Dr. Guy Vingerhoets

Proefschrift ingediend tot het behalen van de academische graad
van Doctor in de Psychologie

2013

UNIVERSITEIT GENT

FACULTEIT PSYCHOLOGIE EN PEDAGOGISCHE WETENSCHAPPEN

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ACKNOWLEDGEMENTS

Wat ik niet zeggen kan
en niet kan schrijven
zal ergens diep in mij
toch bij me blijven.

Ongehoord
maar in een lieve duisternis
verbergt zich iets
dat meer dan woorden is.

(Toon Hermans)

Voor een deel heb ik al afscheid genomen van de vakgroep, ongeveer een jaar geleden. Toch leek het nooit zo. “Natuurlijk zien we elkaar nog terug, ik ben toch nog bezig aan mijn doctoraat?”, “We zien elkaar sowieso op mijn verdediging!”. Maar ook dat échte einde is nu stilaan in zicht. Ik ben opgelucht, absoluut. Maar ik voel nu al een beetje heimwee. Een beetje nostalgie.

Dat ik geen doctoraat kon schrijven in m’n eentje, dat had ik verwacht. Dat ik er zoveel fijne mensen zou leren kennen, daar kon ik alleen maar van dromen, en daar kan ik alleen maar heel erg blij om zijn. Heel wat mensen verdienen het om in de bloemetjes gezet te worden.

Tijdens mijn doctoraat werd ik omringd door mensen die stuk voor stuk specialisten zijn in hun vakgebied. Allereerst een geweldige dankjewel aan mijn promotor **Wouter Duyck**, die toch wel een sprong in het diepe nam met dit onderzoeksproject. Want ik wou wel doctoreren, maar “enkel en alleen als ik patiëntenonderzoek mocht doen.” Wouter, mijn interesse in tweetaligheid en mijn zin om onderzoek te gaan doen, heb ik aan jou te danken. Je leerde me de kneepjes van het vak, en wist me steeds te stimuleren om net dat stapje verder te gaan. Een bezoekje aan jouw bureau zorgde er telkens weer voor dat ik er met nieuwe moed tegenaan ging. Zowel

voor de praktische regelingen als voor theoretische inzichten kon ik altijd bij jou terecht. Dankjewel om me de kans te geven om te proberen, om te ontdekken, om te leren. Dankjewel om me te helpen op de momenten dat ik hulp nodig had, om me te motiveren in moeilijke momenten, maar ook om me de vrijheid te geven om mijn eigen weg te gaan. Dankjewel om me te overtuigen om aan dit doctoraat te beginnen. Dankjewel om met me te springen!

Ik wil ook graag **Miet De Letter** bedanken. Miet, jouw deur stond altijd voor me open. Jouw werkijver, enthousiasme voor onderzoek en vooral je liefde voor patiënten zijn aanstekelijk. Voor alle hulp en tips, dankjewel! Ook **Peter Mariën** verdient een bedanking. Peter, het was een eer om met jou te mogen samenwerken. Je kennis en ervaring zijn onovertroffen, en de bescheidenheid waarmee je ze deelt, deed me naar jou opkijken. Jouw visie op onderzoek was verhelderend, heeft me dikwijls de ogen geopend en de zin gegeven om er voor te gaan. Bedankt!

Ook de andere leden verdienen een woord van dank. **Rob Hartsuiker**, **Dimitri Hemelsoet**, **Patrick Santens** en **Guy Vingerhoets**, jullie hebben dit doctoraat mee naar een hoge niveau getild. Dankjewel voor jullie enthousiasme, jullie kritische en opbouwende kijk op mijn onderzoek, voor de richting die jullie me hebben gegeven, en voor de steun.

Een ongelooflijk grote dank en appreciatie gaan uit naar **alle patiënten** die hebben deelgenomen aan mijn onderzoek, **en hun familie**. Elke keer weer werd ik enthousiast onthaald, elke keer weer was ik van harte welkom met mijn vaak saaie taakjes en aartsmoeilijke oefeningen. Telkens opnieuw merkte ik een ongelooflijke moed om het zo goed mogelijk te doen, om te genezen. Het is niet evident om met afasie te leren leven, noch voor de patiënten zelf, noch voor de familieleden. Ik heb zoveel bewondering voor jullie doorzettingsvermogen, en ik hoop van harte dat ik met dit doctoraat een kleine stap voorwaarts mag betekenen in de afasiediagnostiek en –therapie.

Lieve collega's, wat zou ik gedaan hebben zonder jullie? **Ilse**, **Sarah** en **Hanna**, door jullie voelde op ons bureau komen telkens weer een beetje thuis komen. Een dag alleen op het bureau voelde onwennig en eenzaam.

Ons bureau was veruit de gezelligste, zonnigste, én drukst bezochte plek van het gebouw (en vast en zeker ook het enige waar tomaten groeiden!). Het afronden van mijn doctoraat voelde eenzaam zonder jullie op twee meter afstand van me. Die drie jaar dat ik echter met jullie een bureau heb gedeeld, geven mijn herinnering aan mijn uniefperiode een gouden randje.

Evelyne, **Eva** en **Sofie**, in Utrecht hebben jullie me op sleeptouw genomen nog voor ik officieel jullie collega was. Ook heel wat daaropvolgende taalcongressen deden we samen, stuk voor stuk fijne reizen in geweldig gezelschap. Het voelde goed te weten dat ik steeds bij jullie, maar ook bij **Els**, **Charlotte, Elisah, Maaike** en **Lise** kon aankloppen voor vijf minuutjes ontspanning. Drie jaren lang deelden we elke dag lief en leed tijdens de lunch en tijdens babbeltjes tussendoor. Van taalcollega's werden we vriendinnen. Dankjewel, lieve dames. Ik kan alleen maar trots zijn dat ik in jullie voetsporen mag treden, als ik binnenkort dit doctoraat verdedig. **Michael**, op jouw aankondiging om te gaan lunchen konden we de klok gelijk zetten! Dankjewel voor de bezoekjes en babbeltjes, en voor de hulp bij de analyses! **Evy**, dankjewel voor onze fijne samenwerking, en om me wegwijs te maken op de VUB!

Dankjewel **Louisa**, **Uschi** en **Davy**. Ik trof niet één, maar drie loten uit de loterij met jullie als thesisstudenten. Ook al schrok ik toen Wouter me m'n allereerste werkdag vertelde dat ik drie thesisstudenten zou begeleiden, terwijl ik zelf nog niet eens begonnen was, ik had het niet beter kunnen treffen. Ik heb genoten van de samenwerking met jullie! Dankjewel voor jullie inzet en enthousiasme!

Dankjewel **Christophe**, **Conny**, **Linde** en **Lies**, voor de niet-aflatende ondersteuning en de fijne babbels. Dankjewel **Liliane**, voor het netjes houden van ons bureau, voor jouw aanstekelijk optimisme, en voor het openen van de deur als ik mezelf weer eens buitengesloten had!

Sarah, **Annelies** en **Katja**, jullie waren mijn vaste stop bij elk bezoek aan het UZ. Bij jullie kon ik steeds terecht voor informatie, nuttige tips en zelfs het gebruiken van jullie bureau, maar vooral voor een fijne babbel. Dankjewel!

Op m'n stage op de dienst Neuropsychologie kreeg ik de smaak van patiëntenonderzoek te pakken. **Nathalie**, **Marijke**, **Frederick**, **Iemke, Jehanne en Julie**, dankjewel om van fijn stage een rijke leerervaring, maar vooral een geweldige tijd te maken. **Gudrun**, jij verdient een extra woord van dank. Je was niet alleen mijn stagebegeleidster, maar je werd een goede vriendin. Toen ik besliste om te doctoreren op de vakgroep Experimentele Psychologie, wist ik al snel dat ik het samenwerken met jou zou missen. Maar ook daar pasten we toen een mouw aan, door samen aan een publicatie te blijven werken. Jouw authenticiteit, enthousiasme en bedachtzaamheid typeren je, en zijn voor mij altijd een grote steun geweest. Dankjewel!

Wim, ergens heb ik je altijd beschouwd als mijn soulmate op het werk. Op de één of andere manier voelden we heel snel aan dat we dezelfde ideeën, dezelfde opvattingen hebben over heel wat zaken. Hoe jij werkte en de dingen aanpakte, heeft me vaak gemotiveerd. Dankjewel!

Hyo Jung, sinds de dag dat ik je leerde kennen, ben jij een groot voorbeeld voor me geweest. Je hebt een nuchtere kijk op de dingen, je bent fantastisch goed in wat je doet, je bent een ongelooflijk harde werker en je weet onderzoek en ontspanning perfect te combineren. Eventjes kort bij jou op het bureau binnenspringen werd vaak een lange pauze. Dankjewel voor alle tips, babbels en telefoontjes, en voor de schitterende week Barça!

Eén oktober was m'n eerste werkdag, 18 oktober zat ik op het vliegtuig naar Boston, onderweg naar m'n eerste afasiecongres. **Katrien**, ik kende je al van op m'n stage, maar toen, in Boston, hebben we elkaar beter leren kennen, en heb ik gezien wat voor een ongelooflijk lief en goed persoon jij bent. De manier waarop jij met mensen omgaat, typeert en siert je. Ik prijs me gelukkig dat ik jou toen heb leren kennen, en tot mijn vriendinnen mag rekenen. Dankjewel voor alles!

Nathalie en **Sébastien**, samen zijn we in dit experimentele avontuur gestapt. Samen werkten we ons door talloze lessen, werkjes, afnames, onze thesis en stage, presentaties,... . We maakten er een gewoonte van om ons harde werk steeds te bekronen met een cocktail. Samen namen we de beslissing om te gaan doctoreren, samen vulden we onze beursaanvraag in. Samen wachtten we op goed nieuws. Samen begonnen we aan onze eerste

werkdag op de vakgroep. Ook al zijn onze levens druk bezig een andere wending aan het krijgen, ik kon me geen twee fijnere vrienden wensen om samen dit avontuur mee te maken! Dankjewel! Ik kijk al uit naar onze volgende cocktail!

Ook mijn lieve vrienden en vriendinnen buiten de universiteit verdienen een grote dankjewel. Bij jullie kon ik m'n doctoraat in perspectief zetten. Dankjewel **Linde** & **Kenneth**, **Liv**, **Sofie** & **Wouter**, **Gudrun** & **Bas**, **Frederick** & **Evy**, **Bart** & **Roswitha**, **Michiel** en **Toon**. Bedankt voor alle afleiding, gezelligheid en steuntjes in de rug. Ik beloof plechtig dat ik al m'n schade ga inhalen, en vanaf nu jullie deur platloop!

Dankjewel **meme** en **Roger**, **moemoe** en **pepe**, voor jullie hartverwarmende aanmoedigingen, en voor het steeds met heel veel interesse luisteren naar hoe ik mijn dagen vulde.

Dankjewel, lieve **Lien** en **Els**. Ik ben geweldig blij met zo'n toffe madammen als zussen! Elk op jullie manier hebben jullie me gesteund en geholpen, met lieve kaartjes, fijne uitstapjes, het nalezen van teksten, en zoveel meer. Dankjewel om te zijn wie jullie zijn, ik ben ongelooflijk fier op jullie!

Lieve **papa** en **mama**, opsommen waarvoor ik jullie dankbaar ben, zou minstens een extra hoofdstuk kunnen vormen. Ik kan me voorstellen dat het ook voor jullie een opluchting is dat dit doctoraat er eindelijk is. Van één ding mogen jullie zeker zijn, zonder jullie lag dit boekje hier nu niet. Jullie hebben me opgevangen als ik in de put zat, maar me er ook steeds weer uitgetrokken en gestimuleerd om door te zetten. Voor alle emotionele steun en praktische hulp, voor elk aanmoedigend en troostend woord, maar vooral om me graag te zien en steeds in mij te blijven geloven, ook toen ik dat zelf niet meer kon, bedankt!

Nele

Juni 2013

CHAPTER 1:
INTRODUCTION

Human language is probably the most important skill that humans have developed throughout (cognitive) evolution. Although many other species have a communication system as well, human language makes it possible to describe and refer to objects and events in the far distant past and future, to describe abstract objects and most importantly to communicate through spoken or written information.

When brain damage causes humans to lose this ability to comprehend and produce language, this is called *aphasia*. The term aphasia indicates a general impairment in the comprehension, formulation or use of verbal messages, in spoken and/or written modality. A broad range of specific deficits can occur: aphasic patients can show difficulties in comprehension or production, naming, repetition of words or sentences, or deficits in spontaneous language. The main cause of aphasia is a stroke, for example a haemorrhage or infarction. However, aphasic symptoms can be caused by a tumour, an infection, a degenerative disease, a trauma, or intoxication as well.

When an aphasic patient mastered two or more languages before the onset of aphasia, this is labelled multilingual or polyglot aphasia. It is important to notice here is that these patients are not necessarily balanced bilinguals, i.e. they do not have to master both languages to the same degree[1]. The definition most commonly used in psycholinguistic research is that of Grosjean, who defined bilingualism as 'the regular use of two (or more) languages', and bilinguals 'are those people who need and use two (or more) languages in their everyday lives' (Grosjean, 1992). Based on this definition, it was estimated that more than half of the world's population may currently be considered to be bilingual (Grosjean, 1982). In addition,

[1] Although this definition is used in the literature, all patients included in our studies were balanced bilinguals, highly proficient in both languages.

because strokes are the third most common cause of death (WHO), it becomes obvious that the wide prevalence of bilingual aphasia creates a need for a profound scientific base for the understanding of bilingual aphasia, as a basis for good assessment and therapy.

In the current dissertation, our first goal was to explore language control by non-patient bilinguals as a function of daily language switching (**Chapter two**), because language control will turn out to be crucial in the aphasia studies as well. The second goal was to investigate cross-lingual interactions and cognitive control in patients with bilingual aphasia. We studied cross-lingual interactions on different representational levels: on the word level, describing a case study (**Chapter three**) and a study with a group of patients with bilingual aphasia (**Chapter four**), and on the sentence/syntax level in a group of patients with bilingual aphasia (**Chapter five**). This way, we aimed to clarify the underlying mechanisms of language control in bilingual aphasia, more specifically in it's different forms, differential and selective aphasia, which will be introduced later.

This general introduction is structured as follows: first, we describe behavioural research and models in the field of bilingualism, more specifically concerning cross-lingual interaction at the lexical and syntactical level. We will also discuss non-linguistic (cognitive control) advantages of being bilingual. Subsequently, we discuss neuroanatomical findings and models of the bilingual brain. Finally, we focus on bilingual aphasia, and on the goals of the current dissertation.

BILINGUALISM: BEHAVIOURAL RESEARCH AND MODELS

Cross-lingual interaction at the lexical level

Empirical findings

An important debate in the psycholinguistic literature about bilingualism concerns the nature of the bilingual lexicon (i.e. the memory for lexical, or word-form, representations). Do bilinguals have a separate

lexicon for each language that they master, or are all lexical items stored in one common lexicon?

A large amount of behavioural evidence for the latter model, implying strong cross-lingual interactions, has been found in studies with cognates. Cognates are words with the same meaning and a similar form in both languages (e.g. Dutch: [boek] and English [book]). It has been found that cognates are processed faster compared to non-cognates. This is called the *cognate facilitation effect*, and it has been found to be a very robust effect. It appeared in a wide range of very different experimental tasks and settings, both in language production and comprehension. For example, Costa and colleagues found that pictures with cognate names are named faster than pictures with non-cognate names (Costa, Caramazza, & Sebastian-Galles, 2000; Costa, Santesteban, & Cano, 2005). It was also found that cognates are recognized faster than non-cognates in lexical decision tasks (Caramazza & Brones, 1979; Cristoffanini, Kirsner, & Milech, 1986). The effect was found both in the second language (L2) ànd even when reading in the native language (L1) (van Hell & Dijkstra, 2002). Surprisingly, such a cognate effect even emerged in a strict unilingual context. Using eye tracking, it was shown that Dutch-English bilinguals showed shorter fixations for Dutch-English cognates, even though they only read Dutch sentences, and did not know that English was relevant for the experiment (Van Assche, Duyck, Hartsuiker, & Diependaele, 2009). In addition, "triple" cognates (i.e. words that are similar in three languages) are processed even faster than double cognates (i.e. words that are similar in two languages) (Lemhofer, Dijkstra, & Michel, 2004). The cognate facilitation effect is considered to be a direct consequence of non-selective language activation, and it is explained by spreading activation of semantic, orthographic and phonological representations of the different languages. This cognate effect will also be used as the marker of non-target language activation in some of the present aphasia studies (chapters three and four).

Strong cross-lingual interaction effects have also been found in studies that investigated the recognition of interlingual homographs (i.e. words that share orthography but have a different meaning across languages, e.g. [room], which means *cream* in Dutch, but a part of a house in English). Dijkstra, Timmermans and Schriefers (2000) tested Dutch-English bilinguals

with a lexical go/no-go task: In the second and third experiment, participants were asked to press a button only if the word is a correct English (experiment 2) or Dutch (experiment 3) word. Some of the stimuli were Dutch-English interlingual homographs. In these selective lexical decision tasks, participants reacted slower to the homographs compared to control words, because the selective lexical decision task may not solely rely on lexical activation but instead requires a language decision, and therefore language control.

This evidence for strong interlingual interaction and language non-selective access was also confirmed in other paradigms. For instance, van Heuven et al. investigated the effect of neighbourhood size in bilinguals. The orthographic neighbourhood of a word is defined as the amount of words that share all but one letter in the same position (e.g. *hat*: [English] hot, cat, rat, has, …, but also [Dutch] kat, rat, hal, hut, …) (Coltheart, Davelaar, Jonasson, & Besner, 1977). It was found that word recognition depends on the neighbourhood size of the word in both languages (van Heuven, Dijkstra, & Grainger, 1998). Altenberg and Cairns studied English-German bilinguals performing an English-selective lexical decision task (only English words require a YES-response). Words that were illegal in English but legal in German yielded higher reaction times than words that were illegal in both languages (Altenberg & Cairns, 1983).

Theories and models

Two theoretical models have attempted to conceptualise the bilingual lexicon(s). In their Revised Hierarchical model (RHM), Kroll and Stewart (1994) suggest that bilinguals have a separate lexicon for each language, in which translation equivalents are strongly connected to each other via lexical links, and to their semantic representation via conceptual links (see also *figure 1*).

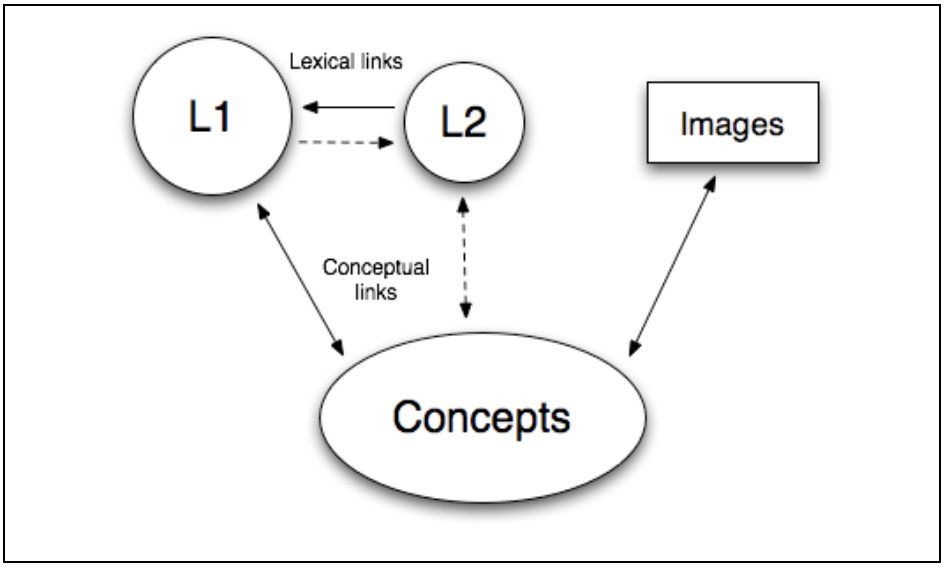

Figure 1. The Revised Hierarchical Model (RHM) of Kroll and Stewart (1994)

The connections are proposed to be bidirectional and asymmetric. The model assumes stronger lexico-semantic links for L1 words compared to L2 words. In addition, lexical links from L2 to L1 are assumed to be stronger than those from L1 to L2. It is hypothesised that the strength of these connections depends on language proficiency. In unbalanced bilinguals (i.e. bilinguals who are significantly more proficient in their L1 compared to L2) L2-L1 lexical connections are stronger compared to L1-L2 links, but the L2 lexico-semantic links are weaker compared to L1 conceptual links. With increasing L2 proficiency, L1-L2 lexical connections and L2 conceptual links become stronger. More recent research however, has also reported strong lexico-semantic links for low-proficient and beginning bilinguals (Duyck & Brysbaert, 2004, 2008). This evidence, and indications supporting a single, integrated lexicon have put the Revised Hierarchical model under pressure as the dominant model of bilingual language representation (for a comprehensive overview and theoretical discussion, see Brysbaert & Duyck, 2010).

The second model, the Bilingual Interactive Activation model (Dijkstra, Grainger, & van Heuven, 1999; Dijkstra & van Heuven, 2002) is a model that assumes one integrated lexicon for both languages (see also *figure 2*). The model focuses on visual word recognition and is in essence a bilingual version of the monolingual interactive activation model (McClelland & Rumelhart, 1981; Rumelhart & McClelland, 1982). It contains different levels: the sensory input firstly activates feature representations, which in turn send activation to letter representations. These letter representations further activate word representations in both languages. In addition, language nodes are also activated. In other words, words are interconnected within and across languages. The model is interactive in the sense that feedback from higher to lower nodes is also possible. In addition, lexical access in this model is assumed to be language non-selective, and the integrated lexicon contains words from both languages.

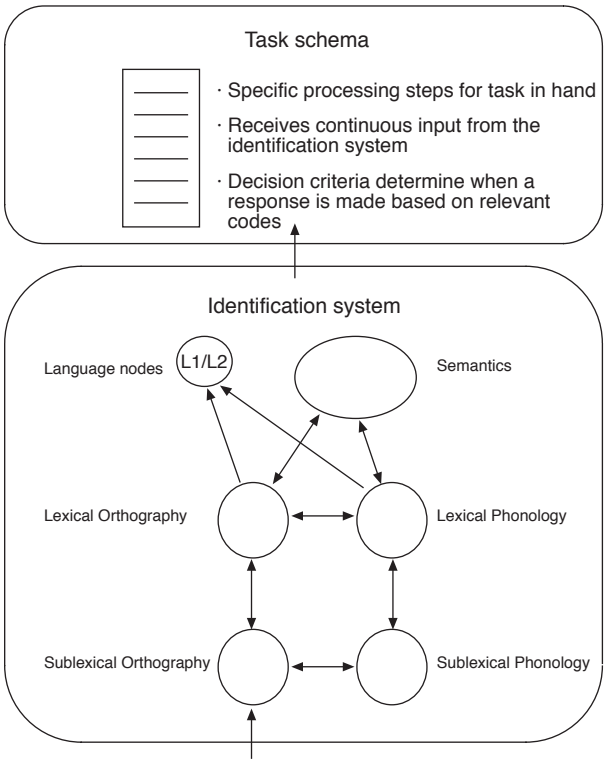

Figure 2. The Bilingual Interactive Activation (BIA+) model (Dijkstra et al., 1999; Dijkstra & van Heuven, 2002)

Cross-lingual interaction at the syntactic level

Empirical findings

In addition to the above cross-lingual interaction effects in visual word recognition at the word level, more scarce recent research on syntactic priming has shown that both languages of a bilingual are also integrated at the syntactic level. Monolingual research had already shown that the processing and production of a sentence is facilitated when the sentence is preceded by a sentence with a similar syntactic structure. For example, after hearing a passive sentence, a person will be inclined to produce a passive sentence rather than an active one. This is called syntactic priming (Bock, 1986). Importantly, syntactic priming effects have also been found across languages. Hartsuiker, Pickering and Veltkamp (2004) found syntactic priming for transitive sentences in a dialogue experiment with Spanish-English bilinguals. They found that the bilinguals produced an English (L2) passive sentence more often after hearing a passive Spanish (L1) sentence than after an active Spanish sentence (see also Hartsuiker & Pickering, 2008; Meijer & Tree, 2003; Shin & Christianson, 2009 for cross-lingual priming with datives). Cross-lingual syntactic priming has also been found in the opposite direction (L2-L1). Schoonbaert and colleagues studied syntactic priming with dative sentences in a group of Dutch–English bilinguals, and found significant priming effects within and across languages (Schoonbaert, Hartsuiker, & Pickering, 2007).

Theories and models

Based on the model of Pickering and Branigan (1998), Hartsuiker et al. (2004) proposed a theoretic framework for cross-lingual syntactic priming effects (see also *figure 3*). Pickering and Branigan claim that the lemma nodes of the words are connected to category nodes, indicating the word type, and combinatorial nodes, representing in which grammatical constructions the word can occur. When a verb can occur in an active and a passive construction, the lemma node of the verb will be connected with two different combinatorial nodes (i.e., an active node and a passive node). Thus, when the verb is used in a passive sentence, both the lemma node of the verb and the passive combinatorial node will become activated. The model further

assumes that these combinatorial nodes are shared between lemma nodes, implying that for instance every verb that can be used in the passive voice will be connected to the passive combinatorial node. Syntactic priming effects are explained as follows: hearing (or producing) a passive sentence will activate the verb and the passive combinatorial node. When the next sentence is produced, the previously activated passive combinatorial node will still be residually active, and will facilitate the subsequent production of a passive sentence. To be able to account for the findings of cross-lingual syntactic priming, Hartsuiker and colleagues extended this model to the bilingual situation, assuming that syntactic information in proficient bilinguals is shared between languages, i.e., lemma nodes of verbs in both languages are connected to the same combinatory nodes.

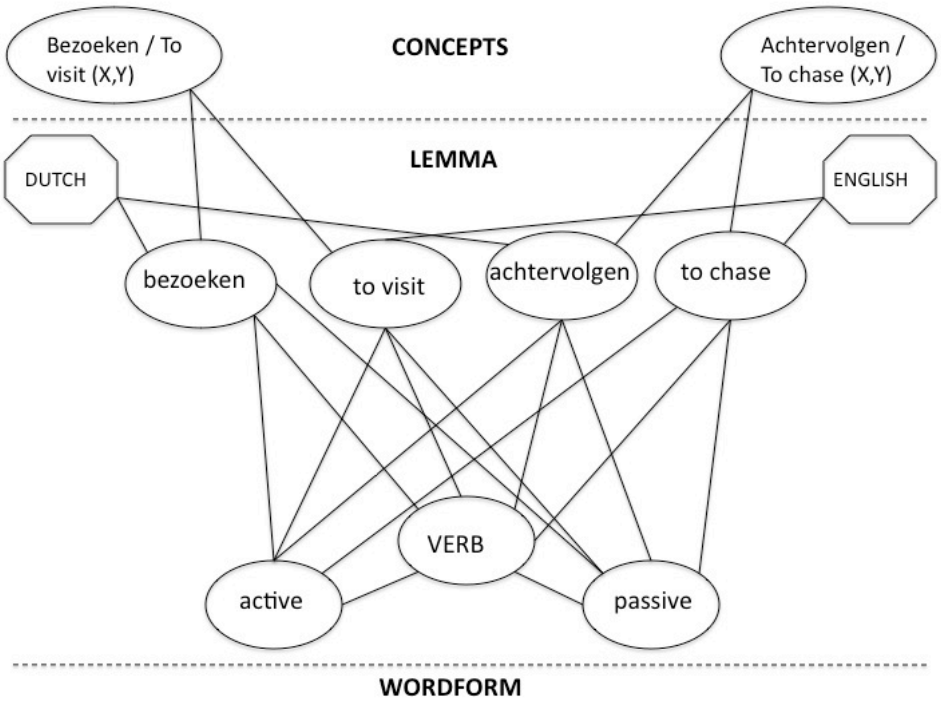

Figure 3. The model for cross-lingual syntactic priming proposed by Hartsuiker et al. (2004) applied to a Dutch-English example. In this model, the lemma nodes of the verbs [bezoeken] / [to visit] and [achtervolgen] / [to chase] are connected to a shared active combinatory node, and a shared passive combinatory node. Each lemma node is also connected to a category node for verb, and a language node (Dutch or English).

Advantages of being bilingual

Empirical findings

Given that the two language of a bilingual are highly interacting, both in word-level and syntactic representations (see above), current research has now shifted towards questions on how bilinguals control this constant dual-language activation. An effect that has yielded a lot of interest in the literature concerning bilingual language control is the bilingual control advantage. Bilinguals seem to outperform monolinguals on tasks tapping in cognitive control, even when the task is not language related. For example, it has been reported that bilinguals outperform monolinguals in a Simon task. In the Simon task, participants are asked to react on the colour of a dot on the screen (press the left button for a green dot, press the right button for a red dot). The dots appear either left or right on the screen. This results in congruent (e.g. a red dot, eliciting a right response, on the right side of the screen) and incongruent trials (i.e. a green dot, eliciting a left response, on the right side of the screen). It has been found that participant react slower on incongruent compared to congruent trials, implying a congruency effect (i.e. the difference between reaction times on incongruent and congruent trials). Interestingly, bilinguals show smaller congruency effects compared to monolinguals, which implies that they are better in suppressing the irrelevant information (i.e. the position of the dot), which is assumed to result from continuous experience in suppressing irrelevant linguistic information due to bilingualism (e.g. Bialystok, 2006; Bialystok et al., 2005; Bialystok, Craik, Klein, & Viswanathan, 2004). Similar results have been found using a Stroop task (Bialystok, Craik, & Luk, 2008) and a flanker task (Costa, Hernandez, & Sebastian-Gallès, 2008) (For a review, see Bialystok, 2009). In addition, executive control functions seem to develop earlier and stronger in bilingual children (Carlson & Meltzoff, 2008; Kovacs & Mehler, 2009; Poulin-Dubois, Blaye, Coutya, & Bialystok, 2011) and stay longer intact in bilingual elderly (Bialystok et al., 2004) compared to monolingual control subjects.

Note that this control advantage is claimed to emerge solely for competitive dual-language activation. This was investigated by Emmorey, Luk, Pyers, and Bialystok (2009), who reported the performance of

bilinguals who know two spoken languages (unimodal bilinguals) and of bilinguals who know both a spoken and a sign language (bimodal bilinguals) in such a flanker paradigm. Interestingly, only the unimodal bilinguals have to inhibit representations in the non-target language to be able to achieve lexical selection for production in the target language. Inhibition is not necessarily required in bimodal bilinguals, because they can produce both the sign and the word at the same time. Indeed, only unimodal bilinguals showed an advantage in the flanker task, suggesting that resolving interlingual competition through inhibition is important for the executive control advantage.

Interestingly, the bilingual executive control advantage was also recently challenged by a large study of Paap and Greenberg (2013). They compared fairly large groups of monolinguals and bilinguals on a wide range of 15 executive processing tasks. Although all of the tasks yielded the expected congruency or inhibition effects, none of these tasks yielded a bilingual advantage, except one task, which actually showed a bilingual disadvantage. This null effect, combined with the observation that most of the reported bilingual advantage reports indeed come from very specific (e.g. Canadian) and a limited number of bilingual populations, suggests that the bilingual advantage does not necessarily emerge from bilingualism, but instead that certain language use characteristics may be crucial for development of the control advantage. Currently however, it is unclear what these language use/learning factors are. It should be noted though that Paap and Greenberg used a L2 proficiency criterion that allowed some L2 functional knowledge, even for 'monolingual' controls. This may be sufficient for the control advantage, and therefore constitute a possible explanation for the null difference that Paap and Greenberg observed between their 'monolinguals' and bilinguals.

Prior and Gollan (2011) provided a rare attempt to identify specific language use factors responsible for the cognitive control advantage. In particular, they studied the role of language use characteristics. They compared the cognitive control performance of a group of English-Spanish bilinguals who regularly switch between languages with the performance of a group of English-Mandarin bilinguals who switch between languages less often. They only found an advantage on task switching in the bilinguals who

often switch languages. Discussing Prior and Gollan (2011), Paap and Greenberg (2013) mention switching as a factor but dismiss it as a crucial determinant, because "… *our bilinguals overwhelmingly report that they use both languages every day and switch every day… our bilinguals switch as often, if not more often, than Prior and Gollan*…". It is true that the bilinguals of Paap and Greenberg probably use their two languages every day (they did not actually assess language switching explicitly), and therefore once in a while must experience a language switch. This is very different however, from the amount of code switching that the Spanish-English bilinguals in San Diego do. In southern California, Hispanics use Spanish and English interchangeably, often multiple times within a sentence. The same occurs in Catalan-Spanish speech in the bilingual population tested by Costa and colleagues (2009; 2008). It is unclear whether this also applies to the San Francisco population of Paap and Greenberg (2013). Although their sample will certainly contain Hispanics similar to those of Prior and Gollan (numbers are not provided for each language pair), it is definitely more diverse, with 30 language pairs for 122 bilinguals, and for most of these languages, repeated code switching may not occur in everyday conversations.

Different results inspiring a range of assumptions and models concerning how a bilingual controls his/her languages have come from language switching studies, in which participants are asked to name items while switching across languages. It was found that bilinguals show a switch cost (i.e. longer reaction times in language switching trials compared to language repeating trials) (e.g. Costa & Santesteban, 2004; Hernandez, Martinez, & Kohnert, 2000). This switch cost can be asymmetric, implying that it takes longer to switch into the dominant L1 compared to the less dominant L2 (Costa & Santesteban, 2004; Meuter & Allport, 1999; Philipp, Gade, & Koch, 2007). This asymmetry seems to disappear as proficiency and use of the two languages becomes more balanced (Costa & Santesteban, 2004). Surprisingly, for speakers who are highly proficient in L1 and L2, but less proficient in L3, there was no asymmetry when these bilinguals switched between L1 and L3 or between L2 and L3 (Costa, Santesteban, & Ivanova, 2006). Because these switching effects fall outside the scope of the current dissertation, we will not go into further detail. However, they have

played a crucial role in the development of models concerning bilingual language control, as will be described in the next section.

Theories and models

In spite of the consensus that access to concepts is a language non-selective process (Dijkstra & van Heuven, 2002; van Heuven et al., 1998) and that this also seems to be the case for word-form (lexical) information (e.g. Dijkstra et al., 2000; Duyck, Van Assche, Drieghe, & Hartsuiker, 2007; Lemhofer & Dijkstra, 2004; van Hell & Dijkstra, 2002; van Heuven et al., 1998), the nature of the mechanism that manages bilingual language control is still a matter of debate.

According to the Inhibitory Control model (IC model, Green, 1998) individuals must resolve competition between language task schemas. Green proposed that the selection of a language involves the inhibition of the unintended language. Green explained switch costs by assuming that it reflects persisting inhibition of the previously irrelevant language. In addition, it is hypothesised that the difference in switching costs between both languages reflects the relative dominance of the two languages. Inhibiting a more dominant language (e.g. L1) requires more cognitive resources than inhibiting a less dominant language (e.g. L2 in an unbalanced bilingual), and switching back into a dominant language will therefore be harder. Green also assumed that the language control mechanism is a part of the general executive control system, managing both language related and language non-related control.

Other models, however, do not assume inhibitory mechanism but rather assume that the lexical selection mechanism is sensitive to the language membership of lexical representations. This is called the language specific selection hypothesis: only the lexical representations that belong to the response language are considered for selection (Costa & Caramazza, 1999; Costa, Miozzo, & Caramazza, 1999). Costa and Santesteban (2004) further tried to reconcile both views, suggesting that the nature of control may depend on proficiency. For instance, individuals who are sufficiently proficient in L2 to access lexical concepts in L2 without L1 mediation (see Kroll & Stewart, 1994), language cues may be sufficient to ensure correct

selection. So, with high levels of proficiency, bilinguals select the relevant language without competition from the other language. However, low-proficient L2 learners still need to make use of inhibitory mechanisms, as was suggested in the IC model.

The present dissertation

An important effect in the literature concerning bilingualism that was highlighted in the introduction is the finding that bilinguals seem to outperform monolinguals on executive control tasks. This effect, however, does not seem to generalize across bilinguals and tasks. The specific factors that determine the advantage are not yet investigated extensively.

In the first empirical chapter of this dissertation we further elaborated the role of language switching in the development of the bilingual executive control advantage **(Chapter two),** in order to achieve a better understanding of language control in bilinguals before assessing the issue of language control in bilingual aphasia. We extended the study of Prior and Gollan (2011), who found that only English-Spanish bilinguals, who often switch languages, showed an advantage on task switching. English-Mandarin bilinguals, who do not often switch languages, did not show a better performance. Our study differed on two crucial elements with the study of Prior and Gollan. First, we investigated whether the frequency of language switching in daily life not only influences task switching, but enhances inhibition as well (by means of a flanker task and a Simon Arrow task). In addition, and more importantly, we also studied the influence of the characteristics of the language pairs. Prior and Gollan assumed that only bilinguals who often language-switch (in this case the English-Spanish bilinguals) train their executive control capacities, yielding better performance on executive control tasks. However, these two experimental groups (English-Spanish and English-Mandarin bilinguals) do not only differ in their amount of switching between languages, but also in the amount of overlap between these languages. Because languages that share orthography (in this case: English and Spanish, both alphabetic languages) and language pairs with a distinct script (English and Mandarin) require different representational structures (Gollan, Forster, & Frost, 1997) and hence also control demands, it is plausible that the bilingual advantages arising from

competition between these two language pairs also differ. Indeed, task switching research has shown that switching between overlapping cognitive tasks (e.g., by using bivalent stimuli) causes a much greater switch cost than switching between tasks that share less task features (Rogers & Monsell, 1995). Therefore, the higher switch cost for the Mandarin-English group in the Prior and Gollan study does not necessarily reflect the fact that they switch less often between languages, but may be alternatively explained by the smaller lexical overlap, between Mandarin and English.

THE BILINGUAL BRAIN: FINDINGS AND MODELS

The neural representation of languages in a bilingual

Neuroimaging and neuroanatomical research has shown that the two languages of a bilingual are not only functionally interaction, but also (at least) integrated at the neural level. Ojemann and Whitaker (1978) used intra-operative cortical stimulation to investigate the localisation of languages in the brain, and found both shared and distinct brain areas for the representation of L1 and L2. This was confirmed by studies that specifically studied reading, counting and word retrieval (Roux & Tremoulet, 2002) and naming (Bello et al., 2006; Lucas, McKhann, & Ojemann, 2004; Walker, Quinones-Hinojosa, & Berger, 2006) (For a review, see Giussani, Roux, Lubrano, Gaini, & Bello, 2007). The larger part of the fMRI studies investigating brain activation during language related tasks in different languages have shown activation in common brain areas for both languages (Briellmann et al., 2004; Chee, Tan, & Theil, 1999; Hernandez et al., 2000; Illes et al., 1999; Kim, Relkin, Lee, & Hirsch, 1997 only in early bilinguals; Mahendra, Plante, Magloire, Milman, & Trouard, 2003; Pu et al., 2001; Vingerhoets et al., 2003). Some studies however found distinct cortical areas representing both languages (Dehaene et al., 1997; Kim et al., 1997 (in late bilinguals)). Similar results have been obtained with older PET technology: both common areas (De Bleser et al., 2003; Klein, Milner, Zatorre, Zhao, & Nikelski, 1999; Klein, Zatorre, Milner, Meyer, & Evans, 1994) and distinct cortical (Perani et al., 1996) areas have been found to be activated during task in both languages.

Taken together, the existing neuroimaging literature suggests that both languages of a bilingual are represented in both shared and distinct brain areas. This is called the amalgamated hypothesis. It has been argued that the variability in the reported results could be due to the variability in *AoA* and *proficiency* of the participants. For example, Kim et al. only found activation in common brain areas in early bilinguals (i.e. bilinguals who acquired both languages before the age of six), whereas in late bilinguals (i.e. bilinguals who learned their L2 after the age of six), distinct brain regions were activated. In addition, various studies report shared patterns of left hemispheric activation in balanced bilinguals (i.e. bilinguals who are equally proficient in both their languages) (Chee et al., 1999; Klein et al., 1999), whereas unbalanced bilinguals often show additional activation, more specifically in prefrontal areas (Briellmann et al., 2004; De Bleser et al., 2003) (See Abutalebi, Cappa, & Perani, 2001 for an overview).

Obviously these two variables, AoA and proficiency, are strongly correlated, which makes it harder to disentangle their role in the activation pattern (See Perani & Abutalebi, 2005 for an overview). Two models concerning the neuroanatomical representation of both languages in a bilingual have been proposed.

Neuroanatomical theories and models concerning bilingual language representation

We discuss two views on how bilingual language control might be achieved in the brain. In his procedural/declarative model, Ullman (Ullman, 2001a) states that languages rely on two separate memory systems. The procedural memory system can be situated left frontally and subcortically in the brain, and is mainly involved in implicit knowledge. More specifically for language this concerns grammatical knowledge of L1, which is typically acquired implicitly and automatically. The declarative memory system is located in bilateral temporo-parietal areas, and is associated with more explicit knowledge, acquired by formal education. In the language domain, this concerns words and their meaning. This model was later extended to second language acquisition, and Ullman proposed that L2 grammar initially relies more strongly on the declarative system, because it consists of a set of rules, often learned by formal education (Ullman, 2001b). However, as L2

proficiency increases, L2 processing will become more automatic and make more use of the procedural memory. However, according to this model, L2 processing will always, compared to L1, continue to appeal more to the declarative memory, leading to a more extended cerebral representation.

A different claim is incorporated in the single adaptive network view: this view assumes that the existing systems mediating syntax or morphology are used right from the start of L2 acquisition (i.e. convergence, Green, 2003). With increasing proficiency, more controlled processing shifts to more automatic language processing (Abutalebi & Green, 2007; Green, Crinion, & Price, 2006; Green, 2003). This is also in line with the view of Paradis (2004) and Lebrun (2002), who also identify differences between L1 and L2 processing in terms of the greater automatisation of L1 and the implicitness of L1 across both lexical and syntactic aspects of language.

A number of neuroanatomical circuits have been identified in bilingual language control. Fabbro et al. (Fabbro, Peru, & Skrap, 1997) describe an organisation of networks, among others a planning network. This network consists of the prefrontal cortex, the caudate, the globus pallidus and the ventral anterior thalamic nucleus. Abutalebi and Green (2007) identified a network of four main regions involved in (bilingual) language control (see also *figure 4*). The main brain region responsible for (language) control is the prefrontal cortex, which is also involved in decision making, working memory, planning, sequencing of behaviour, response inhibition, language and attention. The prefrontal regions are supposed to function as a top-down mechanism, facilitating the processing of task relevant representations, even in the presence of prepotent, irrelevant stimuli. The prefrontal cortex is strongly connected to the parietal cortex, which is important for the selection of competing responses. In addition, they assume that the anterior cingulate cortex (ACC) plays an important role in tasks that tap into selective attention, working memory, language generation and controlled information processing, but most importantly, the ACC is assumed to be involved in conflict processing. The last main region in the control network are the basal ganglia, associated with sequence planning and motor control of language.

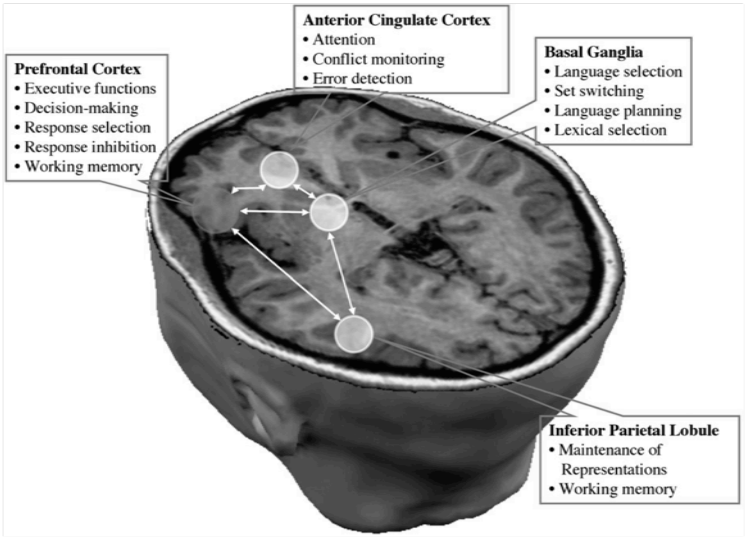

Figure 4. Schematic overview of the bilingual language control network described by Abutalebi and Green (2007)

Following previous research and based on the models of Ullman (Ullman, 2001a, 2001b) and Green (2005) (cfr. suppra), Abutalebi and Green (Abutalebi & Green, 2007) suggest that the representation of L1 and L2 mainly depends on proficiency. In their review they concluded that low L2 proficiency is correlated with higher brain activation in L2 processing, but not only in language related brain areas, but also in cognitive control areas such as the prefrontal and inferior parietal cortex, and the ACC (Liu, Hu, Guo, & Peng, 2010; Yetkin, Yetkin, Haughton, & Cox, 1996). This follows from the need for language control in bilinguals: because both languages of a bilingual are represented in common brain areas, and because both languages are always activated to a certain degree and functionally interacting (see above), a mechanism is needed for selection of the target language and inhibition of the non-target language, in order to solve the competition between translation equivalents (e.g. when naming a picture). Importantly, Green suggested that the mechanism responsible for language control is not a distinct mechanism, but rather a part of a general (executive) control mechanism, involved in both language related and non-language

related control (Green, 1998). Cognitive control processes, or executive control, are mental processes responsible for goal-directed behaviour, for example to be able to stop suddenly, or to adapt the behaviour to environmental circumstances (van den Wildenberg & Ridderinkhof, 2009). Inhibition is the executive function that allows us to stop suddenly, and to suppress initiated behaviour.

BILINGUAL APHASIA: CURRENT STATE OF AFFAIRS

Patterns of impairment and recovery in bilingual aphasia

When brain damage causes humans to lose the ability to comprehend and produce language, it is called aphasia. In contrast with what intuitively might be expected, and also what may be derived from an (functionally and neurally) integrated language processing view, a bilingual suffering from aphasia does not always show the same *impairments* in the different languages that he/she masters, nor do they *recover* in a similar way and in the same time. Paradis desribed six different patterns in which recovery from aphasia in bilinguals can occur (Paradis, 1977). The most common pattern is *parallel recovery*, with both languages recovering in a similar way, given pre-onset proficiency (Alexiadou & Stavrakaki, 2006; Gil & Goral, 2004; Marangolo, Rizzi, Peran, Piras, & Sabatini, 2009; Mastronardi, Ferrante, Celli, Acqui, & Fortuna, 1991). When recovery in one language is more pronounced than in the other, this is called *differential recovery* (Goral, Levy, Obler, & Cohen, 2006; Ibrahim, 2009; Meinzer, Obleser, Flaisch, Eulitz, & Rockstroh, 2007; Vajramani, Akrawi, McCarthy, & Gray, 2008). An extreme case of differential recovery is *selective recovery*. This is the case when recovery is limited to only one language. Aglioti and Fabbro (1993) described such a case, a 70-year-old right-handed woman who suffered from a global mutism caused by a subcortical laesion in the left basal ganglia (Agliotti & Fabbro, 1993). The mutism evolved into selective aphasia, enabling the patient only to communicate in standard Italian, but still showing the mutism in Venetan, a Veronese dialect in which she was raised and which she used for daily communication (See also Agliotti, Beltramello, Girardi, & Fabbro, 1996).

The fourth recovery pattern described by Paradis is *blended recovery*. These patients continuously switch between their languages, and/or mix elements from both languages. For example, S.J. was a Friulian/Italian bilingual man with a lesion in the left prefrontal cortex, a part of the anterior cingulate and the left striatum (Fabbro, Skrap, & Aglioti, 2000). The patient showed normal comprehension in both languages. However, when asked to talk Italian, he repeatedly switched to Friulian and vice versa (See also (Adrover-Roig et al., 2011; Leemann, Laganaro, Schwitter, & Schnider, 2007; Marien, Abutalebi, Engelborghs, & De Deyn, 2005; Riccardi, Fabbro, & Obler, 2004) for more case descriptions of patients showing blended recovery).

Patients in whom one language only starts to recover when the other one has fully recovered, show *successive recovery*. Very sporadically, there seems to be an alternation in recovery in both languages. In this case initially, only one language recovers. However when the other language starts to recover, the firstly discovered language starts to weaken again. This is called *antagonistic recovery*. Both recovery patterns have been hypothesized by Paradis (Paradis, 1977), but to the best of our knowledge, no clinical cases showing these recovery patterns have been described yet.

Data concerning the incidence of these patterns have not yet reached a consensus. Fabbro (1999) only distinguished between parallel and non-parallel recovery, and reported that only 40% of the patients showed parallel recovery. In addition, from the patients that showed non-parallel recovery, 32% showed better recovery in L1, and 28% in L2. Similarly, Paradis (2001) claimed that parallel recovery was the most common recovery pattern (61%), followed by differential recovery (18%), pathological switching and mixing (9%), selective recovery (7%) and successive recovery (5%).

Analogously to the *recovery* patterns described by Paradis, a similar conceptualisation could also be used to describe the impairments in both languages. Recently, Ibrahim (2009) described a 41-year-old right-handed and highly educated man whose first language (L1) was Arabic. Around the age of nine he had acquired Hebrew (L2). He used Hebrew in academic, professional and personal contexts, and therefore developed a very high premorbid proficiency. Due to an infection causing an intracranial and

subdural haemorrhage he suffered from aphasia with non-fluent language production, grammatical impairments and anomia. Remarkably, these deficits were present in both languages, but more pronounced in Hebrew (L2). In analogy with differential recovery (i.e. recovery is more pronounced in one language compared to the other), this pattern of more severe impairments in one language compared to the other could be called *differential aphasia*. Similarly, Vajramani et al. (2008) described a patient with differential aphasia caused by a subdural haemorrhage who had minimal impairments in his mother tongue (Arabic), but global impairments in his L2 (English). Goral et al. described a trilingual aphasic patient who mastered Hebrew (L1), English (L2) and French (L3). He suffered from aphasia caused by a left fronto-temporo-parietal laesion, showing serious word finding difficulties. These were mostly apparent in French, and to a lesser degree in Hebrew and English (Goral et al., 2006). Other cases of differential recovery were described by Adrover-Roig et al. (Adrover-Roig et al., 2011), Hernandez et al. (Hernandez et al., 2008), Mastronardi et al. (Mastronardi et al., 1991) and Laine et al. (Laine, Niemi, Koivuselkasallinen, Ahlsen, & Hyona, 1994). As for *selective aphasia* (i.e. patients who show impairments in *only one* language), Ku, Lachmann and Nagler (1996) described a patient who had lost all comprehension and production in English (L2), but still showed normal language skills in Mandarin (L1). Garcia-Caballero et al. described a 91-year-old woman who was only able to speak in L2 (Spanish), showing a mutism in L1 (Galician) (Garcia-Caballero et al., 2007) (See also Agliotti & Fabbro, 1993). When both language of a bilingual aphasic patient are affected to the same extent, we can call it *parallel aphasia* (Agliotti et al., 1996; Gil & Goral, 2004; Mastronardi et al., 1991).

Patterns of bilingual aphasia: towards a theoretical explanation

Until now, despite much speculation, the factor that determines how, and to what extent, impairment and recovery in both languages will occur is still unknown. For instance, Pitres' law (1895) poses that the most frequently used language will recover the first and the best, irrespective of the fact whether this was learned first or not. According to Ribot's law (1881), the first-acquired language (L1) will always recover best, analogously to

retrograde amnesia, in which the earliest memories will be preserved the best. Other proposed factors were the language spoken in the hospital (Bychowsky, 1919), the language with which the patient has the strongest affective bond (Minkowski, 1928, 1965), or the language that is of most use for the patient (Bay, 1964; Goldstein, 1948). In addition, language status (L1 or L2, language that is most used, …), site or type of the lesion, type of aphasia, how the language was acquired, age, proficiency, type of bilingualism, … have also been proposed to be determining factors. However, none of these factors or laws has been found to provide a sufficient explanation for the development of the impairment and recovery patterns as described in the literature. Especially the cases with selective or much more pronounced damage (differential aphasia) to one of the languages remain difficult to explain.

Within the field of neurolinguistics, the recovery and impairment pattern was assumed to just reflect language specific neural damage. Because it was initially assumed that every language of a multilingual is represented in a distinct brain area (Dehaene et al., 1997; Perani et al., 1996; Pouratian et al., 2000; Simos et al., 2001), cases of differential and selective recovery in patients with bilingual aphasia were typically explained by lesions in the brain area in which the (most) affected language was represented (Albert & Obler, 1978). This has been called the *localised explanation*. For example, the procedural/declarative model (Ullman, 2001a, 2001b) might be able to explain selective impairment of L1 without impairments of L2, when initial pre-pathological L2 proficiency was low: because L1 is represented both in the declarative and in the procedural system, and as the weaker L2 is then only represented in the declarative memory system, damage to the procedural memory (in frontal and basal ganglial areas) will only cause impairments in L1. In this view, specific impairments in L2 processing are much harder to explain, because the declarative system it relies on is also always important for L1. However, claiming a differential representation of grammar in L1 and L2, as in this model, does not seem to be entirely consistent with the existing literature (for an overview, see Green & Abutalebi, 2008; see also the the integrated syntactic representation model of Hartsuiker et al., 2004). In addition, because the bulk of recent behavioural and neuroimaging evidence suggests

that both languages rely on the same (or at least highly overlapping and interacting) brain areas, this localised explanation for selective/differential aphasia seems to be challenged.

Pitres (Pitres, 1895) was the first to associate the different recovery patterns with a problem in language control. Based on the observation that patients recover their "lost" language faster than learning a new one, he stated that the language had not been lost, but instead had become functionally unavailable due to an excessive inhibition. This idea was later incorporated in a dynamical explanation of bilingual aphasia, posing that a problem in cognitive control may underlie the different recovery patterns in bilingual aphasia, rather than only damage to language areas itself.

Recently, Pitres' hypothesis was brought to life again by Green and Price (2001), who also suggested that deficient inhibitory functions might underlie the different impairment and recovery patterns in bilingual aphasia, more specifically a deficit in the selective suppression (and activation) of the languages. As such, selective recovery can be regarded as a continuous activation of the recovered language, reflecting the inability to suppress it. So, the more impaired language is not lost in itself, but rather is the ability the inhibit the recovered, stronger language. Or it can be a continuous inhibition, i.e. a raised activation threshold for the non-recovered language. Successive recovery might be caused by a temporal inhibition of one language, whereas antagonistic recovery might be reflecting a shifting inhibition (Paradis, Goldblum, & Abidi, 1982). The inability to selectively inhibit languages might result in mixed recovery with pathological switching and mixing (Fabbro et al., 2000).

Although this hypothesis may offer a theoretically reasonable explanation for the existence of differential/selective aphasia, empirical evidence supporting this language control account is mostly completely lacking. It is the aim of this dissertation to offer such evidence. If indeed a control deficit underlies selective and differential aphasia, we would expect that patients with selective and differential aphasia, but not patients with parallel aphasia, show impairments in control tasks, not only in language related tasks but in language unrelated tasks, given that language control relies on the same neural system as non-linguistic cognitive control (cfr.

Green, 1998). At the same time, we would expect them to still show effects of cross-lingual interactions, even though one of the languages is severely damaged. That is, even the language that is functionally most impaired should be able to influence processing of the best-preserved language, which is a quite challenging prediction.

The present dissertation

At the domain of behavioural research in bilingualism, the existing literature shows that both languages of a bilingual are constantly activated to a certain level. The knowledge of another language constantly influences language processing, even in L1. These effects are called cross-lingual interactions, and have been found both at the level of lexical (word) processing and at the level of syntax processing.

In the present dissertation, we will investigate cross-lingual interaction in patients with bilingual aphasia, who seem to have lost one of their languages to a greater extent. More specifically, we will study the cognate effect as a marker of cross-lingual interaction at the lexical level (**Chapter three and four**), and the syntactic priming effect as a marker of cross-lingual interaction at the syntactic level (**Chapter five**).

First, we compared the performance of a patient with differential aphasia that we found early during this doctoral project with non-patients controls on a flanker task and two versions of a lexical decision task (**Chapter three**). We included a generalised lexical decision task (press a button if the word is an existing word in any language) and a selective lexical decision task (press a button only if the word is an existing word in the target language), based on the method of Dijkstra and colleagues (Dijkstra et al., 2000). We opted to use both versions of lexical decision because of the difference in control demands. Because a generalised lexical decision task requires a YES-response for words from both languages, whereas words in the non-target language require a NO-response in a selective lexical decision task, these tasks differ in terms of language control demands. The selective lexical decision task imposes much more cross-lingual competition than the general lexical decision task, in which no language selection/decision has to be made. This will allow us to study

cross-lingual interactions in bilingual aphasia at different levels of language control requirements.

Secondly, we compared the performance of a large group of patients with parallel aphasia and a large group of patients with differential aphasia, recruited over the long term, with a group of healthy control subjects on a generalised lexical decision task with cognates and on a flanker task (**Chapter four**). By means of the flanker task, we aimed to investigate the non-linguistic executive control functions of the patients with bilingual aphasia. If an executive control deficit indeed underlies selective and differential aphasia, we would expect that the patients with differential aphasia show a worse performance on the flanker task compared to patients with parallel aphasia. However, because a control deficit rather than damage to language specific brain areas would lead to differential aphasia, we would still expect these patients to show effects of cross-lingual interaction in situations in which language control is not important. This is what we examined using the lexical decision task with cognates. As we described earlier, the cognate effect can be regarded as a marker of cross-lingual interaction. At the one hand, we assessed this cognate effect in a generalised lexical decision task, in which words from any of the bilingual's languages, required a YES-response, and non-words required a NO-response. We chose this task because there is no need to inhibit representations in either of the languages (words in any language require a YES-response). Therefore, the (language) control demands are very low, making this task very suited to selectively tap into lexical interactions, without performance being affected by any control problem. We expected the patients to show cognate effects, just as the control subjects, and we expected both groups of patients (differential vs. parallel) to show similar effects. This pattern of results would support the hypothesis that both languages of a patient with (differential) bilingual aphasia are still intact in terms of neural lexical representation, and that an executive control deficit underlies the differential symptoms in both languages.

Whereas chapters three and four investigate cross-lingual interaction at the word level, the last empirical chapter surpassed the word level and assessed another, more high-level representational layer of language, looking at syntactic priming in patients with parallel and differential aphasia

(**Chapter five**). As was described earlier, syntactic priming is the phenomenon in which individuals are inclined to re-use the previously heard or produced syntactical structure. Only a few studies investigated syntactic priming in patients with aphasia: Saffran and Martin (1997) found significant priming effects with transitive (i.e. active and passive) sentences in patients with impaired sentence production. This was replicated by Hartsuiker and Kolk (1998), who found significant syntactic priming effects with transitive and dative sentences in patients with Broca aphasia. We aimed to extend these findings investigating syntactic priming effects across languages. In the fifth chapter of this dissertation, we asked the following three research questions: (1) Do patients with bilingual aphasia show priming effects within and across languages? (2) Do these priming effects differ from the priming effects observed in control participants? and (3) Does the pattern of priming effects interact with the type of aphasia (parallel and differential aphasia)? Based on previous studies showing cross-lingual interaction in patients with bilingual aphasia, we expected both patient groups to still show syntactic priming, both within and across languages. This would support the hypothesis that also differential aphasia patients retain activation in the most impaired language, also in higher (i.e. syntactic) levels of linguistic representations.

Taken together, the aim of the present dissertation is to to clarify the underlying mechanism in the development of differential vs. parallel aphasia, by further disentangling whether and how both languages of a bilingual aphasic patient interact (at lexical and syntactic levels), and by assessing the role of (non-linguistic) executive control functions.

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CHAPTER 2:
THE INFLUENCE OF LANGUAGE SWITCHING EXPERIENCE ON THE BILINGUAL EXECUTIVE CONTROL ADVANTAGE[1]

It has been found that bilingualism enhances non-linguistic executive control. The goal of this study was to investigate the influence of language switching experience, rather than language proficiency, on this bilingual executive control advantage. We compared the performance of unbalanced bilinguals, balanced non-switching, and balanced switching bilinguals on two executive control tasks, i.e. a flanker and a Simon task. We found that the balanced switching bilinguals outperformed both other groups in terms of executive control performance, whereas the unbalanced and balanced non-switching bilinguals did not differ. These findings indicate that language switching experience, rather than high second-language proficiency, is the key determinant of improved executive control performance in bilingualism.

[1] Verreyt, N., Vandelanotte, D., Szmalec, A., & Duyck, W. The influence of language switching experience on the bilingual executive control advantage. *(submitted to Bilingualism: Language and Cognition)*

INTRODUCTION

About 50% of the world population is considered to be bilingual (Grosjean, 1989). Besides the obvious communicative advantage, several associated and even non-linguistic cognitive benefits of bilingualism have recently been explored. One well-replicated advantage is the finding that bilinguals show improved performance on a broad range of executive control tasks. "Executive control" refers to a range of high-level control functions that support goal-directed behaviour. Three main control functions can be identified: inhibition, updating and shifting (Miyake et al., 2000). In what follows, we will summarise earlier evidence pointing towards bilingual advantages for tasks assessing inhibition and switching functions.

It has been found that bilinguals outperform monolinguals on a range of tasks tapping into inhibition. Bialystok, Craik, and Luk (2008), for example, observed that bilinguals outperform monolinguals on a Stroop task, an inhibition task in which participants have to name the ink colour of colour words (e.g., the word *green* printed in red), while suppressing the natural tendency to read the colour word. Another measure of inhibition is the Eriksen flanker task (Eriksen & Eriksen, 1974). This task requires participants to react to the direction of the central of five arrows (<<><<), while trying to ignore the direction of the four flanking arrows. Bilinguals have been found to outperform monolinguals on this task as well (Costa, Hernandez, & Sebastian-Gallès, 2008). The positive effect of bilingualism on inhibitory control tasks also seems to be an effect that emerges throughout the lifespan. It has been found that bilingual children already show enhanced performance compared to their monolingual peers on tasks tapping into inhibition (Carlson & Meltzoff, 2008). In addition, the advantage remains consistent in bilingual elderly (Bialystok, Craik, Klein, & Viswanathan, 2004; Bialystok et al., 2008).

A highly influential cognitive account of bilingualism and bilingual language control is the Inhibitory Control model (Green, 1998). This model

assumes that bilinguals experience a continuous competition (conflict/interference) between lexical representations of both languages, which are always active to a certain degree in speaking (Hermans, Bongaerts, De Bot, & Schreuder, 1999), reading (Van Assche, Duyck, Hartsuiker, & Diependaele, 2009) and listening (Lagrou, Hartsuiker, & Duyck, 2011). To resolve this competition, control resources are recruited to inhibit the conflicting activation of the non-target language. Importantly, these inhibitory mechanisms seem to be domain-general[2], so that experience in managing competition in the linguistic context also transfers to non-linguistic tasks (Bialystok et al., 2005; Bialystok, Craik, & Ryan, 2006; Colzato et al., 2008; Costa et al., 2008; Martin-Rhee & Bialystok, 2008). The central role for inhibition also becomes clear from a study by Emmorey, Luk, Pyers, and Bialystok (2009), who reported the performance of bilinguals who know two spoken languages (unimodal bilinguals) and of bilinguals who know both a spoken and a sign language (bimodal bilinguals) in such a flanker paradigm. The clever manipulation here implies that only the unimodal bilinguals have to inhibit representations in the non-target language to be able to achieve lexical selection for production in the target language. Inhibition is not necessarily required in bimodal bilinguals, since they can both execute the sign and produce the word, even simultaneously if needed. And, indeed, only unimodal bilinguals showed an advantage in the flanker task, suggesting that resolving interlingual competition through inhibition is important for the executive control advantage.

Interestingly, the bilingual advantage on tasks tapping into inhibition is not only measurable on trials that involve competition between relevant and irrelevant information (like incongruent trials or switch trials) but also on trials that require a simple choice reaction without any cognitive conflict (like congruent trials or non-switch trials) (Costa et al., 2008). This finding suggests that the cognitive benefits of bilingualism are not restricted to one specific executive control function, but may be extended to the entire,

[2] Whether the EC processes put at play by bilingual language control are fully subsidiary of domain-general EC processes is still a matter of debate. Some studies did not find any correlation between linguistic and non-linguistic tasks (Bialystok, Craik, & Luk, 2008; Calabria, Hernandez, Branzi, & Costa, 2012). However, this issue goes beyond the objective of the present article.

domain-general executive control system. Indeed, besides inhibitory control, bilinguals have also been found to show an advantage on tasks tapping into shifting. It has been found that bilinguals show an advantage on task shifting, i.e., showing smaller switch costs[3] compared to monolinguals (Bialystok & Viswanathan, 2009; Garbin et al., 2010; Prior & Gollan, 2011; Prior & MacWhinney, 2010).

Based on the findings that (a) the bilingual advantage does not only appear in conflict trials, but also in non-conflict trials, and that (b) bilinguals also show enhanced performance on other executive functioning tasks, which do not necessarily tap into inhibition, it was suggested that mastering two languages not only enhanced inhibitory control, but leads to improved executive control functions in general.

Importantly, the mere fact of knowing two languages does not always suffice for enhancing executive control functioning. Luk, De Sa and Bialystok (2011) administered a flanker task in a group of monolinguals, late bilinguals and early bilinguals. Only the early bilinguals showed better performance on the control task; no difference was found between the late bilinguals and the early bilinguals. So it seems that being bilingual per se does not suffice to enhance performance on executive control tasks.

Interestingly, the bilingual executive control advantage was also recently challenged by a large study of Paap and Greenberg (2013). They compared fairly large groups of monolinguals and bilinguals on a wide range of 15 executive processing tasks. Although all of the tasks yielded the expected congruency or inhibition effects, none of these tasks yielded a bilingual advantage, except one task, which actually showed a bilingual disadvantage. This null effect, combined with the observation that most of the reported bilingual advantage reports indeed come from very specific (e.g.

[3] It is important here to notice the difference between the switch cost in a task or language switching task, and a "Gratton type" switch cost (i.e. in congruency tasks, the performance on trials following an incongruent trials are worse compared to trials following a congruent trials). It is not yet clear that bilinguals outperform monolinguals on this "Gratton type" switch cost (the ability of going back and forth between different types of trials) (Costa, Hernandez, Costa-Faidella, & Sebastian-Galles, 2009; Costa, Hernandez, & Sebastian-Gallès, 2008).

Canadian) and a limited number of bilingual populations, suggests that the bilingual advantage does not emerge from bilingualism in itself, but instead that certain language use characteristics may be crucial for development of the control advantage. Currently however, it is unclear what these language use/learning factors are.

In the current paper, we aim to further clarify what bilingual parameters may be crucial for development of the bilingual control advantage. More specifically, we further investigated the role of language switching in daily life. It was already suggested in the paper of Emmorey and colleagues (2009) that the amount of (code) switching might underlie the bilingual executive control advantage. It was hypothesised that the difference in control performance between unimodal and bimodal bilinguals could be due to the fact that unimodal bilinguals have to code switch in their communication, whereas bimodal bilinguals prefer to produce both the sign and the word (i.e. code blend), therefore rarely switching between languages. In addition, Prior and Gollan (2011) compared the performance of a group of bilinguals who regularly switch between languages with the performance of a group of bilinguals who switch between languages less often. They only found an advantage on task switching in the bilinguals who often switch languages. Discussing Prior and Gollan (2011), Paap and Greenberg (2013) discuss switching as a factor but dismiss it as a crucial determinant, because "*… our bilinguals overwhelmingly report that they use both languages every day and switch every day… our bilinguals switch as often, if not more often, than Prior and Gollan…*". It is true that the bilinguals of Paap and Greenberg probably use their two languages every day (they did not actually assess language switching explicitly), and therefore once in a while must experience a language switch. This is very different however, from the amount of code switching that the Spanish-English bilinguals in San Diego do. In southern California, Hispanics use Spanish and English interchangeably, often multiple times within a sentence. The same occurs in Catalan-Spanish speech in the bilingual population tested by Costa and colleagues (2009; 2008). It is unclear whether this also applies to the San Francisco population of Paap and Greenberg (2013). Although their sample will certainly contain Hispanics similar to those of Prior and Gollan (numbers are not provided for each language pair), it is definitely more

diverse, with 30 language pairs for 122 bilinguals, and for most of these languages, repeated code switching may not occur in everyday conversations.

Above, we have summarised evidence suggesting that bilinguals develop more performant general control abilities because they must control the continuous interference between lexical representations associated with both languages, and discussed what factor may contribute to this advantage. The primary aim of our study is to gain novel insight into the mechanisms that underlie the bilingual executive control advantage, by investigating the role of language switching experience. From a memory perspective, the interference between languages comprises competition between active lexical representations of those languages in long-term memory. As described in the memory literature (Oberauer, 2009), memory contents have the potential to cause interference when they are in an active state, but once the activation starts to decay, interference effects also rapidly disappear (Szmalec, Verbruggen, Vandierendonck, & Kemps, 2011). Therefore, we predict that the bilingual advantage originating from the competition between languages should primarily occur in bilinguals who show similarly strong activation in lexical representations of both languages at the same time, i.e. bilinguals who use both languages interchangeably within the same context, and often switch languages. By contrast, equally proficient bilinguals who use different languages in different contexts and therefore do not switch that often, should suffer less from interference effects, so that the executive control system is less likely to develop a bilingual advantage. It is the aim of this study to investigate whether high L2 proficiency suffices for developing the bilingual control advantage, or whether a high amount of language switching experience, implying frequent simultaneous high activation in representations from both languages, is necessary. In the present study, we will therefore investigate whether a group of (Brussels) bilinguals that typically switch languages *within discourses* show different control than regular bilinguals, within the same language pair. These regular bilinguals experience language switching also every day, but not as often as the code switching Brussels bilinguals or Catalan-Spanish bilinguals.

The second aim of our study is to investigate to what extent any interaction effect between the bilingual control advantage and switching

experience is task specific or rather extends across executive functions. This is interesting because our primary hypothesis discussed above is supported by a recent study of Prior and Gollan (2011), who showed that bilinguals who often language switch are also better task switchers. This finding is important in the current context but it remains unclear whether experience with language switching also interacts with bilingual advantages in tasks that share less task demands, as was the case for Prior and Gollan, i.e. cognitive control tasks that imply inhibition instead of switching. Obviously, language *switching* experience is much more likely to transfer to non-verbal task *switching* than to *inhibition,* and bilingual advantages across tasks that into different executive functions would suggest a more fundamental and general change to the cognitive system. Therefore, we will use two tasks that primarily measure inhibitory control, namely the flanker task and the Simon arrow task. The distinction between training tasks and training abilities is currently a major debate in the executive control literature. Some findings suggest that cognitive abilities can be trained. Jaeggi, Buschkuehl, Jonides, and Perrig (2008), for example, reported higher fluid intelligence in participants that were trained with an executive control demanding *n*-back task. Other researchers recognise several methodological concerns with such artificial training studies and claim that to this day, not one study has convincingly demonstrated that cognitive abilities can be trained, over and above (strategic) improvements in specific task demands (Shipstead, Redick, & Engle, 2010). In this view, showing that the amount of language switching by bilinguals produces an advantage for tasks with little overlap in task demands while measuring common cognitive (control) abilities, would make a strong case for this discussion in the control literature as well.

The third aim of this study concerns the dissociation of switching experience from language pair characteristics. Prior and Gollan (2011) included Spanish-English bilinguals who regularly language switch and Mandarin-English bilinguals who switch between languages less often. Only the Spanish-English bilinguals showed an advantage on task switching. It was assumed that only bilinguals who often language switch, train their executive control capacities, causing better performance on executive control tasks. However, these two experimental groups do not only differ in their amount of switching between languages, but also in the amount of overlap

between these languages. Because languages that share orthography (in this case: English and Spanish, both alphabetic languages) and language pairs with a distinct script (English and Mandarin) require different representational structures (Gollan, Forster, & Frost, 1997) and hence also control demands, it is plausible that the bilingual advantages arising from competition between these two language pairs also differ. Indeed, task switching research has shown that switching between overlapping cognitive tasks (e.g., by using bivalent stimuli) causes a much greater switch cost than switching between tasks that share less task features (Rogers & Monsell, 1995). Therefore, the higher switch cost for the Mandarin-English group in the Prior and Gollan study does not necessarily reflect the fact that they switch less often between languages, but may be alternatively explained by the smaller lexical overlap, between Mandarin and English.

In summary, our aims are threefold. First, we aim to further disentangle the role of language switching experience in the development of executive functions. Second, we aim to study whether or not the assumed advantage of frequent language switching is task specific. The third and last aim of our study is to investigate the role of language pair characteristics.

We hypothesize that the general control advantage in bilingualism originates from very frequent switching between both languages, within similar contexts and even within sentences or conversations. To test this hypothesis, we tested three different groups of bilinguals: a group of unbalanced bilinguals, a group of balanced non-switching bilinguals, and a group of balanced bilinguals that do often switch languages. Importantly, the bilinguals in the three groups all master the same languages, Dutch (L1) and French (L2). We predict that the switching group will show a better performance on inhibitory control tasks compared to the unbalanced group and the non-switching group that also has high L2 proficiency. We aimed to test only one executive function (i.e. inhibition), and therefore only included a flanker task and a Simon arrow task, two tasks that tap into that specific function.

METHOD

Participants

To be able to include these three different groups of bilinguals, we recruited participants in two different ways: (a) Psychology students of Ghent University, participating for credits, and (b) bilinguals that were recruited through an advertisement on the university website, and who were paid for their participation. All participants had Dutch as their L1, French as L2, and had a good knowledge of English (L3). They were all born in Belgium, highly educated, and differed in their L2 proficiency and the extent of switching. We included three groups: unbalanced (UB), balanced switching (BSB), and balanced non-switching bilinguals (BnSB). The three groups all consisted of both paid and voluntary participants.

Demographic participant information is shown in *table 1*. The UB live in a Dutch-dominant environment and acquired French before the age of 11 at school. After the age of 18, they hardly came in contact with the French language again. The BnSB acquired both languages before the age of six, and are proficient in Dutch and French. However, they are almost never confronted (0.9 days/week) with contexts in which code switching between the two languages occurs. The BSB also acquired both languages before the age of six, are also highly proficient, but actively switch between the languages within sentences/conversations, at least four days a week. The groups were matched on age, sex, and general intelligence based on the Raven Advanced Progressive Matrices. We used a language questionnaire to measure their self-reported language proficiency in Dutch and French, and to assess their switching behaviour.

Materials

In the language questionnaire, participants rated their proficiency for reading, writing and speaking on a seven point Likert scale. We included an additional, more extended questionnaire to assess L2 proficiency, with questions as “I can read instruction in my L2”, “I can answer difficult questions in my L2”. The additional L2 questionnaire consisted of 15

questions that had to be rated on a four point Likert scale. The language switching questionnaire contained 25 questions surveying how often participants switch their languages, whether or not they mix or switch their language on purpose and consciously, whether or not selectively speaking in one language in one context was stimulated during childhood etc. The experiments were run on a standard colour monitor and were programmed and conducted using Eprime. Reaction times were measured with a Cedrus serial USB response box.

Flanker task

The stimuli were white arrows on a black background. One stimulus consisted of five arrows, participants indicated the direction of the arrow by pressing the left or the right button. The arrows could all be pointing in the same direction (congruent trials, e.g. >>**>**>>) or the central arrow could be pointing in the other direction than the flankers (incongruent trials, e.g. >>**<**>>). The proportion congruent/incongruent trials was 75%-25% (Costa et al., 2009).

Simon arrow task

The stimuli were single white arrows on a black background. The arrows could be pointing to the right or the left, and appeared on either the left or the right side of the screen. Trials in which the direction of the arrow corresponded with the side of appearance on the screen are labelled congruent trials; trials in which the direction and the side of appearance did not correspond are incongruent trials. The proportion congruent/incongruent trials was also 75%-25% (Costa et al., 2009).

Procedure and design

The informed consent form, language questionnaire and intelligence test were completed before starting the experiment. The procedure in both experiments was the following: (1) a fixation cross for 400 ms; (2) the experimental stimuli appeared until a response was given, or for maximum 1700 ms; (3) a blank screen for 1000 ms. There were 24 practice trials, followed by three blocks of 96 trials each. Afterwards, participants

completed the Raven Advanced Progressive Matrices. We used a 2 (Congruency) x 3 (Block) x 3 (Group) design with Congruency and Block as within subjects variables and Group as a between subjects variable.

RESULTS

Demographic data

No significant differences were found across groups in male/female ratio, age, or intelligence (Raven) scores (See also Paap & Greenberg, 2013). Participants were asked to rate their proficiency, age of acquisition (AoA) and frequency of use of Dutch and French. There were no significant differences in general proficiency or AoA for Dutch. The UB and the BnSB used Dutch more frequently than the BSB. Significant differences between groups were found for French proficiency: the UB had significant lower L2 proficiency scores than the BnSB ($t(43) = -8.973$, $p = .000$) and the BSB ($t(46) = -15.499$, $p = .000$). Differences in general French proficiency were also found between the two balanced groups ($t(35) = -4.524$, $p = .000$), although L2 proficiency was also very high in the BnSB group. The French AoA of the UB differed significantly from the BnSB ($t(19.548) = 20.679$, $p = .000$) and from the BSB ($t(22.827) = 20.364$, $p = .000$). No differences in AoA were found between the two balanced groups ($t(35) = .375$, $p > .710$). The three groups differed significantly in frequency of use of French, with UB showing a lower frequency of use than the BnSB ($t(17{,}904) = -4.712$, $p = .000$) and the BSB ($t(46) = -18.767$, $p = .000$). In addition, a difference in frequency of use was found between the two balanced groups as well ($t(35) = -5.896$, $p = .000$). The BSB differed significantly from the BnSB ($t(34.77) = -18.78$, $p = .000$) and the UB ($t(46) = -25.15$, $p = .000$) in switching frequency.

Table 1

Demographic data of the three groups

	Group 1 Unbalanced bilinguals (UB)	**Group 2** Balanced non-switching bilinguals (BnSB)	**Group 3** Balanced switching bilinguals (BSB)	**Test**
N	28	17	20	
Male/female ratio	9/19	3/14	4/17	$Chi^2(2) = 2.629$, $p > .269$
Age	20.7 (1.7)	20.9 (3.4)	21.7 (6.1)	$F(2,62) < 1$
Raven	11.0 (1.0)	10.8 (1.4)	10.8 (1.3)	$F(2,62) < 1$
Computer games	2.3 (1.8)	1.9 (0.8)	2.0 (1.7)	$F(2,62) < 1$
Dutch (L1)	7.0 (0.0)	7.0 (0.0)	7.0 (0.0)	No differences
Age of acquisition	0.0 (0.0)	0.0 (0.0)	0.0 (0.0)	No differences
Frequency of use (days)	7 (0.0)	7 (0.0)	6.8 (0.5)	$F(2,62) = 3.302$; $p < 0.043$
French (L2)	2.7 (0.9)	5.2 (0.8)	6.3 (0.8)	$F(2,62) = 120.732$, $p = .000$
Age of acquisition	10.2 (0.7)	1.2 (1.7)	0.9 (1.9)	$F(2,62) = 312.871$, $p = .000$
Frequency of use (days)	0.6 (0.6)	3.0 (2.0)	6.2 (1.7)	$F(2,62) = 103.217$, $p = .000$
Frequency of switching (Days/week)	0.5 (0.6)	0.9 (0.7)	5.8 (0.9)	$F(2,62) = 362.201$, $p = .000$

Experiments

For both experiments we conducted two analyses of variance (ANOVA). We performed a repeated-measures ANOVA with Group as between-subjects factor, and Congruency as within-subjects factor. Because of the difference between the groups concerning French proficiency, we included this variable as a covariate. The dependent variable was the mean RT on correct trials in the first analysis, and the conflict effect (RTs on incongruent trials minus RTs on congruent trials) in the second. To investigate whether the slight French proficiency difference between the two balanced bilingual groups may have contributed to the bilingual advantage, we also ran a regression analysis with the conflict effect as the dependent variable and group and french proficiency as independent variables. RTs that deviated more than 2.5SD from participant mean were removed (0.02% of the total amount of trials). The error rate was 0.05%; incorrect trials were excluded from the analyses.

Flanker task

A significant main effect of Group ($F(2,61) = 5.233$, $p < .008$, MSE = 16746) and a marginally significant effect of Congruency ($F(1,61) = 3.421$, $p = .069$, MSE = 4318) on mean RTs was found. The interaction between Group and Congruency was marginally significant ($F(2,61) = 2.422$, $p = .097$, MSE = 4318). Planned comparisons show no significant differences in mean RTs between UB and BnSB ($t(43) = 0.654$, $p > .517$). The BSB were faster than the BnSB ($t(35) = 4,217$, $p = .000$) and than the UB ($t(46) = 3,239$, $p < .002$) (See also *figure 1*).

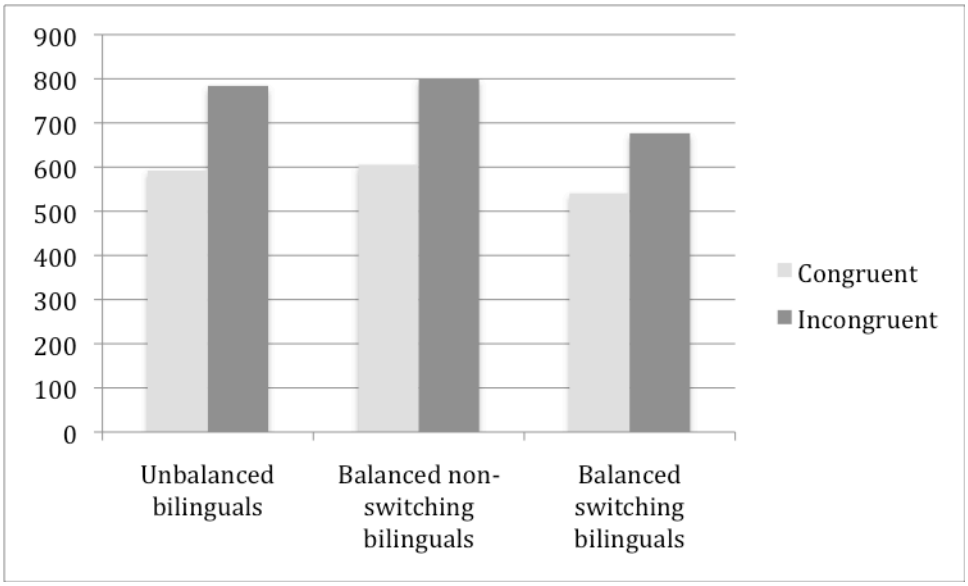

Figure 1. Reaction times on the congruent and incongruent trials in the flanker task

The ANOVA on the conflict effect showed a non-significant effect of Group ($F(2,61) = 2.422$, $p = .097$, MSE = 8637) (See *figure 2*). However, planned comparisons concerning the conflict effect show a significant difference between the UB and BSB ($t(32.266) = 2.381$, $p < .023$) and between the BnSB and BSB ($t(35) = 4.391$, $p = .000$). The UB and the BnSB did not differ significantly ($t(43) = 0.058$, $p > .954$).

Controlling for French proficiency across groups, the regression also revealed a significant difference in the size of the conflict effect across the three groups ($F(2,62) = 3.068$, $p < .05$). There was no significant relation between French proficiency and the size of the conflict effect ($F_{change}(1,61) = 0.663$, *sig* = .419). The positive regression weight even indicated that larger L2 proficiency tended to be associated with worse performance on the conflict tasks (i.e. larger conflict effects). Hence, the bilingual advantage present in the BSB cannot be explained by their slighter higher L2 proficiency.

Concerning the error rates, we only found a main effect of Congruency ($F(1,62) = 65.55$, $p = .000$, MSE = 0.83). No other effects reached significance (F < 1).

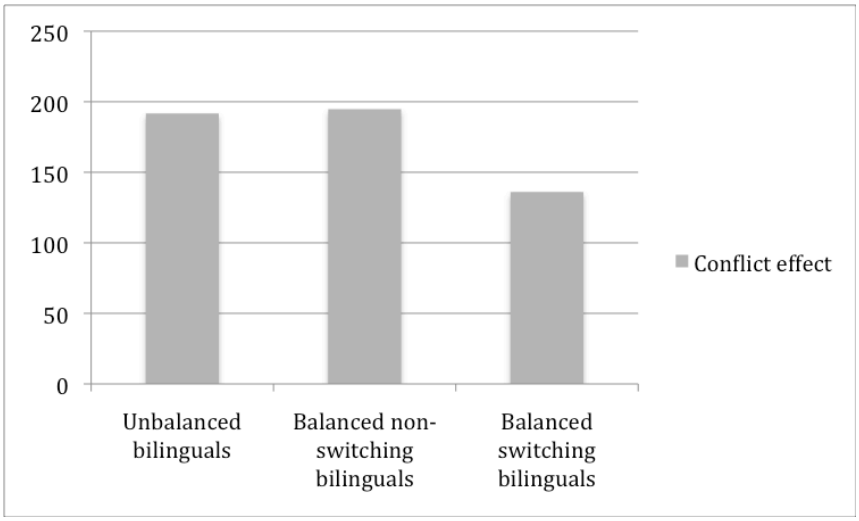

Figure 2. Size of the conflict effect in the flanker task

Simon arrow task

The repeated measures analysis revealed a significant main effect of Group ($F(2,61) = 4.292$, $p < .018$, MSE = 6751), and Congruency ($F(1,61) = 4.101$, $p < .047$, MSE = 721) on the RTs (See *figure 3*). The interaction between Group and Congruency was also significant ($F(2,61) = 6.678$, $p < .002$, MSE = 721). Planned comparisons show no significant differences in RTs between the UB and BnSB ($t(43) = -0.688$, $p > .495$ for congruent trials and $t(43) = -1.599$, $p > .117$ for incongruent trials), nor between the UB and BSB for congruent trials ($t(46) = 1.463$, $p > .150$). For incongruent trials, we found a marginally significant difference ($t(46) = 2.001$, $p > .051$). The BnSB differed significantly from BSB ($t(35) = 2.054$, $p < .047$ for congruent trials and $t(35) = 3.330$, $p < .002$ for incongruent trials).

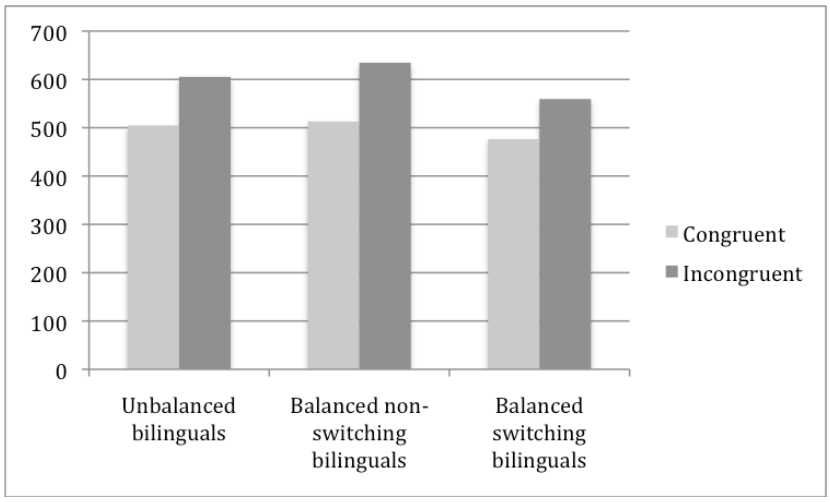

Figure 3. Reaction times on congruent and incongruent trials in the Simon Arrow task

The second analysis showed a main effect of Group ($F(2,62) = 4.900$, $p < .011$, MSE = 1494) on the conflict effect (See *figure 4*). Planned comparisons show a significant difference between the BSB and BnSB ($t(35) = 3.205$, $p < .003$). The UB did not differ significantly from the BnSB ($t(43) = -1.837$, $p > .073$), nor from the BSB ($t(46) = 1.538$, $p > .131$).

With French proficiency as a control predictor, the regression revealed a significant difference in the size of the conflict effect across the three groups ($F(2,62) = 4.90$, $p < .011$), whereas French proficiency itself was not significantly correlated with the size of the conflict effect ($F_{change}(1,61) = 3.238$, $sig = .077$). Thus, the slightly higher L2 proficiency of BSB cannot account for the bilingual advantage.

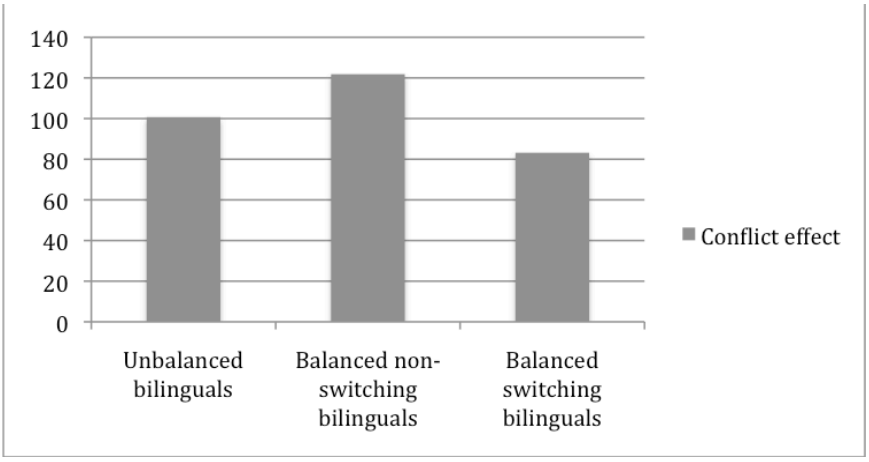

Figure 4. Size of the conflict effect in the Simon Arrow task

DISCUSSION

The aim of this study was threefold. Firstly, we wanted to investigate the influence of language switching experience on the executive control advantage in bilinguals. Second, we aimed to investigate whether any interaction between experience with language switching and the bilingual advantage effect would transfer across tasks that also tap into other executive functions than switching. Third, for the first time, we studied this issue independent from an important difference in language pair characteristics across bilingual groups (Prior & Gollan, 2011). We conducted a flanker task and a Simon arrow task, and compared the performance of unbalanced Dutch-French bilinguals, balanced bilinguals who often switch between languages in their daily lives, and bilinguals who do not often switch between languages.

The results of both experiments point in the same direction: balanced bilingual participants that often switch (BSB) between languages show smaller conflict effects than both unbalanced bilinguals and balanced bilinguals who do not often switch between languages (UB and BnSB), even though these bilinguals also had very high L2 proficiency. This suggests that

executive control advantages are only present when the lexical representations of both languages are often simultaneously active and used or inhibited during frequent code swichting, e.g. in bilinguals who often switch languages, within sentences or conversations. The frequent simultaneous activation between strong lexical representations of different languages causes competition and necessitates the bilinguals to engage their executive control mechanism to select representations in the target language, and inhibit the non-target language. This practice then transfers to non-linguistic cognitive control abilities, tapped into by the Simon and flanker tasks.

The second aim of our study was to investigate whether the advantage of language switching is task specific and only emerges on tasks that involve switching. We found that daily language switching not only affects the switching performance (Prior & Gollan, 2011), but also generalises to other executive control tasks, in this case measuring inhibition (Miyake et al., 2000). This supplements previous work of Prior and Gollan (2011), who showed that language and task switching was only better in bilinguals who regularly switch between languages. However, because they compared switching English-Spanish bilinguals with non-switching English-Mandarin bilinguals, it was yet unclear whether the difference between these groups reflected switching experience or rather different language pair similarity. The present study clearly shows that the bilingual advantage emerges from language switching experience, independent from language pair similarity, and also in tasks tapping into inhibitory executive functions.

The present findings may contribute to an explanation why findings about the bilingual executive control advantage are rather inconsistent. Whereas rather consistent bilingual advantages have been found by Bialystok and colleagues in Canada (e.g. Bialystok, Craik, Klein, & Viswanathan, 2004; Bialystok, 2006; Bialystok & Feng, 2009) and Costa and colleagues in bilingual Barcelona (e.g. Costa, Hernandez, Costa-Faidella, & Sebastian-Galles, 2009; Costa, Hernandez, & Sebastian-Gallès, 2008), a recent study by Paap and Greenberg (2013) failed to find such evidence in any of 15 executive control tasks, testing 122 bilinguals from 30 different language pairs in San Francisco. The present study suggests that active and frequent code switching may the crucial determinant for the

development of the bilingual executive control advantage. Although Paap and Greenberg claim that their bilinguals switch languages daily, it unclear whether this implies just switching languages between contexts (e.g. speaking English at university and Russian at home), or instead active and very frequent code switching within conversations, as is the case for Catalan-Spanish bilinguals, or for the BSB bilinguals in Brussels from this study. Given that the large (30) number of language combinations are unlikely to be used simultaneously in San Francisco, we suspect that their bilingual population is most comparable to the BnSB from this study, which also did not show a bilingual advantage.

An inevitable characteristic of this study is the lack of data about monolinguals. This is a more practical issue, given that everyone in Belgium has at least knowledge of two languages. The positive consequence of this language context is that we were able to compare different groups of bilinguals from the same language pair (Prior & Gollan, 2011). We cannot however exclude that the unbalanced and balanced non-switching bilinguals in this study still show better performance than monolinguals. However, note that also no differences were found between the monolinguals and the non-switching bilinguals in the Prior and Gollan study (2011).

Both language switching and task switching have been found to rely on similar brain regions. Hernandez and colleagues found increased activation in (dorsolateral) prefrontal cortex during language switching (Hernandez, Dapretto, Mazziotta, & Bookheimer, 2001; Hernandez, Martinez, & Kohnert, 2000), a region that has also been found to be involved in task switching (Botvinick et al., 2004; Dove, Pollmann, Schubert, Wiggins, & von Cramon, 2000; Wager, Jonides, & Smith, 2006; Wager, Jonides, Smith, & Nichols, 2005). In addition, Abutalebi and Green have described a general cognitive control mechanism for the selection, inhibition and production of one language by bilinguals (Abutalebi & Green, 2007).

To summarize, this study shows that language switching experience in daily life is a key determinant for the development of a stronger executive control system, underlying the alleged bilingual advantage on executive control tasks. Therefore, this factor should definitely be taken into account in further research investigating executive functioning in bilinguals.

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CHAPTER 3:
COGNATE EFFECTS AND EXECUTIVE CONTROL IN A PATIENT WITH DIFFERENTIAL BILINGUAL APHASIA[1]

We describe a case study of a French-Dutch bilingual aphasia patient with differential aphasia, showing clearly larger impairments in Dutch than in French. We investigated whether this differential impairment in both languages was due to selective damage to language specific brain areas resulting in the 'loss' of the language representation itself, or rather reflects an executive control deficit. We assessed cross-lingual interactions (involving lexical activation in the most affected language) with cognates in a lexical decision (LD) task, and executive control using a flanker task.

We used a generalised LD task (any word requires a YES-response), and a selective LD task in the patient's two languages (only words in a given target language require a YES-response). The cognate data unveil a differential pattern in the three tasks, with a clear cognate facilitation effect in the generalised LD, and almost no cognate effect in the selective LD tasks. This implies that a more impaired language can still affect the processing of words in the best-preserved language, but only with low cross-language competition demands (generalised LD). Additionally, the flanker task showed a larger congruency effect for the patient compared to controls, indicating cognitive control difficulties. Together, these results support accounts of differential bilingual aphasia in terms of language control difficulties.

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INTRODUCTION

During the last decades, bilingualism has gained a lot of interest in the psycholinguistic literature. The fact that this interest developed only recently is surprising, given that it is estimated that more than half of the world population is now bilingual. In this literature, bilinguals are typically individuals who regularly use their two languages (Adrover-Roig et al., 2011; Goral, Levy, Obler, & Cohen, 2006; Ibrahim, 2009; Kurland & Falcon, 2011), but are not necessarily equally proficient in both. An important discussion that has dominated this literature is about the question whether bilinguals have an integrated lexicon (and hence, one neural structure representing both languages) or two separated lexicons, one for each language. Or, otherwise stated, is there always activation in both languages during word recognition, even when only a single language is relevant at that time, or not? During the recent years, behavioural evidence has accumulated supporting language non-selective lexical access: even in unilingual language contexts, words from both languages are activated, and the lexical representations of these different-language words constantly and automatically interact with each other.

An important line of research supporting this hypothesis concerns studies looking at the recognition of cognates. Cognates are words that have the same meaning and a similar orthography/phonology in both languages, for example the Dutch – English word pairs *film-film* (identical) or *boek – book* (non-identical). Several studies have shown that cognates are recognised faster than non-cognates (Duyck, Van Assche, Drieghe, & Hartsuiker, 2007; van Hell & Dijkstra, 2002). This is called the *cognate facilitation effect*. Surprisingly, such a cognate effect even emerges when people are reading unilingual sentences in their native language. Using eye tracking, it was shown that Dutch-English bilinguals showed shorter fixations for Dutch-English cognates, even though they only read Dutch sentences, and did not know that English was relevant for the experiment (Van Assche, Duyck, Hartsuiker, & Diependaele, 2009). These cognate effects are generally considered as a reliable marker for language non-

selective lexical activation, and are commonly explained by convergent activation spreading from the cognate's similar semantic, orthographic, and phonological representations across languages. Non-cognate translation equivalents only share a semantic representation, do not benefit from facilitatory convergation spreading, and therefore are recognised slower. This cognate facilitation effect demonstrated that word processing in one language is affected by other languages, supporting the idea of interacting lexicons and an integrated bilingual language system.

In the current study, we investigated the above cognate facilitation effect, as the most commonly investigated marker of cross-lingual lexical interactions, in a patient with bilingual aphasia. Aphasia is defined as a general impairment in understanding, formulating or using verbal messages, in spoken and/or written modality, caused by brain dysfunction to language-related area. The main cause of aphasia is a stroke, but a tumour, an infection or degenerative brain diseases can also lead to aphasia.

Interestingly, aphasia in bilinguals does not always affect both languages to the same extent. For functional/psycholinguistic theories of bilingual lexical access, this is interesting. If the two languages of a bilingual are represented in a unitary system, as suggested by the behavioural work above, one would expect that both languages rely on the same neural structure. Therefore, one would also expect that damage to that neural structure (aphasia) causes similar functionality loss across both (all) languages represented in that structure. However, in the neuropsychology clinic, it is still a (surprising) fact that some patients still show larger deficiencies in one language than in the other. In addition, language recovery does also not benefit both languages equally (well). Paradis described six different ways in which bilingual aphasia recovery may occur (Paradis, 2004). When recovery occurs similarly in both languages (the most frequent case) it is diagnosed as *parallel* recovery (Marangolo, Rizzi, Peran, Piras, & Sabatini, 2009). When this is not the case and improvement is more pronounced in one language compared to the other, the diagnosis is *differential* recovery. Strikingly, it is not always L1 (i.e. the native language) that recovers best, as was reported by some authors (Goral et al., 2006; Meinzer, Obleser, Flaisch, Eulitz, & Rockstroh, 2007). Aglioti and Fabbro (1993) for instance, described a patient with better recovery in the weakest

language (i.e. L2, the second language). An extreme case of differential recovery is when one language does not recover at all, in which case we speak of *selective* recovery. *Successive* recovery is when one language only starts to recover when the other has fully recovered. The fifth recovery pattern described by Paradis is the case when there is an alternation in recovery: one language starts to recover and then to weaken again when the other becomes stronger. This is called *antagonistic* recovery. Some bilingual aphasic patients uncontrollably switch and mix their languages, in this case we can speak of *blended* recovery (Adrover-Roig et al., 2011; Fabbro, Skrap, & Aglioti, 2000; Leemann, Laganaro, Schwitter, & Schnider, 2007; Marien, Abutalebi, Engelborghs, & De Deyn, 2005; Riccardi, Fabbro, & Obler, 2004).

In analogy with the *recovery* patterns described by Paradis (2004), similar descriptions may also be used to describe the pattern of *impairment* in both languages. For example, a patient with more serious impairments in one language compared to the other, is diagnosed with *differential* aphasia (Adrover-Roig et al., 2011; Aglioti, Beltramello, Girardi, & Fabbro, 1996; Goral et al., 2006; Vajramani, Akrawi, McCarthy, & Gray, 2008), irrespective of the way both languages recover. When only one language is affected, with no apparent impairments in the other, this is called selective aphasia (Ibrahim, 2009). In theory, a patient with differential aphasia (i.e. both languages are damaged to a different extent) might show parallel recovery (both languages recover equally fast), although this distinction is virtually never made in case studies.

Because the first cases of differential and selective aphasia were identified in the neuropsychological literature when the psycholinguistic literature on bilingualism had not yet developed, and reports of cross-lingual lexical interactions did not exist, such aphasias were explained by asymmetrical neural damage: because both languages were assumed to be represented in distinct brain areas, a lesion in the language specific area would then lead to impairments in that particular language, without affecting the other language.

However, as stated above, much evidence has now been found against the idea of language specific brain areas. Both of a bilingual's

languages do not only interact functionally (e.g. cognate effects in the behavioural literature, see above), it has also been confirmed that languages overlap with respect to their neural representation. For instance, Klein and colleagues found largely overlapping brain areas for English and French (Klein, Zatorre, Milner, Meyer, & Evans, 1994) and for English and Chinese (Klein, Milner, Zatorre, Zhao, & Nikelski, 1999) during word production tasks. In addition, Hernandez and colleagues found no difference in the brain activation pattern between picture naming in Spanish and in English (Hernandez, Dapretto, Mazziotta, & Bookheimer, 2001). Vingerhoets and colleagues found that fluency tasks, picture naming and word generation engaged largely the same cerebral areas in Dutch, French and English (Vingerhoets et al., 2003). From this, Green (2005) developed the convergence hypothesis: when learning a second language, the processing of this language will rely on the same neural network and control circuits that are involved in L1 processing.

At first, it seems hard to reconcile these behavioural and neurological demonstrations and models of overlapping/interacting languages (one unitary language system) with the mere existence of differential/selective aphasia. How can a stroke affect only one language if the languages are largely represented in the same areas? Interestingly, Pitres hypothesized already in 1895 that a control deficit might be the cause of selective and differential loss in bilingual aphasia. Pitres stated that *"every language can be independently inhibited, temporarily or permanently. Thus bilingual aphasia is not the result of a lesion in the neural substrate of a language, but rather the result of a functional inhibition of the language."* (Pitres, 1895). In other words, he alludes to a problem in language control, i.e. in the selection of (words in) the intended language, and the inhibition of (words in) not attended languages. Regrettably, his interesting hypothesis was never empirically tested. More recently however, Abutalebi and Green (Abutalebi & Green, 2007) revitalised this idea, describing a neural network for cognitive control and language control, which consists of the prefrontal cortex, the anterior cingulate cortex, the inferior parietal cortex and the basal ganglia. Damage to the components of this network might lead to the language control deficits underlying bilingual aphasia. Hence, in this view, selective language loss is not due to the damage of the language

representations itself, but rather to the cognitive control mechanisms necessary to handle these competing languages.

If this control hypothesis is correct, and the lexical representations themselves are indeed intact, patients with bilingual aphasia could indeed show effects of a language that is heavily damaged onto the processing of another language, even though that language in itself is not very functional. A weak test of this hypothesis has already been reported in *some* bilingual aphasic patients with *parallel* aphasia. For instance, Roberts and Deslauriers (Roberts & Deslauriers, 1999) found that bilingual aphasic patients with parallel aphasia were able to name more pictures of cognates than non-cognates, in both languages. Similarly, Detry and colleagues (Detry, Pillon, & de Partz, 2005) administered a picture-word verification task and a naming task with cognates and non-cognates in a French/English parallel aphasia patient with agrammatism and word finding difficulties. In both tasks, the patient's performance was higher for the cognates compared to the non-cognates. So, even though functionality of the languages in these patients was severely impaired, these languages were still able to even influence processing/activity in another language.

In addition, two studies have investigated the role of cognates in aphasia *treatment* in patients with parallel aphasia. Kohnert (Kohnert, 2004) treated a Spanish/English bilingual patient with severe transcortical motor aphasia, with a parallel impairment in both languages. It was observed that therapy effects generalised across languages to untrained items, but only for cognates. A more recent study (Kurland & Falcon, 2011) studied a similar hypothesis in a Spanish-English bilingual patient with severe expressive aphasia in both languages. Surprisingly, this study revealed detrimental, rather than facilitatory, effects of cognate status in aphasia treatment. Although the reason for this inconsistency is unclear, at least this finding also indicates cross-lingual interactions, and confirms that functionally affected languages may still influence processing in another language.

Although these cognate effects in patients with parallel aphasia are very interesting, a more challenging test for the control hypothesis of Pitres (1895) above is of course the existence of cross-lingual interactions in patients who show *differential* (or selective) aphasia. Is a language that is

more affected than the other still able to influence the best-preserved language? To the best of our knowledge, only one study has yet investigated cognate effects in differential aphasia, about 10 years ago. Lalor and Kirsner (2001) described a balanced English-Italian bilingual aphasic patient who showed larger impairments in Italian (L2) compared to English (L1) on expressive language tasks. They assessed naming in both languages, and found a cognate effect. More important for the current study is that they also administered a generalised lexical decision task (both Italian and English words require a YES-response, non-words require a 'no' response). They found no differences in reaction times (RTs) between cognates and non-cognates, as aphasic patients typically yield highly variable lexical decision RTs. However, the patient showed fewer errors with cognates compared to non-cognates. Although this is an interesting finding in relation the control hypothesis discussed above, Lalor and Kisner did not interpret this effect as such, nor did they assess cognitive control performance of this patient.

AIMS AND METHODS

The aim of the current study was twofold. First, we aimed to gain a more profound insight in how cognates are processed in bilingual aphasia with *differential* language loss, as a marker of cross-lingual interactions. More specifically we aimed to investigate the cognate facilitation effect in relation to language control demands in a French-Dutch bilingual differential aphasia patient, with a larger impairment in L2 (Dutch). We report the data of three different lexical decision (LD) tasks, each yielding different language control demands, with cognates as the critical stimuli. We administered a generalised LD task ("Is it an existing word or not?") and a selective LD task in L1 and L2 ("Is it either a L1/L2 word or not?") (Dijkstra, Van Jaarsveld, & Ten Brinke, 1998). Because a generalised LD requires a YES-response for words from both languages, whereas words in the non-target language require a NO-response in a selective LD, these tasks differ in terms of language control demands. The selective LD imposes much more cross-lingual competition than the general LD, in which no language selection/decision has to be made.

Because the generalised LD does not require inhibiting representations of either of the languages, unlike the selective LD tasks (lexical activation from *any* language requires a YES-response), we expected to find a clear cognate effect in the current patient. We assumed that the most impaired language (Dutch) might still interact with the processing of French words, because the word/non-word decision in the generalised task does not require suppression of any language. This interlingual interaction should yield a cognate facilitation effect. Because the selective LD requires a decision whether the letter string is a word specifically in the target language, more control is needed to map lexical activation in the non-target language to NO-responses. Therefore, we expected a much smaller cognate facilitation effect here. In addition, we expected that the patient would experience less difficulty to suppress his most affected language (in this case: Dutch) compared to the better-preserved language (French). This should lead to differential results in the two selective LD tasks.

The investigation of the cognate effects in generalised versus selective lexical decision and finding different cross-lingual effects would provide indirect evidence for the hypothesis that a control deficit is underlying the differential impairment pattern in our patient. Additionally, we also aimed to investigate this control hypothesis in a more direct way. Thus, the second aim of this study was to directly assess the executive control abilities of our patient. To that end, we also administered a congruency task. If the differential aphasia is caused by an executive control deficit, rather than by damage to a language-selective lexical area, this deficit should be reflected in the congruency task performance. Similar to the study of Green and colleagues (2010), we also used an Eriksen flanker task[2] (Eriksen & Eriksen, 1974). The stimuli of this task typically consist of five stimuli, most often arrows. The participant is required to react to the direction of the central arrow. The direction of the arrows presented next to the central arrow can be the same as the direction of the central arrow (congruent trials) or opposite (incongruent trials). The congruency effect is the difference in error rates or reaction times between congruent and incongruent trials. We expected our patient to show a larger congruency

[2] The Eriksen flanker task is one of the most frequently used tasks to assess cognitive control.

effect in this task than controls. To our knowledge, this is the first study to directly measure performance on a cognitive control task by a differential aphasia patient.

CASE REPORT

We report the data of H.D.M., a right-handed 78-year-old man. He is a French-Dutch bilingual with 15 years of formal education. He worked as a technical engineer until his retirement at the age of 65. His native language is French, but at age 2.5 he started school in Dutch. During his later life, he used both Dutch and French on a daily basis, living in a Dutch-speaking environment, being married to a French-Dutch bilingual woman and raising their children in Dutch. He kept on speaking a lot of French with family and friends, and also watched a lot of French television and read French books and papers. He reported to be equally proficient in both languages[3]. Both the patient and his wife agreed to participate in our study, and an informed consent form was obtained.

In March 2011, H.D.M. suffered an acute left thalamic haemorrhagic stroke (*figures 1&2*) and was admitted to the hospital with complaints of feeling ill and word finding difficulties.

[3] We are aware of the fact that not all people who are able to use two languages can be regarded as fully bilingual, in the sense that language proficiency takes long periods of acculturation and assimilation to reach a deep structural level (Cummins, 1979). Such mastery should not be confused with the simple ability to use a language in social situations such as conversations. However, since the patient described here acquired both languages at a very young age, at kept using both languages equally often in his daily life, we argue that he can be regarded as a fully balanced bilingual, at least for the rather low level of (lexical) language processing that is assessed in this study. We do not assume complete equivalency of all higher linguistic levels.

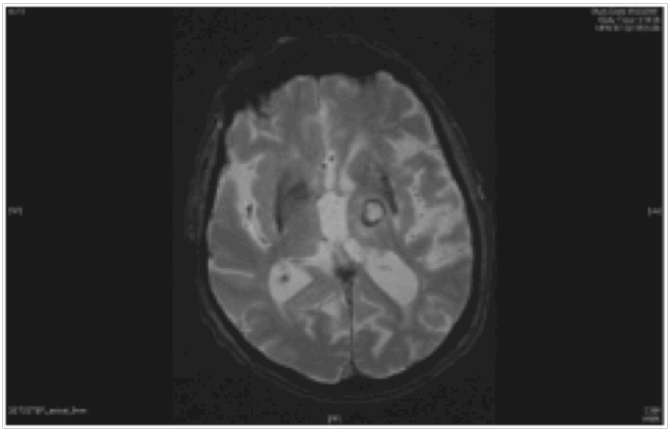

Figure 1. T2* weighted image of the lesion

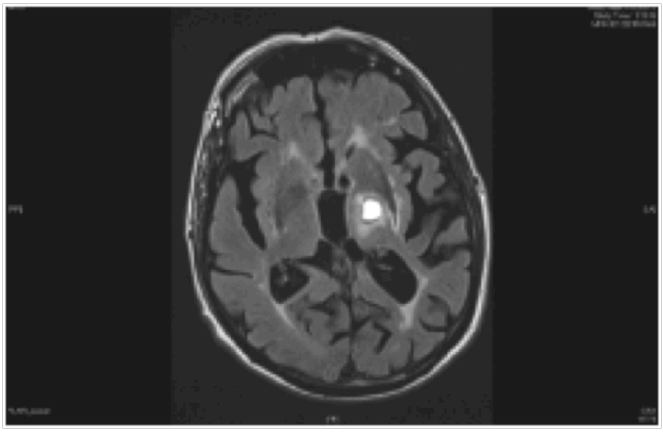

Figure 2. Axial FLAIR image of the lesion

Dutch and French language functions were assessed about three weeks post onset with the Dutch and the French version of the Aachen Aphasia Test (AAT) (Graetz, De Bleser, & Willmes, 1992). Individual scores on each subtest can be found in *table 1*. Significant differences ($p < .05$) between the two languages were found on the subtests Token Test and Naming, for which the patient performed significantly worse in Dutch compared to French. Based on the AAT scores, he was diagnosed with

clinical aphasia in both languages, with a clearly larger impairment in Dutch compared to French. Therefore, the patient received the diagnosis differential aphasia, with a better preservation of his first language. His wife, with whom he is used to communicate both in French and in Dutch, confirmed this. She clearly noted a difference in the communication skills of her husband, reporting more word finding difficulties and semantic paraphasias in Dutch. Showing fluent language production with severe anomia and word finding difficulties, (mild) comprehension problems, writing difficulties, and his repetition being intact, the patient was diagnosed with thalamic aphasia (Fabbro, Peru, & Skrap, 1997). At the moment of testing, the patient did not (yet) receive any speech or language therapy.

In addition to the AAT we administered Part C of the Bilingual Aphasia Test (BAT) (Paradis & Libben, 1987), which concerns passive translation (translation recognition). The score for the French – Dutch part was 4/5, whereas the patient had a perfect score (5/5) for passive translating into French. We also administered the Controlled Oral Word Association Test in Dutch (Miatton, Wolters, Lannoo, & Vingerhoets, 2004), which he found really difficult and frustrating. In the phonological part, he only generated one word, and in the semantic part he was able to give four words. To get an idea of the IQ of the patient, we administered the Coloured Progressive Matrices (Raven, Raven, & Court, 1998). Our patient had a raw score of 21/36, which corresponds with percentile 75.

Table 1

Scores on the subtests of the AAT in French and Dutch

	French AAT	**Dutch AAT**
Spontaneous Speech	26/30	19/30
Token Test (#errors) *	6	19
Repetition	146/150	143/150
Written Language	66/90	69/90
Naming *	113/120	90/120
Comprehension	100/120	94/120

** The patient showed a significant difference between the French and the Dutch score on this subtest ($p < .05$).*

EXPERIMENTAL METHOD

Lexical decision task

We administered three versions of the lexical decision (LD) task: a generalised LD task and a selective LD task in French (L1) and in Dutch (L2). In the generalised LD task, the patient had to indicate if the word is an existing word or not, in any language. In the French selective lexical decision task, he had to indicate if it was a French word or not. Similarly, in the Dutch selective lexical decision he had to make the distinction "Dutch word or not". The three tasks were administered on three separate days, to exclude order effects.

The stimuli used in each LD task were 30 Dutch-French cognates, 30 Dutch non-cognates, 30 French non-cognates and 90 non-words. So, the selective lexical decision tasks also contained words in the non-target language in order to increase language control demands specifically for this task. Different stimuli were used for the three tasks. In the selective LD

tasks, the cognate was presented in the target language (i.e. in Dutch for the Dutch task, in French for the French task). In the generalised LD task, both French and the Dutch cognates were used. Cognates and controls were matched for word length, frequency, neighbourhood size and imageability using the WordGen stimulus generation software (Duyck, Desmet, Verbeke, & Brysbaert, 2004).

Flanker task

Each stimulus of the flanker task consisted of five arrows horizontally presented on the screen. The central arrow could be pointing to the left or to the right, flankers could be pointing in the same direction as the central arrow (congruent trials) or in the opposite direction (incongruent trials). We included 40 congruent and 40 incongruent trials. The patient had to react to the central arrow by pressing a left button (i.e. the *enter* button) or the right button (i.e. the *capslock* button).

RESULTS

Similar to Lalor and Kirsner (2001) and other bilingual aphasia case studies, we will focus on accuracy for interpretations. Because RTs are highly variable in aphasic patients, these are less useful, but will still be reported for the interested reader.

Generalised lexical decision

Error rates

H.D.M. made significantly less errors on cognates (3%) relative to French words (33%) ($t(58) = 3.203$, $p < .002$) and non-words (28%) ($t(118) = 2.888$, $p < .005$). No differences were found between the error rates for cognates (3%) and Dutch non-cognates (3%) ($t(58) = 0.000$; $p > 1.000$). He showed a statistically significant overall cognate effect, making less errors on cognates (3%) compared to non-cognates (18%) ($t(87.751) = 2.483$, $p < .015$). This shows that even the most affected language still interacts with processing in the most preserved language, at least in a task (general lexical

decision) in which language control demands are low. Values are shown in *table 2.*

Table 2
Error rates and reaction times in the generalised lexical decision task

Stimulus	**% errors**	**Reaction Times (ms)**
Cognates	3%	1615
L1 words (French)	33%	1878
L2 words (Dutch)	3%	1952
Non-words	28%	2834

Reaction times

Only the reaction times on correct trials were included in the analyses (RTs). The results of the generalised lexical decision show that on average H.D.M. reacted faster on cognates (1615ms) compared to non-cognates (1915 ms) ($t(76) = -1.216$; $p > .228$). More specifically, he responded much faster for cognates (1615 ms) compared to both French non-cognates (1878 ms) and Dutch non-cognates (1952 ms). These comparisons did however not reach statistical significance ($t(47) = -1.014$; $p > .316$ for the French non-cognates and $t(56) = -1.179$; $p > .243$ for Dutch non-cognates). Response latencies (2834 ms) on non-words were significantly slower than both cognate and non-cognate words (all p's $< .014$). Taken together, in the generalised lexical decision task cognates were recognised 16% faster than L1 words, and 21% faster than L2 words, thus showing a clear cognate facilitation effect. Values are shown in *table 2*.

French selective lexical decision

Error rates

In the French selective lexical decision, the patient had a perfect score on cognates and L1 words, making no errors at all. He made significantly more errors on L2 words (13%) ($t(29) = 2.112$; $p < .043$). In addition, he made more errors on non-words (7%) compared to cognates and L1 words ($t(89) = 2.521$; $p < .013$). Values are shown in *table 3*.

Table 3
Error rates and reaction times in the French (L1) selective lexical decision task

Stimulus	% errors	Reaction Times (ms)
Cognates	0%	1496
L1 words (French)	0%	1537
L2 words (Dutch)	13%	2122
Non-words	7%	4102

Reaction times

H.D.M. responded almost equally fast on (French) cognates (1496 ms) as on French non-cognates (1537 ms) ($t(58) = -0.11$; $p > .912$), showing a cognate "facilitation effect" of only 3%. When giving a NO-response, the patient reacted faster to Dutch non-cognates (2122 ms) compared to the non-words (4102 ms) ($t(108) = -3.795$; $p < .000$). Values are shown in *table 3*.

Dutch selective lexical decision

Error rates

In the Dutch (L2) selective lexical decision, the patient scored equally accurate on (Dutch) cognates (10% errors), L1 non-cognates (10%

errors) and non-words (7% errors) (all *p*'s > .55). He made no errors on the L2 non-cognates. The difference between the error rate on cognates (10%) and the error rate on Dutch non-cognates (0%) was marginally significant ($t(29) = -1.795$, $p < .083$), implying a cognate *interference* effect. Presumably, the cognates activate the French representation more strongly, so that he is inclined to give a NO-response in a Dutch language-seletive lexical decision task, whereas these Dutch cognates require a YES-response (see the *General Discussion*). Values are shown in *table 4.*

Table 4
Error rates and reaction times in the Dutch (L2) selective lexical decision task

Stimulus	**% errors**	**Reaction Times (ms)**
Cognates	10%	3210
L1 words (French)	10%	2185
L2 words (Dutch)	0%	3494
Non-words	7%	3999

Reaction times

The RTs for the (Dutch) cognates (3210 ms) were slightly smaller than the RTs for the Dutch non-cognates (3494 ms) ($t(35.534) = 1.830$; $p < .076$). *NO*-responses were (non-significantly) faster to French non-cognates (2185 ms) than to non-words (3999 ms) ($t(109) = -1.066$; $p > .289$). Taken together, H.D.M. showed a cognate facilitation effect of 8%, which however did not reach statistical significance ($t(82) = .785$; $p > .435$. Values are shown in *table 4*.

In addition, overall RTs in the Dutch selective lexical decision task (3221 ms) were slower compared to the generalised (2070 ms) and the French selective lexical decision task (2314 ms). When comparing the RTs in both languages across the three tasks, we find that the patient reacted

more slowly on the Dutch (2695 ms) compared to the French (1774 ms) stimuli.

Flanker task

H.D.M. made more errors on incongruent trials (12% errors) compared to the congruent trials (2% errors), showing a very big congruency effect of 10%. We also tested 19 control subjects, who were also balanced Dutch-French bilinguals in a separate experiment. None of them made more than 3.7% errors on both incongruent and congruent trials, with a mean of 0.6% errors, resulting in a very small congruency effect on error rates. The 95% confidence interval of the error rates in the control group was [0.0%-1.0%], so, performance in the Flanker task by the patient is significantly and dramatically worse compared to control subjects, even though this is a non-linguistic task. This indicates a clear executive control deficiency.

DISCUSSION

We report the data of a patient with differential aphasia on three versions of the LD task: a generalised LD task and a selective LD task in L1 and in L2. We hypothesised that the pattern would be different in the three tasks, due to the differential need for language control in the generalised versus the selective LD task. In addition, we administered a flanker task to directly assess the executive control functions of the patient. Because we hypothesized that a control deficit might underlie his bilingual aphasia pattern, we expected that he would show a larger congruency effect compared to control subjects.

Because we are aware of the fact that RTs typically show large variance in patients, interpretations were mainly based on error rates. In the generalised lexical decision we found a clear cognate facilitation effect when comparing the performance on cognates with the performance on both L1 and L2 non-cognates. This implies that the most affected language (Dutch) is still able to influence activation in the most preserved language (French), given that cognates were recognised better than L1 (French) non-cognates.

As we argued in the introduction, control demands are lower in the generalised lexical decision task compared to the selective lexical decision task, because the former does not require the participant to inhibit (words in) one language. Because this task requires a much smaller amount of language control, even a bilingual aphasic patient with differential language loss still shows cross-lingual lexical interactions, with activation spreading for the most affect language to the strongest language. RTs showed that cognates were also recognized faster (but not significantly due to high RT variance) than controls in both languages, which corroborates the above finding. These findings are in line with previous studies that reported more efficient processing of cognates compared to non-cognates (Detry et al., 2005; Kohnert, 2004; Roberts & Deslauriers, 1999) in patients with *parallel aphasia.*

Additionally, the performance on the Flanker task showed that H.D.M. performed significantly worse compared to the control group, showing a very large congruency effect, which implies a large amount of interference from the incongruent flankers. This suggests that it was difficult to select the relevant information (i.e. the direction of the central arrow), while ignoring the irrelevant information (i.e. the direction of the flanker arrows), directly supporting our hypothesis of an executive control deficit [4].

In the L1 selective LD task, we did not find a difference between cognates and L1 non-cognates on both RTs and accuracy. However, in the L2 selective LD task, cognates were recognized less accurately compared to L2 non-cognates. This differential pattern in the two versions of the selective LD task can again be explained by the tasks' language control demands, which differ from those in the generalised lexical decision task. Because the patient's most affected language is Dutch, the control hypothesis of bilingual differential aphasia would assume that it is harder for the patient to suppress

[4] We are aware of the fact that the way executive functioning was evaluated in this patient is rather limited (using COWA and flanker task). For further research, we suggest assessing executive functions more profoundly, e.g. using the Winsconsin Card Sorting Task, a switching paragidm, a Go/Nogo task etc (See also Garcia-Molina, Tomos, Bernabeu, Junque, & Roig-Rovira, 2012; Segura et al., 2009).

his French lexicon than to suppress the Dutch. Thus, in the French selective lexical decision the Dutch lexicon is easily suppressed, and influences the recognition of the French cognate only to a very small amount, leading to the absence of a cognate effect in the French selective LD. However, because the French lexicon is not that easily inhibited, it affects the recognition of the Dutch cognates in the Dutch selective LD more strongly. However, because the cognates are likely to activate their French representation more strongly, competition between the activation in that French representation (requiring a NO-response in the Dutch lexical decision) and that in the Dutch representation (requiring a YES-response in the Dutch lexical decision) might cause the cognate *interference* effect in the Dutch (L2) selective lexical decision task. Because the selective lexical decision appeals more to the control system compared to the general lexical decision (cfr. supra), the patient's control deficit leads to the reduction (and inverse) of the cognate effect.

The aphasic symptoms in the patient described here were caused by a subcortical (thalamic) lesion. It was only recently claimed that not only cortical, but also subcortical lesions may cause aphasia (Murdoch, 2004). Structures that have been hypothesised to be involved in linguistic representation are the basal ganglia, the thalamus, the subcortical white matter pathways and the cerebellum. To the best of our knowledge, only four studies have investigated the implications of a subcortical lesion causing aphasia in bilingual patients (Aglioti & Fabbro, 1993; Azarpazhooh, Jahangiri, & Ghaleh, 2010; Fabbro et al., 1997; Reynolds, Turner, Harris, Ojemann, & Davis, 1979). Importantly, seven out of eight bilingual aphasics with subcortical lesions showed differential aphasia, just as the patient described here (Aglioti & Fabbro, 1993; Azarpazhooh, Jahangiri, & Ghaleh, 2010; Fabbro et al., 1997 cases 1&2; Reynolds, Turner, Harris, Ojemann, & Davis, 1979). Additionally, similar to our patient, six of them showed larger impairments in their L2 compared to L1 (Azarpazhooh et al., 2010; Fabbro et al., 1997 cases 1&2; Reynolds et al., 1979). This might suggest an important role of subcortical structures in showing differential impairments in both languages. Because ample evidence has already been found against distinct or spatially separate brain areas representing different languages (see the *Introduction*), we suggest that a deficit in cognitive control might

underlie the differential impairments in both languages (Green, 2005; Paradis, 2004; Pitres, 1895).

It has been shown that subcortical lesions can lead to decreased activation in cortical areas through diaschisis (i.e. a lesion leads to the disfunction of other brain areas through the disruption of the connectivity between the lesioned and the physically intact area) or hypoperfusion (e.g. Hillis et al., 2002). However, because we do not have PET or SPECT data from our patient, we cannot confirm this anatomically. Nevertheless, also based on the executive control problem, we hypothesize that the disruption of the thalamus might cause a hypometabolism in frontal areas, which are known to be involved in language control. For example, an anterior loop (frontal associative cortex, caudate nucleus, globus pallidus, ventral anterior nucleus, frontal associative cortex) has been proposed to be involved in language planning, whereas language selection would rely more on a posterior loop (temporo-parietal cortex, pulvinar, temporo-parietal cortex) (Fabbro et al., 1997). We suggest that the frontal hypometabolism might cause the worse performance on the Flanker task, and might underlie the language control deficits leading to the cognate pattern shown by the patient described here.

As far as treatment is concerned, there is a lack of clear support favouring either training in one language or training in both languages in bilingual patients. Initially it was hypothesised that giving language therapy in both languages might result in a reciprocal inhibition, and might therefore be disastrous for language recovery in any language (Fabbro et al., 1997; Green, 2005; Hilton, 1980; Lebrun, 1988; Paradis, 2004). In addition, it was found that the effects of language therapy in one language might generalise to the untrained language(s) (Edmonds & Kiran, 2006; Filiputti, Tavano, Vorano, De Luca, & Fabbro, 2002; Kiran & Edmonds, 2004; Marangolo et al., 2009; Miertsch, Meisel, & Isel, 2009). However, this generalisation does not always occur (Abutalebi, Rosa, Tettamanti, Green, & Cappa, 2009; Galvez & Hinckley, 2003; Meinzer et al., 2007), so that some authors argue for therapy in all languages (Ansaldo & Marcotte, 2007; Ansaldo, Marcotte, Scherer, & Raboyeau, 2008; Kohnert & Goldstein, 2005). For a more detailed overview, see Kohnert (2004) and Faroqi-Shah et al. (Faroqi-Shah, Frymark, Mullen, & Wang, 2010).

We would like to add a caveat about the limitations of this study. We were only able to estimate premorbid language proficiency. Evidently, we did not have any formal premorbid language proficiency assessments of the patient. However, given the unpredictable nature of stroke and aphasia, this is almost unfeasible. In addition, the pre-morbid language assessment is not meant to reveal slight functional differences between languages. Instead, its aim is to have a rough indication of quasi-equivalent proficiency. To assess executive functioning, we only used a flanker paradigm, given that we already needed a very extensive test battery. We opted for the flanker task since it is the most often used task in cognitive psychology literature to assess executive control functioning. It would however be very interesting to assess executive functioning using more than one task. Evidently, since this is a case study, it should be replicated with a larger group of patients to ensure generalisability. In addition, the data of this group data should be compared to the data of a matched control group. The normative data of the neuropsychological tests used in this patient are also not always suitable for this kind of small datasets (n=1). We also agree a distinction should be made between basic language skills, and fully integrated language performance. It is clear that the present paper focuses on differential loss of quite low-level processes of language use (lexical processing), and that language control problems will probably also affect functioning at other linguistic levels, not assessed in this paper. As far as the bilingual language representations are concerned, results obtained by neural imaging techniques indicate that common representations for different languages are highly unlikely. This alternative however cannot be completely ruled out, but cannot be detected with the spatial resolution of current imaging techniques.

To summarize, we found cognate facilitation and cognate interference effects across three lexical decision tasks, providing evidence for cross-linguistic interactions in a bilingual aphasic patient with differential language loss, even arising from the most affected language. In addition, this patient showed large congruency effects in a flanker task, indicating a deficit in executive control. Together, these results suggest that a control deficit may explain differential aphasia while still assuming an anatomically and functionally integrated bilingual lexicon.

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CHAPTER 4:
COGNATE EFFECTS AND COGNITIVE CONTROL IN PATIENTS WITH PARALLEL AND DIFFERENTIAL BILINGUAL APHASIA[1]

An unresolved hypothesis in the bilingual aphasia literature is the underlying mechanism explaining the selective loss of one language. We investigated whether a control deficit might underlie differential aphasia symptoms. It was suggested that the device involved in language control is part of a general control mechanism, managing both linguistic and non-linguistic cognitive control. If so, patients with differential aphasia should still show cross-lingual interactions, but additionally show a deficit on (non-linguistic) control tasks.

We compared bilinguals with parallel and differential aphasia with a control group. We used a lexical decision task to assess cross-lingual interactions and language control and a flanker task to assess non-linguistic control. We found a cognate effect in the three groups indicating intact cross-lingual lexical interactions. Additionally we found a larger congruency effect on accuracy rates in the patients with differential aphasia, suggesting a general cognitive control dysfunction that may explain the differential language loss.

[1] Verreyt, N., De Letter, M., Hemelsoet, D., Mariën, P., Santens, P., Stevens, M., & Duyck, W. Cognate effects and cognitive control in patients with parallel and differential bilingual aphasia. *(Submitted to Brain and Language).*

INTRODUCTION

Bilingualism is an increasingly growing cultural phenomenon. It is estimated that about half of the world's population is now bilingual. In the literature about bilingualism, bilinguals are typically considered to be individuals who regularly use their two languages (Grosjean, 1989). Given the large amount of bilingual people worldwide, it is not surprising that the occurrence of patients with bilingual aphasia has increased as well. Aphasia is defined here as a general impairment in understanding, formulating or using verbal messages, in spoken and/or written modality, caused by brain dysfunction in language related areas.

Interestingly, bilinguals who suffer from aphasia do not always encounter the same impairments in both languages, nor are both languages always affected to the same extent. In addition, language recovery does not always benefit both languages to the same degree. Paradis (2004) described six different patterns of language recovery in bilingual aphasic patients. When both languages recover in the same way, this is called parallel recovery (Marangolo, Rizzi, Peran, Piras, & Sabatini, 2009). When one language benefits more from recovery compared to the other, this is called differential recovery. It is important to notice that when recovery effects differ across languages, it is not always the case that recovery is more pronounced in L1 (i.e. the native language) (Goral, Levy, Obler, & Cohen, 2006; Meinzer, Obleser, Flaisch, Eulitz, & Rockstroh, 2007). For instance, Agliotti and Fabbro (1993) described a patient who showed better recovery in his second language (L2). Selective recovery is an extreme case of differential recovery. This term is used for patients in whom one language does not recover at all. In some patients, one language only starts to recover when the other has fully recovered. This is called successive recovery. In some patients, there is an alternation in recovery: one language starts to recover and weakens again while the other recovers. A last recovery pattern described by Paradis is when patients uncontrollably switch and mix their languages. This is called blended recovery (Adrover-Roig et al., 2011; Fabbro, Skrap, & Aglioti, 2000; Leemann, Laganaro, Schwitter, & Schnider, 2007; Mariën, Abutalebi, Engelborghs, & De Deyn, 2005; Riccardi, Fabbro,

& Obler, 2004). In this paper, we will not focus on the *recovery* patterns of the patients but on the pattern of *impairments*, for which Paradis' taxonomy can be used as well. For instance, the term parallel aphasia can be used to indicate patients who show similar impairments to the same extent in both languages. Patients that show larger impairments in one language compared to the other present with *differential* aphasia (Adrover-Roig et al., 2011; Agliotti, Beltramello, Girardi, & Fabbro, 1996; Goral et al., 2006; Vajramani, Akrawi, McCarthy, & Gray, 2008).

The occurrence of selective and differential aphasia is interesting for functional as well as neuro-anatomical theories of bilingualism because it suggests that languages can be selectively "damaged" and therefore should probably be represented in distinct areas in the brain. Early accounts of neural representation of bilingualism adopted such a "localised" hypothesis. With languages represented in separate neural regions in the brain, selective impairments in only one language are assumed to be caused by damage to the language specific area representing the (most) affected language. However, a lot of evidence has now been found against this early idea of functionally distinct and anatomically separated lexicons, both in behavioural and in imaging studies, which creates a quite intriguing puzzle.

In the psycholinguistic literature about bilingualism, the most investigated question has exactly been whether bilinguals do have two separate lexicons (one for each language), or do they instead have one language system and one integrated lexicon? And, directly related to this question, are both languages constantly active during language recognition, even in a unilingual context? Recently, quite compelling evidence has been found in favour of language non-selective lexical processing. Even when only one language is relevant, words from both languages are activated, and there is a constant and automatic interaction between the words of different languages, supporting a single lexicon view. An important line of research supporting this view comes from studies investigating cognate effects. Cognates are words that have the same meaning and a similar orthography/phonology in both languages, for example the Dutch – English word pairs *winter-winter* (identical) or *appel – apple* (non-identical). Research has shown that cognates are processed faster than non-cognates, also when only one language is relevant for the experiment, and also when

this is the native language (Duyck, Van Assche, Drieghe, & Hartsuiker, 2007; van Hell & Dijkstra, 2002). This is called the *cognate facilitation effect,* and is typically explained by convergent activation spreading from the cognate's similar semantic, orthographic and phonological representations across languages. Additional behavioural evidence supporting strong cross-lingual interactions comes from studies showing that words are recognised faster when they are preceded by a masked prime translation equivalent in a lexical decision task (Basnight-Brown & Altarriba, 2007; Dunabeitia, Perea, & Carreiras, 2010; Duyck & Warlop, 2009; Perea, Dunabeitia, & Carreiras, 2008; Schoonbaert, Duyck, Brysbaert, & Hartsuiker, 2009). All these studies support a view with a single integrated language system/lexicon.

Even stronger evidence against distinct brain areas representing the different languages of a bilingual has been found in imaging studies. It has been shown that in balanced[2] bilinguals, languages mostly rely on common areas in the brain (Abutalebi, Cappa, & Perani, 2001; Chee, Tan, & Theil, 1999; Kim, Relkin, Lee, & Hirsch, 1997; Klein, Milner, Zatorre, Evans, & Meyer, 1994; Lucas, McKhann, & Ojemann, 2004; Mahendra, Plante, Magloire, Milman, & Trouard, 2003; Price, Green, & von Studnitz, 1999; Vingerhoets et al., 2003).

The evidence for strongly interacting languages represented in overlapping brain areas seems highly contradictory with the occurrence of selective and differential impairments in bilingual aphasia. Interestingly, already long time ago this contradiction emerged. Pitres suggested an integration of these two opinions (1895). He argued that a deficit in language control may underlie selective and differential recovery in bilingual aphasia. He stated that every language can be independently inhibited, temporarily or permanently. According to this view, bilingual aphasia is not the result of a lesion in the neural substrate of a language, but rather the result of a functional inhibition of the language. Recently, this hypothesis gained renewed interest and was further elaborated by Green and Abutalebi (Green

[2] In unbalanced bilinguals, there is evidence for both overlapping and distinct brain regions respresenting both languages (Briellmann et al., 2004; De Bleser et al., 2003). The patients included in this study, however, may all be regarded as balanced bilinguals.

& Abutalebi, 2008; Green, 2005), who assumed two distinct devices for language processing: the bilingual lexico-semantic system, representing word meanings, word forms and syntactic properties, and a device involved in language control. In bilingual language production, it is hypothesised that words of both languages compete for selection. This control device then would be specifically involved in the activation of (words in) the target language, and in the inhibition of (words in) the non-target language. In patients with differential aphasia, it might be the case that this language selection mechanism is damaged, not the language representations itself, or that there is a disconnection between the semantic representation of the words and the language cues (Green, 2005). Importantly, Green (1998) suggested that this control mechanism was not solely responsible for language control but served as a more general control mechanism which was also involved in non-linguistic control. This might suggest that a deficit in this control mechanism not only leads to problems with language control (in this case: differential or selective aphasia) but might also cause control problems on non-linguistic tasks. The aim of this study is to relate differential language loss to such verbal and non-verbal cognitive control and to explore indications of lexical activation in the most affected language and resulting cross-lingual interactions despite (language) control problems.

Only a few studies have already investigated cross-lingual lexical interactions in bilingual aphasic patients by studying cognate effects. The first study assessed picture naming in a trilingual aphasic patient (Stadie, Springer, De Bleser, & Burk, 1995). The cognate status of the word did not influence naming accuracy in L1 but it did increase the amount of correctly named pictures in L2 and L3. Ferrand and Humphreys (1996) tested the matching of spoken English (L1) words to written French (L2) words in a multilingual aphasic patient and also found a better performance for cognates compared to non-cognates. Roberts and Deslauriers (1999) tested confrontation naming in a group of 15 bilingual aphasic patients and 15 control subjects and found a higher naming accuracy for pictures with cognate names compared to pictures with non-cognate names. Detry, Pillon and de Partz (2005) ran a word-picture verification task, a picture naming task and a translation task with cognate and non-cognate stimuli in a French-Italian bilingual aphasic patient. They found better performance for cognates

on the three tasks. These studies demonstrate that at least lexical interactions may emerge between languages in aphasic patients supporting partly preserved lexico semantic functionality. However, none of these studies focused on the relative degree of language loss or differentiated between parallel and differential aphasia and may therefore not answer the question at hand here.

More challenging for the hypothesis that brain regions representing the two languages of a bilingual are still intact and that bilingual aphasia is caused by a control deficit, is to investigate these cross-lingual interactions in patients with *differential aphasia*. We will investigate whether it is possible to observe lexical activation and cross-lingual interactions arising from the most affected language, despite larger functional loss of that language. This is a necessary (but not sufficient) condition to attribute differential aphasia to control. To the best of our knowledge, only one study has investigated cognate effects in a patient with differential aphasia: Lalor and Kirsner (2001) described a Greek-Italian bilingual who suffered from aphasia, showing larger deficits in Italian (L2) compared to Greek (L1). They found a partial cognate effect in naming (i.e. low-frequency Italian cognates with high-frequency translations were recognised faster than low-frequency Italian cognates with low-frequency translations). In addition, they also ran a generalised lexical decision task in which the patient had to decide whether a visually presented word is an existing word in either of the two languages, or not. When comparing cognates and non-cognates they did not find a difference in reaction times (probably because of the high variability), but they did found a difference in accuracy, with more errors for non-cognates compared to cognates. This finding shows that patients with bilingual differential aphasia may show cognate effects, reflecting at least partly preserved cross-lingual lexical interactions. The cognitive control performance of this patient, however, remains unknown.

Only two recent studies investigated whether patients with bilingual aphasia show a control deficit. Green and colleagues (2010) studied control functions in two patients with bilingual aphasia who both showed parallel recovery. An English (L2) lexical decision task was used to assess the ability to handle lexical (word/nonword) conflict, a verbal flanker task to explore the control of conflict with verbal responses, and a non-verbal flanker task to

assess the non-language related control abilities. A different pattern of results was found in both patients. Patient 1 only showed a deficit in verbal control (i.e. abnormally high conflict effects in the lexical decision and in the verbal Stroop task, but not in the non-verbal flanker task). Patient 2 showed abnormally high conflict effects in the non-verbal flanker task and in the lexical decision task, but less abnormal results in the verbal Stroop task. These results indicate that bilingual aphasia might be associated with control problems. However this relation is not unequivocal and the link between control problems and the precise aphasia pattern remains unclear, as only parallel patients were tested. In addition, we recently described the results of a patient with differential aphasia, who clearly showed cognitive control difficulties (Verreyt, De Letter, Hemelsoet, Santens, & Duyck, 2013). The present study will extend this previous work by investigating cognitive control in patients with parallel and differential aphasia.

THE PRESENT STUDY

The hypothesis of Pitres (1895) stating that the mechanism underlying selective and differential aphasia might be a control deficit has never been directly investigated. Only one study examined general cognitive and language control in bilingual aphasics (Green et al., 2010) and a few studies investigated lexical interactions (cognate effects) (Detry et al., 2005; Ferrand & Humphreys, 1996; Lalor & Kirsner, 2001; Roberts & Deslauriers, 1999; Stadie et al., 1995). However, in none of the studies both phenomena were investigated in relation to different patterns of language loss. In the present study we compared fairly large groups of patients with differential or parallel aphasia and a healthy control group. If indeed the two languages of bilinguals are represented in the same brain regions, and a deficit in a general control device underlies selective and differential aphasia, we would expect the patients with differential aphasia (a) to still show evidence for cross-lingual interactions, and (b) to show lower performance on a non-linguistic control task compared to patients with parallel aphasia and compared to a healthy control group.

The first aim of our study was to assess cross-lingual interactions in patients with bilingual aphasia by means of investigating the cognate facilitation effect. As described in the previous section, it has been shown that patients with bilingual aphasia show cognate effects indicating cross-lingual interactions (Detry et al., 2005; Ferrand & Humphreys, 1996; Lalor & Kirsner, 2001; Roberts & Deslauriers, 1999; Stadie et al., 1995). In the present study we assess cognate effects in a lexical decision task. More specifically, we used a generalised lexical decision task, in which words in any language, required a YES-response, and non-words required a NO-response. We chose this task because there is no need to inhibit representations in either of the languages (words in any language require a YES-response). Therefore, the (language) control demands are very low, making this task very suited to selectively tap into lexical interactions, without performance being affected by any control problem. These cross-lingual interactions will be marked by the cognate effect. We expected the patients to show cognate effects, just as the control subjects, and we expected both groups of patients (differential vs. parallel) to show similar effects.

The second aim of this study was to investigate non-linguistic control functions. We aimed to investigate the non-linguistic control functions of the participants by means of an Eriksen flanker task (Eriksen & Eriksen, 1974). The stimuli of this task typically consist of five items, most often arrows. Participants are required to selectively react to the direction of the central arrow. The arrows presented next to the central arrow can be pointing into the same direction of the central arrow (i.e. a congruent trials) or can be pointing into the opposite direction (i.e. an incongruent trial). Research has shown that incongruent trials on average yield more errors and slower reaction times compared to congruent trials. The difference in error rates or reaction times between incongruent and congruent trials is called the congruency effect. We expected the patients with differential aphasia to perform worse (i.e. to show a larger congruency effect) compared to patients with parallel aphasia and compared to the control subjects. To our knowledge, this is the first time cross-lingual interactions were investigated in patients with differential aphasia. In addition, no previous study has contrasted a relatively large number of patients with bilingual aphasia (eight

patients with parallel aphasia, and seven patients with differential aphasia). As such, this study may add to current insights in the bilingual brain, disentangling some important issues concerning bilingual aphasia and language control. We expected that especially differential aphasia patients would show impaired cognitive control, relative to controls and parallel aphasia patients.

METHODS

Participants

All patients were recruited in the University Hospital of Ghent and in ZNA Middelheim Hospital Antwerp (Belgium). The patients were referred to us by the neurologist (D.H.), the speech and language therapist (M.D.L.) or the neurolinguist (P.M.). Inclusion criteria were the following: (1) a very good knowledge of Dutch and French or English before the acute onset of vascular aphasia (as assessed by a language questionnaire); (2) formally diagnosed aphasia based on the Aachen Aphasia Test (AAT, Graetz, De Bleser, & Willmes, 1992) and (3) relatively spared comprehension, also based on aphasia test scores and on the assessment by the speech and language therapist. Patients suffering from a developmental disorder or from a serious cognitive or depressive illness were excluded from the study.

All patients were tested between two weeks and four weeks post-stroke. To examine pre-onset language proficiency and language use, we administered a language proficiency test, which was completed by the patient accompanied by a close family member. To assess language functions in Dutch, we used the Dutch version of the AAT (Graetz et al , 1992). We developed a French experimental version of the AAT to assess French language functions. For English, the Comprehensive Aphasia Test (CAT) was assessed (Howard, Swinburn, & Porter, 2004). In addition, we used part C of the Bilingual Aphasia Test (BAT – Dutch-French and Dutch-English version) to assess passive translation (Paradis & Libben, 1987).

The patients were assigned to the group of differential aphasia if they showed significant differences in their language scores (AAT/CAT scores) across both languages (*table 1 & 2*).

Table 1
Age, sex (F = Female, M = Male), and bilingualism (L1-L2 based on AoA) of the patients with parallel and differential aphasia

Subject	**Age**	**Sex**	**Bilingualism (L1-L2)**
Patients with differential aphasia			
D01	41	F	Dutch-French
D02	24	F	French-Dutch
D03	53	F	Dutch-French
D04	41	M	Dutch-English
D05	77	M	French-Dutch
D06	41	F	Dutch-French
D07	62	M	French-Dutch
Patients with parallel aphasia			
P08	63	M	Dutch-English
P09	80	F	Dutch-French
P10	45	F	Dutch-French
P11	56	M	Dutch-English
P12	41	F	Dutch-French
P13	61	F	Dutch-French
P14	63	M	Dutch-French
P15	59	F	Dutch-French

Table 2

Average percentiles on Aachen Aphasia Test (AAT) for Dutch and French, and Comprehensive Aphasia Test (CAT) for English. Ns.: non-significant difference between the language scores; (*) significant difference between the scores. Caveat: For the subtest "Token Test", the score reflects the amount of errors (so a higher score implies a lower performance); BAT-part C assesses recognition of translation equivalents.

	AAT/CAT			***Aphasia pattern***	***BAT - Part C***	
Sub-ject	*L1 (%ile)*	*L2 (%ile)*	*Significant difference between languages?*		L1-L2 (/5)	L2-L1 (/5)
Patients with differential aphasia						
D01	*78*	*42*	*	Wernicke's aphasia *Differential aphasia* (Dutch better preserved than French)	4	2
D02	*47*	*71,8*	*	Broca's aphasia *Differential aphasia* (Dutch better preserved than French)	5	5

D03	*78,6*	*62,4*	*	Broca's aphasia *Differential aphasia* (Dutch better preserved than French)	5	5
D04	*66,6*	*42,2*	*	Broca's Aphasia *Differential aphasia* (Dutch better preserved than English)	2	1
D05	*86*	*73,6*	*	Amnestic aphasia *Differential aphasia* (French better preserved than Dutch)	5	4
D06	*78,8*	*55,6*	*	Wernicke's (Dutch) and Broca's (French) aphasia *Differential aphasia* (Dutch better preserved than French)	5	5

D07	*66,8*	*46,8*	*	Wernicke's aphasia *Differential aphasia* (French better preserved than Dutch)	5	5
Patients with parallel aphasia						
P08	*77,4*	*73,2*	*Ns.*	Broca's aphasia *Parallel aphasia*	5	5
P09	*86,2*	*85,4*	*Ns.*	Amnestic aphasia *Parallel aphasia*	5	5
P10	*92,6*	*87,8*	*Ns.*	Amnestic aphasia *Parallel aphasia*	5	5
P11	*93,8*	*88,6*	*Ns.*	Amnestic aphasia *Parallel aphasia*	5	5
P12	*92*	*91,2*	*Ns.*	Amnestic aphasia *Parallel aphasia*	5	5
P13	*78,4*	*88,2*	*Ns.*	Amnestic aphasia *Parallel aphasia*	5	5
P14	*57,8*	*70,6*	*Ns.*	Broca's aphasia *Parallel aphasia*	5	5

P15	*10*	*21,2*	*Ns.*	Wernicke's aphasia	5	5
				Parallel aphasia		

We tested 19 control subjects that were recruited among family and friends of the patients and the authors. The control subjects were matched with the patients for age, sex, education, and self-rated proficiency in L1 and L2 (based on the language background questionnaire) (*table 3*).

Table 3
Demographic data of the patients with parallel and differential aphasia, and the control subjects

	Parallel aphasia *N* = 8	**Differential aphasia** *N* = 7	**Control subjects** *N* = 19	*difference*
Male/female	3/5	3/4	4/15	$\chi^{22}_{(2)} = 1.501$; $p>.47$
Age	58.50 (11.98)	48.43 (17.60)	55.68 (12.37)	$F(2,33) = 1.127$; $p > .34$, *MSE* = 181.155
Education	14.75 (2.96)	15.00 (2.45)	15.16 (2.52)	$F(2,33) = .069$; $p > .93$, *MSE* = 6.840
L1 proficiency (pre-onset)	5.00 (0.0)	5.00 (0.0)	5.00 (0.0)	*Nsig.*
L2 proficiency (pre-onset)	3.71 (0.72)	3.95 (0.12)	3.89 (0.80)	$F(2,33) = .273$; $p > .76$, *MSE* = .493

Stimuli and materials

Lexical decision task

We administered a generalised lexical decision (LD) task in which all words (cognates and non-cognates) in either of the patient's both languages required a YES-response (pressing a right button with a green sticker), whereas the nonwords required a NO-response (pressing a left button with a red sticker). Because both Dutch-French and Dutch-English bilinguals were included in the study, we had a Dutch-French and a matched Dutch-English version of the task. The stimuli used in the LD task were 30 Dutch-French/English cognates, 30 Dutch non-cognates, 30 French/English non-cognates and 90 non-words. Both French/English and the Dutch cognates were used. Cognates and non-cognates were matched for word length, frequency, neighbourhood size and imageability using the WordGen stimulus generation software (Duyck, Desmet, Verbeke, & Brysbaert, 2004).

Flanker task

Each stimulus of the flanker task consisted of five arrows horizontally presented on the screen. The central arrow could be pointing to the left or to the right, flankers could be pointing in the same direction as the central arrow (congruent trials) or in the opposite direction (incongruent trials). We included 40 congruent and 40 incongruent trials. The patient had to react to the central arrow by pressing a left button (i.e. the *enter* button) or the right button (i.e. the *capslock* button).

RESULTS

Demographic data are shown in *table 3*. The groups did not differ significantly in male/female ratio, age, years of education, (pre-onset) L1 proficiency and (pre-onset) L2 proficiency.

Lexical decision task

A boxplot of the distribution of the reaction times for the three groups showed a much larger variability in the group of patients with differential aphasia compared to the other two groups. Therefore, we calculated the outlier boundaries for each group separately.

Accuracy

The accuracy rates as a function of group and status can be found in *table 4.* We ran a logistic linear mixed effects analysis with Group (control subjects, patients with parallel aphasia, patients with differential aphasia) and Status (cognate, non-cognate) as the independent variables, accuracy as the dependent variable and participant as the grouping variable. We included a random intercept and a random slope for Status ($\chi^2_{(2)} = 270.54$, $p < 0.001$). The main effect of Group was not statistically significant ($\chi^2_{(2)} = 1.40$, $p = 0.497$), which means that the three groups performed equally correct. The main effect of Status did reach significance ($\chi^2_{(1)} = 66.47$, $p < 0.001$), showing higher accuracy rates on cognates compared to non-cognates. The size of this cognate effect did not significantly differ across groups ($\chi^2_{(2)} = 0.06$, $p = 0.969$), indicating that groups did not differ substantially with respect to cross-lingual lexical interactions *(figure 1)*.

Table 4
Accuracy rates (% correct) and reaction times (ms) for control subjects, patients with parallel aphasia and patients with differential aphasia on cognates and non-cognates.

	Accuracy (% correct)		**Reaction times (ms)**	
	Cognates	**Non-cognates**	**Cognates**	**Non-cognates**
Control subjects	97.22	82.04	850.34	905.91
Patients with parallel aphasia	95.00	75.25	1237.40	1408.54
Patients with differential aphasia	89.52	74.05	1230.05	1338.50

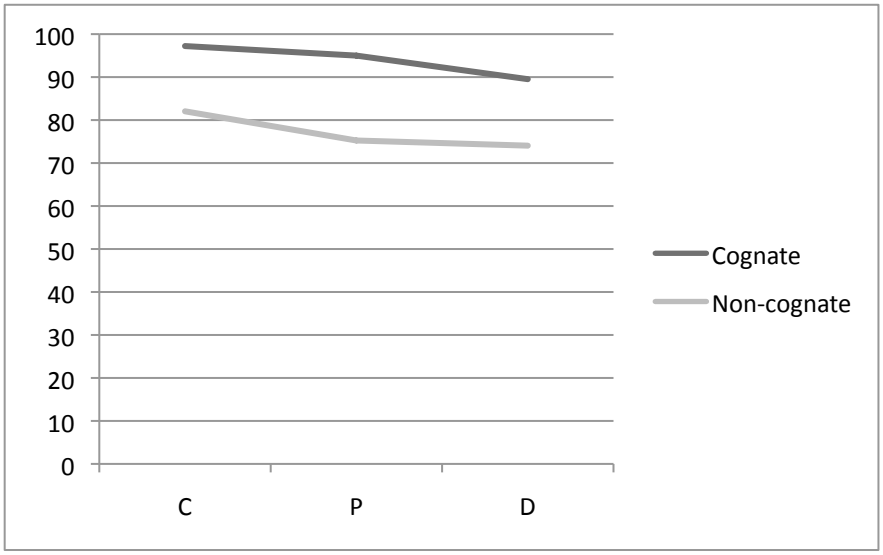

Figure 1. Accuracy rates for cognates and non-cognates for the three participant groups (C = Control group, P = patients with Parallel aphasia, D = patients with Differential aphasia)

Reaction times

We ran a linear mixed effects model with Group (control subjects, patients with parallel aphasia, patients with differential aphasia) and Status (cognate, non-cognate) as independent variables. The dependent variable was reaction times. Only correct trials were included in the analyses. We included a random intercept and a random slope for Status ($\chi^2_{(2)} = 6.03$, $p < 0.048$). The main effect of Group was statistically significant ($\chi^2_{(2)} = 10.24$, $p < 0.006$). Both patient groups differed significantly from the control group ($\beta = 509.93$, $z = 2.58$, $p < 0.010$ for the patients with parallel aphasia, and $\beta = 529.57$, $z = 2.56$, $p < 0.010$ for the patients with differential aphasia), but did not significantly differ from each other ($\beta = 19.65$, $z = 0.082$, $p > 0.934$). A statistically significant main effect of Status was found as well ($\chi^2_{(1)} = 14.35$, $p < 0.001$). Reaction times on cognates were shorter compared to non-cognates, in each of the three groups. This cognate effect did not significantly differ in size across groups ($\chi^2_{(2)} = 3.65$, $p > 0.161$), again indicating a similar degree of cross-lingual lexical interactions *(figure 2)*.

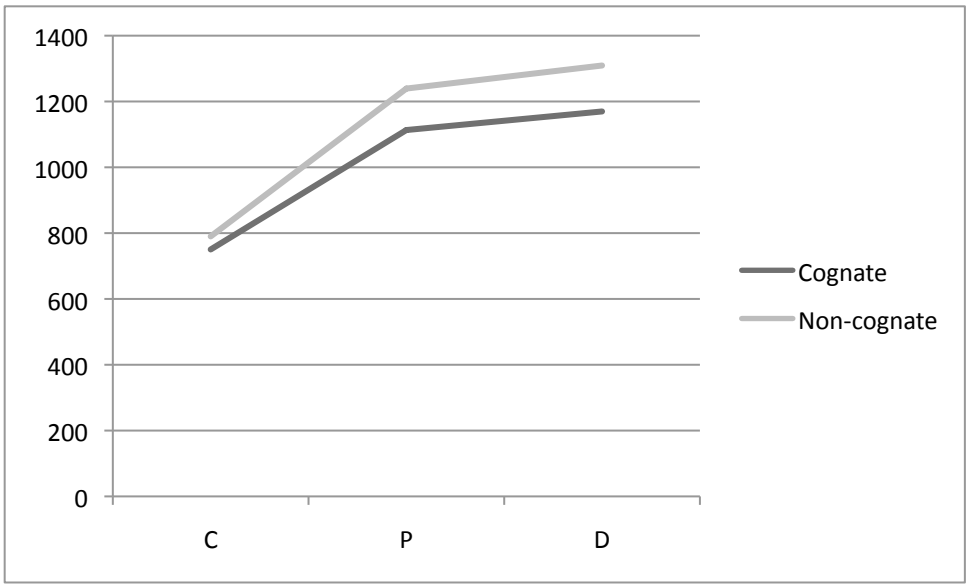

Figure 2. Reaction times on cognates and non-cognates for the three participant groups (C= Control group, P= patients with Parallel aphasia, D= patients with Differential aphasia)

Flanker task

A boxplot of the distribution of the reaction times for the three groups showed a much larger variability in the group of patients with differential aphasia compared to the other two groups. Therefore, we calculated the outlier boundaries for each group separately.

Accuracy

We ran a logistic linear mixed effects model with Group (control subjects, patients with parallel aphasia, patients with differential aphasia) and Status (congruent or incongruent) as independent variables. As the random slope for Status was not significant ($\chi^2_{(2)} = 0.0041$, $p > 0.998$) we only included a random intercept. No main effect reached significance, the interaction between Status and Group was marginally significant ($\chi^2_{(2)} = 5.019$, $p < 0.081$). Further analyses show that both the control subjects and the patients with parallel aphasia showed no differences in accuracy between congruent and incongruent trials ($\beta = 0.18$, $z = 0.49$, $p > 0.621$ for the control subjects, $\beta = 0.16$, $z = 0.33$, $p > 0.740$ for the patients with parallel aphasia), which is probably due to a ceiling effect in these groups. The patients with differential aphasia, however, did show a significant congruency effect ($\beta = -0.94$, $z = -2.32$, $p < 0.020$), showing higher accuracy on congruent trials (99.29%) compared to incongruent trials (96.07%), suggesting weaker cognitive control skills specifically for these patients (*table 5 and figure 3*).

Table 5
Accuracy rated and reaction times (ms = milliseconds) for the three groups of participants on congruent (C) and incongruent (IC) trials, and the congruency effect (CE) for each group, calculated as the difference of the values on IC trials and the values of congruent trials.

	Accuracy rate (% correct)			**Reaction times (ms)**		
	C	**IC**	**CE**	**C**	**IC**	**CE**
Control subjects	99.21	99.47	-0.26	519.02	532.77	13.75
Patients with parallel aphasia	99.06	99.38	-0.32	685.03	697.98	12.95
Patients with differential aphasia	99.29	96.07	3.22	766.86	792.23	25.37

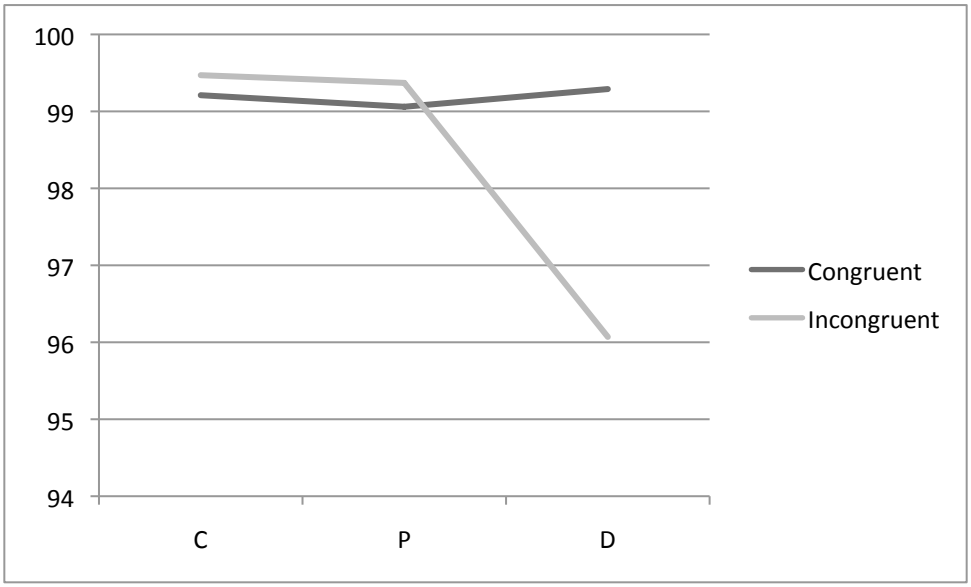

Figure 3. Accuracy for the congruent and the incongruent trials in the flanker task, for the three groups (C= Control group, P= patients with Parallel aphasia, D= patients with Differential aphasia)

Reaction times

We ran a linear mixed effects model with Group (control subjects, patients with parallel aphasia, patients with differential aphasia) and Status (congruent or incongruent) as the independent variables. The dependent variable was reaction times. Only correct trials were included in the analyses. As the random slope for Status was not significant ($\chi^2_{(2)} = 0.04$, $p > 0.976$). We found a significant main effect of Group ($\chi^2_{(2)} = 12.042$, $p < 0.002$). More specifically, both the patients with parallel aphasia (691.51 ms) and the patients with differential aphasia (779.55 ms) showed significant slower reaction times compared to the control group (525.90 ms) ($\beta = 174$, $z = 2.32$, $p < 0.020$ for the patients with parallel aphasia, $\beta = 247$, $z = 3.12$, $p < 0.002$ for the patients with differential aphasia). Both patient groups did not differ significantly from each other ($\beta = 72$, $z = 0.78$, $p > 0.435$). In addition, we also found a significant main effect of Status, showing slower

RTs for incongruent trials (623.48 ms) compared to congruent trials (607.13 ms) ($\chi^2_{(1)}$ = 8.329, p < 0.004). The interaction effect between Group and Status did not reach significance ($\chi^2_{(2)}$ = 1.608, p > 0.448), indicating that the size of the flanker effect (i.e. the difference between RTs on congruent and on incongruent trials) did not differ across groups in terms of RTs, unlike the effect observed for accuracy (see above). *Table 5* and *figure 4* show the reaction times for the three groups.

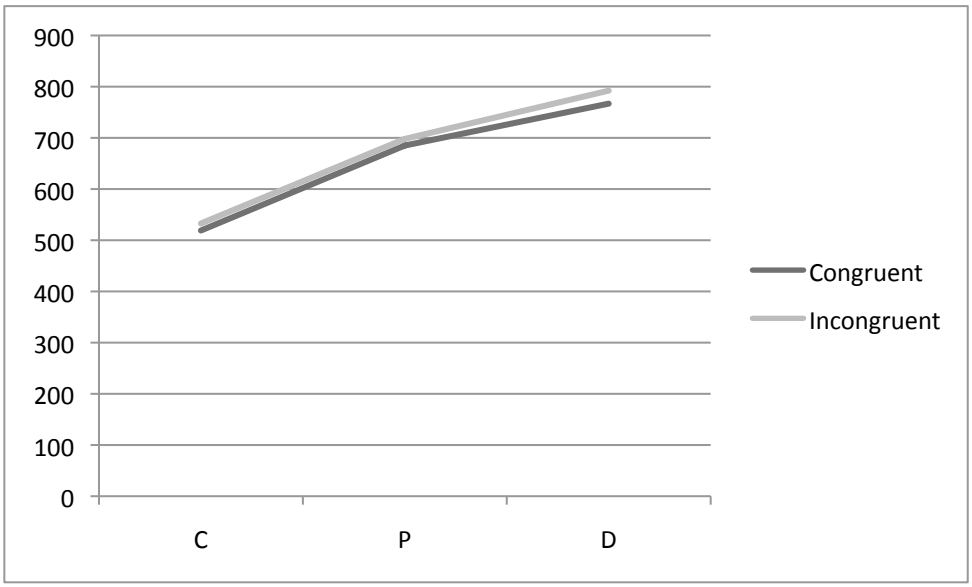

Figure 4. Reaction times for congruent and incongruent trials of the Flanker task in the three groups (C= Control group, P= patients with Parallel aphasia, D= patients with Differential aphasia)

DISCUSSION

Bilingual patients who suffer from aphasia do not always show similar deficits in both languages. It is unclear how this may be reconciled with all the behavioural (Basnight-Brown & Altarriba, 2007; Dunabeitia et al., 2010; Duyck et al., 2007; Duyck & Warlop, 2009; Perea et al., 2008; Schoonbaert et al., 2009; van Hell & Dijkstra, 2002) and neuroimaging

(Abutalebi et al., 2001; Chee et al., 1999; Kim et al., 1997; Klein et al., 1994; Lucas et al., 2004; Mahendra et al., 2003; Price et al., 1999; Vingerhoets et al., 2003) evidence supporting a single, integrated language system for both languages. In this study, we included vascular patients with larger deficits in one language compared to the other (i.e. differential aphasia). It was suggested that a control problem might be underlying the differential damage in both languages. In that case, not the loss of the language, but rather the inability to inhibit and/or activate the language would cause the deficits in the most severely affected language. If this is assumption correct, than patients with differential aphasia should still show effects of cross-lingual interactions. Interestingly, Green (1998) suggested that the system involved in language control is also responsible for control on language unrelated tasks. So, despite intact lexical interactions, we also assumed differential aphasia patients to perform worse on tasks tapping into cognitive control. This is the first time this hypothesis is fully and directly tested, comparing both lexical processing and cognitive control functions between two groups of patients with parallel versus differential aphasia.

To investigate cross-lingual activation, we used a generalised lexical decision task with cognates, which allows to tap into lexical processing, without imposing large language control demands (as is the case in a language-selective lexical decision task). We hypothesised that patients with differential aphasia would show similar performance compared to patients with parallel aphasia and healthy control subjects. Indeed, these predictions were confirmed. A clear cognate facilitation effect was found: all three groups performed better on cognates compared to non-cognates. The size of this cognate effect did not differ across groups: the three groups showed a similar difference in RTs and error rates between cognates and non-cognates. This indicates that the three groups each showed an equal amount of cross-lingual interactions. They all had a similar advantage of the activation of the cognate in both languages. This is of importance to the group with differential aphasia. Finding similar cross-lingual interaction effects compared to healthy control subjects suggests that both languages are still active and able to influence each other. This finding argues against a strict localised account for differential aphasia, which suggests that the selective impairment of one language is due to the selective damage to the language

specific area. This would predict smaller lexical activation in one language and a smaller (or absent) cognate effect. Our findings rather suggest that the representations of both languages are still intact and able to influence linguistic processing in the other language. This is in line with other studies reporting effects of cross-lingual interaction in patients with bilingual aphasia (Detry et al., 2005; Ferrand & Humphreys, 1996; Roberts & Deslauriers, 1999; Stadie et al., 1995) and with the only study that also assessed cognate effects in patients with differential aphasia (Lalor & Kirsner, 2001).

To assess non-verbal cognitive control, we used a flanker task comparing both our patient groups with a healthy control group. Both the patients with parallel aphasia and the control subjects performed almost at ceiling level on both congruent and incongruent trials, yielding no congruency effect. However, patients with differential aphasia made significantly more errors on incongruent trials compared to congruent trials. This confirms our prediction that patients with differential aphasia indeed show worse cognitive control relative to both controls and parallel aphasia patients. They experience more problems inhibiting irrelevant information (i.e. the incongruent flankers). This supports the control hypothesis proposed by Pitres (1895) and elaborated by Green and Abutalebi (2008), which proposes that a deficit in (language) control may underlie differential (and selective) aphasia in bilingual patients.

Neuro-anatomically, the brain regions responsible for control might involve frontal attentional and subcortical mechanisms (Price et al., 1999). Fabbro, Peru and Skrap (1997) have described several circuits involved in language control. For language planning, they proposed a loop consisting of the prefrontal cortex, the caudate nucleus, the globus pallidus, and the ventral anterior thalamic nucleus. In bilinguals, this loop might be specifically important in the selection of the target language and inhibition of the non-target language in language production (Abutalebi, Miozzo, & Cappa, 2000; Mariën et al., 2005). In addition, Stroop-like tasks also show increased activation in the anterior cingulate cortex. An important aim for future research might be to link the pattern of aphasia with the localisation of brain damage, to disentangle neuro-anatomic representations. However, given that none of our patients exclusively showed frontal damage, brain

damage specifically leading to differential aphasia may be hard to detect. The focus should likely be on disconnectivity rather than localised damage, and sophisticated connectivity analyses between frontal areas and language regions may be necessary. Such analyses are not available for the present group of patients. A detailed analysis of a very clear selective or differential aphasia case may test this in the future.

Our findings concerning cognate effects imply that speech and language therapists should be aware of the cognate status of their stimuli, and the possible effects this might have. The effects of cognate status have already been investigated in bilingual aphasia treatment. Kohnert (2004) investigated cognate effects in treatment in a Spanish-English bilingual patient with severe transcortical motor aphasia, showing parallel impairments in both languages. The treatment consisted of two weeks of lexical-semantic therapy in both written and spoken language. During the first week, the therapy language was Spanish, whereas during the second week treatment was given in English. Half of the stimuli that were used in the treatment were cognates. Generalisation of treatment effects from trained to untrained items within one language was found for both cognate and non-cognates. However, generalisation across languages only occurred for cognates. Kurland and Falcon (2011) also studied cognate effects in aphasia treatment but found opposite results. They treated a Spanish-English bilingual with severe expressive aphasia in both languages. Similar to Kohnert (2004), they also treated the patient during the first phase in Spanish and during the second phase in English, but these phases lasted for two weeks (unlike one week in the Kohnert study), and they added a third phase, in which the patient was treated in both Spanish ànd English. Half of the training stimuli were cognates. In the three treatment phases there was an increased naming accuracy for naming non-cognates, but not for cognates. The authors assumed that this effect was due to interference coming from words with a similar phonology and meaning. These two studies imply that practitioners should be aware of the cognate status of the stimuli they use for aphasia treatment. Both the findings of Kohnert (2004) and the findings of Kurland and Falcon (2011) indicate cross-lingual interactions, and confirm that functionally affected languages may still influence processing in another language.

Taken together, these findings seem to provide new insights in the underlying mechanism of differential aphasia, by supporting the hypothesis that differential patterns of bilingual aphasia are not due to selective loss of one (i.e. the most affected) language. Our results provide a two-sided argument for a control deficit underlying differential aphasia. First, the equivalent cross-lingual lexical interactions observed for differential aphasia patients show that these patients do not primarily differ from parallel aphasia patients in terms of lexical interactions, at least when language control demands are low (such as in the generalized lexical decision task). Second, the increased number of errors in the cognitive control tasks instead suggests that the pattern of language loss may instead by attributed to a cognitive control dysfunction that also generalizes to the non-verbal domain.

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CHAPTER 5:
SYNTACTIC PRIMING IN BILINGUAL PATIENTS WITH PARALLEL AND DIFFERENTIAL APHASIA[1]

Background: Syntactic priming is the phenomenon by which the production or processing of a sentence is facilitated when that sentence is preceded by a sentence with a similar syntactic structure. Previous research has shown that this phenomenon also occurs across languages, i.e., hearing a sentence in one language can facilitate the production of a sentence with the same structure in another language. This suggests that syntactic representations are shared across languages.

Aims. The aim of the current study is to investigate this cross-lingual syntactic priming in patients with bilingual aphasia. To address this aim, we asked the following three research questions: (1) Do patients with bilingual aphasia show priming effects within and across languages? (2) Do these priming effects differ from the priming effects observed in control participants? and (3) Does the pattern of priming effects interact with the type of aphasia?

Methods and procedures. We tested two groups of patients: one group had similar impairments in both languages (parallel aphasia); in the other group, the impairments were larger in one of the languages (differential aphasia). We investigated syntactic priming within and across languages by means of a dialogue experiment.

Outcomes and results. We found significant cross-lingual priming effects in both patient groups as well as in a control group. In addition, the effect size of both patient groups was similar to that of the control group.

Conclusion. These findings support models that incorporate shared syntactic representations across languages, and are in favour of a non-localised account of differential aphasia in bilingual aphasia.

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[1] Verreyt, N., Bogaerts, L., Cop, U., Bernolet, S., De Letter, M., Hemelsoet, D., Santens, P. & Duyck, W. (2013) Syntactic priming in bilingual patients with parallel and differential aphasia. *Aphasiology*. DOI:10.1080/02687038.2013.791918

INTRODUCTION

In psycholinguistics, bilinguals are individuals who master and use two or more languages, but are not necessarily equally proficient in both (Grosjean, 1989). As the world's population is becoming more bilingual, it is not surprising that the number of bilingual patients with aphasia increases as well (Lorenzen & Murray, 2008). Aphasia is defined as a general impairment in understanding, formulating or using verbal messages, in spoken and/or written modality, caused by brain dysfunction to language-related areas. The main cause of aphasia is a stroke, but a tumour, an infection or degenerative brain diseases can also lead to aphasia.

Aphasia in bilingual patients does not always affect both languages to the same extent, nor do both languages always recover to the same degree. More specifically, Paradis (2004) described six different recovery patterns in bilingual aphasia (*table 1*).

Analogously to these *recovery* patterns, some impairment patterns might be described using similar terms (i.e., parallel, differential, and selective impairment). For example, a patient with one language that is more affected than the other, or with qualitatively different impairments in one language compared to the other, is diagnosed with differential aphasia (Agliotti & Fabbro, 1993; Goral, Levy, Obler, & Cohen, 2006; Meinzer, Obleser, Flaisch, Eulitz, & Rockstroh, 2007).

Table 1

Recovery patterns described by Paradis (2004)

Recovery pattern	**Description**	**References**
Parallel recovery	Both languages recover with the same speed and/or to the same extent.	(Marangolo, Rizzi, Peran, Piras, & Sabatini, 2009)
Differential recovery	Recovery is more pronounced in one language compared to the other, the recovery in both languages differs qualitatively.	(Aglioti & Fabbro, 1993; Goral, Levy, Obler, & Cohen, 2006; Meinzer, Obleser, Flaisch, Eulitz, & Rockstroh, 2007)
Selective recovery	One language does not recover at all.	
Successive recovery	One language only starts to recover when the other one has fully recovered.	
Antagonistic recovery	An alternation in the recovery of both languages.	
Blended recovery	Patients uncontrollably switch and mix their languages during recovery.	(Adrover-Roig et al., 2011; Fabbro, Skrap, & Aglioti, 2000; Leemann, Laganaro, Schwitter, & Schnider, 2007; Marien, Abutalebi, Engelborghs, & De Deyn, 2005; Riccardi, Fabbro, & Obler, 2004)

Initially, the phenomena of selective and differential aphasia were explained by the idea that languages of a multilingual are represented separately in distinct areas in the brain. It was hypothesised that selective impairment of one language was due to selective damage to the specific brain area representing that particular language. However, much evidence has now been gathered in healthy bilinguals falsifying the hypothesis of language specific brain areas. First, at a functional level, the two languages of a bilingual always seem to be strongly interacting. A strong illustration of this constant interlingual interaction is the cognate effect. Cognates are words with the same meaning and a similar form in different languages (e.g., English–Dutch [film]–[film]). It was found that cognates are processed faster than non-cognates (i.e. the cognate facilitation effect, Dijkstra, Grainger, & Van Heuven, 1999), even if the non-target language should not be activated for the task at hand. For example, Dutch-English cognates are processed faster than non-cognates when reading Dutch sentences in an exclusively Dutch context (Duyck, Van Assche, Drieghe, & Hartsuiker, 2007). The effect also generalizes to word production: pictures with cognate names are named faster than pictures with non-cognate names (Costa, Caramazza, & Sebastian-Galles, 2000). This is commonly explained by convergent activation spreading from the cognate's similar representations across languages. According to cascade models of language production and comprehension, the production or comprehension of a word activates the semantic, orthographic and phonological representation of that word (Dell, 1986). This activation spreads to words with a related meaning, orthography or phonology. In the case of cognates, orthographic and phonologic nodes in both languages become activated, which leads to a higher activation, and therefore faster comprehension or production.

In addition to the behavioural evidence for cross-lingual interactions, neuroscience studies have found that the languages of a multilingual person are represented in overlapping areas in the brain (Hernandez, Martinez, & Kohnert, 2000; Illes et al., 1999). For example, Vingerhoets et al. (2003) investigated brain activation for word generation, word fluency and picture naming in Dutch, English and French within the same subjects, and found largely overlapping brain activation in the three languages (See also Klein, Zatorre, Milner, Meyer, & Evans, 1994).

At first sight, the behavioural and imaging evidence for a single integrated language system may seem contradictory with the existence of differential and selective aphasia: if both languages rely on the same (or highly overlapping) neural structures, how may brain damage have larger effects in one language than in the other? This question may be traced back to a 1895 claim of Pitres, which may account for this discrepancy. He claimed that "*every language could be independently inhibited, temporarily or permanently. Thus bilingual aphasia should not be the result of a lesion in the neural substrate of a language, but rather the result of a functional inhibition of the language."* (Pitres, 1895). In other words, selective or differential aphasia is here explained by a problem of language control, i.e., in the selection of words in the intended language, and the inhibition of words in uninttended languages, rather than to a selective lesion in language specific neural representations themselves.

In the present paper, Pitres' hypothesis will be applied to the syntax level. In addition to cross-lingual interaction effects at the word level, the syntactic priming literature has also revealed cross-lingual interactions at the syntactic level. Research has shown that the processing and production of a sentence is facilitated when the sentence is preceded by a sentence with a similar syntactic structure. For example, after hearing a passive sentence, a person will be inclined to produce a passive sentence rather than an active one. This is called *syntactic priming* (Bock, 1986), and is quite a robust effect. It has been found in a range of paradigms, such as written sentence completion (Pickering & Branigan, 1998), sentence recall (Potter & Lombardi, 1998), and spoken dialogue experiments, in which participants describe pictures to each other (Branigan, Pickering, & Cleland, 2000).

Important for the current study is that syntactic priming has also been found across languages. Hartsuiker, Pickering and Veltkamp (2004) found syntactic priming for transitive sentences in a dialogue experiment with Spanish-English bilinguals. They found that the bilinguals produced an English (L2) passive sentence more often after hearing a passive Spanish (L1) sentence than after an active Spanish sentence (See also Hartsuiker & Pickering, 2008; Meijer & Tree, 2003; Shin & Christianson, 2009 for cross-lingual priming with datives). Cross-lingual syntactic priming has also been found in the opposite direction (L2-L1). Schoonbaert and colleagues studied

syntactic priming with dative sentences in a group of Dutch–English bilinguals, and found significant priming effects within and across languages (Schoonbaert, Hartsuiker, & Pickering, 2007).

One of the mechanisms proposed to account for this cross-lingual priming is that residual activation of the structure of the previously heard or produced sentence might influence the choice for a current structure. Pickering and Branigan (1998) incorporated this assumption in the model of language production by Levelt (Levelt, Roelofs, & Meyer, 1999), to support syntactic priming effects. This model stated that in monolingual language production, the encoding of syntactic information (e.g., the arguments of a verb) is situated on the lemma level. Pickering and Branigan claim that the lemma nodes are connected to category nodes, indicating the word type, and combinatorial nodes, representing in which grammatical constructions the word can occur. When a verb can occur in an active and a passive construction, the lemma node of the verb will be connected with two different combinatorial nodes (i.e., an active node and a passive node). Thus, when the verb is used in a passive sentence, both the lemma node of the verb and the passive combinatorial node will become activated. The model further assumes that these combinatorial nodes are shared between lemma nodes, implying that for instance every verb that can be used in the passive voice will be connected to the passive combinatorial node. Syntactic priming effects are explained as follows: hearing (or producing) a passive sentence will activate the verb and the passive combinatorial node. When the next sentence is produced, the previously activated passive combinatorial node will still be residually active, and will facilitate the subsequent production of a passive sentence.

Previous research has also provided evidence for shared syntactic representations across languages (see above). Therefore, Hartsuiker et al. (2004) extended the model of Pickering and Branigan (1998) to bilinguals. They claim that syntactic information in proficient bilinguals is shared between languages, i.e., lemma nodes of verbs in both languages are connected to the same combinatory nodes (see *figure 1*).

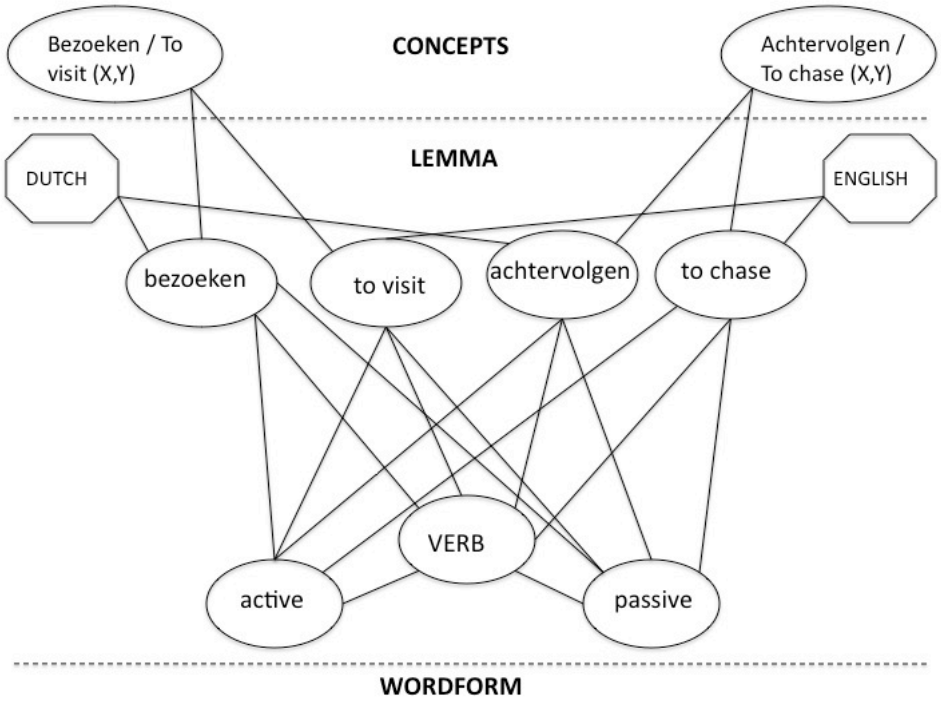

Figure 1. The model for cross-lingual syntactic priming proposed by Hartsuiker et al. (2004) applied to a Dutch-English example. In this model, the lemma nodes of the verbs [bezoeken] / [to visit] and [achtervolgen] / [to chase] are connected to a shared active combinatory node, and a shared passive combinatory node. Each lemma node is also connected to a category node for verb, and a language node (Dutch or English).

In this study, we aimed to investigate whether patients with bilingual aphasia still show cross-lingual syntactic priming effects, and whether such effects depend on the type of aphasia (here: parallel vs. differential loss across languages). Until now, only two studies investigated syntactic priming in aphasic patients. This is regrettable, given the fact that aphasia is often not only characterized by dysfunctions at the lexical level, but also at the grammatical level. Applying this paradigm from monolingual psycholinguistic literature may add to our understanding of these

dysfunctions. Because syntactic priming increases the availability of a grammatical structure, one would assume that patients who show impaired sentence production might benefit from this phenomenon. This is indeed what was found by Saffran and Martin (1997) on syntactic priming in aphasic patients. They investigated syntactic priming in a small group of patients with impaired sentence production. Firstly, participants completed a baseline exercise, in which they were asked to describe pictures that were not preceded by a prime. This was followed by a dialogue experiment. Here, patients heard a prime sentence, which they were asked to repeat. Subsequently they were asked to describe a target picture. This was done both with dative and transitive sentences. After the dialogue experiment the items used in the baseline test were administered again. During the dialogue experiment significant priming effects were observed with transitive, but not with dative sentences. Analysis of the baseline performance showed that patients produced more passives after the dialogue experiment than before. This suggests that there was also a long-term priming effect, illustrating the potential value of this kind of research for therapeutic settings (i.e. extensively practising certain grammatical structures in therapy might enhance daily communication in patients with aphasia).

In the second study, Hartsuiker and Kolk (1998) extended these findings by comparing a group of Broca-aphasic patients with matched control participants. They also used transitive and dative sentences in a dialogue experiment, framed somewhat differently. Firstly, participants were told that they participated in a memory experiment, in which they were asked to indicate whether they had already seen the sentence or picture during the session or not. The participants were asked to read the sentences out loud and to describe the pictures "to facilitate the recognition". No specific instructions were given in the second condition; participants were only asked to describe pictures and read sentences out loud. In the third condition, participants were explicitly asked to use the grammatical structure of the previous sentence. In general, participants had to carry out the same crucial task in each of the three conditions, namely describing pictures that were preceded by prime sentences. The patient group showed significant priming effects for passives, double object datives and prepositional datives, in the three conditions. The size of the priming effects did not differ across

conditions. However, in the control group significant priming effects were only observed in the third condition, in which they were specifically asked to re-use the structure of the previously heard sentence. The authors explained the null priming effects in control subjects as a consequence of task difficulty, or of the use of a relatively small control group.

The finding that aphasic patients show priming effects even though spontaneous production is impaired, suggests that grammatical representations in themselves are not 'lost', but instead harder to functionally access. Both studies showed that aphasic patients used hardly any passive sentences spontaneously, whereas they did produce passives during and after the experiment. This suggests a positive influence of syntactic priming on the quality of language (sentence) production.

THE PRESENT STUDY

With the current study we aimed to go a step further than the existing syntactic priming studies in monolingual aphasia, by investigating *cross*-lingual syntactic priming in *bilingual* aphasic patients. To our knowledge, cross-lingual syntactic priming has never been investigated in relation to aphasia. We also aimed to assess whether such syntactic priming patterns are dependent on relative language loss by comparing a group of patients with parallel aphasia (i.e., having expressive and receptive impairments to the same extent in both languages) and a group of patients with differential aphasia (i.e., having larger impairments in one language compared to the other, or showing different impairments in one language compared to the other). More specifically, we studied the following three research questions: (1) Do patients with bilingual aphasia show priming effects within and across languages? (2) Do these priming effects differ from the priming effects observed in control participants? and (3) Does the pattern of priming effects interact with the type of aphasia[2]? For the latter, we were

[2] Caveat. With "type of aphasia" we do not refer here to the classical distinction between Broca and Wernicke aphasia, but rather to the distinction between parallel and differential aphasia.

specifically interested in the difference between patients with parallel aphasia and patient with differential aphasia. Testing patients with differential aphasia allowed us to investigate whether we could still find syntactic priming from the most affected language as the prime language: are syntactic representations that are most dysfunctional still able to influence production in another language? Within the view that the underlying mechanism of selective and differential aphasia is a problem in cognitive control and not in a brain area representing a single language (Pitres, 1895), we expected that patients with differential aphasia could still show syntactic priming from their most affected language, because the representations themselves are intact, as loss of functionality in spontaneous productions could reflect a language control problem instead. If this hypothesis is correct, production in the most affected language fails because there is a problem in activation or inhibition of the target or non-target language, respectively, however not because the target language representations themselves are dysfunctional. In the syntactic priming paradigm, however, the primes in the most affected language need to be comprehended, not produced. This requires less language control, because language selection is not strictly necessary for comprehension (one may just rely on bottom-up activation from the input), unlike production (Costa & Santesteban, 2004). As such, activation of syntactic representations after comprehension of the prime in the most affected language might still transfer to production of the same grammatical structure in the best-preserved language.

METHOD

We investigated these research questions by using a dialogue experiment in which a confederate and a participant were asked to describe pictures to each other (Bernolet, Hartsuiker, & Pickering, 2007; Branigan et al., 2000). We used four language-conditions: two conditions in which we tested within-language priming (L1-L1, L2-L2) and two conditions in which we tested between-language priming (L1-L2, L2-L1). Firstly, our paradigm and stimuli were piloted in a group of age-matched non-aphasic control subjects, to make sure they elicited priming effects. Consequently, we used

the same paradigm and materials to assess syntactic priming in our patients with parallel vs. differential aphasia.

Participants

The *control group* consisted of 19 Dutch-French bilinguals who were matched with our patient group on age, sex, education, and self-rated proficiency in Dutch and French (*table 2*).

Table 2

Demographic data of the participant groups; L1 and L2 proficiency was rated on a five point Likert scale, for speaking, reading and writing.

	Control group (n=19)	**Patient group (n=6)**	**Group difference**
Age (years)	55.58 (12.38)	59.17 (16.70)	$t_{(23)} = .570$, $p > .574$
Sex (m/f)	4/15	1/5	$\chi^2_{(1)} = .055$, $p > .815$
Education (years)	15.11 (2.49)	14.17 (3.13)	$t_{(23)} = -.758$, $p > .456$
L1 proficiency (pre-onset)	5.00 (0.0)	5.00 (0.0)	Ns.
L2 proficiency (pre-onset)	3.81 (0.76)	3.72 (0.53)	$t_{(23)} = -.243$, $p > .81$

The patient group consisted of six bilingual aphasic patients. All the patients were referred to us by the neurology department of Ghent University Hospital. We used following inclusion criteria: (1) having a very good knowledge of French and Dutch before the onset of aphasia (as assessed by a

language questionnaire[3]); (2) being diagnosed with aphasia based on the Aachen Aphasia Test (AAT); and (3) having relatively good remaining comprehension (also based on AAT scores and on the assessment by the speech and language therapist). Patients suffering from a developmental or neurodegenerative disease, from an infection or tumour, or from a serious cognitive or depressive illness were excluded from the study. None of the patients had had a stroke previous to the one causing the current aphasic symptoms. The vision of all the patients was normal or corrected to normal (see also *table 3* and *table 4*). All patients were early, balanced bilinguals. L1 and L2 were determined based on Age of Acquisition. In the group of patients with differential aphasia, L1 was consistently the best-preserved language.

[3] This questionnaire was filled out based on both the answers of the patients and their closest family member(s), present in the hospital on the day of testing.

Table 3

Demographic data of the patients. All patients are early, balanced bilinguals; L1 and L2 are based on Age of Acquisition; MCA=Middle Cerebral Artery.

Subject	**Age**	**Sex**	**Bilingualism (L1-L2)**	**(previous) profession**	**Aetiology**
Patients with differential aphasia					
N.D.	41	F	Dutch-French	Geriatric nurse	Ischemia in left MCA area
H.D.M.	77	M	French-Dutch	Technical Engineer	Hemorrhage in left thalamus
D.J.	53	F	Dutch-French	Lawyer	Ischemia in left posterior MCA area
Patients with parallel aphasia					
J.C.	80	F	Dutch-French	Housewife	Ischemia in left posterior MCA area
K.H.	45	F	Dutch-French	Secretary	Hemorrhage in left frontal area
I.T.	59	F	Dutch-French	Secretary	Hemorrhage in left parieto-temporal

Table 4

Patient scores and percentiles on Aachen Aphasia Test (AAT) in Dutch and French, difference in Naming scores, aphasia pattern and scores on the Bilingual Aphasia Test (BAT) Part C in both directions.

	AAT					**BAT – part C**	
Subject	Subtest (max. score)	L1 (%ile)	L2 (%ile)	**Naming**	**Aphasia Pattern**	L1-L2 (/5)	L2-L1 (/5)
N.D.	SS (30)	27	15	L1 > L2	Wernicke aphasia	4	2
	TT (50)	7 (90)	31 (53)*		*Differential aphasia* (Dutch better preserved than French)		
	RE (150)	108 (52)	101 (44)				
	WL (90)	80 (83)	25 (27)*				
	NA (120)	**113 (88)**	**58 (36)***				
	LC (120)	98 (77)	84 (50)				
H.D. M.	SS (30)	26	19	L1 > L2	Amnestic aphasia	5	4
	TT (50)	6 (91)	19 (71)*		*Differential aphasia* (French better preserved than Dutch)		
	RE (150)	146 (96)	143 (92)				
	WL (90)	66 (65)	69 (68)				
	NA (120)	**113 (88)**	**90 (67)***				
	LC (120)	100 (80)	94 (70)				
D.J.	SS (30)	22	14	L1 > L2	Broca aphasia	5	5
	TT (50)	29 (57)	34 (48)		*Differential aphasia*		
	RE (150)	138 (84)	124 (69)				

	WL (90) **NA (120)** LC (120)	57 (56) **111 (97)** 118 (99)	49 (49) **82 (55)*** 107 (91)		(Dutch better preserved than French)		
J.C.	SS (30) TT (50) RE (150) WL (90) **NA (120)** LC (120)	25 7 (90) 126 (72) 90 (100) **110 (96)** 96 (73)	24 6 (91) 139 (85) 82 (87) **102 (84)** 100 (80)	Ns.	Amnestic aphasia *Parallel aphasia*	5	5
K.H.	SS (30) TT (50) RE (150) WL (90) **NA (120)** LC (120)	21 11 (84) 148 (98) 90 (100) **113 (98)** 102 (83)	22 15 (77) 146 (96) 88 (97) **113 (98)** 95 (71)	Ns.	Amnestic aphasia *Parallel aphasia*	5	5
I.T.	SS (30) TT (50) RE (150) WL (90) **NA (120)** LC (120)	18 50 (6) 0 (1) 6 (12) **30 (23)** 49 (8)	13 50 (6) 84 (31) 47 (47) **24 (21)** 13 (1)	Ns.	Wernicke aphasia *Parallel aphasia*	5	5

SS = Spontaneous Speech, TT = Token Test, RE = Repetition, WL = Written Language, NA = Naming, LC = Language Comprehension (auditory and reading), (*) significant difference between the scores (p<.05). The

assignment to the parallel or differential aphasia group was based on whether or not the patient showed a significant difference between the two languages in Naming score (in bold). Caveat: For the subtest "Token Test", the score reflects the amount of errors (so a higher score implies a lower performance); BAT-part C assesses recognition of translation equivalents.

Materials

We assessed the proficiency of the patient in speaking, writing and reading in Dutch and French before the onset of aphasia on a five point Likert scale, the context in which they used both languages, and the frequency of use (days/week), in both the patient and the close family member(s). To this end, we used a comprehensive, self-developed questionnaire.

To test the language strengths and deficits we used the Aachen Aphasia Test in Dutch (Graetz, De Bleser, & Willmes, 1992) and a self-developed French parallel version. The patients were assigned to the parallel or differential aphasia group based on the comparison of the scores on the subtest 'Naming' in Dutch and French. If these scores did not differ significantly on a paired t-test (determined by AAT software), the patient was assigned to the parallel aphasia group; if they did differ, the patient was assigned to the differential aphasia group. We opted to base the group assignment on the Naming subtest, because naming proficiency is highly relevant for the experimental task (i.e., describing pictures with a sentence). In addition we administered Part C of the Bilingual Aphasia Test (BAT) for Dutch and French (Paradis & Libben, 1987). This test assesses recognition of translation equivalents.

Before running the experiment, we administered a baseline task to measure how frequently the participants produced active and passive sentences without priming. The baseline task included twenty pictures that the participants were asked to describe without hearing a prime sentence. Ten pictures were supposed to be described in Dutch, ten pictures in French. For every language, eight pictures showed a transitive action, the other two pictures were filler sentences showing an intransitive action.

The syntactic priming task was a dialogue experiment. The *target stimuli* were 296 different pictures. We used pictures from the stimulus set of Bernolet et al. (2009) and from the different language versions of the BAT (of course we excluded the stimuli of the Dutch and the French version of the BAT). We included 132 critical trials, separated by either one or two filler trials. The target pictures depicted an action with a transitive verb (eliciting an active or a passive sentence), and the words needed to describe the picture (agent, patient and infinitive of the verb). We added these words to prevent that word finding difficulties interfered with the focus of this study, accessing syntactic representations (see also Hartsuiker & Kolk, 1998). The words were presented at the right side of the picture so that participants would be more likely to look at the picture first[4]. The order of the words varied across pictures. In addition, all pictures were mirrored relative to the vertical axis, because it was found that the spatial position of the agent might influence the preference for an active or a passive structure (Hartsuiker & Kolk, 1998). Both the original and the mirrored pictures were randomly used. When the patient showed inability to read the words, the experimenter read them aloud. The prime and the target sentence never contained the same verb, to avoid a lexical boost (*figure 2a*).

Since it was shown that animacy of the agent (i.e. the instigator of the action) and patient (i.e. the person (or animal) undergoing the action) might influence syntactic priming (Arai, Van Gompel, & Scheepers, 2007), our stimuli were controlled for this variable. We included equal amounts of three types of pictures: animate-animate (AA – animate agent and patient) pictures, inanimate-animate (IA – inanimate agent and animate patient) pictures, and inanimate-inanimate (II-inanimate agent and patient) pictures. We included 44 trials of each type (11 in each Language condition). In addition to the target pictures, we included 41 *filler pictures* in each language condition, showing an action with an intransitive verb (*figure 2b*).

[4] This was not formally assessed, however the participants quickly understood the importance of firstly looking to the pictures, and then to the words. By only looking at the words, the probability for erroneous sentences was high, because both nouns could act as an agent or a patient.

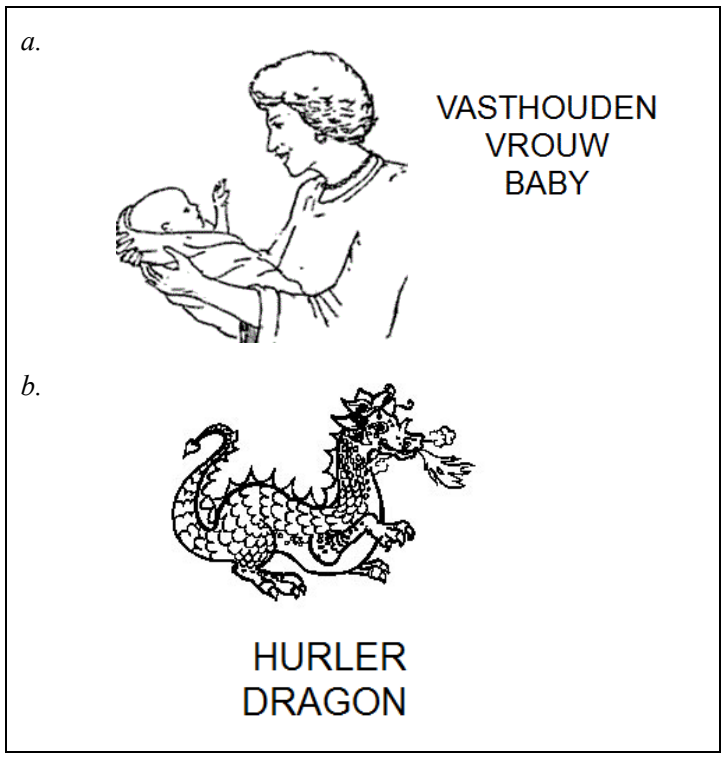

Figure 2. Example of (a) a target picture (with the words "to hold", "woman" and "baby") and (b) a filler picture (with the words "to roar" and "dragon")

Procedure

The study was approved by the Ghent University Hospital Ethics Committee. Before administering the tests, the patients and their closest family member were asked for their permission to be included in the study. Written informed consent was obtained in both the patient and the control group. In the patient group, we first administered the AAT, the BAT and the language questionnaire. The control group filled in the language questionnaire as well. Subsequently we administered the baseline task and the syntactic priming experiment.

The experiment was set up as a dialogue experiment. It was programmed in Eprime (version 2.1). During the experiment, one

experimenter acted as the confederate (i.e. pretending to take part in the experiment as a participant), the other sat next to the participant to guide him through the experiment. The confederate and the participant each sat in front of a computer screen, and the participant was told that they would be describing pictures to each other. To avoid unnecessary complexity, we blocked language trials (L1-L1, L1-L2, L2-L1, L2-L2). Before each language block, the participant was told in which language he/she was supposed to produce a sentence. The sequence of a trial was as follows (see *figure 3*): (a) the confederate read the prime sentence; (b) the participant saw two pictures (one with a blue background, one with a red background), and had to indicate which picture fitted the sentence he just heard by pressing the corresponding button (i.e., *the verification task*); (c) the participant saw a picture he had to describe using the words next to the picture; (d) the confederate coded the target sentence. The confederate coded[5] the target sentences produced by the participant as *active* (sentences with an active surface structure, including when the verb form was morphologically incorrect), *passive* (sentences with a passive surface structure, i.e., having an auxiliary and a past participle, in which the patient takes the function of sentence subject and the agent is expressed as an oblique object), or *other*. In step (c), participants were instructed to firstly look at the picture, and then to the words. We included the verification task (steps a and b) for two reasons. First, we wanted to make sure the participants listened carefully to and comprehended the prime; secondly, the accuracy in the verification task could function as a measure of language comprehension.

[5] All experiment runs were taped and listened to for a second time in case of uncertainty.

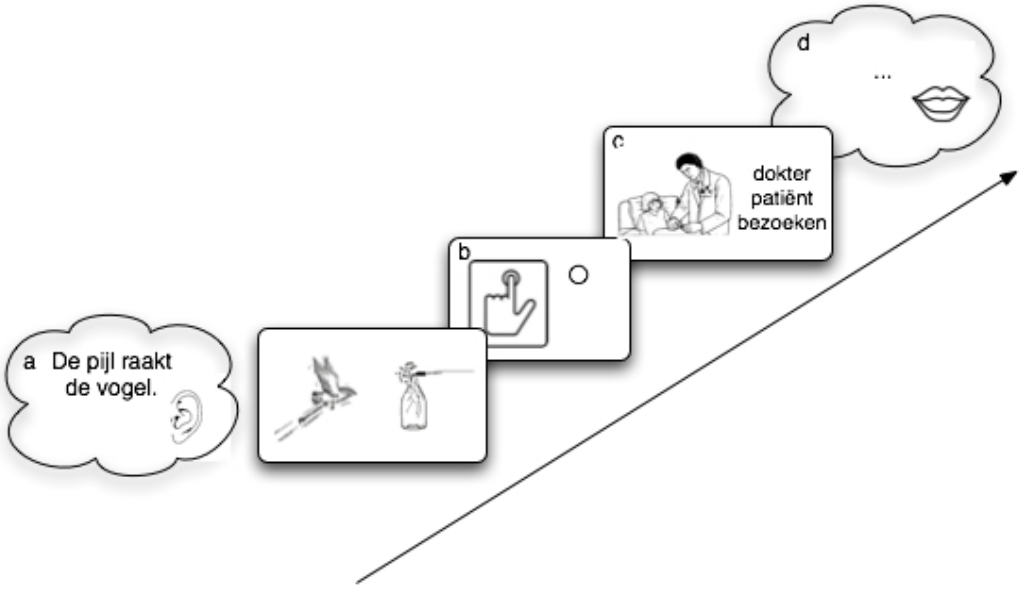

Figure 3. Sequence of a trial as seen by the participant in the dialogue experiment; (a) participants hear a sentence and see two pictures; (b) participants indicate which picture matches the sentence they just heard (= **verification task**); (c) participants see a picture to describe; (d) participants describe the pictures.

Design

The independent variables were *Syntactic structure* of the prime sentence (active vs. passive), *Prime Language* (L1/L2), and *Target Language* (L1/L2). This resulted in four language conditions (L1-L1, L1-L2, L2-L1, L2-L2). The language conditions were administered in blocks, and the order of these language blocks was counterbalanced. The dependent variable was the structure used to describe the target sentence[6].

[6] Analyses with agent and patient agency as an additional factor yielded similar results with respect to the crucial findings described below. Because of the design complexity, the factor is therefore not included in the analyses in the main text.

RESULTS

Control group

Pre-experimental baseline

Baseline results show a low frequency of passive target descriptions. In control subjects, on average 5.3% (SD = 0.08) of the pictures in the Dutch subset were described with a passive sentence, whereas on average 3.3% (SD = 0.09) of the pictures were described with a passive sentence in French. A Chi-square test shows that these average proportions do not differ significantly ($\chi^2_{(3)} = 4.57$, $p > 0.20$).

Verification task

The accuracy on the verification task is very high. The control subjects responded inaccurately in only 1.9% of the trials.

Priming experiment

We used the Lme4 package in R (Version 2.12.2; CRAN project; The R foundation for Statistical Computing, 2009). *Table 5* and *figure 4* show the priming effects in the control subjects. These were calculated as the difference between (amount of passive targets following a passive prime) minus (amount of passive targets following an active prime) (Pickering & Branigan, 1998). Trials in which participants did not use a correct active or passive sentence were excluded from the analysis (3.1% of the trials). We ran a binary logistic regression with one random factor[7] (mixed logit model, see Jaeger (2008). The fixed-effect variables were *Prime* (active/passive), *Prime Language* (L1/L2*),* and *Target Language* (L1/L2). We included the two-way and three-way interactions between these variables. Finally, we included a random intercept for *Subject* (other random effects did not

[7] We first selected a structure for the random effects to then add the fixed effects. Finally the model was reduced by removing non-significant fixed effects and the model diagnostics were assessed.

significantly improve the log-likelihood of the models). The dependent variable was *Target structure*. A significant main effect of *Prime* was found, which is the priming effect (i.e., more passive target sentences after a passive prime compared to an active prime) ($\chi^2(1) = 24.94$, $p < .000$). We also found a significant effect of *Prime Language* ($\chi^2(1) = 4.52$, $p < .034$), and a marginally significant interaction between *Prime* and *Prime Language* ($\chi^2(1) = 3.72$, $p < .054$). Further analyses show that the estimated β is 0.34 with an L1 prime ($p < .042$), whereas β is 0.80 with an L2 prime ($p < .00$). This might suggest that priming effects are somewhat larger with L2-primes, however this effect seems mainly caused by the L2-L2 vs. L1-L2 comparison.

Table 5

Priming effects in the control subjects

		Control Group *(N = 19)*		
Condition	**Prime**	**Active targets (%)**	**Passive targets (%)**	**Priming effect (%)**
L1 – L1	A	90.0	10.0	9.1
	P	80.9	19.1	
L2 – L2	A	78.0	22.0	11.2
	P	66.8	33.2	
L1 – L2	A	81.8	18.2	5.5
	P	76.3	23.7	
L2 – L1	A	82.4	17.7	7.1
	P	75.2	24.8	

"A" = Active prime, "P" = Passive prime; Priming effects are calculated as the difference between (amount of passive targets following a passive prime) minus (amount of passive targets following an active prime) (Pickering & Branigan, 1998).

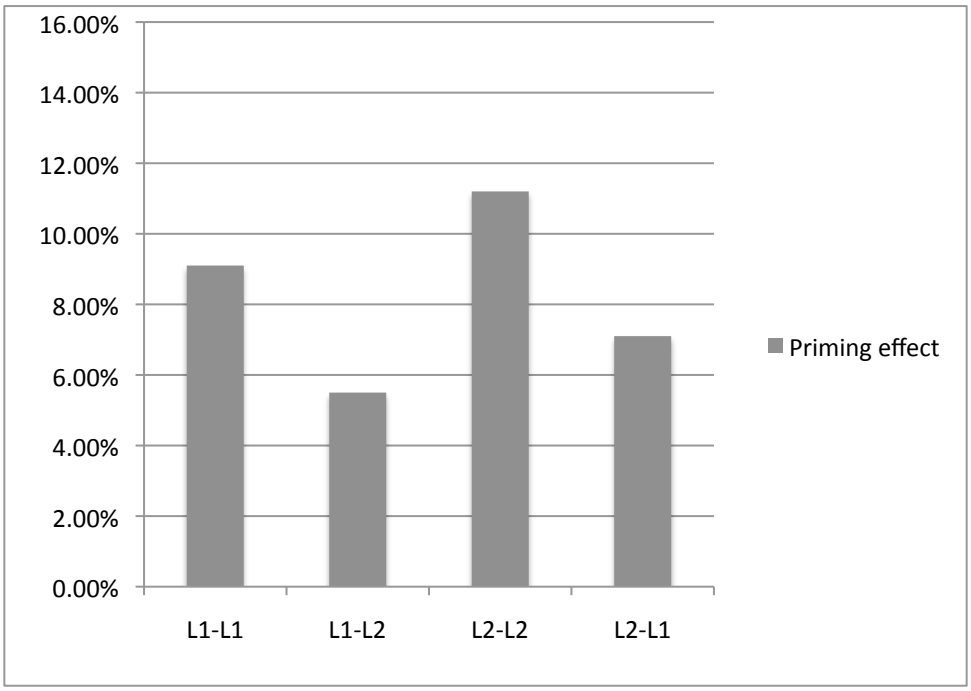

Figure 4. The size of the priming effect in the four language conditions for the control subjects. The priming effects are calculated as the difference between (amount of passive targets following a passive prime) minus (amount of passive targets following an active prime) (Pickering & Branigan, 1998).

Patient groups

Pre-experimental baseline

In the patient groups, on average 10.4% (SD = 0.12) of the target pictures were described with a passive structure in Dutch, in French, this was only the case for on average 4.2% (SD = 0.10) of the target pictures. A Chi-square test shows that the proportion of passive target sentences does not differ significantly between Dutch and French ($\chi^2_{(2,6)} = 1.83$, $p > 0.40$), nor between patients with parallel and differential aphasia ($\chi^2_{(2,6)} = 1.33$, $p < 0.51$). The baseline proportion of passive target descriptions does not differ either between patients or control subjects ($\chi^2_{(36,25)} = 6.90$, $p > 0.08$).

Verification task

The group of patients with parallel aphasia has a mean error percentage of 1.9% (0.5% after L1-primes, 1.4% after L2-primes), patients with differential aphasia 3.3% (1.1% after L1-primes, 2.2% after L2-primes). To analyse the error data we ran a binary logistic regression with one random factor (mixed logit model). The fixed effects variables were *Aphasia type* (parallel/differential aphasia), *Prime* (active/passive), and *Prime Language* (L1/L2). All two-way and three-way interactions were included, as was a random intercept for Subject (other random effects did not significantly improve the log-likelihood of the models). The dependent variable was Accuracy.

The effect of *Aphasia Type* is not significant ($p > 0.1$), suggesting that patients with parallel and differential aphasia do not differ in the amount of errors on the verification task. We find a significant effect of *Prime Language* ($\chi^2_{(1,6)} = 5.49$, ß = -1.51, $p < 0.02$), showing that more errors were made with L2-primes (3.6%) compared to L1-primes (1.6%). No other effects reach significance ($p > 0.08$).

Priming experiment

We ran a fully specified binary logistic regression with one random factor (mixed logit model). The fixed effects variables were *Prime* (active/passive), *Prime Language* (L1/L2), and *Target Language* (L1/L2). We also included a random intercept for *Subject*. The dependent variable was *Target structure*.

We find a significant main effect of *Prime*, implying a syntactic priming effect (more passives after a passive prime than after an active prime; $\chi^2_{(1,3)} = 6.49$, $p < 0.01$). We also find a significant main effect of *Target Language* ($\chi^2_{(1,3)} = 8.40$, $p < 0.004$), meaning that more passives were produced with L2 targets. The interaction between *Prime* and *Target Language* is not significant ($\chi^2_{(1,3)} = 0.1439$, $p > 0.7$) (*figure 6*).

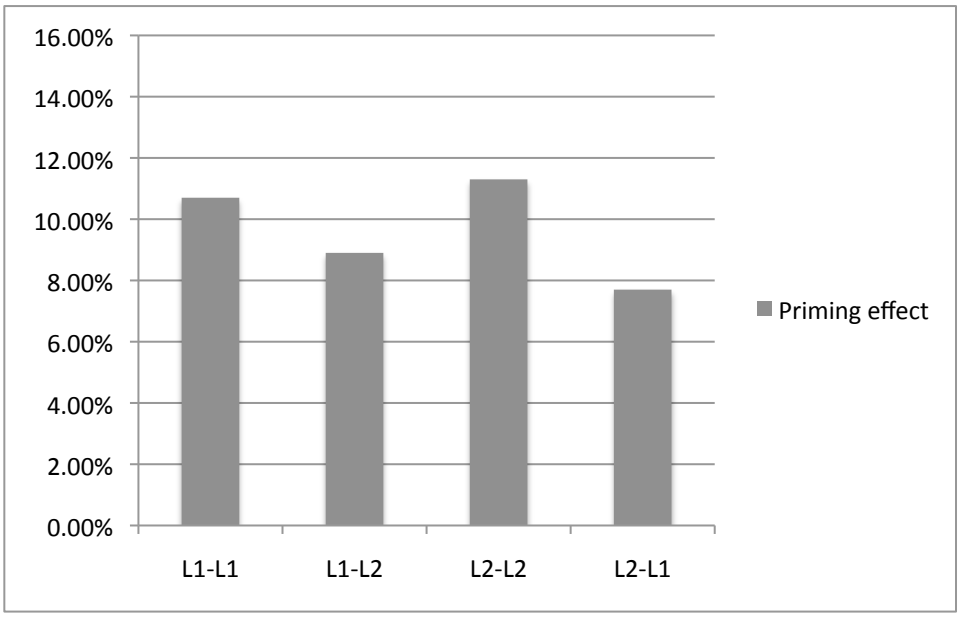

Figure 6. Size of the syntactic priming effect in the group of patients. The indicated values reflect the averages across type of aphasia (patients with parallel and patients with differential aphasia). The priming effects are calculated as the difference between (amount of passive targets following a passive prime) minus (amount of passive targets following an active prime) (Pickering & Branigan, 1998).

Because both our patient groups are very small, we were not able to statistically compare the magnitude of the priming effects in the different language-conditions between the two patient groups. As an alternative we discuss the numerical differences in the magnitude of the priming effects, as if it were a multiple case study.

Table 6 and *figure 5* show that both patient groups show considerable priming effects in each language condition, which are comparable with the priming effects observed in the control group. To be able to compare the priming effects in the patient groups with the control group statistically, we calculated the 95% confidence interval of the

parameter of the factor *Prime* in the control group. The estimated parameter of *Prime* is 0.58 (SE = 0.12), and the confidence interval is [0.34, 0.82]. For the patients with parallel aphasia, the estimated parameter for *Prime* is 0.35 (SE = 0.27) with a confidence interval of [-0.11, 0.81]. For the patients with differential aphasia, the parameter for Prime is 0.69 (SE = 0.3), with a confidence interval of [0.18, 1.20]. This suggests that both patient groups show approximately equally large priming effects across language-conditions compared to the control group.

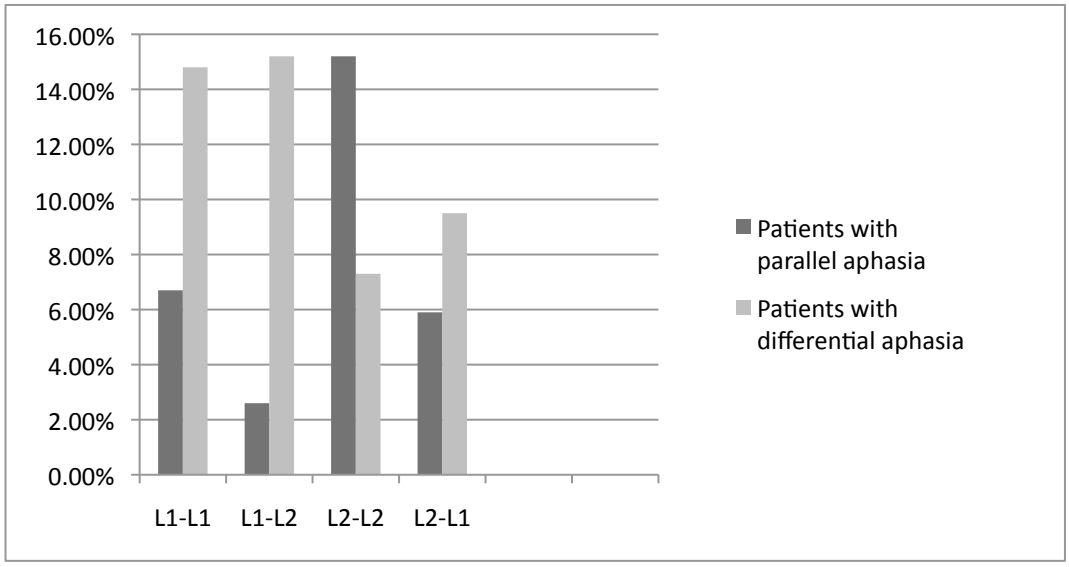

Figure 5. Size of the syntactic priming effect in the group of patients with parallel and differential aphasia. The priming effects are calculated as the difference between (amount of passive targets following a passive prime) minus (amount of passive targets following an active prime) (Pickering & Branigan, 1998).

Table 6

Results of the priming experiment in the patient groups

		Patients with parallel aphasia *N=3*			**Patients with differential aphasia** *N=3*		
Condi -tion	**Prime**	**Active targets (%)**	**Passive targets (%)**	**Priming effect (%)**	**Active targets (%)**	**Passive targets (%)**	**Priming effect (%)**
L1 – L1	A	85.7	14.4	6.7	84.2	15.8	14.8
	P	79.0	21.1		69.4	30.6	
L2 – L2	A	79.5	20.5	15.2	66.7	33.3	7.3
	P	64.3	35.7		59.4	40.6	
L1 – L2	A	66.7	33.3	2.6	82.9	17.1	15.2
	P	64.1	35.9		67.7	32.3	
L2 – L1	A	85.4	14.6	5.9	82.5	17.5	9.5
	P	79.5	20.5		73.0	27.0	

"A" = Active prime, "P" = Passive prime. Priming effects are calculated as the difference between (amount of passive targets following a passive prime) minus (amount of passive targets following an active prime) (Pickering & Branigan, 1998).

The patients with parallel aphasia show more priming with L2-primes (10.6%) compared to L1 primes (4.7%). This pattern[8] was also observed in the control subjects (9.2% with L2 primes vs. 7.3% with L1 primes). Interestingly, the patients with differential aphasia show the opposite effect, with larger priming effects from the best-preserved language (8.4% with L2 primes vs. 15% with L1 primes). When comparing between-language priming (the average effect in the L1-L2 and L2-L1 condition) and within-language priming (the average effect in the L1-L1 and L2-L2 condition), patients with parallel aphasia show more priming when the prime language and the target language are the same (within-language priming, 11%) compared to between-language priming (4.3%). This pattern is again similar in the control group (10.2% within-language priming vs. 6.3% between-language priming). Interestingly, the patients with differential aphasia show almost equal priming effects within languages (11%) and between languages (12.4%).

DISCUSSION

The aim of the current study was to investigate syntactic priming in bilingual aphasia. To address this aim, we asked the following research questions: first, do patients with bilingual aphasia show syntactic priming within and between languages? Secondly, do these priming effects differ across aphasia patterns? We included two types of aphasia patients: to investigate whether relative language loss influences such cross-lingual syntactic interactions, we contrasted patients with parallel aphasia (i.e., having similar impairments in both languages) and patients with differential aphasia (i.e., the impairments in one language are more severe than in the other language). Our third research question was whether these priming effects differ from the priming effects observed in control participants. Therefore we compared the priming effects of the patients with the effects of a group of matched control participants.

[8] We opted not to speculate about differences in the size of the effects, because of the differences in group size, which make it difficult to compare the size and the strength of the priming effects between the patient groups and the control group.

We administered a dialogue experiment in four language conditions: two within-language conditions (L1-L1 and L2-L2) and two between-language conditions (L1-L2, L2-L1). Our results show that patients with bilingual aphasia did show a robust, statistically significant syntactic priming effect: they produced more passive sentences after hearing a passive prime than after hearing an active prime, both within and across languages. This is the first demonstration of cross-lingual syntactic priming in bilingual aphasic patients. Both control subjects and aphasic patients show considerable priming effects in all four language conditions, but some interesting differences across groups also emerged. Within-language priming was stronger than between-language priming for both control participants and patients with parallel aphasia, whereas patients with differential aphasia showed equally strong cross-lingual as intralingual priming. Control participants and patients with parallel aphasia showed stronger priming effects from L2 primes, whereas patients with differential aphasia showed stronger priming effects from the first acquired (and also best-preserved) language (L1). So, patients with parallel aphasia behaved much more similarly to controls than patients with differential aphasia.

Finding syntactic priming effects in patients with bilingual aphasia replicates two previous findings of syntactic priming in aphasic patients (Hartsuiker & Kolk, 1998; Saffran & Martin, 1997). However, Hartsuiker and Kolk (1998) did find larger priming effects in the patient group than in control subjects, which we did not observe. A possible explanation might be that Hartsuiker and Kolk only tested patients with Broca's aphasia, who showed severe syntactic deficits, whereas our patients were diagnosed with different types of aphasia and had relatively smaller syntactic deficits[9]. It is plausible that patients with strong grammatical/syntactic impairment benefit more from syntactic activation triggered by prime sentences when producing sentences. The lack of severe syntactic deficits in our patients might explain why they did not show a larger tendency than the control subjects to rely on the previous structure. This is consistent with our observation that the cross-lingual priming effect for patients with differential aphasia was larger when

[9] It may also be the case that the current study did not have adequate power to detect significant differences. It would definitely be interesting to replicate these findings in larger groups of participants.

producing sentences in the most affected language (15.2%) than the cross-lingual effect for sentence production in the most preserved (but still affected) language (9.5%). Another important difference with Hartsuiker and Kolk, is that they did not find priming effects in the control group. We did observe priming effects in our control subjects, which is in line with previous studies showing cross-lingual priming effects for transitive sentences in control subjects.

Although all groups showed syntactic priming in all language-conditions, one of the most interesting findings in this study is that differential aphasia patients also showed strong syntactic priming effects with L2 primes, even though this is the most affected language. The overall 8.4% priming effect with L2 primes is comparable with the L2 priming effect of patients with parallel aphasia (10.6%) and control subjects (9.2%). Only looking at L2-L1 cross-lingual priming, differential patients even showed stronger priming effects (9.5%) than parallel (5.9%) patients and control (7.1%) participants. This suggests that the most impaired language is not "lost"; syntactic representations in themselves are intact and still able to influence syntactic processing in the other language, if language control demands are low. In the syntactic priming paradigm, the prime in the most affected language (L2) only has to be comprehended, not produced (so that the dominant language does not necessarily need to be inhibited, as is the case in production), so that activation in the syntactic representations triggered by comprehension is strong enough to influence subsequent production in the best-preserved language. However, it remains unclear why the L2-L1 priming effect is larger for the group of patients with differential aphasia compared to the group of patients with parallel aphasia.

In patients with parallel aphasia and control participants, we found that L2 primes (9.9%)[10] elicited larger priming effects compared to L1 primes (6%)[10]. This might be explained by a complexity effect, in which syntactically more complex sentences generalize to syntactically less complex sentences, but the reverse does not occur (Thompson, Shapiro, Kiran, & Sobecks, 2003). One might argue that this complexity effect could

[10] These percentages reflect average priming effects of patients with parallel aphasia and control subjects across language conditions.

also be reflected in more generalization from a less frequent language (L2) to a more frequent language (L1), than vice versa. However, it remains unclear why this effect could not be found in patients with differential aphasia. Replication with larger groups of patients is needed to confirm this pattern of results, and to further elaborate this effect in patients with differential aphasia.

Because the priming effects of parallel aphasia patients were more similar to the effects found in control subjects than the effects of differential aphasia patients, and because the latter still showed strong L2-L1 priming, this provides evidence for a non-localized account of differential language loss, e.g. in terms of language control (see above). A possible network underlying language control was recently described by Abutalebi and Green (2007). The network consists of the prefrontal cortex, the anterior cingulate cortex, the inferior parietal cortex and the basal ganglia. Damage to these components might lead to the control deficits underlying bilingual aphasia. This view is consistent with Pitres' account, which already suggested that differential or selective aphasia may not be due to loss of the language representations themselves, but rather to a problem in controlling languages. To further disentangle the role of each component of this control network in differential bilingual aphasia, additional (imaging) research is needed.

The results are in line with other studies demonstrating cross-lingual syntactic priming (Bernolet et al., 2007; Hartsuiker et al., 2004; Kantola & Van Gompel, 2011; Loebell & Bock, 2003; Schoonbaert et al., 2007; Shin & Christianson, 2009) showing that people tend to re-use syntactic structures, even across languages (see Introduction). In addition, they confirm the predictions based on the bilingual syntactic priming model of Hartsuiker et al. (Hartsuiker et al., 2004), discussed in the introduction, and provide further evidence for shared syntactic representations across languages. In this model, both within- and across-language priming effects are explained in terms of residual activation in syntactic representations after comprehension of the prime. Because the model assumes shared syntactic representations across languages, it predicts cross-lingual priming effects as long as these syntactic representations are intact. As such, this model is compatible with accounts that explain differential aphasia in terms of language control. Important to notice however, is that the model of

Hartsuiker et al. did predict similar priming effect sizes in between- and within-language priming, which is not completely in line with what we found here. Yet, Cai and colleagues recently contested this prediction by assuming that not only the combinatorial node remains activated, but the language node as well, inhibiting other language nodes (Cai, Pickering, Yan, & Branigan, 2011). This would suggest larger within-language than between-language priming effects (as was found in our patients with parallel aphasia and in our control subjects, but not in our group of patients with differential recovery). Further research will be needed to apply these models to syntactic priming effects in patients with bilingual aphasia.

The finding of cross-lingual priming effects is also interesting from a therapeutic perspective, because it implies that training in one language might also be beneficial for the other language. This is in line with previous studies showing that language therapy in one language might generalise to another (untrained) language (Edmonds & Kiran, 2006; Filiputti, Tavano, Vorano, De Luca, & Fabbro, 2002; Kiran & Edmonds, 2004; Marangolo et al., 2009; Miertsch, Meisel, & Isel, 2009; for a conflicting view, see Abutalebi, Rosa, Tettamanti, Green, & Cappa, 2009; Galvez & Hinckley, 2003; Meinzer et al., 2007). Given the current findings, agrammatic symptoms in bilingual patients may benefit from training in, and transfer from, other languages than the dominant language, both for parallel and differential aphasia patients. An interesting finding concerning therapy effects is that aphasia patients seem to show longer lasting priming effects in certain conditions: patients with parallel aphasia produce a passive sentence after an active prime in 33% of the trials of the L1-L2 condition, and patients with differential aphasia produce a passive sentence after an active priming in 33% of the trials of the L2-L2 condition. This never occurred in the control group. This suggests that priming lasts longer in the aphasic group, that is, the passive construction is not inhibited in an active condition, but it is still triggered. In addition, this only seems to occur when the target language is L2, and most often in aphasia patients. Further research is needed to identify the conditions under which training effects last longer and generalize across languages. In addition, the requirements of language therapy should be further investigated. An interesting path for future research would be to sort out why aphasia patients do not benefit from

hearing sentences in daily life, and why they do benefit from language therapy. It might be the case that they do benefit from hearing sentences in daily life, but this is less visible than after therapy. Also, linguistic input for patients with aphasia might be less grammatically diverse, because people take into account possible comprehension problems.

To summarise, this is the first demonstration of cross-lingual syntactic priming in a group of patients with bilingual aphasia. The pattern of the effects was comparable to the pattern observed in a group of matched control subjects. Moreover, patients with differential aphasia also showed cross-lingual priming from the most affected language to the best-preserved language. This shows that the least recovered language can still influence syntactic processing in the other language. Our results are largely in line with the model proposed by Hartsuiker et al. (2004), and support a control-based account of different bilingual aphasia patterns. Nevertheless, these results were obtained in a small group of patients, so further research with more patients is needed to confirm our findings. In addition, the current findings should be replicated with other syntactic structures and language combinations.

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CHAPTER 6:
GENERAL DISCUSSION

Bilingualism is a widespread phenomenon that has received a lot of interest in psycholinguistic research during the last decades. It has been stated that "a bilingual is not just two monolinguals in one person" (Grosjean, 1989). More specifically, bilinguals differ from monolinguals in the fact that they have the possibility to make an utterance in two different languages. Hence, these two different languages need to be stored and controlled, which yields some very interesting questions about their representation, and the way in which (simultaneous) activation in both languages is managed.

The first important issue concerning bilingualism is the nature of the lexicon. Are the two languages of a bilingual stored in distinct lexicons, or are they rather stored in one common lexicon? As indicated in the introduction, this has been the most dominant question in psycholinguistic research about bilingualism during the last decades. By now, a consensus has grown that both languages of a bilingual are represented in at least very strongly interacting lexicons (e.g. Caramazza & Brones, 1979; Costa, Santesteban, & Cano, 2005; Duyck, Van Assche, Drieghe, & Hartsuiker, 2007; Schwartz, Kroll, & Diaz, 2007; Van Assche, Duyck, Hartsuiker, & Diependaele, 2009; van Hell & Dijkstra, 2002). In an attempt to conceptualise the lexicon(s) of a bilingual, older accounts such as that of Kroll and Stewart (1994) still proposed in their Revised Hierarchical model (RHM) that bilinguals have two separated yet connected lexicons. However, as was described in the General Introduction, current research now adopts a single, integrated lexicon, as was assumed in the Bilingual Interactive Activation model (BIA+, Dijkstra, Grainger, & van Heuven, 1999; Dijkstra & van Heuven, 2002). This model assumes different levels of representation, and language nodes connected to the words. It is a bilingual version of the classical interactive activation model, with words from both languages in one and the same integrated lexical system.

Consistent with behavioural and modelling work, neuroimaging research has shown very similar activation when performing tasks in different languages (e.g. Briellmann et al., 2004; Chee, Tan, & Theil, 1999; Hernandez, Martinez, & Kohnert, 2000; Illes et al., 1999; Kim, Relkin, Lee, & Hirsch, 1997 only in early bilinguals; Mahendra, Plante, Magloire, Milman, & Trouard, 2003; Pu et al., 2001; Vingerhoets et al., 2003). These two lines of research indicate that both languages of a bilingual are probably processed in the same brain regions, and word processing therefore yields activation in an integrated lexicon with language non-selective access.

As was explained in the General Introduction, two sorts of models have been proposed to account for the neural representation of both languages of a bilingual. The declarative/procedural model (Ullman, 2001a, 2001b) proposes that in monolinguals, words are represented in a declarative memory system, whereas grammatical rules would be stored in a procedural memory system. In bilinguals who begin to learn a second language, the representation of L2 words would also rely on the declarative system. Unlike L1, L2 grammar would be stored in the same declarative system, and not in the procedural system, as L1 grammatical rules. With increasing L2 proficiency, L2 grammar will gradually rely more on the procedural system.

On the other hand, Green and colleagues proposed a more simple assumption, using the BIA+ model as a starting point. They postulated that the acquisition of a second language would utilise existing devices. The processing of its lexical, grammatical/morphological properties and its prosody will rely on a network shared with L1 (Abutalebi & Green, 2007; Crinion et al., 2006; Green, 2003).

If indeed the two languages of a bilingual are highly interacting, a mechanism is needed to be able to speak, in which activation in representations of both languages is controlled, selecting the intended language and inhibiting the unintended language. Again, two possible views arise from the literature. First, the Inhibitory Control model (IC model, Green, 1998) assumed that language selection occurs through the inhibition of the unintended languages. With increasing proficiency, more cognitive resources would be needed to suppress the language. This control

mechanism would not only manage language control, but is assumed to be a general control mechanism.

Oppositely, the view of Costa and colleagues (Costa & Caramazza, 1999; Costa, Miozzo, & Caramazza, 1999) assumes that language selection is language specific. At least with high levels of language proficiency, it would be possible to only activate one language.

In what follows, we will discuss the studies of the current dissertation, and relate them to the previously described models and literature. In addition, we will discuss implications for therapy, address some limitations of the studies described in this dissertation, and provide some suggestions for further research.

BILINGUALISM AND THE DEVELOPMENT OF EXECUTIVE CONTROL

Recently, a lot of studies have reported that bilinguals outperform monolinguals on a range of control tasks, also on control tasks that tap into cognitive control outside the linguistic domain (e.g. Bialystok et al., 2005; Bialystok, Craik, Klein, & Viswanathan, 2004; Bialystok, Craik, & Ryan, 2006; Costa, Hernandez, & Sebastian-Gallès, 2008; Emmorey, Luk, Pyers, & Bialystok, 2009). It was hypothesised that the constant need for managing competition between lexical representations of both languages, thereby inhibiting (items in) the unintended language and selecting (items in) the intended language, underlies this bilingual executive advantage (Emmorey et al., 2009). Green (1998) suggested that the mechanism responsible for bilingual language control is not language specific, but rather part of a more general executive control mechanism, involved in both language related and language unrelated control tasks. Constantly exercising this general executive control mechanism leads to a boost in cognitive control in bilinguals, which is reflected in the enhanced performance on cognitive control tasks such as Simon or flanker task.

Importantly, the mere fact of mastering two or more languages to a certain degree does not seem to be sufficient to boost the cognitive control

system. For instance, Prior and Gollan (2011) were the first to investigate the role of language switching in the development of the cognitive control advantage (more specifically on task switching). They compared the cognitive control performance of a group of Spanish-English bilinguals who regularly switch between languages with the performance of a group of Mandarin-English bilinguals who switch between languages less often. They only found an advantage on task switching in the bilinguals who often switch languages. In **chapter two**, we also investigated the role of daily language switching in the development of the executive control system. We tested three groups of participants: unbalanced bilinguals, balanced bilinguals that do not often switch languages, and balanced bilinguals that frequently switch between languages. We compared the performance of these three groups on two tasks tapping into executive functions (more specifically inhibition), namely a flanker task and a Simon task.

Our study differed from the study of Prior and Gollan (2011) in two important aspects. The first difference is about the different executive functions that may be influenced by daily language switching. Whereas Prior and Gollan focused on task switching, we went a step further and studied whether language switching enhances inhibition as well. Secondly, and more importantly, our participants were all Dutch-French bilinguals, who only differed in L2 profiency and/or switching frequency. This is different from Prior and Gollan, who tested English-Spanish bilinguals in the frequently switching group, and English-Mandarin bilinguals in the less frequently switching group. Hence, switching was confounded with the similarity within the language pair. Because languages with a distinct script (English and Mandarin) require different representational structures (Gollan, Forster, & Frost, 1997) and hence also control demands, we hypothesised that the degree of overlap between the languages in the study by Prior and Gollan might have influenced the development of the control advantage. Therefore, we opted to include only Dutch-French bilinguals, who only differed from each other in L2 proficiency and/or the amount of language switching in daily life.

In both the Simon task and the flanker task, we found smaller conflict effects in the group of balanced bilinguals that often switch between languages, compared to both the unbalanced bilinguals and the balanced

bilinguals that do not often switch between languages. This suggests that executive control advantages are only present when the lexical representations of both languages are often simultaneously active and used or inhibited during frequent code switching, e.g. in bilinguals who often switch languages, within sentences or conversations. The frequent simultaneous activation between strong lexical representations of different languages causes competition and necessitates the bilinguals to engage their executive control mechanism to select representations in the target language, and inhibit the non-target language. This practice then transfers to non-linguistic cognitive control abilities, tapped into by the Simon and flanker tasks.

In addition, our results show that the bilingual advantage emerges from language switching experience, independent from language pair similarity, and also in tasks tapping into inhibitory executive functions. In this, our study supplements the study of Prior and Gollan (2011). These findings suggest that active and frequent code switching may play a crucial role in the development of the bilingual executive control advantage.

As was described in the General Introduction, the bilingual executive control advantage was recently challenged by a large study of Paap and Greenberg (2013), who compared large groups of monolinguals and bilinguals on a wide range of 15 executive processing tasks. Apart from one task, none of these tasks showed a bilingual control advantage. Based on these null effects, it might be assumed that the bilingual advantage does not necessarily emerge from bilingualism, but instead that certain language use characteristics may be crucial for the development of the control advantage. Our finding that the control advantage only appears in balanced bilinguals who often switch languages in daily life, within conversations or even within sentences, might explain why the advantage cannot consistently be found in studies with bilinguals. Theoretical implications of the current findings will be discussed in detail further in this General Discussion.

BILINGUAL APHASIA

When studying cognitive processes, a common neuropsychological approach is to investigate what happens when the cognitive function is lost. In the present dissertation, this approach was followed for (bilingual) language processing. We investigated what happens when the ability to produce or comprehend language is lost, or when a bilingual individual loses the capability to control his or her languages. This is exactly the case when patients suffer from aphasia. When a bilingual suffers from bilingual aphasia, both languages are not necessarily affected to the same extent, although this is the most common case. When the same impairments occur in both languages, this is called parallel aphasia (Paradis, 1977). When instead impairments are more pronounced in one language compared to the other, it is called differential aphasia, or selective aphasia, when only one language is affected. At first, it was suggested that differential and selective aphasia were caused by the selective damage to language specific brain areas. However, because psycholinguistic and neurolinguistic research has now indicated that both languages of a bilingual strongly interact with each other (e.g. Caramazza & Brones, 1979; Costa et al., 2005; Duyck et al., 2007; Schwartz et al., 2007; Van Assche et al., 2009; van Hell & Dijkstra, 2002), and also are represented in mainly the same/overlapping brain regions (e.g. Briellmann et al., 2004; Chee et al., 1999; Hernandez et al., 2000; Illes et al., 1999; Kim et al., 1997; Mahendra et al., 2003; Pu et al., 2001; Vingerhoets et al., 2003), these localised hypotheses have been put under pressure as an explanation for differential aphasia. With recent findings in the fields of bilingualism and language control, the focus has shifted towards a more dynamic account for the various impairment and recovery patterns in bilingual aphasia, suggesting that a deficit in (language) control might be underlying the asymmetric, functional "loss" of a language (Green & Price, 2001; Pitres, 1895). This has led to the hypothesis that differential and selective aphasia could be a reflection of a problem in language control (Green & Abutalebi, 2008). However, this interesting hypothesis for a puzzling neuropsychological case has never been empirically tested. That was the aim of the present dissertation.

If this hypothesis is correct, and not language specific brain area damage but rather a language control problem underlies selective and differential bilingual aphasia, we would expect patients with selective and differential aphasia to show depraved control performance. Additionally, we would expect them to still show effects of cross-lingual interaction, and even the most affect language should be able to influence the best-preserved language, which is quite a challenging prediction. These two hypotheses were investigated in chapter three to five, at different representational levels of language, going from lexical (word) processing to syntax.

CASE STUDY OF A PATIENT WITH DIFFERENTIAL APHASIA

In **chapter three**, we described the performance of a French-Dutch bilingual patient with differential aphasia, who showed larger impairments in Dutch (L2) compared to French (L1). Intuitively, it would be expected that language production requires much more language control compared to language comprehension, because a selection has to be made between the target words in the different languages, in order to produce just one. This is not the case in comprehension. However, by using different lexical decision tasks, we showed that also comprehension might yield different language control demands, and that aphasia patients are sensitive to the amount of control required by the (comprehension) task.

Cross-lingual interactions

First, we used a generalised lexical decision task, in which words in any language required a YES-response. Secondly, we used a selective lexical decision task, in which only words in one certain language (French OR Dutch) required a YES-response. We argued that the selective lexical decision task imposes much more cross-lingual competition compared to the generalised lexical decision task, because of the need to bind NO-responses to activation in representations of one (i.e. the non-target) language. Therefore, we expected that the generalised lexical decision task allows a purer assessment of cross-lingual interactions in the lexical word

identification system than the selective lexical decision task, which triggers control processes operating on this lexical activation. These cross-lingual interactions were assessed by inserting cognates in the stimulus list. Our predictions were confirmed: in the generalised lexical decision task, the patient showed a clear cognate effect when comparing the performance on cognates with the performance on both L1 and L2 non-cognates. This implies that the most affected language (Dutch) is still able to influence activation in the most preserved language (French), given that cognates were recognised better than matched, L1 (French) non-cognate controls. In the L1 selective lexical decision task, we did not find a difference between cognates and L1 non-cognates. In the L2 selective lexical decision task, cognates were recognised less accurately compared to L2 non-cognates. This differential pattern in the two versions of the selective LD task can again be explained by the language control demands in this task, which differ from those in the generalised lexical decision task. Because the patient's most affected language is Dutch, it might be plausible that it is harder for the patient to suppress his French lexicon than to suppress the Dutch. The amount of inhibition that can be generated might be sufficient to suppress the "weaker" L2 (Dutch), but might not suffice to inhibit the "stronger" L1 (French). This difference in the ability to suppress the lexicon might be reflected in the amount of cross-lingual interaction: the most affected language (Dutch) will be less likely to influence processing in the least affected language, than vice versa. However, also this most affected language has been shown to be able to influence language processing, despite this difference in ability to suppress the language.

The previously described case study was the first to investigate cross-lingual interactions in a patient with differential aphasia. We focused on the variety of lexical tasks, and attempted to attribute the patterns of results to a control deficit, rather than to the selective damage of a language specific brain area.

Cognitive control functions

To support this control deficit hypothesis, we also ran a flanker task. However, because only one differential aphasia patient was available at that time, our only option was to compare his pattern of results with the results of

healthy control subjects. This showed that cognitive control functioning was indeed impaired relative to controls, even though this is not a typical diagnostic marker of aphasia. This comparison supports the language control account for differential aphasia, but can never be conclusive because of the fact that (1) this was necessarily a case study, and (2) the brain damage in the stroke patient makes it difficult to attribute different task performance from healthy controls specifically to cognitive control, independent from other brain functioning differences that may exist because of the trauma. A comparison between different types of aphasia patients was warranted here, specifically linking aphasia type to cognitive control performance.

COGNATE EFFECTS AND COGNITIVE CONTROL IN PATIENTS WITH PARALLEL AND DIFFERENTIAL APHASIA

Therefore, in **chapter four**, we studied similar hypotheses, but now in a larger group of patients. Including both patients with parallel and patients with differential aphasia, and comparing them with a healthy control group, made it possible to compare the patterns of the patients with differential aphasia with patients who suffered a stroke as well, and who also developed aphasia. Eight patients with parallel aphasia and seven patients with differential aphasia were included. We aimed to investigate cross-lingual interaction at the one hand (in a similar way as in the previously described case study), but here we mainly emphasized the difference in control functions across the three groups (as will be described further in this discussion).

Cross-lingual interactions

To assess cross-lingual interactions, participants again performed a generalised lexical decision task with cognates. They showed statistically significant better performance for cognates compared to non-cognates (i.e. the cognate facilitation effect). Importantly, the size of this cognate effect did not differ significantly across groups, indicating that patients with differential aphasia showed an equal amount of cross-lingual interaction

compared to patients with parallel aphasia and control subjects. Again, this also supports the hypothesis that the representations of the languages of a bilingual aphasia patient are strongly interacting, even in aphasia patients. It also shows that the larger functional loss in one of the two languages is due to impaired lexical activation in the most affected language.

These findings are in line with previous research showing cognate effects in patients with bilingual aphasia. For instance, Robert and Deslauriers (1999) investigated the effect of cognate status in early, balanced French-English bilingual aphasic patients and healthy control subjects. They found that cognates were named faster and more accurately, in both languages, in both groups. Similarly, Detry and colleagues (Detry, Pillon, & de Partz, 2005) administered a picture-word verification task and a naming task with cognates and non-cognates in a French-English parallel aphasia patient with agrammatism and word-finding difficulties. In both tasks, the patient's performance was higher for the cognates compared to the non-cognates.

The two studies in this dissertation are the first to investigate cognate effects in patients with differential aphasia. Finding evidence for cross-lingual interaction in these patients suggests that both languages are still active, and able to influence each other, even though they might seem to be lost at the functional level. This finding argues against a strict localised account for differential aphasia, which suggests that the selective impairment of one language is due to the selective damage to the language specific area. Further in this General Discussion, we will extensively elaborate the theoretical implications of the current findings.

Cognitive control functions

In the fourth chapter of this dissertation, we also investigated the control deficit hypothesis by administering a flanker task in our patients with differential and parallel aphasia, and comparing their performance with that of a healthy control group. Both the patients with parallel aphasia and the control subjects performed almost at ceiling level on both congruent and incongruent trials, yielding no congruency effect. However, patients with differential aphasia made significantly more errors on incongruent trials

compared to congruent trials (although this effect was not very large). This confirms our prediction that patients with differential aphasia indeed show worse cognitive control relative to both controls and parallel aphasia patients. They experience more problems inhibiting irrelevant information (i.e. the incongruent flankers). This supports the control hypothesis proposed by Pitres (1895) and elaborated by Green and Abutalebi (2008), which proposes that a deficit in (language) control may underlie differential (and selective) aphasia in bilingual patients. Although the size of the congruency effect of the patients with differential aphasia differed significantly from that of the patients with parallel aphasia and healthy control subjects, the difference was not extremely large (the congruency effect was about 3.5% larger on accuracy, and about 12 ms larger on reaction times). This shows that even relatively subtle deficiencies in cognitive control may already affect the ability to control languages and have selective effects on the use of one of the languages. It is important to note here to that these patients did not have clear and pronounced neural damage in the frontal cortex. Instead, impaired functional cognitive control deficiency probably results from refined neural disturbances. In this view, it is also interesting that these patients were differential aphasia patients, and that clear (but apparently very rare) cases of really selective aphasia might show really larger cognitive control performance (perhaps noticeable in structural neuroimaging). Theoretical implications of the current findings will be more thoroughly explained further in this discussion.

SYNTACTIC PRIMING IN BILINGUAL APHASIA

In **chapter five**, we investigated cross-lingual interactions at the sentence level in patients with bilingual aphasia, studying the syntactic priming effect as a marker of cross-lingual interactions at the syntactical level. The syntactic priming effect is the phenomenon in which people tend to re-use a syntactic structure of a previously heard/produced sentence (Bock, 1986). Important for the current dissertation is that syntactic priming has also been found across languages. Hartsuiker, Pickering and Veltkamp (2004) found syntactic priming for transitive sentences in a dialogue experiment with Spanish-English bilinguals (See also Hartsuiker &

Pickering, 2008; Meijer & Tree, 2003; Shin & Christianson, 2009 for cross-lingual priming with datives). Cross-lingual syntactic priming has also been found in the opposite direction (L2-L1, Schoonbaert, Hartsuiker, & Pickering, 2007).

Only two studies have yet investigated (monolingual) syntactical priming in patients with aphasia (Hartsuiker & Kolk, 1998; Saffran & Martin, 1997). This is regrettable, given the fact that aphasia is often not only characterized by dysfunctions at the lexical level, but also at the grammatical level. For example, patients with aphasia seem to suffer from difficulties understanding and producing passive sentences (Caplan, Waters, Dede, Michaud, & Reddy, 2007; Grodzinsky, 2000; Wassenaar & Hagoort, 2007).

In both the studies of Hartsuiker and Kolk (1998) and Saffran and Martin (1997) significant syntactic priming effects in aphasia patients were found, implying that grammatical representations in themselves are not 'lost', but instead harder to functionally access. Both studies showed that aphasic patients used hardly any passive sentences spontaneously, whereas they did produce passives during and after the experiment. This suggests a positive influence of syntactic priming on the quality of language (sentence) production.

In chapter five, we studied cross-lingual syntactic priming in patients with parallel and differential aphasia. We included six patients with bilingual aphasia (three patients with parallel aphasia and three patients with differential aphasia) in a syntactic priming task, in which they had to describe a picture after having heard the experimenter describing one. The aim of the study was to answer three research questions: (1) Do patients with bilingual aphasia show syntactic priming within and across languages? (2) Does the size of the syntactic priming effect differ across bilingual aphasia patients and healthy control subjects? And (3) Do patients with differential aphasia still show cross-lingual syntactic priming from the most affected language? Testing patients with differential aphasia allowed us to investigate whether we could still find syntactic priming from the most affected language as prime language. Within the view that the underlying mechanism of selective and differential aphasia is a problem in cognitive control and not

in a brain area representing a single language (Pitres, 1895), we expected that patients with differential aphasia could still show syntactic priming from their most affected language, because the representations themselves are intact, as loss of functionality in spontaneous productions could reflect a language control problem instead. If this hypothesis is correct, production in the most affected language fails because there is a problem in activation or inhibition of the target or non-target language, respectively, however not because the target language representations themselves are dysfunctional. In the syntactic priming paradigm, however, the primes in the most affected language need to be comprehended, not produced. This requires less language control, because language selection is not strictly necessary for comprehension (one may just rely on bottom-up activation from the input), unlike production (Costa & Santesteban, 2004a). As such, activation of syntactic representations after comprehension of the prime in the most affected language might still transfer to production of the same grammatical structure in the best-preserved language.

We administered a dialogue experiment with four conditions: two within language conditions (L1-L1 and L2-L2) and two between language conditions (L1-L2 and L2-L1). We found a robust, statistically significant syntactic priming effect in both patient groups, which was in general comparable in size with the effect found in healthy controls. However, some interesting group differences emerged. Control subjects and patients with parallel aphasia showed stronger syntactic priming effects in within language priming compared to the between languages conditions, whereas the patients with differential aphasia did not show a difference in the size of the priming effect within and between languages. In addition, control participants and patients with parallel aphasia showed stronger priming effects from L2 primes, whereas patients with differential aphasia showed stronger priming effects from the best-preserved language (L1). So, parallel aphasic patients behaved much more similar to controls than patients with differential aphasia. The most important finding in this study was that patients with differential aphasia still showed strong syntactic priming effects with L2 primes, even though this is the most affected language. The overall 8.4% priming effect with L2 primes is comparable with the L2 priming effect of patients with parallel aphasia (10.6%) and control subjects (9.2%). Only

looking at the L2-L1 condition, differential patients even showed stronger priming effects (9.5%) than parallel patients (5.9%) and control participants (7.1%). This suggests again that the most impaired language is not "lost". The syntactic representations in itself are still intact, and are able to influence (syntactic) processing in the other language. This is the first time cross-lingual interactions are found at the syntactic level in patients with (parallel and differential) bilingual aphasia.

Together, in chapter three, four and five, we found compelling evidence for cross-lingual interactions in patients with differential aphasia, at the word and at the syntactic level. These findings strongly argue against a localised view on bilingual aphasia, stating that differential (and selective) aphasia is caused by the selective damage to the language specific brain areas.

THEORETICAL IMPLICATIONS

As was described in the General Introduction, two large views on selective and differential aphasia have been proposed in the literature. A localised view postulated that the functional loss of a language is due to the selective damage of a language specific brain area. Oppositely, the more dynamic view argues that functional language loss is rather the result of a deficit in language control, and reflects the inability to inhibit the non-target language or to activate the target language. In the current dissertation, we aimed to empirically test this control deficit hypothesis in patients with parallel and differential aphasia. We assumed that if differential aphasia indeed reflects a control deficit rather than the damage to a language specific brain area, then patients with differential aphasia should show a deficit in performing cognitive control tasks. In addition, they should still show effects of cross-lingual interactions, since both languages are still intact.

Cross-lingual interactions

The Revised Hierarchical model (RHM, Kroll & Stewart, 1994) assumed that bilinguals have a separate lexicon for each language, in which

translation equivalents are strongly connected to each other via lexical links, and to their semantic representation via conceptual links. The strength of the connections is asymmetric (stronger lexical links from L2 to L1 than vice versa, and stronger conceptual links for L1 than for L2), and assumed to be dependent of language proficiency (with increasing proficiency, L1-L2 lexical links and L2 conceptual links strengthen).

Although no specific claims have been made about bilingual aphasia, because the RHM contains two separate lexicons, it would probably support a more localised view on differential and selective aphasia, suggesting that the functional loss of the language is the result of the language specific brain area representing that language. Finding highly interacting lexicons even in patients with differential aphasia (chapter three, four and five) provides evidence against this view, and is therefore not consistent with the RHM or other models that assume localised lexical representations.

In the procedural/declarative model (Ullman, 2001a, 2001b), it is postulated that vocabulary, both in L1 and L2, is represented in the declarative memory systems, whereas L1 grammatical rules are represented in the procedural memory. The representation of L2 grammar depends on L2 proficiency. With low levels of L2 proficiency, L2 grammatical rules will rely on declarative memory systems. However, as L2 proficiency increases and L2 grammar becomes more automatic, it will rely more on procedural memory. Although the model does not make specific claims about bilingual aphasia either, in unbalanced bilinguals, it might provide a plausible explanation for some cases of differential and selective aphasia of L1, assuming that the procedural memory systems are damaged. This would then primarily affect grammatical processing of L1. Furthermore, following the previous interpretation, the model cannot account for selective/differential impairments in L2. Thus, the observation that most of our patients with differential aphasia showed larger impairments in L2 compared to L1 is inconsistent with this prediction. In addition, since in balanced bilinguals, both languages rely on the same memory system, the model predicts that these bilinguals could only suffer from parallel aphasia. Again, this is not in line with our observations: we also included balanced bilinguals who suffered from differential aphasia. The observation of differential aphasia in

balanced bilinguals cannot be explained by the model of Ullman. In addition, since we only included highly proficient and balanced bilinguals, our results cannot provide a straightforward test of the developmental aspect of Ullmans hypothesis, namely the evolution towards procedural memory as L2 proficiency increases.

Unlike the RHM, the BIA+ model assumes an integrated lexicon for both languages mastered by an individual (*figure 1*). The model contains different interconnected levels: a sensory level for feature representations, a level for letter representations, a word level (lexicon) for lexical representations, which are connected to language nodes. Lexical access is assumed to be language non-selective, and the integrated lexicon contains words from both languages.

Our findings indeed support this view, showing that both languages of a patient with bilingual aphasia are still able to influence each other, even when one of the languages seems (partially) "lost" at the functional level. The cross-lingual interactions found in chapter three and four indicate that the word identification system is intact: the presentation of a cognate word activates the cognate in both languages. In the generalised lexical decision task, this activation suffices to activate a YES-response at the task level. However, in the selective lexical decision task, a language choice has to be made: is the activated word an L1 or an L2 word? Thus, a language selection has to be made here, implying a control mechanism. The finding that the patient with differential aphasia did not show a cognate facilitation effect in the selective lexical decision task, whereas he did show this effect in the generalised lexical decision task, supports our hypothesis that this patient suffers from a control deficit rather than from damage to a language specific brain area. This would imply a deficit at the task schema in the BIA+ model. In the next section, we will discuss the theoretical implications of our results for (bilingual) language control models in more detail.

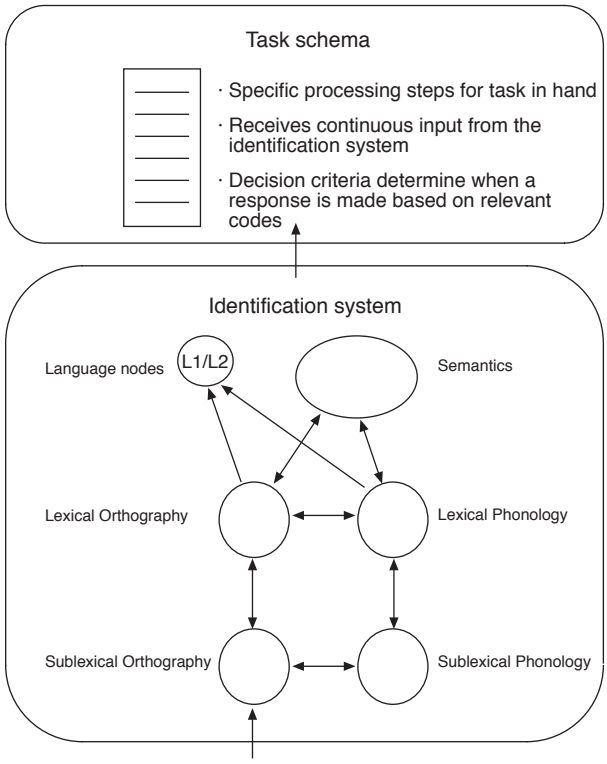

Figure 1. The Bilingual Interactive Activation (BIA+) model (Dijkstra et al., 1999; Dijkstra & van Heuven, 2002)

The results of chapter five, comparable syntactic priming effects in patients with differential aphasia and in patients with parallel aphasia and control subjects, confirm our predictions based on the bilingual syntactic priming model of Hartsuiker et al. (2004) discussed in the General Introduction (*figure 2*).

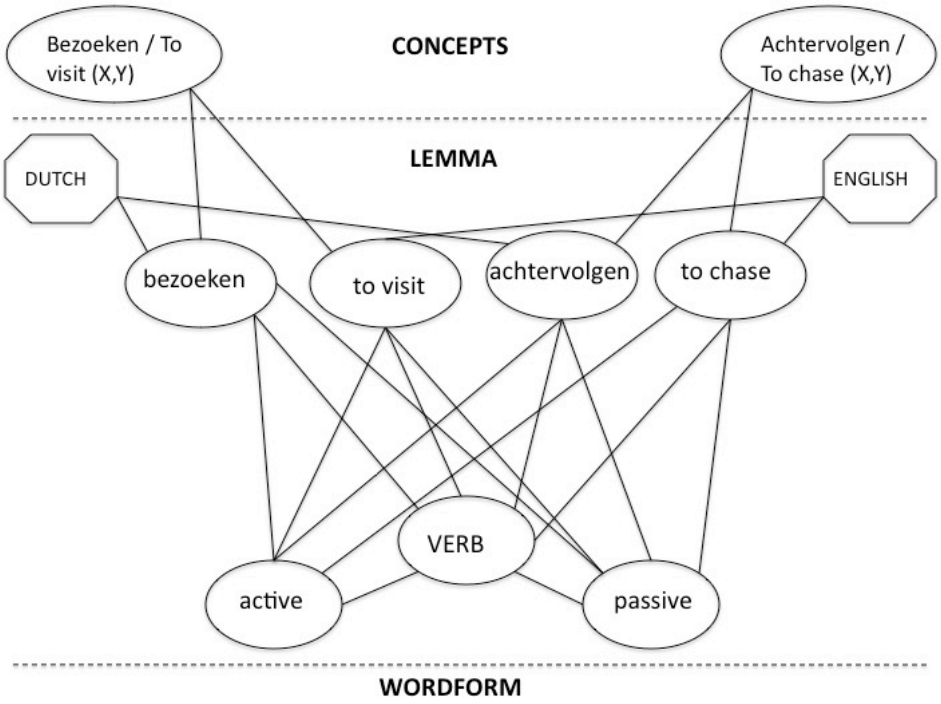

Figure 2. The model for cross-lingual syntactic priming proposed by Hartsuiker et al. (2004) applied to a Dutch-English example. In this model, the lemma nodes of the verbs [bezoeken] / [to visit] and [achtervolgen] / [to chase] are connected to a shared active combinatory node, and a shared passive combinatory node. Each lemma node is also connected to a category node for verb, and a language node (Dutch or English).

In this model, priming effects both within and across languages are explained in terms of residual activation in syntactic representations after comprehension of the prime. For instance, both the verbs “to visit” and “to chase” are transitive, and thus both assumed to be connected to an active and to a passive combinatory node. Hearing a passive sentence (“The patient is visited by the doctor.”) will activate both the lemma node “to visit” and the passive combinatory node. In the production of a following sentence, the residual activation of this passive combinatory node will facilitate the production of a passive sentence, even with another verb.

Because the model assumes shared syntactic representations across languages (i.e. combinatory nodes are connected to all transitive verbs, in either language), it predicts cross-lingual priming effects as long as these syntactic representations are intact. As such, although the model does not make any claims concerning (differential) bilingual aphasia, it is also compatible with accounts that explain differential aphasia in terms of language control. The finding of syntactic priming effects in patients with differential implies that the lemma nodes and the combinatory nodes are still intact. In addition, it is hypothesized that spreading activation of the combinatory nodes results in these effects, and that the combinatory nodes are not dependent of top-down language control. Otherwise, we would have expected no syntactic priming effects from the most affected language in patients with differential aphasia. Further research will be needed to apply these models to syntactic priming effects in patients with bilingual aphasia.

Cognitive control

At first, it seems hard to reconcile the assumption of highly interacting languages represented in overlapping brain regions with the occurrence of selective and differential aphasia. How can brain damage only affect one language, if these languages are represented in largely overlapping brain areas?

Already in 1985, Pitres hypothesised that a control deficit might be underlying selective and differential aphasia in bilinguals. Indeed, problems of control seem to offer a ready account of certain aphasia patterns: e.g.

lexical representations could be intact, but there might be a problem in the activation or inhibition of a language. In addition, Green (1998) stated that the mechanism responsible for (bilingual) language control is not selectively involved in language control, but also in control for non-linguistic tasks. Therefore, we hypothesised that if these patients indeed suffered from a control problem, this would be reflected on language unrelated control tasks.

Green extended the BIA+ model (Dijkstra et al., 1999; Dijkstra & van Heuven, 2002), which is a model for language recognition, to the field of language production. This has lead to the Inhibitory Control model (IC model, Green, 1998). In this model, both languages of a bilingual are, right from the start of the learning process, represented in one integrated lexicon. In order to be able to produce language in the intended language, bilinguals have to select (words in) the target language, and to suppress (words in) the non-target language. Therefore, individuals must resolve competition between language task schemas. Green proposed that the selection of a language involves the inhibition of the lexical representations of the unintended language. More specifically, suppression is applied after a lexical node has been activation from the conceptual system, and is proportional to the level of activation of a particular item (i.e., the more strongly activated the item, the more inhibition is needed to prevent it from being produced). Therefore, inhibiting a more dominant language (e.g. L1) seems to be requiring more cognitive resources than inhibiting a less dominant language (e.g. L2 in an unbalanced bilingual). In addition, Green assumed that the mechanism that manages bilingual language control is part of a general executive control system.

It was suggested that a differential and selective functional language loss in bilingual aphasia is the result of a deficit in this general control system: both languages are still intact, but patients are unable to selectively inhibit the non-target language or to selectively activate the target language. If this is the case, then bilinguals would still show effects of cross-lingual interaction (as was described in the previous section), but also show a deficient performance on tasks tapping into executive control.

This is indeed what was found in the current studies. The patients with differential aphasia included here showed a remarkable deficit on

executive control (assessed using a flanker task), compared to patients with parallel aphasia and healthy control subjects. This is in line with the predictions based on the IC model (Green & Abutalebi, 2008), and suggests that differential aphasia reflects the disability to generate sufficient inhibitory control to suppress the best-preserved language. Given the assumption that more inhibitory control will be needed to suppress the dominant language (in most cases, this is L1), this model also predicts that in differential and selective aphasia, L2 will always be the most affected language. This is however not consistent the aphasia pattern of all our patients. In chapter four, one patient (D02) who mastered French (L1) and Dutch (L2) before the stroke, showed larger impairments in French (L1) than in Dutch (L2). In addition, similar cases of patients with a more severely affected L1 have been described in the literature (Garcia-Caballero et al., 2007, see also Agliotti & Fabbro, 1993). So, the question remains how the IC model can account for patients with bilingual aphasia in whom L1 is most severely affected. One explanation could imply that language dominance, rather than the order of acquisition determines the amount of inhibition needed to inhibit a language (and therefore the direction of differential/selective aphasia). In this view, one could still predict differential aphasia with larger loss of the first acquired language if it is the most dominant one. This is not clear in our patient from chapter four (D02) because she still indicated the first acquired language as the most dominant one. But of course, pre-onset functional measurements of language use are not available in aphasia research, so this remains unclear at this point.

A second view on bilingual language control has been proposed by Costa and colleagues (Costa & Caramazza, 1999; Costa et al., 1999; Costa & Santesteban, 2004a; Costa, Santesteban, & Ivanova, 2006). They postulated that the lexical selection mechanism is sensitive to the language membership of lexical representations. This is called the language specific selection hypothesis: only the lexical representations that belong to the response language are considered for selection. Costa and Santesteban (2004b) further tried to reconcile both views, suggesting that the nature of control may depend on proficiency. For instance, individuals who a sufficiently proficient in L2 (as was also the case for the patients we included), to access lexical concepts in L2 without L1 mediation (see Kroll & Stewart, 1994), then the

language cue may become sufficient to ensure correct selection. So, with high levels of proficiency, bilinguals select the relevant language without competition from the other language. However, low-proficient L2 learners need to make use of inhibitory mechanisms, as was suggested by the IC model. Of course, it is not clear what neural structures this selection/inhibition mechanism would rely on, and as a consequence Costa and colleagues could perhaps also explain differential aphasia in terms of a deficient selection mechanism.

BILINGUAL APHASIA: IMPLICATIONS FOR THERAPY

Finding cross-lingual interaction in patients with bilingual aphasia might have important implications for language therapy. Up until now, language therapy is in most cases provided in only one language. One might wonder if this is always the best option. Would it be better to provide bilingual aphasic patients therapy in both languages, or does therapy in one language suffices? In the latter, which language should we prefer? And could we expect generalisation of treatment to the untrained language? Although the present dissertation was not about aphasia therapy, our findings allow discussing some therapeutical considerations.

Some authors have suggested that providing therapy in both languages might stimulate mutual inhibition, and therefore be detrimental for recovery in each language (Hilton, 1980; Lebrun, 1988). They argue that providing therapy in only one language seems to be preferable (Fabbro, Peru, & Skrap, 1997; Green, 2005; Paradis, 2004). In this case, we implicitly assume that therapy in the trained language will also benefit recovery in the untrained language. However, this is not always the case. Previous literature has shown that indeed training effects generalise to untrained languages, but not in every patient, and only under specific conditions. In general, effects of language therapy seem to be visible in untrained languages as well (Fabbro, 2001; Filiputti, Tavano, Vorano, De Luca, & Fabbro, 2002; Gil & Goral, 2004), however, the trained language still profits most (Junqué, Vendrell, Vendrell-Brucet, & Tobena, 1989). Hinckley (2003) treated a Spanish-English bilingual patient with transcortical motor aphasia in both languages,

thereby focusing on naming pictures. After the training, the patient showed enhanced performance on untrained items in both languages, with a larger advantage for L1 (Spanish). Goral et al. investigated grammatical and lexical skills in a trilingual (Hebrew, English and French) patient with chronic non-fluent aphasia (Goral, Levy, & Kastl, 2007). The therapy language was English, and consisted of two periods of three weeks, focusing on grammatical structures and word finding. Post-therapeutic assessment of grammatical structures, speech speed and fluency indicated that the patient did not only make progress in the trained language (English, L2), but also in Hebrew and French, which were not directly trained. These results show that therapy effects in one language can generalise to untrained languages (Filiputti et al., 2002; Miertsch, Meisel, & Isel, 2009). Marangolo et al. supported this by an imaging study, describing a patient with parallel recovery in a Dutch-Italian bilingual woman with aphasia (Marangolo, Rizzi, Peran, Piras, & Sabatini, 2009). She had phonological impairments in all modalities, and both languages were affected to the same extent. After a six-month therapy in which only Dutch (L1) was trained, the patient showed parallel recovery in both languages. In other words, therapy effects generalised from the trained to the untrained language, suggesting that similar cortical structures were involved in language recovery. Imaging data indicated that recovery was coupled with an extension of the cortical activation in both hemispheres, with identical activation patterns for both languages.

Kiran and Edmonds (2004) reported similar results, however making an important qualification. They applied semantic naming therapy in two English-Spanish bilingual aphasic patients, thereby focusing on strengthening the association between words and their meaning. One of the patients was a balanced bilingual, highly proficient in both languages. He showed generalisation to translation equivalents and semantically related words in the untrained language. No improvement was found in unrelated control words. The second patient was an unbalanced bilingual, with higher proficiency for English compared to Spanish. This patient only showed generalisation to semantically related words in the trained language, but no cross-lingual generalisation to Spanish could be found. In a next study, Edmonds and Kiran (2006) applied semantic naming therapy in three

English-Spanish patients with bilingual aphasia. Patient one was a balanced bilingual, patients two and three were unbalanced bilinguals with a higher proficiency in English. In the balanced bilingual patient, therapy was only provided in Spanish. This patient showed both within-language generalisation to Spanish semantically unrelated words, and between-language generalisation to English semantically related words. Patient two firstly received therapy in English, and afterwards in Spanish. Both patients showed generalisation to English translation equivalents and semantically related English words after Spanish language therapy. Taken together, premorbid proficiency in both languages seems to be an important factor for cross-lingual transfer of therapy benefits. This might reflect the strong interaction across languages in an integrated lexicon (cfr. BIA+ model). As such, activation of a language might generate spreading activation to the interconnected lexical items of another language, leading to generalisation of therapy benefits.

However, generalisation does not always occur. Meinzer et al. described a German-French bilingual aphasic patient who was highly proficient in both languages (Meinzer, Obleser, Flaisch, Eulitz, & Rockstroh, 2007). He only received language training in German, focusing on picture naming. Recovery was limited to German, no generalisation to French was observed. This study was the first to study effects of therapy by means of fMRI. Before and after training, an fMRI scan was made to investigate neural correlates of therapy on language processing in both languages. Post-therapy imaging only showed increased activation compared to pre-training brain activation in language processing in the trained language (German), but did not show any difference in activation pattern pre- and post-training when processing in the untrained language (French). Neural correlates of one-language therapy were also studied by Abutalebi and colleagues (Abutalebi, Rosa, Tettamanti, Green, & Cappa, 2009). Therapy was provided in English. Naming performance only improved in the trained language, which was coupled with an increased activation in brain regions involved in naming and control (mainly the prefrontal cortex). The finding of increased brain activation in control regions might be important for control-based accounts concerning differential aphasia: the increased brain activation in control regions might reflect the increasing activation of the trained

language, thereby increasing the inhibition of the untrained language. As such, not finding generalisation across languages might reflect a deficit to overcome this increased inhibition of the untrained language.

A possibly important factor in obtaining generalisation to untrained languages is provided by Lagarano and Overton-Venet (2001). They stated that generalisation occurs only when the task in both languages appeals to common computational processes and strategies. This was more concretely investigated using cognate stimuli in therapy. Kohnert (2004) applied Spanish language therapy with cognates and non-cognates in a 62-year-old Spanish-English bilingual man with severe non-fluent aphasia. In the trained language, an enhanced performance was observed for both cognates and non-cognates. Oppositely, in the untrained language, improvement was limited to cognates. In terms of computational models of bilingual word recognition, this implies that therapeutic exposure to some words only affects processing and representation of words that are highly overlapping across languages. The beneficial therapeutic activation does not noticeably spread across all representations of the untrained language. Detry et al. also only found cross-lingual generalisation for cognates (Detry et al., 2005). Kurland and Falcon (2011), however, report opposite results using cognates in language therapy. They provided semantic naming therapy in a bilingual patient with chronic expressive aphasia. During the first phase, the training language was Spanish, in the second English, and in the third phase, therapy was given in both languages. In all three phases, the patient showed enhanced performance on trained and related words, but the largest improvement was observed in non-cognates. The authors suggest that the phonological overlap in cognates causes interference, leading to decreased generalisation in cognates.

Because evidence for cross-lingual generalisation of therapy benefits is still quite limited, and this generalisation seems to be depending on various (still unknown) factors, some authors plead for providing therapy in both languages (Ansaldo & Marcotte, 2007; Centeno, 2005; Kohnert, 2004). Given the evidence that both languages of a bilingual interact quite strongly, it even seems evident to train both (Ansaldo, Marcotte, Scherer, & Raboyeau, 2008; Ansaldo & Marcotte, 2007; Kohnert & Goldstein, 2005). In addition, being a multilingual in recovery and therapy might be regarded as

an advantage, in the sense that it creates the possibility to fill up gaps in one language with words in the other.

Based on the existing literature, it is very difficult to draw a conclusion about whether it is really advantageous for the bilingual aphasia patient to receive language training in one or two languages, with or without cognates. However, because lexical representations of both languages seem to be functional to some extent, even in differential aphasia, we argue that cognates may provide a way to access the most affected language. Training with cognates before training with non-cognates in the most affected language might help to gradually activate it, and to overcome the inhibition. In this view, we argue to provide therapy in both languages, in order to prevent that the best-preserved language becomes even stronger, and therefore even harder to suppress in favour of activation of the least-preserved language.

In addition, based on the control hypothesis, we also suggest that it may be advisable to not only focus on language use and recovery in therapy, but instead also train executive functioning, or specifically language control (e.g. by language switching tasks). Although no therapy studies have investigated the influence of executive function training on the recovery of differential and selective aphasia yet, we argue that it is possible that these patients benefit from this kind of therapy as well. Up until now, training executive functions to enhance language abilities in bilingual aphasia has never been studied, however, there are some programs available to train executive functions after a stroke. For example, FRONTOMIX is a combination of exercises that aim to enhance a range of different executive functions (starting and stopping, persisting, planning etc.) in patients who suffered from a stroke (Paemeleire, Heirman, Savonet, & van Beneden, 2009). As far as we know, the impact of this kind of training programs in patients with (differential) bilingual aphasia has never been empirically tested. However, based on the control deficit in patients with differential aphasia, as described in the current dissertation, we suggest that training executive functions might enhance language abilities in those patients as well, especially in the language that shows the largest functional deficiency.

In anticipation of more univocal research, it would be advisable to base the training protocol also on functional factors (which language(s) does the patient need in his daily life?), and to be aware of any beneficial or detrimental effects of the cognate status of the used therapy material.

LIMITATIONS OF THE STUDIES

A first limitation in our studies is the lack of data of monolinguals in the first empirical chapter of this dissertation, where we compared unbalanced bilinguals with balanced switching and non-switching bilinguals. This is a more practical issue, given that everyone in Belgium has at least knowledge of two languages. Including a group of monolinguals might have clarified whether the absence of a control difference between unbalanced and balanced bilinguals would have also generalized to monolinguals.

Secondly, we discuss some limitations inherent in studying patients with bilingual aphasia. As we evidently could not test the patients before the onset of aphasia, premorbid proficiency measures are never available. We are therefore limited to estimate premorbid language proficiency based on self-reports and evaluations from close family members. As such, it is difficult to precisely determine whether the patient was (almost) equally proficient in both languages, and to what extent each language was affected or has recovered. Although this limitation is not easy to solve, we think it is important not to undervalue the estimation of the individual patient. Even when close family and friends do not notice any language problems anymore, the patient is often very conscious of any small language deficits he or she might experience. So relying on self-reports to estimate the premorbid proficiency levels of the patients does not seem to be all that problematic (see also e.g. Agliotti, Beltramello, Girardi, & Fabbro, 1996; Goral et al., 2007; Hernandez et al., 2008).

More specifically for the languages that our bilingual aphasia patients mastered (Dutch, French and English), we experienced difficulties finding a comprehensive aphasia test that was comparable in the three languages. At first, the Bilingual Aphasia Test (BAT, Paradis & Libben,

1987) seemed a valuable possibility, however after closer inspection, this test turned out to be too time-consuming and outdated. For instance, a lot of the pictures were difficult to recognize, the test did not take cognate or homograph status into account, some rare words were included (e.g. “de kikvors” instead of “de kikker”) and the translation did not always occur very accurately (e.g. “de bundel”, instead of “het boek”). Eventually, we opted to use the Aken Aphasia Test (AAT, Graetz, De Bleser, & Willmes, 1992) to assess Dutch language abilities, because this test was used as a standard by the speech and language therapists in Ghent University hospital. However, we could not find a comparable English or French version of the AAT. Therefore we chose to use the Comprehensive Aphasia Test (CAT, Howard, Swinburn, & Porter, 2004) in English, and develop a French version of the AAT ourselves. We are aware of the fact that this is far from the ideal situation to compare language abilities in two languages, and that cross-lingual norms for parallel instruments are preferable. This methodological endeavour however would have threatened the main goals of this paper.

SUGGESTIONS FOR FURTHER RESEARCH

As was mentioned in the Introduction, this is the first time that the hypothesis that a control problem underlies selective and differential aphasia was directly investigated. It would be interesting to use other cognitive control tasks than the flanker task we used. For example: do patients with differential aphasia also show deficits in other cognitive control tasks, such as a Simon task, or task switching? Finding worse performance of patients with differential aphasia on other cognitive control tasks as well, would further support the hypothesis that a control problem underlies the selective impairment of one language, rather than the damage to language specific representations. Or instead, finding a dissociation with other intact cognitive control tasks, would yield a clearer idea about whether perhaps only specific functions may be deficient.

Finally, as already discussed in the previous section about language therapy, the role of bilingual exposure and cognates in language training should definitely be further investigated.

CONCLUSION

The global aim of the current dissertation was to clarify the bilingual language control mechanism and its role in bilingual aphasia, more specifically in differential aphasia. In the first empirical chapter, we further disentangled the development of the executive control advantage in bilinguals, showing that the advantage only emerges in bilingual who often switch between languages in daily life. In the following three empirical chapters, we investigated cross-lingual interactions and cognitive control performance in patients with differential and parallel aphasia. We consistently found evidence for highly interactive languages, even in patients who functionally "lost" one language (at least to a large extent). In addition, these patients with differential aphasia also showed deficient executive control performance. Taken together, our results are in line with models assuming an integrated lexicon representing both languages of a bilingual, and support a more dynamic explanation of differential aphasia, implying that not the selective damage of language specific brain areas, but rather a (language) control deficit gives rise to differential and selective aphasia.

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CHAPTER 7:
NEDERLANDSTALIGE SAMENVATTING

Menselijke taal is wellicht de belangrijkste vaardigheid die mensen hebben verworven tijdens de (cognitieve) evolutie. Echter, verschillende situaties kunnen de oorzaak zijn van het verlies van de mogelijkheid om taal te begrijpen en/of te produceren. Deze aandoening wordt *afasie* genoemd. De belangrijkste oorzaak van afasie is een cerebrovasculair accident (CVA). Daarnaast kunnen ruimte-innemende processen, infecties, degeneratieve aandoeningen, traumata of intoxicaties fatische stoornissen veroorzaken.

Een persoon die voor het letsel twee of meerdere talen beheerste, ontwikkelt na het acute event een polyglotte of meertalige afasie. Het gaat hier niet per definitie om gebalanceerde tweetaligen. In onderzoek wordt veelal de definitie van Grosjean gebruikt, die tweetaligheid als volgt definieert: "*Het regelmatig gebruik van twee talen in het dagelijkse leven. Tweetaligen zijn zij die twee talen in hun dagelijks leven nodig hebben en gebruiken*" (Grosjean, 1989).

Het eerste doel van het huidige proefschrift was om taalcontrole bij tweetaligen te onderzoeken (**hoofdstuk twee**), omdat taalcontrole ook het centrale concept was in onze hypothese voor de verklaring van differentiële tweetalige afasie. Het tweede doel was om cross-linguale interacties en cognitieve controle te onderzoeken bij patiënten met tweetalige afasie. We bestudeerden cross-linguale interactie op verschillende niveaus: op het lexicale niveau, in een gevalstudie (**hoofdstuk drie**) en in een grote groepsstudie bij patiënten met parallelle en differentiële afasie (**hoofdstuk vier**), en op het syntactische niveau (**hoofdstuk vijf**). Op deze manier hebben we geprobeerd om de rol van taalcontrole in de verschillende vormen van tweetalige afasie te doorgronden.

TWEETALIGHEID: HUIDIGE STAND VAN HET ONDERZOEK

Cross-linguale interacties op woordniveau

Een belangrijk debat in het onderzoek naar tweetaligheid concentreert zich rond de aard van het tweetalig lexicon (i.e. het geheugen voor lexicale representaties). Hebben tweetaligen een apart lexicon voor elke taal die ze beheersen, of worden alle woorden gerepresenteerd in een gemeenschappelijk lexicon?

Een paradigma waarin veel evidentie werd gevonden voor de tweede hypothese, namelijk één gemeenschappelijk lexicon met sterk interagerende talen, is het onderzoek met cognaten. Cognaten zijn woorden die in beide talen dezelfde betekenis hebben, en een gelijkaardige vorm (e.g. *boek – book*). Onderzoek heeft aangetoond dat cognaten sneller herkend worden dan niet-cognaten, zelfs wanneer het onderzoek in een zuiver ééntalige context wordt uitgevoerd (Costa, Santesteban, & Cano, 2005; Duyck, Van Assche, Drieghe, & Hartsuiker, 2007; van Hell & Dijkstra, 2002). Bijvoorbeeld, als Nederlands-Engelse tweetaligen een Nederlandse zin lezen waarin het woord *boek* staat, dan zullen ze dit woord sneller herkennen (en er dus minder lang naar kijken) dan een controle woord (e.g. lepel). Dit effect, het cognaat facilitatie effect genoemd, wordt beschouwd als een gevolg van taal niet-selectieve activatie, en wordt verklaard door de spreidende activatie van zowel semantische, orthografische en fonologische representaties over de verschillende talen. Een cognaat heeft immers zowel de betekenis als de spelling en de klanken gemeenschappelijk in verschillende talen, en het is deze overlap die ervoor zorgt dat het cognaat sneller herkend wordt.

Een model dat erg belangrijk is gebleken in de literatuur naar taalherkenning, is het Bilingual Interactive Activation model (BIA+ model, Dijkstra, Grainger, & van Heuven, 1999; Dijkstra & van Heuven, 2002). Dit model veronderstelt één geïntegreerd lexicon voor de representatie van elke taal die een meertalige beheerst. Het bevat verschillende niveaus: de sensorische input activeert eerst kenmerkrepresentaties, die op hun beurt letterrepresentaties activeren. Deze letterrepresentaties sturen activatie naar

lexicale (woord)representaties in verschillende talen. Elke lexicale representatie is bovendien verbonden met een taalknoop. Het model in interactief in de zin dat feedback van hogere naar lagere niveaus mogelijk is. Bovendien werd verondersteld dat lexicale toegang niet taalselectief is.

Ook beeldvormingsonderzoek heeft aangetoond dat beide talen van een tweetalige beroep doen op gelijkaardige en sterk overlappende hersengebieden (Chee, Tan, & Theil, 1999; Hernandez, Martinez, & Kohnert, 2000; Illes et al., 1999; Klein, Milner, Zatorre, Zhao, & Nikelski, 1999; Klein, Zatorre, Milner, Meyer, & Evans, 1994; Pu et al., 2001; Vingerhoets et al., 2003).

Cross-linguale interacties op zinsniveau

Ook op zinsniveau werd evidentie gevonden dat de twee talen van een tweetalige elkaar sterk beïnvloeden. Syntactische priming is het fenomeen waarbij de productie van een zin gefaciliteerd wordt wanneer die zin voorafgegaan wordt door een zin met een gelijkaardige structuur (Bock, 1986). Dit werd niet alleen in ééntalig onderzoek gevonden, maar blijkt ook het geval te zijn tussen talen (Hartsuiker, Pickering, & Veltkamp, 2004; Hartsuiker & Pickering, 2008; Meijer & Tree, 2003; Schoonbaert, Hartsuiker, & Pickering, 2007; Shin & Christianson, 2009). Hartsuiker en collega's stelden een theoretisch kader voor om deze cross-linguale syntactische priming effecten te kunnen verklaren (Hartsuiker et al., 2004; gebaseerd op Pickering & Branigan, 1998). Pickering en Branigan gingen ervan uit dat de lexicale representaties van woorden verbonden zijn met categorische knopen, die coderen voor het woordtype, en met combinatorische knopen, die de mogelijke grammaticale constructies representeren en die gedeeld zijn over lexicale representaties in verschillende talen. Bijvoorbeeld, een transitief werkwoord zal zowel met een actieve als een passieve combinatorische knoop verbonden zijn. Bij het horen van een passieve zin zal zowel de lexicale knoop van het werkwoord als de passieve combinatorische knoop actief worden. Deze restactivatie van de passieve knoop zal er volgens het model voor zorgen dat het produceren van een passieve zin gefaciliteerd wordt. Op deze manier biedt het model een verklaring voor syntactische priming effecten, zowel binnen als tussen talen.

Controle bij tweetaligen

Recent is de aandacht binnen het onderzoek naar tweetaligheid gedeeltelijk verschoven naar het domein van taalcontrole. Gegeven dat beide talen van een tweetalige inderdaad sterk interageren en gerepresenteerd worden in gedeelde hersengebieden, hoe is een tweetalige in staat om deze activatie te controleren? Een effect dat veel interesse heeft uitgelokt binnen het controle onderzoek is de bevinding dat tweetaligen een voordeel lijken te vertonen op cognitieve controletaken. Meer specifiek lijken tweetaligen het beter te doen dan eentaligen op taken die de inhibitie van irrelevante informatie vereisen (e.g. flanker taak, Simon taak) (e.g. Bialystok et al., 2005; Bialystok, Craik, Klein, & Viswanathan, 2004; Bialystok, Craik, & Ryan, 2006), ook als deze informatie niet linguïstisch van aard is. Dit voordeel lijkt het gevolg te zijn van het voortdurend moeten controleren van de activatie in beide talen, om (woorden in) de doeltaal te kunnen selecteren, en (woorden in) de niet-doeltaal te kunnen onderdrukken.

Echter, dit voordeel op controletaken werd niet consistent gevonden bij tweetaligen. Paap en Greenberg (2013) vergeleken de prestaties van grote groepen tweetaligen op 15 cognitieve controletaken, en vonden zo goed als geen voordeel van tweetaligen. Dit onderzoek toont aan dat louter het beheersen van twee talen misschien niet voldoende is om een voordeel in cognitieve controle te ontwikkelen. Inderdaad, Prior en Gollan (2011) vonden reeds dat daarin ook andere factoren meespelen. Zij toonden aan dat enkel tweetaligen die regelmatig switchen tussen talen in het dagelijkse leven, een voordeel vertonen op taakswitchen in een experimentele setting. In hoofdstuk twee van het huidige proefschrift hebben we de rol van taalswitchen verder onderzocht (cfr. infra).

Een mogelijke verklaring voor het controlevoordeel kan gevonden worden in het Inhibitorische Controlemodel van Green (IC model, Green, 1998). Green breidde het BIA+ model, dat in essentie een model voor taalherkenning bij tweetaligen is, verder uit naar het domein van taalproductie. Hij stelde dat er bij productie een competitie plaatsvindt tussen woorden uit verschillende talen. Deze competitie kan worden opgelost door de inhibitie van de irrelevante taal. Bovendien stelt Green dat de mate van inhibitie die nodig is om de taal te kunnen onderdrukken, afhankelijk is van

de taalvaardigheid van de persoon in die taal. Een dominante taal wordt verondersteld moeilijker te onderdrukken te zijn in vergelijking met een minder dominante taal. Het mechanisme dat instaat voor deze inhibitie maakt volgens Green deel uit van een algemeen controlemechanisme, dat zowel taalcontrole als niet-taalgerelateerde controle processen beheert. Deze assumptie vormt de verklaring voor het controlevoordeel dat gevonden wordt bij (bepaalde groepen) tweetaligen: de voortdurende training van het algemeen controlemechanisme voor de taalcontrole leidt tot een betere ontwikkeling van dit mechanisme, wat gereflecteerd wordt in betere prestaties op cognitieve controletaken.

TWEETALIGE AFASIE: HUIDIGE STAND VAN HET ONDERZOEK

In tegenstelling tot wat men misschien intuïtief zou verwachten, worden bij personen met meertalige afasie niet steeds alle talen op dezelfde manier aangetast, noch herstellen ze gelijktijdig of in dezelfde mate. Volgens Paradis kunnen herstelpatronen bij tweetalige afatici zich op verschillende niveaus manifesteren (Paradis, 1977): parallel (beide talen herstellen even goed), differentieel (de ene taal herstelt beter dan de andere), selectief (slechts één taal herstelt), successief (de ene taal herstelt pas nadat de andere volledig hersteld is), antagonistisch (er vindt een afwisseling van herstel in beide talen plaats) en gemengd (de patiënt gebruikt beide talen voortdurend door elkaar). In analogie met de herstelpatronen die vooropgesteld werden door Paradis kunnen we ook de types van taalaantasting bij tweetalige afasiepatiënten op een gelijkaardige manier onderverdelen. In de huidige dissertatie spitsten we ons toe op twee aantastingspatronen, namelijk parallelle afasie (beide talen zijn in gelijke mate aangetast) en differentiële afasie (één taal is sterker aangetast dan de andere). Differentiële afasie is in dat opzicht een minder extreme vorm van aantasting dan selectieve afasie, wat inhoudt dat slechts één taal is aangetast, terwijl de andere volledig intact is gebleven. Aangezien we tijdens de duur van dit doctoraat geen patiënt met selectieve afasie hebben ontmoet, hebben we enkel patiënten met parallelle en differentiële afasie bestudeerd.

Ondanks het feit dat doorheen de jaren heel wat voorstellen worden gedaan, is de bepalende factor voor het herstel- en aantastingspatroon nog steeds onbekend. Taalstatus (L1 of L2, meest gebruikte taal, ...), plaats of type van de laesie, afasietype, de manier waarop de taal geleerd wordt, leeftijd, taalvaardigheid, type van tweetaligheid, ... Geen van deze factoren kan volgens Paradis (1977) een afdoende verklaring bieden voor de herstel- en aantastingspatronen die voorkomen bij patiënten met polyglotte afasie.

Echter, reeds in 1895 stelde Pitres (1895) een probleem in taalcontrole voor als mogelijk onderliggend mechanisme voor de verschillende herstel- en aantastingspatronen in tweetalige afasie, beschreven door Paradis. Afgaand op de observatie dat patiënten sneller hun 'verloren' taal terug verwerven dan dat ze een nieuwe taal zouden leren, stelde hij dat de taal niet meer beschikbaar was omdat ze te sterk geïnhibeerd werd in de vorm van *pathologische inertie*. Deze visie werd later opgepikt en vorm gegeven in een meer *dynamische verklaring* voor tweetalige afasie. Deze dynamische verklaring stelt dat een probleem in cognitieve controle aanleiding geeft tot de verschillende herstelpatronen bij patiënten met tweetalige afasie, en niet zozeer het feit dat hersengebieden die specifiek één van beide talen representeren zouden aangetast zijn, zoals gesteld werd door meer *lokalisatorische verklaringen*.

Indien deze hypotheses kloppen, en differentiële afasie veeleer het gevolg van een probleem in cognitieve controle is dan een reflectie van de beschadiging van het taalspecifieke hersengebied, dan kunnen we verwachten dat patiënten met differentiële afasie nog steeds effecten van sterk interagerende talen vertonen, en dat zij bovendien een verminderde prestatie op controletaken laten zien. Deze hypothese lijkt een meer interessante verklaring te bieden voor het bestaan van differentiële afasie, die verzoenbaar is met het gedrags- en neuroanatomische onderzoek dat geïntegreerde taken suggereert. Vermits deze hypothese echter nooit empirisch onderzocht werd, was dit het primaire doel van dit proefschrift.

OVERZICHT VAN DE BEVINDINGEN

In het eerste empirische hoofdstuk (**hoofdstuk twee**) van dit proefschrift hebben we de rol van taalswitchen in de ontwikkeling van het tweetalig voordeel op executieve functies verder onderzocht. In tegenstelling tot Prior en Gollan (2011), die vaak switchende Engels-Spaanse tweetaligen en minder vaak switchende Engels-Mandarijnse tweetaligen onderzochten, includeerden we enkel Nederlands-Franse tweetaligen, die onderling enkel verschilden in de taalvaardigheid in het Frans (L2) en in de mate van switchen in het dagelijkse leven. Daarenboven onderzochten we niet taakswitchen als afhankelijke variabele, maar onderzochten we of taalswitchen in het dagelijkse leven ook een invloed heeft op inhibitie, door middel van een flanker taak en een Simon pijl taak. Enkel in de groep gebalanceerde tweetaligen die regelmatig switchen tussen talen in het dagelijkse leven, vonden we een voordeel op de inhibitietaken (nl. een kleiner conflicteffect). Dit suggereert dat louter het beheersen van twee talen niet voldoende is om betere prestaties op cognitieve controle taken te vertonen, maar dat de mate van switching tussen talen in het dagelijkse leven cruciaal is. De simultane activatie van de lexicale representaties in beide talen, en het voortdurend gebruiken of inhiberen van de representaties bij het regelmatige switchen tussen talen, zorgt wellicht voor competitie, en vereist het inschakelen van een controlemechanisme om de targettaal te activeren, en de non-targettaal te inhiberen. Dit regelmatig gebruik van het controlemechanisme voor taalcontrole breidt uit naar niet-taalgerelateerde controlefuncties, en wordt gereflecteerd in een kleiner conflicteffect op taken zoals een flanker taak en een Simon pijl taak. Deze bevinding kan een antwoord bieden op de bevindingen van Paap en Greenberg (2013), die er niet in slaagden om het controlevoordeel bij tweetaligen te repliceren, en toont aan dat het controlevoordeel afhankelijk is van taalgebruik karakteristieken van tweetaligen, zoals de frequentie waarmee ze beide talen door elkaar gebruiken.

In **hoofdstuk drie** beschrijven we de prestaties van een patiënt met differentiële afasie op een flanker taak en twee versies van een lexicale decisietaak met cognaten. We gebruikten een gegeneraliseerde lexicale decisietaak (“druk op een knop als het woord bestaat in eender welke taal”)

en een selectieve lexicale decisietaak ("druk op een knop als het woord een bestaand Nederlands/Frans woord is") (Dijkstra, Timmermans, & Schriefers, 2000). We kozen voor deze twee versies vanwege het verschil in controlevereisten: omdat een gegeneraliseerde lexicale decisietaak een "JA" antwoord vereist bij woorden uit beide talen, terwijl dit bij de selectieve lexicale decisietaak enkel het geval is bij een woord uit de doeltaal, vereisen beide taken een verschillende mate van taalcontrole. In de selectieve lexicale decisietaak dient er immers een taalselectie gemaakt te worden, die niet nodig is in de gegeneraliseerde lexicale decisietaak. Dit zorgde ervoor dat we cross-linguale interacties (met het cognaateffect als marker) konden onderzoeken bij verschillende niveaus van controlevereisten. Aangezien we een controledeficiet veronderstelden aan de basis van de differentiële aantasting van de talen in deze patiënt, verwachtten we grotere effecten van cross-linguale interactie in de gegeneraliseerde dan in de selectieve lexicale decisietaak.

Deze verwachtingen werden bevestigd. In de gegeneraliseerde lexicale decisietaak vertoonde de patiënt een duidelijk cognaateffect: cognaten werden beter herkend dan niet-cognaten. Dit impliceert dat de sterkst aangetaste taal (Nederlands) nog steeds de verwerking in de minst aangetaste taal (Frans) kan beïnvloeden. In de Franse selectieve lexicale decisietaak vertoonde de patiënt geen cognaat facilitatie effect, terwijl in de Nederlandse selectieve lexicale decisietaak cognaten zelfs trager werden herkend dan niet-cognaten. Ook deze bevinding kan verklaard worden aan de hand van controlevereisten: Frans lijkt minder sterk aangetast omdat het controlemechanisme er wel nog in slaagt om het Nederlands te onderdrukken, maar niet meer het (dominante) Frans. Op zich vormde deze gevalsstudie reeds ondersteuning voor de hypothese dat een controleprobleem en niet de beschadiging van taalspecifieke hersengebieden aan de basis ligt van differentiële controle. Omdat dit echter een gevalsstudie was, en we geen rechtstreekse vergelijking konden maken tussen patiënten met verschillende soorten tweetalige afasie, hebben we in de volgende studie getracht deze bevindingen uit te breiden.

In **hoofdstuk vier** vergeleken we de prestaties van een grote groep patiënten met parallelle en differentiële afasie met een groep gezonde controlepersonen op een gegeneraliseerde lexicale decisietaak met cognaten

en op een flanker taak. Door middel van de flanker taak wilden we de niet-taalgerelateerde executieve controlefuncties van de patiënten met tweetalige afasie nagaan. Als het inderdaad zo is dat een controleprobleem aan de basis ligt van differentiële afasie, dan verwachtten we dat de patiënten met differentiële afasie slechter zouden presteren op deze controletaak in vergelijking met patiënten met parallelle afasie. Daarenboven verwachtten we dat beide groepen patiënten gelijkaardige effecten van cross-linguale interactie zouden vertonen in een situatie met lage controlevereisten, aangezien de hersengebieden die beide talen representeren, nog steeds intact zijn. We verwachtten dat beide patiëntengroepen een even groot cognaateffect zouden vertonen, vergelijkbaar met de controleparticipanten. We gebruikten een gegeneraliseerde lexicale decisietaak omwille van de lage controlevereisten (cfr. suppra). Onze hypotheses werden bevestigd. De patiënten met differentiële afasie vertoonden significante cognaateffecten die gelijkaardig zijn aan de cognaateffecten van patiënten met parallelle afasie en gezonde controlepersonen. Bovendien vertoonden de patiënten met differentiële afasie een groter conflict effect op de flanker taak in vergelijking met de patiënten met parallelle afasie en gezonde controle personen, wat een deficiet in het controlemechanisme reflecteert. Opnieuw vormen deze resultaten ondersteuning voor een meer dynamische verklaring van differentiële afasie, die uitgaat van een (taal)controleprobleem als onderliggend mechanisme en niet van de beschadiging van de representaties in een taalspecifiek hersengebied.

Terwijl we in hoofdstukken drie en vier cross-linguale interactie op woordniveau onderzochten, bestudeerden we in **hoofdstuk vijf** cross-linguale interacties op zinsniveau. We gingen na of onze patiënten met differentiële afasie nog steeds cross-linguale syntactische priming effecten vertoonden, en of deze effecten in grootte vergelijkbaar zijn met de effecten van patiënten met parallelle afasie en met controlepersonen. Dit bleek het geval: patiënten met differentiële afasie vertoonden gelijkaardige syntactische priming effecten als patiënten met parallelle afasie en gezonde controlepersonen, wat opnieuw ondersteuning bood voor de hypothese dat de representaties in beide talen van een patiënt met differentiële afasie nog intact zijn, en in staat zijn om de verwerking in de andere taal te beïnvloeden.

CONCLUSIE

De algemene doelstelling van het huidige proefschrift was het bestuderen van het tweetalige controlemechanisme, en van de rol ervan in tweetalige afasie, meer specifiek in differentiële afasie. In het eerste empirische hoofdstuk onderzochten we de voorwaarden voor het ontstaan van een controlevoordeel bij tweetaligen, daarbij aantonend dat het voordeel enkel lijkt gevonden te worden bij tweetaligen die regelmatig switchen tussen talen. In de daaropvolgende drie hoofdstukken onderzochten we cross-linguale interactie en cognitieve controle bij patiënten met differentiële en parallelle afasie. We vonden coherente evidentie voor sterk interagerende talen, zowel op lexicaal als syntactisch niveau, zelfs bij patiënten bij wie één taal functioneel sterk aangetast is. Bovendien vertoonden deze patiënten een probleem in controlefuncties. Samengenomen vormen deze resultaten ondersteuning voor modellen die een geïntegreerd lexicon voor beide talen veronderstellen, en zijn ze in lijn met een dynamische verklaring van differentiële afasie, die de sterkere aantasting van slechts één taal situeert in een controleprobleem, en niet in de beschadiging van een taalselectief hersengebied.

De bevindingen van het huidige proefschrift bieden bijkomende ondersteuning voor het BIA+ model (Dijkstra, et al., 1999; Dijkstra & van Heuven, 2002), dat een geïntegreerd lexicon veronderstelt voor de beide talen die een tweetalige beheerst. Het vinden van cross-linguale interactie in hoofdstukken drie en vier toont aan dat het woordidentificatie systeem intact is: het zien van een cognaat activeert het woord in beide talen. Het verschil tussen beide taken (i.e. het vinden van een cognaateffect in de gegeneraliseerde lexicale decisietaak, maar niet in de selectieve lexicale decisietaak) suggereert echter dat het probleem zich bevindt op het niveau van taalcontrole. Volgens het Inhibitorisch Controle model van Green (1998) correspondeert taalcontrole met de mogelijkheid om (het woord in) de niet-doeltaal te onderdrukken. Er werd verondersteld dat het onderliggende probleem bij differentiële afasie ligt in de onmogelijkheid om onvoldoende inhibitie te genereren om één taal te kunnen onderdrukken. De resultaten van de hier beschreven studies tonen inderdaad dat patiënten met differentiële

afasie een controleprobleem vertonen, en liggen in lijn met het model van Green.

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