Cognition 131 (2014) 311–322

Contents lists available at ScienceDirect

Cognition

journal homepage: www.elsevier.com/locate/COGNIT

How embodied is action language? Neurological evidence from motor diseases

Juan F. Cardona [a,b,c,1], Lucila Kargieman [a,b], Vladimiro Sinay [a], Oscar Gershanik [a,b], Carlos Gelormini [a,b], Lucia Amoruso [a,b], María Roca [a], David Pineda [d], Natalia Trujillo [d], Maëva Michon [e], Adolfo M. García [a,b], Daniela Szenkman [a,b], Tristán Bekinschtein [f], Facundo Manes [a,b,g], Agustín Ibáñez [a,b,e,h,*]

[a] Laboratory of Experimental Psychology and Neuroscience (LPEN), Institute of Cognitive Neurology (INECO), Favaloro University, Buenos Aires, Argentina
[b] National Scientific and Technical Research Council (CONICET), Buenos Aires, Argentina
[c] School of Psychology, Catholic University of Pereira (UCP), Risaralda, Colombia
[d] Neuroscience Research Programme, University of Antioquia, Medellin, Colombia
[e] UDP-INECO Foundation Core on Neuroscience (UIFCoN), Diego Portales University, Santiago, Chile
[f] Cognition and Brain Sciences Unit, Medical Research Council, Cambridge CB2 7EF, United Kingdom
[g] Australian Research Council (ARC) Centre of Excellence in Cognition and its Disorders, NSW, Australia
[h] Universidad Autónoma del Caribe, Barranquilla, Colombia

ARTICLE INFO

Article history:
Received 2 May 2013
Revised 15 January 2014
Accepted 4 February 2014

Keywords:
EPD
NMO
ATM
Action language
Representations
Embodied cognition

ABSTRACT

Although motor-language coupling is now being extensively studied, its underlying mechanisms are not fully understood. In this sense, a crucial opposition has emerged between the non-representational and the representational views of embodiment. The former posits that action language is grounded on the non-brain motor system directly engaged by musculoskeletal activity – i.e., peripheral involvement of ongoing actions. Conversely, the latter proposes that such grounding is afforded by the brain's motor system – i.e., activation of neural areas representing motor action. We addressed this controversy through the action-sentence compatibility effect (ACE) paradigm, which induces a contextual coupling of motor actions and verbal processing. ACEs were measured in three patient groups – early Parkinson's disease (EPD), neuromyelitis optica (NMO), and acute transverse myelitis (ATM) patients – as well as their respective healthy controls. NMO and ATM constitute models of injury to non-brain motor areas and the peripheral motor system, whereas EPD provides a model of brain motor system impairment. In our study, EPD patients exhibited impaired ACE and verbal processing relative to healthy participants, NMO, and ATM patients. These results indicate that the processing of action-related words is mainly subserved by a cortico-subcortical motor network system, thus supporting a brain-based embodied view on action language. More generally, our findings are consistent with contemporary perspectives for which action/verb processing depends on distributed brain networks supporting context-sensitive motor-language coupling.

1. Introduction

A major area of debate for neurocognitive theories of language concerns the mechanisms underlying motor-language coupling. Most accounts of action language fit well

* Corresponding author at: Laboratory of Experimental Psychology and Neuroscience (LPEN), Institute of Cognitive Neurology (INECO) and CONICET, Pacheco de Melo 1860, Buenos Aires, Argentina. Tel./fax: +54 (11) 4807 4748.
E-mail address: aibanez@ineco.org.ar (A. Ibáñez).
[1] This work is part of J.F. Cardona's ongoing Ph.D. dissertation at Favaloro University, Argentina.

http://dx.doi.org/10.1016/j.cognition.2014.02.001
0010-0277/

within the embodied cognition framework, which proposes that cognitive processes are essentially grounded in bodily experience (Gallese & Lakoff, 2005; Gallese & Sinigaglia, 2011). Nevertheless, not all embodied cognition theories are conceptually identical, as they feature different views on the cognitive status of representations (for a conceptual review, see Wilson, 2002).

On the one hand, a radical, *non-representational embodied view* rejects traditional accounts based on internal representations (Alsmith & de Vignemont, 2012; Clark, 1997; Gallagher, 2005b; Van Gelder, 1995). This position suggests that peripheral sensory organs (i.e., musculoskeletal structures) automatically and unconsciously provide the necessary feedback for the execution of both gross motor programs and fine tuning, in the absence of semantic representations. In other words, cognitive processes are claimed to depend on the physical body much more than commonly assumed.

According to this view, the availability of perceptual and motor information dispels the need to invoke internal (mental) representations as the constructs that could explain complex behavior. Cognition-action couplings are understood as complex behaviors emerging from interactions among body, environment, and brain, in the absence of computational representations.

Admittedly, this non-representational account proves disfavored in contemporary cognitive neuroscience. However, it has been fruitful to explain phenomena observed in the fields of robotics (Beer, 2003; Brooks, 1999; Pfeifer, Bongard, & Grand, 2007; Pfeifer & Scheier, 1999); coordinated social activity in animals (Ballerini et al., 2008; Barrett, 2011; Reynolds, 1987); visuomotor search, such as the outfielder problem (Bingham, 1988; Fink, Foo, & Warren, 2009; McBeath, Shaffer, & Kaiser, 1995); and developmental changes in object recognition (Thelen, Schoner, Scheier, & Smith, 2001). Moreover, the non-representational account has provided new insights into language-motor coupling (Wilson & Golonka, 2013).

For the non-representational perspective, linguistic information precipitates actions by means of a coupled environment-body system (Wilson & Golonka, 2013). Linguistic, as well as perceptual, information would emerge from situated constraints (Wilson & Golonka, 2013). Therefore, in the absence of word-meaning representations, action-sentence couplings would result from situation-bound processes engaging both relevant linguistic information and musculoskeletal structures (Barwise & Perry, 1983). However, the dearth of empirical research suited to test this hypothesis renders it speculative and, hence, unpopular.

On the other hand, the more lenient *representational embodied view* focuses on the neural mechanisms involved in motor representation (Meteyard, Cuadrado, Bahrami, & Vigliocco, 2012). This hypothesis claims that motor activity and verbal representations of actions are mutually dependent processes at the brain level. Confirmatory evidence comes from several behavioral and neuroimaging studies showing significant overlaps between cortical motor areas engaged in action-related language and action execution (Aziz-Zadeh, Wilson, Rizzolatti, & Iacoboni, 2006; Hauk, Johnsrude, & Pulvermüller, 2004; Pulvermüller, 2005; Tettamanti et al., 2005). Embodied cognition hypotheses are the object of intense discussions (Willems & Francken, 2012). The precise role of brain motor areas and musculoskeletal structures in cognitive domains is still a matter of debate (Calvo & Gomila, 2008). Current models suggest a potential role of supramodal convergence zones in semantic grounding, in addition to sensory-motor circuits (Kiefer & Pulvermuller, 2012). However, the embodied mechanisms underlying action-verb processing remain unknown (Kiefer & Pulvermuller, 2012) and must be empirically established.

In this sense, the opposing views outlined above could be tested by assessing the role of two systems in the grounding of action-verb processing, namely: (a) the peripheral or musculoskeletal system (PMS) and (b) the brain motor system (BMS). Specifically, a comparison of motor-language interactions in patients with injuries compromising either system may shed light on the role(s) that PMS and BMS areas play in action-verb processing (see Section 1.3).

To our knowledge, no previous report has investigated the relative involvement of PMS and BMS in verbal processing or their relevance in language deficits in motor diseases. One direct way to test these hypotheses is to explore motor-language coupling in neuromotor conditions that impair either PMS or BMS structures. A better understanding of this phenomenon may clarify the specific level of body involvement in action language processing.

1.1. The action-sentence compatibility effect

Recent studies have examined the interaction between action semantics and motor performance through the action-sentence compatibility effect (ACE) paradigm (Aravena et al., 2010; Borreggine & Kaschak, 2006; De Vega, Moreno, & Castillo, 2013; De Vega & Urrutia, 2011; Glenberg & Kaschak, 2002). The ACE was originally found by Glenberg and Kaschak (2002). In their study, participants read sentences describing actions which denoted movements towards or away from the body and pressed one of two buttons located either close to, or away from, the body. The ACE is defined as longer reaction times (RTs) for incompatible relative to compatible action sentences. Similarly, Aravena et al. (2010) asked participants to judge sentences describing motor actions typically performed with an open hand (e.g., clapping) or a closed hand (e.g., hammering). Once again, RTs were faster when the hand response was congruent with the action in question. Importantly, Aravena et al. (2010) found brain markers of bidirectional effects between language comprehension and motor processes. More recently, the ACE paradigm was successfully used to tap action-language deficits in a motor disease –namely, early Parkinson's disease (EPD, Ibáñez et al., 2013).

1.2. Motor conditions evaluated in the present study

1.2.1. PMS affectation and BMS preservation: Neuromyelitis optica

Neuromyelitis optica (NMO), also known as Devic's disease, is a demyelinating disease that affects white matter

in the optic nerve and spinal cord (Lin, Yu, Jiang, Li, & Chan, 2007; Yu et al., 2008). It is characterized by varied peripheral motor symptoms (e.g., limb weakness, paralysis) without movement-related cortical (premotor and primary motor areas) or subcortical (e.g., basal ganglia) dysfunctions (Wang, Liu, Duan, & Li, 2011; Wingerchuk, Lennon, Pittock, Lucchinetti, & Weinshenker, 2006). New evidence indicates that while NMO does not involve brain atrophy, there might be some white matter atrophy prevailing in diencephalic and periventricular areas (Pittock et al., 2006; Wang et al., 2011) and regions of the corpus callosum (He et al., 2011; Yu et al., 2008). Preservation of the brain (specially the BMS) in this condition is noteworthy, since the brainstem is compromised in other neurodegenerative diseases. Thus, NMO constitutes a model of PMS impairment with no BMS affectation (although indirect affectation through other non-motor sites is possible).

1.2.2. PMS affectation and BMS preservation: Acute transverse myelitis

Acute transverse myelitis (ATM) is an etiologically heterogeneous syndrome characterized by focal inflammation of the spinal cord and resultant neurological deficits (i.e., weakness, sensory loss, and autonomic dysfunction) (Borchers & Gershwin, 2012; Harzheim, Schlegel, Urbach, Klockgether, & Schmidt, 2004). It is frequently associated with a variety of immunological mechanisms (e.g., infectious or systemic autoimmune diseases), but its etiology remains unknown in numerous cases, which are classified as idiopathic. The typical symptoms in both NMO and ATM include recurrent, stereotypic, and painful spasms of the limbs, weakness in PMS, and spinal or limb dysaesthesias caused by neck flexion (Wingerchuk, Hogancamp, O'Brien, & Weinshenker, 1999; Wingerchuk & Weinshenker, 2003). Cognitive problems associated with ATM remain unclear. ATM constitutes another model of PMS impairment with no BMS affectation (this time, without subtle brain affectation).

1.2.3. BMS affectation and preserved PMS function: Parkinson's disease

Parkinson's disease (PD) is a neurodegenerative disorder characterized by the loss of voluntary movement control (Helmich, Hallett, Deuschl, Toni, & Bloem, 2012; Liu et al., 2006; Rosin, Topka, & Dichgans, 1997). Flexor muscles of the limbs are more affected in the early stages of the disease (Andrews, Burke, & Lance, 1972; Rodriguez-Oroz et al., 2009) due to a deficiency in nigrostriatal dopamine and subsequent functional impairment of the basal ganglia (BG). Recent studies have highlighted speech and action-language disturbances in PD (Bertella et al., 2002; Cotelli et al., 2007; Ibáñez et al., 2013; Peran et al., 2009). Contrary to NMO and MTA, early Parkinson's disease (EPD) provides a model of preserved PMS with BMS affectation (BG and their fronto-striatal connections).

1.3. Experimental hypotheses

A direct comparison of language-action processing in EPD relative to NMO and ATM may provide critical evidence to disentangle the roles of PMS and BMS in motor-language interactions. To this end, the ACE paradigm may prove particularly useful, as it is sensitive to the involvement of current motor responses linked to action verbs. The evidence thus obtained could be used to test conflicting hypotheses derived from the non-representational and representational embodied views, as follows:

1. *Radical, non-representational embodied hypothesis:* Action-language deficits in neurodegenerative motor disorders have their root in the impairment of ongoing motor performance (e.g., absence of fine hand-motor skills). Thus, action-verb processing deficits would emerge from motor diseases compromising both PMS and BMS.
2. *Lenient, representational embodied hypothesis:* Action-language deficits in motor disorders result from impairment of cortical-subcortical areas involved in action planning and execution. This mainstream view in cognitive neuroscience predicts that action-verb processing deficits would be caused by motor diseases compromising BMS, but not PMS.

In line with previous evidence, different interpretations of the above hypotheses are listed below. The ACE in EPD versus NMO and ATM will differ if motor-language interaction is facilitated by BMS and/or PMS. Specifically:

(a) Hypothesis 1 will be supported if ACE is affected in EPD and preserved in NMO and ATM. ACE deficits would thus be associated with BMS, and not PMS, dysfunction, suggesting that cortico-subcortical areas are more relevant than PMS per se for motor-language interaction.
(b) Hypothesis 2 will be supported if ACE is preserved in EPD and impaired in NMO and ATM. ACE deficits would thus be associated with PMS, but not BMS, impairments, suggesting that peripheral responses themselves are crucial for action-language processing.
(c) A non-excluding combination of hypotheses 1 and 2 will be supported if ACE is affected in all motor conditions (EPD, NMO, and ATM). ACE deficits would thus be explained by both BMS and PMS impairments in different clinical manifestations.
(d) Finally, partial support for hypothesis 1 will be obtained if ACE is affected in NMO but not in ATM. Given that NMO involves diffuse non-motor affectation, ACE deficits could then be explained by the recruitment of affected non-motor brain areas required for motor-language integration.

2. Materials and methods

2.1. Participants

The samples' characteristics are summarized in Table 1. Control participants with a history of alcohol abuse, psychiatric or neurological disorders were excluded. All subjects were native Spanish speakers. They participated voluntarily and signed an informed consent in agreement

Table 1
Main demographic and clinical findings for NMO and ATM patients and their control groups.

	NMO group	Controls for NMO	ATM group	Controls for ATM
Sex, F/M	7/3	7/3	3/7	3/7
Handedness (right/left)	9/1	9/1	10/0	10/0
Age (Mean ± SD)	40.60 ± 12.88	40.70 ± 12.70	44.60 ± 14.74	44.80 ± 13.84
Educational level (years)	13.70 ± 2.31	14.90 ± 2.81	16.00 ± 2.49	16.90 ± 2.47
Disease duration	7.40 ± 4.40	NA	6.80 ± 4.62	NA
Positive for NMO-IgG	100%	NA	0%	NA
EDSS score	2.25 ± 1.01	NA	NA	NA

Abbreviations: NMO, neuromyelitis optica; ATM, acute transverse myelitis; EDSS, Expanded Disability Status Scale; NA, not applicable.

with the Helsinki declaration. All experimental procedures were approved by the INECO Ethics Committee.

2.1.1. NMO group and controls (Experiment 1)

Our first experiment involved a group of 10 patients with NMO and 10 healthy participants. The control group (CG) was closely matched to NMO patients for age, sex, and education. NMO patients were diagnosed according to the recently revised diagnosis criteria proposed by Wingerchuk et al. (2006). See "Participants" in Supplementary Data.

2.1.2. ATM group and controls (Experiment 2)

In our second experiment, we studied 10 ATM patients and 10 healthy subjects. The two groups were matched for sex, age, and years of education. Prior to the study, the ATM participants had all been diagnosed with idiopathic ATM according to the Transverse Myelitis Consortium Working Group (Transverse Myelitis Consortium Working Group, 2002) criteria. Only patients with mild bilateral upper limb weakness were included in the study.

2.1.3. PD group and controls (Experiment 3)

Experiment 3 was conducted with 15 EPD patients and 15 healthy volunteers. The two groups were matched for age, level of education, and proportion of males to females (see Table 2). EPD participants who met UK Parkinson Disease Society Brain Bank criteria (Hughes, Daniel, Kilford, & Lees, 1992) were evaluated using part III of the Unified Parkinson's Disease Rating Scale (UPDRS) and Hoehn and Yahr's (1967) stages I or II. Assessment was conducted during the 'on' state of the medication. See "Participants" in Supplementary Data.

2.2. Neuropsychological assessment

All patients and controls were evaluated with: (1) the Addenbrooke's Cognitive Examination Revised (ACE-R, Torralva et al., 2011) to asses early stages of dementia; (2) the Mini Mental State Examination (MMSE) (Bak et al., 2005; Chade et al., 2008; Reyes et al., 2009), as an additional measure of dementia; (3) the INECO Frontal Screening Battery (IFS, Torralva, Roca, Gleichgerrcht, Lopez, & Manes, 2009), to assess executive functions; (4) the picture version of the Pyramids and Palm Trees Test (PPT, Howard & Patterson, 1992), to assess noun processing; and (5) the picture version of the Kissing and Dancing Test (KDT, Bak & Hodges, 2003), to evaluate semantic representation of verbs. In addition, based on current recommendations to assess executive impairment in NMO and ATM

Table 2
Main demographic and clinical data for EPD patients and their control group.

	EPD group	Controls for EPD
Sex, F/M	15(9/6)	15(10/5)
Handedness (right/left)	(15/0)	(15/0)
Age (Mean ± SD)	62.28 ± 6.54	61.33 ± 8.96
Educational level (years)	12.71 ± 4.13	11.73 ± 4.10
Disease duration	3.06 ± 1.85	NA
Hoehn and Yahr	1.21 ± 0.46	NA
UPDRS motor score	15.42 ± 6.33	NA
Onset disease (right/left)	(12/3)	NA
Levodopa Mg/day (range)	267 ± 57.80 (n = 8; 150–350)	NA
Dopamine agonist Mg/day (range)		NA
Pramipexole	.63±.53 (n = 2; .25–1.0)	
Piribedil	175 ± 35.36 (n = 2; 150–200)	
Other antiparkinsonian drugs, Mg/day (range)		NA
Rasagiline	.68±.23 (n = 3; .25–1.0)	

Abbreviations: EPD, early Parkinson's disease; UPDRS, Unified Parkinson's Disease Rating Scale; NA, not applicable.

(Rogers & Panegyres, 2007), we included the Paced Auditory Serial Addition Test (PASAT-Inter-stimulus time: 3 s; Rao, 1990) and the Symbol Digit Modality Test (SMDT; Smith, 1991). See "Neuropsychological assessment" in Supplementary Data.

2.3. ACE task

We used a paradigm previously reported in studies with healthy volunteers, patients with intracranial recordings and EPD patients (Aravena et al., 2010; Ibáñez et al., 2013). The subjects were comfortably seated behind a desk, facing a computer screen. They listened to 52 sentences conveying open-hand (OH) actions, 52 sentences conveying closed-hand (CH) actions, and 52 neutral (N) sentences that did not convey manual actions. The hand position was specifically indicated by a sentence-final verb. Participants indicated as quickly as possible when they understood each sentence by pressing a button using a pre-assigned hand-shape (open or closed). All responses were performed with the dominant hand. However, both hands were held in the required shape to control for possible bilateral hand interference, given that posture has been

shown to modulate semantic processing (Badets & Pesenti, 2010; A. M. Glenberg, Sato, & Cattaneo, 2008; Linderman, Yanagida, Norma, & Hosaka, 2006; Van Elk, Van Schie, & Bekkering, 2008). Also, to ensure that participants were attending to the stimuli, they were told they would be asked about the content of each sentence-final word at the end of the experiment.

Participants were assigned a hand-shape (closed or open) in each of two counterbalanced blocks: OH response block first or CH response block first. Each list of sentences (OH, CH, and N) was divided into two sub-lists of 26 sentences. One sub-list from each pair was randomly assigned for each participant to a response block (OH and CH responses). Participants completed a five-trial training session to become familiar with the task. Each trial began with an ocular fixation cross appearing at the centre of the monitor 300 ms before the beginning of the sentence and disappearing 800 ms after the response. The inter-stimulus interval was set at 150 ms.

Stimuli were third-person Spanish sentences with a critical verb in the preterit tense (*pretérito indefinido*), located in sentence-final position. The sentence lists used in the compatible and incompatible conditions were controlled for relevant linguistic variables, including transitivity, situation aspect, clause content, final target-word frequency, predictability, prototypicality (how representative of the pertinent hand-shape was the manual action encoded by the sentence), and degree of manual specificity (the manual aperture or closure for each sentence). Note that neutral sentences are more predictable in this paradigm, thus eliciting faster RTs. See Aravena et al. (2010) for details about predictability effects.

Mean sentence duration was 4.57 s (SD = .16 s). Audio files were edited so that each trial was preceded and followed by silence periods of 400 ms and 200 ms, respectively. Mean onset-time of the target verb within the sentences was 4.05 s (*SD* = .06; 2.92 s minimum, 5.64 s maximum). Trials were uniformly distributed over the three sentence conditions in a counterbalanced list, ensuring that the same condition did not appear more than two times consecutively. See Aravena et al. (2010) for more details on stimuli features and validation.

3. Data analysis

We used the same statistical analysis procedure for all clinical conditions (NMO, ATM, and EPD) and control groups (CGs). First, repeated measures of analysis of variance (ANOVA) or X^2 were used for neuropsychological assessment and for the ACE paradigm. In the ACE paradigm, mean RTs were calculated for each subject for each type of trial (compatible, incompatible, and neutral) and each type of sentence (OH, CH, and N). Single trials eliciting RTs above 3.5 SD from the mean were considered outliers and therefore excluded from the analyses. The repeated measures ANOVAs had Group as a between-subjects factor (NMO and CG; ATM and CG; EPD and CG) and Compatibility (compatible, incompatible, and neutral) as a within-subject factor. Sentence type (OH, CH, and N) was introduced in the analyses as an additional factor. Moreover, RTs in ACE were normalized by subtracting the mean RT from the neutral trials from the mean RTs from the compatible and incompatible trials. The N sentences are more predictable and frequent than OH and CH sentences, eliciting shorter RTs (Aravena et al., 2010). If any of the neurological conditions evidence preservation of semantic discrimination (N sentences yielding shorter RTs than OH and CH sentences), then the ACE in motor diseases cannot be explained as a general motor impairment.

Tukey's HSD method was used in the calculation of post hoc contrasts, and individual differences in ACE were also explored. A global score of the ACE was defined by the subtraction of mean RT for the incompatible and compatible conditions. The association of global scores with KDT and age was determined through Spearman's rank correlations in both groups.

4. Results

4.1. Demographic, clinical, and laboratory data

Table 1 summarizes demographic, clinical, and laboratory results for NMO and ATM patients as well as their respective controls. Table 2 offers demographic and clinical data for EPD patients and their control group.

4.2. Neuropsychological assessment

Mean scores of all groups on neuropsychological measures are shown in Table 3. See "Neuropsychological results" in Supplementary Data for further details. Cognitive performance in the NMO group was almost comparable to those of its control group (including verbal processing, as assessed through the KDT); however, NMO patients evinced impaired short-term memory, decreased information processing speed, and partially affected executive functions. The ATM group presented no impairments in any domain. Finally, EPD presented impaired working memory and action-verb processing (KDT).

4.3. The role of PMS and BMS in motor-language processing

4.3.1. Experiment 1: Motor-language processing in NMO

4.3.1.1. ACE is preserved in NMO. No group differences or interactions were observed in NMO [F (2.36) = .38; p = .68] (see Fig. 1A). We found a significant compatibility effect [F (2, 36) = 193.54; p < .0001]. A post hoc comparison (MS = 412.66, df = 36.00) showed that, in both NMO and its CG, incompatible trials elicited longer RTs relative to compatible (p < .001) and neutral (p < .001) trials. No differences between responses to compatible and neutral trials were observed (p = .69). A separate ANOVA for each group confirmed these results (see "ACE effects in NMO and CG" and "Table S1" in Supplementary Data).

4.3.1.2. Subtraction analysis. In order to assess the performance of both groups while controlling for their general differences, neutral RTs were subtracted from compatible and incompatible RTs. A significant effect of compatibility was observed [F (1, 18) = 437.75; p < .0001]. After subtrac-

Table 3
Results of neuropsychological tests in NMO, ATM, and EPD patients.

	NMO Group (n = 10)	Controls for NMO (n = 10)	ATM Group (n = 10)	Controls for ATM (n = 10)	EPD Group (n = 15)	Controls for EPD (n = 15)
MMSE	29.50 (.71)	29.70 (.48)	29.80(.36)	29.90 (.31)	29.13(.99)	29.33 (1.11)
ACE-R	90.10 (4.01)*	95.00 (4.85)	94.00 (2.98)	96.50 (3.30)	91.20 (4.64)	92.53 (6.60)
Ineco frontal screening						
Motor programming	3.00 (.00)	3.00(.00)	2.90(.31)	3.00(.00)	2.60(.63)	2.73(.14)
Conflictive instructions	3.00 (.00)	2.80(.42)	2.90(.31)	3.00(.00)	2.67(.49)	2.73(.12)
Go-No Go	2.80 (.42)	2.50(.53)	3.00(.00)	3.00(.00)	2.73(.46)	2.66(.12)
Digits backward	3.50 (1.18)	4.20(.92)	4.40(.84)	4.60(1.17)	**3.80(.68)***	4.60(.25)
Verbal working memory	2.00 (.00)	2.00(.00)	2.00(.00)	2.00(.00)	1.93(.26)	1.87(.88)
Spatial working memory	3.60 (.52)	3.60(.52)	3.90(.31)	3.60(.51)	3.67(.49)	3.80(.18)
Abstraction capacity	3.00 (.00)	2.90(.32)	3.00 (.00)	3.00(.00)	3.00 (.00)	3.00(.00)
Verbal inhibitory control	**3.90 (.11)****	5.70(.48)	5.50(.70)	5.90(.31)	5.73(.46)	5.73(.12)
IFS total scores	**24.80 (1.40)***	26.70 (2.16)	27.10 (1.72)	28.4(1.42)	26.13 (2.36)	27.13(.66)
Kissing & dancing test	50.80 (1.32)	51.60(.70)	51.40(.84)	51.40(.97)	**44.67(3.22)****	51.33(1.18)
Pyramids & palms trees	51.60 (.52)	51.50(.71)	51.80(.42)	51.80(.42)	–	–
SDMT	**53.10 (14.04)***	68.50(18.36)	62.70(25.21)	64.30(19.34)	–	–
PASAT 3s	**36.70 (9.94)****	50.60(4.74)	50.00(10.13)	51.90(3.35)	–	–

Mean (±SD) in each clinical condition vs. control group.
Abbreviations: NMO, neuromyelitis optica; ATM, acute transverse myelitis; EPD, Early Parkinson's disease; MMSE, Minimental State Examination; ACE-R, Addenbrooke's Cognitive Examination; SDMT, Symbol Digit Modalities test; PASAT, Paced Auditory Serial Addition Test. Significant differences are in bold.
* $P < 0.05$ in each clinical condition vs. control group.
** $P < 0.001$ in each clinical condition vs. control group.

tion (Fig. 1B), no significant differences were found between the two groups [Group X Compatibility Interaction, $F(1, 18) = 1.36$; $p = .25$].

4.3.1.3. Preserved motor response to linguistic variables in NMO participants. A stimulus content analysis was performed to evaluate whether the content of each sentence (independent of any ACE) had a differential effect in both groups. As reported previously (Aravena et al., 2010; Ibáñez et al., 2013), and consistent with preceding research showing strong effects of predictability (Fischler & Bloom, 1979; Kleiman, 1980; Kliegl, Grabner, Rolfs, & Engbert, 2004; Kliegl, Nuthmann, & Engbert, 2006), *N* sentences (which have increased levels of predictability) elicited shorter RTs than ON and CH sentences.

Although overall RT means suggested faster responses in CG (880 ms, SD = 37.78) than in NMO (931 ms, SD = 37.79), the main effect of group was not significant [$F(1, 18) = 2.10$, $p = .16$]. Indeed, we found a strong content effect [$F(2, 36) = 62.62$; $p < .0001$]. Post hoc comparisons (MS = 455.91; df = 36.00) showed that both NMO and CG participants responded faster to *N* sentences than to OH ($p < .001$) and CH ($p < .001$) sentences. No differences between responses to OH and CH were observed ($p = .94$; Fig. 1C). In addition, no interaction was observed between type of sentence and group [$F(2, 36) = .0065$; $p = .99$]. See "Stimulus content analysis" and "Table S2" in Supplementary Data.

4.3.2. Experiment 2: Motor-language processing in ATM

4.3.2.1. ACE is also preserved in ATM. As was the case with NMO, no group differences or interactions were observed between ATM and their controls [$F(2, 36) = .009$; $p = .99$] (see Fig. 1D). A main effect of compatibility effect was found [$F(2, 36) = 91.54$; $p < .0001$]. Post hoc comparisons (MS = 1113.0, df = 36.00) showed that, in both ATM and CG, incompatible trials elicited longer RTs than compatible ($p < .001$) and neutral ($p < .001$) trials. No differences between responses to compatible and neutral trials were observed ($p = .12$). These results were confirmed by separate ANOVAs for each group. See "ACE effects in ATM and CG" and "Table S3" in Supplementary Data.

4.3.2.2. Subtraction analysis. A strong ACE was found [$F(1, 18) = 126.13$; $p < .0001$]. No significant differences were observed between the two groups [Group X Compatibility Interaction, $F(1, 18) = .01$; $p = .91$] (see Fig. 1E).

4.3.2.3. Preserved motor response to linguistic variables in ATM participants. Overall, RTs were numerically similar in both ATM (924 ms, SD = 45.25) and CG (920 ms, SD = 44.09), without statistical differences [$F(1, 18) = .013$; $p = .91$]. There were significant differences in RTs for the effect of stimulus content [$F(2, 36) = 45.21$; $p < .0001$]. Post hoc comparisons over this effect (MS = 881.92, df = 36.00) evidenced faster processing of N sentences relative to OH ($p < .001$) and CH ($p < .001$) sentences. No differences between OH and CH sentences were observed ($p = .65$). Additionally, no interaction between type of sentence and group was observed [$F(2, 36) = .05$; $p = .95$; Fig. 1F]. See, "Stimulus content analysis" and "Table S4", in Supplementary Data.

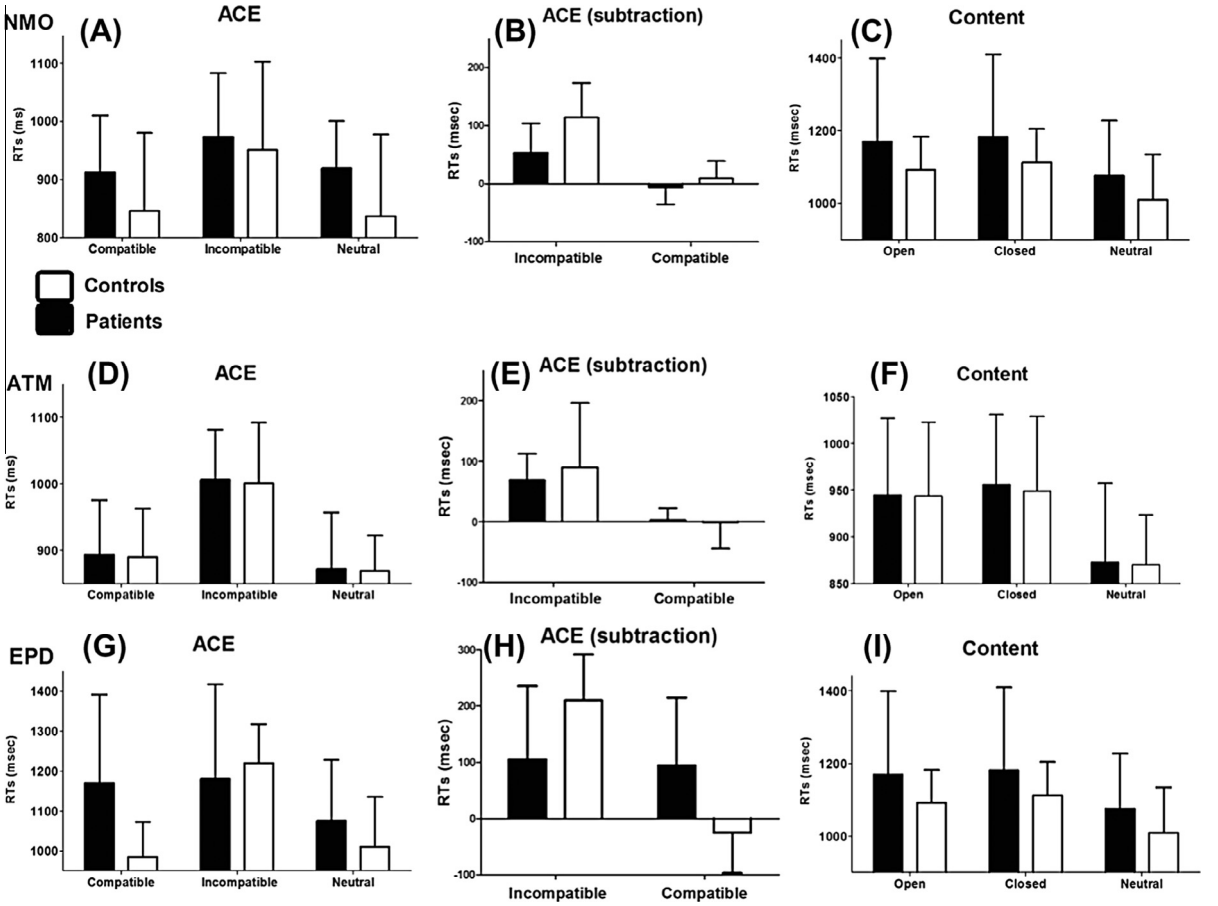

Fig. 1. ACE in NMO, ATM, and EPD. (A). Mean RTs from compatible, incompatible and neutral trials for NMO and CG participants. NMO and CG groups showed a classic ACE (compatible facilitation and incompatible delay of RTs). (B) ACE subtraction. Group comparison of ACE normalized by subtracting mean RT from the neutral trials from the mean RTs from the compatible and incompatible trials. (C) Preserved motor response discrimination for NMO participants. (D) Mean RTs for ATM and CG participants. ATM and CG groups showed an ACE. (E) ACE subtraction. (F) Preserved motor response discrimination for ATM participants. (G) Mean RTs for EPD and CG participants. CG groups showed an ACE, whereas the ACE was absent in EPD participants. (H) ACE subtraction. (I) Preserved motor response discrimination for EPD participants. In all groups, OH and CH sentences yielded longer RTs than N sentences due to a higher frequency and higher cloze probability of the latter. In all panels, the bars depict the SD.

4.3.3. Experiment 3: Motor-language processing in EPD

4.3.3.1. ACE is impaired in EPD. We observed an interaction of Group x Compatibility [F (2, 56) = 31.50, p < .0001] (Fig. 1G). Post hoc comparisons (MS = 3163; df = 83.92) showed an ACE in CG: incompatible trials elicited longer RTs as compared to compatible (p < .001) and neutral (p < .001) trials. Conversely, we found no ACE in EPD patients: here, RTs in neutral trials differed from those of compatible (p < .05) and incompatible (p < .001) trials, but no differences between compatible and incompatible trials were observed (p = .99). A separate ANOVA for each group confirmed these results. See "ACE effects in EDP and CG" and "Table S5" in Supplementary Data.

4.3.3.2. Subtraction analysis. A strong ACE was found after subtraction [F (1, 28) = 906.83; p < .0001] (Fig. 1H). Group differences became larger, as shown by the Group × Compatibility interaction [F (1, 28) = 90.77; p < .0001]. Whereas CG showed a large difference between compatible (M = 24 ms, SD = 25.98) and incompatible (M = 209 ms, SD = 22.47; p < .001) trials, in EPD the means for compatible (M = 94.69 ms, SD = 24.41) and incompatible (M = 104 ms, SD = 26.75; n.s.) trials were quite similar.

4.3.3.3. Preserved motor responses to linguistic variables in EPD. Overall, RTs were numerically faster for CG (1070 ms, SD = 40.9) than EPD participants (1102 ms, SD = 42.00), but the difference was not significant [F (1, 28) = 1.60; p = .20]. Importantly, both EPD and CG participants responded faster to N sentences than to OH and CH sentences [F (2, 56) = 40.04; p < .0001]. Post hoc comparisons (HSD Tukey test, MS = 1944.3, df = 56.00) yielded significant differences of N versus OH (p < .001) and CH (p < .001) sentences. No difference was observed between OH and CH sentences (p = .31) (see Fig. 1I; see also "Stimulus content analysis" and "Table S6" in Supplementary Data). In addition, no interaction was observed between type of sentence and group [F (2, 56) = .20; p = .81].

These results confirm that motor impairment in EPD participants was not so severe as to preclude effects of linguistic variables. Consequently, the ACE deficits in EPD cannot be explained by a general motor or language impairment.

4.4. Correlations

Considering all patient groups and controls, we found a strong association between KDT accuracy and ACE (more KDT accuracy, stronger ACE: $r = .5$; $p < .001$; Fig. 2B). Conversely, no association with age was observed among all participants ($r = -.10$; $p = .54$).

5. Discussion

The present study investigated the role of PMS and BMS in motor-language integration by assessing action-sentence compatibility effects and verbal processing in NMO, ATM, and EPD participants. Interestingly, ACE performance was affected as a result of BMS impairment only (EPD group). To our knowledge, this is the first report testing both the representational and non-representational views of embodiment in the domain of motor diseases.

5.1. Action-verb processing and the PMS

In experiments 1 and 2, ACE performance was similar for patients with PMS impairments triggered by demyelinating-inflammatory syndromes of the spinal cord (NMO and ATM) and their controls: RTs were longer for incompatible than for compatible action sentences. Thus, the ACE task is unlikely to reflect PMS activity. In addition, KDT results indicate adequate action/verb processing in both groups. These findings suggest that action-language processing does not directly depend on PMS activation. Instead, motor-language interaction is most likely subserved by the preserved motor brain areas.

5.2. Action-verb processing and the BMS

Conversely, participants with EPD (Experiment 3) exhibited impaired ACE compared to healthy participants, replicating previous reports (Ibáñez et al., 2013). Likewise, EPD patients were selectively impaired for action/verb processing. These results suggest that the cortico-subcortical motor system is directly involved in motor-language integration. The contrast between experiments 1 and 2, on the one hand, and 3, on the other, indicates that ACE perfor-

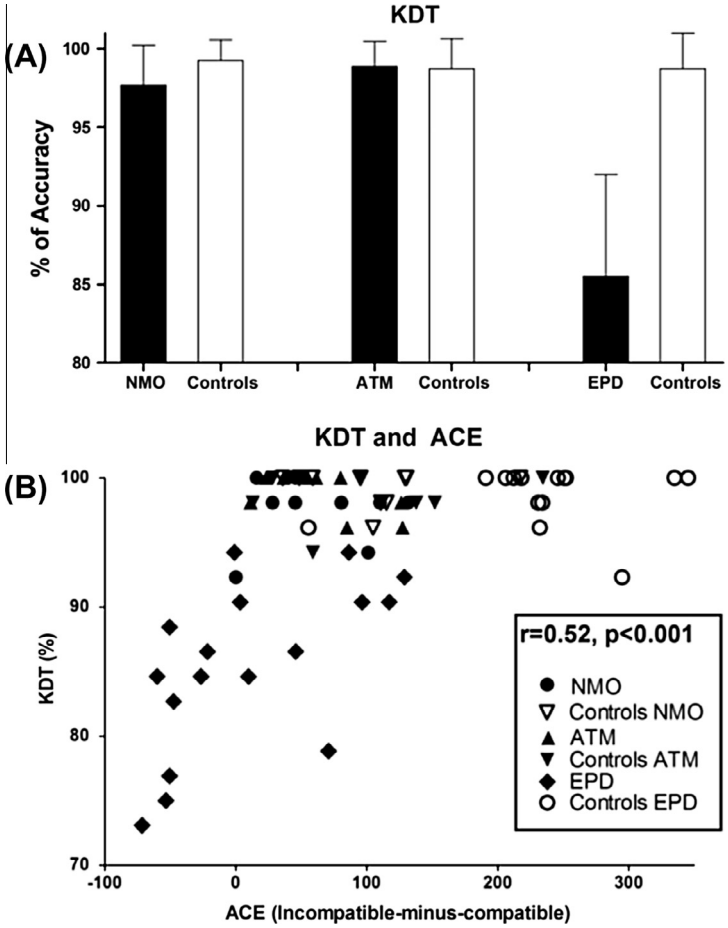

Fig. 2. Verbal processing (KDT) in NMO, ATM, and EPD. (A) KDT scale denotes percentage of correct responses. (B) KDT is associated with the ACE. The ACE is computed as the subtraction between incompatible and compatible trials (the higher the time interval, the stronger the ACE).

mance depends on BMS integrity, and not on peripheral activation. These findings are clearly inconsistent with the non-representational embodied view, which assumes that the PMS is directly involved in motor-cognitive integration. Similarly, the semantic grounding of action words seems to depend on cell assemblies in sensory-motor areas (Kiefer, Sim, Liebich, Hauk, & Tanaka, 2007).

5.3. Testing different ACE hypotheses

Regarding ACE performance, we proposed two main hypotheses about the possible roles of PMS and BMS in motor-language coupling. These, in turn, gave rise to the four interpretations recapitulated below:

(a) H1 ⇒ ACE is affected in EPD and preserved in NMO and ATM.
(b) H2 ⇒ ACE is preserved in EPD and impaired in NMO and ATM.
(c) Partial H1 and H2 ⇒ ACE is affected in EPD, BMO, and ATM.
(d) Partial H1 ⇒ ACE is affected in NMO but not in ATM.

In the present study, EPD patients exhibited impaired ACE as compared with healthy and other neurological (NMO and ATM) participants. This pattern is consistent with postulate (a) and constitutes strong evidence that action/verb processing is embodied but dependent primarily on BMS activation. This representational view of embodiment indicates that action-language coupling depends on a network distributed throughout partially overlapping motor-language areas (Arevalo, Baldo, & Dronkers, 2012).

For its own part, postulate (b) suggests that ACE manifestation depends primarily on peripheral activation, with minor participation of BMS. However, our results rule out this hypothesis by showing quite the opposite effect. This finding speaks against the non-representational embodied view.

In a similar way, postulate (c) posits that the ACE equally depends on both peripheral and cerebral components. However, our results showed that the ACE is preserved in NMO and ATM, but not in EPD, suggesting that it depends specifically on brain motor-language coupling. This finding disconfirms hypothesis (c).

Finally, regarding postulate (d), different studies have revealed early cognitive impairment associated with cortical affectation in NMO (Blanc et al., 2008; He et al., 2011). However, those studies did not explore the motor-language domain. Our results showed that action/verb processing is preserved in these patients. Therefore, slight and diffuse non-motor brain affectation in NMO seems to have no significant impact on motor-language interaction.

Recently, we proposed a hypothetical model of motor-language coupling to understand the specific action-language impairment in EPD (Cardona et al., 2013). We suggest that a distinct BG-thalamocortical circuitry might be involved in the motor-language integration observed in action/verb processing. The proposed model includes two major mechanisms: a motor component (BG-frontal loop), involved in the processing of motor simulation and action patterns in cortical areas; and a semantic component, which would play a major role in the grounding of abstract conceptual knowledge. Behavioral studies on action priming (Helbig, Graf, & Kiefer, 2006; Helbig, Steinwender, Graf, & Kiefer, 2010; Kiefer, Sim, Helbig, & Graf, 2011) and interference effects (Myung, Blumstein, & Sedivy, 2006) also offer convergent evidence for the interplay between action-related and conceptual information. Moreover, converging evidence from PD suggests a more complex relationship between the language and motor systems by showing a bidirectional influence of motor and language areas, including subcortical motor and non-motor regions (Cardona et al., 2013; Ibáñez et al., 2013; Peran et al., 2009). Results from the present study also support this hypothetical model and are in line with previous reports (Glenberg & Kaschak, 2002). In sum, these findings reinforce the notion that BMS activation is a crucial mechanism underlying facilitation in the ACE paradigm.

Furthermore, motor-language integration would involve an internal simulation implicating the brain areas which subserve the execution of a particular action (Garagnani, Wennekers, & Pulvermüller, 2007). According to our findings, the BG would provide a trigger for simulation of action generation and motor habit activation in action language (Alegre, Guridi, & Artieda, 2011; Cardona et al., 2013). Taken together, these findings support the involvement of the motor system in action-word processing, indicating that the semantic grounding of such representations involves an emergent and distributed cortical-subcortical network.

Notice that this model does not (seek to) explain how abstract knowledge is represented. Other models (e.g., Kiefer and Pulvermuller, 2012) have made theoretical efforts to explain the representation of abstract concepts within modality-specific sensory, motor, and emotional brain circuits. The latter proposal is consistent with our notion of a bidirectional coupling between motor and language areas (Aravena et al., 2010; Cardona et al., 2013; Ibáñez et al., 2013). However, unlike our model, it posits that representations are organized in a modality-specific and somatotopic fashion, an assumption that has been recently challenged (Cardona et al., 2013).

5.4. Theoretical implications

The opposition between the non-representational and representational embodied views foregrounds the involvement of corporeal and cerebral motor components in cognitive processes. This debate does not challenge the notion of embodiment. Instead, it concerns the presence or absence of brain-specific involvement in grounded cognition. The non-representational embodiment view states that peripheral sensory organs and effectors are crucial in structuring information flow (Clark, 1997; Gallagher, 2005a, 2005b). Our results rule out the possibility that cognitive processing exclusively depends on the PMS, as proposed by non-representational embodied views. Instead, they support contemporary theoretical perspectives for which cognitive functions are served by distributed and interactive brain networks (Fischer & Zwaan, 2008; Uttal, 2003). Notice, however, that the PMS may indeed play an important role in certain aspects of verbal comprehension, particularly when emotional aspects are involved (Foroni &

Semin, 2009; Havas, Glenberg, Gutowski, Lucarelli, & Davidson, 2010).

Our study lends further support to the dominant embodied view, for which semantic grounding depends on brain motor areas. More generally, another implication of our findings concerns the nature of the perception–action system. It is likely that similar cortical-subcortical circuits play a cardinal role in the initiation of both action/verb processing and movement execution. In neurodegenerative diseases, contextual coupling impairments may reflect damage to these circuits (Ibáñez & Manes, 2012; Ibáñez et al., 2013).

5.5. Limitations and suggestions for further research

This study presents important limitations that should be addressed in future research efforts. First, our relatively small sample size may have influenced the results. Notwithstanding, our data does systematically show that BMS – as opposed to PMS – impairment is directly related with ACE and action/verb processing failures.

Another potential limitation is that motor symptoms can vary in the three neurological conditions considered. However, the level of motor affectation in superior limbs was assessed with the UPDRS (in EPD) and EDSS (in NMO and ATM), and it was also controlled by two neurologists specialized in motor disorders (OG and VS).

Assessment was conducted during the 'on' state of the medication. In this time-period, antiparkinsonian medication temporarily diminishes motor symptomatology in EPD patients, facilitating their participation in the experiment. However, since levodopa has been shown to improve verbal processing in a percentage of PD subjects (Mattis, Tang, Ma, Dhawan, & Eidelberg, 2011), any observed impairment of ACE or verbal processing cannot be explained by medication effects.

Also, our patients were investigated only with routine MRI recordings, as was the case in other cognitive studies on PD (for a recent overview, see Cardona et al., 2013) and NMO (Blanc et al., 2008). Regarding ATM patients, brain MRI abnormality was an exclusion criterion (Borchers & Gershwin, 2012). Further volumetric and fMRI studies may provide additional insights about the relationship between the location of cortical-subcortical involvement, the pattern of cognitive impairment, and the specifics of high-order motor dysfunction in these conditions.

6. Conclusions

The main finding of this study is that patients with PMS impairment (ATM and NMO) showed preserved action-language processing, while those with BMS affectation (EPD) did not. Our results speak against non-representational embodied views as well as completely disembodied theories. On the contrary, they are consistent with an embodied view in which semantic content requires close interaction with brain motor systems. Specifically, the motor system might be intricately involved in language comprehension, with BG circuitry playing an important role in the cognitive loop that enables motor-language coupling.

Acknowledgments

This work was partially supported by grants from CONICET, CONICYT/FONDECYT Regular (1130920), COLCIENCIAS (1115-545-31374, contract: 392), FONCyT-PICT 2012-0412, FONCyT-PICT 2012-1309 and INECO Foundation.

Appendix A. Supplementary material

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.cognition.2014.02.001.

References

Alegre, M., Guridi, J., & Artieda, J. (2011). The mirror system, theory of mind and Parkinson's disease. *Journal of the Neurological Sciences, 310*, 194–196.

Alsmith, A., & de Vignemont, F. (2012). Embodying the mind and representing the body. *Review of Philosophy and Psychology, 3*, 1–13.

Andrews, C. J., Burke, D., & Lance, J. W. (1972). The response to muscle stretch and shortening in Parkinsonian rigidity. *Brain, 95*, 795–812.

Aravena, P., Hurtado, E., Riveros, R., Cardona, J. F., Manes, F., & Ibáñez, A. (2010). Applauding with closed hands: Neural signature of action-sentence compatibility effects. *PLoS ONE, 5*, e11751.

Arevalo, A. L., Baldo, J. V., & Dronkers, N. F. (2012). What do brain lesions tell us about theories of embodied semantics and the human mirror neuron system? *Cortex, 48*, 242–254.

Aziz-Zadeh, L., Wilson, S. M., Rizzolatti, G., & Iacoboni, M. (2006). Congruent embodied representations for visually presented actions and linguistic phrases describing actions. *Current Biology, 16*, 1818–1823.

Badets, A., & Pesenti, M. (2010). Creating number semantics through finger movement perception. *Cognition, 115*, 46–53.

Bak, T. H., & Hodges, J. R. (2003). Kissing and dancing – A test to distinguish the lexical and conceptual contributions to noun/verb and action/object dissociation. Preliminary results in patients with frontotemporal dementia. *Journal of Neurolinguistics, 16*, 169–181.

Bak, T. H., Rogers, T. T., Crawford, L. M., Hearn, V. C., Mathuranath, P. S., & Hodges, J. R. (2005). Cognitive bedside assessment in atypical parkinsonian syndromes. *Journal of Neurology, Neurosurgery & Psychiatry, 76*, 420–422.

Ballerini, M., Cabibbo, N., Candelier, R., Cavagna, A., Cisbani, E., Giardina, I., et al. (2008). Interaction ruling animal collective behavior depends on topological rather than metric distance: Evidence from a field study. *Proceedings of the National Academy of Sciences of the United State of America, 105*(4), 1232–1237.

Barrett, L. (2011). *Beyond the brain: How body and environment shape animal and human minds*. Princeton, NJ: Princeton University Press.

Barwise, J., & Perry, J. (1983). *Situations and attitudes*. Cambridge, Mass.: MIT Press.

Beer, R. D. (2003). *The dynamics of active categorical perception in an evolved model agent* (Vol. 11, pp. 209–243).

Bertella, L., Albani, G., Greco, E., Priano, L., Mauro, A., Marchi, S., et al. (2002). Noun verb dissociation in Parkinson's disease. *Brain and Cognition, 48*, 277–280.

Bingham, Geoffrey P. (1988). Task-specific devices and the perceptual bottleneck. *Human Movement Science, 7*(24), 225–264.

Blanc, F., Zephir, H., Lebrun, C., Labauge, P., Castelnovo, G., Fleury, M., et al. (2008). Cognitive functions in neuromyelitis optica. *Archives of Neurology, 65*, 84–88.

Borchers, A. T., & Gershwin, M. E. (2012). Transverse myelitis. *Autoimmunity Reviews, 11*, 231–248.

Borreggine, K. L., & Kaschak, M. P. (2006). The action-sentense compatibility effect: Iťs all in the timing. *Cognitive Sciences, 30*, 1097–1112.

Brooks, R. A. (1999). *Cambrian intelligence: The early history of the new AI*. Cambridge, Mass.: MIT Press.

Calvo, P., & Gomila, A. (2008). *Handbook of cognitive science: An embodied approach*. Amsterdam: Elsevier Science Ltd..

Cardona, J. F., Gershanik, O., Gelormini-Lezama, C., Houck, A. L., Cardona, S., Kargieman, L. et al. (2013). Action-verb processing in Parkinson's disease: new pathways for motor-language coupling. *Brain Structure and Function*.

Chade, A., Roca, M., Torralva, T., Gleichgerrcht, E., Fabbro, N., Gómez-Arévalo, G., et al. (2008). Detecting cognitive impairments in patients with Parkinson's disease using a brief cognitive screening tool: Addenbrooke's Cognitive Examination (ACE). *Dementia & Neuropsychologia, 2*, 197–200.

Clark, A. (1997). *Being there: Putting brain, body, and world together again*. Cambridge, Mass: MIT Press.

Cotelli, M., Borroni, B., Manenti, R., Zanetti, M., Arevalo, A., Cappa, S. F., et al. (2007). Action and object naming in Parkinson's disease without dementia. *European Journal of Neurology, 14*, 632–637.

De Vega, M., Moreno, V., & Castillo, D. (2013). The comprehension of action-related sentences may cause interference rather than facilitation on matching actions. *Psychological Research, 77*, 20–30.

De Vega, M., & Urrutia, M. (2011). Counterfactual sentences may activate motoric processes. An action-sentence compatibility effect study. *Journal of Cognitive Psychology, 23*, 962–973.

Fink, P. W., Foo, P. S., & Warren, W. H. (2009). Catching fly balls in virtual reality: A critical test of the outfielder problem. *Journal of Vision, 9*(13), pp. 14 1–8.

Fischer, M. H., & Zwaan, R. A. (2008). Embodied language: A review of the role of the motor system in language comprehension. *Quarterly Journal of Experimental Psychology, 61*, 825–850.

Fischler, I., & Bloom, P. A. (1979). Automatic and attentional processes in the effects of sentence contexts on word recognition. *Journal of Verbal Learning and Verbal Behavior, 18*, 1–20.

Foroni, F., & Semin, G. R. (2009). Language that puts you in touch with your bodily feelings: The multimodal responsiveness of affective expressions. *Psychological Science, 20*, 974–980.

Gallagher, S. (2005a). Dynamic models of body schematic processes. In De Preester, H. & Knockaert, V. (Eds.), *Body image and body schema: Interdisciplinary perspectives on the body* (pp. 233–250). Amsterdam: John Benjamins.

Gallagher, S. (2005b). *How the body shapes the mind*. Oxford: Clarendon Press.

Gallese, V., & Lakoff, G. (2005). The Brain's concepts: The role of the Sensory-motor system in conceptual knowledge. *Cognitive Neuropsychology, 22*, 455–479.

Gallese, V., & Sinigaglia, C. (2011). What is so special about embodied simulation? *Trends in Cognitive Sciences, 15*, 512–519.

Garagnani, M., Wennekers, T., & Pulvermüller, F. (2007). A neuronal model of the language cortex. *Neurocomputing, 70*, 1914–1919.

Glenberg, A. M., & Kaschak, M. P. (2002). Grounding language in action. *Psychonomic Bulletin & Review, 9*, 558–565.

Glenberg, A. M., Sato, M., & Cattaneo, L. (2008). Use-induced motor plasticity affects the processing of abstract and concrete language. *Current Biology, 18*(7), R290–291.

Harzheim, M., Schlegel, U., Urbach, H., Klockgether, T., & Schmidt, S. (2004). Discriminatory features of acute transverse myelitis: A retrospective analysis of 45 patients. *Journal of the Neurological Sciences, 217*(2), 217–223.

Hauk, O., Johnsrude, I., & Pulvermüller, F. (2004). Somatotopic representation of action words in human motor and premotor cortex. *Neuron, 41*, 301–307.

Havas, D. A., Glenberg, A. M., Gutowski, K. A., Lucarelli, M. J., & Davidson, R. J. (2010). Cosmetic use of botulinum toxin-a affects processing of emotional language. *Psychological Science, 21*, 895–900.

He, D., Wu, Q., Chen, X., Zhao, D., Gong, Q., & Zhou, H. (2011). Cognitive impairment and whole brain diffusion in patients with neuromyelitis optica after acute relapse. *Brain and Cognition, 77*, 80–88.

Helbig, H. B., Graf, M., & Kiefer, M. (2006). The role of action representations in visual object recognition. *Experimental Brain Research, 174*(2), 221–228. http://dx.doi.org/10.1007/s00221-006-0443-5.

Helbig, H. B., Steinwender, J., Graf, M., & Kiefer, M. (2010). Action observation can prime visual object recognition. *Experimental Brain Research, 200*(3–4), 251–258. http://dx.doi.org/10.1007/s00221-009-1953-8.

Helmich, R. C., Hallett, M., Deuschl, G., Toni, I., & Bloem, B. R. (2012). Cerebral causes and consequences of parkinsonian resting tremor: A tale of two circuits? *Brain, 135*, 3206–3226.

Hoehn, M. M., & Yahr, M. D. (1967). Parkinsonism: Onset, progression and mortality. *Neurology, 17*, 427–442.

Howard, D., & Patterson, K. (1992). *Pyramids and palm trees: A test of semantic access from pictures and words*. Bury, St. Edmuds: Suffolk.

Hughes, A. J., Daniel, S. E., Kilford, L., & Lees, A. J. (1992). Accuracy of clinical diagnosis of idiopathic Parkinson's disease: A clinico-pathological study of 100 cases. *Journal of Neurology, Neurosurgery & Psychiatry, 55*, 181–184.

Ibáñez, A., Cardona, J. F., Dos Santos, Y. V., Blenkmann, A., Aravena, P., Roca, M., et al. (2013). Motor-language coupling: Direct evidence from early Parkinson's disease and intracranial cortical recordings. *Cortex, 49*, 968–984.

Ibáñez, A., & Manes, F. (2012). Contextual social cognition and the behavioral variant of frontotemporal dementia. *Neurology, 78*, 1354–1362.

Kiefer, M., & Pulvermuller, F. (2012). Conceptual representations in mind and brain: Theoretical developments, current evidence and future directions. *Cortex, 48*(7), 805–825.

Kiefer, M., Sim, E. J., Helbig, H., & Graf, M. (2011). Tracking the time course of action priming on object recognition: Evidence for fast and slow influences of action on perception. *Journal of Cognitive Neuroscience, 23*(8), 1864–1874. http://dx.doi.org/10.1162/jocn.2010.21543.

Kiefer, M., Sim, E. J., Liebich, S., Hauk, O., & Tanaka, J. (2007). Experience-dependent plasticity of conceptual representations in human sensory-motor areas. *Journal of Cognitive Neuroscience, 19*(3), 525–542. http://dx.doi.org/10.1162/jocn.2007.19.3.525.

Kleiman, G. M. (1980). Sentence frame contexts and lexical decisions: Sentence-acceptability and word-relatedness effects. *Memory & Cognition, 8*, 336–344.

Kliegl, R., Grabner, E., Rolfs, M., & Engbert, R. (2004). Length, frequency, and predictability effects of words on eye movements in reading. *European Journal of Cognitive Psychology, 16*, 262–284.

Kliegl, R., Nuthmann, A., & Engbert, R. (2006). Tracking the mind during reading: the influence of past, present, and future words on fixation durations. *Journal of Experimental Psychology: General, 135*, 12–35.

Lin, F., Yu, C., Jiang, T., Li, K., & Chan, P. (2007). Diffusion tensor tractography-based group mapping of the pyramidal tract in relapsing-remitting multiple sclerosis patients. *American Journal of Neuroradiology, 28*, 278–282.

Linderman, W., Yanagida, Y., Norma, H., & Hosaka, K. (2006). Wereable vibrotactile systems for virtual contact and information display. *Virtual Reality, 9*, 203–213.

Liu, W., McIntire, K., Kim, S. H., Zhang, J., Dascalos, S., Lyons, K. E., et al. (2006). Bilateral subthalamic stimulation improves gait initiation in patients with Parkinson's disease. *Gait & Posture, 23*, 492–498.

Mattis, P. J., Tang, C. C., Ma, Y., Dhawan, V., & Eidelberg, D. (2011). Network correlates of the cognitive response to levodopa in Parkinson disease. *Neurology, 77*, 858–865.

McBeath, M. K., Shaffer, D. M., & Kaiser, M. K. (1995). How baseball outfielders determine where to run to catch fly balls. *Science, 268*(5210), 569–573.

Meteyard, L., Cuadrado, S. R., Bahrami, B., & Vigliocco, G. (2012). Coming of age: A review of embodiment and the neuroscience of semantics. *Cortex, 48*, 788–804.

Myung, J. Y., Blumstein, S. E., & Sedivy, J. C. (2006). Playing on the typewriter, typing on the piano: Manipulation knowledge of objects. *Cognition, 98*(3), 223–243.

Peran, P., Cardebat, D., Cherubini, A., Piras, F., Luccichenti, G., Peppe, A., et al. (2009). Object naming and action-verb generation in Parkinson's disease: A fMRI study. *Cortex, 45*, 960–971.

Pfeifer, Rolf., Bongard, Josh., & Grand, Simon. (2007). *How the body shapes the way we think: A new view of intelligence*. Cambridge, Mass.: MIT Press.

Pfeifer, Rolf, & Scheier, Christian (1999). *Understanding intelligence*. Cambridge, Mass: MIT Press.

Pittock, S. J., Weinshenker, B. G., Lucchinetti, C. F., Wingerchuk, D. M., Corboy, J. R., & Lennon, V. A. (2006). Neuromyelitis optica brain lesions localized at sites of high aquaporin 4 expression. *Archives of Neurology, 63*(7), 964–968.

Pulvermüller, F. (2005). Brain mechanisms linking language and action. *Nature Reviews Neuroscience, 6*, 576–582.

Rao, S. M. (1990). *A manual for brief, repeatable battery of neuropsychological tests in multiple sclerosis*. Milwaukee, WI: Section of Neuropsychology, Medical College of Wisconsin.

Reyes, M. A., Perez-Lloret, S., Roldan, G. E., Martin, M. E., Leiguarda, R., & Merello, M. (2009). Addenbrooke's Cognitive Examination validation in Parkinson's disease. *European Journal of Neurology, 16*, 142–147.

Reynolds, C. W. (1987). Flocks, herds, and schools: A distributed behavioral model. *Computer Graphics, 21*(4), 25–34.

Rodriguez-Oroz, M. C., Jahanshahi, M., Krack, P., Litvan, I., Macias, R., Bezard, E., et al. (2009). Initial clinical manifestations of Parkinson's disease: features and pathophysiological mechanisms. *The Lancet Neurology, 8*, 1128–1139.

Rogers, J. M., & Panegyres, P. K. (2007). Cognitive impairment in multiple sclerosis: Evidence-based analysis and recommendations. *Journal of Clinical Neuroscience, 14*, 919–927.

Rosin, R., Topka, H., & Dichgans, J. (1997). Gait initiation in Parkinson's disease. *Movement Disorders, 12*, 682–690.
Smith, A. (1991). *Symbol digit modalities test*. Los Angeles, CA: Western Psychological Services.
Tettamanti, M., Buccino, G., Saccuman, M. C., Gallese, V., Danna, M., Scifo, P., et al. (2005). Listening to action-related sentences activates fronto-parietal motor circuits. *Journal of Cognitive Neuroscience, 17*, 273–281.
Thelen, E., Schoner, G., Scheier, C., & Smith, L. B. (2001). The dynamics of embodiment: A field theory of infant perseverative reaching. *Journal of Behavioral and Brain Science, 24*(1), 1–34.
Torralva, T., Roca, M., Gleichgerrcht, E., Bonifacio, A., Raimondi, C., & Manes, F. (2011). Validation of the Spanish version of the addenbrooke's cognitive examination-revised (ACE-R). *Neurologia, 26*, 351–356.
Torralva, T., Roca, M., Gleichgerrcht, E., Lopez, P., & Manes, F. (2009). INECO Frontal Screening (IFS): A brief, sensitive, and specific tool to assess executive functions in dementia. *Journal of the International Neuropsychological Society, 15*, 777–786.
Transverse Myelitis Consortium Working Group (2002). Proposed diagnostic criteria and nosology of acute transverse myelitis. *Neurology, 59*, 499–505.
Uttal, W. R. (2003). *The new phrenology. The limits of localizing cognitive process in the brain*. Cambridge, MA: MIT Press.
Van Elk, M., Van Schie, H. T., & Bekkering, H. (2008). Semantics in action: An electrophysiological study on the use of semantic knowledge for action. *Journal of Physiology – Paris, 102*, 95–100.
Van Gelder, T. (1995). The distinction between mind and cognition. In Houng, Y. H. & Ho, J. C. (Eds.), *Mind and cognition* (pp. 57–81). Taipei: Academia Sinica.
Wang, F., Liu, Y., Duan, Y., & Li, K. (2011). Brain MRI abnormalities in neuromyelitis optica. *European Journal of Radiology, 80*, 445–449.
Willems, R. M., & Francken, J. C. (2012). Embodied cognition: Taking the next step. *Frontiers in Psychology, 3*, 582.
Wilson, M. (2002). Six views of embodied cognition. *Psychonomic Bulletin & Review, 9*, 625–636.
Wilson, A. D., & Golonka, S. (2013). Embodied cognition is not what you think it is. *Frontiers in Psychology, 4*, 58.
Wingerchuk, D. M., Hogancamp, W. F., O'Brien, P. C., & Weinshenker, B. G. (1999). The clinical course of neuromyelitis optica (Devic's syndrome). *Neurology, 53*, 1107–1114.
Wingerchuk, D. M., Lennon, V. A., Pittock, S. J., Lucchinetti, C. F., & Weinshenker, B. G. (2006). Revised diagnostic criteria for neuromyelitis optica. *Neurology, 66*, 1485–1489.
Wingerchuk, D. M., & Weinshenker, B. G. (2003). Neuromyelitis optica: Clinical predictors of a relapsing course and survival. *Neurology, 60*, 848–853.
Yu, C., Lin, F., Li, K., Jiang, T., Qin, W., Sun, H., et al. (2008). Pathogenesis of normal-appearing white matter damage in neuromyelitis optica: Diffusion-tensor MR imaging. *Radiology, 246*, 222–228.