COGNITIVE NEUROPSYCHOLOGY, 2015
VOL. 32, NOS. 7–8, 427–430
http://dx.doi.org/10.1080/02643294.2016.1154832

Routledge
Taylor & Francis Group

COMMENTARY

Simulation of impairment: Commentary on Nickels, Rapp, and Kohnen (2015)

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ABSTRACT

Nickels, L., Rapp, B., and Kohnen, S. (2015. Challenges in the use of treatment to investigate cognition. *Cognitive Neuropsychology, 32,* 91–103) argue that impairment and treatment may be used to test computational models of cognition. They state that, contrary to their view, the authors of the WEAVER++ model of spoken word production have explicitly rejected simulation of impairment [i.e., Levelt, W. J. M., Roelofs, A., & Meyer, A. S. (1999b). Multiple perspectives on word production. *Behavioral and Brain Sciences, 22,* 61–69]. Here, I argue that this incorrectly characterizes the position of Levelt et al. Moreover, I further clarify this position, which holds that simulation of impairment requires both a theory of the intact system and assumptions about the underlying deficit, which is a widely accepted view. To demonstrate this position, I outline the approach taken in WEAVER++ simulations of aphasic performance reported in Roelofs, A. (2014. A dorsal-pathway account of aphasic language production: The WEAVER++/ARC model. *Cortex, 59,* 33–48). These simulations not only prove that the developers of WEAVER++ endorse simulation of impairment, but also highlight the importance of integrating psycholinguistic, functional neuroimaging, and tractographic evidence in testing models of impaired performance.

ARTICLE HISTORY
Received 16 December 2015
Accepted 6 February 2016

KEYWORDS
Aphasia; cognition; impairment; model; simulation; theory

In a recent article, Nickels, Rapp, and Kohnen (2015) make the case that not only impairment but also treatment may be used to test computational models of cognition. In reviewing models in the literature, they state that

> it is worth noting that authors of some influential computational models explicitly reject the simulation of impairment. For example, regarding the WEAVER++ implementation of Levelt, Roelofs, and Meyer's influential theory of spoken word production (Levelt, Roelofs, & Meyer, 1999a, 1999b), Levelt et al. (1999b, p. 68) state that "we feel it as [*sic*] a bridge too far to expect a patient's behaviour to conform to our theory", continuing "there is little a priori reason to suppose that an impaired system performs according to an intact theory" (although see Roelofs, 2004). (Nickels et al., 2015, p. 93)

The aim of this commentary is to make clear that the developers of WEAVER++ do not reject simulation of impairment but instead argued for a particular approach (in response to Ferrand, 1999). I show that the quotation from Levelt et al. (1999b) misses a crucial sentence. In particular, Levelt et al. argued that application of a theory of normal performance

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to impairments requires making additional assumptions about the nature of the underlying deficit, which is a widely held view. In a number of articles (i.e., Roelofs, 2003, 2004, 2014), such additional assumptions were made, and the model was applied to impaired performance through simulation. For example, in Roelofs (2014), I reported the results of computer simulations showing that by assuming particular underlying deficits, WEAVER++ successfully accounts for the typical patterns of impaired and spared language performance associated with classic acute-onset and progressive aphasias. Moreover, the model was shown to account for evidence on impaired production performance as a consequence of damage to certain white-matter pathways. This simulation work proves that the developers of WEAVER++ endorse the application of their model to impairment. In the remainder, I first clarify the real position of Levelt et al. and then illustrate their view by briefly outlining the approach to simulation of impairment adopted in Roelofs (2014), which complements the approach proposed by Nickels et al. (2015).

To start with the quoted sentences, it is important to note that the quotation from Levelt et al. (1999b) conveys an incorrect message because a final crucial sentence is missing. Following the quoted sentences (which were surrounding a response by Levelt et al., 1999b, to Ferrand, 1999, concerning a specific anomic patient), Levelt et al. ended by saying that "The real theoretical challenge is to create and test a theory of the impaired system (as is done, e.g., by Dell, Schwartz, Martin, Saffran, & Gagnon, 1997)" (p. 68). In accounting for aphasic language performance, Dell et al. (1997) explicitly distinguished between a theory of the intact system and assumptions about the underlying deficit. Their theory of normal word production assumed that lexical access is a two-step process (i.e., the first step involving a mapping between semantic features and lemmas, and the second step mapping between lemmas and phonemes) including both forward and backward spreading of activation (i.e., interactivity). In creating a theory of the impaired system, Dell et al. made three additional assumptions, namely that brain damage may reduce connection weights (i.e., reduce spreading rate) between network levels, that damage may reduce representational integrity (i.e., increase decay rate), and that these variations in connection weight and decay rate are global (i.e., the decay increase is the same for semantic features, lemmas, and phonemes, and the weight reduction holds for all network connections). This model of the impaired system is referred to as the weight-decay model.

It is important to note that, according to this view, theories of normal word production and assumptions about the underlying deficits are to some extent independent. For example, Foygel and Dell (2000) proposed the same theory of normal word production as Dell et al. (1997) but made different assumptions about the underlying deficit, referred to as the semantic–phonological model. In creating this new model, Foygel and Dell dropped the globality assumption and instead assumed that damage may be different for the two steps of lexical access. In particular, they assumed that damage may lead to a reduced spreading rate between semantic features and lemmas, between lemmas and phonemes, or both. The theories about impaired performance of Dell et al. and Foygel and Dell corroborate the view of Levelt et al. (1999b) that application of a theory of normal word production (which does not differ between Dell et al., 1997, and Foygel & Dell, 2000) to impaired performance requires making additional assumptions about the underlying deficit (these assumptions differ between Dell et al., 1997, and Foygel & Dell, 2000). Note that the theories of the impaired system included the theory of the intact system as a proper part. By dropping the globality assumption, the semantic–

phonological model of Foygel and Dell could simulate the impaired performance of particular patients that could not be simulated by the weight-decay model of Dell et al. Instead of assigning the failure to simulate these patient data to the theory of the intact system of Dell et al. (as Ferrand, 1999, did regarding Levelt et al., 1999a), Foygel and Dell adopted different assumptions about the underlying deficit. This is exactly the point that Levelt et al. tried to make in response to Ferrand (1999): Applying a theory of the intact system to impaired performance requires making assumptions about the underlying deficit, and a failure to account for patient data may therefore be due to the specific assumptions made about the deficit rather than due to the theory of the intact system.

In a recent study (i.e., Roelofs, 2014), I followed the approach to the simulation of impaired performance endorsed by Levelt et al. (1999b) and exemplified by the work of Dell et al. (1997). The starting point for the simulations was a computationally implemented psycholinguistic model of the functional processes underlying spoken word production, comprehension, and repetition, namely WEAVER++, which accounts for a wide range of behavioural findings concerning normal performance. In addition, an extensive meta-analysis of neuroimaging studies was used to localize the functional processes assumed by the psycholinguistic model (i.e., WEAVER++) to grey-matter areas of the human brain. Moreover, functional processes were related to white-matter connections in the human brain by using tractographic evidence. Furthermore, lesion–symptom analyses were used to relate anatomical evidence concerning damaged brain areas and connections in patients to characteristics of their impaired language performance. In particular, brain damage was related to behavioural symptoms in classic acute-onset aphasia types, including Broca's, Wernicke's, conduction, transcortical motor, transcortical sensory, and mixed transcortical aphasia. Also, evidence about the locus of brain atrophy in neurodegenerative disease was used in simulations of impaired language performance in semantic dementia, which is a type of primary progressive aphasia caused by degeneration of the anterior temporal lobes. Finally, specific assumptions were made about the exact functional nature of the underlying deficits caused by brain damage. In particular, damage severity was simulated by manipulating connection weights or decay rate at specific network loci, following Foygel and Dell (2000). The results of the computer simulations revealed that the model successfully accounts, with a broad stroke, for the empirically observed patterns of impaired and spared production, comprehension, and repetition performance. In summary, the WEAVER++ simulations of impaired performance employed both the functional assumptions in the model concerning the intact system and additional assumptions about corresponding grey- and white-matter substrates and lesion–symptom relationships.

This description of the simulation approach adopted in Roelofs (2014) should make clear that the simulation of impairment did not involve a direct application of an intact WEAVER++ model to impaired performance. Rather, the intact model was extended by implementing theoretical assumptions about the deficit, exactly in line with the approach to impaired performance that Levelt et al. (1999b) argued for. To make explicit that the model of impaired performance included several additional assumptions, the acronym ARC was added to the name of the model (yielding WEAVER++/ARC), which served to highlight a new central claim in the model about the role of the arcuate fasciculus in accounting for aphasic performance (i.e., ARC stands for arcuate repetition and conversation). The simulations demonstrate the importance of integrating evidence from psycholinguistic, functional neuroimaging, and tractographic studies in testing a computational model of impaired performance (cf. Shallice & Cooper, 2011).

To summarize, I have shown that the developers of WEAVER++ do not reject simulation of impairment but instead argued for a particular approach. Moreover, I have demonstrated this approach by outlining WEAVER++ simulations of aphasic language performance. These simulations not only prove that the developers of WEAVER++ endorse simulation of impairment, but also highlight the importance of integrating psycholinguistic, functional neuroimaging, and tractographic evidence in testing models of impaired performance.

Disclosure statement

No potential conflict of interest was reported by the authors.

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