Language and the brain - Michael T. Ullman

1) Testing for domain-specificity and domain-generality

The issue of *domain-specificity* is generally examined in the same way as is separability – namely, by testing for double dissociations. For example, one might show double dissociations between grammar and movement, between grammar and attention, between grammar and ... the list goes on. As you can imagine, it is very difficult if not impossible to demonstrate domain-specificity: How can we test every possible function, with every possible task? Thus one should be very wary of any claims of domain-specificity, and check carefully the basis on which they are made. However, domain-generality can *also* be difficult to show, for the same reasons as are described in Box 7.1 regarding a lack of dissociations: That is, a null result (i.e., no dissociation) is always potentially problematic. Nevertheless, one can provide relatively clear evidence for shared biocognitive substrates between particular language and non-language functions, such as grammar and motor function, by demonstrating *associations* between these functions, moreover with concomitant *dissociations* between these and *other* language and non-language functions. For example, the existence of common neurocognitive substrates for grammar and motor function would be suggested if the two pattern together in their brain activity, their impairment to brain damage, their reaction to drugs, and so on, while they both show different patterns across the board from lexical or conceptual-semantic functions. So, although both domain-specificity and broad domain-generality are difficult to demonstrate, narrower "domain-commonality" between particular language and non-language functions is feasible to test for.

2) A single-mechanism model of regular/irregular morphology

Double dissociations between regular and irregular morphological forms have generally posed a problem for single-mechanism language models. However, a relatively recent model has tried to overcome this limitation (Joanisse & Seidenberg, 1999). The model contains distinct representations for semantics and for input and output phonology, each being subserved by a separate set of units. These units and the pathways between them are assumed to be neuroanatomically distinct, and can therefore be lesioned independently. Although the model claims distinct representations and pathways, it is a single-mechansism model in that it assumes a uniformity of processing mechanisms: all representations and pathways underlie the computation of both regular and irregular morphological forms. It is posited that the inconsistent phonological patterns of irregulars result in their computation relying more on semantics than on phonology. Regulars, in contrast, do not show this bias, and novel verbs actually show the opposite pattern, relying for their computation on phonology but not on semantics.

Simulations of damage to the semantic representation led to worse performance producing irregular than regular or novel past tenses. Simulations of damage to output phonology led to worse performance producing novel than regular and irregular past tenses, but no difference between regulars and irregulars. So, the model revealed double dissociations between irregular and novel verbs but, crucially, not between irregular and regular forms even from lesions to output phonology.

The predictions of this model, and specifically the results from this simulation, do not seem to fit important empirical data, including several findings reported in the text of this chapter: the pattern of worse performance at regular than irregular forms in patients with frontal or basal ganglia insults, even when controlling for a variety of factors; the pattern of activation in Broca's area for regular as compared to irregular forms; and the spatio-temporal dynamics of irregulars and regulars, which fits closely with independently-identified processes in a dual-system model. Additionally, recent studies have reported that irregular impairments can be found in the absence of semantic deficits, and conversely, that semantic deficits do not necessarily lead to irregular impairments, contrary to the predictions of the single-mechanism model described above (Miozzo, 2003; Tyler, 2004).