BEHAVIORAL AND BRAIN SCIENCES (1999) **22,** 571–575
Printed in the United States of America

Continuing Commentary

Commentary on **Ralph-Axel Müller (1996). Innateness, autonomy, universality? Neurobiological approaches to language. BBS 19:611–675.**

Abstract of the original article: The concepts of the innateness, universality, species-specificity, and autonomy of the human language capacity have had an extreme impact on the psycholinguistic debate for over thirty years. These concepts are evaluated from several neurobiological perspectives, with an emphasis on the emergence of language and its decay due to brain lesion and progressive brain disease.

Evidence of perceptuomotor homologies and preadaptations for human language in nonhuman primates suggests a gradual emergence of language during hominid evolution. Regarding ontogeny, the innate component of language capacity is likely to be polygenic and shared with other developmental domains. Dissociations between verbal and nonverbal development are probably rooted in the perceptuomotor specializations of neural substrates rather than the autonomy of a grammar module. Aphasiological data often assumed to suggest modular linguistic subsystems can be accounted for in terms of a neurofunctional model incorporating perceptuomotor-based regional specializations and distributivity of representations. Thus, dissociations between grammatical functors and content words are owing to different conditions of acquisition and resulting differences in neural representation. Human brains are characterized by multifactorial interindividual variability, and strict universality of functional organization is biologically unrealistic.

A theoretical alternative is proposed according to which (1) linguistic specialization of brain areas is due to epigenetic and probabilistic maturational events, not to genetic "hard-wiring," and (2) linguistic knowledge is neurally represented in distributed cell assemblies whose topography reflects the perceptuomotor modalities involved in the acquisition and use of a given item of knowledge.

Can current methods of pathonormal inference tell us anything about modularity?

David W. Gow[a] and Philip C. Rodkin[b]

[a]*Neuropsychology Laboratory, Massachusetts General Hospital, Boston, MA 02114;* [b]*Department of Psychology: Social and Health Sciences, Duke University, Durham, NC 27708.* **gow@helix.mgh.harvard.edu**
pcr@email.unc.edu

Abstract: This commentary on Müller's target article is designed to help bridge the gap between method and theory in neurobiological approaches to language. We focus on pathonormal inference as reflective of broader conceptual issues concerning universality and modularity. Our commentary directs attention to how current methodological and analytic procedures are less than optimal and minimize possibilities for reaching theoretical consensus. We make suggestions about alternative analytic strategies that provide a direction for future scientific progress.

A tension exists in aphasiology between the richness of linguistic theory and the neuropsychological methods available to assess, correct, and revise its central tenets. Müller (1996) suggests that the standard model of pathonormal inference in aphasiology and cognitive neuropsychology presupposes that all humans have the same functional organization of language (universality) and entails that language processes show modular organization. The use of case studies presupposes universality because it depends on the assumption that the functional organization in one individual is representative of other individuals. Müller also implies that the assumptions of subtraction and transparency of processing in pathonormal inference set an unacceptably low standard for identifying instances of dissociation, given the typically graded and noncircumscribed nature of demonstrated behavioral and physiological deficits.

These concerns have critical implications for the practice of neuropsychology. The two main analytic strategies available to neuropsychologists are to rely on very select case studies and to perform group studies of patients with similar but nonidentical deficits. [See also *BBS* multiple book review of Shallice's *From Neuropsychology to Mental Structure, BBS* 14(3) 1991.] Both strategies are problematic. Investigations of rare "pure cases" in which a small circumscribed lesion produces an equally circumscribed (and measurable) functional deficit have significant limitations. Researchers must focus their energies on a small number of pure cases without being able to test how representative the cases are of human brains as a whole. The generalizability of case study results is an empirical issue and can only be evaluated by comparing large sets of case studies using similar exacting measures.

Given that dissociation and double dissociation are the empirical currency of neuropsychological inference, researchers are faced with a different problem when examining less pure cases in group studies. In these kinds of investigation, researchers look for commonalities among patients with less-circumscribed, overlapping, but nonidentical deficits. Depressed performance on a small number of measures with relatively preserved performance on a similarly small set of closely related measures is generally accepted as evidence of dissociation and thus of some degree of modularity for the dissociated process. However, such a pattern may be consistent with either modular or nonmodular organization depending on whether additional assumptions can be made about compensatory phenomena and the functional and physiological dependencies between processors.

Standard pathonormal inference models assume a simple transparent mapping between a behavioral deficit and neurological damage. This assumption is clearly unfounded in interactive systems, but they may also be inappropriate in modular systems. For example, suppose that spoken word recognition depends on three closely localized, connected, but nonoverlapping processors car-

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https://doi.org/10.1017/S0140525X99002058 Published online by Cambridge University Press

rying out auditory, acoustic-phonetic, and lexical analyses with strict feedforward organization. Patients are typically tested on some combination of measures showing, for instance, that they have a deficit in acoustic-phonetic processing but normal hearing. One simple interpretation is that patients share some degree of damage to the acoustic-phonetic processing module, with the degree of damage proportional to the severity of their impairment. But there are other possibilities. Deficits could result from disconnection between auditory and acoustic-phonetic processors. Or deficits might reflect slow, noisy processing in acoustic-phonetic or auditory modules as a function of hypometabolism from lesions in white matter tracts connected to these regions. If an inhibitory tract regulates the output of either module, disruption to the tract may impair its interpretation. Finally, impaired performance by the acoustic-phonetic module may be masked by compensation in the lexical module. Given these possibilities, even physically circumscribed functions may be hard to localize under the assumption of linear lesion-deficit association.

Conventional statistical analyses encourage the assumption that similar language deficits are a direct function of damage to a common processor. Consider the exclusive use of overlap analysis, correlations, regressions, and patient group comparisons to relate lesion sites and aphasic syndromes. Nonlinearities are difficult to detect because effect sizes are strictly linear. Synchrony between processes is hidden in regression when adjustments to effect sizes eliminate the influence of all other measures. Local environments are neglected when focus is on the unique, acontextual effect of a variable. Sources of variability other than those hypothesized are quashed, placed into error, and usually not explored. Hence, individual deviations from a sample mean, or individual patients' deviations from their overall mean, are rarely investigated. Similarly, variability in lesion sites and syndromes is underemphasized. These problems are exacerbated when inferential tests decide a variable's importance. The small sample size typical in aphasiology can reveal only very large and coarse effects, and the independence assumption is at odds with ideas of interactivity. For all these reasons, what are important matters to Müller will be "nonessential complicating factors" to others.

Analyses should be supplemented with procedures that are less reliant on linearity, significance, and independence. For example, researchers might use indices such as distances in cluster or multidimensional scaling analyses to determine a finite number of conceptually meaningful linguistic profiles. The profile is a frequent, nonlinear combination of many variables, not the acontextual, linear impact of any one variable. Patients grouped into profiles have similar configurations of linguistic deficits and strengths. Patient profiles might then be associated with lesion data through techniques such as multiple discriminant analysis that allow spatial representations of how various neurological variables differentiate linguistic profiles. Subsidiary analyses could examine how lesion sites account for variability within a profile. The procedure could also be reversed, with lesion profiles related to language variables. This kind of analysis, particularly when supplemented with longitudinal, multi-level data, steps away from considering only linear relations between static, unitary syndromes and brain regions.

Our suggestion is that Müller's theoretical contribution eventually needs to be supplemented with methodologies appropriate for dynamic, interactive, living systems. As this occurs, debates over the innateness, modularity, and universality of language might give way to analyses of why language appears innate, modular, and universal. This could also help us understand why linguistic phenomena do not perfectly conform to these ideals.

Territorial song and facial gesture: A language precursor in apes

Maria Ujhelyi

Institute of Behavioural Sciences, Semmelweis University of Medicine, 1089 Budapest, Hungary. **ujhmar@net.sote.hu** **www.sote.hu/magtud**

Abstract: The natural communication system of chimpanzees has some unique characteristics rooted in two possible ways of producing call variants in primates. The chimpanzee call repertoire contains variants available to all group members. The transfer presupposes voluntary control and learnability. Chimpanzee vocalization (or its homologue in the common ancestor of chimpanzee and man) seems to represent a real precursor of human language.

As a part of his argument against the nativist view of language, Müller (1996t) devotes a section to communication in monkeys and apes (sect. 3.1.1). In his short review of data showing possible homologies between monkey communication and human language, Müller mentions the calls of titi monkeys containing variously ordered call sequences as a possible example of "protosyntax."

This type of vocalization, in which several small units are combined into a longer sequence, is more widely distributed in primates. It occurs also in indris (Thalmann et al. 1993), in tamarins (Cleveland & Snowdon 1982), and in each species of the lesser apes, the gibbons (Geissmann 1993). In addition, recent field studies demonstrate that chimpanzees and bonobos also utter long calls, which are built from acoustically distinct segments (Clark & Wrangham 1993; Hohmann & Fruth 1994). Although essentially the same set of units are utilized, syntactically different calls emerge due to the different arrangements of the units. Naturally existing syntactical ability is the background of the laboratory performance of Kanzi, who understands and produces meaning differences arising from different word orders (Savage-Rumbaugh et al. 1993).

Searching for the adaptive advantage of producing variable calls, we find that most of the species producing them are special territory defending monogamous primates who use acoustic signals to mark their territories. Because the primary sounds are limited in number and genetically fixed, the differences needed for marking territory individually can only be achieved by combining the elementary, invariant sounds and varying their arrangement. According to Mitani and Marler's (1989) analysis, the territorial song variants of gibbons realize individual differences. The syntactically variable compound call seems to evolve first under the constraint of labelling individual territory.

Present day chimpanzees and bonobos both live in large, multimale promiscuous groups in which the individuals do not mark or defend territories against other group members. However, it cannot be excluded that their long, composite, syntactically variable call, which also features individual differences, has an antecedent in vocal territorial marking. Gorillas have a call type that they emit only when they encounter another group (Fossey 1972). Comparing this call and chimpanzees' long calls, Mori (1983) suggested the possibility of a common ancestral call with territorial function. De Waal (1988) also observed the similarity between the long call and the gibbon territorial song. Furthermore, the call presentation strongly reminds one of territorial song, which is generally presented in duet form, with the members of a pair performing a mutually cooperative and coordinated display. Coordinated call display also exists in great apes. In common chimpanzees, the males often call together (Mitani & Brandt 1994), and in bonobos, male-female pairs perform duets (Hohmann & Fruth 1994) despite the seemingly strong correlation between monogamy and duet performance (Haimoff 1986).

Although some features of the earlier territorial song are preserved in group living conditions, the functions both of producing individual differences in vocalization and that of duetting changed. Instead of spacing, the long call of chimpanzees and bonobos serves to maintain the connection between the group

https://doi.org/10.1017/S0140525X99002058 Published online by Cambridge University Press

members, between definite individuals bridging great distances. As the exclusive nature of marking an individual territory is absent, duetting (or chorusing) can serve another function; the social transmission of call variants. Mitani and Brandt (1994) observed that chimpanzees attempt to match the acoustic characteristics of one another's vocalizations when calling together. They appear to alter the acoustic structure of their calls when chorusing with different partners. Boesch (1991) reported that males started to use the individually specific calls typically emitted by other group members who had disappeared from the group.

Living in large groups gave rise to further possible changes in vocal capacity. In primates with complex social interactions, facial gestures play an important role in expressing emotions: they have an immediate communicative function. However – as a secondary result – acoustic (articulatory) variations arise when an animal changes its facial gesture during vocalizing. In this way, even in the absence of a human-like vocal tract, articulatory nuances are enhanced by facial gestures, modified by mandibular position or lip configuration (Hauser et al. 1993; Richman 1976).

By integrating two types of primate vocalization systems, chimpanzee and bonobo communication establishes a structurally and functionally distinct level compared to lower animal communication. This stage of the evolution of communication might be present in the common ancestor of chimpanzee and man, which seems to represent a real precursor of human language.

Territorial vocalization supplies a possible foundation for the origin of syntax, while the syntactic variation becomes socially transferable in both species of chimpanzees. Among them, individual or pair territories no longer exist; instead they live in complex social networks. Both preconditions seem necessary however, because neither monogamous species nor group living species without a long call (with its possible background in territorial vocalization) could achieve the evolutionary level of communication that can be found in great apes. Human language, however, underwent further radical structural and functional reorganizations in the course of its evolution.

Author's Response

Homology, neurogenetic imprecision, and lesional complexity

Ralph-Axel Müller

Department of Cognitive Science, University of California, San Diego, San Diego, CA 92037. **amueller@ucsd.edu**

Abstract: The two commentaries appear supportive of the target article. **Ujhelyi's** commentary can be complemented with recent evidence supporting continuity of language and cognitive evolution in hominids. **Gow & Rodkin's** caveats regarding "pathonormal inference" and the single-case methodology are discussed from a developmental neurobiological perspective. Early structural brain lesion and developmental disorders can serve as pathological models of normal neurofunctional variability resulting from neurodevelopmental imprecision. A final point concerns the advantages of integrating multiple structural and functional imaging modalities in neuropsychological studies.

Ujhelyi argues that "protosyntactic" qualities are found more widely in primate vocalizations than my cursory discussion indicated. However, by virtue of its hierarchical organization, human syntax involves more than differential concatenation of vocalizations. Ujhelyi acknowledges "radical reorganization" in hominid evolution of language, but her use of the term "syntax" seems to downplay these fundamental differences. Ujhelyi's discussion is nonetheless valuable in highlighting potential homologies between certain elementary aspects of human grammar and primate vocalization systems.

Parallels with the issue of "protosyntactic" vocalizations can be found in other lines of animal search. Examples (though not directly related to language) are recent studies on numerosity in rhesus monkeys (Brannon & Terrace 1998) that seem to indicate an unexpected potential for the acquisition of numbers and ordinal rules. [See also Davis & Pérusse: "Numerical Competence in Animals" *BBS* 11(4) 1988.] This does not imply that monkeys are budding mathematicians, but it suggests that there could be homologies in nonhuman species with some cognitive capacities underlying human mathematical thinking. For another example, categorical auditory perception has been demonstrated in a number of nonhuman species, even in invertebrates such as crickets (Wyttenbach et al. 1996). Such findings do not imply homology between human and cricket auditory systems, but they do show that categorical perception can be achieved by relatively simple auditory systems and that its occurrence in human phonology does not call for a discontinuity model of language emergence.

In addition to the studies cited in the target article, there is new neuroanatomical evidence compatible with a continuity model.[1] Gannon et al. (1998) recently reported larger plana temporale in the left than in the right hemisphere in chimpanzees. In humans, such asymmetry has been related to left-hemisphere dominance for language (Foundas et al. 1994; Galaburda 1984; Geschwind & Levitzky 1968). The precise functional significance of the chimpanzee findings remains unclear, but it has now become possible to examine such questions by means of functional magnetic resonance imaging in awake primates (Dubowitz et al. 1998; Stefanacci et al. 1998).

Gow & Rodkin expand on the problematic implications of "pathonormal inference" for cognitive neuropsychology. I sympathize with their discussion of study designs examining the nonlinear interaction of neuropsychological, clinical, and demographic variables, but I do not entirely agree that dissociations are the "empirical currency" of neuropsychology. It is historically accurate that inferences about the (lesioned) brain used to be almost exclusively based on behavioral data. With the advent of noninvasive imaging techniques, however, a purely behavioral approach needlessly limits (and indeed impedes) the study of brain-behavior relationships.

Gow & Rodkin also criticize the cognitive neuropsychological single-case methodology that is based on the assumption of neurofunctional universality. [See also *BBS* multiple book review & Shallice's *From Neuropsychology to Mental Structure*, *BBS* 14(3) 1991.] Functional neuroimaging in disorders of brain development provides insight into the maturational plasticity of neurofunctional organization, which constrains universality and thus limits conclusions drawn from single-case studies. Behavioral, electrophysiological, and animal lesion studies have long indicated the brain's potential for fundamental reorganization after early lesions (Kolb 1995). With functional neuroimaging, such reorganization can now be observed more directly in human subjects (Chugani & Müller, in press). Recent neuroimaging studies in patients with unilateral lesions – congenital or from the first years of life – have documented

https://doi.org/10.1017/S0140525X99002058 Published online by Cambridge University Press

the brain's potential for inter- and intrahemispheric reorganization in the language (Müller et al. 1998a; 1999a; 1999b) and motor domains (Müller et al. 1997; 1998b; Seitz et al. 1998).

Anomalies of functional organization are also found in developmental disorders, which are not typically characterized by localized structural brain damage. Individual variability associated with these neurofunctional anomalies appears considerable. For example, functional neuroimaging studies of autism have generated a wide range of often incompatible findings (Minshew 1994; Rumsey 1996). Diagnostic criteria for patient selection and differences in experimental conditions clearly contribute to inconsistencies in the literature, but epigenetically based individual variability within disorders is likely to play an important role (Courchesne et al., in press). There is increasing evidence that pervasive developmental disorders involve spectra of deficits, including low-functioning autism, high-functioning forms and Asperger's syndrome (Szatmari et al. 1995), and a "broader phenotype" located within the normal spectrum of neuropsychological variability and outside the diagnostic criteria of autism proper (Le Couteur et al. 1996). A recent study by Kates et al. (1998), for example, showed striking differences in regional neuroanatomic measurements (above all in the mediotemporal lobe and in lobules VI and VII of the cerebellar vermis) in monozygotic twins who were discordant for autism proper (twin B fit the criteria of the "broader phenotype"). The notion of individual variability in an autistic spectrum that blends into the normal spectrum is paralleled by findings suggesting that developmental dyslexia is situated at the lower tail of a normal distribution of reading abilities (Shaywitz et al. 1992).

Disturbances of brain development are thus associated, not simply with predestined long-term dysfunction, but with individual variability of neurofunctional organization. When induced by congenital or early postnatal structural brain damage, this variability will be related to onset, site, and size of lesion. When it occurs in the context of a developmental disorder, it may result from epigenetic mechanisms following an initial pathogenic event. The principles underlying such variability in patients with early brain damage and developmental disorders can be conceptualized as interacting effects of *vulnerability* (for example, neurotoxic exposure in utero or perinatal trauma) and *compensation* (for example, axonal sprouting or regional functional reorganization; Müller & Courchesne, in press).

According to conventional wisdom, effects of vulnerability and compensation are found only in *pathological* development. However, this view is based on an understanding of brain development as a genetically prespecified and perfectly accurate series of events. An alternative is to view ontogenesis as a series of probabilistic events. Neurodevelopmental imprecision (Clarke 1981), which is subject to repair and compensatory reorganization on a microscopic scale, creates multiple sources of neurofunctional variability within the "normal" spectrum. For example, from a developmental perspective neocortical architecture is not precise. Instead the initial architecture of what O'Leary (1989) calls the "protocortex" is underspecified and highly malleable. Relative universality of neurofunctional organization emerges in an epigenetic process, in which activity of afferent connections and ultimately stimulation from and interaction with the environment play a crucial role (Kandel & Jessell 1991; Katz & Shatz 1996; Quartz & Sejnowski 1997). These influences are not preprogrammed, nor are they precisely the same for every individual. The malleability of cortical functional organization is underlined by studies showing correlation of experiential with structural (Amunts et al. 1997; Schlaug et al. 1995) and functional (Elbert et al. 1995) cortical measures. It is conspicuous in studies of subjects with loss of a sensory modality early in life (as, for example, in blindness; Sadato et al. 1998). My criticism of the universality concept, however, does not imply a denial of the obvious gross invariance of neurofunctional organization in the vast majority of human brains. Instead, it is directed against the notion of universality based on genetic predetermination.

Finally, **Gow & Rodkin** propose to correlate neuropsychological and demographic patient profiles with lesion data. Gow & Rodkin also use the term "lesion profile," but the issue requires more emphasis. In neuropsychological studies, lesions are usually grossly classified based on structural imaging data (CT or MRI), if such data are operationalized at all. However, *functional* neuroimaging shows (often severe) effects *far beyond* a structural lesion (Metter & Hanson 1994). A structural lesion apparently triggers multiple (partly distal) metabolic and biochemical events, all of which have potential effect on cognitive-behavioral outcome. Thus, in a patient with a given structural lesion, functional lesions of differing size and site may be identified by PET using different radioisotopes or by proton magnetic resonance spectroscopy (cf. Chugani et al. 1997; Duncan 1997; Preul et al. 1997). All in all, there are many reasons to operationalize brain lesions as comprehensively as neuropsychological profiles. Greater use of functional neuroimaging in cognitive neuropsychology may help explain individual variation in focal structural lesion effects in terms of less focal functional impairments.

NOTE

1. I am indebted to James T. Heaton for pointing out an error in section 3.1.2 of the target article (p. 615) concerning asymmetries in song-control nuclei in birds. In fact, there have been findings of *functional* asymmetries (lateralization of vocal control) in the *absence* of anatomical asymmetry.

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Letter "r" before author's initials stands for Author's Response to the Continuing Commentary.

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https://doi.org/10.1017/S0140525X99002058 Published online by Cambridge University Press