Genetic factors and normal variation in the organization of language
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1. Introduction and summary

In this essay we present two themes. The first is a factual review of the behavioral and neurological differences in language and cognition between people with and without familial left handedness: these differences begin to justify the claim that there is a continuum of how language and cognition are represented in the brain, reflecting a quantitative difference in the role of the right hemisphere, and consequent potential qualitative differences.

The second theme involves the implications of this finding. Various cases of rare neurological organization for language have called into question the idea that there is a single form of representation: these include cases of left-hemispherectomy in which the patients with a lone right hemisphere can grow up to be normal linguistically (Curtiss et al., 2001; Devlin et al, 2003) with normal developmental stages (Curtiss & Shaeffer, 1997) as well as unique instances such as the infamous formerly hydrocephalic mathematician whose neocortex was a thin layer of tissue lining the skull (Lewin, 1980) – clearly the topology and connections of different cortical areas are very different in these cases from the norm. Even classic and recent studies call into question the unique location and function of a linguo-central structure such as Broca's and Wernicke's areas (Penfield and Roberts, 1959; Bogen and Bogen, 1976; Anderson, 2010; Ragalsky and Hickok, 2011). But people with familial left-handedness comprise 40% of the population, so we cannot consign their unique behavioral and neurological structures to an odd distaff "minority".

A profound implication for language of these considerations is the possibility that the existence of language is not causally dependent on any particular unique neurological organization. Rather, especially the sentence construction mechanism of syntax is a computational type that recruits different neurological structures. On this view the possibility for syntax emerges as a function of the availability of propositional relations, combined with an explosive growth in the number of lexical items that can externalize the internally

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represented categories. The syntactic computational architecture is represented neurologically via co-option and integration of multiple brain regions that are collectively suited to the type of computation that language requires. On this view, there can be significant lability of how language will be represented in an individual's brain, if there is significant variability in how the computationally relevant areas function or are interconnected.

We will show that there is significant variability in functional cortical organization differentiating individuals with familial sinistrality (FS+) and those without (FS-), along a continuum of genetic effects associated with left-handedness. The steps in this argument are first to review some general considerations about the basis for cerebral asymmetries for higher functions in general; we then turn to a brief review of current evidence that people with and without familial left-handedness (sinistrality) have characteristic differences for language behavior – people with familial left-handedness appear to access lexical items more readily than other; these behavioral differences are reflected in some recent studies of brain imaging, which show both qualitative and quantitative effects. We suggest that the apparent qualitative effects may result from the quantitative differences, but leave open the possibility that the qualitative differences reflect real differences in how language is realized neurologically.

2. Cerebral asymmetries – Computational differences in the hemispheres and alternative explanations

A number of cognitive functions and processing characteristics have been traditionally ascribed to a specific cerebral hemisphere, notably verbal, relational and/or sequential processing in the left and nonverbal, spatial, prosodic and holistic processing in the right. It has become increasingly clear that these dichotomies are not entirely accurate and show considerable individual variability. Since substantial and quantitatively predictable shifts from left hemisphere language processing may be present in up to 40% of the general population, consideration of these differences is not only theoretically illuminating but also relevant to biolinguistic research programs in practice. That is, these individual differences in hemispheric specialization can provide considerable insight into the computational flexibility of the brain and computational options of how behavioral interfaces might be represented. We first review some of the more reliable evidence for moderate hemispheric specialization in several cognitive and linguistic domains in relation to theories of lateralization. Then we discuss the relation between functional and behavioral asymmetry and evidence for a common genetic influence. Finally, we consider how variable cerebral asymmetries may inform theories of syntax-semantic and syntax-phonology interfaces.[1]

[1] Some theories of cerebral lateralization hinge exceptionally on a close connection between language and motor function, for instance suggesting that the population-level bias for right-handedness (from e.g. mother-child bonding or a hand-to-hand combat advantage) and an essential link between fine motor skill in speech (for articulation) ultimately establishes a left hemisphere bias for language processing (e.g., Jonas & Jonas, 1976). We omit a discussion of such theories since

It is uncontroversial to assume that hemispheric specialization is largely driven by the anatomical separation of the brain by the longitudinal fissure and interconnecting axon tracts--the corpus callosum, connecting cortical areas and the anterior commissure, connecting subcortical areas. The corpus callosum is a dense tract of approximately 200 million topographically organized axons connecting the two hemispheres, with many of the fibers dedicated to motor cortex. The corpus callosum introduces a significant conduction delay in interhemispheric communication since the human brain is larger than other primate brains, but axons in the human corpus callosum are not proportionally larger (Olivares et al. 2001). Morphological changes in the corpus callosum diameter have been associated with left-handedness (Dunham & Hopkins, 2006), developmental (Duara et al. 1991; Hynd et al. 1995) and psychiatric disorders (see Innocenti et al. for a review).

Ringo et al. (1994), among others, proposed a "Callosal Distance" hypothesis, that interhemispheric communication delays through the corpus callosum are critical to functional lateralization, particularly in large brains where conduction delays may be on the order of tens of milliseconds (Aboitiz et al. 2003). This theory receives substantial support from a range of neuroanatomical evidence and computational models. Using simple self-organizing neural networks, Levitan and Reggia (Levitan & Reggia, 2000; 1999; Shevtsova & Reggia, 1999) demonstrated that lateralization of self organized neural maps, akin to functional specialization, depended on the properties of a connecting simulated corpus callosum, in addition to the number of neurons each hemisphere and their dynamical properties (e.g. excitability). Many fibers in the corpus callosum are thought to be inhibitory, such that a functional bias in one hemisphere will inhibit recruitment of corresponding regions in the opposite hemisphere for the same function.

Empirically, recent studies suggest that a measure as simple as overall skull size can predict the strength of behavioral asymmetries – smaller brains have less lateralization for language (but not visual processing) than large brains, as would be predicted by the callosal distance hypothesis (Tzourio-Mazoyer et al. 2010). This distinction between language and vision is also consistent with connectionist models that suggest the degree of hemispheric specialization may depend on the complexity of the task, showing greater emergent unilateral involvement for complex tasks (e.g. language; Monaghan & Pollmann, 2003). The 'callosal distance hypothesis' thus provides a clear neuroanatomical mechanism for establishing functional cerebral asymmetry. In the next section, we consider possible mechanisms for a left-hemisphere bias in language lateralization.

2.1. Processing Speed and Relational/Holistic Processing

Language is arguably the most complex of cognitive processes, requiring rapid analysis and integration at multiple levels of complex structure to support natural speech. Thus, many historically important ideas about cerebral

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they lack both evidence and utility, instead focusing on lateralization theories that address the connection between brain function and computational properties.

asymmetries start with language as the critical case. In the late 19th century, Hughlings Jackson suggested that the basis for language asymmetries is not modality-specific, but rather follows from an essential difference between the hemispheric 'styles' of processing: the left hemisphere is "propositional", while the right hemisphere is "associative" (Hughlings-Jackson, 1878, 1879). Bever (1975) reformulated this as a more general distinction between "relational" and "holistic" processing, relating it to the relative number of representations that are integrated at one time. Bever (1980) suggested that the basis for such an essential difference could be resolved to a (potentially very small) left hemisphere superiority in computational power - in conjunction with a developmental lateralization process involving complementary inhibition, this computational difference would produce the observed left hemisphere superiority for relational processing. Relational processing requires at least two distinct representations to be stably maintained in order to be interrelated: by definition, this involves more representations than holistic processing, which can be processed one representation at a time.

Greater 'computational power' in the left than right hemisphere could be reflected in anatomic asymmetries that might suggest more computational power. An obvious candidate for this is the asymmetry of the planum temporale, a region of auditory cortex that is often substantially larger in the left hemisphere than the right. Early (around 30 weeks gestation) anatomical asymmetries of the planum temporale (Geschwind & Levitsky, 1968), in addition to more developed cortical folding in the right hemisphere partially motivated the Geschwind & Galaburda (1985) theory of cerebral asymmetry. The Geschwind-Galaburda theory rests on the apparent developmental origin of cerebral asymmetry—namely that the left hemisphere matures more slowly than the right—and the hypothesis that rate of maturation is mediated by the intrauterine environment. Noting that left-handedness is more prevalent in males, Geschwind and Galaburda hypothesized that testosterone is a key environmental factor that influences the maturation of the fetal brain. This hypothesis also offers an explanation for (Geschwind & Behan, 1982; 1984) and others' finding that immune and developmental disorders are more prevalent in left-handers and individuals with familial left-handedness.

The testosterone hypothesis has broad implications beyond cerebral asymmetries that could support it: they note that the development prevalence of asthma in each sex reverses around puberty (from being more common in males during childhood to more common in post-pubescent females), consistent with an immune-testosterone link. However, Vink et al. (2010) found no association between hormone levels and the change in the prevalence of asthma in a large Dutch sample and suggested that other sex-dependent developmental factors, such as differential lung development or obesity, may account for the reversal. While not fatal to the Geschwind-Galaburda theory, this evidence undermines a major feature of the theory, suggesting that non-hormonal sex effects may be responsible for the apparent link between left-handedness and immune disorders.

Using hemispheric neural network models, Shevtsova & Reggia (1999) found that lateralization is biased towards larger networks, consistent with the

Geschwind-Galaburda theory. On the other hand, there are well-known examples of neural growth in response to usage, even in adults (Maguire et al., 2000). Since fetal hearing develops as early as 24 weeks (Birnholz & Benacerraf, 1983), some six weeks before planum temporale asymmetry emerges, it is plausible that this asymmetry reflects an earlier predisposition for left-hemisphere language and/or auditory processing.

Aside from differences in the anatomical size of specific brain structures, a number of empirical hypotheses suggest that the functional basis for a computational superiority of the left hemisphere could be increased processing speed. Differential processing speed between cerebral hemispheres, has been proposed as an adaptive energy-conserving mechanism that will naturally arise when a split neural network must support at least one highly demanding task while minimizing energy consumption (Grushin, 2005). Empirically, some support has been found for the view that a deficiency in processing speed may explain certain cases of Specific Language Impairment behaviors (Leonard et al. 2007).

Poeppel (2003) has focused on differences in processing speed for auditory input in particular: he suggested that language lateralization is related to intrinsic differences in hemispheric specialization for processing auditory input on different timescales. On his "asymmetric sampling in time" (AST) hypothesis, initial auditory processing occurs bilaterally, with later resolution of auditory input into laterally distinct timescales of information integration: a short (20-40ms; gamma EEG frequency band) time window more prominent in the left hemisphere and a long (150-250ms; theta band) window more prominent in the right hemisphere. These timescales reflect two components of the speech signal: rapid spectral changes associated with formant information and slower spectral changes associated with prosodic information. To provide support for this hypothesis Poeppel and colleagues have shown greater gamma band activity in the left than right auditory cortex and greater theta band activity in the right auditory cortex at rest (Giraud et al. 2007). Boemio et al. (2005) also found greater sensitivity to long (>85ms) frequency modulated segments in the right superior temporal sulcus than the left. Gamma band oscillations have been suggested to have a critical role in the binding problem (e.g., Engel et al. 1991; Singer et al. 1995; Miltner et al. 1999). Since a 'faster' hemisphere might be expected to bind representations more rapidly, increased gamma activity in the left hemisphere could be consistent with differential processing speed.

Greater processing speed could be the result of a larger number of parallel computation units (e.g. more neural assemblies), faster low-level processing (e.g. at a higher oscillator frequency, say gamma vs. theta) or an increase in the efficiency of processing and reduction in the time needed to converge to a stable state in a neural network. We now consider an alternative to the processing speed/capacity as the sole explanation for the hypothesized computational superiority of the left hemisphere: the critical distinction between the hemispheres may be the relative level of neural noise in processing that results from functional differences. Conceptually, if noise effects each representation equally, the effect on relational tasks involving many simultaneous

representations will increase geometrically, compared with minimal effect on holistic tasks involving only one representation at a time.

The signal-to-noise ratio (SNR) describes the power of a stimulus-induced electrophysiological response to the non-induced fluctuations around the signal (not to be confused with the ratio of induced signal power to resting activity). More generally, this is the fidelity with which an encoding signal can be reproduced. SNR can be non-invasively measured with EEG by comparing the average evoked response to some repeated stimulation to the level of trial-by-trial variability (Möcks et al. 1988). SNR has important theoretical implications for neural processing, for instance bounding the information capacity of a channel, under certain assumptions (Shannon & Weaver, 1949), the memory capacity of a neural population (Ganguli et al. 2008) and coupling properties of neural oscillators. Thus, it is directly relevant that SNR has already been shown to be higher in the left hemisphere than the right (Winterer, 1999).

High SNR is generally considered to be a desirable property for computational efficiency. However, the optimal level of SNR, may not always be the highest, but can be intermediate. In the brain, neural noise has been proposed as beneficial to neural processing by inducing stochastic resonance. Stochastic resonance is the slightly counterintuitive phenomenon in which noise actually increases SNR by essentially lowering the neural response threshold, thus increasing sensitivity (and response to) to low amplitude signals. For example, in the human visual system, monocular subthreshold visual stimulation was found to evoke changes in scalp potentials when accompanied by visual noise (to the other eye, but overlapping visual field), suggesting that the brain does benefit from stochastic resonance, at least in sensory processing (Mori & Kai, 2002). Language processing is poorly described in terms of low amplitude thresholds at this level of abstraction, but neural computation properties such as SNR do have relatively direct relevance for dynamical systems models of language acquisition (Andrews, 2003; Hancock, 2009) and processing (Tabor & Hutchins, 2004; Tabor & Tanenhaus, 1999) and general theories of binding in vision science and linguistics. Conceptually, language may be preferentially lateralized to the hemisphere having (close to) a computationally optimal SNR.

Hemispheric differences in the modulatory action of dopamine in the cortico-striatal-thalamic loop present a possible, and theoretically appealing source of SNR asymmetry. The corticostriatal loop, consisting of largely ipsilateral parallel 'direct' and 'indirect' pathways within the basal ganglia, plays a key role in most aspects of cognitive processing and motor control. Thus, asymmetries in this pathway may have broad effects on functional lateralization and an intrinsic link to motor lateralization. The dominant types of striatal dopamine receptors, D1 and D2, have excitatory and inhibitory modulatory effects on cortical projections, respectively, and together provide a contrast enhancement mechanism affecting cortical SNR.

We propose that a left-right asymmetry in D2 receptor activity produces a corresponding asymmetry in cortical SNR, thus affecting the degree to which noise-sensitive cognitive processes are lateralized. Several lines of indirect evidence support this hypothesis:

1. In normal, right-handed adults, there is evidence of greater D2 activity in the left striatum than the right (Larisch et al. 1998; Vernaleken et al. 2007).
2. Dopamine activity, especially D2 activity, is known to bias motor activity (e.g. turning preference) in animals (Giorgi & Biggio, 1990) and may be similarly linked to hand preference in humans (Mohr & Lievesley, 2007; Mohr et al. 2003)
3. Dopamine D2 function is closely linked to a number of psychiatric disorders that have been linked to non right-handedness and reduced cerebral lateralization (e.g., Abi-Dargham et al. 2000).
4. The major candidate gene associated with handedness (LRRTM1) is notably expressed in the human striatum, where it may interact with dopaminergic synapses.

While non-dopaminergic neural changes could produce similar effects (e.g. increased cortical excitability as proposed in ADHD), these effects would not be expected to have the same close link to motor lateralization.

3. Familial handedness and heritable variation in the neurological representation of language.

The usual reasons in today's science to study genetic effects on language is to demonstrate evidence that language is "innate" in some interesting sense, that differentiates it from heritability of "general cognition", "communicative capacity" and so on. Thus, there are alleged investigations of spared syntactic capacity in Williams' Syndrome children (Bellugi et al. 1994; Clahsen, 1998; Zukowski, 2004), as well as children with severe motor disabilities: conversely, there are forms of selective impairment of language in, e.g., Turners syndrome (Curtiss, 2012) and "FoxP2" (Watkins et al. 2002) children. In each of these cases, the usual method (in principle) is to isolate a particular genetic abnormality, and relate it to the selective sparing or selective impairment of language ability, thereby making more specific the claim that language is "innate".

In our behavioral research of many decades and recent neurolinguistic studies, we have adopted a different method to provide converging information about the heritability of how language is used and represented. In particular, we have tracked the effects of familial left-handedness in right-handers. Many thousands of questionnaires have shown that about 40% of all college students are right-handers with familial left-handedness, and an equal percentage of right-handers without familial left-handedness. Thus, we can use familial handedness as a tool to explore differences in how language is used and represented in two equally large "normal" populations. Of course, there is no guarantee that there is any effect, any more than would be found by differentiating people by height. But we have in fact found significant effects of familial handedness: this presumably is mediated by differences in neurological organization, particularly asymmetries, as affected by genetic tendencies towards cerebral symmetry, even in phenotypic right-handers. Below we review some findings from others as well as our research.

Loss of linguistic ability results from damage to specific areas of the left neocortex. The fact that normal language depends on (rather small) specific areas suggests that it may be critically 'caused' by those areas. However, certain aspects of language may have considerable latitude in their neurological representation. For example, Luria (1970) and colleagues noted that right-handed patients with left-handed relatives (FS+) recover faster from left-hemisphere aphasia, and show a higher incidence of right-hemisphere aphasia than those without familial left-handers (FS-). They speculated that FS+ right-handers have a genetic disposition towards bilateral representation for language, which often surfaces in their families as explicit left-handedness. In individuals of both left- and right hand preference, familial sinistrality may account for some of the variability seen in aphasia symptoms (Ettlinger et al. 1956, Subirana 1958) and language symptoms in individuals with unilateral lesions (Hécaen et al. 1981). Individuals with crossed aphasia show symptoms similar to those with left-hemisphere damage, but have a higher incidence of FS+ (Coppens et al. 2002).

We have found a consistent behavioral difference between the two familial groups in how language is processed, which may explain Luria's observation. Normal FS+ people comprehend language initially via individual words, while FS- people give greater attention to syntactic organization. A simple demonstration is that FS+ people read sentences faster and understand them better in a visual word-by-word paradigm than a clause-by-clause paradigm: the opposite pattern occurs for FS- people. Another example is that if words in a short essay alternate in isolation between the ears at a normal rate, FS+ people understand the essay better than if the words are presented all monaurally: the converse is true for FS- people. Bever et al. interpreted this as a result of the relative segregation of each word from the adjacent ones in the alternating ear condition, making it easier for FS+ people to recognize each word separately (these studies and others are reported in Bever et al. 1987; Bever, 1988). In another set of studies, Townsend and colleagues reported that recognition of an auditory probe word from a just-heard sentence fragment is faster in FS+ people than FS- people, while the latter are more sensitive to the overall grammatical structure of the sentence fragment (main vs. subordinate clause; Townsend et al. 2001).

The bilateral representation of language in FS+ people may be specific to lexical knowledge, since acquiring that is less demanding computationally than syntactic structures, and hence more likely to find representation in the right hemisphere. On this view, FS+ people have a more widespread representation of individual lexical items, and hence can access each word more readily and distinctly from syntactic processing than FS- people (Bever et al. 1987; Bever et al. 1989). This hypothesis would explain the relative ease of processing lexical items in FS+ people.

This leads to a prediction: lexical processing is more bilateral in FS+ right-handers than FS- right-handers, but syntactic processing is left-hemisphered for all right-handers. Recently, we tested this using fMRI brain imaging of subjects while they are re-ordering word sequences according to syntactic constraints or according to lexico-semantic relations between the words. We found suggestive evidence that the lexical tasks activated the language areas bilaterally in FS+

right-handers, but activated only the left hemisphere areas in the FS- right-handers: all subjects showed strong left-hemisphere dominance in corresponding syntactic tasks (Chan, 2007). This confirms our prediction, and supports our explanation for Luria's original clinical observations. It also demonstrates that there is considerable lability in the neurological representation of important aspects of language.

Familial handedness and the critical period offers further evidence for the genetic differentiation of language representation. The notorious case of the critical period is syntactic knowledge of an explicit language, which is neither determined by sensory/motor learning nor related directly to universals of thought. Bever has argued that the critical period for syntax learning is a natural result of the functional role that syntax plays in learning language – namely, it assigns consistent computational representations that solidify perceptual and productive behavioral systems, and reconciles differences in how those systems pair forms with meanings (Bever, 1975; 1981). On this view, the syntactic derivational system for sentences is a bilateral filter on emerging perceptual and productive capacities: once those capacities are complete and in register with each other, further acquisition of syntax no longer has a functional role, and the syntax acquisition mechanisms decouple from disuse, not because of a biological or maturationally mechanistic change (see Bever 1988) for a demonstration of the hypothesis that grammars act as cognitive mediators between production and perception in adult artificial language learning).

This interpretation is consistent with our recent finding that the age of the critical period differs as a function of familial handedness: FS+ deaf children show a younger critical age for mastery of English syntax than FS- children (Ross & Bever, 2004). This follows from the fact that FS+ people access the lexical structure of language more readily, and access syntactic organization less readily than FS- people: FS+ children are acquiring their knowledge of language with greater emphasis on lexically coded structures, and hence depend more on the period during which vocabulary grows most rapidly (between 5 and 10 years; itself possibly the result of changes in social exposure, and emergence into early adolescence).

4. The Genetics of Cerebral Asymmetries

Remarkably little is known about the genetics of asymmetries as reflected in phenotypic handedness. Part of the difficulty lies in the paucity of reliable behavioral measures of asymmetries other than subject-reported or measured hand dominance and the associated debate over whether handedness should be viewed as a strictly categorical trait or a quantitative trait, and if so, how to measure the continuum. Dichotic listening tests (Kimura, 1961) have been widely used as a non-invasive, behavioral method of assessing cerebral dominance at a cerebral level: these measures are generally consistent with sodium amytal tests and fMRI measures of functional language lateralization (Zatorre, 1988; Hund-Georgiadas et al, 2002). Overall, the majority of subjects typically show a right ear advantage (REA) in dichotic listening studies, with a tendency for reduced REA in left-handed subjects. Zurif & Bryden (1969) found that the latter effect was

moderated by familial sinistrality: both right-handed and FS- left-handed subjects showed a REA while FS+ left-handed subjects showed minimal asymmetry effects. Zurif and Bryden (and Hines & Satz, 1971) also found similar visual field dominance effects, with reduced right visual half field (VHF) superiority for digits in left-handed FS+ than FS-.

Studies of familial sinistrality effects on complex cognitive functions have yielded extremely mixed results, likely reflecting the statistically underpowered nature of many studies. Considering the moderate heritability (~20-30%) of non-right-handedness, unreliability of self-report and problems introduced by variable family size (Bishop, 1990), sample sizes of several hundred subjects are needed to attain acceptable statistical power (>.8). Power can be increased substantially with the use of non-categorical measures of familial sinistrality (Corey & Foundas, 2005), but these are not widely used. Even when more genetically informed familial handedness measures are used, these are sometimes based on a particular theory of genetic transmission and expression, thus confounding familial handedness effects with a specific, and likely incorrect, genetic model (e.g., McManus, 1995).

Two major genetic models of handedness have been proposed: Annett's Right Shift theory and McManus's Dextral Chance theory. Both models propose that a single locus, dominant for left-hemisphere specialization, controls asymmetry, with random factors or minor alleles producing right-hemisphere shift.

The Right Shift theory (Annett, 1985) proposes that a single gene, rs, handicaps language processing in right hemisphere (through unspecified mechanisms). The majority of the population is expected to be rs++, homozygous for the right shift allele, and thus strong right-handed with left-language hemisphere. Heterozygotes and those lacking the allele (rs--) have a reduced left hemisphere handicap and lateralization becomes subject to random factors. Later versions of the right shift theory include an 'agnosic' modification to the right shift gene that removes the specificity to the right hemisphere in an attempt to account for the possible links between left-handedness and autism and schizophrenia (Annett, 1999).

The Dextral Chance model (Annett & Alexander, 1996; McManus, 1985; 1985) proposes dextral (D) and chance (C) alleles (the latter being the minor allele). Only one allele (with equal chance of being expressed) contributes to the phenotype in this model: the D allele produces right-handedness and left language lateralization; the C allele produces random handedness and lateralization, independently.

Neither the Dextral Chance or Right-Shift theories have been supported by complex segregation analysis of family and twin data (Medland et al., 2009; 2006), nor have candidate genes for handedness been robustly identified. The absence of a candidate gene, despite genome wide association efforts, suggests that a simple, single locus model of direct genetic influence on handedness is inadequate and complex polygenic models of small effects (Francks et al. 2002; 2007; Medland et al., 2009) should be pursued.

Rather than relying on single-gene models of handedness, we have applied a more general Bayesian multifactorial model to estimate the genetic effects of

familial handedness in subjects. Categorical phenotypes can be mapped to a continuous latent variable using a standard multifactorial threshold model (Falconer, 1965), a particularly useful method when Mendelian inheritance patterns are not observed, as in handedness. Under this model, the probability of expressing a phenotype in a given category is function of an unobserved liability for a phenotype, in part the sum of additive genetic effects at an unknown number of loci. A variety of maps from liability to phenotype are possible; we use the simplest case in which a phenotype is categorically expressed (or not) if the liability is above (below) a threshold[2]. We have applied such a binary threshold model to proband-reported handedness pedigrees, estimating genetic effects and latent liabilities (see Sorensen & Gianola 2002 for a technical description of the algorithm). This model produces two useful results: an estimate of the heritability of handedness under a basic genetic model and estimated liabilities (and underlying genetic effects) for our experimental subjects. As expected, this method estimates the heritability[3] of handedness at h^2=.22-.36 (95% CI), consistent with Medland's (2009) maximum likelihood estimate of .20-.27 from a much larger twin study.

The use of estimated liabilities, like other methods of quantifying the degree of familial sinistrality (Corey & Foundas, 2005; Karev, 2010), yields a substantial power increase over dichotomous FS+/- methods and does so with minimal genetic assumptions. In addition, the Bayesian nature of our method produces liability distributions, rather than point estimates of familial sinistrality. This not only produces an implicit measure of uncertainty for each individual, but also avoids the common confounding of familial sinistrality measure and family size (Bishop, 1990), since dispersion, rather than the mean, is largely affected by family size. Emerging results from our laboratory using this measure, in conjunction with EEG measures promise to identify familial handedness effects more robustly than previous behavioral studies. In an event-related potential (ERP) version of the Townsend et al. (2001) word probe study, we have found evidence for variability mediated by familial sinistrality in the lateralization of the P2 ERP component, a possible marker for early lexical processing (Hancock and Bever, 2010). This lends initial support and validity to this approach, and to its significance for functional processing of language.

Of course, there are differences in neurological organization mediated by familial sinistrality in modalities other than language. For example, we recently found that an early right hemisphere negativity (ERAN) to musical anomalies is significantly stronger in FS- than FS+ right-handed subjects (Sammler et al 2012). The same FS- subjects showed only a trend for a stronger early left hemisphere negativity (ELAN) to grammatical anomalies. However, almost all FS- subjects showed both an ERAN for music and an ELAN for language, while almost no FS+ subjects showed this differential pattern. This suggests further that the neurological organization for complex behaviors is less differentiated and

[2] Since liability is unobserved, a threshold for a binary trait may be chosen for convenience, e.g. zero.

[3] Heritability here is the ratio of variance explained by genetic factors to variance explained by genetic, familial and environmental factors.

lateralized in FS+ right-handers (for recent empirical findings related to our research, see also Fisher et al 2012; Hancock 2012).

Why are there these effects of familial sinistrality? In the case of language, our recent findings lend support to Bever et al's (1987) hypothesis that lexical representations are relatively more available in the right hemisphere in FS+ people. That hypothesis reasoned that the lexicon may be more susceptible to widespread neurological representation than syntactic processing: the latter is more demanding computationally, and thus may be relegated to the computationally more powerful hemisphere. But if FS+ people have less lateralized brains, as suggested by various facts, this would allow for more right hemisphere representation and processing for the simpler aspects of language, in this case the lexicon. In a more general framework, the SNR concept of the basis for cerebral asymmetries would suggest that genetically-mediated more equilateral dopamine D2 activity in FS+ people reduces the bias towards left hemisphere language function typical in FS- people. Under this model, non-linguistic effects of familial handedness are also expected, based on the extent to which the basal ganglia are involved in non-linguistic tasks.

5. Implications of genetic variation in language organization and representation.

The empirical premise behind the differences between FS+ and FS- people is that FS+ people have reduced left hemisphere lateralization, and correspondingly weaker differential lateralization for language and other complex behaviors: in some cases, this (by hypothesis) initially quantitative difference results in apparent qualitative categorical effects. There are several different kinds of implications of our findings that support these results.

a) Implications for clinical research and therapies.

Virtually every clinical study of language dysfunctions and special language behaviors reports the handedness of the patients. Yet, despite Luria's classic findings on aphasia in FS+ patients, and the established association between familial left-handedness and psychiatric disorders, almost no studies of language dysfunction report familial handedness of the patients. Our 30 years of behavioral research and our recent modeling and imaging results argue strongly that it is critical to differentiate patients according to the familial-handedness-based likelihood that they are, or would have been, left-handed. Our current model of family pedigree effects offers an opportunity to enrich clinical research in this way.

b) Implications for experimental research and theory.

Psycholinguistic behavioral and neurolinguistic research continues today in attempts to build models of language acquisition and language performance. The behavioral differences between FS+ and FS- people we have outlined is sufficient reason alone to keep track of this variable: if FS+ people consistently access lexical items more readily than FS- people, and conversely for syntactic patterns, this will surely interact with many specific kinds of experiments. Our recent neurological findings go further to substantiate the importance of familial

handedness, since today's model building often refers to potential neurological concomitants of the postulates of the models.

c) Implications for the genetics of handedness: a refined phenotype

As we have noted, remarkably little is known about the genetics of left-handedness, despite its frequency and substantial heritability. In part this may be because handedness in general is multiply determined; it is also made more difficult to study because at least some left-handedness has been shown to be the result of acquired brain damage in people who are genetically right handed: conversely, many "right" handed people may be genetically left handed, but forced by social pressures to be right-handed. In sum, the phenotypic differentiation of left and right-handedness is not sharp, which complicates any search for specific polymorphisms or other genetic effects that influence handedness. An outcome of our research, using the model that estimates additive genetic effects associated with left-handedness as a function of family background, will be to sharpen the cognitive and neurological phenotypes of explicit left-handers, as well as explicit right-handers, with high and low genetic loadings for left-handedness. The result will be a better chance that DNA assays will reveal polymorphisms associated with handedness phenotypes than current case-control studies that consider only phenotypic hand preferences.

d) Implications for linguistic theory and the biological foundations of language

Finally, it is clear that there are different mechanisms for the expression of language in behavior, at least in the quantitative contributions and interactions between the hemispheres. This raises the question for linguistic theory and the genetics of language as to whether the quantitative differences result in actual qualitative differences in how language is represented neurologically and processed computationally. To put it bluntly:

i) is there more than one "normal" form of neurological representation for language?

ii) if so, is there more than one "normal" computational architecture for grammars?

iii) if so, is there more than one normal system for language behavior?

To decide the answer to (i) and (ii) requires a fuller analysis of what kind of lexical information is in fact relatively strongly represented or accessed in the right hemisphere in FS+ people. It could be information directly relevant to syntactic representations, such as lexical category, morphological structure, and relevant to phonological theory if it is represented in abstract phonological terms. In this case, we would have to conclude that indeed there is more than one normal form for neurological representation of language. On the other hand, the relevant "lexical' information in the right hemisphere of FS+ people could be associative information, information that would facilitate lexical access in behavior, but not be directly relevant for syntactic computations.

There are corresponding options for the implications of the differences for the actual structure of linguistic grammars. For example, a current controversy in linguistic theory has to do with whether lexical representations are simply special cases of idioms which in turn are special cases of sentence constructions and

conversely (e.g., Goldberg, 1996; Boas and Sag, 2012; Fillmore et al. 1988; see also Culicover and Jackendoff, 2006). On such theoretical views of grammar, different representational systems for relating the lexicon to larger compositional structures would definitely imply different kinds of computational architectures in different groups of people.

The largest question has to do with the implications of our findings for the causal relation between neurological structures and the structure of language. It is often implicit in biolinguistic discussions that a critical contributor to the structure of language is in the details of the biological basis for its acquisition, representation and use. On this view, the biopsychoneurological basis for language exists (whether by exaptation, saltation, or selection) in such a way to make possible language as we see it neurologically organized Our findings of the considerable lability of that organization suggest another possibility: that the basis for language lies not in any specific neurological set of centers and connections, but in the availability of various cerebral components that can carry out the kind of computations required for the external mapping of sequences of internal propositional relations.

This view is a particular implementation of the recent "minimalist" program, on which syntax is a direct expression of a system that efficiently relates propositional structures to externalized serial representations (Chomsky, 2003; Boeckx, 2006). On this view, the neurological organization for the best mapping system follows otherwise available computational centers and connections between them – the neurological organization in each case is itself the best available implementation. But what is the "best" implementation will differ as a function of larger tendencies and constraints on how the different computational components of the brain are "best" connected. Our research suggests that there is systematic normal variation in what is "best".

There are at least two implications of this idea for the biological foundations for language. The more conservative assumption would be that all people share a fundamental form of neurological capacity for language, but differ in the emphasis on lexical versus compositional mapping processes. This would mean that attested languages must convey substantial structural information both in their lexicon and in syntactic patterns, to accommodate to each of the major normal populations. This may underlie the apparent fact that languages are often structurally redundant in the corresponding way – both lexical and compositional structures are evident in actual sentences.

A more radical interpretation of the normal variation in neurological organization for language is that the unique biological capacity for language is rooted more deeply in human neurophysiology than in specific computational centers and connections between them. While this may seem mysterious or at least radically speculative, stranger things have turned out to be true. (For more extensive discussion of these issues, see Bever (in press)).

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