Uniquely human developmental timing may drive cerebral lateralization and interhemispheric collaboration

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Abstract

Cerebral lateralization is intertwined with virtually every cognitive function that we think makes us human. Yet a clear dichotomy has never been explained: lateralized processing suggests independent, local development of neural circuits, but the complementary nature of lateralized functions and extremely strong functional coupling between homologous areas suggest robust interhemispheric interactions. Here, we review literature and present modeling evidence that this dichotomy can be explained by the uniquely steep trajectory of human post-natal brain growth. This drastic volumetric change cause most long distance, interhemispheric connections to be more unreliable than shorter, intrahemispheric connections, leading to lateralization. Strong interhemispheric collaboration is enabled by the later maturation and myelination of long-distance callosal connections. We also review and reanalyze a well-cited modeling paper (Ringo, Doty, Demeter, and Simard (1994)) thought to show a relationship between the degree of hemispheric coordination and length of conduction delays, showing that previous claims have a clear alternative explanation.

Keywords: corpus callosum; lateralization; asymmetry; conduction delays;

Introduction

A single concept, supported by a single paper, has dominated thought as to the origins of cerebral lateralization. The modeling work of Ringo et al. (1994) has been exclusively and extensively cited to support the notion that large magnitude conduction delays, due to the large human brain size, enable cerebral lateralization. This *delay magnitude hypothesis* has intuitive appeal, as it supports another long-held notion: that some combination of large brains and functional lateralization have made us human.

There is no denying the importance of functional lateralization in human cognitive abilities; we are functionally lateralized in virtually all cognitive functions that we think are special to our species, including language, high-precision manual use of tools, spatial processing abilities, and even our emotional processing (Gazzaniga, 2000; Craig, 2005). There is also no denying, however, that the lateralized hemispheres are also tightly coupled in terms of both their complementary abilities (Gazzaniga, 2000; Hellige, 2006) and their functional coupling (Stark et al., 2008). This dichotomy is simply not captured by the delay magnitude hypothesis. Nor does the hypothesis account for the anatomical and functional asymmetries that appear throughout the animal kingdom (Rogers & Andrew, 2002; Rogers, 2009) in organisms with small

brains. Whether or not the delay magnitude hypothesis is correct, it certainly is not complete.

We hypothesize that functional lateralization is not caused by the magnitude of conduction delays. First, we'll review literature that supports our hypothesis. We'll present a re-analysis of Ringo et al. (1994) that severely restricts the scope of their results. We'll propose a new hypothesis that the developmental trajectory of human brains enables functional lateralization–specifically, that the vast and accelerated post-natal expansion of brain size and delayed maturation of the corpus callosum causes *unreliable* timing of interhemispheric information in pre-adult humans. We'll review literature supporting this hypothesis, then we'll present our own model supporting the plausibility of our developmentalhypothesis. Finally, we'll summarize our results and discuss implications of our findings to the general phenomena of lateralization, asymmetry, and cognition.

The failure of conduction delay magnitude

Callosal axons are especially long in humans, due to their need to traverse through our large, highly gyrified brains to connect to the opposite hemisphere. Because the average conduction velocity of axons does not sufficiently compensate for the additional axon lengths when compared to smaller-brained animals, the resulting interhemispheric transmission delay over the majority of callosal axons is longer in human brains. The delay magnitude hypothesis suggests that this increased delay would cause less interhemispheric collaboration and therefore enable cerebral asymmetry.

While the anatomy and physiology of callosal axons is well-established, their seemingly intuitive effects on interhemispheric collaboration is supported by a single model in a single paper (Ringo et al., 1994). Here, we argue against the delay magnitude hypothesis in two parts. First, we present 4 results from the literature that are inconsistent with the delay magnitude hypothesis. Second, we show that the model itself does not support the hypothesis.

1. Increased interhemispheric collaboration is associated with an increase in *slow* fibers

Larger corpus callosum size is associated with less lateralization. This is true for regions of the corpus callosum, as well as the corpus callosum as a whole. The midbody of the callosum, which carries fibers to and from motor

cortex, is larger for individuals with less lateralization in handedness (Witelson, 1989; Luders et al., 2010). Callosal cross-sectional area is proportionally larger for left-handers (Witelson, 1985), who show less functional lateralization than right-handers.

Within humans, larger corpus callosum size is associated with a larger number of thin fibers, not with the thickness of fibers (Aboitiz, Scheibel, Fisher, & Zaidel, 1992). This suggests that those with more interhemispheric collaboration have significantly more *slow* fibers–just the opposite of what the delay magnitude hypothesis would predict.

2. Homotopic areas show functional coupling

The corpus callosum largely connects corresponding (homotopic) areas between left and right cerebral hemispheres. Thus, according to the delay magnitude hypothesis, homotopic areas connected with slow, thin fibers (Aboitiz & Montiel, 2003) should show weak functional connectivity. In fact, this is not the case at all. For example, when examining interhemispheric correlations through resting-state fMRI, Stark et al. (2008) found very strong interhemispheric correlations between association areas. Reduced interhemispheric coherence (measured with EEG) at locations away from primary sensory/motor cortices has been measured in mental disabilities or diseases, such as dyslexia (Dhar, Been, Minderaa, & Althaus, 2010) and schizophrenia (Hoptman et al., 2012).

Hellige (2006) points out that functional specializations tend to be complementary. For example, visual processing of the left hemisphere seems biased towards high frequency processing, while the right hemisphere seems biased towards low-frequency processing (Sergent, 1982; Ivry & Robertson, 1998). If there is less interhemispheric integration due to more independent processing, then why would the two hemispheres show any type of relationship at all? The delay magnitude hypothesis offers no answer.

3. Longer delays may support coordination

The corpus callosum in larger brains doesn't simply have longer conduction delays; it also has a broader range of conduction delays. Innocenti (2011) reviewed data suggesting that a broader range of conduction delays supports a broader range of oscillations across the corpus callosum (Caminiti, Ghaziri, Galuske, Hof, & Innocenti, 2009), which may increase the stability of those oscillations (Roberts & Robinson, 2008). The current belief is that these oscillations are necessary for binding of information between two distant cortical areas (Fries, 2005); stabilization of inter hemispheric oscillations would presumably enhance interhemispheric communication. Thus, longer delays may be associated with improved ability to coordinate interhemispheric integration.

4. Shorter delays are detrimental in development

Many ideas of how the human brain may be unique have been debunked, including suggestions that the human brain is specially gyrified or has a unique fundamental asymmetry. Human brains are clearly unique developmentally–as precocial

mammals (born with our eyes open), we are the only species known to extend the accelerated rate of prenatal brain growth well beyond birth (Martin, 1983). This means that the rate of brain growth is especially high in humans.

Lewis and Elman (2008) used a version of Ringo et al.'s model to show that, the steeper the developmental brain growth curve, the more detrimental interhemispheric connections are to learning. This is due to the fact that, as brain size changes more quickly, the conduction delays change more as well, and those larger changes are more detrimental to learning. As their model "matured", even though the magnitude of delays were longer, because they were more stable, they promoted interhemispheric collaboration.

The delay magnitude hypothesis only addresses mature, adult brains. We suggest that taking a developmental angle to this problem may give more general results.

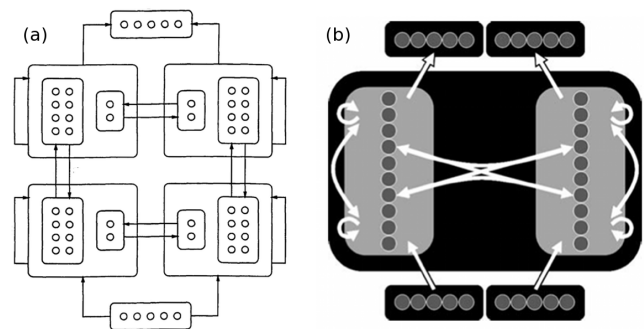## 5. Delays only affect the onset of communication

Figure 1: (a) The model architecture of Ringo et al. (1994). Information flows from bottom to top; left model hemisphere is to the left, and right model hemisphere is to the right. Arrows represent full connections between pre- and post-synaptic units. All delays are 1 time-step, except the interhemispheric ("callosal") connections, whose delay were varied across conditions. Note the shared output nodes, which allow an (unintended) path for fast interhemispheric coordination independent of the "callosal" connections.

(b) The model architecture of Lewis and Elman (2008) simplifies the structure and splits the inputs and outputs.

The model failed to control all interhemispheric transfer
Although Ringo et al. aimed to separate interhemispheric communication through long conduction delays, their model setup failed to do so (Fig. 1a). In addition to their "callosal" connections that were varied with short and long delays, their model also had converging connections from the hemispheres to a shared bank of output nodes, whose delays were always short. Thus, even if they re-trained their models without any "callosal" connections, the hemispheres would still show interhemispheric dependence; one hemisphere would not be able to complete the task without the other.[1] This issue is

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[1]We in fact verified this through simulations using their model.

an important confound in interpreting their results.

This issue was addressed in the only paper to follow-up the Ringo et al. study, by simply splitting the output nodes into two separate banks (Lewis & Elman, 2008), as depicted in Fig. 1b. All modeling work in this paper uses this same split-output architecture.

The results are often misunderstood and misinterpreted

Fig. 3 describes the Ringo et al. methods; Fig. 3a shows the original results. Citations to this paper are often made to support the notion that functional lateralization is inevitable, given the human brain size. This is a misrepresentation of the Ringo et al. results. In the paper, the authors only claim that lateralization *at short settling times* is caused by long delays. Tasks that allow "multiple passes" across the callosum were interpreted to show indistinguishable results across delays.

In fact, the original Ringo et al. results do not show anything except a *static delay* in interhemispheric coordination, of exactly the value of a single pass across the model corpus callosum. When we transpose the results from the two models by this value (9 time steps), the two models are indistinguishable (see Fig. 2 for details). This suggests that the only change in interhemispheric interactions found by their model is a simple, static delay in onset of interhemispheric communication, of a value equal to the time it takes for information to move from one hemisphere to the other. Note that this onset of activity may be mediated by "gigantic" callosal fibers–the largest 0.1% of fibers that do vary with brain size (Olivares, Montiel, & Aboitiz, 2001; Wang, 2008), an effect not captured in this model.

The model does not provide any evidence of qualitatively reduced interhemispheric interaction, only weak evidence for a (slightly) delayed one.

Our hypothesis: changes in timing reliability

The delay magnitude hypothesis fails to explain the basic dichotomy of how functional areas become both lateralized and functionally coupled. The developmental time-course of the thin callosal fibers suggest a two stage process to us: an initial stage where *all* associative / pre-frontal white-matter connections are immature, favoring local processing, and then a later stage were white-matter connections mature and come on-line. This pattern is well-supported in white matter in general, with some support in the corpus callosum as well (e.g. the anterior, frontal portion) (Jernigan, Baar, Stiles, & Madsen, 2011). Current imaging technologies can only detect particular types of maturational changes, with those that we outline below (relatively small changes in fiber diameter) currently excluded.

As mentioned above, previous work by Lewis and Elman suggested that unreliability in conduction delays could affect interhemispheric processing. However, though human postnatal brain growth is fast compared to other species, it is still quite slow compared to the time-scale of plasticity in the brain. Based on their work, we suggest that any factor that
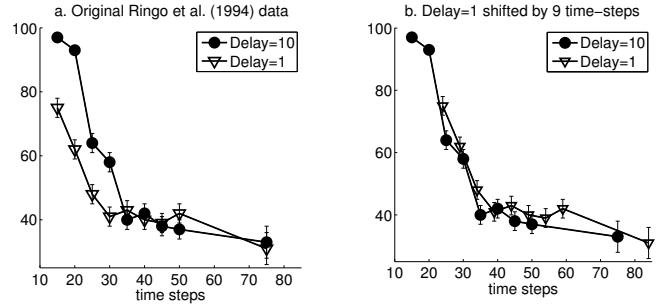

Figure 2: (a) Original data from Ringo et al. (1994), showing performance of networks after lesioning interhemispheric fibers, for two networks with different interhemispheric delays (1 time-step vs. 10 time-steps). Different networks were required to process across a range of times (x-axis; 15-75 time-steps), while they were trained to output binary strings that were associated with particular input binary strings. After training, "callosal" connections were lesioned, and network performance was measured. The network with the shorter interhemispheric delays (D=1; empty triangles) shows poorer performance on networks running for fewer time-steps (x-axis=15-30 time-steps); this was interpreted as indicating less interhemispheric interaction.

(b) We expect a network with delay=1 and delay=10 to have a difference of 9 time-steps to the onset of hemispheric interaction. We shifted the D=1 curve by 9 time-steps later (right on the x-axis) to allow us to visualize any qualitative difference in the interhemispheric interaction outside of this difference in onset. The overlap of the curves suggest that there is no other variation in interhemispheric communication besides this simple static delay.

disrupts the reliability of timing between cortical areas will have a detrimental effect on their coordination.

In reviewing the developmental literature, we did find one source of variability in the coordination of timing that is relevant to the corpus callosum: unreliable conduction delays in unmyelinated fibers with a thickness less than $0.5\mu m$ (Wang, 2008). Interestingly, in all adult animal species, callosal fibers are rarely found with a thickness below $0.5\mu m$ (Aboitiz & Montiel, 2003). However, neonates have a preponderance of such fibers[2] (Berbel & Innocenti, 1988; LaMantia & Rakic, 1990). While some of these small-diameter, unmyelinated fibers persist into adulthood, many become myelinated and all become more reliable by increases in their diameter (Aboitiz & Montiel, 2003).

Thus, we have a mechanism that fits all of our criteria: initially thin axon diameters decrease the reliability of the timing of information on a timescale relevant to neural processing (individual spikes), and follow a developmental trajectory that would initially support more independence (through less

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[2]This may be due to the need to compress brain size for successful birthing, and lower compressability of grey matter.

reliable timing), with developmental maturation tending towards interhemispheric collaboration (more reliable timing).

We hypothesize that association areas develop with decreased interhemispheric contributions, due to the unreliability of interhemispheric signals through small, unreliable fibers . This allows for the hemispheres to develop independently, which enables asymmetries to develop. Developmental changes in these fibers (diameter increases and myelination) make them reliable, and interhemispheric coordination comes online. In the discussion section, we'll expand this hypothesis to show exactly how it can address each of the questions laid out above.

Methods

We implemented a version of Lewis and Elman's model (Fig. 1b), which uses rate-coded leaky-integrator units. We suggest this is plausible: asymmetries are linked to higher-order cortical areas (Sergent, 1982; Schenker, Sherwood, Hof, & Semendeferi, 2007) which tend to interconnect over the corpus callosum using slow fibers (Aboitiz & Montiel, 2003) that are suggested to use rate-coding, rather than spike-time coding employed by thicker, faster fibers (Wang, 2008).

All connections in the model carry a delay; in all simulations cited and implemented, intrahemispheric delays are set to 1 time-step. Each hemisphere consists of 5 input units, fully connected to 15 hidden units. The hidden units have full recurrent self-connections, as well as full feed-forward connections to 5 output units. 3 hidden units from each hemisphere connected fully and reciprocally to each other as a model "corpus callosum"; these were the only shared connections between the hemispheres[3]. For all simulations, only these interhemispheric connections were manipulated.

As in the previous studies, the task for the network was to learn associations between input binary strings and output binary strings. We used a version of backpropagation through time appropriate for learning with conduction delays (Pearlmutter, 1989), for calculating our error gradients, and used resilient backpropagation for computing our gradient updates (Riedmiller & Braun, 1993). We used the sum-squared error function function[4].

We found that learning in the networks with published learning rates was slow; we also found that the degree of interhemispheric communication was dependent on parameters that were not varied in each study. We chose parameters to optimize learning speed, while balancing between interhemispheric and intrahemispheric dependencies[5]. Since the purpose of this study was to examine changes in intrahemispheric and interhemispheric processing, and not their actual magnitude, this seemed a reasonable approach.

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[3]Lewis and Elman used 10 hidden units and 2 interhemispheric units; we increased these numbers to facilitate task-learning. Select simulations run with their parameters showed similar effects.

[4]We used a gradient $((y-t)^3)$ that penalized local minima where a many patterns were learned quickly by sacrificing a few patterns which were not learned at all.

[5]Parameters: $T = 5, \alpha = 10^{-3}, \kappa = 10^{-2}$

For this study, we set a fixed total time (30 time-steps) and interhemispheric delay (10 time-steps).

Experimental Setup

We measured two values for performance: the **classification error** was the percentage of output nodes that were not within 0.5 of their expected output value ($+1$ or -1), and the **training error** was the average (sum-squared) error at each output node. For each of these values, the **lesion-induced error** was computed as difference between performance of the intact and lesioned network, as was done in Lewis and Elman. Again following that paper, our dataset contained both **intra**hemispheric patterns (50% of the input patterns), one hemisphere could determine its output without receiving any information from the other, as well as **inter**hemispheric patterns (50% of the input patterns), one hemisphere had to receive information from the other hemisphere to choose between 4 possible output strings. This allowed a more nuanced analysis of network performance differences.

Importantly, we operationalized unreliable conduction delays as Gaussian noise of the activity (instantaneous firing rate) transmitted over fixed (reliable) delays. In a rate-coding system, variation in the arrival of individual spikes, or a missing spike, leads to jitter in the instantaneous firing rate. We implemented this jitter directly in our rate-coded network as Gaussian noise on the activity, on a per-synapse basis. The jitter was a function of the delay at each synapse[6].

We ran 25 **no-noise** networks first (without any noise introduced on interhemispheric connections), to establish baseline measures. The networks were trained until they had zero classification error or until 1000 training epochs elapsed. Every 100 epochs, we measured error in the in-tact networks, as well as lesion-induced error. We then duplicated this procedure for 25 **noise** networks, which were identical except for having random Gaussian noise (2% of average unit activity) injected on the interhemispheric connections.

We thought that introducing Gaussian noise would cause interhemispheric information to be less reliable than intrahemispheric information, causing intra-hemispheric patterns to be learned more independently of the other hemisphere, and delaying learning of interhemispheric patterns. Therefore, we predicted that (1) the learning trajectory of the network with interhemispheric noise would be more gradual (i.e. have a smaller slope), and asymptote earlier. We also predicted that lesion-induced error would be lower in the *noise* vs. *no-noise* networks.

Results

Fig. 3a shows learning trajectories of classification error, for no-noise and noise networks on both in-tact and lesioned conditions. As predicted, learning in the noise networks was slower and reached asymptote at a higher error than the no-noise networks. We show these results for comparison to pre-

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[6]$noise = \mathcal{N}(2*10^{-4}*delay, 1*10^{-4})$; constant chosen such that μ is $\approx 2\%$ of average activity over all units.

vious papers. Fig. 3b shows the learning trajectories for learning error, on the same set of networks. Notice that, consistent with our predictions, lesion-induced error (the difference between corresponding intact and lesioned curves) is smaller for the noise networks vs. the no-noise networks on both measures. Interestingly, for learning error (a more nuanced measure of network performance), noise networks had less error in the lesioned networks than the no-noise networks.
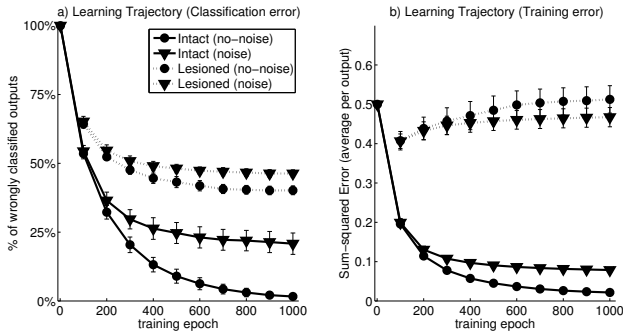

Figure 3: Changes in (a) classification error and (b) training error over training epochs, for noise and no-noise networks in-tact and lesioned conditions.

In order to examine these results more closely, we computed lesion-induced error on for the noise and no-noise networks for training error, then separated them into intrahemispheric and interhemispheric patterns (Fig. 4a). The noise networks showed less lesion-induced error for both interhemispheric and intrahemispheric patterns. Looking more closely at the differences between noise and no-noise networks (Fig. 4b), we find the surprising result that noise networks had much less lesion-induced error for intrahemispheric patterns than the non-noise networks, while the two had relatively equal levels of lesion-induced error for interhemispheric patterns.

These results indicate that the network with noisy interhemispheric fibers tried to accomplish the task (as much as possible) intrahemispherically, particularly in cases where both hemispheres are necessary to complete a task.

Discussion

We argued that current thought on interhemispheric integration fails to explain a basic dichotomy: how interhemispheric segregation may be necessary for developing asymmetries, but must be overcome to produce interhemispheric coupling found in adults. We showed that ideas based on the magnitude of conduction delays cannot explain interhemispheric segregation, nor can they explain strong interhemispheric coupling. We suggested that the especially steep developmental gradient of humans may hold the key. Our literature review revealed that two properties of these fibers might cause such a pattern: their changing length and their changing reliability in timing. The latter is relevant on the necessary timescale, so we focused our work here on examining the effects of changes in the reliability of the timing of information.
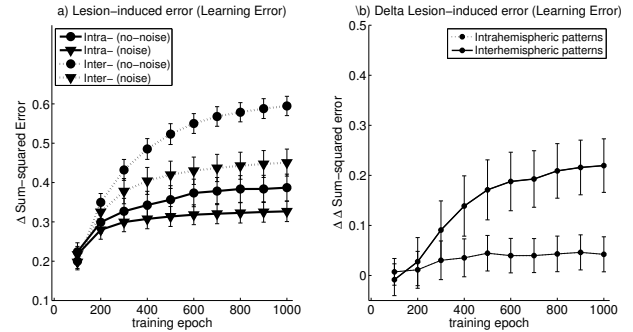

Figure 4: (a) Lesion-induced error for both noise and no-noise networks, split into interhemispheric and intrahemispheric patterns. (b) Difference between noise and no-noise networks for lesion-induced error; positive values mean more lesion-induced error in no-noise networks.

The results of our computational experiments showed that timing unreliability, in the form of Gaussian noise of our instantaneous firing rate, could induce more independent development of the cerebral hemispheres. This effect need not be related to the magnitude of conduction delay–but in mammalian brains it is: longer fibers have both longer delays and more jitter in their timing. These effects are also temperature-dependent (Wang, 2008); perhaps a mechanism leading to the large number of asymmetries found in cold-blooded species (Rogers & Andrew, 2002).

Our simulations also hinted at how asymmetry may emerge. We found that noisy callosal fibers led networks to try and use local, intrahemispheric processing, even for patterns requiring interhemispheric information. This type of early local processing is an indication that these networks may encourage developmental asymmetries.

Finally, we note a few recent papers on the benefits of noise in learning (Ermentrout, Galn, & Urban, 2008; Faisal, Selen, & Wolpert, 2008; Vincent, Larochelle, Lajoie, Bengio, & Manzagol, 2010). We intend to investigate whether initially noisy interhemispheric interactions facilitate both generalization and specialization of the hemispheres. Shared processing of highly salient features may allow each hemisphere to select secondary features that it is more specialized to process.

Acknowledgments

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