Autism and dyslexia: A spectrum of cognitive styles as defined by minicolumnar morphometry

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SUMMARY

There is a continuum of cognitive styles amongst humans, defined by differences in minicolumnar numbers/width and arcuate/commissural white matter connectivities. Specifically, it is the connectivity within and between modular cortical circuits that defines conditions such as autism and developmental dyslexia. In autism, a model of local hyperconnectivity and long-range hypoconnectivity explains many of the behavioral and cognitive traits present in the condition, while the inverse arrangement of local hyperconnectivity and long-range hyperconnectivity in dyslexia sheds light on that condition as well. We propose that the cognitive styles present in autism and developmental dyslexia typify the extremes of a minicolumnar spectrum in humans.

Encephalization

It has been proposed that the underlying cause for corticalization throughout the mammalian lineage is the addition of minicolumns to the neocortex [1, 2]. Concerning primate neocortical expansion, Jolly [3] and Humphrey [4] placed encephalization within the framework of the social intellect. Terminology Machiavellian intelligence by Byrne and Whiten [5], and more recently popularized by Dunbar [6] as The Social Brain Hypothesis, it states that neocortical expansion within the primate line has been driven by group size and complexity and, in particular, the complexity of social relationships. It is difficult to quantitatively measure "social complexity" because of the broad and super-inclusive nature of the term; however, it has been assumed that as group size grows so does hierarchical and social complexity [7]. Given this increase there arises the need for a brain which can accommodate the greater number of nuanced social relations in order to confer continuing reproductive success. In an attempt to concretely measure group complexity, Dunbar [7] found a direct correlation across various primate species between variables of group size and cranial capacity. Earlier work by Aiello and Dunbar [8] had also shown that because the neocortex ratio is a direct function of cranial capacity, it can be used to calculate neocortical volume. Therefore, the larger the group size (i.e., the greater the social complexity), the larger the neocortex. Of relevance to human studies, Homo sapiens are simultaneously one of the most socially complex species and have one of the greatest brain-to-body ratios [9, 10] (Table 1).

While measurements of encephalization have been used for studies of comparative anatomy between species, intraspecies phylogenetic comparisons have traditionally been avoided within the realm of the evolutionary sciences. This is largely due to the assumption that variations within a given species are either negligible or nonexistent and any reported variation is due to measurement error. As Ives and colleagues [11] argue, despite the statistical pitfalls, the expectation of negligible phenotypic variation of traits within a species is unrealistic. Therefore, some measurements and indicators of encephalization, while traditionally utilized for between-species comparison, can be just as applicable for studying encephalization from within the human population and not solely as tools of comparative anatomy.

Phenotypes which exhibit additive genetic/epigenetic effects across a single species typically resemble a bell curve pattern [12], with the majority of individuals regressing towards the mean. In the case of encephalization, while the broader term includes more than a single variable which cumulatively determines the level of neocortical expansion (e.g., the number of minicolumns and differences in arcuate and commissural projections [13, 14]), it may be possible to study these variables as a single generalizable unit due to their extreme interrelation [15]. For instance, a related increase in minicolumnar numbers and arcuate fibers and a decrease in commissural white matter tracts are noted in autism [16].

In every normal distribution, there will be two tail ends for the trait under study. In the case of minicolumnar morphometry, if this trait is in fact normally distributed across the human species, then one would expect behavioral correlates to its varied phenotypes given the sensitivity of the mechanics to even the slightest of changes. We suggest this is the case with autism and dyslexia.
hyperconnectivity and hyperreactivity within the circuit itself is sacrificed, shorter corticocortical tracts are reinforced, leading to a decrease in long-range connectivity. Due to an economy of wiring, limitations placed upon the gyral window may not be the only factor contributing to that of controls, indicating that the increase in cortical cell density is due to a greater overall number of minicolumns rather than an increase in cell numbers per column [17].

From a macroscopic viewpoint, there are alterations in the gyral window in autism. The gyral window is a plane through which afferent and efferent fibers connect the cortex to its underlying white matter [18]. Because an increase in the number of minicolumns also means an increase in cortical surface area [18], one would expect an increase in gyration and a decrease in the size of the gyral window, and this is what has been found with autism [19–22].

Herbert et al. [23] have reported an increase in the outer radiate white matter in autism. Casanova [24] suggests that this volumetric increase of arcuate projections is due to an increase in the numbers of minicolumns, with connecting fibers scaling to the number of modular units. Yet there is not only an increase in arcuate connectivity, there is a decrease in commissural tracts as well [21,25]. However, due to a volumetric constriction of the gyral window which affects the developing commissural white matter, the constraint of the gyral window may not be the only factor contributing to a decrease in long-range connectivity. Due to an economy of wiring, limitations placed upon total cell size, including the length, size, and number of local/distant connections, all plays an important role in determining total connectivity [26]. In other words, there is a “save wire” approach concerning the commissural fibers and number of minicolumns, in which the physical placement of neural connections and their numbers/size are determined by both the active use of synaptic connections [27,28] and other factors controlling the economy of wiring [29,30].

The relationship between inter- and intracortical connectivity is a delicately balanced one. As minicolumn numbers and short-range corticocortical connections increase, due to physical constraints placed upon the gyral window and save-wire constraints, the levels of long-range connectivity are reduced and modular integration is incrementally sacrificed [14,26,29,30]. As long-range connectivity is sacrificed, shorter corticocortical tracts are reinforced, leading to poorly integrated, hyper-independent modules which exhibit hyperconnectivity and hyperreactivity within the circuit itself [14,31–33]. On a behavioral level, this leads to poorer multimodal coordination during tasks which require a high level of functional integration, e.g., language and socialization, but can lead to unimpaired or even exceptional functioning during tasks that depend on individual modular functions [31]. Therefore, in autism, the underlying modular arrangement of the cortex promotes poorer intercortical integration and a specialization of functions, leading to the cluster of behavioral traits that typifies the condition.

### Autism

While it is currently defined via behavioral criteria, recent research has brought to light some basic cytoarchitectural characteristics which underlie the cluster of behaviors known as autism. Casanova et al. [16] have found significant differences in the minicolumnar morphometries between the brains of nine autistics and nine matched controls. Compared to controls, autistics exhibit reduced minicolumnar width and peripheral neuropil spacing and increased mean cell spacing. Despite the increase in intracolumnar cell spacing, the number of cells per minicolumn appears comparable to that of controls, indicating that the increase in cortical cell density is due to a greater overall number of minicolumns rather than an increase in cell numbers per column [17].

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### Dyslexia

Like autism, dyslexia is currently defined via behavioral criteria. While some agencies such as the World Health Organization and the American Psychiatric Association fail to differentiate between reading disabilities in comprehension and those in phonological decoding [34,35], the literature on developmental dyslexia has consistently defined the condition by deficits in the latter [36,37]. As with autism, research investigating the cytoarchitectural underpinnings of dyslexia has found characteristic differences in minicolumnar arrangements and connectivity as compared to controls.

While minicolumnar and peripheral neuropil width are all reduced in autism as compared to controls, dyslexics exhibit increased minicolumnar and neuropil width [38]. On a macroscopic level, reduced brain size and gyration have been noted [39] and as would be expected with a decrease in cortical surface area, an enlargement of the gyral window is present in dyslexics [40].

Because lesser constraints are placed upon the gyral window in dyslexia as compared to autism and control groups, a greater number of commissural fibers are possible, leading to an increase in volume of tracts such as the corpus callosum [40–42]. Additionally, a lesser number of minicolumns necessitates fewer local connections if the inner and outer radiate compartments in dyslexia scale respective to the reduction in minicolumns, which they appear to do [40]. With a reduction in local connections the cortex decreases its feature extraction capabilities which can cause, amongst other traits, a deficit in phonological decoding: the defining trait of dyslexia, suggesting atypical corticalization. So while in autism the specific underlying modular arrangement promotes poorer global integration and an increase in modular specialization, the modular arrangement in dyslexia produces an increase in global integration and a decrease in feature extraction which cause the classic deficits in word recognition.

### Discussion

Conditions defined by the fluid continuum of intra-/intercortical connectivity will also present on behavioral and cognitive continuums and therefore should not be considered discrete conditions unrelated to the general population [43–45]. Instead, as proposed in this paper, underlying aspects of conditions like...
autism and dyslexia should be considered as polar extremes surrounding the full human bell curve of minicolumnar morphometry. As Casanova and coauthors [38] state, “it appears that minicolumns exist within a phenotypic spectrum that intertwines the inhibitory/excitatory flow of neocortical information with a tweaking of the signal-to-noise ratio relevant to feature extraction” (p. 110). Because intra- and intercortical coordination is a finely tuned relationship of these signal-to-noise ratios [46], with extreme signal at one end of the bell curve and weak signal at the other, extremes of either pole can disrupt functional fluidity. This can cause two people to exhibit similar skill profiles despite opposing underlying etiologies, e.g., autism and dyslexia. And in this respect, it is plausible to propose that these two conditions simultaneously share aspects which are cortical inversions of one another and yet can also explain why some autistic people may present with various reading disorders similar to those seen in dyslexia.

With this in mind, the authors have proposed a spectrum of cytoarchitectural morphometry in humans, where variations within this spectrum are characterized by aspects which are key to mammalian encephalization; namely, alterations in the number of radial units to the neocortex. As proposed by various evolutionary theorists, encephalization amongst primates has been driven by social competition selecting for increasingly intelligent organisms which can better accommodate the accruing complexity of social demands.

Within a given species a range of phenotypic variety is expressed. In humans in particular, variations in the minicolumnar phenotypes affect cortex-wide connectivity, their numbers a factor in determining both arcuate and commissural white matter numbers and size. This connectivity determines the levels of parcellation and specialization of modular circuits within the neocortex, affecting the level of neocortical integration versus independence of regions. Ultimately, this integration versus independence affects behavioral phenotypes in characteristically specific ways. As proposed by various evolutionary theorists, encephalization amongst primates has been driven by social competition selecting for increasingly intelligent organisms which can better accommodate the accruing complexity of social demands.

As far back as 1983, Frith and Snowling [47] suggested that autism and dyslexia were complementary in their reading deficits, despite that they did not propose the two conditions to be neurological complements. While we have presented a more complicated behavioral picture of these two conditions, one in which dyslexia can both stand alone and co-occur with autism, we do not propose behavior to be so simply categorized. However, aspects of the cytoarchitecture which underlie these conditions seem to present with an elegant dichotomy, one which is reminiscent of the human trend as well as the larger mammalian one.

Conflicts of interest statement

Neither author has a financial or personal relationship with another person or organization that could inappropriately influence (bias) her or his work. This work was prepared without sponsorship from any funding agency.

References


