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Consistency and variation in the placement of cortical folds: A perspective

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ABSTRACT

Cortical folds, known as gyri and sulci, are prominent features of the human brain that play a crucial role in its function. These folds exhibit both consistency and variation within and across individuals and species, presenting a scientific challenge to our understanding of the underlying mechanisms. In this perspective paper, we summarize current knowledge about fold development and placement. We discuss the temporal and anatomical differences between primary, secondary, and tertiary folds, highlighting the consistency of primary folds and the increasing variation in later-developing folds. We explore the biological and mechanical factors that influence fold placement, including gene expression, tissue growth, axonal tension, curvature, thickness, and stiffness, which likely work together in a complex, coupled manner. We also highlight the need for advanced computational modeling approaches to unravel the mechanisms of precise placement of primary folds and further our understanding of brain complexity.

Statement of significance: Understanding the factors driving both the consistency and variation in fold patterns is essential for unraveling the functional implications and potential links to neurological and psychiatric disorders. Ultimately, gaining deeper insights into fold development and placement could have significant implications for our fundamental understanding of the brain, as well as mental health research and clinical applications.

1. Introduction

Much like the rest of our bodies, our brains have common anatomical features shared between nearly all humans, but also individual differences, due to genetic and/or environmental factors, that set us apart. Recently, there have even been suggestions that the pattern of cortical folds in each brain is as unique as a snowflake (Fig. 1) or a fingerprint, and could be used to identify individuals [1]. However, the shared features are also very important. Interestingly, cortical folds exhibit striking similarities not only among humans, but across our primate order and even throughout mammalia [2]. Recently, a community of neuroscientists, physicists, engineers, and others have been working to develop models of brain development that capture the contributions of genetics [3,4], cell behavior [5,6], axon connectivity [7–10], mechanics [11–14], and other factors to the formation of cortical folds. One of the many challenges confronting these modeling efforts is the need to explain both the consistency and variation seen in cortical folding patterns within and across species.

In this perspective, we aim to summarize our current understanding of fold development and placement, and offer suggestions to overcome the associated scientific challenges. We first discuss the temporal and anatomical differences between folds, and the consistency, or lack thereof, that is seen in humans and mammals. Then we discuss the scientific challenges in the search for an explanation for both consistency and variation, and the drivers of fold placement that have already been suggested. Finally, we conclude with a brief look at the potential approaches that could lead to new understanding of the factors that drive both consistency and variation in fold patterns.

2. Background

Cortical folds emerge at different stages of gestation: The development of the human brain is a complex process that occurs over a span of several years [15], beginning during embryonic development and continuing through childhood and adolescence. Cortical folds are the result of surface area expansion, which outpaces the volumetric expansion; the folds accommodate this extra surface area and additionally enhance the efficiency of the connections between neurons. The primary inner and outer folds of the brain, called sulci and gyri, respectively, begin to form around gestational weeks 24–31 [16,17]. As the brain continues to develop, primary folds get longer and deeper, and secondary folds

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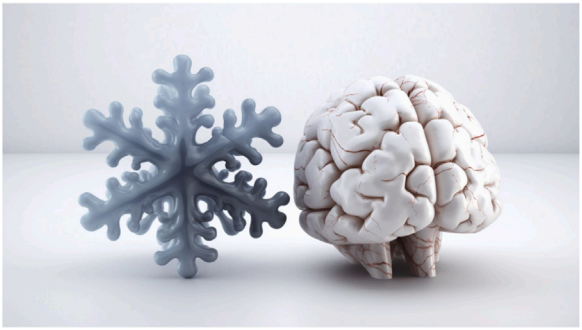

Fig. 1. Each brain, like a snowflake, has consistent primary structures alongside intricate variations in the fine details.

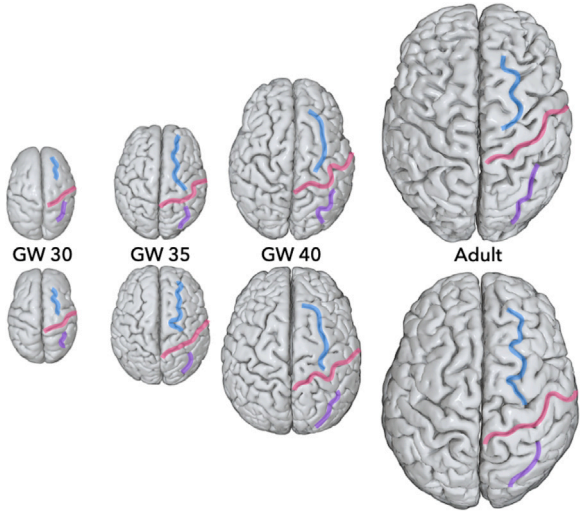

Fig. 2. Consistency and variation in fold placement in humans. Two representative human brains are shown at each timepoint: gestational week (GW) 30, 35, and 40, and adulthood. The central sulcus (pink) and intraparietal sulcus (purple) tend to be consistently placed throughout development and across individuals, while the superior frontal sulcus (blue) emerges later and shows more variation. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

begin to form from gestational week 32 to term [17]. Finally, around gestational weeks 37-40, tertiary folds begin to form, continuing to develop until adulthood [18,19]. These folds allow for even more intricate connections between neurons and facilitate higher-level cognitive processes such as reasoning and problem-solving. To some extent, the division between conserved and varied patterns falls along temporal lines, where early primary folds are the most consistent, and secondary and tertiary folds form later with more variation [20].

In this sense, primary folds are like the six sides of a snowflake, which is seen consistently across all specimens (Fig. 1). However, like the smaller branching patterns that make each snowflake unique, each brain also has its own variations in the secondary and tertiary folds.

Primary, secondary, and tertiary folds differ anatomically: In addition to the chronological differences, there are anatomical differences between primary, secondary, and tertiary folds [21]. Primary folds are often deeper [22] and buckle radially, forming gyri and sulci, while secondary and tertiary folds are shallower and tend to form more complex morphologies via in-plane folds and bends [23]. During development, primary folds also continue to deepen, increasing the overall folding amplitude of the cortex [21,24]. Secondary and tertiary folds are narrower than primary folds, with higher curvature, and tend to decrease the average wavelength of cortical folds. Tertiary folds are the shallowest and smallest of all folds [19,25]. Interestingly, in regional studies, no significant cortical thickness difference has been found between primary and tertiary sulci [25]. To distinguish between these developmental stages, researchers use surface measurements like sulcal length, width [17], and depth [25,26]; curvature [21]; surface area [26]; volume [18]; gyrification index; and folding wavelength. For instance, Dubois et al. [21] used spectral decomposition of mean curvature to determine emerging wavelengths of folding during development, and used that to classify developmental periods. Similarly, Mallela et al. [18] explored primary and secondary cortical folding patterns through a Jacobian volumetric analysis. No relative volumetric change was observed after gestational week 32, corresponding to the emergence of secondary folding patterns.

Consistency and variation are seen across different individuals: Primary folds are grossly conserved across individuals. For example, the central sulcus is a deep fissure that separates the parietal lobe from the frontal lobe and is responsible for some fine motor control of the hand. It is one of the most prominent structures of the human brain (Fig. 2, shown in pink), and shows high genetic heritability [19,27]. Another example is the intraparietal sulcus (Fig. 2, shown in purple), which is located at the lateral side of the parietal lobe and is important for coordination of perception, visual, and motor functions such as grasping. It emerges early in development, around gestational week 26 in humans and embryonic day 100 for crab-eating macaques [17], and its placement is mostly conserved across individuals while it consistently increases in length during development. This spatial stability of primary sulci, in both location and orientation, has been quantified by looking at average curvature maps [28].

Despite these similarities, brains are different enough that the idea of a unique 'brainprint' to identify individuals has been suggested [1]. Many people have attempted to understand the functional implications of differences in brain morphology; the interest in Einstein's brain, which may [29,30] or may not [31] be unusual, is an extreme case. But even setting aside Einstein, variation is seen across the human race, particularly in secondary and tertiary folds, which form later in development. For example, the superior frontal sulcus (Fig. 2, shown in blue) is highly adaptive, both functionally and structurally, and matures at different rates across individuals. Neither the length and shape of the superior frontal sulcus nor its location follow a consistent trajectory across individuals and development. Even among primary folds, their shape, length, and depth may vary across individuals [32]. This can be seen in the 'hand knob' of the precentral gyrus (adjacent to the central sulcus), which is responsible for the hand motor function, and is located more dorsally in right-handed individuals than left-handed [33]. Individual-specific sulci patterning are also evident for other primary sulci, such as the superior temporal and cingulate sulci [32].

Consistency and variation are also seen across different species: The similarities and differences seen between individuals are also noticeable across different species. Larger species generally have larger and more gyrencephalic (folded) brains compared to smaller species [34–36], as surface area increases much more than cortical volume [37]. But even as the degree of folding changes, gyral and sulcal consistencies, called homologies, are present among different species. In general, the shallow folds or dimples of the prefrontal cortices of non-human primates, for instance in the frontal eye field in rhesus macaques [38], have deepened over the course of evolution to form the deep primary sulci in humans [25]. The central sulcus, for instance, is also observed in non-human primates (Fig. 3), and even the central portion (the 'hand knob') is homologous in chimpanzees and humans, although there are also structural differences (i.e. surface area, depth) within primate species [39]. Sulcal organization is not random [40]; on the contrary, it is thought to follow a phylogenetic trend in primates [41]. Consistency is most noticeable in regions that evolved the earliest, showing evidence for evolutionary stability [42]. Conversely, differences between species

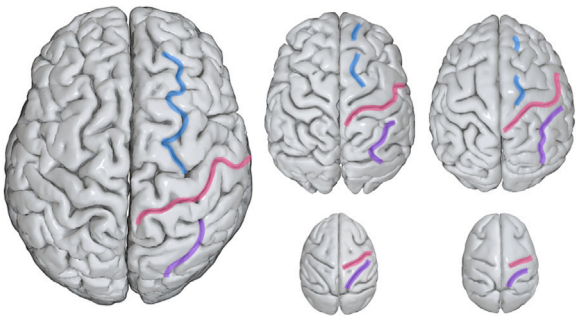

Fig. 3. Consistency and variation in fold placement in primates. Clockwise from left: human, bonobo, chimpanzee, tufted capuchin, and rhesus macaque. The central sulcus (pink) and intraparietal sulcus (purple) tend to be consistently placed across different primate species, while the superior frontal sulcus (blue) emerges only in more highly folded brains and shows more variation. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

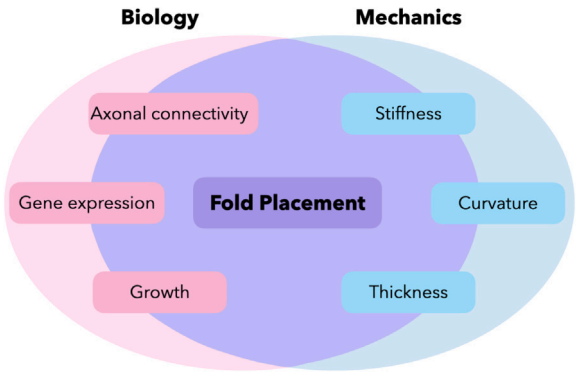

Fig. 4. A variety of biological and mechanical factors affect the placement of cortical folds. Biological factors include heterogeneous gene expression, the geometry and density of axonal connections, and heterogeneous expansion driven by growth. Mechanical factors include variations in tissue stiffness, cortical thickness, and curvature. These mechanisms are not necessarily competitive, but might be complementary and strongly coupled.

might reflect evolutionary changes. This can be seen in the primary sulci of crab-eating macaques and humans, which are grossly spatially and temporally homologous with the exception of the cingulate sulcus, which is associated with higher-order cognitive functions specific to humans [17].

As with inter-individual differences, the greatest variation across species is observed in tertiary folds. Tertiary sulci are found only in great apes, although some non-human primates have shallow dimples that are considered to be homologus to tertiary sulci [25]. Even within the great apes, only some species or individuals might have a given tertiary fold [40]. For example, the paracingulate sulcus is found in humans and chimpanzees, but not baboons, macaques, or gibbons, and even then only in 23% to 50% of individuals [43]. However, even within homologous sulci, patterns (e.g. length, depth, shape) can be highly variable across species. For instance, the superior frontal sulci of great apes are shorter and more irregular in shape in comparison to humans [44] (Fig. 3, shown in blue).

3. What factors drive fold placement?

The outstanding challenge to our deeper understanding of the form and function of primary, secondary, and tertiary folds is the multiple mechanisms that could plausibly affect the placement of cortical folds (Fig. 4). It is important to note that these mechanisms are not mutually exclusive; rather than being competing theories, they are likely complementary and highly coupled.

Biological mechanisms of fold placement: Multiple biological mechanisms have been proposed to explain the folding process, spanning the molecular, cellular, and tissue scales. At the molecular level, genes and gene expression have been identified as regulators of cortical folding [45]. Heterogeneous gene expression drives different levels of proliferation, differentiation, and migration of neural progenitors and neurons, which in turn drive the development of cortical folds [46]. Studies in ferrets and cats have long shown the consistent primary folds locations [47], and more recent genetic studies in humans and ferrets have shown the consistency of differential expression in hundreds or even thousands of genes underneath the sites of developing gyri and sulci [48,49]. In general, genes are thought to be a stronger driver of primary fold placement than secondary or tertiary, which are likely more environmentally influenced [27,50,51]. The former is seen in the high heritability of primary fold position and shape [20], while the latter is clearly seen in the differences between twins [52].

Relatedly, tissue growth is known to be heterogeneous across the cortex [53,54], potentially due to heterogeneous gene expression as described above, as well as other factors. Increased growth in one region makes that region more likely to form part of a gyrus [48,49,55–57] while gyri also likely grow more after formation [13].

On the cellular scale, axonal tension has also been suggested as a determinant of fold placement. It has been shown experimentally that axons are under substantial axial tension [58], and this tension has been hypothesized to drive cortical folding [7]. In an analysis of the macaque brain, it was found that a gyrus consistently formed along the border between two large interconnected areas. Based on this, it was theorized that strong axonal connection between regions leads to the formation of a gyrus, pulling the two regions closer to together, while a weaker connection allows the two regions to drift further apart on opposite sides of a sulcus [7,59]. While this hypothesis as a *major* driver of cortical folding has been challenged by later studies demonstrating contradictory stress patterns in the developing mouse [60] and ferret [61], axon tension – whether pathway-specific or ubiquitous tethering [62,63] – could still be a contributing factor to fold placement.

On the other hand, important cellular processes during development are known to associate with cortical folding [64]. It is well-known that radial glial cells provide a substrate and pathway for neuronal migration from the proliferative ventricular zone towards the cortical plate [65,66]. In gyrencephalic species along all orders of mammalia, the subventricular zone is comprised of two distinct zones: a thin apical (inner) and a thicker basal (outer) zone [67]. The proliferation of basal radial glial cells (bRGCs) differ between lissencephalic and gyrencephalic species. Intermediate (basal) progenitor cells are more abundant in the outer subventricular zone in gyrencephalic species [64,68], leading to further proliferation of bRGCs, which have processes extending to the cortical plate but not to the ventricular zone [64,69]. In lissencephalic species, the bRGCs directly produce neurons, while in gyrencephalic species they undergo multiple divisions (symmetric and asymmetric) to expand the bRGC population [70]. The abundance of bRGCs at the outer subventricular zone then causes tangential dispersion of migrating neurons in a divergent (fanning-out) trajectory through radial intercalation between classical apical radial glial cells [64,68,71], resulting in areal expansion of the cortex at the expense of thickness [67], which drives cortical folding [45]. Furthermore, the morphology of migrating neurons are vital; it is suggested that the processes of radially-migrating neurons are highly branched with wider angles for gyrencephalic species, for instance ferrets, facilitating tangential dispersion and cortical folding [72]. The divergent trajectory also prevents overcrowding of the migratory pathways and migration delay [64]. Further, divergent trajectories are significantly found under prospective gyri, but not sulci, providing evidence for their role in cortical folding and fold placement [64].

Mechanical mechanisms of fold placement: A number of studies have also shed light on the role of mechanical factors, such as curvature, thickness, and stiffness, in determining the placement of gyri and sulci. While cortical expansion likely drives the formation of cortical folds, the intricate patterns are highly sensitive to geometrical and mechanical perturbations [11], particularly the location of the folds. For example, folding initially occurs in regions of the lowest curvature [73]. Simulations on an ellipsoidal geometry resulted in a structure resembling the early central sulcus in fetal development [74], suggesting that the initial nonhomogeneous geometry of the fetal brain could strongly affect fold placement. Later, secondary and tertiary folds might be affected by the geometry of the primary folds that formed earlier [24,75].

Cortical thickness and stiffness are also crucial parameters in the folding of the cortex. Globally, cortical thickness correlates with the depth and frequency of sulci in both human and non-human primates [76–79]. A thick cortex tends to form fewer gyri and sulci [77,80,81], while a thin cortex tends to form many more folds. These correlations can be seen both in human pathology (for instance, lissencephaly and polymicrogyria) and in different species (for instance, the manatee's thick and barely folded cortex, and the dolphin's thin and highly convoluted cortex). Heterogeneous thickness, which could result from either differential gene expression or from mechanosensitive feedback [13], and stiffness, related to microstructural or cytoarchitectural differences, could also affect where folds form. A small region with greater thickness or lower stiffness than the surrounding cortex tends to end up on a gyrus [82]. Periodic or other nonuniform patterns of heterogeneities are thus capable of generating complex and hierarchical folding patterns [77,83]. Global mechanical properties, including viscoelasticity, likely also play a role in regulating folding morphologies across species. In general, a brain tissue with a higher average shear modulus would have more resistance to folding, whereas a more viscous tissue has the tendency of deformation under sustained force. However, these properties are dependent on factors like age, health condition, brain region, and microstructure, and have been found to vary based on different experimental protocols [84–86]. Nonetheless, an experimental study of viscoelasticity of mouse, rat and pig brains provide the direct comparable data [87]. The results indicate that the pig cortex is the softest of the three, which is consistent with the fact that the pig has a more convoluted cortex.

Coupling of biological and mechanical mechanisms: Of course, the relationship between biological factors and the mechanics involved is not unidirectional but rather fully coupled [13]. This type of coupling gives rise to a complex dynamic biological and mechanical system, in which gene expression, celluar mechanisms, and mechanical forces are fully coupled [as reviewed by [3]]. The human brain displays subtle variations in shape, tissue properties, and growth rates determined by biology. These small inter-individual differences can impact mechanical buckling – a nonlinear process that is sensitive to geometrical and mechanical properties – and potentially contribute to the variability observed in gyrification patterns. For example, recent modeling studies have explored the migration of neurons and associated mechanics, shedding light on the complex interplay between biology and mechanics [5,88–90].

4. Conclusion

A comprehensive, quantitative analysis of cortical morphology is intractable because of the sheer complexity and diversity of folding patterns [51]. While the exact placement of cortical folds may provide helpful information about brain development and functionality, many studies are more concerned with either global structural properties (e.g. average cortical thickness or gyrification index) or cellular-scale neuronal activity and circuitry. Even on the lobal or regional level, many analyses of growth, thickness, connectivity, etc. can be averaged or smoothed over a length scale that allows the exact position of folds to be ignored [12,22].

However, there is an urgent need to understand this and other aspects of the brain's complexity. The highly conserved patterns across individuals and species suggest that fold placement, particularly of primary folds, is tightly regulated and likely serves an important functional role. Increasing evidence suggests that the placement and patterns of folds are linked to meaningful functional differences across individuals and species [19,91]. Furthermore, deviations in cortical fold placement are associated with various neurological and psychiatric disorders, such as schizophrenia, autism spectrum disorder, and epilepsy [92–94]. Advanced knowledge in this area could help our understanding of mental health — etiology, diagnosis, prognosis, treatment, etc. [95]. For instance, disruptions in neurodevelopmental processes that result in changes in the primary folding pattern, perhaps via sulcal pits [20], could be detected in early fetal development [96]

While extensive computational modeling efforts have focused on the cortical folding process, only a few models have attempted to reproduce the consistent patterns and orientations of primary folds. Instability analysis in particular, whether implemented in a finite-element framework or carried out analytically, can only calculate the instability point and general pattern, and is intrinsically unable to predict the *location* of instabilities. Only a few studies have directly investigated realistic perturbations that might affect fold placement, such as initial brain shape [11,74,97], heterogeneous cortical growth rate [53], heterogeneous stiffness [57], and axonal connectivity [8].

Another obstacle to further understanding is the relative scarcity of expertly parcellated specimens that would be needed for the analysis of fold placement in actual brains. Segmentation refers to the separation of e.g. white and gray matter in magnetic resonance images, while parcellation refers to the identification of meaningful anatomical or functional units [98]. While segmentation can be performed semi- or fully automatically on a huge range of specimens using a number of different software options [37], parcellation methods are built on carefully labeled atlases of a specific group with limited variation — e.g. healthy adolescent and adult humans in the case of Freesurfer [99], or rhesus macaques in the case of CIVET-Macaque [100]. The identification of homologous gyri and sulci across species, for instance, is often limited by the challenge of identifying anatomically and functionally similar structures in dissimilar brains.

In conclusion, a deeper understanding of the mechanisms and importance of cortical fold placement hinges on advances both in the segmentation of actual brain specimens and in the realistic simulation of the biomechanical process of cortical folding. Multiple paths forward present themselves. For one, in the future it might be possible to spare neuroanatomists the painstaking work that has provided reliable parcellations up to now — as ever-more intelligent software packages are developed and trained on already available data, extrapolation to larger ranges of species, ages, and health status might become feasible. On the simulation front, many potential contributing mechanisms remain underexplored, particularly the interactions between multiple complementary or competing factors, regionally- or time-specific mechanisms, and pathways that might explain abnormal cortical folding in the case of certain pathologies. On the experimental front, one of the most important resources to answer questions about consistent and variable folding patterns is longitudinal imaging studies. While some longitudinal studies of infants do exist [101], there is currently not sufficient longitudinal data to validate existing hypotheses of folding locations. Longitudinal imaging of fetal brains could shed light on the temporal evolution of folding patterns and white matter microstructure changes. The evolution of folding patterns can be quantified in terms of volume, surface area, cortical thickness, and gyrification index [102], both globally and locally. Collaborations between experimentalists and computationalists could incorporate data on regional microstructure [103] into calibrated models in order to uncover the mechanism behind

consistent cortical folding patterns. As the next stage of brain development research, we call for an increase in longitudinal studies and data in the field. Using this integrated framework, researchers from diverse disciplines, such as neuroscience and biomechanics, could gain mutually beneficial insights and collaboratively tackle the challenges associated with the study of brain folding.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

No data was used for the research described in the article.

Declaration of Generative AI and AI-assisted technologies in the writing process

The authors used Midjourney to generate Fig. 1. After preparing the manuscript, the authors used Chat GPT to draft the abstract. After using these tools, the authors reviewed and edited the content and take full responsibility for the content of the publication.

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