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Review

Computational models of cortical folding: A review of common approaches

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ABSTRACT

The process of gyrification, by which the brain develops the intricate pattern of gyral hills and sulcal valleys, is the result of interactions between biological and mechanical processes during brain development. Researchers have developed a vast array of computational models in order to investigate cortical folding. This review aims to summarize these studies, focusing on five essential elements of the brain that affect development and gyrification and how they are represented in computational models: (i) the constraints of skull, meninges, and cerebrospinal fluid; (ii) heterogeneity of cortical layers and regions; (iii) anisotropic behavior of subcortical fiber tracts; (iv) material properties of brain tissue; and (v) the complex geometry of the brain. Finally, we highlight areas of need for future simulations of brain development.

1. Introduction

The human brain starts small and smooth before growing and folding into its characteristic shape between 25 and 40 weeks of gestation (Fernández et al., 2016). The form of the brain is the result of coupled biological and mechanical processes, whereby cells exert forces on the surrounding tissue, and in turn respond to the forces they experience. Alterations in cortical folding are associated with various neurological disorders, including schizophrenia (Wisco et al., 2007; Cachia et al., 2015), autism spectrum disorders (Kates et al., 2009; Monterrey et al., 2017), Williams syndrome (Essen et al., 2006), attention deficit hyperactivity disorder (Wolosin et al., 2009), and bipolar disorder (Sarrazin et al., 2018). Proper diagnosis and treatment of these disorders requires a deeper understanding of how the brain folds. Improved characterization of gyrification patterns would enable early diagnosis and potentially identify pathways for the development of effective interventions.

Scientists have studied brain morphology for well over a hundred years. Many of the early investigations focused on comparisons between humans and other primates and mammals (Baillarger, 1845; Le Gros Clark, 1945; Hofman, 1985; Welker, 1990), studying the allometric relationships governing brain morphology across species. For instance, it was found that brain size increases exponentially with body size (Jerison, 1973) and that the cortical surface area increases linearly with the volume of the brain (Baillarger, 1845), while the cortical thickness remains relatively constant (Hofman, 1988). Other early investigators focused on the geometry of the folded brain. They found that the volume of the cortical columns and layers remains consistent throughout the gyri and sulci (outer and inner folds, respectively) (Bok, 1929), and that folding aligns with directions of minimal curvature (Todd, 1982). Recently, cellular, molecular, and genetic factors of brain development have offered new insights to the study of cortical folding (Rakic, 1988; O'Leary et al., 2007; Rakic, 2009; Matsumoto et al., 2020; Franchini, 2021). For instance, studies have shown that tissue microstructure (Llinares-Benadero and Borrell, 2019; Alexander-Bloch et al., 2020) and gene expression (de Juan Romero et al., 2015) affect folding patterns and functional connectivity in specific regions (Gautam et al., 2015; Schmitt et al., 2021). In many cases, these investigations have been focused on not only understanding basic questions about the ontology of the brain, but also on the mechanisms responsible for cortical malformations and mental disorders (Ross and Walsh, 2001; Parrini et al., 2016; Kawasaki, 2017).

As the rapid growth and folding of the human brain takes place during the third trimester of gestation, *in vivo* experiments and even close observation are difficult. In response to this challenge, a number of computational models have been developed in order to represent the process of gyrification *in silico*, often accompanied by *in vivo* imaging, *ex vivo* dissections, and physical analogue experiments (Xu et al., 2009, 2010; Bayly et al., 2013; Tallinen et al., 2016; Holland et al., 2018). Computational modeling has greatly increased our understanding of brain development, providing powerful tools to simulate complex, coupled, and nonlinear physical and biological interactions in order to predict realistic folded morphologies (Tallinen et al., 2016). These

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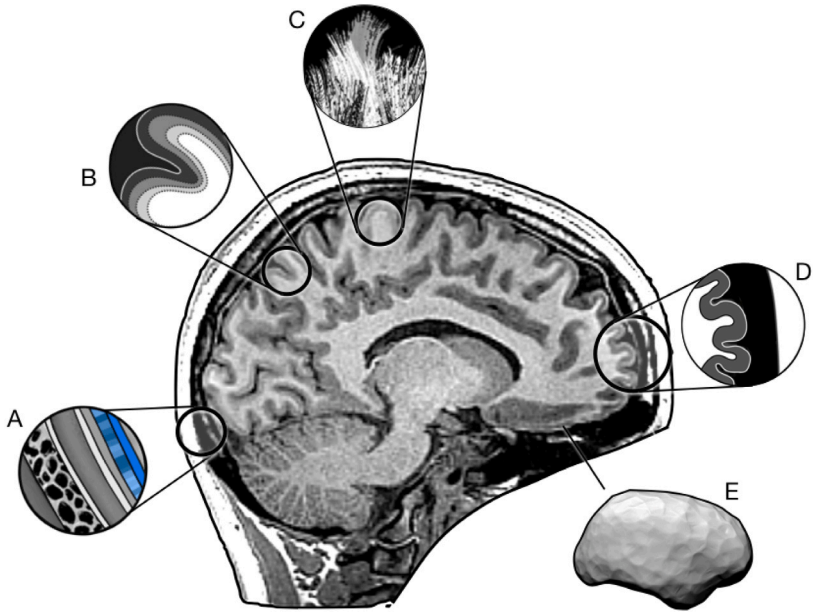

Fig. 1. Features of the human brain and head relevant to simulations of brain development. (A) Layers surrounding the brain (from outermost to innermost): scalp, skull, periosteal dura mater, dural venous sinus, meningeal dura mater, arachnoid mater, subarachnoid space, cerebrospinal fluid (shown in blue), and pia mater. (B) Cortex, consisting of six distinct layers (shown in shades of gray). (C) Axonal white matter fiber tracts, consisting of bundles of axons covered in myelin sheaths, connecting different regions of the brain and spinal cord. (D) Gray and white matter, distinct tissues with different properties. (E) Complex geometry of the brain, which features regions of high and low curvature. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

models have enabled researchers to test and evaluate different hypotheses and to observe the effects of multiple parameters, such as cortical thickness (Tallinen et al., 2014) and curvature (Toro and Burnod, 2005), on gyrification. *In silico* approaches have the advantage of being able to simulate long-term developmental processes quickly and at comparatively low cost.

Computational simulations of cortical folding require robust and scaleable numerical techniques, particularly given the material, geometrical, and contact nonlinearities that can arise. Many simulations are carried out using commercially available finite element software packages such as ABAQUS (Holland et al., 2015; Razavi et al., 2017; Wang et al., 2020a) and COMSOL Multiphysics (Xu et al., 2009, 2010; Filas et al., 2012; Bayly et al., 2013), while others use in-house codes (Toro and Burnod, 2005; Tallinen et al., 2014; Budday et al., 2014; Tallinen and Biggins, 2015; Tallinen et al., 2016; Holland et al., 2018). Commercial tools offer lower barriers to entry but place more limits on customization. For instance, researchers can innovate certain parts of the simulation or solution algorithm, such as UMAT for custom constitutive material behaviors in Abaqus Standard (Bayly et al., 2013; Holland et al., 2015; Wang et al., 2020a), but certain things are difficult or impossible in this framework, such as nonlocal behavior. On the other hand, custom codes offer infinite customizability to the problem at hand, although this flexibility is accompanied by the challenge of construction and maintenance. Such frameworks have been developed using multiple programming languages such as C++, FORTRAN, Python, MATLAB, Mathematica, etc. These languages have their own advantages and disadvantages; for example, MATLAB already has many useful built-in functions related to the finite element method, from gridding to solvers, while C++, FORTRAN, and Python are free and open-source. In addition to the model improvements discussed in this review, researchers are constantly working to increase the stability, efficiency, and accuracy of the applied numerical methods, along with enhancing the flexibility and usability of the available software.

Computational and experimental approaches need to progress in tandem, particularly for validation of numerical models. Computational models have been evaluated both qualitatively, through visual comparisons with brain morphology (Budday et al., 2015d), and quantitatively. From MR data, brain volume (de Rooij and Kuhl, 2018), cortical thickness (Holland et al., 2018, 2020a; Wang et al., 2020a), and fold wavelength (Heuer et al., 2019) can be calculated and used for both calibration and validation. In addition to comparisons with human brain morphology, ferrets (Tallinen and Biggins, 2015; Xu et al., 2009, 2010), non-human primates (Zhang et al., 2017; Ge et al., 2018), and organoids (Karzbrun et al., 2018) have been used. Non-human subjects also allow for a wider range of experimental assays, including cuts that reveal residual stress distributions in the brain (Xu et al., 2009, 2010). Furthermore, non-biological experiments such as swelling gels (Tallinen et al., 2016; Greiner et al., 2021) and stretched polymers (Holland et al., 2018; Budday et al., 2017) have also been used for calibration and validation of computational simulations.

In this review, we focus specifically on the implementation of theoretical and computational models of cortical folding. We begin with a brief overview of the underlying biology and mechanics of brain development (Section 2). Then we focus on five elements which affect brain development (Fig. 1) and how they have been represented in computational models: extra-cerebral tissues including the skull, meninges, and cerebrospinal fluid) (Section 3); gray matter (Section 4); white matter (Section 5); tissue properties (Section 6); and initial geometry (Section 7). Finally, we conclude and suggest a number of future research directions (Section 8).

2. Biomechanics of brain development

2.1. Biology of brain development

During early development, glial cells originating from progenitor cells in the ventricular zone travel between the ventricle and cortical plate, providing a scaffold of glial fibers on which neurons, produced from the same progenitor cells, migrate outwards (Rakic, 2009). These migrating neurons eventually establish the cortical plate, also known as the cortex or cortical gray matter (Fig. 2). In humans, cortical folding begins around the 25th week of gestation (Lagercrantz and Changeux, 2009), after the completion of neuronal proliferation and migration (Childs et al., 2001; Habas et al., 2012). After migrating, neurons develop dendrites and axons to connect to other cells. Along with the extracellular matrix, the bulk of the subcortical white matter is made

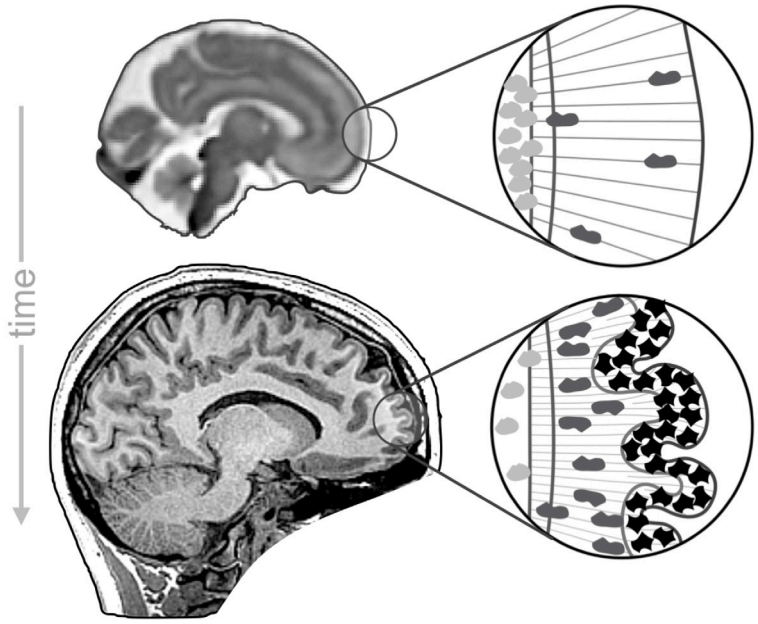

Fig. 2. Neurogenesis and cortical folding during development. Top: Early in development, neural progenitor cells (light gray) originate in the ventricular zone. Radial glial cells (gray) arise from the progenitor cells and migrate towards the cortical plate, forming radial glial fibers (gray lines). Bottom: Later, neurons (black) arise from the same progenitor cells and migrate outward along the radial glial fibers to accumulate on the cortex.

up of bundles of axons, which increase in cross-sectional area during development (Partridge et al., 2004; Dimond et al., 2020). For thorough reviews on the cellular processes that accompany brain development, we suggest Taverna et al. (2014), Llinares-Benadero and Borrell (2019).

2.2. Biomechanical theories of brain development

The main theories of gyrification can be divided into those that attribute folding to intrinsic forces and those that implicate extrinsic forces. The latter was briefly favored, inspired by the observation that the surface area of the brain is much larger than that of the cranium (Papez, 1929; Le Gros Clark, 1945). Later, the volume constraint of the skull was largely disregarded as an explanation for cortical folds after an early experiment found that folding continued normally even when some brain tissue was removed (Barron, 1950).

Subsequently, most research has focused on the role of forces intrinsic to the cerebrum itself. In fact, some of the earliest publications on gyrification claimed that folding occurred because the growth of the outer cortical layer outpaced that of the inner white matter, resulting in instability and folding (His, 1874; Retzius, 1891). Later, in one of the first quantitative theories of gyrification, folding was predicted to occur as the result of differential growth between cortical layers (Richman et al., 1975). This differential growth hypothesis has also been supported by recent studies on developing brain organoids (Karzbrun et al., 2018).

A competing theory of intrinsic forces in cortical folding points to axonal tension as the driver of gyrification, with gyri resulting from areas with more axonal connections (Van Essen, 1997) and folding scaling with connectivity (Herculano-Houzel et al., 2010). These hypotheses have been supported by studies that demonstrate the substantial impact of axonal tension on the shape of convolutions (Hilgetag and Barbas, 2005). However, experiments have also revealed that stress patterns in the brain do not match those predicted to result from axonal tension (Xu et al., 2010). While this theory has fallen out of favor recently, it is likely that elements of all three theories combine to modulate the expansion of gray and white matter, determine the placement of individual folds, and shape the surface of the cortex (Garcia et al., 2018a). For excellent reviews on the competing theories, we refer interested readers to Welker (1990), Bayly et al. (2014), Ronan and Fletcher (2015a), Garcia et al. (2018a).

2.3. Mechanical framework for growth

The majority of studies of brain development have based their models on the theory of finite growth (Rodriguez et al., 1994), in which material is added or removed in the stress-free state, and residual stress arises from an elastic deformation needed to preserve continuity. Mathematically, this is represented by the multiplicative decomposition of the deformation gradient,

$$\boldsymbol{F} = \frac{\partial \boldsymbol{x}}{\partial \boldsymbol{X}} = \boldsymbol{F}^{\mathrm{e}} \cdot \boldsymbol{F}^{\mathrm{g}} \left(\vartheta^{\mathrm{g}}\right), \tag{1}$$

where $\boldsymbol{x}$ and $\boldsymbol{X}$ are the current and reference coordinates of a material point, respectively, $\boldsymbol{F}^{\mathrm{e}}$ is the energy-storing, reversible elastic part of the deformation tensor, $\boldsymbol{F}^{\mathrm{g}}$ is an irreversible growth tensor, and ϑ^{g} is the growth variable describing the amount of volume growth that has taken place (Fig. 3). Further details are presented in later sections where relevant. For a more thorough discussion of the theory of finite growth, we refer interested readers to Taber (1995), Kuhl et al. (2003), Menzel and Kuhl (2012), Holland (2018). In addition to the many studies who have used this framework, a few have represented cortical growth using isotropic osmotic (Geng et al., 2009) or thermal expansion (Wang et al., 2019a).

3. Cranial modeling: from the brain in isolation to the brain–meninges–skull complex

Early studies of cortical convolutions were particularly interested in investigating the mechanical constraint of the skull (Papez, 1929; Le Gros Clark, 1945), but since an experimental study showed that brain folding is not dependent on running out of room inside the skull (Barron, 1950), research has mostly focused on factors intrinsic to the brain. Thus, the majority of models of brain development represent the gray and white matter in isolation (Fig. 4A). Simulations of brains growing in complete isolation have led to a fundamental understanding of the forces intrinsic to the cerebrum that can give rise to gyri and sulci. These studies focus on the effects of differential growth between cortical layers, inducing mismatch strain which then results in instabilities and folding (Richman et al., 1975). From this work, we know that increasing the cortical growth rate increases gyrification (Tallinen et al., 2014), while increasing cortical stiffness or thickness decreases gyrification (Razavi et al., 2015b).

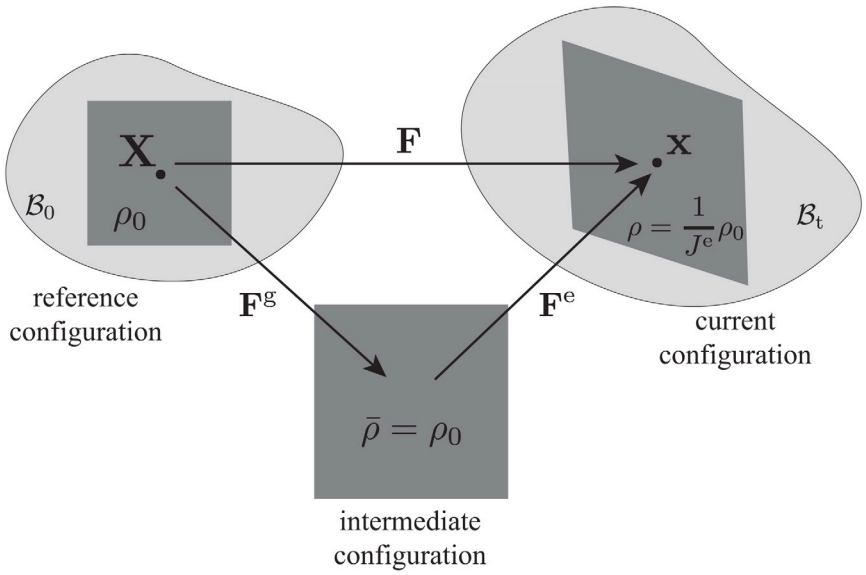

Fig. 3. Configurations in the theory of finite growth. The dark gray boxes are infinitesimal neighborhoods of the points $\boldsymbol{X}$ in the reference configuration $\mathcal{B}_0$, and $\boldsymbol{x}$ in the deformed configuration $\mathcal{B}_t$.

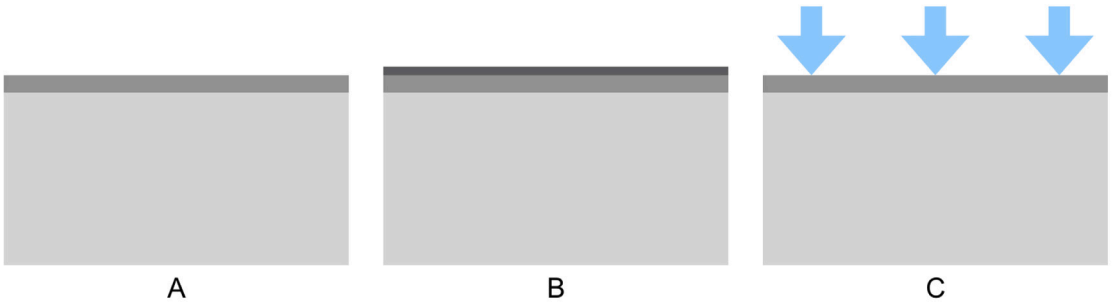

Fig. 4. Constraints of the skull, meninges, and cerebrospinal fluid in models of brain development. In most studies, cortical folding is assumed to result from forces intrinsic to the brain, generally between the cortical and subcortical layers (A), while only a few studies have investigated the role of extrinsic tissues such as the pia mater, via a trilayer model (B, Wang et al., 2019b), or cerebrospinal fluid, via a surface pressure boundary condition (C, Darayi and Holland, 2020).

3.1. Constraint of the skull

While the constraint of the skull is not necessary for the formation of cortical folds, it does appear to influence gyrification and fold geometry. When a cranial constraint is included, curvature increases much faster, resulting in a more highly convoluted brain and earlier emergence of gyri and sulci (Nie et al., 2010). Similarly, it has been suggested that the skull may influence the geometry of the gyri, flattening them as they make contact with the skull (Tallinen et al., 2014; Striedter et al., 2015). Abnormalities of the developing skull also affect brain development. For example, the premature fusion of cranial sutures (craniosynostosis) can drastically alter the size and shape of the skull and brain (Kapp-Simon et al., 2007; Kirmi et al., 2009; Collmann et al., 2005). However, even though the effect of cranial suture fusion on the growing brain has been investigated (Weickenmeier et al., 2017), the overall role of the skull in brain development must be further studied.

3.2. Constraint of the meninges

The meninges consist of the pia mater, arachnoid mater, and dura mater. While the meninges are known to play a biochemical role in signaling between the brain and the skull (Gagan et al., 2007), their mechanical role is less clear. When the brain is modeled as a trilayer, with a layer of pia mater on top of the gray and white matter (Fig. 4B), the thickness, stiffness, and growth rate of the pia mater was found to affect cortical folding, decreasing the number of folds and increasing the wavelength (Wang et al., 2019b). As meninges dominate the brain's mechanical behavior in some loading cases (Sharp et al., 2009), their impact on folding mechanics should be more rigorously investigated.

3.3. Constraint of cerebrospinal fluid

The cerebrospinal fluid (CSF) surrounds the brain, supplying nutrients, removing cellular waste products, and, importantly, protecting the brain from damage (Orešković and Klarica, 2010). Disruption of normal CSF circulation during brain development can result in neurodevelopmental disorders such as hydrocephalus (Owen-Lynch et al., 2003; Miyan et al., 2006), a common disorder resulting in ventricle enlargement, increased intracranial pressure, decreased neuronal migration, and decreased cortical thickness (Miyan et al., 2003; Kang et al., 2018; Roy et al., 2019). Normally, the CSF exerts a pressure of 1.5 mmHg to 6 mmHg (200 Pa to 800 Pa) on the full-term infant brain and 10 mmHg to 15 mmHg (1300 Pa to 2000 Pa) on the adult brain (Dunn, 2002). Several studies have pointed out the importance of these physiological pressures for normal brain development broadly (Miyan et al., 2003; Roy et al., 2019; Kang et al., 2020), but the effect of CSF pressure on the gyrification process specifically is poorly understood. When CSF was added as a pressure constraint, predictions of absolute Gaussian curvature improved in comparison to an unconstrained model (Chen et al., 2010). Our recent work has also shown that the addition of pressure on the outer surface of a bilayer system (Fig. 4C), such as that

exerted by the CSF, reduces the stability of the system, particularly in cases of soft materials such as the brain (Darayi and Holland, 2020).

3.4. Future directions

While experimental evidence suggests that cranial constraints are not the main cause of folding, they may be one necessary ingredient among others for the development of the typical shape of the brain. For example, it has been suggested that gyri become flattened from pressing against the meninges and skull (Striedter et al., 2015). Thus, while perhaps playing a negligible role in the initiation of folds, the effect of external constraints on later gyrification is worth further investigation, such as a model of post-buckling behavior that includes contact between cortical and meningeal tissues. To better understand what this constraint and contact would look like, further experimental studies are necessary to describe the behavior of the meninges, particularly their growth trajectory throughout development. Additionally, many questions remain about the interaction of the brain, meninges, and skull in a variety of neurological conditions. Computational simulations of gyrification in the case of premature sutural fusion in craniosynostosis, ideally in conjunction with medical imaging, could lead to a deeper understanding of the coupled growth of the brain and skull. This could, in turn, result in new insights into other neurological conditions such as microcephaly, of great interest because of its association with Zika virus. Finally, unlike studies of traumatic brain injury (Zemlan et al., 1999, 2002; Panzer et al., 2012; Minta et al., 2020), CSF has been severely understudied in brain development. Addressing this will require accurate measurements of changes in CSF properties and pressure throughout development. We also note that the ventricles, interior reservoirs of CSF, have not been included in models of gyrification, and thus their role in cortical folding is unknown. A combined computational approach with neuroimaging data would be particularly powerful in the investigation of both physiological and pathological (i.e., ventriculomegaly) ventricular pressures.

4. Cortical modeling: from homogeneity to heterogeneity

The earliest models of brain folding assumed a homogeneous cortex experiencing homogeneous growth (Fig. 5A), partly due to limitations of early analytical approaches (Richman et al., 1975). This growth is generally assumed to be driven by morphoregulatory genes or molecules (Taber, 1995), which is often modeled as a simple time-dependent process, e.g.

$$\dot{\vartheta}^{\mathrm{g}} = G\,, \tag{2}$$

where G is a simple constant growth rate (Bayly et al., 2013; Budday et al., 2015c). More complex functions have also been used (e.g. Wang et al., 2021a), but it is important to note that nonlinear functions and changes in the growth rate are only relevant when other time-dependent behaviors such as subcortical growth are included. While many studies have modeled the cortex with isotropic volumetric growth (Budday et al., 2014; Holland et al., 2015; Razavi et al., 2015a; Holland et al., 2018; Chavoshnejad et al., 2021),

$$\boldsymbol{F}^{\mathrm{g}}\left(\vartheta^{\mathrm{g}}\right) = (\vartheta^{\mathrm{g}})^{1/3}\boldsymbol{I}\,, \tag{3}$$

there is strong evidence that the surface area of the cortex grows transversely isotropically (Finlay and Darlington, 1995; Essen, 2007; Mota and Herculano-Houzel, 2015; Wang et al., 2021b), i.e.

$$\boldsymbol{F}^{\mathrm{g}}\left(\vartheta^{\mathrm{g}}\right) = \sqrt{\vartheta^{\mathrm{g}}}\boldsymbol{I} + (1-\sqrt{\vartheta^{\mathrm{g}}})\boldsymbol{n}_0 \otimes \boldsymbol{n}_0\,, \tag{4}$$

where $\boldsymbol{n}_0$ is normal to the plane of growth. Models with cortical homogeneity have illuminated the effects of growth rate on surface morphology (e.g. Budday et al., 2014), showing that increased cortical growth, relative to the growth of the subcortex, leads to shallower folds, whereas increased cortical thickness leads to longer and wider folds (Budday et al., 2015c). In spherical domains, cortical thickness was also found to influence the onset of buckling in the case of isotropic cortical growth, while transversely isotropic cortical growth was insensitive to initial thickness (Razavi et al., 2015a). In our own work, we modeled a simple rectangular bilayer with homogeneous thickness and growth in the cortex to show that gyri thicken and sulci thin as a natural result of the forces generated during folding (Holland et al., 2018). These results show that these thickness variations, a striking feature of physiological cortical thickness patterns (Brodmann, 1909; Fischl and Dale, 2000; Razavi et al., 2015b), can emerge from a perfectly homogeneous system.

4.1. Heterogeneity

However, studies on neuronal migration and composition have revealed heterogeneity in the evolution of the cortex (Lavdas et al., 1996; Kriegstein et al., 2006; Rajagopalan et al., 2011; Van Essen et al., 2018; Garcia et al., 2018b). The simplest approach to introducing cortical heterogeneity is to include small perturbations (Fig. 5B), often in the form of a localized increase or reduction in cortical thickness, stiffness, or growth. This is done to trigger the formation of instabilities on otherwise-perfectly uniform surfaces, particularly rectangular and spherical domains. Several of these studies have found that gyri are more likely to form at locations of higher growth or stiffness, while sulci tend to appear at points of lower growth or stiffness (Toro and Burnod, 2005; Zhang et al., 2016; Bayly et al., 2013; Razavi et al., 2015b; Budday and Steinmann, 2018). Very few studies have gone further, varying cortical properties throughout the cortical surface (Fig. 5C). For example, sinusoidal variations in cortical growth (Budday and Steinmann, 2018) and thickness (da Costa Campos et al., 2021) led to more complex wrinkling patterns similar to experimental observations. Additionally, increased cortical heterogeneity decreased the stability of the bilayer system, with buckling occurring in the thinnest regions. Recently, we developed a novel gyrification model where sulci and gyri (differentiated by mean curvature) grow at different rates (Wang et al., 2020a), concluding that gyri grow faster than sulci.

4.2. Future directions

The greatest challenge in cortical modeling is the distance that currently exists between our biological understanding and our computational models. For example, while the balance of mass includes both sources and fluxes,

$$\dot{\rho}_0 = \mathrm{Div}\,\boldsymbol{j}_0 + h_0\,, \tag{5}$$

mass transport is generally ignored ($\mathrm{Div}\,\boldsymbol{j}_0 = 0$). One study addressed this by modeling the migration of cells outward from the subventricular zone to the cortical plate with a locally changing cell density (de Rooij and Kuhl, 2018); a more recent study modified this slightly by coupling cell density and stiffness (Zarzor et al., 2021). Further advances should consider how to model volume growth after folding begins, due to dendritic arborization, axonal myelination, and synaptogenesis (Cafiero et al., 2019). Additionally, incorporating the effects of gene expression on these processes could provide a natural source of heterogeneity in cortical properties and growth (de Juan Romero et al., 2015; Ronan and Fletcher, 2015b). Overall, future studies should move beyond the simple patterns considered thus far, and attempt to include heterogeneity based on data such as initial cortical thicknesses in fetal brains (Holland et al., 2020b), potentially alongside simultaneous changes in tissue stiffness. Finally, the cortex is also known to vary significantly in the radial direction, with six distinct layers or laminae. Laminar differences in cytoarchitecture (Waehnert et al., 2014) and gene expression (Shinmyo et al., 2017; Matsumoto et al., 2017) may also produce differences in tissue properties and growth. Although radial changes in tissue properties have been investigated in the subcortex (Budday and Steinmann, 2018), this has not been explored in the cortical laminae.

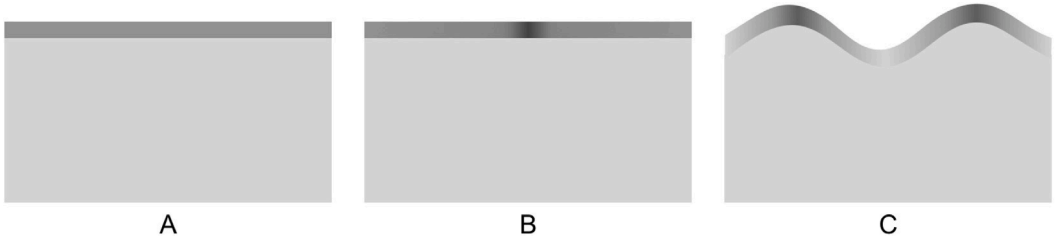

Fig. 5. Models of cortical growth. Homogeneous cortical growth (A) is most common in studies of brain development, although local perturbations (B, where the darker region indicates higher growth) are sometimes included to initiate folding on otherwise perfectly uniform domains. A few studies have investigated more complex heterogeneous patterns (C), including both pre-patterned variation (Budday and Steinmann, 2018) and variation dependent on the formation of cortical folds (Wang et al., 2020a).

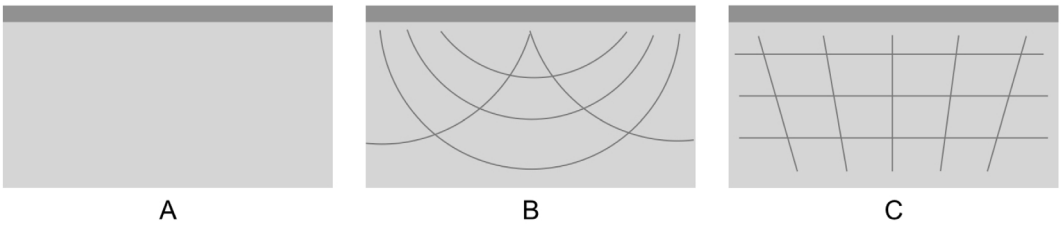

Fig. 6. Models of subcortical growth. Many models of cortical folding have included a simple subcortical layer (A) that either does not grow (Xu et al., 2010; Tallinen and Biggins, 2015; Razavi et al., 2015a; Zhang et al., 2017; Holland et al., 2018) or grows isotropically in response to mechanical stimuli (Xu et al., 2009; Budday et al., 2014, 2015e; Razavi et al., 2015b; Zhang et al., 2017). Only a few models have incorporated axonal-based anisotropy in the subcortex, using different patterns of axon alignment (B and C; Holland et al., 2015; Razavi et al., 2017; Garcia et al., 2020).

5. Subcortical modeling: from isotropy to anisotropy

Historically, more focus has been placed on the cortex than on the other structures in the brain. In fact, many studies have analyzed cortical convolutions using a model consisting of a bi- or multilayer with a purely elastic subcortex (e.g. Xu et al., 2010; Tallinen and Biggins, 2015; Razavi et al., 2015a; Zhang et al., 2017; Holland et al., 2018), while others assume a simple stretch-driven isotropically growing subcortex (e.g. Budday et al., 2014, 2015e; Razavi et al., 2015b; Zhang et al., 2017; Riccobelli and Bevilacqua, 2020), e.g.

$$\dot{\vartheta}^{\mathrm{g}} = G\left(J^{\mathrm{e}} - J^{0}\right), \tag{6}$$

where $J^{\mathrm{e}} = J/J^{\mathrm{g}}$ is the elastic component of volume change and J^{0} is the homeostatic set point for elastic volume stretch. These studies showed that subcortical growth is positively correlated with the degree of cortical folding, while increasing the shear modulus of subcortical regions results in smooth shallow folding patterns (Budday et al., 2015c,e; Holland et al., 2015; Razavi et al., 2015b,a).

5.1. Anisotropy

Strongly aligned bundles of axons and heterogeneous cell division and migration have been identified in white matter (Harrison, 1935; Bray, 1984; Rakic, 2003; Rana et al., 2019). The presence of these aligned fibers has revealed material anisotropy with respect to the local neuroarchitecture of white matter (Prange and Margulies, 2002). Given that axons are known to grow under prolonged tension (Weiss, 1941; Bray, 1984; Smith, 2009), this anisotropy could play a crucial role in both the elastic and growth behavior of brain tissue. Based on these observations, researchers have proposed multiple models to simulate white matter growth during development. For example, Bayly et al. (2013) assumed that the subcortex could grow differentially in response to radial and tangential stresses,

$$\boldsymbol{F}^{\mathrm{g}}\left(\vartheta^{\mathrm{g}}\right) = \sum_{i}^{3} \vartheta_{i}^{\mathrm{g}}\, \boldsymbol{e}_{i} \otimes \boldsymbol{e}_{i}, \qquad \text{with} \qquad \dot{\vartheta}_{i}^{\mathrm{g}} = G\left(\sigma_{i} - \sigma_{i,0}\right)\vartheta_{i}^{\mathrm{g}}, \tag{7}$$

where $\boldsymbol{e}_i$ are a set of orthonormal vectors, and σ_i and $\sigma_{i,0}$ are the Cauchy stress and the 'target' stress, respectively, in the specified direction. This model led to stress distributions that were in good agreement with previous *ex vivo* experiments (Xu et al., 2010). We further explored the effects of axonal elongation and orientation in a model of transversely isotropic white matter, which elongates along a preferred axonal direction in response to applied stretch (Fig. 6B and C),

$$\boldsymbol{F}^{\mathrm{g}}\left(\vartheta^{\mathrm{g}}\right) = \boldsymbol{I} + \left(\vartheta^{\mathrm{g}} - 1\right)\boldsymbol{a}_{0} \otimes \boldsymbol{a}_{0}, \qquad \text{with} \qquad \dot{\vartheta}^{\mathrm{g}} = G\left(\lambda^{\mathrm{e}} - \lambda^{0}\right), \tag{8}$$

where $\boldsymbol{I}$ is the identity tensor, $\boldsymbol{a}_0$ is the undeformed fiber direction, $\lambda^{\mathrm{e}} = \|\boldsymbol{a}\|/\vartheta^{\mathrm{g}}$ is the elastic stretch along the deformed fiber direction $\boldsymbol{a} = \boldsymbol{F}\cdot\boldsymbol{a}_0$, and λ^{0} is the homeostatic set point for fiber length. We found that increased axonal growth rates along specific orientations led to an increase in sulcal depth, gyral wavelength, and irregular morphology (Holland et al., 2015). In another study, gyral regions tended to form in regions of dense glial and axonal fibers growing faster than the surrounding white matter (Razavi et al., 2017; Chavoshnejad et al., 2021). Most recently, a new model incorporates both growth and reorganization of radial and tangential subcortical fibers (Fig. 6C) in response to stress (Garcia et al., 2020),

$$\dot{\vartheta}_{i}^{\mathrm{g}} = f_{i} G\left(\sigma_{i} - \sigma_{i,0}\right)\vartheta_{i}^{\mathrm{g}}, \tag{9}$$

where f_i is the volume fraction of fibers oriented in direction i. These simulations predict that fibers will be predominantly oriented radially in gyri and tangentially under sulci, which is similar to the fiber organization seen in real brains.

5.2. Future directions

Based on diffusion tensor and MR imaging studies that have demonstrated structural anisotropy and nonuniform growth in white matter (Nagy et al., 2004; Østby et al., 2009; Song et al., 2015), it is important to include realistic subcortical behavior in models of cortical folding. This is particularly important in the case of neurological disorders where connectivity is implicated as a possible cause, such as Autism Spectrum Disorder (Barnea-Goraly et al., 2004; Hendry et al., 2006; Cheng et al., 2010). One of the next steps in subcortical modeling should be to include multiple fiber directions or fiber dispersion models, (Qiu et al., 2015; Eskandari et al., 2020), as done in the elastic behavior of white matter in the field of traumatic brain injury (Giordano and Kleiven, 2014). In the case of brain development, anisotropy can appear in both elastic and growth properties, but only the latter

Fig. 7. Stiffness contrast between cortical and subcortical layers. Early models assumed that gray matter is much stiffer than white matter (A, where darker colors represent stiffer material). However, more recent data suggest that the stiffness contrast between the two tissues is much lower (B), and potentially that white matter is even slightly stiffer than gray matter (C).

has been investigated. While fiber directions can be observed from neuroimaging studies, it is important to consider that this data is taken at a single time point. As it is known that axons respond significantly to mechanical stimulus (Lamoureux et al., 2010), their ability to reorient should be investigated (Himpel et al., 2008). Finally, we note that a traditional finite element approach is not capable of modeling true connectivity across elements; existing models of 'axons' create discrete vector fields of preferred directions in individual elements (Holland et al., 2015). The direct representation of axonal bundles would open up new possibilities in the study of cortical folding.

6. Tissue stiffness: from contrast to similarity

Questions about the material properties of brain tissue have plagued the study of gyrification since its inception. The gray-white stiffness ratio has enormous implications on simulations of instabilities, as wrinkling, creasing, and folding are known to emerge in different regimes (Li et al., 2012). The earliest attempts at simulating brain folding (e.g. Richman et al., 1975) were based on studies of instabilities in non-biological multilayered systems (Biot, 1963b,a), which generally had very high (one or two orders of magnitude) stiffness contrast (Fig. 7A) and predicted sinusoidal wrinkling. A growing body of literature has examined the buckling instabilities of multilayered systems based on Biot's linear perturbation approach (Huang et al., 2005; Holland et al., 2017; Colin et al., 2019; Alawiye et al., 2019), although experimental studies tend to predict lower critical strains in comparison, likely because of the formation of surface self-contact in small localized regions (Gent and Cho, 1999; Ghatak and Das, 2007). These studies have shown that the stiffness ratio of the cortex to the subcortex is proportional to the wavelength (Dervaux and Ben Amar, 2011). High stiffness contrast has also been shown to delay the onset of secondary bifurcation points such as period-doubling and period-tripling (Budday et al., 2015a) and increase the generation of residual stresses in white matter (Xu et al., 2009). More recently, the stiffness contrast has been recognized as one of the most important factors in brain development.

6.1. Low stiffness contrast

Recent progress on the material characterization of brain tissue has revealed the low stiffness contrast between gray and white matter (Arbogast and Margulies, 1998; Miller and Chinzei, 2002; Miller et al., 2000; Kruse et al., 2008; Christ et al., 2010). Experimental and numerical data suggest that the stiffness contrast between the cortex and the subcortex ranges from 1 to 6 (Chatelin et al., 2010). Some data from mechanical characterization studies of brain tissue have even suggested that the white matter is stiffer than gray matter (Kruse et al., 2008; van Dommelen et al., 2010; Budday et al., 2015b). Differences in reported values could be due to multiple factors such as species, age, post-mortem time, specimen size, testing procedure, and regional variations (Prange and Margulies, 2002; Jin et al., 2013; Sack et al., 2009; Hiscox et al., 2020). Based on these experimental findings, many studies of cortical folding (e.g. Xu et al., 2010; Bayly et al., 2013; Tallinen et al., 2014; Budday et al., 2015c; Holland et al., 2015; Razavi et al., 2015b; Tallinen et al., 2016; Riccobelli and Bevilacqua, 2020) use gray-white stiffness ratios between 1 and 3 (Fig. 7B), with a few exploring ranges of stiffness ratios up to or beyond 10 (e.g. Budday et al., 2014, 2015a,e; Budday and Steinmann, 2018; Wang et al., 2020a; da Costa Campos et al., 2021). Studies have found that a growing bilayer favors creasing before wrinkling when the stiffness ratio is close to one, and tends to wrinkle as the stiffness ratio increases (Tallinen and Biggins, 2015; Razavi et al., 2015b; Jin et al., 2015). Our previous studies have found that, for stiffness contrasts below 10, the source of compressive forces in the film (i.e., growth vs. substrate prestretch) affects results significantly (Holland et al., 2017), and that even low pressures from the surrounding CSF can decrease stability (Darayi and Holland, 2020). To date, very few studies (Xu et al., 2009; Tallinen and Biggins, 2015; Wang et al., 2019a) have simulated the developing brain with stiffness ratios below one (Fig. 7C).

6.2. Future directions

Advances in computational studies of folding are highly dependent on concurrent advances in the experimental characterization of brain tissue properties. Of course, it is challenging to test the brain, an extremely soft and biologically sensitive tissue surrounded by the thick bone of the skull; this challenge is even greater *in utero*, where the properties most relevant to gyrification are found. Magnetic resonance elastography is promising for capturing *in vivo* properties (Weickenmeier et al., 2018), although spatial resolution is limited; this is an important limitation to overcome, especially given the radial variations in the cortex discussed earlier. While much of the work in this area has been focused on identifying regional variations in properties, it should be noted that properties are also changing throughout development, for instance stiffening as myelination progresses (Weickenmeier et al., 2016). A potential direction for future study would be to model stiffness as a time-varying property; in the study of airway instabilities, for instance, this has been shown to lead to intricate instability patterns (Eskandari et al., 2016). Finally, when comparing tissue properties between simulations and experiments, it is important to remember that loading and growth can change the effective stiffness of a material, or its resistance to further deformation (Holland et al., 2017). Because of this, models that define low initial stiffness contrast between gray and white matter will, through cortical growth, increase the stiffness contrast in their simulations.

7. Geometries: from simple to complex

Analytical and many numerical simulations of the developing brain have used simple, often two-dimensional, domains. The most commonly used geometry in the literature for modeling the brain folding is a rectangular plane strain (e.g. Xu et al., 2009, 2010; Bayly et al., 2013; Tallinen et al., 2014; Budday et al., 2014; Holland et al., 2015; Budday et al., 2015a; Razavi et al., 2015b; Holland et al., 2018; Budday and Steinmann, 2018; Riccobelli and Bevilacqua, 2020) or 2D bilayer (e.g. Tallinen et al., 2014; Budday et al., 2014; Holland et al., 2015; Budday et al., 2015a; Holland et al., 2018; Budday and Steinmann,

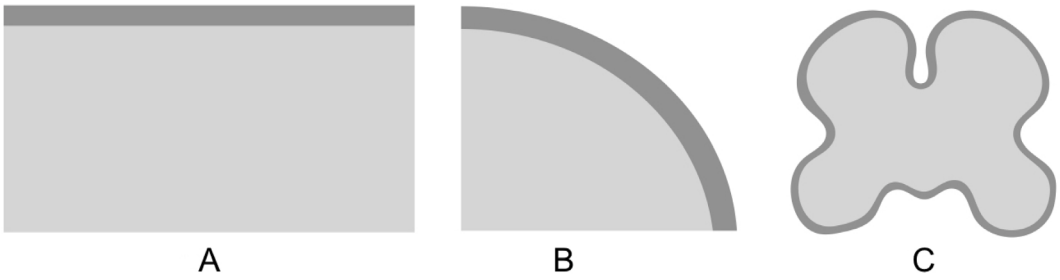

Fig. 8. Model geometries in simulations of brain development. Simple rectangular (A) and elliptical (B) models have been used in many studies of cortical folding, while more realistic geometries such as those reproduced from fetal MR images (C), have shown promise in reproducing physiological folding patterns (Nie et al., 2010; Tallinen et al., 2016).

2018), representing a small subsection of the brain in which curvature can be neglected (Fig. 8A). Researchers have used these models to investigate the influence of various factors such as stiffness ratio, layer thickness, and growth rate. Other 2D models, such as semi-ellipse and circular bilayer, have also been considered (Xu et al., 2009, 2010; Budday et al., 2014; Razavi et al., 2015b; Budday et al., 2015e), often focusing on curvature, which changes across the surface of an ellipse (Fig. 8B). Convolutions first appear at the minor axis of ellipsoids before propagating to the sides with shallower folding patterns (Toro and Burnod, 2005; Budday et al., 2014), and longer wavelengths emerge on the curved surface of elliptical models in comparison with rectangular domains (Bayly et al., 2013). From observations of different mammalian species, brain size and shape strongly influence folding patterns (Hofman, 1989), for example with longitudinal folding patterns more likely to be identified on longer brains (Budday et al., 2015d). Studies on rectangular, elliptical, and eventually ellipsoidal domains of brain tissue have been hugely successful in focusing on non-geometric effects, such as the role of growth and tissue properties.

7.1. Complex geometries

While simple geometries have provided considerable insight into brain folding, there is still a need for more complex and realistic models, which to date have only been used in a handful of studies. Cortical folding patterns are strongly influenced by the initial geometry of the cortex, affecting the shape, size, and orientation of folds. Even before gyrification begins, the fetal brain has a complex shape with significant variations in curvature across its surface (Todd, 1982; Bohi et al., 2019; Shishegar et al., 2021) (Fig. 8C). An early surface model of cortical folding was based on MR images of fetal brains at 22 weeks of gestation, and explored the effects of cranial constraints and regional variations in growth (Nie et al., 2010). Later, another study based on real fetal brain geometries demonstrated the sensitivity of gyrification patterns to variations in the initial geometry and demonstrated qualitative and quantitative similarities to human brains such as the variation of gyrification index during development, gyral and sulcal locations, and the appearance of primary convolutions (Tallinen et al., 2016). Recently, an inverse modeling approach based on scans of six normal fetuses between gestational weeks 21 and 25 was used to quantify local volume changes in the developing brain, finding evidence of heterogeneous growth shortly before the emergence of the central sulcus (Wang et al., 2020b).

7.2. Future directions

The ultimate goal of gyrification simulations is to begin with a fetal brain geometry and simulate the entire process of cortical folding, producing real patterns of the cortical topology including sulcal root maps and 'plis de passage' (Mangin et al., 2019). This is particularly important for appropriate comparisons with the many neuroimaging studies focused on physiological and disordered development (Marsh et al., 2008; Mackes et al., 2020; Hashem et al., 2020). While this has been done with significant success, it has only been possible by modeling only the brain surface (Nie et al., 2010) or by fixing the cortical thickness to a non-physiological value (Tallinen et al., 2016). Future studies will need to devise numerical approaches to handle the huge size and shape changes that occur throughout development, such as remeshing to maintain optimal size and aspect ratios in growing elements. Another challenge related to fetal geometries is the limited resolution of *in utero* fetal neuroimaging (Scott et al., 2011; Clouchoux et al., 2012); using preterm infants instead is not ideal due to the possibility of abnormal development (Dubois et al., 2008; Lefèvre et al., 2015). Significant advances could be made by focusing on the brains of non-human primates and other mammals, such as macaques (Young et al., 2017) and ferrets (Barnette et al., 2009), which exhibit folding but to a lesser extent than humans. An added benefit of modeling brain development in animals is the large variety in shapes and sizes of brains across the animal kingdom (Heuer et al., 2019), which can lead to a broader investigation of the factors that contribute to brain folding writ large. Incremental steps towards the ultimate goal of simulating folding throughout development could focus on aspects of brain geometry that have been understudied to this point. For example, while a number of models have investigated the effect of varying curvature, such as that found on an ellipse, no simplified geometry has included concave areas. Of course, the surface of the brain is largely convex, but local concavities (such as the region that becomes the sylvian fissure and the occipital lobe near the cerebellum) may affect folding in their vicinity. Finally, the majority of studies have used 2D models, which are not capable of capturing the full three-dimensional pattern of folding. Intersections (Ge et al., 2018; Zhang et al., 2018; Huang et al., 2019; Razavi et al., 2021) and tangential folds in gyri and sulci are important features in highly folded brains, such as those of humans.

8. Conclusion

Tremendous progress has been made in the study of cortical folding *in silico* over the last several decades. In this review, we have discussed how researchers have, over time, advanced simulations beyond their limited beginnings. We focused on the areas of cranial, cortical, and subcortical modeling, with special attention to the tissue properties and model geometries used. However, each of these aspects remains in need of further investigation if we are to continue to deepen our understanding of the developing brain. While all computational models rely on simplifying assumptions to make complex biological problems tractable, there are many opportunities for the brain biomechanics community to advance towards more accurate and realistic models of the developing brain, both in the short and long term. As gyrification is the result of coupled biological and mechanical behavior, we divide our suggestions for future work into the two complementary domains of mechanics and biology. In the realm of mechanics, simulations should explore, for instance, the far post-buckling behavior, where gyri potentially interact with the meninges and skull (Striedter et al., 2015), as well as the formation of tangential folds and intersections (Razavi et al., 2021). As models get larger, more complex, and run for longer

periods of time, they will become more expensive and require additional computing resources and perhaps novel numerical approaches for solving. In the realm of biology, there are many *in vivo* and *in vitro* studies on the molecular, cellular, and genetic determinants of folding that should be better incorporated into our *in silico* models. This would include direct modeling of neuronal migration; incorporation of other sources of volume growth such as myelination and dendritic arborization; differential gene expression as a source of regional and temporal heterogeneity; modeling cortical laminae as distinct layers with differing properties; and representing white matter with multiple fiber directions and varying connectivity between regions.

In these and other areas, future studies should investigate the 'edge cases' of development, outside the bounds of typical human physiology, both in the huge variety of neurological diseases and disorders, and within the diversity of the animal kingdom. Conditions such as craniosynostosis, Zika-associated microcephaly (Vesnaver et al., 2017), and lissencephaly in Miller-Dieker syndrome (Bershteyn et al., 2017) highlight the role of different processes that also occur in typical development. Similarly, animals possess brains of strikingly different sizes and shapes, exhibiting varying degrees of folding. While the ferret is frequently studied because its cortical folding process takes place after birth, other animals offer unique examples of brain development worthy of further study. For example, dolphin brains are even more highly folded than humans, and the brains of manatees and beavers are peculiarly smooth for animals of their size (Welker, 1990). Brain organoids, developed from human induced pluripotent stem cells, are another particularly promising area for the study of both physiological and pathological brain development (Vaez Ghaemi et al., 2018; Karzbrun and Reiner, 2019; Kyrousi and Cappello, 2020).

Of course, advances in computational simulations of gyrification must be accompanied by advances in experimental studies. The main areas of need currently are better characterization of tissue properties and increased and improved neuroimaging studies. The stiffness of white and gray matter is still not settled, and even more questions remain regarding regional variations and temporal changes throughout development, particularly during gestation. Similarly, future simulations may require tissue properties for the meninges and values for CSF pressure both in the ventricles and against the pial surface. In the field of neuroimaging, huge improvements have occurred recently with the proliferation of 7T imaging, allowing for detailed images of structures as fine as the cortical laminae (Allen et al., 2021; Wagstyl et al., 2020) and offering hope for refinements in techniques such as MR elastography.

In light of recent advances in both computational and experimental investigations of brain development, our understanding of the brain continues to deepen. However, it remains our most complex organ, and further improvements are necessary to unlock the many remaining questions about how our brains form and function.

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.

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