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TOPICAL REVIEW

# Development of the cerebral cortex and the effect of the intrauterine environment

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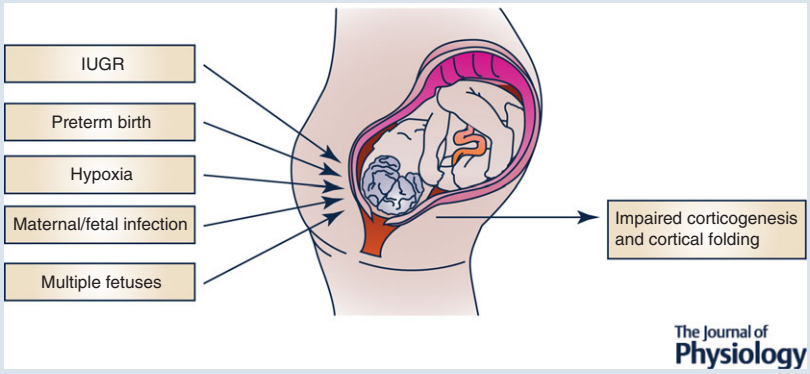


**Abstract** The human brain is one of the most complex structures currently under study. Its external shape is highly convoluted, with folds and valleys over the entire surface of the cortex. Disruption of the normal pattern of folding is associated with a number of abnormal neurological outcomes, some serious for the individual. Most of our knowledge of the normal development and folding of the cerebral cortex (gyrification) focuses on the internal, biological (i.e. genetically driven) mechanisms of the brain that drive gyrification. However, the impact of an adverse intrauterine and maternal physiological environment on cortical folding during fetal development has been understudied. Accumulating evidence suggests that the state of the intrauterine and maternal environment can have a significant impact on gyrification of the fetal cerebral cortex. This review summarises our current knowledge of how development in a suboptimal intrauterine and maternal environment can affect the normal development of the folded cerebral cortex.

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**Abstract figure legend** Common pregnancy-related conditions such as intrauterine growth restriction (IUGR), preterm birth, hypoxia, maternal/fetal infections and multiple fetuses, can have an impact on the normal development and folding of the cerebral cortex.

## Introduction

The development of the cerebral cortex is a complex but organized process that is common to all mammals, although the timing of the stages of cortical development differs among them (Clancy *et al.* 2009). From a single layer of cells, the cerebral cortex starts developing at approximately 33 days post-conception in humans in an inside-out fashion, creating a diverse and complex architecture that has been the subject of intense study (reviewed in Bystron *et al.* 2008). But although the 'inside-out' development of the cerebral cortex is similar for all mammals, specific aspects of the neuronal architecture differ between species. For example, while some species have cortical hemispheres that show complex folding with 'hills' (gyri) and 'valleys' (sulci), and are therefore referred to as *gyrencephalic*, others have almost completely smooth cortical surfaces and are referred to as *lissencephalic* (Welker, 1990; Defelipe, 2011). The gyrification index (GI) is used to describe the degree and complexity of the folding. The GI is low in lissencephalic animals such as the mouse (GI = 1.03) and in less complexly folded brains such as baboons (GI ∼1.89), and high in more complex brains such as the human brain (GI = 2.53).

In humans the process of gyrification begins *in utero* (Welker, 1990), and continues throughout the early years of life, peaking at early adolescence, stabilizing during adulthood and subsequently declining with the ageing process (Raznahan *et al.* 2011). Abnormal and/or disrupted gyrification during brain development has been reported in association with a wide range of adverse outcomes for the neonate and infant, including aggressive traits in children (Thijssen *et al.* 2015), schizophrenia (Liu *et al.* 2016), autism (Bos *et al.* 2015; Schaer *et al.* 2015; Ecker *et al.* 2016), inclination to alcohol consumption (Kuhn *et al.* 2016), anorexia nervosa (Favaro *et al.* 2015), cognitive deficits (Daamen *et al.* 2015; Docherty *et al.* 2015; Gautam *et al.* 2015) and many others (Piao *et al.* 2004; Mochida, 2009; Megraw *et al.* 2011; Caglayan *et al.* 2014; Chen *et al.* 2014; Hu *et al.* 2014; Kaindl, 2014; Squier & Jansen, 2014; Sun & Hevner, 2014; Passemard *et al.* 2016). In general, gyrification results from differential expansion of the cerebral cortex, which is dictated by genetic, environmental, biochemical and physical events. Disruptions to this precisely orchestrated sequence of events during brain development results in altered brain structure, and therefore potentially altered brain function, and proper understanding of the process in order to prevent these outcomes is paramount.

## Cortical development and folding

The cerebral cortex arises from the rostral end of the neural tube from a single layer of cells referred to as the ventricular zone (VZ), which divide to firstly produce the preplate (PP), and then the second germinal zone referred to as the subventricular zone (SVZ). The neurons produced by the VZ and SVZ start migrating outwards, accumulating and positioning into a layer referred to as the cortical plate (CP). These processes of neuron generation (Reillo *et al.* 2011; Nonaka-Kinoshita *et al.* 2013) and subsequent migration from the germinal zones (Pang *et al.* 2008; Kato, 2015; Moffat *et al.* 2015) are crucial for the gyrification process occurring later in development. At the same time, Cajal-Retzius cells present in the PP migrate to the surface of the cortical hemispheres producing a layer of cells known as the marginal zone (MZ), while the rest of the PP evolves into what becomes known as the subplate (SP). The space between the SVZ and the SP is referred to as the intermediate zone (IZ) and harbours the newly migrating neurons and the radial glial cells (RGCs) that aid neuronal migration. This cortical development process creates the six-layered structure (VZ, SVZ, IZ, SP, CP and MZ) that is common to all mammals (Bystron *et al.* 2008). However, only some mammals – including humans – then undergo the complex development of gyri and sulci.

In humans, gyrification starts during the second trimester of gestation and is mostly complete by term. This process comprises a series of interconnected steps which ultimately result in a folded cerebral cortex (Welker, 1990), and although a number of theories on how and why the cerebral cortex folds have been proposed (Richman *et al.* 1975; Van Essen, 1997; Ronan *et al.* 2014; Striedter *et al.* 2015), the exact mechanisms that drive gyrification are still unknown. Many studies adhere to the theory that gyrification is an inherent (i.e. genetically driven) process (Van Essen, 1997; Budday *et al.* 2014; Ronan *et al.* 2014; Sun & Hevner, 2014; Ronan & Fletcher, 2015; Striedter *et al.* 2015; Fernandez *et al.* 2016), and this has led to the view that programmed expression of specific growth factors such as fibroblast growth factor (FGF) 2 (Rash *et al.* 2013) and $\beta$-catenin (Chenn & Walsh, 2002) have a pivotal role in driving cortical folding. Indeed, many

studies have demonstrated inheritability of the folding pattern (Armstrong *et al.* 1995; Lohmann *et al.* 1999; Atkinson *et al.* 2016), showing that gyrification has a high degree of genetic determination.

## Impaired cortical folding following development in an adverse prenatal environment

A large proportion of the literature on cortical folding has focused on the gyrification process under normal intrauterine conditions, but little attention has been paid to how this process might be affected by an adverse intrauterine environment. Intrauterine growth restriction (IUGR), preterm birth, prenatal hypoxia, malnutrition, maternal and fetal infection, and multiple fetuses per pregnancy are among the most common pregnancy-related conditions associated with adverse outcomes, but they have not been adequately studied in animal models suitable for the study of cortical gyrification (i.e. in gyrencephalic species). In this review we will address the existing literature concerning the impact of a suboptimal intrauterine and maternal physiological environment on brain development, specifically gyrification.

**Intrauterine growth restriction.** Normal fetal growth is dependent on efficient transfer of nutrients – amino acids, glucose, and oxygen in particular – from the maternal uterine circulation to the fetus via the placenta. Inadequate transfer of oxygen, glucose, essential amino acids and triglycerides restricts fetal growth (McMillen *et al.* 2001). IUGR describes the condition where fetal growth falls below the population-based normograms for size, and is most commonly attributed to poor placental function, i.e. placental insufficiency. A number of follow-up studies show that IUGR is associated with significant neurodevelopmental disabilities, including abnormalities in fine and gross motor skills, cognitive function, language, memory, concentration, attention, mood and school performance (Raz *et al.* 2012; de Bie *et al.* 2015), and lower IQ scores (Rogne *et al.* 2015). These adverse outcomes in IUGR are often correlated with the severity of the growth restriction (Delcour *et al.* 2012; Fung *et al.* 2012).

IUGR affects brain structure and, in particular, adversely impacts the cerebral hemispheres and cerebellum (Miller *et al.* 2016; McDougall *et al.* 2017). In the rat, IUGR leads to delayed neuronal migration in the cerebral cortex (Sasaki *et al.* 2000; Tashima *et al.* 2001). These abnormalities in neuronal migration and/or positioning are linked to cortical folding malformations such as lissencephaly, polymicrogyria, cortical dysplasia and microcephaly (Pang *et al.* 2008; Kato, 2015; Moffat *et al.* 2015). Given that neuronal migration plays an important role in the gyrification process, it is not unexpected that the disrupted neuronal migration seen in the IUGR brain might cause alterations in gyrification.

The normal development of the SVZ has an essential role in gyrification (Toda *et al.* 2016). In the IUGR guinea pig fetus there is an inverse correlation between cell proliferation in SVZ and brain weight (Tolcos *et al.* 2015), indicating that IUGR directly impacts this neurogenic zone. Considering that in gyrencephalic brains the manipulation of the proliferation rate in the outer SVZ can modify size and shape of cortical folds (Reillo *et al.* 2011; Nonaka-Kinoshita *et al.* 2013), these findings strongly suggest that IUGR has an impact on gyrification via alterations on cell proliferation in the SVZ.

Imaging studies performed in IUGR children show reduced grey matter volume in the temporal, parietal, frontal and insular regions and a reduction in white matter volume throughout the brain, (Padilla *et al.* 2011), changes that are still present at 1 year of age (Tolsa *et al.* 2004; Eixarch *et al.* 2008; Padilla *et al.* 2011). In the cerebral cortex, magnetic resonance imaging studies show that IUGR newborns have a significant reduction in the volume of the cranium and the cerebral cortical grey matter (Tolsa *et al.* 2004; Inder *et al.* 2005), and accelerated cortical development reaching maturation of the sylvian fissure, parieto-occipital and cingulate sulci earlier than the normally developing group (Businelli *et al.* 2014). These IUGR babies also show altered cortical morphometry, including reduced insular cortical thickness and smaller insular cortical volume (Egana-Ugrinovic *et al.* 2014). Indeed, IUGR babies have significant changes to their gyrification patterns compared to appropriately grown for gestational age (AGA) newborns. Specifically, in IUGR compared to AGA babies, cortical gyrification is reduced with lower surface area and lower sulcation index – another measure used to study brain folding – when compared with babies of the same age, but higher sulcation index in comparison with babies with similar cortical surface measurements (Dubois *et al.* 2008). Furthermore, IUGR newborns show deeper fissure measurements of the left and right insula, and a deeper left cingulate fissure (Egana-Ugrinovic *et al.* 2013).

To our knowledge there has been no study addressing the effects of IUGR on cortical folding in animals with a gyrencephalic brain, as most studies of IUGR have been performed in lissencephalic species such as rats and guinea pigs (Basilious *et al.* 2015). It is now important that we study the impact of IUGR on gyrification using appropriate animal models (e.g. IUGR sheep) to identify, and better understand, the molecular and cellular mechanisms that are impaired in the IUGR gyrencephalic brain to potentially inform clinical management of IUGR babies.

**Preterm birth.** Preterm birth is associated with brain injuries/lesions, the most common being periventricular leukomalacia (PVL), accompanied by neuronal and

axonal deficits involving the cerebral white matter, thalamus, basal ganglia, cerebral cortex, brainstem and cerebellum. These neuropathologies have been associated with profound cognitive and behavioural impairments that persist into adulthood (Govaert *et al.* 2006; Soria-Pastor *et al.* 2008; Zubiaurre-Elorza *et al.* 2009; Groeschel *et al.* 2014; Abbott, 2015). Some of these behavioural deficits can be quite subtle, such as reduced accuracy and response time of individuals during certain tests (Daamen *et al.* 2015), impaired executive control (Urben *et al.* 2017), poor performance on language-based tests (Nam *et al.* 2015), and overall IQ when assessed at different life stages (Skranes *et al.* 2013; Nosarti *et al.* 2014; Solsnes *et al.* 2015; Karolis *et al.* 2016).

Preterm birth is also associated with a lower GI and a lower cortical surface area, with the insula, superior temporal sulcus and ventral portions of pre- and post-central sulci in both hemispheres the most affected regions (Engelhardt *et al.* 2015). In a recent study, pattern differences in the bilateral superior frontal, occipital and basal temporal cortices, as well as the bilateral para-hippocampal gyri were found in very preterm neonates born at 24–33 weeks postmenstrual age (PMA), and assessed at 26–36 weeks PMA (i.e. when clinically stable), and again prior to discharge at late preterm age (32–40 weeks PMA) (Kim *et al.* 2016). Another study of very preterm babies (24–28 weeks PMA at birth) found a decreased GI at 30 and 40 weeks PMA (Moeskops *et al.* 2015). Similar reports in young children born preterm showed shallower anterior superior temporal sulci, smaller relative surface area (relative to the total hemisphere area) in both inferior sensorimotor cortex and posterior superior temporal cortex, and a shorter or interrupted cingulate sulcus (Zhang *et al.* 2015). These developmental deficits are long-lasting; for example, preterm children studied at 8 years of age showed an increase in temporal lobe gyrification and reduction in cortical surface area in superior and inferior frontal gyri (Kesler *et al.* 2006), while a separate study in preterm babies with white matter injury reported delayed myelination and brain folding (Ramenghi *et al.* 2007). Also, microcephaly, lissencephaly and polymicrogyria have been correlated with preterm birth (Brown, 2009), although higher global GI (i.e. increased gyrification) in preterm individuals has also been reported (Lefevre *et al.* 2016).

The molecular mechanisms by which preterm birth affects gyrification remain to be fully identified. Preterm birth reduces neurogenesis in the cortical SVZ (Malik *et al.* 2013), and considering that neurogenesis in both VZ and SVZ affects gyrification (Reillo *et al.* 2011; Nonaka-Kinoshita *et al.* 2013), this is a potential mechanism by which preterm birth affects gyrification. Although the mechanisms underlying the changes in gyrification patterns and structure of the cerebral cortex remain to be elucidated, the persistence of changes into late childhood indicates that conditions present early in brain development determine the long-term trajectory of anatomical and functional cortical development. A better understanding of the underlying mechanisms involved in gyrification might provide promising insight into how best to treat the neurodevelopmental deficits that are frequently a legacy of preterm birth.

**Hypoxia.** Clinical evidence suggests that bouts of hypoxia or asphyxia *in utero* are not uncommon, and may arise from temporary or partial obstruction of umbilical blood flow, placental abruption, as well as severe hyopxia or even asphyxia during parturition and immediately after birth (Sunshine, 2003). As a result of perinatal hypoxia, different patterns of brain injury can occur determined by the severity, duration and timing of hypoxia, and the effect on particular populations of cells, and the vascular immaturity of the brain (Myers, 1972). Even short periods of perinatal hypoxia can cause neuronal death, damage to the white matter and reduced growth of neural processes (Rees & Inder, 2005), and can have devastating neurodevelopmental consequences such as epilepsy, learning disabilities, mental retardation, cognitive and memory deficits, visual dysfunction, and cerebral palsy (Vannucci & Perlman, 1997; Dirnagl *et al.* 1999; Rennie *et al.* 2007).

Of relevance to cortical structure, two recent neuroimaging studies have shown reduction in brain volume concomitant (Bregant *et al.* 2013) and associated (Smith *et al.* 2015) with abnormal folding patterns of the cerebral cortex in young adults with a history of perinatal hypoxia. However, the cellular processes that underlie these changes cannot be identified using MRI. In rodents, fetal and neonatal hypoxia leads to reduced neuronal number in the cerebral cortex (Chung *et al.* 2015), to cortical inflammation and hypomyelination (Ortega *et al.* 2016), and to reduced cortical neuron and glial size (Schwartz *et al.* 2004). In addition, genes known to be involved in brain development, neuronal migration and gyrification, such as *DCX*, are affected by hypoxia: while acute hypoxia increases expression of *DCX*, chronic hypoxia reduces it (Schneider *et al.* 2012).

In addition to acute episodes of hypoxia, chronic fetal hypoxemia can arise as a consequence of placental insufficiency. This has been extensively modelled in pregnant sheep and guinea pigs during the second half of gestation in terms of the attrition of fetal growth (Mallard *et al.* 1999; Rees & Inder, 2005), but the consequence of placental insufficiency for cortical development during fetal life has not been closely examined, with one or two exceptions. For example, sheep fetuses exposed to mid-gestation hypoxaemia during the time that gyrification occurs rapidly (80–90 days' gestation) resulted in abnormal cortical folding of the frontal lobe, with a reduced folding index, a delay in neuronal migration,

and a decrease in neuronal density (Rees *et al.* 1997). Other studies have shown the SVZ to respond to hypoxia with either increased (Fagel *et al.* 2006; Felling *et al.* 2006; Yang *et al.* 2007) or decreased (Romanko *et al.* 2007; Spadafora *et al.* 2010) cell proliferation rates. Considering that manipulation of cell proliferation rates in the outer SVZ can clearly affect gyrification (Reillo *et al.* 2011; Nonaka-Kinoshita *et al.* 2013), it is probable that hypoxia impacts folding of the cerebral cortex via its effects on the SVZ. Also, chronic hypoxia modifies both FGF ligands and FGF-responsive RGCs in the perinatal brain (Ganat *et al.* 2002). Thus, considering that both FGF (Rash *et al.* 2013; Matsumoto *et al.* 2017) and RGCs (Reillo *et al.* 2011; Betizeau *et al.* 2013; Pilz *et al.* 2013) play a role in gyrification, this may be another mechanism by which chronic fetal hypoxia has an impact on cortical folding. However, to date, neither of these mechanisms has been assessed directly in models of perinatal hypoxia using gyrencephalic species.

**Infection.** Both viral and bacterial infections occur during pregnancy, and both have consequences for the structure of the developing brain. Viral infection has gained attention due to the recent Zika virus (ZIKV) outbreak, which appears to provoke microcephaly (Musso & Gubler, 2016). In humans, cytomegalovirus (CMV) during pregnancy has been consistently associated with major changes in brain structure (Picone *et al.* 2014). For example, CMV infection showed fusion of gyri and disarray of neuronal lamination (Jay *et al.* 1997), microcephaly (Perlman & Argyle, 1992) and polymicrogyria (Turkelson & Martin, 2009), but precisely how these major structural changes are brought about is unknown.

One of the main targets of CMV is the RGCs (van Den Pol *et al.* 1999), which are of critical importance for the proper development of the brain and play a significant role in the folding of the cerebral cortex (Reillo *et al.* 2011; Betizeau *et al.* 2013; Pilz *et al.* 2013). This strongly suggests a mechanism by which CMV infections cause cortical folding abnormalities, although it is yet to be validated experimentally in a model of maternal CMV infection using gyrencephalic species. This highlights the importance of exploring the consequences of perinatal CMV infections on brain folding to make an accurate prognosis and develop integral treatments that can ameliorate the effects of the infection on cortical folding.

Bacterial infections also adversely affect fetal brain development. Modelling maternal bacterial infection by administration of LPS reduced the mitotic index (Stolp *et al.* 2011; Carpentier *et al.* 2013) and cell cycle re-entry in neural progenitor cells (Carpentier *et al.* 2013), and altered the angle of their mitotic cleavage, suggesting an alteration in the symmetry of cell division in these cells (Stolp *et al.* 2011). LPS administration during pregnancy also reduced fetal brain weight, and altered the cortical patterning of neuron subtypes, changing the distribution and density of the different subtypes of pyramidal neurons during development (Carpentier *et al.* 2013). As proper control of neural progenitor cell cycle critically affects both cerebral cortical size (Rakic, 1995; Chenn & Walsh, 2002) and folding (Reillo *et al.* 2011; Nonaka-Kinoshita *et al.* 2013), these findings suggest a mechanism by which bacterial infections might impair the normal development and folding of the cerebral cortex.

**Multiple pregnancies.** The impact of multiplicity on fetal and neonatal development remains controversial. Although some agree that there is an influence of multiple pregnancies on perinatal development, others believe that there is no direct evidence suggesting that adverse outcomes are attributed to factors associated with multiple pregnancy such as preterm birth and IUGR (reviewed in (Ingram Cooke, 2010).

Twins, in general, are less encephalized – i.e. they have a lower brain/body mass ratio – compared to singletons (Hofman, 1984), and they also have a higher risk of cerebral palsy and microcephaly (Bejar *et al.* 1990). Overall, twins also have an increased risk of developing behavioural problems, possibly as a consequence of both prematurity and IUGR, which is more common in twins (reviewed in Ingram Cooke, 2010).

Newborns from multiple pregnancies show delayed cortical maturation, with reduced surface area and a reduced sulcation index when compared to singletons. Although delayed, these gyrification patterns followed the same developmental trajectory as seen in singletons and were not considered abnormal (Dubois *et al.* 2008). Similarly, in twins, the development of folding between 19 and 32 weeks PMA was delayed by 2–3 weeks when compared with singletons, although these differences disappeared after 33 weeks (Chi *et al.* 1977). Interestingly, no differences in prefrontal gyrification patterns have been observed in monozygotic compared to dizygotic twins, suggesting that environmental factors might have a similar or even greater impact on gyrification compared to genetic factors (Hasan *et al.* 2011).

## Conclusion

The study of the brain architecture and specifically the pattern of cortical folding has been an area of extensive and intensive investigation for many years. This review has covered what is known about the impact of abnormal intrauterine and maternal conditions during pregnancy on brain development with the purpose of demonstrating that we know little about how these affect the process of cortical gyrification. The process of folding of the

cerebral cortex is not only of fundamental interest, but impaired gyrification arises from many causes, and has many implications for the neonate and infant, including schizophrenia, autism, cognitive deficits and many others listed in this review.

There is ample knowledge on the molecules that play a role in gyrification (see Sun & Hevner, 2014; Fernandez *et al.* 2016). However the actual mechanism that drives gyrification remains elusive, and currently there are a few widely cited theories on why it is necessary for the cortical surface to become folded at all (reviewed in Ronan & Fletcher, 2015; Striedter *et al.* 2015). Nonetheless, it is important to be aware of the limitations and context of our experimental approaches when studying the gyrification process of the brain. Gene knockout/knockin studies are of limited value because rodents do not have gyrencephalic cortices, and for knockout studies, the absence of gene expression throughout the whole brain development process, long before gyrification starts, may be particularly misleading. In studies of gyrification there is a need to keep in mind the domino effect of disrupting global brain development before gyrification has started to properly address the nature of the hypothesis at hand.

Our understanding of the process of how the brain surface turns from a completely smooth entity in early stages in embryonic development towards an intricately and puzzling masterpiece is incomplete. Regrettably at present, we lack good animal models to study this fundamental process and therefore efforts should now focus on moving this field forward using gyrencephalic and not lissencephalic species. This should include broadening our scope to study the impact of environmental factors and physiological context surrounding the process of cortical folding. Study of not only the genetically driven mechanisms of cortical folding but also the impact of intrauterine and maternal physiology on this process is paramount to understand gyrification completely. This will surely prove an invaluable tool for better diagnosis and prevention of the consequences of a misfolded cerebral cortex.

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## Additional information

### Competing interests

None declared.

### Author contributions

All authors have read and approved the final version of this manuscript and agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All persons designated as authors qualify for authorship, and all those who qualify for authorship are listed.

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