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Interaction between genes and the relational environment during development of the social brain

Humans have complex brains. These have evolved over a vast phylogenetic history. Scientists are discovering genetic innovations that may have contributed to brain development over evolutionary time. The science of comparative genomics reveals when during evolution each such formative genomic event occurred and the mechanism by which it arose. However, genetics are necessary but not sufficient to account for our mental capacities. For example, our ability to interact as persons (to practise theory of mind) is not genetically encoded, but is learned. During infancy and childhood, brains cannot follow normal developmental trajectories in the absence of attentive, loving caregiving. Human brain development and function require personal input. We share in the fullness of being human by interpersonal relationship, and a Christian interpretation of this fact is that human flourishing requires that people know, and are known by, God.

Keywords: social interaction, theory of mind (ToM), child neglect, stress, cerebral development, language, nurture, being human, knowing.

Popular understandings of science have attributed to genes an almost unlimited capacity to determine the development of brain and mind. But our personal histories are the outcome of a complex interplay of genetics and environment, as has been reviewed comprehensively by Alexander.¹ The present paper will consider four issues. It will present illustrative data describing genetic change in the evolutionary development of the human brain. It will review recent studies investigating the role of language and the importance of nurture (as revealed by studies of neglected children) in the formation of each individual's social brain. It will conclude by considering implications for a biblical view of humanity as a social being destined for fellowship with God.

1. Evolution

Our genetics underlie everything that we may achieve with respect to our mental and personal capacities. After all, we have a human genome and not that of a wombat; and some very small mutations can have devastating impacts on brain and mind. Human brain tissue shows distinctive features early in fetal development and in cultured organoids – increased cellularity, deeper cerebral folds,

¹ Alexander, D. *Genes, Determinism and God*, Cambridge: Cambridge University Press (2017).

long-distance connections, lengthened mitoses in progenitor cells and delayed development.² Our behaviour is intimately connected with our biochemistry. The hormone oxytocin communicates between brain cells and sustains social behaviour, such as bonding with mates and parenting – and thus provides the conditions that underlie acts of kindness.³ However, parenting styles and personal histories also affect the way the oxytocin system operates.⁴ Genes and experience interact to form us as personal beings.

Our brains have formed over a vast history of evolutionary development. The evidence for this established paradigm has accrued from multi-species genome comparisons, a field that has flourished from about the turn of the century. Molecular geneticists are identifying many genetic changes that are associated with brain development.⁵ Supplementary material provides illustrative examples of when and how candidate genetic innovations arose through our evolutionary history. The most recent events described occurred in the lineage specific to humans; the earliest, in an ancestor of all mammals. Christians must integrate this evolutionary history into their reflections on our status as biological creatures – anthropoid primates – beloved of, and called by, God.

An unexamined philosophical geneticism posits that genes underlie all our behaviour and values. I have heard undergraduate students argue that natural selection underlies all human virtue. The idea of the omnipotent gene has been promulgated by the media, and in high school and undergraduate education.⁶ Perhaps the myth of genetic determinism endures because of its connections with the promise of a technologically-attainable utopia: ‘Genomic science, moving within a space of neoliberal markets, makes the rhetoric of hype and hope indispensable precisely because credible promises generate cycles of investment and value. The determinist gene (or the determinist genome) is part of this social machinery of expectations and profits.’⁷ The ‘selfish gene’ may in fact be the ‘neo-liberal gene’, or the ‘social control gene’. Or even the ‘money-

2 as described in the meeting report, Gibbons, A. ‘New tools offer clues to how the human brain takes shape’, *Science* (2017) 358, 705; Bermudez-Mora, F., Badsha, F., Kanton, S. et al. ‘Differences and similarities between human and chimpanzee neural progenitors during cerebral cortex development’, *eLife* (2006) 5, e18683.

3 Preston, S.D. ‘The rewarding nature of social contact’, *Science* (2017) 357, 1353.

4 Rilling, J.K. & Young, L.J. ‘The biology of mammalian parenting and its effect on offspring social development’, *Science* (2014) 345, 771; Baker, M., Lindell, S.G., Driscoll, C.A. et al. ‘Early rearing history influences oxytocin receptor epigenetic regulation in rhesus macaques’, *Proc Natl Acad Sci USA* (2017) 114, 11769.

5 Mitchell, C. & Silver, D.L. ‘Enhancing our brains: Genomic mechanisms underlying cortical evolution’, *Seminars in Cell and Developmental Biology* (2017), in press.

6 Carver, R.B., Castera, J., Gericke, N. et al. ‘Young adults’ belief in genetic determinism, and knowledge and attitudes towards modern genetics and genomics: the PUGGS questionnaire’, *PLoS One* (2017) 12, e0169808.

7 Esposito, M. ‘Expectation and futurity: the remarkable success of genetic determinism’, *Stud Hist Philos Biol Biomed Sci* (2017) 62, 1.

for-genetics research gene’.

An overweening estimation of the power of genes may influence scientists. Researchers have sought to ascertain how human and chimp brains differ in gene activity, by characterising the set of RNA molecules generated in cells (the transcriptome).⁸ These approaches are inherently ambiguous because they compare systems that differ by two independent variables. Firstly, what is the difference in brain gene activity resulting from genetics? To address this, we would need to study brain transcriptomes of chimps and chimp-reared humans. Secondly, what is the difference in gene activity resulting from social and cultural environment? To address this, we would have to compare brain transcriptomes of humans who have been denied all social contact with those of humans who have been raised in richly interactive societies. But we cannot banish infants to the wild, only to biopsy their brains. Such experiments are forbidden.

An attempt to perform the ‘forbidden experiment’ was attributed to Frederick of Hohenstaufen II (1194-1250). He sought to raise children under conditions of socioemotional (but not physical) deprivation. To quote his chronicler, Friar Salimbene, Frederick ordered ‘foster-mothers and nurses to suckle and bathe and wash the children, but in no wise to prattle or speak with them; for he would have learnt whether they would speak the Hebrew language (which had been the first), or Greek, or Latin, or Arabic, or perchance the tongue of their parents of whom they had been born’. It appears that children raised in the absence of verbal communication failed to flourish and soon died. ‘But he laboured in vain, for the children could not live without clappings of the hands, and gestures, and gladness of countenance, and blandishments.’⁹

The effects of social interaction on brain development can be studied in less radically intrusive ways. Some children are raised with reduced socioemotional input. How does deafness affect children’s understanding of other people’s mental states, including their feelings, intentions and beliefs? How does childhood neglect affect mental and neural development?

2. Language and theory of mind

The highly developed human ability to interpret other people’s mind states is known as theory of mind (ToM), and its development during childhood is called mentalisation. Preschool children with specific language impairment struggle with ToM. They show deficits in cognitive ToM: the ability to share a focus of intention with another, and to perceive that someone else may understand

8 Sousa, A.M.M., Meyer, K.A., Santpere, G. et al. ‘Evolution of the human nervous system function, structure and development’, *Cell* (2017) 170, 226.

9 Masson, G., *Frederick II of Hohenstaufen*, London: Secker & Warburg (1957), p. 230; quote available at <http://sourcebooks.fordham.edu/halsall/source/salimbene1.html>

a situation incorrectly and act consistently on that misunderstanding. They also show deficits in affective ToM: the ability to interpret facial expressions and emotions. A hypothesis that naturally arises from this association is that language facilitates the development of ToM.¹⁰ In support of this suggestion, teacher-led conversations about mental states with primary school children facilitated the development of the children's ToM.¹¹ The capacity for ToM is learned, and communication with people who possess ToM is the medium by which it is transmitted across generations.

If exposure to mental-state language is required for the development of ToM, then deafness (in the absence of compensating modes of communication) would lead to defective ToM acquisition. Dutch children with moderate hearing loss showed delayed development of ToM relative to children with normal hearing. The hearing-impaired children showed normal understanding of other people's intentions, but delayed understanding of others' desires and beliefs – presumably because they were exposed to less social communication ('mental-state talk').¹² Japanese children with hearing loss also showed delays in the development of ToM. The deficit was related to vocabulary and appreciation of sentence structure (syntax), not to age or non-verbal intelligence. The development of ToM requires that infants engage in conversation that conveys information about other people's thoughts and feelings.¹³ In deaf adults who communicate only through a private sign language, ToM remains underdeveloped, even though memory and comprehension are normal, and visual capacities are heightened. A lifetime of social interaction cannot generate ToM in the absence of 'participation in a shared linguistic community' and exposure to 'mental-state verbs'.¹⁴

It has been argued that the capacity for ToM (or mind reading) is learned to the same extent as the capacity to read print. 'Most, possibly all, human neurocognitive skills are shaped by culture and many are culturally inherited but the parallels between mind reading and print reading are extraordinary...' (as exemplified, Table 1). 'If a group of human infants managed to survive on a desert island, in a cruel Lord of the Flies-like experiment, they would be no more likely to develop a theory of mind and become explicit mind readers than

10 Vissers, C. & Koolen, S. 'Theory of mind deficits and social emotional functioning in preschoolers with specific language impairment', *Front Psychol* (2016) 7, 1734.

11 Bianco, F. & Lecce, S. 'Translating child development research into practice: Can teachers foster children's theory of mind in primary school?', *Br J Educ Psychol* (2016) 86, 592.

12 Netten, A.P., Rieffe, C., Soede, W. et al. 'Can you hear what I think? Theory of mind in young children with moderate hearing loss', *Ear Hear* (2017) 38, 588.

13 Fujino, H., Fukushima, K. & Fujiyoshi, A. 'Theory of mind and language development in Japanese children with hearing loss', *Int J Pediatr Otorhinolaryngol* (2017) 96, 77.

14 Gagne, D.L. & Coppola, M. 'Visible social interactions do not support the development of false belief understanding in the absence of linguistic input: evidence from deaf adult home-signers', *Front Psychol* (2017) 8, 837.

to develop a writing system and become literate print readers.’¹⁵

Table 1. Parallels between print reading and mind reading (from ref. 15)

	print reading	mind reading
brain areas	occipito-temporal	medial prefrontal
developmental disorders	dyslexia	autism spectrum
capacity develops to	late adolescence	late adolescence
cognitive effort needed	executive processes	executive processes
individual variation	environmental > genetic	environmental > genetic
teaching is needed to relate	print to referents and pronunciation	mental states, situations and behaviour

It is easy to assume in our technologically driven society that the fullness of humanness is genetically specified. Rather it is acquired in proportion to the quality of our nurture as social beings. Severe hearing deficiency in infancy leads to compromised incidental learning, difficulties in expressing and regulating emotions, problems in socialisation, and internalising (depression, anxiety) and externalising (disruptive behaviour, aggression) behaviour. In children with normal hearing and in hearing-impaired children provided with cochlear implants, language proficiency is related to psychosocial development.¹⁶ When infants are exposed to language, they do not learn that language; rather, language forms their social brains.¹⁷

3. Neglect and the social brain

The neglect of young children – the absence of devoted input from parents or other caregivers – is a widespread phenomenon with haunting echoes of Frederick’s ‘forbidden experiment’. A child’s genetic potential is sensitive to the quality of attentive nurturing. Multiple interacting processes – stress and endocrine responses, inflammation and epigenetic change – mediate between early life stress, the development of brain structure and mental well-being.¹⁸

Childhood abuse, including emotional and physical neglect, influences the

15 Heyes, C.M. & Frith, C.D. ‘The cultural evolution of mind reading’, *Science* (2014) 344, 1357.

16 Netten, A.P., Rieffe, C., Ketelaar, L. et al. ‘Terrible twos or early signs of psychopathology? Developmental patterns in early identified preschoolers with cochlear implants compared with hearing controls’, *Ear and Hearing* (2018), 39, 495.

17 Halpern, M. ‘How children learn their mother tongue: they don’t’, *J Psycholinguist Res* (2016) 45, 1173.

18 Gonzalez-Mariscal, G. & Melo, A.I. ‘Bidirectional effects of mother-young contact on the maternal and neonatal brains’, *Adv Exp Med Biol* (2017) 1015, 97; Nemeroff, C.B. ‘Paradise Lost: the neurobiological and clinical consequences of child abuse and neglect’, *Neuron* (2016) 89, 892.

development of personality traits (such as fearfulness) that lead to major depression, both in the general population¹⁹ and in people with schizophrenia.²⁰ Functional magnetic resonance imaging (MRI) of the brains of depressed or schizophrenic adults engaged in ToM reasoning indicates that, in each case, childhood maltreatment, including neglect, correlates with altered brain activity. In depressed people, the amygdala (a region involved in processing emotions and anxiety) is activated. Perhaps hurtful memories are used as lenses by which depressed people interpret ongoing social interactions.²¹ In schizophrenic people, the dorsomedial prefrontal cortex (PFC) (a region associated with the interpretation of other's intentions) is activated. This may indicate difficulties in inferring others' mind-states.²²

Optimal emotional development requires the formation of connections between the medial PFC and the amygdala. Parental nurture contributes to regulation of infant emotions and responses to stress (parental buffering), and may facilitate the development of medial PFC-mediated control of the amygdala during a critical period of neuroplasticity. Once formed, the neural circuits remain stable. Parental separation or neglect may lead to accelerated formation of adult-like medial PFC-amygdala circuitry (possibly with loss of plasticity), internalising symptoms (withdrawal), deficits in emotion regulation and potentially, psychopathologies.²³

Childhood abuse, including neglect, is a risk factor for self-inflicted injury²⁴ and for suicidal behaviour.²⁵ It promotes externalising behaviours that are related to lower age of initiation of alcohol and cannabis consumption.²⁶ Adverse

19 Dannehl, K., Rief, W. & Euteneuer, F. 'Childhood adversity and cognitive functioning in patients with major depression', *Child Abuse Negl* (2017) 70, 247; Otsuka, A., Takaesu, Y., Sato, M. et al. 'Interpersonal sensitivity mediates the effects of child abuse and affective temperaments on depressive symptoms in the general adult population', *Neuropsychiatric Disease and Treatment* (2017) 13, 2559.

20 Okubo, R., Inoue, T., Hashimoto, N. et al. 'The mediator effect of personality traits on the relationship between childhood abuse and depressive symptoms in schizophrenia', *Psychiatry Res* (2017) 257, 126.

21 Hentze C, Walter H, Schramm E. et al. 'Functional correlates of childhood maltreatment and symptom severity during affective theory of mind tasks in chronic depression', *Psychiatry Res* (2016) 250, 1.

22 Quide, Y., Ong, X.H., Mohnke S. et al. 'Childhood trauma-related alterations in brain function during a theory-of-mind task in schizophrenia', *Schizophrenia Res* (2017) 189, 162.

23 Gee, D.G. 'Sensitive periods of emotion regulation: influences of parental care on fronto-amygdala circuitry and plasticity', *New Dir Child Adolesc Dev* (2016) 153, 87.

24 Kavurma, C., Tas, F.V., Demirgoren, B.S. et al. 'Do serum BDNF levels vary in self-harm behavior among adolescents and are they correlated with traumatic experiences?', *Psychiatry Res* (2017) 258, 130.

25 Liu, J., Fang, Y., Gong, J. et al. 'Associations between suicidal behaviour and childhood abuse and neglect: a meta-analysis', *J Affect Disord* (2017) 220, 147.

26 Proctor, L.J., Lewis, T., Roesch, S. et al. 'Child maltreatment and age of alcohol and marijuana

childhood experiences, especially neglect, have been associated with poor adjustment to high school learning environments,²⁷ with elevated high school dropout rates²⁸ and, by affecting executive functions (the capacity to plan and focus), with difficulties in adapting to university life.²⁹

Memories of childhood abuse, including physical and emotional neglect, during pregnancy are associated with depression,³⁰ and with subtle changes in thyroid function (as measured by altered levels of thyroid-stimulating hormone).³¹ Compromised thyroid hormone function may affect neural development in the fetus. Indeed, childhood emotional neglect has been associated with increased rates of stillbirth.³² Childhood maltreatment, most commonly neglect, tends to be transmitted across generations.³³ Mothers who were neglected as children tend to be less attentive to their own infants. Functional MRI shows that such mothers showed heightened responses to their children's cry in the cingulate and insular cortices – areas responsible for empathy and emotion processing. The exaggerated neural activity may represent an aversive response; but regardless of the mechanism, it is clear that early neglect is encoded in long-term neural activity.³⁴

Neglect changes brain structure. A study using structural MRI showed that childhood neglect leads to reduced right and left caudate volumes (in females, not males). These regions of the subcortex are associated with regulation of

initiation in high-risk youth', *Addict Behav* (2017) 75, 64-69; Scheidell, J.D., Quinn, K., McGorray, S.P. et al. 'Childhood traumatic experiences and the association with marijuana and cocaine use in adolescence through adulthood', *Addiction* (2018) 113, 44.

27 Oh, I. & Song, J. 'Mediating effect of emotional/behavioural problems and academic competence between parental abuse/neglect and school adjustment', *Child Abuse Negl* (2017), in press.

28 Morrow, A.S. & Villodas, M.T. 'Direct and indirect pathways from adverse childhood experiences to high school dropout among high-risk adolescents', *J Res Adolesc* (2018), 28, 327.

29 Welsh, M.C., Peterson, E. & Jameson, M.M. 'History of childhood maltreatment and college academic outcomes: indirect effects of hot execution function', *Front Psychol* (2017) 8, article 1091.

30 Yildiz Inanici, S., Inanici, M.A. & Yoldemir, A.T. 'The relationship between subjective experience of childhood abuse and neglect and depressive symptoms during pregnancy', *J Forensic Leg Med* (2017) 49, 76.

31 Moog, N.K., Heim, C.M., Entringer, S. et al. 'Childhood maltreatment is associated with increased risk of subclinical hypothyroidism in pregnancy', *Psychoneuroendocrinology* (2017) 84, 190.

32 Freedman, A.A., Cammack, A.L., Temple, J.R. et al. 'Maternal exposure to childhood maltreatment and risk of stillbirth', *Ann Epidemiol* (2017) 27, 459.

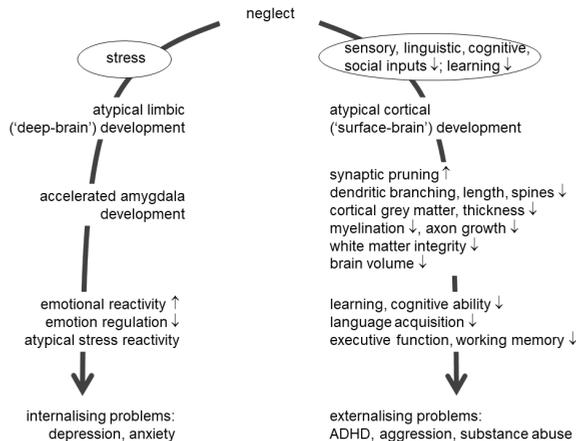
33 Plant, D.T., Jones, F.W., Pariante, C.M. & Pawlby, S. 'Association between maternal childhood trauma and offspring childhood psychopathology: mediation analysis from the ALSPAC cohort', *Br J Psychiatry* (2017) 211, 144.

34 Wright, D.B., Laurent, H.K. & Ablow, J.C. 'Mothers who were neglected in childhood show differences in neural response to their infant's cry', *Child Maltreat* (2017) 22, 158.

cognitive and emotional processes.³⁵ People who were maltreated as children and who suffer from bipolar disorder possessed decreased volumes of grey matter (cell bodies) in the PFC. Neglect in particular was associated with reduced volumes of grey matter in thalamic regions which relay information regarding potential threats between the PFC and amygdala, thus perturbing PFC-thalamic-amygdala circuitry.³⁶ Abuse, including neglect, is a risk factor for eating disorders. Women with eating disorders who were neglected as children possessed reduced grey matter volumes and white matter (cell connections) integrity in particular areas relative to those who were not maltreated. For example, a fold called the left inferior temporal gyrus processes information regarding body image and shows decreased volume.³⁷

In summary, neglect affects multiple circuits in the developing brain. The circuitry considered above emphasises the limbic ('deep-brain') network. Stress causes accelerated development of the amygdala, leading to abnormal regulation of emotion and stress responses. Sequels to these deficits include various forms of internalising psychopathology (Figure 1, left).³⁸

Figure 1. Models relating childhood neglect to abnormal development of brain and mind. Abstracted from ref. 37



35 Frodl, T., Janowitz, D., Schmaal, L. et al. 'Childhood adversity impacts on brain subcortical structures relevant to depression', *J Psychiatr Res* (2017) 86, 58.

36 Duarte, D.G.G., de C. Neves, M., Albuquerque, M.R. et al. 'Gray matter brain volumes in childhood-maltreated patients with bipolar disorder type I: a voxel-based morphometric study', *Journal of Affective Disorders* (2016) 197, 74.

37 Monteleone, A.M., Monteleone, P., Esposito, F. et al. 'The effects of childhood maltreatment on brain structure in adults with eating disorders', *World J Biol Psychiatry* (2017), in press.

38 As summarised by McLaughlin, K.A., Sheridan, M.A. & Nelson, C.A. 'Neglect as a violation of species-expectant experience: neurodevelopmental consequences', *Biol Psychiatry* (2017) 82, 462.

Studies of childhood neglect as described above reveal major effects on socio-emotional and cerebral development, but some children have been exposed to conditions of deprivation that are even closer to those of the 'forbidden experiment'. The effects of time spent in understaffed, severely neglectful orphanages (such as those of the Communist era of Romania), and of subsequent adoption into stable families, have been studied in both the UK and the US.

Children who spent more than six months in an orphanage before adoption (relative to children adopted before six months) showed persistently higher incidences of autism spectrum disorder, social disinhibition, inattention and overactivity. Intellectual ability (as measured by IQ) and ToM was suppressed. There was evidence of epigenetic change (DNA methylation) in the late-adopted group. With time from placement in foster care, such children showed recovery in cognitive performance, although they achieved less educationally. However, a surge of emotional difficulties occurred during young adulthood.³⁹

An MRI method (diffusion tensor imaging) has been used to investigate the structural integrity of white matter tracts in the brain. These are cell extensions, such as axons, that mediate communication between cells. Neglect impaired the integrity of white matter tracts, especially of the fronto-striatal circuitry (external capsule) and the corpus callosum. White matter abnormalities may reflect defects in the myelination (insulation) of axon fibres. This damage was associated with the development of depression and anxiety. Girls, but not boys, who were fostered into supportive families at two years, showed improvements in emotional symptoms between eight and twelve years.⁴⁰

Children reared in orphanages are deprived of sensory, linguistic, cognitive and social stimulation. The absence of such inputs may suppress the development of cortical ('surface-brain') circuits. Neglect may lead to extreme pruning of synapses (connections between brain cells), loss of dendritic spines, and suppression of dendritic branching and length. Structural MRI studies showed that institutionalised children had decreased volumes of grey matter, with subsequent thinning of the brain cortex; and of white matter. Loss of myelination leads to loss of white matter integrity and communication between brain regions. Brain volumes are reduced. The result is loss of learning, language acquisition, cognitive functions and (via loss of working memory) executive

39 Kumsta, R., Marzi, S.J., Viana, J. et al. 'Severe psychosocial deprivation in early childhood is associated with increased DNA methylation across a region spanning the transcription start site of *CYP2E1*', *Transl Psychiatry* (2016) 6, e830; Sonuga-Barke, E.J.S., Kennedy, M., Kumsta, R. et al. 'Child-to-adult neurodevelopmental and mental health trajectories after early life deprivation: the young adult follow-up of the longitudinal English and Romanian Adoptees study', *Lancet* (2017) 389, 1539.

40 Bick, J., Zhu, T., Stamoulis, C. et al. 'Effect of early institutionalization and foster care on long-term white matter development', *JAMA Pediatrics* (2015) 169, 211; Bick, J., Fox, N., Zeanah, C. & Nelson, C.A. 'Early deprivation, atypical brain development, and internalizing symptoms in late childhood', *Neuroscience* (2017) 342, 140.

function (the ability to act purposefully) (Figure 1, right). After adoption into supportive families, by eight years of age, only white matter volume recovered to normal levels.⁴¹

All sorts of claims are made for genetic control of our being. However, genes do not dictate the formation of the brain and of the way people relate, make love, defend their territories, organise their societies, practise virtue or visit savagery upon each other. The multimillion year-long evolution of genes supporting neural systems can produce only a neural primordium, poised to respond to social inputs that are fragile, variable over short timescales (a generation or two) and potentially ephemeral. A common question encountered is, 'Are we still evolving?' The answer is, 'Of course – but imperceptibly slowly.' Of infinitely greater urgency is the fact that the nurturing of each generation, and the continued vitality of society and of a moral humanity, are highly vulnerable to excessive material expectation, preoccupation with hedonistic pursuits, substance abuse, ideological whim, social conflict, and the neglect of moral imperatives (of which many have been informed by the gospel of Jesus). The myth of genetic determinism is a fantasy.

4. Beyond genes: creating social brains

The organisation of matter in human brains is not a simple outcome of genetic specification. Brains develop in response to dispositions, values and visions that science cannot measure, such as love. One might include the fruit of the Spirit (Gal. 5:22-25) in the inventory of cues regulating cerebral gene expression. Several imperatives follow.

1. Bringing up our children

The growth of children's prosocial behaviour (their socialisation) is not innate but learned. A virtue such as helping is facilitated not merely by verbal encouragement and rewards, but by the way children observe those who have learned to be helpful before them.⁴² The problem-solving determination of eighteen-month-old infants is enhanced by watching adults persist with a problem.⁴³

41 Tibu, F., Sheridan, M.A., McLaughlin, K.A. et al. 'Reduced working memory mediates the link between early institutional rearing and symptoms of ADHD at 12 years', *Front Psychol* (2016) 7, article 1850; Bick, J. & Nelson, C.A. 'Early experience and brain development', *WIREs Cogn Sci* (2017) 8, e1387; McLaughlin, K.A., Sheridan, M.A. & Nelson, C.A. 'Neglect as a violation of species-expectant experience: neurodevelopmental consequences', *Biol Psychiatry* (2017) 82, 462.

42 Dahl, A. 'How, not whether: contributions of others in the development of infant helping', *Curr Opin Psychol* (2018) 20, 72.

43 Leonard, J.A., Lee, Y. & Schultz, L.E. 'Infants make more attempts to achieve a goal when they see adults persist', *Science* (2017) 357, 1290.

Even attending to faces is itself learned by visual exposure to faces, which enables the formation of a dedicated brain region, located in the superior temporal sulcus.⁴⁴ Genes can provide only the conditions that make learned social behaviour possible.

The most God-like, creative thing humans can do is to provide the conditions of love and support that optimally support neural and mental (including social) development in their children. From birth (if not during development in utero, when they learn to recognise their mothers' voices),⁴⁵ children's brains are formed by the environment of love, faith and hope in which they are nurtured. We are able to love, show compassion and pursue moral visions only because we learned our humanity from others.

Self-perpetuating cycles of parental neglect have produced multitudes of children who are endowed with enormous genetic potential but whose mental and socio-emotional development is disrupted. Childhood maltreatment is a global epidemic. A recent study in Germany found that almost one quarter of people in the general population had experienced some form of childhood maltreatment, with the most common being emotional (7.1%) and physical (9.0%) neglect.⁴⁶ Much of the neural and mental damage inflicted in the first few years of life is poorly reversible. Herein lies a priority for pastoral mission in a broken society.

2. The viability of society

The nature-versus-nurture, genes-versus-environment dichotomy is dead. Genes are necessary but insufficient when it comes to the creation of social brains. The possession of human genomes cannot guarantee the perpetuation of human society. The evolution of genes or changes in gene frequencies – or indeed targeted editing of the germline – can have infinitesimally small effects on overall human well-being.

Intense socio-emotional input is required for optimal language acquisition, cognition, ToM, mental health, grey and white matter formation, and brain volume. It follows that the geneticist claims sometimes made for genes (that they control human reciprocity, ethics, virtue, love ...) must be seen as ungrounded mythology. Genes in human brains produce virtuous and loving human persons *only* when their expression is regulated by the nurture of those who have

44 As shown by a study of macaques; Arcaro, M.J., Schade, P.F., Vincent, J.L. et al. 'Seeing faces is necessary for face-domain formation', *Nature Neuroscience* (2017) 20, 1404.

45 Ferrari, G.A., Nicolini, Y., Demuru, E. et al. 'Ultrasonographic investigation of human fetus responses to maternal communicative and non-communicative stimuli', *Front Psychol* (2016) 7, 354.

46 Witt, A., Brown, R.C., Plener, P.L. et al. 'Child maltreatment in Germany: prevalence rates in the general population', *Child Adolesc Psychiatry Ment Health* (2017) 11, 47.

themselves learned and practised virtue and love. The data reviewed above indicate that an optimal social environment optimises expression of those genes that underlie the social brain.

Westerners live and think in the shadow of Enlightenment individualism. There is great wisdom in the African proverb *umuntu ngumuntu ngabantu*, which may be translated, 'a person is a person through other persons'.⁴⁷ Perhaps Westerners have lost the sense that the potent dynamic of communal cohesion creates persons. The presupposition that every man is an island, a bundle of rights to be heroically defended, must be seen as idiosyncratic and self-defeating. From an African perspective, the saying, 'a person is a person through persons' is 'opposed to all kinds of individualism' and 'collectivism of a European kind'. It stresses that community is not 'an aggregated sum of individuals'. There is an 'ontological independence to human society'. African thought starts with society and moves to individuals.⁴⁸

An English equivalent might be 'humans making humans human'.⁴⁹ Such communitarian thinking should be native to Christians. Whereas Descartes famously stated that 'I think therefore I am', we can affirm that 'I am known therefore I am'. But the immateriality of 'knowing' means that it can be overlooked by scientists and journalists (and consumers) who focus on measurable quantities. CS Lewis, in his Narnia stories, seemed to point to the vulnerability of our social being. When Aslan called the Talking Beasts into existence, he gave them Narnia and himself, but warned them: 'The Dumb Beasts whom I have not chosen are yours also. Treat them gently and cherish them but do not go back to their ways lest you cease to be Talking Beasts. For out of them you were taken and into them you can return'.⁵⁰ The implication is clear. Just as Narnia's Beasts could lose their gift of sapience, so human sociality could be lost through neglect of self-giving and self-forming relationality.

3. Knowing God

If knowing other humans confers upon us the richness of humanness, then it might be suggested analogously that knowing God confers upon us the life of the New Age, the *zoe aionios*. The medium through which knowledge of the

47 De Gruchy, J.C. *Christianity and Democracy*, Cambridge: Cambridge University Press (1995), p. 191.

48 Villa-Vicencio, C. & De Gruchy, J. *Doing Ethics in Context*, Maryknoll: Orbis and Cape Town: David Philip (1994), p. 29.

49 Evans, J.H. *What is a Human?*, New York: Oxford University Press (2016), p. 159.

50 Available at <https://gutenberg.ca/ebooks/lewiscs-magiciansnephew/lewiscs-magiciansnephew-00-h.html> (Ch X); sequel at <https://gutenberg.ca/ebooks/lewiscs-lastbattle/lewiscs-lastbattle-00-h.html> (Chs X, XIV).

divine Mind (ToM) is imparted is the divine Word, Jesus.⁵¹

Key to this understanding is the prayer of Jesus in which he stated that 'eternal life is to know you, the only true God, and to know Jesus Christ whom you sent' (John 17:3). Here is a clear statement that knowing and living are inseparable, if not equivalent. Indeed, the 'biblical understanding of life connects it with knowing – existential knowing. It thus implies entering into relationship – with God, with other persons and, to a lesser extent with things'. The fullness of life then 'consists in cognitive and responsive relationships'⁵² with those who were persons before us: our parents, families, communities and ultimately the God who is love. Life is not a property of an object existing in lonely isolation.⁵³

Similarly, when St Paul articulated his deepest yearnings (Phil. 3:10-12), he gave first place to knowing God, and linked that with the experience of knowing the power of Christ's resurrection (transformation within daily life), and ultimately with the hope of resurrection (transformation to New Age life). Wright's translation stresses the knowing-living connection: Paul's passion is 'knowing him, knowing the power of his resurrection, and knowing the partnership of his sufferings...that I may arrive at the final resurrection of the dead'.⁵⁴ The theme of knowing God is deeply rooted in the Hebrew Scriptures; and the claim is made also in the opposite direction: we are known by God.⁵⁵ The author of Psalm 139 connected God's knowledge of him closely to his life-long development from unformed substance *in utero* to his thoughts, speech and actions. You know me; you know everything I do; you know all my actions; you know what I will say; when my bones were being formed in secret ... you knew.⁵⁶ Steane thus describes Christian faith as a dawning realisation that I am known by someone.⁵⁷

Human genes generate a neural substrate which becomes organised into a social brain only following reception of scheduled environmentally (socially)-sourced morphogenic signals. Human brain development is not autonomous

51 As emphasised in the Johannine literature (Jn 1:1, 14; 1Jn 1:1; Rev. 19:13); and underlying St Paul's claim that 'we however have the mind of Christ' (1 Cor. 2:16).

52 Spanner, D. *Biblical Creation and the Theory of Evolution*, Exeter: Paternoster Press (1987), p. 71.

53 I once observed, in a suspension of cells from a patient's lung cancer in a plastic dish, a little block of ciliated bronchial epithelium being propelled in sweeping arcs through the medium. It was both beautiful (those synchronously beating cilia!) and hauntingly meaningless (isolated from the whole body) – wondrously living but in a desolate minimalistic sense.

54 Wright, N.T. *Paul and the Faithfulness of God*, London: SPCK (2013), p. 832.

55 *ibid.*, 987-988; Old Testament instances of this theme of knowing include Isa. 11:9; Jer. 9:24, 31:33-34.

56 Psa. 139:1-5, 23 (knowing); cf 13-16 (creation, being); New Testament emphasis on God's knowledge of his people is found in Gal. 4:9; 1Cor. 8:1-3; 13:12; 2Tim. 2:19.

57 Steane, A. *Faithful to Science*, Oxford: OUP (2014), 110.

but is contingent upon species-expectant experience,⁵⁸ the anticipation of necessary developmental cues that arise outside the individual. Longitudinal studies indicate that human flourishing ('complete human well-being') requires social inputs. These include participation in a religious community.⁵⁹ We are incomplete in ourselves, and this is reminiscent of St Augustine's realisation that 'God ... you have made us for yourself, and our hearts are restless till they find their rest in you.'⁶⁰ A Christian interpretation of the requirements for flourishing is that knowing God is a vital instance of species-expectant experience: growth towards the full potentiality of each individual awaits the transformative experience of encounter with God.

A hope-filled observation is that, in adulthood, the human brain retains plasticity – the capacity to undergo reorganisation – during training in socio-affective skills such as compassionate attitudes, and in socio-cognitive skills such as ToM. This has been shown by structural MRI studies of brains of people who have engaged in mental exercises for three-month periods. Areas of the cerebral cortex increased in thickness.⁶¹ From this analogy, it may be posited that the experience of knowing God has an enduring capacity to change, mould, reconfigure our brains and minds. Some may find this approach excessively brain- or matter-focused. I am not qualified to speculate on all the ways by which the Spirit of God may transform people and leave the divine imprint on their minds. But the way by which relationship forms brain and mind is at the very least a powerful analogy.

To conclude, human brains require human genes. But the development of the human social brain, and the ability to function in a human society, are contingent upon species-expectant interpersonal knowing. Jesus extended the formative effect of 'knowing' beyond human society: 'Now that you have known me, you will know my Father also' (Jn 14:7). The structure of my brain, from which my personhood, values, loves and yearnings arise, will endure beyond the dissolution of my body to find new form in the New Creation. It has been to some extent formed by God's knowledge of me and by my painfully clouded and incomplete knowledge of him.

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58 McLaughlin, Sheridan & Nelson *op. cit.*, (41).

59 Vanderweele, T.J. 'On the promotion of human flourishing', *Proc Natl Acad Sci USA* (2017) 114, 8148; human flourishing includes physical and mental health, meaning, purpose, character, virtue, and close social relationships.

60 Lane, T. *The Lion Concise Book of Christian Thought*, Tring: Lion (1984), p. 43.

61 Valk, S.L., Bernhardt, B.C., Trautwein, F.-M. et al. 'Structural plasticity of the social brain: differential change after social-affective and cognitive mental training', *Sci Adv* (2017) 3, e1700489.