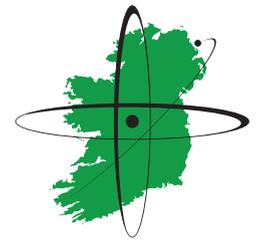


Human Genetics and the Image of God

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Abstract. Some people still deny that extensive evolutionary change (macroevolution) has occurred. They deny that the existing mammalian orders or the primates (including *Homo sapiens*) could have evolved from founding progenitors. Others deny that evolutionary mechanism is compatible with the biblical assertion that God is Creator of the spectacularly diverse biosphere of which we are a part. We are told that once we understand the mechanism of our evolution, we can no longer think of ourselves as having a divinely ordained purpose. An answer to the 'how' question supposedly eliminates the 'why' question.

This paper argues that such controversies are eminently resolvable. Although genome science has generated compelling evidence for the evolutionary origins of human beings, genetics cannot describe fully what makes us human. Our humanity is the outcome of three kinds of adaptive history: the influence of biological evolution through the genes, the evolution of neural circuits through experience, and the influence of cultural evolution. Just as we have human life through our relationships with other people, so we receive spiritual life in knowing the Word of God, spoken in Jesus Christ.

Setting the Scene

It is ironic that opposition to the idea of human evolution has reached a crescendo even as evidence for human evolution has laid the issue to rest. We might expect overarching theories to be based on complex arguments comprehensible only to the specialist. However genome science has generated accessible evidence that dispels uncertainty as to human evolutionary origins.

Our genetic connectedness to (other) apes cannot call into the question the primary datum of our lives: that we are relational beings. Indeed it is our relationships with other people (and, I believe, with God) that constitute us as human. The basis of Christian faith is that a personal God has revealed himself in personal terms as a person, Jesus of Nazareth.

Our Retroviral Heritage

In the early 1980s, the dramatic story of the first human cancer-causing retrovirus was unfolding. Japanese researchers were investigating an aggressive leukaemia that afflicted adults in south-west Japan. They discovered the human T-cell leukaemia virus (HTLV-1).¹

When HTLV-1 infects cells, its genetic material is copied from RNA into DNA by a viral enzyme (a *reverse transcriptase*). The freshly made DNA is spliced into the chromosomal DNA of the infected (host) cell. The insertion event is initiated by another viral enzyme (an *endonuclease*), which recognizes a short sequence of bases in the DNA of the host cell, and cuts each DNA strand a few bases apart. This creates a gap into which the viral DNA is inserted. The resulting viral DNA insert is recognized by its characteristic set of genes, and flanking short *target site duplications* (TSDs) of host DNA (Figure 1).

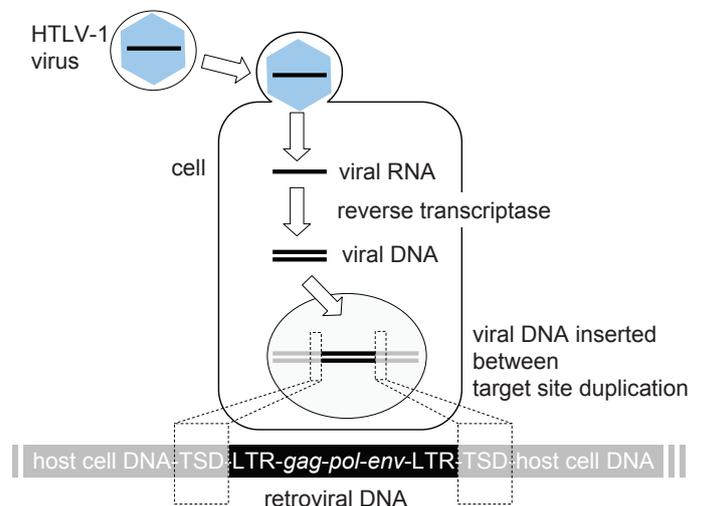


Figure 1. How an infectious retrovirus inserts its genome into that of a cell.

These insertion events occur randomly in the host cell genome. Early in infection, myriad distinguishable retroviral DNA inserts are found in a cell population. But in a leukaemia that develops years later, every leukaemic cell possesses the same inserted segment of viral DNA.²

Why should all the cells in a cancer have the same retroviral DNA insert? The impulse to develop into a cancer was initiated in *one* cell harbouring one particular HTLV-1 insert. This cell and its offspring were driven by the retrovirus (and by later genetic changes) into a programme of uncontrolled multiplication, producing an expanding clone of descendants, all of which *inherited* the original viral DNA insert from the founding cell. The billions of cancer cells possess the same inserted segment of viral DNA because they inherited it from the one progenitor cell.

At about this time, scientists discovered that *everyone* possesses inserted retroviral sequences (or *endogenous retroviruses*, ERVs) as a normal component of their DNA. These ERVs did not enter our DNA by the familiar process of infection. Rather, we inherited them from our parents. All humans have inherited the same 400,000 ERV segments, and they constitute 8% of our DNA.³ Our genome is a historical record of past retroviral insertions into the DNA of our forbears.⁴

For all humans to possess the *same set* of ERVs as part of their genetic endowment, the germ-line cells sustaining the original infections must have been ancestral to us all. When did the common ancestor(s) live? In the 1980's some reports suggested that humans and chimpanzees possessed ERVs at the same sites in their respective genomes.⁵ If humans and chimps did in fact possess the same ERV inserts, then both species must have inherited their collection of ERVs from the same ancestors.

The fact that humans and chimps - and indeed other primates - share a set of ERVs was confirmed by a study of six ERVs. Three of the ERVs are common to the African great apes. The other three are present in Old World Monkeys and apes including humans, but not New World Monkeys. Each ERV established that humans, chimps and other primates that possess it are descended from the one reproductive cell that sustained the insert (Figure 2).⁶

Confirmations accumulated in the literature (Figure 3).⁷ ERVs have been instrumental in structuring our genomes.⁸ Genome sequencing has shown that more than 99.9% of the ERVs in the human genome are shared with chimps. We share a long and unbroken pedigree with (other) primates.⁹

Self-Propagating Segments of DNA in our Genomes

Our genomes are populated by many other self-propagating units of genetic material (or *transposable elements*). They are recognized as defined segments of DNA that multiply by copying-and-pasting themselves.

Long interspersed elements (LINEs) are self-propagating segments of DNA of unknown origin. LINEs produce a protein that acts as a reverse transcriptase (to copy an RNA version of their genome into DNA) and an endonuclease (to initiate the insertion event) (Figure 4).¹⁰

Some families of LINE elements are very ancient, and particular inserts are shared widely with other mammals. Others are found only in primates. The youngest family is present only in humans.¹¹ It is currently generating new inserts in the human gene pool at a rate of one per twenty births.¹² LINEs are unpredictable insertional mutagens that cause disease,¹³ although some have been recruited to provide genetic function.

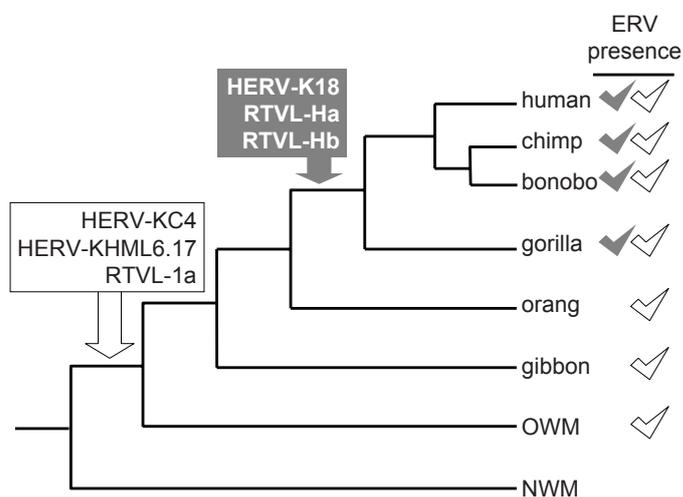


Figure 2. How endogenous retrovirus (ERVs) demonstrate that African great apes (humans, chimps and gorillas) derive from a common ancestor; and that Old World Monkeys (OWMs) and apes derive from a common ancestor not shared with New World Monkeys (NWMs).⁶ Ticks indicate the presence of particular ERVs; arrows indicate their inferred times of insertion.



Figure 3. The insertion site of an ERV. The uninterrupted target site and its duplications are in bold, and shaded.^{6,7}

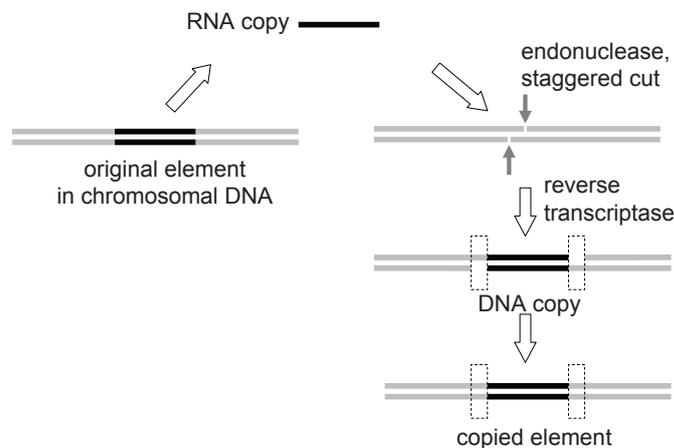


Figure 4. How transposable elements such as LINEs and SINEs multiply in genomes. The double grey lines represent chromosomal DNA; the short black lines represent transposable element RNA (single line) and DNA (double lines).

There are more than 900,000 segments of DNA recognisable as LINES in our genome (20% of our DNA). When the insertion sites occupied by some 500 human-specific LINE elements were investigated in non-human primates, in no case was an independent insertion found at the corresponding sites in those other species. The probability of two LINE elements independently inserting into the same site in two different species is thus negligible.¹⁴ If two species possess the same insert they have inherited it from the same progenitor. LINE elements are powerful markers for delineating lines of descent. At least 99% of the LINES we possess are *shared* with the chimps – a staggering demonstration of common ancestry.¹⁵

The LINE reverse transcriptases sometimes generate LINE copies that include fragments of unrelated DNA. These randomly and uniquely generated inserts are perfect markers of evolutionary relatedness. Over eighty of these patchwork LINE inserts are present in our genome. Many are shared by other species. They outline an unambiguous evolutionary history congruent with that derived from other genetic markers (Figures 2, 5).¹⁶

Short interspersed elements (SINEs) possess no genes. They use the enzymes made by LINES in order to multiply in host genomes. The most abundant SINEs in our genome are the primate-specific *Alu elements*, of which there are many subfamilies¹⁷ and 1.1 million inserts (11% of our DNA). The mechanism by which Alu elements copy-and-paste themselves is partially understood.¹⁸

Alu elements multiply through the genome largely by the activity of a few highly active 'master' elements.¹⁹ They are still copying-and-pasting themselves in peoples' genomes. New Alu elements arise in the human gene pool once in every 20 births.¹² Alu elements are insertional mutagens and may cause disease.¹³

When over 2500 insertions of human-specific Alu families were characterised, in no case was an independent insertion found in any of these sites in non-human species.²⁰ If multiple species possess a particular insert, those species are descended from the one individual in which that unique insert occurred. Nearly all (>99%) of the Alu elements in our DNA are shared with other primates.

One research group has identified nine Alu elements found only in humans and chimps, 45 shared by only humans, chimps and gorillas, and 38 shared by humans, chimps, gorillas and orangutans. Sixty-five Alu inserts are common to the great apes and the lesser apes (gibbons) (Figure 5). The apes are derived from the single ancestor that sustained the DNA insertion of each one of these Alu elements.²¹

Apes and monkeys are simian primates. More distant primate relations, the prosimians, include

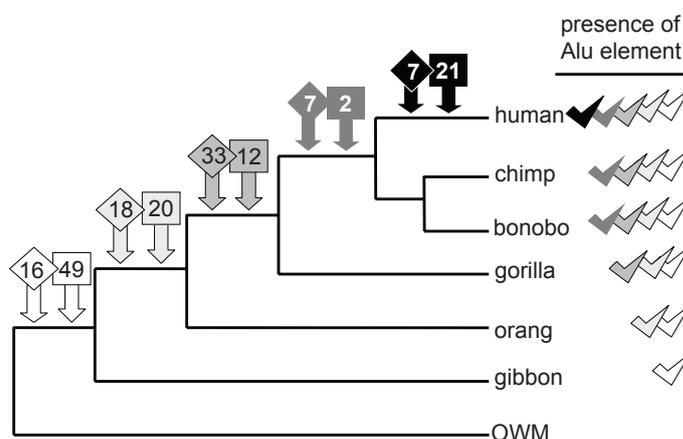


Figure 5. How a study of Alu elements has delineated the relationships of the apes. Diamonds and squares represent two different families of Alu elements; numerals within symbols represent the numbers of individual elements studied.²¹ Ticks indicate the presence of particular Alu inserts in different species. Arrows indicate the inferred time when they were inserted into primate DNA.

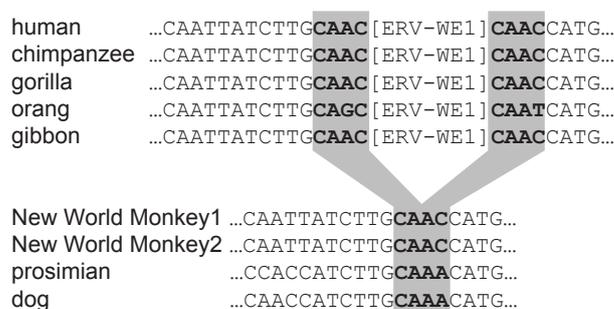


Figure 6. The insertion site of ERV-WE1.²⁴

the tarsiers and the galago-loris-lemur group. The relationships between these groups have been controversial for a long time. The presence of four ancient Alu inserts in simians and tarsiers but not in lorises or lemurs has established that simians and tarsiers are derived from a common lineage.²²

Genes from Junk

ERVs and parasitic genetic elements have provided us with essential genes. The *env* gene of infectious retroviruses enables the virus particles to stick to cells. The human genome possesses sixteen ERVs that retain *env* genes with the capacity to produce intact proteins. These *env* genes have remained intact far longer than would be expected if they were junk.²³

The ERV-WE1 insert (Figure 6) is common to Old World Monkeys and apes.²⁴ Its *env* protein is made by cells of the placenta, and causes the cells to fuse. This is necessary for the formation of the placenta – and for our development.

The insertion of an *Hsmar1* transposable element into the pre-existing SET gene has generated the novel *SETMAR* gene that is involved in repairing damaged DNA. The insertion event occurred in an ancestor of the simian primates.²⁵ Accumulated random and unique genetic events generate novelty and complexity.

The random activity of LINE enzymes generates potentially novel gene copies (*retrogenes*) from pre-existing genes. The *GLUD2* retrogene produces an enzyme called glutamate dehydrogenase in the brain. This gene was copied-and-pasted in an ape ancestor (Figure 7).²⁶ Over 100 such retrogenes have been discovered in the human genome.²⁷ Irreducible complexity arising *de novo* is a recurring theme.

Genes that Make Us Human

In summary,

- 50% of our DNA has been contributed by retroviruses and transposable elements;
- unique inserts shared by humans and other species establish common ancestry;
- ancestral primate genomes have been transformed into ours by the incremental accumulation of DNA changes, all of which reflect familiar mechanisms;
- some of our genes have been contributed by randomly self-propagating genetic units.

The publication of the human and other primate genomes has meant that 'scientists can address anthropology's fundamental question at a new level: what are the genetic changes that make us human?'²⁸ Many features characterise us as human,²⁹ but given that 'our cognitive abilities, more than anything else', have defined our distinctive evolutionary niche,³⁰ 'there is a general consensus that it is our brain and its unusual talent for complex thought that is the most significant.'³¹

The task of relating our genome to our biology is daunting. Human DNA differs from that of chimps by base substitutions (35 million), inserts and deletions (five million),³² and other rearrangements.³³ The contribution of any one of these is not obvious. Most changes will represent random drift; half will reflect chimp-specific alterations. The significant changes may be found in coding genes, regulatory sequences, or the vast expanse of intergenic DNA.³⁴ The genetic roots of humanness must be sought in an evolutionary framework that encompasses the order Primates.³⁵

Carroll wrote:³⁶ 'It seems unlikely that the traits that interest us most – bipedalism, skeletal morphology, craniofacial morphology, brain size and speech – were the products of the selection of just a few genes'. It may be the interacting indivisible entirety of our genome that specifies the indivisible entirety of our biology. Lahn has said: 'If you didn't tell me that humans were special biologically, I wouldn't have predicted it looking at the human genome.'³⁷

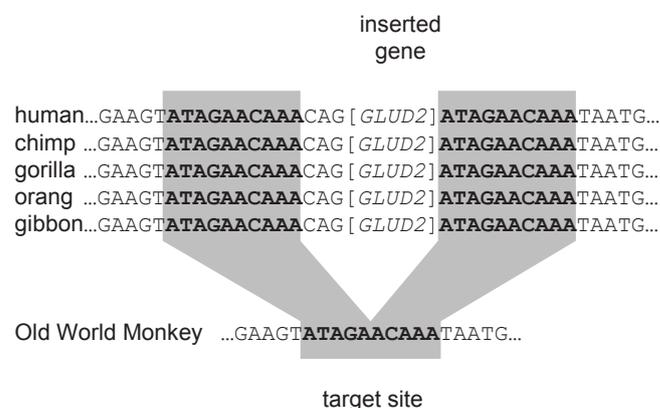


Figure 7. The insertion site of the *GLUD2* gene.²⁶

Genetics cannot describe fully what makes us human. A complete understanding of uniquely human traits will include more than DNA. We must address 'those long-debated traits of sophisticated language, culture and technology, in which nurture as well as nature plays a leading role. We're in the age of the genome, but we can still recognise that it takes much more than genes to make the human.'²⁸ Humanness involves our biology as well as cognition, behaviour, symbolic communication, and culture.²⁹ Darwin's friend and supporter, the Harvard botanist Asa Gray, said that man 'is as certainly and completely an animal as he is certainly something more.'³⁸ Another American biologist, Rick Colling, has said: 'We are fully biological but also much more.'³⁹

Genes for Religion

Wolpert has suggested that there are 'circuits in our brain set up by the genes that predispose us to have religious and mystical beliefs'.⁴⁰ If that were completely true, it would have no bearing on whether a transcendent being, the focus of such beliefs, exists independently of our minds. The spiritual faculty – an awareness of the transcendent – may have been of selective advantage because God is the source of the physical and moral order that makes our existence intelligible. A mental faculty that arises by natural selection need not for that reason be unconnected to an independent objective reality.

Augustine said no less than Wolpert. 'Man is one of your creatures Lord and his instinct is to praise you ... you made us for yourself and our hearts find no peace until they rest in you'. Colling argues that 'the universal desire for relationship appears to extend beyond mere human interaction'; we are 'wired for God'. Our minds desire relational connections to achieve both biological survival and relationship with God.⁴¹ Any discovery that an innate yearning for God it is genetically encoded cannot undermine the assertion that God ordained that it be there. The action of God is not an alternative to the molecular biological mechanisms disclosed by science.

However, Wolpert's thesis is problematic. He compares the tendency to 'religious belief' (considered to be genetically encoded) with science (that is 'not programmed in our brains').⁴² This provides the underlying structure to his book, and the basis of the exalted status given to science.

However, 'science' and 'religion' are not comparable concepts. The equivalent of 'science' is 'theology'. It is these that are scholarly activities that transcend genetics. Science seeks understanding of natural history; Christian theology seeks understanding of God's actions in human history. The basis of each discipline is the empirical world. The equivalent of 'religion' (causal beliefs regarding the spiritual world) is that set of causal beliefs that interprets the material world. The capacity to entertain all such beliefs may well reflect genetically encoded, specifically human neural circuits.

Genes provide the neural substrate upon which experience acts to mould our brains. With a faculty as basic even as that of sight, it is *experience* that turns potentiality into actuality. 'If babies born with cataracts fail to have them removed within their first year or so, it avails little to remove them, for the vision tuning program has shut down...'.⁴³ This reflects experience-dependent circuit refinement during an all-important critical period: 'the seemingly innocuous act of covering an eye can profoundly alter the physical structure of the brain'.⁴⁴ The brains of musicians, jugglers, taxi drivers, and university students develop anatomically in response to environmental inputs.⁴⁵

Genes and Experience

The physical structure and the function of the brain, genetics and all, is moulded early in life by the attention and love of those to provide care. Neural and behavioural abnormalities occur in young children who have suffered socio-emotional deprivation.⁴⁶

In the brain of a neonate, 'environmental cues mediated by the senses play a major role in determining how neurons will differentiate, sprout dendrites, form and maintain synaptic connections, and create the final neural networks that convey functionality. By adolescence, the majority of the changes that are taking place in the brain of that child are determined by experience, not genetics'.⁴⁷

Sensory neglect in childhood leads to abnormal brain development. 'In the development of socio-emotional functioning, early life nurturing appears to be critical. If this is absent from the first three years of life, and then a child is adopted and begins to receive attention, love and nurturing, these positive experiences may not be sufficient to overcome the malorganisation of the neural system mediating socio-emotional functioning'.⁴⁸

These 'experiments' of nature cannot control all variables (alcohol exposure *in utero*, nutrition). However, experiments in non-human primates have established that boring environments make for plain-looking brains, and enriched environments 'lead to dramatic increases in both neurogenesis and the density of neuronal dendrites.' Environments influence brain structure.⁴⁹ In the study of brain pathology, environmental enrichment exerts 'a range of dramatic effects'.⁵⁰

Thus the capacity to form relationships results from the experience-based expression of an underlying genetic potential. The somatosensory environment provided by a loving caregiver provides the sensory cues necessary to express the genetic potential of an infant to develop healthy relationships.⁵¹

An *a priori* commitment to describe humanity in exclusively physical (genetic) terms is problematic. Our brains, minds and humanity arise not from our genes only, but from non-quantifiable ('spiritual') qualities such as love, joy, peace, patience, kindness, goodness, faithfulness, gentleness, and self-control – all of which are invisible to science. That which is spiritual must be admitted as constitutive of our neural development as human persons.

The apparently tangible reality of human 'life' requires the apparently intangible conditions of relationship and words. This is indicated by Jesus at his temptation:

Man shall not live by bread alone, but needs every word that God speaks. (Matt 4:4; from Deut 8:3);

and by Peter when Jesus asked whether his disciples would leave him:

You have the words that give eternal life. (John 6:68)

Supremely, John uses the concept *logos*, meaningful to both Jewish and Greek readers, to refer to Christ, who as the 'Word' was God's personified self-expression to humanity. The Word was the source of Life (John 1:4; also 1 John 1:1). As God's Word he is life-giving. Spiritual life as life in relation to God comes from experiencing the grace of the One into whose society we are graciously included. The concreteness of life arises from the apparent abstractness of personal relationship.

Humanity and 'the Gracious Other'

Our humanity is not guaranteed by the possession of a human genome. The development of our humanity requires the non-material and vulnerable reality of inter-personal relationship. The medieval Emperor Frederick II of Hohenstaufen sought to understand the origins of language by demanding that some children be raised from birth in complete silence. His experiment was performed by 'bidding 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. But he laboured in vain, for the children could not live without clappings of the hands, and gestures, and gladness of countenance, and blandishments.⁵²

It seems that the children never learnt to speak any language. Their genes for humanity and for religion did not kick in. They died in infancy. We live and develop as human, not according to some deterministic genetic blueprint, but by being immersed in the nurture of a prior human community. 'The untouched newborn may literally die.'⁵³

Much may be learned from children who have spent their earliest years apart from human company, either because of severe neglect (Genie), or because they were separated from society and raised by animals (Amala and Kamala).⁵⁴ When introduced into society, such children do not learn to speak, or develop a sense of 'I' as a human being, or become socialised. The neglected child Genie, for example, could not grasp the meaning of pronouns. She used 'I', 'me', and 'you' interchangeably.⁵⁵ Language is learned only in the companionship of others. It may be necessary for human consciousness.⁵⁶

When it comes to being fully human, genetics counts for nothing if an infant is separated from humanity. Our humanity is the outcome of 'three kinds of adaptive history': the influence of biological evolution through the genes, the evolution of neural circuits through experience, and the influence of cultural evolution. It seems that 'culture and speech are essential for the making of the human mind'. Indeed, a social trigger at the right moment may be needed for certain genetic programmes to kick in.⁵⁷

Steeves has said that defining 'human' by means of distant hairy relatives or genetic tests is as unfulfilling as defining 'human' as a creature with a chin. During development, 'the burgeoning consciousness of the infant will not necessarily develop into human intentionality on its own but rather requires the presence of a Significant Other who is human. This "gracious act of attention" is thus responsible for "creating" a human-person ... Humans we know are not defined genetically or anthropologically ... Being human is being treated by humans as human.'⁵⁸ The Xhosa saying 'umuntu ngumuntu ngabantu' (a person becomes a person through persons) encapsulates this insight brilliantly.⁵⁹

Genes are necessary but insufficient to define our humanity. Our status as human beings arises from personal knowledge. Genetics give us the ability to receive the love of others which in fact confers upon us our full humanity. This is congruent with the words of Christ:

And eternal life means knowing you the only true God, and knowing Jesus Christ, whom you sent. (John 17:3; also 1 John 5:20)

In being known and loved we know and love. It seems that we are socialised into the community of God only by the communication of his grace. No extra material is added to our humanity, but just as we have human life through knowing, so we receive spiritual life in knowing the Word of God, spoken in Jesus Christ.

I believe that the words of Paul, written to the Christian community at Philippi, reveal the apogee of authentic humanity:

All I want is to know Christ and to experience the power of his resurrection, to share in his sufferings, and become like him in his death, in the hope that I myself will be raised from death to life. (Phlp 3:10-11).

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About the Author

Dr Graeme Finlay studied for a PhD in cellular immunology, and then joined the Auckland Cancer Society Research Centre, New Zealand, where he has been working for the past 20 years. This laboratory is involved in the development of novel anti-cancer agents, and is the largest group of this nature in the southern hemisphere. Research focus has centred on DNA-binding agents that poison the DNA-organising enzyme topoisomerase II. Areas investigated have included effects on the cell division cycle, mechanisms of cell death and the activity of the tumour suppressor gene encoding p53 in cell death pathways. Since 2000 Dr Finlay has also been Senior Lecturer in General Pathology in the Department of Molecular Medicine and Pathology, University of Auckland.



Two very different currents of thought directed him into the study of comparative evolutionary genetics. The first was the explosive growth in the understanding in cancer genetics that occurred since the early 1980s. The second was the wholesale importation of American creationist ideas into New Zealand. These developments intersected in fascinating ways. They generated a writing programme designed to identify some of the extraordinary developments in genetics described in the scientific literature, and present them in terms accessible to non-biologists. The resulting booklets were all published in 2004 by Telos Books (Auckland) with the titles:

- 'Evolving Creation' 46pp. ISBN 0-476-00650-3,
- 'God's Books: Genetics and Genesis' 75pp. ISBN 0-476-00651-1,
- 'A Seamless Web: Science and Faith' 59pp. ISBN 0-476-00816-6.

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