Critical Focus

Complexity in the Human Cardiovascular System

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Abstract

The failure of evolution to explain biological complexity is becoming increasingly obvious. Using the language of intelligent design and informed by systems biology, this paper describes the complexity of the human cardiovascular system. The sophisticated mechanisms needed to control pulsatile blood flow, the presence of stored information, and the planning necessary to realize the human cardiovascular system are examples of biological complexity. Other elements of design that are discussed are beneficence and attention to detail, both of which are necessary to achieve an unusual feature of *Homo sapiens*: a prolonged post-reproductive lifespan. Because the most common cause of death in *Homo sapiens* is myocardial infarction and stroke, a cardiovascular system capable of supporting life is necessary to allow a prolonged post-reproductive lifespan. Darwinism offers no convincing explanation for a prolonged post-reproductive lifespan. Specified complexity is discussed as a basis for recognizing intelligent design. Several shortcomings of evolution are discussed, such as the unlikelihood that humans evolved along with chimpanzees from a common ancestor. Finally, we discuss the irreducible complexity of the mammalian heart.

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INTRODUCTION

The mainstream theory of the origin of biological complexity—Darwinian evolution—holds that biological complexity developed over eons through the unguided accumulation of beneficial random mutations, with teleology playing no role. A common tenet of Darwinian evolution is that, given enough time, any organism, no matter how complex, can evolve because small, random mutations can give rise to any biological structure [1].

Scientists have never directly witnessed the evolution of a new species, though scientists did not observe the overwhelming majority of the history of life on Earth. A species is here defined as the largest group of organisms in which any two individuals of the appropriate sexes or mating types can produce fertile offspring. This lack of scientific observation makes the theory of evolution non-falsifiable. The philosopher of science Sir Karl Popper argued that non-falsifiable theories are merely beliefs, unworthy of a loftier designation.

What has been observed are numerous examples of phenotypic adaptations, such as the variation in beak size of "Darwin's finches" [2] and the mouthparts of the cichlids of the Rift Lakes of Africa [3], both of which demonstrate phenotypic plasticity within a genome. At least some of these finches and cichlid species can produce fertile hybrids. Further evidence of genomic plasticity is seen in *Canis familiaris*: Would a naive observer think a Great Dane and a chihuahua were the same species? *Canis familiaris* demonstrates that the phenotypic plasticity within a species can be surprisingly large and that a marked difference in appearance is an insufficient basis for designation as a new species.

Trillions of *Plasmodium falciparum* protozoa, human immunodeficiency viruses, and *Escherichia coli* bacteria have been observed over thousands of generations. Minor changes have been observed, many caused by mutations that cause loss of function of a gene. These loss-of-function mutations can

slightly improve competitiveness by eliminating unnecessary functions in vitro, but no new molecular machinery has evolved to provide the selective advantage [1]. Despite this, as Michael Behe notes, Darwinism has been quite impervious to falsification [1].

As Stephen Meyer wrote in the prologue to his classic *Darwin's Doubt* [4]:

> The technical literature in biology is now replete with world-class biologists routinely expressing doubts about various aspects of neo-Darwinian theory, and especially about its central tenet, namely the alleged creative power of the natural selection and mutation mechanism.
>
> Nevertheless, popular defenses of the theory continue, rarely if ever acknowledging the growing body of critical scientific opinion about the standing of evolution. Rarely has there been such a great disparity between the popular perception of a theory and its actual standing in the relevant peer-reviewed science literature.

This disparity was the impetus for a 2016 conference sponsored by The Royal Society—one of the world's most prestigious scientific organizations—to address developments in evolutionary biology and adjacent fields that questioned the standard theory of evolution. The opening presentation of that conference was given by the world-class biologist and Austrian evolutionary theorist Gerd Müller, who seemed to agree with Meyer's point about the creative power of random mutation. He noted several fundamental "explanatory deficits" of the textbook neo-Darwinian theory, including explaining phenotypic complexity and novelty.[1]

Intelligent design is a competing theory of the origin of complex biology. Its central claim is that only the input of intelligence can adequately explain the existence of complex, information-rich organisms [5]. Features such as control systems, information storage and feedback are offered as suggestive of design [6]. Of particular note, William Dembski has proposed that "specified complexity" is a feature of design in an organism [5]. Specified complexity is characterized by two properties: a small probability of occurring at random and a succinct description [5].

Teleology is recognized as a driving force in intelligent design. To adherents, teleology is obvious in features such as reproduction, growth, camouflage, the loss of hemoglobin in Antarctic icefish to reduce blood viscosity in near-freezing water [7] and flagella.

In an essay provocatively titled "Evolutionary Biologist Concedes Intelligent Design Is the Cutting Edge," well-known evolutionary biologist Bret Weinstein recently described the relationship of the competing fields by saying: [2]

> If you decide… that your challengers aren't entitled to a hearing because they're motivated by the wrong stuff, then you do two things. One, you artificially stunt the growth of your field, and you create a more vibrant realm where your competitors have a better field to play in because you've left a lot of holes in the theory ready to be identified, which I think is what's going on. The better intelligent design folks are finding real questions raised by Darwinism, and the Darwinists, instead of answering those questions, [are] deciding it's not worthy of their time. And that it is putting us on a collision course.

This paper describes the complexity of the human cardiovascular system and argues that this system is most easily explained as the result of intelligent design. It will focus on qualities seen in successful design, such as robust control, information storage, planning, beneficence, and attention to detail. Of these features, beneficence in particular is difficult to explain by evolution. This paper surveys evidence of design in the control of pulsatile flow, the determination of the necessary quantity of cardiac power and the requirements for a prolonged post-reproductive lifespan. Finally, we argue that many elements in the human cardiovascular system are specified, particularly its set points and materials.

DISCUSSION

Robust control of blood flow

Quantities such as peak blood velocity, peak acceleration, and systemic vascular resistance change with cardiac output, which itself varies in physiologic or pathologic states. The pulsatility of blood flow dictates that blood velocity and acceleration change constantly. Consequently, monitoring and controlling blood flow are more complex than they are in systems with continuous flow. These tasks are made even more difficult because blood is a non-Newtonian fluid, meaning that its viscosity varies with its shear rate. Information about three aspects of blood flow are sensed: systemic vascular resistance [8], wall shear stress [9], and erythrocyte deformation [10]. All three quantities are strictly correlated with blood viscosity.

The systemic vascular resistance response (SVRR) was described in 2015 [8]. Systemic vascular resistance is detected by myocardial mechanoreceptors in the left ventricle. This information modulates the release of B-type natriuretic factor and downregulates the expression of erythropoietin, the hormone that drives the production of erythrocytes. The hematocrit, which is the percentage of the volume of whole blood composed of erythrocytes, is the strongest intrinsic determinant of blood viscosity.

The detection of wall shear rate by endothelial shear receptors in the carotid sinus, a dilatation of the proximal internal carotid artery, was recently described [9,11]. The shape of the carotid sinus is tailored to create an area of flow recirculation, which increases the amplitude of wall shear stress. This creates "gain" in detecting wall shear stress. This information is carried to the central nervous system via the glossopharyngeal nerve

[1] https://evolutionnews.org/2017/01/1_happy_new_yea/

[2] https://evolutionnews.org/2024/06/evolutionary-biologist-concedes-intelligent-design-is-the-cutting-edge/

(CN IX). After processing via multiple interneurons, output from the brainstem modulates the activity of the renin-angiotensin-aldosterone system (RAAS).

The RAAS is the physiologic antagonist of the SVRR [8,12]. The SVRR promotes natriuresis while the RAAS promotes sodium retention. Historically, the effect of the RAAS on erythropoietin has been less widely recognized. Angiotensin II is a secretagogue of erythropoietin and a growth factor for erythroid progenitors. The most dramatic example of the RAAS on erythropoiesis is posttransplant erythrocytosis.

Thus, erythropoietin production, red blood cell mass, hematocrit, and blood viscosity are not determined by tissue oxygen demand in the absence of chronic hypoxia. Instead, they are modulated to maintain wall shear stress in the carotid sinus [11]. There is no set point for hematocrit per se.

Findings in the dog species *Canis familiaris* supports the idea that hematocrit has no set point. With the exception of the greyhound, the plasma concentrations of molecules of clinical interest are similar in different breeds, making general canine reference ranges possible.[3] However, blood viscosity and hematocrit differ significantly among dog breeds. The mean blood viscosity of the golden retriever at a high shear rate (125/sec) in one study was 3.93 centipoise, the greyhound's was 6.00 centipoise, and the Irish wolfhound's was 6.21 centipoise. The mean hematocrit of the golden retriever in that study was 40.5%, and the greyhound and Irish wolfhound's were each 58.8% [13]. These results are consistent with the theory that blood viscosity and hematocrit are modulated to control vascular wall shear stress and are not a genetically determined set point. Wall shear stress is affected by numerous anatomic and hematologic factors (vide infra).

Recently, the authors described how blood viscosity is tailored to local hemorheological (blood flow) conditions via an array of both long- and short-acting hormones, including epinephrine, prostaglandin E2 and nitric oxide [14]. Epinephrine decreases erythrocyte deformability, thereby increasing blood viscosity and reducing the chances of developing turbulence, at the same time that it increases cardiac output, blood velocity and peak acceleration. Prostaglandin E2 (PGE2) decreases erythrocyte deformability, thereby increasing blood viscosity in the left ventricular outflow tract and aortic arch, regions where blood velocity is high and the direction of blood flow changes almost constantly (Figure 1). PGE2 is produced by alveolar type 2 pneumocytes, and its activity is limited by its short half-life (making it a short-acting hormone).

Nitric oxide likewise modulates blood viscosity and can be produced both by the endothelium and by the autocrine system. In arterioles with slower blood flow, endothelial production of nitric oxide increases erythrocyte deformability and lowers blood viscosity. Erythrocytes sense their own deformation and modulate their stiffness in response to autocrine production of nitric oxide [10]. This appears to be a key factor in allowing erythrocytes to traverse capillaries that have a diameter smaller than their own. This response may play a role in the

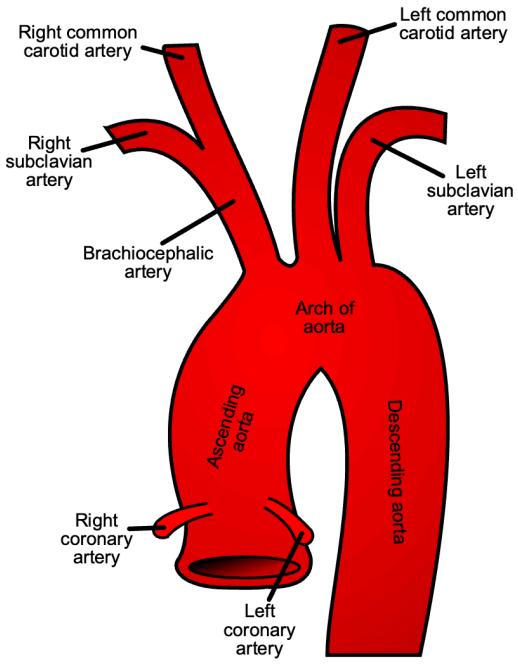

Figure 1. Aortic arch. High velocity blood leaves the left ventricle and enters the ascending aorta, where the direction of flow constantly changes. Public domain. **doi:**10.5048/BIO-C.2025.1.f1

Fåhræus-Lindqvist effect, i.e., the progressive decrease in blood viscosity as tube diameter decreases from slightly greater than 0.3 mm to 0.04 mm [12].

Because blood viscosity is controlled so closely, we argue that blood viscosity should be considered part of what the eminent physiologist Claude Bernard called the "milieu intérieur." All the sensors involved in the control of blood flow must be calibrated to recognize when the cardiovascular system is at homeostasis. If the set point of one sensor is incorrect, its output will constantly be at odds with the others and homeostasis will never occur. Coordinating the output of three separate sensors poses a formidable stochastic barrier.

Information Storage

A second characteristic of designed systems is information storage [8]. Set points are examples of stored information, and the human cardiovascular system has many of these. The set point for wall shear stress in the common carotid artery of humans is 11–14 dyn/cm^2 [15], and the set point for systemic vascular resistance is 800–1200 dynes-sec/cm^{-5} [16]. Normal ranges are also examples of set points. Reference clinical laboratories measure the concentration of hundreds of different molecules. The concentration of each must be within the normal range to maintain homeostasis.

Control of plasma concentrations of some of these different molecules is complex. For example, glucose homeostasis requires the coordinated activity of the liver, pancreas, skeletal muscle (which takes up most circulating glucose following a meal), multiple hormones and normal blood viscosity. High blood viscosity reduces perfusion of skeletal muscle reducing

[3] https://www.idexx.com/files/diagnostic-update-greyhound-specific-reference-intervals.pdf

glucose uptake by skeletal muscle and prolonged elevated glucose concentrations in blood. This leads to glucose intolerance and ultimately diabetes mellitus type 2 and it many pathologic changes, such as microvascular disease, premature loss of aortic compliance, and accelerated atherosclerosis [17]. Furthermore, the activity of most metabolic and synthetic biochemical pathways is controlled. Establishing appropriate set points that involve these pathways would require information about the necessary concentrations of their end products and the availability of precursors.

The resting membrane potential of a cell (V_{rest}) is another example of a set point. It is determined by the intrinsic selective permeability of the cell membrane to ions and charged molecules, embedded charged molecules in the plasma membrane (both in the inner and outer leaflets of the lipid bilayer) and the osmotic equilibrium of plasma. A wide variety of proteins transport and pump ions to generate concentration gradients that result in voltage differences, while ion channels allow ions to move across the membrane down those gradients. Thus, V_{rest} is another quantity that depends on the coordinated activity of several different processes.

An additional layer of complexity in the cardiovascular system is establishing and maintaining the V_{rest} of each cell type in the human cardiovascular system, which requires its own V_{rest}.

The V_{rest} of erythrocytes is approximately –10mV [18]. The V_{rest} of smooth muscle cells is –55mV, the V_{rest} of neurons is –65mV, and the V_{rest} of cardiac muscle cells is –80mV. In contrast, the V_{rest} of skeletal muscle cells is –90mV.4

The plasma concentration of clinically significant molecules differs in rabbits, hamsters and other small laboratory animals [19], while the normal laboratory reference ranges for these same molecules are similar in chimpanzees and humans [20]. Where did the information to determine these set points originate? Was every possible set point field-tested as life ostensibly evolved from the earliest cells, leading to different plasma concentrations of clinically significant molecules in rabbits and hamsters and different membrane potentials in cardiac muscle and skeletal muscle cells?

Comparing Human and Chimpanzee Hearts

Adherents of Darwinian evolution can easily argue that the information necessary to determine the cardiovascular set points in *Homo sapiens* was largely inherited from a closely related ancestor. This argument is attractive to Darwinists because the stochastic barrier to actualizing *Homo sapiens* by evolution is lower if it requires only microevolution from a pre-existing animal. For this reason, it is instructive to compare the cardiovascular system of *Homo sapiens* with that of the chimpanzee, with which humans share a "last common ancestor" according to adherents of Darwinian evolution.

The human left ventricle is longer, less spherical, less trabeculated, relaxes more quickly in early diastole and exhibits greater twisting and untwisting with contraction than the heart of the chimpanzee. These changes result in greater stroke volume and cardiac output. The human heart is thought to be specialized for endurance physical activity such as hunting and gathering or farming, while the chimpanzee's is specialized for resistance activity such as climbing and fighting [21].

Detailed knowledge at the "nuts-and-bolts molecular level"—to use Behe's phrase [1]—of the morphogenesis of the hearts of the human, chimpanzee, and the putative last common ancestor is necessary to know if actualization of the human heart by evolution is feasible. Presently, the existence of a last common ancestor between humans and chimpanzees is speculation. It has been argued that common descent from an ape-like ancestor is unlikely because it would require multiple mutations to occur simultaneously. For example, an enlarged brain would require changes to the skull, mandible and teeth [22]. A similar argument has been offered against the possibility that icefish could have evolved because the elimination of hemoglobin required the adoption of an entirely new high-output, low-pressure, low-resistance cardiovascular system, antifreeze glycoprotein and aglomerular kidneys [7].

Planning

Planning is the process of specifying the quantities and composition of the components of a system needed to make it successful. Planning requires foreknowledge. In the cardiovascular system, there must be quantitative knowledge of the following in order to specify the amount of cardiac power needed to provide adequate cardiac output for the design to work: tissue metabolic demands, blood pressure, heart rate, stroke volume, the energy lost to friction caused by 10 to 60 trillion endothelial cells [23], resistance due to blood viscosity, vascular tone and the drag caused by every vascular branch and curve. These values combine to determine wall shear stress and peripheral vascular resistance.

The fact that blood viscosity is an emergent phenomenon makes specifying cardiac power even more difficult. An emergent phenomenon is one that becomes fully manifest only when the components of a system interact. The viscosity of blood is the cumulative result of the hematocrit, shear rate, erythrocyte deformability, erythrocyte surface charge, osmolarity, plasma ionic strength and the size, shape, charge, and concentration of every plasma protein. The energy lost to friction also varies with blood viscosity. As noted above, blood viscosity varies in different parts of the circulation. The main explanation for this variation is that blood is a non-Newtonian fluid, which means that viscosity changes when blood flow (shear rate) varies. The viscosity of Newtonian fluids is constant at different shear rates (Figure 2).

Further, it must be determined if the energy needed to generate that power is available when constrained by the metabolic rate of the organism and food supply. For example, in icefish, an estimated 22% of their basic metabolic rate is devoted to cardiac work, compared to 0.5% to 5.0% in temperate-zone fish [7]. Both designs are successful.

Beneficence

Beneficence in design can be defined as including a feature that is a boon to an individual but, in Darwinist terms, does

[4] https://www.kenhub.com/en/library/anatomy/membrane-potential.

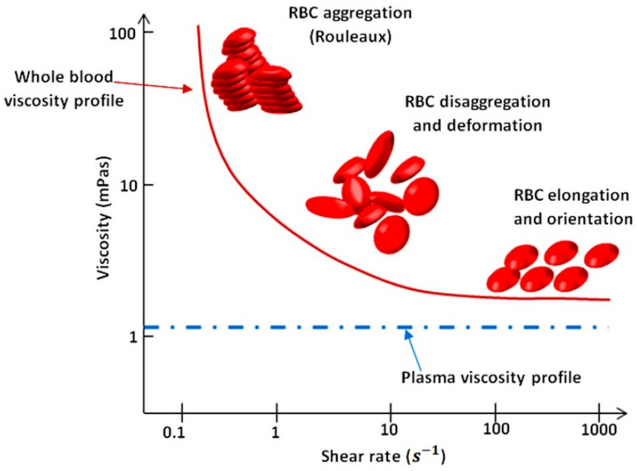

Figure 2. The non-Newtonian viscous behavior of blood compared to plasma. Due to the presence of proteins plasma demonstrates very mild non-Newtonian characteristics. The combination of cells and aggregating proteins such as fibrinogen and low-density lipoprotein makes blood a non-Newtonian fluid. Its viscosity increases almost exponentially as the shear rate decreases. This increased viscosity increases the risk of thrombosis. Reproduced from ref [24] under open access license CC BY 4.0. **doi:**10.5048/BIO-C.2025.1.f2

not confer a competitive advantage to a species. It is a design choice. An example is a prolonged post-reproductive lifespan (PPRL). This subject was recently reviewed by Croft et al. who wrote [25]:

> Why females of some species cease ovulation before the end of their natural lifespan is a longstanding evolutionary puzzle. Any decline in reproductive function is detrimental to Darwinian fitness and thus classical evolutionary theory predicts the early termination of reproduction should be selected against. Reproductive decline long before the end of life in humans has generated tremendous medical and evolutionary interest.

Those authors concluded that evidence showing that a PPRL has adaptive value, such as by increasing the survival of kin, is currently unconvincing, although some Darwinists disagree.

The most common cause of death worldwide according to the World Health Organization is ischemic heart disease. The second most common is cerebrovascular disease. Delaying these outcomes, thereby allowing a PPRL, requires optimization of the cardiovascular system.

According to the Arterial Compliance Theory of Aging, one factor that can limit lifespan is loss of aortic compliance.[5] Normal aortic compliance allows a portion of each stroke volume to be stored in systole and then propelled in diastole. This is called the "Windkessel" effect and serves to dampen peak arterial blood pressure and velocity and increase diastolic flow. Diastolic flow is important for perfusing the coronary arteries and myocardium because the resistance caused by the contracting cardiomyocytes restricts coronary artery flow during systole.

Most mammals have approximately one billion or fewer heartbeats in a lifetime. *Homo sapiens* is an exception. A human with a heart rate of 60 beats per minute will have around 2.5 billion heartbeats in 80 years. An extraordinary number of heartbeats is probably necessary for a PPRL. Repetitive deformation causes mechanical fatigue and fracture of the elastic elements in the aorta, primarily the protein elastin. It is replaced by collagen, which is stiffer. This eventually leads to aortic stiffening, which increases vascular impedance, increases pulse pressure (the difference between systolic and diastolic blood pressure) [26], and increases turbulence in the proximal aorta [27], a consequence of higher peak systolic blood velocity. A recent abstract reported that minimal and delayed age-related increases of arterial stiffness might contribute to the very low observed levels of coronary atherosclerosis and dementia in the Tsimane [28], a population that has been called the "healthiest in the world".[6]

Loss of aortic compliance contributes to hypertension, which then accelerates the loss of compliance, resulting in a vicious cycle [29]. Increased blood pressure increases peak blood velocity and Reynolds number in the carotid arterial system. This can result in loss of laminar blood flow and flow separation, where abnormally high-velocity arterial flow is separated from abnormally slow arterial flow [30]. Endothelial nitric oxide production is decreased and blood viscosity is increased in these areas of slow flow making them prone to thrombosis. Similar changes occur in the coronary arteries [31]. According to the thrombogenic theory of atherosclerosis, organization of these thrombi forms an atherosclerotic plaque [32]. Thus, it is easy to see how loss of aortic elasticity leads to ischemic heart disease and cerebrovascular disease.

Older literature suggested that the elastin production in man, like other animals, stops when growth ceases. [33] More recent data shows that tropoelastin, the precursor to elastin, is synthesized throughout life in humans, raising the possibility that the ability to synthesize elastin extends throughout life in *Homo sapiens*, albeit at decreased levels. Tropoelastin expression decreases by 50% each decade [26]. Prolonged production of elastin could be a key attribute in allowing a PPRL.

The normal architecture of the aorta is necessary for normal compliance

Aortic stiffening results in premature death in Hutchinson-Gilford progeria. Progeria patients typically die in the second decade of life of atherothrombosis despite having blood lipid levels comparable to controls. Accelerated aortic stiffening is a constant finding in this condition. This results in increased peak blood velocity, loss of laminar blood flow, and accelerated atherothrombosis.

The normal aortic tunica media consists of lamellae of smooth muscles separated by lamellae of extracellular matrix. For poorly

[5] https://medium.com/@bigdaddypathologist/the-vascular-elasticity-theory-of-aging-f70d25fb2652

[6] https://www.medscape.com/viewarticle/worlds-healthiest-arteries-found-be-most-elastic-2023a1000wup?ecd=wnl_dne1_240101_MSCPEDIT_etid6211574&uac=137319ET&impID=6211574

understood reasons, the molecular defect that causes progeria interferes with the deposition of the normal aortic extracellular matrix, of which elastin is an important component. Autopsy studies of patients with progeria have demonstrated severe loss of smooth muscle cells in the aortic media with replacement by collagen. In one study, progeria patients with an average age of seven had aortic stiffness expected in those aged 60 to 69 [34]. Elastin is also an important component of the dermis, explaining the aged appearance of patients with progeria.

A PPRL requires another aspect of intelligent design: attention to detail. There are at least two possible configurations of the coronary arteries that are capable of supporting life through the reproductive years. A coronary artery originating from the opposite coronary sinus is considered to be an anomaly because of its rarity [35]. This anomaly imparts an increased risk of a major adverse coronary event which becomes marked after age 40. This would not have adversely affected the reproductive fitness of paleolithic humans, who had an estimated life expectancy of approximately 33 years [36]. Thus, this attribute is unlikely to have been selected against. An alternative explanation for why a coronary artery originates from the nearest coronary sinus is that this configuration is necessary for a PPRL. According to the theory of intelligent design, it was specified in the design of *Homo sapiens*. This is an example of attention to detail.

Specified complexity

The word "specify" is used in this paper in the same way as it is used in architecture. Architectural specification writers specify the type of materials that should be used, how they should be installed and the quality standards to which these materials must adhere. While a drawing might show where a door goes, the specifications will detail the type of wood, the finish, the hardware and even the method of installation.[7] Specifying the type of materials and method of installation is vital for normal aortic function. Obviously, specification writing in architecture requires the input of intelligence.

Similarly, William Dembski argues that specification implies design of an object [5]. A pattern of specification allows one to reject the possibility that chance resulted in the appearance of the object. He quoted the mathematician Abraham de Moivre who wrote in 1718:

> The same Arguments which explode the Notion of Luck, may, on the other side, be useful in some Cases to establish a due comparison between Chance and Design: We may imagine Chance and Design to be, as it were, in Competition with each other, for the production of some sorts of Events, and may calculate what Probability there is, that those Events should be rather owing to one than to the other.

Dembski argues that parity of reasoning demands that if large probabilities vindicate chance and defeat design, then small probabilities should vindicate design and defeat chance.

This aptly describes biological set points. For example, the possible values for blood glucose in humans range from 0 to 2,656 mg/dL.[8] Only a small minority of these values are compatible with homeostasis. The probability that a possible value is compatible with homeostasis is small. The description of this set is simple: the "normal range." The glucose concentration in humans meets Dembski's criteria for specified complexity.

Specified complexity is seen in the human cardiovascular system in the set points for normal wall shear stress and systemic vascular resistance, the different transmembrane electrical potentials in the various cell types in the cardiovascular system, the quantity of cardiac power, the morphologic uniqueness of the human heart compared to the heart of its closest living relative, the chimpanzee, the arrangement and components of the aortic media and the origin of the coronary arteries. All cells have hundreds of synthetic pathways that are controlled by feedback or some other mechanism, which is determined by a set point. The human genome encodes 1,391 DNA-binding transcription factors that regulate the expression of more than 20,000 human genes [37]. To adherents of intelligent design, the origin of set points and the other elements of specified complexity are only compatible with design.

Set points may not even give a complete picture of the control of biological pathways. According to the National Human Genome Research Institute:[9]

> Researchers are learning that biological pathways are far more complicated than once thought. Most pathways do not start at point A and end at point B. In fact, many pathways have no real boundaries, and pathways often work together to accomplish tasks. When multiple biological pathways interact with each other, they form a biological network (Figure 3).

Irreducible Complexity

Irreducible complexity is one of the foundational concepts supporting the theory of intelligent design. According to Michael Behe, an object is irreducibly complex if all components are needed for the object to function but individually add no selective advantage and would not be maintained during evolution.

The mammalian heart can be considered to consist of three individual components: the electrical conduction system, the four cardiac valves and the myocardium. The conduction system is useless without a myocardium to stimulate. Without valves, heart failure would occur immediately. Without the tricuspid valve, contraction of the right ventricle would lead to retrograde flow of blood through the right atrium and both vena cavae. This would lead to a drastic decrease in cardiac output and pulmonary blood flow, incompatible with prolonged survival. Obviously, a myocardium without a conducting system or heart valves would not produce cardiac output or blood

[7] https://www.archisoup.com/architecture-specifications

[8] https://www.guinnessworldrecords.com/news/2023/5/miraculous-survival-of-boy-with-blood-sugar-level-21-times-higher-than-normal-746164

[9] https://www.genome.gov/about-genomics/fact-sheets/Biological-Pathways-Fact-Sheet

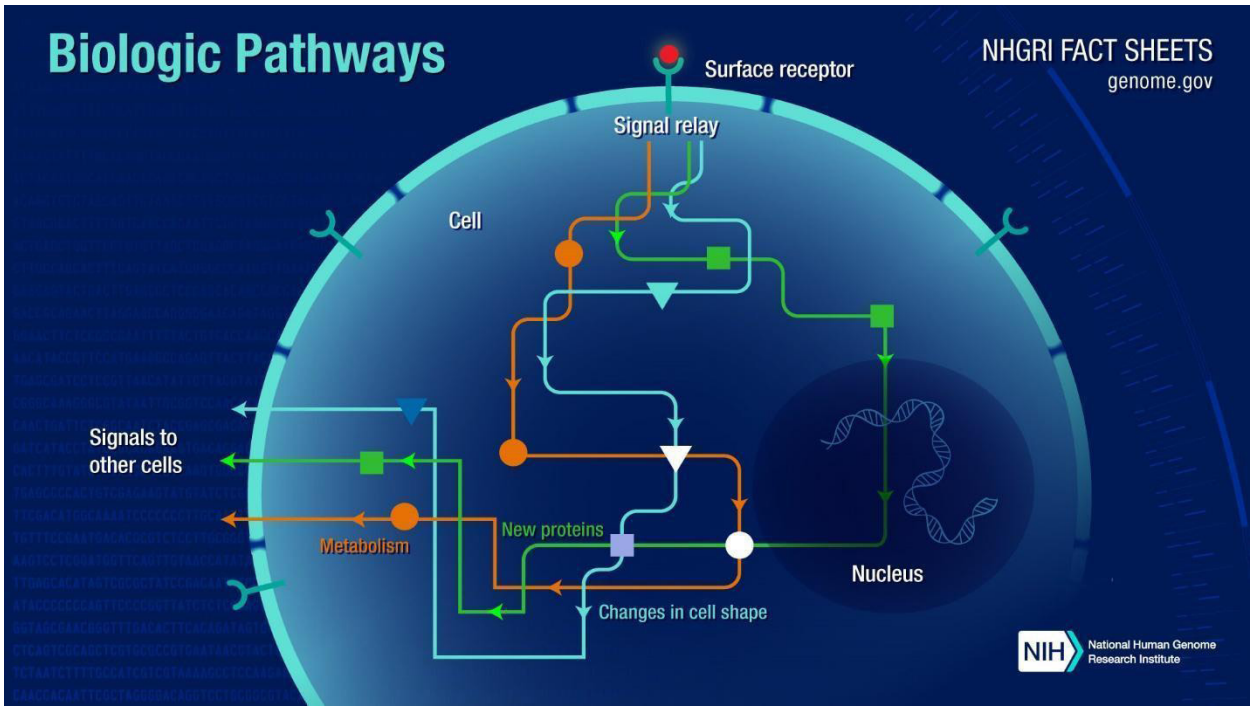

Figure 3. The complexity of biologic pathways. A schematic figure hinting at the complexity of biologic pathways. Courtesy of the National Institute of Health National Human Genome Institute. Courtesy of the National Institute of Health National Human Genome Institute (https://www.genome.gov/about-genomics/fact-sheets/Biological-Pathways-Fact-Sheet). **doi:**10.5048/BIO-C.2025.1.f3

pressure sufficient to maintain life. Thus, the heart can be considered irreducibly complex.

CONCLUSION

A critical examination of Darwinism reveals growing skepticism of the theory, and the search for an alternative theory is being actively pursued.

The more levels of complexity that are identified in an organism, the less likely it is that that organism was the product of blind luck. The human cardiovascular system is irreducibly complex, requiring sophisticated sensing mechanisms to control pulsatile blood flow and stored information about the different resting voltage potentials of the different cell types in the cardiovascular system and the normal ranges of biologically important molecules. A successful, unique animal requires extensive planning so that it has adequate cardiac power when constrained by the available sources of nutrition. Moreover, the human cardiovascular system meets the cardiovascular requirements for the prolonged post-reproductive lifespan of *Homo sapiens,* for which natural selection has no selective power. The idea of specified complexity supports the contention that humans were designed. Finally, the irreducible complexity of the mammalian heart argues for the intelligent design of *Homo sapiens* and against evolution.

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