EDITORIAL

PALAETIOLOGICAL SCIENCE AND CULTURAL POWER

"Palaetiological science" is a term coined in 1837 by William Whewell,[1] to define "those researches in which the object is, to ascend from the present state of things to a more ancient condition, from which the present is derived by intelligible causes." In other words, palaetiological sciences are those which use the evidence of the present in reconstructing the past. Whewell's term has been used infrequently, but was recently promoted by an internet group calling themselves the "Darwin-L discussion group."[2]

The palaetiological sciences are more generally referred to as the "historical sciences." Two examples Whewell used were philology (the study of the history of languages) and geology. Other examples include cosmology, paleontology, paleoanthropology, evolutionary biology, systematics, and historical biogeography. Such "historical sciences" contrast with such "experimental sciences" as physics and chemistry, which do not generally attempt to reconstruct the past. (Historical science might be too broad a term — some of the "historical sciences" may have subspecialties that are more experimental than historical in nature.)

One might wonder why it is so important to make such a distinction, separating palaetiological (historical) sciences into a separate category. One reason is that there seems to be a suspicion that the historical sciences are more subjective, and thus less dependable, than the more prestigious experimental sciences such as physics and chemistry. Arthur Shapiro, a population biologist, once wrote:

> *Popperism* [falsification] *is also widely invoked in certain schools of systematics and biogeography. Why in those places? Because all of those fields have reputations as soft, fuzzy, and ill-defined*.[3]

Evolutionary biology has been severely criticized as unscientific, because it has no recognized universal laws or deductive theory.[4] Paleontology has also been criticized in this respect. Nobel prize-winning physicist Luis Alvarez once said, in an interview with the *New York Times*:

> *I don't like to say bad things about paleontologists, but they're really not very good scientists. They're more like stamp collectors.*[5]

This statement raised a storm of indignation among paleontologists, but the response by Stephen Jay Gould acknowledges the problem by its

title: "A plea for the high status of natural history."[6] One does not plead for what one already has. Since Alvarez was a physicist rather than a paleontologist, and notorious for his ill temperament, his statement could plausibly be dismissed as a misunderstanding.

A similar explanation does not apply to recent statements by Henry Gee — a science writer for *Nature*, a vertebrate paleontologist who has worked at the British Museum (Natural History), and a practitioner of the cladistic method of systematics. He can hardly be called an outsider to paleontology. According to Gee:

> *To see palaeontology as in any way 'historical' is a mistake in that it assumes that untestable stories have scientific value. But we already know that Deep Time does not support statements based on connected narrative, so to claim that palaeontology can be seen as an historical science is meaningless: if the dictates of Deep Time are followed, no science can ever be historical.*[7]

Again, from Gee:

> *For example, the evolution of Man is said to have been driven by improvements in posture, brain size, and the coordination between hand and eye, which led to technological achievements such as fire, the manufacture of tools, and the use of language. But such scenarios are subjective. They can never be tested by experiment, and so they are unscientific. They rely for their currency not on scientific test, but on assertion and the authority of their presentation.*[8]

Not surprisingly, such statements do not go down well with historical scientists. The journal *Geology* published a paper[9] that responded to Gee's statements by claiming that "historical science is not inferior to experimental science when it comes to testing hypotheses." However, the persuasiveness of this claim was significantly weakened by further statements in the paper that scientists do not really practice Popperian falsification anyway. Hypotheses that seem to fail testing are frequently salvaged by sacrificing an auxiliary assumption:

> *Moreover this difficulty cannot be circumvented by varying the conditions under which a hypothesis is tested, given that the number of auxiliary conditions involved in any real-world situation is unknown and potentially infinite; it is impossible to control for them all.*[10]

If this is true, it would seem that the underlying hypothesis itself has not been tested; only the auxiliary assumption has been tested. If this is

true of experimentally testable hypotheses, it is even more applicable to hypotheses about unrepeatable historical events. Numerous scholars have noted that predictive theory is not a part of (evolutionary) biology.[11]

Hence it seems that hypotheses in historical science may not necessarily be testable. Yet those who practice historical science often ask for equal recognition with the experimental sciences. This might suggest a question: Why do the practitioners of historical sciences seem to want so badly for their activities to be recognized as "science?" Why not just use the term "natural history" or something similar? Is there something magic about the use of the term "science?" The following quotation may point toward the answer:

> *Scientists engaging in turf battles for legitimation, authority, domination, money, power, students, laboratory space, or glory, often invoke the legitimacy reason in arguments to secure places in a 'pecking order' for different sciences or to reject hierarchies of authority and social status altogether.*[12]

It seems the motivation for seeking "the high status of natural history" may have as much to do with sociological factors than with discovering how nature operates. There is power in the telling of history. As has been pointed out,[13] those individuals with the authority to tell the creation story for their society function as the priests of that society, and derive from that position a great deal of power over how members of the society view themselves and their world. In our society, the authority to describe reality has been largely given over to the scientists, and it is within the "palaetiological sciences" that the credibility of various origins stories is discussed. Hence, the apparent desire by historical scientists to occupy the status of scientist-priest.

The conflict between creation and evolution, it seems, is not strictly a scientific debate. It may not even be primarily about science at all. After all, the conflict is not about experimental data, but about historical explanations. The point of contention is the authority to tell the creation story in our culture, and, thereby, to influence the direction of that culture:

> *What is at stake, therefore, in the interpretation of Genesis cannot be merely the historicity of ancient narratives, or the doctrine of biblical inspiration, or even the systems of theology based on an inspired historical record of Creation, Fall, and Deluge. From a critical perspective it can be argued that the ultimate issue is nothing less than the social order, its character and sanctions, as dependent on human nature, created and corrupt*.[14]

As various groups compete for acceptance of their ideas of Earth history, it would be well to ask whether the issues might have more to do with personal philosophy than what we ordinarily consider to be "science."

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ENDNOTES

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3. Shapiro AM. 1993. Did Michael Ruse give away the store? NCSE Reports 13(1):20-21.
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6. Gould (see Note 5).
7. Gee H. 1999. In search of deep time: beyond the fossil record to a new history of life. Ithaca NY: Comstock Publishing, p 8.
8. Ibid., p 5.
9. Cleland CE. 2001. Historical science, experimental science, and the scientific method. Geology 29:987-990.
10. Ibid.
11. Murray (see Note 4).
12. Griesemer JR. 2002. Some concepts of historical science. http://philo.ucdavis.edu/zope/home/jrgriese/phil108/assets/Griesemer/1996a.pdf
13. Phillip Johnson, in lecture presented at Loma Linda University, 3 February 2001.
14. Moore JR. 1986. Geologists and intepreters of Genesis in the nineteenth century. In: Lindberg DC, Numbers RL, editors. God and Nature: Historical Essays on the Encounter Between Christianity and Science. Berkeley CA: University of California Press, p 322-350 (quote on p 327).

ARTICLES

RUSHING TO JUDGMENT: FUNCTIONALITY IN NONCODING OR "JUNK" DNA

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WHAT THIS ARTICLE IS ABOUT

The 1960s discovery that much nuclear DNA in eukaryotic cells does not code for proteins was quickly interpreted as evidence for the evolution of eukaryotic genomes. Papers were published suggesting a nomenclature reflecting evolutionary assumptions about this "junk DNA." Noncoding DNA was also used as evidence for the selfish gene theory popularized by Richard Dawkins and others. As many important functions played by noncoding DNA have come to light, the assumption can no longer be made that it represents DNA potsherds of evolution. Now the assumption of functionality in what was once called junk DNA is widespread, but its interpretation within a Darwinian framework remains. Thus, what was once touted as evidence of life's evolutionary history because of its lack of function is now interpreted as evidence of the same thing because it is functional. This experience calls into question how much data actually unambiguously support Darwinian evolution, what evolutionary theory actually predicts, and how data can be used to check its predictive power.

INTRODUCTION

During the late 1960s papers began appearing that showed eukaryotic DNA contained large quantities of repetitive DNA which did not appear to code for proteins (i.e., Britten and Kohne 1968). By the early 1970s, the term "junk DNA" had been coined to refer to this noncoding DNA (i.e., Ohno 1972). Junk DNA seemed like an appropriate term for DNA cluttering up the genome while contributing in no way to the protein coding function of DNA; yet there seemed to be so much of this noncoding DNA that its significance could not be ignored. One measure of the importance attributed to these noncoding sequences was the awarding of the 1993 Nobel Prize in Medicine and Physiology

to Richard Roberts and Phillip Sharp recognizing their 1977 discovery of introns (Chow et al. 1977, and Berget, Moore and Sharp 1977). These DNA sequences interrupt coding sequences and do not code for proteins themselves. In recent lists introns have been categorized as junk DNA along with other noncoding DNA (i.e., Nowak 1994).

Two lines of evidence pointed toward noncoding DNA's lack of functionality: first, significant variation in noncoding DNA is evident between closely related species and even within species (i.e., Zeyl, Bell and Green 1996). This variation is so great that it is used to produce DNA fingerprints that can differentiate between individual humans and individuals in many other species (Moxon and Wills 1999, Higgins 1999, Baker et al. 1993, Turner et al. 1992, Smith et al. 1990, Jeffreys 1988, Jeffreys, Wilson and Thein 1985). Conservation of protein (and thus DNA) sequences is a hallmark of coding functionality (Lewin 2000); consequently the presence of variability is assumed to mean a given stretch of DNA is noncoding. Initially it was assumed that if DNA did not code for protein, or certain specific RNAs, it was nonfunctional. The second line of evidence was somewhat more direct: mutation of some noncoding DNA did not produce significant changes in phenotype (Nei 1987 discusses this, but also points out that there are some constraints on evolution of noncoding regions). If the DNA did anything, then changing it should change the organism in some way. Some small change in DNA sequences may have little impact, but major and extensive mutation could reasonably be expected to impact any coding function. In the case of some "junk" DNA sequences, seemingly major changes produced no apparent impact.

This paper will document changes in the perceived meaning and role of noncoding DNA, starting with the initial view that an organism's genome could be viewed in much the same way as archaeologists view middens containing refuse from the past. In this view, noncoding DNA represents the broken remains of old genes that no longer function, mixed in with dust-like repetitive sequences that have blown in and multiplied. Thus, noncoding DNA, as long as it lacks function, may be mined for evidence of life's distant past and support for the argument that a Designer was not involved in creation.

It is important to emphasize that this logic hinges on lack of knowledge concerning function of noncoding DNA. Functional sequences come under the influence of selection which biases the data to such an extent

that a clear interpretation of past history is impossible. Selection may act like grave robbers who remove all precious metals and stones, biasing the record to appear that a culture lacked access to these resources. A second assumption deals with the nature of the Designer, who, if He exists, cannot have cluttered up the genomes of organisms with superfluous sequences. Finally, the mechanism of evolution — the "Blind Watchmaker", as Richard Dawkins calls it (1986) — could, and in fact would, produce cobbled-together genomes full of bits and pieces, some functional, and many simple clutter. This paper will demonstrate that, as data have accumulated, clear roles for non-coding DNA once thought to lack function have been found, and show that noncoding DNA is as consistent with design theory, perhaps more so, than it ever was with Darwinism.

WHAT IS JUNK DNA?

Because of confusion about the definition of junk DNA, the topic is difficult to discuss unless we first develop a working definition. In the most general use of the term, "junk DNA" is noncoding DNA — DNA that does not directly code for a protein product or specific RNA products like tRNA and rRNA. This noncoding DNA was initially assumed to be functionless, and thus noncoding DNA will be used as the definition of junk DNA in this paper. The meaning of "junk DNA" has become restricted significantly in recent years as the functionality of much of what was once considered junk has become obvious. Most modern genetics and biochemistry texts avoid the term. Even when junk DNA is mentioned, it is frequently given significantly different definitions. For example, Lodish et al. (1995, p 307) called it "extra DNA" for which no function has been found, and then footnotes the comment, "we do not use this term." Two dictionaries of biological terms (Stenesh 1989, and King and Stansfield 1990) call it "selfish DNA." In the early 1990s the term "selfish DNA," coined in the early 1980s (Orgel and Crick 1980, Orgel, Crick and Sapienza 1980), was popularized by Richard Dawkins (1989, p 366) in his book *The Selfish Gene*.

TYPES OF NONCODING DNA

At least nine classes of DNA were once thought to be functionless. Each of these has been referred to at one time or another as junk, and all were included in a list of types of junk DNA compiled by Nowak

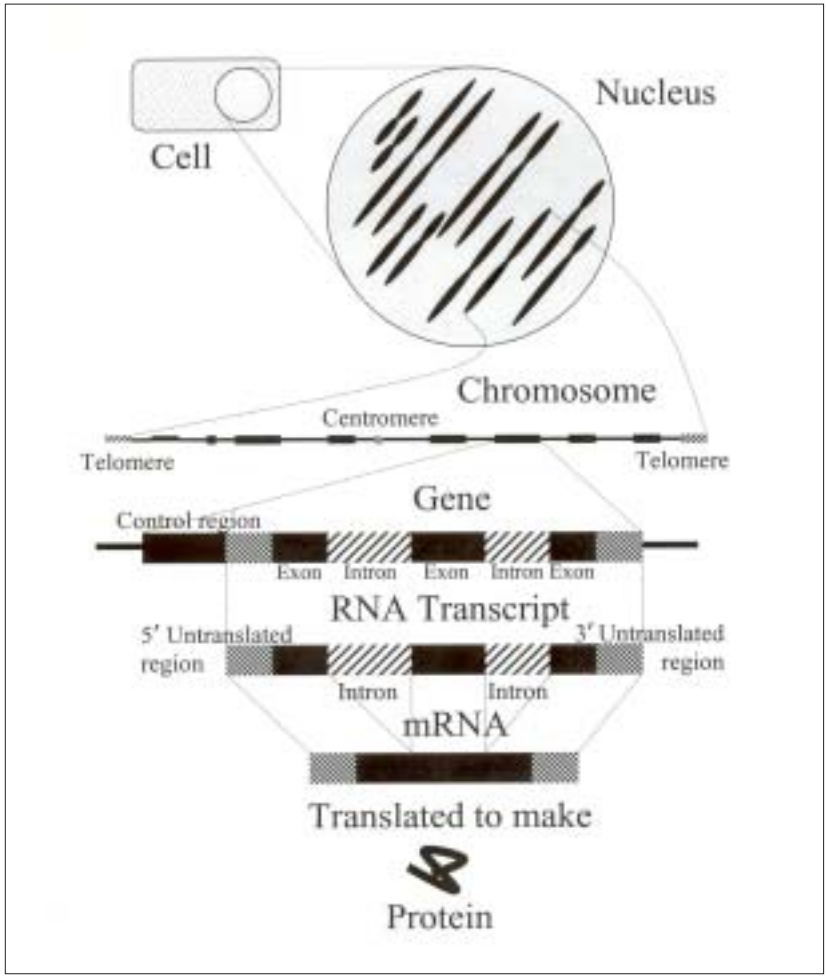

Figure 1. Only a small portion of DNA codes for proteins. Chromosomes, which are found in the cell nucleus, contain long linear stretches of DNA. Within the DNA are genes, but stretches of DNA between the genes do not code for proteins. At the beginning of genes (and frequently elsewhere) are control regions which play a role in regulating gene expression. Following the control region is the part of the gene which is transcribed to RNA. The RNA transcript starts with a 5' untranslated region followed by exons and introns and ends with a 3' untranslated region. Introns are removed to make the exons contiguous, and these contiguous sets of exons code for eukaryotic proteins. The protein is made by ribosomes which translate the RNA message contained in exons.

(1994). These nine types can be grouped into three larger groups: 1) untranslated parts of RNA transcripts, 2) repetitive DNA sequences, and 3) other noncoding sequences.

1. Untranslated Parts of RNA Transcripts

Not all RNA transcribed from DNA actually codes for protein (see Figure 1). Initial eukaryotic RNA transcripts produced by RNA polymerase II are called heterogeneous nuclear RNA (hnRNA). Before hnRNA can be exported from the nucleus as mRNA, it must first be processed to remove introns and make other modifications. Parts of the hnRNA that are removed do not code for the protein being produced, but even parts of the mature mRNA do not code for protein. Three noncoding parts of hnRNA are never translated: 1) introns (removed during RNA processing); 2) the 5' untranslated region; and 3) the 3' untranslated region. The latter two leave the nucleus as part of the mRNA. It is only the coding portion of mRNA, referred to as exons because they exit the nucleus, that carry genetic information defining a protein's amino-acid sequence. This code is translated into a protein in the cytoplasm of eukayrotic cells. Introns that are removed from hnRNA were thought to be junk cluttering the transcript which must be cast aside before the useful coding part of RNA transcripts can be translated.

Nucleic acids are always read in a specific direction, starting at the 5' end, and proceeding toward the 3' end. The 5' and 3' untranslated regions lie at each end of mRNA. Ribosomes, the organelles that translate the coding portion of mRNA into protein, attach first to the 5' end and slide along mRNA in the 3' direction until they reach a start codon signaling the beginning of a protein. Translation from mRNA to protein by ribosomes continues in the 3' direction from the start codon to the first stop codon. It seems reasonable to assume that the mRNA 5' end must play an important role in providing a ribosome attachment site and, this has been demonstrated (Lewin and Siliciano 1997). A function is not as immediately obvious for the mRNA 3' end which follows the stop codon signifying the end of the protein coding region. These 3' untranslated regions, because of their apparent lack of obvious function, have been classified as junk DNA.

2. Repetitive DNA

A surprisingly large proportion of eukaryotic DNA is made up of short sequences repeated many times. These repeated sequences seem

too short to code for proteins and are not known to be transcribed. There are five commonly recognized major classes of repetitive DNA:

1) Satellites, also called simple-sequence DNA. These are made up of many (up to 10^5) tandem repeats of a short DNA sequence, and seem to be concentrated in heterochromatin at the ends (telomeres) and centers (centromeres) of chromosomes. There are at least 10 types of human satellite DNA. Typically they make up 10-15 % of mammals' genomes.

2) Minisatellites are similar to satellites, but are scattered throughout the genome in clusters of fewer repeats.

3) Microsatellites are shorter still than minisatellites.

4) Short Interspersed Elements (SINEs), like mini- and micro-satellites, are found distributed throughout the genome, but differ in being single units of DNA about 300 bp (base pairs) in length, instead of repeated shorter units. An example is the human Alu SINE which occurs in the range of 300,000 times (Lewin 2000) making up about 5 % of the human genome (Deininger 1989). One of the interesting properties of SINEs is that they appear to move about in the genome.

5) Long Interspersed Elements (LINEs) are longer than SINEs, up to 7,000 bp — but typically about 6,500 bp — and, like SINEs, may move about in the genome. In mammal genomes there are 20,000-50,000 copies of L1, the most common LINE family (Lewin 2000).

3. Other Noncoding Sequences

Pseudogenes resemble genes, but are not known to be translated into functional proteins. Two classes of pseudogenes have been identified. The first class, unprocessed pseudogenes, resemble normal eukaryotic genes in all respects, but appear to have mutated and become functionless. Processed pseudogenes constitute a second class of pseudogenes. These unexpressed sequences resemble known genes with the introns removed. They appear to have been first transcribed as hnRNA from a functional gene, then processed into mRNA with the introns cut out and then reverse transcribed to make a DNA copy which was then inserted into the organism's genome at locations independent

of the original gene. Both classes of pseudogenes typically contain stop codons within all possible reading frames; thus only fragments of the protein they seem to code for would be produced if they were expressed.

It is not known if pseudogenes are expressed in any way, and because of their assumed history, Lewin (2000) refers to them as "dead ends of evolution." Interestingly, there are no obvious explanations for why certain gene families have pseudogenes while others do not. More commonly expressed genes may be more likely to have more pseudogenes. The problem with this explanation is that to be inherited, the events necessary to produce a processed pseudogene must occur in the germ cells. It seems unlikely that genes would be expressed in germ cells, which are not known to actively transcribe genes for which processed pseudogenes have been identified. In one exceptional case, a mouse ribosomal protein gene has approximately 15 related processed pseudogenes.

A diverse set of noncoding DNA is represented by heterogeneous nuclear RNA, a mixture of RNAs of varying lengths found in the nucleus. According to Nowak (1994) approximately 25% of the hnRNA is pre-mRNA that is being processed; the source and role of the remainder is unclear.

PROBLEMS WITH JUNK DNA

Noncoding DNA makes up a significant portion of the total genomic DNA in many eukaryotes. For example, older sources estimate 97 % of the human genome to be noncoding DNA (Yam 1995), while the recently published sequence data increases the estimates to 98.9% noncoding DNA (Venter et al. 2001). These estimates present problems for both intelligent design and naturalistic/evolutionary models of the history of life.

THE PROBLEM FOR DESIGN THEORY

It is difficult to imagine a Designer creating organisms exhibiting elegant efficiency at the gross level, but scattering superfluous molecular debris throughout DNA coding for higher levels of organization. Such inconsistency contradicts the argument that organisms are complex and efficient to such a degree that intelligent design, rather than random events coupled with natural selection, best explains their origin. If design predicts efficiency and noncoding DNA is nonfunctional, then noncoding

DNA must be evidence against genome design and for a more haphazard mechanism of origin.

Prominent evolutionists have eagerly proclaimed noncoding DNA to be molecular debris left over by the process of evolution. As mentioned previously, Dawkins (1989) and, much earlier, Orgel and others (1980; and Orgel, Crick and Sapienza 1980) proposed that evolution does not occur at the phenotypic level, but at the molecular level. Successful genes are "selfish" in that they "care" only about perpetuation of their own sequence. In perpetuating themselves, "genes" that do not compromise their "host's" fitness will proliferate relative to those that decrease host fitness. In this scheme some genes behave in a parasitic manner, perpetuating themselves while not significantly impacting host fitness. This view deconstructs organisms to the point that they are merely conduits for the preservation and proliferation of some segments of DNA, which runs directly contrary to the belief that the constituent parts of organisms all work together to enhance fitness of the whole. Highly repetitive and repetitive DNA sequences, including LINEs, SINEs, and the various satellite DNAs, are assumed to represent these *functionless* "selfish genes" that exist only for self-perpetuation.

Brosius and Gould (1992) moved boldly during the early 1990s to define the terms used for noncoding DNA in such a way that the data are first interpreted as evidences of evolution and then named. If their terminology had been adopted, any interpretation of the data outside the Darwinian paradigm would first have required a redefinition of the terminology used in discussing the data. They stated: "We wish to propose a general terminology that might aid the integrated study of evolution and molecular biology." Their proposed system of nomenclature assumed that noncoding DNA represents what was once functional may be functional again, but is *currently functionless*.

This "genomenclature" was challenged and even greeted with some ridicule at the time of publication. Graur (1993), in what must be one of the most amusing letters ever published in *Nature*, called genomenclature: "A cruel joke at the expense of the vocal chords of molecular biologists and the integrity of the English language." Brosius and Gould used terms like "nuon," meaning any definable stretch of nucleic acids, and "protonuon," meaning a stretch of nucleic acids with the potential to be recruited as a new gene via mutation and selection. Because these terms (and others) sound suspiciously like the physics terms "muon"

and "proton," Grauer went on to accuse Brosius and Gould of "a clear fit of physics envy." Grauer's main objection, however, was to the tongue-twisting nature of genomenclature, not the underlying assumption that noncoding DNA represents debris from the evolutionary past (this is pointed out in Brosius and Gould 1993).

Despite the ridicule, Gould continued to see noncoding DNA as both debris left over during the process of evolution and raw material for future evolution. In a *New York Times* opinion piece on the Human Genome Project, he stated:

> *Our 30,000 genes make up only 1 percent or so of our total genome. The rest — including bacterial immigrants and other pieces that can replicate and move — originate more as accidents of history than as predictable necessities of physical laws. Moreover, these noncoding regions, disrespectfully called 'junk DNA,' also build a pool of potential for future use that, more than any other factor, may establish any lineage's capacity for further evolutionary increase in complexity* (Gould 2001).

Walter Gilbert and others (Gilbert and Glynias 1993; Dorit and Gilbert 1991; Dorit, Schoenbach and Gilbert 1990) have promoted the idea that the exon-intron arrangement in eukaryotic genes represents a means of rapid evolution of functional genes that overcomes the problems represented by the incredible improbability of producing functional proteins via mutation of initially random sequences. In this model, each exon represents a functional domain; and by combining together different, already functional domains, new functional proteins can be made relatively easily. In other words, exons are the prefabricated nuts and bolts that can be used to make any number of functional molecular machines. Introns are functionless DNA that just happen to fall between functional exons. This view appears to be endorsed by Lewin (2000, p 58-62) in *Genes VII*, among the most respected molecular biology texts available.

THE PROBLEM FOR DARWINISTS

While Darwinists trumpeted noncoding DNA as prima facie evidence against design, they ignored the fact that efficiency is also accepted within the evolution paradigm as a hallmark of organisms. Efficiency is presumed to increase as natural selection eliminates less-efficient members of a population. As inefficiency increases, the burden it imposes is assumed to impact "fitness." When the impact on fitness

becomes biologically significant, selection will eliminate those organisms with systems relatively less efficient than others competing for the same resources. Only efficient organisms can survive in a selective environment.

The large amount of noncoding DNA in eukaryote genomes seems very inefficient. One would think that a trend would be evident in organisms, going from less to more efficient use of DNA. Ironically, the simpler the organism, the greater its efficiency in DNA use, not the opposite (Lewin 2000). The simplest organisms have little or no noncoding DNA. Alternatively, if noncoding DNA provides grist for the evolutionary mill, one might predict that organisms with more noncoding DNA would evolve more rapidly than those with less "extra" DNA as raw material to work with. This has not been demonstrated. Bacteria with relatively compact genomes are known to adapt to environmental changes at startling rates via rapid mutation. It is true that bacteria have very short generation times, and this may contribute to their rapid adaptation. It is also true that some different mechanisms may be in place in bacteria to direct genetic change, but the reality remains that in this diverse group of organisms whose genetic behavior has been extensively studied, biochemical adaptation to changing environments does not seem to require noncoding DNA.

Relative abundance of noncoding DNA can vary significantly between closely related organisms (see Martin and Gordon 1995, and Sessions and Larson 1987 for examples of this), indicating that changes in the amount of noncoding DNA is an easy evolutionary step. If it is easy to change the quantity of noncoding DNA, the question arises, "Why are those with more than the average amount of noncoding DNA not selected against?" It could be argued that the difference in efficiency between two individuals with varying amounts of noncoding DNA would not be large enough to impact the individual's reproductive success, but this is a troubling argument that is unsupported by the data.

Making and maintaining DNA requires significant energy input on the part of cells. Not only does the cell have to provide the deoxynucleotide building blocks for extra unneeded DNA, but also enzymes to polymerize and proofread newly made DNA, gyrases to unwind the template DNA, DNA repair enzymes, and so on. Factor all that into the 75 trillion cells in an average human with six billion bases in each nucleus,

and the cost becomes potentially significant, even though the cost of other cellular activities may have a much greater direct cost in terms of energy.

The problem of wasted energy would be so much greater if some "junk" DNA were translated, an apparent requirement if it is to serve as a resouce for evolution of novel new proteins. Akashi and Gojobori (2002) discuss the cost of polypeptide production and ways in which proteins, particularly those most commonly expressed, are optimized to utilize amino acids with the lowest metabolic cost possible. Clearly, if selection is sensitive enough to adjust specific amino acids within proteins to lower the energy cost of their production, then it should be sensitive enough to eliminate production of any "junk" proteins. It also follows that any DNA sequences that do not provide a selective advantage, especially if they constitute a significant majority of an organism's genome, should represent a significant metabolic cost and thus be selected against.

It cannot be argued that genome size has no phenotypic impact. Sessions and Larson (1987) have shown that, at least in some closely related salamander species, genome size is negatively correlated with the rate of development. Martin and Gordon (1995) suggest that the large amount of DNA in the nucleus of obligate neotenic salamanders slows development, increases cell size and slows metabolism which they suggest improves survival in cold-water environments. Supporting the theory that increased genome size slows development, Jockusch (1997) showed that genome size is positively correlated with embryonic development time.

Another example of phenotypic change correlated with variation in nuclear DNA size is evident in populations of the flowering plant *Silene latifolia*. In this plant, genome size shows a significant negative correlation with calyx diameter, a trait of clear ecological importance (Meagher and Costich 1996). Vinogradov (1997) has shown that resting metabolic rate in passerine birds is negatively correlated with increased nuclear DNA when body size is held constant. It is noteworthy that these papers emphasize the supposed evolutionary significance of noncoding DNA, and contradict the assumption that it lacks function. This at least partially disqualifies the previous argument that lack of function in noncoding DNA supports the idea that it is molecular debris of the evolutionary process. Whatever the source, much DNA appears to have a significant

phenotypic impact upon which selection may act, whether or not it directly codes for proteins or controls their expression.

Having unneeded DNA presents a potential danger to cells. It is not inconceivable that mutations could occur, resulting in production of noncoding RNA, some of which may interfere with production of essential — or at least beneficial — RNAs and, if they code for them, proteins. If "junk proteins" were made, their production would, at best, waste a cell's resources or, at worst, alter the activity of other proteins. Darwinists suggest that production of new proteins from old noncoding DNA is the very mechanism by which some new genes were produced. This postulated production of "junk" proteins via genes whose expression is not tightly controlled presents a potential danger to cells both by sapping the resources of the cell for a non-productive task and also because the protein may have functions that interfere with the normal function of other essential components of the cell. Nyolase produced by *Flavobacterium* has been presented as an example of a new functional protein arising from a sequence (in this case assumed to be a formerly unread reading frame) which did not previously code for any protein (Ohno 1984). If functional proteins can spring forth from previously noncoding sequences, they need not all be adaptive; in fact, harm to the cell appears a far more likely outcome.

Loss of functionless DNA would seem to be a relatively easy evolutionary step. Gaining DNA may be more difficult, although data exist which are consistent with the theory that increases in the number of copies of some DNA stretches has occurred as a result of imperfect crossing over during meiosis prophase I. Alternative explanations of these repeats may be equally consistent with the data, but the important point for this argument is that DNA which is not a normal part of an organism's genome has been shown to be rapidly lost. For example, Petrov and Hartl (1998) have shown that, at least in *Drosophila* species, functionless DNA disappears after only a few generations. This appears to be analogous to the vision loss observed in some fish and other organisms that live in caves, or the loss of flying ability observed in birds that live on isolated islands. The conventional explanation is that without selective pressure to maintain them, these abilities are lost. In caves where there is no light, sight provides no selective advantage. Similarly, flight provides little advantage in the absence of predators and presence of abundant marine food around islands. Apparently, at least in *Drosophila*, extra

DNA, like sight and flight, will not be maintained in the absence of selective pressure to maintain it.

The fact that DNA not normally part of a specific genome is easy to lose, combined with evidence that increases in genome size significantly impact phenotype, calls into question the idea that noncoding DNA does not impact fitness enough for natural selection to work on it. These data, combined with the logical inference that noncoding DNA may produce RNA or protein products that negatively impact fitness, all call into question the idea that noncoding DNA represents a currently functionless record of the phylogenetic history of organisms which has been passed down over many generations.

For both intelligent design theorists and Darwinists, noncoding DNA presents a problem if it is really functionless. Intelligent design assumes that a wise Designer would not add functionless rubbish to His creation. Evolutionists assume some function, exemplified in Brosius and Gould's (1992) nomenclature, if not in the present, at least as remnants of past functionality and raw material for the future. Assuming that noncoding DNA lacks function appears to violate the basic scientific assumption that what is seen in nature exhibits some purpose which can be determined through observation and experimentation. Enthusiasm for absence of function in noncoding DNA appears to have sprung more from philosophical presuppositions, than a careful analysis of data and their implications for Darwinism. If any functionality was to be assigned to noncoding DNA, it was to be done within the context of its role in evolution, not on the basis of any immediate benefit to the organism bearing it in its nucleus.

EVIDENCE OF FUNCTIONALITY IN NONCODING DNA

Both direct and indirect evidence show that functionality is present in some noncoding DNA. One way to look for potential function is to see if DNA sequences exhibit characteristics of other sequences known to be functional. Using this approach, sequences known to code for proteins and those that do not have both been shown to exhibit characteristics of an information carrying code. Searls (1992, 1997) suggested that DNA exhibits all the characteristics of a language, including a grammar. As early as 1981 (Shulman, Steinberg and Westmoreland. 1981) and in later papers (i.e., Michel 1986), statistical methods were published for obtaining coding sequences out of the morass of noncoding DNA. More recently statistical studies utilizing neural networks have been used to

locate protein coding regions (Uberbacher and Mural 1991, Granjeon and Tarroux 1995). This work concentrates on finding statistical patterns to distinguish coding from noncoding sequences; they do not show that noncoding sequences still contain information, only that they exhibit a statistical signature. More direct work has been reviewed by Yam (1995). Mantegna et al. (1994 and 1995; also see Flam 1994, Havlin et al. 1995, and Peng e al 1995) applied a method for studying languages (Zipf approach) to the study of DNA sequences and suggested "noncoding regions of DNA may carry biological information." While this paper has not gone unchallenged (see Tsonis, Elsner and Tsonis 1997; Konopka and Martindale 1995; Yam 1995; Chatzidimitriou-Dreismann, Streffer and Larhammar 1996), it does suggest that DNA should be examined for functions other than protein coding.

Aside from protein coding, DNA sequences may include signals controlling replication and other aspects of the cell lifecycle. Manuelidis (1990) suggests that during interphase (ordinary cell activity) chromosomes are localized in specific parts of the nucleus in different cell lines due to three-dimensional structure imparted to them by folding of "junk" DNA. This three-dimensional structure may also "index different genetic compartments for orderly transcription and replication." More recent work by Macera et al. (1995) has shown that noncoding DNA may play a role in the suppression of genes and suggests that some clinical conditions result from changes in noncoding DNA. Reinhart et al. (2000) have shown that a short RNA sequence regulates developmental timing in *Caenorhabditis elegans*. Eyre-Walker (1999) has shown evidence for selection on noncoding DNA that varies its GC content. Earlier work by Martin et al. (1984) discussed a mouse interspersed repeat that, "…evolves as if it encodes a protein." This seems to imply some level of functionality. If selection is operating on a noncoding DNA region, this region must have some impact on fitness. Related to this thought is research reported by Koop and Hood (1994) showing surprising sequence homology between long regions of corresponding mouse and human noncoding DNA, again implying function and selection to maintain the sequence.

After the excitement about noncoding DNA in the early 1970s, many special examples of functional noncoding sequences have been found. Every untranslated part of hnRNA and mRNA has been found to have a function in at least some transcripts. Some introns contain

other genes that are expressed independently of the exons they separate. Thus, as long as the coding strands for both genes are the same, they will always be transcribed together. In addition, the work of Thomas Cech (Cech 1985, Kruger et al. 1982, Zaug, Grabowski and Cech 1983) has shown that introns are not noncoding stretches of RNA transcribed from equally functionless DNA but, in some cases, act in complex ways resembling protein enzymes as they splice themselves out of pre-mRNA. These segments of DNA, once thought of as merely interrupting the important parts of eukaryotic genes, are now found to play an active role in removing themselves from the gene transcript. This does not show a coding role for the introns, but reveals a level of complexity and potential functionality previously unanticipated. Not all introns have been shown to contain these "ribozymes," but ribozymes should encourage caution before writing off introns as having no function.

It is also worth noting that there is a significant trend toward increased gene size when bacterial genes are compared to those in single-celled eukaryotes. The trend continues when yeast genes are compared with nematode or fly genes, and when these relatively "simple" organisms' genes are compared with genes from humans and other complex multicelled eukaryotes. While gene size goes up dramatically, only a very small proportion of the increase results from increases in the size of exons which code for protein. The bulk of the increase can be attributed to increase in the number and size of introns (Lewin 2000). This correlation between an increase in introns and an increase in apparent phenotypic complexity needs to be explored further before the role of the introns is assumed to be insignificant.

Specific functions for some introns have been discovered. For example, many introns also code for small nuclear RNAs (snRNAs). These accumulate in the nucleolus, and may play a role in ribosome assembly. Thus the introns that are cut out of the pre-mRNA may play a role in either producing or regulating machinery that translates mRNA's codons into protein. Zuckerkandl (1997) reviews work showing introns, along with other noncoding DNA, play an important role in repression of genes and the sequential switching of genes during development, suggesting that up to 15% of "junk DNA" functions in this vital role. A specific example of regulation of expression by an intron sequence involved the suppression of rat osteocalcin gene by the sequence TTTCTTT within

the first intron of the osteocalcin gene (Goto et al. 1996). The repressor sequence serves as a negative feedback on expression of the gene.

In artificial settings, RNA has been shown to be capable of repressing the expression of specific genes. This repression has been demonstrated in a heritable manner in the roundworm *Caenorhabditis elegans* (Grishok, Tabara and Mello 2000). The extent, if any, of RNA inhibition (RNAi) in nature has not been established, but serves as another example of an unanticipated role for RNA which may be related to the kind of negative feedback seen in the TTTCTTT sequence in the osteocalcin intron. A general review of the nature and role of introns when viewed from a design standpoint is given by Bergman (2001).

The obvious role of the 5' untranslated region of mRNA in signaling for ribosome binding has already been mentioned. Untranslated regions at the 3' end of mRNAs have been found to play an important role in the regulation of some gene activity (Wickens and Takayama 1994) and thus clearly engage in an important function. A specific example of function for the 3' untranslated region has been demonstrated in regulation of the human luteinizing hormone/chorionic gonadotropin receptor gene (Lu and Menton 1996). In this case several different mRNA transcripts for the receptor gene are known. The mRNA species with a long 3' untranslated region repress expression of the gene by reducing affinity for ribosomes and reducing the mRNA cytoplasmic half-life. The mRNA species with short 3' untranslated regions increased protein expression, apparently as a result of some other post-transcriptional mechanism of regulation. As new roles played by RNA are discovered and understanding increases of the enzyme-like properties of some RNAs, dismissing hnRNAs which are not precursors of mRNA as lacking in immediate function seems premature. It may be reasonable to predict that as we learn more about the roles of noncoding RNA sequences inside and outside of the nucleus, particularly in control of gene expression, ever-decreasing amounts of it will be consigned to speculative roles in an organism's evolution.

At the centromere, satellite DNA sequences play a role in assembly of the kinetochore and attachment of spindle fibers during mitosis (Wells 1966). Satellite sequences play an equally dramatic role at chromosome ends where a few nucleotides are lost from telomeres during each replication cycle. Given enough replications, telomeres are eliminated unless the lost nucleotides are replaced. Loss of the telomeres leads to chromo-

some shortening, and further replication and shortening results in loss of important functional genes. Damage or loss of these genes may lead to cell senescence. It is speculated that telomere loss is a partial mechanism for aging (see Hodesa 1999 for a review of the relationship between telomeres and aging). In some — but not all — cells, special enzymes called telomerases add satellite DNA to the ends of chromosomes so that there is little or no loss of DNA after replication. Thus, noncoding satellite DNA in telomeres plays an important role in preserving the ends of chromosomes and maintaining functional cell lines. Some suggestions about the role of natural selection working on the length of DNA segments and favoring tandem repeats have been made by Stephan and Cho (1994). In this case, the function of some tandem repeats may be to regulate length, not to code or signal anything else.

Long and Short Interspersed Elements (LINEs and SINEs) appear at first examination to almost perfectly fit Dawkin's definition of "selfish DNA." Because of their transposon activity, they seem to pose a hazard to normal gene function. As they move around in the genome they may insert into functional genes disrupting protein coding, or destroying control regions. A number of documented genetic diseases have been shown to be caused by movement of SINEs and LINEs. Individual cases of neurofibromatosis-1 (elephant man disease) are associated with insertion of a SINE, while some instances of hemophilia and Duchenne muscular dystrophy appear to result from disruption of genes by LINES. Aside from destroying genes as they move around in genomes, potential function for at least one SINE has been demonstrated: the Alu SINE has been shown to play a role in control of protein synthesis when cells are stressed (Chu et al. 1998). A role in X chromosome inactivation has also been proposed for the most common LINE, L1 (Lyon 2000). This is based on the observation that, compared to its frequency in autosomes, L1 appears at almost twice the frequency on X chromosomes and is particularly concentrated around the region where chromosome inactivation starts (Bailey et al. 2000). Most recently a potentially very important role in repairing breaks in DNA has been demonstrated for L1 by Morrish et al. (2002).

The absence of a recognized general role for microsatellites may be because this designation is based on sequence characteristics, not function. While the characteristics of different sequences may categorize them as microsatellites, their functions may vary dramatically. Nadir

et al. (1996) show evidence that the Alu SINE is associated with A-rich microsatellites and suggest a role for this class of microsatellites in providing targets for Alu insertion. According to this interpretation, A-rich microsatellites act as markers for Alu retroposition, thus playing a role in preventing gene disruption by insertion of Alu at inappropriate locations. This may be an important role given the already noted diseases caused by movement of SINEs and LINES.

Clearly, if this interpretation is correct, microsatellites play an important role in the organization of chromatin and, in cooperation with the Alu SINE, may act as part of an elaborate mechanism for the regulation of gene expression. A separate role for microsatellites in organization of chromosomes within the nucleus is suggested by the observation of Gasser and Laemmli (1987) who noted that A and T boxes resembling A-rich microsatellites are found associated with the nuclear scaffold. Attachment of chromosomes to the nuclear scaffold, possibly involving these A and T boxes, is believed to be responsible for arrangement of DNA within the nucleus.

Defects in microsatellites are associated with some types of cancer, although this is assumed to be an indicator of susceptibility to replication errors rather than a cause of cancer (Moxon and Wills 1999). Increase in the number of repeats within microsatellites making up part of the coding portion of some genes has been associated with Huntington's disease and a number of rare neurological disorders. Variation in the size of triplet repeat microsatellites within genes has been shown to affect gene expression. Interestingly, Moxon and Wills suggest that rather than being the molecular debris of evolution, microsatellites play an active role in the adaptation of bacteria to potentially lethal changes in their environment. Because of the role played by microsatellites in phase variation, Moxin and Wills call bacterial microsatellites "true evolutionary adaptations." They go on to suggest that microsatellites may play a similar role in the rapid adaptive regulation of eukaryotic genes. This represents a major shift from viewing this class of noncoding DNA as lacking function or as selfish DNA, although it still illustrates the imposition of an evolutionary framework on how data are interpreted.

At least one microsatellite sequence — AGAT — has a demonstrated function in regulation (Weiss and Orkin 1995). This shows that different subclasses of microsatellites may play significantly different, but important, roles (Nadir et al. 1996).

The role of pseudogenes, if there is one, remains problematic. Unprocessed pseudogenes appear to be copies of normal genes which mutated and lost their function over the course of time. Processed pseudogenes appear to be degenerate genes. These present a more problematic picture, particularly in the light of their association with retroposons.

Despite the poor record of the assumption that noncoding DNA is functionless, papers published relatively recently invoke the term "junk DNA" when describing DNA for which no function has yet been determined (see Gardiner 1997 for an example of this). Still, the assumption that noncoding sequences lack function seems to be going out of vogue, and calls are being made to investigate potential functions for even the most unpromising simple repeats (for example see Epplen, Maeueler and Santos 1998). Because the term "junk DNA" is still used to refer to noncoding DNA, much of which is clearly functional, there is some discussion of completely abandoning the term, although no obvious replacement is evident (Kuska 1998a,b), the efforts of Brosius and Gould (1992) having been ignored. However, the term remains in use and a cursory search using PubMed reveals at least 10 instances of its use in titles of papers published in major journals between 1997 and 2001. All of these papers either deal with technical issues associated with noncoding DNA in the general study of DNA sequences, or suggest functions for it. Clearly, while specific functions for all noncoding DNA have not been discovered, the assumption of lack of function no longer dominates the thinking of molecular biologists.

CONCLUSIONS

Much of the excitement surrounding noncoding DNA appears to have been misdirected. In many respects the history of noncoding DNA resembles that of vestigial organs. Evolutionists accepted the assumed lack of function of noncoding DNA as evidence supporting their worldview, even though lack of function is not necessarily a logical deduction from evolutionary theory. Furthermore, an assumption of function does not have to follow from the idea of design. In claiming that noncoding DNA supports evolutionary theory, predictions of functionality reasonably based on that theory had to be ignored.

Darwinists defined what they thought a Designer would do and then presented noncoding DNA as violating that prediction. In doing this three mistakes were made:

1) Terms of the argument were unfairly constrained by defining the Designer in a way that seemed to be contradicted by the evidence. If a Designer exists, He is not compelled to fit any definitions His creatures may want to impose, especially not those definitions that preclude His existence on the basis of what He created. Designers can do whatever pleases them. If this were not so, it would be reasonable to question that automobiles with functionless fins from the 1950s were designed by intelligent beings.

2) A second error involved treating the hypothesis that noncoding DNA lacked function as if it were well-supported by the data, when there were little data. Worse still, the hypothesis was invoked as if it were a fact instead of a tentative interpretation. If noncoding DNA is functional, then the argument that a Designer would not have included functionless junk in the design becomes irrelevant.

3) The final failure was neglecting to examine evolutionary theory to be sure that it does not predict functionality. This failure resulted in a false dichotomy between the predictions made by design versus those made by Darwinism. It might be argued that in this final error some latitude can be given, as evolutionary theory does not always make clear predictions. In fact, it frequently appears to be more robust than other ideas because it can be adjusted to "predict" whatever the data happen to say. As long as noncoding DNA appears functionless, that is what evolutionary theory predicts, but if it is functional, then evolutionary theory provides an equally accommodating framework in which to fit the data.

The history of noncoding DNA serves as a cautionary tale illustrating the danger inherent in ignoring the predictive value of one's paradigm. Careful evaluation is needed before jumping on a new trend and claiming that it supports one side or the other of the creation-evolution debate. In attempting to discredit creationists, Darwinists ignored the prediction of functionality made by their own theory and the lack of supporting data. Rushing to judgment is never a wise first step when examining the predictions of competing theories in the absence of sufficient data.

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ANNOTATIONS FROM THE LITERATURE

DESIGN: TRILOBITE COMPLEXITY

Chadwick, AV, DeHaan RF. 2000. The trilobite: enigma of complexity. A case for intelligent design. Perspectives on Science and Christian Faith 52:233-241.

Summary: Trilobites are extinct arthropods, with a level of complexity similar to that of living arthropods. It seems reasonable to infer that they shared biological processes seen in arthropods today. This means that trilobites had a complex genetic system, cell division, a nervous sytem, a developmental system, sense organs including eyes, etc. These systems imply the existence of the same biomolecules seen in living arthropods, including DNA, histones, and other associated proteins, microtubules, neurotransmitters, regulatory proteins, and many others. But there is no evidence in the fossil record of the source of this complexity. Trilobites are found in Cambrian sediments, without a trace of ancestry in the underlying sediments. Intelligent design theory provides an explanation for the source of this complexity, while ordinary evolutionary theory does not. Speculation on the identity of the designer goes beyond the boundaries of science.

Comment: It seems apparent that design can be seen throughout the history of life on this planet. Trilobites, as well as living organisms, illustrate this design as well.

GEOLOGY: CHANGES IN EARTH'S AXIS

Prevot M, Mattern E, Camps P, Daignieres M. 2000. Evidence for a 20^0 tilting of the Earth's rotation axis 110 million years ago. Earth and Planetary Science Letters 179:517-528.

Summary: The author used paleomagnetic measurements from continental rocks to infer that Earth's axis shifted through the Cretaceous, with an abrupt shift of about 20^0 during the mid-Cretaceous. This may have reflected a major shift in distribution of Earth's mass during the Lower Cretaceous. Effects of the axial shift on distribution of mantle material might be linked to high plume activity and low frequency of geomagnetic reversals during this time.

Comment: The breakup of Pangaea is thought to have occurred largely in the Cretaceous. An abrupt change in the angle of Earth's axis could produce sufficient force to move the continents. How these processes are linked remains an important research question.

MOLECULAR EVOLUTION: DEGENERATION OF DUPLICATED GENES

Lynch M, Conery JS. 2000. The evolutionary fate and consequences of duplicate genes. Science 290:1151-1155.

Summary: Gene duplication and divergence is widely regarded as the source of new genetic information. The theory is that a duplicated gene might evolve to a new function. Alternatively, duplicated genes might experience crippling mutations and degenerate into useless DNA sequences. By prospecting for duplicate genes in genomic sequences from several eukaryotic species, Lynch and Conery concluded that gene duplications occur much more frequently than expected, with rates similar to those of point mutations. The vast majority of duplicated genes become nonfunctional. Very few duplicated genes retain any function, and those that survive experience strong selective pressures. Different fates of duplicated genes in different populations could contribute to the process of speciation.

Comment: An interesting implication of this study is that it indicates duplicated genes are highly unlikely to produce new functions. It is much more likely that duplicated genes will degenerate and contribute nothing to evolution. The high rate of duplication inferred in this study is based on the supposition that only a single gene copy existed at the origin of the species. This supposition might be testable by comparing genomic sequences among closely related species to see whether they vary in the number of gene copies. If some genes were created in duplicate, the actual rate of gene duplication might be overestimated.

ORIGIN OF LIFE: RNA CAN'T TAKE THE HEAT

Moulton V, Gardner PP, Pointon RF, Creamer LK, Jameson GB, Penny D. 2000. RNA folding argues against a hot-start origin of life. Journal of Molecular Evolution 51:416-421.

Summary: Deep-sea hydrothermal vents have been proposed as a likely location for the origin of life. The presence of charged metallic surfaces, particularly iron pyrite, might facilitate chemical reactions

leading to the production of RNA and other biologically important macromolecules. Both theoretical and experimental results show that high temperatures such as exist around hydrothermal vents strongly reduce RNA folding. RNA molecules showed no secondary structure at temperatures above about 70^0 C. Furthermore, high temperatures are destructive to RNA molecules. At 100^0 C, an RNA molecule of 2000 nucleotides is expected to experience one break every 26 seconds. Cytosine is especially unstable at high temperatures. These features make it highly unlikely that life originated in a high-temperature environment, such as around hydrothermal vents.

Comment: These results reinforce previous reports that macromolecules are desroyed rather than created in hot aqueous environments. The presence of archaea in extreme environments is not a reflection of the origin of life, but of its diversity.

PALEONTOLOGY: DINOSAURS ON THE BEACH?

Lopez-Martinea N, Moratalla JJ, Sanz JL. 2000. Dinosaurs nesting on tidal flats. Palaeogeography, Palaeoclimatology, Palaeoecology 160:153-163.

Summary: Sauropod and hadrosaur dinosaurs have been variously interpreted as aquatic or terrestrial reptiles, with most paleontologists now favoring the terrestrial interpretation. However, dinosaur fossils are often found in sediments interpreted as marine, sometimes scavenged by sharks, or even encrusted with oysters. Dinosaur tracks are often located in sediments interpreted as margins of aquatic environments.

Now a group of dinosaur eggs has been found in an Upper Cretaceous tidal flat environment in Spain. The eggshell fossils have a high porosity. If the original eggshells were this porous, they would become dehydrated if left in a dry environment. This points to a periaquatic habitat for these dinosaurs. Modern crocodiles lay their eggs near water, and cover them with vegetation and mud. It is not known whether these dinosaurs followed a similar behavioral pattern. The identity of the dinosaurs is not known. Sauropod bones have been found in a nearby mine, and many eggshell fragments are found in the general area, along with fossils of mostly marine species.

Comment: It seems likely that some types of dinosaurs lived in or near aquatic habitats, while others did not. The preservation of such a

large number of eggshells and other fossil material must have required special circumstances. Examples such as this provide interesting problems regarding the conditions under which the fossils were assembled and preserved.

PALEONTOLOGY: PERMIAN BACTERIA BROUGHT TO LIFE

Graur D, Pupko T. 2001. The Permian bacterium that isn't. Molecular Biology and Evolution 18(6):1143-1146.

Summary: The small differences in DNA sequence between the "Permian" bacteria and contemporary bacteria indicate a short history of divergence and point to a more recent contamination of the salt deposits.

Comment: Contamination is a potential problem, but the investigators took great pains to carefully select salt crystals that appeared uncontaminated. A criticism based on expectation of great divergence in DNA sequence seems less than compelling in the absence of any empirical basis for suspecting contamination.

Vreeland RH, Rosenzweig WD, Powers DW. 2000. Isolation of a 250 million-year-old halotolerant bacterium from a primary salt crystal. Nature 407:844-845. For reactions, see Nature 411:155 (2001).

Summary: Bacteria were cultured from a salt crystal recovered from the Permian Salado Formation near Carlsbad, New Mexico. Care was taken to avoid contamination with modern bacteria at every step of the process. The salt crystal did not show signs of recrystallization or other alteration. The bacteria were identified as a *Bacillus* species. Sequence comparisons of 16s ribosomal DNA showed close similarity to *B. marismortui* (99% identity), and *Virgibacillus panthothenticus* (97.5% identity). The previous accepted record for oldest viable bacterial spores was based on bacteria cultured from 25-30 million-year-old amber.

Comment: This is a remarkable claim. If the bacteria were truly deposited with the salt, the spores have been quiescent since the time of deposition of the salt. According to the molecular clock hypothesis, one would expect a sequence divergence far greater than 1% over a period of 250 million years. For example, a recent experiment on *E. coli* over 10,000 generations showed an average detected rate of genetic

divergence of approximately 10^{-3} per generation. At this rate, an average divergence of 1% would be reached in 1,000 generations, about 150 days. The number of generations in 250 million years should be on the order of 10^{11}. Clearly interesting research questions are raised by the report of viable Permian bacteria.

LITERATURE REVIEWS

Readers are invited to submit reviews of current books or journal articles relating to origins.Please submit contributions to: ORIGINS, Geoscience Research Institute, 11060 Campus St., Loma Linda, California 92350. The Institute does not distribute the publications reviewed; please contact the publisher directly.

DARWIN HIMSELF

Reviewed by Henry Zuill
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ANNIE'S BOX: CHARLES DARWIN, HIS DAUGHTER, AND HUMAN EVOLUTION. Randal Keynes. 2001. London: Fourth Estate, a division of HarperCollins Publishers. 331 p. 34 plate + 19 text illustrations. Cloth, £16.99. ISBN 1-84115-060-6.

Randal Keynes, author of *Annie's Box*, is the great-great-grandson of Charles and Emma Darwin. Thus, he has direct access to family papers and other materials pertaining to the life of Charles Darwin. Some of these historical records are new to the public. He has drawn on these, and other contemporary writings, to reconstruct the Darwin family's daily life, and show how it contributed to Charles' thinking as he developed ideas about evolution, particularly human evolution.

The book is historically interesting, but recommending it requires qualifications based on what readers anticipate. Some may hope Kenyes has new insights into Darwin's ideas. However, anyone, who anticipates new understandings about evolution, will be disappointed. Others may look for confirmation of an often-rumoured late-life conversion. Likewise, this will not be found. If it happened at all, it is not recorded here.

If one is curious about the mindset of Darwin, however, then *Annie's Box* will be helpful. The book gives an intriguing description of the man himself, and how and why he reached his opinions. Often, Darwin is either eulogized or demonised; here, however, we find neither. In this balance, I believe Keynes makes a contribution.

Annie's Box is arranged chronologically from when Darwin was beginning his career and marriage, through the years of child rearing, to

old age, and finally death. Charles Darwin was a complex man, and as presented in *Annie's Box*, he embodied a peculiar mix of emotions — gentleness, egocentricity, timidity, determination, and even bigotry. There is some of each of these qualities in every person. Seeing them here reminds readers that objectivity is hard to achieve.

Several threads weave through *Annie's Box* and sometimes intertwine. One thread deals with Charles' spiritual doubts in contrast with Emma's steady faith. When Charles' learned about vast geological ages, he began to doubt the biblical creation account, and in turn, this produced doubts about the rest of the Bible. For example, he saw the God of the Old Testament as a vengeful tyrant. Thus that part of Scripture was dismissed. The New Testament, based on Old Testament prophecies, likewise had to go. His upbringing and theological training had not given him an active faith, and when science raised serious questions, he was swept away. Eventually this led him to reject all of Scripture as divine revelation.

Sometimes he doubted his doubts, but always returned to them. He eventually saw himself as an agnostic as opposed to being an atheist, but he seems to have given the Creator little, if any, benefit of the doubt. Agnostics are often difficult to distinguish from outright unbelievers.

Darwin was determined to make a scientific name for himself, and focused on natural selection as a mechanism to not only explain evolution, but also serve as his vehicle on the road to fame. Though a careful observer of nature, he was obsessed with his species theory, and often extrapolated far beyond the limits of data. The picture that comes through is that, as a scientist, he was considerably less than objective. He was convinced that he was right and once referred to his theory as "all gospel." In his view, natural selection had unrestricted powers.

Perhaps we can be more understanding of Darwin, given his limited view of nature. On the other hand, today's scientists who do the same thing in the light of far more extensive understanding of such things as the limiting nature of genes, and the fossil record, cannot be so easily dismissed.

Another intriguing theme concerns Darwin's views of suffering in nature. At that time, children often died early. Three of the ten Darwin children did not reach adulthood. Two died in infancy, but Annie, for whom the book is titled, was ten when she succumbed to what appears to have been tuberculosis. Readers will be charmed by the description

of this little girl, and will share in the grief that followed the family, particularly Charles and Emma, for the rest of their lives.

In addition to such great and grievous loss, Charles Darwin was often ill. He knew suffering from personal experience.

His grief and illness highlighted the problem of human and animal pain. Emma saw suffering as, in the end, producing a greater good. Charles struggled with this problem for years, and always ended at the same place: that there was no God active in nature. We would do well to re-examine this problem. Each thinking person must grapple with it; established faith, to some degree, depends upon the answer.

Chronic ill-health is another part of the Darwin puzzle. I wondered how prevailing medical misinformation and its horrific applications at that time might have contributed to his problems. His condition seemed to worsen under stress, however; and even at the best of times, whatever it was that so often plagued him never seemed far away. For around twenty years Darwin kept his species theory secret, fearing to make it public. He felt this stressful hesitancy contributed to his bad health — and it probably did. It was a high price to pay.

It is evident that Darwin wanted to be accepted and well thought of. Thus he delayed publishing his theory, clearly recognising its potential for rejection, and himself along with it. That it was so quickly accepted was surprising, possibly even to him. It could not have been because it was so persuasive. Many of his fellow scientists saw its weaknesses. Certainly, the idea of species fixity was easily rejected in light of what was being learned about nature; but its rejection, given its theological and ecclesiastical implications, must have contributed to weakened faith, as it did for Darwin. Moreover, could a general lack of faith, and rejection of moral accountability, have been a major deciding factor in acceptance of Darwin's theory?

It is ironic that the man whose theories did so much to undermine faith is buried in Westminster Abbey.

In summary, *Annie's Box* is a revealing peak into the mind of Darwin. I believe readers will gain new insights into, not only what drove Darwin, but also the social milieu in which his ideas took root.

GENERAL SCIENCE NOTES

THE MIOCENE/PLEISTOCENE CONTACT IN THE COLUMBIA BASIN: TIME IMPLICATIONS

Harold G. Coffin
The Dalles, Oregon

INTRODUCTION

Approximately 14 million years is thought to have elapsed between the laying down of the last flow of most of the Columbia River Basalts and the deposition of the wind-blown glacial silts of the Palouse soil (Baksi 1989, Fryxell and Cook 1964). If 14 million years were involved, the amount of erosion should be profound, cutting down into many basalt beds of the Columbia River Plateau. The purpose of this research was to examine this contact for evidence of 14 million years of erosion.

Many so-called time gaps exist in the geologic record. Some of these time gaps show little erosion, even though many millions of years are said to have elapsed between the laying down of the lower bed and the deposition of the upper stratum. Roth (1988) has commented on the significance of this phenomenon. The geology in this part of Eastern Washington State seemed suitable for a close examination of one of these gaps, even though the time gap is not as great as has been seen at some other locations. The gap exists between the uppermost deposit of Miocene Columbia River Basalt and the Pleistocene Palouse loess that overlies it. The bed below the gap was laid down as molten lava. If no long period of gradual deposition was involved, the contact should be sharp, unless eroded. Another factor favoring this study was the great difference between the two beds — one being hard, dark volcanic material and the other being light, soft loess of sedimentary origin.

LOCATION AND GEOLOGY

The area studied is a plateau that lies in Washington State between Walla Walla on the south and Spokane on the north, and between the Idaho border on the east and U.S. Highway 395 on the west (Figure 1). The topography of most of this area is rolling hills that are the result of the warping and doming of the basalt, and several canyons that cut

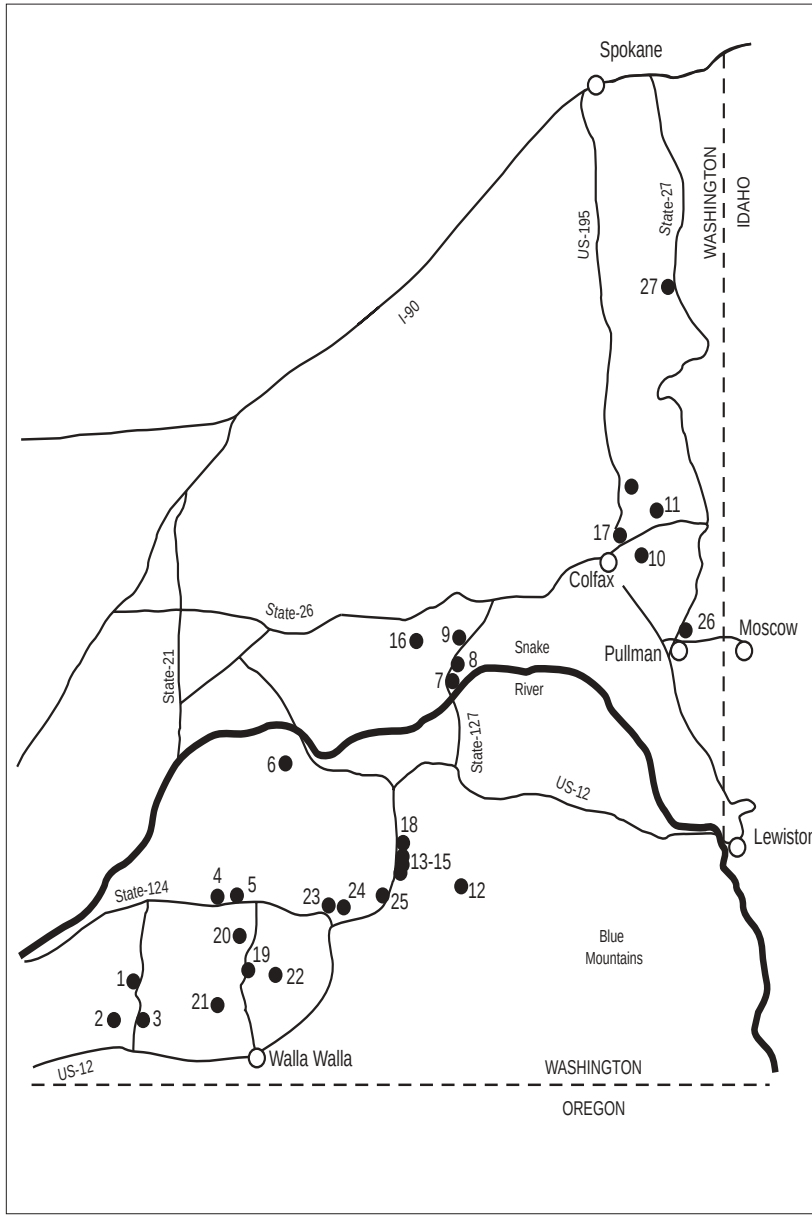

FIGURE 1. A map of the Palouse county of Southeastern Washington. The collection sites are numbered according to the chronological order in which they were discovered and examined during three separate trips to the area.

deep into the plateau. The elevation gradually drops from about 850 m on the east to around 150 m on the west. Several topographic highs on the east side are composed of the Precambrian granites that underlie the basalt. The best-known of these are Steptoe Butte (front cover photo) and Kamiak Butte, 1101 m and 1110 m high respectively.

Mantled over the basalt (but not on the steep granite buttes) is a thick layer of fine sediment considered to be wind-blown glacial silt (Busacca 2001). The silt feathers out toward the north and west and disappears among the forested foothills of the Rocky Mountains of Idaho. Except along the edges, it provides an excellent soil up to 5 m deep that is utilized for both dry wheat farming and irrigated crops.

The Missoula Flood (sometimes called the Bretz Flood, or the Spokane Flood) that resulted from the breaking of an ice dam that produced huge Lake Missoula in the intermountain valleys of Northern Idaho and Western Montana removed the loess from long northeast-southwest trending tracts and scoured out deep canyons in the underlying basalt (Orr, Orr and Baldwin 1992).

Many flows of lava (over 300) that arose from fissure eruptions in Southeastern Washington, Northeastern Oregon and adjacent Idaho were laid down in the Columbia Basin before the deposition of the loess (Tolan et al. 1989). These eruptions are interpreted to have commenced around 17 million years ago. Nine-tenths or more of the total lava had been ejected by 14 million years (Tolan et al. 1989). Three major suites of basalt, the Saddle Mountains, Wanapum, and Grande Ronde (Reidel 1983) make up the basalt beds of the research area. The uppermost basalt bed at all the 27 sites examined and sampled was within the Wanapum basalt group. (See Table 1 for details for each site.) All site samples are located in basalt that has been radiometrically dated at 15.3 to 14.5 million years old.

METHODS

In this research area, 27 satisfactory exposures of the Miocene/Pleistocene contact in road cuts and quarries were located during searches along approximately 2400 km of highways and country roads.

The following criteria were established for each site:

The basalt/loess contact must be clearly visible for at least 50 m.

The location must not have been affected by Missoula Flooding erosion.

TABLE 1. Details of the Research Sites

Formation,[1] Site	Erosion	Sponge Spicules[2]	Unusual Features
1. Sentinal Gap Quarry	none	+	Water reaction products
2. Sentinal Gap Quarry	none	+	Doming of basalt
3. Sand Hollow Quarry	into canyon	nsc	No erosion parallel to canyon
4. Lyons Ferry Quarry	none	nsc	Basalt dips toward east
5. Lyons Ferry Quarry	sheet	+	Smooth erosion
6. Lyons Ferry Roadcut	sheet?	nsc	Gravel between basalt and loess
7. Priest Rapids Roadcut	?	nsc	Massive vesicular basalt
8. Priest Rapids Roadcut	sheet	nsc	2 ash layers between 1st and 2nd basalt beds
9. Priest Rapids Quarry	none	+	1 ash layer between 1st and 2nd basalt beds
10. Priest Rapids Quarry	none	+	Doming of basalt bed
11. Priest Rapids Quarry	none	+	Doming of basalt bed
12. Rosa Quarry	none	+	Site parallel to canyon wall
13. Rosa Roadcut	none	+	Doming of basalt bed
14. Rosa Quarry	none	+	Small questionable site
15. Rosa Roadcut	gully	+	Free floating boulders in loess

TABLE 1. (continued)

Formation,[1] Site	Erosion	Sponge Spicules[2]	Unusual Features
16. Priest Rapids Quarry	sheet	nsc	Eroded dome
17. Priest Rapids Quarry	?	+	Slight irregularities along contact
18. Rosa Roadcut	into canyon	nsc	Erosion probably post-loess deposition
19. Sand Hollow Quarry	sheet	+	Erosion into canyon likely
20. Sand Hollow Quarry	none	+	Quarry cut into canyon wall
21. Sand Hollow Quarry	none	nsc	Basalt bed follows topography
22. Sand Hollow Roadcut, Quarry	none	+	Doming of basalt
23. Rosa Quarry	into canyon	+	Red lava on top basalt bed
24. Rosa Quarry	none	+	Several basalt beds visible
25. Rosa Quarry	into canyon	+	Red lava on top basalt bed
26. Priest Rapids Quarry	none	+	Bedding of loess
27. Priest Rapids Quarry	none	+	Molds of plant roots in loess

1. Formation ages in "millions of years" (from Tolan and others): Priest Rapids and Rosa = 14.5; Lyons Ferry and Sentinal Gap = 14.5+; Sand Hollow = 15.3.

2. nsc = No sample collected at this site

The sites should not be in or affected by modern canyon cutting and stream erosion.

Table 2 illustrates the data sought for each site. Twenty-two loess samples were collected. Loess was not collected at several sites for two reasons. A deep irrigation ditch prevented close access to one site. The loess of several sites with high vertical cliffs could not be reached either from the bottom or the top. When collected, the sample was taken from just above the contact, not from the ground surface at the top of the cliff.

It was originally planned that where possible the surface of the basalt would be excavated for examination. In most cases this was impossible because seldom was the loess above the basalt less than 2 m deep. The amount of excavation to reach the surface of the basalt was impractical. At Site 1 an erosional gully in the loess reached almost to the basalt. Excavation to the basalt surface was achieved. Bulldozing activity or natural erosion at several quarries had removed most of the overlying loess without much disturbance of the underlying basalt. These locations gave opportunity to examine the basalt surface (Figure 2).

Every effort was made to avoid the modern erosion coulees and canyons or those produced by the Missoula Floods, but some quarries had been cut back into the hillside from a stream or river canyon. In such cases it was difficult to determine if the quarry face that ran parallel

FIGURE 2. At Site 26, the equipment is sitting on the smooth, flat surface of the basalt. The loess, visible in the background, has been removed.

TABLE 2. The suite of data desired at each site

DATA SHEET

A. General Information

1. Map location .....
.....
2. Site geology .....
.....
3. Extent of contact exposure .....
.....
4. Unusual features.....
.....

B. Palouse Soils

1. Formation name.....
2. Nature of soil (bedding, burrows, pebbles, etc.) .....
.....
.....

C. Basalt

1. Formation name.....
2. Surface features
   a. Erosion (degree and nature) .....
   .....
   .....
   b. Appearance of surface .....
   .....
3. Nature of basalt
   a. Water reaction .....
   b. Vesicles .....
   c. Phenocrysts .....
   d. Columnar ..... or Entablature .....
4. Thickness of flow (if base visible) .....

D. Photos and Samples

1. Photo of contact .....
2. Photo of surface .....
3. Sample of palouse soil .....
4. Site number .....

FIGURE 3. A quarry (Site 9) showing two beds of basalt and overlying loess. A thin band of gray volcanic ash lies between the top and second beds of basalt.

FIGURE 4. This large active quarry (Site 17) clearly shows the sharp unbroken contact line between basalt and loess.

to the canyon had pushed past the erosion crest of the canyon wall. With one exception, all gully erosion seen at the sites was associated with erosion that was post-loess deposition. The one exception was a road cut in U.S. 12, about 11 km north of Dayton, Washington. The road cut was high on a deep canyon wall, and there was a question as to whether it met the criteria of a suitable site. It had some unique features that will be discussed later.

RESULTS

Fifteen of the 27 sites had sharp contacts exhibiting no or slight erosion (Figures 2-4 illustrate three such sites). In general, the sharp, straight contact between the basalt and the loess was striking. However, the exposed surfaces of the basalts, where visible, did not have a knife

FIGURE 5. A long road cut showing the smooth contact between the basalt and the overlying loess. There was 10-20 cm of gravel between the top of the basalt bed and the loess.

edge of intact basalt, but the surfaces were blanketed with a thin layer of loose, angular basalt rocks usually less than fist size (Figure 5). Five, perhaps six, sites displayed sheet erosion. This could be determined in two ways. First, where the bottom of the top basalt bed was visible, thinning by erosion could be seen by tracing horizontally along the bed. In some cases, the bed, as seen in cross section in an anticlinal dome,

FIGURE 6. Site 5. Sheet erosion that appears to cut down through several basalt beds.

gradually and smoothly feathered laterally on both sides of the anticline, and sometimes the erosion continued cutting down into the lower beds (Figure 6). Second, in cases where the bottom of the top basalt bed was not visible, one could assume that if basalt columns were not at right angles to the top surface of the bed, erosion was the cause, rather than bending or warping of the flow.

Ten sites showed either doming of the basalt, or dipping of the surface. The modern loess topography often matched the doming or slope of the underlying basalt.

Talus from the Pleistocene Missoula Flood erosion and modern erosion and weathering is clearly visible in many areas. No examples of large beds of pre-Pleistocene erosional deposits were seen in the research

1. The Dalles Formation in north central Oregon, the Eagle Creek Formation in the northern Cascade Mountains, and the Ellensburg Formation in south central Washington are massive deposits of conglomerates and breccias. Some of them contain exotic pebbles and boulders from outside the Columbia River Basalt Plateau mixed with volcanic rocks eroded from the Plateau.

area. There are, however, large deposits of such material outside the research area.[1]

As indicated in Table 1, all samples of loess collected showed broken sponge spicules. The source of the spicules is not known, but they are derived from marine sponges.

Normally the loess is homogenous, but closer examination does show some minor bedding. *Occasionally, fist-sized or larger rocks are found free-floating in the loess* (Figure 7).

DISCUSSION

The three issues of greatest interest for this study are the relative lack of gully erosion in the basalt, the cause of the sheet erosion and the origin of the sponge spicules. The questions raised by these observations can be better understood by referring to the following sequence of geological events in the research area:

1. Emplacement of the granite.
2. Erosion of the granite into knobs and hills as seen along the northern edge of the Columbia River Basalt Plateau and in the steptoes protruding above the basalt.
3. Eruption of lava and the laying down of numerous beds of basalt

FIGURE 7. Site 15. Gully erosion of basalt along the edge of a deep canyon. Pebbles and rocks are free-floating in the loess above the basalt.

all the way to the Pacific Ocean off the Oregon and Washington coasts.

4. Occasional thin beds of sediment and minor erosion between basalt beds (Items 3 and 4 would have been contemporaneous).
5. Warping and doming of basalt beds.
6. Sheet erosion on a minor scale (Items 5 and 6 may have been contemporaneous).
7. The laying down of the Palouse loess with fair abundance of sponge spicules.
8. Major erosion from the Missoula Floods.
9. Minor erosion and slumping of Palouse loess due to weathering and modern agricultural activities.

Note that the sheet erosion (Item 6) occurred before the laying down of the loess (Item 7).

Considering the length of time normally calculated to have passed between the laying down of the last basalt bed and the depositing of the loess, erosion should have been profound. Although some erosion exists, it is infinitesimal compared to what should be seen. The uneroded surface of the uppermost bed below the loess appears to repeat what we see in the underlying beds as revealed in the walls of the deeper canyons of the area. In the area of Palouse Falls, which is located in the major Cheney-Palouse erosion tract of the Missoula Floods, several valleys were stripped of the overlying loess, but with little or no erosion of the underlying basalt.

Modern small streams, such as seen in the region today, cut V-shaped channels into the basalt, or if large enough, U-shaped channels with vertical walls. The Pleistocene Missoula Flood cut major canyons. What is the explanation for the sheet erosion? The absence of gully erosion superimposed on the sheet erosion suggests that the sheet erosion was not the result of prolonged weathering. Talus slopes or piles of rock debris resulting from pre-Missoula Flood erosion were not seen in the research area. Apparently these sediments were transported elsewhere. Thus the cause and time of the sheet erosion, though insignificant compared to the erosion expected in 14 million years, remains a question for further research.

The presence of sponge spicules in the loess throughout the research area is an enigma (Table 1). Although freshwater sponges are known, the abundance of the observed spicules and their siliceous composition indicates their sources to have been marine sponges. It seems difficult to postulate a marine origin for the loess. To my knowledge, no marine sediments adjacent to the Columbia River Basalts are likely sources for these organic remains.

CONCLUSION

Examination of the contact between the Miocene Columbia River Basalt and the Pleistocene Palouse loess revealed no gully erosion except for Missoula Flood and modern erosion, both of which were excluded from the study. A thin layer of what appears to be a weathering profile mantles the top of the last basalt flow. Pre-Missoula Flood erosion consisted of a minor amount of smooth, broad sheet erosion, the cause of which has yet to be determined. A lapse of 14 million years between the last lava flow and the deposition of the loess is not supported by the data from this research.

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