

Compelling Data for Common Descent from Matching Redundant DNA Sequences

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1. DEFINITION OF TERMS

- General evolution theory- All life developed progressively. New species were derived from a modification of their ancestors.
- Fundamental evolution theory- All life developed progressively through purely natural processes. New DNA sequences came about by chance mutations.
- General creation theory- An intelligent designer somehow intervened to cause certain life to form on earth.
- Fundamental creation theory- Major groups if not species were independently created by an intelligent designer.
- Theistic evolution or progressive creation theory- Is a mix of the general evolution theory and the general creation theory. All life developed progressively. New species were derived from a modification of their ancestors. An intelligent designer intervened at certain points in time to somehow modify the DNA of old species to eventually produce new species.
- Amino acid
- NA- Nucleic Acid
- DNA- DeoxyriboNA
- Organism- DNA based self-replicator

2. INTRODUCTION

Creation and evolution are the two possible theories involved with the debate on the origins of Biology. Futuyma, a leading evolutionist states in ref. 1, "Creation and Evolution, between them, exhaust the possible explanations for the origin of living things. Organisms either appeared on the earth fully developed or they did not. If they did not, they must have developed from preexisting species by some process of modification. If they did appear in a fully developed state, they must indeed have been created by some omnipotent intelligence". The Theory of Evolution proposes that life evolved over time in progressive stages of old organisms being modified and passing on their genes to their descendants. At points in time, enough variation within a species population develops in a local group such that this local group stops or is unable to reproduce with the rest of the population. These points when a local group forms a new species are the nodes on an evolution tree. This results in the pattern of continuous and connected ancestral lines through time shown in Figure 1. According to evolution this process explains the great diversity of life today. Fundamental Creation involves the intervention of a super natural intelligence at some point in time shown. Figure 1 shows the fundamental creation diagram. Creation events are shown by the dot followed by essentially vertical lines which means insignificant variation over time.

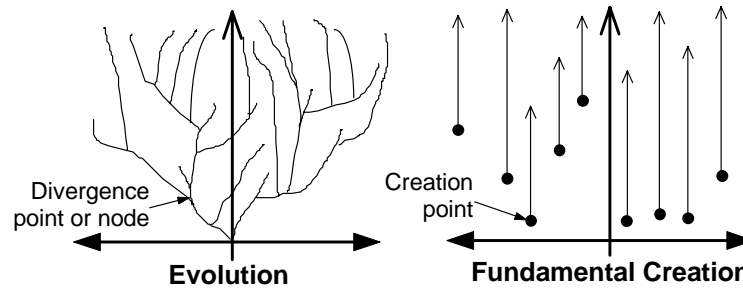


Figure 1 Schematics of Evolution and Fundamental Creation Theories

The evolution theory proposes that all organisms progressively developed by passing on their genes which changed sometimes. The fundamental evolution theory proposes that all these changes occurred by chance with no purpose at all as explained by the leading evolutionist, Richard Dawkins (5).

“All appearances to the contrary, the only watchmaker in nature is the blind forces of physics, albeit deployed in a very special way. ... Natural selection, the blind, unconscious, automatic process which Darwin discovered, and which we now know is the explanation for the existence and apparently purposeful form of all life, has no purpose in mind. It has no mind and no mind’s eye. It does not plan for the future. It has no vision, no foresight, no sight at all. It can be said to play the role of the blind watch maker.”

In contrast, the fundamental creation theory propose an intelligent designer which at least thought out what it takes for an organism to survive, created this organism on earth at a certain point in time. This means that the designer would have had to think of the chemistry which would allow the organism to function and survive and then created the proper DNA sequence which define this organism.

As Figure 1 shows, the evolution theory claims the different organisms are all related to some common ancestor in past history. This makes a prediction that the closer the organisms are related the more they would share in common. The key component that is passed down through the generations is the DNA which defines the chemistry of the organism. This article compares this DNA sequences of Humans, Chimpanzees and Mice and claims that these comparison make a solid and strong case that these organisms share a common physical ancestor as the evolution theory predicts.

3. DNA INFORMATION AND PROTEIN FUNCTION

The DNA sequence is basically a 4 letter language. 4 different nucleotides are used to form molecular chains that make up the DNA. They are adenine (A), guanine (G), thymine (T) and cytosine (C). These DNA chains are the source of information, or the blueprint, that is used to define all the rest of the chemistry that builds a living animal or plant. DNA and RNA are nucleic acids. Proteins are the basic molecules that make up the cells which are the basic building blocks of life. Proteins are a chain of amino acids that are defined by the DNA through a 2 step process. As shown in Figure 2, the first step in the copying process is the transcription process where RNA is formed from DNA. The RNA polymerase enzyme facilitates the transcription of DNA to RNA. Then, as shown in Figure 2 and Figure 3, this RNA is translated into protein molecules. The detail picture in Figure 3 shows how a triplet or set of three nucleotides defines or is translated into a single amino acid which is part of a strand of amino acids which forms a protein. The 20 different amino acids used throughout life are shown in Table 1. The DNA triplets which are translated into the different amino acids are listed in Table 1. The "t" in Table 1 is the "u" in Figure 3.

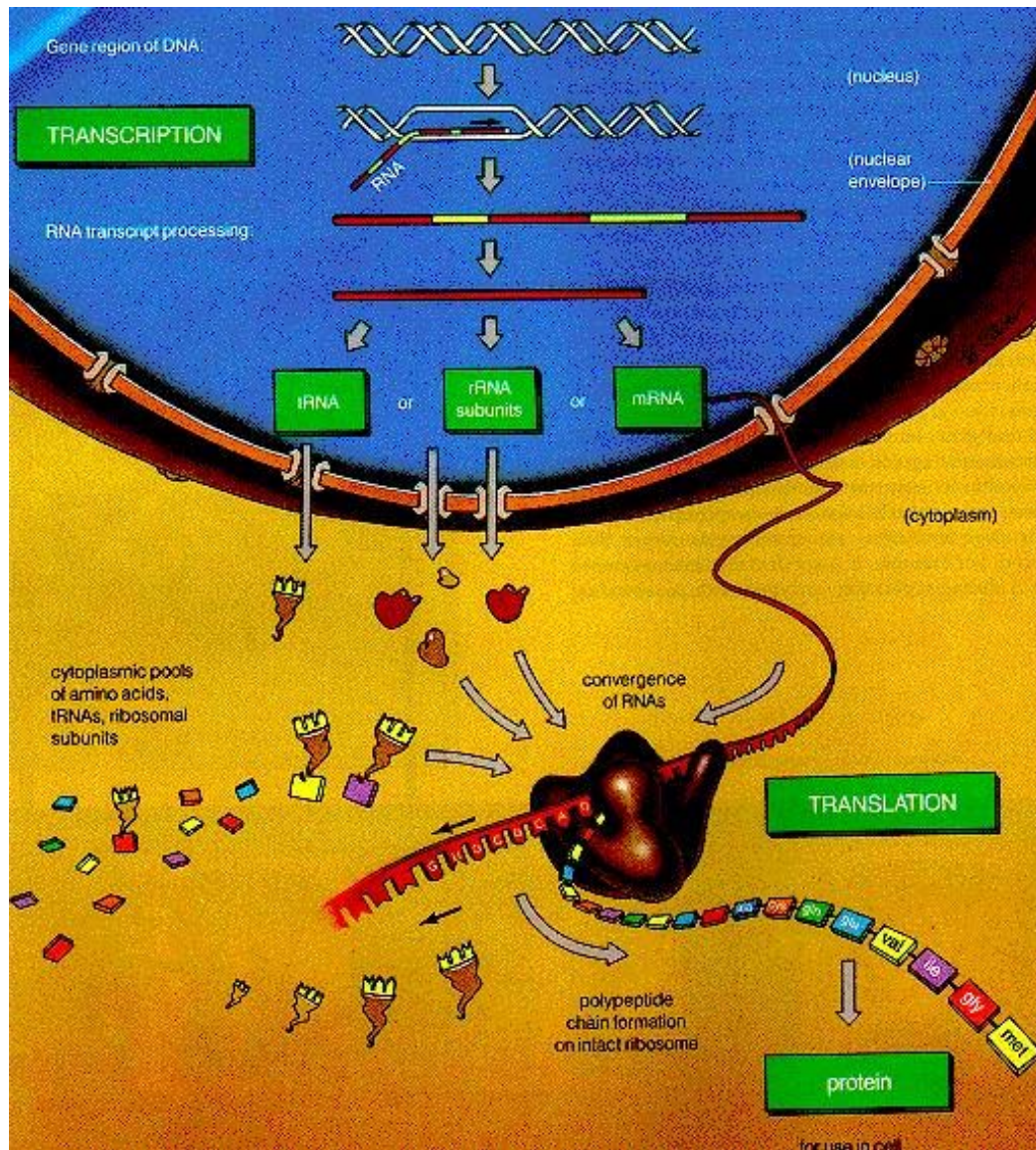


Figure 2 Flow of Genetic Information in Protein Synthesis of Eukaryotic Cells

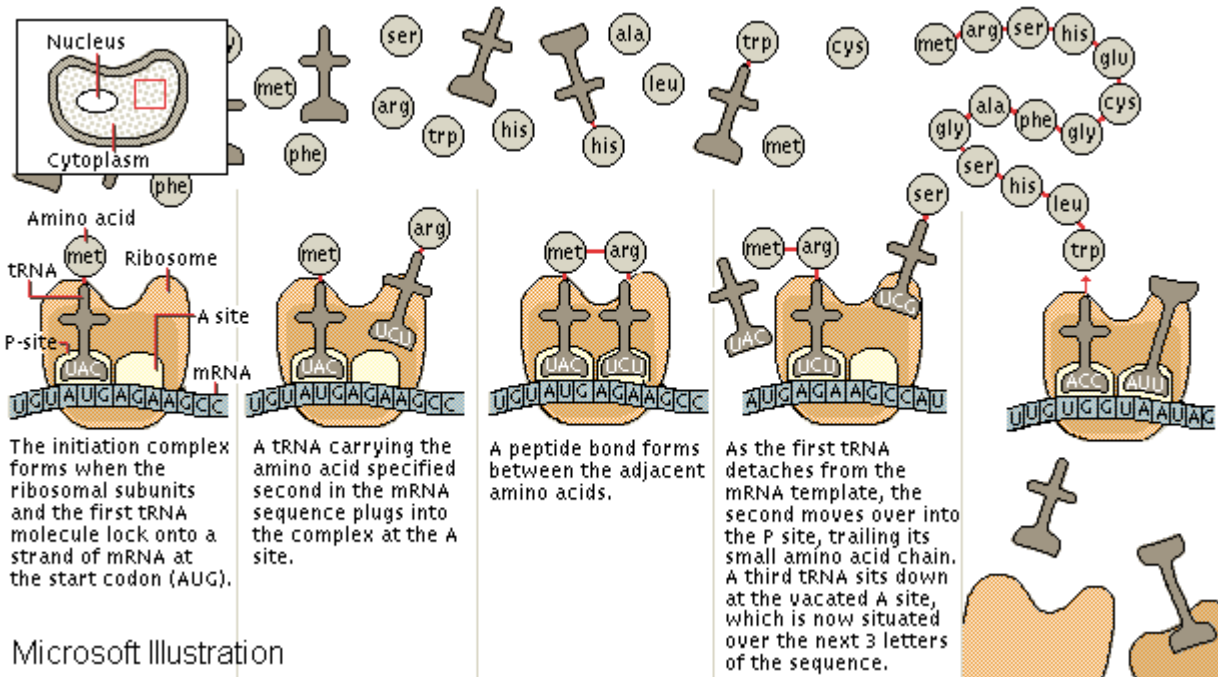


Figure 3 Translation Process of RNA to Proteins

Table 1 AA and DNA relationship

AA Let.	AA Abrv.	# of triplets (d#)	DNA triplets
a	Ala	4	gct,gcc,gca,gcg
c	Cys	2	tgt,tgc
d	Asp	2	gat,gac
e	Glu	2	gaa,gag
f	Phe	2	ttt,ttc
g	Gly	4	ggt,ggc,gga,ggg
h	His	2	cat,cac
i	Ile	3	att,atc,ata
k	Lys	2	aaa,aag
l	Leu	6	tta,ttg,ctt,ctc,cta,ctg
m	Met	1	atg
n	Asn	2	aat,aac
o	Stop	3	taa,tag,tga
p	Pro	4	cct,ccc,cca,ccg
q	Gln	2	caa,cag
r	Arg	6	cg,t,gcg,cga,cgg,aga,agg
s	Ser	6	tct,tcc,tca,tcg,agt,agc
t	Thr	4	act,acc,aca,acg
v	Val	4	ggt,gtc,gta,gtg
w	Trp	1	tgg
y	Tyr	2	tat,tac

The protein molecules make up the structure and perform the functions of the cell. A protein molecule, after being formed, spontaneously folds into a specific 3-D geometry that allows it to perform its specific chemical functions, as shown in the example of a sperm whale myoglobin in Figure 4. The protein molecule is contained in gray. Figure 4 shows how the myoglobin acts on the heme. The 3-D shape matches well with other molecules that it acts on so it can hold them in the proper position long enough to allow for electron transfer to occur to make new chemical bonds. This is the typical function of a protein, to help facilitate a chemical reaction; thus, it is called a catalyst or enzyme. This is accomplished by grooves or cavities in the 3-D structure. Certain amino acids strategically located within the 3-D structure react with other molecules in order to bring the proper change to their chemistry. When not being copied, the nucleic acids, DNA and RNA, also naturally forms a coiled-up 3-D chemical structure. The DNA in storage consist of two complimentary strands that are paired together. These

double strands circle about a center axis to form a double helix. The double helix also naturally forms a coiled-up 3-D chemical structure.

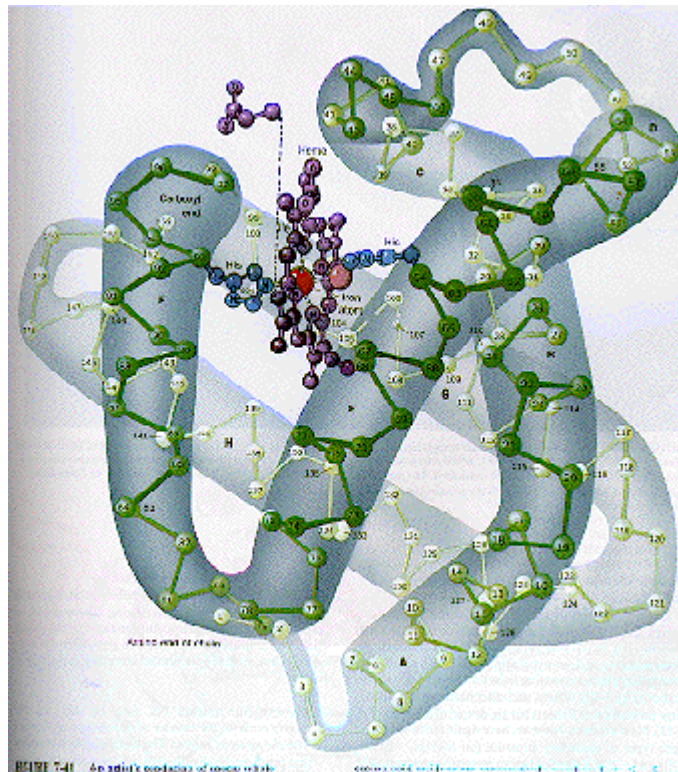


Figure 4 3-D Geometric Shape of Sperm Whale Myoglobin Protein

The DNA sequences are the key to defining the biology of each organism; thus, it is of interest to investigate it's implication concerning the origins debate. To do so the following section describes how relevant probability calculations are made.

4. PROBABILITY FOR MATCHING SEQUENCES

4.1 Method for Calculation Probabilities

In random actions which are repeated, each of the outcomes of the individual actions is supposedly independent of the previous action. For example, the outcome of a dice role is supposedly independent of the previous rolls. Thus, defining an outcome as special because it matches the previous outcome would meet the detachable requirement. For example, it would be appropriate to associate a low probability with a fair dice that kept rolling the same number.

In cases of repeated random actions the appropriate probability to associate with them is the level of success probability. This probability for achieving a certain percent success rate or higher. This section explains how to calculate the probability for a certain level of success occurring by chance through random selection of variables whose different possibilities are all equal. For example, the 6 different possibilities of a fair die each have equal probabilities. The probability of rolling a six on one try is 1/6. The probability of rolling two sixes in a row is (1/6)²=1/36. Calculating the probability for a getting a mix of success and failures is more complicated and requires the formulas below.

Xi: Number of different possible individual outcomes for ith condition

Ni: Number of attempts for ith condition

Si: Number of successes for ith condition

Fi: Number of failures for ith condition (Ni-Si)

ith condition: Condition where one specific outcome out of Xi possibilities occurs Si times out of Ni tries

Li: Total number of possible configurations that satisfy ith condition

Mi: Total number of possible configurations in complete set determined Xi, Ni

Pi: Probability for satisfying the ith condition

C(A,B): number of combinations for a given number of items; A: number of items; B: number of items in each combination. C(A,B)= A!/(B!(A-B)!) where N!=1*2*3...*N

$$Li = (Xi-1)^{Fi} * C(Ni,Fi)$$

$$Mi = Xi^{Ni}$$

$$Pi = Li / Mi \quad (\text{assumes each different possibility of } Xi \text{ has equal probability of occurring})$$

Pi is fundamentally a function of Xi, Ni and Fi.

For example, consider condition 1 (i=1) the case where rolling a fair die 6 times; thus, N1=6. A fair dice has 6 different equally probable possibilities; thus, X1=6. The probability of getting a success rate of exactly 50% or rolling 3 or more sixes (S1=3) is calculated below.

$$L1=(6-1)^3 * C(6,3) = 2500, M1 = 6^6 = 46656, P1 = L1 / M1 = 2500/46656 = 0.054$$

Thus; there is a probability of 0.054 or a chance of 1 in 18.7 that one would roll 3 or more sixes in 6 tries. This is a low number as expected.

To ensure that the calculated probability is not unconservative but accurate for relating to the conclusion one is trying to support, the probability for the level of success (PS) should be determined. Meeting a certain percent success requirement is obtained by achieving the certain percentage or higher. For example, with dice a 50% level of success means that at least 50% of the rolls were sixes. Thus, rolling 3, 4, 5 or 6 sixes out of 6 tries would each qualify as achieving a 50% level of success. The probability for meeting a certain percent success is determined by summing up the probabilities for all the possibilities that have at least the designated level of success. This sum is shown by the cumulative curves in Figure 5 and the formula for calculating the probability for a certain level of success is listed in the following equation.

$$PS = \sum_{F1m=0}^{F1m=F1} P1(X1,N1,F1m)$$

The probability distribution curve in Figure 5 shows relative probabilities of the different possibilities for the different conditions labeled in the graph. These curves were calculated using the formulas above and normalized so that the area under the curve is equal to one so that the sum of the probabilities for all possibilities is equal to one. Notice that that as the number of tries increases the more unlikely it is to get a high percentage of success. In other words, the more times one rolls the dice the less likely a high total percentage of rolled sixes would occur.

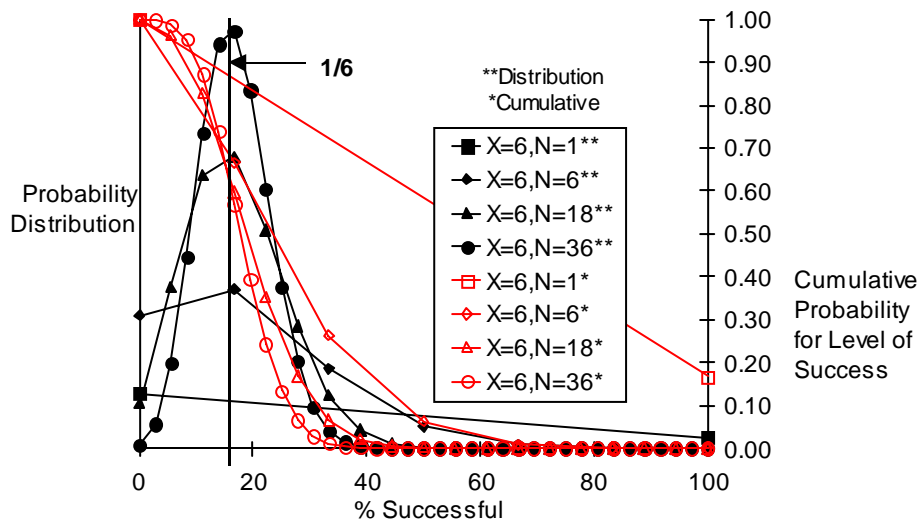


Figure 5 Probability Diagram for X=6

The formula for determining the total probability (PT) for a mix of conditions (n different ones) is listed below. Mixed conditions are where there are variables involved with different X values such as the case where one is rolling dice (X1=6 different possibilities for a dice) and also tossing of coins (X2=2 possibilities for a coin). For example, the formula below could be used to determine the total probability (PT) for rolling at least 2 (S1=2) sixes out of 3 (N1=3) tries and tossing at least 3 (S2=3) heads out of 4 (N2=4) tries. The formula below calculates a probability of 0.023 for achieving this level of success.

$$PT = \sum_{F1m=0}^{F1m=F1} \sum_{F2m=0}^{F2m=F2} \dots \sum_{Fnm=0}^{Fnm=Fn} PTn \quad \text{where } PTn = P1(X1,N1,F1m) * P2(X2,N2,F2m) \dots Pn(Xn,Nn,Fnm)$$

4.2 Neutral Example of Matching Sequences

The buttons on a phone have 3 letters and 1 number as shown in Table 2. Thus, for defining a specific phone number there is a redundancy of three letters but no redundancy of the numbers. Consider that two different people brought to you a sequence of letters which defined the phone number for a pizza store you had asked for. The first person brought kugswcgwrf and the second person brought ktgsxcgwrf. These sequences match at 8 of the 10 sites. However, based on the requirement for performing the same function of defining the phone number 594-772-4963, the letter sequences do not have to match due to the redundancy in how the letters relate to the numbers. Thus, the requirement for accomplishing the same function does not explain the high level of matching (80%) between kugswcgwrf and ktgsxcgwrf. The probability for the sequences to match at this level, when the letters are selected randomly from letters which define the phone number 594-772-4963 ($X=3, N=10, S=8$ then $PS= 0.00340$), is 1 out of $1/.00340$ or 294. Since this is a very small probability, one would not expect it to happen by chance. Rather, one would suspect that something other than chance caused it. The only other plausible explanation is that there was a common source of these two letter sequences. Most likely, this original common source would at least have had the same letters where the sequences do match, k_gs_cgwrf. Based upon this information one would not be sure what the original letters at site 2 and 5 were, but at least in the past one would suspect that a sequence of these letters existed from which both kugswcgwrf and ktgsxcgwrf were derived. Thus, one would at least conclude that the two letter sequences had a common source. Once they diverged from their common source, the letters at site 2 and 5 were changed somehow, but the changes that were passed down still had to perform the function of defining the phone number 594-772-4963. If the letter sequence, while being passed on from one person to the next, got changed to a sequence that did not define the phone number for the pizza store, then it would lose its original function. The person that had it would discover it did not work; thus, would not be interested in passing it on; therefore, the nonfunctional sequences would die out.

Table 2 Number and Letters on Phone Buttons

Number	Letter
2	abc
3	def
4	ghi
5	jkl
6	mno
7	prs
8	tuv
9	wxy

This above example has some important similarities to the theory of evolution. According to this theory DNA sequences are passed down from one generation to the next. Sometimes there are mistakes in the DNA copying process and there is a change in the DNA sequence. If the change does not affect the function of the proteins it defines, then the change does not affect new generations. If the change does affect the organism, then most likely it is a detrimental effect. However, sometimes a functional change can be neutral or for the better. If enough of these changes occur, eventually a new species can develop. By comparing the DNA sequences one could determine the level of matching between different species and investigate if a common ancestor is inferred: just as a common source was inferred in the above example. The next section shows how the conclusion of a common ancestor is inferred when the redundant DNA sequences of mice, chimpanzees and humans are compared.

5. COMPARISON OF MICE, MONKEY AND HUMAN DNA SEQUENCES

5.1 Extron DNA

The cytochrome C protein has been extensively studied and exists in most animals; thus, is a good candidate for evaluation. It performs the function of electron transport involved with oxidative phosphorylation. Studies have been done to determine what different amino acids could exist at the 110 different AA sites in this protein and the molecule still effectively perform the function of electron transport involved with oxidative phosphorylation. Any AA for a certain site is considered functionally equivalent if when it exists at that certain site the molecule still performs its biochemical function. The functionally equivalent AA for cytochrome C according to Ref. 2 are listed in the second column of Table 3 for each AA site # for the cytochrome C protein. Actually, I had to personally consult with the author of Ref. 2 to get the complete list. This list of functionally equivalent AA may not be perfectly accurate. Ref. 2 points out that about half of the functionally equivalent AA sites are known, while the others are theoretically predicted. In fact at sites 8, 76, 79, 93 and 113 of the actual AA in the genome are not even listed as a functionally equivalent AA in Table 3. Thus, the list of functionally

equivalent AA in Table 3 is not perfect, but should still be good enough for estimating the order of magnitude of functionally equivalent sequences occurring by chance. In Table 3, along with the number of redundant functionally equivalent AA (column 3) is also listed the corresponding number of redundant functionally equivalent DNA triplets (column 4). Because in most cases as shown in Table 1 several different DNA triplets define the same amino acids, the number of redundant triplets is greater than the number of redundant AA.

According to Table 3, the level of AA redundancy is quite high. At some places up to 19 of the 20 AA are functionally equivalent. However, at 14% of the sites only one AA could exist at a certain site. These sites must be key locations where the molecule takes an important bend to form its functional shape or it is the part of the molecule that actually interacts to cause the proper electron transfer resulting in the proper breaking and making of chemical bonds. The level of redundant DNA triplets is very high. In some cases 60 out of the 64 possible triplets define a functionally equivalent AA.

The DNA triplets and the corresponding AA in the human (column 5-7) and mice (column 8-10) genes is also listed in Table 3. This genome information for humans and mice comes from Ref. 3 and 4, respectively. It should be pointed out that the chimpanzee AA sequences are exactly the same as the human sequence; thus, it is expected that the chimpanzee DNA sequence will better match with the human sequence, than the mouse sequences do. The DNA site #'s listed in Table 3 correspond to the continuous numbering in Ref. 3 and 4. The last two columns mark if the DNA triplets or AA for the human and mouse genes match for that site. The d# (column 11) is the number of DNA triplets which according to Table 1 define the actual human AA for that site.

Table 3 Cytochrome C DNA and AA Information

AA Site#	Functionally Equivalent AA	Redun.		Human			Mice			Matching		
		AA	DNA	Site #	DNA	AA	Site #	DNA	AA	d#	DNA	AA
4	acdefgiklmnpqrstvw	18	57	1214	tta	l	371	tta	l	6	Y	Y
5	cdefgiklnpqstwy	14	41	1217	gaa	e	374	gaa	e	2	Y	Y
6	acdefghiklnpqrstvwy	19	60	1220	tta	l	377	tta	l	6	Y	Y
7	acdefgiklmnpqrstvw	18	57	1223	aat	n	380	aaa	k	2	N	N
8	acdefghiklnpqrstvw	18	58	1226	atg	m	383	atg	m	1	Y	Y
9	acdegiklnpqrstvw	16	54	1229	ggt	g	386	ggt	g	4	Y	Y
10	acdefgiklmnpqrstvw	18	57	1232	gat	d	389	gat	d	2	Y	Y
11	acdefghiklnpqrstvwy	19	60	1235	ggt	v	392	ggt	v	4	Y	Y
12	acdefghiklnpqrstvwy	19	60	1238	gag	e	395	gaa	e	2	N	Y
13	acdegiklnpqrstvw	16	54	1241	aaa	k	398	aaa	k	2	Y	Y
14	g	1	4	1244	ggc	g	401	ggc	g	4	Y	Y
15	acdegiklnpqrstvw	16	54	1247	aag	k	404	aag	k	2	Y	Y
16	acdegknpqrstw	13	41	1250	aag	k	407	aag	k	2	Y	Y
17	acdefghiklnpqrstvw	18	58	1253	att	i	410	att	i	3	Y	Y
18	f	1	2	1256	ttt	f	413	ttt	f	2	Y	Y
19	cdegiklnpstvw	13	42	1259	att	i	416	ggt	v	3	N	N
20	acdefgiklmnpqrstvw	18	57	1262	atg	m	419	cag	q	1	N	N
21	kqr	3	10	1265	aag	k	422	aag	k	2	Y	Y
22	acgilpstvy	10	39	1268	tgt	c	425	tgt	c	2	Y	Y
23	acdegiklnpstvw	14	46	1271	tcc	s	428	gcc	a	6	N	N
24	acdefghiklnpqrstvwy	19	60	1274	cag	q	431	cag	q	2	Y	Y
25	c	1	2	1277	tgc	c	434	tgc	c	2	Y	Y
26	h	1	2	1280	cac	h	437	cac	h	2	Y	Y
27	agilpstv	8	35	1283	acc	t	440	act	t	4	N	Y
28	acdefghiklnpqrstvwy	19	60	1286	ggt	v	443	gtg	v	4	N	Y
29	acdefghiklnpqrstvwy	19	60	1289	gaa	e	446	gaa	e	2	Y	Y
30	acdegiklnpqrstvw	16	54	1292	aag	k	449	aag	k	2	Y	Y
31	acdegiklnpstvw	14	46	1295	gga	g	452	gga	g	4	Y	Y
32	adegilpstvw	11	40	1298	ggc	g	455	ggc	g	4	Y	Y
33	acdegiklnpstvw	14	46	1301	aag	k	458	aag	k	2	Y	Y
34	dehknqrs	8	24	1304	cac	h	461	cat	h	2	N	Y
35	cegklnqstwy	11	33	1307	aag	k	464	aag	k	2	Y	Y
36	acdegiklnpstvw	14	46	1310	act	t	467	act	t	4	Y	Y
37	agilptv	7	29	1313	ggg	g	470	gga	g	4	N	Y
38	p	1	4	1316	cca	p	473	cca	p	4	Y	Y
39	adegilnpstvw	12	42	1319	aat	n	476	aat	n	2	Y	Y
40	l	1	6	1322	ctc	l	479	ctc	l	6	Y	Y
41	acdefghiklnpqrstvwy	18	56	1325	cat	h	482	cac	h	2	N	Y
42	g	1	4	1328	ggt	g	485	ggt	g	4	Y	Y
43	afilvy	6	21	1331	ctc	l	488	ctg	l	6	N	Y
44	cdefghiklnpqrstvwy	18	56	1334	ttt	f	491	ttc	f	2	N	Y
45	cgilnstvw	9	32	1337	ggg	g	494	ggg	g	4	Y	Y
46	r	1	6	1340	cgg	r	497	cgg	r	6	Y	Y
47	acdeghiklnpqrstvw	17	56	1343	aag	k	500	aag	k	2	Y	Y
48	acdegiklnpstvw	14	46	1346	aca	t	503	aca	t	4	Y	Y
49	g	1	4	1349	ggt	g	506	ggc	g	4	N	Y
50	degknpqst	9	28	1352	cag	q	509	cag	q	2	Y	Y
51	adegilpstvw	11	40	1355	gcc	a	512	gct	a	4	N	Y
52	acdegiklnpstvw	14	46	1358	cct	p	515	gct	a	4	N	N
53	gs	2	10	1361	gga	g	518	gga	g	4	Y	Y
54	defgnpsy	8	24	1364	tac	y	521	ttc	f	2	N	N
55	acdegiklnpstvw	14	46	1367	tct	s	524	tct	s	6	Y	Y
56	hkrswy	6	19	1370	tac	y	527	tac	y	2	Y	Y
57	st	2	10	1373	aca	t	530	aca	t	4	Y	Y
58	acdegiklnpstvw	14	46	1376	gcc	a	533	gat	d	4	N	N

Table 3 (Continued)

Site #	Functionally Equivalent AA	Redun.		Human			Mice			Matching		
		AA	DNA	Site #	DNA	AA	Site #	DNA	AA	d#	DNA	AA
59	agilptv	7	29	1379	gcc	a	536	gcc	a	4	Y	Y
60	imn	3	6	1382	aat	n	539	aac	n	2	N	Y
61	acdegiklnpqrstvw	16	54	1385	aag	k	542	aag	k	2	Y	Y
62	acdegiklnpqrstvw	16	54	1388	aac	n	545	aac	n	2	Y	Y
63	acdefgiklmnpqrstvw	18	57	1391	aaa	k	548	aaa	k	2	Y	Y
64	adegilnpstvw	12	42	1394	ggt	g	551	ggt	g	4	Y	Y
65	acdegiklnpstvw	14	46	1498	atc	i	658	atc	i	3	Y	Y
66	acdegiklnpstvw	14	46	1501	atc	i	661	acc	t	3	N	N
67	flwy	4	11	1504	tgg	w	664	tgg	w	1	Y	Y
68	acdegiklnpstvw	14	46	1507	gga	g	667	gga	g	4	Y	Y
69	acdefghiklnpqrstvwy	19	60	1510	gag	e	670	gag	e	2	Y	Y
70	acdegiklnpstvw	14	46	1513	gat	d	673	gat	d	2	Y	Y
71	cdeghknpqrstvw	14	43	1516	aca	t	676	acc	t	4	N	Y
72	cfgilmstvw	10	33	1519	ctg	l	679	ctg	l	6	Y	Y
73	fhmy	4	7	1522	atg	m	682	atg	m	1	Y	Y
74	acdefghiknpqrstvwy	18	54	1525	gag	e	685	gag	e	2	Y	Y
76	l	1	6	1528	tat	y	688	tat	y	2	Y	Y
77	cegklnpstvw	11	37	1531	ttg	l	691	ttg	l	6	Y	Y
78	den	3	6	1534	gag	e	694	gag	e	2	Y	Y
79	gipstv	6	25	1537	aat	n	697	aat	n	2	Y	Y
79	gipstv	6	25	1540	ccc	p	700	ccc	p	4	Y	Y
80	deknqrs	7	22	1543	aag	k	703	aaa	k	2	N	Y
81	ekn	3	6	1546	aag	k	706	aag	k	2	Y	Y
82	fhly	4	12	1549	tac	y	709	tac	y	2	Y	Y
83	cilmvw	6	17	1552	atc	i	712	atc	i	3	Y	Y
84	glpstv	6	28	1555	cct	p	715	cct	p	4	Y	Y
85	degkns	6	18	1558	gga	g	718	gga	g	4	Y	Y
86	gnpst	5	20	1561	aca	t	721	aca	t	4	Y	Y
87	k	1	2	1564	aaa	k	724	aaa	k	2	Y	Y
88	m	1	1	1567	atg	m	727	atg	m	1	Y	Y
89	agilpstv	8	35	1570	atc	i	730	atc	i	3	Y	Y
90	f	1	2	1573	ttt	f	733	ttc	f	2	N	Y
91	agiptv	6	23	1576	gtc	v	736	gct	a	4	N	N
92	g	1	4	1579	ggc	g	739	gga	g	4	N	Y
93	cfgmstvw	8	24	1582	att	i	742	att	i	3	Y	Y
94	deknqqs	7	20	1585	aag	k	745	aag	k	2	Y	Y
95	acegiklnpstvw	13	44	1588	aag	k	748	aag	k	2	Y	Y
96	acdefghiklnpqrstvwy	19	60	1591	aag	k	751	aag	k	2	Y	Y
97	acdegiklnpstvw	14	46	1594	gaa	e	754	gga	g	2	N	N
98	deq	3	6	1597	gaa	e	757	gaa	e	2	Y	Y
99	cdegklnpqrstvw	14	47	1600	agg	r	760	agg	r	6	Y	Y
100	acdegiklnpstvw	14	46	1603	gca	a	763	gca	a	4	Y	Y
101	dehknq	6	12	1606	gac	d	766	gac	d	2	Y	Y
102	ilv	3	13	1609	tta	l	769	cta	l	6	N	Y
103	ilv	3	13	1612	ata	i	772	ata	i	3	Y	Y
104	acdegiklnpstvw	14	46	1615	gct	a	775	gct	a	4	Y	Y
105	cefgklnpqstwy	13	39	1618	tat	y	778	tat	y	2	Y	Y
106	lm	2	7	1621	ctc	l	781	ctt	l	6	N	Y
107	acdeghiklnpqrstvw	17	56	1624	aaa	k	784	aaa	k	2	Y	Y
108	acdegiklnpstvw	14	46	1627	aaa	k	787	aag	k	2	N	Y
109	acdegiklnpstvw	14	46	1630	gct	a	790	gct	a	4	Y	Y
110	acdegiklnpstvw	14	46	1633	act	t	793	act	t	4	Y	Y
111	acdegiklnpstvw	14	46	1636	aat	n	796	aat	n	2	Y	Y
112	acdegiklnpstvw	14	46	1639	gag	e	799	gag	e	2	Y	Y
113	e	1	2	1642	taa	o	802	taa	o	3	Y	Y

Since the theory of evolution predicts that mice and humans share a common ancestor it is of interest to compare the DNA sequences of the humans and mice to see how much they match. The second to the last columns of Table 3 show that the DNA triplets match at most of the sites, 82 out of 110, while they are only

required to match at AA site #88. This is expected from the theory of evolution, because the mice and humans sequences would have been passed down from some common ancestor in the distant past.

An intelligent designer that understood what is required to make a functional cytochrome C molecule would be aware of the many different possible sequences which would produce the cytochrome C molecule function. Thus, the intelligent designer when selecting the DNA sequence for the cytochrome C molecule be in a position of choosing one sequence from all the many different functionally equivalent sequences. There is no reason based upon biological function for the designer to pick the same sequence as some other organism's cytochrome C molecule. Is it likely that an intelligent designer would by chance select the same sequence as some other organism? Assuming that the level of possible functionally equivalent sequences according to Table 3 are equally probable, the equations presented in Section 4.1 can be used to calculate the probability of the sequences matching by chance.

To reduce the computational effort, the number of possible results for each site (X) are grouped in multiples of 6 under the Cytochrome C AA columns in Table 4. For example, if a site could have 15 different DNA triplets it would be counted in the i=2 (add 1 to N2) group because it rounds down to X2=12. If the DNA triplets matched at that site then 1 would be added to S2. The value for the probability of obtaining the level of success listed for the corresponding set of X's, N's and S's in Table 4 is listed as PT. Use of the rounded down data is conservative because it calculates a higher probability. The PT value of 5.49E-88 for the matching of the DNA triplets is very small. Thus, based upon the assumptions involved with these calculation, the sequences are not expected to match by chance.

Table 4 Probability Calculation Information

i	Cytochrome C AA			Cytochrome C DNA			Histone DNA			Oxidase DNA		
	Xi	Ni	Si	Xi	Ni	Si	Xi	Ni	Si	Xi	Ni	Si
1	6	13	11	1	4	4	1	2	2	1	1	1
2	12	5	4	2	48	40	2	30	21	2	53	34
3	18	7	4	3	7	7	3	6	3	3	25	10
4	24	9	6	4	30	23	4	41	13	4	42	15
5	30	5	4	6	11	8	6	24	8	6	37	20
6	36	7	6									
7	42	26	18									
8	54	18	14									
9	60	8	6									
PT	5.49E-88			1.96E-22			4.98E-5			1.53E-10		

If the assumption of functional equivalent AA is removed there still is a significant level of redundancy because of the multiple number of DNA triplets which define the AA as shown in Table 1. Different DNA triplets can define the exact same protein sequence; thus, definitely the exact same biological function outside of the DNA structure.

Some fundamental creationist could claim that there may be some other function that requires the high level of DNA sequence matching. I believe while the DNA is not being copied it lies essentially dormant in the cell. The DNA structure these triplets form I believe has essentially the same double helix structure. Each codon has the same sugar backbone; thus, there appears no reason for different DNA sequences of the same length to cause any different biological function within the DNA structure when it is not being copied. This article has already shown that there is amino acid sequence redundancy in the function of proteins. Proteins are the key functioning molecules, so if protein sequences have redundancy then it would be expected that DNS sequence have redundancy.

DNA copying occurs during the transcription process, mitosis or meiosis in germ cells. The polymerase emzyne facilitates the reading of the DNA during the transcription process. The polymerase functions to translate many different sequence; thus, it is straight forward to expect there to be no significant difference on the transcription efficiency of the different Cytochrome C DNA sequences defined functional equivalent in Table 6. Meiosis and mitosis involves the replication of all the DNA sequences in the replicating cell; thus, most any sequence can be replicated; therefore, it appears quite ad-hoc to think that different functionally equivalent Cytochrome DNA sequences would make a significant difference on the efficiency of meiosis or mitosis. The high level of matching of the redundant DNA appears to occur throughout much of the DNA. Thus, if there is some functional requirement for this high level of matching it would have to be some general primary phenomenon not just some local or hard to detect secondary effect. Thus, during the copying and non-copying times of the DNA life there appears to be typically no significant functional difference between different functionally equivalent DNA sequences of the same length. Therefore, there appears no good reason to doubt

that the relationship between the DNA triplets and the AA in Table 1 truly represents an essentially redundant relationships.

The redundancy for the DNA triplet to AA relationship in the cytochrome C molecule is shown by the d# column in Table 3. The corresponding set of X's, N's and S's for this relationship is listed under the Cytochrome C DNA column in Table 4. The DNA sets listed in Table 4 are just for the locations where the DNA triplets define the same AA. Locations where the DNA triplets do not define the same AA are left out of the probability calculation because they do not provide a basis for evaluating the redundancy specifically involved with the translation of DNA to AA. For Cytochrome C, 100 of the AA match out of the total 110 sites. The PT value of $1.96E-22$ for the cytochrome C molecule is still very low which still strongly supports the conclusion that the sequences are not expected to match by chance. The Histone DNA set is for a DNA triplet comparison made between the DNA sequence for the human (Ref. 7) and mice (Ref. 8) Histone H4 protein. For this comparison, all of the AA match at the total 103 sites. The oxidase DNA set is for a DNA triplet comparison made between the DNA sequence the human (Ref. 9) and mantled howler monkey (Ref. 10) cytochrome oxidase subunit II (COII) protein. For this comparison, 153 of the AA match out of the total 227 sites. The AA functional equivalence was not know for these molecule so just the basic DNA redundancy probability calculation could be made. Their results are listed in the third and fourth sets in Table 4. Low PT values are still determined. As explained in section 2.2.4 of Ref. 6 probabilities in the range of $1.0E-6$ or lower, I find compelling; thus, based upon this rationale criterion the histone and cytochrome oxidase comparisons also provide strong evidence that humans, mice, monkeys and chimpanzees share a common ancestor. Apparently, since there is higher percent difference for the histone and cytochrome oxidase DNA sequences compared to the cytochrome C sequence, the histone and cytochrome oxidase DNA sequence must have gotten mutated more than the DNA sequence for the Cytochrome C protein. Evolutionist have published vast amounts of sequence comparisons in journals such as The Journal of Molecular Evolution, that they claim consistently support that all organisms share common ancestors.

5.2 Intron DNA

The portion of the DNA that gets translated into the Cytochrome C protein is actually split into two parts along both the human and mice gene. This means splicing needs to be done in the DNA to protein translation process. The place where they are split is at the same AA site # 64 (note that the DNA site #'s jump at this location) which is another match that makes no functional difference but expected from evolution; therefore, this match of the splice location qualifies as more evidence for a common ancestor relationship. The portion that is expressed or translated into a protein is called an exon and the portion that is never expressed is called an intron or junk DNA. The first straight strand of DNA in Figure 2 still contains the introns while the second straight strand does not. An comparison of the mice (104 nucleotides) and human (101 nucleotides) intron is shown in Table 5. Notice that the DNA site #'s for these introns exactly cover the gap in the DNA site #'s in Table 3. The intron comparisons are made by spacing out the individual DNA nucleotides so the matching is maximized. Under this condition there is a large amount of matching for site numbers 1439 and 594 on. 47 of the 64 sites (73%) of the last approximately 65% of the intron. Since the nucleotides are spaced out the chances for matching are increased slightly over the straight forward number of 25%. However, the 73% is still much higher than expected to occur by chance; thus, this high level of matching in this functionless junk DNA is more evidence for a common ancestor relationship.

Table 5 Cytochrome C Intron DNA Information

Human		Mouse			Human		Mouse			Human		Mouse		
Site #	DNA	Site #	DNA	?	Site #	DNA	Site #	DNA	?	Site #	DNA	Site #	DNA	?
1397	a	554	a		1432	a	582	a	Y	1463	t	619	t	Y
1398	a	555	a	Y	1433	g	583	g	Y	1464	g	620	g	Y
1399	g	556	c	N	1434	g	584	g	Y	1465	t	621	t	Y
1400	a	557	g	N	1435	a	585	t	N	1466	g	622	a	N
1401	g	558	g	Y	1436	a	586	t	N	1467	a	623	a	Y
1402	t	559	g	N	1437	t	587	g	N	1468	a	624	a	Y
1403	c	560	g	N			588	c	N	1469	a	625	a	Y
1404	a	561	g	N			589	t	N			626	c	N
		562	g	N			590	t	N			627	a	N
		563	a	N			591	g	N			628	c	N
		564	g	N			592	g	N			629	t	N
1405	c	565	c	Y			593	g	N	1470	t	630	t	Y
1406	t	566	t	Y			594	t	N	1471	a	631	a	Y
1407	t	567	g	N					Y	1472	a	632	a	Y
1408	g	568	c	N	1438	a			N	1473	c	633	c	Y
1409	t	569	t	Y	1439	t	595	t	Y	1474	c	634	c	Y
1410	t	570	g	N	1440	a	596	a	Y	1475	g	635	t	N
1411	a	571	t	N	1441	a	597	a	Y	1476	a	636	c	N
1412	a	572	c	N	1442	c	598	c	Y	1477	t	637	t	Y
1413	a	573	a	Y	1443	a	599	c	N	1478	g	638	g	Y
1414	t	574	g	N	1444	t	600	a	N	1479	c	639	c	Y
1415	a			N	1445	g	601	g	Y	1480	a	640	a	Y
1416	a			N	1446	t	602	t	Y	1481	t	641	t	Y
1417	a			N	1447	g	603	g	Y			642	c	N
				Y	1448	g	604	c	N	1482	t	643	t	Y
1418	a			N	1449	c	605	a	N	1483	c	644	c	Y
1419	c			N	1450	a	606	g	N	1484	t	645	t	Y
1420	a			N	1451	a	607	a	Y	1485	t	646	t	Y
1421	a			N	1452	a	608	a	Y	1486	t	647	t	Y
1422	c	575	c	Y	1453	c	609	t	N	1487	c	648	c	Y
1423	a	576	a	Y	1454	t	610	t	Y	1488	t	649	t	Y
1424	c	577	c	Y	1455	a	611	a	Y	1489	t			
1425	a	578	a	Y	1456	t	612	c	N	1490	g	650	g	Y
1426	a			N	1457	c	613	c	Y	1491	t	651	t	Y
1427	a			N	1458	a	614	a	Y	1492	t	652	t	Y
1428	a			N	1459	g	615	g	Y	1493	t	653	t	Y
1429	t	579	g	N	1460	g	616	g	Y	1494	a	654	a	Y
1430	g	580	a	N	1461	a	617	t	N	1495	g	655	g	Y
1431	c	581	c	Y	1462	g	618	g	Y	1496	g	656	g	Y
										1497	c	657	c	Y

Table 6 shows the estimated levels of junk DNA in a sampling of organisms. For some of the smaller organism all of the DNA is expressed. However, in the larger organisms most of the DNA is not used. For example, in humans only 18% is expressed and in the fritillaria only 0.02% is expressed. These large levels of apparently functionless intron or junk DNA fit well with the evolution hypothesis that organisms collected mutations over many generations, but does not fit well with the fundamental creation hypothesis. Some evolutionist and creationist think that there may be some biological function that the unexpressed DNA performs like providing a certain beneficial molecular structure to the DNA. This may explain some of the junk DNA because there may be some requirement for the junk DNA to be a certain size to make the DNA molecule have a certain molecular shape. However, it is difficult to see how this could explain all the junk DNA for such a range and variation as that in Table 6. In addition, as explained in Section 5.1, the resulting DNA molecular structure is the same for the different nucleotides; thus, there is no reason for there to be a biological requirement for

sequence matching within the junk DNA. Thus, there appears a solid case that the fundamental creation position cannot successfully explain the sequence matching in the junk DNA, but it is just what is expected from evolution.

Table 6 Used and Unused DNA (nucleotides x 10⁻⁹) in Sampling of Organisms

Organism	Total	Used	% Used
bacterium (escherichia coli)	0.004	0.004	100.000
yeast (saccharomyces)	0.009	0.006	70.000
nematode (caenorhabditis)	0.090	0.023	25.000
fruit fly (drosophila)	0.180	0.059	33.000
newt (triturus)	19.000	0.570	3.000
human	3.500	0.630	18.000
lungfish (protoperus)	140.000	1.120	0.800
flowering plant (arabidopsis)	0.200	0.062	31.000
flowering plant (fritillaria)	130.000	0.026	0.020

6. OTHER EXAMPLES OF COMMON ANCESTOR CHARACTERISTICS

Humans have one less chromosome pair than apes. It turns out according to Ref. 21, that the one chromosome (#2) in humans that is quite different from all the ape chromosomes is actually a combination of the two ape chromosomes that are quite different from all the other human chromosomes. The difference between these chromosomes is essentially just their length because the combination of these two ape chromosomes have as a total sequence about the same sequence as the human #2. Thus, it appears the human #2 is an actual combination of these two ape chromosomes implying common ancestors.

Fossils show the bone structure or morphology of extinct species. Of the structures that do perform a function, common characteristics are also found. For example, during the embryo development the bones of the human hand grow out of the same tissue the bones of a bat's wing or a whale's flipper does and they share in their mature form many identifying features (muscle insertion points, ridges). There are many more examples of these sorts of common characteristics, some of them are listed in Ref. 11, 12 and 22. Ref. 18 shows the real difficulty fundamental creationists have in drawing the line between humans and fossil remains of other extinct hominids.

For many of these common morphological characteristics there appears to be no functional requirement for them to be the same. For example, what appears to be the rudiments of legs from an ancient land mammal can be seen in whale skeletons (11,12). This results in multiple amounts of data pointing to the same common descent conclusion resulting in a converging argument for common ancestry. Some creationists claim for these apparently functionless morphological structures that there may be some unknown biological reason that caused the intelligent designer to make the designs similar, or the designer just wanted to keep consistent. One can always appeal to the unknown; however, examples that evolutionists present appear to me to be a strong case for the biological feature in question to truly have no function.

Evolutionists have used all the data from the morphology of ancient fossils and the DNA of modern species to put together a whole history of the sequential development of life. According to evolutionists common characteristics are found at all levels amongst species that are suspected to share a common ancestor. By using objective tools such as cladistics evolutionists have developed phylogenetic relationships or evolution trees.

Comparisons of the morphology of fossils is bound to involve an element of subjectivity. There is no way of measuring the exact distance in strictly mathematical terms based upon morphological differences between two different organisms. DNA sequence comparisons provide the information from which quantitative measurements of the distance between different organisms can be made. This allows for mathematical calculations to be made which provide an objective way to evaluate claims about the relationship of species. Section 5 presents a probability calculation that makes a strong case that humans, chimpanzees and mice share a common ancestor. The example of redundancy in the DNA triplet is a solid case for a common characteristic which makes no functional difference because the basic function that the DNA triplet is involved with is a translation process which can define the exact same protein sequence and function from different redundant triplets.

The evidence for common ancestor appears to confirm that at least the general evolution theory is correct. However, it does not necessarily confirm that the fundamental evolution theory is correct. Obviously, there are differences between species which means that there are protein functions that are different between the species. The fundamental evolution theory claims that all these differences between all species are just a result

of chance from mistakes in the DNA copying process and the purposeless direction provided by natural selection. Scientists have not yet determined if all the differences can be explained by chance. I believe this is done by considering the Markov process (2) which evaluates random walks through the protein function space between the old protein function and the new protein function. For most cases, I believe this takes a lot of work to determine because there are so many variables and unknowns involved. Considering the high level of complexity involved with many of the biological functions in organisms it seems implausible that all of the DNA definition for the chemistry of life could have developed just by chance. Ref. 20 gives some remarkable examples that appear to have irreducible complexity.

7. ATTEMPTS TO DISMISS THE EVIDENCE FROM REDUNDANT SEQUENCE COMPARISONS

7.1 Missing ancient ancestor allegedly implied by lack of modern ancestor

Attempts to dismiss the argument that DNA sequence comparisons imply common descent have been published by critics of Evolution. The most popular one is explained by Denton in Ref. 13 and was used in the popular creation textbook Ref. 14. Denton presents Table 7 of 21 different organisms which shows the percent of the number of AA which are different amongst all of the AA sites in the Cytochrome C molecule for each of these 21 organisms. Table 3 shows that of the 110 AA sites in the Cytochrome C, 10 AA sites are different so Denton's table would report a $100/110=91\%$ value for the human to mouse comparison. The 21 organisms in Denton's table essentially cover the whole range from humans to bacteria. Denton orders the organisms in his table according to the time from the proposed divergence from a common ancestor with the most recent ones on the top of the table and the most ancient divergence at the bottom. Thus, moving up the table means that according to evolution the species are expected to be more closely related and developed from a common ancestor more recently according to evolution. Since the relatively simple bacteria are considered some of the first organisms to evolve and the more complicated humans are some of the most recent, Denton's table provides an opportunity to investigate the trend through time for the proposed sequences of development of organisms through evolution.

Table 7 Denton's Protein Sequence Comparison

Denton acknowledges his table does indicate that the percent differences get smaller the more closely the organisms are related. Denton points out that the general pattern from the sequences indicates the same standard hierarchical topological categories that biologist Linnaeus came up with before Darwin proposed the theory of evolution. For example, within jawed vertebrates the group of terrestrial (land) organisms, amphibians, reptiles and mammals are more closely related than non-terrestrial organisms (fish). Within these groups such as mammals, there are groups of mammals such as rodents or hoofed animals that are consistently more closely related to each other than other groups of mammals. Denton and evolutionists would agree that the DNA sequences imply a pattern which is consistent with the standard hierarchical topological categories. The disagreement comes from Denton's claim that the pattern implies no transitional forms; therefore, the pattern does not indicate evolution.

Denton makes the case for no transitional forms being implied by pointing out that no sequence or group of sequences can be designated as intermediate with respect to other groups. "Of the remaining Eukaryotic cytochromes, ... all exhibit a sequence divergence between 64 and 67 percent." Since all the sequences have about the same difference in this comparison Denton correctly points out that this indicates that none of them stands out as a transitional form, "... It means that no Eukaryotic cytochromes is intermediate between the bacterial cytochrome and the other Eukaryotic cytochromes" Denton goes on to say that this implies there is no transitional form; thus, the "missing links" are truly missing. The fundamental flaw in this argument is that the sequence comparisons made in Denton's table are from modern organisms not extinct ancient ones. The DNA sequences are taken from organisms that are alive today. Evolution proposes the common ancestor of the modern bacterial cytochrome and the other Eukaryotic cytochromes lived hundreds of millions of years ago. This would be some ancient bacteria which diverged from the path that led to the modern bacteria and started the path that led to the other Eukaryotic cytochromes. If this ancient bacteria could be compared to the other bacteria it diverged from then their sequence would be quite similar as Denton expects. The problem is Denton was expecting the modern organisms to have similar sequences which is not appropriate for this case because evolution proposes that the divergence from the bacteria occurred hundreds of millions of years ago. Because of the redundancy in the Cytochrome C AA sequence there is no requirement from biological function that would keep the sequences from changing to some other functionally equivalent sequence. Naturally, the Cytochrome C AA sequences have been continuing to change between all the different species since the time they diverged. Thus, there is no reason to expect any of the modern species compared to the modern bacteria to have an AA sequence that matches more closely to the modern bacteria. Therefore, the reason why Denton

did not find the missing link in his table is because his table only has modern organisms. The transitional Cytochrome C AA sequences if it did exist most likely became extinct hundreds of millions of years ago.

Denton is aware that it is the ancient organisms that are expected to have the most similar sequences, but claims that there is no evidence that this assumption is correct. There is good reason to expect that the more ancient organisms are expected to have more similar sequences. Based on the reasonable assumption that organisms have always developed mutations, it is expected that organisms collected more and more variation over time even if their morphology remained the same over time because of the high level of redundancy in the DNA and AA sequences. Since it is very difficult if not impossible to get the sequences for these ancient organisms because they died out a long time ago, it is not appropriate to expect to study these ancient sequences directly. However, they can be implied. Even though no common ancestor or transitional organism is found in the table, Denton's Table does imply a common ancestor because going up the table the sequences consistently become more similar. Evolution predicts this trend because going up the tables means the proposed common ancestor is more recent. Some creationist would object to this by arguing that this is also expected from fundamental creation because the more similar the organisms the more similar the sequences should be. While this may be true when comparing all the DNA of the different organism; however, there is no biological reason for this to be true when comparing just the DNA sequence for the Cytochrome C protein. As previously pointed out in section 5, many different cytochrome C AA sequence produce the same function; thus, there appears to be no requirement for the designer to specifically make the cytochrome C AA sequence similar. In fact only 14% of the sites are required to be the same according to Table 3.

Denton goes onto to point out that evolution could explain his table of data if there is a sequence change or mutation rate that is constant over time. The theory that mutation rates are fairly constant over time; thus, sequences difference can be used to measure time from divergence has been labeled "molecular clock". Denton points out that the mutation rate is not expected to be constant for the organisms in his table because they involve species with a very large variation of reproduction rates. Denton expects that mutation rates would be related to the number of generations which means that those species which regenerate quickly such as flies will develop mutations in the population in a much shorter amount of time than humans would. Since Denton's table indicates that the mutation rate was constant with time rather than related to the number of generations, he concludes that the data in his table cannot be successfully explained by evolution. It appears to me that evolutionist have not yet figured out the molecular clock. Determining what caused mutations when they occurred and how often is very complicated problem; thus, it is not surprising that evolutionist have not yet developed a mature understanding of how the differences in the sequences came about. However, the determination of common ancestors does not require having this issue be resolved. As explained in section 5 it is possible to infer common ancestors from the similarities in the sequences.

Denton doesn't present or discuss the results of the DNA sequence comparisons. Denton does not inform the reader the level of redundancy involved with the cytochrome C AA or DNA sequences or explain that the sequences in the junk DNA also match significantly. Denton does not evaluate the success of the alternative hypothesis, creation, at explaining the sequence comparisons. Another critic of Denton can be found at Ref. 19.

7.2 Creation of High Level of Matching by Intelligent Designer

Some fundamental creationist could claim that the sequence matching is also expected from an intelligent designer. I think from a fundamental creation position it is appropriate to claim that more similar species would have more similar protein functions, but this does not translate into a requirement for a high level of DNA sequence matching because the same protein function can be obtained by many different DNA sequences. For example, for Cytochrome C there is only one site, #88 where it is required for the codons to match. Thus, there is no biological reason for the sequences to match at such a high level and they are not expected to match by chance. Therefore, the only other reason or hypothesis left for the fundamental creation hypothesis is that the intelligent designer just intended to make them match. But this is clearly ad-hoc because it has the intelligent designer going through the additional unnecessary effort of creating life in such a way to make it look like the DNA sequence for the cytochrome C molecule for humans and mice were both derived from some common sequence implying a common ancestor. But this is the opposite of what the fundamental creation theory proposes, that organisms were created independently. Therefore, proposing such additional effort by the intelligent designer would clearly qualify as ad-hoc as defined in section 4.1.2 of Ref. 6. According to section 4.1.2 of Ref. 6, the rationale approach does not prefer theories that are determined as ad-hoc over other theories that are not ad-hoc. Even the famous Christian philosopher, Francis Schaeffer, did not consider explanation of this type satisfactory, "that God created the fossils in the earth in order to fool fools. This is totally out of character with the God of the Bible" (22). God creating the fossils has God doing extra unnecessary super natural intervention (not mentioned in Genesis) to make it appear that God did not super naturally intervene as described by a straight forward interpretation of Genesis. God creating the unnecessarily high level of DNA

sequence matching has God doing extra unnecessary super natural intervention (not mentioned in Genesis) to make it appear that God did not super naturally intervene as described by the most straight forward interpretation of Genesis.

In addition, the sequences don't completely match; thus, the fundamental creation hypothesis that the intelligent designer intended to make the sequences match is not consistent with all the data. Some fundamental creationists could propose that the sequence variation developed after the organism was created. However, I suspect a significant percentage of the locations in the sequence of humans and chimpanzees that do not match with mice would match in humans and chimpanzees. In the hypothetical example in Figure 6, at site #1 where the human and ape sequence do not match with the mice sequence, the human and ape letter do match. This does not prove but supports the view that "B" or "A" would generally work at site #1 for that protein function in at least most any mammal. Thus, this evidence would support the view that there is no functional requirement for a designer to make "B" occur in both the humans and chimp #1 site. Thus, there appears no good explanation from the fundamental creation hypothesis, but the data is just what is expected from evolution.

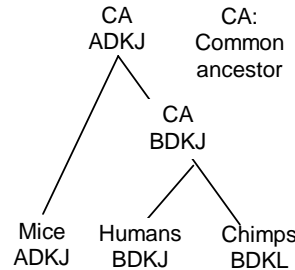


Figure 6 Hypothetical cladogram for the sequences of some protein function for modern species and their common ancestor.

7.3 Summary

A scientific creation argument involves comparing the objective predictions made by the intelligent design hypothesis to the relevant data. Section 5 makes the case that the sequence comparison cannot be successfully explained by the fundamental creation hypothesis because it fundamentally requires an ad-hoc claim about the intelligent designer. Since the DNA sequences are consistent with the expectation for a common ancestor, the evolution theory should be considered as the one of the two possible basic hypothesis supported by the data. Even the author, (Michael Behe, a biochemist) of the book, Darwin's Black Box, which is considered the best book written which makes the case for creation based upon the level of complexity in the chemistry of life acknowledges evidence for common descent. Behe points out that similarities between the mitochondria in modern eukaryotic cells and other bacteria imply that the mitochondria were once single cell bacteria which merged with some ancient eukaryotic cell that formed a multi-cell organism.

8. PHILOSOPHICAL AND PERSONAL REFLECTIONS AND COMPARISONS

It is my hope that there is some good purpose for humans. Also, I hope that humans do have a free will so that they are free to make choices that I hope are motivated by a good purpose. Just because there is strong evidence for evolution being the correct theory, does not make it impossible for evidence to exist that indicates a super natural intelligence intervened to cause humans to exist. However, hoping for something does not make it true.

Chance and the deterministic principles that typically govern the natural world cannot cause a free will. Dawkin's quote in Section 2 points out that there is no intelligent purpose involved with the fundamental evolution process. The strong evidence for common ancestors indicates that at least the general evolution theory is correct; however, scientist have not yet determined if the fundamental theory of evolution is correct for reasons explained in Section 6.

There is some evidence that indicates that more than chance and the natural deterministic laws has caused the universe to result in producing complex organisms on earth. Using the same probability equations that indicates today's species share common ancestors, result in probabilities that also indicate that the super natural has intervened into the universe to cause life to develop by evolution and specifically humans to develop on earth. Ref. 15 presents that the probability of the universe expanding at the rate that allows life to form is approximately 10^{-54} . Ref. 16 claims that the first cell forming a very improbable, implying it was caused by an intelligent designer. As for as I am aware, the lowest probability (when estimated conservatively) associated with any sacred book of any religion is 0.0071. Religions typically claim that there is a supernatural intelligence with a purpose in mind for humans. The conservative probability calculation is presented in Ref. 17.

9. CONCLUSION

The DNA sequences which define the chemistry of life have a high level of redundancy. Even with this high level of redundancy, there is still a significantly high level of matching of the sequences between the different species. Because of this high level of redundancy, there is no requirement for a designer to make the sequences have such a high level of matching. In addition, the level of matching could not be explained by chance. The pattern of matching is just what is expected from evolution; thus, the sequence matching strongly supports the evolution theory over the fundamental creation theory. This matching is found at different level such as at the AA level and the DNA level. This matching is also found throughout different portions of the expressed DNA and even in the intron or junk DNA. Thus, there are converging results that amounts to a compelling argument that at least the general theory of evolution is true.

Just because there is strong evidence for evolution being true, does not make it impossible for evidence to exist that indicates a super natural intelligence intervened to cause humans to exist. By use of the same probability equations in this article that indicate common ancestors, there is also evidence that indicates that more than chance and the natural deterministic laws has caused the universe to result in producing complex organisms on earth. Thus, it appears that neither the fundamental evolution or fundamental creation theory is correct, rather it appears that the correct theory is the theistic evolution or progressive creation..

10. REFERENCES

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