

July 2005

## GUINEA PIGS & HUMANS – WE HAVE A LOT MORE IN COMMON THAN EVOLUTIONISTS WOULD THINK

By Jeff Gift, Ph.D.

Toxicologists like myself make a living out of evaluating the impact of chemical exposures and other insults on the health of laboratory animals (we can't test humans after all). Rats and mice, members of the evolutionary order Rodentia, make up a large majority of these experimental animals. Ken Boschert, a veterinarian with Washington University's division of comparative medicine and the operator of a Web site called Net Vet ([netvet.wustl.edu/](http://netvet.wustl.edu/)) estimates that 99 percent of experimental animals nowadays are rats and mice, which are small, cheap to feed, and reproduce quickly. Rats and mice are also believed to share a closer evolutionary lineage to humans than other non-primate mammals. Yet, another familiar mammal, guinea pigs, are in many ways toxicologically and genetically more like humans than rats, mice and even our closest evolutionary cousins, the chimpanzee.<sup>1</sup> This article will relate evidence from personal experiences and readings that suggest that guinea pigs are more likely to have shared a common designer than a common ancestor with humans.

### **Guinea Pigs and Humans are Resistant to the Toxic Effects of EGBE**

Ethylene glycol butyl ether (EGBE; also called 2-Butoxyethanol) is a clear solvent used in formulating cleaning products (e.g., window cleaners) and protective coatings (e.g., the inside of soda and beer cans). If EGBE were as toxic to humans as it is to most other mammalian species it would likely be banned from use in these cleaning products and in can manufacturing. According to the US Environmental Protection Agency,<sup>2</sup> EGBE causes life threatening hemolysis, the destruction of red blood cells, and other secondary effects in rats, mice, rabbits and, to a lesser extent, monkeys at exposure concentrations not much higher than that which a worker might experience while cleaning the kitchen floor with an EGBE-based cleaning solvent. Yet, humans and guinea pigs appear to be completely resistant to the hemolytic effects of EGBE. A surprising aspect of human and guinea pig resistance to EGBE is that it is not shared by closer evolutionary relatives such as the rhesus mon-



key, indicating that human red blood cells are in this sense more like that of guinea pigs than monkeys and other rodents.

Humans share other unique traits with guinea pigs. Among other things guinea pigs have an immune system that is very similar to ours (see [netvet.wustl.edu/species/guinea/gpmodel.txt](http://netvet.wustl.edu/species/guinea/gpmodel.txt) for a fuller discussion of guinea pigs and their laboratory uses). These similarities suggest similarity between human and guinea pig genes that belie the supposed distance between them on the evolutionary tree. Another strong indication of this surprising similarity comes from studies in the field of genetics of pseudogenes.

### **What are Pseudogenes?**

Pseudogenes are usually regarded as the disabled copies of protein-coding genes. For nearly twenty years, the evolutionist Edward Max<sup>3</sup> has been highlighting pseudogenes as an insurmountably powerful argument for organic evolution and against special creation. According to John Woodmorappe,<sup>4</sup> this argument rests on the truth of the following three premises: 1) pseudogenes lack function, 2) while an Intelligent Designer may plausibly re-use the same designs for functional structures, it is unreasonable to suggest that an Intelligent Designer would create non-functional genes, let alone ones that

share the same lesions from organism to organism, 3) owing to such 'shared mistakes', pseudogenes containing them could not have originated from independent inactivation events that occurred subsequent to the creation, but can only be explained through the common evolutionary ancestry of the organisms that bear them.

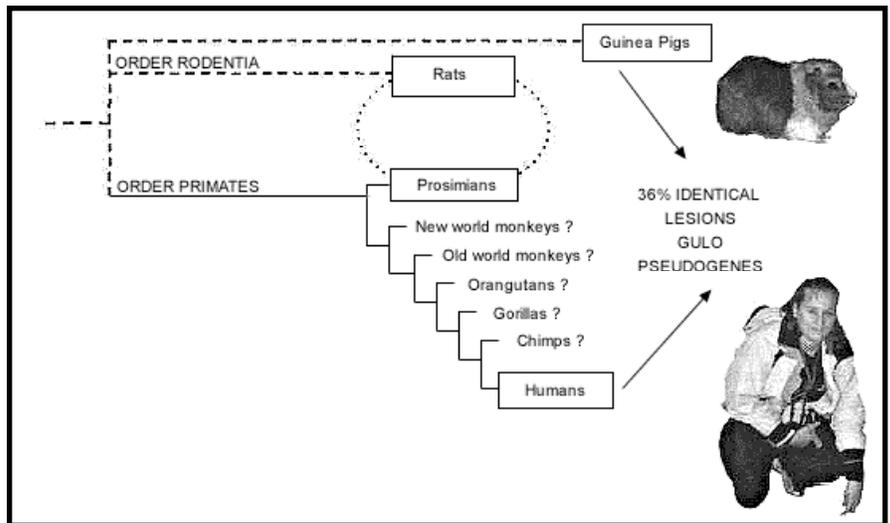
Woodmorappe describes the growing evidence that undermines these premises, particularly the first and third premises. For example, the non-functionality of pseudogenes is not an established fact.<sup>5</sup> There is a growing body of evidence to the contrary. In addition, even if pseudogenes are in fact largely non-functional, lesions within them can originate independently. In his December, 2004 report Woodmorappe focuses on primates' olfactory receptor pseudogenes, the urate oxidase pseudogenes and the gulonolactone (L-) oxidase (Gulo) pseudogenes. (Functional Gulo genes are important for the synthesis of vitamin C.) We will focus on this latter set of pseudogenes because of the discovery that the independently-derived guinea pig and human Gulo pseudogenes have an astounding 36% identical 'disablement' and because Woodmorappe claims that this is "as close as one can get to a resounding disproof of the entire evolutionistic 'shared mistakes' argument."

**The Gulo pseudogenes of Humans and Guinea Pigs: numerous 'shared mistakes' without common ancestry**

Ascorbic acid (vitamin C) is an essential micronutrient that performs a variety of functions in the body. Humans, simian primates (apes and monkeys), and guinea pigs are incapable of synthesizing their own vitamin C and so require dietary sources of this vitamin. The human intake of vitamin C recommended by the Food and Drug Administration is an order of magnitude below the amount synthesized by the vast majority of those mammals capable of producing it.<sup>6</sup>

Many of those mammals found unable to synthesize ascorbic acid have regions of their genome that are believed to correspond to parts of the functional Gulo gene that is found in those mammals found capable to synthesizing Gulo and thus vitamin C. Evolutionists have cited these apparently vestigial remnants of Gulo to make arguments against an Intelligent Designer. However, previous studies of the Gulo gene and pseudogene have focused on those parts of a few exons that appear to correspond between humans and rats. A recent and more comprehensive study<sup>7</sup> discovered an unexpected high degree of identicalness between the 'lesions' of the guinea pig Gulo pseudogene and those of its counterpart

in the higher primates (including humans). Guinea pig and human Gulo pseudogenes show an astonishing 36% identical nucleotide substitutions (relative to the intact rat Gulo gene), despite the fact that the two pseudogenes could not possibly have arisen from a common ancestral pseudogene (see the figure below from Dr. Woodmorappe's report). Furthermore, not only are the inactivations of Gulo in the guinea pig and primates clearly independent events based on the phylogenetic analysis shown in the figure, but also on inferred evolutionistically believed times of inactivation. Other authors<sup>8</sup> have estimated that the guinea pig lost Gulo function less than 20 million years ago. In contrast, the separate inactivation of the Gulo gene in primates allegedly occurred between the time of simian-prosimian divergence (50–65 million years ago) but before the Old/New world monkey divergence (35–45 million years ago).



Dr. Woodmorappe goes on to describe other recent studies that document the astounding similarities between the 'lesions' of the guinea pig Gulo pseudogene and humans. In fact, the similarities he describes suggest that humans are more closely related to guinea pigs than to prosimian primates or to other rodents. (Many scientists argue that guinea pigs are not rodents like rats, shrews and mice.) There is no apparent reason to question the validity of this new information, which certainly seems to falsify the pseudogene 'shared mistakes' argument.<sup>3</sup> Logic now dictates that 'shared mistakes' be approached in terms of parallel mutations rather than common evolutionary ancestry.

**Conclusions**

Here we have seen examples in the fields of toxicology and genetics that contradict the major evolutionary premise that common features and functionality are due to common evolutionary ancestry. Some toxicologists

are beginning to reconsider the broad relevance of rats and mice to humans. For many complex systems such as the hematologic and immunologic systems, there may indeed be a call for use of the more evolutionarily “distant” guinea pig as human “guinea pigs” in laboratory testing. The striking degree of identicalness between the ‘lesions’ of presumably non-functional pseudogenes, unrelated by evolutionary ancestry, clearly dispenses with organic evolution as a necessary explanation for this overall phenomenon. Moreover, it reopens the consideration of such pseudogenes being one-time functional genes that became independently disabled sometime after the Fall. As Dr. Woodmorappe points out, however, much more must be learned about the thousands of pseudogenes in various genomes before detailed generalizations about them can be made in a scientific creationist context. Evolutionary scientists believe that all of science can be understood by examining present processes and this is a worthy quest, but as Christian scholars, we also need to be prepared to accept the possibility that the world and all that is in it has changed for reasons and via processes that may no longer exist and are only known to our omnipotent, omnipresent and omniscient creator God (Job 38:4; II Peter 3:3-6). ❧

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<sup>1</sup> Primates are believed to have made the evolutionary split from rodents about 80 million years ago; humans are believed to have split from monkeys 20 million to 25 million years ago. (Gianaro, C, University of Chicago researchers discovered that humans are a ‘privileged’ evolutionary lineage, [www.eurekalert.org/pub\\_releases/2004-12/uocm-uoc122304.php](http://www.eurekalert.org/pub_releases/2004-12/uocm-uoc122304.php), 28 Dec, 2004.)

<sup>2</sup> US Environmental Protection Agency. (1999) Toxicological Review of Ethylene Glycol Butyl Ether (EGBE), [www.epa.gov/iris/toxreviews/0500-tr.pdf](http://www.epa.gov/iris/toxreviews/0500-tr.pdf)

<sup>3</sup> Max, E.E. (2003) Plagiarized errors and molecular genetics, [www.talkorigins.org/faqs/molgen/](http://www.talkorigins.org/faqs/molgen/)

<sup>4</sup> Woodmorappe, J. Potentially decisive evidence against pseudogene ‘shared mistakes’, [www.answersingenesis.org/tj/v18/i3/mistakes.asp](http://www.answersingenesis.org/tj/v18/i3/mistakes.asp), first published (2004) *Creation Ex Nihilo Technical Journal*, **18**(3): 63–69

<sup>5</sup> Woodmorappe, J., (2000) Are pseudogenes ‘shared mistakes’ between primate genomes? *Creation Ex Nihilo Technical Journal* **14**(3): 55–71

<sup>6</sup> This has prompted controversial suggestions (e.g., Linus Pauling) that humans should take vitamin C at daily gram-level doses.

<sup>7</sup> Inai, Y., Ohta, Y. Nishikimi, M. (2003) The whole structure of the human non-functional L-gulonolactone oxidase gene—the gene responsible for scurvy—and the evolution of repetitive sequences thereon. *J Nutritional Science and Vitaminology* **49**(5): 315–319

<sup>8</sup> Nishikimi, M., Yagi, K. (1996) Biochemistry and molecular biology of ascorbic acid biosynthesis. *Subcellular Biochemistry* **25**:17–39

## MEETING NEWS

Twenty-five attendees at our June 9, TASC meeting were updated by our speaker and Vice Chairman, Dan Reynolds, PhD, on how starlight from distant galaxies could have traveled billions of light years by day four of creation week. Dr. Reynolds reviewed physicist Russell Humphreys’ work on starlight and time by discussing and showing portions of videos titled *Starlight and Time Revisited*, *Black Holes and White Holes*, and *Quantized Red Shifts*. This helped us to understand how millions of light years of time could have passed in the cosmos compared to days of earth time. Dr. Humphreys’ “white hole cosmology” is based on scripture, general relativity, and observational evidence which overturns the underlying assumptions of the big bang model (homogeneity and isotropy). We are grateful to Dan for helping us to understand the concepts involved in starlight and time.

## COMING EVENTS

**Thursday, July 14, 7:30 P.M., Providence Baptist Church, 6339 Glenwood Ave., Raleigh**

View and discuss video, *What Does The Scientific Evidence Say About A Creator?* Dr. Gerald Van Dyke, Professor of Botany at NC State, will lead this discussion. Discover astonishing new evidence for yourself. Best-selling author and former-atheist, Lee Strobel, takes you on a remarkable investigation into how the universe began and introduces you to the Intelligent Design Movement and the mind-stretching discoveries from several scientific fields that present astonishing evidence for a Creator including cosmology, cellular biology, DNA research, astronomy, and physics. Plan now to attend!

**Thursday, August 11, 7:30 P.M., Providence Baptist Church, 6339 Glenwood Ave., Raleigh**

We will complete the viewing of the video, *What Does The Scientific Evidence Say About A Creator?* and Dr. Gerald Van Dyke, Professor of Botany at NC State, will lead our discussion following the video.

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