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Occasional Papers of the BSG

ALL CREATION GROANS: THE PROBLEM OF NATURAL EVIL

PROCEEDINGS OF THE SIXTH BSG CONFERENCE

edited by
Roger W. Sanders

Occasional Papers of the BSG

About the BSG: Founded in 1996, BSG: A Creation Biology Study Group is a fellowship of researchers and scholars who seek to

1. Develop a new view of biology that is consistent with the Biblical record.
2. Encourage high-quality creation biology and baraminology research.
3. Sponsor conferences and other appropriate activities to promote creation biology.
4. Develop a community of creation biologists who share these goals.

BSG membership is open to any who share these goals. For more information, visit the official BSG website, http://www.bryancore.org/bsg/, or email info@bryancore.org.

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Manuscripts should be double-spaced in 12-point Times font. Figures and tables should be included at the end of the manuscript, preceded by a separate section of figure and table legends. Do not integrate figures or legends directly into the text. Title page should include the following items (in order): The title of the paper, the names and affiliations of all authors, and contact information (mailing address, email, phone and FAX number) for the corresponding author. All papers (reviews and research papers) must include an abstract of 250 words or less. References should be made by author-date parenthetical notation and listed alphabetically at the end of the paper (before the figure/table legends). References to books must include pages (e.g. Marsh 1941, p. 100). For reference style, see *OPBSG* #1.

All manuscripts must be submitted electronically to the *OPBSG* editor in one of the following formats: Word, WordPerfect, PDF, or RTF.

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All Creation Groans: The Problem of Natural Evil: Proceedings of the Sixth BSG Conference

Contents

Welcome

On behalf of the Center for Creation Studies and the Department of Biology and Chemistry at Liberty University, I would like to welcome you to the "All Creation Groans: The Problem of Natural Evil" 2007 BSG conference. We are excited to be able to host this interesting conference where creation biologists and others can explore the living world from a perspective that is generally ignored today.

The Center for Creation Studies supports the broader mission of Liberty University which is to "train young champions for Christ." Liberty University is committed to the inerrancy of Scripture including the opening chapters of Genesis. The Center for Creation Studies has a three part mission: teaching, research and outreach.

Our greatest effort is in the area of teaching. We offer two courses on the origins controversy. One is an advanced course CRST 390 which is a small, discussion based course targeted to biology majors and those with a passion for creation. Our other course, CRST 290 History of Life is required of all Liberty undergraduates. It is a comprehensive introduction to the origins controversy from a Biblical creation perspective. Nearly 3,000 students a year take the course in residential and distance formats.

Conferences such as this are so important because they bring people together from various places and a wide array of disciplines. Creation science and creation biology has come a long way in the last few decades. In large part, this is because of the many new people who have gotten involved. Indeed, it is imperative that we encourage more to get involved with creation and help them along the way. As we strive to work together, encourage one another, and respect each other, we can demonstrate to the world what Christian scientists can do.

Again, welcome to Liberty University and Lynchburg, Virginia. I trust that the Lord will use this conference to encourage you in faith and spur you on to greater service for His Kingdom.

David A. DeWitt
Director, Center for Creation Studies
Liberty University

Introduction: Wrestling with Evil

In *Origin of Species*, Charles Darwin tried to confront a culture accustomed to seeing the benevolence of God in creation. He wrote,

> We behold the face of nature bright with gladness, we often see superabundance of food; we do not see, or we forget, that the birds which are idly singing round us mostly live on insects or seeds, and are thus constantly destroying life; or we forget how largely these songsters, or their eggs, or their nestlings, are destroyed by birds and beasts of prey; we do not always bear in mind that though food may be now superabundant, it is not so at all seasons of each recurring year. (Darwin 1859, p. 62)

This was a marked contrast to the rosier view offered by the likes of Paley and Ray. John Ray, for example, found the kindness of God even in lice, which "deterr Men and Women from Sluttishness and Sordidness, and ... provoke them to Cleanliness and Neatness" (Ray 1717, p. 309). Darwin's focus on the unpleasant realities of suffering and death flows naturally from his own life of suffering with a mysterious illness and the loss of several of his children, including his beloved daughter Anne (see Desmond and Moore 1991).

If we creationists intend to offer a meaningful account of creation, we must confront the problem of evil with much greater seriousness than Paley or Ray. Our starting point is a little more realistic than the natural theologians, because we recognize the reality of a fallen, cursed world. This world is not the perfect harmony that God originally created. It has changed, and we believe that change traces back to the Fall of man into sin. To explain the existence of natural evil in the biological world, we must seriously address the questions: How did this come to be so fallen? In what way was the world cursed?

A good place to begin is the Curse itself, as recorded in Genesis 3:14-19. Here we find the origin of painful childbirth, marital strife, hard labor, thorns and thistles, and physical death. Creationists have read much into this brief passage, blaming the curse for predation, parasites, pathogens, and poisons, all with the idea that these things were originally good and were perverted by sin into the harmful and frightening things they are now. What interests me about the Curse though is how it is not just a random selection of punishments. God almost seems to be systematically stymying our ability to obey Him, and thereby love Him (John 14:23). Consider the parallel between His commands and His curses: God commanded, "be fruitful and multiply" (Gen. 1:28). God cursed, "I will greatly multiply thy sorrow... in sorrow thou shalt bring forth children" (Gen. 3:16). God commanded, "have dominion ... over every living thing that moveth upon the earth" (Gen. 1:28). God cursed, "I will put enmity between [the snake] and the woman, and between [its] seed and her seed" (Gen. 3:15). God put Adam in the garden to "dress it and to keep it" (Gen. 2: 15). God cursed the ground, "in sorrow shalt thou eat of it all the days of thy life; Thorns also and thistles shall it bring forth to thee" (Gen. 3:17-18). Death is the ultimate hindrance to our ability to carry out God's commands. It casts its shadow over everything we do, so that even those who manage to obey God in some small way still die. The parallels here do not indicate a haphazard cursing but rather a careful deliberation on the part of God.

What can we make of this? One of creationism's favorite devices for explaining biological evil is degeneration. This is nothing new in the church, as it was a favorite device of even early modern Christians. In seventeenth century England, Matthew Hale thought that degeneration explained how there could be so many different modern animals and yet so little room on Noah's Ark: The modern animals were degenerated forms of the more perfect Ark animals (see Browne 2003). In modern creationism, our first impulse to explain biological evil is usually an appeal to degeneration, namely that some evil phenomenon (e.g. predation, parasitism, etc.) was originally good (e.g. herbivory, mutualism, etc.) and just gradually fell apart sometime after the Fall. Some creationists go so far as to claim that *all* biological changes are inherently degenerative (e.g. Price 1924, p. 108; Bergman 2005). I have also used this degeneration model to explain several biological phenomena (Wood 2001, 2002; Mace et al. 2003).

When I look at the deliberative Curse, it is hard for me to continue justifying this degenerative model, as if God just let things fall apart. To borrow a term, the Curse seems to be a very intelligent design. Other passages of Scripture seem to support a more active role for God in creating natural evil. God sent the plagues on Egypt, including the locusts, flies, lice, frogs, and boils (Ex. 7-13). God sent the fiery serpents to punish the complaining Israelites (Num. 21:6). God feeds the predators (Ps. 104:21). God creates disasters (Is. 45:7). As Kennard (2007)

argues elsewhere in these proceedings, God uses chaos to fight chaos. Maybe predators, parasites, pathogens, and poisons look so beautifully designed because they *are* so beautifully designed.

This is not to say that degeneration should have no place in our studies of natural evil. Rather, we must realize that degeneration is but one possible explanation among several. In some cases, such as the highly degenerated genome of *Mycoplasma genitalium* (Wood 2001), it seems to fit the data well. In other cases, such as the anthrax lethal toxin (Wood 2002) or schistosomes (Mace et al. 2003), degeneration seems much more forced as an explanation. To claim that all biological change is degenerative would certainly be an exaggeration, at least beyond what we have actually tested. The emergence of C_4 photosynthesis, for example, seems to be one example of a nondegenerative change (Wood and Cavanaugh 2001; Wood 2003).

If we admit nondegenerative explanations of biological evil into our theoretical arsenal, what would such explanations look like? I would recommend that we simply try to be creative and see what works. No one has seriously examined Wilson's (2004) dual gene hypothesis (one set of genes for "good" and one set for "bad"), which here he extends to prey defenses (Wilson 2007). Many pathogenic bacteria exhibit pathogenicity islands, where all the genes needed for infection are located. These islands appear to be alien insertions in the genomes where they occur. This should be explored with Wilson's ideas in mind. If these pathogenicity islands are not native to their host genomes, then where do they come from?

Other possibilities include direct creation at the Fall. One of my papers at this conference explores the curious existence of carnivorous plants (Sanders and Wood 2007), which seem not only to resist plants' primordial commission of rendering food to animals, but actually reverse that role and kill animals for food. While the pitcher plants might be explicable on the basis of degeneration, the elaborate snap traps of the Venus Flytrap and the sticky traps of the sundews appear to be marvelous designs for the purpose of catching bugs. There is no variation in the carnivory of the flytrap/sundew family (Droseraceae), which might otherwise indicate an original, beneficent condition. These plants appear invariantly designed to catch bugs. Maybe that's exactly what God intended when He made them.

Overall, I think the prospects for understanding biological evil within creationism are quite good. We already have a strong set of ideas to explain a great number of phenomena, and with a bit of creative work, we should be able to develop and test even more. As we do so, I would hope that we can begin to think clinically about the natural evil of disease. Surely, if we are correct about natural evil, we should be able to develop new and more effective treatments for disease. Perhaps it will be a creationist theory that solves the problem of antibiotic resistance, by proposing a treatment not for the human but for the bacterial pathogen!

Todd Charles Wood
Bryan College

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Wilson, G. 2007. Designed for defense: reptiles and amphibians thwarting predators in a fallen world. *Occas Papers of the BSG* 10:22-23.

Wood, T.C. 2001. Genome decay in the mycoplasmas. *Impact* 340:i-iv.

Wood, T.C. 2002. The terror of anthrax in a degrading creation. *Impact* 345: i-iv.

Wood, T.C. 2003. Mediated design. *Impact* 363:i-iv.

Wood, T.C. and D.P. Cavanaugh. 2001. A baraminological analysis of subtribe Flaveriinae (Asteraceae: Helenieae) and the origin of biological complexity. *Origins* 52:7-27.

Conference Schedule

Unless specified, all meetings will take place in DeMoss Hall 1090.

Wednesday, June 13

8:30 a.m.
Registration opens

9:00 a.m.
Workshop Session 1

12:00 noon
Lunch

1:30 p.m.
Workshop Session 2
See handout for location of afternoon workshops.

5:30 p.m.
Cookout

7:30 p.m.
Welcome and announcements
Roger Schultz
Dean, College of Arts and Sciences
Liberty University

7:45 p.m.
Plenary: "All Creation Groans: A Cumulative Case Theodicy for Natural Evil"
Edward N. Martin
Liberty University

Thursday, June 14

9:00 a.m.
Plenary: "Toward a Pre-Lapsarian and Post-Lapsarian Biblical Philosophy of Nature"
John T. Baldwin
Andrews University

10:15 a.m.
Break

10:30 a.m.
Contributed Paper Session I: Theology

C17. Wise, "The Superiority of a Young-Age Creation Theodicy"
C7. Kennard, "God Uses Chaos to Fight the Rebellion of the Fall"
C3. Davis, "Thorns in the Metanarrative of the Bible: From the Curse of Eden to the Crown of Thorns"
C4. Demme, "Does the Curse on the Serpent in Genesis 3.14-15 Have Direct Implications for Creation Biology?"

12:00 noon
Lunch

1:15 p.m.
Plenary: "Design, Optimality, Goodness"
Paul Nelson
Biola University

2:30 p.m.
Break

2:45 p.m.
Contributed Paper Session II: Design

C16. Wilson, "Designed for Defense: Reptiles and Amphibians Thwarting Predators in a Fallen World"
C6. Gollmer, "Degradation of Design and Anti-Patterns"
C2. Cavanaugh, "A Systems Biology Paradigm for Cellular Pathways and Organismic Populations: Insights from Principles of Systems Engineering"
C8. Kennard, "Potential Evil in the Biblical Account of the Original Good Creation"

4:00 p.m.
Break

4:15 p.m.
Contributed Paper Session III: Biology

C5. Gillen & Hubbard, "Developing *Serratia marcescens* as a Model to Elucidate Aspects of Germ Genesis Exemplified by *Mycobacterium leprae*"
C10. Kim, "Viral Attenuation (Reduction of Pathogenicity) and Its Link to Innate Oncolytic Potential: Implications of a Perfect Original Creation"
C1. Brophy & Kramer, "Preliminary Results from a Baraminological Analysis of the Mole Salamanders (Caudata: Ambystomatidae)"
C18. Wood, "Evidence that Some Toothed Mysticetes are Archaeocetes (Mammalia: Cetacea)"

5:30 p.m.
Dinner

7:00 p.m.
BSG Business Meeting

7:30 p.m.
Poster Session

Friday, June 15

9:00 a.m.
Plenary: "The Origin of Pathogenic Bacteria by Degeneration or Design"
Todd Wood
Bryan College

10:15 a.m.
Break

10:30 a.m.
Contributed Paper Session IV: Faith/Science Integration

C11. Lightner, "Were There Deserts Before the Curse?"
C13. McConnachie & Brophy, "A Biblical Word Analysis for the Landfowl (Aves: Galliformes)"
C14. Sanders, "Taxonomic Distribution of 'Thorns and Thistles'"

12:00 noon
Lunch

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Plenary Abstracts

P1. Toward a Pre-Lapsarian and Post-Lapsarian Biblical Philosophy of Nature

J.T. Baldwin
Andrews University

From a historical/theological *consequentialist* (Russell 2004) understanding of Genesis 1-11, pre-lapsarian conditions represent what might be called a first dominion order (completely foreign in comparison to present biological and geological conditions. The first dominion order was totally good and curse-free. It seems to have been a predation-free habitat (Gen 1:30). If so, the principle was that no animal was to serve as another animal's food. Humans were to be subject to death only upon transgression (Gen 2:17) hence suggesting a causal relation between sin and death. Perhaps the original creation was fundamentally a death free world. If so, God had some wonderful way, unrevealed to us in the Scriptures, of addressing the overpopulation issue in a world populated by living things that procreate. Perhaps decomposers were not needed as they are today? This world changed with the Fall.

A first post-lapsarian divine curse rests on the animal kingdom in the following sense: God cursed the serpent "more than all behemoths" (Gen 3:14). Critical scholars deny that the Hebrew for "more than" (Gen 3:14) suggests that the curse on the serpent included other lower creatures (Hengstenberg 1858, p. 14). However, Genesis 3:1 says that the serpent was "more" crafty than any beast. Here the same Hebrew term is used in a comparative sense. Could the same be said for Genes 3:14? If so, God's creative curse may be genetically retooling the whole biological world. God may have been turning the world into a predation ecology subject to recycling, decomposition, and so on. This would be a temporary knowledge of evil until the restoration in the new creation (Rev 22:3), where this curse is to be reversed or removed.

A second post-lapsarian divine curse rested on the vegetable kingdom. What about plant death? When Genesis 1:29 states that all seed plants are given for food, where do the noxious ones come from? The text infers that expressions of natural evil appear in the form of thorns and thistles following a divine curse (Gen 3:17). The influence of Satan may be at work in the degenerating design seen in this kingdom. Noxious weeds, says Jesus represent the work of the enemy (Matt 13:27-28).

A third post-lapsarian dreadful divine curse rested upon the mineral kingdom at the time of the global *mabbul* (catastrophic destruction by water), when the face of the earth, the crust of the earth, was destroyed and reformed (Gen 6:13; 7:11; 8:21; 9:ll). Probably here we find the origin and first appearance of plate tectonics, earthquakes, volcanoes, desertification, draughts, killer lightening and wind storms.

The curses including things like death, suffering, and predation are reversed in the new creation to the glory of God.

Russell, J.R. 2004. "Eschatology and Scientific Cosmology: From Conflict to Interaction." *CTI Reflections* 8:2-37
Hengstenberg, E. W. 1858. *Christology of the Old Testament* vol. 1. T. & T. Clark, Edinburgh.

P2. All Creation Groans: A Cumulative Case Theodicy for Natural Evil

E.N. Martin
Liberty University

Although the problem of natural evil might be allowed by the Christian theist to count *prima facie* against theistic belief (though this itself should be carefully questioned), the theist can never allow it to count *conclusively*. Thankfully, the theist has a large number of rational, existential, moral/intuitive insights and developed theories that cumulatively form a significant theodicy (justification for God's allowance of evil). But a significant theodicy for which problem of evil, seeing that there are many? Clearly, the problem of evil can be parsed out at these four different levels: Why does God allow *any* evil? Why does God allow the *types* and *kinds* of evil? Why does God allow the *amount* of evil (also here, read *magnitude, intensity, distribution, duration*)? Why does God allow the *particular* evils? I argue that the theist, seeing that there are straightforward answers for level one, is best to focus his energies on theodical levels two and three, and especially level two. For if the theist can, given that Theism (or Christian Theism) is true, provide some not irrational reasons (for all he knows) to think that God allows the types and kinds of evils that he does, then since token evils (level four) fall under types of evils (level two), the theist, by providing justification for types of evils (level two) will have provided some good

grounds for supposing that the good God has a reason for the particular evil God allows or causes (level four). But arguably second-level theodicies (or cumulative theodical suggestions) also bear important relevance to level three theodicies, or, that the former can be extended to have obvious theodical bearing on successfully answering atheistic appraisals of the problem of the amount (or intensity, etc.) of natural evil.

Of particular centrality to any problem of natural evil are a small host of atheistic questions (whether justified or not), drawn mainly from David Hume: Why is there any pain at all? Why didn't God make the world a better place? Why isn't God a massive interventionist? Why does God (or at least God's reasons for allowing evil) remain hidden from us? I propose a set of six theodical suggestions, drawn in some part from contemporary literature, focusing mainly on global goods (goods related to certain types or kinds of evils allowed). These goods may actually obtain by the allowance of evils, or, may be made possible by the same allowance. An important question is whether meticulous providence is true (i.e., whether there is some particular good that God has as a morally sufficient reason for each particular evil he allows). Minimally this point signals that one's own theological orientation will determine the particular topology of the landscape of evils that needs justifying within that system. Maximally this point may signal another reason why not to look to level four theodicy (because there *is* no reason why some particular evil *had to happen as it did, when it did,* in all of its particularity). The six types of theodical suggestion I propose to synthesize are elements drawn from (i) the glory and purpose of God in creating a world including both humans and animals; (ii) the impossibility of a best possible world; (iii) the Fall and the existence of indeterministically free willed finite agents; (iv) the regularity of nature and natural law; (v) the potential soul-making benefits from suffering within both human and animal worlds; and, (vi) the severe limitations in the post-Fall world on our ability to identify the justifying reasons why the hidden God (cf. Is 59:2) allows (specific kinds or amounts of) evil.

P3. Design, Optimality, Goodness

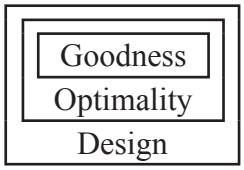
P. Nelson
Biola University

The problem of so-called "natural evil" has been central to the origins debate since antiquity. When considering the birth of modern evolutionary theory in the mid-19th century, many commentators have argued that Darwin's aesthetic and theological revulsion at both the apparent imperfections of organisms, and at their seemingly malevolent "contrivances" (designs), played a key role in his search for a naturalistic account of descent with modification. Nor is this motivation strictly historical: the current work and arguments of such senior evolutionary theoreticians as George Williams (SUNY-Stony Brook) or Jerry Coyne (University of Chicago) are replete with empirical and theological claims about the apparent suboptimality or malevolence of organisms. Their questions are familiar: Why do humans run the risk of choking? Why are our retinas wired backwards? Who designed HIV, and why?

Anyone seeking clear insights when confronted with such questions must be troubled by the conceptual confusions widespread in the literature. Often, naturalistic challenges are posed to a theistic (specifically, Christian and creationist) account of origins, which that account has more than adequate explanatory resources to handle. Eyelessness in cave fauna, for instance, poses no taxing difficulty for a well-articulated theory of creation. In such instances, the challenge reflects little more than ignorance of the opposing theory, or biological naiveté.

In other cases, however, challenges to a creationist understanding of the natural order presuppose a theology that wants careful examination. These challenges typically employ counterfactual or hypothetical (ideal) organisms to establish a metric of optimality, along which real organisms are then located, as putatively suboptimal. These metrics may also presuppose value terms – e.g., "goodness" – whose content must be derived from somewhere other than evolutionary theory itself.

I sort these issues by proposing the nested categories of



I argue that confusion about these categories, their support (or lack of support) in biological evidence, and their roots in science, philosophy, and theology, can lead to a deeply muddled understanding of how theories of creation, and theories of naturalistic evolution, should be weighed. Much of what is currently understood as "evidence" disconfirming theories of creation is not evidence at all. Knowing how to sift observational puzzles into their correct bins – is this *designed*? – is it *optimal*? – is it *good*? (and how one would know in each case) – can do much to relieve misplaced worries about the explanatory strength of theories of creation.

P4. The Origin of Pathogenic Bacteria by Degeneration or Design

T.C. Wood
Bryan College

In *Literal Meaning of Genesis*, Augustine suggested that harmful animals could have originated at Creation as benign creatures that became dangerous after the Fall (Hill 2002, p. 230). Modern creationists widely support this concept, invoking degeneration as the mechanism by which pathogens originate (e.g. see Wood 2001; Gillen 2007). Alternatively, Wilson (2004) argued that many traits associated with pathogenic organisms must have been designed to do harm, although the genes for those traits might not have been expressed until after the Fall. Since the past decade has seen a dramatic increase in the number of finished bacterial genomes in the public domain, I attempted to test the degeneration hypothesis using 486 bacterial chromosomes published in GenBank as of April 5, 2007. I assumed that degeneration would manifest itself as smaller chromosomes, fewer genes, or more pseudogenes in pathogens when compared to nonpathogenic bacteria (e.g. Cole et al. 2001). I first classified the organisms as free-living (189 chromosomes),

opportunistic pathogen (196 chromosomes), obligate pathogen (63 chromosomes), or symbiont/commensal (38 chromosomes). I found that opportunistic and obligate pathogens had significantly smaller chromosomes and fewer protein genes than free-living bacteria. Symbionts/commensals also had significantly smaller chromosomes and fewer protein genes than free-living bacteria. Only obligate pathogens had significantly fewer RNA genes than free-living bacteria. The number of RNA genes in opportunistic pathogens and symbionts/commensals was not significantly different from the number in free-living bacteria. The fraction of coding sequence in opportunistic and obligate pathogens was significantly lower than the coding fraction in free-living bacteria. Surprisingly, the number of pseudogenes in opportunistic pathogens was not significantly different from pseudogenes in free-living bacteria, and obligate pathogens had significantly fewer pseudogenes than free-living bacteria. Thus, my survey of bacterial chromosomes revealed that the chromosome size, gene count, and coding fraction were significantly smaller in pathogens than in free-living bacteria, supporting the degeneration hypothesis. The pseudogene count did not support the degeneration hypothesis. Since the published bacterial genomes are biased towards bacteria of medical, bioremediation, or metabolic interest, I reduced my sample to include only genera that included both free-living and pathogenic (opportunistic or obligate) species. This sample did not reveal a bias towards reduced genomes in pathogenic bacteria. Indeed, in 58% of the pairwise comparisons, the pathogen's genome was larger than the free-living, and in 52%, the pathogen had more protein genes than the free-living. In 56% of the comparisons, the pathogen had fewer pseudogenes than the free-living. The only signs of degeneration that I found were in RNA gene count and coding fraction. In 57% of the comparisons, the free-living bacteria had more RNA genes than the pathogen, and in 35% of the comparisons, the coding fraction of the pathogen was less than the coding fraction of the free-living. I conclude that DNA loss and pseudogenization might not be a major mechanism for the origin of pathogenic bacteria. In at least half of the pairwise comparisons, the pathogenic genomes were larger than their nonpathogenic congeners. This implies that a mechanism of acquisition of DNA, via plasmids or transposons, should be seriously considered as an alternative to degeneration. The smallness of the pathogenic genomes in the larger sample might be accounted for by sample selection bias (e.g. for small genomes of mycoplasmas or rickettsias).

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Wilson, G. 2004. The origins of natural evil. *Occas. Papers of the BSG* 4:8.

Wood, T.C. 2001. Genome decay in the mycoplasmas. *Impact* 340:i-iv.

Contributed Abstracts

C1. Preliminary Results from a Baraminological Analysis of the Mole Salamanders (Caudata: Ambystomatidae)

T.R. Brophy[1] & P.A. Kramer[2]
[1]*Liberty University*
[2]*Independent Scholar*

The mole salamander family (Caudata: Ambystomatidae) consists of 32 extant species in the single genus *Ambystoma* and is widely distributed throughout most of North America (Petranka, 1998; Frost et al., 2006; Pauly et al., 2007). We analyzed a published morphological dataset (Kraus, 1988) using baraminic distance correlation (BDC) and classical multidimensional scaling (MDS) on uncorrected distance matrices. The dataset consists of 32 characters from 14 extant ambystomatids (U.S. bisexual species) and nine extant outgroup taxa (genera from the Dicamptodontidae, Rhyacotritonidae, Plethodontidae, and Salamandridae) but excludes the ambystomatid species from the Mexican radiation (Shaffer, 1984; Reilly and Brandon, 1994; Shaffer and McKnight, 1996). We analyzed the following subsets of data: 1) BDC and MDS on complete dataset (character relevance = 0.85; 21 characters; 3D stress = 0.16); 2) BDC on dataset minus four *Ambystoma* species from the subgenus *Linguaelapsus* (character relevance = 0.85; 19 characters); 3) BDC and MDS on dataset including all *Ambystoma* species, but with *Dicamptodon* and *Rhyacotriton* (the most closely related outgroups according to conventional taxonomy; Tihen, 1958; Sever, 1992; Good and Wake, 1992; Petranka, 1998) as the only outgroups (character relevance = 0.95; 29 characters; 3D stress = 0.09); 4) BDC on dataset minus four *Ambystoma* species from the subgenus *Linguaelapsus*, but with *Dicamptodon* and *Rhyacotriton* as the only outgroups (character relevance = 0.95; 30 characters). A consistent pattern emerges from these various analyses. First, most of the subgenus *Linguaelapsus* displays both internal continuity and discontinuity with other groups in BDC, and forms distinct and well separated clusters in 3D-MDS. Second, most of the remaining *Ambystoma* (all but *A. gracile*) display internal continuity but lack discontinuity with all outgroups in BDC, and form only poorly separated clusters in 3D-MDS. Finally, *A. gracile* is continuous with *Dicamptodon* and *Rhyacotriton* in both BDC and 3D-MDS. An analysis of hybridization within this family reveals 33 unique interspecific crosses. Ten of these confirm the internal continuity of the second *Ambystoma* group in this study and four connect it with the subgenus *Linguaelapsus*. Based on the results of BDC, MDS, and hybridization, we conclude that all *Ambystoma* in this study (with the possible exception of *A. gracile*) represent one monobaramin. Additionally, seven interspecific crosses establish the internal continuity of the Mexican ambystomatids, two connect the subgenus *Linguaelapsus* to the Mexican ambystomatids, and ten connect the Mexican ambystomatids with the second *Ambystoma* group from this study. Based on these hybridization results and the fact that the entire Mexican ambystomatid radiation is thought to be a monophyletic assemblage related to *A. tigrinum* (Shaffer, 1984; Shaffer and McKnight, 1996), we conclude that the entire family Ambystomatidae represents a single monobaramin. The presence of discontinuity below the genus level in this study (between the subgenus *Linguaelapsus* and the other groups) may be due to the nature of the characters selected for analysis. One of the purposes of Kraus' (1988) paper, after all, was to elucidate relationships within the genus *Ambystoma*. This apparent discontinuity may also be due, however, to insufficient numbers of characters in the analyses and/or the potentially close relationship between *Ambystoma* and the outgroup taxa (particularly *Dicamptodon* and *Rhyacotriton*). Future analyses will utilize a dataset containing 115 characters for the same group of taxa, and the potential for different groupings of outgroup taxa (Kraus, 1987).

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C2. A Systems Biology Paradigm for Cellular Pathways and Organismic Populations: Insights from Principles of Systems Engineering

D.P. Cavanaugh
Independent Scholar

Biology and its theoretical apparatus are largely guided by the axiom of materialistic reductionism (Autumn 2002; Auyang 1998; Robert *et al*. 2001); understanding complex systems achieved by breaking them down into smaller objects/components, whose individual explanation lead to the system explanation. The reductionist paradigm now hinders the development of both systems and cellular biology (Autumn 2002; Robert *et al*. 2001; Srere 2000). Reductionist over simplifications is seriously hampering understanding of diversification of biological populations and dynamics of ecosystems (Auyang 1998, Autumn 2002). Aristotle (Dwyer 1999; Lee 2003; Lennox 2000; Srere 2000) put forth the idea of synergy (Corning 1995, 1996, 1998, 2003, 2005; Dwyer 1999; Robert *et al*. 2001) as a paradigm to make sense of the world.

Synergy provides a framework for defining systems biology and for understanding of biological phenomena. Within systems engineering (Fink 1996) and cybernetics (Ashby 1957), a system may be thought of as a synergistic, complex, coadapted, codependent arrangement of objects/components (also subsystems), where the final effect/function/performance greatly exceeds the sum of the parts. System level behaviors result from the information flows (form, fit, and function) and synergistic interactions between corresponding functional components (Fink 1996).

Analogies with engineered software/hardware systems (Auyang 1998; Fink 1996) provide insight into biological systems and a framework for systems biology research; spanning biology from cellular systems, to biological populations, onto entire ecosystems. Problems in control, locomotion, signal transduction and signal processing have similarities to those encountered in hardware, like robotic systems. Systems biology provides a better framework for understanding natural history than atomic level, mechanistic explanations taken in isolation (Autumn 2002). System engineering principles provide tools to investigate the adaptive, feedback control and maintenance of complex systems operating in a robust, dynamic equilibrium within changing environments, accomplished through flows of system signals/information.

Hybrid computing systems are digital (discrete)/analog (variable) in nature. Regulating interlocking cellular metabolite processing control systems and signal transduction pathways represent hybrid computing/control. Metabolites' concentrations are analogous to voltages, and metabolite flow rates through enzymatic processing systems are analogous to current flow. Regulatory control of enzymes may be modeled with analog elements such as transistors, where gain is analogous to enzyme turn over rates, and comparators, where voltage thresholds are comparable to enzyme substrate dissociation constants (50% up progress curve, Lehinger 2004).

Cellular control systems using "random number generation" (thermal noise) for Monte-Carlo combinatorial optimization algorithms (simulated annealing, Cavanaugh 1988) are exemplified by copying errors producing the hyper-variable regions of antibodies through a sloppy DNA polymerase (1:100 to 1:1000, Lehinger 2004). A self directed, constrained DNA hyper-mutation in response to environmental stress or abundance might cause significant variation within basic kinds. Evidence of diversification trajectories have appeared in baraminological studies, providing evidence of ahistorical, perhaps adaptive (epigenetic? Jablonka 2005) morphogenetic systems. Such patterns could be explained by a neo-Lamarckian/systems theory (Lindberg 1998) diversification paradigm, resulting in a successful framework for understanding natural history and relationships among organisms within ecosystems.

Lamarckian vitalism may be understood as the synergistic interaction of interlocking molecular sub-systems maintaining a robust, dynamical equilibrium (Srere 2000). Lamarckian orthogenesis may be understood as behavioral or structural adaptation to ecological environments/niches by self sorting through learned behaviors, imprinting or organismic preferences (Corning 1995; Robert *et al*. 2001) derived from genetic/epigenetic variation, with reproductive isolation from historical contingency/canalization. Orthogenetic variation could arise from environmental feedback (environmental stress or opportunity) stimulating individual morphogenetic systems within biological populations to go to a state of self directed, hyper-mutation of germ line genetic information, resulting in correlated suites of characters achieving either trajectories or "orbits" about Chaotic strange attractors in morphospace.

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C3. Thorns in the Metanarrative of the Bible: From the Curse of Eden to the Crown of Thorns

C.J. Davis
Bryan College

Thorns play a prominent role in the metanarrative of the Bible. They result from the curse of Eden (Gen. 3:18). The וְקֹוץ וְדַרְדַּר generic, "thorns and thistles" of the Old Testament in the Greek translation of the Old Testament (the LXX) become ἀκάνθας καί τριβόλους "thorns and briers." Jesus wore a crown of thorns ἀκάνθῶν ([same word], see Matt. 27:29 and parallels).

This paper argues that God intended a clear connection between Jesus' death and the idea of thorns in the Bible. Beyond the clear linguistic connections above, there are many other texts in the Bible in which "thorn/thistle" appears in prominent theological settings. The burning bush in Exod. 3:2 הַסְּנֶה, is a type of thorn bush. This connection is recognized in the early Jewish interpretation of the passage right down to the present day critical commentaries. Prominent ancient Jewish and Christian interpreters connected Mount Sinai, with this thorn image of the burning bush, and this connection, though the minority view at present, is allowed as one of the two possible etymologies for Sinai by the most prominent lexicographical resources even today (e.g., Koehler & Baumgartner 1994-2000). The Tabernacle is made of acacia wood which is a thorn tree, עֲצֵי שִׁטִּים (Exod. 25:5 etc.). This connection of the Tabernacle and thorns was recognized in antiquity and universally today. The last station of the Israelites before they entered the Promised Land was Abel-Shittim, "the field of thorns" אָבֵל הַשִּׁטִּים (Num. 33:49).

Differing views of Biblical hermeneutics approach the Bible with different presuppositions. This paper will follow the ancient and recent suggestions that a metanarrative approach to the Bible is 1) consistent with the New Testament's usage of the Old Testament, and 2) rightly honors God as the ultimate author of Scripture. In short, Biblical exegesis goes beyond trying to determine what the human author intended for his original audience. Rather, it is legitimate to investigate God's understanding of connections in the Bible. If one supposes that God has a metanarrative reason for so much in the Bible about thorns, very interesting theological ideas come forth.

The following conclusions are suggested: God dwelt in a place of thorns covered with gold to signify His dwelling in the midst of redeemed sinners whose sin is covered by God's grace. The people under Moses, the lawgiver, lived in the field of thorns until Joshua/'Ιησοῦς/Jesus came to underline the idea of the perfection demanded by the law versus the grace offered in Christ's death. The burning bush and perhaps Sinai have "thorn" connections to emphasize the cursing function of the law yet the preserving nature of grace. Jesus wore a crown of thorns to point back to the thorns of Eden. It is possible that Abraham's ram is caught in a thicket associated with thorns. If so, this connection is a further metanarrative connection with Jesus' death.

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C4. Does the Curse on the Serpent in Genesis 3.14-15 Have Direct Implications for Creation Biology?

I. Demme
Independent Scholar

Christian and Jewish interpretations and translations of Genesis 3.14-15 have varied widely, not only with respect to the identity and nature of the serpent, the serpent's offspring, and the woman's offspring, but also with respect to how and to whom the various curses apply. Some see the crawling on the belly and eating dust in verse 14 as an etiology of physiology and behavior for animals of suborder Serpentes (Gunkel 1997, p. 21; Hartley 2000, p. 69), while other interpreters see these terms as metaphors for defeat of a fully spiritual being. (Kaiser 1978, p. 77-78; Waltke 2001, p. 93) Just who it is who does something to whose head, and what he/she/they do is an even older and more controversial question, going back to the translation efforts of the Vulgate, the Septuagint, and the Targums (each showing a very different reading of the Hebrew text).

More important for our purposes in creation biology is the question of whether the hostility placed between the woman and the serpent, his offspring and her offspring, is describing a relationship between humans and animals, or between groups of humans, or between a human and a spiritual being.

Isaiah 11.8 and 65.25 may be interpreting the hostility as one between humans and animals, as does the pseudepigraphal Apocalypse of Moses 10-12 (Life of Adam and Eve 37-39).

Revelation 12 and Romans 16.20, on the other hand, appear to be interpreting the hostility as between the Satan and Mary/Jesus and the Church.

Also flowing into the imagery of Revelation 12 is the well-known Divine Warrior motif which is connected to creation in passages like Psalm 74.14, 89.9-12, Job 9.13, 26.12-13, Isaiah

51.9-10, and to the eschaton in Isaiah 27.1.

Putting all these passages together, it would seem that the immediate message of Genesis 3.14-15 is that creation, which has risen in rebellion against humanity, will be engaged in warfare with humanity until a time when it will be defeated. In the prophetic literature it is made clear that this defeat of rebellious creation will be complete, ushering in a new Eden, and will occur by the agency of YHWH himself. The authors of the New Testament then reveal that it is through the incarnation, death, and resurrection of Jesus that this conquest is engineered, and that the warfare extends beyond that of humans and animals to that between Israel and the Satan.

Given the breadth and complexity of the theological and exegetical issues surrounding Genesis 3.15 and Revelation 12 in particular, biblical creationists may never (this side of the resurrection) reach a consensus on the precise identity of the serpent or the scope of the curse placed upon it and its offspring. At a minimum, however, we can follow the example of biblical authors who see a connection between the hostility between Eve and the serpent and the current hostility between humanity and the animal kingdom and thus gain a better appreciation for the part played by animal life in the redemption of the creation from the Fall.

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C5. Developing *Serratia marcescens* as a model to elucidate aspects of germ genesis exemplified by *Mycobacterium leprae*

A.L. Gillen & R. Hubbard

Liberty University

For creationists, Biblical studies and current microbiological research suggests that factors leading to pathogenicity in bacteria may be attributed to the fall of man and the curse on the original "good" creation that is now undergoing decay. The origin of bacterial diseases is complex and multifaceted and may be explained by a combination of factors including, at least: mutations (deletion, insertions, inversions, translocations), mobile genes, and man's protective defense mechanisms (Wood 2001; Gillen 2007). This paper focuses on the hypothesis that degeneracy (loss mutations) in the metabolic pathways, in part, leads to pathogenesis.

The best example of a bacterium undergoing genomic decay is *Mycobacterium leprae* (Eiglmeier 2001). The leprosy bacillus has a very slow growth rate and is totally dependent upon animal or human cells for survival. Comparative genomics of *M. leprae* with *M. tuberculosis* have revealed that *M. leprae* has lost more than 2000 genes (~25% of its total genome). This discovery has reinforced the evidence that *M. leprae* originated from a *M. tuberculosis* type ancestor but has undergone extensive loss mutations that cause serious energy limitations, leaving only a minimal genome for replication in animal hosts (Seifert and DiRita 2006).

M. leprae is not practical for study in most microbiology labs because it must be grown in armadillos or specialized mice (Tortora *et al.* 2007). In contrast, the bacterium *Serratia marcescens* is easily grown in the laboratory, and may serve as a model for genomic decay leading to pathogenicity. *Serratia marcescens* is noted for the production of a bright red pigment called prodigiosin. It produces a wide diversity of color morphs, depending on the partial or complete synthesis of prodigiosin (Williams 1956, 1973). The metabolic pathways involved in prodigiosin production are numerous and complex, so mutations affecting any of these pathways could result in loss of pigment production (Lim 2003). Most (70-93 %) *Serratia* infections are caused by naturally occurring white mutants, which also often bear antigenic flagella and appear to be more host dependent (Ball *et al.* 1977; Ding and Williams 1983). Williams and Quadri (1980) determined that UV irradiation could transform prodigiosin producing organisms into non-producing white mutants.

Though in the initial stages, we seek to develop and elucidate that research model using *Serratia marcescens*. In preliminary experiments to maximize random mutations correlated with loss of prodigiosin, radiation time influenced mortality and the number of white mutants that grew. When exposed to ultraviolet light for 30 seconds, bacterial mortality was 49% (no white mutants); 60 seconds, 93% (some white mutants); 90 seconds, 93% (more white mutants); 120 and 180 seconds, 96-99% (all white mutants). The degeneracy of prodigiosin production in our preliminary experiments have some similarity (i.e. some UV white mutants have flagella and have lost some metabolic capabilities) to that in clinical strains (933 and WF), suggesting a possible link to pathogenicity. The initiation rate and degree of pathogenicity in the UV white mutants is unknown, but those that develop flagella are more likely to become pathogenic via some undetermined mechanism(s). Future investigations will explore this inverse relationship of prodigiosin production with motility (i.e. flagella), pathogenicity, and host dependency.

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C6. Degradation of Design and Anti-Patterns

S. Gollmer
Cedarville University

Since the fall of creation, it has been in a state of decay. The beauty of the creation is ever present; however, it is diminished by multiple generations of adaptation to a sin cursed world. The prevalence of "evil" in the creation has led some to conclude that living systems are not designed, but are the result of random processes. The challenge for a creationist is to demonstrate good design by a creator in the context of a fallen world.

One means of recognizing good design is through the use of design patterns. Design patterns are best practice solutions to particular computer programming tasks. Although design patterns originated in the context of object-oriented programming, the design goals of modularity, reusability, and robustness are applicable to any complex system of objects that inter-operate in a reliable fashion. Design patterns accomplish these goals by encapsulating behavior, which is most likely to change, into loosely coupled objects. These objects have prescribed interactions, which makes it easier to anticipate behavior when the environment of the object changes. Although computer programs control the interaction of objects through sequential operations, well-defined interfaces, and message passing, the encapsulation of behavior in biological systems is not as easily achieved. However, encapsulation is prevalent in biology as illustrated by the variety of organs and tissues of an organism and the organelles and metabolic pathways in a cell.

If design patterns embody good design practices for complex systems, then anti-patterns embody design practices that fight against the goals of modularity, reusability, and robustness. One way an anti-pattern can arise is from the repeated use of a single solution to solve a variety of problems (aka. To a hammer, everything is a nail syndrome.) Also monolithic systems that can not be easily decomposed into simpler objects manifest another anti-pattern structure. When these two anti-patterns, as well as others, are present in a complex system, the system can manifest unpredictable behavior when changes are made.

The goal of this paper is to identify the presence of design patterns and anti-patterns in biochemical systems. Beginning with a portion of the carbon-utilization regulon in *Saccharomyces cerevisiae*, a search is made for matches to design patterns. With each match, an evaluation is made to determine the completeness of the pattern implementation. In the case of incomplete implementation, a conjecture is made as to possible complete implementations, which infer that the current implementation is a degraded form of a design pattern. When enough data are gathered, a comparison will be made between potential degraded design patterns and anti-patterns to determine any differences and similarities. The observed differences between these patterns will provide a framework for distinguishing between inherent bad design and good design in a fallen world. In addition, with knowledge on how good design is degraded, it may be possible to anticipate how good design can possibly be restored.

C7. God Uses Chaos to Fight the Rebellion of the Fall

D. Kennard
Bryan College

The sovereign God fights chaos with chaos by instituting futility within the created order, as evident in the divine oracles of judgment (Gen. 3:14–19; Rom. 8:20–21). This futility draws upon the chaos of hostility, intensification, and confusion of categories already present within the creation account to permeate and frustrate all relations in judgment. One of the main contributions of this paper is to: 1) identify the chaos metaphors apparent a) in the creation account and b) in ancient Near Eastern mythology to 2) show how they get appropriated within God's judgments. These chaotic metaphors are corralled in God's good creation but now let loose as an evil to deal with human sin. This futility leaves the blessings (be fruitful, multiply, fill the earth, subdue, and rule) intact, but now mingles the blessings with the experiential knowledge of evil, such as increased pain in childbirth and increased effort in accomplishing labor (Gen. 1:28; 3:5; 9:1–7). Likewise, humans still retain the initial tasks like cultivating the ground but now in a manner that subjects them to futility with thorns and thistles and much labor (Gen. 3: 17–19, 23). Darkness and the deep house many of these chaos actors, like Leviathan (Gen. 1:21; Ex. 7:9-12; Deut. 32:33; Neh. 2:13; Job 7:12; Pss. 74:13; 91:13; 148:7; Isa 27:1; 51:9; Jer. 51: 9; Lam. 4:3; Gunkel 1895, pp. 171-398, esp. 379-98; Lambert and Parker, 1966; Jacobsen 1976, pp. 167-91; Deiterich 1891, pp. 117-22; Bousset 1906, pp. 350-56; Fontenrose *et al.* 1959, p. 210; Aune 1998, pp. 670-93; Lucan 1685, sect. 60.5.79-81; Lucian 1882, sect. 300.9) or the one who sheds blood (Gen. 9: 5-6; Ex. 21:28-29; Num. 21:6-9; Deut. 32:24; Isa 18:6; Jer. 15: 3; 16:4; 19:7; 30:6; 34:20; Ezek. 14:21; Micah 5:7). Animals running about carnivorously limit and thwart the control humans bring to the creation (Gen. 9:5-6; Ex. 21:28-29; Num. 21:6-9; Deut. 32:24; Isa 18:6; Jer. 15:3; 16:4; 19:7; 30:6; 34:20; Ezek. 14: 21; Micah 5:7; cf. Kennard 2007). Even the extreme measures of God utilizing the waters of chaos to attack the sin-dominated condition of the earth kills nearly all but leaves the remnant as preserved in blessing and work (Gen. 9:1–7, 20).

One of the most devastating features of this chaos futility is that of death. Soul (*nephesh*) is synonymous with life (Brown *et al.* 1953, p. 659), so it is chaotic that souls die. God warned Adam (Gen. 2:17). God's oracle of judgment brought death (Gen. 3:19), which reigned and imputed death to humans, further dominating man by death (Rom. 5:12–21; Eph. 2:2–3).

Animal death begins with human death at the fall (Gen. 3: 15, 21 and parallel with Eccl. 3:18-22 and Gen 2:17 and Rom 8:20; Terreros 1994, 2003, pp. 150-75). The oracle of judgment indicates that within the hostility between serpents and humans that both shall kill the other (Gen. 3:15). For example, the bite of Egyptian adders kill Israelites in the wilderness until God treated it by the bronze serpent (Num. 21:6–9). Likewise, humans crush the heads of such serpents (Gen. 3:15; Von Rad 1972, p. 87; Westermann 1974, pp. 237-8; Skinner 1930, pp. 71-4, 79-82; Speiser 1986, pp. 25-28; Wenham 1987, pp. 72-3, 78-9; Bauer 1957, pp. 612-13; Jewett, 2007, p. 994; Freedman

and Simon 1977, Gotsmich 1941, pp. 844-79; Malaise 1972, pp. 179-80; Siculus 1976, sect. 17.100.8). Ecclesiastes compares human and animal death to be alike in bringing a release of their spirit (*ruḥ*/רוּחַ), bringing a vain end to their body in the decaying process (Eccl. 3:18–22). Additionally, animals died for God to make clothes from animal skins to better cover the humans than the leaves did, though nothing is said about this being a sacrifice (Gen. 3:21). With the global flood all souls, including animals, are killed in the global flood except those in the ark (Gen. 7: 21–23). As a chaos metaphor, death is used by God in judgment. God fights chaos with chaos in the creation.

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C8. Potential Evil in the Biblical Account of the Original Good Creation

D. Kennard
Bryan College

Four examples demonstrate the thesis that God includes potential evil within His good creation.

1) God creates a tree of the experience of good and evil within His good creation that when its fruit was appropriated initiated evil consequences (Gen. 1:12; 2:9; 3:6-12; Num. 24:16).

2) Some animals likely have carnivorous tools (teeth and claws) before the Fall, after the Fall, and into Kingdom. God created the animals as within the good creation (Gen. 1:12; 2: 9; 3:6-12) and blessed them with vegetation as food. Many of these described animals become carnivorous with no Biblically described modification process. That is, the same terms are used to describe them pre-fall as post-fall as in Kingdom. Such a continuity of term would imply a continuity of defining traits like: claws, teeth and strength pre-fall as in post-fall as in Kingdom**.** For example, "beasts" (*bhmh*/בְּהֵמָה; Gen. 1:24-26) includes carnivorous animals like lions destroying flocks (Micah 5:7; Isa. 30:6; Pr. 30:30) and eating dead as covenant curse (Deut. 32:24; Isa. 18:6; Jer. 15:3; 16:4; 19:7; 34:20). Ezekiel calls these beasts, which God uses in judgment, *evil* (*hraym*/ הָרָעִים ; Ezek. 14:21). As a term, "beasts" (*bhmh*/בְּהֵמָה) even includes poisonous serpents (*nahash*/נָחָשׁ below; Isa. 30:6; Num. 21:6-9). Ultimately, these same animals with no described modification (except an exclusion of carnivorous activity) are incorporated within Kingdom (Isa 11:6-9).

3) Great Sea Monsters (tanînm/תַּנִּינִם) are threatening as a category of animal, including allusions to carnivorous activity, and at times mythological chaos monsters, as indicated through a word study of *tanînm*/תַּנִּינִם (Gen. 1:21; Ex. 7:9-12; Deut. 32: 33; Neh. 2:13; Job 7:12; Pss. 74:13; 91:13; 148:7; Isa 27:1; 51: 9; Jer. 51:9; Lam. 4:3; Gunkel 1895, pp. 171-398, esp. 379-98; Lambert & Parker 1966; Jacobsen 1976, pp. 167-91; Deiterich 1891, pp. 117-22; Bousset 1906, pp. 350-56; Fontenrose *et al.* 1959, p. 210; Aune 1998, pp. 670-93; Lucan 1685, sect. 60.5.79-81; Lucian 1882, sect. 300.9).

4) The tempter serpent (nahash/נָחָשׁ) is a venomous snake, which will crawl on its belly, not a satan (Gen. 3:1-5, 13-15; Num. 21:6-7; Deut. 8:15; Pss. 58:4; 91: 13; 140:3; Prov. 23:32; Ecc. 10:8, 11; Jer. 8:17; Amos 5:19; 9:3; 1 Tim. 2:14; 2 Cor. 11: 3; Rom. 16:20; Jn. 2:11; 6:64; 8:25, 44; 15:27; 16:4; 1 Jn. 1:1; 2:7, 13-14, 24; 3:8, 11; Rev. 12:9; 20:2; Von Rad 1972, p. 87; Westermann 1974, pp. 237-8; Skinner 1930, pp. 71-4, 79-82; Speiser 1986, pp. 25-28; Wenham 1987, pp. 72-3, 78-9; Bauer 1957, pp. 612-13; Jewett 2007, p. 994; Beale 1999, pp. 661-2; 1 Macc. 3.22-3; 4.10, 30, 36; 5.7, 21, 43; 7.42-43; 8.4-6; 9.7, 15-16, 68; 10.52-53, 82; 13.51; 14.13; Wisdom of Solomon 2: 24; Josephus 80, *Antiquities* 1.45-50.4). No Biblical text places Satan in the Genesis 3 account; instead the Satan tradition comes from Zoroastrianism and Jewish syncretism (Russell 1977, pp. 207–9; Westergaard 1993, *Zendavesta Th.* 3, pp. 54-55, 62; Charlesworth 1983, *The Life of Adam and Eve* 33; 1 Enoch 69:6). *Serpents* (*nahash*/נָחָשׁ) in Mosaic theology of the Pentateuch are: a) affirmed as good channels of the work of God (like a blessing to

Dan, Moses rod, and the healing bronze snake), or b) threatening crawling animals (Gen. 3:1, 14; 49:17; Ex. 4:3; 7:15; Num. 21: 6-9; Deut. 8:15; 2 Kgs. 18:4; 1 Cor. 10:9). Evil is not invading on the back of one greater than man, but rather a subordinate snake is rebelling against man. "Serpent" as a snake whose bite is deadly (Gen. 3:15; Num. 21:6-9) raises the possibility that God created venomous serpents.

Therefore, actual evil of the fall and of post-fall animal structure and behavior is potential within God's good creation and the pre-fall condition.

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C9. Reovirus: Orphan Virus and Its Implication in Original Creation

L. Kim

Independent Scholar

Due to an evolutionary perspective, viruses are largely considered to be selfish parasites, propagating more viruses in order to assure survival, and to serve no beneficial purpose. Creationists have suggested that viruses originally were created to have a benign or beneficial function (Bergman 1999, Francis 2003, Gruenke *et al*. 2004, Kim 2006). Thus, it is of great interest that recent studies clearly show REOviruses (Respiratory Enteric Orphan viruses) as having a potential innate beneficial oncolytic role in humans. The term "orphan virus" means a virus that is not associated with any known human disease.

Reoviruses are ubiquitous, non-enveloped viruses containing 10 segments of double-stranded RNA (dsRNA) as their genome, and common isolates of the respiratory and gastrointestinal tract of humans, but they are not associated with any known diseases and are therefore considered to be benign (Tyler, 2001). Rosen *et al*. (1963) concluded that reoviruses possibly play an etiologic role in the generation of some minor respiratory/enteric illnesses, but in general reovirus infections are asymptomatic. Hashiro *et al*. (1977) was able to demonstrate that certain virally and spontaneously transformed murine cell lines were susceptible to reovirus infection, whereas normal human and subhuman primate cells, primary mouse cells, normal rat kidney cells, and baby hamster kidney cells were not. Duncan et al. (1978) also found that normal and transformed cells exhibited different sensitivities to reovirus infection, with cytopathology observed only in the transformed cells and not in normal cells, which nonetheless produced virus for a sustained period. Collectively, these observations suggested that reovirus infection efficiency is somehow linked to the transformed state of the cell. When cells are transfected with oncogenes such as Ras, Sos, and v-erbB, they became susceptible to reovirus infection (Strong and Lee 1996; Strong *et al*. 1998), indicating an oncogenic Ras signaling pathway is exploited by reovirus. Because hyper-activating mutations of the proto-oncogene Ras occur in about 30% of all human tumors (Bos 1989), the orphan virus is used in clinical trials as a powerful anti-cancer agent against Ras oncogenic tumors (Norman *et al*. 2005). In addition, because reovirus, a double-stranded RNA virus, is an efficient inducer of type I interferon, it is likely that a host-interferon response also plays an important role in reovirus oncolysis in vivo (Steel and Cox 1995; Steel and Hauser 2005).

Initially classified as orphan virus, the reovirus is, however, not entirely benign to animal models. Several recent studies showed that reovirus caused so-called "black feet syndrome" in immunecompromised animals (Loken *et al*. 2004; Kim *et al*. 2007a, 2007b, 2007c). Because cancer patients often undergo immune suppression due to heavy chemo/radiation treatments or advanced tumor progression, this viral pathogenicity can be a major hurdle in reovirus-anticancer therapy. However, a genetically attenuated reovirus variant derived from persistent reovirus infection performed a potent anti-tumor activity with a significantly reduced viral pathogenesis in immune-compromised animals (Kim *et al*. 2007a, 2007b). Importantly, reovirus attenuation does not affect its viral oncolytic potential while significantly reducing viral pathogenesis *in vivo*.

Creation biology studies are needed to understand the relationship among native vs. attenuated reoviruses, their respective hosts, their genetic structures and expression, and their mode of transfer. Once this is achieved, reoviruses can be developed as a model to understand, in part, how pathenogenic viruses originated.

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C10. Viral Attenuation (Reduction of Pathogenicity) and Its Link to Innate Oncolytic Potential: Implications of a Perfect Original Creation

L. Kim
Independent Scholar

Creation biology must grapple with the relationship of virus as beneficial genetic entity, 'pathogenic unattenuated selfish DNA/RNA genetic entity,' and benign/attenuated genetic entity. Many studies have clearly shown viruses play an important role in the ecosystem (Bergman 1999; Francis 2003; Gruenke *et al.* 2004; Lucas and Wood 2006) and function to maintain the normal host physiology, including apoptotic induction (necessary for the development of the immune system and turnover of epithelia cells like those found in the skin and gut) (Stanier 1979, 385–387; Braithwaite & Russell 2001; Clarke *et al.* 2005; Laine *et al.* 2005), host immune activation (Steele and Cox 1995; Al-Sheboul *et al.* 1996), differentiation (such as placenta differentiation; Rote *et al.* 2004). Presumably, these would have been the major functions in a pre-Fall world. Because genome replication and transcription of most viruses lack proofreading potential, mutation rates can be 1000-fold higher than in DNA-based genomes (Doming *et al.* 1995; Wells *et al.* 2001; Gay *et al.* 2006). And as mutations are accumulated in viral genomes during replication, viruses lose their biological functions resulting in defective viruses (Ahmed and Fields 1981), suggesting genetic integrity is necessary for their normal biological functions. Thus, it is likely that altered viruses derived from original viruses, in some cases, may be or become pathogenic, which is probably a post-Fall phenomenon. Therefore, comparisons of pathogenic viruses and closely-related attenuated strains, which can be induced by targeted mutation or deletion of the viral virulent genes (Kirn 2001; Thorne *et al*. 2005; Kim *et al*. 2007a, 2007b), may suggest genetic causes of pathogenicity.

Many naturally attenuated viruses have been shown to specifically target cancer cells while sparing normal counterparts, which ultimately led to the use of these viruses in clinical trials as potent anti-cancer agents (Duncan *et al.* 1978; Nemunaitis 1999; Ring 2002; Varghese and Rabkin 2002; Everts and Poel 2005; Norman and Lee 2005; Roberts *et al.* 2006; Kim *et al.* 2007b). Two dominant models attempting to explain oncolysis have been proposed as follows: 1) Oncolytic viruses utilize abnormal cellular signaling pathways only developed in cancers during carcinogenic progression in order to actively replicate and lyse them. Carcinogenesis is a multi-step process involving the accumulation of mutations of normal proto-oncogenes and tumor suppressor genes. Interestingly, oncolytic viruses exert enhanced viral tropism toward cancers cells containing accumulated carcinogenic mutations. Examples include: reovirus vs. cells with hyper-activating mutations in the Ras proto-oncogene (Coffey *et al.* 1998; Takai *et al.* 2001; Duursma and Semin 2003; Norman and Lee 2005; Kim *et al.* 2007;), myxoma (rabbit poxvirus) virus vs. various human carcinogeneses, especially oncogenic Akt signalling pathways (Lun *et al.* 2005; McFadden 2005; Wang *et al.* 2006), and adenovirus vs. tumor suppressor (p53, RB) defective cancer cells (Bischoff *et al.* 1996; Carroll *et al.* 1999; Heise *et al.* 2000; Morris 2002). 2) Oncolytic viruses utilize faulty interferon response pathways only developed in cancers during carcinogenic progression. The low pathogenicity of oncolytic viruses is due in large part to intact anti-viral responses to interferons, which confer protection on normal tissues (Stojdl *et al.* 2000). Examples include: Vesicular stomatitis virus (VSV) from insects and livestock vs. various human tumors (Stojdl *et al.* 2000, 2003; Ahmed *et al.* 2004; Ebert *et al.* 2005), measles and vaccinia virus vs. various human tumors (Nakamura and Russell 2004; Heinzerling et *al.* 2005; Thorne *et al* 2005).

Because the presence in the pre-Fall world of both tumors or pre-tumor activity and viral oncolysis is unlikely, post-Fall viral oncolytic activity probably arose as a phenotypic extension or transfer of normal viral activities (possibly like tissue apoptosis) of pre-Fall beneficial genetic entities. However, oncolysis presents intriguing variation to the biology of such entities. To use attenuated oncolytic virology in creation modeling of the origin of pathogenicity, first, direct comparisons of the genomes of oncolytic attenuated viruses and related non-attenuated virulent

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C11. Were There Deserts Before the Curse?

J.K. Lightner
Independent Scholar

Genesis documents that God created everything very good (Genesis 1:31). Since deserts can be very hostile toward life, this brings up the question as to whether or not desert biomes existed prior to the Curse. It would seem that their existence would be inconsistent with the initial very good creation. There is other scriptural evidence that supports this view as well. First,

the sparseness of life in the desert seems inconsistent with the directives to fill the earth as well, as with the declaration that God intends the earth to be inhabited (Genesis 1:22, 28; 8:17, 9:1, 7; Isaiah 45:18). This is particularly so because God is associated with life and is known for his abundance (Deuteronomy 30:20, John 1:4; 10:10; 14:6, Psalm 36:7-8, Ephesians 1:8, 1 John 3:1).

Secondly, prophetic literature describing judgment often refers to desertification and areas becoming uninhabited, at least by humans (Isaiah 13:19-22; 34:8-15; Jeremiah 4:26-29; 50: 12-13; 51:43; Ezekiel 29:8-12; Hosea 2:3; Zephaniah 2:8-15). Additionally, thorns (קוֹץ, qôṣ) and thistles (דַּרְדַּר, dardar) are mentioned as effects of the Curse (Genesis 3:17-18, Kohlenberger and Swanson 1997). The Hebrew for thorns occurs 11 more times in Scripture and refers to something that grows in the desert (מִדְבָּ ר, midbâr, Judges 8:7, 16, Kohlenberger and Swanson 1997), easily catches fire (Exodus 22:6), is often intentionally burned (2 Samuel 23:6-7; Isaiah 33:12) and burns quickly (Psalm 118:12). The word thistle (דַּרְדַּר, dardar) only occurs one other time, again with thorns (קוֹץ, qôṣ), in a passage relating God's impending judgment on Israel's sin (Hosea 10:8). Never is either word used with a positive connotation (Isaiah 32:13, Jeremiah 4:3, Ezekiel 28:24).

Finally, pre-Curse conditions are inferred from prophetic literature concerning a future restoration. A passage detailing healing and safety for the redeemed also describes the desert blooming and being filled with abundant water and plant life (Isaiah 35:1-10, מִדְבָּ ר, midbâr appears in verses 1 and 6; other Hebrew words describing deserts occur too). This transformation of the desert appears in several other passages as well (Isaiah 32:15; 41:17-20; 44:3; 51:3; 55:12-13). Scripturally, water is associated with life and with God (Jeremiah 2:13, John 4:10, 13-14; 7:37-38; 1 Corinthians 10:3-4; Revelation 21:6; 22:1, 17).

Objections could be raised that post-Flood, especially post-Mosaic, contexts apply only narrowly. On this basis, one might posit that deserts (and even thorns) were created outside of Eden or that man and animals originally had wider tolerances and were more able to cope in arid environments. However, most desert creatures today are very specialized compared to closely related non-desert species, presumably those in the same baramin. Similar specialization within a monobaramin has been documented (Lightner 2007). This implies that species adapted (in the sense of having ecologically specialized traits) to live in the desert were derived from creatures that were not so specialized. If, indeed, deserts give a physical representation of judgment that follows sin, then these desert-adapted species may be a picture of God's grace and provision even in adverse circumstances. More research on the biological and paleontological implications of this view would be helpful.

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C12. Endogenous Retroviruses: Remnants of Germline Infection or Created in the Cell?

Y. Liu
Maranatha Baptist Bible College

The genomes of all vertebrates and humans harbor multiple copies of endogenous retroviruses (ERVs), DNA sequences that have genes and gene organizations homologous to those of retroviruses. While some ERVs are expressed and some even assembled into intracellular viral particles, most of them are deficient and are rarely transmitted horizontally. Conventional biologists have developed the endogenization theory from an evolutionary perspective to explain the origin of ERVs, i.e, they diverged from exogenous retroviruses that infected the host's ancestors, inserted into their germlines, and then degenerated. Under old earth assumptions, the best evidence, among others, for the endogenization theory is that modern exogenous viruses can infect the germline and be inherited like Mendelian genes (Jaenisch 1976).

However, several facts about ERVs suggest that young-earth creation models of their origin can be developed, which may give us insights into the origin of viral diseases and other genetic issues. 1. Endogenization of modern exogenous retroviruses is rarely observed in *nature*. 2. Most modern ERVs are not actively replicating even within the cell. At least all human ERVs appear fixed in numbers and positions; although some mouse ERVs are capable of expanding in the host genome (Cooper and Hausman 2004, p. 225). Are the human ERVs older, therefore more degenerated and less active? If the human race is indeed younger than the murine race, there is no reason to suppose that the human ERVs are older than those of the mouse. 3. Xenotropic ERVs reside in cells that have no receptor for them (Coffin, 1996). 4. Essential beneficial functions of some ERVs and irreducibly complex coordination between ERVs and host DNA sequences argue against the possibility of historical acquisition of ERVs followed by positive selection (see below).

Some ERVs are expressed at certain stages of the host's lifetime to the benefit of the host. Recently, there has been much investigation on the function and regulation of syncytins, which are the *env* proteins of human endogenous viruses (HERVs) (Frendo *et al*. 2003; Mallet *et al.* 2004). Syncytin-1 has been shown to be critical for the formation of syncytiotrophoblast and its secretion of human chorionic gonadotropin (hCG) (Frendo *et al*. 2003). Efficient tissue-specific expression of syncytin-1 requires cis-acting elements in both the ERV and sequences (including an enhancer) upstream of the ERV (Prudhomme *et al*. 2004). Furthermore, expression of syncytins is under the control of host sex hormones. If the syncytins are indeed essential for human reproduction, they appear to be components of an irreducibly complex system that have to be created together to perform the intended function (Okulicz and Ace 2003). Recently, ERV *env* genes similar to syncytins have been shown to be essential for placenta development of sheep (Dunlap *et al*. 2006).

These data are consistent with an assumption of a recent creation. At least some ERVs must have been incorporated into the initial design of eukaryotic life. The degenerative nature of

most mutations argues against the evolution of infectious viruses (especially complex retroviruses) from ERVs. Exogenous viruses might have been created simultaneously with their endogenous counterparts during the creation week. Transmission and propagation of infectious retroviruses among the host population could have helped in maintenance of the endogenous viral sequences via recombination, in a way similar to recombinational DNA repair and modern gene therapy.

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C13. A Biblical Word Analysis for the Landfowl (Aves: Galliformes)

M. McConnachie & T.R. Brophy
Liberty University

The Old Testament Scriptures are a valuable source of baraminological information, both in setting apobaraminic limits and suggesting the rate/mode of post-Flood diversification (Wood, 2002). We performed a biblical word analysis as part of a larger baraminological study of the landfowl. The landfowl include such well known birds as chickens, turkeys, pheasants, grouse, quail, partridges, and peacocks. We first compiled a list of 31 words that had the potential to be found in the English Old Testament. Next, we used *Bible Gateway* (http://www.biblegateway.com) and the *University of Virginia's Electronic Text Center* (http://etext.virginia.edu) to locate specific uses in the following translations: KJV, NKJV, NASB, NIV, and RSV. Finally, we used *BibleWorks* (2003), three concordances (Strong, 1984; Goodrick and Kohlenberger, 1990; Kohlenberger and Swanson, 1998), and several lexicons (Brown et al., 2005; Koehler and Baumgartner, 1958; Holladay, 1974; Tregelles, 1979; VanGemeren, 1997) to verify our understanding of the original Hebrew/Aramaic words. Seven of the 31 words are found in at least one English translation. These are used 94 times and correspond to 16 Hebrew/Aramaic words. Eighty-one occurrences come from Hebrew/Aramaic words (*'ôp*, *'ôpā*, *ṣippôr*, *ṣippar*, *'ayiṭ*, *yâqûš*, *yqš*, *yâqôš*) that refer to a general fowl/bird category or to birds of prey, and are therefore of little value in delimiting landfowl baramins. Seven occurrences come from Hebrew words (*barbur*, *tinšemet*, *'ls*, *tukkiyyîm*, *motnayim*, *zarzîr*) for which translation differences exist in the English text. In most of these cases, the lexicons are also uncertain of translation, and generally give multiple possibilities. Six occurrences, however, probably refer to members of the landfowl order. The Hebrew word *qōrē'*, found in both I Samuel 26: 20 and Jeremiah 17:11, is translated "partridge" in all English versions. All lexicons agree that *qōrē'*, named for its call, is usually translated "partridge". Aharoni (1938) identifies this bird as *Ammoperdix hayi* (sand partridge), which is native to the Dead Sea region of Palestine. In I Samuel 26:20, *qōrē'* is hunted in the mountains. Similarly, sand partridges are game birds frequently found in hilly regions. Jeremiah 17:11 refers to the tendency of two sand partridge females, one of whom is eventually displaced, to lay their eggs in a single hole. The most interesting references to landfowl in the Old Testament, however, come in connection with God's sending of quail to the Israelites in the wilderness (Exodus 16:13, Numbers 11:31-32, Psalm 105:40). The Hebrew word *śᵉlâw* is translated "quail" in all English versions. All lexicons agree that *śᵉlâw*, named for its fatness, is translated "quail". Four of the lexicons (all but VanGemeren, 1997) refer to this bird as *Coturnix*. Large flocks of quail (*Coturnix coturnix*) still migrate north over the Red Sea and arrive at the Sinai Peninsula after wintering in Africa. Such flocks are frequently so weakened after this journey, that they fall to the ground in exhaustion and can easily be caught by hand (Meier, 1991; Klemm, 1993). In God's timing and by His direction, quail were sent as both an expression of God's graciousness (Exodus 16) and wrath (Numbers 11) towards the Israelites (Kiuchi, 1997). Psalm 78:26-30 does not specifically mention quail, but clearly retells the Numbers 11 account of God's judgment. These events suggest that the migratory habits of *C. coturnix*, which descended from some other species on the Ark, were fully established within one millennium after the Flood (Ussher, 1658; Dryer, 1983). Although interesting and inherently edifying, none of these Old Testament references are useful for setting baraminic limits in the landfowl.

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C14. Taxonomic Distribution of "Thorns and Thistles"

R.W. Sanders
Bryan College

Conventional theory holds that plant thorns and similar structures evolved in ancestors of lineages ranging from single species to whole families. In these lineages, mutations arose and were selected to allow structures to be modified as piercing weapons, as follows: entire stems (thorns), the leaf or leaf parts (spines), or epidermis (prickles and stinging hairs). Accordingly, the selective pressure is protection from herbivorous vertebrates. These structures are alluded to by "thorns and thistles" in the curse on Adam (Gen. 3:17-19) and, therefore, must figure into any Biblical understanding of the origin of natural evil. The Hebrew *qôs* is best rendered as thornbush, *dardar* as a type of thistle; together as a pair they intensify thorniness (Younger 1997). Thus, the intended reference is to armed plants with which physical contact is unpleasant or harmful, as commonly understood by botanists ("armed: possessing sharp projections, such as prickles, spines, or thorns," Diggs *et al*. 1999, p. 1424). With reference to building a creationist model of the origin of plant armature, the objective of this study is to lay the foundation for an understanding of the distribution of physical armature among plant baramins.

The families of flowering plants were surveyed in the literature for the relative occurrence of species bearing thorns, spines, prickles, or stinging hairs (Hansen & Rahn 1969; Cronquist 1981; Goldberg 1986, 1989; Gentry 1993). The number of baramins represented is not known; however, previous baraminic studies suggest that even the larger families comprise only one to a few holobaramins (Wood 2006). Armed species apparently occur in only 110 of the 252 families recognized. Thorns, spines, and prickles are widely distributed among the 110 families, but stinging hairs are limited to only four families. There were 58 families, including two of the largest (7,000 and 10,000 species), with only one or a few isolated armed species or genera. Armed plants are common (but not predominant) in 35 small to large families (50 to 5,000 species each), whereas armed species predominate in only 11 small to medium families (26 to 2,000 species each). Strikingly, only six families are universally armed or nearly so. Five of these are relatively small (Fouquieriaceae [10 spp], Didieriaceae [15 spp], Smilaceae [575 spp], Pandanaceae [700 spp], and Agavaceae [700 spp]), but only one, Cactaceae (2,000 spp), is moderately sized. Within the families in which armed species are common or predominant, the armed species are usually concentrated in a few large genera or groups of related genera. Of special interest is that, within some families (e.g., Rosaceae), one form of armature occurs in multiple, distantly related groups (prickles in roses and blackberries) and other structures occur in yet other separated groups (thorns in hawthorns and plums). Taken together, these data suggest that entire baramins probably were not created armed. Since plant baramins likely survived the Flood as multiple individuals and certain pre-Flood monobaramins were armed, some currently armed monobaramins may represent descendants of pre-Flood "thorns and thistles," whereas others probably do not. Post-Flood speciation mechanisms are required to account for the diversity and number of the currently armed species.

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C15. Creation and Carnivory in the Pitcher Plants of Nepenthaceae and Sarraceniaceae

R.W. Sanders & T.C. Wood
Bryan College

Genesis 1:29-30 indicates that God gave plants to animals and people for food, but today there are over 600 species of carnivorous plants that "eat" animals for food. Baraminological analysis can assist us in understanding the origins of plant carnivory, either as the original design or a post-Fall adaptation. All species produce modified leaves or stems ("traps") that capture and digest small animals (mostly arthropods) as a supplementary source of nitrogen, the benefits of which vary according to species (Ellison 2006). Types of traps include pitchers, flypaper, bladder traps, snap traps, or corkscrew traps. Carnivorous species occur in eleven angiosperm families (pitcher plants in only four). Traditional classifications of these families, based on their atypical flowers and vegetative bodies, are contradictory but have been eclipsed recently by molecular phylogenies (Soltis et al. 2005, p. 256ff), which place the pitcher plants in the Caryophyllales (Nepenthaceae, ~90 spp.), Ericales

(Sarraceniaceae, 25 spp.), Oxalidales (Cephalotaceae, 1 sp.), and Poales (Bromeliaceae, only 3 of the 2110 spp.). To establish baraminic relationships of Nepenthaceae and Sarraceniaceae, we surveyed interspecific hybridization from published records (Clarke 1997, 2001; Clarke and Lee 2004; D'Amato 1998; McPherson 2007; Pietropaolo and Pietropaolo 1986; Steiner 2002; Wistuba et al. 2002), the International Carnivorous Plant Society database, and catalogues of carnivorous plant dealers (www.flytraps.com, www.exoticaplants.com.au, www.alohanepenthes.com, www.wistuba.com). Within the single genus *Nepenthes* of Nepenthaceae (~90 species), we found records of 227 hybrids between species. These hybrids connect 79 *Nepenthes* species into a single monobaramin. Within Sarraceniaceae, all pairwise hybrid combinations among the eight species of *Sarracenia* are known. Hybrids were also reported from the genus *Heliamphora* (15 spp.), but since *Heliamphora* species are still being described, hybrids are less well known. Twelve of 15 *Heliamphora* species are linked by hybridization, but we anticipate that hybrids of the remaining species will be discovered or produced artificially in the future. We found no records of intergeneric hybrids connecting these two genera or the third Sarraceniaceae monotypic genus *Darlingtonia*. Thus, while all three genera of Sarraceniaceae can be considered monobaramins, there is no evidence of continuity or discontinuity between them. If pitcher plants originated from noncarnivorous ancestors, we might expect to see some variation in the carnivory habit, but no such variation is observed. In both families, the carnivorous habit is found in all species. Nevertheless, commensal and possibly mutualistic relationships within the pitcher exist as well. *Sarracenia purpurea* serves as a host to the commensal pitcher-plant midges (*Metriocnemis knabi*) and mosquitoes (*Wyeomyia smithii*) (Heard 1994), and tree frogs have been observed in the pitchers of *Nepenthes mirabilis* (Hua and Kuizheng 2004). Most intriguing of all, three species of nitrogen-fixing bacteria have been found living in *Sarracenia purpurea* (Prankeicius and Cameron 1991). The presence of nitrogen-fixing bacteria implies a possible mutualism and suggests that the digestion of insect prey may be a secondary source of nitrogen. This potential mutualism should be investigated further. Present data are insufficient to determine if pitcher plant carnivory is the original design of the plants or a secondary post-Fall adaptation.

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C16. Designed for Defense: Reptiles and Amphibians Thwarting Predators in a Fallen World

G. Wilson

New St. Andrews College

Since sin would bring death into the world and that God foreknew that the fall was imminent, I proposed that God genetically designed pathogens, parasites, and predators with the capacity to develop malignant features at the time of the fall. In other words, these features arose by the induction of pre-existing genes created prior to the fall (Wilson 2004). Have the prey been overlooked; whether they are herbivores or mid-trophic predators? I suggest that because many mid-trophic animals need to defend themselves against predation after the fall, they were genetically front-loaded during Creation with various defense mechanisms, which were induced by the fall.

I will briefly discuss one or more examples from the various types of defense mechanisms that occur among reptiles and amphibians. These include: 1) structural armor in various reptiles (Greene 1988), 2) defensive secretions in the Australian gecko (Rosenberg and Russell 1980) and European fire salamander (Brodie and Smatresk 1990), 3) cryptic coloration and immobility in amphibians and reptiles (Brodie *et al*. 1974; Greene 1988), 5) aposematic (warning) coloration in various frogs and salamanders (Weldon 1990; Daly 1998), 6) startling and/or threatening displays in various reptiles (Greene 1988), 7) autotomy in skinks (Dial 1981; Dial and Fitzpatrick 1983), 8) convulsing and/or feigning death in the hognose snake (Greene 1988), and 9) frightening morphology (pit vipers).

Frightening morphology (as opposed to threatening displays) has been largely overlooked in the literature as a defense mechanism probably because of its subjective nature and the problems associated with scientifically measuring frightening appearances and the fear it may induce. Is being frightened by a particular morphology only a learned response or could certain morphologies and their associated stress response be an intrinsic design feature? I propose that this could be tested empirically by measuring the change in heart rate of a mammalian predator (e.g., a house cat) when exposed (through glass) to a live and dangerous reptilian mid-trophic prey item of frightening appearance (e.g., a rattlesnake). Increase in heart rate is the most easily measured stress response. The predator used would be unconditioned (i.e., has not had a prior harrowing encounter with the dangerous prey species). Exposure to live, harmless primary consumer prey species of similar size (e.g., a rat) would be used as a control.

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C17. The Superiority of the Young-Age Creation Theodicy

K.P. Wise
Southern Baptist Theological Seminary

The purpose of a theodicy is to reconcile the existence of a benevolent God with the existence of evil – both moral evil and 'natural evil'. Examples of natural evils are climatological (*e.g.* tornados, hurricanes), geological (*e.g.* volcanoes, earthquakes), and biological (*e.g.* death, pathology, carnivory, natural selection).

Categories of observational data which a theodicy must explain include: A) widespread intuitional data that associates causes of animal and/or human suffering as both evil and undeserved, but causes of plant injury as usually not evil; B) all natural evil is currently inevitable; C) many natural evils are currently essential for maintenance and/or optimality; and D) natural evils are evidenced throughout most of the stratigraphic record. Categories of Scriptural data include: E) God's nature (*e.g.* good, omniscient, omnipotent); F) the sin of spirit beings seems to post-date the Creation Week and is (understandably, given they are not given dominion) nowhere recorded as the cause of any curse on the physical creation; G) the creation was 'very good' at the end of the Creation Week; H) except for Christ, nothing in the present creation preexisted this creation; and (especially in Genesis 1-3, Isaiah 11 & 65; Romans 8; Revelation 21-22), I) the pre-Fall animal world was without death, carnivory, and hurt; and J) God did respond to man's sin with a curse imposed on the whole physical creation which is to be lifted only with the new creation.

Theodicies which deny that God is all-good or suggest He doesn't know, doesn't care, and/or can't do anything are contrary to E. Theodicies which deny the evil of 'natural evils' are contrary to A. Theodicies which trace evil to a previous existence are contrary to H. Theodicies which claim evil is inherent in the physical world are contrary to G. Theodices which trace evil to angelic/Satanic sin are contrary to F. Thus natural evil was introduced into the creation as the result of man's moral evil, something also consistent with J. God introducing natural evil because of man's sin but preemptively (Dembski, 2006) is without example in Scripture, counter to a reasonable sense of justice and against G, H, and I. Natural evil was thus introduced into the creation because of man's sin and chronologically *following* man's sin. Since young-age creationists believe the fossil record of animals post-dates man's Fall, but old-age creationists believe the opposite, the only acceptable Christian theodicy is consistent with D only in the young-age creation model. Old-age creationism lacks a satisfactory Christian theodicy and would reject G, H, and I.

B and C require a pre-Fall creation structure radically different from that observed in the present. Caution is required projecting current patterns back into this period, and a reminder of the importance of considering Biblical data *before* utilizing science is emphasized.

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C18. Evidence that Some Toothed Mysticetes are Archaeocetes (Mammalia: Cetacea)

T.C. Wood
Bryan College

Previous baraminological research indicated that the Archaeoceti are discontinuous with modern toothed (Odontoceti) and baleen (Mysticeti) whales (Mace and Wood 2005; Wood 2006). These earlier studies suffered from a lack of fossil whales from Odontoceti and Mysticeti, which could theoretically connect the archaeocetes with modern whales. The description of the toothed mysticete *Janjucetus hunderi* included a phylogeny with a detailed sampling of extinct cetacean taxa (Fitzgerald 2006). The dataset consisted of nine dental characters, 256 craniomandibular characters, and one postcranial character, for a total of 266 morphological characters. The 26 taxa included extant and extinct mysticetes and odontocetes, archaeocetes, and two artiodactyls. To maximize the number of characters for baraminic distance calculations, I eliminated *Micromysticetus*, *Agorophius*, *Aetiocetus*, *Archaeodelphis*, and the unnamed fossil mysticete ChM PV4745, all of which had less than 66% of their characters known. The reduced dataset had 104 characters with character relevance $a > 0.95$. The 3D MDS (stress = 0.26) exhibited features of both previous studies. The mysticetes and odontocetes were well-separated (average MDS distance 0.476), as in Mace and Wood's (2005) results. As in Wood's (2006) study, archaeocetes were adjacent to the mysticetes and odontocetes but were not interposed between them and the artiodactyls. Most striking of all was a separation between the mysticetes, with four extinct mysticete taxa (*Janjucetus*, *Chonecetus*, *Mammalodon*, and ChM PV5720) grouping with the archaeocetes *Zygorhiza* and *Georgiacetus*. Baraminic distance correlation analysis confirmed that these four mysticetes were significantly positively correlated with the archaeocetes and significantly negatively correlated with other mysticetes. These four mysticetes are considered "archaic toothed" mysticetes and originate from Upper Oligocene or Lower Miocene sediments (Fordyce and Barnes 1994). Fitzgerald's (2006) phylogeny placed these

four taxa basal to a mysticete clade that included *Eomysticetus*, *Diorocetus*, *Pelocetus*, and four extant mysticetes. To confirm my results, I performed a baraminic distance analysis on a second dataset from Bouetel and Muizon (2006). This dataset consisted of 86 craniomandibular and 15 dental characters. The taxa included two archaeocetes (*Zygorhiza* and *Dorudon*), three toothed mysticetes (*Eomysticetus*, *Aetiocetus*, *Chonecetus*), and 18 other mysticetes. To maximize the number of characters used in baramnic distance calculations, I eliminated three taxa from the dataset that had less than 50% known characters (*Mixocetus*, *Nannocetus*, and *Metopocetus*), and I used a character relevance cutoff of 0.9. The baraminic distances were calculated from 73 characters and showed a correlation pattern with two groups. One group contained archaeocetes and the toothed mysticetes, and the remaining mysticetes formed the second group. Both groups showed significant positive baraminic distance correlation for within group comparisons and significant negative correlation for between group comparisons. Taken together, these results imply that the toothed mysticetes *Janjucetus*, *Chonecetus*, *Mammalodon*, *Aetiocetus*, and *Eomysticetus* are cobaraminic with the archaeocetes *Zygorhiza*, *Georgiacetus*, and *Dorudon*. It is possible that Eocene archaeocetes and the Oligocene/Miocene "toothed mysticetes" form a stratomorphic series similar to the fossil equids (Cavanaugh et al. 2003), although a biological trajectory (Wood and Cavanaugh 2003) was not observed in MDS results. All present analyses confirmed the discontinuity between mysticetes *sensu stricto*, odontocetes, and archaeocetes and "toothed mysticetes."

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An Inside Look at the BSG Editorial Process

TODD CHARLES WOOD[1]

[1]*Center for Origins Research, Bryan College, Dayton, TN, USA*

A decade ago, when we started the BSG, we quickly discovered the value of meeting personally to develop ideas and discuss research. Though we began as a strictly email discussion group, we accomplished more in our first meeting in the summer of 1997 than we could have done in months of email. Our conferences soon became our main activity, and we opened the conference to public submissions in 2001. Submitted abstracts required editing, and that opened up a whole new can of worms. We developed our editorial policy by trial and error, mostly by addressing problems as they arose. We've definitely made mistakes, which we've hopefully learned from. Since this is our fifth conference with open submissions, I thought it would be a good idea to look back at our previous conferences and to give an overview of the editorial process as it currently stands.

Roger Sanders, the current editor of *OPBSG* and my colleague at Bryan College, is preparing a more thorough and formal philosophy and theology of peer review. That document will give a justification of peer review in general, but for this article, I want to give an inside look at the how and why, the nuts and bolts, of BSG editing. I will also offer advice to authors on how to navigate the submission and review process by avoiding the most common problems. I'll conclude the article with some statistics on the abstracts we've processed, to give us an idea of how successful we've been and where we need to improve.

Our goal in editing is to make the abstract as good as we can. We want as many outstanding and innovative contributions as we can get. To achieve that goal, we have very high editorial standards. Since we get comparatively few abstracts (compared to the hundreds or thousands at some conferences), we have the luxury to take our time and try to improve even the best of the submissions. As a result, almost everyone who submits an abstract is asked to revise it, myself included. One year, I even had to rewrite an abstract completely when the reviewer pointed out that I had written nothing that hadn't already been published elsewhere.

Why bother with such high standards? What is to be gained? We become better scholars and better writers. That's why we're a "study group" instead of a "society" or "fellowship." Societies get together to present (and debate) research, while fellowships often exist merely for mutual support and encouragement. Both of those purposes are important components of what we do at BSG, but our main purpose goes beyond that. We want to develop and grow as we work towards a creationist understanding of biology. Hence, the abstract submission process isn't merely a chance to report ideas or research results. It's an opportunity to improve our work in the safety of a confidential evaluation. It gives us the chance to correct factual mistakes, muddled writing, and errors of reasoning before the abstract becomes public, when such mistakes would be much more embarrassing. After reading this year's submissions prior to revision, I believe that our regular contributors have become better writers over the years. They've learned how to get the most out of just 500 words.

When the abstracts come in, they are read by Roger Sanders (as editor) and me (as assistant editor). Any abstracts that might be a conflict of interest for us are reassigned to another member of the *OPBSG* editorial board. We've only had two conflicts of interest, namely abstracts written as a critique of something we've both written or abstracts written by one or both of us. For the remainder of the abstracts, I might advise Roger with a few brief comments or a recommended reviewer. For this 2007 conference, I made comments only on four of the 22 abstracts submitted. All four of those abstracts were also sent to external reviewers. Roger, as the editor, makes the final decision on all abstracts, except on those with a conflict of interest. In those cases, the assigned editor makes the final decision. Roger or the editor decides what reviewers to use, what recommendations to pass along to the authors, and what kind of changes will be expected.

Reviewers are chosen to match the expertise of the abstract. Molecular biologists review molecular biology abstracts. Bible scholars review Bible abstracts. Paleontologists review paleontology abstracts, and so on. Abstracts that Roger and I feel good about are generally reviewed by only one other reviewer. More challenging abstracts are sent to more than one reviewer. These challenging abstracts might be abstracts that we disagree about, or abstracts that received an excessively negative review. In the latter case, we think it's appropriate to get a second or even third opinion, before the author is given major revisions to work on, or in the worst case, the abstract is rejected.

What do we look for in abstracts? The first thing is making sure that the abstract conforms to our organizational goal of developing a creationist model of biology. Practically

speaking, that means we avoid apologetics and anti-evolutionary arguments. That doesn't mean that we don't make arguments against evolution or apologetics for creation. We merely insist that the primary purpose of the abstract be to build a positive creationist interpretation or theory. If in the course of developing a creationist theory we can make observations about evolution or apologetics, that's fine. We've always felt that opening the conferences to anti-evolutionism or apologetics could swamp our efforts to develop uniquely creationist theories and distract us from the very purpose the BSG was started in the first place.

The next thing we look for is originality. This derives from our value of original research. There are many publication outlets in creationism, not to mention the hundreds of creationist books now available. Dozens of ideas have been proposed and repeated again and again in these publications. What we're looking for is something original, some attempt to validate or falsify some creationist idea. This is part of building a creationist biology model. We need to sift through all of these many ideas, take out the good stuff and discard that which is probably not going to work.

For original abstracts that fit our goal of constructing creationist theories, the most common problem we encounter in abstracts is bad writing. It's very easy to do. Sometimes information gets left out. Sometimes sentences are written in an awkward way. Sometimes sentences are not logically connected, and we have difficulty following the reasoning. Occasionally, the hypothesis being tested is left out entirely! Authors are so close to their ideas that they forget to dumb it down for the rest of us. This is the problem I personally wrestle with the most. I usually have my assistant read my articles and abstracts before I show them to anyone else. She identifies the awkward phrasing, superfluous adverbs, and generally muddled wording, and I correct it as best I can. It usually takes a number of revisions before I can get my point across.

More serious but less frequent problems include factual errors or errors in reasoning. Sometimes the hypothesis isn't valid. Sometimes the methods are not appropriate. Sometimes the results have been misinterpreted. These problems require additional reading or research on the part of the author. As I said above, when such problems occur we usually try to get several reviewers' opinions on the abstract, just to make sure that there really are problems before asking the author to make extensive revisions.

Once the editor gets the reviews in, he examines the abstracts again and decides what kind of revisions he will request from each author. To the authors, this can be pretty aggravating. It's really hard to put a lot of work into an abstract only to be told that it's basically not good enough. I remember the review of one of my abstracts that concluded, "there's absolutely nothing new" in the abstract. I was so disappointed that I asked Roger to reject it so I wouldn't have to revise it. But he insisted that I at least try to revise it, and I'm glad I did. As I noted above, this kind of review makes us better authors and scholars. It takes some humility to accept the requested revisions, but the revisions always improve the abstracts. Getting a better abstract and becoming a better writer is worth the temporary aggravation.

Unfortunately, occasionally an author will become extremely offended at the reviews or requested revisions. Sometimes these offended authors will respond with accusations against the editor or even the whole BSG. These kinds of accusations put us in an awkward position. Obviously, if we've done something wrong, we want to make it right, but it's hard to determine who's at fault when the accusations are laced with bitterness or insults. This is more unfortunate because several of these incidents were caused by simple misunderstandings. Resolution could have been achieved easily, but overcoming hurt feelings is much more difficult.

What should you do if you think you've been treated unfairly by the reviewers or editor? Tell the editor, but don't attack him. He's on your side. Remember that our main goal is getting as many outstanding abstracts as we possibly can, and we all experience the same kind of aggravation dealing with revisions. The BSG is definitely not looking for excuses to reject abstracts arbitrarily. The best way to express your dissatisfaction is to state your perception of the requested revisions and why you think they are unfair. Say something like, "I perceive that the reviewer is attacking my position when he says ____ [be specific], and I object to revising my abstract according to his suggestions." That will help the editor identify precisely what the problem is. As I said, misunderstandings often spark these disagreements, and the most common misunderstanding is to assume that we're asking for a conceptual revision when what we really want is clearer writing. If you think the editor or reviewer doesn't understand your abstract or is responding inappropriately, it's probable that polishing the writing will fix the problem without ever changing the content of the abstract.

Since I've submitted eleven different abstracts to these conferences and been on the inside track of almost all of the editing, I'd like to share some tips on getting an abstract through the review process. I first recommend that potential authors really need to understand what we mean by building creationist theories. This is not the same as critiquing evolution or making an argument for design. Read our past proceedings carefully to get an idea of what we're interested in. Don't think that you have to propose something shockingly new or that you have to resolve some major problem. This is just an abstract. We're perfectly content with work that is, frankly, dull. Even if you haven't finished your research, but you want to describe your hypothesis and your preliminary results, that's fine too. As long as it fits the overarching goal of developing a creationist understanding of biology, go for it!

My next recommendation for those who haven't submitted before is to run your idea past the editor before you submit an abstract. This could save you a lot of aggravation. Most of us on the executive council routinely do this, and the editor can then make recommendations on how to write the abstract in the first place. It saves an amazing amount of time, since the editor is usually pretty happy with the end product, and the review process becomes much less painful. Sending an abstract out of the blue could be risky.

The next thing that every author needs to remember is to write a good abstract. Sounds simple, but unfortunately, it's not. Every abstract needs to be a complete summary of what you're going to say. For science papers, that means you need a short introduction to the hypothesis, an explanation of methods, a description of results (as complete as necessary), and a few sentences of

discussion and conclusion. Philosophical or theological papers should be similarly complete in their coverage. One error we've seen somewhat frequently is submitting an abstract that is actually just the introductory section of a longer paper. The last sentence might read, "In my paper I will discuss how I tested these ideas and what I found." That is *not* an abstract. Don't do that. You have to say in the abstract what you did, what you found, and what you concluded.

As you write your abstract, remember that you only have five hundred words. Use them wisely. If the abstract runs over 500 words, it will probably be sent back to be shortened, so be ruthless as you edit your own work. Make sure that every word you use is essential to the "story" of the abstract. Are there phrases that you could shorten? Could you delete some adverbs or adjectives and keep the original meaning? Are there any sentences that could be eliminated? I often find that I overload the introductory part of the abstract, and I don't have enough room to present the results and conclusions. Then I have to go back and cut down the intro material to be able to adequately explain the results within 500 words. This can be quite difficult to do, especially when it's your own work. I always think that what I'm writing is important to the abstract, which it might be. There's a big difference between "important" and "essential," though. "Important" ideas might have to give way to "essential" ones in the final abstract. I don't think this is much to worry about, though, since you can always put back the "important" things into a paper (which you could submit to the *OPBSG*). Keep in mind too that the 500-word limit only applies to the text of the abstract and not to the references cited.

Also as you try to write a good abstract, remember that this abstract will be evaluated by someone with expertise in the area in which you are writing. BSG reviewers aren't just some "good old boy" network. We are interested in being as good as possible, and that often requires seeking reviews from outside our editorial board and our "usual" reviewers. Consulting only our "buddies" is a recipe for stagnation. In a typical year, we will consult several reviewers who have never reviewed for us before. Because of the expertise of the reviewers, writing outside of your own expertise is almost a guarantee of substantial revisions. That doesn't mean it's impossible to do (see Bartlett 2007). If the reviewer suggests revisions, and the editor is convinced that you've met the standard of the reviewer, he will gladly accept your work. But it's best to know going in that something that is not your field will probably need extra work on your part to meet the standards.

Finally, let me emphasize that we all need to take the review process in stride. Remember that this is just an abstract. Revising a 500-word abstract really isn't that big of a deal. Clear and calm communication with the editor is probably the best way to get through the review process successfully.

So how have we done? Despite the occasional unpleasantries, I think that the results of the editing process have been very good. We've received 75 abstracts, and we accepted 65 of them. That's an 87% acceptance rate. That's amazingly high. We've only rejected four abstracts (5%), and six abstracts were withdrawn. We started out with only eight abstracts submitted in 2001, and we received 22 abstracts for this year's conference. Our growth rate is quite healthy also, both in gross numbers of abstracts and in recruiting new authors. The number of submitted abstracts has grown by an average of 31% at each conference. After the first conference in 2001, an average of 39% of the abstracts at each conference were authored or co-authored by someone who had not previously participated in a BSG conference.

Our "customer satisfaction" also seems good. Not counting the first HybriDatabase abstract from 2001 (it's thirty co-authors were mostly members of an Origins class at Bryan College), we've had 44 different individuals author or co-author abstracts, of which 21 (48%) have written more than one abstract. That's an average of 2.1 abstracts for each author. In addition, the BSG has been good for developing collaborations. Of the 75 submissions, 17 (23%) had multiple authors. BSG seems to bring people back and encourages them to work together.

Where can we improve? Well, we've published 18 contributed abstracts in this year's conference proceedings, and that's a record for the BSG. But 18 is still pretty small compared to many other conferences. Our size could be a result of the very narrow focus we have, but our growth rate, if it can be sustained, should remedy this situation in another five to ten years. I would love to see the BSG conference become so large that we have to expand to another day or split up into tracks!

We also can improve the way we edit. I mentioned previously that Roger Sanders is developing a paper on peer review (which, of course, will be peer reviewed). We're hoping this work will give us a better idea of how and why we peer review and perhaps even suggest means of improving the process. In addition, we need to develop a formal editorial procedure manual with clearer guidelines. As I said, we've mostly learned how to do abstract editing the hard way, by trial and error. By now, we think we have a good method worked out, and we just need to write it down. With clearer guidelines for everyone involved (editor, reviewer, author), we should be able to avoid misunderstandings in the future. We will make this new editorial manual available at the BSG website before the 2009 conference.

Looking back and reflecting on our journey is always beneficial. It's good to remind ourselves of how far we've come, and even the distasteful memories show us what to avoid in the future. I'm also excited as I look forward to our future conferences. The BSG Executive Council is already developing an aggressive plan for the future. Our next conference in 2008 will be held in San Diego in conjunction with the International Conference on Creationism. We will accept abstracts for the 2008 conference, and they will be due at the usual time in February. The 2008 theme will be "Looking to the Future: Creationist Biology in the 21st Century." We're still working out the order, but conferences thereafter will be themed around Noah's Ark, design, paleontology, and ecology and biogeography. The future is bright indeed. May God continue to bless us, and may we continue to humbly seek Him and His glory in all we do.

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