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PRESIDENT'S MESSAGES*

Lamarck Lives!

Dr. Rebecca Reiss

You probably have heard the Lamarckian inheritance theory explained this way: giraffes have long necks because they reach for leaves on trees and this is passed on to their offspring, who have even longer necks. Dismissed because of lack of evidence, it turns out that Lamarck wasn't completely wrong; he just had the wrong order of magnitude. Perhaps you have heard the recent news that the daughters of obese male rats exhibit symptoms of diabetes even if they are fed a low fat diet. This is an example of the growing evidence for the inheritance of acquired characteristics that is revolutionizing our views about DNA and natural selection.

Jean-Baptiste Lamarck (1744-1829) is best known for his theory for the inheritance of acquired characteristics, but this was not his only contribution to the elucidation of evolutionary principles. Lamarck predicted that organisms lose organs for which they have no use, exemplified by sightless cave dwelling creatures that have no use for eyes as they do not normally encounter light in their environment. New species of sightless insects and fish are still being discovered as we explore underground and deep-water environments.

Is the existence of sightless organisms evidence for an 'intelligent designer?' Only if one hypothesizes that since these organisms don't have eyes, then they don't have genes for eyes. The evidence does not support this hypothesis; the genes still exist, but they are shut off and are not expressed. So the argument will always come back to distinguishing between natural phenomena and divine intervention. There is a naturalistic explanation for the silencing of genes for unused organs that requires modern molecular genetic techniques to detect.

There has long been subtle evidence for the inheritance of acquired characteristics, but only recently has the mechanism been determined. Chemical flags are added or removed from DNA that in turn change the configuration of the chromosomes, resulting in differences in levels of gene expression. If changes in these chemical flags occur on the chromosomes in egg or sperm, then the trait is passed on to the next generation. Epigenetics is the term given to this process and its implications for human health are just beginning to be realized.

The finding that maintaining rats on a high-fat diet influences the health of the next generation provides one explanation for the increase of obesity in human populations and provides evidence for Lamarckian inheritance. The environment marks our DNA and this is an aspect of natural selection that we are just beginning to understand. Epigenetic changes are now implicated in cancer progression, post-traumatic stress syndrome, as well as the obesity epidemic. We have a long way to go before we completely understand how the environment changes our DNA, but evolution by natural selection doesn't just occur over geological time, it is a continuous process that is occurring in your cells even as you read this essay.

Continued on Page 2

**In this issue, we print two president messages. Since we have a real genetics professor for a President this year, we are taking advantage of her knowledge and ability to "get the point across" to those of us who are not quite so expert in the field. ed.*

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WEBMASTER: Jesse Johnson

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Continued from Page 1

If progress is to continue, it is necessary that science education focus on critical thinking skills and testable hypotheses rather than promoting irreducible complexity as an explanation. To deny evolution by natural selection is to deny that cures for scourges of human health such as cancer can be discovered. The well being of generations yet unborn depends on how we educate today's students.

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THE DEATH OF JUNK DNA

In the 1970s, the development of techniques to sequence DNA in our chromosomes revealed that over 50% of the DNA appeared inert, without function. Composed of repeated sections, it was branded by one researcher as 'junk DNA' since no function was detectable with techniques of the time. I remember discussing this term with my colleagues in graduate school at Cornell in the 1980s, and we all had the same two reactions; how do you know its junk if you don't know what it does; and what if it is repetitive because it has an extremely important function? The term "junk" may have influenced some scientists, but in 1983, Dr. Barbara McClintock received the Nobel Prize for her discovery of transposable elements. Commonly known as "jumping genes" because they can move around the genome and change expression of other genes. Transposable elements are part of the repetitive faction of our genomes. The functions of other classes of repeated DNA are being revealed with the latest technology. It turns out that these repeated regions are active and exhibit sensitivity to environmental signals, such as a high fat diet. The American Association for the Advancement of Science renamed "junk DNA" as genomic dark matter and ranks it as one of this past decade's most important findings.

So how does the intelligent design community view the rapid progress in genetics and convince some people that ID has a place in today's classroom? In his January lecture in Albuquerque, Dr. Richard Sternberg from the Biologic Institute provided insight into how the intelligent design movement is trying to use the genome dark matter to prove their case. The main argument falls under the category of "we told you so." ID predicts that junk DNA isn't junk and that there is more to the genome than just genes. Dr. Sternberg constantly alluded to the idea that the genome isn't the only thing in our cells that controls cellular function, yet he never offered any hypothesis as to what else **is** in control. It is common knowledge among biologists that our chromosomes carry genetic information from generation to generation, and their complex structure includes the DNA genome (a special carbohydrate) and protein encased in a protective envelope comprised of fat. All of these molecules play a role in the transmission of information necessary for cell function. The second argument was that of "irreducible complexity," described as a black box in which cellular functions occur and we can only attempt to simulate. But he suggested that simulations devel-

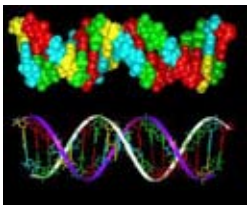
Continued on Page 3

oped at the Biologic Institute could explain cell function. The take-home message was: don't think about this too much, it's just too complicated.

The most recent legislative session in NM included a bill that could have allowed intelligent design to be taught in public schools as an alternative to science. Fortunately, this bill never made it to the floor of the legislature through the efforts of individuals dedicated to maintaining New Mexico's strong science education standards. A similar bill has passed the Tennessee House of Representatives, so the state that gave us the Scopes Trial and the Tennessee creation museum is trying to give voice to religious teaching in the science class. Despite the outrage of the scientific community in Tennessee, the bill is on track to become law. [*Since this was written, the bill has just died the when the session ended ed.*]

New Mexicans can be proud that the National Center for Genome Resources in Santa Fe maintains state of the art instrumentation that makes the new genetic revolution responsible. Research projects at NM research universities and private institutions are making strides in tracing the signals that led to obesity, heart disease, and mental illness. While the opportunities for the next generation of scientists are astounding, so are the ethical questions that will arise as findings are applied to human health. The educational challenges are daunting and if New Mexico is going to continue to lead in this area, an educated workforce is necessary. Assaults on science education in public schools only serve to distract from this challenge of establishing a skilled workforce and an educated citizenry. As we learn more about this mysterious dark matter in our DNA we increase our understanding of natural selection and what appears to be "irreducibly complex" to some represents the next important discovery.

Dr. Reiss is the current President of CESE serving for the 2010/2011 term. She is an Associate Professor at New Mexico Tech, which specializes in technology and science teaching and research. It has often been a consistent top 10 contender in various rankings as one of the best technical schools in the nation. Dr. Reiss specializes in genetics.



BEHE IN BRITAIN, MILLER'S MOUSETRAP, AND THE ORIGINS OF MALARIA©

Professor Michael Behe (yes, him again) has toured the United Kingdom, as the guest of a new obscurantist organization calling itself the Centre for Intelligent Design (C4ID). The Centre's president, Prof Norman Nevin, believes that Genesis 1 through 11 (garden, talking snake, Noah's Ark, the lot) is literally and historically true, and the Centre's list of friends is a roll call of religiously motivated UK creationists. And three of Prof Behe's lectures were delivered in churches, one in a biblical literalist church in Belfast, one in London's Notting Hill (preceded by hymn singing)[1], and one in Westminster. According to the published itinerary, the last of these was held "in association with Premier Christian Radio." However, C4ID, like Prof Behe himself, assures us that Intelligent Design concerns itself with science, not religion, and has nothing to do with creationism. At the lectures, the Discovery Institute's fake[2] textbook *Explore Evolution* was on sale, alongside copies of Ben Stein's thoroughly discredited[3] movie, *Expelled; no Intelligence Allowed*. The Centre nonetheless assures us that it is completely independent of the Discovery Institute, which has merely supplied its ideas, its materials, its inaugural speaker and, one fears, its standards of intellectual integrity.

Reports are in of the actual content of Prof Behe's lecture[4], and I feel as if I have travelled backwards in time. He spoke about the "irreducible complexity" of a mousetrap, an argument that first appeared in *Darwin's Black Box*, way back in 1996. Ken Miller's hilarious deconstruction (of the mousetrap, as well as the argument) is available online[5], as is Prof Behe's lame attempt[6] to put it back together. His next exhibit, believe it or not, was the bacterial flagellum. But what about all the evidence linking it to its simpler precursors, he was asked. Irrelevant, because until it became a complete flagellum, it was not functioning as such, so it is indeed irreducible. A rather unconvincing semantic trick, worked by changing the meaning[7] of "irreducible" in mid-argument. We also got the claim that since mutations are the result of copying errors, they must involve degradation, or loss of function, so they couldn't explain the elaboration of function anyway. The flood of counterexamples that immediately comes to mind merely serve to illustrate the effects of Intelligent Design. When asked about Kitzmiller v Dover School Board, he explained that the judge was not in a position to give an informed opinion, since he was

not a scientist, and was taking his opinions from the scientific establishment. The same scientific establishment that, he said, stops him from publishing his views in the peer-reviewed literature. ID, it seems, comes with its own conspiracy theory. Prof Behe, I conclude, is completely sincere, unsinkable, and fact-proof.

Last December (2010)[8], Prof Behe published a paper in "Quarterly Reviews of Biology" in which he aims, rather unsuccessfully, to minimize the constructive role of mutation combined with selection. He does this by confining attention to prokaryotes grown in isolation, by introducing his own asymmetric criteria as to what would count as constructive, and by admitting for consideration only those relatively few cases where the mutation and its operation are understood at the molecular level. Limiting the playing field, tilting the playing field, and moving the goalposts closer together. Even so, he has to admit some cases of gain of function, any one of which would suffice to destroy the argument for design. By a delightful coincidence, if such it be, the same issue of the journal contains a detailed philosophical analysis[9] of the logical errors and rhetorical devices used by Prof Behe and his associates, some of which I have mentioned here.

Malaria, unlike the mousetrap, does not merit an index entry in *Darwin's Black Box*, but featured in his UK lecture, and plays a major role in his 2007 book, *The Edge of Evolution.* Here he explains at some length[10] why he does not consider the emergence of chloroquine resistance in the malaria parasite to be a Darwinian process. This book, too, has been mercilessly dissected by reviewers far more qualified than I am[11], as has the specific claim regarding chloroquine resistance[12]. So I'll content myself at this stage with the observation that if chloroquine resistance really is the result of intelligent design, that tells us something rather disconcerting about the Designer.

Malaria itself is a parasitic disease involving two separate species, a vertebrate host and an insect vector. The full life cycle involves a number of separate phases depending on the exact species[13], with infection of the host by an insect bite, migration to the liver and thence to red blood cells, asexual reproduction within the red blood cells, and formation of male and female gametes. When the insect takes a blood meal from a host, the gametes recombine within its stomach, giving rise to a new generation and a new cycle. The parasite is a magnificent (if that is the correct word) example of adaptive evolution in action. Like any infectious agent, it needs to evade the host's immune system. It does this, first by hiding in the liver, and later by hiding within the red blood cells. It prolongs the bodily residence time of the infected blood cells by increasing their stickiness, causing them to cling to the walls of blood vessels, rather than making their way to the spleen, which would remove them. This, incidentally, is among the ways in which it induces weakness in the hosts, making them less capable of defending themselves against the insect carriers. Since the parasites cannot remain fully hidden as they migrate, hosts (including humans) do tend to build up immunity over time. The parasite counters this by the position of the sexual phase in its life cycle. The function of sex, as always, is to juggle information (sex, after all, is not necessary for reproduction), so that each new infection will bring parasites with rearranged genomes, coated with proteins that the host has not seen before.

Malaria type parasites have been identified in the abdominal cavity of a biting midge trapped in early Cretaceous amber. The midge seems to be adapted to feeding on cold-blooded animals, and indeed it has been suggested that malaria was among the many diseases afflicting the dinosaurs[14]. Molecular evidence[15] suggests an even older origin for the disease, around 130 million years ago, with malaria both in mammals and in birds having originated from a form parasitic on reptiles. The vectors for human malaria are several species of Anopheles mosquitoes, and the parasites that they carry are closely related to those causing similar diseases in the other great apes. It was long believed that the protozoan Plasmodium falciparum that is responsible for the most virulent form of human malaria came from chimpanzees, but the most recent studies[16] show that its closest relative is one that infects gorillas. Either way, malaria, like HIV, is among the human diseases that have found their way to us from our close relatives. P. falciparum is most prevalent in hot, damp climates, and appears (from molecular studies) to have started spreading widely among humans around 6000 years ago, perhaps as a result of the higher population densities and irrigation practices associated with agriculture[17].

Like all parasites, Plasmodium must at some stage have evolved from free-living organisms. It seems plausible that these were aquatic, and acquired the ability to recognize, feed on, and finally live within insect larvae. Every parasite has the problem of moving from one individual host animal to another, and for this genus, the problem is solved by transfer of fluids when the vector insect is feeding. A necessary cost of the parasitic lifestyle is exposure to extreme changes of environment, which parasites deal with by adopting different phases at different stages in their life cycle. This is an option open to protozoans and to multicellular parasites, which have large enough genomes to include the instructions for all the necessary quick change acts, and which respond to environmental clues that determine which set is activated. The evolution of chloroquine resistance in P. falciparum is a matter of enormous practical importance, since malaria kills something like 1 million people a year. The precise genetic mechanism, involving a sequence of changes to one particular protein, is under active investigation, as is the intriguing fact that a parasite lineage that has acquired

resistant to one drug can have increased sensitivity to others[18]. Such are the fruits of the "materialistic explanations" that Behe and his colleagues wish to replace[19]. with "theistic understanding".*

"Intelligent design" or effective science? There is not room for both.

Addendum: Shortly after Behe's review appeared, an extensive study of the evolutionary history of 3,983 gene families across the three domains of life (Archaea, Eubacteria, Eukaryotes) was published in Nature[20]. To quote the MIT press release. "The work suggests that the collective genome of all life underwent an expansion between 3.3 and 2.8 billion years ago, during which time 27 percent of all presently existing gene families came into being." As Figure 1 (publicly available; see Footnote 20) clearly shows, the generation of new function outstrips and precedes the loss of older function. This is a massive refutation of Behe's entire line of argument, and of the use that Creationists and their allies would wish to make of it.

This article has also been posted on the website of the British Centre for Science Education

(http://bcseweb.blogspot.com/2010/12/behe-in-britain-millers-mousetrap-and.html)

Endnotes:

[1]Craig Lucas in http://poddelusion.co.uk/blog/2010/11/26/episode-61-26th-november-2010/

[2]See e.g. http://ncse.com/creationism/analysis/explore-evolution and http://bcseweb.blogspot.com/p/evolution-exposed.html

[3]http://www.expelledexposed.com/index.php/the-truth/id

[4]See e.g. http://www.thetwentyfirstfloor.com/?p=1434

[5]http://www.youtube.com/watch?v=ieKDLtrBXs0 among other places

[6]http://www.arn.org/docs/behe/mb_responsetokmiller0101.htm

[7]More exactly, the referent; it is the flagellicity, not the complexity, that on Behe's terms re ally is irreducible

[8]The Quarterly Review of Biology, December 2010, Vol. 85, No. 4, 419-445.

[9]Ibid., 473-482

[10]The usual statistical sleight-of-hand, pretending that sequential changes in a highly variable protein have to happen simultaneously.

[11]For a particularly scathing and detailed analysis by Jerry Coyne, see http://www.talkreason.org/articles/Mutator.cfm

[12]Matzke NJ, The edge of creationism, Trends in ecology and Evolution 22, 566, 2007, and references therein

[13]See e.g. http://www.cdc.gov/malaria/about/biology/index.html for details

[14]Poinar G, jr and Telford, SR Parasitology 131, 79, 2005; What Bugged the Dinosaurs?, Poinar, G., jr and Poinar, R., Princeton University Press, 2008.

[15]From analysis of cytochrome B: Yotoko, KSC and Elisei, C. Malaria parasites and their relationships with their hosts.. Journal of Zoological Systematics and Evolutionary Research 44, 265, 2006

[16]Liu W et al. Origin of the human malaria parasite Plasmodium falciparum in western gorillas. Nature 467, 420, 2010.

[17]Hume JCC, Lyons EJ and Day KP, Malaria in antiquity: a genetics perspective, World Archaeology 35, 180, 2003

[18]See e.g. Johnson DJ et al., Evidence for a Central Role for PfCRT in Conferring Plasmodium falciparum Resistance to Diverse Antimalarial Agents, Molecular Cell, 15, 867, 2004,

[19]www.antievolution.org/features/wedge.pdf, p4, "Governing Goals"

[20]Nature 469, 93-96 (19 December 2010); Abstract, and figure referred to, publicly available at http://www.nature.com/nature/journal/v469/n7328/full/nature09649.html

* Stop the press: The mechanism by which the sickle cell allele protects against malaria has now been elucidated. See http://www.sciencedaily.com/releases/2011/04/110428123931.htm, retrieved May 19, 2011; Ferreira et al., Cell, 145(3) 398-409, 2011]
Sickle hemoglobin leads to increased expression of an enzyme that oxidises heme, producing carbon monoxide in the brains of carriers, and this protects against the development of cerebral malaria. But no doubt Behe would dismiss this as an example of loss of function, since it depends ultimately on the inhibition of destructive metabolic pathways. Ah well.

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HUMAN FAMILY TREE (WEB) SPROUTS NEW BRANCH (THREAD)

Sequencing of ancient DNA from a finger bone and a tooth found in Siberia has revealed a new hominin population that is neither Neanderthal nor modern human, but closer to the Neanderthals. This group, named "Denisovans" after the cave where their remains were discovered, interbred with modern humans, and contributed specifically to the ancestry of present-day Melanesians.

References:

Reich, David, et al. "{Genetic history of an archaic hominin group from Denisova Cave in Siberia}." Nature. 468 (2010): 1053-1060; News and Views non-technical commentary, ibid. 1044-1045; see also Krause, Johannes, et al. "The complete mitochondrial DNA genome of an unknown hominin from southern Siberia." Nature. 464 (2010): 894-897.

http://johnhawks.net/weblog/reviews/neandertals/neandertal_dna/denisova-nuclear-genome-reich-2010.html (with discussion of background)
Commentaries and discussions at

http://www.nytimes.com/2010/12/23/science/23ancestor.html?_r=1&hp (article by Carl Zimmer)

http://pandasthumb.org/archives/2010/12/denisovans.html

http://news.nationalgeographic.com/news/2010/12/101222-new-human-species-dna-nature-science-evolution-fossil-finger/

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WHERE'S THE SCIENCE AND MATH REFORM?

Terry Dunbar, Ph.D.
Vice president, CESE

CESE has been working for many years discussing ways to influence the quality of math and science instruction in New Mexico. We have devised ways to compare schools, a critical initial step in the process of transferring best practice from one school to another. The facts that the transfer of best practice has not occurred and that our students have shown little test score progress in either math or science have caused considerable consternation among our members. Why have our schools not moved forward in preparing students for technical careers, especially since science and engineering play such an important role in our state's economy? Why do some schools show dramatic improvements while others stagnate or actually slip backward? Improvements are often short-lived. Where do the problems lie?

We know that demographic factors (ethnicity, poverty, and other characteristics) can predict the performance of schools. Public schools have almost no control over these factors. The chart below, courtesy of CESE statistician Walt Murfin, shows the proportion of variance in reading scores explained by demographics. As can be seen, percent minority and FRPL (free and reduced-price lunch – a measure of poverty) explain the majority of variation. The chart for math is similar.

Public schools have no control over the demographic makeup of their students. All students who live inside a school's boundary area and who are not a danger to other students are accepted. Every student who walks through the door is accepted into the classroom. Demographics predict the average outcomes, but do not necessarily cause results for individual schools. Some schools with high proportions of underachieving minorities, poor students, and students with disabilities have managed to raise their test scores significantly above predicted, but many have not. The shortcoming must be somewhere in the way schools are organized, or in the standard three-legged stool of K-12 classroom education – curriculum, assessment, and instruction.

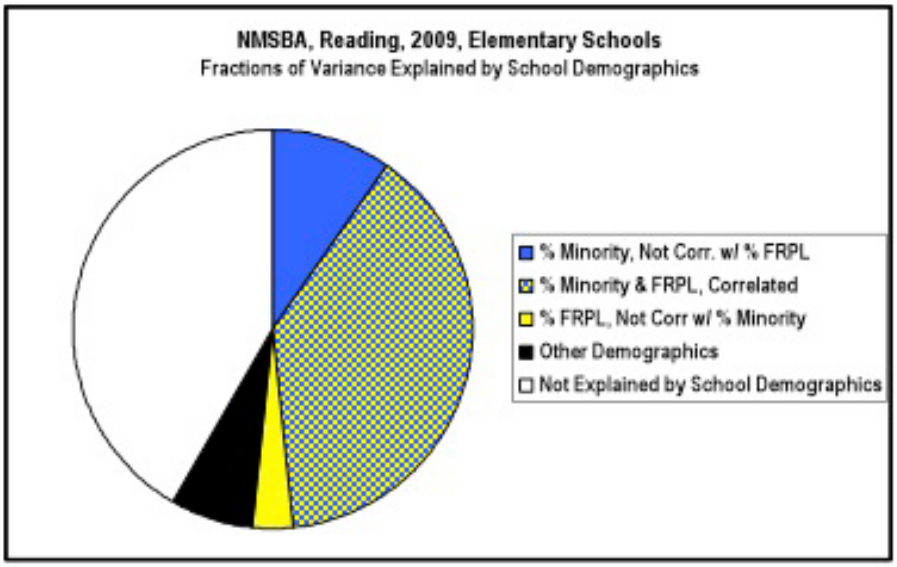
This article is meant to examine what is known about the instructional leg and its relation to school reform. We will first look at math and science instruction separately, and later at the context of the school as a whole. It's not that we don't have data-based insight into what to do in the classroom. Multiple studies have been performed. Robert Marzano (Marzano, 2001) conducted a meta-analysis of thousands of classroom research studies. Effect sizes for various classroom strategies were calculated. The table below shows some of the instructional strategies and their effect sizes. An effect size of 1.0 means a difference of one standard deviation from the mean. Teacher behavior can have a huge effect on student performance (Marzano, 2007).

Categories of Instructional Strategies that Affect Student Achievement	
Category	**Average Effect Size**
Identifying similarities and differences (compare and contrast)	1.61
Summarizing and note taking	1.00
Reinforcing effort and providing recognition	.80
Homework and practice	.77

Research on specific curriculum areas provides clear guidance on what works and what doesn't work in the classroom. In math, the National Research Council (2005a) advises that teachers at all levels avoid math instruction that is nothing but "drill and kill" (computation-based) and instead emphasize:

- Combating common preconceptions about mathematics (math is only for the select few, there is only one correct way to solve a math problem, etc.)
- Engaging students' prior knowledge
- Encouraging problem-solving skills
- Developing error-finding and error-correction skills
- Encouraging math conversations
- Encouraging the use of metacognitive processes (problem-solving, communication, reasoning) to facilitate knowledge and skill construction
- Use of representation (manipulatives, drawings, diagrams) for concept development

Recommendations for science instruction (National Research Council, 2005b) are similar. Invoking prior knowledge before and during a classroom investigation helps students make important connections. During science classroom activities, it is important to address students' scientific misconceptions as they arise. Asking students critical questions to clarify their conjectures helps them give more complete answers and explanations, and fosters engagement and learning. Students



should be provided multiple opportunities to summarize observed results and to generate conclusions based on those results.

Too much classroom science instruction consists of memorization of facts and the teaching of science as history, as a sequence of discoveries by famous scientists. School science chiefly differs from professional science by the rare occurrence of scientific inquiry. Feynman (1995) decried the lack of real inquiry in school classrooms. He characterized the scientific method in three words: observation, reason, and experiment. True, guided inquiry, then, is distinguished from most current classroom practice by students identifying questions, making hypotheses, and then designing experiments to test the hypotheses.

Why doesn't more of this best practice occur in schools? If we examine schools as systems, we can understand that a school must be structured in such a way that these reforms are not only possible, but encouraged and supported. Various structures have been studied, and we know now more than ever what works in schools. A school functions as a system. Human systems (schools, companies, churches, etc.) are complex entities with multiple interactions. Teasing out what works is difficult, but some features come up over and over again in research on successful schools.

CESE members have advocated for matching schools with similar demographics (and thus presumably similar challenges). Those schools whose students perform best on the New Mexico standards-based assessment (SBA) must have something worthy of sharing with the schools that produce lower test scores. So, for example, if visits are arranged (school A visits school B because school B has significantly higher test score), then teachers and administrators from school A should have some foreknowledge of what to look for. Of course, given sufficient money, an outside group could observe both schools and determine those relevant best practices that cause success and the relevant worst practices that cause failure.

Educational researchers have looked at schools with an eye toward teasing out the hallmarks of instructional success. Many studies find commonalities among successful schools regardless of level, background of students, and size of the school. Schmoker (2006) cites "a guaranteed and viable curriculum" (focused teaching to the standards) as the major factor that affects student achievement in schools. Here's a short list of other instructional initiatives found in successful schools. Some factors, such as parent involvement and student motivation, are highly correlated with school success, but schools don't always have control over them. The factors below are all under the direct control of the school staff.

- The three Rs: rigor, relevance, and relationships
- High expectations, pressure to achieve
- Data-based decision making
- Emphasis on time on task (efficient use of instructional time)
- Frequent monitoring of student progress
- Strong classroom management
- Teacher collaboration (professional learning communities, teaming (secondary teachers sharing the same group of kids, shared vision)
- High-quality, relevant, coherent professional development
- High-quality leadership
- Constructivist teaching and learning (*)

* Constructivism is a theory of learning based on the idea that knowledge is constructed by the learner based on mental activity. Learning is an active process in which meaning is developed on the basis of experience.

If in the future comparison of schools in New Mexico does happen, we should be able to tease out some of the factors that permit successful schools to overcome the demographic odds. Since New Mexico is unique in many respects, we can not know ahead of time all the factors that are associated with high achievement. Nevertheless, research on school organization gives us some clues as to what researchers in New Mexico are likely to find. Successful schools set goals and monitor progress toward achievement (Marzano, 2003). Successful schools tend to have strong leaders who have their fingers on the pulse of all facets of school life. Other features may appear at schools that have improved their test scores. Some successful schools have raised test scores by strengthening students' test-taking skills. Many have favored math and reading instruction at the expense of all other curriculum areas, since the No Child Left Behind federal act was passed (NCLB), judges and compares schools on the basis of only reading and math test scores.

It should be clear that there are many explanations for the slow and unsteady progress in New Mexico schools. Principals must have a comprehensive understanding of what needs to be done and how it is to be accomplished. Underachieving schools must do everything possible to increase positive parental involvement. It may take several years to reverse the effects of inaction, lack of focus, and other poor school practices. Will we find leaders who can step into our failing schools and turn them around? Will those leaders be able to recruit and retain teachers willing and able to work together in coherent, focused, and comprehensive teams? Will we be able to recognize and replicate the practices of successful schools at less successful

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