**Re: Update for the Mach-Gaensslen Foundation - 2008**
**Dr. Lynn Megeney, The Mach-Gaensslen Chair in Cardiac Research**.

The year 2008 was an exciting and compelling transition year for my laboratory and research program. In late 2007, the biotechnology start-up company of which I was a founding scientist (StemPath Inc.) was poised to receive substantial venture capital based financing. Unfortunately, the lead investor within the group was unable to complete the financing arrangement which precipitated the dissolution of StemPath Inc. However, through a concerted effort with the other co-founding scientist of StemPath Inc. (Dr. Michael Rudnicki), we established a new Biotechnology start-up company, Verio Therapeutics Inc. (July 2008). In addition to the strong scientific credentials, Verio Therapeutics has a truly international business strength with a renowned biotech business persona, Frank Gleeson serving as the CEO, as well as Dr. Cal Stiller serving as the Chair of the board for Verio Therapeutics. Verio is now securing a financing base to move the original cardiac repair program toward a full pre-clinical assessment and we anticipate a final office action to secure the patent protection for our compound within the coming 3-4 months. The cardiac repair program is based on original basic science observations from my laboratory demonstrating that delivery of a proprietary compound/protein to infarcted hearts was sufficient to promote repair and regeneration of the damaged myocardium. The support from the Mach-Gaensslen Foundation has been instrumental in both the initial efforts to establish this line of research and to the continued development of basic science toward a clinically relevant application.

In addition to the strong translational research program, we have made a number of remarkable discoveries related to cardiac muscle development and adaptation. For example, in 2002 we reported that cell death proteins and pathways were in fact conserved features of skeletal muscle differentiation and were likely to be a conserved element for most forms of cellular differentiation (Fernando et al. 2002, Proc.Natl.Acad.Sci.USA). Although this observation was deemed to be very controversial at the time, similar observations have now been reported by dozens of labs around the globe. Early in 2008, we began to compile experimental evidence that similar cell death proteins are required for the normal maturation process in cardiac muscle cells. This is a provocative observation as cell death proteins and pathways have been uniformly considered to be key elements that promote cardiac muscle cell death. As such, our observations will require a major reconsideration in the field of basic cardiac research and suggest that the standard therapeutic targeting of such proteins and pathways may have significant unintended consequences for normal function and adaptation. We have presented these observations at a number of venues and have been encouraged by the very strong interest and excitement that has been generated. We are currently preparing the manuscript for submission to a high profile journal and anticipate publication/acceptance for publication within the coming year.