25 November 2010

Report to the Mach Gaensslen Foundation – 2010

Dr Lynn Megeney, The Mach Gaensslen Chair in Cardiac Research

Dear Board Members;

It gives me great pleasure to provide a highlight of the research activities from my laboratory over the preceding year. 2010 has been a most exciting time for the Megeney research group. In summary, my research team has published a number of landmark papers in 2010, some of which have garnered international attention. These papers follow the general theme of exploring the role of cell death proteins, specifically we are investigating the role that these critical factors have beyond simply killing cells. This is an area of research that we have pioneered during my tenure as the Mach Gaensslen Chair. We are now recognized as the global leaders in this most critical research area.

Early in the year we reported a very novel and exciting observation with significant implications for many fields of bio-medicine. In the paper of Larsen et al. (2010) we describe the observation that muscle stem cells require extensive DNA damage to mature into adult muscle cells/fibers (Published in prestigious science journal The Proceedings of the National Academy of Sciences USA). Briefly, we noted that during the cell maturation process there is widespread DNA damage and that if the damage process is blocked (by blocking the activity of cell death proteins/genes) the maturation process is dramatically curtailed. Prior to our observations it has been generally accepted that DNA damage causes either cell death outright or acts as a preemptor for turning a normal healthy cell into a cancer cell. Our observations suggest that DNA damage is a part of the normal cell maturation process, a finding that has already begun to dramatically alter our understanding of normal developmental processes as well as the origin of many cancer cell types. The paper was extensively covered in the national lay press and the international scientific community. One example of the former was an interview I conducted with the CBC science program Quirks and Quarks (which can be accessed from the CBC online site).

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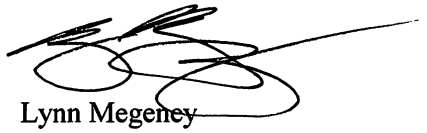
Following the work above, we published a second provocative manuscriptin which we describe a second unanticipated role for a cell death protein. In Lee et al. (2010) we report that cell death proteins in simple organisms like yeast are actually required for preventing the excessive build up or sticking together of normal proteins inside the yeast cell (This paper was also published in The Proceedings of the National Academy of Sciences USA). When we removed one cell death protein in particular, proteins piled up inside the cell and caused premature aging and cell death. These observations have significant implications for a number of human disease conditions in which the pathology arises from the formation of too many protein plaques inside the cell, including Alzheimer's, Hungtingtons and other related conditions. As such, our work suggests that a viable therapeutic strategy to treat these devastating conditions may involve targeted activation of these cell death proteins. As with the above paper of Larsen et al, this publication has generated considerable excitement in the field and spurned invitations to speak at a number of international scientific meetings.

Finally, I would like to extend a most sincere thank you to the Mach-Gaensslen Foundation for the support. The financial trust the Foundation has provided in the investment of my research program has been instrumental in supporting the advances we have made to date and will continue to make over the years to come.

Best Regards,



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