



The Role of MeCP2 in Neuron Development and Bone Homeostasis
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Rett Syndrome is a severe neurodevelopment disorder affecting young females. It is characterized by apparently normal early development followed by loss of purposeful use of the hands, slowed brain and head growth, seizures, osteoporosis, loss of speech, and mental retardation. Mutations in the gene, encoding methyl-CpG-binding protein 2 (MeCP2), are the main cause of this syndrome. Although Rett Syndrome is being studied worldwide, it is not known what specific role MeCP2 plays in neuron development and bone homeostasis. The focus of this project was to optimize staining procedures that would allow successful research in synapse formation and bone development in models of Rett syndrome. Immunofluorescence was used to differentially stain myocytes, which are used in co-culture with sympathetic neurons to study synapse formation. Several factors influencing staining outcome were examined to determine their effect on specificity and clarity of the fluorescence signal generated by an antibody to the muscle protein, actinin. In bone, histological techniques, such as safranin O/fast green staining, were used to analyze differences between wild-type mice and mice with a targeted deletion of the *Mecp2* gene. The results of these initial histological studies on bone will lead to further exploration in several areas of bone homeostasis. Detailed histological studies of these clinically relevant tissues will provide insight into how loss of MeCP2 affects the development and homeostasis of cells that are directly involved in the pathogenesis of this disorder. This project was funded by INBRE - Supported by NIH grant 2 P20 RR016472-06 from the NCRR and the Schanen/Twiss lab at A. I. Dupont Hospital for Children.



Tumor Necrosis Factor Alpha (TNF- α) Regulation of Reticulocalbin (RCN-1) Cell Surface Expression and Capillary-like Formation in Bone Marrow Endothelial Cells (BMECs)

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The goal of this project is to determine the effect that Tumor Necrosis Factor Alpha (TNF- α) has on Bone Marrow Endothelial Cells (BMECs). Specifically, Reticulocalbin (RCN-1) surface expression and capillary-like formation of the BMECs were examined. TNF- α is a pro-inflammatory cytokine known to be involved in inflammation and angiogenesis, while RCN-1 is a recently discovered surface protein whose function has not yet been reported. It is hypothesized that RCN-1 levels on the cell surface will be increased by TNF- α , while its effect on angiogenesis of BMECs is unknown. To determine RCN-1 surface expression, BMECs were given three different treatments of TNF- α . One group was given none, another group was treated for four hours with TNF- α and the third group was treated for twenty-four hours. The cells were treated with an RCN-1 anti-body, and then treated with a secondary fluorescent anti-body. Analysis with Fluorescence Activated Cell Sorting (FACS) shows that the levels of RCN-1 were noticeably increased on the BMECs treated with TNF- α . To determine the effect of TNF- α on capillary-like formation, BMECs were left untreated and treated with TNF- α for four hours. Each group was then grown on Matrigel® for twenty-four hours in plain media and media containing TNF- α . The cells were examined after the growth period, and only the BMECs that never were exposed to TNF- α showed significant capillary-like formation. This, along with the previously mentioned RCN-1 levels, suggests that up-regulation of RCN-1 on the BMEC cell surface by TNF- α results in a drastic decrease in angiogenesis. Funding for this project was provided by INBRE.

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