CDMD Muscle Cell Biology and Disease
Annual Scientific Retreat
Sponsored by the Center for Duchenne Muscular Dystrophy at UCLA
Tuesday November 29th, 2016 9AM-4:30PM

The retreat will start with coffee and pastries followed by research and clinical presentations, lunch, an afternoon poster session, and research award presentations. A networking happy hour with cocktails will end the retreat.

WE ARE NOW SOLICITING ABSTRACTS FOR POSTERS AND ORAL PRESENTATIONS.
DEADLINE TO SUBMIT IS NOVEMBER 2, 2016

ABSTRACT FORMAT:

1. Use arial 11pt font, single-spaced, left alignment.
2. Title, **in bold**, should not exceed 150 characters including spaces.
3. Title at the top of the abstract followed by authors.
4. Include all names of contributing authors in the format shown in the attached example (first name followed by last name for each author). **Presenting author should be in bold.**
5. Affiliations should be indicated with superscript numbers following each author. The affiliations should appear below the list of authors.
6. Abstract should follow affiliations and is limited to 300-words, including references.
7. Abstract should include background, objectives, approach, results, and conclusions although these headings are not required. NO figures or diagrams.
8. Include a blank line between each of the following: heading, author list, affiliations, abstract.
9. File should be a word document.
10. Filename should be in all caps: YOUR LAST NAME_YOUR FIRST NAME.doc
11. Complete the attached Abstract Submission Form for CDMD Annual Scientific Retreat.
12. Email the abstract as a word document and the completed form to amymartin@ucla.edu before 5PM November 2, 2016.
13. Late abstracts will NOT be accepted.

PLEASE BRING YOUR 4X4 POSTER TO THE RETREAT BY 9:00 AM on November 29, 2016.

We hope you will join us to learn about the latest research going on within the CDMD and share your research with others!

Identifying human- and disease-specific differences in human muscle glycosylation that affect muscle function.

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Adhesion complexes, including the dystrophin glycoprotein complex (DGC), the utrophin glycoprotein complex (UGC), and the α7β1 integrin heterodimer, connect the muscle cell membrane, or sarcolemma, with extracellular ligands. Specific and regulated glycosylation of molecules in these adhesion complexes is crucial for proper muscle function. For example, aberrant glycosylation of α-dystroglycan (α-DG), the laminin binding glycoprotein in both the DGC and UGC, results in sarcolemma instability, and mutations in genes required for proper α-DG glycosylation contribute to a spectrum of disorders known as dystroglycanopathies including Walker-Warburg syndrome and muscle-eye-brain disease.

While α-DG glycosylation has been fairly well characterized, studies profiling global differences in glycosylation between healthy and dystrophic muscle are very limited, and research done to date has only examined rodent muscle. However, specific differences in muscle glycosylation between humans and mice exist. For example, the plant lectin *Wisteria floribunda* agglutinin (WFA) has been used to correlate localization of the UGC at the mouse sarcolemma. However, in human muscle, WFA distribution at the sarcolemma does not correlate with utrophin localization. Thus, the primary goal of my project is to identify human- and disease-specific differences in muscle cell glycosylation, examining all glycoproteins on the sarcolemma. To identify differentiation- and disease-specific differences in glycosylation, the cell surface glycome of normal and patient-derived muscle cells, provided by the UCLA CDMD cell repository, was profiled using a ratiometric lectin microarray. In addition, glycosylation-related transcript expression in healthy and dystrophic myotubes and myoblasts was analyzed. As I identify differentiation- and disease-specific differences in human muscle glycosylation and glycosylation-related genes, I will determine how these differences affect muscle function, with the goal of identifying therapeutic approaches that will restore proper human muscle glycosylation, membrane stability, and sarcolemmal adhesion.