**RINH Invited Speaker Form**
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| **Name & job title:** | Professor David A. Mills, Peter J. Shields Endowed Chair, Department of Food Science & Technology |
| **Institution:** | University of California (Davis, CA) |
| **Talk title:** | Microbial colonization patterns in mammalian neonates—how milk "farms" the infant gut microbiota |
| **Date:** | Tuesday August 30th |
| **Seminar Abstract:** (provide URL if easier) | Human milk contains numerous components that shape the microbial content of the developing infant gastrointestinal tract. A prominent feature of milk is an array of complex glycans that serve a passive immune function by sequestering and deflecting pathogens while simultaneously enriching a protective, milk-oriented microbiota (MOM) often dominated by bifidobacteria. Recent research suggests the timing of establishment, and proper function of, a MOM is critical for infant development. An infant's MOM is initially established through environmental transfer to the gut and subsequently shaped by diet (milk) and host genetics. Once established, MOMs dominated by bifidobacteria exhibit low residual milk glycans and higher levels of short chain fatty acids in the faeces, suggesting a strongly saccharolytic colonic microbiota.<br><br>The mechanistic basis for milk glycan consumption by bifidobacteria has been the subject of active research. Different infant-borne bifidobacteria contain specific glycosidases and transport systems required to utilize free glycans or glycoconjugates. Consumption of milk glycans enhances specific bifidobacterial interaction with the infant host through both direct and indirect routes. Growth on free milk glycans results in increased bifidobacterial binding to epithelial cells and beneficially modulates intestinal function. In addition, metabolites generated during growth on milk glycans dampen inflammation and strengthen gut barrier function. In aggregate, these studies suggest a co-evolutionary relationship between mammalian milk glycans, infant-borne bifidobacteria and the infant host resulting in a programmed enrichment of a protective bifidobacterial-dominant MOM during a critical stage of infant development. Importantly, disruption of this programmed enrichment, by poor environmental transfer, antibiotic use, or infection, can lead to a "poorly functioning" MOM that may pose a risk for negative health outcomes. Further analysis of this naturally evolved system will shed light on effective pre- and probiotic tools that support and ensure a protective MOM for all at risk infants. |

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| **Speaker Biography:** (provide URL if easier) | http://mills.ucdavis.edu/david-mills |
| **Image:** |  |
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