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36TH CARDIOVASCULAR NURSING & NURSE PRACTITIONERS MEETING

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Origin and perpetuation of chronic low-grade inflammation as a driver for cardiovascular complications during metabolic syndrome

Statement of the Problem: Obesity associated cardiometabolic risk factors (CMetRF) is both a US and a worldwide epidemic and a major burden to healthcare system. Chronic low-grade inflammation (CLGI) is a well-established characteristic of the obese-human condition and conventionally, research has focused on the CLGI of liver and adipose tissue as a driver. Though, the gastrointestinal (GI) mucosa is the first tissue that interacts with dietary components and luminal microbiota both of which are known to regulate obesity associated CMetRF, the research on the role of GI-mucosa in obesity associated CMetRF has been ignored.

Methodology & Theoretical Orientation: Recent novel findings from my lab support a key role of Janus kinase 3 (Jak3), a non-receptor tyrosine kinase, in intestinal and systemic CLGI associated CMetRF in both an animal-model and in humans. De-identified and discarded tissue samples from CMetRF were analyzed for the markers of CLGI. The human model was reconstituted in mouse using tissue-specific genetic manipulation of non-receptor tyrosine kinase Jak3.

Findings: Our data show that in human CMetRF are associated with compromises intestinal expression and localization of not only Jak3 but also drug transport proteins. In mouse model, intestinal loss of Jak3 leads to colonic dysbiosis associated CLGI, obesity, and metabolic syndrome. Mechanistically, we show that Jak3 mediates intestinal tolerance through suppressed-expression and limited activation of intestinal TLR4/2. Moreover, pharmacological manipulation of both PI3K and TLR pathways in intestine leads to amelioration of CLGI and improvement of CMetRF.

Conclusion & Significance: This study showed that CLGI is associated with CMetRF where localized intestinal inflammation may play a major role in systemic manifestation of the inflammation leading to increased CMetRF. Moreover, intestinal regulation of inflammation may provide new avenue for a long-term reduction of CMetRF.

Biography

Narendra Kumar is a tenured Associate Professor and Chair of Graduate Program Committee at the College of Pharmacy at the Texas A&M University. He has completed his PhD from the Indian Institute of Technology (IIT) and postdoctoral training from the University of Tennessee Health science center. Previously, he held a joint appointment as instructor of Physiology and Pediatric Gastroenterology at the University of Tennessee HSC and Le Bonheur Children Medical Center respectively. He has rated among top 25% in the college by the students, he teaches immunology, biochemistry, pharmacogenomics, and autoimmune diseases under Pharm D program. In research he is the recipient of several national awards, such as Crohn's and Colitis Foundation of America (CCFA) Research Fellowship Award, CCFA Career Development Award, National Institute of Health (NIH) Basic Scientist Career Development Award, NIH small business innovation award (SBIR) and American Gastroenterology Association (AGA) Research Scholar Award to name a few. He has published more than 25 peer reviewed research articles, 50 abstracts, and a book chapter. He also has three patents to his credit, two of which are commercialized while the third is under negotiation for licensing.

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