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Alpha-enolase is up regulated on the cell surface and responds to plasminogen activation in mice expressing a delta133p53 alpha mimic

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The p53 tumor suppressor is an intrinsic part of the cellular stress response. To add to the complexity of understanding p53 function 12 p53 isoforms are produced, many of which have tumor promoting properties. The delta133p53alpha isoform lacks the N-terminal 133 amino acids due to an alternative promoter in intron four and it is aberrantly expressed in multiple tumors including breast and colon. Previous work attributed pro-inflammatory and proliferative properties todelta133p53alpha using a mouse model expressing a delta133p53alpha mimic (delta122p53). To identify the mechanism by which delta122p53 triggers inflammation the current study used a proteomic-based approach. The bone marrow, thymus, and lung proteome from Δ122p53, wild-type mice (p53+), and p53 null mice (p53-) were compared using two dimensional fluorescence difference gel electrophoresis and western blotting. In the bone marrow alpha-enolase was increased in delta122p53 cells. Further analysis showed alpha-enolase was increased in the cytosol and cell surface of delta122p53 bone marrow and peripheral blood mononuclear cells. Alpha-enolase on the delta122p53 peripheral blood mononuclear cell surface acted as a plasminogen receptor, with tumor necrosis factor alpha induced upon plasminogen stimulation. Taken together, these data identified new proteins associated with p53 function. One of which, alpha-enolase, is regulated differently by delta122p53.Increased cell surface alpha-enolase function with delta122p53 provides a possible explanation for the model's pro-inflammatory features and suggests the delta133p53 alpha isoform may direct an inflammatory response by increasing the amount of alpha-enolase on the cell surface.

Biography

Tania Slatter completed her Doctorate in Biochemistry at the University of Otago, New Zealand in 2007. Her current position is as a Senior Research Fellow in the field of molecular pathologyuses animal models and human clinical samples to investigate how inflammation contributes to different pathologies including cancer. She has published 18 papers in reputed journals.

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Protective anti-inflammatory responses mediated by a commensal microbial molecule require both innate and cognate interactions

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The microbiota is critical in shaping the mammalian host's immune system. Polysaccharide A, the archetypical immunomodulatory microbial molecule of the gut commensal Bacteroides fragilis, induces regulatory T cells to secrete the anti-inflammatory cytokine interleukin 10. We show, in a model of colitis, that PSA requires both innate and adaptive immunity to generate protection. Dendritic cells mediate PSA's effect on IL-10 production. Unlike conventional DCs, plasmacytoid DCs exposed to PSA do not produce the proinflammatory cytokines tumor necrosis factor-α and IL-12 but PDCs do specifically stimulate IL-10 secretion by CD4+ T cells and efficiently mediate PSA-mediated immunoprotection. PSA induces and preferentially ligates Toll-like receptor 2 on PDCs but not on CDCs. Compared with other TLR2 ligands, PSA better enhances PDC expression of co-stimulatory molecules required for protection against colitis. PDCs orchestrate beneficial immunoregulatory interaction of commensal microbial molecules with CD4+ T cells through both innate and adaptive immunity.

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