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POSTER PRESENTATIONS

P-0867

Impaired respiratory burst contributes to infections in PKCδ-deficient patients

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Patients with inherited autosomal recessive (AR) protein kinase C δ (PKCδ) deficiency suffer from childhood-onset autoimmunity. They also suffer from recurrent infections that overlap with those observed in patients with chronic granulomatous disease (CGD). Although their immunosuppressive treatment is a cofounding factor, we hypothesized that PKCδ-deficient patients may be intrinsically susceptible to infections. We studied the NADPH oxidase activity in EBV-B cells, primary and monocyte-derived phagocytes in a large international cohort of PKCδ-deficient patients. The patients' EBV-B cells produced little amounts of reactive oxygen species (ROS) and did not phosphorylate p40*phox* normally after PMA or opsonized *S. aureus* stimulation. Both phenotypes were restored by retrotransduction with wild-type *PRKCD* cDNA. The patients' circulating and monocyte-derived phagocytes displayed low levels of ROS production as well as a markedly reduced neutrophil extracellular trap formation and impaired phosphorylation of p40*phox*. Our results suggest a role for PKCδ in the activation of the NADPH oxidase complex through phosphorylation of p40*phox*. Patients with AR PKCδ deficiency have an impaired NADPH oxidase activity in various myeloid subsets, which may contributes to their CGD-like infectious phenotype.

Keywords: Immunodeficiency, infectious disease, molecular immunology

P-0868

Innate and adaptive immune activation contribute to radiotherapy-mediated tumour control in lung cancer

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Radiotherapy is widely used in lung cancer treatment but its role in regulating immune responses is only more recently appreciated. While activation of the adaptive immune system has been shown to be important in multiple mouse models, this has not been well characterised in the context of lung cancer. Furthermore, the innate immune response to lung radiotherapy remain unclear. Our work explored the activation and interplay of innate and adaptive immunity in response to targeted radiotherapy in the lung. We established a regiment of targeted lung irradiation using the Small Animal Radiation Research Platform (SARRP), which allowed specific targeting of the left lung lobe in mice, and used flow cytometry to quantify immune changes upon targeted radiotherapy. We demonstrated the effectiveness of this regiment in inducing significant reduction in tumour burden in the B16-OVA model of lung metastasis, and activation of dendritic cells (DCs) and adaptive CD4+ and CD8+ T-cells were observed by flow cytometry. Targeted radiotherapy in RAG-deficient mice led to reduced anti-tumour responses, confirming the importance of adaptive immunity. Additionally, we observed activation of innate natural killer (NK) cells, and antibody-mediated depletion of NK cells reduced the effectiveness of radiotherapy. We also identified activation of macrophages and Group 2 innate lymphoid cells (ILC2s). Our results highlighted the importance of both innate and adaptive immunity in the anti-tumour response induced by lung radiotherapy. These findings may inform therapeutic strategies to target these cell types alongside radiotherapy in lung cancer. We are now investigating potential crosstalk between these cell types.

Keywords: Adaptive immunity, cancer immunology, innate immunity

P-0870

Oral cadmium increases contact hypersensitivity reaction in rats

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Cadmium (Cd) in food and drinking water presents a health risk to the general population. We have shown previously that orally-acquired Cd affects basal immune homeostasis in the skin. In this study, we examined the effect of 30-days oral exposure of inflammatory disease-prone Dark Agouti (DA) rats to two environmentally relevant Cd doses (5 and 50 ppm) on contact hypersensitivity reaction (CHS) induced by topical 0,4% dinitrochlorobenzene (DNCB). Both Cd doses increased proinflammatory epidermal cell response (IL-1, TNF and IL-6 production) to DNCB sensitization, as well as epidermal cells' potential to stimulate naïve lymphocytes *ex vivo* (increased IFN-γ and IL-17 production in co-cultures). The proinflammatory milieu of epidermal cells induced by sensitization was accompanied by increased hapten-specific production of IFN-γ (at a lower Cd dose) and IL-17 (at both Cd doses) by draining lymph node (DLN) cells, compared to Cd non-treated animals. During the challenge phase of CHS, oral Cd increased ear swelling response and skin inflammation (edema, mononuclear and neutrophil cell infiltration) at both Cd doses, what correlated with increased innate (TNF) and hapten-specific effector (IFN-γ, IL-17) cytokine response by ear cells. Even in Albino Oxford (AO) rats generally less prone to inflammation, oral Cd increased the proinflammatory response of epidermal cells following sensitization, however, DLN cell responses were absent. Ear swelling response to hapten challenge was observed in AO individuals which acquired a higher Cd dose. Presented data imply the potential of food- and water-borne Cd to be risk factors for skin disease development and/or its exacerbation.

Keywords: Animal models, inflammatory disease, modification allergic responses, skin diseases