

ISEV2021 Abstract Book

About ISEV

The International Society for Extracellular Vesicles is the leading professional society for researchers and scientists involved in the study of microvesicles and exosomes. With nearly 1,000 members, ISEV continues to be the leader in advancing the study of extracellular vesicles. Founded in 2012 in Sweden, ISEV has since moved its Headquarters to the United States. Through its programs and services, ISEV provides essential training and research opportunities for those involved in exosome and microvesicle research.

Mission Statement

Advancing extracellular vesicle research globally.

Vision

Our vision is to be the leading advocate and guide of extracellular vesicle research and to advance the understanding of extracellular vesicle biology.

ISEV2021 Annual Meeting

The International Society for Extracellular Vesicles is the premier international conference of extracellular vesicle research, covering the latest in exosomes, microvesicles and more. With an anticipated 1,000 attendees, ISEV2021 will feature presentations from the top researchers in the field, as well as providing opportunities for talks from students and early career researchers.

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Summary/Conclusion: We set up a combination of two simple in vitro functional assays, representative of both innate and acquired immunity, to assess the immunomodulatory effects of MSC-EVs. Although these tests need to be further evaluated on a large scale, we propose that the use of cell lines with a positive internal control (Dex) should ensure both adequate precision and robustness.

PS01.12 | Immunomodulation by extracellular vesicles from *Trichinella spiralis* muscle larvae: increasing tolerogenic properties of human dendritic cells

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Introduction: Excretory-secretory products (ES) of parasitic worms (helminths) shift hosts' immunological balance toward Th2 and regulatory responses thus acting beneficial in chronic inflammations, i.e. autoimmune diseases. As shown in several helminths, extracellular vesicles (EVs) are active immunomodulatory component of ES. We found that *Trichinella spiralis* produces EVs (TsEVs) which influence cytokine production by PBMC. Now we aim to show how TsEVs influence human dendritic cells (DC), as key players in initiation, progression and regulation of immune response.

Methods: EVs were enriched from conditioned medium of *T. spiralis* muscle larvae (ES L1) by differential centrifugation. Human monocyte derived dendritic cells (DCs) were treated with TsEVs and subsequently co-cultivated with allogenic T cells. Phenotypes and cytokine production of DC and T cells were determined by flow cytometry.

Results: TsEVs induce stable tolerogenic phenotype of DCs, reflected in the expression of surface markers (HLA-DR, CD-40, CD-86) almost at the level of the control, except for slight elevation in the surface CD-83, and significantly increased ILT-3 and CCR-7. Stimulated DCs produce significant amounts of IL-10 and TGF- β , and polarize immune response of T cells towards Th2 and regulatory type. T cells co-cultured with TsEVs stimulated DCs show significant increase in the production of IL-4 and IL-10 with the production of IFN- γ at the level of control. Moreover, TsEVs stimulated DCs induce expansion of CD4+CD25+Foxp3+ regulatory T cells.

Summary/Conclusion: TsEVs influence viability, differentiation, maturation potential of DCs and their capacity to regulate T cell-mediated immune response, similar as ES L1 of *T. spiralis* do. They induce tolerogenic phenotype of DCs and regulatory response of T cells. Starting from this capacity of TsEVs to convey immunomodulatory properties of ES L1, new therapeutics, based on TsEVs could be designed as novel therapy for autoimmune diseases.

PS01.13 | Air pollution Particulate Matter and EVs: involvement of PM-fraction and PM-activated toxic signaling pathways in EVs released by pulmonary epithelial cells

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Introduction: Poor air quality associated with high levels of particulate matter (PM) is one of the five greatest environmental risks for health, causing millions of premature deaths each year (cardiovascular diseases, cancer, COPD,...). PM is composed of hundreds of different chemicals, such as organic (Polycyclic Aromatic Hydrocarbons, PAHs) and inorganic or hydrosoluble compounds (ions and metals), as well as biological species (bacteria). Due to their diameter $< 2.5 \mu\text{m}$, PM_{2.5}, or fine particles, can penetrate deep into the lung alveoli. Exposure of lung epithelial cells to PM_{2.5} triggers the activation of toxic pathways such as: (1) the AhR signaling pathway involved in the metabolic activation of PAHs, (2) the TLR4 signaling pathway involved in the inflammatory response, and (3) the production of Reactive Oxygen Species (ROS), which cause severe damage to cellular macromolecules. Numerous studies also show that PM_{2.5} induces the secretion of EV by exposed cells, but neither the fraction of PM nor the signaling pathways involved are known. Answering these questions is the objective of this study.

Methods: First, BEAS-2B lung epithelial cells were exposed to PM_{2.5} and their organic and hydrosoluble extracts for 24 and 48 hours. Second, in order to determine the signaling pathways involved, BEAS-2B cells were pre-treated prior to exposure to PM_{2.5} with optimized concentrations of the following three specific inhibitors: CH223191 (AhR antagonist), TAK-242 (TLR4