



POSTER PRESENTATION

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Acrolein: blocking antibody formation: pro tumor, anti-allergy

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Background

Allergic sensitization has been linked to active and passive smoking in exposed individuals and even their pets. We here investigated the contribution of acrolein, a compound generated in large amounts during smoking, during nasal sensitization and – based on the surprising preliminary results – on tumor growth. As a model antigen we used KLH with or without acrolein.

Methods

BALB/c mice were nasally sensitized 5 times in biweekly intervals with KLH alone or with KLH in conjunction with acrolein. Airway hyperreactivity was measured according to change of enhanced pause and KLH-specific anaphylactic reaction was monitored in vivo. Levels of specific antibodies as well as cytokine profile of KLH-stimulated splenocytes were analyzed by ELISA. Further, mouse D2F2-tumor cells were grafted to the flanks and tumor growth monitored in mice previously exposed to acrolein or buffer.

Results

Nasal application of KLH as model antigen induced specific IgG1-, IgG2a-, IgA- and IgE-levels. The same mice secreted elevated levels of IL5, IL13, IL10 and IFN- γ . They showed increased airway-hyperreactivity and had a significant drop in body temperature upon allergen challenge. Pointing towards tolerance, and against our expectations, presence of acrolein in the KLH-antigen significantly reduced specific antibody-titers, resulted in lower splenocyte cytokine production and prevented

anaphylaxis. However, the impaired immune response simultaneously led to a significantly higher tumor growth in mice exposed to acrolein than in the control group.

Conclusion

Acrolein in smoke – best known for its carcinogenic effect - decreases the risk of sensitization towards a specific antigen by inhibiting immune activation. Our data further suggest that Acrolein via the same mechanism acts tumor promoting in smokers.

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