



MEETING ABSTRACT

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OR11-005 - Mast cells respond to pathogen signals with IL-1 β

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Introduction

Mast cells, key effector cells of allergic and innate immune responses, have recently been reported to be an important source of IL-1 β in patients with autoinflammatory conditions such as cryopyrin-associated-periodic-fever syndromes (CAPS). CAPS patients show IL-1 β -driven systemic inflammation together with non-histamine dependent urticarial rash, which are caused by activating mutations of the inflammasome, a multiprotein oligomer responsible for the initiation of inflammatory responses to pathogens.

Objectives

To determine if mast cells can produce and release IL-1 β in response to pathogenic signals that target the inflammasomes NLRP3, NLRC4, or AIM2.

Methods

Peritoneal mast cells (PMCs) were obtained through lavage from adult (>8 weeks) C57BL/6 mice and WBB6F1 Kit^{+/+} mice, purified via CD117⁺ bead selection (>96 % purity) and cultured for 7-14 days. 10⁵ cells/well were primed with LPS (100ng/ml) for 15 hrs. Then the PMCs were stimulated with 10 μ M Nigericin (NLRP3), 5mM ATP (NLRP3), 100 μ M R837 (NLRP3) for 45 min or for 4 hours with 600 ng Flagellin (NLRC4) transfected with DOTAP or 200 ng polydAdT (AIM2) transfected with Lipofectamine. IL-1 β production was measured in the supernatants by Elisa.

Results

PMCs produced significant amounts (mean \pm SEM) of IL-1 β upon stimulation with Nigericin (467 \pm 41pg/ml), ATP (152 \pm 88pg/ml), R837 (21 \pm 2 pg/ml), Flagellin

(245 \pm 44pg/ml) and polydAdT (571 \pm 194pg/ml) as compared to no stimuli (7,1 \pm 0,8 pg/ml) only.

Conclusion

We show that mouse mast cells incubated with inflammatory activators produce significant amounts of IL-1 β ex vivo. Our data suggest that inflammasome-driven mast cell activation and subsequent IL-1 β production and release may importantly contribute to innate immune responses to pathogens.

Competing interests

None declared.

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