

Sekresi interleukin 13, interleukin 10, interferon y, dan indoleamine 2,3-dioxygenase dari kultur sel mononuklear darah tepi pasien asma alergi yang dipajan alergen dermatophagoides pteronyssinus = Secretion of interleukin 13, interleukin 10, interferon y, and indoleamine 2,3-dioxygenase from peripheral blood mononuclear cell culture of allergy asthma patients exposed by dermatophagoides pteronyssinus allergen / Cityta Putri Kwarta

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Abstrak

ABSTRAK

Asma alergi merupakan penyakit atopi degeneratif yang disebabkan alergi atau hipersensitifitas tipe-1. Lebih dari 50% penderita asma alergi disebabkan adanya reaksi hipersensitif terhadap alergen Tungau Debu Rumah (TDR). Skrining subjek penelitian berdasarkan manifestasi asma dan Skin Prick Test (SPT) didapatkan 25 subjek atopi asma yang disebabkan alergi terhadap alergen TDR dan 21 subjek nonatopi. Respon imunitas seluler dievaluasi melalui teknik kultur Kultur sel mononuklear darah tepi (SMDT) yang diisolasi dari darah menggunakan teknik ficoll gradient. Kultur SMDT dari masing-masing subjek distimulasi dengan Alergen TDR, PHA (kontrol positif), dan RPMI (kontrol negatif) selanjutnya diinkubasi dalam inkubator CO₂ 5%, 37°C selama 72 jam. Dengan metode multiplex assay, supernatan hasil kultur dilakukan pengukuran IFNγ untuk menilai mediator proinflamasi tipe-1, Interleukin 13 (IL-13) untuk menilai mediator proinflamasi tipe-2, dan IL-10 sebagai anti inflamasi serta kadar Indoleamine 2,3-Dioxygenase (IDO) diukur dengan metode ELISA Sandwich. Terdapat peningkatan rasio sitokin proinflamasi tipe-2 (IL13) terhadap anti inflamasi (IL10) dan penurunan rasio sitokin proinflamasi tipe-1 (IFN) terhadap proinflamasi tipe-2 (IL-13) yang dihasilkan dari kultur SMDT pada kelompok atopi asma dibandingkan dengan kelompok nonatopi. Perubahan pola keseimbangan mediator pro inlamasi tipe-1, tipe-2 dan anti inflamasi pada subjek asma alergi diduga mempengaruhi produksi IDO yang ditemukan secara signifikan lebih rendah dibanding subjek non atopi.

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ABSTRACT

Allergic asthma is degenerative atopy caused by type 1 allergic or hypersensitivity. More than 50% of people with allergic asthma are caused by hypersensitivity reactions to house dust mite allergens (HDM). Screening of research subjects based on asthma manifestations and Skin Prick Test (SPT) found 25 subjects with atopic asthma caused by allergies to TDR allergens and 21 nonatopic subjects. The cellular immune response was evaluated through a culture of peripheral blood mononuclear cell culture (PBMC) technique isolated from blood using the ficoll gradient technique. PBMC cultures from each subject were stimulated with HDM allergens, PHA (positive control), and RPMI (negative controls) then incubated in a 5% CO₂ incubator, 37°C for 72 hours. With the multiplex assay method, IFNγ measurements were carried out by the culture supernatant to assess type 1 proinflammatory mediator, Interleukin 13 (IL-13) to assess type 2 proinflammatory mediators, and IL-10 as anti-inflammatory and Indoleamine 2,3-Dioxygenase levels (IDO) is measured by the ELISA Sandwich method. There was an increase in the ratio of type-2

(IL13) proinflammatory cytokines to anti-inflammatory (IL10) and a decrease in type-1 (IFN) proinflammatory cytokine to proinflammatory type-2 (IL-13) resulting from PBMC culture in the asthma atopy group compared to the nonatopic group. Changes in the balance pattern of type 1, type-2 and anti-inflammatory pro-inflammatory mediators in allergic asthma subjects suspected to affect IDO production were found to be significantly lower than non-atopy subjects.