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(Jessica Hefiama Jaya, Achmad Basori, Sudarno)

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SOOT PARTICULATE EXPOSURE INCREASES CD54/INTERCELLULAR ADHESION MOLECULE-1 (ICAM-1) EXPRESSION IN CARDIOVASCULAR DISORDER

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Abstract

Exposure of air pollution has become a risk factor for cardiovascular disease. One mechanism thought to contribute is oxidative stress, which will increase the reactive oxygen species resulting in release of pro-inflammatory cytokines and increased expression of adhesion molecules such as intercellular adhesion molecule-1 (ICAM-1). The aim of this study was to describe the effects of soot particulate (carbon black powder) exposures on ICAM-1 expressions in mechanism of cardiovascular disorders. Experiments were performed on white female rats (Rattus novergicus) divided into 3 groups: control group (P0) (n=12), without exposure to soot particulate; an treatment group 1 (P1) (n=12), exposed to soot particulate concentration of 532 mg/m3 one hour/day for 30 days; and an treatment group 2 (P2) (n=12), exposed to soot particulate concentration of 1064 mg/m3 one hour/day for 30 days. Immunohistochemical staining was used to quantify the expression of ICAM-1 in cardiac tissues. We quantified the expressions of ICAM-1 with the number of immunoreactive cells in 5 fields of view. The mean ICAM-1 expressions were different between P0, P1 and P2 (4.9, 21.47, 36.33). Expression of ICAM-1 was statistically significant between three groups (p=0,001). The increase of ICAM-1 expression in P0 compared to P1 (p=0,019), P0 compared to P2 (p=0,000) and P1 compared to P2 (p=0.045) were significant.

Exposures to soot particulate matter increased the ICAM-1 expression in subjects significantly. Our findings suggest a key role of activation inflammatory pathway in response to soot particulate exposure in cardiovascular disease.

Keyword : soot, particulate, cardiovascular, disorders, oxidative, stress, ICAM-1,

Daftar Pustaka :