[PA4-29] [04/20/2001 (Fri) 10:30 - 11:30 / Hall 4]

Down-regulation of naringenin on iNOS, IL-1βand IL-6, in murine macrophages

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In this study, we investigated the effect of naringenin on the regulation of iNOS and proinflammatory cytokines, such as, IL-1β, IL-6, in murine macrophages. Naringenin alone did not affect the expression of iNOS and proinflammatory cytokines; in contrast, treatment of suppressed the LPS-induced gene expression of IL-1β, IL-6 and iNOS, in a dose-dependent manner as determined by RT-PCR analysis. NO production was assessed by measurement of nitrites in the medium. The level of NO was found to correlate well with a decrease in transcripts of iNOS. Since the promoter in IL-1βand iNOS gene contains binding motifs for NF-kB, the effect of naringenin on the inactivation of this transcripts factor was determined by transient transfection assay. Employing a transfection and reporter gene expression system with p(NF-kB)3-Luciferase, the treatment of naringenin produced a dose-dependent inhibition of luciferase activity in RAW 264.7 murine macrophages cell line. These results suggest that suppression of iNOS, IL-1β, and IL-6 gene expression by naringenin might be mediated by the inhibition of NF-kB activation[This work was supported by KFDA Grant and RCPM from KOSEF].

[PA4-30] [04/20/2001 (Fri) 10:30 - 11:30 / Hall 4]

Suppression of nonylphenol on iNOS, IL-1βand IL-6 in murine macrophages

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4-Nonylphenol is a degradation product of a widely used non-ionic surfactant group, alkylphenol polyethoxylates that are mainly found as an intermediate in the chemical manufacturing industry. In this study, we investigated the effect of nonylphenol on the regulation of iNOS and proinflammatory cytokines, such as, IL-1β, IL-6 in murine macrophages. Nonylphenol alone did not affect the expression of iNOS and proinflammatory cytokines; in contrast, treatment of suppressed the LPS-induced gene expression of IL-1β, IL-6, and iNOS, in a dose-dependent manner as determined by RT-PCR analysis. NO production was assessed by measurement of nitrites in the medium. The level of NO was found to correlate well with a decrease in transcripts of iNOS. Since the promoter in IL-1βand iNOS gene contains binding motifs for NF-kB, the effect of nonylphenol on the inactivation of this transcripts factor was determined by transient transfection assay and electrophoretic mobility shift assay (EMSA). Employing a transfection and reproter gene expression system with p(NF-kB)3-Luciferase, the treatment of nonylphenol produced a dose-dependent inhibition of luciferase activity in RAW 264.7 murine macrophages cell line. Using DNA fragments containing the NF-kB binding sequence, nonylphenol was shown to inhibition the protein/DNA binding of NF-kB to its cognate site as measured by EMSA. These results suggest that suppression of iNOS, IL-1β, and IL-6 gene expression by Nonylphenol might be mediated by the inhibition of NF-kB activation[This work was supported by KFDA Grant and RCPM from KOSEF].

[PA4-31] [04/20/2001 (Fri) 10:30 - 11:30 / Hall 4]

Suppression of bisphenol A on iNOS, IL-6 and TNF-αin murine macrophages

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In this study, we investigated the effect of bisphenol A on the regulation of iNOS and proinflammatory cytokines, such as, IL-6 and TNF-αin mouse peritoneal macrophages. Bisphenol A alone did not affect the expression of iNOS and proinflammatory cytokines; in contrast, treatment of bisphenol A suppressed the LPS-induced gene expression of IL-6, TNF-α and iNOS, in a dose-dependent manner as determined by RT-PCR analysis. Bisphenol A was shown increased TNF-α gene expression in a dose-dependent manner. NO production was assessed by measurement of nitrites in the medium. The level of NO was found to correlate well with a decrease in transcripts of iNOS. Since the promoter in TNF-α and iNOS gene contains binding motifs for NF-kB, the effect Bisphenol A on the inactivation of this transcripts factor was determined by transient transfection assay and electrophoretic mobility shift assay (EMSA). Employing a transfection and reporter gene expression system with p(NF-kB)3-Luciferase, the treatment of bisphenol A produced a dose-dependent inhibition of luciferase activity in RAW 264.7 murine macrophages cell line. Using DNA fragments containing the NF-kB binding sequence, nonylphenol was shown to inhibition the protein/DNA binding of NF-kB to its cognate site as measured by EMSA. These results suggest that suppression of iNOS and IL-6 gene expression by bispheonl A might be mediated by the inhibition of NF-kB activation [This work was supported by KFDA Grant and RCPM from KOSEF].

[PA4-32] [04/20/2001 (Fri) 10:30 - 11:30 / Hall 4]

Down-regulation of iNOS, TNF-a and IL-6 gene expression by genistein in murine macrophages.

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The phytoestrogen and principal isoflavone in soy, genistein, has adverse effects on reproductive physiology in rodents. Therefore, in this study, we investigated the effect of genistein on the of iNOS and proinflammatory cytokines, such as, IL-6 and TNF-αin murine macrophages. Genistein alone did not affect the expression of iNOS and proinflammatory cytokines; in contrast, treatment of genistein suppressed the LPS-induced gene expression of IL-6, TNF-αand iNOS, in a dose-dependent manner as determined by RT-PCR analysis. NO production was assessed by measurement of nitrites in the culture medium. The level of NO was found to correlate well with a decrease in transcripts of iNOS. Since the promoter in TNF-αand iNOS gene contains binding motifs for NF-kB, the effect genistein on the inactivation of this transcripts factor was determined by transient transfection assay. Employing a transfection and reporter gene expression system with p(NF-kB)3-Luciferase, the treatment of genistein produced a dose-dependent inhibition of luciferase activity in RAW 264.7 murine macrophages cell line. These results suggest that suppression of iNOS, TNF-α, and IL-6 gene expression by genistein might be mediated by the inhibition of NF-kB activation [This work was supported by KFDA Grant and RCPM from KOSEF].

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Down-regulation of murine Cyp1a-1 in mouse hepatoma Hepa-1c1c7 cells by genistein

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