



POSTER PRESENTATION

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Modulation of oxidative stress and TNF α secretion by peritoneal macrophages of arthritic rats fed with a flavonoid-enriched diet

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Introduction

The inflammatory response increases the oxidative stress and cytokine production, such as TNF α , in macrophages. Flavonoids, present in vegetables, have potential antioxidant and anti-inflammatory properties. Previous studies have shown that a flavonoid-enriched cocoa extract added to macrophages *in vitro* decreases the secretion of NO and TNF α [1].

Aim

The objective of the present study was to ascertain the production of oxidant products and a pro-inflammatory cytokine (TNF α) by peritoneal macrophages obtained from arthritic rats after a long-term cocoa diet.

Methods

Female LOU rats were fed with a 10% cocoa diet or standard chow. After 2 weeks of diet, collagen-induced arthritis (CIA) was induced in part of animals from each group. One month later, peritoneal macrophages were obtained by injecting ice-cold sterile PBS into the peritoneal cavity. Cells were cultured and stimulated by addition of 1 μ g/mL LPS. TNF α secretion was quantified by ELISA in 24 h supernatants. In the same samples, NO $_2^-$ concentration (stable end product of NO) was measured by a modification of Griess reaction. To determine intracellular ROS production, macrophages were incubated with reduced 2',7'-dichlorofluorescein diacetate (H $_2$ DCF-DA) for 30 min at 37 °C. Fluorescence of DCF oxidized by ROS was measured by fluorimetry every 30 min up to 2 h.

Results

Macrophages from CIA animals fed cocoa decreased TNF α and NO production by ~57% and ~36% with respect to those from CIA reference group under LPS-stimulation conditions (P<0.05). ROS production by macrophages from CIA animals fed standard diet was higher with respect to healthy reference animals (P<0.05). Cocoa diet avoided ROS secretion increase in CIA animals (P<0.05), raising similar values to healthy animals.

Conclusion

A long-term cocoa diet reduced the production of oxidants and TNF α in peritoneal macrophages obtained from collagen-induced arthritic rats.

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Reference

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