Cellular uptake and anti-inflammatory effects of palm oil-derived delta (δ)-tocotrienol in microglia

ABSTRACT

Tocopherols long dominated studies on vitamin E, although interest has shifted to tocotrienols. It was previously shown that δ-tocotrienol derived from palm oil reduced nitric oxide released by BV2 microglia as early as 18 h after lipopolysaccharide stimulation. The current study measured δ-tocotrienol uptake by BV2 over a 24 h incubation period and its anti-inflammatory effects on primary microglia. Uptake of 17.5 μg/mL δ-tocotrienol by BV2 microglia began as early as 5 min and rose steeply to 21 ± 3% of the amount administered at 24 h. The amount of δ-tocotrienol retained in the lipopolysaccharide-stimulated microglia at 24 h was 14 ± 2%, with no substantial difference seen in unstimulated microglia. The same δ-tocotrienol regimen reduced nitric oxide levels by 82% at 24 h after lipopolysaccharide stimulation ($p < 0.05$). This was accompanied by decreased inducible nitric oxide synthase protein expression by 67 ± 5% compared to untreated controls ($p < 0.05$). In primary microglia, δ-tocotrienol downregulated IL-1β production, but TNF-α and IL-6 were not affected. δ-Tocotrienol also reduced prostaglandin E2 production by ~78%% and decreased transcription of COX-2 and 5-LOX, but not COX-1. This study showed the anti-inflammatory effects of δ-tocotrienol derived from palm oil and opens up interest for tocotrienol supplementation to reduce the effects of inflammatory conditions.

Keyword: Microglia; Inflammation; Palm oil; Delta-tocotrienol