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Objectives: This study investigated the anti-inflammatory effects of anthocyanins and flavan-4-ols (precursor of phlobaphenes) within a whole-food matrix against dextran sulfate sodium (DSS)-induced colitis using four maize near-isogenic lines (NILs) that differ only in a single class of flavonoids. The four NILs are A (lacks anthocyanins and phlobaphenes), B (phlobaphenes +), C (anthocyanins +) and D (anthocyanins + and phlobaphenes +).

Methods: Conventional male C57BL6 mice were subjected to control diet 25% A, B, C, and D supplemented corn diet. Germ-free (GF) C57BL6 mice were subjected to either control or 25% D supplemented diet. Colitis was induced in both conventional and GF mice by 3% and 1.25% (w/v) DSS in the drinking water, respectively. Intestinal permeability was measured using FITC-dextran. RT-PCR was used to analyze the gene expression levels and 16S rRNA for bacterial relative abundance.

Results: In conventional mice, supplementation of A, B, C, and D prevented DSS-induced colon shortening and body weight loss compared to mice on the control diet ($P < 0.05$). Mice supplemented with B, C, D diets had lower gut permeability than DSS mice ($P < 0.05$). The mRNA expression level of pro-inflammatory interleukin-6 (IL-6) was suppressed in B, C and D supplemented mice whereas, interleukin 1 β (IL-1 β) expression was lowered in mice supplemented with C and D diets only. Supplementation of the four NILs decreased the abundance of the genus *Pseudomonas* in colitic mice ($P < 0.05$). In GF mice, D diet ameliorated DSS-induced colon shortening and elevated gut permeability. The expression level of IL-6 and IL-1 β were downregulated in GF mice fed with D diet ($P < 0.05$).

Conclusions: In summary, flavan-4-ols and/or anthocyanins in the whole-food matrix exerted their protective effects against DSS-induced chronic inflammation and concomitantly restored intestinal barrier function. GF mice experiment results demonstrated that above mentioned beneficial effects from bioactive corn compounds were partially independent of gut microbiota.

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