Food and Chemical Toxicology
Volume 158, December 2021, 112654

MiR-182–5p/TLR4/NF-κB axis contributes to the protective effect of caffeic acid phenethyl ester against cadmium-induced spleen toxicity and associated damage in mice

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Received 15 September 2021, Revised 23 October 2021, Accepted 26 October 2021, Available online 29 October 2021.

Handling Editor: Dr. Jose Luis Domingo

https://doi.org/10.1016/j.fct.2021.112654

Highlights

- Cadmium causes spleen damage via inducing inflammation and apoptosis in mice.

- Cd exposure increases the levels of TLR4 and decreases the level of miR-182–5p.
- CAPE plays a protective role against Cd induced spleen injury via TLR4/NFκB pathway.
- MiR-182–5p targets TLR4 to involve in the protective process of CAPE.

Abstract

Cadmium (Cd) is a toxic heavy metal pollutant that can be accumulated in organs including the spleen, thereby threatening human health. In this study, the effect of caffeic acid phenethyl ester (CAPE, a bioactive component of honeybee propolis) on $CdCl_2$-induced spleen toxicity and underlying mechanisms were examined in mice. Histological examinations revealed that CAPE (10 μmol/kg/day b.w.) could mitigate spleen damage induced by $CdCl_2$ (1.5 mg/kg/day b.w.) in mice. Compared to the mice treated only by $CdCl_2$, CAPE administration increased the body weight while decreasing the spleen weight, spleen Cd content and spleen to body ratio of the $CdCl_2$-treated mice. Western blot and ELISA tests revealed that CAPE suppressed $CdCl_2$-induced inflammation (indicated by the decreases in the levels of inflammatory indictors). TUNEL and Western blot results showed that CAPE suppressed $CdCl_2$-induced apoptosis through reducing the percentage of TUNEL-positive cells and regulating apoptosis factors. The antagonistic effect of CAPE against $CdCl_2$-induced spleen toxicity was realized by increasing miR-182–5p expression to regulate the TLR4/NF-κB pathway. Therefore, CAPE could be a food-derived spleen protector to counteract Cd-induced spleen toxicity through alleviating apoptosis and inflammation *via* the miR-182–5p/TLR4/NF-κB axis.

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Keywords

Caffeic acid phenethyl ester; miR-182–5p; TLR4; Cadmium; Spleen damage

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