LETTER TO THE EDITOR

Berberine reduces insulin resistance: the roles for glucocorticoid receptor and aryl hydrocarbon receptor

To the Editor:

We read with interest the article by Zhao et al. (1) that showed synthetic glucocorticoid dexamethasone-induced insulin resistance in theca cells is diminished by berberine.

We speculate that the effects of berberine described by Zhao et al. could be explained by berberine interactions with the aryl hydrocarbon receptor (AhR) and consequently could involve crosstalk between AhR and the glucocorticoid receptor (GR). The following facts support our hypothesis.

First, the biologic effects of dexamethasone occur primarily through the GR by transcriptional regulation of various genes. It has been demonstrated that berberine has no effect on the expression and transcriptional activity of human GR (2). Therefore, the effects of berberine on dexamethasone-induced insulin resistance probably do not involve direct interaction between berberine and GR.

Second, berberine is an activator of AhR, and it induces expression of AhR-dependent genes such as CYP1A1 in human and rat cells (3).

Third, mutual interactions between AhR and GR have been described in HepG2 and HeLa human cancer cell lines (4, 5) and in human hepatocytes (6, 7). It was reported that AhR activation by compounds such as 2,3,7,8-tetrachlorodibenzo-p-dioxin influences the expression and activity of GR and vice versa—that is, activation of GR by dexamethasone alters the expression and functions of AhR (4, 5).

We hypothesize that the diminution of dexamethasone-induced insulin resistance by berberine could be due to the activation of AhR by berberine and consequent interaction between AhR and GR signaling.

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REFERENCES