2017-08-17

Dietary Phytochemicals as Inhibitors of Primary Amine Oxidase

Padraig Shanahan
Dublin Institute of Technology, d11125149@mydit.ie

Jeffrey O'Sullivan
Trinity College Dublin, Ireland, JOSULLI@tcd.ie

Keith F. Tipton
Trinity College Dublin, Ireland, ktipton@tcd.ie

Gemma Kinsella
Dublin Institute of Technology, gemma.kinsella@dit.ie

Barry J. Ryan
Dublin Institute of Technology, barry.ryan@dit.ie

See next page for additional authors

Follow this and additional works at: http://arrow.dit.ie/schfsehart

Part of the Biochemistry Commons, Molecular Biology Commons, and the Other Biochemistry, Biophysics, and Structural Biology Commons

Recommended Citation

This work is licensed under a Creative Commons Attribution-Noncommercial-Share Alike 3.0 License
Dietary phytochemicals as inhibitors of primary amine oxidase

P. Shanahan¹, J O’Sullivan², KF Tipton³, GK Kinsella¹, B Ryan¹, GTM. Henehan¹

1. Applied Enzymology Group, Food Science and Environmental Health, Dublin Institute of Technology, Dublin 1, Ireland.

2. School of Dental Sciences, Trinity College Dublin, Dublin, Ireland.

3. School of Biochemistry and Immunology, Trinity College Dublin, Dublin, Ireland.

Phytochemicals such as methylxanthines, catechins and polyphenols show health benefits in a range of diseases although their mechanism of action is not fully understood. Primary Amine Oxidase (PrAO) is widely recognised as a therapeutic drug target for the treatment of inflammatory, vascular and neurodegenerative diseases. Previous work in our laboratories showed that caffeine inhibited bovine PrAO activity with a Ki of 1.0mM. In the present study we examined a range of methylxanthines and catechins as inhibitors of bovine PrAO. The methylxanthines tested were caffeine, paraxanthine, theophylline, theobromine and 7-methylxanthine. Of these, only theobromine was an inhibitor with an IC50 of ca. 300µM. Calculations indicated that theobromine in foods could inhibit PrAO activity by 20%. The effect of dietary catechins; epicatechin, epicatechin gallate and epigallocatechingallate was even more significant with IC50 values in the micromolar region. However, inhibition by catechins was complicated by apparent activation of PrAO at high concentrations although this was not significant at physiologically attainable levels. Nonetheless, these findings indicate that a range of dietary phytochemicals could affect PrAO activity in vivo. We suggest that the health benefits associated with consumption of certain phytochemicals may be attributed to PrAO inhibition.