**Ganoderma lucidum aumenta a apoptose na leucemia via Akt e Erk**

_04/06/11_

Ganoderma lucidum induced apoptosis in NB4 human leukemia cells: involvement of Akt and Erk.
Calviño E, Manjón JL, Sancho P, Tejedor MC, Herráez A, Diez JC.

Source
Departamento de Bioquímica y Biología Molecular, Campus Universitario, Universidad de Alcalá, 28871 Alcalá de Henares, Madrid, Spain.

Abstract
AIM OF THE STUDY:
The final goal of this work was to study the toxic and apoptosis effects induced by fractions from Ganoderma lucidum [Ganoderma lucidum (Curtis) P. Karst.; Ganodermataceae Donk] on NB4 human leukemia cells.

MATERIALS AND METHODS:
Two aqueous extracts and a methanol-extracted column-chromatography semipurified fraction were obtained from Ganoderma lucidum fruiting body. Flow cytometry analyses were used to measure cell viability, cell cycle and DNA fragmentation and to quantify apoptosis. Western-blot analyses were used to quantify changes in apoptosis proteins and intracellular kinases.

RESULTS:
Aqueous extracts slightly reduce cell viability and induce DNA fragmentation in NB4 cells. Methanol-extracted semipurified fraction at dilutions down to 15% or 40% of the initial fraction concentration reduced significantly the viability of these leukemia cells (treated for 19h) with induction of DNA fragmentation and induction of apoptosis. Overmore, the dilution down to 15% of the initial E3 concentration induced a reduction of p53 levels, of the Bcl2/Bax relationship as well as reduced levels of both unphosphorylated and phosphorylated Akt (Protein kinase Akt, protein kinase B) and Erk (Erk1 and 2).

CONCLUSIONS:
Induction of apoptosis and alterations in signal transduction kinases (Akt and Erk) are produced by active fractions from Ganoderma lucidum on human leukemia cells. These data could be of important relevance from the viewpoint of antitumor actions of compounds from Ganoderma lucidum. Eventual therapy applications in leukemia cells might be developed.

Copyright (c) 2009 Elsevier Ireland Ltd. All rights reserved.

PMID:
20036724