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Gan To Kagaku Ryoho. 1989 Apr;16(4 Pt 2-2):1483-9.

1. **[Induction of phenotypic reverse transformation by plant glycosides in cultured cancer cells].**

[Article in Japanese]
Odashima S[1], Ota T, Fujikawa-Yamamoto K, Abe H.

Author information

Abstract

Crude ginsenosides extracted from the root of Panax ginseng C.A. Meyer inhibited the growth and colony forming ability of Morris hepatoma cells in soft agar suspension culture, and stimulate the serum protein synthesis of these cells, thus converting the cell characteristics both functionally and morphologically to those resembling original normal liver cells. We have called such a phenomenon "reverse transformation" or "redifferentiation" which can be regarded as decarcinogenesis. In this report, the results of our recent investigations are presented with particular reference to reverse transformation of B16 melanoma cells induced by ginsenoside Rh2 isolated from the methanol extract of crude ginseng saponin fraction and action mechanisms of ginsenoside Rh2 are also discussed.

PMID: 2658830

[Indexed for MEDLINE]

Publication type, MeSH terms, Substances

Eur J Cancer. 1979 Jun;15(6):885-92.

2. **Induction of phenotypic reverse transformation by ginsenosides in cultured Morris hepatoma cells.**

Odashima S, Nakayabu Y, Honjo N, Abe H, Arichi S.

PMID: 227695

[Indexed for MEDLINE]

MeSH terms, Substances

Eur J Cancer. 1996 Jul;32A(8):1420-8.

3. # In vitro induction of differentiation by ginsenoides in F9 teratocarcinoma cells.

Lee YN[1], Lee HY, Chung HY, Kim SI, Lee SK, Park BC, Kim KW.

Author information

Abstract

The aim of this study was to determine the ability of the ginsenosides, extracts of Panax ginseng C.A. Meyer, to cause differentiation of F9 teratocarcinoma stem cells as a model system. F9 stem cells cultured in the presence of the ginsenosides together with dibutyryl cyclic AMP (dbcAMP) became parietal endoderm-like cells. Moreover, the expression of differentiation marker genes, such as laminin B1 and type IV collagen, was increased after treatment with the ginsenosides. Among the various purified ginsenosides, Rh1 and Rh2 were the most effective at causing differentiation of F9 cells. Since ginsenosides and glucocorticoid hormone have similar chemical structures, we examined the possibility of the involvement of a glucocorticoid receptor (GR) in the differentiation process induced by the ginsenosides. According to Southwestern blot analysis, a 94 kDa protein regarded as a GR was detected in F9 cells cultured in the medium containing the ginsenosides Rh1 or Rh2. In addition, F9 stem cells treated with the ginsenosides Rh1 or Rh2 and with RU486, a glucocorticoid antagonist with a high affinity for the GR, did not differentiate into endoderm cells morphologically, and the expression of laminin B1 gene was not induced in these cells. In a gel mobility shift assay, protein factors capable of binding to the glucocorticoid responsive element (GRE) specifically were detected in nuclear extracts of the ginsenoside-treated F9 cells. Moreover, overexpression of GR by cotransfection of GR expression vector and GRE-luciferase vector enhanced the transactivation activity of GRE promoter in the presence of ginsenosides Rh1 or Rh2 and was further augmented by dbcAMP. In addition, ginsenosides Rh1 and Rh2 bound to a GR assessed by whole-cell binding assay, even though the specific binding affinity was weaker compared to dexamethasone. Based on these data, we suggest that the ginsenosides Rh1 and Rh2 cause the differentiation of F9 cells and the effects of ginsenosides might be exerted via binding with a GR or its analogous nuclear receptor.

PMID: 8869109

[Indexed for MEDLINE]

Publication type, MeSH terms, Substances

Yao Xue Xue Bao. 1996;31(10):742-5.

4.

[Differentiation of B16 melanoma cells induced by ginsenoside RH2].

[Article in Chinese]

Xia LJ[1], Han R.

Author information

Abstract

The effect of ginsenoside Rh2, a constituent isolated from Panax ginseng C. A. Meyer, on the growth of tumor cells in vitro was investigated. The results showed that Rh2 inhibited the growth of B16 cells at the concentration of 10 micrograms.ml-1 (IC50: 4.1 micrograms.ml-1). Rh2 was found to significantly induce the activity of differentiation of B16 cells at the concentration of 10 micrograms.ml-1 in vitro. The melanin synthesis of Rh2 in treated B16 cells was increased. Morphologically, the Rh2 induced B16 cells turned to be epithelioid cells. B16 cells became dendrite shaped morphologically at higher concentration of Rh2. Flow cytometry demonstrated that the B16 cells treated with Rh2 were blocked at G1 phase.

PMID: 9863241

[Indexed for MEDLINE]

Publication type, MeSH terms, Substances

J Ethnopharmacol. 2013 Nov 25;150(2):700-7. doi: 10.1016/j.jep.2013.09.036. Epub 2013 Oct 2.

5.

Korean red ginseng extract induces proliferation to differentiation transition of human acute promyelocytic leukemia cells via MYC-SKP2-CDKN1B axis.

Jo S[1], Lee H, Kim S, Lee CH, Chung H.

Author information

Abstract

ETHNOPHARMACOLOGICAL RELEVANCE: Korean red ginseng has been used as traditional medicine in East Asia. Recent scientific research revealed multiple effects of Korean red ginseng, including anticancer activity. To evaluate the effect of Korean red ginseng extract (KRGE) in acute promyelocytic leukemia (APL) and elucidate its molecular mechanism.

MATERIALS AND METHODS: NB4 cells were treated with 1mg/ml KRGE for 48 h and examined for cell proliferation and differentiation. Cell cycle distribution of KRGE-treated cells was analyzed and the expression level of G1 phase regulators was determined. MYC was overexpressed by retroviral transduction and its effect on SKP2 and CDKN1B gene expression, cell proliferation, cell cycle and differentiation was evaluated in KRGE-treated cells.

RESULTS: KRGE alone was sufficient to induce granulocytic differentiation accompanied with growth inhibition. KRGE treatment resulted in cell cycle arrest at the G1 phase with augmented Cdkn1b proteins without changes in transcript levels. Cycloheximide treatment revealed reduced degradation of Cdkn1b protein by KRGE. In addition, KRGE treatment reduced expression of MYC and SKP2 genes, both at mRNA and protein levels. Upon ectopic expression of MYC, the effect of KRGE was reversed with lesser reduction and induction of SKP2 gene and Cdkn1b protein, respectively. Taken together, these results suggest a sequential molecular mechanism from MYC reduction, SKP2 reduction, Cdkn1b protein stabilization, G1 phase arrest to granulocytic differentiation by KRGE in human APL.

CONCLUSIONS: KRGE induces leukemic proliferation to differentiation transition in APL through modulation of the MYC-SKP2-CDKN1B axis.

KEYWORDS: AML; APL; ATO; ATRA; Acute promyelocytic leukemia (APL); BSA; CDKN1B; CHX; Differentiation; FBS; KRGE; Korean red ginseng extract; Korean red ginseng extract (KRGE); NBT; P/D transition; PBS; PCR; PML-RARA; RT-PCR; SKP2; WST; acute myeloid leukemia; acute promyelocytic leukemia; all-trans retinoic acid; arsenic trioxide; bovine serum albumin; cycloheximide; fetal bovine serum; nitro blue tetrazolium; phosphate-buffered saline; polymerase chain reaction; proliferation/differentiation transition; promyelocytic leukemia-retinoic acid receptor alpha; reverse transcription- polymerase chain reaction; water-soluble tetrazolium salt

PMID: 24095829 DOI: 10.1016/j.jep.2013.09.036

[Indexed for MEDLINE]

Publication type, MeSH terms, Substances

6. Yakugaku Zasshi. 2011;131(6):993-1000.

Induction of differentiation by panaxydol in human hepatocarcinoma SMMC-7721 cells via cAMP and MAP kinase dependent mechanism.

Wang ZJ[1], Song L, Guo LC, Yin M, Sun YN.

Author information

Abstract

Panaxydol (PND) is one of the main non-peptidyl small molecules isolated from the lipophilic fractions of Panax notoginseng. The present study was carried out to demonstrate the potential effects of panaxydol on the induction of differentiation of human liver carcinoma cell lines SMMC-7721. Cell viability was evaluated by MTT method and Trypan blue exclusion assay respectively. The changes of morphology were detected by transmission electron

microscope. Inhibitors were applied to detect the signaling pathway of differentiation. The level of intracellular cyclic AMP was determined by radioimmunoassay. The expression of p-ERK, Id1, and p21 were determined by Western blot. We found that panaxydol inhibit the proliferation of SMMC-7721 cells and caused the morphology and ultrastructure changes of SMMC-7721. Moreover, panaxydol dose-dependently increased the secretion of albumin and alkaline phosphatase activity, and decreased the secretion of AFP correspondingly. These changes of differentiation markers in SMMC-7721 can be reversed by the protein kinase A inhibitor RpcAMPS and by MAP kinase kinase 1/2 inhibitor U0126 or sorafenib. Intracellular cAMP was elevated by panaxydol in SMMC-7721 cells. Panaxydol dose-dependently decreased the expression of regulatory factors Id1 and increased the protein levels of p21 and p-ERK1/2 correspondingly. It suggested panaxydol might be of value for further exploration as a potential anti-cancer agent via cAMP and MAP kinase-dependent mechanism.

PMID: 21628989

[Indexed for MEDLINE] **Free full text**

Publication type, MeSH terms, Substances

Chem Biol Interact. 2009 Sep 14;181(1):138-43. doi: 10.1016/j.cbi.2009.04.015. Epub 2009 May 18.

7. ## Panaxydol inhibits the proliferation and induces the differentiation of human hepatocarcinoma cell line HepG2.

Guo L[1], Song L, Wang Z, Zhao W, Mao W, Yin M.

Author information

Abstract

Panaxydol, a polyacetylene compound isolated from Panax ginseng, exerts anti-proliferative effects against malignant cells. No previous study, however, has been reported on its effects on hepatocellular carcinoma cells. Here, we investigated the effects of panaxydol on the proliferation and differentiation of human hepatocarcinoma cell line HepG2. We studied by electronic microscopy of morphological and ultrastructural changes induced by panaxydol. We also examined the cytotoxicities of panaxydol against HepG2 cells using the 3-(4,5-dimethyl-2-thiazolyl)-2,5-diphenyl tetrazolium bromide assay and the effect of panaxydol on cell cycle distributions by flow cytometry. We investigated the production of liver proteins in panaxydol-treated cells including alpha-fetoprotein and albumin and measured the specific activity of alkaline phosphatase and gamma-glutamyl transferase. We further investigated the effects of panaxydol on the expression of Id-1, Id-2, p21 and pRb by RT-PCR or immunoblotting analysis. We found that panaxydol inhibited the proliferation of HepG2 cells and caused morphological and ultrastructural changes in HepG2 cells resembling more mature forms of

hepatocytes. Moreover, panaxydol induced a cell cycle arrest at the G(1) to S transition in HepG2 cells. It also significantly decreased the secretion of alpha-fetoprotein and the activity of gamma-glutamyl transferase. By contrast, panaxydol remarkably increased the secretion of albumin and the alkaline phosphatase activity. Furthermore, panaxydol increased the mRNA content of p21 while reducing that of Id-1 and Id-2. Panaxydol also increased the protein levels of p21, pRb and the hypophosphorylated pRb in a dose-dependent manner. These findings suggest that panaxydol is of value for further exploration as a potential anti-cancer agent.

PMID: 19450571 DOI: 10.1016/j.cbi.2009.04.015

[Indexed for MEDLINE]

ELSEVIER FULL-TEXT ARTICLE

MeSH terms, Substances

J Clin Neurosci. 2009 Mar;16(3):444-8. doi: 10.1016/j.jocn.2008.05.014.

8.

Phosphatidylinositol 3-kinase activity is required for the induction of differentiation in C6 glioma cells by panaxydol.

Hai J[1], Lin Q, Lu Y.

Author information

Abstract

Panaxydol isolated from the lipophilic fractions of Tienchi ginseng (Panax notoginseng) induces growth inhibition and differentiation of rat C6 glioma cells. The underlying molecular mechanisms are not completely understood. In the present study, we identified phosphatidylinositol 3-kinase (PI 3-K) as a necessary enzyme for the differentiation of C6 cells treated with panaxydol. The specific PI 3-K inhibitor wortmannin resulted in attenuated differentiation of C6 cells induced by panaxydol, and was associated with perinuclear localization of glial fibrillary acidic protein expression and a diminished process formation. These data suggest that induction of differentiation in C6 cells by panaxydol could be mediated through a PI 3-K-dependent pathway.

PMID: 19179079 DOI: 10.1016/j.jocn.2008.05.014

[Indexed for MEDLINE]

ELSEVIER FULL-TEXT ARTICLE

Publication type, MeSH terms, Substances

Zhong Xi Yi Jie He Xue Bao. 2007 Sep;5(5):570-2.

9. [Effects of total saponins of Panax japonicus on human leukemic HL-60 cells].

[Article in Chinese]

Yuan D[1], Zuo R, Zhang CC.

Author information

Abstract

OBJECTIVE: To study the effects of total saponins of Panax japonicus (TSPJ) on human leukemic HL-60 cells.

METHODS: Human leukemic HL-60 cells were cultured in vitro. The cancer cell vigor was detected by using cell counting kit-8. Nitroblue tetrazolium (NBT) was used for measuring cell reduction. The cell cycle and the expression of differentiation antigen CD11b were detected by flow cytometry.

RESULTS: Compared with the negative control group, TSPJ in different concentrations could decrease the vigor of HL-60 cells and the number of cells in S phase and up-regulate the CD11b expression, while the numbers of NBT positive cells and cells in G(0)/G(1) phase in the different concentrations of TSPJ-treated groups were increased.

CONCLUSION: TSPJ can inhibit the HL-60 cell growth in vitro. Its mechanism may be related to inhibiting proliferation and inducing cell differentiation and cycle arrest.

PMID: 17854562

[Indexed for MEDLINE] Free full text

Publication types, MeSH terms, Substances

Neurol Res. 2008 Feb;30(1):99-105.

10. Growth inhibition and induction of differentiation by panaxydol in rat C6 glioma cells.

Hai J[1], Lin Q, Lu Y, Yi J, Zhang H.

Author information

Abstract

OBJECTIVES: Panaxydol is a naturally occurring non-peptidyl small molecule isolated from the lipophilic fractions of Panax notoginseng, a well-known Chinese traditional medicine. In

this study, we aimed to investigate the effects of panaxydol on growth inhibition and its mechanisms in C6 rat glioma cells.

METHODS: The effects of panaxydol on cell proliferation, morphologic changes, glial fibrillary acidic protein (GFAP) expression and cell cycle regulation in rat C6 cells were evaluated using 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyl tetrazolium bromide (MTT) assay, hematoxylin and eosin (HE) staining, immunocytochemistry, flow cytometric analysis and Western blot respectively.

RESULTS: Panaxydol markedly inhibited the proliferation of C6 cells in a dose-dependent manner with IC50 of 39.5 +/- 2.3 microM. In addition, the cell morphologic changes and increased expression of GFAP in C6 cells in the presence of panaxydol implied a cellular differentiation. Flow cytometric analysis revealed that panaxydol-treated cells accumulated in G0/G1 phase with a marked decrease in the number of C6 cells at S phase. Western blot analysis demonstrated that panaxydol resulted in an increase in the protein expression of p27 in C6 cells as early as 3 hours after treatment consistent with the differentiation response, but protein expression of p53, p21, p16 and pRb remained unchanged.

CONCLUSION: These findings suggest that panaxydol inhibits the proliferation of C6 cells via G0/G1 cell cycle arrest in association with induction of p27 expression and differentiation.

PMID: 17767808 DOI: 10.1179/016164107X228697

[Indexed for MEDLINE]

View full text

Publication type, MeSH terms, Substances

Ai Zheng. 2004 Dec;23(12):1655-9.

11. ## [Effect of telomerase on ginsenoside Rh2-induced differentiation of hepatocarcinoma cell line SMMC-7721].

[Article in Chinese]
Zeng XL[1], Tu ZG.

Author information

Abstract

BACKGROUND & OBJECTIVE: Recently we found that 20 microg/ml of ginsenoside Rh2 (G-Rh(2)) can inhibit growth, and induce differentiation of hepatocarcinoma cell line SMMC-7721. Re-activation of telomerase may play an important role in carcinogenesis, and cellular immortalization. This study was to explore the mechanism of G-Rh(2)-induced differentiation of SMMC-7721 cells by detecting telomerase activity.

METHODS: After treated with 20 mug/ml of G-Rh(2), telomerase activity in SMMC-7721 cells was detected by polymerase chain reaction (PCR)-based telomeric repeat amplification

protocol (TRAP) coupled with enzyme-linked immune sorbent assay(ELISA); mRNA levels of human telomerase reverse transcriptase (hTERT), and p21 were measured by reverse transcriptase-polymerase chain reaction (RT-PCR); changes of cell cycle, and expression of Cyclin D1, Cyclin E,P16 protein were detected by flow cytometry (FCM).

RESULTS: G-Rh(2) inhibited telomerase activity of SMMC-7721 in a time- dependent manner: the activity decreased from 1.105 to 0.765 (P< 0.01) after 1-day treatment, while from 1.152 to 0.326 (P< 0.01) after treated for 5-day treatment. The mRNA expression of hTERT was significantly reduced by 20 microg/ml of G-Rh(2). Cell cycle assay showed that 20 microg/ml of G-Rh(2) increased the proportion of SMMC-7721 cells in G1 phase, and decreased those in S, and G2/M phases, the increase of G1 phase after 1-day treatment was the most apparent (from 60.85% to 78.53%,P< 0.05). Furthermore,G-Rh(2) weakened the expressions of positive-regulating factors(Cyclin D1,Cyclin E),and increased the expressions of negative-regulating factors (P16 protein, p21 gene) in SMMC- 7721 cells.

CONCLUSIONS: G-Rh(2) may effectively reduce telomerase activity through affecting transcription level of hTERT,and arresting cell cycle progression. The down- regulation of telomerase activity in SMMC-7721 cells may be closely related to G-Rh(2)-induced differentiation.

PMID: 15601555

[Indexed for MEDLINE]

Publication type, MeSH terms, Substances

Ai Zheng. 2004 Aug;23(8):879-84.

12. ## [Induction of differentiation by ginsenoside Rh2 in hepatocarcinoma cell SMMC-7721].

[Article in Chinese]
Zeng XL[1], Tu ZG.

Author information

Abstract

BACKGROUND & OBJECTIVE: Up to now, searching for non-toxic and natural origin substances that induced the differentiation of cancer cells is a key for anticancer therapy. Ginseng is one of the most widely used natural tonics in oriental countries for thousands of years and has been reported to have various biological effects. Ginsenosides are thought to be the major effective ingredients in ginseng. Among them, ginsenoside Rh2(G-Rh2) has been suggested to have a cell-growth suppressive effect on various cancer cells, but the mechanism is unclear. This study was to investigate the induced differentiative effects of G-Rh2 on SMMC-7721 hepatocarcinoma cells.

METHODS:

Effects of G-Rh2 on cell viability was analyzed by MTT assay. Cell morphology was examined by a light and electronic microscope. Alpha-fetoprotein (AFP) in plasma was determined qualitatively and quantitatively with immunohistochemistry and ELISA. The specific activities of alkaline phosphatase (ALP) and heat-resistant ALP in plasma were assayed by ALP kit based on Bessey method. The specific activity of gamma-glutamyltranspeptidase (gamma-GT) was measured with gamma-GT kit.The secretory amount of AFP or albumin was detected with radioimmunoassay kit.

RESULTS: G-Rh2 inhibited the proliferation of SMMC-7721 cells in dose and time-dependent manners. The inhibition rate was 50.87% after 6-day treatment with 10 microg/ml G-Rh2 while 46.84% after 4-day treatment with 20 microg/ml G-Rh2. Twenty microg/ml G-Rh2 induced the mature and normality of morphology and ultrastructure in SMMC-7721 cells. After treated with 10 microg/ml or 20 microg/ml G-Rh2, the production of AFP was significantly reduced (P< 0.05), and the secretory amount of AFP was reduced from 6.60+/-0.30 to 2.35+/-0.06 (P< 0.01), and the specific activities of gamma-GT and heat-resistant ALP were remarkably declined (P< 0.01); while the secretory amount of albumin and ALP activity were remarkably enhanced (P< 0.01).

CONCLUSION: G-Rh2 could induce the SMMC-7721 cell differentiation tending to normal.

PMID: 15301707

[Indexed for MEDLINE]

Publication type, MeSH terms, Substances

Ann Acad Med Singapore. 2000 Jan;29(1):42-6.

13. ## Panax (ginseng)--panacea or placebo? Molecular and cellular basis of its pharmacological activity.

Ong YC[1], Yong EL.

Author information

Abstract

INTRODUCTION: The use of ethnobotanical drugs amongst Asians as complementary medicine is prevalent and is also gaining increasing popularity in the West. The most well-known herb traditionally used as a drug is the root of the ginseng species. There are many traditional and anecdotal claims to the therapeutic properties of ginseng. In recent years, there have been systematic efforts to analyse the bioactivities of ginseng saponins.

METHODS: A comprehensive review of published literature covering molecular and cellular research as well as animal and human studies on ginseng and its derivatives.

RESULTS AND CONCLUSION: Current published data would serve as a framework to understand the pharmacology of ginseng in its entirety, from its molecular action to actual

therapeutic effects observed in human use. A new paradigm is emerging whereby the pharmacological effects of traditional herbs such as ginseng can be understood in the light of their polyvalent actions as demonstrated by ginseng saponins with their positive anti-mutagenic, anti-cancer, anti-inflammatory, anti-diabetes and neurovascular effects. With increasing understanding, evidence-based incorporation of traditional herbs as complementary medicine into mainstream medical science can be achieved in the near future.

PMID: 10748963

[Indexed for MEDLINE]

Publication type, MeSH terms, Substance

Zhongguo Zhong Xi Yi Jie He Za Zhi. 1993 Dec;13(12):722-4, 708.

14. **[Inductive differentiation effect of ginsenosides on human acute non-lymphocytic leukemic cells in 58 patients].**

[Article in Chinese]
Yi RL[1], Li W, Hao XZ.

Author information

Abstract

Ginsenosides are the main active component of Panax ginseng. It has been shown that ginsenosides have antineoplastic, antiaging, immunologic function enhancing and other pharmacological actions. In this article, result of experimental studies showed ginsenosides extracted from stem and leaf of Panax ginseng (GSL) has inductive differentiation effect on all types of acute nonlymphocytic leukemia cells in primary culture. The effect on M5, M4 was most potent, followed by M1, M2 and the least, on M3. Through analysis, it was considered that the inductive differentiation effect of ginsenosides might be due to the comprehensive effect of increasing intracellular cAMP and inducing interferon. Since GSL have some other important actions, therefore, if it could be used as a differentiation inducer in clinical practice or combined with other antineoplastic drugs, it would show co-antineoplastic actions in many aspect.

PMID: 8136644

[Indexed for MEDLINE]

Publication type, MeSH terms, Substances