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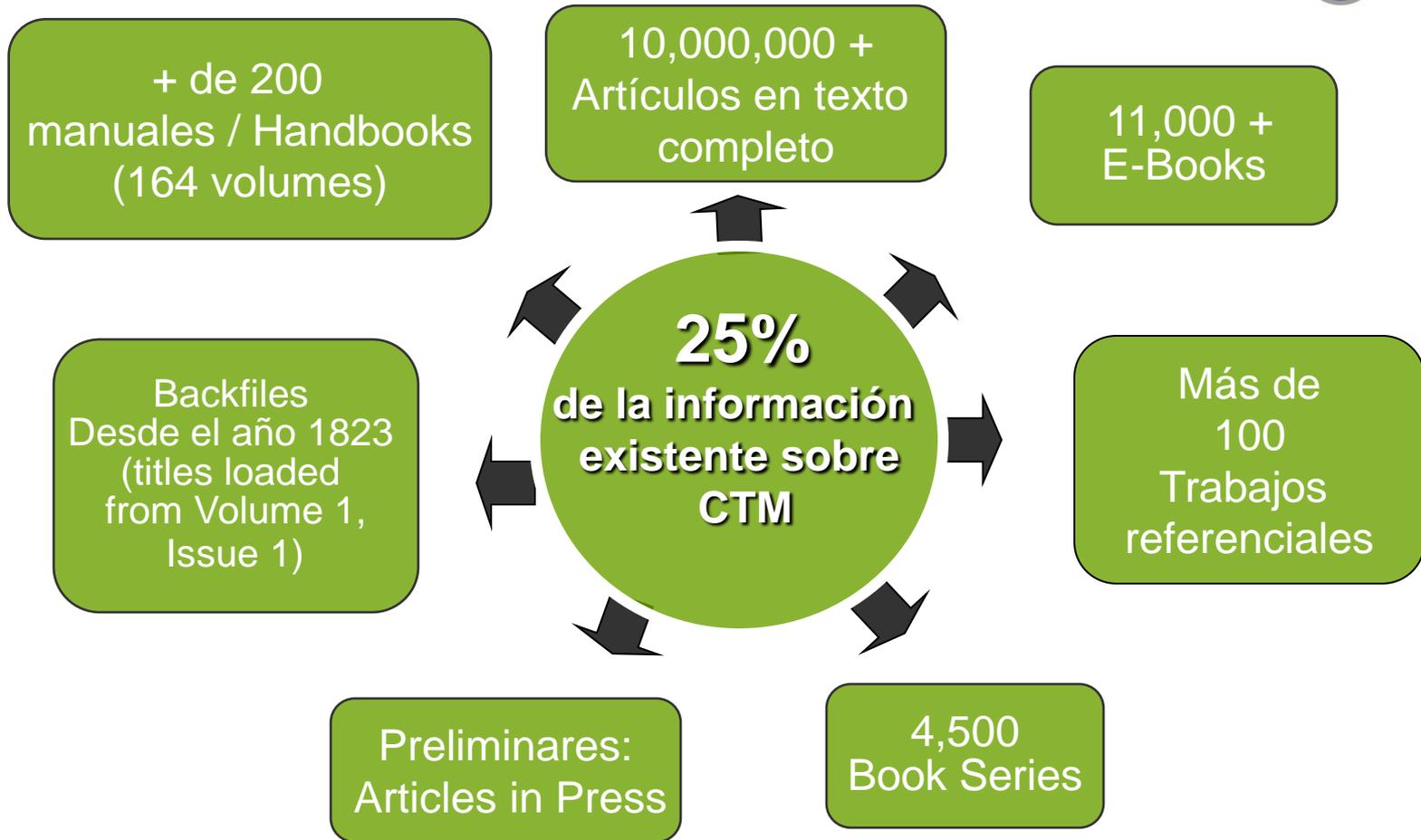


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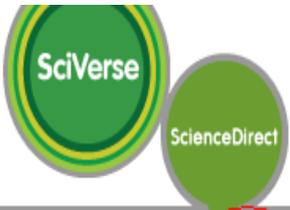
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Topic: dna damage (5,173), dna repair (2,296), dna (1,949), radio therapy (1,892), antibody (1,319)

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- Induction of DNA damage by risk factors of colon cancer in human colon cells derived from biopsies**
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- p53 Mediated tumor cell response to chemotherapeutic dna damage: A preliminary study in matched pairs of breast cancer biopsies**
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- Putative colon cancer risk factors damage global DNA and TP53 in primary human colon cells isolated from surgical samples**
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- Processing of clustered DNA damage in human breast cancer cells MCF-7 with partial DNA-PKcs deficiency**
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- High-dose combination alkylating agents with autologous bone-marrow support in patients with breast cancer: preliminary assessment of DNA damage in individual peripheral blood lymphocytes using the single cell gel electrophoresis assay**
Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis and Related Subjects, Volume 574, Issue 2, 1999, Pages 104-110



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Gaoliang Ouyang, Luming Yao, Kai Ruan, Gang Song, Yubin Mao, Shideng Bao
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R. Supino, P. Perego, U. Le...
- Induction of DNA damage in normal and cancer cells**
Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis, Volume 668, Issues 1-3, February 2011, Pages 1-10
B. L. Pool-Zobel, U. Le...
- p53 Mediated tumor cell death in normal and cancer cells**
Human Pathology, Volume 40, Issue 1, January 2009, Pages 1-10
Ute M. Moll, Anne G. Os...
- Putative colon cancer cell lines**
Food and Chemical Toxicology, Volume 47, Issue 1, January 2009, Pages 1-10
A. Schaeferhenrich, W...
- Processing of clustered DNA damage**
Cancer Letters, Volume 188, Issue 1, January 2005, Pages 1-10
Prakash Peddi, Dave C...
- High-dose combination of DNA damage agents**
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Abstract 1: Genistein is a major isoflavonoid in dietary soybean, commonly consumed in Asia. Genistein exerts inhibitory effects on the proliferation of various **cancer cells** and plays an important role in **cancer** prevention. However, the molecular and cellular mechanisms of genistein on human ovarian **cancer cells** are still little known. We show that exposure of human ovarian **cancer HO-8910 cells** to genistein induces DNA **damage**, and triggers G2/M phase arrest and apoptosis. Furthermore, we also found that checkpoint proteins ATM and ATR are phosphorylated and activated in the **cells** treated with genistein. It is also shown that genistein increases the phosphorylation and activation of Chk1 and Chk2, which results in the phosphorylation and inactivation of phosphatases Cdc25C and Cdc25A, and thereby the phosphorylation and inactivation of Cdc2 which arrests **cells** in G2/M phase. Moreover, genistein enhances the phosphorylation and activation of p53, while decreases the ratio of Bcl-2/Bax and Bcl-xL/Bax and the level of phosphorylated Akt, which result in **cells** undergoing apoptosis. These results demonstrate that genistein-activated ATM-Chk2-Cdc25 and ATR-Chk1-Cdc25 DNA **damage** checkpoint pathways can arrest ovarian **cancer cells** in G2/M phase, and induce apoptosis while the cellular DNA **damage** is too serious to be repaired. Thus, the antiproliferative, DNA **damage**-inducing and pro-apoptotic activities of genistein are probably responsible for its genotoxic effects on human ovarian **cancer HO-8910 cells**.

Abstract 2: Zinc plays an essential role in a wide range of cellular processes, including defense against free radicals and maintaining genomic stability. The presence of zinc in some proteins is fundamental for their functioning as transcription factors. Little is known about interaction between zinc and DNA, which can be important in light of reports on the role of zinc in **cancer** transformation and sometimes contradictory character of these reports. In the present study we studied cyto- and genotoxicity of zinc sulfate (ZnSO₄) in normal human lymphocytes and human myelogenous leukemia K562 **cancer cells** in the presence of zinc and hydrogen peroxide (H₂O₂). Zinc at concentrations from the range 10–1000 μM decreased the viability of **cancer cells** and this effect, especially for low concentrations of the element, was much more pronounced than in normal **cells**. Zinc did not induce DNA **damage** in normal **cells**, but did so in **cancer cells**. We observed a key difference between the action of zinc in normal and **cancer cells** in the presence of H₂O₂, since the element exerted a protective effect against cyto- and geno-toxic action of H₂O₂ in the former, whereas it increased such action in the latter. Zinc inhibited the repair of DNA **damage** induced by H₂O₂ in **cancer cells**. The results suggest that zinc may protect normal **cells** against DNA-damaging action and increase this action in **cancer cells**, which indicates the dual action of this element in dependency of target **cells** and can be useful in **cancer** therapy.

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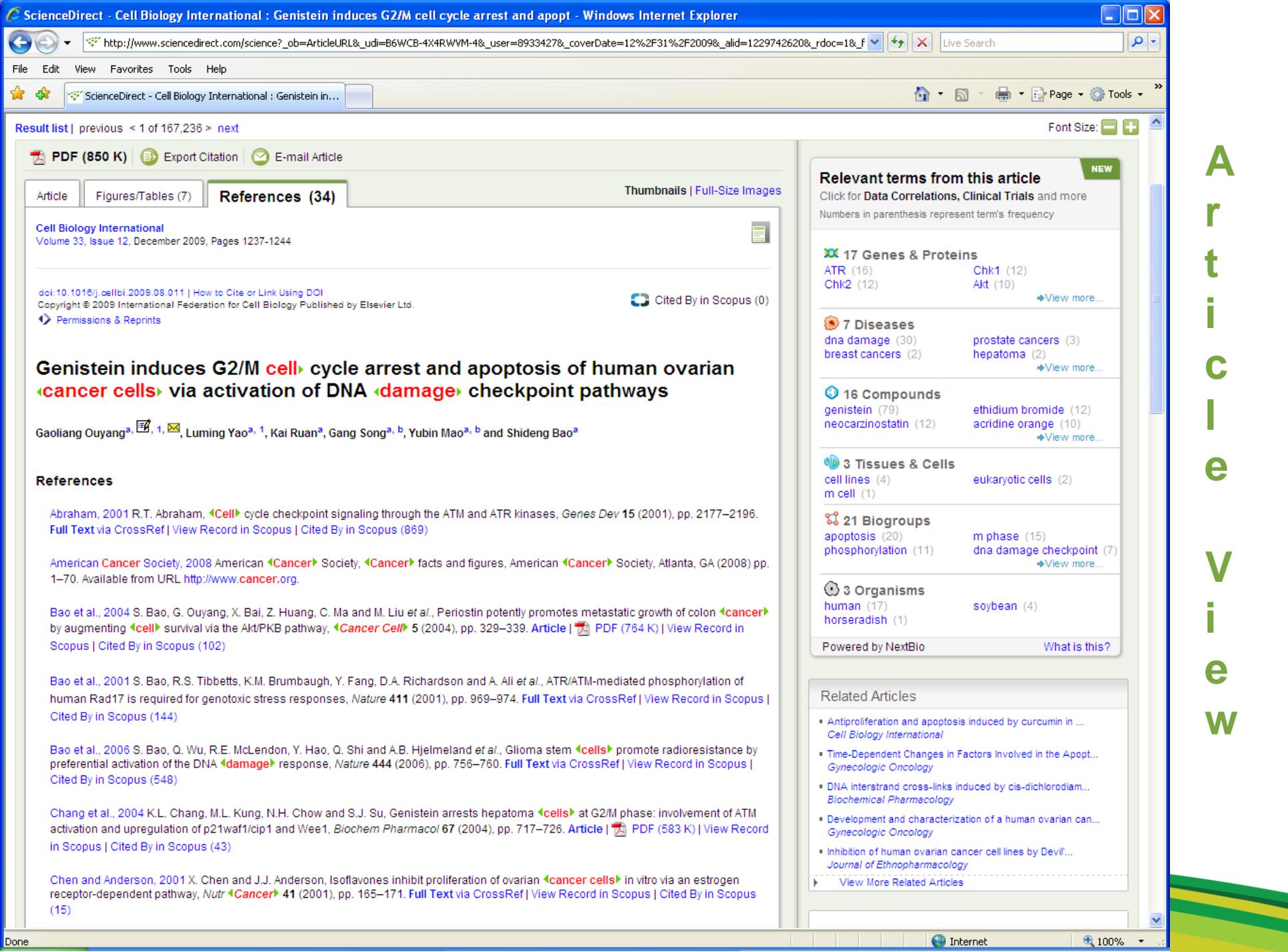
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Genistein induces G2/M cell cycle arrest and apoptosis of human ovarian cancer cells via activation of DNA damage checkpoint pathways

Gaoliang Ouyang^a, Luming Yao^{a, 1}, Kai Ruan^a, Gang Song^{a, b}, Yubin Mao^{a, b} and Shideng Bao^a

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Abstract

Genistein is a major isoflavonoid in dietary soybean, commonly consumed in Asia. Genistein exerts inhibitory effects on the proliferation of various cancer cells and plays an important role in cancer prevention. However, the molecular and cellular mechanisms of genistein on human ovarian cancer cells are still little known. We show that exposure of human ovarian cancer HO-8910 cells to genistein induces DNA damage and triggers G2/M phase arrest and apoptosis. Furthermore, we also found that checkpoint proteins ATM and ATR are phosphorylated and activated in the cells treated with genistein. It is also shown that genistein increases the phosphorylation and activation of Chk1 and Chk2, which results in the phosphorylation and inactivation of phosphatases Cdc25C and Cdc25A, and thereby the phosphorylation and inactivation of Cdc2 which arrests cells in G2/M phase. Moreover, genistein enhances the phosphorylation and activation of p53, while decreases the ratio of Bcl-2/Bax and Bcl-xL/Bax and the level of phosphorylated Akt, which result in cells undergoing apoptosis. These results demonstrate that genistein-activated ATM-Chk2-Cdc25 and ATR-Chk1-Cdc25 DNA damage checkpoint pathways can arrest ovarian cancer cells in G2/M phase, and induce apoptosis while the cellular DNA damage is too serious to be repaired. Thus, the antiproliferative, DNA damage-inducing and pro-apoptotic activities of genistein are probably responsible for its genotoxic effects on human ovarian cancer HO-8910 cells.

Keywords: Genistein; Apoptosis; DNA damage checkpoint; Ovarian cancer cell

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1. Introduction
2. Materials and methods
 - 2.1. Materials
 - 2.2. Cell culture and genistein treatment
 - 2.3. MTT assay
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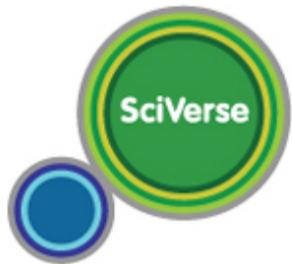
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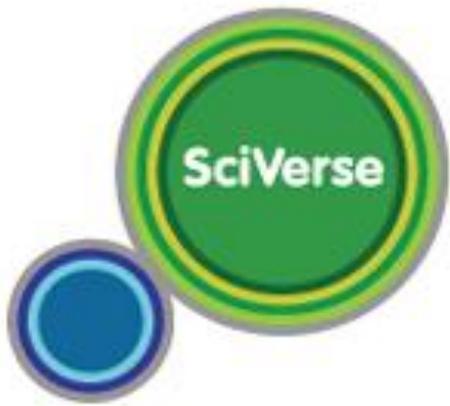
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