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A Response to Article "Selenium-Modified Chitosan Induces HepG2 Cell Apoptosis and Differential Protein Analysis" [Letter]

Sela S Mariya^(D), Silmi Mariya², Novaria SD Panjaitan^(D)

¹Center for Biomedical Research, Research Organization for Health, National Research and Innovation Agency (BRIN), Cibinong, West Java, Indonesia; ²Primate Research Center (PRC), IPB University, Bogor, West Java, Indonesia

Correspondence: Sela S Mariya, Center for Biomedical Research, Research Organization for Health, Genomic Building, Cibinong Science Center, Jl. Raya Bogor No. 490, Cibinong, West Java, 16911, Indonesia, Email sela002@brin.go.id

Dear editor

We have read with great interest an article by Sun et al,¹ a well-written important work which studied the effect of Selenium-Modified Chitosan-induced HepG2 cell apoptosis and differential protein analysis. This study is very important as a basic analysis to develop candidate bioactive compounds has potency as anti-cancer drugs. We would like to give our perception, particularly on the data interpretation and method utilized to analyze cell apoptosis.

Firstly, we are really interested in the author's data about how the Selenium-Modified Chitosan-induced the process of programmed cell death cell via an intrinsic pathway. The inhibition effects of SMC on HepG2 cells were well provided, but the information for normal/non-cancer cells was neither presented nor available to readers to prove that the tested concentrations were not toxic to the normal/non-cancer cells and only showed the inhibition activity on the growth of cancer cells. We also found ourselves missed some information in the figures presented in the results section. Figure 2 in their recently published report showed the inhibitory effects of SMC on HepG2 cells with different concentrations (25-800 µg/mL) and time (24 h, 36 h, 48 h). However, there is a yellow red dash line appears which could confuse the readers. The meaning and detailed explanation of this dash line should be provided by writing a figure legend to the histogram to make it easier for readers to understand it. As shown in Figure 3, the morphological observation results of HepG2 cells after SMC treatments. The authors gave notes: (A) 0 h, (B) 24 h, (C) 36 h, (D) 48 h. To our knowledge, cells undergoing apoptosis show several typical morphological features, including shrinkage of the cell,² fragmentation into membrane-bound apoptotic bodies, rapid phagocytosis by neighboring cells, nuclear chromatin condensation, cytoplasmic vacuolation, and plasma membrane isolation.³ How the different morphological characteristics of shrunken apoptotic bodies of HepG2 cells in the 24 h, 36 h, and 48 h after HepG2 cell treatments with SMC was not clearly labeled in Figure 3. Our suggestion about this figure is to provide a description of the differences in morphological observation in the control, 24 h, 36 h, and 48 h after HepG2 cell treatments with SMC and the measurement or magnification could be put on each figure.

Assay of protein expression levels in this study supported the hypothesis that SMC-induced apoptosis might be mediated through the mitochondrial apoptotic pathway. However, we think that the analysis of biomarker extrinsic apoptotic pathways should be validated in this study to demonstrate that SMC actually induced apoptosis through the intrinsic pathway. In this study, it is not clear whether SMC could increase or decrease the biomarker extrinsic apoptotic pathway. The gene expression method can be performed as an alternative method to analyze all biomarkers of apoptosis, not only the extrinsic pathway but also the intrinsic pathway. Gene expression can support the data in analyzing a profile of gene regulation which has play role in the diseases such as cancer field,⁴ Alzheimer,⁵ allergy,⁶ etc. This method is also used to determine the pattern of apoptotic protein production.⁷ We hope that our inputs can be taken into consideration to increase information in future studies regarding the study of apoptosis induced by bioactive compounds.

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Author Contributions

All authors contributed to data analysis, drafting or revising the article, have agreed on the journal to which the article will be submitted, gave final approval of the version to be published, and agree to be accountable for all aspects of the work.

Disclosure

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