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**Cholestane rhamnosides from *Ornithogalum saundersiae* bulbs and their cytotoxic activity against HL-60, A549 and TIG-3 cells**

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*Ornithogalum saundersiae* (Liliaceae) is native to South Africa and cultivated as an ornamental plant in the world. Previously, we have reported a total of 29 cholestane glycosides, including OSW-1, isolated from the MeOH extract of *O. saundersiae* bulbs and their cytotoxic activity against several malignant tumor cells. In this presentation, we wish to report focus on cholestane rhamnosides isolated from the MeOH extract of *O. saundersiae* bulbs. The concentrated MeOH extract of *O. saundersiae* bulbs was passed through a Diaion HP-20 column eluted with 20% MeOH, EtOH, EtOAc, successively. The EtOH eluate fraction was subjected to column chromatography on silica gel and ODS silica gel, as well as preparative HPLC to give 19 Cholestane Rhamnosides including 12 new naturally occurring compounds. The structures of the new compounds were determined by spectroscopic analysis mainly based on one and two-dimensional NMR and the results of hydrolysis. Cholestane rhamnosides 3, 7-12, 15-17 and 19 exhibited cytotoxic activity against HL-60 human promyelocytic leukemia cells and A549 human lung cancer cells with IC<sub>50</sub> values ranging from 0.05 M to 7.72 M and 0.27 M to 2.41 M, respectively. HL-60 cells treated with (22S)-3, 22-Dihydroxycholest-5, 24-dien-16-yl-L-Rhamnopyranoside (11), which had an IC<sub>50</sub> value of 0.16 M, displayed the hallmark indicators of apoptosis, such as fragmented and condensed nuclear chromatins and activation of caspase-3. In the HL-60 cells treated with 11, the accumulation of sub-G1 cells and G2/M phase cells was shown by flow cytometry analysis. These results suggested that 11 arrests HL-60 cell proliferation in the G2/M phase induces apoptotic cell death. Furthermore, the loss of the mitochondria membrane potential and release of cytochrome c to the cytosol were not observed in the HL-60 cells treated with 11. In conclusion, 11 may induce apoptosis in HL-60 cells via a mitochondria-independent pathway.

### Biography

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