Identifikasi dan mekanisme aksi senyawa sitotoksik pada tanaman sisik naga pyrrosia piloselloides l mg price terhadap sel kanker mcf 7 t47d dan widr = Identification and cytotoxicity mechanism sisik naga pyrrosia piloselloides l mg price cytotoxic compounds on mcf 7 t47d and widr cancer cell

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Abstrak

Kanker payudara menempati urutan pertama banyaknya kejadian kanker pada wanita. Kebutuhan obat antikanker payudara yang mampu menghambat dan mematikan pertumbuhan sel kanker payudara tanpa mempengaruhi sel normal sangat penting. Secara empiris tanaman sisik naga digunakan sebagai obat kanker payudara dan kanker kolon. Ekstrak daun sisik naga mempunyai sifat sitotoksik terhadap MCF-7 dengan IC50 sebesar 83,63 µg/mL. Kandungan kimia sisik naga yang kemungkinan aktif sitotoksik adalah flavonoid, saponin, steroid, tanin dan terpenoid. Flavonoid, saponin dan tanin larut dalam pelarut polar sedangkan terpenoid dan aglikon saponin larut dalam pelarut organik non polar.
Penelitian ini bertujuan mencari aktivitas sitotoksik senyawa hasil isolasi tanaman sisik naga terhadap sel kanker payudara (MCF-7 dan t47D) dan kanker kolon (WiDr) dibandingkan sel normal (Vero). Pengamatan mekanisme kematian sel diamati dengan pengecatan double stanning etidium bromida-akridin orange, % ekspresi p53 dan caspase 3, serta FTIC annexin v-propidium iodida. Ekstraksi kandungan bioaktif sitotoksik menggunakan pelarut n-heksan, diklorometana dan metanol kemudian dilanjutkan fraksinasi dan isolasi menggunakan kromatografi kolom dengan fase diam silika gel 60 dan fase gerak n-heksan, etil asetat dan metanol. Identifikasi struktur dilakukan terhadap senyawa hasil isolasi terpilih yang paling sitotoksik.
Hasil penelitian diperoleh ekstrak diklorometana, fraksi II ekstrak diklorometana dan senyawa I2 hasil isolasi fraksi II ekstrak diklorometana tanaman sisik naga paling toksik terhadap sel MCF-7, t47D, WiDr dan non toksik pada sel Vero. Pemberian senyawa I2 konsentrasi IC50 terhadap sel MCF-7, t47D, dan WiDr menyebabkan peningkatan kematian sel, % ekspresi p53, % ekspresi caspase 3 dan jumlah sel mati karena apoptosis tetapi tidak menimbulkan efek pada sel Vero. Hasil identifikasi spektrum IR, 1H-NMR, 13C-NMR dan MS senyawa I2 adalah -sitosterol (C29H50O). Penapisan in silico nampak -sitosterol berikatan dengan CDK2 dengan sisi aktif pada Leu83 akibatnya fase S dipersingkat kembali menuju G1 dan terjadi apoptosis.
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Breast cancer is the first rank in the occurrence of cancer in women. Therefore, breast cancer drugs that inhibit cell growth and kill breast cancer cells selectively without affecting the normal cells are very important. Sisik naga was empirically used as medicine for breast and colon cancer. Sisik naga leaf extract was revealed to be cytotoxic against MCF-7 with an IC50 of 83.63 µg/ mL. Sisik naga chemical constituents that have cytotoxicity are flavonoid, saponin, steroid, tannin and terpenoid. Flavonoid, saponin and tannin are the soluble in polar solvent, while terpenoid and saponin aglycone are soluble in non polar organic solvent.
This study aims to find the cytotoxic activity of isolated compounds from sisik naga against breast cancer (MCF-7 and t47D) and colon cancer (WiDr) compared to normal (Vero) cells. Observation of the

mechanism of cell death was observed by ethidium bromide-acridine orange double stanning, p53 and caspase 3 expression, and FTIC annexin v-propidium iodide stanning. Cytotoxic bioactive were extracted using hexane, dichloromethane and methanol solvent respectively, followed by fractionation and isolation using column chromatography with silica gel 60 as stationary phase and n-hexane, ethyl acetate and methanol as mobile phase. Identification of the isolated compounds structure with most cytotoxicity was done.

The results showed that dichloromethane extract, fractions II of dichloromethane extract and I2 compounds isolated from fraction II dichloromethane extracts of sisik naga were most cytotoxic to MCF-7, t47D, and WiDr, while non cytotoxic to Vero cells. Exposure to compound I2 at IC50 concentration on MCF-7, t47D, and WiDr caused increased cell death, p53 and caspase 3 expression, and increased of dead cell due to apoptosis, but showed no effect on Vero cell. Identification of IR, 1H-NMR, 13C-NMR spectrum and MS spectrum of I2 compound showed that the compound were -sitosterol (C29H50O). The in silico screening showed that -sitosterol appeared to bind on the active site of CDK2 at Leu83, which cause acceleration from S phase to G1 phase and apoptosis.