

Pengaruh alfa mangostin pada Epithelial-Mesenchymal Transition (EMT) pada sel lestari kanker hati, HepG2 yang tahan terhadap sorafenib melalui jalur TGF-/SMAD = The effect of alpha mangosteen on Epithelial Mesenchymal Transition (EMT) on human hepatocellular carcinoma HepG2 cells surviving sorafenib via TGF-/SMAD pathway

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Abstrak

Epithelial Mesenchymal Transition (EMT) adalah salah satu mekanisme resistensi Sorafenib pada kanker hepatoseluler. Alfa Mangostin diketahui dapat menurunkan aktifitas jalur TGF- \hat{I}^2 pada hepatic stellate cells. Penelitian ini dilakukan untuk mengetahui pengaruh Alfa Mangostin pada Epithelial Mesenchymal Transition (EMT), HepG2 yang tahan terhadap Sorafenib melalui jalur TGF- \hat{I}^2 /SMAD. Sel lestari kanker hati, HepG2 dibagi menjadi enam kelompok perlakuan, yaitu kelompok DMSO 0,01%, Alfa Mangostin20 $\frac{1}{4}$ M, Sorafenib10 $\frac{1}{4}$ M, Sorafenib10 $\frac{1}{4}$ M+Sorafenib10 $\frac{1}{4}$ M, Sorafenib10 $\frac{1}{4}$ M-Alfa Mangostin20 $\frac{1}{4}$ M, dan Sorafenib10 $\frac{1}{4}$ M +Sorafenib10 $\frac{1}{4}$ M - Alfa Mangostin20 $\frac{1}{4}$ M selama 24 jam. Sel dihitung menggunakan trypan blue exclusion method. Kadar TGF- \hat{I}^2 medium diukur menggunakan ELISA. Ekspresi TGF- \hat{I}^2 ,TGF- \hat{I}^2 RI, SMAD3, SMAD7, E-cadherin dan Vimentin diukur dengan qRT-PCR. Pemberian Alfa Mangostin pada sel HepG2 yang tahan terhadap Sorafenib dapat menurunkan jumlah sel hidup. Namun terdapat peningkatan ekspresi TGF- \hat{I}^2 , kadar TGF- \hat{I}^2 aktif, TGF- \hat{I}^2 RI, SMAD3, dan Vimentin serta penurunan ekspresi SMAD7 dan E-cadherin setelah pemberian Sorafenib dan Alfa Mangostin. Alfa Mangostin menurunkan jumlah sel hidup namun tidak menghambat EMT melalui jalur TGF- \hat{I}^2 /SMAD pada sel HepG2 yang tahan terhadap Sorafenib.

Epithelial Mesenchymal Transition (EMT) is one of resistance mechanism through Sorafenib in hepatocellular carcinoma. Alpha Mangosteen is known to have reduced TGF- \hat{I}^2 /SMAD pathway in hepatic stellate cells. This research purpose is to explore the effect of Alpha Mangosteen on Epithelial Mesenchymal Transition (EMT) on human hepatocellular carcinoma HepG2 cells surviving Sorafenib via TGF- \hat{I}^2 /SMAD pathways. Immortalized HCC cell line, HepG2 cells, were divided into 6 groups: DMSO0,01% group, Alpha Mangosteen 20 $\frac{1}{4}$ M group, Sorafenib10 $\frac{1}{4}$ M group, Sorafenib 10 $\frac{1}{4}$ M-Sorafenib10 $\frac{1}{4}$ M group, Sorafenib10 $\frac{1}{4}$ M + Alpha Mangosteen 20 $\frac{1}{4}$ M group, and Sorafenib10 $\frac{1}{4}$ M + Sorafenib10 $\frac{1}{4}$ M - Alpha Mangosteen 20 $\frac{1}{4}$ M group for 24 h. Cells were harvested and counted by trypan blue exclusion method. TGF- \hat{I}^2 medium concentration was evaluated by ELISA. Expression of TGF- \hat{I}^2 ,TGF- \hat{I}^2 RI, SMAD3, SMAD7, E-cadherin and Vimentin measured by qRT-PCR. Alpha Mangosteen administration on HepG2 surviving Sorafenib cells reduced mean live cells. However, the expression of TGF- \hat{I}^2 , TGF- \hat{I}^2 active concentration, TGF- \hat{I}^2 R, SMAD3, and Vimentin were elevated. Alpha Mangosteen also decreased SMAD7 and E-cadherin expression. Alpha Mangosteen reduced live cells but did not have effect on preventing EMT activation through TGF- \hat{I}^2 /SMAD pathways on HepG2 surviving Sorafenib cells.