

Efek Avidin terhadap Viabilitas dan Proliferasi Sel Kanker Kolorektal HT-29 sebagai Kandidat Anti-Kanker = The Effect of Avidin on Viability and Proliferation of Colorectal Cancer HT-29 Cells as Anti-Cancer Candidate

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Abstrak

Sel kanker adalah sel yang berproliferasi secara progresif, dan salah satu dasar pengendaliannya hingga saat ini yaitu dengan menghambat kemampuan proliferasinya melalui intervensi sintesis nukleotida purin/pirimidin menggunakan analog purin/pirimidin. Avidin, suatu protein yang ditemukan pada putih telur, diketahui dapat mengikat biotin dengan sangat kuat, yang merupakan koenzim pada reaksi karboksilasi, suatu tahapan penting di biosintesis de novo nukleotida purin. Studi sebelumnya membuktikan bahwa viabilitas dan proliferasi sel mononuklear darah tepi (SMDT) dapat dihambat dengan penambahan avidin yang diakibatkan gangguan ketersediaan biotin. Studi ini bertujuan melihat efek pemberian avidin terhadap sel kanker kolorektal HT-29 dilihat dari viabilitas, proliferasi, ekspresi gen dan protein cyclin D1, serta siklus sel. Penelitian dilakukan dengan mengultur sel kanker kolorektal HT-29 dengan avidin, lalu dianalisis viabilitas, proliferasi, ekspresi gen dan protein cyclin D1, serta siklus selnya pada 24, 48, dan 72 jam. Didapatkan hasil bahwa avidin menghambat viabilitas dan proliferasi sel HT-29, serta menurunkan ekspresi gen dan protein cyclin D1 pada sel HT-29, namun tidak memengaruhi transisi fase G0/G1 ke fase S siklus sel HT-29.

.....Cancer cells are progressively proliferating cell, and up to now, one way to control its proliferation is by intervening the formation of purine/pyrimidine nucleotide using its purine/pyrimidine analog. Avidin, a protein from white egg, known to bind biotin strongly, whereas biotin is an important coenzyme in carboxylation reaction, a key step in purine nucleotide de novo pathway. Previous study showed that viability of peripheral blood mononuclear cells (PBMC) was reduced and its proliferation was inhibited caused by lack of biotin due to avidin administration. This study aims to observe the effect of avidin administration to HT-29 cells viability, proliferation, cyclin D1 gene and protein expression, also the cell cycle. The experiment done by culturing HT-29 cells, then its viability, proliferation, cyclin D1 gene and protein expression, also the cell cycle analyzed at 24, 48, and 72 hours. The result showed that avidin halted HT-29 cells viability and proliferation, also lower its cyclin D1 gene and protein expression, but did not affect the transition between G0/G1 phase to S phase on HT-29 cell cycle