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Penghambatan proliferasi sel mononukleus darah tepi (SMDT) manusia oleh avidin, pascastimulasi fitohemaglutinin (PHA) dan interleukin-2 (IL-2) = Inhibition of peripheral blood mononuclear cell (PBMC) proliferation by avidin pascastimulation of phytohemagglutinin (PHA) and interleukin-2 (IL-2)

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Abstrak

[ABSTRAK
Proliferasi sel merupakan peningkatan dalam jumlah sel sebagai hasil dari pertumbuhan dan pembelahan sel. Selain terjadi pada sel normal pembelahan sel juga terjadi pada sel kanker yang ditandai dengan proliferasi tak terkendali. Banyak di antara penghambatan proliferasi dilakukan dengan cara menghambat sintesis DNA, yaitu mengintervensi pembentukan basa nukleotida purin atau pirimidin. Mengingat dalam sintesis purin de novo terdapat peran biotin yang merupakan koenzim dalam proses karboksilasi, maka penambahan avidin diduga kuat dapat mengikat biotin dengan afinitas yang sangat tinggi. Penelitian ini bertujuan untuk mempelajari potensi avidin dalam kemampuannya mengikat botin untuk menghambat mitosis. Pada penelitian ini SMDT dikultur dalam medium yang distimulasi oleh PHA, IL-2, serta PHA dan IL-2 dengan dan tanpa avidin. Efek dari penambahan avidin ini dilihat pada jam-jam tertentu dan dilakukan analisis terhadap proliferasi, viabilitas, serta siklus sel. Berdasarkan hasil penelitian, avidin menghambat proliferasi SMDT serta menurunkan viabilitas SMDT baik pada kultur yang distimulasi PHA maupun pada kultur yang distimulasi PHA dan IL-2. Penambahan avidin juga menghambat masuknya progresi SMDT yang dikultur selama 72 jam dari fase G0/G1 ke fase S. Penelitian ini menunjukkan bahwa avidin dapat mengikat biotin yang ada dalam medium sehingga proliferasi sel menjadi terhambat.
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ABSTRACT
Cell proliferation is the increment of cell number as a result of cell growth and cell division. Cell division occurs not only in normal cells but also in cancer cells which undergo uncontrolled cell division. Most of the cell proliferation inhibition was done by inhibiting the DNA synthesis by which intervening the formation of purine or pyrimidine nucleotide bases. Considering the role of biotin in purine de novo synthesis as a coenzyme in the carboxylation reaction, it was assumed that avidin can bind biotin with very high affinity. The aim of this research is to study the potential of avidin to bind biotin for inhibit mitosis. In this study PBMC was cultured in a medium that stimulated by PHA, IL-2, PHA and IL-2 with and without avidin. The effect of the addition of avidin was observed at certain hours

for the analysis of proliferation, viability, and cell cycle. This study suggest that avidin inhibits proliferation and decreases viability of PBMC both of PBMC stimulated by PHA and stimulated by PHA and IL-2. The addition of avidin also inhibits the entry of progression of PBMC when cultured for 72 hours from phase G0/G1 to S phase. Based on these data, we propose that avidin might bind extracellular biotin in the medium therefore the cell proliferation was inhibited;Cell proliferation is the increment of cell number as a result of cell growth and cell division. Cell division occurs not only in normal cells but also in cancer cells which undergo uncontrolled cell division. Most of the cell proliferation inhibition was done by inhibiting the DNA synthesis by which intervening the formation of purine or pyrimidine nucleotide bases. Considering the role of biotin in purine de novo synthesis as a coenzyme in the carboxylation reaction, it was assumed that avidin can bind biotin with very high affinity. The aim of this research is to study the potential of avidin to bind biotin for inhibit mitosis. In this study PBMC was cultured in a medium that stimulated by PHA, IL-2, PHA and IL-2 with and without avidin. The effect of the addition of avidin was observed at certain hours for the analysis of proliferation, viability, and cell cycle. This study suggest that avidin inhibits proliferation and decreases viability of PBMC both of PBMC stimulated by PHA and stimulated by PHA and IL-2. The addition of avidin also inhibits the entry of progression of PBMC when cultured for 72 hours from phase G0/G1 to S phase. Based on these data, we propose that avidin might bind extracellular biotin in the medium therefore the cell proliferation was inhibited, Cell proliferation is the increment of cell number as a result of cell growth and cell division. Cell division occurs not only in normal cells but also in cancer cells which undergo uncontrolled cell division. Most of the cell proliferation inhibition was done by inhibiting the DNA synthesis by which intervening the formation of purine or pyrimidine nucleotide bases. Considering the role of biotin in purine de novo synthesis as a coenzyme in the carboxylation reaction, it was assumed that avidin can bind biotin with very high affinity. The aim of this research is to study the potential of avidin to bind biotin for inhibit mitosis. In this study PBMC was cultured in a medium that stimulated by PHA, IL-2, PHA and IL-2 with and without avidin. The effect of the addition of avidin was observed at certain hours for the analysis of proliferation, viability, and cell cycle. This study suggest that avidin inhibits proliferation and decreases viability of PBMC both of PBMC stimulated by PHA and stimulated by PHA and IL-2. The addition of avidin also inhibits the entry of progression of PBMC when cultured for 72 hours from phase G0/G1 to S phase. Based on these data, we propose that avidin might bind extracellular biotin in the medium therefore the cell proliferation was inhibited]