Colesterol. As estatinas podem aumentar o risco de câncer

Statins, low-density lipoprotein cholesterol, and risk of cancer.

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OBJECTIVES: We sought to assess whether statin-mediated reductions in low-density lipoprotein cholesterol (LDL-C) are associated with an increased risk of cancer. BACKGROUND: We recently reported an inverse association between on-treatment LDL-C levels and incident cancer in statin-treated patients enrolled in large randomized controlled trials, raising concern that LDL-C lowering by statins may increase cancer risk. However, meta-analyses suggest a neutral overall effect of statins on incident cancer. METHODS: A systematic literature search identified 15 eligible randomized controlled trials of statins with >or=1,000 person-years of follow-up that provided on-treatment LDL-C levels and rates of incident cancers (19 statin and 14 control arms, 437,017 person-years cumulative follow-up, and 5,752 incident cancers). RESULTS: In the statin arms, meta-regression analysis demonstrated an inverse association between on-treatment LDL-C and incident cancer, with an excess of 2.2 (95% confidence interval: 0.7 to 3.6) cancers per 1,000 person-years for every 10 mg/dl decrement in on-treatment LDL-C (p=0.006). The corresponding difference among control arms was 1.2 (95% confidence interval: -0.2 to 2.7, p=0.09). Compared with the control arms, the statin regression line was significantly shifted leftward, such that similar rates of incident cancer were associated with lower on-treatment LDL-C (p<0.05). Meta-regression demonstrated that statins lack an effect on cancer risk across all levels of on-treatment LDL-C. CONCLUSIONS: There is an inverse association between on-treatment LDL-C and incident cancer. However, statins, despite producing marked reductions in LDL-C, are not associated with an increased risk of cancer.

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Independent associations between low-density lipoprotein cholesterol and cancer among patients with type 2 diabetes mellitus.

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BACKGROUND: The risk association between low-density lipoprotein (LDL) cholesterol and cancer remains controversial and largely unexplored for people not receiving statin therapy. METHODS: We examined the risk association between LDL cholesterol and cancer among patients with type 2 diabetes mellitus who were free of cancer at enrolment and whose statin use was known. We considered a variety of nonlinear relationships in our analysis. RESULTS: During a median follow-up period of 4.90 years, cancer developed in 270 (4.4%) of 6107 patients. Among the 3800 patients who did not receive statin therapy, the risk association between LDL cholesterol and cancer was represented by a V-shaped curve. Compared with patients whose LDL cholesterol was at least 2.80 mmol/L but less than 3.80 mmol/L, the risk of cancer, death from any cause or the composite outcome of cancer or death was greater among those with an LDL cholesterol level of less than 2.80 mmol/L (hazard ratio for cancer 1.87, 95% confidence interval [CI] 1.29-2.71) and those with an LDL cholesterol level of 3.80 mmol/L or greater (hazard ratio for cancer 1.87, 95% CI 1.19-1.99) among patients not using statins; the hazard ratio was reduced to 1.24 (1.01-1.53) for the entire sample (statin users and those not using statins). These associations persisted after adjustment for covariates and exclusion of patients with less than 2.5 years of follow-up. INTERPRETATION: Among patients with type 2 diabetes, the association between LDL cholesterol and cancer was V-shaped, whereby both low and high levels of LDL cholesterol were associated with elevated risk of cancer.

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Statins and cancer: A systematic review and meta-analysis.

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BACKGROUND: Systematic reviews on the association between statin therapy and cancer have focused on randomised trials without assessing the quality of evidence. We aimed to review the overall evidence taking study quality into consideration. METHODS: Publications of original studies on the effect of statin treatment on cancer in adult patients were searched on MEDLINE, EMBASE and CENTRAL databases up to October 2007. Our search yielded 37 eligible original studies out of 3607 references. Five studies were additionally found through manual search. Thus, 42 studies were included in the analyses: 17 randomised controlled trials, 10 cohort studies, and 15 case-control studies. FINDINGS: Statins had no effect on the overall incidence of cancer (median risk ratio (RR) 0.96, range 0.72 to 1.2), or on the incidence of lung (median RR 0.92, range 0.83 to 3.0), breast (median RR 1.04, range 0.47 to 19) or prostate cancer (median RR 0.96, range 0.33 to 1.7). They seemed to protect from stomach (median RR 0.59, range 0.40 to 0.88) and liver cancer (median RR 0.62, range 0.33 to 1.2), and from lymphoma (median RR 0.74, range 0.28 to 2.2). They increased the incidence of both melanoma (median RR 1.5, range 1.3 to 1.7) and non-melanoma skin cancer (median RR 1.6, range 1.2 to 2.2). The effect varied, yet inconsistently, by statin type. The median follow-up time was 4 years. The strength of evidence was mostly weak. INTERPRETATION: The evidence suggests that statins do not have short-term effects on cancer risk. The evidence on potentially protective or harmful effects is inconclusive. High quality cohort studies with long follow-up are needed to resolve the issue.

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Non-cardiovascular mortality, low-density lipoprotein cholesterol and statins: a meta-regression analysis.

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BACKGROUND: As of today, the effect of statins on non-cardiovascular mortality is still being debated. Single studies have not been able to provide definite answers. We performed a meta-regression analysis on randomized statin trials in order to provide evidence that non-cardiovascular mortality is related to statin treatment and low-density lipoprotein (LDL) cholesterol plasma level. METHODS: We selected 29 randomized controlled trials of statins versus placebo, a total of 90,480 patients, with a follow-up of >12 months. Baseline and follow-up LDL levels and all-cause, cardiovascular and non-cardiovascular mortality were recorded. Weighted linear regression analysis was carried out separately for placebo and treatment groups. RESULTS: LDL level was inversely related to overall mortality (p = 0.0105) and non-cardiovascular mortality (p = 0.0171) in the treatment group. By contrast, in the placebo group only non-cardiovascular mortality was inversely correlated to LDL (p = 0.0032). The regression lines have similar slopes and run almost parallel to each other, with the treatment line lying below the placebo line. To identify the threshold of risk for starting statin therapy, we analysed the relationship between baseline cardiovascular risk and overall mortality in the two groups. Both correlations are highly significant and regression lines intersect at a risk of 0.29% per year. This implies that the effects of statins are favourable when the baseline cardiovascular risk exceeds approximately 3% in 10 years. CONCLUSIONS: A trend of increased non-cardiovascular mortality with decreased LDL exists both in placebo and treatment groups.

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As estatinas diminuem os níveis de PSA e complicam a detecção do câncer de próstata
By lowering PSA levels, statins may complicate cancer detection, although further studies are needed to quantify the clinical significance of this effect.