PENGARUH TEPUNG LABU KUNING TERHADAP EKSPRESI SREBP-1C JARINGAN HEPAR DAN ADIPOSA PADA TIKUS MODEL DYSLIPIDEMIA

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INTISARI

Latar Belakang: Peningkatan ekspresi SREBP-1c akibat diet tinggi lemak dan fruktosa dapat meningkatkan proses lipogenesis *de novo*. Lipogenesis yang terjadi terus menerus dapat menyebabkan terjadinya dislipidemia serta meningkatkan peroksidasi lipid dan membentuk *reactive oxygen species* (ROS), *singlet oxygen* (O^-) dan hidrogen peroksida (H_2O_2). Labu kuning adalah bahan makanan yang mengandung karoten dan berfungsi sebagai antioksidan. Penelitian sebelumnya menunjukkan pemberian karotenoid dapat menurunkan ekspresi SREBP-1c. Karoten pada labu kuning memiliki potensi yang besar dalam menurunkan ekspresi SREBP-1c di dua jaringan utama lipogenesis yaitu hepar dan adiposa putih.
Tujuan: Mengetahui pengaruh pemberian tepung labu kuning terhadap ekspresi SREBP-1c di jaringan hepar dan adiposa putih.
Metode: Tikus *Sprague Dawley* dibagi dalam 5 kelompok (Kelompok kontrol normal : tikus sehat; Kelompok kontrol dislipidemia : diet tinggi lemak & fruktosa (DTLF) ; Kelompok tepung labu kuning 0,16g/200g BB: DTLF + tepung labu kuning 0,16g/200g BB ; Kelompok tepung labu kuning 0,16g/200g BB:: DTLF + tepung labu kuning 0,32g/200g BB; Kelompok tepung labu kuning 0,64g/200g BB: DTLF + tepung labu kuning 0,64g/200g BB). Pemberian diet tinggi lemak & fruktosa (DTLF) dilakukan selama 25 hari, selanjutnya dilakukan pemeriksaan kadar trigliserida. Apabila kadar trigliserida >100 mg/dL, tikus diberi tepung labu kuning selama 4 minggu. Pada akhir penelitian dilakukan pengambilan jaringan hepar dan adiposa putih untuk pemeriksaan ekspresi SREBP-1c dengan metode *real time* PCR. Data dianalisis dengan uji parametrik *one-way* ANOVA dan uji t tidak berpasangan. Korelasi dosis tepung labu kuning dengan tingkat ekspresi SREBP-1c menggunakan uji korelasi Pearson. Perbedaan dinilai bermakna apabila nilai probabilitas $p<0,05$.
Hasil: Tingkat ekspresi SREBP-1c di jaringan hepar dan adiposa putih pada tikus dislipidemia tanpa perlakuan adalah 1,30±0,04 and 1,07±0,0. Sementara kelompok tikus dislipidemia yang diberikan tepung labu kuning dosis 0,32g/200g BB berturut-turut adalah 1,13±0,01 dan 0,99±0,04. Nilai ini mendekati ekspresi SREBP-1c pada tikus normal yaitu 1,11±0,01 dan 0,99±0,04 dengan perbedaan yang bermakna antar kelompok. Kekuatan korelasi (r) di jaringan hepar dan adiposa putih berturut-turut adalah -0,662 dan -0,757 ($p<0,05$)
Kesimpulan: Ekspresi SREBP-1c lebih rendah pada tikus yang diberikan tepung labu kuning di kedua jaringan dengan dosis optimal yaitu 0,32g/200g BB. Ekspresi SREBP-1c di hepar lebih tinggi dibandingkan adiposa. Terdapat korelasi negatif antara dosis tepung labu kuning dengan ekspresi SREBP-1c yang bermakna secara statistik.

Kata kunci : dislipidemia, *sterol regulatory element binding protein*-1c (SREBP-1c), labu kuning, karotenoid.

EFFECT OF PUMPKIN POWDER ON HEPATIC AND WHITE ADIPOSE TISSUE SREBP-1C EXPRESSION IN DISLIPIDEMIA RATS MODEL

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ABSTRACT

Background: Increased expression of SREBP-1c because of high fat and fructose diet can improve the process of lipogenesis de novo. Lipogenesis that occurs continuously can cause dyslipidemia and increase lipid peroxidation and form reactive oxygen species (ROS), singlet oxygen (O^-) and hydrogen peroxide (H_2O_2). Pumpkin is a food that contains carotenoids act as antioxidants. Several previous studies have shown the provision of carotenoids can reduce the expression of SREBP-1c. The content of carotenoids in pumpkin has great potential in reducing the expression of SREBP-1c in the two major tissues for lipogenesis, the liver and white adipose tissues.
Objective: Knowing the effect of pumpkin powder on the expression of SREBP-1c in liver and white adipose tissue.
Method: Sprague-Dawley rats divided into five groups (normal control group: healthy rats; control group of dyslipidemia: high fat-fructose diet (HFFD); dose 1 group: HFFD+ 0.16g / 200g of weight pumpkin powder; dose 2 group: HFFD + 0.32g / 200 g of weight pumpkin powder; dose 3 group: HFFD+ 0.64 g / 200g of weight pumpkin powder). Rats were maintained control normal diet (CND) and high fat-fructose diet (HFFD) for 25 days. After 25 days, rats were examined for its triglycerides level. After their triglycerides level exceeded 100 mg/dL, rats were maintained with semipurified diet only (either normal and dyslipidemia control group) and semipurified diet+ different dose of pumpkin powder for 4 weeks. At the end of the study, liver and white adipose tissue were collected for subsequent SREBP-1c determination using real time PCR. Data were analyzed using parametric test one-way ANOVA and unpaired t test. Pearson correlation test used for analyze correlation between dose of pumpkin flour and the level of expression of SREBP-1c. Differences assessed significant when the probability value of $p < 0.05$.
Result: SREBP-1c expression levels in liver and white adipose tissue in dyslipidemia rats without treatment, respectively, was 1.30±0.04 and 1.07±0.01. While SREBP-1c expression in liver and white adipose tissue with treatment of 0.32g / 200g of weight pumpkin powder, respectively, was 1.13 ± 0.01 and 0.99 ± 0.04 were lower than rats group without treatment and approaching the SREBP-1c expression in normal rats 1.11 ± 0.01 and 0.99 ± 0.04 with a significant difference between groups. The strength of the correlation (r) in the liver and white adipose tissue, respectively, were -0.662 and -0.757 ($p < 0.05$)
Conclusion: SREBP-1c expression was lower in rats given pumpkin powder on both tissues. The optimal dose to suppress SREBP-1c close to or the same as the normal is 0,32g / 200g of weight. SREBP-1c expression in liver are higher than white adipose tissues. There is a negative correlation between the dose of pumpkin flour with the expression of SREBP-1c and statistically significant.

Keyword: dyslipidemia, sterol regulatory element binding protein-1c (SREBP-1c), pumpkin, carotenoid.