Efek Proteksi 6-Gingerol terhadap Protein Penanda Inflamasi Tumor Necrosis Factor Alpha (TNF-@) di Jaringan Jantung Tikus Sprague-Dawley Model Sindrom Metabolik = Effect of 6-Gingerol on Inflammatory Biomarker TNF-Î± in Cardiac Tissue of Sprague-Dawley Rats with Metabolic Syndrome Model

Syifa Nurfitriyanti, author

Abstrak

Latar Belakang
Sindrom metabolik secara signifikan dikaitkan dengan peningkatan risiko penyakit kardiovaskular. Inflamasi kronis merupakan salah satu mekanisme yang diusulkan memiliki peran penting dalam perkembangan sindrom metabolik menjadi penyakit kardiovaskular. Suatu penelitian eksperimental menunjukkan bahwa penargetan spesifik dari proses inflamasi terbukti dapat mengurangi perkembangan penyakit ini. Sitokin proinflamasi Tumor Necrosis Factor- (TNF-) telah diidentifikasi sebagai regulator utama respon inflamasi dan dianggap sebagai sitokin alarm yang bertanggung jawab dalam menginisiasi dan mempertahankan kondisi inflamasi sehingga TNF- dipilih sebagai target pertama dalam cytokine-targeted approach. 6-Gingerol terbukti memiliki beberapa aktivitas farmakologis, termasuk anti-inflamasi. Penelitian dilakukan untuk mengetahui efek proteksi 6-Gingerol terhadap proses inflamasi jantung yang disebabkan oleh sindrom metabolik melalui penurunan protein penanda inflamasi TNF-.
Metode
Tikus jantan Sprague-Dawley dikategorikan menjadi lima kelompok, yaitu normal sehat, sindrom metabolik (MetS), MetS + 6-Gingerol 50 mg/kgBB, MetS + 6-Gingerol 100 mg/kgBB, serta MetS + 6-Gingerol 200 mg/kgBB. Dilakukan pemberian diet tinggi lemak-tinggi fruktosa selama 16 minggu serta injeksi streptozotocin intraperitoneal (22 mg/kg) pada minggu ke-8 untuk menginduksi model sindrom metabolik. Di akhir penelitian, hewan diterminasi dan dilakukan pengambilan sampel jantung. Tingkat ekspresi TNF- pada jaringan jantung diukur menggunakan BioEnzy© ELISA kit (Rat TNF- ELISA Kit).
Hasil
Penelitian menunjukkan penurunan ekspresi TNF- secara signifikan pada kelompok tikus MetS dengan pemberian 6-Gingerol dosis 200 mg/kgBB ($p<0.001$) dibandingkan dengan kelompok tikus MetS.
Kesimpulan
6-Gingerol berpotensi untuk memperbaiki proses inflamasi jantung pada sindrom metabolik pada jantung tikus dengan sindrom metabolik melalui penurunan ekspresi protein TNF-.
.....Introduction
Metabolic syndrome (MetS) is significantly associated with an increased risk of developing cardiovascular diseases (CVDs). Chronic inflammation seems to be essential players in the progression of MetS and its subsequent transition to CVDs. Specific targeting of these processes in experimental models has been shown to reduce disease progression. Tumor Necrosis Factor- (TNF-) has been identified as a major regulator of inflammatory responses and considered as alarm cytokines which initiate and maintain inflammation, therefore, it was selected as the first target in the cytokine-targeted approach. 6-Gingerol has been reported to have a myriad of promising pharmacological activities including notable anti-inflammatory

potential. Hence, research was conducted to determine the protective effect of 6-Gingerol in cardiac inflammatory process induced by metabolic syndrome through reducing the inflammatory marker TNF-.
Metode
Male Sprague-Dawley rats were categorized into five groups: standard commercial diet, metabolic syndrome (MetS), MetS + 6-Gingerol 50 mg/kgBW, MetS + 6-Gingerol 100 mg/kgBW, and MetS + 6-Gingerol 200 mg/kgBW. Rats were fed with a high-fat high-fructose diet for 16 weeks and at Week 8, single-dose low-dose streptozotocin (22 mg/kg) were intraperitoneally injected to induce MetS. After all animals were terminated, cardiac tissue was harvested to measure TNF- levels. TNF- levels was measured using BioEnzy© ELISA kit (Rat TNF- ELISA Kit).
Hasil
This study shows significant decrease of TNF- levels in cardiac tissue in the MetS group administered with a dose of 6-Gingerol at 200 mg/kgBW ($p<0.001$) as compared to the MetS group.
Kesimpulan
6-gingerol potentially attenuates inflammation process in cardiac tissue of syndrome metabolic by their ability to reduce TNF- protein expression