

## Negative impact of inflammation and insulin resistance on the biogenesis of HDL-c in Indonesian men with metabolic syndrome

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### Abstrak

Tujuan Mempelajari keterkaitan antara inflamasi dan resistensi insulin dengan gangguan biogenesis HDL yang menyebabkan rendahnya konsentrasi HDL. Metode Penelitian ini menggunakan desain potong lintang dengan jumlah subyek 163 pria dewasa berusia 25-60 tahun dengan sindroma metabolik (kriteria IDF, 2005), tanpa gangguan fungsi hati dan ginjal. Penelitian ini dilakukan di Jakarta pada tahun 2007-2009. Indikator-indikator yang diukur adalah apolipoprotein A-1 (apoA-1), prebeta-1 HDL, cholesteryl ester transfer protein (CETP), kolesterol HDL, berat badan, tinggi badan, lingkar perut (LP), tekanan darah sistolik (TDS), tekanan darah diastolik (TDD), glukosa darah puasa (GDP), dan trigliserida serum. Rasio apoA-1/HDL-c diambil sebagai indikator maturasi HDL, sedangkan rasio CETP/HDL-c dan CETP/TG menunjukkan katabolisme HDL. high sensitivity-CRP (hsCRP), HOMA-IR digunakan sebagai indikator inflamasi dan resistensi insulin. Data dianalisis dengan menggunakan analisis univariat, bivariat, dan multivariat. Hasil Hasil penelitian menunjukkan bahwa hsCRP berkorelasi positif dengan CETP ( $r_s = 0,200$ ,  $p = 0,042$ ), dan rasio CETP/HDL-c ( $r_s = 0,188$ ,  $p = 0,013$ ); HOMA-IR berkorelasi positif dengan rasio apoA-1/HDL-c ( $r_s = 0,190$ ,  $p = 0,016$ ) dan berkorelasi negatif dengan rasio CETP/TG ( $r_s = -0,162$ ,  $p = 0,04$ ). Hasil analisis general linear model (GLM) menunjukkan hsCRP memiliki kontribusi terbesar terhadap rasio CETP/HDL-c, apoA-1, dan CETP (berturut-turut  $p = 0,009$ ;  $0,016$ ;  $0,054$ ). Kesimpulan Penelitian ini menyimpulkan adanya hubungan antara inflamasi dan resistensi insulin dengan gangguan biogenesis HDL pada pria dengan SM. Inflamasi berkaitan dengan peningkatan katabolisme kolesterol HDL, sedangkan resistensi insulin berkaitan dengan penurunan maturasi dan peningkatan katabolisme kolesterol HDL, yang akhirnya berkontribusi terhadap rendahnya konsentrasi kolesterol HDL. Inflamasi memiliki kontribusi yang lebih bermakna terhadap faktor biogenesis HDL daripada resistensi insulin.

*Aim* To find out the relationship between inflammation and insulin resistance with impaired HDL biogenesis that cause low HDL-c concentration *Methods* Using a cross-sectional design, this study involved 163 adult men, aged 25-60 years old with metabolic syndrome (IDF criteria, 2005), without liver and kidney dysfunction. This study was undertaken in Jakarta in the year 2007-2009. Measured indicators were serum apolipoprotein A-1 (apoA-1), prebeta-1 HDL, cholesteryl ester transfer protein (CETP), HDL cholesterol (HDL-c), body weight, height, waist circumference (WC), systolic blood pressure (SBP), diastolic blood pressure (DBP), fasting blood glucose (FBG), and triglyceride. The apoA-1/HDL-c ratios were taken as indicator of HDL maturation, whereas CETP/HDL-c and CETP/TG ratios were indicated HDL catabolism. high-sensitivity CRP (hsCRP) and HOMA-IR were taken as indicator of inflammation and insulin resistance, respectively. Data were analyzed by using univariate, bivariate, and multivariate analysis. Results Positive correlations were found between hsCRP and CETP ( $r_s = 0.200$ ,  $p = 0.042$ ), and CETP/HDL-c ratios ( $r_s = 0.188$ ,  $p = 0.013$ ). HOMA-IR positively correlated with apoA-1/HDL-c ratios ( $r_s = 0.190$ ,  $p = 0.016$ ) and negatively correlated with the CETP/TG ratios ( $r_s = -0.162$ ,  $p = 0.04$ ). Results of general linear model analysis showed that serum hsCRP concentration had the highest contribution to CETP/HDL-c ratios, apoA-

1, dan CETP (p= 0.009; 0.016; 0.054, respectively). Conclusions Inflammation and insulin resistance related to dysfunction of HDL biogenesis in men with metabolic syndrome. The inflammation correlated with increased HDL catabolism, whereas the insulin resistance correlated with decreased HDL maturation and increased HDL catabolism. These may lead to low HDL-c concentration. Inflammation had higher contribution to HDL biogenesis factors than insulin resistance.</i>