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# Improvements in cholesterol efflux capacity of HDL and adiponectin contribute to mitigation in cardiovascular disease risk after bariatric surgery in a cohort with morbid obesity

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## Abstract

**Background:** Bariatric surgery can alleviate cardiovascular risk via effects on cardiovascular disease (CVD) risk factors such as diabetes mellitus, hypertension, and dyslipidemia. Our study aimed to assess the cholesterol efflux capacity (CEC) of HDL as a negative risk factor for CVD in individuals with obesity and identify the factors associated with improvement in CEC 3 months following bariatric surgery.

**Methods:** We recruited 40 control individuals (mean BMI of 22.2 kg/m<sup>2</sup>) and 56 obese individuals (mean BMI of 45.9 kg/m<sup>2</sup>). The biochemical parameters, inflammatory status and CEC of HDL was measured for the obese individuals before bariatric surgery and at 3 months after surgery. The CEC was measured using a cell-based cholesterol efflux system of BODIPY-cholesterol-labelled THP-1 macrophages.

**Results:** A significant reduction in BMI (− 17%,  $p < 0.001$ ), resolution of insulin sensitivity (HOMA2-IR = − 23.4%,  $p = 0.002$ ; Adipo IR = − 16%,  $p = 0.009$ ) and inflammation [log resistin = − 6%,  $p = 0.07$ ] were observed 3 months post-surgery. CEC significantly improved 3 months after surgery [Pre:  $0.91 \pm 0.13$ ; Post:  $1.02 \pm 0.16$ ;  $p = 0.001$ ] despite a decrease in HDL-C levels. The change in CEC correlated with the change in apo A-I ( $r = 0.39$ ,  $p = 0.02$ ) and adiponectin levels ( $r = 0.35$ ,  $p = 0.03$ ).

**Conclusion:** The results suggest that improvements in CEC, through improvement in adipose tissue health in terms of adipokine secretion and insulin sensitivity could be an important pathway in modulating obesity-related CVD risk.

**Keywords:** Bariatric surgery, Cardiovascular risk, Dyslipidemia, Insulin resistance, Cholesterol efflux capacity, Adiponectin

## Introduction

Cardiovascular disorders (CVD) account for about one-third of the global deaths. One of the modifiable risk factors for the development of CVD is obesity [1]. According to the World Health Organization, about 13% of the world's adult population in 2016 was plagued by obesity. The obesity-induced metabolic alterations predispose an

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