Sleep disordered breathing (SDB), an entity characterized by repetitive upper airway obstruction, represents a prevalent disorder affecting millions of individuals. The implications of untreated SDB are far-reaching including decrements in quality of life and furthermore, based upon recent data, increased cardiovascular risk and mortality. Hyperlipidaemia and derangements of lipids such as low density lipoprotein (LDL) and high density lipoprotein (HDL) are also common with an estimated prevalence of 54.9 per cent for men and 46.5 per cent for women, and are major risk factors for coronary heart disease. Cholesterol profiles of atherogenic dyslipidaemia are key components of the metabolic syndrome which encompasses substantive cardiovascular risk factors, and overall connotes increased cardiovascular risk and mortality. Given the common underlying microenvironnement, the term Syndrome Z has been coined to incorporate SDB with other commonly recognized metabolic syndrome risk factors including hypertension, central obesity, insulin resistance and measures of atherogenic dyslipidaemia- specifically hypertriglyceridaemia (>150 mg/dl) and low HDL levels (<40 mg/dl in men and <50 mg/dl in women). Although it is plausible that SDB, through mechanisms of enhanced intermittent sympathetic nervous activation, intermittent hypoxia, ventilatory overshoot hyperoxia, and sleep disruption may lead to alterations in lipid metabolism; the clarity of these mechanistic pathways and degree by which obesity mediates these relationships continue to be active areas of investigation.

In this issue Chou and colleagues report on a Taiwanese referral-based study demonstrating that unlike the apnoea hypopnoea index; the oxygen desaturation index defined as the frequency of 4 per cent oxygen desaturation episodes per hour of sleep was significantly associated with hypercholesterolaemia (>200 mg/dl) and hypertriglyceridaemia (>150 mg/dl) even after taking into account confounding factors such as obesity as defined by body mass index. The findings from Chou and colleagues add to growing literature highlighting the importance of the specific role of SDB-related intermittent hypoxia in lipid metabolism. Intermittent hypoxia may impact key steps of hepatic lipid metabolism including enhancing lipid biosynthesis and lipoprotein secretion and affecting reverse cholesterol transport. Provocative animal experiments have demonstrated that intermittent hypoxia may be important means by which dyslipidaemia may occur. Specifically, exposure of lean mice to 5 days of intermittent hypoxia increased serum cholesterol and phospholipid levels, upregulated pathways of triglyceride and phospholipid biosynthesis and inhibited pathways of cholesterol uptake in the liver. Interestingly, in this study, intermittent hypoxia increased total cholesterol and HDL, but not LDL. The same group has identified intermittent hypoxia-related upregulation of hepatic stearoyl coenzyme A desaturase-1 (SCD-1), an enzyme that, in a hypoxic environment, is expected to increase triglyceride synthesis more than cholesterol and is expected to enhance hepatic release of lipids, and has also described the development of steatohepatitis in severe obesity in response to hypoxic stress. Clinical research data are sparse and discrepant regarding the presence of an association between SDB and dyslipidaemia, and whether any of the noted relationships are independent of obesity. Data from the Cleveland Family Study have demonstrated that over a 5 yr period of time, cholesterol is a risk factor for incident SDB defined by the apnoea hypopnoea index. These data suggest that individuals with elevated cholesterol levels may represent a phenotype of augmented SDB risk.

In summary, given the disparity in results published so far, more clinical research as provided by...
Chou and colleagues\textsuperscript{7} is needed to better characterize the relationships between SDB and dyslipidaemia, particularly from epidemiologic cohorts enrolling community dwelling individuals that would have limited referral bias. In particular, examination of measures of intermittent hypoxia as it relates to cholesterol profiles would be of interest as only limited data are published in this regard. Investigating the relationship of intermittent hypoxia and oxidative stress mechanisms as it relates to lipid metabolism would be of interest to identify culprit pathways resulting in atherosclerosis. Understanding other mechanisms linking SDB and lipid dysregulation are of great importance as well, particularly the role of sympathetic nervous system activation as most research thus far has focused upon intermittent hypoxia pathophysiology.

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