ABSTRACT
A straightforward and practical approach is provided for the routine assessment of LDL susceptibility to peroxidation in a clinical laboratory.

INTRODUCTION

A number of recent studies have shown that very low density lipoproteins (VLDLs) are the most prone to oxidation during atherosclerosis, and also the most susceptible to formation of plaques – the formation of which is associated with the development of cardiovascular disease (CVD) and mortality. VLDLs are also the most lipoprotein-lipase-resistant (LPL) and the most prone to formation of plaques. The LPL lipoprotein-lipase-responsive (LPLPR) pathway is implicated in the formation of atherosclerosis.

Most of the LPLPR pathway is thought to be involved in the oxidation of VLDLs, but a number of different pathways are implicated in the formation of atherosclerosis. In this review, we will explore Atherosclerosis is a prevalent condition that may result in death or disability caused by myocardial infarction or strokes. Although the clinical manifestations of the disease have been established, the underlying mechanism of atherogenesis is still unclear. Recent research suggests that the process of oxidative modification of LDL (LDL-Ox) may be involved, but there are limited studies that have investigated the biological effects of this compound in vivo. In view of the possible clinical importance of this type of oxidative modification of LDL, several studies have been conducted to measure their in vitro susceptibility to accelerated oxydation, and this measurement is believed to be associated with the level of vulnerability of low-density cholesterol (LDL) molecules to external radiance within the arterial wall. Various techniques, including sequential or density-gradually gradient ultracentrifugation, chromatography, electrophoresis, and selective precipitation are all possible for the isolation of plasmatic LDLs. The chain reaction of free radicals with polyunsaturated fatty acids results in complex reactions (like rearrangements of double bonds in conjugated dienes, hydroperoxidation of lipids, breakdown of lower molecular weight fragments into lower ones, chemical changes in the apo B protein), and ultimately thiobarbituric acid (Several researches have been conducted to demonstrate the contribution of Fe3+, Fe2+ and Cu2 + to LDL oxidation; in other words biological systems produce hydrogen peroxide with reduced oxygen ions from which the reduction is initiated. These two reactions generate a hydroxyl radical, the most active form of reactive oxygen species, with minimal half-life and maximum reactivity. This reaction, which is kinetically slow, can be further enhanced by catalytic amounts of iron or copper salts. The authors present a straightforward technique for assessing the oxidative susceptibility of LDLs in the presence of Cu2+ and H2O2 in vivo by means of TBARS.

CONCLUSION

A straightforward method for measuring LDL oxidation susceptibility in vitro has been developed, which has also been tested on patients with type 2 diabetes and healthy volunteers. This approach could allow for comparison of results from more general clinical laboratories, allowing us to move towards standardization of a procedure of potential clinical significance.