Atherosclerosis is the predominant feature of cardiovascular diseases (CVD) and currently accounts for most of the morbidity and mortality worldwide. Retinoids (naturally and synthetic vitamin A analogs) have been found to ameliorate atherosclerosis through inhibition of smooth muscle cell proliferation, migration, enhanced apoptosis, prevention of foam cell formation and eventually inhibition of atherosclerosis development. Elimination of retinoids takes place through action of CYP26 members, CYP26A1, B1, and C1, whereof CYP26B1 has been the focus of this thesis.

The present thesis shows that CYP26B1 is induced by retinoic acid in vascular cells and in the vessel wall. The retinoic acid levels in the cell may be altered via functional genetic variants of the CYP26B1 gene and via blockage of CYP26B1, which implies that CYP26B1 may be a potential future therapeutic target to treat vascular disorders.