

Oral Contraception and Cardiovascular Diseases, Biologic effects

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Cardiovascular diseases, i.e. venous thrombosis (DVT), myocardial infarction (AMI) and stroke, are multicausal diseases (genetics and environment). Studies have demonstrated that when risk factors act simultaneously, the joint effect may exceed the separate effects, and points to the presence of subpopulations, where a risk factor, e.g. oral contraceptives, has a much higher effect than on its own. Oral contraceptives increase the risk of DVT about 2-4 fold. Oral contraceptives also increase the risk of arterial diseases 3-5 fold. Numerous studies have tried to give a biological explanation of the attributable risk fraction of oral contraceptives and thrombosis. Indeed oral contraceptives have effects on the haemostatic balance, with an overall net-effect towards a procoagulant state, which in thrombosis prone individuals can cause increased susceptibility and development of thrombosis. This was suggested and reported in women with familiar antithrombin deficiency more than a decade ago (Jespersen 1987), and subsequently reported in cases with other defects of the natural anticoagulant system. This model has successfully been applied on the venous site and operational from the clinical point of view. The mechanisms behind the effect of oral contraceptives are not clear. Changes in synthesis (liver and endothelium) rather than clearance have been shown (Petersen 1999). The multicausality in arterial disease is even more complex. AMI, stroke, and oral contraceptives are in particular associated when smoking and hypertension are present. Smoking, age, and oral contraceptives have been shown to have a negative influence on the antithrombotic system in current users. Types of progestin are also of importance.