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Differential effects of the type of oral contraceptive and menstrual phase on endothelial function in healthy young women

A. K. Thompson, A. Przemska, D. Vasilopoulou, A. L. Turner, K. J. Newens and C. M. Williams
Department of Food Biosciences, University of Reading, Reading RG6 6AP, UK

Changes in endothelial function have been shown to correlate with risk of CVD[1]. Studies investigating endothelial function in premenopausal women must be carefully designed to account for cyclic changes in estrogen, a potent vasoactive hormone[2]. A small number of studies have used flow mediated dilatation (FMD) to investigate the effect of menstrual cycle phase on endothelial function in premenopausal women; both free-cycling[3], and those taking two different formulations of oral contraceptives (OC)[4,5]. These studies confirmed the oestrogen-correlated variation in FMD response in free-cycling women, but have also indicated that the response in women taking OC depends on the type of progestin. The current study aimed to compare three types of OC on endothelial function using a range of endothelial function measurements.

Twenty-nine healthy young women who had been taking OC for at least 3 months were recruited. All were non-smokers, with normal BMI and blood pressure. The women were taking OC containing 150 μg levonorgestrel (Microgynon, *n* 10), 150 μg desogestrel (Marvelon, *n* 10) or 3 mg drospirenone (Yasmin, *n* 9). All OC regimes involved no pills during days 1–7 (corresponding to the menstrual phase) then active pills during days 8–28 (progestin plus 30 μg ethinylestradiol). Subjects had four visits, one during days 5–7 (pill-free phase) and one during days 26–28 (active-pill phase) for two consecutive menstrual cycles.

FMD response was significantly higher during the active-pill than pill-free phases in women receiving desogestrel and drospirenone (both $P<0.001$), but not in those receiving levonorgestrel. This was also seen using endothelium-dependent vasodilation as measured by laser Doppler iontophoresis (LDI-ACh) (both $P<0.02$). No significant differences between phases were observed in any group using other techniques (pulse wave analysis, pulse wave velocity, digital volume pulse or endothelial-independent vasodilation measured using LDI). Women receiving drospirenone had larger changes in FMD between the pill phases than women on desogestrel ($P<0.001$), and women taking levonorgestrel had lower LDI-ACh values during their active-pill phase than women on the other OC ($P<0.02$).

These results confirm the previous report of a negative effect of levonorgestrel on endothelial function, indicating an antagonism of the effect of estrogen. Neither desogestrel nor drospirenone appear to have this effect. FMD and LDI measures also confirmed these differences between pill phases, but other techniques are either lacking in adequate sensitivity or else are measuring aspects of vascular reactivity that do not change during between pill phases. This information may help researchers understand the variation in different endothelial function measurements that occur in premenopausal women taking different OC. This may begin to provide researchers with more confidence regarding the inclusion of premenopausal women in their cohorts, and address the lack of research into vascular function in this subject group.

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