The endometrium is a highly hormone dependent structure. Estradiol is the main mitogen on the endometrial glands, stroma and vessels. Progesterone inhibits the proliferative effects of estradiol and induces differentiation in preparation to the implantation. It is well known from more than 50 years that anovulation or dysovulation are associated with an increased risk in endometrial cancer. At that time it was shown that norethisterone was able to prevent the occurrence of hyperplasia and endometrial cancer. These early observations were further confirmed by studies on the endometrium of women using oral contraception, the bioactive IUD and hormone therapy for menopause (HRT). The highest the progestin administered the lowest the rate of endometrial cancer. The prevention of endometrial cancer was also highlighted recently by the decrease observed in obese women with a combined continuous HRT. Some new data suggest that the progesterone receptor (PR) isoforms/progesterone receptivity could be abnormal in women with PCOS and endometriosis (1). The role of insulin in the resistance in women with PCOS could be an explanation. The importance of the androgen receptor in the endometrium has been recently studied although insufficiently (2); it could suggests that the androgenic component of the progestin could play a role in the protective effect of some progestins. At last, some studies have highlighted the place of synthetic progestins in the treatment and potential prevention in atypical hyperplasia/well differentiated endometrial cancer(2). The place of progestins in the prevention of endometrial cancer including women with high risk such as Lynch syndrome has to be better defined and reported especially in perimenopausal women at the age of menstrual cycle disorders.

1) Li X et al., Endometrial progesterone resistance and PCOS. J Biomed Sci. 2014 Jan 9;21(1):2