The role of growth hormone (GH) in female reproduction has become a topic of increasing interest over the last decade. The addition of GH has been proposed to improve follicle development in poor responders of assisted reproductive technology. Nevertheless, the role of GH in the human endometrium is still largely unknown. We had the first mondial case of a hypophysectomised patient which experienced multiple IVF/embryo transfer failures with inadequate endometrial development. The use of GH replacement therapy followed by conventional controlled ovarian hyperstimulation, enabled good endometrial development and better ovarian response to gonadotrophines, leading to a successful pregnancy.

Women with panhypopituitarism usually present a decreased ovarian response to exogenous gonadotrophines, which results in an augmentation of the dose and duration of gonadotrophines use for ovarian stimulation. Receptors for both GH and IGF-I have been identified on human granulosa cells and GH directly or maybe indirectly through the IGF-1 augments the ovarian response to gonadotrophine stimulation. Indeed, to maximize pregnancy rates, studies suggest a minimal endometrial thickness of 7.0 mm and preferably >9.0 mm. Because it is not possible to study the implantation process in women in vivo due to ethical and technical issues, most of the existing data have been derived from animal studies. The induction of proliferation and decidualisation of endometrium of hypophysectomised mice was found to be partly mediated by an upregulation of oestradiol receptors, in case of GH supplementation. A concurrent elevation of GH may be related to the positive local effect of GH or its mediators on uterine receptivity. Studies have demonstrated a direct relationship between the levels of oestradiol and GH during spontaneous cycling or ovarian stimulation and therefore suggesting that IGF1 may be an important local mediator of estrogen action in the uterus.