Egg-sharing in return for subsidized fertility treatment: a possible solution for therapeutic cloning?

Sir,

We read with great interest the recent advances in therapeutic cloning made by Hwang et al. (2005), in which the efficiency of therapeutic cloning for the derivation of embryonic stem cells was dramatically improved. Previously, only one in 200 attempts were successful, now the success rate stands at around one in twelve. Further rapid progress in this technology is likely, and there is a good chance of therapeutic cloning becoming a routine medical treatment procedure in the future. Nevertheless, a major bottleneck of this new technology is the severe shortage of human donor oocytes.

A possible solution that appears particularly promising is ‘egg-sharing’ in return for subsidized fertility treatment (Blyth, 2002). The cost of IVF treatment is particularly high, which is often a huge financial burden for any childless couple contemplating fertility treatment (Garceau et al., 2002), even for those in the middle-income group. To partially offset the high costs, a fraction of the patient’s eggs may be donated in return for subsidized fertility treatment. Indeed, such egg-sharing schemes to aid women who are unable to produce any oocytes of their own have been going on for some time (Blyth, 2002).

It can be strongly argued that egg-sharing is ethically justifiable, because it reaps great benefits for both parties involved. The financial burden for one childless couple is greatly eased, while the new hope is given to another childless woman who is unable to produce any oocytes of her own. Compared to the rampant commercialization that is inherent in the direct sale and purchase of donor oocytes, egg-sharing appears to be more morally palatable.

Likewise, it would also be more ethically justifiable for egg-sharing to be utilized in providing donor oocytes for therapeutic cloning. On one hand, a childless couple is being aided financially; while on the other hand, a new lease of life may be given to a terminally sick patient. No doubt, it may be argued that by giving a fraction of her oocytes away, the fertility treatment of the donor may be compromised to some extent. However, it is very often the case that the use of gonadotrophins in fertility treatment leads to a production of excess oocytes and hence supernumerary embryos, which are frozen, stored for several years and then eventually destroyed (Forster, 1998). Indeed, recent evidence would suggest that egg-sharing does not significantly compromise the success of fertility treatment (Thum et al., 2003). Moreover, there is a tendency towards replacement of a single embryo to reduce the chance of multiple pregnancy (Martikainen et al., 2001). Hence the issue of excess embryos will become more of a problem later. Perhaps it would make more ethical sense to utilize excess oocytes in therapeutic cloning.

However, there will be a need for further and more stringent screening for possible infective agents, and the need for embryology laboratories involved in the oocyte collection to have ‘good manufacturing practice’ certification, especially if therapy is contemplated in the long run.

References


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GnRH agonist (buserelin) or HCG for ovulation induction in GnRH antagonist IVF/ICSI cycles: a prospective randomized study

Sir,

We read with interest the study by Humaidan et al. (2005) involving the use of GnRH agonist to trigger the final stages of oocyte maturation and wish to commend the authors for conducting this important trial. Previous studies have suggested that the use of GnRH agonist to trigger oocyte maturation may be effective in preventing the development of ovarian hyperstimulation syndrome (OHSS) (Lewit et al., 1996; Itskovitz-Eldor, 2000). Although the primary objective was not to evaluate its effect on the prevention of OHSS, this study is still important because it validates the effectiveness of using GnRH agonist to achieve oocyte maturation after co-treatment with GnRH antagonist during ovarian stimulation for IVF. Furthermore, results of this study as well other previous studies (Fauser et al., 2002; Nevo et al., 2003) have shown without question that the use of GnRH agonist to trigger oocyte maturation alters the luteal phase steroidogenic profile which may, indeed, adversely affect implantation if the appropriate supplementation is not utilized. If this strategy is to be adopted routinely for the

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