J. Reprod. Fert. (1987) 80, 531–535

Printed in Great Britain

Fertilization *in vitro* of rat oocytes undergoing maturation in response to a GnRH analogue

N. Dekel and R. Shalgi*

*Department of Hormone Research, The Weizmann Institute of Science, Rehovot 76100, and *Department of Embryology and Teratology, Tel Aviv University School of Medicine, Tel Aviv, Israel*

Summary. Oocytes were exposed to GnRHa to induce their maturation both *in vivo*, by administration of the hormone to hypophysectomized rats, and *in vitro*, in cultures of intact ovarian follicles. Mature oocytes obtained under both these conditions were then exposed *in vitro* to a sperm suspension for fertilization. Fertilization of control groups of oocytes, isolated from intact or hypophysectomized PMSG-primed hCG-induced ovulators, was 88·3 ± 3·3% (n = 331) and 90·0 ± 2·8% (n = 427), respectively, as compared to 82·8 ± 3·2% (n = 413) for oocytes isolated from hypophysectomized PMSG-primed GnRHa-induced ovulators. Fertilization rate in oocytes treated by GnRHa *in vitro* was 78·5 ± 3·1% (n = 247) as compared to 79·3 ± 4·1% (n = 261) in LH-treated oocytes. These results demonstrate that fertilizability of oocytes undergoing maturation in response to GnRHa is similar to that of oocytes induced to mature by LH. No differences could be detected in the proportions of abnormal oocytes (polyspermic, fragmented and dead) and the zygotes obtained after fertilization of GnRHa- or LH-treated oocytes showed similar ability to cleave.

Introduction

Until fairly recently it was generally accepted that the pituitary, which is stimulated by gonadotrophin-releasing hormone (GnRH) to synthesize and secrete gonadotrophins, is the only target for this hormone. However, later experiments have shown that GnRH and its agonist analogues can also elicit ovarian responses *in vitro*, in isolated ovarian cells (Clayton *et al.*, 1979; Hsueh & Erickson, 1979; Labrie *et al.*, 1979; Behrman *et al.*, 1980; Clark *et al.*, 1980; Hsueh & Jones, 1981; Knecht *et al.*, 1983) and *in vivo* in hypophysectomized rats (Ying & Guillemin, 1979; Hsueh *et al.*, 1980; Corbin & Bex, 1981; Ekholm *et al.*, 1981; Dekel *et al.*, 1983, 1985; Erickson *et al.*, 1983; Naor *et al.*, 1983). These findings, together with the demonstration of ovarian receptors for GnRH (Clayton *et al.*, 1979; Jones *et al.*, 1980; Clayton & Catt, 1981; Seguin *et al.*, 1982), suggested that GnRH can interact with the ovary in a direct manner.

It is known that the follicular oocyte in mammals is arrested at the diplotene stage of prophase of the first meiotic division (Austin, 1961) and that the physiological stimulus for resumption of meiosis is provided by the preovulatory surge of luteinizing hormone (LH) (Ayalon *et al.*, 1972). The observation that GnRH could mimic LH action, inducing meiosis resumption, was initially reported by Hillensjö & LeMaire (1980), who found that exposure of isolated ovarian follicles to GnRH or its agonist analogues *in vitro* results in maturation of the oocytes. The direct stimulatory action of GnRH on the ovary has also been demonstrated *in vivo*. Both oocyte maturation and ovulation were induced in hypophysectomized rats after administration of GnRH agonists (Hsueh & Erickson, 1979; Corbin & Bex, 1981; Ekholm *et al.*, 1981; Dekel *et al.*, 1983, 1985; Erickson *et al.*, 1983; Naor *et al.*, 1983).

The studies discussed above demonstrated that GnRH, like LH, can stimulate the oocyte to