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THE STUDY OF OVULATION

— AND EARLY PREGNANCY

Edited by Y. Boutaleb and A. Gzouli

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List of principal contributors

T. Aono
Dept. of Obstetrics and Gynecology
University of Tokushima
School of Medicine
Kuramoto-cho 3-18
Tokushima 770
Japan

K. Azuma
Dept. of Obstetrics and Gynecology
University of Tokushima
School of Medicine
Kuramoto-cho 3-18
Tokushima 770
Japan

Y. Boutaleb
11 Angle rue Chateaubrian & rue Galilee
Casablanc
Morocco

J.H. Check
Dept. of Obstetrics and Gynecology
University of Medicine and Dentistry of New Jersey
Three Cooper Plaza
Second Floor
Suite 211
Camden
New Jersey 08103
USA

S. Daya
Dept. of Obstetrics and Gynecology
McMaster University
1200 Main Street West
Hamilton
Ontario
Canada L8N 3Z5

M. Dhont
Dept. of Gynecology
De Pintelaan 185
B-9000 Ghent
Belgium

P. Franchimont
Dept. of Endocrinology
Radioimmunoassay Laboratory
CHU-B 35
University of Liege
4000 Liege
Belgium

M. Garofalo
First Clinic of Obstetrics and Gynecology
University of Rome 'La Sapienza'
155, Viale del Policlinico
Policlinico Umberto 1
00161 Rome
Italy

A. Gzouli
Av-Cote D'Emeraude
Rue V
Anfa
Casablanc
Morocco

M. Irahara
Dept. of Obstetrics and Gynecology
University of Tokushima
School of Medicine
Kuramoto-cho 3-18
Tokushima 770
Japan

J. Iwamasa
Dept. of Obstetrics and Gynecology
Kumamoto University Medical School
Kumamoto
Japan

S. Kuwabara
Dept. of Obstetrics and Gynecology
Kanazawa Medical University
Uchinade
Kahoku-Gun
Ishikawa-Ken 920-02
Japan

L. Loiudice
1st Obstetrics & Gynecology Institute
University of Bari
Bari
Italy

B. Lunenfeld
Institute of Endocrinology
Sheba Medical Center
Tel-Hashomer 52621
Israel

L. Marianowski
2nd Dept. of Obstetrics and Gynecology
University School of Medicine
Bielanski Hospital
Warsaw
Poland

A.R. Martinez
Dept. of Obstetrics and Gynecology
Free University Hospital
PO Box 7057
1007 MB Amsterdam
The Netherlands

G.B. Melis
Istutio de Ginecologica & Ostetrica
Universita degli Studi di Pisa
Via Roma 67
56100 Pisa
Italy

K. Nowroozi
1015 Chestnut Street
Suite 1020
Philadelphia
PA 19107
USA

S. Pellegrini
Dept. of Obstetrics and Gynecology
University of Florence
Viale Morgagni
85-50134 Firenze
Italy

J. Pulcrano
Bioself Distribution, S.A.
7, ave. de Thonex
CH-1226 Thonex
Geneva
Switzerland

M. Ribič-Pucelj
Dept. of Obstetrics and Gynecology
University of Ljubljana
Ljubljana
Yugoslavia

R.S. Samil
Dept. of Obstetrics and Gynecology
Faculty of Medicine
University of Indonesia
Dr. Cipto Mangunkusumo Hospital
Jakarta
Indonesia

E.-M. Schindler
Dept. of Gynecology
University of Essen
Hufelandstraße 55
D-4300 Essen
Germany

H.-R. Tinneberg
Dept. of Obstetrics and Gynecology
University of Tubingen
Schleichstraße 4
7400 Tubingen 1
Germany

H.M. Vemer
Scientific Development Group
Organon Int. B.V.
Pharma Division
Oss
The Netherlands

G. Vlassis
32 Ethnikis Aminis St.
54621 Thessaloniki
Greece

G. Wilke
Dept. of Obstetrics and Gynecology
University of Göttingen
D-3400 Göttingen
Robert-Koch Straße 40
Germany

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Foreword

Whilst preparing for the XIIIth World Congress of Fertility and Sterility, I could not help reflecting on the possibility that perhaps the Palais de Congrès might not be completed in time – and that for the first time in the history of the IFFS, the Congress might have to be held in tents! However, I need not have worried; after a very rapid construction schedule, this splendid new building was finished shortly before the Congress opened – and provided an ideal setting for the distinguished gathering of scientists from all over the world that met in Marrakesh for the IFFS Congress.

I should like to thank all those who took part in the XIIIth World Congress; their presentations made an important contribution to our growing understanding of all aspects of reproductive medicine, an understanding which is helping to develop new approaches to therapy around the world.

Finally, I should like to pay a special tribute to Dr Jean Cohen and Professor Robert Harrison, from the IFFS Committee, who together went to so much effort to ensure the success of the scientific program. I am indebted to them for their help – and I trust that these published Proceedings will provide a useful record of this very important scientific occasion.

Y. Boutaleb
President of the Congress

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SECTION 1

Mechanisms and timing of ovulation

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1

Some basic mechanisms of ovulation

P. Franchimont, M.-T. Hazee-Hagelstein, C. Charlet-Renard, J.-M. Jaspar and G. Hennen

Ovulation consists of the rupture of the apex follicle wall allowing extrusion of a mature oocyte. This event appears a few hours after the luteinizing hormone (LH) and follicle stimulating hormone (FSH) midcycle peak.

During the time interval between LH and FSH midcycle burst and the rupture of the follicle with the release of the oocyte, several processes develop such as:

(1) Activation of proteolytic enzymes within and in the vicinity of the dominant follicle;
(2) Changes in cumulus and corona radiata cellular density and interconnection;
(3) The resumption of meiosis; and
(4) The beginning of granulosa cell luteinization.

The mechanisms known today, to explain the rupture of the follicle and the expulsion of the oocytes, are:

(1) The LH-induced activation of proteolytic enzymes concomitant with a reduction of connective tissue component synthesis, both phenomena leading to weakened follicular wall;
(2) An increase in osmotic pressure in the follicle causing water drainage into the follicle, and a subsequent augmentation of hydrostatic pressure. The latter leads to the rupture of weakened connective tissue wall.

This chapter will consider the known metabolic mechanisms of fragilization of follicular wall and its rupture. Then, phenomena associated with the ovulatory period (i.e. from LH and FSH midcycle peak to extrusion of oocyte) will be discussed.

CONNECTIVE ORGANIZATION OF THE FOLLICLE

Ovarian follicles are surrounded by a basement membrane (BM) between the granulosa and theca interna layers. Basement membrane of rat follicles

contains type IV collagen (collagen IV), heparin sulfate proteoglycans and laminin. Type III (interstitial) collagen (collagen III) has been localized in the mesenchyme of the theca interna layers of rat ovarian follicle[1] where it constitutes a fibrous capsule surrounding the follicle[2]. Furthermore, the connective structures help to maintain the intercellular cohesion in the follicle. Moreover proteoglycans together with hyaluronic acid (HA) are probably responsible for the viscosity of the follicular fluid (FF) and may, to some degree, contribute to its osmotic pressure.

Origin of constituents of connective tissue within and surrounding follicles

Granulosa cells produce intrafollicular and BM proteoglycan collagen IV, whereas collagen III and proteoglycans of the fibrous capsule surrounding theca externa layer are produced by fibroblasts of mesenchymal areas of the ovary, mainly of theca externa fibroblasts.

Regulation of their production

Synthesis of proteoglycans by granulosa cells is under the influence of FSH, while LH exerts no effect or even reduces proteoglycan synthesis[3–6]. The stimulating effect of FSH on proteoglycan biosynthesis is increased by insulin-like growth factor I (IGF 1[7]).

Transforming growth factor-β (TGF-β) is produced by theca interna cells when estrogen levels are low[8]. This growth factor stimulates BM collagen IV in granulosa cells, and external fibrous capsule collagen III in theca externa fibroblasts.

Normally, during the early follicular phase, when FSH levels are high and estrogen levels are still low or moderate, the production of connective matrix components in follicles in BM, and in fibrous external capsule is elevated.

During the late follicular phase, estrogen secretion is high, FSH production very low and LH slowly increases[9]. This hormonal status is associated with a reduction in collagen and proteoglycan synthesis.

ENZYME ACTIVATION

During the last steps of follicular maturation, in several species, a reduction of connective tissue components is observed in the follicle and in BM. This decrease is most probably linked with a reduction in collagen and proteoglycan synthesis, as well as with increased activity of proteolytic enzymes which degrade the connective macromolecules. Thus, FSH and LH trigger production and activation of proteolytic enzymes capable of degrading collagen and proteoglycans: plasminogen activator (PA) proteoglycanase, collagenase, and β-glucoronidase[10–12].

The reduction of connective component is illustrated in the rat preovulatory follicle, where there is a reduction in collagen content and proteoglycan

production[13-15]. We have also shown that a reduction of human follicular fluid proteoglycan levels is related to follicular size and inversely correlated to progesterone content[16].

We suggest that in women, slow LH increase during the late follicular phase and the midcycle peaks of FSH and LH are associated with the production and activation of proteases which degrade connective tissue structures.

Simultaneously, enzyme inhibitors of metallo and serine proteases which were secreted during the early and midfollicular phase decline, allowing activation of latent enzymes. Milwidsky *et al.* (1989)[17] demonstrated that the maturation process is accompanied by a reduction in the level of protease inhibitory activity in FF and an increase in the conversion of latent to active proteoglycans. It is likely that this conversion is mediated by endogenous plasmin- or trypsin-like protease, the inhibition of which is declining during folliculogenesis.

The role of the enzyme inhibitors clearly appears in the recent study of Pellicer *et al.* (1988)[18]. These authors demonstrated that the early injection of serine protease inhibitors into ovarian bursas of rat impaired the entire process of follicular development as well as ovulation.

The interval between LH peak or luteinizing hormone–human chorionic gonadotropin (LH–hCG) injection and the ovulation is short and associated with morphological changes at the apex of the follicular wall. Thus, in women, the LH midcycle peak is followed by the rupture of the follicle 8–10 h later[19].

Curry *et al.* (1985)[11] have demonstrated that human chorionic gonadotropin (hCG)-induced collagenolytic activity in immature, pseudopregnant (pregnant mare serum gonadotropin-(PMSG) induced) rats, is at its maximum after 8 h. These results correspond to those obtained by Morales *et al.* (1983)[14] who reported that collagen content of rat follicles decreased 6–10 h after the liberation of LH. Furthermore, observation of rabbit follicles 4 h after hCG injection revealed a mild dissociation of granulosa cells as well as moderate dislocation of the internal theca apical cells and of the fibroblasts of the external theca. Signs of BM rupture are observed 9 h later and a complete rupture is obtained 9.30 h after hCG stimulation.

Sources of enzymes

Latent enzymes are produced by granulosa cells and by fibroblasts of mesenchymal tissue of the ovary. Enzyme inhibitors are produced by the same cells in the follicle.

Regulation of enzyme production

As said previously, LH and to some extent FSH release proteolytic enzymes, and mainly FSH, but also LH, release and activate PA produced by Graafian follicles which in turn activate proteolytic enzymes: elastase, proteoglycanase, Cathepsin G, collagenase, etc. Moreover, the estrogens may play a direct role in this process as demonstrated by the ability of 17-β-estradiol (E_2) to

stimulate the production of PA[20,21]. Estrogen-induced enzymatic degradation of proteoglycans may explain the inverse correlation between proteoglycans and E_2 or $E_2 + E_1$ (estrone) in follicular fluid during human menopausal gonadotropin (hMG)-induced follicular maturation[22].

The action of LH appears to be mediated by prostaglandins prostaglandin E_2 (PGE_2) and prostaglandin F2α ($PGF_{2\alpha}$) which release enzymes from all cell types: granulosa cells, theca cells, externa theca fibroblasts.

Relaxin is also capable of stimulating the production of several enzymes implicated in connective tissue degradation[10]. In pig ovary, relaxin is present within the cells of theca interna[23,24]. Its synthesis is under the influence of LH and increases as the preovulatory follicle develops[23]. We can propose a hypothesis explaining why PA is released after a latent period following LH stimulation. Luteinizing hormone would act on theca interna cells by stimulating prostaglandins and relaxin, responsible for the secretion of proteolytic enzymes by granulosa cells.

OVULATION: MECHANICAL CONSEQUENCE OF CONNECTIVE MACROMOLECULE BREAKDOWN

The breakdown of proteoglycans and glycosaminoglycans and to a lesser extent collagens and hyaluronic acid, due to enzymatic degradation, results in the addition of molecules to follicular fluid and leads to increased intrafollicular osmotic pressure and follicular swelling which is related to water drainage into the follicle. Upon ovulation, this may contribute further to the rupture of the follicular wall, already weakened by proteolytic enzymes.

If enzymatic activation is reduced or absent, an anovulation can be observed. Such biochemical lesion could explain the syndrome of unruptured follicle.

EVENTS ASSOCIATED TO OVULATION PERIOD

In the interval between FSH and LH midcycle peak and ovulation several events appear.

Morphological changes of corona radiata and of cumulus oophorus

After gonadotropin midcycle peaks, cells surrounding oocytes are limited to a single layer or a few layers, whereas cumulus cells are dissociated and extended[25]. The role of activated enzymes in this process seems to be likely.

Meiosis resumption

In primates, the first steps of meiosis of the oocyte are carried out during late fetal life and the first months of life. The process arrests at the prophase

of the first mitosis of meiosis. A membrane isolates the nucleus material which constitutes the germinal vesicle. The oocytes remain quiescent in a dominant follicle until the LH midcycle peak. After this signal, there is resumption of meiosis with germinal vesicle breakdown, then achievement of the first mitosis with first polar body emission and arrest of meiosis at metaphase stage of the second mitosis.

Luteinizing hormone stimulates oocyte maturation presumably by disrupting the functional communications maintained by basal levels of cyclic adenosine monophosphate (cAMP) between the oocytes and the cumulus cells[26,27]. Furthermore, a local action on oocyte maturation is dependent on IGF-1, TGF-β and TGF-α.

Insulin-like growth factor-1 has been implicated in the complex control of follicular growth and ovulation[28]. Production of IGF-1 in granulosa cells has been shown to be stimulated by FSH, LH and E_2. Its concentration is higher in dominant follicle and increases further after the LH surge. Furthermore, it clearly stimulates the resumption of meiosis *in vivo* and *in vitro*[28 30]. Thus we may speculate that IGF-1, locally produced, is of main importance in the maturation of oocyte by a short loop intraovarian action: FSH and LH midcycle peak → IGF-1 → meiosis.

Transforming growth factor-β also stimulates meiotic maturation of the rat oocyte but the mechanism by which it is secreted remains unclear[29].

Finally follistatin, also called FSH suppressing protein, which has an inhibin-like activity (but is completely different in 2 subunits from inhibin molecule) accelerates the rate of meiotic maturation of rat oocyte in culture[31]. Regulation of FSH suppressing protein secretion is still an enigma.

REFERENCES

1. Christiane, Y., Demoulin, A., Gillain, D., Leroy, F., Lambotte, R., Lapiere, Ch-M., Nusgens, B. and Foidart, J.M. (1988). Laminin and type III procollagen peptide in human preovulatory follicular fluid. *Fertil. Steril.*, **50**, 48
2. Espey, L.L. (1967). Ultrastructure of the apex of the rabbit Graafian follicle during the ovulatory process. *Endocrinology*, **81**, 267
3. Mueller, P.L., Schreiber, J.R., Lucky, A.W., Schulman, J.D., Rodabard, D. and Ross, G.T. (1978). Follicle-stimulation hormone stimulates ovarian synthesis of proteoglycans in the estrogen-stimulated hypophysectomized immature female rat. *Endocrinology*, **102**, 824
4. Gebauer, H., Lindner, H.R. and Amsterdam, A. (1978). Synthesis of heparin-like glycosaminoglycans in rat ovarian slices. *Biol. Reprod.*, **18**, 350
5. Ax, R.L. and Ryan, R.J. (1979). FSH stimulation of 3H-glucosamine-incorporation into proteoglycans by porcine cells *in vitro*. *J. Clin. Endocrinol. Metab.*, **49**, 646
6. Schweitzer, M., Jackson, J.C. and Ryan, R.J. (1981). The porcine ovarian follicle. VIII. FSH stimulation of *in vitro* 3H-glycosamine incorporation into mucopolysacharides. *Biol. Reprod.*, **24**, 332
7. Adashi, E.Y., Resnick, C.E., Svoboda, M.E., Van Wyck, J.J., Hascall, V.C. and Yanagishita, M. (1986). Independent and synergistic actions of somatomedin-C in the stimulation of proteoglycan biosynthesis by cultured rat granulosa cells. *Endocrinology*, **118**, 456
8. Skinner, M.K., Keski-Oja, J., Osteen, K.G. and Moses, H.L. (1987). Ovarian thecal cell produce transforming growth factor-beta which can regulate granulosa cell growth. *Endocrinology*, **121**, 786

9. Bomsel-Helmreich, O., Gougeon, A., Thiebault, A., Saltarelli, D., Milgrom, E., Frydman, R. and Papiernik, E. (1979). Healthy and atretic human follicles in the preovulatory phase: differences in evolution of follicular morphology and steroid content of follicular fluid. *J. Clin. Endocrinol. Metab.*, **48**, 686
10. Too, C.K., Bryant-Greenwood, G.D. and Greenwood, F.C. (1984). Relaxin increases the release of plasminogen activator, collagenase, and proteoglycanase from rat granulosa cells *in vitro*. *Endocrinology*, **115**, 1043
11. Curry, T.H., Dean, D.D., Woessner, J.F. and Lemaire, W.J. (1985). The extraction of a tissue collagenase associated with ovulation in the rat. *Biol. Reprod.*, **33**, 981
12. Canipari, R. and Strickland, S. (1986). Studies on the hormonal regulation of plasminogen activator production in the rat ovary. *Endocrinology*, **118**, 1652
13. Yanagishita, M. and Hascall, V.C. (1979). Biosynthesis of proteoglycans by rat granulosa cells cultured *in vitro*. *J. Biol. Chem.*, **254**, 12355
14. Morales, T.I., Woessner, J.F., Marsh, J.M. and Lemaire, W.J. (1983). Collagen, collagenase and collagenolytic activity in rat Graafian follicle during follicular growth and ovulation. *Biochem. Biophys. Acta*, **756**, 119
15. Reich, R., Tsafriri, A. and Mechanic, G.L. (1983). Studies on the involvement of collagenase and of plasmin in ovulation. *Biol. Reprod.*, **30**, 93
16. Franchimont, P., Demoulin, A. and Valcke, J.C. (1988). Endocrine, paracrine and autocrine control of follicular development. *Horm. Metabol. Res.*, **20**, 193
17. Milwidsky, A., Kaneti, H., Finci, Z., Laufer, N., Tsafriri, A. and Mayer, M. (1989). Human follicular fluid protease and antiprotease activities: a suggested correlation with ability of oocytes to undergo *in vitro* fertilization. *Fertil. Steril.*, **52**, 274
18. Pellicer, A., Lightman, A., Ariza, A., De Cherney, A.H., Naftolin, F. and Littlefield, B.A. (1988). Follicular development is impaired by inhibitors of serine proteases in the rat. *Am. J. Obstet. Gynecol.*, **158**, 670
19. Okamura, H., Takenata, A., Yafima, Y. and Nishimura, T. (1980). Ovulatory changes in the wall of the human Graafian follicle. *J. Reprod. Fertil.*, **58**, 153
20. Reich, R., Miskin, R. and Tsafriri, A. (1986). Intrafollicular distribution of plasminogen activators and their hormonal regulation *in vitro*. *Endocrinology*, **119**, 1588
21. Wang, C., Leung, A. (1987). Estrogens, progestogens, and androgens enhance the follicle-stimulating hormone-stimulated plasminogen activator production by cultured rat granulosa cells. *Endocrinology*, **120**, 2131
22. Franchimont, P., Hazee-Hagelstein, M.T., Hazout, A., Gysen, Ph., Pirson, F., Schatz, B. and Demerlé, F. (1990). Correlation between follicular fluid content and the results of *in vitro* fertilization and embryo transfer. III. Proteoglycans. *Biol. Reprod.*, in press
23. Evans, G., Wathes, D.C., King, E.L., Armstrong, D.T. and Porter, D.G. (1983). Changes in relaxin production by the theca during the preovulatory period of the pig. *J. Reprod. Fertil.*, **69**, 677
24. Bagnell, C.A., Frando, L.B., Bryant-Greenwood, G.D., Downey, B.R., Tsang, B.K. and Ainsworth, L. (1986). Localization of relaxin in the pig follicle during preovulatory development. *Biol. Reprod.*, **34**, suppl. 1, 116
25. Veeck, L.L. (1988). Oocyte assessment and biological performance. *Ann. NY Acad. Sci., USA*, **541**, 259
26. Gilula, N.B., Epstein, M.L. and Beers, W.H. (1978). Cell to cell communication and ovulation. *J. Cell. Biol.*, **78**, 58
27. Dekel, N., Lawrence, T.S., Gilula, N.B. and Beers, W.H. (1981). Modulation of cell to cell communication in the cumulus-oocyte complex and the regulation of oocyte maturation by LH. *Dev. Biol.*, **86**, 356
28. Holly, J.M.P. and Wass, J.A.H. (1989). Insulin-like growth factors, autocrine, paracrine or endocrine? New perspectives of the somatomedin hypothesis in the light of recent developments. *J. Endocrinol.*, **122**, 611
29. Feng, P., Catt, K.J. and Knecht, M. (1988). Transforming growth factor-beta stimulates meiotic maturation of the rat oocyte. *Endocrinology*, **122**, 181
30. Hansson, H.A., Billig, H. and Isgaard, J. (1989). Insulin-like growth factor I in the developing and mature rat testis: immunohistochemical aspects. *Biol. Reprod.*, **40**, 1321
31. O., W.S., Robertson, D.M. and de Kretser, D.M. (1989). Inhibin as an oocyte meiotic inhibitor. *Mol. Cell. Endocrinol.*, **62**, 307

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