A RELATION OF COLLAGEN DEGRADATION IN MOUSE UTERUS TO FETECTOMIZED PLACENTA

K. Shimizu, T. Sato and J. Yamada

Department of Anatomy, Tokyo Medical College, 6-1-1 Shinjuku, Shinjuku-ku, Tokyo 160, Japan

The uterus grows adaptively to fetal growth during pregnancy. Following parturition, both the weight and protein content of the uterus show a rapid decrease, which is known as postpartum involution. A conspicuous feature in the postpartum involution is collagen degradation, since this represents one of the fastest known rates in any connective tissues. Because the uterine collagen degradation starts after expelling the placenta from the uterus, a factor secreted by the placenta may inhibit the uterine collagen metabolism from turning into a catabolic state. It has still been obscure what cell in the placenta secretes an inhibiting factor. In the fetectomized placenta, the non-endocrine elements degenerate but the structural integrity and metabolic activity of the endocrine elements remain. To determine what cell secretes an inhibiting factor, we observed a relation of collagenolysis in the uterus to the fetectomized placenta.

The animals used were female mice of the IVCS strain. At 7 weeks of age the right oviduct was ligated. After one week they were mated. Fetectomy was performed on day 16 of pregnancy. Animals were killed on postoperative 3 days (day of parturition). With bilaterally pregnant mice, fetectomy was performed on the left uterine horn on day 18 of pregnancy (last pregnant day). They were killed on postoperative 1 or 4 days (postpartum day 3). The non-pregnant uterine horn (NPH) from the unilaterally pregnant mice and the right postpartum uterine horn from the bilaterally pregnant ones, respectively, was removed. Sections were stained with picrosirius red and then viewed with polarizing light.

The trophoblastic giant (G) cells in the basal zone of the fetectomized placenta on the day of parturition (postoperative 3 days) showed similar in appearance to those of the normal placenta on the last pregnant day. When the contralateral uterine horn had the fetectomized placenta, the endometrial collagen degradation in the NPH did not occur on the day of parturition (postoperative 3 days). On the other hand, when the contralateral uterine horn had not placenta, the endometrial collagen in the NPH degraded on the day of parturition (postoperative 3 days). However, the fetectomized placenta did not affect the postpartum collagen degradation in the endometrium of the contralateral parturient uterine horn. When compared with the normal placenta on the last pregnant day, most of G cells in the basal zone of fetectomized placenta on postpartum day 3 (postoperative 4 days) degenerated.

Since G cells can secrete several hormones, our results indicate that a factor secreted by G cells may inhibit the uterine collagen metabolism from turning into a catabolic state and that elimination of a factor by expelling the placenta at parturition and by removing artificially the placenta, respectively, initiate the uterine collagen degradation.

INVolvement OF INTERLeUKIN 6 IN Premature RUPTURE OF MEMBRANES

Kazuo Masaki, Masanobu Tanaka, Satoshi Ishizaki, Ikuo Morita*,
Seitu Murota* and Shun Hirakawa

First Department of Obstetrics and Gynecology, Toho University School of Medicine,
6-11-1 Omorinishi, Ohta-ku, Tokyo 143, Japan
*Section of Physiological Chemistry, Graduate School, Tokyo Medical and Dental University,
1-5-45 Yushima, Bunkyo-ku, Tokyo 113, Japan

Chorioamnionitis has been thought to be one of the causes for premature rupture of membranes (PROM). Recently, it is focused on interleukin 6 (IL-6) in inflammation. In this paper we investigate the involvement of IL-6 in PROM. Fetal membranes were obtained from healthy women undergoing full term cesarean section and from normal women after spontaneous vaginal delivery. Chorionic cells and amnion cells, prepared by enzymatic dispersion of isolated chorion and amnion, were cultured. IL-6 in the cultured media were measured...