Murakami, Akiko Kanto, Hisamitsu Takaya, Yasushi Kotani, Hidekatsu Nakai, Isao Tsuji, Ayako Suzuki, Noriomi Matsumura. *Department of Obstetrics & Gynecology, Kindai University*

**Objective**: Retained products of conception (RPOC) is associated with postpartum hemorrhage and intrauterine infection. Although critical obstetric hemorrhages occur in some cases, conservative treatment was possible in recently reported cases. Against this background, we conducted this retrospective study for cases of RPOC treated at our hospital.
**Methods**: Consecutive 33 cases who were diagnosed with RPOC between 2000 and 2018 were retrospectively analyzed. The major axis of the remaining tissue was measured on ultrasound and/or MRI images taken at the time of diagnosis, and compared. After that, S-hCG levels were observed from 22 cases, and their outcome was evaluated.
**Results**: 8 cases were classified as Group A, who required blood transfusion before hemostasis was achieved soon after delivery of the baby. 4 cases belonged to Group B, who experienced massive hemorrhage, and required blood transfusion during the period of conservative treatment. Group C is comprised of the other 21 cases, who experienced no major trouble throughout the period of conservative management. The retained placenta of Group A was bigger than group C, although there was no significant difference between group A and B. During the conservative treatment, the levels of S-hCG gradually decreased with the half-life of approximately five days, disappeared rapidly after hemostatic therapy or removal of the retained placenta. Importantly, no patient needs hysterectomy.
**Conclusion**: RPOC cases who require hysterectomy seem to be very rare, although some patients experience massive hemorrhage.

## 5.
## IN VIVO IMAGING TO VISUALIZE FETO-MATERNAL INTERFACE IN PREGNANCY-ASSOCIATED HYPERTENSION MOUSE

Anna Sato [1], Jun-dai kim [2], Akiyoshi Fukamizu [2], Atsuo Itakura [1]. [1] *Department of Obstetrics and Gynecology, Juntendo University;* [2] *Life science center for survival dynamics, Tsukuba University*

**Object**: Hypertensive disorders of pregnancy (HDP) is thought that placental dysfunction is important in the pathogenesis. The morphological change is important, but it has not been well documented the fetoplacental vasculature. I try to reveal the detail of vascular system to understand HDP, and I decide to use the in vivo imaging (two-photon microscopy) technology and emerging tissue-clearing technologies (the Scale system).
**Methods**: As the model of HDP, we generated a transgenic mouse model that developed pregnancy-associated hypertension (PAH) by the overproduction of Ang II in maternal circulation during late pregnancy. To figure out vascular formation, I used three-dimensional (3D) observation of whole tissues and tissue-clearing technology, the Scale system. I prepare for R26GRR mice that is a ROSA26 knock-in Cre-reporter exhibiting green emission before and red after Cre-mediated recombination. Then I combined R26GRR mice and Tie2-Cre-PAH mice (Tie2 gene is vascular endothelial cell-specific expression.). Then I combine placenta of R26GRR/Tie2-Cre/PAH mice together with the microscopy to form 3D imaging.
**Result**: I established the method for visualizing placental vessels with fluorescence. It revealed that the vascular networks of PAH mice were rough than WT mice by two-photon microscopy.
**Conclusion**: I successfully observed feto-maternal inter face using tissue-clearing agent and in vivo 3D imaging. This technique will contribute that to elucidate the feto-maternal interface with normal pregnancy and HDP including effects of anti-hypertensive drugs.

## 6.
## LNCRNA *1600012P17RIK* IS A MOUSE PLACENTA-SPECIFIC LNCRNA

Junxiao Wang [1], Aya Misawa [1], Shan-shun Luo [2], Toshihiro Takizawa [1]. [1] *Department of Molecular Medicine and Anatomy, Nippon Medical School;* [2] *Department of Geriatrics, the First Hospital of Harbin Medical University*

**Objective**: Long non-coding RNAs (lncRNAs) exert functions in regulating various biological processes. However, there is little information available on the expression and function of mouse placenta-derived lncRNAs. The purpose of this study was to identify mouse placenta-specific lncRNAs.
**Methods**: For in silico lncRNA expression pattern analysis, we analyzed the lncRNA expression profile in E10 and E17 mouse placenta using the FANTOM5 database. For in vivo lncRNA expression analysis, we performed real-time PCR to examine whether selected lncRNAs were associated with the placenta using B6D2F1 mouse placentae at different stages (from E7.5 to E18.5) and adult organs.
**Results**: In silico analysis revealed that approximately 350 lncRNAs were expressed in both E10 and E17 mouse placentae. In the 10 most highly expressed lncRNAs in these placentae, we found 2 mouse placenta-specific lncRNA candidates that were exclusively or predominantly expressed in the placenta compared with adult organs. Among these candidates, we selected *1600012P17Rik* lncRNA that was the highest expression lncRNA in E17 placenta (55%). In vivo real-time PCR analysis showed that *1600012P17Rik* was expressed exclusively in the mouse placenta but not in any other organs used in this study. During placenta development, *1600012P17Rik* was hardly detectable at E7.5 and began to increase thereafter, reaching peak expression at E16.5.
**Conclusion**: LncRNA *1600012P17Rik* was a mouse placenta-specific lncRNA and was expressed in a developmental stage-specific manner.

## 7.
## GLUCOCORTICOID WEAKENS FETAL MEMBRANES VIA IL-1B PRODUCTION BY AMNIOTIC FLUID MACROPHAGE

Hikaru Kiyokawa [1,2], Haruta Mogami [2], Yusuke Ueda [2], Yousuke Kawamura [2], Mai Sato [2], Yoshitsugu Chigusa [2], Eiji Kondoh [2], Masaki Mandai [2]. [1] *Department Obstetrics and Gynecology, Kurashiki Central Hopital;* [2] *Department Gynecology and Obstetrics, Kyoto university*

**Objectives**: Glucocorticoid (GC) use during pregnancy is known to increase the risk of preterm birth and preterm premature rupture of membranes (pPROM). Here, we investigated the mechanism of how GC weaken the fetal membranes (FM).
**Methods**: The thickness of FM between control pregnant women and patients treated with GC was measured. Corticosterone (C) was subcutaneously injected to pregnant mice daily from 12 to 18 dpc. FM were collected and used for immunofluorescence and quantitative RT-PCR. The thickness of FM was measured. Primary human amnion mesenchymal cells (hAMC) were incubated with hydrocortisone (HC) or IL-1B for 24 or 48 hours.
**Results**: The amnion mesenchymal layer was significantly thinner in GC treated pregnant patients and in C-injected mice than in cotrol groups. *COL1A1* mRNA was decreased and *COX2* mRNA and PGE2 synthesis were increased by C. Prolifiration and migration of macrophages (M) were observed around C-injected amnion. In immunofluorescence, IL-1B was localized to these migrated M. In hAMC, HC did not change *MMP* and *COX2* mRNA expressions, but treatment of IL-1B significantly increased *MMP* and *COX2* mRNA levels. Futhermore *COL1A1* mRNA levels was decreased by both HC and IL-1B.
**Conclusion**: GC weakens amnion via collagen degradation by MMP and supression of collagen synthesis. Induction of MMP and prostaglandin synthesis woud be mediated by IL-1B from recruited amniotic fluid M, and both released IL-1B and GC decrease transcription of collagen genes. Collectively, these results indicated that GC plays a pivotal role in the pathogenesis of pPROM.

## 8.
## ANALYSIS OF UTERINE DCS BEFORE IMPLANTATION

Ippei Yasuda [1,2], Mizuki Ueda [2], Taiki Moriya [2], Ryoyo Ikebuchi [2,3], Yutaka Kusumoto [2], Akemi Ushijima [1], Tomoko Shima [1], Akitoshi Nakashima [1], Shigeru Saito [1], Michio Tomura [2]. [1] *Department of Obstetrics and Gynecology, University of Toyama;* [2] *Laboratory of Immunology, Faculty of Pharmacy, Osaka Ohtani University;* [3] *Research Fellow of Japan Society for the Promotion of Science*