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Urinary Excretion of Oestrone Sulphate and Cortisol in Early Pregnant Gilts Treated with Glucocorticoids

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Madej A, Romanowicz K, Einarsson S, Forsberg M, Barcikowski B: Urinary excretion of oestrone sulphate and cortisol in early pregnant gilts treated with glucocorticoids. Acta vet. scand. 1998, 39, 61-70. – The aim of this study was to determine rates of urinary excretion of oestrone sulphate and cortisol in early pregnant gilts that were untreated or treated with either dexamethasone, corn oil or hydrocortisone. Twenty Polish Landrace gilts were used. They were grouped immediately after mating as follows: Experiment I – Group 1 (5 gilts), control animals and Group 2 (5 gilts), injected i.m. with dexamethasone (30 μg/kg) at 12-h intervals from day 13 to day 22 of pregnancy; Experiment II – Group 3 (5 gilts), injected i.m. with corn oil at 12-h intervals from day 13 to day 22 of pregnancy and Group 4 (5 gilts), injected i.m. with hydrocortisone acetate (250 mg) at 12-h intervals from day 11 to day 20 of pregnancy. Gilts were placed in metabolic cages, and 24-h urine aliquots were collected from day 6 to day 32 of pregnancy. On days 34-36 of pregnancy gilts were slaughtered and clinical data were collected. Rates of urinary excretion of oestrone sulphate and cortisol were determined by enhanced chemiluminescence immunoassays. The urinary excretion of oestrone sulphate expressed in nmol/24 h and μmol/mol creatinine were significantly correlated. There was no correlation between cortisol expressed in nmol/24 h and μmol/mol creatinine ($p>0.5$). A first significant increase of urinary oestrone sulphate excretion, expressed in nmol/24 h, on days 13-14 and a second one on days 19-20 of gestation occurred in control untreated and oil-treated gilts. The urinary excretion of oestrone sulphate reached maximum values between days 25 and 32 of gestation. In dexamethasone-treated gilts cortisol excretion significantly decreased on day 16, i.e. 3 days after injections of dexamethasone had commenced. The treatment with hydrocortisone resulted in a significantly increased cortisol excretion after the last injection of hydrocortisone. There were no relations between levels of urinary oestrone sulphate excretion expressed in nmol/24 h and the number of foetuses. When the urinary excretion of oestrone sulphate was expressed in mol/mol creatinine we found a positive relation between concentrations on day 20 of pregnancy and the number of foetuses. In one untreated gilt with a relatively high urinary excretion of cortisol (more than 200 nmol/24 h) a lower number of foetuses was found at autopsy.
In conclusion, both dexamethasone and hydrocortisone treatment seemed to delay the first observed peak in oestrone sulphate in gilts without affecting the embryonic survival and the number of viable foetuses.

stress; foetus; chemiluminescence immunoassay.

Introduction

In the pig establishment of pregnancy begins about 11-12 days after mating (*Geisert et al.* 1990). There is strong evidence that prolongation of the functional life of the corpora lutea in

the pig depends on the synthesis and release of oestrogens from the conceptus. It appears that the synthesis and release of oestrogens from porcine blastocysts is biphasic, with the first increase occurring on day 11 of pregnancy and the second between days 14 and 18 (*Geisert et al.* 1990). The conceptus also secretes catechol oestrogens, prostaglandins (*Geisert et al.* 1990), and interferon α and γ (*Bazer et al.* 1994) which could interact with oestrogens to prevent luteolysis. The mechanism of maternal recognition of pregnancy is very complex, and exact timing of all signals is of importance for embryonic survival. The prolificacy of pigs, regardless of the breed, is primarily attributable to an embryo survival until days 20-22 of pregnancy (*Galvin et al.* 1993). The further embryo mortality until day 30 of pregnancy was found to be negligible (*Ashworth et al.* 1992). Poor conception rate and reduced litter size are also assumed to be a result of the manifestations of stress (*Varley & Stedman* 1994). *Behrens et al.* (1993) reported that elevated cortisol levels in pseudopregnant gilts altered endocrine and uterine functions related to pregnancy. In our previous study (*Tsuma et al.* 1996) we reported cortisol to be increased in the fasted early pregnant sows, which had numerically lower embryonic survival than the control animals. The aim of this work was to monitor the urinary excretion of oestrone sulphate and cortisol in pregnant control gilts and gilts given repeated injections of dexamethasone, corn oil and hydrocortisone acetate.

Materials and methods

Animals

All gilts with body weight ranging from 88 to 119 kg, age (9 to 10 months) and the same genetic background (Polish Landrace) were housed at the Kielanowski Institute of Animal Physiology and Nutrition, Jablonna near Warsaw, Poland. The barn was illuminated for 12 h (lights on at 0700 h, lights off at 1900 h). Room temperature varied between 15 and 17 °C. The gilts were fed 2.5 kg/day of a diet containing 12.5% crude protein and 11.5 MJ ME/kg. Water was available ad libitum. Experiment I was performed in February/March 1993 and experiment II in March/April. Gilts were bred according to the system normally used in the experimental farm and placed in individual metabolism cages on day 5 after mating. The following groups were studied: Experiment I – Group 1 (5 gilts), untreated animals and Group 2 (5 gilts), injected i.m. with dexamethasone (30 μg/kg) at 12-h intervals from day 13 to day 22 of pregnancy; Experiment II – Group 3 (5 gilts), injected i.m. with corn oil at 12-h intervals from day 13 to day 22 of pregnancy and Group 4 (5 gilts), injected i.m. with hydrocortisone acetate (250 mg) at 12-h intervals from day 11 to day 20 of pregnancy.

Urine samples (24-h collection) were collected from day 6 to day 32-33 after mating. Urine was separated from faeces in the collection funnel, filtered through glass wool, and collected in 10-l flasks. After 12 h, collected urine was transferred to a temperature below 8 °C. Then, the total volume of urine (24 h collection) was measured. An aliquot (10 ml) of 24 h collection was pipetted to the tube that contained 125 mg of boric acid and was then frozen at –20 °C until assayed for oestrone sulphate and cortisol.

The general health status of the gilts was recorded all the time from mating until slaughter. On days 34-36 after mating gilts were slaughtered. Clinical findings are presented in our previous paper (*Madej et al.* 1997).

Analytical methods

Urine concentrations of cortisol and oestrone sulphate were quantified by enhanced chemiluminescence immunoassays. In brief, hormone present in the sample and horse-radish peroxi-

dase (HRP) labelled hormone compete for binding sites on a hormone specific antibody. The antibody is presented in the liquid phase and is captured during the assay incubation by a second antibody coated onto the wells. At the end of the incubation, unbound tracer is removed by washing and aspiration of the wells. Signal reagent, containing luminescence substrate and enhancer, is added to the wells to initiate the HRP-catalysed light-emitting reaction. The wells are read in a luminometer 5 min after adding the signal reagent.

Oestrone sulphate: The specificity of the antibody has been reported previously (*Wright et al.* 1978). The antibody was diluted with PBS-0.1%gelatin buffer to an initial dilution of 1:10000. Oestrone-3-hemisuccinate-HRP (MILAB, Helsingborg, Sweden) was diluted in PBS-0.1% BSA-0.05% Tween buffer to an initial dilution of 1:200000. Standards 1.6–200 nmol/L were prepared in PBS-BSA-Tween. Urine samples were diluted 1:10 in distilled water. Second antibody-coated wells, washing media and signal reagent were purchased from Kodak Clinical Diagnostics Ltd (Amersham, England). Duplicate samples of unknown urine and standards (25 µl), oestrone-free porcine urine (25 µl), oestrone-3-hemisuccinate-HRP (100 µl) and antibody (100 µl) were pipetted into the wells. Wells were agitated and incubated for 1 h at 37 °C. After washing (4 times) signal reagent was added and the wells were read in a luminometer (Amerlite analyser, Kodak Clinical Diagnostics Ltd, Amersham, England) after 5 min. Serial dilutions of porcine urine containing high oestrone sulphate concentrations produced displacement curves parallel to the standard curve. The intra-assay coefficient of variation calculated from 32 precision profiles was below 13% for concentrations ranging from 1.6 to 200 nmol/l. Inter-assay coefficients of variation based on 20 different assays were 26.0% (6.8 nmol/l), 11.1% (59.0 nmol/l) and 8.4% (224.0 nmol/l). Urinary oestrone sulphate was expressed in nmol/24 h. Additionally, hormone concentrations were divided by the concentration of urinary creatinine for each sample and expressed in mol per mol creatinine (CR).

Cortisol: Urine concentrations of cortisol were measured using a commercial kit (Amerlite, Kodak Clinical Diagnostics Ltd, Amersham, England). Samples (250 µl) and standards were extracted with diethyl ether (2.5 ml) (Merck, Darmstadt, Germany) and subsequently washed twice in 1 ml of distilled water. After evaporation samples and standards were resuspended in 250 µl of PBS and stored at +4 °C overnight. All further steps followed the procedure recommended by the manufacturer. The intra-assay coefficients of variation calculated from 19 precision profiles were below 13% for concentrations ranging from 9.8 to 1120.0 nmol/l. Inter-assay coefficients of variation were 22.1% (15.6 nmol/l), 19.1% (120.5 nmol/l) and 10.4% (485.8 nmol/l) based on 20 different assays. Urinary excretion of cortisol was expressed in nmol/24 h as well as µmol/mol CR.

Creatinine: Concentrations of CR in urine were determined by a kinetic picrate method (Jaffè reaction) without deproteinization (*Bartels & Böhmer* 1971, *Fabiny et al.* 1971) in a computerized multichannel spectrophotometer (Cobas Mira; Hoffman-LaRoche & Co, Basel, Switzerland).

Statistical analysis

The statistical analysis of the oestrone sulphate and cortisol data was carried out on logarithmic values using the General Linear Model procedure from SAS (Statistical Analysis Institute, Inc., 1987). Within-group variation between

gilts was used as an error term when testing the differences between groups. Means are expressed as ± s.e.m. Probabilities <0.05 were considered statistically different.

Results

A correlation between urinary excretion of oestrone sulphate expressed in nmol/24 h and μmol/mol CR was high and significant (range from r = 0.42 to r = 0.89, p<0.01). For cortisol there was no correlation between the nmol/24 h and μmol/mol CR values (p>0.5).

Fig. 1 depicts urinary oestrone sulphate excretion expressed in nmol/24 h in untreated (control) and dexamethasone-treated gilts from day 6 to day 33 of gestation. In control gilts the first increase in oestrone sulphate (866 ± 203 nmol/24 h) occurred on day 13 and the second (1224 ± 474 nmol/24 h) on day 17. In dexamethasone-treated gilts oestrone sulphate did not change until day 19 of pregnancy when it increased to 559 ± 191 nmol/24 h. In both groups oestrone sulphate excretion reached maximum values between days 27 and 32 of gestation (Fig. 1). The first increase of oestrone sulphate excretion in oil- and hydrocortisone-treated gilts was noted on day 14 (728 ± 376 nmol/24 h) and day 19 (1904 ± 1049 nmol/24 h) of pregnancy, respectively (Fig. 2). In oil-treated gilts oestrone sulphate excretion reached maximum values between days 25 and 32 of gestation (Fig. 2). In hydrocortisone-treated gilts oestrone sulphate excretion reached maximum values between days 22 and 26 of gestation (Fig. 2) and then significantly decreased until day 31. Regarding the urinary excretion of cortisol (Fig. 3), control gilts showed an increase between days 6 and 7, whereafter it tended to decrease until day 33 of pregnancy. In dexamethasone-treated gilts cortisol excretion significantly decreased on day 16, i.e. 3 days after injections of dexamethasone had commenced. Fig. 4 shows the urinary excretion of cortisol which was low 2 days (37 ± 7 nmol/24 h) before the oil injections and significantly increased 2 days (182 ± 106 nmol/24 h) afterwards. In hydrocortisone-treated gilts cortisol excretion significantly increased after the last injection of hydrocortisone, i.e. from 91 ± 19 nmol/24 h on day 20 of pregnancy to 210 ± 29 nmol/24 h on day 23 (Fig. 4).

A detailed examination of the urinary excretion of oestrone sulphate expressed in μmol/mol CR from untreated gilts revealed a positive relation between the concentration on day 20 of gestation and the number of foetuses, i.e. 65, 241, 1128, 3475 and 9250 μmol/mol CR corresponded to 8, 11, 14, 16 and 18 foetuses, respectively. Results obtained from the oestrone sulphate and cortisol determinations in urine from 2 control pregnant gilts are shown in Fig. 5 and 6, respectively. Gilt no. 934 had 18 corpora lutea and 18 foetuses. Urinary oestrone sulphate excretion in this gilt reached 10 000 μmol per mol CR on day 20 while the corresponding excretion rate in gilt no. 935 was still below 100 mol per mol CR (Fig. 5). At slaughter, we found 18 corpora lutea but only eight foetuses in gilt no. 935. As regards gilt no. 935, cortisol excretion was high between days 7 and 22 after mating (Fig. 6). Gilt no. 934 showed a relatively low rate of cortisol excretion that did not exceed 200 nmol/24 h between days 6 and 33 after mating.

Discussion

It was previously found that the urinary excretion of oestrone sulphate increases dramatically around day 20 after mating and reaches a maximum around days 25-28 in sows (*Seren et al.* 1983, *Choi et al.* 1987). In the present study we observed a significant increase of urinary oestrone sulphate excretion, expressed in nmol/24

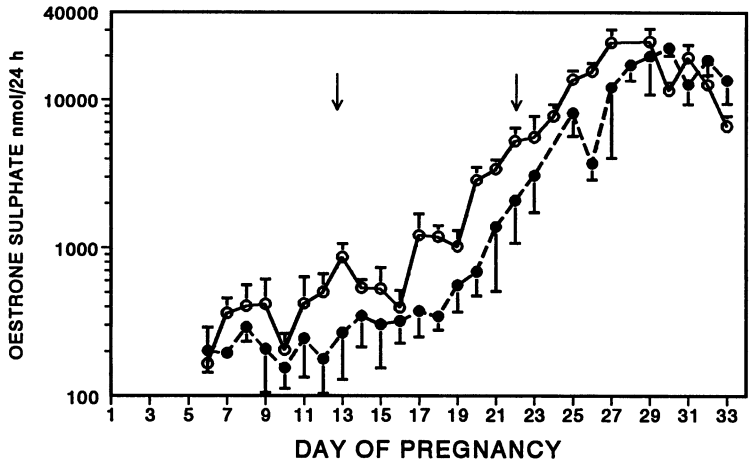

Figure 1. Urinary excretion of oestrone sulphate (nmol/24 h ± s.e.m.) in 5 control untreated gilts (○——○) and in 4 gilts treated with dexamethasone (●——●). Arrows indicate first and last day of dexamethasone injection.

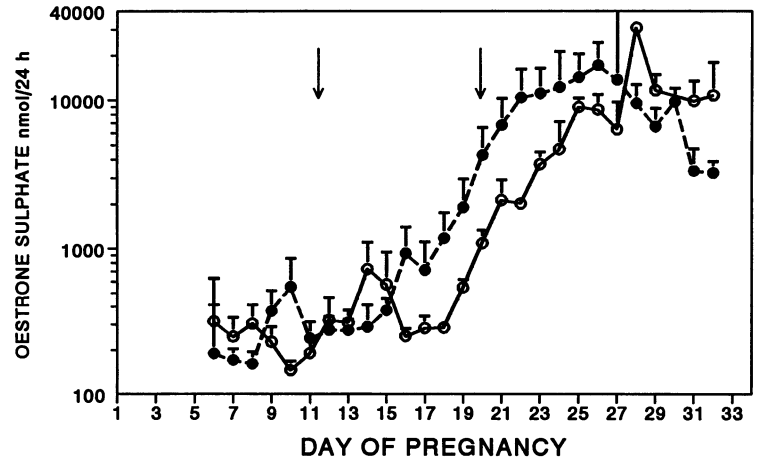

Figure 2. Urinary excretion of oestrone sulphate (nmol/24 h ± s.e.m.) in 3 control, oil-treated gilts (○——○) and in 4 gilts treated with hydrocortisone (●——●). Arrows indicate first and last day of oil or hydrocortisone injection.

h, on days 13-14 followed by a second one on days 19-20 of gestation in both untreated and oil-treated gilts. This is in relatively good agreement with the suggestion made by *Geisert et al.* (1990) that the synthesis and release of oestrogens from porcine blastocysts are biphasic, with the first increase on day 11 of pregnancy and the second one between days 14 and 18 (*Geisert et al.* 1990). Oestradiol is oxidized to oestrone in a reaction catalysed by dehydrog-

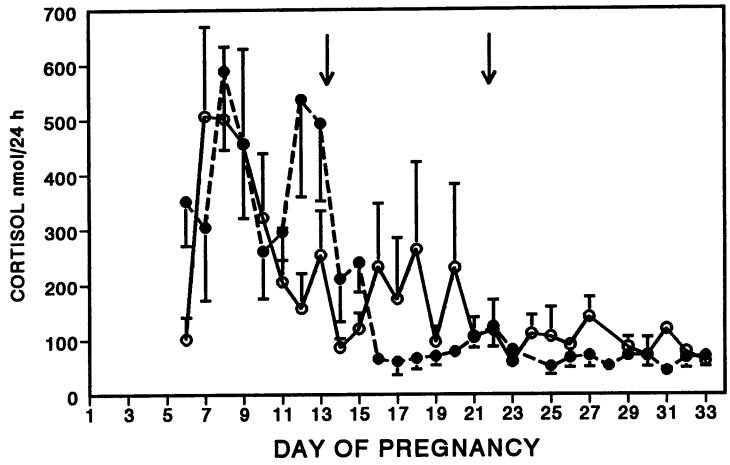

Figure 3. Urinary excretion of cortisol (nmol/24 h ± s.e.m.) in 5 untreated control gilts (○——○) and in 4 gilts treated with dexamethasone (●——●). Arrows indicate first and last day of dexamethasone injection.

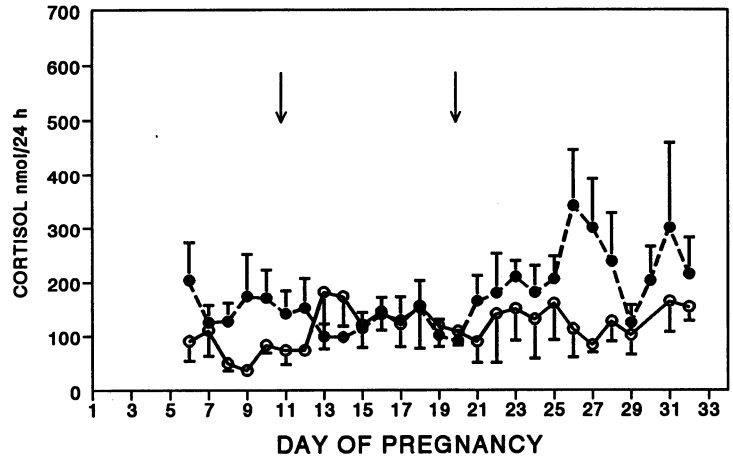

Figure 4. Urinary excretion of cortisol (nmol/24 h ± s.e.m.) in 3 control, oil-treated gilts (○——○) and in 4 gilts treated with hydrocortisone (●——●). Arrows indicate first and last day of oil or hydrocortisone injection.

enase, and free oestrone is probably then sulfated by endometrial sulfotransferase (see *Roberts et al.* 1993 for discussion).

We could not find any positive relation between levels of urinary oestrone sulphate excretion expressed in nmol/24 h and the number of fetuses. Previously, *Stoner et al.* (1986) found a positive correlation between plasma concentrations of oestrone sulphate on day 30 of pregnancy and litter size at parturition in swine.

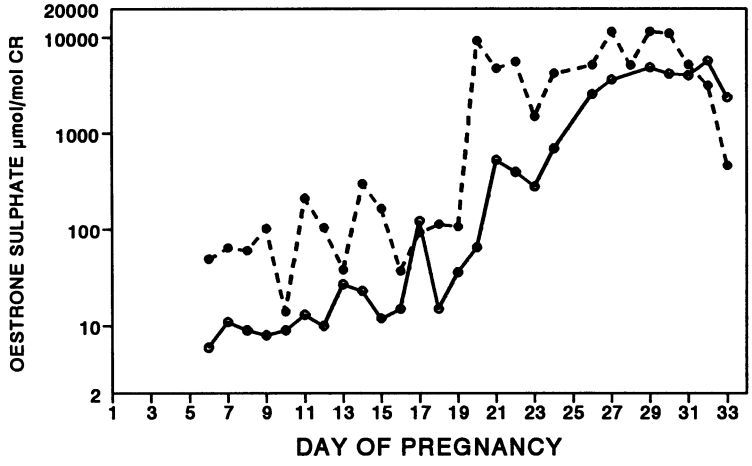

Figure 5. Urinary excretion of oestrone sulphate (µmol/mol CR) in 2 control untreated gilts (●——●, no. 934 with 18 foetuses and ○——○, no. 935 with 8 foetuses).

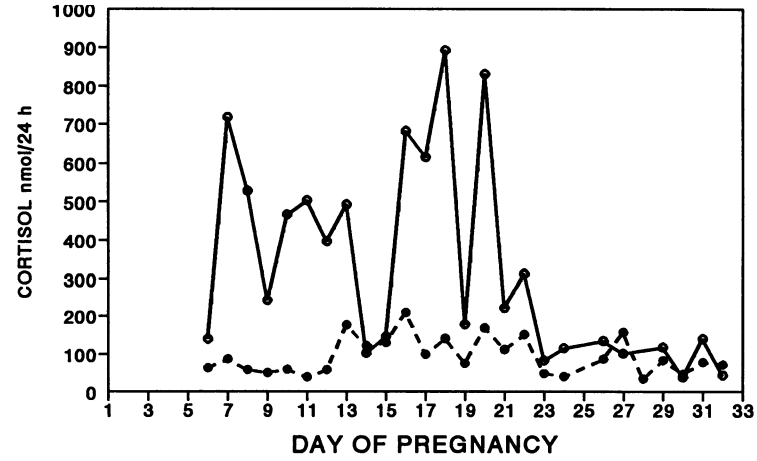

Figure 6. Urinary excretion of cortisol (nmol/24 h) in 2 control untreated gilts (●——●, no. 934 with 18 foetuses and ○——○, no. 935 with 8 foetuses).

However, *van der Wiel et al.* (1992) reported that such a correlation is too low to be used for reliably predicting the expected number of piglets at birth. In fact, earlier findings of *Edqvist et al.* (1980) suggested that the plasma oestrone sulphate could be used in practice for early pregnancy diagnosis in pigs but not for predicting litter size. On the other hand, *Moenter et al.* (1992) reported that pregnant pigs with plasma concentrations of oestrone sulphate greater

than 0.4, 0.5, 0.67, 0.85, 1.04 or 1.75 ng/ml on days 20, 21, 22, 23, 24, 25 or 26, respectively, were classified as having 8 or more foetuses. Animals with lower concentrations were classified as having 7 or fewer foetuses. Litter size classification was correct in 73% of the cases (*Moenter et al.* 1992). There were no differences in serum concentrations of total conjugated and unconjugated oestrone up to day 30 of gestation between Meishan and Large White hybrid gilts (*Hunter et al.* 1994). In the present study we also tried to relate the urinary excretion of oestrone sulphate expressed in μmol/mol CR to the number of foetuses. The only relation was found on day 20 of pregnancy in control untreated gilts. This observation needs to be corroborated in the further studies.

Both dexamethasone and hydrocortisone treatment seemed to delay the first observed peak of oestrone sulphate. *Liptrap & Cummings* (1991) treated sows with dexamethasone from day 9 to day 14 of the oestrous cycle. The main finding was the low androstenedione and oestradiol concentrations in follicular fluid on day 12 during treatment with dexamethasone. Elevated levels of glucocorticoids resulted in fewer 5 mm follicles as well as in a reduction of their steroidogenic capacity. *Gee et al.* (1991) also concluded that exogenous glucocorticoids are able to affect follicular steroidogenesis through suppression of oestradiol secretion in the pig. Recently, *Tsuma et al.* (1996) found that food deprivation during early pregnancy elevated cortisol concentrations concomitant with decreased oestradiol-17β concentrations.

Urine cortisol is widely accepted as an integrator of circulating cortisol and represents the average level of free cortisol between urinations (*Miller et al.* 1991). In the present study dexamethasone treatment depressed the urinary excretion of cortisol within 3 days. In contrast, hydrocortisone injections had no influence on the urinary excretion of cortisol. The dose of hydrocortisone acetate used in our study was similar to that used by *Barb et al.* (1982) who reported a threefold elevation in plasma cortisol levels in gilts in connection with 2 daily injections of 250 mg hydrocortisone acetate for the first 12 days of the oestrous cycle. It is, however, interesting to note that 3 days after the last injection of hydrocortisone in the present study the urinary excretion of cortisol increased. Long-term ACTH treatment resulted in elevated cortisol excretion in bighorn sheeps (*Miller et al.* 1991). Additionally, we reported that ingestion of polychlorinated biphenyls resulted in a relatively high urinary excretion of cortisol in pregnant mink (*Madej et al.* 1992). It is interesting to note that 2 untreated gilts with the same number of corpora lutea but different numbers of foetuses had completely different rates of urinary cortisol excretion. Indeed, this supports the findings of *Behrens et al.* (1993) who used the pseudopregnant gilts as a model and concluded that cortisol could be involved in reproductive failure.

Barnett & Hemsworth (1986) reported that pregnant pigs in individual cage-stalls showed no evidence of a chronic stress response compared with group housed animals. Short- or long-term stress frequently influence growth rate, reproductive performance, the immune system and welfare status, although the effects vary from case to case. Thus consequences of stress need to be seriously considered in the design and implementation of experiments. The fact that embryonic survival as well as the number of viable foetuses were not affected by glucocorticoid treatment (see *Madej et al.* 1997) suggests that early pregnant gilts are capable of overcoming even such a stress.

Acknowledgements

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References

Ashworth CJ, Haley CS, Wilmut I: Effect of Regumate on ovulation rate, embryo survival and conceptus growth in Meishan and Landrace× Large White gilts. Theriogenology 1992, *37,* 433-443.

Barb CR, Kraeling RR, Rampacek GB, Fonda ES, Kiser TE: Inhibition of ovulation and LH secretion in the gilt after treatment with ACTH or hydrocortisone. J. Reprod. Fert. 1982, *64,* 85-92.

Barnett JL, Hemsworth PH: The impact of handling and environmental factors on the stress response and its consequences in swine. Lab. Anim. Sci. 1986, *36,* 366-369.

Bartels H, Böhmer M: Eine Mikromethode zur Kreatininbestimmung. (Method for determination of creatinine). Clin. Chim. Acta. 1971, *32,* 81-85.

Bazer FW, Ott TL, Spencer TE: Pregnancy recognition in ruminants, pigs and horses: Signals from the trophoblast. Theriogenology 1994, *41,* 79-94.

Behrens MJ, Kattesh HG, Baumbach GA: Plasma and uterine cortisol, progesterone and protein changes in pseudopregnant gilts treated with hydrocortisone acetate. Theriogenology 1993, *40,* 1231-1241.

Choi HS, Kiesenhofer E, Gantner H, Hois J, Bamberg E: Pregnancy diagnosis in sows by estimation of oestrogens in blood, urine or faeces. Anim. Reprod. Sci. 1987, *15,* 209-216.

Edqvist L-E, Einarsson S, Larsson K: Early pregnancy diagnosis in pigs by assay of oestrone sulphate in peripheral blood plasma. Proc. Int. Pig Vet. Soc. 1980, p. 27.

Fabiny DL, Ertinghausen G: Automated reaction-rate method for determination of serum creatinine with the CentriChem. Clin. Chem. 1971, *17,* 696-700.

Galvin JM, Wilmut I, Day BN, Ritchie M, Thomson M, Haley CS: Reproductive performance in relation to uterine and embryonic traits during early gestation in Meishan, Large White and crossbred sows. J. Reprod. Fert. 1993, *98,* 377-384.

Gee CM, Geissinger HD, Liptrap RM: Morphometric and steroid hormone changes associated with experimental anovulatory follicles in the sow. Can. J. Vet. Res. 1991, *55,* 206-211.

Geisert RD, Zavy MT, Moffatt RJ, Blair RM, Yellin T: Embryonic steroids and the establishment of pregnancy in pigs. J. Reprod. Fert. 1990, Suppl. *40,* 293-305

Hunter MG, Faillace LS, Picton HM: Intrauterine and peripheral steroid concentrations and conceptus development in Meishan and Large White hybrids gilts. Reprod. Fertil. Dev. 1994, *6,* 783-789.

Liptrap RM, Cummings E: Steroid concentrations in follicular fluid of sows treated with dexamethasone. Anim. Reprod. Sci. 1991, *26,* 303-310.

Madej A, Forsberg M, Edqvist L-E: Urinary excretion of cortisol and oestrone sulphate in pregnant mink fed by PCB and fractions of PCB. AMBIO 1992, *21,* 582-585

Madej A, Romanowicz K, Forsberg M, Barcikowski B, Einarsson S: Effect of glucocorticoid treatment on biochemical and hormonal blood parameters in early pregnant gilts. Acta vet. scand. 1997, *38,* 263-273.

Miller MW, Hobs NT, Sousa MC: Detecting stress responses in Rocky Mountain bighorn sheep (*Ovis canadensis canadensis*): reliability of cortisol concentrations in urine and feces. Can. J. Zool. 1991, *69,* 15-24.

Moenter SM, Webel SK, Dziuk PJ: Pregnancy detection an litter size classification by oestrone sulfate measurements in swine under farm conditions. Anim. Reprod. Sci. 1992, *27,* 161-167.

Roberts RM, Xie S, Trout WE: Embryo-uterine interactions in pigs during week 2 of pregnancy. J. Reprod. Fert. 1993, Suppl., *48,* 171-186.

Seren, E, Mattioli M, Gaiani R, Tamanini C: Direct estimation of urine oestrone conjugate for a rapid pregnancy diagnosis in sows. Theriogenology 1983, *19,* 817-822.

SAS Institute, Inc.: SAS/STAT® Guide for Personal Computers, Version 6 Edition. SAS Institute, Inc., Cary, NC, 1987.

Stoner CS, Bazer FW, Thatcher WW, Wilcox CJ, Combs GE, Knight JW, Wettemann RP, White CE: Relationship between oestrone sulfate in plasma and litter size at farrowing for sows and gilts. Theriogenology 1986, *25,* 709-720.

Tsuma VT, Einarsson S, Madej A, Kindahl H, Lundeheim N: Effect of food deprivation during early pregnancy on endocrine changes in primiparous sows. Anim. Reprod. Sci. 1996, *41,* 267-278.

van der Wiel DEM, Vos EA, te Brake JHA, van der Lende T: Pregnancy diagnosis in sows: enzyme immuno-assay measurement of oestrone sulphate in plasma on Day 25-30 after insemination in comparison to ultrasound scanning on Day 28. Livestock Prod. Sci. 1992, *32,* 323-330.

Varley M, Stedman R: Stress and reproduction. In Cole DJA, Wiseman J, Varley MA, (eds.), Principles of Pig Sciences, Nottingham University Press, 1994, pp. 277-296.

Wright K, Collins DC, Musey PI, Preedy JRK: A specific radioimmunoassay for oestrone sulfate in plasma and urine without hydrolysis. J. Clin. Endocrinol. Metab. 1978, *47,* 1092-1098.

Sammanfattning

Urinutsöndringen av östronsulfat och kortisol under tidig dräktighet hos gyltor med eller utan glukokortikoidbehandling.

Studien omfattade 20 polska Lantrasgyltor, som uppstallades vid The Kielanowski Institute of Animal Physiology and Nutrition, Jablonna, Polen. Efter betäckning placerades gyltorna i metabolismburar. För att undvika den stress som kan uppkomma vid blodprovstagning samlades urinprover för att följa insatt behandling. Vi behandlade dräktiga gyltor med upprepade doser av dexametason, vegetabilisk olja respektive hydrokortison från dag 11-13 t.o.m. dag 20-22 efter betäckning. Urinprover, som togs från dag 6 t.o.m. dag 32, analyserades med avseende på östronsulfat, kortisol och kreatinin. Det förelåg en hög korrelation mellan urinutsöndringen av östronsulfat uttryckt i nmol/24 timmar och μmol/mol kreatinin. Däremot fanns det ingen korrelation när det gäller exkretionen av kortisol. Den första ökningen av östronsulfat, uttryckt i nmol/24 timmar, påvisades dag 13-14 och den andra dag 19-20 av dräktigheten i de båda kontrollgrupperna. Resultatet av studien tyder på att den första ökningen av östronsulfat hämmades efter dexametason- och hydrokortisonbehandlingen. Urinutsöndringen av östronsulfat uppnådde sitt maximum mellan dag 25 och 32 av dräktigheten. Urinutsöndringen av kortisol hos dexametasonbehandlade gyltor sjönk dag 16, dvs. 3-5 dagar efter påbörjad behandling med dexametason. Behandling av gyltor med hydrokortison resulterade i en ökad utsöndring av kortisol efter den sista injektionen. Det fanns ingen relation mellan östronsulfatmängden i urinen, uttryckt i nmol/24 timmar, och antalet funna foster. Däremot kunde man påvisa en sådan korrelation mellan östronsulfat uttryckt i mol/mol kreatinin under dag 20 av dräktigheten. Hos en kontrollgylta, vars exkretion av kortisol var högre än 200 nmol/24 timmar fanns, endast 8 levande foster av 18 möjliga. Hos en annan kontrollgylta, där alla 18 foster var levande, var exkretionen av kortisol väsentligt lägre.

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