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Glucocorticoid exposure before and after birth alters blood profiles in piglets

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Elevated maternal glucocorticoid levels during pregnancy may influence fetal development and postnatal physiology of offspring. The aim of this study was to evaluate the effects of prenatal and neonatal dexamethasone exposure on hematological parameters in piglets. The experiment was conducted on 24 clinically healthy Large White Polish sows randomly assigned to two prenatal treatment groups: dexamethasone-treated (DEX) or physiological saline-treated controls (PhS). Piglets were subsequently allocated to four experimental groups according to prenatal and neonatal exposure (PhS/PhS, PhS/DEX, DEX/PhS, DEX/DEX) in a 2 × 2 factorial design. Blood samples were collected from six piglets per treatment group at each sampling time point (postnatal days 3, 14, and 30).

Significant main effects of prenatal and neonatal dexamethasone exposure were observed in several erythrocyte parameters. On day 3, both prenatal and neonatal dexamethasone significantly increased RBC ($p<0.001$) and decreased MCV and MCH ($p<0.001$), with a significant interaction between factors. On day 14, neonatal dexamethasone increased RBC, HCT, and Hb values ($p\leq0.030$), whereas prenatal exposure significantly affected MCHC and MCH ($p\leq0.010$). By day 30, dexamethasone exposure was associated with lower Hb, HCT, MCH, and MCHC values ($p\leq0.010$), indicating microcytic and hypochromic erythrocyte changes. White blood cell counts were not consistently affected by the treatments.

These findings demonstrate that prenatal and neonatal glucocorticoid exposure modifies early erythropoietic development in piglets and influences hematological adaptation during the suckling period.

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The intrauterine environment plays a key role in shaping mammalian development. Adverse maternal conditions, including stress associated with elevated glucocorticoid exposure during gestation, may induce long-lasting physiological changes in offspring, a phenomenon known as prenatal programming [Reddout-Beam *et al.* 2024]. In pigs, prenatal stress has been linked to alterations in hypothalamic-pituitary-adrenal (HPA) axis function, behavior, immune competence, and the development of the skeletal and gastrointestinal systems [Śliwa *et al.* 2005]. Reduced lymphocyte proliferation and thymus size have also been reported, potentially increasing susceptibility to neonatal morbidity [Tuchscherer *et al*. 2002]. Together, these findings demonstrate that the gestational environment can induce persistent adaptations across multiple physiological systems in offspring [Merlot *et al*. 2008, Otten *et al*. 2015].

Glucocorticoids (GCs) are major mediators of prenatal stress effects on the fetus and play a central role in developmental programming. Physiological increases in endogenous cortisol during late gestation contribute to fetal organ maturation; however, excessive or prolonged glucocorticoid exposure may adversely affect fetal development [Merlot *et al.* 2008]. Among synthetic glucocorticoids, dexamethasone (Dex) is widely used in experimental models because of its strong affinity for glucocorticoid receptors and potent biological activity. Dexamethasone readily crosses the placenta and can influence fetal development through activation of glucocorticoid signaling pathways [Baisden *et al.* 2007]. In swine, exogenous glucocorticoids have been used experimentally to modify the timing of parturition or to investigate the effects of enhanced glucocorticoid signaling on fetal development and neonatal growth [De Rensis *et al*. 2002, Śliwa *et al.* 2009, Śliwa *et al.* 2010, Śliwa *et al.* 2006, Carroll 2001]. Thus, dexamethasone administration in experimental studies primarily serves as a model of increased glucocorticoid exposure rather than a routine practice in pig production, although its periparturient administration has been investigated in commercial sow management systems [Ward *et al.* 2020, Will *et al.* 2023].

Despite increasing interest in prenatal glucocorticoid exposure in pigs, hematological parameters in piglets exposed to dexamethasone remain insufficiently characterized. Neonatal hematological indices, including red blood cell count, hemoglobin concentration, hematocrit, and leukocyte profiles, are important indicators of physiological status, oxygen-carrying capacity, and immune competence in newborn animals. Altered glucocorticoid exposure during fetal development may affect hematopoiesis and lead to measurable changes in blood parameters during early postnatal life [Wasfi *et al.* 1990]. In veterinary physiology, exogenous glucocorticoids are known to induce a characteristic steroid leukogram consisting of neutrophilia and lymphopenia, often accompanied by leukocytosis [Amdi *et al.* 2020, Flaming *et al.* 1994]. Consequently, chronic maternal glucocorticoid exposure during gestation may alter leukocyte profiles in the offspring [Hong 2022]. In addition, glucocorticoids may influence erythropoiesis by affecting erythroid progenitor proliferation and

erythropoietic activity. Evidence from other species suggests that glucocorticoid exposure can increase erythropoietic activity or hemoglobin levels [Fjelkner *et al.* 2024]; however, such effects remain poorly characterized in swine.

Early postnatal life is a critical period for hematopoietic development in piglets, during which erythropoiesis expands rapidly, and the immune system undergoes functional maturation. Therefore, this study evaluated the effects of prenatal and/or neonatal dexamethasone exposure on hematological parameters. We hypothesized that dexamethasone exposure during the prenatal and/or early postnatal period would alter hematological profiles, resulting in changes in erythrocyte indices and leukocyte counts during the suckling period.

Material and methods

Ethical approval

The animal study protocol was approved by the Local Ethical Committee for Animal Experiments in Lublin (Resolution No. 1105).

Animals, breeding, and experimental design

Twenty-four clinically healthy Large White Polish sows (second or third parity) were used in this study. All sows had previously raised litters with comparable maternal behavior and an average pre-weaning mortality of one to two piglets per litter. The sows were individually housed in separate pens under standard husbandry conditions: controlled ambient temperature and humidity, a 12:12 h light-dark cycle, ad libitum access to fresh water, and a commercial diet provided twice daily (2.3 kg/day). The feed was nutritionally adequate for pregnant and lactating sows, containing 12.96 MJ ME/kg and 16.35% crude protein. Farrowing room temperature was maintained at 22-23°C, while creep area for piglets 32-35°C.

Sows were naturally mated using boars of the same breed, age class, and genetic line. To minimize genetic variability, only Large White Polish boars from the same breeding herd were used. Each boar serviced no more than two sows per week, and their use was balanced across experimental groups to avoid systematic sire effects.

Gestational day 0 was defined as the day of first mating. On gestational day 91, a total of 24 clinically healthy sows were randomly assigned to two prenatal treatment groups: DEX group (n = 12) – sows receiving dexamethasone during late gestation; and PhS group (n = 12) – control sows receiving physiological saline.

From gestational day 91 to day 115, sows in the DEX group received intramuscular injections of dexamethasone (Dexamethasone pro inj. 0.2%, Eurovet Animal Health B.V., Bladel, The Netherlands) at a dose of 3.0 mg every second day, resulting in a total dose of approximately 36 mg per sow. The dose was administered as a fixed amount because the sows were of similar parity and body mass. Control sows received an equivalent volume of physiological saline (PhS) according to the same schedule.

Following parturition, litters within each prenatal treatment group were randomly allocated to neonatal treatment, resulting in four experimental groups. Within both the prenatal DEX group and the control group, six sows were assigned to neonatal dexamethasone treatment and six to neonatal saline, producing the following four treatment combinations: DEX/DEX (n = 6 sows) – prenatal dexamethasone + neonatal dexamethasone; DEX/PhS (n = 6 sows) – prenatal dexamethasone + neonatal saline; PhS/DEX (n = 6 sows) – prenatal saline + neonatal dexamethasone; PhS/PhS (n = 6 sows) – prenatal saline + neonatal saline.

All piglets within each litter received the neonatal treatment assigned to their dam. Piglets in the DEX/DEX and PhS/DEX groups received dexamethasone intramuscularly at 0.5 mg/kg body weight every second day from postnatal day 2 to day 30. Piglets in the DEX/PhS and PhS/PhS groups received physiological saline according to the same schedule and route of administration. All injections were administered at 9:00 a.m. into the cervical neck musculature caudal to the ear using standard swine injection procedures. Body weight was recorded every second day to ensure accurate dose calculation.

The dosing regimen was based on previously published experimental studies in pigs, including earlier work by our group, in which comparable dexamethasone protocols produced consistent biological effects without adverse maternal outcomes [Śliwa *et al.* 2005, Śliwa *et al*. 2009]. Although dexamethasone is rapidly absorbed after intramuscular administration and has a relatively short elimination half-life in pigs (approximately 0.8-1.1 h) [Wyns *et al*. 2013], the every-second-day dosing interval was selected to provide repeated glucocorticoid exposure during early postnatal development while minimizing the frequency of animal handling.

Gestation length did not differ between groups and averaged 114.8±0.7 days in the PhS group and 115.2±0.6 days in the DEX group (mean±SE). Mean litter size at birth was 9.1±1.4 in the PhS group and 9.3±1.2 in the DEX group, with no significant differences between treatments. The sex distribution of piglets was comparable across groups, and neonatal mortality remained low (<5%). All piglets were free of congenital defects and remained with their biological dams throughout the study period. No cross-fostering, litter equalization, or culling procedures were applied, allowing piglets to remain under their original maternal environment from birth until the end of the experiment. Body weights were recorded every second day to ensure accurate dose calculation for DEX-treated piglets.

For hematological analysis, one piglet per litter was sampled at each time point, with each sow contributing one piglet at every age examined. Blood samples were collected at postnatal days 3, 14, and 30, resulting in six piglets per treatment group at each sampling time point (n = 6).

Piglets were not offered solid feed and were not treated with any medications during the 30-day suckling period. Iron supplementation was intentionally omitted to maintain a uniform experimental model and to allow evaluation of hematological responses under conditions of limited iron availability during early postnatal life.

Blood sample collection

All piglets within each litter were treated according to their assigned prenatal and neonatal experimental group. For hematological analyses, a subset of piglets was selected for blood sampling, with six piglets per treatment group sampled at each time point (postnatal days 3, 14, and 30). A cross-sectional sampling design was applied, and different piglets were used at each sampling time so that each animal was sampled only once during the study.

Piglets were not fasted before blood collection. At each sampling time point, one piglet was randomly selected from each litter for blood collection to minimize potential selection bias. Different piglets were sampled at each age, and each animal was sampled only once during the study. Blood was collected by venipuncture of the external jugular vein and transferred immediately into EDTA-coated tubes. Piglets were gently restrained during sampling, and all procedures were performed by experienced personnel using standard swine handling techniques. The sampling procedure was completed rapidly (within a few minutes per animal) to minimize potential stress-related alterations in hematological parameters. Hematological parameters were measured using an automated veterinary hematology analyzer Mythic 5 Vet Pro (Orphée SA, Geneva, Switzerland), routinely employed for whole blood analysis in swine at the University of Life Sciences in Lublin. The following parameters were assessed: red blood cell count (RBC), hemoglobin concentration (Hb), hematocrit (HCT), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), and white blood cell count (WBC).

Statistical analysis

Blood morphology parameters were analysed using a general linear model (GLM) including prenatal treatment (PhS or DEX administered to sows) and neonatal treatment (PhS or DEX administered to piglets) as fixed factors and their interaction. Because one piglet per litter was sampled at each age, the individual piglet constituted the observational unit for statistical analysis at a given sampling time point. Litter was included in the model as a random effect to account for potential litter-related variation. Data were analysed separately for each sampling age (postnatal days 3, 14, and 30) because different piglets were sampled at each time point. When significant effects were detected, pairwise comparisons were performed using Tukey's post hoc test. Statistical significance was set at $p<0.05$, and values between 0.05 and 0.10 were considered a trend towards significance. All analyses were performed using Statistica 13.3 (TIBCO Software Inc., Palo Alto, CA, USA).

Results and discussion

Significant effects of prenatal and/or neonatal dexamethasone exposure were observed for RBC, MCV, MCHC, and MCH in 3-day-old piglets (Tab. 1). Prenatal dexamethasone significantly increased RBC counts ($p=0.001$), and a similar effect

Table 1. Hematological parameters in piglets on postnatal day 3 following prenatal and/or neonatal dexamethasone administration (mean±pooled SEM)

Dexamethasone exposure[1]		RBC (10^{12}/L)	HCT (L/L)	Hb (mmol/L)	MCV (fl)	MCHC (mmol/L)	MCH (fmol)	WBC (10^9/L)
prenatal[2]	neonatal[3]							
					Main factors			
-		3.06^{b}	0.194	4.34	64.60^{b}	22.54^{a}	1.46^{a}	7.16
+		3.70^{a}	0.220	4.58	59.67^{a}	20.78^{b}	1.32^{b}	6.22
	-	3.03^{b}	0.198	4.21	66.52^{b}	22.75^{a}	1.51^{a}	6.35
	+	3.73^{a}	0.215	4.43	57.75^{a}	20.57^{b}	1.19^{b}	7.03
					Treatment[4]			
-	-	2.48^{b}	0.174	4.15	70.42	23.96^{a}	1.68^{a}	6.35
-	+	3.63^{a}	0.214	4.51	58.77	21.12^{b}	1.24^{bc}	7.97
+	-	3.57^{a}	0.222	4.79	62.62	21.53^{a}	1.35^{b}	6.34
+	+	3.83^{a}	0.217	4.35	56.72^{c}	20.02^{b}	1.14^{c}	6.09
SEM[5]		0.172	0.001	0.230	1.229	0.458	0.046	0.656
					p-value			
Prenatal		0.001	0.052	0.306	<0.001	0.001	<0.001	0.166
Neonatal		<0.001	0.122	0.873	<0.001	<0.001	0.001	0.309
Prenatal x neonatal		0.018	0.052	0.105	0.030	0.166	0.021	0.170

[1]Number of observations for calculation of treatment effects n = 6, for main factors n = 12; [2]Piglets exposed to dexamethasone prenatally; [3]Piglets exposed to dexamethasone neonatally; [4]Different superscript letters (a, b, c) indicate significant differences between experimental groups within the same hematological parameter ($p<0.05$); [5]SEM = pooled standard error of the mean derived from the two-way ANOVA model; − Indicates no dexamethasone exposure; + Indicates dexamethasone exposure; symbols refer separately to prenatal and neonatal treatment.

was observed for neonatal dexamethasone (p=0.001). A significant prenatal × neonatal interaction was detected for RBC (p=0.018). Both prenatal and neonatal dexamethasone exposure significantly reduced MCV and MCH values (p<0.001 for both factors). Significant prenatal × neonatal interactions were also observed for MCV (p=0.030) and MCH (p=0.021). Prenatal dexamethasone exposure showed a tendency to affect HCT values (p=0.052). In addition, MCHC values were reduced by both prenatal (p=0.001) and neonatal (p<0.001) dexamethasone exposure.

In 14-day-old piglets (Tab. 2), neonatal dexamethasone exposure increased RBC (p=0.005), HCT (p=0.010), and Hb (p=0.030). Prenatal dexamethasone significantly affected erythrocyte indices, increasing MCHC (p<0.001) and MCH (p=0.010) and decreasing WBC counts (p=0.003). Significant prenatal × neonatal interactions were detected for RBC (p=0.003), HCT (p=0.005), Hb (p=0.003), MCHC (p=0.001), MCH (p=0.039), and WBC (p=0.037).

On day 30, significant effects were observed for RBC, HCT, Hb, MCV, MCHC, and MCH (Tab. 3). Neonatal dexamethasone exposure decreased RBC (p=0.006) and HCT (p=0.001). The prenatal × neonatal interaction showed a tendency for RBC (p=0.051), whereas no interaction was detected for HCT (p=0.110). Significant prenatal × neonatal interactions were detected for Hb (p = 0.010), MCV (p=0.008), MCHC (p<0.001), and MCH (p<0.001), whereas no interaction was observed for WBC (p = 0.797). Significant prenatal effects were found for MCV (p=0.001), MCHC

Table 2. Hematological parameters in piglets on postnatal day 14 following prenatal and/or neonatal dexamethasone administration (mean±pooled SEM)

Dexamethasone exposure[1]		RBC (10^{12}/L)	HCT (L/L)	Hb (mmol/L)	MCV (fl)	MCHC (mmol/L)	MCH (fmol)	WBC (10^9/L)
prenatal[2]	neonatal[3]							
					Main factors			
-		4.46	0.222	3.85	49.64	17.03	0.84	8.22
+		4.41	0.221	4.21	50.16	19.03	0.95	5.92
	-	3.82	0.197	3.51	50.01	18.14	0.91	7.43
	+	5.06	0.252	4.55	49.79	17.92	0.89	6.71
					Treatment[4]			
-	-	3.19^{b}	0.157^{b}	2.17^{b}	49.50	16.39^{c}	0.81^{b}	9.67^{a}
-	+	5.73^{a}	0.287^{a}	5.13^{a}	49.77	16.67b^{c}	0.88^{ab}	6.76^{b}
+	-	4.44^{ab}	0.223^{ab}	4.45^{ab}	50.52	19.88^{a}	1.00^{a}	5.18^{b}
+	+	4.38^{ab}	0.218^{ab}	3.97^{ab}	49.80	18.17^{b}	0.90^{ab}	6.65^{b}
SEM[5]		0.172	0.003	1.342	0.715	0.787	0.039	0.624
					p-value			
Prenatal		0.899	0.947	0.427	0.684	<0.001	0.01	0.003
Neonatal		0.005	0.010	0.030	0.861	0.583	0.699	0.294
Prenatal x neonatal		0.003	0.005	0.003	0.701	0.001	0.039	0.037

[1]Number of observations for calculation of treatment effects n = 6, for main factors n = 12; [2]Piglets exposed to dexamethasone prenatally; [3]Piglets exposed to dexamethasone neonatally; [4]Different superscript letters (a, b, c) indicate significant differences between experimental groups within the same hematological parameter (p<0.05); [5]SEM = pooled standard error of the mean derived from the two-way ANOVA model; − Indicates no dexamethasone exposure; + Indicates dexamethasone exposure; symbols refer separately to prenatal and neonatal treatment.

Table 3. Hematological parameters in piglets on postnatal day 30 following prenatal and/or neonatal dexamethasone administration (mean±pooled SEM)

Dexamethasone exposure[1]		RBC (10^{12}/L)	HCT (L/L)	Hb (mmol/L)	MCV (fl)	MCHC (mmol/L)	MCH (fmol)	WBC (10^9/L)
prenatal[2]	neonatal[3]							
					Main factors			
-		5.93^{ab}	0.286^{ab}	4.69	48.87	17.96	0.88	12.97
+		5.87^{ab}	0.255^{ab}	4.14	43.06	16,00	0.69	9.49
	-	6.51^{a}	0.303^{a}	5.06	47.29	18.43	0.88	10.61
	+	5.30^{b}	0.238^{b}	3.77	44.64	15.53	0.75	11.08
					Treatment[4]			
-	-	6.94	0.350^{a}	5.97^{a}	52.52^{a}	20.64^{a}	1.09^{a}	12.16
-	+	4.92	0.222^{b}	3.41^{b}	45.22^{b}	15.28^{b}	0.68^{b}	13.77
+	-	6.07	0.256^{b}	4.15^{b}	42.06^{b}	16.22^{b}	0.68^{b}	9.06
+	+	5.67	0.254^{b}	4.12^{b}	44.05^{b}	15.77^{b}	0.70^{b}	9.92
SEM[5]		0.172	0.030	1.40	0.713	0.970	0.05	0.657
					p-value			
Prenatal		0.879	0.187	0.229	0.001	0.001	<0.001	0.025
Neonatal		0.006	0.001	0.008	0.106	<0.001	<0.001	0.400
Prenatal x neonatal		0.051	0.110	0.010	0.008	<0.001	<0.001	0.797

[1]Number of observations for calculation of treatment effects n = 6, for main factors n = 12; [2]Piglets exposed to dexamethasone prenatally; [3]Piglets exposed to dexamethasone neonatally; [4]Different superscript letters (a, b, c) indicate significant differences between experimental groups within the same hematological parameter (p<0.05); [5]SEM = pooled standard error of the mean derived from the two-way ANOVA model; − Indicates no dexamethasone exposure; + Indicates dexamethasone exposure; symbols refer separately to prenatal and neonatal treatment.

(p=0.001), MCH (p<0.001), and WBC (p=0.025). Neonatal treatment significantly affected Hb (p=0.008), MCHC (p<0.001), and MCH (p<0.001).

Age-related patterns of hematological changes

Although blood samples were collected from different piglets at each time point, an age-related pattern of hematological changes could be identified at the group level. Because different animals were sampled at each age, these observations are descriptive and no statistical comparisons between ages were performed. In all experimental groups, RBC counts increased between postnatal days 3 and 14. Piglets exposed to neonatal dexamethasone showed a greater increase in RBC values on day 14 compared with control piglets. By day 30, RBC, hemoglobin, and hematocrit values were lower in dexamethasone-exposed groups compared with controls. In contrast, no clear age-related directional pattern was observed for WBC counts across sampling times.

Information on the hematological consequences of prenatal and/or neonatal dexamethasone exposure in pigs remains scarce, and current knowledge is based largely on studies in other species and human neonates. In the present study, dexamethasone administration was used as an experimental model of repeated glucocorticoid exposure during early life to evaluate potential effects of glucocorticoid signaling on postnatal hematological parameters in piglets. Although such an experimental approach does not directly replicate clinical conditions, it provides a controlled framework for investigating potential glucocorticoid-mediated influences on early postnatal physiological adaptation.

The selected sampling time points correspond to key stages of early postnatal development in piglets. Postnatal day 3 represents the immediate neonatal period, characterized by rapid physiological adaptation, including stabilization of erythropoiesis, early immune maturation, and the establishment of autonomous endocrine regulation [Huang *et al.* 2024]. Day 14 corresponds to the mid-suckling period, during which hematological parameters increasingly reflect both prenatal influences and postnatal environmental conditions [Yu *et al.* 2019].

The present results indicate that dexamethasone exposure was associated with dynamic changes in erythrocyte parameters during early postnatal life. Increased RBC values were observed on postnatal days 3 and 14, although the magnitude and pattern of this response differed between these time points. Because erythropoiesis requires several days for the generation of new erythrocytes, the early increase observed on day 3 most likely reflects acute physiological responses such as redistribution of circulating erythrocytes, transient changes in plasma volume, or splenic mobilization of erythrocytes rather than newly formed cells.

By contrast, the increase in RBC observed on day 14 in piglets receiving neonatal dexamethasone may reflect a delayed physiological response consistent with the period of rapid postnatal expansion of erythropoiesis in piglets. Glucocorticoids are known to influence erythropoiesis, and similar glucocorticoid-associated increases

in erythropoietic activity have been described in experimental models [Hanssen and Iskander 2025, Voorhees *et al*. 2013]. However, because direct indicators of erythropoietic activity were not measured in the present study, such mechanisms remain speculative.

Interestingly, prenatal dexamethasone exposure alone did not sustain the elevated RBC values observed early after birth, whereas neonatal dexamethasone treatment was associated with higher RBC counts on day 14. This pattern may suggest that prenatal glucocorticoid exposure modifies the subsequent responsiveness of hematological parameters to postnatal glucocorticoid stimulation. Comparable long-term effects of prenatal stress and glucocorticoid exposure on offspring physiology have been described previously [Merlot *et al.* 2008, Otten *et al*. 2015, Hong 2022]. Previous studies in pigs have also reported short-term hematological responses following dexamethasone administration [Li *et al.* 2019]. In the present study, hematological measurements were performed several days after treatment initiation, which may explain the detection of delayed or secondary responses rather than immediate pharmacological effects.

At later stages of the suckling period, the direction of the erythrocyte response changed. By day 30, lower RBC, HCT, and Hb values were observed in dexamethasone-exposed piglets. These later changes were accompanied by reductions in erythrocyte indices (MCV, MCH, and MCHC), which are consistent with iron-limited erythropoiesis. Piglets are born with limited iron reserves, and sow milk contains insufficient iron to support the high demands associated with rapid postnatal growth. Consequently, iron deficiency commonly develops during the suckling period in the absence of supplementation [Fjelkner *et al*. 2024, Ventrella *et al*. 2016]. In the present experimental model, iron supplementation was intentionally omitted to maintain uniform experimental conditions. Therefore, the hematological pattern observed at later stages of development likely reflects iron-restricted erythropoiesis associated with rapid growth rather than a direct pathological effect of dexamethasone itself. Under such conditions, reductions in MCV, MCH, and MCHC are consistent with the development of microcytic and hypochromic erythrocytes typical of iron deficiency [Fjelkner *et al.* 2024, Ventrella *et al*. 2016].

Glucocorticoids may also influence erythroid maturation dynamics. Experimental studies suggest that glucocorticoids can stimulate proliferation of erythroid progenitors while temporarily delaying terminal differentiation of erythroblasts [Hanssen and Iskander 2025, Voorhees *et al.* 2013]. Such mechanisms could potentially contribute to transient changes in erythrocyte indices; however, the absence of reticulocyte counts or direct markers of erythropoietic activity in the present study limits interpretation of these processes.

In contrast to erythrocyte parameters, leukocyte responses were relatively modest. Prenatal dexamethasone exposure was associated with lower total WBC values at postnatal days 14 and 30, whereas neonatal dexamethasone administration did not significantly affect total leukocyte counts. Glucocorticoids are known to influence leukocyte distribution and immune function [Amdi *et al.* 2020, Flaming *et al.* 1994].

Consequently, chronic maternal glucocorticoid exposure during gestation may alter leukocyte profiles in the offspring [Hong 2022]. Because differential leukocyte counts were not determined in the present study, potential changes in leukocyte subpopulations cannot be excluded despite relatively stable total WBC values.

The present findings suggest that prenatal and/or neonatal dexamethasone exposure is associated with transient alterations in erythrocyte parameters during early postnatal life. At later stages of the suckling period, hematological changes appear to be strongly influenced by physiological limitations in iron availability characteristic of rapidly growing piglets without iron supplementation. An important limitation of the present study is that differential leukocyte counts were not determined. Consequently, potential dexamethasone-induced alterations in specific leukocyte populations, including neutrophils and lymphocytes, could not be evaluated, and interpretation was limited to total WBC counts.

In conclusion, prenatal and/or neonatal dexamethasone exposure modifies hematological parameters in piglets during early postnatal life. Neonatal dexamethasone administration was associated with transient increases in erythrocyte parameters during the early suckling period, whereas prenatal exposure appeared to influence the subsequent responsiveness of these parameters to postnatal glucocorticoid treatment. At later stages of the suckling period, reductions in RBC, HCT, Hb, and erythrocyte indices were observed, consistent with iron-limited erythropoiesis in rapidly growing piglets without iron supplementation. Prenatal dexamethasone exposure was also associated with lower total leukocyte counts at some time points. Together, these findings indicate that glucocorticoid exposure during prenatal and/or early postnatal life can transiently influence hematological adaptation in piglets, although later changes appear to be strongly shaped by physiological iron limitation during the suckling period.

Conflict of interest

The authors declare that they have no conflicts of interest.

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