



## Concentrations of chemerin and leptin in healthy and sick newborns

### Stężenie chemeryny i leptyny u noworodków zdrowych i chorych

Alicja Nawrat , Justyna Czubińska-Lada , Anna Sienko , Anna Szymańska

Department of Neonatal Intensive Care and Pathology, Faculty of Medical Sciences in Zabrze,  
Medical University of Silesia, Katowice, Poland

#### ABSTRACT

**INTRODUCTION:** Chemerin and leptin are adipokines involved in the regulation of metabolism and the inflammatory response. In the neonatal period, they may reflect both fetal nutritional status and early adaptive disturbances associated with infection or perinatal risk factors.

**MATERIAL AND METHODS:** The study included 127 term, appropriate-for-gestational-age newborns, divided into groups: Ia – early-onset infection (n = 40), Ib – perinatal risk factors without infection (n = 36), and II – control group (n = 51). Chemerin and leptin concentrations were measured in peripheral venous blood serum between the 3rd and 7th day of life. Associations between hormonal, metabolic and clinical parameters were analyzed.

**RESULTS:** Serum chemerin and leptin concentrations were significantly higher in newborns with early-onset infection and in those burdened with perinatal risk factors compared with healthy infants.

**CONCLUSIONS:** Chemerin and leptin appear to be markers of metabolic and inflammatory disturbances in the perinatal period. Measurement of their concentrations in peripheral venous blood serum may constitute a valuable adjunctive tool for identifying newborns who require increased clinical surveillance.

#### KEYWORDS

chemerin, leptin, newborn, early-onset infection, perinatal risk factors

#### STRESZCZENIE

**WSTĘP:** Chemeryna i leptyna są adipokinami uczestniczącymi w regulacji metabolizmu i odpowiedzi zapalnej. W okresie noworodkowym mogą odzwierciedlać zarówno stan odżywienia płodu, jak i wczesne zaburzenia adaptacyjne związane z zakażeniem lub czynnikami ryzyka okołoporodowego.

**MATERIAŁ I METODY:** Badaniem objęto 127 noworodków donoszonych, podzielonych na grupy: Ia – z zakażeniem wczesnym (n = 40), Ib – z czynnikami ryzyka okołoporodowego (n = 36), II – kontrolną (n = 51). Stężenia chemeryny i leptyny w surowicy krwi żyłnej oznaczono między 3. a 7. dobą życia. Analizowano zależności między parametrami hormonalnymi, metabolicznymi i klinicznymi.

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**Address for correspondence:** dr n. med. Alicja Nawrat, Klinika Intensywnej Terapii i Patologii Noworodka, Wydział Nauk Medycznych w Zabrze ŚUM, ul. 3 Maja 13, 41-800 Zabrze, tel. +48 32 370 42 91, e-mail: anawrat@szpital.zabrze.pl



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**WNIKI:** Stężenia chemeryny i leptyny były istotnie wyższe u noworodków z wczesnym zakażeniem i obciążonych perinatalnymi czynnikami ryzyka niż u zdrowych.

**WNIOSKI:** Chemeryna i leptyna są markerami zaburzeń metabolicznych i zapalnych w okresie okołoporodowym. Oznaczenie ich stężeń w surowicy obwodowej krwi żyłnej może stanowić cenne narzędzie w identyfikacji noworodków wymagających zwiększonego nadzoru klinicznego.

## SŁOWA KLUCZOWE

chemeryna, leptyna, noworodek, wczesne zakażenie, czynniki ryzyka okołoporodowego

## INTRODUCTION

Adipose tissue cells synthesize and secrete numerous adipokines, including chemerin and leptin, which play important roles in carbohydrate and lipid metabolism, angiogenesis, fetal and neonatal development, and the pathogenesis of infections [1]. Elevated levels of chemerin have been reported in individuals with metabolic syndrome, obesity, diabetes mellitus, chronic obstructive pulmonary disease, chronic pancreatitis, psoriasis, neoplastic diseases, and in children with epilepsy [2,3,4,5,6,7]. In inflammatory conditions, chemerin exerts antimicrobial activity and stimulates macrophages, natural killer (NK) cells and dendritic cells [1,8,9,10]. Leptin is a better-characterized adipokine. In newborns, its relationships with gender, anthropometric parameters and gestational age, as well as its involvement in early-onset infections, have been partially elucidated [11,12,13,14,15]. In the present study, we evaluated chemerin and leptin concentrations in healthy and sick term newborns. The aim of the study was to determine whether early-onset infections affects the concentrations of these hormones and to analyze correlations between chemerin and leptin levels and selected inflammatory markers in affected newborns.

## MATERIAL AND METHODS

A total of 127 full-term newborns of any gender, appropriate for gestational age and aged 3–7 days, participated in the study. Based on the presence of early-onset infection and perinatal risk factors, the infants were divided into two main groups:

**Group I (study group)** – 76 newborns, including:

**Ia** – 40 newborns with early-onset infection diagnosed within the first 72 hours of life

**Ib** – 36 newborns without infection but burdened with perinatal risk factors:

- maternal and sociomedical: maternal age under 16 years, alcohol abuse during pregnancy, maternal nicotine use, history of recurrent miscarriages, pregnancy-induced hypertension, first-trimester vaginal bleeding, maternal anemia, oligohydramnios, urinary and genital

tract infections, zoonotic infectious diseases, group B streptococcus colonization

- labor-related: operative delivery (including caesarean section), premature rupture of membranes with amniotic fluid leakage > 18 hours, meconium-stained amniotic fluid
- neonatal: need for resuscitation procedures, endotracheal intubation and mechanical ventilation

**Group II (control group)** – 51 healthy newborns from uncomplicated pregnancies, delivered vaginally after spontaneous onset of labor.

All newborns from the study group were treated at the Department of Neonatal Intensive Care and Pathology in Zabrze, while 51 healthy infants from the control group were born during the same period in the Obstetrics and Neonatology Unit in Gliwice. Among the diagnosed infections, the following were identified: pneumonia (11 cases), urinary tract infection (9 cases), sepsis (6 cases), concurrent omphalitis, skin infection and conjunctivitis (6 cases), osteomyelitis (2 cases), and single cases of enteritis, meningitis, extensive oral and pharyngeal candidiasis, systemic inflammatory response syndrome, mastitis and generalized staphylococcal skin infection.

Perinatal risk factors primarily included abnormal, non-physiological labor (26 cases) and adverse socio-economic conditions or maternal pregnancy pathology (24 cases). Neonatal risk factors accounted for 14% of cases (5 newborns).

Chemerin and leptin concentrations in venous blood serum were measured using the ELISA method between the 3rd and 7th day of life. For laboratory analysis, 1.5 mL of venous blood was collected during routine diagnostic blood sampling. The study was approved by the Bioethics Committee of the Medical University of Silesia in Katowice (Resolution No. KNW/022/KB1/1/I/16). All patients had written informed consent obtained from their parents or legal guardians.

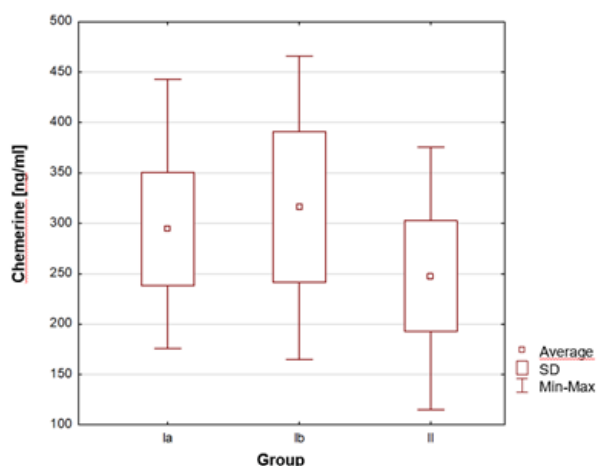
Clinical data were collected from medical records and interviews with parents or legal guardians. Diagnoses of early-onset infections and perinatal asphyxia were established based on widely accepted criteria. Clinical data and laboratory results were analyzed using the licensed software Statistica 12.0 (StatSoft Inc., Tulsa, OK, USA).



## RESULTS

### Assessment of serum chemerin concentrations in both groups of newborns

In the entire cohort of 127 newborns, serum chemerin concentrations ranged from 115.2 to 466.1 ng/mL, with a mean value of  $281.8 \pm 67.79$  ng/mL. In newborns with early-onset infection (group Ia), the mean concentration of this hormone was  $294.30 \pm 56.25$  ng/mL (range: 175.6–442.8 ng/mL) and was significantly higher ( $p < 0.001$ ) than in the control group (group II), in which the mean concentration was  $247.6 \pm 54.92$  ng/mL (range: 115.2–375.5 ng/mL). In newborns without infection but with perinatal risk factors (group Ib), chemerin concentrations ranged from 164.8 to 466.1 ng/mL, with a mean value of  $316.3 \pm 74.86$  ng/mL. This value was also significantly higher ( $p < 0.001$ ) than in the control group (group II). The mean concentration of chemerin in group Ib did not differ significantly from that observed in newborns with infection (group Ia). The results of the analysis are shown in Figure 1.



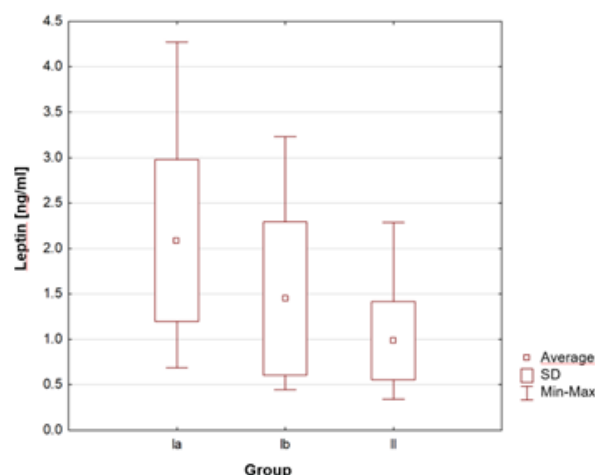
**Fig. 1.** Comparison of mean serum chemerin concentrations in newborns with early-onset infection (group Ia), newborns with perinatal risk factors (group Ib) and the control group (group II)

### Assessment of serum leptin concentrations in both groups of newborns

In the entire cohort of 127 newborns, serum leptin concentrations ranged from 0.342 to 4.273 ng/mL, with a mean value of  $1.464 \pm 0.86$  ng/mL. In newborns with early-onset infection (group Ia), the mean concentration of this hormone was  $2.088 \pm 0.89$  ng/mL (range: 0.687–4.273 ng/mL), which was significantly higher ( $p < 0.001$ ) than in the control group (group II), where the mean concentration was  $0.985 \pm 0.43$  ng/mL (range: 0.342–2.285 ng/mL).

In group Ib, leptin concentrations ranged from 0.447 to 3.229 ng/mL, with a mean value of  $1.449 \pm 0.84$  ng/mL. This value was also significantly higher ( $p < 0.05$ ) than in group II.

The mean leptin concentration in newborns with early-onset infection was also significantly higher ( $p < 0.001$ ) than in newborns without infection but with perinatal risk factors. The results of the analysis are presented in Figure 2.



**Fig. 2.** Comparison of mean serum leptin concentrations in newborns with early-onset infection (group Ia), newborns with perinatal risk factors (group Ib), and the control group (group II)

### Assessment of chemerin and leptin concentrations in both groups of newborns according to gender

Table I presents a comparison of chemerin concentrations between male and female newborns in the study and control groups. In healthy female newborns mean chemerin concentrations was significantly higher than in male newborns ( $p < 0.05$ ). No such significant differences were observed in newborns with early-onset infection ( $p = 0.599$ ) or in newborns with perinatal risk factors ( $p = 0.110$ ).

**Table I.** Analysis of serum chemerin concentrations in both groups of newborns according to sex

Group		Male	Female	p
Ia n = 40	N	26	14	0.599
	Average	287.8	287.8	
	SD	51.27	66.08	
	Range	196.9–442.8	175.6–390.1	
Ib n = 36	N	25	11	0.1104
	Average	329.6	286.2	
	SD	70.79	78.44	
	Range	190.2–466.1	164.8	
Control n = 51	N	27	24	0.0116
	Average	229.6	267.9	
	SD	57.23	45.25	
	Range	115.2–314.0	165.8–375.5	

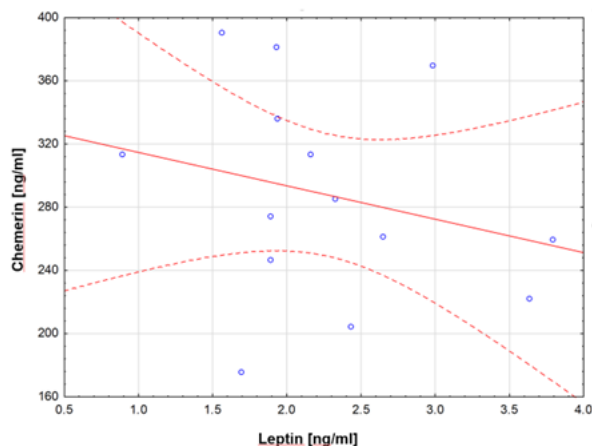


Table II presents a comparison of mean leptin concentrations between male and female newborns in the study and control groups. Gender did not have a significant effect on leptin concentrations in healthy newborns, in those with early-onset infection, or in those with perinatal risk factors.

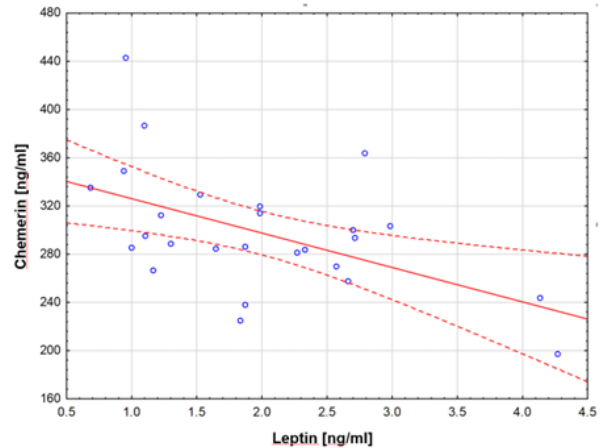
**Table II.** Analysis of serum leptin concentrations in both groups of newborns according to gender

Group		Male	Female	p
Ia n = 40	N	26	14	0.343
	Average	1.989	2.273	
	SD	0.938	0.79	
	Range	0.687–4.275	0.896–3.795	
Ib n = 36	N	25	11	0.216
	Average	1.586	1.137	
	SD	0.85	0.77	
	Range	0.447–3.136	0.497–3.229	
Control n = 51	N	27	24	0.177
	Average	0.901	1.079	
	SD	0.378	0.47	
	Range	0.342–1.587	0.414–2.285	

Taking into account gender within each group, a detailed analysis was performed and revealed significant negative correlations between chemerin and leptin concentrations in newborns with early-onset infection (group Ia). The correlation analysis, presented in Figures 3 and 4, indicates that in both female and male newborns with early-onset infection, higher chemerin levels are associated with lower leptin concentrations.



**Fig. 3.** Correlation analysis between serum chemerin and leptin concentrations in female newborns with early-onset infection



**Fig. 4.** Correlation analysis between serum chemerin and leptin concentrations in male newborns with early-onset infection

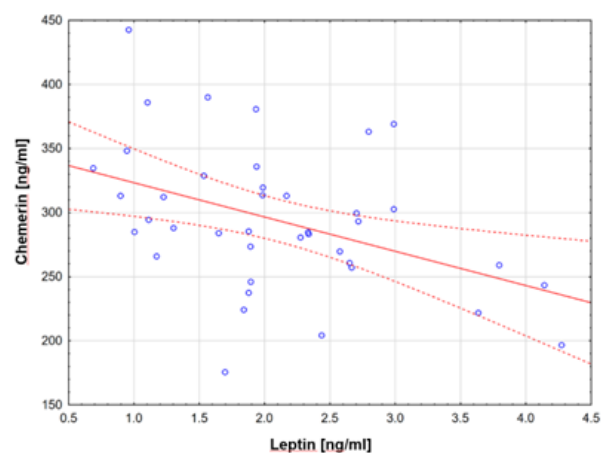
### Correlation analysis between chemerin and leptin concentrations with consideration of gender in the study and control groups

The results of the correlation analysis between chemerin and leptin with consideration of gender in the study and control groups are presented in Table III.

**Table III.** Results of the correlation analysis between serum chemerin and leptin concentrations according to gender in the study and control groups

Group	Gender	Correlation	
		r	p
Ia	Female	-0.2527	0.383
	Male	-0.522	0.006
Ib	Female	-0.067	0.845
	Male	-0.177	0.396
Control	Female	0.276	0.191
	Male	0.117	0.560

A negative correlation ( $p < 0.05$ ) was found in male newborns with diagnosed early-onset infection between chemerin and leptin concentrations (Figure 5).



**Fig. 5.** Correlation analysis between serum chemerin and leptin concentrations and male gender in newborns with diagnosed early-onset infection



### Correlation analysis between chemerin and leptin concentrations according to selected biochemical and hematological indicators in the study groups

The results of the correlation analysis between chemerin and leptin concentrations and selected biochemical and hematological markers in newborns with early-onset infection (group Ia) are presented in Table IV, and in newborns with perinatal risk factors (group Ib) in Table V.

**Table IV.** Correlation analysis between serum chemerin and leptin concentrations in newborns with early-onset infection and selected biochemical and hematological indicators

Indicator	Chemerin		Leptin	
	r	p	r	p
CRP	0.1673	0.302	-0.1160	0.476
Hematocrit	0.2604	0.105	0.0432	0.791
Hemoglobin	0.2513	0.118	0.0087	0.958
Red blood cells	0.1996	0.217	0.0824	0.0613
White blood cells	-0.1491	0.359	0.1859	0.251
Platelets	0.2016	0.212	0.0215	0.895

CRP – C-reactive protein

**Table V.** Correlation between serum chemerin and leptin concentrations and selected biochemical and hematological indicators in newborns exposed to perinatal risk factors

Indicator	Chemerin		Leptin	
	r	p	r	p
CRP	-0.2290	0.179	-0.2241	0.189
Hematocrit	-0.2986	0.077	-0.1836	0.284
Hemoglobin	-0.2732	0.107	-0.2108	0.217
Red blood cells	-0.2656	0.117	-0.2096	0.220
White blood cells	-0.2809	0.097	-0.1567	0.362
Platelets	0.1870	0.275	-0.0635	0.713

CRP – C-reactive protein

The performed analysis did not reveal any statistically significant correlations between serum chemerin and leptin concentrations and the laboratory parameters assessed in the study groups, either in newborns with early-onset infection or in those exposed to perinatal risk factors.

## DISCUSSION

Research conducted in recent years has significantly advanced our understanding of adipose tissue, its structure, metabolism, and interactions with other organs. It has been confirmed that adipose tissue is a complex system of metabolically active cells and functions as an endocrine organ [16,17,18]. Its components – including adipocytes, connective tissue stroma, vascular stromal cells, immune cells, and

neural elements – work together to produce a variety of biologically active adipokines that act locally (autocrine/paracrine) or on distant organs (endocrine effects) [3,16,17,18]. Among them, leptin, first described in 1994 as an anorexigenic factor is the best characterized [12,15,16,19,20]. Another adipokine that has recently gained considerable interest is chemerin, discovered more than 20 years ago [1,3,8,16,21,22,23,24]. Both hormones play important roles in fetal and neonatal development [13,25,26,27].

Normal growth early in life is regulated by numerous factors reflecting complex interactions between the fetus, the mother and the placenta. Proper development depends not only on adequate delivery of oxygen, glucose and protein to the fetus but also on the ability to utilize these substrates in growth processes. These mechanisms are influenced by numerous hormones – including thyroid hormones, growth hormone, insulin, cortisol, adiponectin, leptin, chemerin and somatotropin – whose regulation during the perinatal and neonatal periods remains poorly understood [12,13,26,27,28,29]. Developmental disturbances are only partly due to genetic factors; more often they are the result from maternal systemic diseases, harmful habits, and complications during pregnancy and delivery [30,31,32,33,34,35].

In our study of 127 term eutrophic newborns, we demonstrated that early-onset infections – systemic or localized, primarily affecting the respiratory and urinary systems – caused a significant increase in serum chemerin concentration compared with healthy term newborns. This finding suggests that chemerin is actively involved in infection-related inflammation or supports the hypothesis that early-onset infection in a eutrophic term newborn stimulates chemerin production and secretion. Chemerin is known to have multiple biological functions, including immunologic activity through the stimulation of chemotaxis of macrophage and immature dendritic cells [1,19]. Its exact role in inflammatory processes remains unclear despite numerous experimental and clinical studies [9,10,22,25,36]. The role of chemerin in immune responses is complex, as its effects varies depending on the length of its polypeptide chain and may be both pro- and anti-inflammatory. The largest form (chemerin-157) participates in early inflammatory responses and has a potent chemotactic activity [37], while the 154-amino-acid form suppresses macrophage activation, exerting an anti-inflammatory effect [37]. Elevated chemerin levels are observed in adults with chronic gastrointestinal, renal and joint diseases and during exacerbations of chronic obstructive pulmonary disease [25,38]. In recent years, Godlewska et al. [8] demonstrated antibacterial and anti-*Candida albicans* activity of recombinant chemerin and the chemerin-derived peptide p4 in adults. Eichelmann et al. [16] also demonstrated an association



between chemerin and inflammatory markers (C-reactive protein, tumor necrosis factor  $\alpha$ , interleukin 6).

Our research also showed that perinatal risk factors significantly increased chemerin concentrations in newborns without infection compared with healthy infants from uncomplicated pregnancies. This suggests that pregnancy pathology (excluding endocrine disorders and maternal hypertension), cesarean delivery, and associated perinatal stressors may contribute to increased chemerin production in early neonatal life. In these newborns, mean chemerin concentrations were higher not only compared with healthy controls but even (albeit insignificantly) compared with those with early-onset infection. Hyperchemerinemia in newborns from non-physiological pregnancies or delivered by cesarean section – likely resulting from increased placental synthesis and release – may in the future be recognized as a marker of metabolic risk. However, the lack of similar observations in adolescents, children and newborns limits the ability to determine the extent to which early-onset infections activate the immature immune system. It can only be assumed that severe infectious pathology during the first month of life plays an important role in chemerin secretion, and that its overproduction (hyperchemerinemia) may adversely affect hormonal development and predispose to metabolic disturbances later in life.

Leptin, like chemerin, is an adipokine essential for metabolism and growth and an indicator of nutritional status. Concentrations of both hormones are influenced by numerous exogenous factors [12,15,16,24]. In the present study, we confirmed a significant impact of early-onset infection on the increase of leptin concentration, which indicates a substantial stimulation of leptin synthesis and secretion during infection. The observed hyperleptinemia supports the role of leptin – due to its cytokine-like structure – as a hormone involved in inflammatory processes and immune activation [16,20,39]. Leptin is known to activate macrophages and monocytes, stimulate the proliferation of endothelial cells and naïve T-cells, and promote the migration of immunocompetent cells. It may inhibit proliferation of the memory T-cells and influence phagocytic function [16,39]. Korek and Krauss [40] suggest that adipokines, including leptin, which is important in obesity and metabolic dysregulation, may adversely affect inflammation by increasing the synthesis of interleukin 6 and tumor necrosis factor  $\alpha$ . Reports on leptin levels in neonatal infections are inconsistent. In 1996, Frazer-Llado et al. [41] found only minimal expression of the leptin gene and no significant changes in serum leptin levels in newborns

with severe infections. Conversely, Orbak et al. [42] reported higher leptin concentrations in newborns with bacterial sepsis than in healthy infants and documented a strong correlation between leptin and other inflammatory markers (C-reactive protein, neutrophil index), suggesting its potential as a future diagnostic marker of severe neonatal infections. In our study, no such correlations were observed between chemerin or leptin and C-reactive protein, platelet count or leukocyte count in newborns with or without infection who had perinatal infectious or non-infectious risk factors.

As with chemerin, we found higher mean leptin concentrations in newborns with perinatal risk factors and no infection than in healthy infants. Numerous studies have emphasized the impact of pregnancy and delivery pathology on hormonal changes in newborns with normal postnatal adaptation [13,14,42]. Determining whether gender influences hormone levels in healthy and ill newborns has long been a subject of interest to researchers. In our study, chemerin concentrations were higher in healthy female newborns compared with males; however, early-onset infection and perinatal risk factors offset this difference, reflecting substantial hormonal disturbances even in eutrophic term newborns born after pathological pregnancies or deliveries. We also demonstrated a significant negative correlations between chemerin and leptin in newborns with early-onset infection – both girls and boys – suggesting biological interactions between these two hormones.

We did not find any significant gender-related differences in leptin concentrations among term newborns, whether healthy or ill, which is consistent with findings by Bury et al. [13]. However, Sadownik et al. [43] showed that newborns of various gestational ages with intrauterine infection had higher leptin concentrations in girls than in boys. Similarly, Stojewska et al. [14] reported significantly higher leptin levels in peripheral blood in healthy female newborns compared with male newborns.

Present findings, combined with the latest neonatal research on the influence of perinatal risk factors and infection on hormonal and lipid profile alterations, confirm that this issue remains unresolved. The neonatal period is a particularly vulnerable phase, during which harmful factors – related to infection, hypoxia and anatomical or physiological immaturity – can have profound long-term consequences. There is a clear need for further research to determine the roles of various disease processes, not only infectious ones, in the hormone secretion mechanisms, their mutual interactions and potential long-term health effects.



## CONCLUSIONS

1. Early-onset infections, non-physiological pregnancy and labor in the mother contribute to increased concentrations of serum chemerin and leptin in peripheral venous blood of eutrophic term newborns, regardless of gender.

2. Healthy female newborns born at term exhibit higher chemerin concentrations than male newborns.

3. In both female and male newborns with early-onset infection, there is a correlation between chemerin and leptin concentrations is observed, despite the no differences in leptin levels between healthy and sick newborns of either gender.

## Authors' contribution

Study design – A. Nawrat

Data collection – A. Nawrat, A. Szymańska

Data interpretation – A. Nawrat, J. Czubińska-Łada, A. Sienko, A. Szymańska

Statistical analysis – A. Nawrat

Manuscript preparation – J. Czubińska-Łada

Literature research – A. Sienko

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