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THE RELATIONSHIP BETWEEN CRP GENE POLYMORPHISM AND THE SERUM CONCENTRATIONS OF C-REACTIVE PROTEIN, TOTAL CHOLESTEROL AND HDL CHOLESTEROL IN SUCKLING PIGLETS*

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Abstract

The relationship between CRP gene (1271 G/A, 3'UTR) polymorphism and the serum levels of C-reactive protein (CRP), total cholesterol (Ch-T) and high density lipoprotein cholesterol (HDL-ch) was analysed in suckling crossbred [Polish Large White × Polish Landrace (♀) × × Duroc × Pietrain (♂)] piglets. CRP genotypes were identified by PCR-RFLP with HinfI restriction enzyme. The levels of CRP, Ch-T, HDL-ch and white blood cell (WBC) counts were determined in blood samples collected from younger (21±3 days of age) and older piglets (35±3 days of age). There was a relationship between CRP gene (1271 G/A, 3'UTR) polymorphism and variations in the serum levels of CRP in piglets with normal WBC counts. The above relationship did not manifest itself in piglets with elevated WBC counts. The studied genotypes differed in their response to elevated WBC counts, and the noted differences were more pronounced in older piglets. The response of genotypes with weak CRP expression caused an increase in CRP levels and a decrease in the serum concentrations of Ch-T and HDL-ch. Such a response was not observed in the genotype with strong CRP expression.

Key words: CRP gene, C-reactive protein, cholesterol, HDL-ch, piglets

C-reactive protein (CRP) is a positive acute phase reactant in humans (Black et al., 2004; Hage and Szalai, 2007, 2009) and pigs (Llamas Moya et al., 2006; Heegaard et al., 2011). During systemic defense responses to infections, inflammations or injuries, serum CRP levels increase rapidly and dramatically (Black et al., 2004; Llamas Moya et al., 2006). CRP recognizes antigens, forms complexes with them and initiates their elimination by activating humoral (complement system) and cel-

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lular (phagocytes) factors. CRP concentrations quickly return to normal in response to treatment (Black et al., 2004; Llamas Moya et al., 2006).

Slightly elevated serum CRP level indicates immunological stimulation. CRP is not merely a marker of inflammation but actively participates in diseases. CRP has pleiotropic effects including both anti-inflammatory and pro-inflammatory activities (Black et al., 2004; Hage and Szalai, 2007, 2009).

In the body, CRP occurs in two conformationally distinct forms, pentameric (pCRP) and monomeric (mCRP). Dissociation of pentameric to monomeric CRP takes place on activated platelets and cells exposing bioactive lipids (Eisenhardt et al., 2011). Both CRP forms have pro-inflammatory properties, but mCRP is mainly associated with pathophysiological effects in inflammatory diseases (Eisenhardt et al., 2011).

Elevated serum CRP concentrations are noted in many human pathological conditions such as cardiovascular disease, metabolic syndrome and type 2 diabetes (Black et al., 2004; Hage and Szalai, 2007, 2009). Apart from slightly elevated CRP levels, low-grade chronic systemic inflammation triggers other pathophysiological responses, including a two- to three-fold increase in proinflammatory cytokine levels, leukocytosis (Petersen and Pedersen, 2005), disturbances in lipid and lipoprotein metabolism (Feingold and Grunfeld, 2010; Khovidhunkit et al., 2004). During infection/inflammation in human, serum triglyceride level is increased but HDL-ch markedly decreases. In turn, serum total cholesterol decreases or does not change (Khovidhunkit et al., 2004).

Many different mutations have been identified in the human CRP gene. Some of them, including in the 3'UTR region, affect serum CRP levels (Hage and Szalai, 2007, 2009; Shen and Ordovas, 2009). Martinez-Calleja et al. (2012) stated that 3'UTR region of CRP gene is associated with increased mRNA stability in T allele carriers of SNP +1444T/C and therefore increased expression of CRP. Furthermore, the TT genotype was more prevalent in subjects with obesity and diabetes type 2. In these patients, CRP and triglyceride levels, and leukocyte counts were higher compared with healthy ones.

The human CRP gene has been studied extensively, while the literature regarding the porcine CRP gene is limited. Chomdej et al. (2004) found two mutations in the 3'UTR region, Pareek and Życzko (2006) analysed the correlation between CRP gene polymorphism and the mortality rates of suckling piglets, whereas Łaszyn and Życzko (2010) studied the relationship between CRP gene mutations and CRP serum levels in sows.

The objective of this study was to determine the effect of CRP gene polymorphism on CRP expression and correlations with selected lipid profile parameters in piglets.

Material and methods

The experimental materials comprised suckling crossbred [Polish Large White × × Polish Landrace (♀) × Duroc × Pietrain (♂)] piglets (n = 295) with no visible dis-

ease symptoms, raised in a private farm. The piglets were divided into two groups: younger animals (n = 186), the offspring of sows (n = 22) at 21±3 days of lactation, and older animals (n = 109), the offspring of sows (n = 23) at 35±3 days of lactation. In the first week of their life, Suibiofer SE was administered to piglets (Biowet, Drwalew SA), they had their tails docked and teeth clipped, and boars were castrated. Upon the approval of the Local Animal Ethics Committee (No. 9/2008N), blood was sampled from the jugular vein, excluding piglets whose body weights were significantly different from the group average. DNA for molecular analyses was isolated from whole blood, using the MasterPure™ Genomic DNA Purification Kit (Epicentre, USA). Mutation at position 1271 (G/A) in the 3'UTR region of the CRP gene (GenBank accession no. AY714055) was identified by PCR-RFLP with HinfI restriction enzyme (Fermentas), as described by Chomdej et al. (2004). The levels of CRP, total cholesterol (Ch-T), high density lipoprotein cholesterol (HDL-ch), and white blood cell (WBC) counts were determined at a veterinary laboratory. In accordance with the instructions supplied by Biosystems Reagents and Instruments (Spain), CRP content was determined using a suspension of latex particles coated with anti-human CRP antibodies, Ch-T levels were measured with the use of cholesterol oxidase/peroxidase, and HDL-ch levels were determined by the direct method (Detergent). The following equipment was used: Epoll 20 photometer (CRP, HDL-ch), Hitachi 902 analyser (Ch-T), NS4 automatic blood analyser (WBC). For the purpose of a comparative statistical analysis, younger and older piglets were further subdivided into those with normal WBC counts of 11 to 22×10^3/µl, and elevated WBC counts of 23 to 40×10^3/µl (Quintero-Gutiérrez et al., 2008). The division was applied to exclude piglets with possible infection/inflammation without clinical symptoms. The data were processed statistically using the following tests: the Kolmogorov-Smirnov test and the Lilliefors test to compare the values of the analysed parameters with a normal distribution, the Mann-Whitney test and the Kruskal-Wallis test with a post-hoc analysis to determine the significance of differences between the two and three compared groups, respectively. The Spearman coefficient (r_s) was used to assess correlation between variables. All calculations were performed using STATISTICA 9.0 software. The results are presented as arithmetic means ($\bar{x}$), standard deviations (s), medians (me), and minimum and maximum values.

Results

The identified CRP genotypes, AA, AG and GG, accounted for 7.8%, 48.8% and 43.4% of all genotypes, respectively.

As shown in Table 1, in younger piglets CRP gene polymorphism was correlated with serum CRP concentrations only in animals with normal leukocyte counts. Piglets with the GG genotype, in comparison with AA and AG, had considerably higher CRP levels. No significant differences were found between AA and AG piglets in this respect. The serum concentrations of Ch-T and HDL-ch were similar in all genotypes.

Table 1. Relationship between CRP gene (1271 G/A) polymorphism and the serum levels of CRP, total cholesterol and HDL cholesterol in younger piglets with WBC counts within and above the normal range

Indices	WBC (10^9/l)		Piglet genotypes			P value
			AA	AG	GG	
CRP (mg/l)	within normal range	N	10	86	72	
		$\bar{x}$	7.0±7.40	9.4±7.84	12.4±6.39	<0.0001
		Me	5.35 A (1.10–26.20)	7.40 A (1.20–50.40)	11.85 B (1.90–41.0)	
	above normal range	N		11	7	0.7511
		$\bar{x}$		8.9±4.85	9.8±5.38	
		Me		9.0 (1.71–15.30)	9.6 (4.30–16.80)	
P value				0.6490	0.3836	
Ch-T (mmol/l)	within normal range	N	10	86	72	
		$\bar{x}$	2.8±0.84	2.8±0.97	2.6±0.70	0.5460
		Me	2.55 (1.86–4.64)	2.52 (1.64–6.54)	2.52 (1.34–5.32)	
	above normal range	N		11	7	
		$\bar{x}$		2.0±0.38	2.6±0.60	0.0550
		Me		2.06 (1.50–2.70)	2.50 (1.96–3.56)	
P value				0,0021	0.8134	
HDL-ch (mmol/l)	within normal range	N	10	86	72	
		$\bar{x}$	1.3±0.36	1.3±0.30	1.3±0.30	0.6909
		Me	1.18 (0.94–-2.06)	1.24 (0.60–2.12)	1.27(0.58–1.92)	
	above normal range	N		11	7	
		$\bar{x}$		1.1±0.26	1.1±0.30	0.7852
		Me		1.10 (0.74–1.72)	1.08 (0.74–1.66)	
P value				0.1060	0.1838	

A, B – values in rows with different letters differ significantly ($P \leq 0.01$).

Table 2. Relationship between CRP gene (1271 G/A) polymorphism and the serum levels of CRP, total cholesterol and HDL cholesterol in older piglets with WBC counts within and above the normal range

Indices	WBC (10^9/l)		Piglet genotypes			P value
			AA	AG	GG	
CRP (mg/l)	within normal range	N	9	33	30	
		$\bar{x}$	7.5±3.60	8.7±5.65	13.6±6.66	0.0036
		Me	8.10 a (2.40–12.50)	7.70 aA (1.20–23.20)	12.40 bB (3.10–31.30)	
	above normal range	N	4	14	19	0.9274
		$\bar{x}$	10.7±5.53	13.0±6.16	13.8±6.65	
		Me	10.80 (4.20–16.90)	10.90 (4.70–25.00)	11.3 (5.30–26.50)	
P value				0.018	0.9754	
Ch-T (mmol/l)	within normal range	N	9	33	30	
		$\bar{x}$	2.1±0.47	1.9±0.40	1.8±0.40	0.3357
		Me	1.98 (1.46–3.00)	1.94 (1.22–3.04)	1.76 (1.12–2.58)	
	above normal range	N	4	14	19	
		$\bar{x}$	1.7±0.37	1.6±0.19	2.2±0.77	0.0150
		Me	1.61 a (1.28–2.14)	1.66 a (1.40–2.00)	2.1 b (1.32–4.16)	
P value				0,0080	0.1027	
HDL-ch (mmol/l)	within normal range	N	9	33	30	
		$\bar{x}$	1.0±0.16	1.1±0.24	1.0±0.20	0.4017
		Me	1.04 (0.78–1.24)	1.08 (0.58–1.60)	0.97 (0.66–1.48)	
	above normal range	N	4	14	19	
		$\bar{x}$	0.9±0.14	0.9±0.08	1.1±0.23	0.0110
		Me	0.83 a (0.78–1.08)	0.88 a (0.74–1.02)	1.08 b (0.80–1.58)	
P value				0.0007	0.6005	

a, b – values in rows with different letters differ significantly ($P \leq 0.05$).
A, B – as above for $P \leq 0.01$.

The AG and GG genotypes were compared in the group of younger piglets with elevated WBC counts, as the AA genotype was not represented. They were found to differ only with regard to Ch-T levels which were higher in GG piglets and lower in AG individuals. No significant differences were noted in the serum concentrations of CRP and HDL-ch between genotypes.

A comparison of younger piglets with elevated and normal WBC counts revealed no differences in the studied parameters in the GG genotype, whereas AG individuals had considerably lower Ch-T levels, and showed a tendency towards increased CRP concentrations.

Similarly as younger animals, older piglets with normal WBC counts differed only with respect to serum CRP levels (Table 2) which were higher in the GG genotype, compared with AA and AG individuals. In the group of piglets with elevated WBC counts, higher Ch-T and HDL-ch levels were noted in the GG genotype, compared with AA and AG animals.

A comparison of older piglets with elevated and normal WBC counts (Table 2) revealed no significant differences in the serum levels of CRP, Ch-T and HDL-ch in the GG genotype, similarly as in younger animals. In contrast to the GG genotype, AG piglets were marked by substantially higher CRP concentrations and lower Ch-T and HDL-ch levels. Such trends, although less pronounced, were also observed in younger piglets. Similar tendencies to those noted in the AG genotype were also reported for AA piglets, but due to a too small number of animals they were not validated statistically.

Table 3. Values of Spearman's rank correlation coefficient (r_s) between the studied indices

Correlation between	WBC (10^9/l)		Piglet genotypes					
			younger			older		
			AA	AG	GG	AA	AG	GG
CRP and Ch-T	within normal range	r_s	0.042	0.220	0.064	–0.650	–0.078	–0.354
		p	0.907	0.043	0.597	0.058	0.663	0.054
	above normal range	r_s		–0.030	0.321		–0.563	–0.245
		p		0.934	0.498		0.030	0.311
CRP and HDL-ch	within normal range	r_s	–0.201	0.070	0.080	–0.426	–0.336	–0.251
		p	0.578	0.525	0.507	0.251	0.056	0.181
	above normal range	r_s		–0.155	0.464		–0.487	–0.091
		p		0.649	0.302		–0.083	0.709
Ch-T and HDL-ch	within normal range	r_s	0.778	0.544	0.685	0.803	0.617	0.555
		p	0.008	7×10^{-8}	$<1\times10^{-4}$	0.009	1×10^{-4}	14×10^{-4}
	above normal range	r_s		0.738	0.929		0.644	0.857
		p		0.015	0.007		0.010	3×10–6

Table 3 presents the values of the coefficients of correlation (rs) between the serum levels of CRP, Ch-T and HDL-ch in the studied genotypes. Irrespective of the age and genotype of piglets and their blood leukocyte counts, the serum concentrations of Ch-T and HDL-ch were strongly positively correlated. In the serum of younger piglets, the relationships between the other parameters were not significant, except for a positive correlation between CRP and Ch-T levels in AG individuals with normal WBC counts.

In older piglets with the AA and GG genotypes and normal WBC counts, CRP and Ch-T levels were negatively correlated (p values close to the significance limit). Such a relationship was also observed in AG piglets with elevated WBC counts. A tendency towards a negative correlation between CRP and HDL-ch concentrations was also noted in the AG genotype, regardless of leukocyte counts.

Discussion

Our results regarding serum CRP levels in piglets partially confirmed the findings of Bürger et al. (1992). Considerable variations in CRP levels were noted (Tables 1 and 2). Variations in blood CRP levels, including in human subjects, were found to be heritable in 35 – 40% (Hage and Szalai, 2007). CRP gene expression is also modified by environmental factors. The reference ranges for the baseline serum concentrations of CRP in pigs are 3.6 to 183 mg/l (Diack et al., 2011).

The serum concentrations of Ch-T and HDL-ch determined in piglets in the present study are similar to the values noted by Gallardo et al. (2008) and lower than those reported by Poracova et al. (2011).

The relationship between CRP gene polymorphism in the 3'UTR region (position 1271 G/A) and CRP gene expression, observed in piglets with normal WBC counts, validates findings from an earlier study of sows (Łaszyn and Życzko, 2010). The current results correspond to those obtained in human subjects. According to many authors, mutations in the 3'UTR region of the CRP gene considerably affect CRP levels in humans (Marsik et al., 2006; Hage and Szalai, 2007, 2009; Shen and Ordovas, 2009). The 3'UTR is disproportionately long in the CRP gene, which could indicate regulatory role, such as through influence on mRNA stability (Suk Danik and Ridker, 2007).

The relationship between CRP gene polymorphism and expression, noted in piglets with normal WBC counts, did not manifest itself in individuals with elevated WBC counts (resulting presumably from infection or inflammation). The above results are partially consistent with those reported by Marsik et al. (2006) who demonstrated that CRP gene mutation (+1444 C/T) in the 3'UTR region affects the serum levels of CRP, interleukin 6 (IL-6) and tumor necrosis factor (TNF). In a group of healthy young men, baseline CRP concentrations were 64% higher and IL-6 levels were two-fold lower in the TT genotype than in the CC genotype. Blood TNF levels were similar in TT and CC individuals. Within 24 hours after LPS infusion inducing inflammation, both genotypes demonstrated comparable CRP concentra-

tions, but IL-6 and TNF levels were significantly higher in CC individuals than in TT individuals. According to the cited authors, the CC genotype exerts protective anti-inflammatory effects in human endotoxemia. Shen and Ordovas (2009) reported that also responses to fenofibrate (a lipid lowering drug) treatment are dependent on CRP gene polymorphism. Genotypes characterized by a high baseline inflammatory status are more resistant to the above drug.

In the present experiment, an analysis of each genotype with respect to WBC counts yielded different results in younger and older piglets. In the group of younger animals, AG (with normal and elevated WBC counts) and GG piglets differed only in cholesterol levels. In the group of older piglets, WBC counts affected CRP, Ch-T and HDL-ch concentrations in AA and AG individuals, while none of the above parameters was influenced by WBC counts in GG piglets. It seems that the absence of differences between genotypes (Table 1) was related to the piglets' age. Poracova et al. (2011) observed dynamic changes in lipid concentrations in piglets aged 21 to 28 days; Ch-T and HDL-ch levels decreased, and triglyceride and LDL-ch concentrations increased. In older AA and AG piglets, differences in the values of the studied indicators correlated with WBC counts could have resulted from the response of the above genotypes to inflammation or infection. Blood cholesterol levels decrease during inflammation and infection, and the drop is stimulated by TNF and IL-2. IL-6 enhances cholesterol synthesis, but it also reduces its secretion. HDL-ch metabolism is induced by TNF-α and IL-1 via triggering endothelial lipase overexpression. In chronic conditions, endothelial lipase levels are positively correlated with CRP and IL-6 concentrations (Khovidhunkit et al., 2004; Lamarche and Paradis, 2007; Feingold and Grunfeld, 2010). Verma et al. (2002) noted a positive feedback between CRP and IL-6 which involved CRP synthesis induced by IL-6, mostly in hepatocytes. Increased CRP secretion induces IL-6 release in monocytes and epithelial cells, thus enhancing its proinflammatory effects.

Our results provide new insights into the previously unknown effects of CRP gene polymorphism in pigs, which may be modified by the expression of CRP-linked and CRP-unlinked genes. Chomdej et al. (2004) found the CRP gene locus between markers S0001 (proximal) and S0214 (distal) on SSC4.q13. Uddin et al. (2011) detected QTLs for the HDL/LDL ratio in the above region. FABP5 (fatty acid binding protein), which plays a key role in regulating lipid metabolism, was also identified in this region. Berg et al. (2006) detected a QTL for fatness and growth, denoted FAT1 (between microsatellites S0001 and SW217), orthologous to a region in the human genome located on chromosome 1q23.3 containing gene loci associated with type 2 diabetes whose strong predictor is CRP.

There was a relationship between CRP gene (1271 G/A, 3'UTR) polymorphism and variations in the serum levels of CRP in piglets with normal WBC counts. The above relationship did not manifest itself in piglets with elevated WBC counts. The studied genotypes differed in their response to elevated WBC counts. The response of genotypes with weak CRP expression involved an increase in CRP levels and a decrease in the serum concentrations of Ch-T and HDL-ch. Such a response was not observed in the genotype with strong CRP expression.

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Związek polimorfizmu genu CRP z koncentracją kodowanego białka C-reaktywnego, cholesterolu całkowitego i HDL-ch w surowicy krwi ssących prosiąt

STRESZCZENIE

Analizowano związek między polimorfizmem genu CRP (poz. 1271 G/A, 3'UTR) a zawartością białka C-reaktywnego (CRP), cholesterolu całkowitego (Ch-T) i HDL-ch w surowicy krwi ssących prosiąt ras [Wielka Biała Polska × Polska Biała Zwisłoucha (♀) × Duroc × Pietrain (♂)]. Genotypy CRP identyfikowano metodą PCR-RFLP używając enzymu restrykcyjnego HinfI. Oznaczono poziom CRP, Ch-T i HDL-ch oraz liczbę białych krwinek (wbc) w krwi. Prosięta podzielono na młodsze (21±3 dni życia) i starsze (35±3 dni życia). Stwierdzono wystąpienie związku między polimorfizmem genu CRP (poz. 1271 G/A, 3'UTR) i zróżnicowaniem poziomu kodowanego białka w surowicy krwi prosiąt z liczbą wbc w normie fizjologicznej. Związek ten był maskowany u prosiąt z podwyższoną liczbą wbc. W odpowiedzi na podwyższoną liczbę wbc ujawniły się różnice między genotypami. Objawiły się one wyraźniej u starszych niż u młodszych prosiąt. Genotypy u których ekspresja CRP była słabo wyrażona reagowały podwyższeniem poziomu CRP i obniżeniem poziomu Ch-T i HDL-ch. U genotypu o silnie wyrażonej ekspresji CRP nie stwierdzono takiej reakcji.