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Association between the human immune response and body mass index

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ABSTRACT

The aim of this study was to determine the strength of the association between the human immune response and body mass index (BMI) and whether differences exist in the effects of obesity on selected immune parameters between men and women. Two hundred ninety participants were divided into groups according to sex and BMI. Parameters CD3, CD4, CD8, CD16+56, CD19, HLADR, CD11b, CD11c, and CD54 were quantified. Leukocyte and differential counts were performed. We observed elevation with regard to the normal weight group in the parameters of white blood cells, neutrophils, monocytes, CD3, CD4, CD19, and CD11b for the whole study group. A decrease was observed in the expression of CD16+56. The effect of BMI on the immune system was much more apparent in women. BMI was correlated with the majority of the measured parameters, reflecting a strong association between BMI and the human immune system.

1. Introduction

Obesity is the result of a chronic imbalance between greater energy intake and lesser expenditure. Obesity is associated with many pathologies, including type 2 diabetes, cardiovascular diseases, insulin resistance, inflammation, and tumors [1]. Nowadays, it is well known that adipose (fat) tissue, metabolism, and the immune system cooperate closely. Substantial evidence indicates that obesity is associated with inflammation, including changes in peripheral blood lymphocyte subpopulations, elevated inflammatory cytokine production, acute-phase proteins, and activation of inflammatory signaling pathways [2,3]. Systemic inflammation represents a common underlying factor in the pathogenesis of serious obesity-related metabolic disorders and related complications [4]. The adipose tissue is a primary site for initiating inflammation, although other metabolically active tissues are also involved, mainly the liver [1]. Changes in fat metabolism, such as progressive fat tissue infiltration by lymphocytes and macrophages, insulin resistance development and glucose intolerance, and secretion of proinflammatory cytokines, adipocytokines, and chemotactic factors, may lead to impaired homeostasis and blood reversal. Thereafter, changes in the level of circulating factors influence other organs such as the liver, blood vessels, heart, and pancreas. Evidence indicates that obesity is linked to the potential development of asthma and atopic and autoimmune diseases, caused by decreased immunological tolerance to antigens [5]. However, the risk of development of obesity and further related chronic diseases is highly individual. In some subjects, a moderate adiposity increase may dramatically elevate cardiovascular and metabolic risk, whereas others may not experience any complications with an even more severe degree of adiposity [6]. Prevalence of overweight and obesity closely depends on sex and age; accordingly, their combination with other diseases, such as hyperglycemia and hypertension, is significantly higher in the female population and in older cohorts [7]. A variable effect of weight on the different sexes is apparent from a young age [5]. The purpose of this study was to determine whether differences exist in the effects of obesity on selected immune parameters between men and women.

2. Subjects and methods

2.1. Subjects

With the aim of gaining participants for the study, we posted advertisements in frequented public locations. Criteria for inclusion included providing informed consent, age between 40 and 45 years, no blindness or hearing disability, no acute physical illness or unstable physical condition, and consumption of >50 units of alcohol/week. Subjects were recruited via health practitioners in accordance with ethical committee requirements and completed a detailed nutritional and lifestyle questionnaire and a battery of psychological tests. The study included a total of 290 individuals in different stages of obesity. The subjects were divided into 5 groups according to their body mass index (BMI), a measure of obesity (Table 1). The age mean was 42.3 ± 0.2 years. The group with normal BMI was considered a control group. All groups outside the

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Table 1
Characteristics of the study participants

	BMI range (kg/m^2)	Women	Men	All
Normal weight	19–24.99	73	24	97
Overweight	25–29.99	49	46	95
Obese	30–39.99	54	26	80
Extremely obese	>40	10	4	14

normal BMI range were compared with this normal weight group and between themselves. Underweight individuals were excluded from this comparison as a group because the number of participants was too small (only 4), but they were included in the whole study group for correlations with BMI. Table 2 summarizes the differences between groups with regard to smoking habits, disease status, and medication. All individuals were informed about the aim and procedures of the study and gave their consent. Early-morning fasted blood samples were taken using EDTA vacutainer tubes. Samples were processed a maximum of 2 hours after collection.

2.2. Flow cytometry analysis

The human leukocyte differentiation antigens CD3, CD4, CD8, $CD16^+56$, CD19, HLADR (Beckman Coulter, Marseille, France), CD11b, CD11c, and CD54 (Becton Dickinson Biosciences, San Jose, CA) and negative control IgG1/IgG2a were quantified with monoclonal antibodies conjugated to fluorescein isothiocyanate, phycoerythrin, phycoerythrin–cyanine 5, and phycoerythrin–cyanine 7. Five microliters of appropriate monoclonal antibody was added to 50 μL of a whole-blood sample and incubated for 15 minutes at room temperature. Thereafter, the erythrocytes were lysed with 125 μL of a lysing solution, OptiLyse C, for 10 minutes. The reaction was stopped by the addition of 250 μL phosphate-buffered saline. The samples were analyzed by flow cytometry using Cytomics FC 500 and CXP software (Beckman Coulter).The leukocyte subsets were defined by forward- and side-scatter pattern. The negative control value was determined by a fluorescence background and antibody-nonspecific staining.

2.3. Analysis of peripheral blood cells

The analysis of peripheral blood cells (*e.g.*, total and differential count) was performed on a Beckman Coulter AcT 5diff hematology analyzer. The values are expressed in percentages and absolute numbers.

Table 2
Disease status of participants and their medications at the time of study

	Normal (%)	Overweight (%)	Obese (%)	Extremely obese (%)
Smoking	19.6	24.2	26.3	28.6
Any cardiovascular disease	2.1	1.1	8.8	14.3
Hypertension	6.2	4.2	18.8	35.7
Diabetes	0	1	1.3	7.1
Elevated cholesterol	2.1	4.2	2.5	0
Allergy	24.7	27.4	31.3	14.3
Asthma	3.1	4.2	6.3	0
Bronchitis	0	1.1	3.8	7.1
Pollinosis	10.3	15.8	15	14.3
Analgesics	9.3	9.5	12.5	7.1
Antibiotics	15.5	11.6	8.7	7.1
Sulfa drugs	0	0	0	7.1
Antirheumatics	2.1	7.4	2.5	14.3
Antidiabetics	0	0	1.3	7.1
Antihistamines	2.1	7.4	11.3	7.1

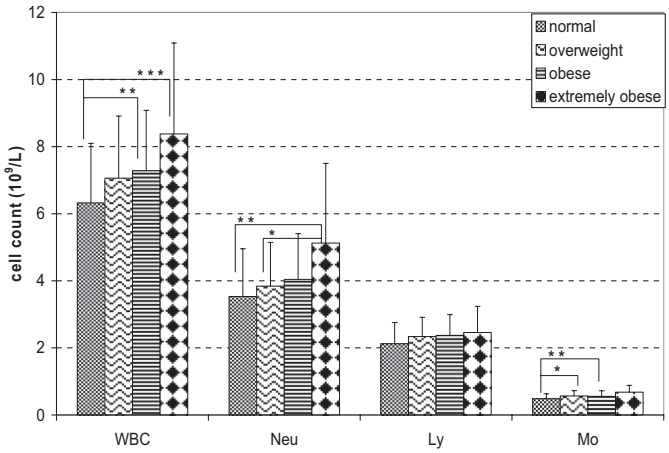

Fig. 1. Comparison of leukocyte count between BMI groups in peripheral blood. $^*p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$, cell count 10^9/L. WBC, white blood cells; Neu, neutrophils, Ly, lymphocytes; Mo, monocytes. For WBC, Neu, and Ly we used 1-way analysis of variance (ANOVA) and Bonferroni's test; for Mo we used 1-way ANOVA and Tamhane's test. Data are presented as means ± SD.

2.4. Statistical analysis

The statistical analysis was performed to assess differences between BMI groups. Normality was verified using the Kolmogorov–Smirnov test. To compare BMI groups with regard to continuous variables, 1-way analysis of variance was used. Multiple comparisons were followed by Bonferroni's or Tamhane's post hoc test. The Kruskal–Wallis test was used for comparing nonnormally distributed data. The relationship between continuous variables and BMI was assessed by Pearson or Spearman rank correlation. All data were expressed as the mean ± SD. $p < 0.05$ indicated statistical significance.

3. Results

3.1. Immune cell counts

The number of white blood cells was significantly elevated in obese (7.3×10^9/L, $p < 0.01$) and extremely obese (8.4×10^9/L, $p < 0.001$) individuals when compared with normal controls ($6.3 \times 10^9/\mu$L; Fig. 1). This elevation was caused by an elevation of the total neutrophil count (extremely obese [5.1×10^9/L] versus normal controls [3.5×10^9/L, $p < 0.01$] and versus overweight individuals [3.8×10^9/L, $p < 0.05$]) and monocytes (normal control [0.49×10^9/L] versus overweight [0.56×10^9/L, $p < 0.01$] and obese individuals [0.55×10^9/L, $p < 0.05$]). In the obese group the total number of lymphocytes was higher, but not statistically significantly. The group of all participants exhibited a positive correlation between BMI and leukocyte subpopulations, namely lymphocytes, monocytes, neutrophils, eosinophils, and basophils (Table 3). Women demonstrated a positive correlation of BMI with all parameters of differential count, whereas the BMI of men did not correlate with lymphocyte, monocyte, and eosinophil counts. By contrast, men's BMI exhibited a closer relationship with basophil count. The cell counts variances were more apparent in female than in male groups.

3.2. Phenotype analysis of peripheral blood—CD3, CD4, CD8, CD19, HLA DR, and $CD3^-/CD16^+/CD56^+$

Our analysis demonstrated that an abundance of CD3, CD4, and CD19 lymphocyte subpopulations in the peripheral blood of the whole study population increased with BMI (Fig. 2, Table 3). All monitored subpopulations in all groups were still within the normal range. However, we observed statistical differences between normal and obese women ($p < 0.05$) for CD3 and CD4 total counts.

Table 3
Comparison of the results of linear regression analysis with body mass index

	Women (*r*)	Men (*r*)	All (*r*)
White blood cell count	0.326**	0.260**	0.290**
Neutrophil count	0.257**	0.255*	0.226**
Lymphocyte count	0.215**	NS	0.195**
Monocyte count	0.268**	NS	0.235**
Eosinophil count	0.179*	NS	0.164**
Basophil count	0.181*	0.232*	0.205**
CD3 count	0.231**	NS	0.177**
CD4 count	0.236**	NS	0.214**
CD4:CD8 ratio	0.184*	NS	NS
$CD3^-CD16^+56$ (%)	−0.233**	NS	−0.167**
CD19 (%)	0.272**	NS	0.211**
CD19 count	0.378**	NS	0.279**
CD11b lymphocyte count	NS	NS	0.131*
CD11b monocyte count	0.358**	NS	0.238**
CD11b granulocytes (%)	NS	NS	0.148*
CD11c monocyte count	0.179*	NS	0.151*
CD54 lymphocytes (%)	−0.178*	NS	−0.162**

Significance was calculated by Spearman or Pearson correlation (2-tailed).
*$p < 0.05$, **$p < 0.01$. NS, not significant; *r*, correlation coefficient.

The change in $CD8^+$ lymphocyte count was not significant (Table 4). Similarly, associations of CD3 and CD4 number with BMI were significant ($p < 0.01$, Table 3). Although we determined that $CD3^+$ and $CD4^+$ cells numbers were elevated, they did not indicate an activation status—the number of $CD3^+HLA\text{-}DR^+$ cells did not change (Table 4) and did not correlate with BMI. The percentage of expression of CD3, CD4, CD8, and HLA-DR antigens and the ratio of CD4:CD8 did not change between groups for both sexes. We did not detect any significant associations between the percentage of expression and BMI other than the CD4:CD8 ratio ($p < 0.05$) of women (Table 3). For the female group the total count of CD19 expression (Fig. 2, Table 4), a marker of B lymphocytes, exhibited the most significant changes in the overweight group ($p < 0.01$) and the obese group ($p < 0.01$) when compared with the control group with normal BMI. Regarding the expression of CD19 expressed as a percentage, the results were the same but at a lower level of significance ($p < 0.05$). CD19 expression positively correlated with BMI (total count and percentage, $p < 0.01$; Fig. 3). The percentage of expression of natural killer (NK) cell marker $CD3^-/CD16^+/CD56^+$ decreased with increased BMI. A clear tendency for decline in the expression of this marker was visible in the overweight group. A statistically lower expression could be seen in the obese group (10.9%, $p < 0.05$) when compared with the normal control group (13.3%). This trend remained in extremely obese participants (normal 13.3% > overweight 11.3% > obese 10.9% [$p < 0.05$] > extremely obese 10.7%). In addition, the expression of $CD3^-/CD16^+/CD56^+$ negatively correlated with BMI ($r = -0.167$, $p < 0.01$) and with C-reactive protein, an inflammation marker (data not shown; $r = -0.135$, $p < 0.05$). When using male/female discrimination, these associations were even stronger (BMI $r = -0.233$, $p < 0.01$; C-reactive protein $r = -0.177$, $p < 0.05$) with regard to the female population (Fig. 4).

3.3. Expression of adhesion molecules

The expression of 3 types of adhesion molecules, CD11b, CD11c, and CD54, on lymphocytes, monocytes, and granulocytes was evaluated. On granulocytes, none of these molecules exhibited altered expression when comparing the different BMI groups. Although expression was unchanged, there was a positive correlation between granulocyte CD11b (%) and BMI in the entire study population ($r = 0.148$, $p < 0.05$). Concerning monocytes, the population of women exhibited an increase in the total amount of CD11b-positive cells in the overweight, obese, and extremely obese groups (Table 4) and an association with BMI ($p < 0.01$; Fig. 5). Female serum levels of interleukin-6, which stimulates adipocyte expression of monocyte chemoattractant protein-1, correlated with BMI as well ($r = 0.401$, $p < 0.01$, data not shown). We denote the impact of obesity on female blood monocyte CD11c expression as a correlation of the total number CD11c-positive monocytes with BMI ($p < 0.05$). There was an increasing trend without statistical differences between groups (Table 3). We did not observe either a statistically positive association with BMI or a significantly increased expression of CD11b and CD11c molecules on circulating monocytes in men (Table 4). Lymphocytes exhibited a negative association of the relative amount of their $CD54^+$ marker in the group of women (Table 3, $p < 0.05$). The trend of a negative association with BMI remained in the men, but was not significant.

4. Discussion

Obesity is associated with the modulation of immune parameters. Weight reduction has a profound effect on many of these variables [8]. Several authors have reported a chronic inflammation status in individuals with higher BMI [9–11]. Our parameters affirmed a proinflammatory tendency of obesity by elevated amounts of white blood cells, neutrophils, and monocytes in the blood of all participants with BMI higher than that of the control group. Despite this elevation, total counts were still in the normal range but more or less correlated with BMI. The macrophage infiltration of white adipose tissue is an important feature of low-grade inflammation as part of obesity. BMI is positively correlated with the number of macrophages in adipose tissue [12]. Our findings indicated a sex-dependent association between BMI and cell subpopulation counts in peripheral blood. The cell count variances were more apparent in female than in male groups. The BMI of women correlated with all leukocyte subpopulations; the BMI of men correlated with neutrophil and basophil count only. In the whole tested population we denote an increase of eosinophil and basophil counts with elevated BMI. This increase can be a risk factor for atopy, development of allergic reactions, and asthma symptoms or bronchial hyperreactivity. Recent studies have reported that BMI is an independent risk factor for allergy in teenage girls, but not in boys [6]. A relationship exists between obesity and asthma, but the way in which obesity affects the lungs has not yet been determined. A positive relationship between the percentage of neutrophils and eosinophils in venous blood and BMI may indicate an increased risk for development of airway inflammation in obese individuals. Scott et al. [13] described a positive association of the percentage of

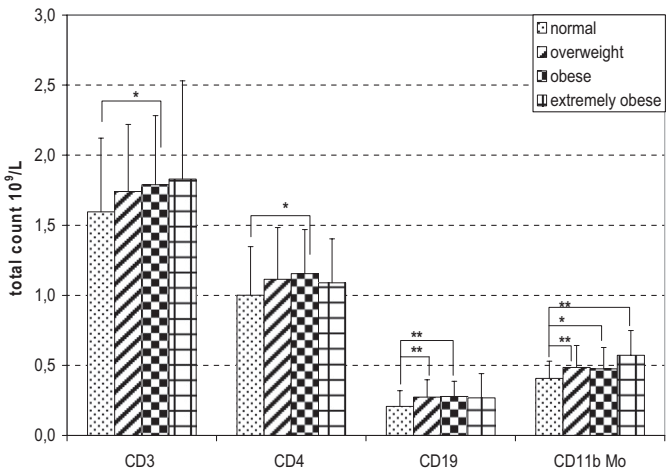

Fig. 2. Total count of CD3-, CD4-, and CD19-positive lymphocytes and CD11b-positive monocytes in peripheral blood for the whole study population. Statistical analysis was performed using 1-way analysis of variance and Bonferroni's test or Kruskal–Wallis test, *$p < 0.05$, **$p < 0.01$. Data are presented as means ± SD.

Table 4
Changes in lymphocyte subpopulation count depending on body mass index

	N	Female (10^9/L)	SD	*N*	Male (10^9/L)	SD	*N*	Together (10^9/L)	SD
CD3 count									
Normal	73	1.54	0.44	24	1.77	0.71	97	1.60	0.53
Overweight	49	1.71	0.48	46	1.78	0.47	95	1.74	0.48
Obese	53	1.76*	0.49	26	1.84	0.50	79	1.79*	0.49
Extremely obese	10	1.94	0.80	4	1.56	0.30	14	1.83	0.70
Normal range		0.77–2.20			0.59–1.99				
$CD3^+CD4^+$ count									
Normal	73	0.98	0.31	24	1.06	0.43	97	1.00	0.35
Overweight	49	1.11	0.36	46	1.12	0.39	95	1.11	0.37
Obese	53	1.16*	0.31	26	1.13	0.33	79	1.15*	0.32
Extremely obese	10	1.15	0.34	4	0.93	0.17	14	1.09	0.31
Normal range		0.39–1.34			0.39–1.34				
$CD3^+CD8^+$ count									
Normal	73	0.53	0.20	24	0.68	0.36	97	0.57	0.26
Overweight	49	0.57	0.24	46	0.64	0.22	95	0.60	0.23
Obese	53	0.55	0.24	26	0.68	0.24	79	0.60	0.25
Extremely obese	10	0.66	0.37	4	0.63	0.25	14	0.65	0.33
Normal range		0.50–0.99			0.50–0.99				
$CD3^+$HLA-DR count									
Normal	73	0.08	0.06	24	0.08	0.05	97	0.08	0.06
Overweight	49	0.07	0.03	46	0.08	0.03	95	0.07	0.03
Obese	53	0.08	0.04	26	0.08	0.04	79	0.08	0.04
Extremely obese	10	0.09	0.06	4	0.07	0.02	14	0.08	0.05
Normal range		0.04–0.38			0.04–0.38				
CD19 count									
Normal	73	0.19	0.08	24	0.25	0.18	97	0.21	0.11
Overweight	49	0.27**	0.12	46	0.27	0.13	95	0.27**	0.12
Obese	53	0.28**	0.11	26	0.26	0.11	79	0.28**	0.11
Extremely obese	10	0.29	0.20	4	0.20	0.07	14	0.27	0.17
Normal range		0.10–0.53			0.10–0.53				
CD11b Mo count									
Normal	66	0.39	0.10	22	0.45	0.16	88	0.41	0.12
Overweight	47	0.48**	0.14	39	0.49	0.17	86	0.48**	0.16
Obese	47	0.48*	0.15	26	0.46	0.16	73	0.47*	0.15
Extremely obese	9	0.59**	0.16	3	0.50	0.24	12	0.57**	0.18
Normal range		0.14–0.64			0.14–0.63				
CD11c Mo count									
Normal	66	0.16	0.13	22	0.20	0.19	88	0.17	0.15
Overweight	47	0.19	0.16	39	0.19	0.16	86	0.19	0.16
Obese	47	0.24	0.19	26	0.23	0.20	73	0.23	0.19
Extremely obese	9	0.22	0.17	3	0.31	0.36	12	0.24	0.22
Normal range		0.07–0.30			0.07–0.30				

Mo, monocytes; *N*, number of participants. Data are presented as means ± SD.
*$p < 0.05$, **$p < 0.01$, compared with the normal group.

sputum neutrophils with BMI in asthmatic females. Neutrophilic asthma was present in a greater proportion of obese compared with nonobese females. Increasing BMI leads to an increasing risk of asthma development for both men and women, but for women this also includes the risks of wheezing, allergic rhinoconjuctivitis, and atopic dermatitis [6,14].

Human visceral adipose tissue has a larger quantity of T lymphocytes per gram in obese subjects. This can be a result of T-cell proliferation and influx [15,4]. Proinflammatory T cells present in visceral adipose tissue may sustain local inflammatory cell activation before the appearance of macrophages [16]. We agree with the suggestion of Kintscher et al. [16] that adipose tissue infiltration of $CD3^+CD4^+$-positive cells increases with body weight because we observed an increased number of $CD3^+$ and $CD3^+CD4^+$ lymphocytes in the peripheral blood of obese women correlating with BMI. The correlation of CD19 with BMI for the whole study population implies stimulation of proliferation and survival of B cells in individuals with a higher BMI than normal, similar to what occurs in autoimmune diseases or infections. Karlsson et al. [17] in their review discussed possible influences of these parameters such as age, gender, number of participants, and comorbidities upon immunity. Our study included age-balanced subjects (40–45 years old), did not exclude persons with obesity-related diseases (which could also influence the immunity status), and included a large number of participants (besides the extremely obese group). Participants in our study exhibited a significant effect of sex on lymphocyte subpopulations and BMI. Although we observed an increase in CD3, CD4, and CD19 counts in the obese group of women, increasing BMI did not affect the lymphocyte subpopulation in men (neither statistical differences between groups nor a correlation with BMI existed).

NK cells, $CD3^-/CD16^+/CD56^+$, are first-line cells for the clearing of virus-infected and tumor cells from humans. Chronic inflammation induced by obesity can affect both tumor initiation and tumor progression [18]. Based on these facts, we can assume that a decreased amount of circulating NK cells and their correlation with enhanced inflammation marker (C-reactive protein) and BMI indicates a real risk for developing cancer and increased susceptibility to infections. Our findings are in line with the meta-analysis of Renehan et al. [19], who demonstrated an association of obesity with 25–40% of certain malignancies in both obese men and women. Our data suggest that women are at greater risk of developing cancer because this marker exhibited no association with BMI and C-reactive protein for the male population and differences

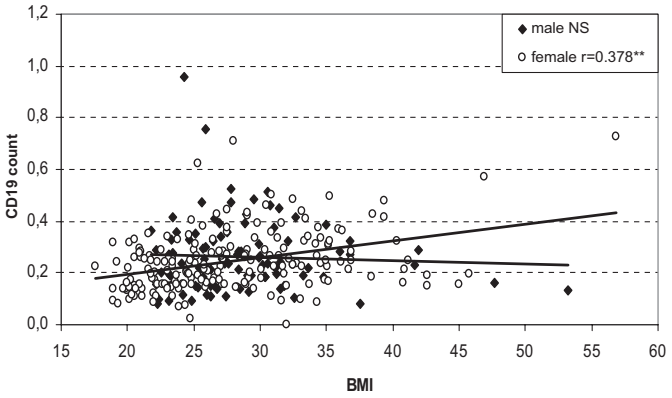

Fig. 3. Correlation of CD19 count with body mass index (BMI). Linear regression analysis of total CD19-positive lymphocyte count (10^9/L) from individuals with different BMI. Results were calculated by Pearson's correlation (2-tailed), $p < 0.01$. NS, not significant; *r*, correlation coefficient.

between groups were not significant. However, epidemiological data did not exclude obese men from susceptibility to cancer.

It is interesting that CD19 on B lymphocytes is the only parameter exhibiting an association with BMI, both as a total amount and as a percentage of expression, with the total amount correlating more closely. On the contrary, NK cell markers exhibited changes in the expression percentage but not as a count of positive cells. CD3 and CD4 correlated with BMI only as a total amount (Table 3), as well as with all parameters of blood count. Most parameters exhibited a correlation with BMI as a total amount.

Adhesion molecules are surface membrane glycoproteins, which enable cells to adhere to others cells. Changes in their expression contribute to the manifestation of a variety of diseases [20]. Because of the long-term low inflammation caused by obesity, we monitored the expression of some adhesion molecules on peripheral blood cells. Our finding of an association of granulocyte CD11b with BMI is in line with the results of Viardot et al. [21]. These authors reported that the expression of CD11b on granulocytes decreased with mean total weight loss in obese subjects with type 2 diabetes or prediabetes. Mastej and Adamiec [22] observed a positive correlation between neutrophil CD11b and BMI, as well as with leptin in the group with type 2 diabetes. Because of the increasing total neutrophil count within our BMI groups, a positive correlation of granulocyte CD11b with BMI and indication of activated endothelium (going by positive correlation of soluble E-selectin serum levels with BMI, for the whole study population, $r =$

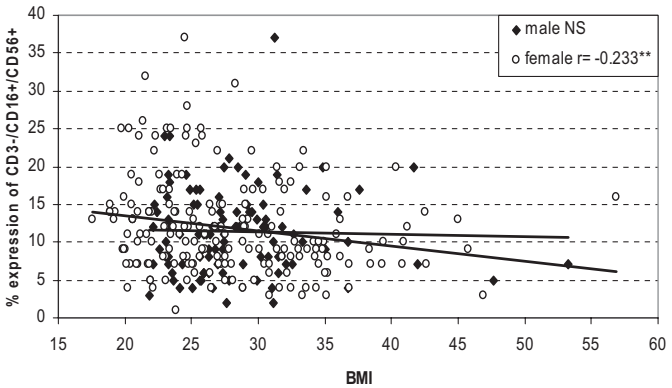

Fig. 4. Correlation of the expression of $CD3^-/CD16^+/CD56^+$ with body mass index (BMI). Linear regression analysis of the percentage of $CD3^-/CD16^+/CD56^+$ from individuals with different BMI. The regression results were calculated by Spearman's nonparametric correlation, 2-tailed, $p < 0.01$. NS, not significant; *r*, correlation coefficient.

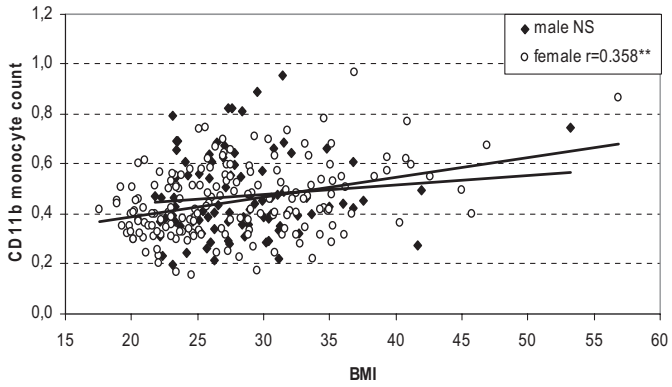

Fig. 5. Correlation of CD11b monocyte count with body mass index (BMI). Linear regression analysis of CD11b monocyte count with BMI from individuals with different BMI, $^{**}p < 0.01$. NS, not significant; *r*, correlation coefficient.

0,341, $p < 0.01$, data not shown), we can affirm the tendency of extravasation of neutrophils from blood vessels to tissue with increasing BMI. CD11b plays an important role in monocyte adhesion to the vascular wall [6]. CD11c has been used as an activation marker for monocytes/macrophages and plays an important role in T-cell accumulation and activation in adipose tissue [23,24]. CD11c also contributes to monocyte adhesion to inflamed endothelial cells [25]. Wu et al. [23] reported an increased amount of CD11c on blood monocytes in obese humans (1 male and 8 females, BMI 49.9) with metabolic syndrome and a reduced number of CD11c after diet-induced weight loss. We observed increased $CD11b^+$ monocyte counts in all 3 groups of women with higher BMI than normal. Along with the elevated levels of interleukin-6 (a stimulator of fat cell expression of monocyte chemoattractant protein-1) observed in our study population and increasing BMI, this can indicate better conditions for the transition of monocytes into adipocyte tissue. In conclusion, the activity of peripheral blood monocytes of women increases with increasing BMI. The positive correlation between BMI and cell adhesion molecule expression (CD11b, CD11c) on monocytes/granulocytes may point to the ongoing active low-inflammation mechanism in study participants. The response of lymphocytes to metabolic changes and activation of the immune system in our study is characterized by a negative correlation of the relative amount of $CD54^+$ lymphocytes with BMI, which can be seen in changes in immune response in the recognition and discernment of extraneous antigens and the altered cytotoxicity of effector cells. CD54 is a leukocyte adhesion molecule expressed on the top of T lymphocytes, among other places, and is necessary for transendothelial transport of lymphocytes from the blood stream to tissues. We suspect that decreased CD54 expression on lymphocytes with increasing BMI occurs because most lymphocytes that express CD54 might have already migrated into the adipose tissue. A mouse model indicated an early lymphocyte infiltration into tissue that was even more enhanced [17].

Our results indicated that the impact of BMI on the evaluated parameters is strongly sex dependent. The question is whether this dependency is because the group of men was about two thirds smaller or because the immune system of women is indeed more vulnerable to BMI changes and whether the risk of adipose tissue inflammation development with increasing BMI is greater for females. The number of men is a limitation of this study; however, other authors cited in this paper [6,14] also mention BMI as a sex-dependent factor with regard to immunity, especially allergic diseases.

5. Conclusion

The present study demonstrates the effect of obesity on the chosen parameters of immunity and differences of this effect in

men and women. We confirmed the proinflammatory status of obese individuals (elevated amounts of white blood cells, neutrophils, and monocytes with regard to the normal weight group). The increase was evident in the count of $CD3^+$ and $CD3^+CD4^+$ cells in the obese group, the count of $CD19^+$ lymphocytes in the overweight and obese groups, and the count of $CD11b^+$ monocytes in all 3 groups (*i.e.*, overweight, obese, and extremely obese) with regard to the entire study population. Only the $CD3^-/CD16^+/CD56^+$ count exhibited a decrease in expression, which was significant in the obese group. Our results indicate that the impact of BMI on evaluated parameters is apparent and strongly sex dependent. After discriminating between men and women, these changes were notable in the group of women but not in men. However, BMI correlated with the majority of measured parameters in the entire study group, which implies that obesity might seriously affect the regulation of the immune and inflammation response and, among other factors, may be responsible for decreased immunity tolerance, allergy and asthma development, and other immune-mediated diseases.

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