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Innate and adaptive immune parameters and adiposity in non-obese adults

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Abstract

Introduction
Excess body weight, mainly resulting from high body fat mass, is known to negatively affect immune system functioning due to the proinflammatory and prooxidative roles of adipose tissue. In general, most studies have documented the immunosuppressive role of adiposity by comparing selected immune markers between individuals of normal weight and those with obesity.

Study aims
This study investigates whether the amount of adipose tissue observed in overweight individuals may already negatively affect immune function.

Methods
We examined a broad panel of immunological parameters – both innate (WBC, neutrophil count, phagocytic uptake, respiratory burst, complement activity, lysozyme activity) and adaptive (total IgA, total IgG, CD3 count, CD19 count, the strength of antibody response to flu vaccination), in 85 women and 98 men aged 18–37 years, with a BMI between 18.5 and 29.99. As measures of adiposity, we used BMI on a continuous scale, BMI categorized as normal weight or overweight, and body fat mass percentage.

Results
In women, CD3 count was positively associated with continuous BMI ($p = 0.03$) and categorized BMI ($p = 0.006$). CD19 was positively associated with continuous BMI ($p = 0.03$), categorized BMI ($p = 0.01$), and fat mass percentage ($p = 0.02$). In men, categorized BMI was positively associated with phagocytic uptake ($p = 0.04$). However, none of these relationships would remain statistically significant after applying corrections for multiple comparisons.

Conclusion
Our results suggest that there is no relationship between adiposity and the analyzed immunity markers in non-obese (normal weight and overweight) adults.

Keywords: BMI, body fat percentage, waist to height ratio, innate immunity, adaptive immunity

Original article
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Introduction

Obesity, defined as excessive fat deposition that can impair health (WHO, 2006), is a known risk factor for various infectious diseases, including influenza (Karki et al., 2018; Louie et al., 2011; Maier et al., 2021), COVID-19 (Alberca et al., 2021; Stefan et al., 2020), cellulitis (Cheong, 2019; Taira et al., 2024), and urinary tract infections (Alhabeeb et al., 2021; Nseir et al., 2015; Semins et al., 2012). Obesity is also correlated with greater severity and/or worse outcomes of influenza (Fezeu et al., 2011; Martin et al., 2013; Ren et al. 2013; Ribeiro et al., 2015; Vaillant et al., 2009;) and COVID-19 (Arulanandam et al., 2023; Gao et al., 2020; Tamara & Tahapary, 2020; Poly et al., 2021; Yang et al., 2021a). It is additionally associated with more frequent surgical site infections (Koutsoumbelis et al., 2011; Mehta et al., 2012; Olsen et al., 2008; Waisbren et al., 2010), as well as to more frequent carriage of *Staphylococcus aureus* (Befus et al., 2015; Campbell et al., 2013, Erikstrup et al., 2019; Herwaldt et al., 2004; Olsen et al. 2013) which is one of the most common causes of such infections (Bucataru et al., 2023; Saadatian-Elahi et al., 2008; Zarb et al., 2012).

The relationship between adiposity and infection rate appears to be U-shaped – not only obesity, but also underweight, is associated with an increased risk compared to normal weight (Dobner & Kaser, 2018). In studies on adults, this pattern was observed in relation to the risk of hospitalization from influenza (Moser et al., 2019), the risk of influenza-related pneumonia (Phung et al., 2013), respiratory infections in general (Harpsøe et al., 2016; Yang et al., 2021b), the risk of septicemia (Yang et al., 2021b), and the risk of death from COVID-19 (Guglielmi et al., 2022; Wu et al., 2021).

Human defense mechanisms against pathogens are categorized into two groups: innate (specific) immunity, which serves as the body's initial line of defense, and adaptive (non-specific) immunity, which functions as the secondary line of defense (Wang et al., 2024). Among leukocytes (white blood cells, WBC), neutrophils are the predominant subtype, comprising 50–70% of leukocytes (Leliefeld et al., 2016). Neutrophils kill pathogens through phagocytosis (Boero et al., 2021). The killing of engulfed microbes can be performed by using antibacterial proteins or reactive oxygen species (Kolaczkowska & Kubes, 2013), the latter process being called respiratory burst (Dahlgren & Karlsson, 1999). While neutrophils are cellular components of innate immunity, humoral components include the complement system and lysozyme (Wang et al., 2024). The other subtype of WBC, lymphocytes, participate in adaptive immune mechanisms (Alberts et al., 2002). T lymphocytes drive the cellular immune response by reacting directly against foreign antigens, while B lymphocytes are involved in the humoral immune response by secreting immunoglobulins (Ig) (Alberts et al., 2002). For quantitative analysis of lymphocyte subsets, cell surface proteins termed cluster of differentiation (CD) are used; CD3 is a marker for T lymphocytes and CD19 is a marker for B lymphocytes (Hongbao et al., 2015).

Elevated leukocyte levels are crucial markers of inflammatory conditions, including obesity-related inflammation (Dixon & O'Brien, 2006). In adult studies, higher WBC counts were found in obese individuals relative to nonobese individuals (Al-Sufyani & Mahassni, 2011;

Ilavská et al., 2012; Kullo et al., 2002; Marzullo et al., 2013; Nieman et al., 1999; Womack et al., 2007), with the exception of two studies conducted on women, which found no difference (Mahassni, 2020; Mahassni & Bashanfar, 2019). Neutrophil count was also higher in obese than in non-obese subjects (Al-Sufyani & Mahassni, 2011; Ilavská et al., 2012; Marzullo et al., 2013; Nieman et al., 1999), with the exception of studies by Scully et al. (2017) and Kullo et al. (2002). In the study on women, Nieman et al. (1999) showed that obese women had higher levels of phagocytic uptake and respiratory burst compared with non-obese women, while in the study on both sexes, no difference in phagocytosis was observed (Scully et al., 2017). The complement system is considered to be one of the key factors contributing to inflammation and obesity-associated metabolic disorders (Ahmad & Al-Domi, 2017; Moreno-Navarrete & Fernández-Real, 2019). Studies on complement and adiposity yielded mixed results. Some studies observed that serum complement factors (i.e., C3 protein) rise in parallel with BMI ((Hernandez-Mijarez et al., 2007; Muscari et al., 1998; 2000), while others found that C3 levels were lower in obese patients than in non-obese ones (Gottschlich et al., 1993), or no differences in selected serum complement components were observed between lean, overweight, and obese individuals (Błogowski et al., 2013).

Lymphocytes also play an important role in obesity-associated inflammation (Chatzigeorgiou et al., 2012). In the study by Nieman et al. (1999), obese women had higher CD3 and CD19 levels than non-obese women. Ilavská et al. (2012) also observed a significant positive correlation between BMI and CD3 and CD19 in women but not in men. Tanaka et al. (1993) analyzed the proliferation of mitogen-induced B- and T-cells in obese subjects and non-obese controls. Proliferation was significantly lower in obese participants. Furthermore, T lymphocyte responses to mitogen increased in these individuals after weight reduction (in B lymphocytes increase was not statistically significant) (Tanaka et al., 1993). Reduced lymphocyte proliferation among obese women, relative to nonobese women, was also documented by Nieman et al. (1999). Total levels of immunoglobulins IgG (Gottschlich et al., 1993; Marzullo et al., 2013) and IgA (Marzullo et al., 2013) were compared between obese and non-obese subjects, with no difference found. Studies have demonstrated a reduced response to the hepatitis B vaccine (Averhoff et al., 1998; Fan et al., 2016; Liu et al., 2017; Young et al., 2013) and the rabies vaccine (Banga et al., 2014) in obese adults compared to non-obese individuals, whereas mixed results were found regarding the response to the influenza vaccination (Callahan et al., 2014; King et al., 2024; Neidich et al., 2017; Sheridan et al., 2012).

In most studies examining the relationship between adiposity and immunity, adiposity is measured using BMI. BMI is recommended as a costless, easy, and reliable tool to assess adiposity in young adults and middle-aged people (Adab et al., 2018; Kato et al., 1996; Mohajan & Mohajan 2023; NHLBI, 1998); however, it has some limitations. It does not measure the amount of body fat directly, as it is based not on fat tissue mass but on total body weight, which is also influenced by muscle mass. As a result, people with high muscle mass may be misclassified as overweight or even obese (Ode et al., 2007; Witt & Bush, 2005).

In the study by Alasagheirin et al. (2011), men with similar average BMI fell into a different category of adiposity based on fat mass percentage. Moreover, misclassification in both directions was observed – some participants were overclassified as obese, while others were underclassified as normal-weight. Additionally, while BMI serves as a measure of total body fat, it does not provide information about fat distribution. However, the biggest health risk was found to be associated with fat excess in the abdominal region, even in individuals classified as non-obese based on their BMI (Emery et al., 1993; Eyben et al., 2003; Lukács et al., 2019; Matsuzawa et al., 1995; Silva et al., 2009; Von Kawamoto et al., 2005). The waist-to-height ratio (WHtR) has been proposed as an indicator of abdominal obesity (Ashwell & Clarke, 2009; Ashwell & Lejeune, 1996; Hsieh & Yoshinaga, 1995).

Correlations between adiposity and immunological parameters are usually presented as comparisons between two groups: obese vs. non-obese (Gottschlich et al., 1993; Nieman et al., 1999; Tanaka et al., 1993) or obese vs. normal weight (Al-Sufyani & Mahassni, 2011; Ilavská et al., 2012; Scully et al., 2017; Sheridan et al., 2012; Young et al. 2013). Studies where BMI was analyzed on a continuous scale often included BMI values classified as obesity (Ilavská et al., 2012; Nieman et al., 1999; Sheridan et al., 2012; Quach et al., 2023). As a consequence, there is limited data on the correlation between adiposity and immunity within the non-obese group.

The aim of this paper is to analyze the relationship between adiposity and various parameters of innate and adaptive immunity in men and women with a BMI range of 18.5–29.99. In addition to BMI, body fat percentage and WHtR were included as measures of adiposity.

Materials and Methods

Participants

We recruited a total of 126 women aged 18.6–36.1 years and 134 men aged 18.9–36.7 years. The criteria for participation in the study included: willingness to receive an influenza vaccine, absence of chronic diseases or hormonal problems, and, for women, no hormonal contraception. On the day of the first visit, participants confirmed the absence of any symptom of an ongoing infection, and an additional current health assessment was performed by a physician to qualify participants for vaccination. When preparing the database for analyses, the following exclusion criteria were applied: incomplete immunological data, use of antibiotics (including between two visits), and elevated inflammation markers (WBC $> 10^3/\mu$l or/and CRP > 5 mg/l). From the remaining sample, we also excluded participants with a BMI value indicating underweight, i.e., <18.49 (eight women, four men), or obesity, i.e., ≥ 30.00 (one woman, two men). The final sample comprised 85 women aged 18.6–36.1 years (mean=25.2, SD=4.4) and 98 men aged 19.0–36.7 years (mean=27.4, SD=4.7).

Study procedure

The study protocol received approval from the Bioethics Commission of the Lower Silesian Chamber of Physicians and Dentists (2/PB/2013). All participants provided written informed consent.

The study included influenza vaccination; therefore, it was conducted during the "flu season" – that is, during the months of high influenza prevalence when influenza vaccination is recommended, i.e., from September to February

(with the last follow-up appointments in March), across two consecutive years.

For each participant, the study protocol included two visits with a 28-day interval. Blood sampling procedures necessitated scheduling both visits between 7:30 and 9:00 AM in a private healthcare facility. The first visit included a medical examination (to confirm eligibility for vaccination), blood sampling, body measurements, questionnaire completion, and administration of the influenza vaccine (Vaxigrip, Sanofi Pasteur).

The second visit included blood sample collection and completion of questionnaires.

On each day of the study, blood samples were transported within two hours of collection to a certified analytical laboratory (for morphological and biochemical tests), and to the university laboratory to immediately perform tests on whole blood. Additionally, serum was separated and and stored at -80°C for further analyses.

Adiposity measures

Adiposity was assessed using three indicators: BMI, body fat percentage (BF%), and WHtR. Height was measured to the nearest 0.1 cm with a Martin anthropometer, and weight to the nearest 0.01 kg with a Radwag® electronic scale. BMI values of 18.0–24.99 were classified as normal weight, and 25.0–29.99 as overweight. A bioimpedance analyzer (Bodycomp MF; Akern®) was used to measure body fat percentage (BF%) and calculated to 0.1 accuracy using the manufacturer's software (BodyGram 1.2; Akern®). WHtR was calculated as the ratio of waist circumference (cm) to body height (cm), with waist circumference measured with a flexible tape to an accuracy of 0.1 cm.

Immunological measures

The immunological parameters assessed included:

1) Innate immunity measurements: neutrophil count, phagocytic uptake, respiratory burst, complement activity, lysozyme activity;
2) Adaptive immunity measurements: total IgA level, total IgG level, CD3 (marker of T lymphocytes), CD19 (marker of B lymphocytes), strength of response to the influenzavaccine, and seroconversion after the vaccine.

Additionally, WBC was measured, comprising cells that participate in both innate and adaptive immune responses.

Postvaccination response strength was calculated as the fold rise in antiinfluenza IgG antibody titer from baseline (day of vaccination) to day 28. A fold increase of ≥ 4 was classified as positive (seroconversion), while a fold increase < 4 was classified as negative (lack of seroconversion) (Cauchemez et al., 2012). The immunological procedures were described in detail in a previous paper (Nowak et al., 2018).

Control variables

There is sufficient evidence that human immune functioning changes with age (Kumar & Burns, 2008; Shaw et al., 2014); for example, a decline in phagocytic uptake (Emanuelli et al., 1986; Wenisch et al., 2000) and in respiratory burst (Di Lorenzo, et al. 1999; Fülöp et al., 1985; Nagel et al., 1982) was observed (although the samples had a different age range than in this study – they included individuals >60 years old).

Since testosterone is considered to be an immunosuppressant (Furman et al., 2014; Trigunaite et al., 2015), free testosterone level (fT) was included as a potential confounding factor. Free testosterone was measured in serum using

a commercial ELISA kit (Demeditec®). Considering that our study was conducted over two consecutive flu seasons, we checked if there were differences in immunological measures between seasons. Taken together, we used age, fT, and season as potential cofounders.

Statistical analyses

Statistical analyses were performed in Statistica 13.3 (TIBCO Software Inc., USA). A significance threshold of $p < 0.05$ was set. Normality of data distribution was checked using the Shapiro–Wilk test. Because most variables did not show normal distribution, nonparametric tests were performed. A Mann–Whitney test was applied for group comparisons and a Spearman's rank correlation for associations. To account for potential confounders, we applied multiple regression analyses, treating immunity markers (excluding seroconversion) as dependent variables and adiposity measures as predictors. Variables lacking normal distribution were used in regression analyses transformed using the Box-Cox formula.

BMI was analyzed both as a continuous and categorical variable, with normal weight coded as 0 and overweight as 1. The strength of the post-vaccination response was used as both a continuous and categorical variable, with a distinction between an absence of seroconversion (coded as 0) and seroconversion (coded as 1). Among the control variables, study season was categorical: the first season was coded as 0 (47 women, 30 men) and the second was coded as 1 (38 women, 68 men).

Because seroconversion was a categorical variable, the Mann–Whitney test was used to examine its associations with continuous adiposity measures, and the chisquare test was applied to assess its relationship with categorized BMI.

Results

Table 1 presents descriptive statistics for adiposity measures, immune parameters, and control variables analyzed on a continuous scale. Men and women differed significantly in age, fT, BMI, BF%, WHtR, phagocytic uptake, respiratory burst, and CD19 (Mann-Whitney test: $p < 0.001$). Seventy-one women and 69 men had a BMI within the normal range, while 14 women and 29 men were overweight. Seroconversion was observed in 57 women and 76 men, and no seroconversion was found in 28 women and 22 men.

Table 1. Descriptive statistics of the studied variables and comparisons between women and men

	Women (N = 85)		Men (N = 98)		Mann-Whitney test	
	Mean (SD)	Min-max	Mean (SD)	Min-max	Z	p
Age [years]	25.25 (4.36)	18.64–36.10	27.36 (4.68)	18.97–36.72	–2.98	0.003
fT [pg/ml]	5.67 (8.20)	0.19–52.95	24.79 (11.01)	2.53–65.23	–10.72	< 0.001
BMI [kg/m^2]	22.39 (2.63)	18.55–28.94	23.79 (2.66)	18.71–29.88	–3.60	<0.001
BMI in normal weight group (N = 140)	21.52 (1.83)	18.55–24.93	22.40 (1.60)	18.71–24.90	–3.02	0.003
BMI in overweight group (N = 43)	26.81 (1.21)	25.00–28.94	27.09 (1.51)	25.05–29.88	–0.38	0.71

	Women (N = 85)		Men (N = 98)		Mann-Whitney test	
	Mean (SD)	Min-max	Mean (SD)	Min-max	Z	p
BF [%]	27.25 (5.83)	14.8–41.5	20.70 (4.77)	9.4–32.5	7.00	<0.001
WHtR	0.43 (0.04)	0.30–0.59	0.46 (0.04)	0.39–0.59	–5.28	<0.001
WBC [$10^3/\mu l$]	5.88 (1.46)	3.2–9.6	5.84 (1.40)	3.4–9.8	0.14	0.89
Neutrophil count [$10^3/\mu l$]	3.14 (1.12)	1.5–6.3	2.94 (1.00)	1.2–6.5	1.08	0.28
Phagocytic uptake[1]	237.6 (113.3)	63.3–583.7	174.4(64.9)	54.3–430.6	4.05	<0.001
Respiratory burst[2]	6.43 (4.2)	1.39–24.56	9.11 (9.10)	2.67–60.08	–3.66	0.001
Complement activity [ng/ml]	187506 (72014.7)	50082.2–341366.7	186901.7 (52583.1)	98462.2–287527.8	0.52	0.60
Lysozyme activity[3]	0.38 (0.08)	0.20–0.54	0.37 (0.08)	0.08–0.64	1.39	0.16
IgA [g/l]	1.79 (1.05)	0.55–7.01	1.82 (1.01)	0.57–7.34	–0.37	0.71
IgG [g/l]	11.20 (3.72)	4.23–23.41	11.99 (4.47)	4.16–26.95	–0.94	0.34
CD3 [cells/μl]	1472.62 (639.10)	412.08–4481.33	1476.17 (525.33)	538.13–3422.81	–0.34	0.74
CD19 [cells/μl]	195.9 (95.39)	39.5–459.4	237.78 (119.26)	45.33–639.93	–2.58	0.01
Strength of post-vaccination response [fold increase]	7.05 (7.21)	1–32	8.23 (9.66)	1–64	–0.99	0.32

[1] Mean fluorescence intensity measured in blood phagocytes following phagocytosis of fluorescently labelled bacteria.

[2] Mean area under the chemiluminescence curve (AUC) for the stimulated sample, expressed relative to the control AUC.

[3] The absorbance difference between control samples (bacterial suspension lacking lysozyme) and test samples (bacteria exposed to serum containing lysozyme).

All three measures of adiposity were correlated with each other (BMI and BF%: women: $r_s = 0.75$, $t_{(83)} = 10.44$, $p < 0.001$; men: $r_s = 0.50$, $t_{(96)} = 5.66$, $p < 0.001$; BMI and WHtR: women: $r_s = 0.72$, $t_{(83)} = 9.56$, $p < 0.001$; men: $r_s = 0.87$, $t_{(96)} = 16.96$, $p < 0.001$; WHtR and BF%: women: $r_s = 0.63$, $t_{(83)} = 7.39$, $p < 0.001$; men: $r_s = 0.59$, $t_{(96)} = 7.08$, $p < 0.001$).

Zero-order correlation tests showed no significant relationship between adiposity measures and immune parameters in women. Among the controlled factors, only fT was related to neutrophil count ($r_s = 0.25$, $t_{(83)} = 2.31$, $p = 0.02$), phagocytic uptake ($r_s = -0.21$, $t_{(83)} = -1.99$, $p = 0.0497$), and lysozyme activity ($r_s = 0.23$, $t_{(83)} = 2.15$, $p = 0.03$) (Table 2). In men, only post-vaccination response was negatively corelated to BMI ($r_s = -0.23$, $t_{(96)} = -2.28$, $p = 0.02$) and WHtR ($r_s = -0.21$, $t_{(96)} = -2.15$, $p = 0.03$). Free testosterone was positively associated with the strength of the post-vaccination response ($r_s = 0.31$, $t_{(96)} = 3.13$, $p = 0.002$), age was negatively

correlated with CD3 (r_s = -0.30, $t_{(96)}$ = -3.03, p = 0.003), CD19 (r_s = -0.22, $t_{(96)}$ = -2.19, p = 0.03), and the strength of the post-vaccination response (r_s = -0.30, $t_{(96)}$ = -3.06, p = 0.003) (Table 3).

Table 2. Correlation of immunological parameters with adiposity measures and continuous control variables in women – r_s values

	Adiposity measures			Controlled factors	
	BMI	BF%	WHtR	Age	fT
WBC	-0.04	-0.001	-0.002	0.11	0.20
Neutrophil count	0.05	0.06	0.11	0.18	**0.25***
Phagocytic uptake	0.003	-0.06	-0.02	-0.04	**-0.21***
Respiratory burst	0.005	-0.03	-0.13	-0.08	-0.18
Complement activity	-0.02	-0.02	-0.07	0.08	-0.08
Lysozyme activity	0.05	0.13	-0.03	-0.01	**0.23***
IgA	-0.09	-0.04	-0.01	0.22	0.04
IgG	-0.02	0.03	-0.13	-0.14	0.12
CD3	0.12	0.06	0.09	0.12	0.01
CD19	0.12	0.14	0.10	0.06	0.03
Strength of post-vaccination response	-0.18	-0.15	-0.14	-0.02	-0.02

* p < 0.05

Statistically significant relationships (α = 0.05) are bolded.

Table 3. Correlation of immunological parameters with adiposity measures and continuous control variables in men – r_s values

	Adiposity measures			Controlled factors	
	BMI	BF%	WHtR	Age	fT
WBC	-0.03	0.04	0.02	-0.19	0.12
Neutrophil count	-0.03	0.08	0.07	-0.10	0.09
Phagocytic uptake	0.20	0.03	0.16	-0.001	-0.14
Respiratory burst	0.02	-0.06	-0.08	-0.03	0.16
Complement activity	-0.08	0.04	-0.09	0.07	-0.03
Lysozyme activity	-0.16	-0.06	-0.10	-0.07	0.04
IgA	0.04	-0.05	0.06	0.18	0.0002
IgG	-0.05	-0.05	-0.13	-0.11	0.16
CD3	-0.06	0.04	-0.09	**-0.30****	-0.0002
CD19	0.08	-0.01	0.04	**-0.22***	-0.04
Strength of post-vaccination response	**-0.23***	0.03	**-0.21***	-0.30**	0.31**

* p < 0.05; ** p < 0.01

Statistically significant relationships (α = 0.05) are bolded.

A simple comparison between two BMI categories showed that overweight women had lower CD3 ($p = 0.03$) and CD19 ($p = 0.04$) levels compared to normal-weight women. Meanwhile, overweight men had lower phagocytic uptake ($p = 0.03$) and higher lysozyme activity ($p = 0.04$) compared to normal-weight men (Table 4). There was also a trend ($p = 0.06$) toward a weaker post-vaccination response in overweight men compared to normal-weight men.

Table 4. Results of Mann-Whitney's test with immunological parameters as dependent variables and categorized BMI (normal weight vs. overweight) as independent variable

	categorized BMI			
	Women		Men	
	Z	P	Z	P
WBC	-0.63	0.53	0.75	0.45
Neutrophil count	-1.10	0.27	0.74	0.46
Phagocytic uptake	-0.82	0.41	-2.20	**0.03***
Respiratory burst	0.67	0.50	0.32	0.75
Complement activity	0.34	0.74	0.67	0.50
Lysozyme activity	1.49	0.14	2.02	**0.04***
IgA	0.21	0.84	-0.85	0.40
IgG	-0.28	0.78	0.51	0.61
CD3	-2.14	**0.03***	-0.36	0.72
CD19	-2.08	**0.04***	-0.57	0.57
Strength of post-vaccination response	0.66	0.51	-1.88	0.06

*$p < 0.10$

Statistically significant results (= 0.05) are bolded.

In order to conduct multiple regression analyses, we assessed which of the potential cofounders should be included in the model. For each immune parameter, we conducted a series of three simple regression analyses, with age, fT, and season as independent variables. Variables found to be significant predictors were included in multiple regression models (Table 5 and Table 6).

The regression analyses in women showed that none of the innate immunity parameters, nor the antibody variables (IgA and IgG) or post-vaccination response were predicted by any of the morphological measures. There was only a positive trend for phagocytic uptake in relation to categorized BMI ($\beta = 0.18$, $t_{(81)} = 1.87$, $p = 0.06$), but not to continuous BMI ($\beta = 0.08$, $t_{(81)} = 0.82$, $p = 0.42$) (Table 5). CD3 was positively associated with BMI, both continuous ($\beta = 0.22$, $t_{(82)} = 2.16$, $p = 0.03$) and categorized ($\beta = 0.28$, $t_{(82)} = 2.83$, $p = 0.006$), but not with fat mass percentage ($\beta = 0.11$, $t_{(82)} = 1.09$, $p = 0.28$) or WHtR ($\beta = 0.13$, $t_{(82)} = 1.22$, $p = 0.23$). CD19 was positively associated with all quantitative measures of adiposity: continuous BMI ($\beta = 0.23$, $t_{(82)} = 2.24$, $p = 0.03$), categorized BMI ($\beta = 0.27$, $t_{(82)} = 2.63$, $p = 0.01$), and BF% ($\beta = 0.24$, $t_{(82)} = 2.30$, $p = 0.02$), but not to WHtR ($\beta = 0.16$, $t_{(82)} = 1.52$, $p = 0.13$).

In men, phagocytic uptake was significantly predicted only by categorized BMI in relation to phagocytic uptake ($\beta = 0.20$, $t_{(95)} = 2.06$, $p = 0.04$); there was also a positive trend for continuous BMI ($\beta = 0.18$, $t_{(95)} = 1.83$, $p = 0.07$) (Table 6). For lysozyme activity, there was a negative trend in relation to categorized BMI ($\beta = -0.18$, $t_{(95)} = -1.78$, $p = 0.08$) but not with continuous BMI ($\beta = -0.14$, $t_{(95)} = -1.42$, $p = 0.16$). There was also a positive trend for the relationship between CD19 and WHtR

(β = 0.20, $t_{(94)}$ = 1.87, p = 0.06), but not between CD19 and any other measure of adiposity (BMI continuous: β = 0.16, $t_{(94)}$ = 1.55, p = 0.13, BMI categorised: β = 0.11, $t_{(94)}$ = 1.10, p = 0.27, BF%: β = 0.06, $t_{(94)}$ = 0.59, p = 0.56).

Table 5. Beta-coefficients (with Standard Errors) of regression analyses in women (N = 85). BMI categorical means comparison between normal weight and overweight

	continuous BMI	categorized BMI	BF%	WHtR
WBC	-0.06 (0.11)	0.07 (0.11)	-0.04 (0.11)	0.03 (0.11)
Neutrophil count[1]	0.003 (0.11)	0.08 (0.11)	0.04 (0.11)	0.10 (0.11)
Phagocytic uptake[2]	0.08 (0.10)	0.18^ (0.10)	0.03 (0.10)	0.06 (0.10)
Respiratory burst[3]	0.08 (0.11)	-0.02 (0.11)	0.05 (0.11)	-0.08 (0.11)
Complement activity	-0.02 (0.11)	-0.03 (0.11)	-0.03 (0.11)	-0.08 (0.11)
Lysozyme activity	0.04 (0.11)	-0.13 (0.11)	0.13 (0.11)	-0.04 (0.11)
IgA	-0.09 (0.11)	-0.01 (0.11)	-0.05 (0.11)	-0.02 (0.11)
IgG	-0.02 (0.11)	0.03 (0.11)	0.05 (0.11)	-0.11 (0.11)
CD3[3]	**0.22*** **(0.10)**	**0.28**** **(0.10)**	0.11 (0.10)	0.13 (0.10)
CD19[3]	0.23* (0.10)	0.27* (0.10)	0.24* **(0.10)**	0.16 (0.10)
Strength of post-vaccination response	-0.16 (0.11)	-0.05 (0.11)	-0.12 (0.11)	-0.11 (0.11)

bold = p < 0.10; bold* p < 0.05; bold** p < 0.01

[1] controlled for fT; [2] controlled for season and fT; [3] controlled for season.

Statistically significant results (α = 0.05) are bolded.

Although the Spearman correlation analysis showed associations between the strength of the post-vaccination response with continuous BMI and WHtR, it did not remain significant after controlling for age, fT, and season (BMI continuous: β = -0.09, $t_{(92)}$ = -0.93, p = 0.36, WHtR: β = -0.06, $t_{(92)}$ = -0.56, p = 0.58), while the models including these confounders were significant (model with BMI continuous: adj R^2 = 0.15, $F_{(4,92)}$ = 5.09, p < 0.001, model with WHtR: adj R^2 = 0.14, $F_{(5,91)}$ = 4.92, p = 0.001).

The Mann-Whitney test showed no difference in continuous measures of adiposity between individuals with positive and negative responses to vaccination (i.e., with seroconversion and without seroconversion), in either women (BMI: Z = 1.44, p = 0.15, BF%: Z = 0.84,

p = 0.40, WHtR: Z = 0.98, p = 0.33) or men (BMI: Z = 1.72, p = 0.09, BF%: Z = -1.22, p = 0.22, WHtR: Z = 1.06, p = 0.29). The chi-square test also showed no statistically significant association between seroconversion and categorized BMI – seroconversion was not less frequent in overweight individuals (women: $\chi 2 = 0.06$, p = 0.81, men: $\chi 2 = 1.74$, p = 0.19).

Table 6. Beta-coefficients (with Standard Errors) of regression analyses in men (N = 98). BMI categorical means comparison between normal weight and overweight

	continuous BMI	categorized BMI	BF%	WHtR
WBC[1]	0.07 (0.11)	-0.01 (0.10)	0.07 (0.10)	0.16 (0.11)
Neutrophil count	-0.04 (0.10)	-0.09 (0.10)	0.05 (0.10)	0.03 (0.10)
Phagocytic uptake[2]	0.18^ (0.10)	**0.20*** **(0.10)**	0.05 (0.10)	0.16 (0.10)
Respiratory burst[2]	0.13 (0.09)	0.07 (0.09)	0.01 (0.09)	0.06 (0.10)
Complement activity[2]	-0.14 (0.10)	-0.13 (0.10)	0.01 (0.10)	-0.15 (0.10)
Lysozyme activity[2]	-0.14 (0.10)	-0.18^ (0.10)	-0.04 (0.10)	-0.08 (0.10)
IgA[3]	-0.07 (0.11)	-0.002 (0.10)	-0.05 (0.10)	-0.01 (0.11)
IgG[2]	-0.01 (0.09)	0.01 (0.09)	-0.01 (0.09)	-0.06 (0.09)
CD3[1]	0.09 (0.10)	0.14 (0.10)	0.12 (0.10)	0.11 (0.11)
CD19[1]	0.16 (0.10)	0.11 (0.10)	0.06 (0.10)	0.20^ (0.11)
Strength of post-vaccination response[4]	-0.09 (0.10)	-0.06 (0.10)	0.13 (0.10)	-0.06 (0.11)

bold: $p < 0.10$; bold* $p < 0.05$;

[1] controlled for age and season; [2] controlled for season; [3] controlled for age; [4] controlled for age, fT, and season

Statistically significant results ($\alpha = 0.05$) are bolded.

Discussion

None of the adiposity measures used in our study were related to white blood cell count or neutrophil count. The lack of difference in WBC and neutrophil count between normal weight and overweight individuals is consistent with the results obtained in the series of studies on Saudi women (Al-Sufyani & Mahassni, 2011; Mahassni, 2020; Mahassni & Bashanfar, 2019), in the study on men (Kullo et al., 2002), and in the study on both sexes combined (Ilavská et al., 2012). On the contrary, Womack et al. (2007) observed higher WBC in overweight women

compared to normal-weight ones. A positive correlation between continuous BMI and WBC was observed in some studies; however, the study sample also included much older participants than in our study – the age range was 45–64 years (Neto et al., 1992), 30–74 years (Schwartz & Weis, 1991), and 18–89 years (Panagotiakos et al., 2005).

A positive correlation between body fat percentage and both WBC and neutrophil count, after adjusting for sex, was observed in a study by Marzulo et al. (2013). Their sample consisted of a non-obese group and an obese group (categorized by BMI), the latter with a mean BF% of 45.1% (SD 6.8); thus, their data included higher values of BF% compared to the present study (see Table 1). It is possible that the correlation of WBC and neutrophil count with fat mass may be observed only over a wider range of body fat mass, when obese individuals are also included. In other words, an excess of body fat does not affect leukocyte count until it reaches a certain threshold, with values above this threshold being typical of obesity.

The absence of a correlation between WHtR and WBC aligns with findings by Mahassni and Bashanfar (2019), who likewise reported no differences in WBC across groups defined by low, moderate, and high waistcircumference–based healthrisk categories. In the study on women by Mahassni (2020), a significant difference in WBC was observed only between extreme categories of waist circumference, i.e., between low and high risk but not between low and moderate risk.

After controlling for confounders, phagocytic uptake of *E. coli* by neutrophils was positively associated with BMI both in men (continuous and categorized BMI) and in women (only categorized BMI). Meanwhile, lysozyme activity was negatively associated only with categorized BMI in men. The positive association found between BMI and phagocytic uptake aligns with the findings by Nieman (1999), who reported enhanced phagocytic uptake of *S. aureus* by granulocytes in obese women compared to their non-obese counterparts. These findings indicate that overweight and obese women both show differences from normal-weight women in this immune measure. Although Scully et al. (2017) found no difference in phagocytic uptake of *S. aureus* by neutrophils between non-obese healthy controls and obese subjects, it is worth noting that they compared only 12 controls (4 males) with 20 obese subjects without MetS (12 males), and 20 obese subjects with MetS (14 males) of different ethnicity.

We found no association between measures of adiposity and complement activity or respiratory burst. Our results contrast with general remarks that complement activity (measured as the levels of its components, C3 and C4) is positively associated with adiposity measures in adults (Gabrielsson et al., 2003; Nilsson et al., 2014; Qin et al., 2014), as well as in children (Cianflone et al., 2005) and adolescents (Wärnberg et al., 2006) , when comparing obese and lean individuals. However, to our knowledge, this is the first study in which total serum complement activity was compared between normal-weight and overweight individuals. In previous research examining complement C3 levels in different BMI categories, comparisons were made between non-obese versus obese individuals (Gottschlich et al., 1993).

No correlation between BMI and respiratory burst in peripheral blood mononuclear cells (i.e., phagocytes other than

neutrophils) was observed in the study by Pangrazzi et al. (2020), where the BMI range was 20.2–43.5. It needs to be mentioned, however, that the age of partcipants differed from our sample – the range was 31–89 years with a mean of 69.7 years (SD: 12.9). Contrary to the study by Panrazzi et al. (2020), Nieman et al. (1999), who compared middle-aged non-obese and obese women, found that the respiratory burst of granulocytes was significantly higher in obese women. Thus, as with WBC and neutrophil count, we can conclude that complement activity and respiratory burst are likewise altered mainly in obese subjects while remaining unaffected or only slightly affected in overweight subjects.

After adjusting for confounding variables, Tlymphocyte count (CD3) showed a positive association with BMI in women but not in men. B lymphocyte count (CD19) in women was positively correlated with BMI and BF% but not with WHtR. In men, there was a positive correlation with WHtR but not with BMI or BF%. The observed sex difference regarding the correlation of CD3 and CD19 with BMI is consistent with the study by Ilavská et al. (2012), where significant positive correlations were found in women (CD3: $r = 0.23$; CD19: $r = 0.38$) but not in men. It is worth noting that although both our study and the study by Ilavská et al. (2012) showed a linear correlation of BMI with CD3 and CD19 in women, BMI classified as underweight was not included in the analyses. Thus, it is possible that across a wider range of BMI, with values lower than those categorized as normal weight, the relationship would no longer be linear. This has been shown in some studies comparing individuals with a BMI outside the normal range (18.5–24.99), which indicated an increased susceptibility to infection in individuals with both higher and lower than normal weight (Harpsøe, et al. 2016; Moser et al., 2019; Phung et al., 2013; Yang et al., 2021b).

CD3 and CD19 levels were higher in overweight compared to normal-weight women. Differences in these parameters between normal weight and other BMI categories were also analyzed in Slovakian men and women aged 40–45 years (Ilavská et al., 2012), Pakistani men aged 18–29 years (CD19 only) (Alam et al. 2012), Saudi women aged 24–52 years (Mahassni, 2019), and Saudi women aged 17–26 years (Al-Sufyani & Mahassni, 2011). Ilavská et al. (2012) found a significant difference in CD3 in obese women but not in overweight women, while the difference in CD19 was significant in both overweight and obese women. No difference was observed in men. In the study on men, there was no difference in CD19 between normal weight and other BMI categories (underweight, overweight, obese) (Alam et al., 2012). In the study by Mahassni (2019), obese women had a significantly higher T lymphocyte count compared to normal-weight women, while overweight women demonstrated a positive trend ($p = 0.068$). For CD19, no difference was found. In the study on women by Al-Sufyani and Mahassni (2011), no difference in T lymphocyte count was observed between five BMI categories (from underweight to highly obese), while for B lymphocyte count, the differences were marginally significant ($p = 0.057$). Prechtl et al. (2023) found no association between total body fat and visceral fat with B lymphocyte count, but they observed a positive correlation with one subset of B lymphocytes (IgD^+ B cells).

Although the results are mixed, some of the findings (including ours) suggest that being overweight may lead to an increase in CD3 and CD19 levels.

No relationship was found between any measure of adiposity and total immunoglobulin levels, including IgA and IgG. This agrees with the results of Mahassni (2019) in women, where no difference was found in total IgG or total IgA levels between underweight, normal weight, overweight and obese groups, and between waist circumference ranges classified as low, moderate, and high health risk. In the study by Marzullo et al. (2013), neither IgA nor IgG differed between obese versus non-obese, and no correlation was found with BF%.

No measure of adiposity was related to influenza vaccination effectiveness, regardless of whether effectiveness was assessed via the fold increase in anti-influenza IgG antibody titers or by seroconversion status. Although simple correlations in men showed negative associations between BMI and WHtR and the strength of the postvaccination response, these relationships were not significant after adjusting for age, fT, and season. Our results agree with Neidich et al. (2017), where the risk of influenza and influenza-like illness among vaccinated subjects was significantly lower in normal-weight individuals compared to obese individuals, but not compared to overweight individuals. Moreover, there was no difference in seroconversion rates between four BMI categories (from underweight to obese). Callahan et al. (2014) found no difference in the rate of seroconversion between five categories of BMI (from underweight to morbid obesity) in the analysis of influenza vaccination effectiveness in adults. Thus, being overweight appears to not be a risk factor for the non-effectiveness of the influenza vaccine.

Considering the immunomodulatory role of adipose tissue (Grant & Dixit, 2015; Pond et al., 2005), we might expect a relationship between the analyzed immune parameters and body fat mass percentage rather than with BMI. Our results, however, showed otherwise. We did not find any correlation between immune parameters and body fat distribution (measured as WHtR), either. Existing evidence may suggest to use various measures of adiposity in future studies. For example, Yoshimura et al. (2015) observed no correlation of WBC or neutrophil count and body fat percentage; however, they found a positive correlation with the visceral fat thickness measured by ultrasonography. Prechtl et al. (2023) measured adiposity using BMI, total body fat mass percentage, visceral fat mass percentage (assessed by bioelectrical impedance), and WHtR. They obtained mixed results regarding correlations with B lymphocytes, depending on the cell subset and method of measurement (gated vs. ungated B lymphocytes).

Owing to the distinct anatomical and functional characteristics of visceral versus subcutaneous fat depots (Ibrahim 2010), the immunomodulatory effects of adipose tissue—especially in overweight individuals—would likely be mediated predominantly by visceral fat (VAT), a parameter not captured in our study. Some studies showed that waist circumference (an anthropometric measure of VAT amount) is the best measure of physiological disturbances associated with fat amount, including immunity changes (Mahassini, 2020).

Considering the evolutionary aspect and hypotheses related to the signal-

ing of biological condition in the mate market, our results do not suggest that overweight status in young and middle-aged adults affects the biological quality related to immunity. Our results may also support the Immunity Priority Hypothesis (Pawlowski et al., 2017). This is because, despite various factors affecting biological condition (and attractiveness – at least within the broad range of normality), an organism should always secure enough energy for immune function. It is, however, likely that being overweight may impair some immune parameters in older individuals compared to those studied here, as the organism typically has a lower biological condition and is not able to compensate for the physiological costs of being overweight. Furthermore, our study does not support the 'good genes' hypothesis, which postulates that physical attractiveness can be an honest signal of underlying biological quality in the studied age range. We should also remember that in the past, to secure energy reserves for pregnancy and lactation, selection may have favored a tendency toward excess weight in women (during the reproductive period of life), and therefore also may have driven the selection of adequate immune function within this BMI range.

It should be noted that among the relationships identified through regression analyses, only one—BMI categorized with CD3 in women—was significant at α 0.01. For the others, p -values ranged between 0.01 and 0.04. This means that after applying corrections for multiple comparisons, no relationship would remain significant.

In conclusion, our results imply that associations between adiposity and immunological parameters, which were observed in the studies using comparisons between obese and non-obese individuals, are weak or not present within a group of non-obese adults.

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None.

Contributions from individual authors

All authors participated in designing the study. BB: participants recruitment, anthropometric data collection, statistical analysis, writing of major parts of manuscript. NJ: hormonal and immunological data collection, critical revision and writing of minor parts of manuscript; PB: conception of the study, critical revision and writing of minor parts of manuscript.

Data availability statement

Data are available from the corresponding author upon request.

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Conflict of Interest

All authors have no competing interests to declare.

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