Original paper

# Prevention of obesity and carbohydrate metabolism disorders in the population at an industrial facility

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## Abstract

**Introduction:** One of the urgent tasks of modern healthcare is the development of measures aimed at preventing obesity in the able-bodied population, including in the Republic of Kazakhstan, which would reduce economic losses and increase the coefficient of life expectancy in the population.

**Aim:** To identify measures for the early prevention of excess weight, obesity, and carbohydrate metabolism disorders in the able-bodied population at an industrial facility in Kazakhstan.

**Material and methods:** The study involved 662 people (574 men and 88 women) aged 19 to 64 years. The subjects were divided into 2 groups: the main group – persons with obesity (242 people) and the control group – relatively healthy persons (422 people). The diagnosis of obesity was made based on the medical history of a patient, external examination, and physical, laboratory, and instrumental assessment.

**Results:** Three main factors influencing the development of obesity have been identified: a significant level of inactivity; serum glucose; the presence of comorbidities. Other factors (gender and age, smoking) do not have a significant effect on the increase in the body mass index of persons working at an industrial facility.

**Conclusions:** Accordingly, a priority in the prevention of obesity is a healthy lifestyle, involving regular physical activity and a healthy balanced diet.

## Introduction

Obesity is a complex, multifactorial, and largely preventable disease, which, along with excess weight, is now detected in more than one-third of the world’s population [1]. By 2030, 38% of the world’s adult population will be overweight, and another 20% will be obese [2]. The prevalence of obesity and obesity-related diseases is increasing worldwide [3, 4]. The body mass index (BMI) allows the assessment of the ratio of body weight and height of a person, and their compliance, and it allows prediction of the risk of obesity and diseases developing against this background. According to WHO estimates, there are more than 650 million obese people worldwide (BMI ≥ 30 kg/m$^2$). Over the past 3 decades, the prevalence of overweight and obesity in the world has increased by almost 30–50% among adults and children, respectively. According to World Health Organisation (WHO) data, 82 environmental and lifestyle factors contributing to the development of obesity have been identified. The presence of risk factors at any stage of the disease affects the rate of its progression and the severity of complications, and requires correction [5].

The prevalence of obesity in developing countries continues to increase to the level of the United States (US) [6]. Obesity significantly increases the risk of developing a number of chronic diseases, including sleep apnoea, type 2 diabetes mellitus (DM2), gallbladder diseases, osteoarthritis, and diseases of the cardiovascular system [7–9]. In 2015, approximately 4 million deaths worldwide were associated with high BMI, of which 2.7 million were associated with cardiovascular diseases and 0.9 million with diabetes mellitus [10]. Data on the relationship between smoking and obesity are contradictory. The results of a regression analysis of a study conducted on 16,412 participants aged 18–79 years showed a close relationship between waist circumference and smoking habits in women [11]. In a study conducted on 499,504 adults aged 31 to 69 years, it was found that the greatest risk of developing obesity is observed in respondents who quit smoking, and this risk remains high for 30 years after quitting [12]. Other studies have shown that smoking may not affect body weight indicators or be associated with weight loss [13]. Several studies have reported that smoking was associated with lower body weight and BMI [14]. It is also known that smoking is associated with insulin resistance and DM2 [15]. Obesity is associated with various metabolic disorders. One of the main causes of excess weight and obesity are poor diet (excessive energy consumption) and hypodynamia (low energy expenditure). Secondary obesity, which is an integral part of various genetic, endocrine diseases, and diseases of the central nervous system, is no more than 5%. The basis of carbohydrate metabolism disorders in obesity is secondary insulin resistance, hyperinsulinaemia develops against its background, which leads to an increase in body weight [16].

Numerous studies show the influence of intestinal hormones and microbiota on the development of obesity. In particular, the authors [17] demonstrate that the microbiome-abundant environment attenuates exogenous chemical-induced health risks by remodeling the intestinal microbiota, improving the intestinal ecosystem, and preventing intestinal epithelial leakage. In turn, a study [18] revealed that endocrine-disrupting chemicals induce gut microbiota changes through the gut-brain-microbiota axis conferring susceptibility to obesity and neurodevelopmental disorders. In other works [19, 20] the authors also note the therapeutic potential of microbiota in the treatment of obesity.

The total cost of treating obesity and related comorbidities is very high and continues to grow, which is reflected in the indirect impact on the well-being of society and its productivity [21]. According to Khan *et al.* [22] in the US, on average, spending per person in old age is significantly higher among overweight participants (12,390 USD [US Dollars] 95% confidence interval [CI] USD 10,427–14,354) and people with grade I and II obesity (USD 23,396, 95% CI USD 18,474–28,319) compared with participants with normal BMI ($p < 0.001$). Against the background of the COVID-19 pandemic, obesity was considered a clinically significant risk factor for severe disease [23]. Numerous studies have supported this theory, and several systematic reviews and meta-analyses on this issue have been published to date [24]. The study by Williamson *et al.* [25] has shown that a BMI of 30 kg/m$^2$ or higher is associated with a slightly higher risk of death from COVID-19 than a BMI of less than 30 kg/m$^2$. Accordingly, the prevention of obesity in the able-bodied population is also important for reducing the mortality rate from COVID-19. In addition, such efforts to achieve a normal weight should help reduce the risk of developing DM2, cardiovascular diseases, and certain types of cancer, which persisted throughout the pandemic and constantly put a burden on the health system [24]. Thus, one of the priorities of modern healthcare is the development of measures aimed at preventing the development of obesity, especially in children and the able-bodied population. This would lead to a significant reduction in cases of sudden death, and an increase in well-being and the coefficient of quality life expectancy in the population.

## Aim

The purpose of the study is to identify measures for the early prevention of overweight, obesity, and carbohydrate metabolism disorders in the able-bodied population at an industrial facility in Kazakhstan.

## Material and methods

The study involved 662 people (574 men and 88 women) aged 19 to 64 years. Inclusion criteria: age: 19–64 years, working in the industrial sector, the presence of secondary obesity, providing written consent to participate in the survey.

Exclusion criteria: age ≤ 19 years or ≥ 64 years, unemployed, secondary obesity in past medical history, lack of written consent to participate in the survey.

All subjects were divided into 2 groups: the main group –persons with obesity (242 people) and the control group – relatively healthy persons (422 people). This sample of respondents is almost evenly distributed in terms of the ratio of sick and healthy male and female respondents. The proportion of obese among men was 35% ±2%, and among women it was 43 ±5% ($z = -1.39$; $p \geq 0.05$) (Table I).

The diagnosis of obesity was made based on the medical history of the patient (the time of occurrence of obesity, the dynamics of weight changes, the presence of obesity in relatives, lifestyle, physical activity, diet, and nature of nutrition), an external examination, and physical, laboratory, and instrumental analysis. The study took into account glucose levels, BMI values, the presence of bad habits, and concomitant diseases (Tables II–IX). A questionnaire was used to collect data on physical activity and smoking.

In the study, it was required first to determine the state of health, i.e. whether the respondent was ill or healthy. Medical history, examination data, and clinical and physiological parameters such as age, gender, body mass index, level of inactivity, etc. were used as predictors.

**Table I.** Comparative characteristics of the distribution of those surveyed according to gender

| Group | X | ± m | *z* | *P* > \|*z*\| | 95% confidence interval | |
|---|---|---|---|---|---|---|
| Men | 0.35 | 0.02 | | | 0.32 | 0.39 |
| Women | 0.43 | 0.05 | | | 0.33 | 0.53 |
| Difference | –0.08 | 0.06 | | | –0.19 | 0.03 |
| | $H_0$ | 0.05 | –1.39 | 0.166 | | |

*$n_1$ (men) = 574, $n_2$ (women) = 88; difference = proportion (men) – proportion (women) – z = –1.39; $H_0$: difference = 0; Ha: difference < 0; Ha: difference != 0; Ha: difference > 0; Pr (Z < z) = 0.08; Pr (|Z| > |z|) = 0.16; Pr (Z > z) = 0.92.*

**Table II.** Comparative characteristics of the distribution of smoking respondents of both genders

| Group | *X* | ± m | *z* | *P* > \|*z*\| | 95% confidence interval | |
|---|---|---|---|---|---|---|
| Men | 0.33 | 0.02 | | | 0.29 | 0.37 |
| Women | 0.19 | 0.04 | | | 0.11 | 0.27 |
| Difference | –0.14 | 0.04 | | | 0.05 | 0.23 |
| | $H_0$ | 0.05 | 2.60 | 0.009 | | |

*$n_1$ (men) = 574, $n_2$ (women) = 88; difference = proportion (men) – proportion (women) – z = 2.60; $H_0$: difference = 0; Ha: difference < 0; Ha: difference! = 0; Ha: difference > 0; Pr (Z < z) = 0.99; Pr (|Z| > |z|) = 0.009; Pr (Z > z) = 0.005.*

**Table III.** Comparative characteristics of the distribution of persons of both genders with BMI > 26 kg/m$^2$

| Group | *X* | ± m | *z* | *P* > \|*z*\| | 95% confidence interval | |
|---|---|---|---|---|---|---|
| Men | 0.43 | 0.02 | | | 0.39 | 0.47 |
| Women | 0.52 | 0.05 | | | 0.42 | 0.63 |
| Difference | –0.09 | 0.06 | –1.63 | 0.104 | –0.20 | 0.02 |
| | $H_0$ | 0.05 | | | | |

*$n_1$ (men) = 574, $n_2$ (women) = 88; difference = proportion (men) – proportion (women) – z = -1.62; $H_0$: difference = 0; Ha: difference < 0; Ha: difference! = 0; Ha: difference > 0; Pr (Z < z) = 0.05; Pr (|Z| > |z|) = 0.10; Pr (Z > z) = 0.95.*

The protocol corresponding to the World Medical Association Declaration of Helsinki: ethical principles for medical research involving human subjects [26] and approved by the Ethics Committee of the Medical University provided for physical and clinical laboratory examination of patients in compliance with methodological requirements.

### Statistical analysis

Statistical analysis was performed using Stata 15 (Stats Corp LLC, TX, US, No. 301506315475), MedCalc Statistical Software version 19.6.1 (Medcalc Software Ltd, Ostend, Belgium), and Statistica 13.0 for Windows (StatSoft Inc. No. JPZ804I382130ARCN10-J). Quantitative values are presented as the arithmetic mean and 95% confidence interval (M [95% CI]), median and interquartile range (Me [Q25; Q75]), and qualitative indicators as absolute and relative frequencies (*n* [%]). The difference in the groups by quantitative indicators was determined by nonparametric statistics using the Mann-Whitney U test, and by qualitative values – the Yates-corrected $\chi^2$ criterion, Pearson coefficient, Cramer's V coefficient, and the Kendall correlation coefficient. The reliability of the differences is at the level of $p < 0.05$.

## Results and discussion

The study found that the level of glucose in the blood serum of 74.2% of the women in the control group was below 5.5 mmol/l, and in 25.8% it was above 5.6 mmol/l. In the men of the control group, in 93.8% of cases, the glucose level was below 5.5 mmol/l, and in 6.2% it exceeded the value of 5.6 mmol/l. In the men of the main group, in 76.9%, the glucose level was below 5.5 mmol/l, and in 23.1% it was above 5.6 mmol/l. In the women of the main group, the glucose level was lower than 5.5 mmol/l in 55.3%, and it exceeded 5.6 mmol/l in 44.7%. The evaluation of the quantitative relationship of the gender of the respondents of the control group with the BMI showed that it is weak and insignificant in terms of statistical reliability. Thus, the critical value of Pearson's $\chi^2$ at the significance level $p \geq 0.614$ is 1.8, and the p-calculated criteria of gamma, Kendall's tau-b, and Cramer's V showed a weak relationship between the studied nominal and interval variables at the level of –0.23, –0.07, and 0.07, respectively. When assessing the relationship between the gender of the respondents of the main group and the BMI, it was revealed that it is weak in strength and statistically unreliable. The critical value of Pearson's $\chi^2$ at the signifi-

**Table IV.** Distribution of respondents of both genders and different age groups (relatively healthy) depending on BMI levels (%)

| BMI level | Age | *X* | ± m | 95% confidence interval | |
|---|---|---|---|---|---|
| < 21 | 19–24 years old | 41.94 | 8.86 | 26.09 | 59.64 |
| < 21 | 25–29 years old | 31.43 | 5.55 | 21.64 | 43.20 |
| < 21 | 30–34 years old | 33.80 | 5.61 | 23.76 | 45.55 |
| < 21 | 35–39 years old | 36.07 | 6.15 | 25.03 | 48.80 |
| < 21 | 40–44 years old | 12.20 | 5.11 | 5.15 | 26.21 |
| < 21 | 45–49 years old | 16.67 | 7.61 | 6.37 | 37.01 |
| < 21 | 50–54 years old | 15.38 | 10.01 | 3.85 | 45.20 |
| < 21 | 55–64 years old | 0.00 | – | – | – |
| 22–25 | 19–24 years old | 58.06 | 8.86 | 40.36 | 73.91 |
| 22–25 | 25–29 years old | 65.71 | 5.67 | 53.87 | 75.88 |
| 22–25 | 30–34 years old | 64.79 | 5.67 | 53.02 | 75.00 |
| 22–25 | 35–39 years old | 59.02 | 6.30 | 46.32 | 70.62 |
| 22–25 | 40–44 years old | 78.05 | 6.46 | 62.86 | 88.19 |
| 22–25 | 45–49 years old | 75.00 | 8.84 | 54.27 | 88.35 |
| 22–25 | 50–54 years old | 84.62 | 10.01 | 54.80 | 96.15 |
| 22–25 | 55–64 years old | 90.91 | 8.67 | 55.95 | 98.75 |
| 26–29 | 19–24 years old | 0.00 | – | – | – |
| 26–29 | 25–29 years old | 2.86 | 1.99 | 0.71 | 10.77 |
| 26–29 | 30–34 years old | 1.41 | 1.40 | 0.20 | 9.39 |
| 26–29 | 35–39 years old | 3.28 | 2.28 | 0.82 | 12.24 |
| 26–29 | 40–44 years old | 9.76 | 4.63 | 3.70 | 23.34 |
| 26–29 | 45–49 years old | 8.33 | 5.64 | 2.08 | 27.99 |
| 26–29 | 50–54 years old | 0.00 | – | – | – |
| 26–29 | 55–64 years old | 9.09 | 8.67 | 1.25 | 44.05 |
| 30 < | 19–24 years old | 0.00 | – | – | – |
| 30 < | 25–29 years old | 0.00 | – | – | – |
| 30 < | 30–34 years old | 0.00 | – | – | – |
| 30 < | 35–39 years old | 1.64 | 1.63 | 0.23 | 10.81 |
| 30 < | 40–44 years old | 0.00 | – | – | – |
| 30 < | 45–49 years old | 0.00 | – | – | – |
| 30 < | 50–54 years old | 0.00 | – | – | – |
| 30 < | 55–64 years old | 0.00 | – | – | – |

*$n_1$ = 322.*

cance level $p \geq 0.28$ is 3.8, and the p-calculated criteria of gamma, Kendall's tau b, and Cramer's V showed an insignificant relationship between the studied variables at the level of –0.09, –0.04, and 0.13, respectively.

Analysis of the quantitative relationship between the smoking habits of patients in the control group and the BMI showed that it is insignificant in strength and insignificant in statistical reliability. Thus, the critical value of Pearson's $\chi^2$ at the significance level $p \geq 0.683$ is 1.49, and the p-calculated criteria of gamma, Kendall's tau b, and Cramer's V showed a very weak relationship between the studied nominal and interval variables at the level of –0.03, –0.01, and 0.07, respectively. The association between smoking in patients of the main group and BMI showed that it is insignificant in strength and in statistical reliability. The critical value of Pearson's $\chi^2$ at the significance level $p \geq 0.921$ is 0.49, and the p-calculated criteria of gamma, Kendall's tau b, and Cramer's V showed a zero relationship between the studied variables at the level of –0.0016,

**Table V.** Distribution of respondents of both genders and different age groups (obesity) depending on BMI levels (%)

| BMI level | Age | *X* | ± m | 95% confidence interval | |
|---|---|---|---|---|---|
| < 21 | 19–24 years old | 0.00 | – | – | – |
| < 21 | 25–29 years old | 0.00 | – | – | – |
| < 21 | 30–34 years old | 0.00 | – | – | – |
| < 21 | 35–39 years old | 0.00 | – | – | – |
| < 21 | 40–44 years old | 0.00 | – | – | – |
| < 21 | 45–49 years old | 3.13 | 3.08 | 0.43 | 19.27 |
| < 21 | 50–54 years old | 0.00 | – | – | – |
| < 21 | 55–64 years old | 0.00 | – | – | – |
| 22–25 | 19–24 years old | 0.00 | – | – | – |
| 22–25 | 25–29 years old | 4.00 | 3.92 | 0.55 | 23.73 |
| 22–25 | 30–34 years old | 2.70 | 2.67 | 0.38 | 16.99 |
| 22–25 | 35–39 years old | 0.00 | – | – | – |
| 22–25 | 40–44 years old | 2.22 | 2.20 | 0.31 | 14.28 |
| 22–25 | 45–49 years old | 0.00 | – | – | – |
| 22–25 | 50–54 years old | 4.00 | 3.92 | 0.55 | 23.73 |
| 22–25 | 55–64 years old | 0.00 | – | – | – |
| 26–29 | 19–24 years old | 40.00 | 21.91 | 9.94 | 80.10 |
| 26–29 | 25–29 years old | 68.00 | 9.33 | 47.73 | 83.18 |
| 26–29 | 30–34 years old | 54.05 | 8.19 | 38.05 | 69.26 |
| 26–29 | 35–39 years old | 70.00 | 6.48 | 55.96 | 81.08 |
| 26–29 | 40–44 years old | 60.00 | 7.30 | 45.17 | 73.20 |
| 26–29 | 45–49 years old | 53.13 | 8.82 | 36.06 | 69.49 |
| 26–29 | 50–54 years old | 52.00 | 9.99 | 32.99 | 70.45 |
| 26–29 | 55–64 years old | 47.83 | 10.42 | 28.71 | 67.60 |
| 30 < | 19–24 years old | 60.00 | 21.91 | 19.90 | 90.06 |
| 30 < | 25–29 years old | 28.00 | 8.98 | 13.92 | 48.33 |
| 30 < | 30–34 years old | 43.24 | 8.14 | 28.38 | 59.43 |
| 30 < | 35–39 years old | 30.00 | 6.48 | 18.92 | 44.04 |
| 30 < | 40–44 years old | 37.78 | 7.23 | 24.89 | 52.66 |
| 30 < | 45–49 years old | 43.75 | 8.77 | 27.82 | 61.08 |
| 30 < | 50–54 years old | 44.00 | 9.93 | 26.21 | 63.47 |
| 30 < | 55–64 years old | 52.17 | 10.42 | 32.40 | 71.29 |

*$n_3$ = 242.*

**Table VI.** Distribution of healthy respondents of both genders depending on glucose level (%)

| Glucose level | Gender | *X* | ± m | 95% confidence interval | |
|---|---|---|---|---|---|
| < 3.9 | Men | 1.03 | 0.59 | 0.33 | 3.16 |
| < 3.9 | Women | 0.00 | – | – | – |
| 4.0–5.5 | Men | 92.78 | 1.52 | 89.17 | 95.26 |
| 4.0–5.5 | Women | 74.19 | 7.86 | 56.18 | 86.57 |
| 5.6 < | Men | 6.19 | 1.41 | 3.92 | 9.62 |
| 5.6 < | Women | 25.81 | 7.86 | 13.43 | 43.82 |

*$n_1$ = 322.*

**Table VII.** Distribution of obese responders of both genders depending on glucose level (%)

| Glucose level | Gender | *X* | ± m | 95% confidence interval | |
|---|---|---|---|---|---|
| 4.0–5.5 | Men | 76.96 | 2.95 | 70.65 | 82.25 |
| 4.0–5.5 | Women | 55.26 | 8.07 | 39.38 | 70.14 |
| 5.6 < | Men | 23.04 | 2.95 | 17.75 | 29.35 |
| 5.6 < | Women | 44.74 | 8.07 | 29.86 | 60.62 |

*$n_3$ = 242.*

**Table VIII.** Distribution of respondents of different age groups (healthy) depending on glucose levels (%)

| Glucose level | Age | *X* | ± m | 95% confidence interval | |
|---|---|---|---|---|---|
| < 3.9 | 19–24 years old | 3.23 | 3.17 | 0.45 | 19.76 |
| < 3.9 | 25–29 years old | 2.86 | 1.99 | 0.71 | 10.77 |
| < 3.9 | 30–34 years old | 0.00 | – | – | – |
| < 3.9 | 35–39 years old | 0.00 | – | – | – |
| < 3.9 | 40–44 years old | 0.00 | – | – | – |
| < 3.9 | 45–49 years old | 0.00 | – | – | – |
| < 3.9 | 50–54 years old | 0.00 | – | – | – |
| < 3.9 | 55–64 years old | 0.00 | – | – | – |
| 4.0–5.5 | 19–24 years old | 90.32 | 5.31 | 73.85 | 96.86 |
| 4.0–5.5 | 25–29 years old | 92.86 | 3.08 | 83.91 | 97.01 |
| 4.0–5.5 | 30–34 years old | 94.37 | 2.74 | 85.88 | 97.88 |
| 4.0–5.5 | 35–39 years old | 91.80 | 3.51 | 81.72 | 96.56 |
| 4.0–5.5 | 40–44 years old | 90.24 | 4.63 | 76.66 | 96.30 |
| 4.0–5.5 | 45–49 years old | 79.17 | 8.29 | 58.57 | 91.08 |
| 4.0–5.5 | 50–54 years old | 84.62 | 10.01 | 54.80 | 96.15 |
| 4.0–5.5 | 55–64 years old | 90.91 | 8.67 | 55.95 | 98.75 |
| 5.6 < | 19–24 years old | 6.45 | 4.41 | 1.61 | 22.52 |
| 5.6 < | 25–29 years old | 4.29 | 2.42 | 1.38 | 12.51 |
| 5.6 < | 30–34 years old | 5.63 | 2.74 | 2.12 | 14.12 |
| 5.6 < | 35–39 years old | 8.20 | 3.51 | 3.44 | 18.28 |
| 5.6 < | 40–44 years old | 9.76 | 4.63 | 3.70 | 23.34 |
| 5.6 < | 45–49 years old | 20.83 | 8.29 | 8.92 | 41.43 |
| 5.6 < | 50–54 years old | 15.38 | 10.01 | 3.85 | 45.20 |
| 5.6 < | 55–64 years old | 9.09 | 8.67 | 1.25 | 44.05 |

*$n_1$ = 322.*

–0.0007, and 0.04, respectively. An assessment of the quantitative relationship between the glucose level in the control group respondents and the BMI found that it was weak in strength and significant in statistical reliability. Thus, the critical value of Pearson's $\chi^2$ at the significance level $p \leq 0.01$ is 15.96, and the p-calculated criteria of gamma, Kendall's tau b, and Cramer's V showed a weak relationship between the studied nominal and interval variables at the level of 0.33, 0.09, and 0.15, respectively. Evaluation of the relationship between glucose levels in patients of the main group and BMI also showed that it is weak in strength, and significant in statistical reliability. Thus, the critical value of Pearson's $\chi^2$ at the significance level $p \leq 0.001$ is 6.76, and the p-calculated criteria of gamma, Kendall's tau b, and Cramer's V presented a weak relationship between the studied nominal and interval variables at the level –0.15, 0.07, and 0.17, respectively.

The characterisation of the quantitative relationship between the level of inactivity in the control group respondents and BMI revealed that it is closer to moderate in strength and significant in statistical reliability.

**Table IX.** Distribution of respondents of different age groups (obese) depending on glucose levels (%)

| Glucose level | Age | *X* | ± m | 95% confidence interval | |
|---|---|---|---|---|---|
| 4.0–5.5 | 19–24 years old | 100.00 | 0.00 | – | – |
| 4.0–5.5 | 25–29 years old | 84.00 | 7.33 | 64.19 | 93.89 |
| 4.0–5.5 | 30–34 years old | 78.38 | 6.77 | 62.27 | 88.84 |
| 4.0–5.5 | 35–39 years old | 76.00 | 6.04 | 62.26 | 85.88 |
| 4.0–5.5 | 40–44 years old | 68.89 | 6.90 | 54.01 | 80.68 |
| 4.0–5.5 | 45–49 years old | 78.13 | 7.31 | 60.60 | 89.24 |
| 4.0–5.5 | 50–54 years old | 68.00 | 9.33 | 47.73 | 83.18 |
| 4.0–5.5 | 55–64 years old | 52.17 | 10.42 | 32.40 | 71.29 |
| 5.6 < | 19–24 years old | 0.00 | – | – | – |
| 5.6 < | 25–29 years old | 16.00 | 7.33 | 6.11 | 35.81 |
| 5.6 < | 30–34 years old | 21.62 | 6.77 | 11.16 | 37.73 |
| 5.6 < | 35–39 years old | 24.00 | 6.04 | 14.12 | 37.74 |
| 5.6 < | 40–44 years old | 31.11 | 6.90 | 19.32 | 45.99 |
| 5.6 < | 45–49 years old | 21.88 | 7.31 | 10.76 | 39.40 |
| 5.6 < | 50–54 years old | 32.00 | 9.33 | 16.82 | 52.27 |
| 5.6 < | 55–64 years old | 47.83 | 10.42 | 28.71 | 67.60 |

$n_3 = 242$.

Thus, the critical value of Pearson's $\chi^2$ at the significance level $p \leq 0.001$ is 89.34, and the p-calculated criteria of gamma, Kendall's tau b, and Cramer's V presented moderate feedback between the studied nominal and interval variables at the level –0.43, –0.17, and 0.37, respectively. The association between the level of inactivity in patients of the main group and BMI turned out to be closer to strong in strength and significant in statistical reliability. Thus, the critical value of Pearson's $\chi^2$ at the significance level $p \leq 0.001$ is 154.69, and the p-calculated criteria of gamma, Kendall's tau b, and Cramer's V presented strong feedback between the studied nominal and interval variables at the level –0.88, –0.67, and 0.56, respectively.

The quantitative relationship between the health status of the control group respondents and the BMI turned out to be closer to weak in strength and insignificant in statistical reliability. Thus, the critical value of Pearson's $\chi^2$ at a significance level of $p \geq 0.095$ is 29.83, and the p-calculated criteria of gamma, Kendall's tau b, and Cramer's V showed a weak relationship between the studied nominal and interval variables at the level 0.28, 0.17, and 0.18, respectively. The assessment of the relationship between the health status of patients in the main group and BMI turned out to be closer to weak and statistically unreliable. Thus, the critical value of Pearson's $\chi^2$ at the significance level $p \geq 0.71$ is 17.02, and the p-calculated criteria of gamma, Kendall's tau b, and Cramer's V showed a weak relationship between the studied variables at the level 0.11, 0.07, and 0.15, respectively.

The calculated values of the odds ratio (OR) (Table X) of the presented predictors provide useful information for understanding the quantitative relationship between the level of inactivity, body mass index, gender, and age with the probabilistic state of health of the respondents (sick/healthy). The body mass index is a strong predictor of a person's health status and the ratio of chances of being sick is 21.4 times higher in people with increased body weight than in healthy people with normal weight ($z = 9.61$; $p \leq 0.001$), which corresponds to data obtained by Khan *et al.* In other words, there is a direct relationship between the level of morbidity and increased body weight of the studied groups of people. The remaining predictors are much less powerful than BMI, and they are statistically unreliable ($p \geq 0.05$).

The calculated regression coefficients (RC) of the above predictors are also useful for understanding the quantitative relationship between the level of inactivity, body mass index, biological sex and age, and the probabilistic state of health of the respondent (sick/healthy). Moreover, these coefficients can be used to construct a mathematical and statistical equation for predicting a particular state of health of respondents in response to changes in the levels of predictors/factors (Table XI).

For a more visual characterisation of the quantitative relationship of the level of inactivity, body mass index, biological age, and smoking with the probabilistic level of the respondent's BMI (0 = < 25 and 1 = > 26), the calculation of the OR values of the presented

**Table X.** Comparative analysis of the quantitative influence of some biological, functional, and clinical factors on the health status (sick/healthy) in respondents of both genders using logistic regression (odds ratio)

| Group (sick/healthy) | Odds ratio | ± m | *z* | *P* > \|*z*\| | 95% confidence interval | |
|---|---|---|---|---|---|---|
| Hypodynamia | 0.64 | 0.18 | –1.56 | 0.119 | 0.36 | 1.12 |
| BMI | 21.44 | 6.84 | 9.61 | 0.001 | 11.47 | 40.08 |
| Gender | 1.34 | 0.51 | 0.76 | 0.449 | 0.63 | 2.83 |
| Age | 0.91 | 0.07 | –1.36 | 0.174 | 0.78 | 1.05 |
| Constant | 0.00072 | 0.00097 | –5.35 | 0.001 | 0.00005 | 0.01 |

*n (ill/healthy) = 662; LR* $\chi^2$ *= 486.6; p ≤ 0.001; pseudo* $R^2$ *= 0.56.*

**Table XI.** Comparative analysis of the quantitative influence of some biological, functional, and clinical factors on the health status (sick/healthy) in respondents of both genders using logistic regression (regression coefficients)

| Group (sick/healthy) | Regression coefficient | ± m | *z* | *P* > \|*z*\| | 95% confidence interval | |
|---|---|---|---|---|---|---|
| Hypodynamia | –0.44644 | 0.2867144 | –1.56 | 0.119 | –1.00839 | 0.1155099 |
| BMI | 3.06551 | 0.3190968 | 9.61 | 0.001 | 2.440092 | 3.690928 |
| Gender | 0.2903335 | 0.3837317 | 0.76 | 0.449 | –0.4617669 | 1.042434 |
| Age | –0.09911265 | 0.0729113 | –1.36 | 0.174 | –0.24203 | 0.043777 |
| Constant | –7.241089 | 1.354148 | –5.35 | 0.001 | –9.895171 | –4.587008 |

*n (ill/healthy) = 662; LR* $\chi^2$ *= 486.6; p ≤ 0.001; pseudo* $R^2$ *= 0.56.*

predictors was carried out using a nested (hierarchical) logistic regression in the form of sequential blocks. Each block provides information for understanding the reliable significance of the quantitative influence of a predictor (independent variable) on the variable under study. Thus, in the first block, the influence of one biological age as a predictor of the respondent's BMI level is investigated, and it can be stated that it is statistically reliable: $z = 4.88$, $p \le 0.001$. The biological age of the respondent is a weak predictor of a BMI level and the ratio of the chances of being obese, and prediabetes is only 1.2 times higher in people with increasing age. In the second block, in addition to assessing the influence of age, which is also statistically significant at the level of $p \le 0.001$, smoking is also considered, although its effect on the respondent's BMI level is already statistically unreliable ($z = 1.18$, $p \ge 0.24$). The body mass index is a strong predictor of a person's health status and the ratio of the chances of being obese, and prediabetes is 22.6 times higher in people with increased body weight than in healthy people with normal weight ($z = 9.70$, $p \le 0.001$), considering the influence of 2 other factors. Thus, there is a direct link between the disease and the increased body weight of the studied groups of people (Wald $\chi^2 = 160.9$, $p \le 0.001$). The remaining predictors are much weaker than BMI and statistically unreliable ($p \ge 0.05$) (Table XII–XV).

The calculated values of the odds ratio of the following predictors/factors provide useful information for understanding the quantitative relationship of gender and age, diagnosis, smoking, the level of inactivity, and the concentration of glucose in the blood of respondents with the level of their BMI (0 = < 25 and 1 = > 26). As can be seen, the diagnosis, the level of inactivity, and age are statistically reliable predictors of the respondents' BMI level, and their odds ratio varies between 0.05 and 2.3 ($z = –11.2–10.1$, $p \le 0.05–0.001$). In other words, there is a direct relationship between the above factors and the increased body weight of the studied groups, and for hypodynamia, the relationship is inversely proportional, i.e., the lower the respondent's physical activity scores, the higher the body mass index. The remaining predictors are much smaller in their strength, and they are statistically unreliable ($p \ge 0.05$) (Tables XVI, XVII).

The calculated regression coefficients of the studied predictors are also necessary to understand the quantitative relationship between the level of inactivity, diagnosis, biological age, and the respondent's BMI level (0 = < 25 and 1 = > 26). In addition, these coefficients can be used to construct a mathematical and statistical equation for predicting a particular BMI level of respondents in response to changes in the levels of predictors/factors (Table XVIII).

For a review assessment of the quantitative relationship between the level of inactivity, age, and diagnosis with the probabilistic level of the respondent's BMI (0 = < 25 and 1 = > 26), a calculation of the values of the OR of the studied predictors was carried out using a nested (hierarchical) logistic regression in the form of sequential blocks. Each block provides information for understanding the reliable significance of the quantitative influence of a predictor (independent variable) on the variable under study. Thus, in the first block, the influence of one biological age as a predictor of the respondent's BMI level is investigated, and it can be stated that it is statistically reliable: $z = 6.94$, $p \leq 0.001$. The biological age of the respondent is a weak predictor

**Table XII.** Comparative analysis of the quantitative influence of some biological, functional, and clinical factors in the form of blocks on the BMI in respondents of both genders using nest logistic regression (odds ratio, block 1)

| BMI | Odds ratio | ± m | *z* | *P* > \|*z*\| | 95% confidence interval | |
|---|---|---|---|---|---|---|
| Age | 1.23 | 0.05 | 4.88 | 0.001 | 1.13 | 1.34 |
| Constant | 0.23 | 0.04 | –7.06 | 0.001 | 0.16 | 0.35 |

*n (BMI) = 662; LR $\chi^2$ = 52.4; p ≤ 0.001; pseudo $R^2$ = 0.056.*

**Table XIII.** Comparative analysis of the quantitative influence of some biological, functional, and clinical factors in the form of blocks on the BMI in respondents of both genders using nest logistic regression (odds ratio, block 2)

| BMI | Odds ratio | ± m | *z* | *P* > \|*z*\| | 95% confidence interval | |
|---|---|---|---|---|---|---|
| Age | 1.24 | 0.05 | 4.97 | 0.001 | 1.14 | 1.35 |
| Smoking | 1.24 | 0.22 | 1.18 | 0.24 | 0.87 | 1.75 |
| Constant | 0.16 | 0.06 | –4.76 | 0.001 | 0.075 | 0.34 |

*n (BMI) = 662; LR $\chi^2$ = 436.9; p ≤ 0.001; pseudo $R^2$ = 0.48.*

**Table XIV.** Comparative analysis of the quantitative influence of some biological, functional, and clinical factors in the form of blocks on the BMI in respondents of both genders using nest logistic regression (odds ratio, block 3)

| BMI | Odds ratio | ± m | *z* | *P* > \|*z*\| | 95% confidence interval | |
|---|---|---|---|---|---|---|
| Age | 0.92 | 0.07 | –1.08 | 0.28 | 0.80 | 1.07 |
| Smoking | 1.62 | 0.45 | 1.73 | 0.08 | 0.94 | 2.81 |
| BMI | 22.64 | 7.28 | 9.70 | 0.001 | 12.06 | 42.52 |
| Hypodynamia | 0.66 | 0.19 | –1.45 | 0.15 | 0.38 | 1.16 |
| Constant | 0.00024 | 0.00036 | –5.64 | 0.001 | 0.00001 | 0.0044 |

*n (BMI) = 662; LR $\chi^2$ = 580.8; p ≤ 0.001; pseudo $R^2$ = 0.64.*

**Table XV.** Comparison of blocks indicators

| Block | Wald $\chi^2$ | Degree of freedom | *P* > *F* |
|---|---|---|---|
| 1 | 23.77 | 1 | 0.001 |
| 2 | 1.40 | 1 | 0.24 |
| 3 | 160.91 | 2 | 0.001 |

**Table XVI.** Comparative analysis of the quantitative influence of some biological, functional, and clinical factors on the level of BMI in respondents of both genders using logistic regression (odds ratio)

| BMI | Odds ratio | ± m | *z* | *P* > \|*z*\| | 95% confidence interval | |
|---|---|---|---|---|---|---|
| Gender | 1.39 | 0.62 | 0.75 | 0.454 | 0.58 | 3.34 |
| Diagnosis | 2.27 | 0.18 | 10.11 | 0.001 | 1.94 | 2.67 |
| Smoking | 0.99 | 0.31 | –0.02 | 0.984 | 0.54 | 1.84 |
| Hypodynamia | 0.048 | 0.01 | –11.19 | 0.001 | 0.03 | 0.082 |
| Age | 1.16 | 0.09 | 1.95 | 0.052 | 0.99 | 1.36 |
| Glucose | 1.06 | 0.41 | 0.15 | 0.880 | 0.49 | 2.26 |
| Constant | 22.71 | 27.007 | 2.63 | 0.009 | 2.21 | 233.58 |

*n (BMI) = 662; LR $\chi^2$ = 581.4; p ≤ 0.001; pseudo $R^2$ = 0.64.*

**Table XVII.** Comparative analysis of the quantitative influence of some biological, functional, and clinical factors on the level of BMI in respondents of both genders using logistic regression (odds ratio)

| BMI | Odds ratio | ± m | z | *P* > \|*z*\| | 95% confidence interval | |
|---|---|---|---|---|---|---|
| Diagnosis | 2.30 | 0.18 | 10.38 | 0.001 | 1.97 | 2.69 |
| Hypodynamia | 0.049 | 0.013 | –11.34 | 0.001 | 0.029 | 0.08 |
| Age | 1.17 | 0.09 | 2.06 | 0.039 | 1.008 | 1.36 |
| Constant | 33.21 | 23.09 | 5.04 | 0.001 | 8.49 | 129.81 |

*n (BMI) = 662; LR* $\chi^2$ *= 580.8; p ≤ 0.001; pseudo* $R^2$ *= 0.64.*

of a person's BMI level, and the odds ratio of having an increased weight is only 1.3 times higher in people with increasing age. In the second block, in addition to assessing the influence of age, which is also statistically significant at the level of $p \leq 0.001$, the level of inactivity is investigated, and this effect on the respondent's BMI level is also statistically significant: $z = -14.10$, $p \leq 0.001$. The diagnosis is a relatively weak predictor of a person's BMI level, although its contribution is higher than that of the other 2 factors, but the odds ratio of being overweight is only 2.3 times higher ($z = 10.38$, $p \leq 0.001$), considering the influence of the other 2 factors. Thus, there is a direct relationship between the studied predictors and the increased body weight of the studied groups of people (Wald $\chi^2$ = 107.8–198.8, $p \leq 0.001$) (Tables XIX–XXII).

Thus, having quantitatively analysed all of the above factors (predictors) of the BMI level of the studied respondents, both sick and relatively healthy, it is possible to reliably build their ordinal series according to the degree of influence on the dependent variable (BMI) in the following form: first place – the level of inactivity, which corresponds to the data obtained by Gray *et al.* [27]; second place – blood glucose level, which is confirmed by Naguib *et al.* [28]; third place – the health status of respondents (concomitant diagnosis). Other factors, such as gender, age, and smoking habits, do not significantly affect the distribution of BMI of individuals, as confirmed by Wang [13]. However, according to Yi *et al.* [29] women have a lower BMI than men, especially at a younger age. BMI increased with age, which was more pronounced in women than in men. Age and gender optima, as a rule, exceeded the current normal weight (BMI 18.5–24.9 kg/m$^2$), with the exception of women younger than 50 years of age. Obesity develops as a result of a violation of the energy balance of the body, when the energy intake from food exceeds the energy expenditure of the body. The causes of obesity are diverse; some of them, including genetic factors, are not subject to external influence [30], while others, such as diet, physical activity [31], and socio-economic circumstances (poverty) [32], can be corrected. Recent studies show the connection of obesity with the influence of environmental factors [33] and the peculiarities of life in megacities [34]. In addition, all these factors probably work in tandem, creating a complex set of conditions under which obesity persists. However, the research data by Hsiao *et al.* [35] show that there is no clear relationship between food consumption patterns and BMI or waist circumference in the elderly.

The paper revealed a close relationship between body mass index indicators and the presence of comorbid conditions, these data are consistent with the data by Khan *et al.* [22] obtained during a prospective cohort study of 29,621 patients. Based on the data obtained, it was found that lack of physical activity is one of the main factors contributing to the obesity epidemic, and it is often the goal of intervention, because it is amenable to change at the individual level, which does not contradict the data presented by Wiklund [36]. Accordingly, public health to implement obesity prevention measures should focus on promoting physical activity rather than healthy eating, but the former does not preclude the latter. Environmental factors also affect the

**Table XVIII.** Comparative analysis of the quantitative influence of some biological, functional, and clinical factors on the level of BMI in respondents of both genders using logistic regression (regression coefficients)

| BMI | Regression coefficient | ± m | z | *P* > \|*z*\| | 95% confidence interval | |
|---|---|---|---|---|---|---|
| Diagnosis | 0.8331114 | 0.0802342 | 10.38 | 0.001 | 0.6758553 | 0.9903675 |
| Hypodynamia | –3.011142 | 0.2655676 | –11.34 | 0.001 | –3.531645 | –2.490639 |
| Age | 0.1588813 | 0.0770622 | 2.06 | 0.039 | 0.0078422 | 0.3099204 |
| Constant | 3.502811 | 0.6955859 | 5.04 | 0.001 | 2.139488 | 4.866135 |

*n (BMI) = 662; LR* $\chi^2$ *= 580.8; p ≤ 0.001; pseudo* $R^2$ *= 0.64.*

**Table XIX.** Comparative analysis of the quantitative influence of some biological, functional, and clinical factors in the form of blocks on the level of BMI in respondents of both genders using nested logistic regression (odds ratio, block 1)

| BMI | Odds ratio | ± m | *z* | *P* > \|*z*\| | 95% confidence interval | |
|---|---|---|---|---|---|---|
| Age | 1.35 | 0.059 | 6.94 | 0.001 | 1.24 | 1.47 |
| Constant | 0.22 | 0.044 | –7.45 | 0.001 | 0.14 | 0.32 |

*n (BMI) = 662; LR* $\chi^2$ *= 52.4; p ≤ 0.001; pseudo* $R^2$ *= 0.056.*

**Table XX.** Comparative analysis of the quantitative influence of some biological, functional, and clinical factors in the form of blocks on the level of BMI in respondents of both genders using nested logistic regression (odds ratio, block 2)

| BMI | Odds ratio | ± m | *z* | *P* > \|*z*\| | 95% confidence interval | |
|---|---|---|---|---|---|---|
| Age | 1.26 | 0.081 | 3.64 | 0.001 | 1.11 | 1.43 |
| Hypodynamia | 0.041 | 0.009 | –14.10 | 0.001 | 0.026 | 0.064 |
| Constant | 545.08 | 328.8 | 10.44 | 0.001 | 167.06 | 1,778.4 |

*n (BMI) = 662; LR* $\chi^2$ *= 436.9; p ≤ 0.001; pseudo* $R^2$ *= 0.48.*

**Table XXI.** Comparative analysis of the quantitative influence of some biological, functional, and clinical factors in the form of blocks on the level of BMI in respondents of both genders using nested logistic regression (odds ratio, block 3)

| BMI | Odds ratio | ± m | *z* | *P* > \|*z*\| | 95% confidence interval | |
|---|---|---|---|---|---|---|
| Age | 1.17 | 0.090 | 2.06 | 0.04 | 1.008 | 1.36 |
| Hypodynamia | 0.049 | 0.013 | –11.34 | 0.001 | 0.029 | 0.08 |
| Diagnosis | 2.30 | 0.18 | 10.38 | 0.001 | 1.97 | 2.69 |
| Constant | 33.21 | 23.09 | 5.04 | 0.001 | 8.49 | 129.81 |

*n (BMI) = 662; LR* $\chi^2$ *= 580.8; p ≤ 0.001; pseudo* $R^2$ *= 0.64.*

**Table XXII.** Comparison of blocks indicators

| Block | Wald $\chi^2$ | Degree of freedom | *P* > *F* |
|---|---|---|---|
| 1 | 48.19 | 1 | 0.001 |
| 2 | 198.85 | 1 | 0.001 |
| 3 | 107.82 | 1 | 0.001 |

level of physical activity and, accordingly, the development of obesity (air pollution, lack of a green zones, etc.) [35].

A large-scale study by Dicker *et al.* [37] investigated perceptions, behaviours, and barriers to effective coping with obesity by interviewing 14,502 adult participants with BMI ≥ 30 kg/m$^2$ from 11 countries. It was found that even though 65% of patients and 80% of medical professionals perceive obesity as a chronic disease, only 55% of obese people officially establish such a diagnosis and only a quarter of them receive long-term controlled treatment. According to 79% of doctors and 63% of patients, a complete lifestyle change is required to reduce body weight. All methods of treating obesity, including non-drug, drug, and surgical, are aimed at reducing the intake and/or increasing energy consumption. Proper nutrition is central to the treatment of obesity because it helps reduce blood glucose levels, which, according to the results of this study, is a prerequisite for reducing body weight. Based on the data obtained in the study, the blood glucose level ranks second in terms of the degree of influence on BMI, which is confirmed by Naguib *et al.* [28]. Accordingly, only a gradual, long-term change in nutrition, established eating habits, and not a temporary restriction of the use of certain foods can lead to successful weight loss. Karlsson *et al.* [38] established a close relationship between increased BMI and the level of dementia in middle-aged people, which again must be considered when promoting the prevention of obesity as a factor affecting the quality of the production result, including at industrial facilities in Kazakhstan.

Thus, based on the data obtained, it is possible to determine the main directions of obesity prevention among able-bodied citizens working at an industrial enterprise:

- mandatory availability of industrial gymnastics;
- organisation of amateur sports teams in various sports;
- organisation of internal sports competitions with mandatory encouragement of employees to strengthen motivation and introduce a healthy lifestyle;
- changing the diet in places catering at work (canteens, buffets);
- conducting regular thorough medical examinations for early detection of diseases that contribute to the development of obesity;
- regular monitoring of blood glucose levels.

## Conclusions

Based on a quantitative analysis of all of the above factors (predictors) of an increase in the BMI of the respondents studied, namely, data from physical, clinical, laboratory examinations, determination of serum glucose levels, BMI values, the presence of bad habits, concomitant diseases, both sick and relatively healthy individuals, it is possible to reliably build their ordinal a number according to the degree of influence on the dependent variable (BMI) in the following form: first place – the level of inactivity; second place – the level of glucose in the blood; third place – the state of health of respondents (concomitant diseases). Other factors, such as gender, age, and smoking history, do not significantly affect the distribution of BMI in persons working at an industrial facility. The paper found that the biological age of the respondent is a weak predictor of an increase in the level of BMI and the ratio of the chances of developing obesity; prediabetes is only 1.2 times higher in people with increasing age.

Analysis of the quantitative relationship between smoking in both the control and main group patients and BMI showed that it is insignificant in strength and insignificant in statistical reliability, which indicates that this factor is not an obligate predictor of obesity. Based on the data obtained during the study, 3 main directions of obesity prevention in the able-bodied population at an industrial facility in Kazakhstan were identified: promotion and active implementation of physical activity, i.e. industrial gymnastics, regular screening analysis of blood glucose levels, dietary changes, regular thorough medical examinations. The strong links revealed between the level of inactivity, the level of serum glucose, the degree and rate of increase in BMI, and the further development of alimentary obesity, allow the development of an individual programme for weight loss for each patient. A limitation of the study is that the authors did not collect data on the number of cigarettes smoked, which could have influenced the results.

## Conflict of interest

The authors declare no conflict of interest.

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