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CHANGING BODY MASS INDEX IN THE FIRST YEAR AFTER STARTING STANDARD GLUCOSE THERAPEUTIC MEASURES IN PATIENTS WITH DIABETES MELLITUS

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Although Body Mass Index (BMI) is not a perfect indicator of nutritional status, a large number of studies showed a direct correlation between the BMI and mortality from all cause, making it an important prognostic factor for the evolution of diabetes mellitus. The aim of this study is to show the evolution of BMI in the first year after initiation of antidiabetic therapy in diabetic patients that were not treated up to the time of registration. The study was conducted on 1603 patients (842 women, 761 men) of whom 803 had data on the BMI after one year (447 women, 356 men). Patients received nutritional counselling, oral and/or injection antidiabetic therapy. It analyzed the influence of different psycho-socio-medical factors on BMI evolution. Female gender, Romanian ethnicity, urban areas origin, lack of alcohol consumption, superior professional training, increased monthly income, higher value of initial BMI, daily consumption of fruits and vegetables, the presence of hypertension and family history of diabetes were associated with a reduction in average BMI at 1 year follow-up. There was no correlation between BMI evolution and the marital status, smoker/non-smoker status, stress, physical activity, the association of dyslipidemia, history of fetal macrosomia in women. An increased BMI has led to a therapy intensification after one year. BMI is an indicator influenced by many factors. A psycho-socio-medical approach of risk factors leads to a lower BMI that results in a better evolution and prognosis of diabetes mellitus.

Key words: diabetes mellitus, body mass index, lifestyle intervention.

BACKGROUND AND AIMS

Management of weight control in patients with newly diagnosed diabetes is beneficial in the treatment and prognosis[1–2]. A lifestyle intervention in the early evolution of diabetes, leads to a better metabolic response[3–6]. Numerous studies have shown a decrease of morbidity and mortality from any cause by reducing BMI[7–14]. Patients with diabetes mellitus due to metabolic stress of obesity could benefit of glycemic control only by weight loss[15–17].

There are many controversies in the theory of "obesity paradox" (which highlight lower risk of cardiovascular mortality in overweight/obese patients compared to those of normal weight[18–21]), because the data are often deficient in terms of physiological differences in diabetes genotypes, presence of other risk factors in diabetic patients with normal body weight (smoking, alcohol consumption), fat distribution, comorbidities, lack of information about the exact causes of mortality.

The prevalence of overweight in world population in 2014 in people over 18 years was 1.8 billion of which more than 600 million obese[22]. The prevalence of diabetes among patients with BMI ≥ 40 kg/m^2 as SHIELD (The Study to Help Improve Early evaluation and managementof risk factors Leading to Diabetes) was 25%, and 27% according to NHANES (National Health and Nutrition Examination Surveys). The prevalence of overweight diabetics was 87% (of which 59% obese) in SHIELD and 82% (of which 51% obese) in NHANES[23].

In Romania, the results of ORO study (epidemiological study of the prevalence of obesity and risk factors of obesity in adults – 2014) have shown an obesity prevalence of 21.3% (9.9% in the category 18–39 years, 30% between 40–59 years and 41.6% over 60 years)[24].

Although the importance of reducing BMI is unquestionable, the core problem in obese people is the rhythm of weight loss, influenced by many factors, such as poor medical education, lack of compliance, social environment, various addictions, limited exercise capacity, age, secondary obesity etc.

MATERIALS AND METHODS

This is a prospective study developed in Municipal Hospital Ploiesti, Department of Diabetes. Between August 2013 and September 2015 there were consecutively registered 1603 patients (842 women, 761 men) newly diagnosed with diabetes or with a history of increased blood sugar levels (glycemia ≥ 126 mg/dl), that had no dietary or therapeutic intervention to registration. Only 803 patients (447 women, 356 men) had data on the BMI to one year follow-up (476 patients did not returned, 98 died and 226 patients had missing data of BMI evolution). Patients received nutritional counseling, oral and/or injection antidiabetic therapy based on American Diabetes Asociation guidelines. We collected data from the medical record about the following psycho-socio-medical factors in order to analyse the relationship with the evolution of BMI: gender, age, social environment, ethnicity, professional status, marital status, socioeconomic status, status of smoking, drinking alcohol consumption, fruits and vegetables consumption, physical activity, episodes major stress, baseline BMI, BMI maximum combination hypertension, dyslipidemia, family diabetes history, personal history of fetal macrosomia, initial treatment and adjustments therapy.

BMI was calculated after the formula: weight in kilograms divided by the square of height in meters.

STATISTICAL ANALYSIS

All statistical analyses were carried out using R, version 3.1.3[25]. Normality was assessed primarily visually using histograms (of studentized residuals), quantile-quantile plots and boxplots, and secondarily by the omnibus d'Agostino test (as implemented in the fBasics R package[26]). Homogeneity of variance across groups was also assessed graphically (by plotting the distribution of residuals) and inferentially by the Levene test (as implemented in the "car" package[27]). Two-group comparisons of continuous variables were performed by Welch t Student test (*i.e.* assuming unequal variances) when the normality assumption was satisfied and by the Mann-Whitney test when that assumption was not valid; univariate, multiple group comparisons were done with (one way) Welch ANOVA ("car" package). For effect size was used Hedges's g in the case of two group parametric comparisons and Cliff's delta in case of two group non-parametric comparisons ("effsize" R package[28]); to estimate effect size in ANOVA omega squared was used, computed manually in R based on the results returned by the "car" package, using the standard formula from the literature (as no package is available for its automatic computation)[29,30]. Quantile regression as a form of robust regression was performed using the "quantreg"[31] package and robust regression using Huber's M estimation method was performed using R package "MASS"[32].

All variables independently associated with changes in BMI ($p < 0.05$) from the univariate analyses were entered in a multivariate backward stepwise selection regression model. Standardization of regression coefficients was carried out using the package "QuantPsyc"[33]. To adjust for baseline BMI and the fact that this was an observational study, we did not model the BMI at 12 months as such, but the change in BMI from baseline to one year (delta BMI). The correlation between two continuous variables was assessed with Pearson's product moment correlation coefficient. Graphs were generated with package ggplot2[34].

RESULTS AND DISCUSSIONS

After one year of the initial diagnosis 5% of the patients had a decrease of 4.00 kg/m^2 in their BMI, 30% had a reduction of at least 1.40 kg/m^2, 50% a decrease of at least 0.40 kg/m^2 and in 60% of the patients BMI remained unchanged or decreased to different extents. Decreasing BMI is assumed to be associated with a decrease in health costs, as evidenced by a Spanish study reporting that decreasing one BMI unit in one year was associated with an 8% decrease in health-costs among those patients not increasing their BMI in one year (whereas increasing BMI with one unit among those patients whose BMI increased in one year was associated with a 20% rise in costs)[35].

Gender. In our dataset, female patients had initially a mean BMI of 33.32 kg/m^2 (95% CI 32.71-33.92), whereas at one year the mean BMI

had slightly decreased to 32.76 kg/m^2 (95% CI 32.18-33.33). Similarly, the BMI of male patients in this data set was initially 30.68 (95% CI 30.16-31.20), but after one year it was almost unmodified, 30.58 (95% CI 30.07-31.10). Thus, whereas in one year the mean BMI of women decreased by 0.56 kg/m^2, that in men only decreased by 0.09 kg/m^2 ($p = 0.004$, Hedge's $g = 0.20$).

The difference in BMI between the two genders is in line with what is known about the relationship between this derivative measure and diabetes: men tend to be diagnosed with diabetes at a BMI lower by 1–3 kg/m^2 than women, partly assumed to be related to a rise of automation and a reduction in physical work in men (more than in women)[36]. However, a large study on the variation of BMI across countries found large differences between various countries (with a higher between-country variance for women than for men), the lowest average BMI in both men and women being registered in Vietnam (20.3 and 19.8, respectively), the highest mean BMI in men in Mali (32.7) and the highest mean BMI in women in South Africa (30.9)[37]. It is not clear why women in this study tended to decrease their BMI within one year, whereas it mostly did not change in men. However, this should not be interpreted that all male patients had roughly the same BMI; rather, in some subjects BMI decreased and in others increased, with a null average. The central tendency was towards lower BMI in females, unlike males.

Age. As expected, there were large variations regarding BMI and age, with no simple [linear] relationship. However, the data tended to indicate a decrease in BMI with age ($r = –0.14$, $p < 0.001$), although the relationship is far from being simply linear (Fig. 1) and the decrease is driven primarily by women. Despite the complex character relationship of this relationship, it does not seem to stem from the natural evolution of BMI, as a cohort study carried out in UK on 1,003 women found that in 58% of females the BMI increased with age, in 30.6% it did not change and in only 11.4% it diminished with age[38]. It was to be expected, therefore that the majority of females in our study would have an increase in BMI; however, the follow-up period was of only one year and it would be worth examining whether over a longer period the BMI of subjects in our study tends to decrease or at least remain unchanged. A large study on adult subjects of both genders had similar findings with respect to weight loss (not directly BMI, but because they were adults, it is to be expected that changes in weight mirror changes in BMI): for a follow-up period of 13 years, weight loss was only seen in 12.3% and stable weight in 38.3%, whereas the rest of the patients had weight gains[39].

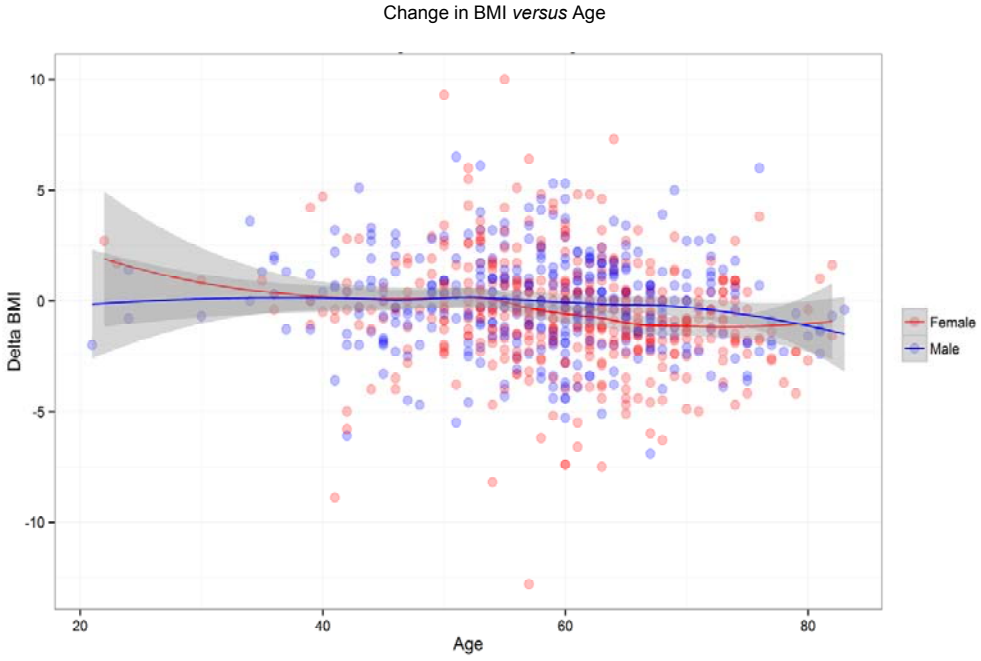

Fig. 1. The relationship between delta BMI and age. The shaded regions correspond to confidence interval for the two losess (local polynomial regression) lines for females and males. More intense colours correspond to several points with identical values.

Urban versus *rural environment*. Although the mean difference between the BMI of patients from rural and urban environments examined at one year from diagnosis is small (31.60 and 31.90 kg/m^2, respectively), looking into the change in BMI showed that in rural patients the mean BMI was almost unchanged (mean of delta BMI 0.07 kg/m^2), whereas it decreased by 0.76 kg/m^2 in those from urban localities ($p < 0.001$, Hedges's $g = 0.36$). Looking in the initial values of BMI, the mean values were 31.52 for rural patients and 32.66 kg/m^2 ($p = 0.01$, Hedges's $g = 0.19$). Studies from other geographic regions and cultures reported different findings for the differences in BMI between the two living environments, rural and urban. A cross-sectional study from Peru has also reported lower BMI for populations from rural environments than those from urban settings, as well as a higher tendency of rural inhabitants to underestimate their weight[40]. Instead, in United States rural adults tended to have a higher frequency of obesity than urban adults, even after controlling for demographic, food and physical exercise variables[41]. In India, lower values were reported for blood pressure, lipid levels and glycemia in rural men but not in rural women, when compared with persons from urban environments[42]. It is not clear why in our study people from urban environments seemed more sensitive to the importance of decreasing BMI than those from rural settings.

Ethnicity. The majority of the patients in the data set were of the Romanian nationality and a small subgroup ($n = 49$) belonged to the Romani minority ethnic group. At the time of diagnosis, the mean BMI was 32.02 in the Romanian patients and 33.26 in the Romani patients, but the difference was not statistically significant ($p = 0.22$). This lack of statistical significance may be related to the low statistical power and an increase in sample size might make it reach the conventional threshold of significance. There were similar findings in connection with the change in BMI along the 12 month period: the mean difference in BMI was of 0.37 kg/m^2 (decrease) in the Romanian subgroup, whereas in the Romani subgroup it increased on average by 0.1 kg/m^2), but the difference was also not statistically different ($p = 0.23$).

Educational status. Observational data suggest interesting and complex relationships between education and BMI, for both men and women, in different countries from different habitats: in countries with lower levels of urbanicity men with higher education tend to have a higher BMI, whereas in the most urban countries (except for Mexico and Brazil) men with higher education have generally a lower BMI, and similar findings were valid for women, where the relationship was stronger[43]. In advanced economies this inverse relationship between education (most often as a proxy for the socioeconomic level) and BMI has been consistently reported in the literature for women, and to a less extent for men[44–50]. In our data set, the relationship between education and BMI was not as strong, although there was a trend in this direction (the mean BMI was 32.66 ± 6.20 kg/m^2 for those reporting no studies, 32.33 ± 6.10 kg/m^2 for those with a low level of education, 31.67 ± 5.74 kg/m^2 in those with medium education and 31.16 ± 4.99 kg/m^2 for those with higher education, $p = 0.08$). With respect to the change (decrease) in BMI at 12 months, the relationship with education was not as straightforward: the largest average loss of BMI (–0.88 ± 2.31 kg/m^2) was indeed registered among the patients with higher education, but the smallest mean change (–0.25 ± 2.42 kg/m^2) was seen in those with a low level of education (not in those with no studies, for which the mean BMI loss over 12 years was –0.51 ± 2.68) ($p = 0.10$) ($\omega 2 = 0.001$). The subgroup of persons with no formal education was (as expected) very low ($n = 16$), and the number of those with higher studies was also relatively small ($n = 48$).

Marital status. No statistically significant difference was found among the patients with different marital status: married, single (not married), widow, divorced, remarried, but except for the married people, who were predominant, the other subgroups were underrepresented and the statistical power was insufficient to allow the detection of potential differences. When pooling widows, divorced and remarried people together with those married ("married extended group"), a difference was seen at the initial time of diagnosis between this group and the single patients in the mean BMI (32.13 and 30.93 kg/m^2), but even so it was not statistically significant ($p = 0.28$), possibly because of the low number ($n = 27$) of single patients in the data set. Earlier publications (in the 1990s) reported that marriage tends to associate with higher body weight, whereas in divorced or widowed people, body weight was found rather to decrease. The majority of later publications tended to emphasize that it is not the marital status per se that is relevant in this respect, but rather the change in marital status[51].

Socioeconomic status. There was a stronger association between the socioeconomic status as measured by the average income and the change in BMI over the 12 month period. At the time of diagnosis no significant difference was found in the BMI between the patients with low and middle-income and those with a high income ($p = 0.87$), which is not the typical pattern for general populations in affluent economies (which is generally the same seen for education, as educational attainment and income are relatively well correlated)[52–53]. However, in the univariate analysis income was shown to be strongly associated with the change in BMI across the one year period: those reporting a low income had a slight increase in the mean BMI (0.08 ± 2.92 kg/m^2), those with a medium income had a decrease in the mean BMI of 0.42 ± 2.12 kg/m^2), and those with a high income had a larger decrease in their mean BMI (0.95 ± 2.38 kg/m^2). This finding is interesting, because it shows that socioeconomic inequalities, although not expressed in the BMI at the time of diagnosis tend to become manifest in adopting measures intended to improve the BMI, but this effect is very small ($\omega^2 = 0.009$).

Smoking status. No significant difference was seen in the BMI at the diagnosis or after 12 months between non-smokers, current smokers and former smokers in our study ($p = 0.16$ for delta BMI, $p = 0.87$ for the initial BMI). A number of studies have shown that smokers tend to have a lower BMI than non-smokers or past smokers, but the difference is relatively small, and thus larger sample size is needed to reach the threshold of statistical significance (for instance, in one large study in Japanese patients the mean BMI of never-smokers was 23.3 kg/m^2, that of current smokers 23.0 kg/m^2 and that of former smokers 23.4 kg/m^2 [54]. Although there are biologically plausible mechanisms to explain a possible weight decrease by smoking, it has also been argued that smoking hinders exercise consecutively to its constraints on respiratory functions and hence the effect of smoking on body weight has been characterized as ambiguous[55].

The fact that in our sample no significant difference was seen in BMI between the three categories may be in line with this ambiguity, indicating that if there are differences in BMI among the three categories, then they are small, but allowance has to be made for confounding factors. Similarly, there is no convincing evidence that smokers or non-smokers had an advantage in decreasing their BMI over a 12-month period. Despite a hypothetical (and controversial) beneficial effect on BMI of smoking, there is convincing evidence that smoking increases the risk of developing T2DM; according to a meta-analysis, if the relationship between smoking and T2DM was causal, it would explain about 11.7% of cases in males and 2.4% in females[56,57].

Alcohol consumption. Unlike smoking, for which no clear pattern could be detected, there was a clear relationship in our sample between alcohol consumption and BMI at the diagnosis, as well as between alcohol consumption and the decrease in BMI over a period of 12 months. In agreement with similar findings in other publications[58,59] (but not in all studies[60]) the mean BMI at diagnosis was lessening with increasing amounts of alcohol consumption, from 33.50 kg/m^2 in non-consumers to 29.99 kg/m^2 in heavy drinkers. The largest mean decreases were observed in patients not consuming alcohol (mean delta BMI 0.54 kg/m^2), followed by patients claiming consumption of small amounts of alcohol (mean delta BMI 0.36 kg/m^2); the decrease in BMI was substantially lower in patients with a moderate of alcohol (0.15 kg/m^2), whereas the mean delta BMI increased by 0.08 kg/m^2 in those with a strong consumption of alcohol ($p = 0.01$, $\omega^2 = 0.04$). As also indicated by ω^2, despite this relatively strong relationship, alcohol consumption only explained less than 1% of the total variance seen in the change in BMI (adjusted $R2 = 0.006$) and considering the observational character of the study and the potential impact of other confounding factors this finding has to be interpreted with caution. It seems reasonable though, to expect more decrease in BMI among the non-drinkers, in whom BMI was higher, than in heavy drinkers, where the BMI was already comparatively lower.

Consumption of fruits and vegetables. The patients reporting daily consumption of fruits and vegetables had a lower mean BMI at diagnosis than the remainder of the patients (mean BMI 31.52 kg/m^2 *versus* 32.53 kg/m^2), although the difference between the two groups was lower than the one between those practicing physical exercise or not. In patients with a regular healthy diet, the mean decrease in BMI tended to be larger than in those without such a diet (0.51 kg/m^2 *versus* 0.21 kg/m^2, $p = 0.07$). There was a certain degree of correlation between physical exercise and the daily consumption of fruits and vegetables, but it was rather limited ($r = 0.10$, $p = 0.006$).

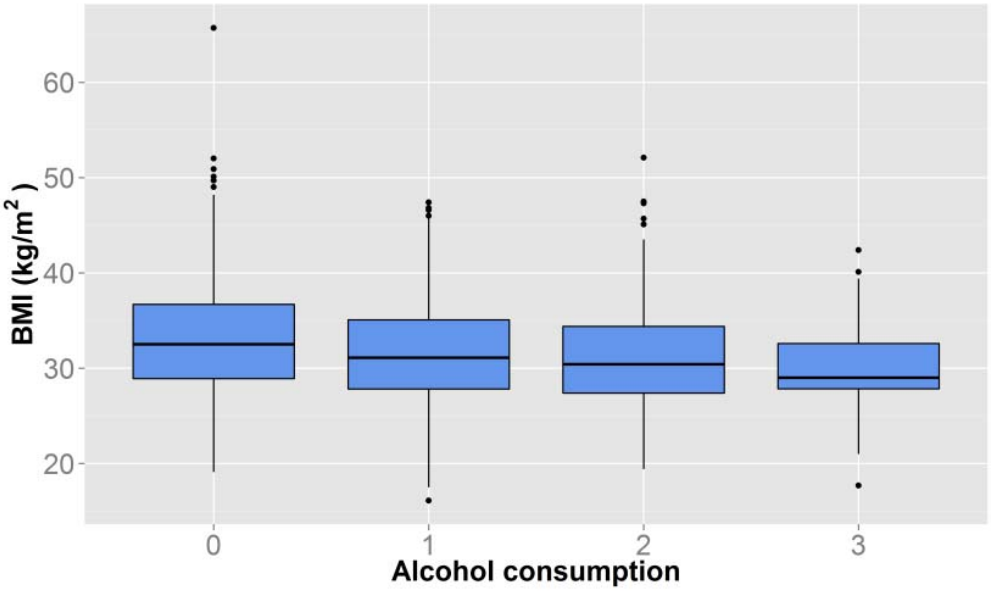

Fig. 2. Variation of BMI by the degree of alcohol consumption (0 – no alcohol consumption; 1 – low level of alcohol consumption; 2 – moderate consumption; 3 – high levels of alcohol consumption).

Physical exercise. At the time of diagnosis, the subgroup of patients that included physical exercise in their lifestyle had a mean BMI of 31.08 kg/m^2, whereas those not practicing physical exercise had a mean BMI of 34.37 kg/m^2 ($p < 0.001$, Cliff's delta 0.30). After 12 months, those already practicing physical exercise had a mean decrease in BMI of only 0.20 kg/m^2, while the other group had a mean decrease in BMI of 0.66 kg/m^2 ($p = 0.01$, Cliff's delta 0.11), suggesting that it is more difficult to achieve decreases in BMI in those patients already having an active life.

Perceived stress. Stress, as perceived by patients was not associated with any significant difference in BMI at the time of diagnosis (31.77 *versus* 32.28 kg/m^2, $p = 0.23$), neither was the decrease in BMI over one year related to stress as perceived by patients (delta BMI 0.31 *versus* 0.36 kg/m^2, $p = 0.75$).

Initial BMI. A high initial BMI tended to associate with more weight loss than a lower initial BMI ($r = –0.29$, $p < 0.001$). This was seen especially when comparing severely obese patients in the dataset (BMI ≥ 40 kg/m^2) with the rest of the patients (BMI < 40 kg/m^2); in the former, the mean decrease in BMI was 1.69 kg/m^2, whereas in the remainder of the patients, it was only about one eighth, 0.20 kg/m^2. In terms of percentages of the initial BMI, in those with severe obesity, the median decrease of BMI was 2.75%, whereas in the rest of the patients it was 1.04% ($p = 0.001$).

Maximum BMI. There was a limited correlation between maximal BMI and the change in BMI over one year: the decrease in BMI tended to be larger in those patients reporting a higher maximal BMI ($r = –0.13$, $p < 0.001$). Similarly to the age, the effect was stronger in females than in males (Fig. 3).

Hypertension. Non-hypertensive patients in our data set had a lower mean BMI (29.27 kg/m^2) at the diagnosis than those with hypertension (32.58 kg/m^2, $p < 0.001$, Cliff's delta 0.35). No significant difference was seen in BMI between the patients suffering of any cardiovascular diseases and those not affected by such diseases (31.92 *versus* 32.48 kg/m^2, $p = 0.24$), nor was any difference seen in the change in BMI at one year between the two categories of patients ($p = 45$). After one year, the median change in BMI in the non-hypertensive group was positive (*i.e.* it increased with 0.4 kg/m^2), while in the hypertensive patients it was negative, with a median decrease of 0.4 kg/m^2 ($p = 0.004$, Cliff's delta 0.17), suggesting that these patients were more motivated than the non-hypertensive ones to lose weight.

Atherogenic dyslipidemia. About three quarter of the patients in the data set (73.10%) had atherogenic dyslipidemia, and only 22.54% did not have such dyslipidemia (for 4.36% data on its presence or absence were not available). The former patients had a higher mean BMI (32.45 kg/m^2) than the latter (31.27 kg/m^2, $p = 0.008$, Cliff's delta 0.13). No relationship was detected between the presence or absence of atherogenic dyslipidemia and the decrease of BMI across one year.

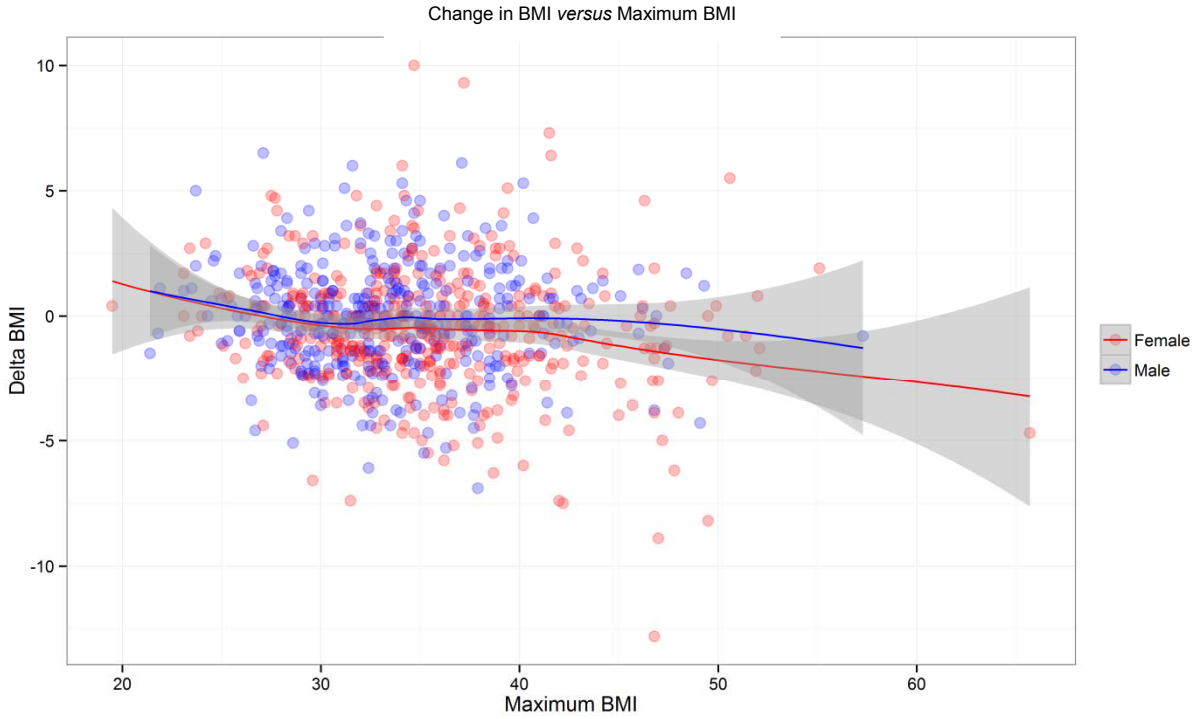

Fig. 3. Variation of change in BMI over one year against maximum BMI for male and female patients.The shaded regions correspond to confidence interval s for the two loess (local polynomial regression) lines for females and males. More intense colors correspond to several points with identical values.

Family diabetes history. The mean BMI of the patients with first degree relatives having a history of type 2 diabetes mellitus (DM2) was higher than that of patients without such relatives (31.71 *versus* 33.20 kg/m^2, p = 0.008, Cliff's delta 0.12). As for second degree relatives, the difference was too small to have any statistical significance (32.00 *versus* 32.44 kg/m^2, p = 0.40). Comparing patients with a family history of DM2 irrespective of degree (1–4), a significantly lower BMI was found in those having relatives with DM2 than in the remainder of the patients (31.53 *versus* 32.85 kg/m^2, p = 0.005, Cliff's delta 0.11). The decrease in BMI at one year tended to be less in those with no family history of DM2 (mean delta BMI 0.22 kg/m^2) than in those with such a history (mean delta BMI 0.50 kg/m^2, p = 0.09, Hedge's g 0.12).

Fetal macrosomia history. A significant difference was seen at the time of diagnosis between the mean BMI of the patients reporting fetal macrosomia (34.19 kg/m^2) and the mean BMI of the remainder of the patients (31.86 kg/m^2, p = 0.003, Cliff's delta 0.21). However, no relationship was evident between the decrease in BMI and the fetal macrosomia status of the patients (mean delta BMI 0.62 and 0.31 kg/m^2 for patients with and without fetal macrosomia, p = 0.29); this lack of statistical significance may be partially related to the relatively small statistical power (n = 80 for patients with fetal macrosomia), but even so, this indicates that the effect of macrosomia on the decrease in BMI at one year is relatively small).

Initial treatment. Excluding the 33 patients in whom treatment was limited to diet, we divided the patients into two groups, according to the effect of the treatment on body weight: those treated with medications considered neutral with respect to weight loss and those treated with medications known to increase body weight (mixed cases were also included in the latter group). The initial mean BMI was 33.55 ± 5.83 kg/m^2 in the first group and 29.62 ± 5.33 kg/m^2 in the second. After one year of treatment the mean BMI decreased in the first group with 1.05 ± 2.04 kg/m^2, whereas in the second the mean BMI increased with 0.91 ± 2.33 kg/m^2 (p < 0.001, Cliff's delta 0.50). 55.42% of all patients in the dataset were treated with a single oral antidiabetic product and 32.75% were treated with two oral antidiabetic medications or more. Also, 1.12% received one insulin product, 3.49% received one insulin besides an oral antidiabetic, 3.11% received two insulins or more, with or without oral antidiabetics and 4.11% were recommended dietary measures only. Both linear and robust regression models (based on Huber's M

estimation method and quantile regression) indicated that patients treated with insulins, with or without oral antidiabetics, had a higher BMI than those treated by diet alone, whereas those treated with a single oral antidiabetic had a lower BMI ($p < 0.001$, adjusted $R2 = 0.15$). Because the subgroups for several treatments were rather small, we also grouped the treatments involving insulins (with or without oral antidiabetics) and associations of two or more oral antidiabetics in a single subgroup (heavily treated/intensified treatment) and compared it with those receiving diet or a single oral medication (lightly treated/conventional treatment); the mean BMI increased in the first group by 0.69 kg/m^2, whereas it decreased in the second by 1.04 kg/m^2 ($p < 0.001$).

Change of treatment. We classified the treatment approach at 6 months by defining three categories of patients: those for whom no change in treatment was recommended, those for whom the treatment was changed upwards (increase of dose, change or addition of another drug product because of insufficient control of glycaemia) and those for whom the treatment was changed downwards (decrease of dose, change or removing of a medicine). The mean BMI of patients for whom the treatment was changed upwards was higher (32,57 kg/m^2) than that of patients whose treatment was kept unmodified (31,75 mg/kg), whereas the mean BMI of patients for whom the treatment was changed downwards was lower (30.10 kg/m^2) than the one of patients whose treatment was kept unmodified ($p = 0.002$, adjusted $R2 = 0.01$).

We compared the characteristics of the patients of the data set analyzed (for which data on BMI after 12 months of treatment were available) and those of the patients for which data on BMI after one year were not available. No significant differences were seen in the distribution by gender, rural or urban environment, smoking status, alcohol consumption, income, ethnicity, marrital status, but the patients for which BMI data were not available tended to be slightly (but significantly) older (mean age 62.08 ± 12.15 *versus* 59.21 ± 9.89 years) and to have lower BMI (29.97 ± 8.70 *versus* 32.09 ± 5.96 kg/m^2) and lower maximal BMI (32.53 ± 9.11 *versus* 34.58 ± 5.77 kg/m^2); there were also small differences regarding the educational status (a slightly lower proportion of persons with medium studies in the patients for which BMI data were not available, but slightly higher proportion of patients with higher education in the same group).

CONCLUSIONS

A multiple regression model ($p < 0.001$, adjusted $R2 = 0.18$) based on backward selection was developed and it included the initial treatment, sex, age at diagnosis, rural or urban environment, income and a family history of diabetes. As in the univariate analysis, the model indicated that heavily treated patients had their BMI increased over one year, whereas those treated with single/dual oral antidiabetic medication had a decrease in BMI. Being male is associated with a tendency toward an increase in BMI, whereas living in an urban environment, having a higher income, having a family history of DM2 and being older tended to associate with a reduction in BMI. Based on the standardized coefficients, the strongest contribution was that of two insulins or more (8.37), followed by that of income (–1.59), whereas the smallest was that of age (–0.008).

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