

- 37 Silventoinen K, Jelenkovic A, Sund R, et al. Genetic and environmental effects on body mass index from infancy to the onset of adulthood: an individual-based pooled analysis of 45 twin cohorts participating in the Collaborative project of Development of Anthropometrical measures in Twins (CODATwins) study. *Am J Clin Nutr* 2016;104:371–9.
- 38 Ventura AK, Birch LL. Does parenting affect children's eating and weight status? *Int J Behav Nutr Phys Act* 2008;5:15.
- 39 Scaglioni S, De Cosmi V, Ciappolino V, et al. Factors influencing children's eating behaviours. *Nutrients* 2018;10:706.
- 40 Wells JCK, Chomtho S, Fewtrell MS. Programming of body composition by early growth and nutrition. *Proc Nutr Soc* 2007;66:423–34.

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## The contribution of obesity to the population burden of high metabolic cardiovascular risk among different ethnic groups. The HELIUS study

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**Background:** The burden of cardiovascular risk is distributed unequally between ethnic groups. It is uncertain to what extent this is attributable to ethnic differences in general and abdominal obesity. Therefore, we studied the contribution of general and abdominal obesity to metabolic cardiovascular risk among different ethnic groups. **Methods:** We used data of 21 411 participants of Dutch, South-Asian Surinamese, African-Surinamese, Ghanaian, Turkish or Moroccan origin in Healthy Life in an Urban Setting (Amsterdam, the Netherlands). Obesity was defined using body-mass-index (general) or waist-to-height-ratio (abdominal). High metabolic risk was defined as having at least two of the following: triglycerides  $\geq 1.7$  mmol/l, fasting glucose  $\geq 5.6$  mmol/l, blood pressure  $\geq 130$  mmHg systolic and/or  $\geq 85$  mmHg diastolic and high-density lipoprotein cholesterol  $< 1.03$  mmol/l (men) or  $< 1.29$  mmol/l (women). **Results:** Among ethnic minority men, age-adjusted prevalence rates of high metabolic risk ranged from 32 to 59% vs. 33% among Dutch men. Contributions of general obesity to high metabolic risk ranged from 7.1 to 17.8%, vs. 10.1% among Dutch men, whereas contributions of abdominal obesity ranged from 52.1 to 92.3%, vs. 53.9% among Dutch men. Among ethnic minority women, age-adjusted prevalence rates of high metabolic risk ranged from 24 to 35% vs. 12% among Dutch women. Contributions of general obesity ranged from 14.6 to 41.8%, vs. 20% among Dutch women, whereas contributions of abdominal obesity ranged from 68.0 to 92.8%, vs. 72.1% among Dutch women. **Conclusions:** Obesity, especially abdominal obesity, contributes significantly to the prevalence of high metabolic cardiovascular risk. Results suggest that this contribution varies substantially between ethnic groups, which helps explain ethnic differences in cardiovascular risk.  
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### Introduction

Type-2 diabetes (T2D) and cardiovascular disease (CVD) often affect ethnic minority groups more than European host populations.<sup>1–3</sup> For example the age-adjusted odds of T2D was 3–12 times higher among ethnic minority groups in the Netherlands relative to the Dutch.<sup>3</sup> Similarly, some ethnic minority groups have shown a particularly high incidence of CVD relative to majority population.<sup>1,2</sup>

Studies suggest that obesity contributes substantially to the high prevalence of CVD and T2D globally via several mechanisms [e.g. by alterations in lipids, blood pressure (BP) and inflammation].<sup>4–7</sup> Estimates of this contribution have varied widely.<sup>8–10</sup> For example, studies have estimated that 3–83% of the prevalence of diabetes, and 7–44% of CVD can be attributed to obesity when obesity is defined

by body-mass-index (BMI).<sup>8–11</sup> Moreover, previous work has suggested that abdominal obesity may be a stronger risk factor for CVD and T2D than general obesity, and may therefore contribute even more to CVD and T2D.<sup>12,13</sup>

High rates of general and abdominal obesity are found in ethnic minority groups.<sup>3,14</sup> Thus, part of the ethnic disparities in the prevalence of CVD and T2D may be attributable to ethnic disparities in obesity. However, the magnitude of the contribution of obesity to cardiovascular risk and, to a lesser extent T2D, is currently unknown among various ethnic minority groups, especially for abdominal obesity. This uncertainty stems from the fact that this contribution does not only depend on ethnic disparities in the prevalence of obesity, but also on disparities in the association between obesity and metabolic risk.<sup>15</sup> The aim of this study is to provide more insight in the contribution of obesity to cardiovascular and T2D

risk. To this end, we used data from the Healthy Life in an Urban Setting (HELIUS) study among participants of several ethnic groups to estimate the contribution of general and abdominal obesity to metabolic risk factors associated with CVD and T2D.

## Methods

The HELIUS study is a large-scale, multi-ethnic cohort study on health and health care utilization among different ethnic groups living in Amsterdam, the Netherlands. The aims and design of the HELIUS study have been published.<sup>16</sup> Briefly, potential participants aged 18–70 years living in Amsterdam were randomly sampled via the municipality register, stratified for ethnicity. Baseline data were obtained in 2011–15 via questionnaires and physical examination. A total of 90 019 subjects were invited. Approximately 55% responded either by response card or after a home visit by an ethnically matched interviewer. Of those, 24 789 agreed to participate, resulting in response rate of 28% (ranging from 21% among Moroccans to 35% among Ghanaians). The study protocols were approved by the AMC Ethical Review Board, and all participants provided written informed consent.

### Ethnicity

Participants' ethnicity was defined according to the country of birth of the participant as well as that of his/her parents [for a full discussion of the concept of ethnicity in the Netherlands (and this study), see Stronks et al.<sup>17</sup>]. Specifically, a participant was considered as of non-Dutch ethnic if he/she fulfilled either of the following criteria: (1) he/she was born abroad and has at least one parent born abroad (first generation); or (2) he/she was born in the Netherlands but both his/her parents were born abroad (second generation). Of the Surinamese immigrants in the Netherlands, approximately 80% are either African origin or South-Asian origin. Surinamese subgroups were classified according to self-reported ethnic origin. For the Dutch sample, we invited people who were born in the Netherlands and whose parents were both born in the Netherlands.

### Anthropometric measures

Weight was measured in light clothing using a Seca 877 digital scale to the nearest 0.1 kg. Height was measured without shoes using a portable stadiometer (Seca 217) to the nearest 0.1 cm in upright position. Waist circumference was measured using a flexible tape measure at the level mid-way between the lower rib margin and the iliac crest. All measures were taken in duplicate and the mean was used in the analyses. If the discrepancy between the duplicate measures differed more than 0.5 cm for height, 0.5 kg for weight or 1 cm for waist circumference, a third measurement was taken. The two measures which were most similar were used to calculate the mean.

General obesity was based on BMI, calculated as weight in kilograms divided by squared height in meters and defined using WHO cut-off values ( $BMI \geq 30 \text{ kg/m}^2$ ) without ethnic-specific cut-off values consistent with current practice in the Netherlands.<sup>18</sup> Abdominal obesity was based on weight-to-height ratio (WHtR), calculated as the waist circumference in centimeters divided by height in centimeters. We used the cut-off value proposed by Ashwell et al.<sup>12</sup>, namely  $WHtR \geq 0.5$ , as there is no WHO cut-off value for obesity based on WHtR. WHtR was chosen as the abdominal obesity measure because, of the abdominal obesity measures, WHtR may be the most robust across ethnic groups.<sup>12</sup>

### Metabolic risk

For this study, we defined high metabolic risk similar to the Adult Treatment Panel III definition of metabolic syndrome [i.e. triglycerides, high-density lipoprotein (HDL) cholesterol, fasting

glucose and BP, but not LDL and smoking], but without the abdominal obesity criterion.<sup>19</sup> Thus, we defined high metabolic risk as having at least two of the following four criteria: high triglycerides ( $\geq 150 \text{ mg/dl}$ ), low HDL cholesterol ( $< 40 \text{ mg/dl}$  for men,  $< 50 \text{ mg/dl}$  for women), high fasting glucose ( $\geq 100 \text{ mg/dl}$ ) and a high BP ( $\geq 130/85 \text{ mmHg}$ ). Using medication related to a criterion was considered as a fulfillment of that criterion.

BP was measured using a validated automated digital BP device (WatchBP Home; Microlife AG) on the left arm in a seated position after the person had been seated for at least 5 min. BP measurements were conducted in duplicate and the average BP was used for analysis. Fasting blood samples were drawn, and lipids and glucose were determined with by enzymatic colorimetric spectrophotometry and enzymatic spectrophotometric (UV) method respectively (Roche Diagnostics, Japan).

### Study population

Baseline data collected by both questionnaire and physical examination were available among 22 165 participants. We excluded participants with a Javanese Surinamese ( $n = 233$ ), 'other/unknown Surinamese' ( $n = 267$ ) or unknown/other ethnic background ( $n = 48$ ) due to small sample sizes. Furthermore, we excluded participants with missing data regarding risk factors used to define high metabolic risk (i.e. triglycerides, HDL, glucose and/or BP,  $n = 177$ ), as well as participants with missing data regarding anthropometric measures ( $n = 29$ ). This resulted in a study population of 21 411 participants.

### Statistical analyses

Ethnic groups may differ in the obesity prevalence and associated metabolic risk. To integrate both aspects, we estimated the population attributable fraction (PAF) of obesity to high metabolic risk. We first conducted Poisson regression analyses to determine the prevalence ratio of obesity to high metabolic risk in each subgroup. These analyses were adjusted for age, and were conducted separately for BMI and WHtR. Next, we estimated the PAF of obesity for high metabolic risk via an adjusted PAF algorithm;  $PAF = P(PR - 1)/PR \times 100$ , where  $P$  is the prevalence of obesity among those with high metabolic risk and  $PR$  is the prevalence ratio of obesity on high metabolic risk, adjusted for age (for a detailed discussion regarding the PAF formula, see Rockhill et al.<sup>20</sup>).

We then estimated the prevalence of obesity-related metabolic risk per ethnic group, separately for general obesity and abdominal obesity. To this end, we first estimated the prevalence of high metabolic risk at the mean age of each subgroup via binary logistic regression. Next, we multiplied this prevalence estimate with the previously calculated PAF to determine the obesity-related prevalence of high metabolic risk.

## Results

Ethnic groups differed in mean age (40.9–48.1 years in men and 39.9–46.1 years in women; table 1). In men, the prevalence of BMI-defined general obesity ranged from 10.1% among Dutch men to 28.1% among Turkish men. In women, the prevalence ranged from 10.1% among Dutch women to 44.4% Ghanaian women. The prevalence of WHtR-defined abdominal obesity was higher and ranged from 54.5% to 79.2% among men and 45.1% to 84.4% among women. The prevalence of high metabolic risk was lowest among the Dutch, especially among women. The pattern and prevalence of individual components used to define high metabolic risk also differed between ethnic groups. For a list of additional cardiovascular risk factors per ethnic group, we refer the reader to Supplementary table S1.

**Table 1** Characteristics (mean (SD) or percentage) of the study population, by ethnicity and sex

	Dutch	South-Asian Surinamese	African Surinamese	Ghanaian	Turkish	Moroccan
<b>Men</b>						
<i>N</i>	2075	1362	1594	897	1618	1504
Age (years)	46.9 (13.8)	44.8 (13.6)	48.1 (12.9)	46.9 (11.5)	40.9 (12.1)	42.1 (12.7)
BMI (kg/m <sup>2</sup> )	25.2 (3.8)	25.83 (4.17)	26.28 (4.14)	26.72 (3.76)	27.85 (4.37)	26.69 (4.01)
Waist-to-height ratio	0.51 (0.07)	0.55 (0.07)	0.52 (0.07)	0.54 (0.07)	0.56 (0.07)	0.54 (0.07)
BMI obesity (%)	10.1	13.7	17.2	17.4	28.1	19.2
WHtR obesity (%)	54.5	74.7	60.6	71.5	79.2	74.2
High metabolic risk (%)	38.2	57.0	39.1	38.9	48.9	40.7
High triglycerides (%)	23.7	40.2	18.1	15.5	35.4	21.4
Low HDL cholesterol (%)	20.9	44.6	21.1	17.8	42.0	31.9
High blood pressure (%)	51.5	58.1	64.9	74.0	49.0	44.1
High glucose (%)	36.2	50.9	35.9	36.1	37.5	41.3
<b>Women</b>						
<i>N</i>	2452	1663	2496	1418	1957	2375
Age (years)	45.6 (14.2)	46.1 (13.2)	47.8 (12.3)	43.4 (10.7)	39.9 (12.1)	39.4 (12.9)
BMI (kg/m <sup>2</sup> )	24.4 (4.5)	26.7 (5.3)	28.8 (5.9)	29.6 (5.3)	29.1 (6.5)	28.1 (5.8)
Waist-to-height ratio	0.50 (0.08)	0.57 (0.09)	0.57 (0.09)	0.58 (0.08)	0.58 (0.08)	0.57 (0.10)
BMI obesity (%)	10.1	23.4	37.7	44.4	40.8	35.2
WHtR obesity (%)	45.1	77.8	75.9	84.4	75.6	73.6
High metabolic risk (%)	18.6	43.1	34.1	28.9	30.5	25.6
High triglycerides (%)	11.7	25.3	12.4	7.3	19.0	11.3
Low HDL cholesterol (%)	18.6	47.1	30.9	21.8	42.5	38.7
High blood pressure (%)	28.4	46.1	57.7	62.2	30.2	24.5
High glucose (%)	16.5	34.8	27.1	23.7	20.9	24.1

Notes: Data are mean (SD) or percentages. BMI, body mass index; SD, standard deviation; WHtR, waist to height ratio; HDL, high-density lipoprotein cholesterol.

Among men, the contribution of general obesity to high metabolic risk ranged from 7.1% among South-Asian Surinamese to 17.8% among Turkish men, vs. 10.1% among the Dutch (table 2). The contribution of abdominal obesity to high metabolic risk was higher than the contribution of general obesity ranging from 52.1% among African Surinamese to 92.3% among Moroccans, vs. 53.9% among the Dutch. Among women, we observed a more heterogeneous contribution of general obesity to high metabolic risk; among South-Asian Surinamese, African Surinamese and Ghanaian women, this contribution varied between 14.6% and 26.6% vs. 20% among the Dutch, whereas among Turkish and Moroccan women this contribution was substantially higher (41.2% and 41.8%, respectively). The contribution of abdominal obesity to high metabolic risk showed a similar pattern, varying between 68.0% and 73.9% among South-Asian Surinamese, African Surinamese and Ghanaian women vs. 72.1% among the Dutch, whereas this contribution among Turkish and Moroccan women was 82.3% and 92.8%, respectively. We then estimated the potentially achievable health gain in the population in each ethnic group if all participants were to have non-obese levels. Adjusted for age, the prevalence of high metabolic risk varied between 32% and 59% across the ethnic minority men vs. 33% among Dutch men (figure 1, upper panels). If all men were to be non-obese based on general obesity, the prevalence of high metabolic risk would be 28% to 54% among ethnic minority men vs. 30% among the Dutch. If all men were to be non-obese based on abdominal obesity, the prevalence of metabolic risk would be 3% to 27% among ethnic minority men vs. 15% among the Dutch.

Among ethnic minority women, the age-adjusted prevalence of high metabolic risk varied between 24% and 35% vs. 12% among the Dutch (figure 1, lower panels). If all ethnic minority women were to be non-obese based on general obesity, the prevalence of high metabolic risk would be 16% to 30% among ethnic minority women vs. 10% among the Dutch. If all ethnic minority women were to be non-obese based on abdominal obesity, the prevalence of metabolic risk would be 2% to 9% vs. 3% among the Dutch).

## Discussion

### Key findings

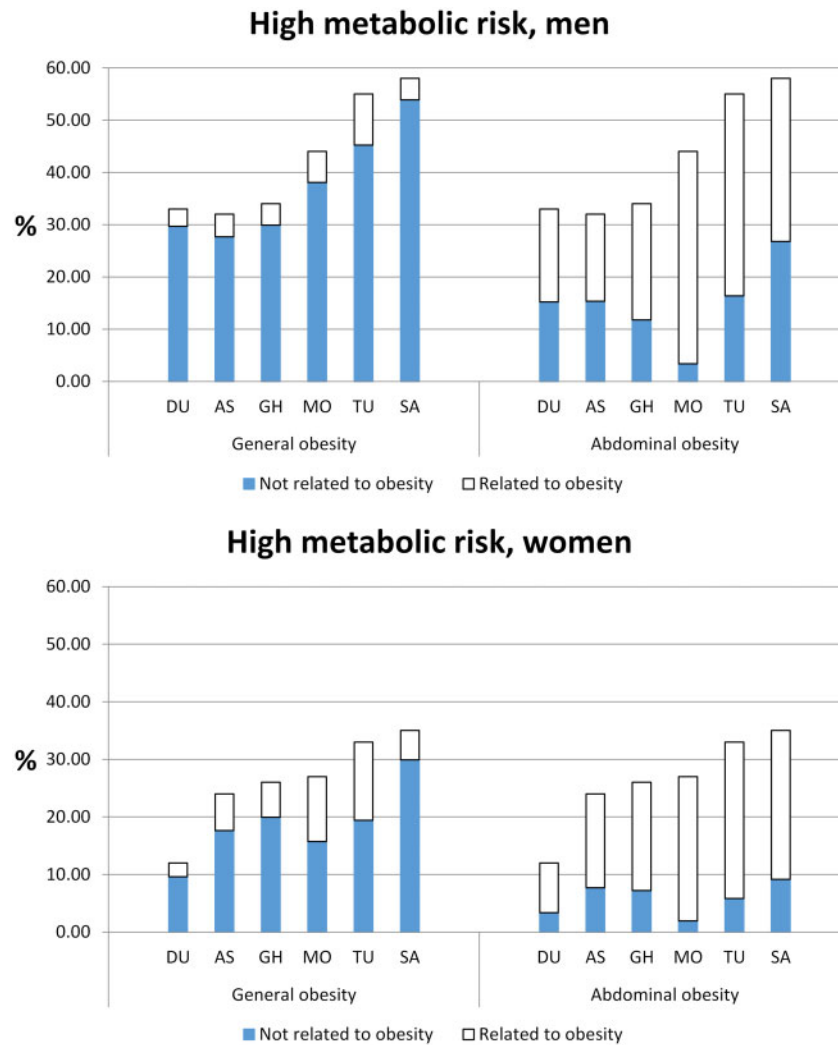
Obesity, especially abdominal obesity, contributes substantially to the prevalence of high metabolic risk. Our results suggest that this contribution is generally higher among ethnic minority groups than among the Dutch majority population. Hence, reducing the prevalence of obesity, particularly abdominal obesity, may reduce the prevalence of high metabolic risk among all ethnic groups and reduce some of the metabolic risk differences between ethnic minority groups and the Dutch.

### Evaluation of potential limitations

As with all cohort studies, some selection bias may have occurred due to non-response. The data that was available among non-responders showed only small SES and age differences between responders and non-responders.<sup>12</sup> Although SES and age are known to be related to metabolic health, this non-response data did not include measures regarding CVD risk or adiposity. So selection bias is less likely, but we cannot truly evaluate whether selection bias occurred and, if so, how this bias has affected our results.

Due to the cross-sectional design, causal inferences regarding obesity and high metabolic risk should be made with caution. Although it is widely assumed that a causal relation between fat accumulation and metabolic disease exists, a high occurrence of metabolic risk factors may also affect susceptibility for weight gain and obesity.<sup>21,22</sup> If so, this may have led to an overestimation of the contribution of obesity to the prevalence of high metabolic risk, and the potential health gain related to weight loss.

We used measures of prevalent cardiovascular risk, based on components of the metabolic syndrome, as a proxy for overall cardiovascular risk. Although high metabolic risk can be considered an inferior outcome measure, the association between components of metabolic syndrome and CVD has been well established.<sup>23</sup> Nevertheless, this association may differ between



**Figure 1** Prevalence of high metabolic risk related and not related to obesity

**Notes:** The prevalence of high metabolic risk by ethnicity and sex, adjusted for age, and split for obesity related and obesity unrelated prevalence based on general obesity (body mass index (BMI)  $\geq 30\text{kg/m}^2$ ), or abdominal obesity (waist-to-height ratio (WHtR)  $\geq 0.5$ ). DU, Dutch; AS, African Surinamese; GH, Ghanaian; MO, Moroccan; TU, Turkish; SA, South-Asian Surinamese

ethnic groups, for example due to ethnic disparities in the age-of-onset of these risk factors.<sup>3</sup> Thus, it would be of value to determine, in future studies, how the contribution of obesity to metabolic and CVD disease incidence varies between ethnic groups.

Obesity was measured using anthropometric measures. More sophisticated measures to determine adiposity mass and distribution (e.g. Dual-Energy X-Ray Absorptiometry) would be preferable because these measures may be more accurate and may better reflect ethnic variations in fat-distributions.<sup>24</sup> However, these measures are impractical for both large cohort studies and daily clinical practice.

In order to estimate the population contribution of obesity to high metabolic risk, we did not exclude participants with prior CVD. However, our results are similar after exclusion of participants with prior CVD, suggesting that our results are also applicable to a strictly primary prevention setting (Supplementary table S2).

### Discussion of key findings

Although the contribution of general obesity to high metabolic risk was similar between most ethnic groups, the basis of these contributions did differ, with the Dutch showing a relatively low prevalence, but relatively strong association between general

obesity and high metabolic risk, whereas the ethnic minority groups showed a weaker association and a higher prevalence of general obesity. This is in accordance with previous studies which also reported higher prevalence rates of general obesity, but weaker associations between general obesity and cardiovascular metabolic disease among ethnic minority groups relative to ethnic majority groups.<sup>25,26</sup> Our results suggest that, despite a similar contribution of general obesity to high metabolic risk, a similar absolute reduction in the prevalence of general obesity may not result in a similarly strong reduction of metabolic risk among all ethnic minority groups. Thus, among ethnic minority groups, relatively large reductions in the prevalence of obesity prevalence may be necessary to reduce disparities in high metabolic risk.

Due to ethnic differences in fat accumulation, distribution and in the associations between obesity and disease, it has been suggested to apply different obesity thresholds for different ethnic groups, especially for BMI.<sup>15,27–29</sup> Controlling for these differences by applying lower, ethnic-specific BMI cut-off values did not strongly affect our results regarding ethnic disparities in the contribution of general obesity to high metabolic risk (results not shown), suggesting that ethnic disparities in fat distribution do not contribute substantially to our results.

**Table 2** Age adjusted estimated contribution of general obesity or abdominal obesity to high metabolic risk

	General obesity (BMI) <sup>a</sup>			Abdominal obesity (WtHR) <sup>b</sup>		
	P <sup>c</sup>	PR <sup>d</sup>	PAF <sup>e</sup>	P <sup>c</sup>	PR <sup>d</sup>	PAF <sup>e</sup>
<b>Men</b>						
Dutch	21.2	1.9 (1.6; 2.3)	10.1	82.2	2.9 (2.4; 3.5)	53.9
South-Asian Surinamese	19.9	1.6 (1.3; 1.9)	7.1	90.6	2.4 (1.9; 3.2)	53.8
African Surinamese	28.9	1.9 (1.6; 2.2)	13.5	83.8	2.6 (2.1; 3.3)	52.1
Ghanaian	28.7	1.7 (1.4; 2.2)	12.1	92.0	3.5 (2.3; 5.2)	65.4
Turkish	42.9	1.7 (1.5; 2.0)	17.8	95.1	3.8 (2.7; 5.3)	70.2
Moroccan	31.9	1.7 (1.5; 2.1)	13.5	92.3	3.0 (2.2; 4.1)	92.3
<b>Women</b>						
Dutch	31.4	2.8 (2.3; 3.4)	20.0	88.1	5.5 (4.1; 7.4)	72.1
South-Asian Surinamese	37.1	1.7 (1.4; 1.9)	14.6	96.1	4.3 (2.9; 6.4)	73.9
African Surinamese	57.9	1.8 (1.6; 2.1)	26.6	94.5	3.6 (2.6; 4.8)	68.0
Ghanaian	62.1	1.6 (1.3; 2.0)	23.8	97.6	3.9 (2.0; 7.3)	72.3
Turkish	71.3	2.4 (2.0; 2.9)	41.2	97.3	6.5 (3.9; 10.8)	82.3
Moroccan	69.7	2.5 (2.1; 3.0)	41.8	99.0	16.0 (7.1; 36.0)	92.8

Notes: <sup>a</sup>General obesity is defined as a body mass index (BMI)  $\geq 30$  kg/m<sup>2</sup>.

<sup>b</sup>Abdominal obesity is defined as a waist-to-height ratio (WtHR)  $\geq 0.5$ .

<sup>c</sup>Prevalence of obesity among participants with high metabolic risk.

<sup>d</sup>Age-adjusted prevalence ratio (PR) and 95% confidence interval of high metabolic risk between obese and non-obese.

<sup>e</sup>Population attributable fraction (PAF), calculated as  $P((PR-1)/PR) \times 100$ .

In our study, general obesity contributed between 7.1% and 26.6% to high metabolic risk for most ethnic groups. This contribution is similar to contributions found in earlier studies among the general population in several countries.<sup>8–10</sup> For example, a study from Australia among men and women from the general population found that BMI-defined obesity contributed to 15.7% of all cases of hypertension, 32.4% of all cases of diabetes and 18.8% of all cases of dyslipidemia.<sup>10</sup>

Turkish and Moroccan women showed a much stronger contribution of general obesity to high metabolic risk (41.1% and 41.8%, respectively) than women in the other ethnic groups. Earlier studies already found a particularly high prevalence of dyslipidemia among Turkish and Moroccans relative to other ethnic groups.<sup>30</sup> Our results suggest that obesity contributes substantially to this relatively high prevalence of dyslipidemia and overall metabolic risk.

For WtHR-defined obesity, we found that contributions to high metabolic risk varied between 52.1% and 92.8%. Earlier studies have reported lower contributions of abdominal obesity to the prevalence of cardiovascular risk at population level.<sup>31,32</sup> For example, an Australian study on type 2 diabetes, low HDL, increased triglycerides and hypertension found that abdominal obesity contributed to 17–38% of these risk factors among men and 30–47% among women.<sup>31</sup> Our reported contributions were higher, in part because our participants were from a different ethnic group with a higher prevalence of abdominal obesity. However, this does not explain the higher contribution among the Dutch. Alternatively, these differences may be related to the use of waist circumference to define abdominal obesity in the previous study, as waist circumference may be associated less strongly with metabolic risk factors relative to WtHR.<sup>12</sup>

Among some ethnic groups (e.g. Dutch, South-Asian Surinamese), the contribution of abdominal obesity to high metabolic risk varied between 50% and 80% whereas among the Moroccan ethnic group (and, to a lesser extent, Turkish women) this contribution was substantially higher. Thus, only for some ethnic groups, (factors related to) abdominal obesity explain almost all of the high metabolic risk. It is unclear why this is the case only for some ethnic groups.

Guidelines promote several strategies to reduce the prevalence of obesity at both population and individual level.<sup>33,34</sup> In multi-ethnic settings, it may be possible to increase the effectiveness of these strategies by adapting these strategies to the specific ethnic groups

and, possibly, initiating such preventive interventions from a younger age than among the ethnic majority group.<sup>3,35</sup> This may not only be beneficial due to ethnic differences in the prevalence and determinants of obesity, but also because the preferred interventions to reduce weight may differ between ethnic groups (e.g. for cultural reasons) and these interventions may differ in the effectiveness for reduction of metabolic risk.<sup>36</sup>

## Conclusions

Obesity contributes substantially to cardiovascular risk across ethnic groups in the Netherlands. Reducing the prevalence of obesity, in particular abdominal obesity, could potentially reduce both the risk of CVD in all populations and may affect ethnic disparities in cardiovascular risk.

## Supplementary data

Supplementary data are available at *EURPUB* online.

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*Conflicts of interest:* None declared.

## Key points

- High metabolic risk is particularly common among Turkish and South-Asian Surinamese.

- Obesity contributes to high metabolic risk among all ethnic groups.
- This contribution is much higher for abdominal obesity than general obesity.
- This contribution is particularly high among Turkish and Moroccans.
- Reducing obesity may affect ethnic disparities in metabolic risk.

## References

- 1 van Oeffelen AA, Agyemang C, Stronks K, et al. Incidence of first acute myocardial infarction over time specific for age, sex, and country of birth. *Neth J Med* 2014;72:20–7.
- 2 Agyemang C, van Oeffelen AA, Norredam M, et al. Ethnic disparities in ischemic stroke, intracerebral hemorrhage, and subarachnoid hemorrhage incidence in the Netherlands. *Stroke* 2014;45:3236–42.
- 3 Snijder MB, Agyemang C, Peters RJ, et al. Case finding and medical treatment of type 2 diabetes among different ethnic minority groups: the HELIUS study. *J Diabetes Res* 2017;2017:1.
- 4 Smith U. Abdominal obesity: a marker of ectopic fat accumulation. *J Clin Invest* 2015;125:1790–2.
- 5 Kachur S, Lavie CJ, de Schutter A, et al. Obesity and cardiovascular diseases. *Minerva Med* 2017;108:212–28.
- 6 Saltiel AR, Olefsky JM. Inflammatory mechanisms linking obesity and metabolic disease. *J Clin Invest* 2017;127:1–4.
- 7 Van Gaal LF, Mertens IL, De Block CE. Mechanisms linking obesity with cardiovascular disease. *Nature* 2006;444:875–80.
- 8 Jiang Y, Chen Y, Mao Y, et al. The contribution of excess weight to prevalent diabetes in Canadian adults. *Public Health* 2008;122:271–6.
- 9 Dal Grande E, Gill T, Wyatt L, et al. Population attributable risk (PAR) of overweight and obesity on chronic diseases: south Australian representative, cross-sectional data, 2004–2006. *Obes Res Clin Pract* 2009;3:1–IV.
- 10 Tanamas SK, Permatasari V, Ng WL, et al. Estimating the proportion of metabolic health outcomes attributable to obesity: a cross-sectional exploration of body mass index and waist circumference combinations. *BMC Obes* 2016;3:4.
- 11 Flegal KM, Panagiotou OA, Graubard BI. Estimating population attributable fractions to quantify the health burden of obesity. *Ann Epidemiol* 2015;25:201–7.
- 12 Ashwell M, Gunn P, Gibson S. Waist-to-height ratio is a better screening tool than waist circumference and BMI for adult cardiometabolic risk factors: systematic review and meta-analysis. *Obes Rev* 2012;13:275–86.
- 13 Cheong KC, Ghazali SM, Hock LK, et al. The discriminative ability of waist circumference, body mass index and waist-to-hip ratio in identifying metabolic syndrome: variations by age, sex and race. *Diabetes Metab Syndr* 2015;9:74–8.
- 14 Modesti PA, Bianchi S, Borghi C, et al. Cardiovascular health in migrants: current status and issues for prevention. A collaborative multidisciplinary task force report. *J Cardiovasc Med (Hagerstown)* 2014;15:683–92.
- 15 Ntuk UE, Gill JM, Mackay DF, et al. Ethnic-specific obesity cutoffs for diabetes risk: cross-sectional study of 490, 288 UK biobank participants. *Diabetes Care* 2014;37:2500–7.
- 16 Snijder MB, Galenkamp H, Prins M, et al. Cohort profile: the healthy life in an urban setting (HELIUS) study in Amsterdam, The Netherlands. *BMJ Open* 2017;7:e017873.
- 17 Stronks K, Kulu-Glasgow I, Agyemang C. The utility of ‘country of birth’ for the classification of ethnic groups in health research: the Dutch experience. *Ethn Health* 2009;14:255–69.
- 18 Cardiovasculaire risicomanagement (Tweede herziening), 2012 (1 January 2016, date last accessed).
- 19 Grundy SM, Brewer HB Jr, Cleeman JI, et al. Definition of metabolic syndrome: report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition. *Arterioscler Thromb Vasc Biol* 2004;24:e13–18.
- 20 Rockhill B, Newman B, Weinberg C. Use and misuse of population attributable fractions. *Am J Public Health* 1998;88:15–19.
- 21 Landsberg L, Aronne LJ, Beilin LJ, et al. Obesity-related hypertension: pathogenesis, cardiovascular risk, and treatment—a position paper of the Obesity Society and The American Society of Hypertension. *Obesity (Silver Spring)* 2013;21:8–24.
- 22 Langley-Evans SC. Nutrition in early life and the programming of adult disease: a review. *J Hum Nutr Diet* 2015;28(Suppl 1):1–14.
- 23 O’Neill S, O’Driscoll L. Metabolic syndrome: a closer look at the growing epidemic and its associated pathologies. *Obes Rev* 2015;16:1–12.
- 24 Seabolt LA, Welch EB, Silver HJ. Imaging methods for analyzing body composition in human obesity and cardiometabolic disease. *Ann N Y Acad Sci* 2015;1353:41–59.
- 25 Jackson CL, Wang NY, Yeh HC, et al. Body-mass index and mortality risk in U.S. blacks compared to whites. *Obesity (Silver Spring)* 2014;22:842–51.
- 26 Taylor HA Jr, Coady SA, Levy D, et al. Relationships of BMI to cardiovascular risk factors differ by ethnicity. *Obesity (Silver Spring)* 2010;18:1638–45.
- 27 Kohli S, Sniderman AD, Tchernof A, et al. Ethnic-specific differences in abdominal subcutaneous adipose tissue compartments. *Obesity (Silver Spring)* 2010;18:2177–83.
- 28 Low S, Chin MC, Ma S, et al. Rationale for redefining obesity in Asians. *Ann Acad Med Singapore* 2009;38:66–9.
- 29 Consultation WHOE. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet* 2004;363:157–63.
- 30 Perini W, Snijder MB, Peters RJG, et al. Ethnic disparities in estimated cardiovascular disease risk in Amsterdam, the Netherlands: the HELIUS study. *Neth Heart J* 2018;26:252–62.
- 31 Cameron AJ, Dunstan DW, Owen N, et al. Health and mortality consequences of abdominal obesity: evidence from the AusDiab study. *Med J Aust* 2009;191:202–8.
- 32 Xue H, Wang C, Li Y, et al. Incidence of type 2 diabetes and number of events attributable to abdominal obesity in China: a cohort study. *J Diabetes* 2016;8:190–8.
- 33 Piepoli MF, Hoes AW, Agewall S, et al. 2016 European Guidelines on cardiovascular disease prevention in clinical practice: the Sixth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (constituted by representatives of 10 societies and by invited experts) developed with the special contribution of the European Association for Cardiovascular Prevention & Rehabilitation (EACPR). *Eur Heart J* 2016;37:2315–81.
- 34 Jensen MD, Ryan DH, Apovian CM, et al. 2013 AHA/ACC/TOS guideline for the management of overweight and obesity in adults: a report of the American college of cardiology/American heart association task force on practice guidelines and the obesity society. *J Am Coll Cardiol* 2014;63:2985–3023.
- 35 Perini W, Snijder MB, Agyemang C, et al. Eligibility for cardiovascular risk screening among different ethnic groups: the HELIUS study. *Eur J Prev Cardiol* 2019;2047487319866284.
- 36 Church TS, Blair SN, Cocroham S, et al. Effects of aerobic and resistance training on hemoglobin A1c levels in patients with type 2 diabetes: a randomized controlled trial. *JAMA* 2010;304:2253–62.

# The effect of prenatal maternal physical activity and lifestyle in perinatal outcome: results from a Greek study

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**Background:** Several maternal characteristics, including lifestyle, have been associated with perinatal outcomes and birth anthropometric characteristics of the offspring. This study aimed to identify whether physical activity (PA) and other lifestyle parameters of the mother are associated with the pregnancy outcomes or with the infant's birth anthropometric characteristics. **Methods:** Participants were recruited in Mitera Maternity Hospital, Athens, Greece. Socio-demographic, medical history and anthropometric assessment took place. PA during pregnancy was assessed with the Pregnancy Physical Activity Questionnaire. Dietary assessment was conducted with the Food Frequency Questionnaire and adherence to the Mediterranean diet was evaluated with the MedDiet score. Birth weight and gestational age data were also collected. **Results:** Sedentary-intensity activity scores increased with increased educational level, while moderate-intensity activity scores decreased with increased educational level. Pregnant women who delivered large for gestational age infants had lower sports activity score. Higher vigorous and sports activity score was demonstrated in cases with a vaginal delivery compared with caesarean section. PA score was significantly and positively correlated with several nutrient intakes and PA was higher in women with a healthier nutritional pattern. Increased MedDiet scores were found in mothers with increased educational level. **Conclusions:** Overall, PA was higher in women with a healthier nutritional pattern. An increased vs. a low activity level during pregnancy is positively associated with vaginal delivery and with the size of the offspring.

## Introduction

Intrauterine growth and development is considered a vulnerable process and may have a significant influence in later life of the offspring. The birth weight of a neonate is a reliable index of intrauterine growth and a sensitive predictor of its chances of survival, growth and long-term physical and psychosocial development.<sup>1</sup> Pregnancy malnutrition, excessive weight gain during pregnancy and low birth weight are associated with perinatal morbidity and predisposition to diabetes and cardiovascular disease in adulthood.<sup>2,3</sup>

Several maternal characteristics (i.e. height, pregravid weight, weight gain during pregnancy, education) have been associated with perinatal outcomes, such as foetal growth, considered as excellent predictors of neonatal size.<sup>4</sup> Mother's body weight is one of the major factors. As such, obese women are more likely to give birth to children with larger body size, even when they have lower average pregnancy weight gain and normal glucose tolerance.<sup>5</sup> Also, physical activity (PA) during pregnancy seems to influence perinatal outcomes. Weight-bearing exercise of moderate-intensity enhances foetoplacental growth<sup>6</sup> and decreases the risk of chronic diseases for both mother and child.<sup>7</sup> Exercise performed during pregnancy has been shown to shift the gestational age distribution slightly upward resulting in reduced preterm births and slightly increased post-term births.<sup>8</sup> In a randomized clinical trial, previously sedentary women who began exercising at 12th to 14th week improved fitness and birth outcomes.<sup>9</sup> Added to PA, the nutritional status of the mother plays an important role in child's health. Poor maternal nutritional status is related to adverse birth outcomes, such

as low birth weight, preterm birth and intrauterine growth restriction.<sup>2</sup>

The aim of this study was to identify whether PA and nutrition of the mother (daily nutrient intakes and the level of adherence to Mediterranean diet) is associated with the pregnancy outcomes or with the infant's birth anthropometric characteristics.

## Methods

### Study participants

This is a descriptive epidemiology approach on a Greek sample of women postpartum. The study design was approved by the Bioethics Committee of Mitera Maternity Hospital, Athens, Greece. All participants were recruited between December 2016 and July 2017. Parents were approached postnatal by a research assistant with experience in interview administration and data collection. Selection criteria used to determine eligibility were: age >18 years, Greek ethnicity and use of Greek language. Women were ineligible if they had serious complications of pregnancy and surgical complications (e.g. severe bleeding, any infection, etc.) or multiple pregnancies. After obtaining a written informed consent, a planned interview followed to collect all data. If parents were unable to attend, data were collected via telephone interviews.

### Socio-demographic, medical history and anthropometric assessment

Socio-demographic data, parental weight and height and perinatal data were reported by the parents or taken from the children's birth