Published in final edited form as:
*Ann Epidemiol*. 2010 December ; 20(12): 883–889. doi:10.1016/j.annepidem.2010.05.002.

# SHORT SLEEP DURATION IS ASSOCIATED WITH THE DEVELOPMENT OF IMPAIRED FASTING GLUCOSE: THE WESTERN NEW YORK HEALTH STUDY

**Lisa Rafalson, PhD**[1], **Richard P. Donahue, PhD, MPH**[2], **Saverio Stranges, MD, PhD**[3], **Michael J. LaMonte, PhD, MPH**[2], **Jacek Dmochowski, PhD**[4], **Joan Dorn, PhD**[2,5], and **Maurizio Trevisan, MD, MS**[2,6]

[1]Department of Family Medicine, School of Medicine and Biomedical Sciences, State University of New York, University at Buffalo, Buffalo, NY, USA

[2]Department of Social and Preventive Medicine, School of Public Health and Health Professions, State University of New York University at Buffalo, Buffalo, NY, USA

[3]Health Sciences Research Institute, University of Warwick Medical School, Coventry, United Kingdom

[4]Department of Mathematics and Statistics, University of North Carolina at Charlotte, Charlotte, NC

[5]Department of Exercise and Nutrition Sciences, School of Public Health and Health Professions, State University of New York University at Buffalo, Buffalo, NY, USA

[6]Health Sciences System, Nevada System of Higher Education, Las Vegas, NV, USA

## Abstract

**Purpose—**To examine whether sleep duration was associated with incident impaired fasting glucose (IFG) over six years of follow-up in the Western New York Health Study.

**Methods—**Participants (n= 1,455, 68% response rate) who were free of type 2 diabetes and known cardiovascular disease at baseline (1996-2001) were reexamined in 2003-2004. A nested case-control study was conducted. Cases had fasting plasma glucose (FPG) < 100 mg/dl at baseline and 100 to 125 mg/dl at follow-up: controls (n=272) had FPG <100 mg/dl at both exams. Cases (n=91) were individually matched to three controls (n=272) on sex, race, and year of study enrollment. Average sleep duration was categorized as short < 6h, mid-range 6 to 8h, and long-sleep > 8h.

**Results—**In multivariate conditional logistic regression after adjustment for several diabetes risk factors the Odds Ratio (OR) of IFG among short sleepers was 3.0 (95% CI 1.05, 8.59) compared to mid-range sleepers. There was no association between long sleep and IFG: OR 1.6 (95% CI: 0.45.-5.42). Adjustment for insulin resistance attenuated the association only among short sleepers OR 2.5 (95% CI: 0.83, 7.46).



Corresponding author: Lisa Rafalson PhD, Department of Family Medicine, School of Medicine and Biomedical Sciences, State University of New York at Buffalo, ECMC, 462 Grider Street, Buffalo, NY 14215, (716) 898-5195, rafalson@buffalo.edu.

DISCLOSURE: NONE

**Conclusion—**Short sleep duration was associated with an elevated risk of IFG. Insulin resistance appears to mediate this association.

### Keywords

impaired fasting glucose; glucose metabolism; sleep duration; nested case-control study; population-based

## INTRODUCTION

Sleep is a restorative process required for metabolic homeostasis (1). Although sleep is necessary for normal homeostatic function, the amount one gets is in part discretionary. In the United States, the average number of hours of sleep for adults has declined since the mid 1900s from about 9 hours a night to current estimates of 7 hours a night (2;3;4) Observational epidemiologic studies have shown that inadequate sleep is associated with obesity(5), hypertension (6), coronary heart disease (7), and overall mortality(8;9).

Prospective studies have demonstrated a U or J-shaped association between sleep duration and incident type 2 diabetes mellitus. For example, after 12 years of follow-up in 1100 men 40-70 years of age, the Massachusetts Male Aging Study reported the relative risk of developing type 2 diabetes: compared to people who slept 7h a night, participants who slept 6h had a relative risk (RR) of 1.95 (95% CI:1.06-3.58) and those who slept >8h had a RR of 3.12 (95% CI: 1.53-6.37) (10). In the 10 year follow-up, the Nurse's health study (n=70,026 females, age 30-55 years) reported an elevated risk of developing type 2 diabetes among long sleepers (≥9h) RR: 1.29 (95% CI: 1.05-1.59) compared to sleeping 8h a night, but not among short sleepers (11). Cross-sectional studies have yielded similar results (12;13).

The Western New York Follow-up Study is a prospective, community-based cohort of men and women that was designed to examine biomarkers that predicted incident type 2 diabetes. Within the prospective follow-up we conducted an individually matched, nested, case-control study to test the hypothesis that short sleep duration at baseline would be associated with an increased likelihood of developing impaired fasting glucose (IFG) independent of diabetes risk factors and several confounding variables. We also examined the role of insulin resistance on this association.

## MATERIALS AND METHODS

### Study Population

The study design and methodology of the original Western New York Health Study have been previously published (14). Participants in this report were originally enrolled as healthy control participants in the Western New York Health Study, an epidemiologic case–control investigation of patterns of alcohol intake and coronary heart disease in Erie and Niagara Counties, New York, conducted from 1996 to 2001. In brief, the initial control cohort was randomly selected from drivers' license and Health Care Finance Administration lists. Eligible participants for the current study were male and female control subjects aged 35–79 years selected from the baseline examination without known clinical cardiovascular disease (self-report) or type 2 diabetes (fasting plasma glucose >125 mg/dl or self-report) and who were capable of completing the study protocol (n = 2,652). Exclusion criteria included self-report of any medical condition that would prohibit participation (e.g., all cancers except skin cancer, type 1 diabetes, physical or mental impairment that would prevent completion of the protocol (n=165), permanent change in residence to out of state (n=39), deceased (n=67), or inability to contact and determine eligibility (n=242). This left 2,139 persons eligible, of whom 1,455 (68.0%) completed the full clinical follow-up examination in 2003–2004. The mean follow-

up time was 5.9 ± 0.8 years Of the 1,455, 528 had prevalent IFG and were therefore not considered in this analysis. This left 927 subjects available who were normoglycemic at baseline of whom 91 cases were identified for matching to n=272 controls. Subjects who developed type 2 diabetes were not included in this report because at baseline only n=6 were "at risk" (normoglycemic): the majority had IFG at baseline.

Compared to persons who declined to participate, those who completed the follow-up examination were less likely to report being a smoker at baseline and were more likely to have completed high school. There were no significant differences in fasting blood glucose concentrations, BMI or sex ratio (data not presented). The protocol was approved by the University at Buffalo Health Science Institutional Review Board and all participants provided written informed consent prior to participation.

## Study protocol

At both the baseline and six-year follow-up examinations, all participants completed a clinical examination that included measures of resting blood pressure, height, weight, and abdominal height according to standardized protocols (15). Blood pressure was measured according the American Heart Association's recommendations. The mean of the second and third measures were used in the analyses. Hypertension was defined as blood pressure ≥ 140/90 mmHg or regular use of antihypertensive medication (16). Study subjects provided a fasting blood sample (at least 10 h overnight) and were asked to refrain from smoking or vigorous physical activity for 24 h prior to their exam. Several standardized questionnaires were administered including cigarette and alcohol use, general health and well-being, personal and family health history, and socioeconomic status. Current alcohol consumption was defined as a person who had consumed at least one alcoholic beverage in the 30 days preceding the interview. Smoking status was categorized as ever smoker (current within the past 30 days or former smoker) or never smoker. Physical activity and sleep duration were ascertained with the Stanford Seven –Day Physical Activity Recall questionnaire (17). Physical activity in the last week was calculated as the number of hours/day in activities that were moderate or vigorous intensity. Sleep duration was ascertained by posing the question, "On average, how many hours did you sleep each night during the last five weekday nights (Sunday-Thursday)?" Response categories were collapsed into three groups: short sleep duration (<6 hours), mid-range or reference group (6-8hours), and long sleep duration (>8 hours). This classification is consistent with the classifications used in previous studies on the health effects of habitual sleep duration (6;12; 13). The presence of depressive symptoms was assessed by the Center for Epidemiologic Studies Depression Scale (CES-D) using the cut point ≥ 16 to identify depression (18). A family history of type 2 diabetes was defined as a positive report in a first-degree relative. Homeostasis model assessment insulin resistance was calculated as fasting insulin × fasting glucose / 22.5 (19).

## Case and Control Definitions

For this nested case-control study a case of IFG was defined as an individual with fasting plasma glucose (FPG) <100 mg/dl at the baseline examination and between 100 and 125 mg/dl at the follow-up examination: 91 cases were identified for matching at follow-up. Each case was individually matched with up to three controls (n=272) based upon sex, race (White vs. other), and year of baseline interview (to equalize follow-up time). One control was missing sleep data. All controls had normoglycemia (FPG <100 mg/dl) at both exams.

## Laboratory Methods

Fasting glucose concentrations were determined by the glucose oxidase method (Beckman instruments, Fullerton, CA). The interasssay coefficient of determination was below 5%. Fasting insulin was assayed from a kit provided by Linco Research, Inc that has minimal cross

reactivity with human proinsulin. The assay has a lower detection limit of 2 μU/ml with interassay CV of 3.6-8.4% and an intrassay CV from 2.2-4.4%.

### Statistical Analysis

For this report, data were compared between IFG cases and matched control subjects. Chi-square tests, analysis of variance or unpaired t tests were used as appropriate. Multivariable logistic regression analyses (conditioned on the matching criteria) was performed to estimate the odds ratio (OR) and 95% confidence interval (CI) of IFG comparing short (<6h) and long (>8h) duration of average weekday sleep versus the midrange or referent category (6-8h) after adjusting for the matching factors plus several covariates presented in two models. Model 1 is a risk factor model which includes the matching factors sex, race, and year of baseline interview plus age, abdominal height (cm), weight change (kg) and baseline weight, family history of diabetes (yes/no), smoking (ever/never), hypertension (yes/no), and depression (CES-D ≥ 16 vs. <16). Model 2 is a mediator model and includes insulin resistance (HOMA-IR) in addition to the matching criteria and variables in Model 1. Risk factor selection was made *a priori* based on reported findings in the literature. Retention in the model was based upon likelihood ratio tests: variables that significantly improved the -2 log likelihood when comparing a model with and without the covariate of interest were retained. Likelihood ratio tests were also conducted to test for interactions by comparing the log likelihood between two nested models, one with only the main effects and the other with both the main effects and the interaction terms in the model. No significant interactions were noted in the terms tested (sex*sleep and race*sleep). All statistical tests were two-sided, and $p < 0.05$ was considered statistically significant. P-values represent overall significance level. Analyses were carried out using SPSS for Windows (version 16.0; SPSS, Chicago, IL).

## RESULTS

Table 1 presents the mean (standard deviation) or N (percentage) of selected baseline characteristics by IFG case/control status at follow-up. There were 91 cases of impaired fasting glucose matched to 272 control participants (1 control was missing sleep data). At baseline, compared to controls IFG cases were significantly older (58 years vs. 54 years, p =0.005), had a larger abdominal height (21.3 vs. 20.1 cm, p=0.005) and BMI (p=0.04). Mean fasting glucose was higher among cases 94.3 mg/dl than controls 90.5 mg/dl (p<0.001), and mean fasting insulin was slightly, but significantly higher among cases than controls 15.0 uU/ml vs. 13.3 uU/ml, p=0.04). Cases were more insulin resistant than controls (HOMA-IR) 3.5 vs. 3.0, p=0.01. The average number of hours of weekday sleep duration was 6.8h vs. 7.1h (p=0.019) for cases and controls, respectively. Compared to matched controls, cases were less likely to have completed more than a high-school education (p=0.04). A positive family history of type 2 diabetes, current smoking, and the presence of hypertension were more prevalent among cases than controls (p< 0.05 for all). There were no differences between cases and controls with respect to physical activity, drinking habits, or resting heart rate.

Table 2 reports the mean (standard deviation) or N (percentage) of baseline characteristics by sleep duration category. The distribution of sleep was: 6.6% (n=25) short sleep, 86.5% (n=314) mid-range sleep and 6.6% (n=24) long sleep. Only age significantly differed between the sleep duration categories (p=0.026) where short sleepers were youngest and long sleepers were oldest. There were no differences in BMI, abdominal height or weight change, fasting glucose or insulin concentrations, HOMA-IR, physical activity or heart rate. Although short-sleepers reported the highest prevalence of current alcohol consumption, smoking, and family history of diabetes, the differences were not significant.

Table 3 presents the results of the multivariate conditional logistic regression analysis. OR (95% CI) of IFG are presented for short and long sleep compared to the mid-range sleep

duration category. In the risk factor model (Model 1) adjusted for the matching criteria plus age, abdominal height, weight change and baseline weight, family history of type 2 diabetes, smoking, hypertension, and depression the OR of IFG among short sleepers was 3.0 (95% CI: 1.05, 8.59) whereas the OR for long sleep was 1.60 (95% CI: 0.45,5.42) To examine the role of insulin resistance on the association between sleep and IFG we included HOMA-IR in Model 2 (mediator model). Among short sleepers, the association between sleep and IFG was attenuated and no longer significant OR 2.5 (95% CI: 0.83, 7.46) and unchanged among long sleepers. The addition of physical activity, alcohol consumption and education to the models had no effect on the sleep-IFG association (data not shown) and were therefore not further included as covariates.

## DISCUSSION

In the present study after six years of follow-up, short sleep was associated with a significant, three-fold increased likelihood of developing IFG even after considering several putative diabetes risk factors. This estimate was slightly attenuated but no longer statistically significant when the role of insulin resistance on this observed association was examined, suggesting that insulin resistance explains some but not all of the association. To our knowledge no other studies have reported specifically on the sleep-IFG association but the present study's estimates are in accord with previous studies that have examined the sleep-impaired glucose tolerance (IGT) association. However, other studies have been hampered by their cross-sectional design. For example, the Quebec Family Study examined the association between average sleep and the combined endpoint of (IGT) and type 2 diabetes in 223 Canadian families (n=740) (12). The odds of developing a glucose impairment was twice as high among short sleepers and 58% greater among long sleepers compared to mid-range sleepers ($p<0.05$ for both). The Sleep Heart Health Study (13) reported the OR of type 2 diabetes was 2.5 times greater among the shortest sleepers (<5h night), and about 70% greater among persons sleeping <6h or ≥ 9h a night vs. 7-8h. Similarly, people who slept 6h carried a 60% excess likelihood of having IGT ($P<0.001$ for all). Like these studies and others that have examined the sleep-type 2 diabetes association(10) long duration was also found to be a significant predictor of disordered glucose metabolism. The present study found that long sleep duration >8h increased the likelihood of IFG by 60%, however, our findings were not statistically significant, perhaps because of the limited number of case subjects (n=5) in this sleep category. Although short and long sleep duration have both been found to elevate the risk of disordered glucose metabolism, the underlying mechanisms are thought to differ.

There are several plausible biologic pathways through which sleep loss can lead to disordered glucose metabolism. One mechanism is through alterations in the neurohormonal regulation of feeding behaviors. Experimentally, acute sleep curtailment resulted in a 28% increase in mean levels of the appetite stimulating hormone ghrelin, and an 18% decrease in the anorexigenic hormone leptin. Moreover, sleep restriction was significantly associated with self-reported increased hunger and appetite especially for calorie dense foods, despite normalizing caloric intake via a continuous glucose infusion(20).

Another possible pathway by which reduced sleep may lead to type 2 diabetes is through the activation of the sympathetic nervous system (SNS). Experimental studies have shown that sleep restriction decreases glucose tolerance, and increases cortisol levels and heart rate variability (21;22). These neuroendocrine changes interfere with mechanisms that regulate plasma glucose, resulting in a higher steady state plasma glucose concentration (23).

The activation of inflammatory pathways may also play a role in the observed association between short sleep duration and impaired glucose metabolism. Experimental studies have

shown that reduced sleep is associated with increased TNF-α, IL-6 and CRP (24;25). Moreover, it is well established that inflammation predicts subsequent type 2 diabetes (26;27).

Finally, the possibility that short sleep duration may represent a risk marker rather than a causal risk factor for diseases cannot be ruled out at the present time. In fact, short sleepers are likely to be characterized by a distinctive pattern of socio-demographic, lifestyle, and co-morbid medical conditions that may confound the observed associations (28). Data on sleep quality were not available therefore we were unable to assess the presence of sleep disorders such as obstructive sleep apnea. It is possible that differences in the reasons for short sleep duration (eg. self- imposed sleep restriction, sleep loss due to family or job demands, or underlying illness) might affect the observed sleep- IFG association, but we were unable to directly examine this issue.

One limitation of the present study is that sleep duration was self-reported, not objectively measured which may result in a systematic over-reporting of sleep duration (8;29). Over-reporting would most likely be random, non-differential, which would dilute the sleep-IFG association. Another limitation is that we did not control for some comorbid conditions that may inhibit longer sleep duration such as, incontinence, esophageal reflux, or arthritis. However, adjustment was made for several diseases that have been shown to be associated with diabetes including hypertension and depression. In addition, reliance on a single FPG measure at the baseline and follow-up examinations to categorize normoglycemic and IFG subjects could have resulted in an under-diagnosis of IFG as well as misclassification: the directionality of the potential bias is unknown. Fasting glucose measures are highly correlated over time (our own data show the correlation coefficient is r=0.60 for repeat FPG measures over a six year period). Nevertheless the American Diabetes Association supports the use of one fasting measure in epidemiologic studies. (30).

There are several notable strengths of this study: Participants were randomly selected from the general population of Erie and Niagara counties and are racially representative of these counties. In addition, the matched design improves internal validity by decreasing variability between cases and controls, resulting in an analysis that is more sensitive to the association of interest. Furthermore, the selection of three control participants per case rather than just one enhanced statistical power. Also, these data include both men and women and a wide age range (35-79 years). Nevertheless, because of the matched design and lack of racial diversity these findings may not be generalized to other populations.

In summary, persons who self-reported short sleep duration were three times more likely to develop IFG compared to those whose average sleep was 6-8 h a night. This association can be explained in part by insulin resistance.

## Acknowledgments

The authors would like to acknowledge Ms Mya Swanson, B.A. for her assistance with database management and statistical analyses. This study was supported by a grant from NIH NIDDK RO1 DK60587. Abstract published in Circulation 119(10) March 17,2009 p86.

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## List of Abbreviations and Acronyms

| | |
|---|---|
| **BMI** | Body mass index |
| **CES-D** | Center for Epidemiologic Studies Depression Scale |
| **CI** | Confidence interval |
| **FPG** | Fasting plasma glucose |
| **HOMA-IR** | Homeostasis model assessment insulin resistance |
| **HTN** | Hypertension |
| **IFG** | Impaired fasting glucose |
| **IGT** | Impaired glucose tolerance |
| **OGTT** | Oral glucose tolerance test |
| **OR** | Odds Ratio |
| **PA** | Physical activity |
| **RR** | Relative risk |

## Table 1

Mean (sd) or N(%) of baseline characteristics by case -control status at follow-up

| | Cases N=91 | Matched Controls N=272 | P-value |
|---|---|---|---|
| | *Mean (sd)* | *Mean (sd)* | |
| **Age (years)** | 58.0(11) | 54.0 (11) | **0.005** |
| **BMI (kg/m$^2$)** | 28.1 (5.2) | 26.9 (4.7) | **0.04** |
| **Abdominal height (cm)** | 21.3 (3.3) | 20.1(3.3) | **0.005** |
| **Weight change (kg)*** | 2.5 (5.4) | 1.4 (6.4) | **0.13** |
| **Fasting glucose (mg/dl)** | 94.3 (4.4) | 90.5 (5.4) | **<0.001** |
| **Fasting insulin (uU/ml)** | 15.0 (7.5) | 13.3 (6.7) | **0.04** |
| **HOMA-IR** | 3.5 (1.7) | 3.0 (1.6) | **0.01** |
| **Resting heart rate (bpm)** | 70.3 (9.5) | 68.4 (9.4) | **0.96** |
| **Physical activity (MET hrs/wk)** | 9.3 (13.3) | 10.2 (12.5) | **0.57** |
| **Daily sleep (hrs/d)** | 6.8 (1.2) | 7.1 (1.0) | **0.019** |
| | ***N(%)*** | ***N(%)*** | |
| **Male** | 39(42.9) | 117 (42.9) | **1.0** |
| **White, non-Hispanic** | 87(95.6) | 261 (95.6) | **1.0** |
| **Formal education >12 years** | 54(59.3) | 194 (71.1) | **0.038** |
| **Family history of T2DM** | 36(42.9) | 69 (26.7) | **0.005** |
| **Hypertension** | 34 (38.6) | 56 (20.8) | **0.001** |
| **CESD ≥16** | 9 (10.8) | 18 (6.7) | **0.22** |
| **Current alcohol drinking** | 56 (61.5) | 188 (69.9) | **0.14** |
| **Smoking:** | | | **0.017** |
| **Never** | 34(37.4) | 141(51.8) | |
| **Ever** | 57 (62.6) | 131 (48.2) | |

*Weight change – weight at follow-up visit minus weight at baseline visit

Physical Activity- total weekly MET hours in ≥ moderate activity in the past seven days

Hypertension (HTN) defined as SBP ≥140 mmHG or DBP ≥90mmHg or taking antihypertensive medication

## Table 2

Mean (SD) or N(%) of baseline characteristics by sleep duration category

| | Short <6h | Mid-range 6-8h | Long >8h | P-value[!] |
|---|---|---|---|---|
| ***Total N (%)*** | ***25 (6.6)*** | ***314 (86.5)*** | ***24 (6.6)*** | |
| *Cases N (%)* | *11 (12.1)* | *75 (82.4)* | *5 (5.5)* | 0.07 |
| *Matched controls N (%)* | *14 (5.1)* | *239 (87.9)* | *19 (7.0)* | |
| ***Mean (sd)*** | | | | |
| Age (years) | 50.3 (7.3) | 55.3 (11.1) | 58.5 (11.0) | 0.026 |
| BMI (kg/m2) | 27.2 (4.3) | 27.1(4.9) | 28.3 (4.9) | 0.51 |
| Abdominal height (cm) | 20.8 (3.6) | 20.3 (3.3) | 21.0 (3.0) | 0.47 |
| Weight change (kg)[*] | 1.3 (6.5) | 1.7 (6.3) | 1.7 (4.6) | 0.95 |
| Fasting glucose (mg/dl) | 91.0 (5.3) | 91.4 (5.5) | 91.8 (4.3) | 0.87 |
| Fasting insulin (uU/ml) | 15.9 (8.1) | 13.5 (6.9) | 14.4 (7.2) | 0.23 |
| HOMA_IR | 3.5 (1.8) | 3.1 (1.6) | 3.2 (1.6) | 0.30 |
| Resting heart rate (bpm) | 72.3 (9.1) | 68.6 (9.5) | 69.7 (8.1) | 0.16 |
| Physical activity (MET hrs/wk) | 10.6 (16.4) | 10.3 (12.7) | 4.4 (4.2) | 0.09 |
| ***N (%)*** | | | | |
| Male | 12 (48.0) | 135 (43.0) | 8 (33.0) | 0.56 |
| White | 22 (88.0) | 303 (96.5) | 22 (91.7) | 0.09 |
| Education >12 years | 14 (56.0) | 218 (69.4) | 15 (62.5) | 0.32 |
| Family history of T2DM | 11 (47.8) | 86 (29.1) | 8 (36.4) | 0.14 |
| [±]HTN | 7 (29.2) | 76 (24.7) | 7 (29.2) | 0.80 |
| CES-D ≥16 | 2 (8.3) | 23 (7.6) | 2 (8.7) | 0.98 |
| Current alcohol drinker | 18 (72.0) | 213 (68.5) | 12 (52.2) | 0.24 |
| Smoker ever | 17 (68.0) | 160 (51.1) | 11 (45.8) | 0.22 |

[*]Weight change – weight at follow-up visit minus weight at baseline visit

Physical Activity- total hours per week in ≥ moderate activity

[±]Hypertension (HTN) defined as SBP ≥140 mmHG or DBP ≥90mmHg or taking antihypertensive medication.

[!]P-values represent overall significance level.

## Table 3

Multivariate conditional logistic regression adjusted OR (95% CI) of IFG by sleep duration category

| Sleep hour per weekdays | Model 1 | Model 2 |
|---|---|---|
| **<6** | 3.0 (1.05,8.59) | 2.5 (0.83,7.46) |
| **6-8** | 1.0 (referent) | 1.0 (referent) |
| **>8** | 1.6 (0.45, 5.42) | 1.6 (0.45, 5.70 |

**Model 1 Risk factor** model adjusted for the matching criteria sex, race (white vs. other, and year of baseline interview plus age, abdominal height, weight change and baseline weight, family history of diabetes, smoking (ever/never), hypertension, and depression (ces-d ≥16)

**Model 2 Mediator model** adjusted for Model 1 plus HOMA-IR