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Diabetes And Glucose Metabolism

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Metabolic And Neuroendocrine Adaptability Following One Night Of Partial Sleep Restriction In Dutch Males

Iris Pelsma, MD, MSc[1], *Rosa van den Berg, MD, PhD*[1], *Sander Kooijman, PhD*[1], *Marieke Snel, MD*[2], *Gewei Zhu, MSc*[3], *Alison Moore, MSc*[4], *Jenny Pople, MSc*[4], *Sjan Lavrijsen, MD, PhD*[1], *Mark Birch-Machin, PhD*[3], *Ranjit Bhogal, PhD*[4], *Abdoelwaheb El Ghalbzouri, PhD*[1], *Borja Martinez-Tellez, PhD*[1], *Timo Giesbrecht, PhD*[5], *Sander Kersten, PhD*[6], *Michiel van Weeghel, PhD*[7], *Onno Cornelis Meijer, PhD*[8], *Alberto Martin Pereira Arias, MD,PhD*[2], *David Gunn, PhD*[4], *Patrick Rensen, PhD*[1], *and Nienke Biermasz, MD,PhD*[9]

[1]Leiden University Medical Center, Leiden, Netherlands; [2]Leiden University Medical Center, Leiden, Netherlands; [3]Newcastle University Medical School, Newcastle upon Tyne, United Kingdom; [4]Research and Development, Unilever, Sharnbrook, Bedford, United Kingdom; [5]Research and Development, Unilever, Sharnbrook, Bedford, Netherlands; [6]Wageningen University, Wageningen, Netherlands; [7]Amsterdam Univeristy Medical Center, Amsterdam, Netherlands; [8]Leiden University Medical Centre, Leiden, Netherlands; [9]Leiden University Medical Center, Oegstgeest, Netherlands

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Introduction: Chronic and acute short sleep associate with many adverse effects. Metabolic and neuroendocrine effects during the day following short sleep, however, are unknown. **Methods:** Randomized cross-over study using short sleep (4h) *vs* normal sleep (8h) in 31 males (31 years (IQR 23-47, BMI 26.0±4.2 kg/m^2 (range 20.1-35.0). Glucose, lipid, incretin, ACTH, and cortisol levels, as well as respiratory exchange ratio (RER; indirect calorimetry) were assessed following standardized mixed meals. RNAseq of morning skeletal muscle biopsies and pathway analysis on differentially expressed genes was performed. **Results:** Short sleep increased fasting free fatty acids (0.34 mmol/L (IQR 0.26 - 0.38) *vs* 0.47 mmol/L (IQR 0.38 - 0.70), P<0.0001). Cortisol levels peaked upon early wake-

up, which eliminated a peak at normal wake-up. Morning postprandial glucose peaked later (time $P<0.001$, levels $P=0.106$, pattern $P=0.031$), whilst insulin was unaffected. Afternoon postprandial insulin was lowered (time $P<0.001$, levels $P=0.044$, pattern $P=0.419$), whilst glucose was unaffected. Morning and afternoon postprandial TC, and LDL-c were lowered (morning: TC, time $P<0.0001$, levels $P=0.009$, pattern $P=0.289$; LDL-c, time $P<0.001$, levels $P=0.038$, pattern $P=0.797$; afternoon: TC, time $P<0.001$, levels $P=0.007$, pattern $P=0.92$; LDL-c, time $P<0.001$, levels $P=0.055$, pattern $P=0.991$). Pathway analysis of muscle revealed increased fatty acid oxidation (FAO), reflected by increased Oxidative Phosphorylation and Mitochondrial Dysfunction pathways, accompanied by lowered fasting RER (0.87 (IQR 0.86 - 0.92) *vs* 0.86 (IQR 0.84 - 0.87), $P=0.0033$). **Conclusions:** Acute partial sleep restriction subtly and temporarily changed glucose dynamics (morning), and insulin levels (afternoon), and lowered TC and LDL-c levels in the morning and afternoon, with concurrent increased FAO and mitochondrial dysfunction pathways in skeletal muscle, and calorimetric shifting from glucose towards lipid oxidation (fasted and postprandial), potentially highlighting increased delipidation of chylomicrons following sleep restriction.

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