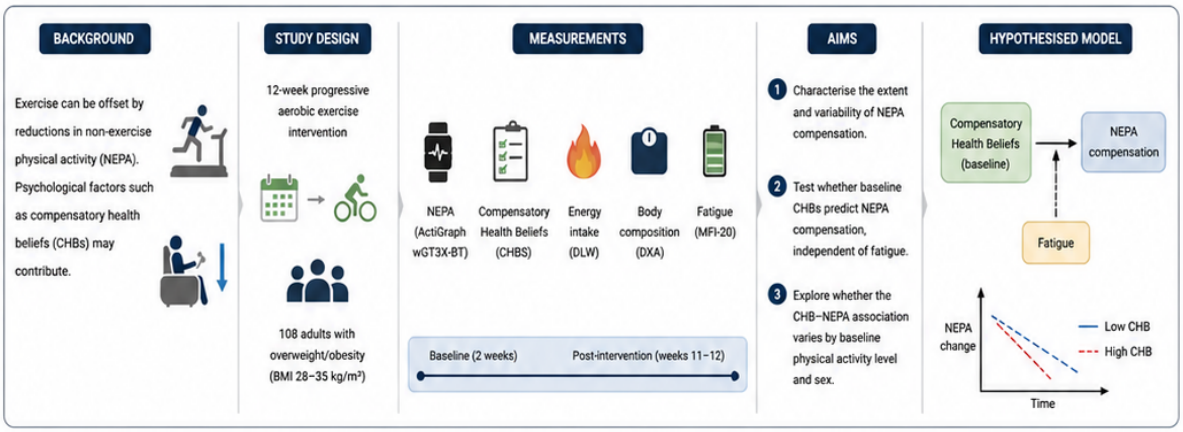
Are compensatory health beliefs associated with non-exercise physical activity compensation after a 12-week aerobic exercise intervention? Matthew Hawke and Christoph Theveßen



Introduction – Background & Rationale. The worldwide prevalence of obesity is high and increasing[1]. In Germany, a 2019/2020 self-reported survey found 19% of adults were obese and 53.4% overweight[2]. This is a major public health concern, as obesity is a risk factor for type 2 diabetes, cardiovascular diseases, several cancers, and musculoskeletal disorders, and is associated with premature death[3],[4].

Physical activity is a key pillar of multimodal obesity therapy[5], yet exercise-induced weight loss is often smaller and more variable than predicted[6]. One explanation is energy compensation: increases in exercise energy expenditure may be partially offset by physiological or behavioural adaptations[7]. For example, after exercise, individuals have been shown to compensate behaviourally through eating more (increased energy intake)[8] or moving less during the rest of the day (reduced non-exercise physical activity, or NEPA)[9],[10]. NEPA refers to bodily motion outside volitional exercise, including activities of daily living such as fidgeting, posture maintenance, and ambulation[11]. Substantial inter-individual variability in energy compensation is consistently observed. One systematic review found a mean energy compensation of 18%, but with a standard deviation of 93%[12], meaning group-level means may obscure substantial individual compensation[6].

Determinants of energy compensation remain poorly understood, though it appears most reliably in longer interventions[12]. Psychological factors associated with energy compensation have received little attention so far. One potential mechanism is compensatory health beliefs (CHBs), e.g., "I worked out this morning, so I can rest now." CHBs have been empirically associated as a contributor to energy intake compensation[8], however their relationship with NEPA compensation remains unclear.

Gap of knowledge. Compensatory Health Beliefs (CHBs) are beliefs that the negative effects of an unhealthy behaviour can be compensated for, or "neutralised," by a healthy one, for example, "I can eat this cake now because I will exercise this evening"[13]. Higher CHB scores have been linked to more maladaptive health behaviours, reported illness symptoms, and higher BMI[13]. To date, only one study has examined CHBs in relation to NEPA compensation as a result of an exercise intervention. Gray et al.[14], in a qualitative study of older adults who reduced NEPA during a four-week programme, reported the licensing of subsequent inactivity as one psychological mechanism of physical activity compensation, alongside physiological factors such as fatigue[14]. While inferred from participant

interviews, CHBs were not measured with a validated instrument[14]. To our knowledge, no study has yet quantitatively examined whether individuals with stronger CHBs are more likely to compensate in their NEPA after an exercise intervention.

Our hypothesis is that baseline compensatory health beliefs (CHBs) will be associated with non-exercise physical activity (NEPA) compensation in response to an exercise intervention. We will test this through 3 aims which first establish observed compensation, test our core assumption and finally consider its generalisability:

- **Aim 1:** Characterise the extent and inter-individual variability of NEPA compensation in response to the intervention.
- **Aim 2:** Determine whether baseline CHBs are associated with NEPA compensation, and whether this association is independent of fatigue.
- **Aim 3:** To explore whether the CHB–NEPA association varies by baseline physical activity level and sex.

Sample and recruitment. A sample of 108 participants will be recruited to allow for approximately 20% attrition, yielding a target analysed sample of 88. This provides 80% power ($\alpha = 0.05$) to detect an effect anchored to previously reported associations between CHB scores and health-risk behaviours ($r \approx 0.28$–0.29)[13]. A study with a comparable exercise intervention reported an attrition/exclusion rate of 13% supporting our estimate as reasonable and conservative[15]. The sample will include men and women aged 20-40 years, with BMI of 28-35kg/m², who are sedentary ($VO_2max \leq 45$ ml·kg⁻¹·min⁻¹ measured via graded maximal exercise test on a cycle ergometer), generally healthy (blood pressure ≤140/90 mmHg, fasting blood glucose ≤6.1 mmol/l, no regular medication), not currently engaged in weight-loss, and with no contraindications to participating in vigorous aerobic exercise. To mitigate selection bias, recruitment will be performed through multiple channels. Alongside general outreach (advertisements), we will establish cooperation agreements with general practices and occupational-health services, in which physicians identify potentially eligible patients and invite them to opt in. Where feasible, we will also pursue register-based recruitment, inviting a random sample of age-eligible residents drawn from the residents' registration office, to reach individuals who would not otherwise self-select into exercise research. Recruitment source will be recorded for every participant to describe the characteristics of the achieved sample and to allow post-hoc examination of channel-related differences.

Study Design and Intervention. We are proposing a single-group, within-subject design with a pre-intervention baseline period serving as the control condition. To reduce the risk that awareness of the primary outcomes biases participant behaviour, the study will be presented as an investigation into cardiovascular and metabolic responses to supervised aerobic training. The specific hypotheses regarding compensatory behaviours will remain unknown until after the data collection. This partial blinding is intended to minimise changes in non-exercise activity or dietary compensation that could occur if participants knew these were the outcomes of interest. Participants will perform supervised aerobic exercise five sessions per week across a 12-week intervention, with the prescribed dose anchored to exercise-induced energy expenditure and ramped progressively to improve tolerability and

adherence (See table 1). Exercise intensity will be monitored via a heart rate monitor (Polar RS400) during each session. The heart rate monitor will use sex, heart rate, body weight (adjusted at weeks 3, 6 and 9), and age to estimate energy expenditure during the workout[16].

Phase	Weeks	Target kcal/week	Target kcal/session	Intensity
Ramp in	1-2	1500	300	Mod (~45-55%HRR)
Progression	3-6	2000	400	Mod (~55-65%HRR)
Target	7-12	2500	500	Vig (~65-75%HRR)

Table 1: Planned progressive exercise intervention protocol

Measurements and analysis. The primary outcome is NEPA, quantified as the change in the two-week baseline to weeks 11–12. NEPA will be measured using a research-grade tri-axial accelerometer (ActiGraph wGT3X-BT) provided to each participant, worn on the right hip during all waking hours. To isolate NEPA, accelerometer measurements coinciding with supervised exercise sessions will be excluded, so that only activity outside prescribed exercise contributes to the outcome. The primary predictor, Compensatory Health Beliefs (CHBs), is measured at baseline via the 17-item CHBS. In brief, measures include CHBs, NEPA, resting metabolic rate, energy intake, body composition, weight and fatigue. Instruments, timing and rationale are detailed in Table 2.

For **Aim 1**, we will characterise the extent and inter-individual variability of NEPA compensation descriptively (mean change, SD, and the proportion of participants showing reductions). As in prior work[12], we expect the average reduction to be small but to vary widely between people, with a subgroup compensating strongly compared to baseline. For **Aim 2**, linear regression will test the association between baseline CHBs and NEPA compensation. A second model will then be used to control for variance in fatigue to determine whether the association is independent of it. We expect higher baseline CHBs to predict NEPA compensation, with the association attenuated but retained after fatigue adjustment. For **Aim 3**, we will add baseline physical activity level and sex as moderators to explore whether the CHB–NEPA association varies across these subgroups. This analysis is exploratory and expected to be hypothesis-generating rather than confirmatory given power constraints. Resting metabolic rate, energy intake (intake-balance method) and body composition are measured to position NEPA compensation within the total energy-balance equation and to rule out that intake or metabolism-driven changes are misattributed to NEPA[7,8].

The proposed project timeline presents a realistic plan to complete the study within the 3-year funding period. Key strengths include a 5-month recruitment phase, which allows for 5 different entry points to begin the intervention, and 6-month time buffer to accommodate unforeseen challenges and delays.

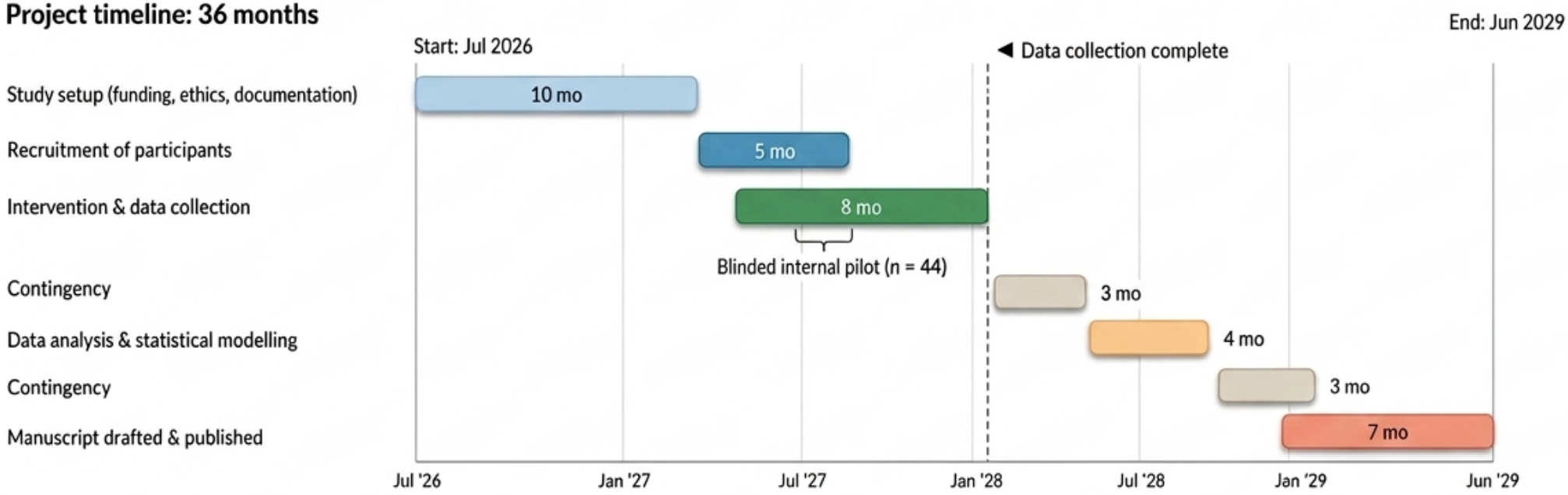


Table 2. Outcome measures, instruments, assessment timing, and rationale.

What	How	When	Rationale
Compensatory health beliefs	*17 item Compensatory Health Belief Scale* (CHBS)	Baseline	Validated CHB measure with good internal consistency ($\alpha = 0.80$) and test-retest reliability[13].
NEPA	*ActiGraph* wGTSX-BT	2-week baseline, week 11–12	Validated tri-axial accelerometer with established criterion validity for step counting and energy-expenditure/intensity classification against indirect calorimetry; hip placement provides the most accurate intensity and energy-expenditure estimation[17].
Resting metabolic rate	*Indirect calorimetry*	Baseline and post intervention	Gold-standard, non-invasive method for quantifying resting energy expenditure via respiratory gas exchange[19].
Energy intake	*Doubly labeled water*	Baseline and week 11–12	Gold-standard measure of total energy expenditure in free-living individuals, combined with change in body energy stores, allows estimation of energy intake vin the intake-balance method[19,20].
Weight	*Calibrated electronic scale*	Baseline, week 3, 6, 9 and post intervention	Weight is measured on an electronic scale under standardized conditions.
Body composition	*Dual-energy X-ray absorptiometry* (DXA)	Baseline and post intervention	Validated, reliable method for measuring body composition, combined with DLW expenditure to estimate energy intake (intake-balance method)[21,22].
Fatigue	*Multidimensional Fatigue Inventory* (MFI-20)	Baseline, week 3, week, 7, week 12.	A validated, multidimensional self-report measure of fatigue[23] and used extensively in exercise interventions[24–26].

Risks and controls. The primary risk is poor adherence and low recruitment. This is controlled through exercise prescription and recruitment strategy. As the single-group design does not treat exercise as an independent variable, intensity can be gradually ramped, allowing a regimen that is both clinically recommended and more readily adhered to[27]. Second, the five-month recruitment phase provides five separate entry points, enabling contingency recruitment at later stages if required.

A further consideration is whether measurable NEPA compensation occurs. The hypothesis concerns inter-individual variability rather than a group-level mean reduction. Prior work reports small mean compensation but very large between-person variability[12], the pattern our design is intended to capture. Aim 1 is framed descriptively so it yields an informative result irrespective of direction. Quantifying the extent and variability of NEPA compensation in a well-characterised overweight/obese cohort, using gold-standard energy-balance measures, is a contribution independent of the CHB findings. Aim 2, however, depends on people differing in how much they compensate. If everyone compensates similarly, there is little variation for baseline Compensatory Health Beliefs (CHBs) to explain and the CHB–NEPA analysis loses power. Features of our design control against this. We have a well-characterised cohort and gold-standard measures keep measurement error low, so genuine differences are not lost in measurement error. Furthermore, NEPA compensation is a common response to exercise training, reported in roughly two-thirds of studies, with several reporting large inter-individual differences[10], including at durations comparable to ours[6]. Meaningful between-person variation is therefore likely.

Once half the sample is completed, they will also serve as a blinded internal pilot. This means observed NEPA-compensation variance will be used to re-estimate the required sample size for subsequent recruitment rounds, without examining the CHB–NEPA association, to ensure the study remains adequately powered. Blinded variance re-estimation does not inflate Type I error and is well established[28]. As a contingency against the high cost and burden of the energy-balance battery (doubly labelled water, DXA), these could instead be collected in a randomly selected subsample should budget or retention require it.

Significance and Impact. Achieving and maintaining weight loss remains a major treatment challenge impeded by energy compensation[29],[30]. Improving the efficacy of weight-loss treatment is of large public health interest, given that even moderate weight loss meaningfully improves metabolic health[31]. This study explores a plausible psychological determinant of NEPA compensation, CHBs, while accounting for relevant covariates, offering a novel contribution to understanding who compensates and why. If our hypothesis is supported, the CHB scale could serve as a low-cost screening tool to identify individuals at elevated risk of behavioural compensation, and would motivate trials testing whether directly targeting CHBs reduces compensation and improves the weight-loss response to exercise. By situating a health-psychology concept within physiological weight management research, the project encourages a more holistic and interdisciplinary approach to understanding weight management.

References

1. Worldwide trends in underweight and obesity from 1990 to 2022: a pooled analysis of 3663 population-representative studies with 222 million children, adolescents, and adults. *Lancet* 2024; 403: 1027–1050, https://pubmed.ncbi.nlm.nih.gov/38432237/ (2024).

2. Schienkiewitz A, Kuhnert R, Blume M, et al. Overweight and obesity among adults in Germany - Results from GEDA 2019/2020-EHIS. *J Health Monit* 2022; 7: 21–28.

3. Carmienke S, Freitag MH, Pischon T, et al. General and abdominal obesity parameters and their combination in relation to mortality: a systematic review and meta-regression analysis. *Eur J Clin Nutr* 2013; 67: 573–585.

4. Flegal KM, Kit BK, Orpana H, et al. Association of all-cause mortality with overweight and obesity using standard body mass index categories: a systematic review and meta-analysis. *JAMA* 2013; 309: 71–82.

5. German Obesity Society (DAG). S3 Guideline on the Prevention and Treatment of Obesity. Frankfurt: Association of the Scientific Medical Societies in Germany (AWMF); 2024. AWMF Registry No.: 050-001. Available from: https://register.awmf.org/de/leitlinien/detail/050-001

6. King NA, Hopkins M, Caudwell P, et al. Individual variability following 12 weeks of supervised exercise: identification and characterization of compensation for exercise-induced weight loss. *Int J Obes (Lond)* 2008; 32: 177–184.

7. Pontzer H, Durazo-Arvizu R, Dugas LR, et al. Constrained Total Energy Expenditure and Metabolic Adaptation to Physical Activity in Adult Humans. *Curr Biol* 2016; 26: 410–417.

8. Martin CK, Johnson WD, Myers CA, et al. Effect of different doses of supervised exercise on food intake, metabolism, and non-exercise physical activity: The E-MECHANIC randomized controlled trial. *Am J Clin Nutr* 2019; 110: 583–592.

9. Silva AM, Júdice PB, Carraça EV, et al. What is the effect of diet and/or exercise interventions on behavioural compensation in non-exercise physical activity and related energy expenditure of free-living adults? A systematic review. *Br J Nutr* 2018; 119: 1327–1345.

10. Mansfeldt JM and Magkos F. Compensatory Responses to Exercise Training As Barriers to Weight Loss: Changes in Energy Intake and Non-exercise Physical Activity. *Curr Nutr Rep* 2023; 12: 327–337.

11. Levine JA. Nonexercise activity thermogenesis (NEAT): environment and biology. *Am J Physiol Endocrinol Metab* 2004; 286: E675-85.

12. Riou M-È, Jomphe-Tremblay S, Lamothe G, et al. Predictors of Energy Compensation during Exercise Interventions: A Systematic Review. *Nutrients* 2015; 7: 3677–3704.

13. Knäuper B, Rabiau M, Cohen O, et al. Compensatory health beliefs: scale development and psychometric properties. *Psychology & Health* 2004; 19: 607–624.

14. Gray P, Murphy M, Gallagher A, et al. A qualitative investigation of physical activity compensation among older adults. *Br J Health Psychol* 2018; 23: 208–224.

15. Rosenkilde M, Auerbach P, Reichkendler MH, et al. Body fat loss and compensatory mechanisms in response to different doses of aerobic exercise—a randomized controlled trial in overweight sedentary males. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology* 2012; 303: R571-R579.

16. Keytel LR, Goedecke JH, Noakes TD, et al. Prediction of energy expenditure from heart rate monitoring during submaximal exercise. *J Sports Sci* 2005; 23: 289–297.

17. Santos-Lozano A, Santín-Medeiros F, Cardon G, et al. Actigraph GT3X: validation and determination of physical activity intensity cut points. *Int J Sports Med* 2013; 34: 975–982.

18. Olivas-León CU, Olivas-Aguirre FJ, Chávez-Guevara IA, et al. Using Respiratory Gas Analyzers to Determine Resting Metabolic Rate in Adults: A Systematic Review of Validity Studies. *Sports (Basel)* 2025; 13.

19. Westerterp KR. Doubly labelled water assessment of energy expenditure: principle, practice, and promise. *Eur J Appl Physiol* 2017; 117: 1277–1285.

20. Westerterp KR. Exercise, energy expenditure and energy balance, as measured with doubly labelled water. *Proc Nutr Soc* 2018; 77: 4–10.

21. Kuriyan R. Body composition techniques. *Indian J Med Res* 2018; 148: 648–658.

22. Racette SB, Das SK, Bhapkar M, et al. Approaches for quantifying energy intake and %calorie restriction during calorie restriction interventions in humans: the multicenter CALERIE study. *Am J Physiol Endocrinol Metab* 2012; 302: E441-8.

23. Smets EM, Garssen B, Cull A, et al. Application of the multidimensional fatigue inventory (MFI-20) in cancer patients receiving radiotherapy. *Br J Cancer* 1996; 73: 241–245, https://idp.nature.com/authorize/casa?redirect_uri=https://www.nature.com/articles/bjc199642&casa_token=ikbkqow3qh4aaaaa:xsprk8q3nh_w0pqtds9trxeg30a8ve7dyy-cnhidulybtig7qt4pz_iop65xuvpq7jvv9ufcj2yfvrtl (1996).

24. Ericsson A, Palstam A, Larsson A, et al. Resistance exercise improves physical fatigue in women with fibromyalgia: a randomized controlled trial. *Arthritis Res Ther* 2016; 18: 176.

25. Salehi F, Dehghan M, Mangolian Shahrbabaki P, et al. Effectiveness of exercise on fatigue in hemodialysis patients: a randomized controlled trial. *BMC Sports Sci Med Rehabil* 2020; 12: 19.

26. Neuzillet C, Vergnault M, Bonnetain F, et al. Rationale and design of the Adapted Physical Activity in advanced Pancreatic Cancer patients (APACaP) GERCOR (Groupe Coopérateur Multidisciplinaire en Oncologie) trial: study protocol for a randomized controlled trial. *Trials* 2015; 16: 454.

27. Riebe D, Ehrman JK, Liguori G, et al. *ACSM's Guidelines for Exercise Testing and Prescription*: American College of Sports Medicine, 2018.

28. Friede T and Kieser M. Sample size recalculation in internal pilot study designs: a review. *Biom J* 2006; 48: 537–555.

29. Doucet É, McInis K and Mahmoodianfard S. Compensation in response to energy deficits induced by exercise or diet. *Obes Rev* 2018; 19 Suppl 1: 36–46.

30. Hays HM and Flack KD. Energy Compensation with Exercise and Dietary-Induced Weight Loss. *Journal of Clinical Exercise Physiology* 2021; 10: 51–61.

31. Magkos F, Fraterrigo G, Yoshino J, et al. Effects of Moderate and Subsequent Progressive Weight Loss on Metabolic Function and Adipose Tissue Biology in Humans with Obesity. *Cell Metab* 2016; 23: 591–601.

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