A cognitive signature of metabolic health in effort-based decision-making

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Abstract

Survival necessitates a delicate balance between expending effort to obtain resources, and energy conservation. Individual differences in motivation, the tendency to expend effort, play a fundamental role in society, affecting health, education, and economic outcomes. Previous theories explain variations in motivation via dopaminergic function. However, insulin resistance could also alter motivated behaviour by shifting the balance toward energy conservation. In a preregistered experiment, we investigated whether blunted motivational tendency— reduced tendency to exert effort, quantified using economic decision-making models, and previously linked to neuropsychiatric symptoms— is present in type-2 diabetes. We found subjects with type-2 diabetes showed this cognitive signature of blunted motivational tendency, compared to matched non-diabetic controls. Across a large sample with and without diabetes, we found that increasing risk for diabetes linearly predicted blunted motivational tendency. Diabetic patients treated with semaglutide did not show restored motivational tendency. Metabolic ill-health is associated with a shift towards energy conservation, potentially contributing to comorbidity between metabolic disease and mental illness.

Keywords: Motivation, effort, computational modelling, type-2 diabetes, semaglutide

Introduction

Motivation underpins every facet of human behaviour. Higher motivation is associated with better outcomes across domains including academic performance, employment status, IQ score and life satisfaction[1], while reduced motivation (variously termed anhedonia, apathy or avolition) is a key transdiagnostic symptom of neurological and psychiatric conditions including depression, schizophrenia, Parkinson's disease, and stroke[2]. Patients often describe reduced motivation as being among the most negative impacts on their quality of life[3]. Empirical work suggests a neurocognitive mechanism underpins motivational differences: the integration of costs and benefits during decision-making (effort-based decision-making)[4].

Growing evidence suggests that motivation and metabolic health are closely linked. Metabolic disorders like type-2 diabetes (T2D) are highly comorbid with neuropsychiatric disorders like depression or schizophrenia, and motivational symptoms are postulated to play a mediating role in the relationship[5]. An overall shift towards greater energy conservation, manifesting partly as reduced effort expenditure or 'blunted motivational tendency', may constitute a common risk factor across mental and metabolic health. This suggests the existence of an overarching energy regulation phenotype spanning cognitive and physical health disorders[6].

Motivation depends primarily on dopaminergic signalling in the brain, particularly projections from the ventral tegmental area (VTA) to the ventral striatum

via the mesolimbic pathway[2]. In humans, dopamine D2/3 receptor availability has been shown to correlate with motivational tendency, and dopamine agonists may increase motivational tendency[7]. Neurons along the mesolimbic pathway also express receptors for insulin. While the effects of insulin on the brain are complex, a main effect seems to be an increase in dopamine release[8]. As well as enhancing motivation, striatal dopamine has a number of effects relevant to energy homeostasis, including inhibiting appetite and suppressing endogenous glucose production[9]. By extension, insulin insensitivity could potentially disrupt motivation. For instance, empirical work suggests the relationship between depression and blunted motivation is mediated by overweight status, possibly due to elevated rates of insulin resistance[10].

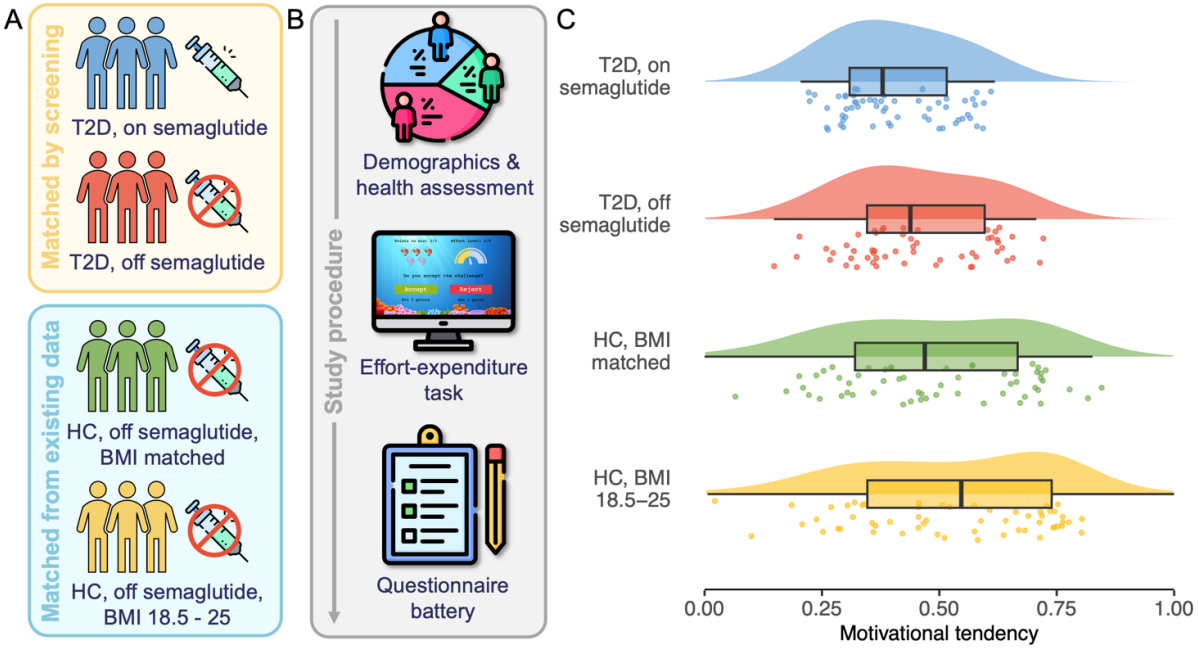
A common, effective treatment for diabetes and obesity is the glucagon-like peptide 1 (GLP-1) analogue semaglutide. Semaglutide works by augmenting insulin secretion and regulating appetite, but also affects the brain where, like insulin, it targets dopaminergic neurons in mesolimbic pathways[11].

It is unclear whether poor metabolic health, like mental and neurological health, is associated with blunted motivational tendency, and whether effective treatments work to ameliorate this blunting. We set out to investigate how a cognitive signature of motivational tendency is affected by metabolic disease and treatment, focussing on T2D and semaglutide. We hypothesised that people with T2D would exhibit blunted motivational tendencies and that this would be restored in patients undergoing semaglutide treatment.

Results

We recruited four study groups, differing in metabolic health and medication status, but matched by age, gender, and physical activity: subjects with T2D taking semaglutide, subjects with T2D not taking semaglutide, non-diabetic control subjects matched on body mass index (BMI), and control subjects with low metabolic risk (including restricting BMI to between 18.5 and 25; Fig. 1A). Participants completed a gamified effort-based decision-making task online[12], as well as self-report questionnaires targeting general health and neuropsychiatric symptoms (Fig. 1B). Using computational modelling, we quantified subject-level parameters governing participants' effort-based decision-making.

The four study groups did not differ in age, gender, physical activity, and, for the first three groups, BMI (Table 1). Effort-discounting effects during the task were found in all groups (*all* $p < .001$). To obtain subject-level parameter estimates describing participants' effort-based decision-making, we used hierarchical Bayesian modelling to fit six economic decision theory models to the task data (with weakly informative Gaussian priors and Markov-Chain Monte Carlo sampling, implemented in cmdstanr[13]). We checked all models for model convergence and chain mixing, using numeric diagnostics of ESS and split R-hats, and visual



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105 **Figure 1. Study groups, design, and group differences in motivational tendency. A**:
106 Type-2 diabetic (T2D) patients on (n = 58) and off (n = 54) semaglutide treatment
107 were identified by screening. Healthy controls (HC; i.e. non-diabetic) were selected
108 from an existing dataset (1) and either matched to the T2D groups by BMI (n = 58), or
109 restricted to a BMI range of 18.5 – 25 (n = 58). All groups are matched by age, gender,
110 and physical activity. **B**: Participants completed an online study consisting of three
111 components. **C**: Comparison of a computational *motivational tendency* parameter
112 between our four study groups.

113

114 inspection of trace plots. The winning model (identified by formal model
115 comparison) entailed three free parameters: effort sensitivity (weighting of effort
116 magnitude), reward sensitivity (weighting of reward magnitude), and motivational
117 tendency (overall decision bias towards exerting effort). Computational modelling
118 results replicated previous results, showing parabolic discounting of rewards by
119 effort across participants[14] – validating this instantiation of the model.

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Table 1. **Demographic characteristics of the sample, by study group.**

	T2D on semaglutide	**T2D off semaglutide**	**HC BMI matched**	**HC BMI 18.5-25**
Sample size, number (%)	58 (25.44)	54 (23.68)	58 (25.44)	58 (25.44)
Demographics				
Age, mean (SD, range)	44.7 (13.09; 19-74)	45.9 (11.37; 25-68)	48.3 (13.58; 21-69)	45.4 (14.33; 21-74)
Gender, number (%)				
Female	31 (53.45)	34 (62.96)	28 (48.28)	40 (68.97)
Male	26 (44.83)	20 (37.04)	30 (51.72)	17 (29.31)
Non-binary	1 (1.72)	0 (0)	0 (0)	1 (1.72)
BMI, mean (SD)	37.6 (8.53)	37.8 (10.0)	35.0 (6.60)	22.3 (1.80)
Physical activity				
IPAQ, mean sum score (SD)	2403 (4185)	3363 (4987)	2516 (2272)	2887 (2582)
Psychiatric questionnaires				
AES, mean sum score (SD)	54.4 (10.7)	55.1 (11.0)	56.3 (9.52)	57.3 (8.52)
SHAPS, mean sum score (SD)	10.4 (7.71)	8.83 (6.73)	9.76 (6.82)	8.31 (6.27)

Note. T2D, Type-2diabetes; HC, Healthy control (i.e., non-diabetic); BMI, Body mass index; SD, Standard deviation; IPAQ, International Physical Activity Questionnaire; AES, Apathy Evaluation Scale; SHAPS, Snaith-Hamilton Pleasure Scale.

A cognitive signature of blunted motivational tendency in T2D subjects

In our preregistered analyses, we found a main effect of group on motivational tendency ($F(3, 224) = 5.025$, $p = .002$, $\eta^2 = 0.063$), such that non-diabetic, low-BMI controls had the highest drive to exert effort for rewards, and diabetic patients on semaglutide the lowest (Fig. 1C). Post-hoc comparisons show the group effect was driven by differences between the T2D group on semaglutide and both the non-diabetic, BMI-matched controls ($t(90.501) = 2.451$, $p = 0.016$, $d = 0.455$) and the non-diabetic, low-BMI controls ($t(87.572) = 3.803$, $p < .001$, $d = 0.706$), as well as the difference between the T2D group off semaglutide and low-BMI controls ($t(100.08) = 2.309$, $p = .023$, $d = 0.431$). Note that only the effect between the T2D group on semaglutide and the low-BMI controls remained significant after Bonferroni correction for multiple comparisons. The group effect on motivational tendency was robust to controlling for antidepressant use (main effect of group: $F(3, 223) = 5.017$, $p = .002$; main effect of antidepressant use: $F(1, 223) = 0.632$, $p = .428$), a variable that significantly differed between groups (highest in T2D on semaglutide, lowest in non-diabetic groups; $X^2(3) = 25.191$, $p < .001$, Table 1). Furthermore, our results were not driven by differences in neuropsychiatric symptoms – we found no group differences in neuropsychiatric questionnaire scores (apathy: $F(3, 224) = 0.555$, $p = .645$; anhedonia: $F(3, 224) = 1.212$, $p = .306$, Table 1).

An increasing risk for diabetes linearly predicts blunted motivational tendency

In an exploratory analysis, we tested the relationship between motivational tendency and metabolic risk score across study groups in a generalized linear model. Metabolic risk significantly predicted motivational tendency (β = -0.129, t = -2.489, p = .014, η^2 = 0.027), with a higher metabolic risk score predicting a lower motivational tendency parameter. The effect of metabolic risk on motivational tendency remained significant when controlling for antidepressant use (β = -0.109, t = -2.011, p = .046, η^2 =.017).

In our sample of type-2 diabetic patients on semaglutide injections, time since semaglutide injection did not affect motivational decision-making (within-subjects comparison of one to six days since injection, n = 25: $t(24)$ = 0.237, p = 0.814).

Discussion

In this study, we found that type-2 diabetes (T2D) is associated with altered effort-based decision-making. Specifically, participants with T2D exhibited a lower motivational tendency compared to non-diabetic controls. This main result was echoed by our finding that increased risk of diabetes is associated with decreased motivational tendency.

Our findings align with a broader framework regarding the brain's involvement in energy regulation. In T2D, reduced sensitivity to insulin could be conceptualised as shifting the body's homeostatic set point, favouring energy conservation over expenditure[15]. Cognitively, this homeostatic shift corresponds to a

168 shift away from effortful behaviour, potentially owing to alterations in dopamine
169 signalling[2].
170 The cross-sectional nature of this study means we cannot draw any
171 conclusions about whether changes in motivation precede, or are a consequence of,
172 T2D. However, an important implication of our results is the putative existence of a
173 positive feedback loop: decreased motivational tendencies and a behavioural bias
174 towards energy conservation could potentially inhibit behaviours such as physical
175 exercise, leading to weight gain and the continued maintenance or progression of
176 metabolic dysfunction.
177 As semaglutide's effects on weight loss are mediated by the brain, and
178 through dopaminergic signalling in particular[11] which in turn influences behaviour[2],
179 we had hypothesized semaglutide would enhance motivation. We were surprised,
180 therefore, to find semaglutide did not significantly affect motivation – in fact, the
181 T2D group on semaglutide had numerically (though not significantly) the lowest
182 motivational tendency across all four groups. We consider two explanations for this
183 result. First, it is possible that despite our group matching approach, sampling
184 differences between T2D subjects on and off semaglutide resulted in systematic
185 differences between the groups. For instance, participants who had been prescribed
186 semaglutide may have had worse glucose control or metabolic health before
187 semaglutide prescription, making them less comparable with our other groups. Note
188 however, that groups were matched carefully on a number of variables including
189 BMI. Alternatively, semaglutide may genuinely have no effect on motivational

tendency in T2D. Further work, ideally a randomised controlled trial on the cognitive effects of semaglutide, will be needed to understand its effects on motivation fully.

Our study results should be seen in light of several limitations, the most important of which is that the cross-sectional nature of our study precludes any assessment of a causal relationship between metabolic health and motivation. Longitudinal epidemiological studies suggest that T2D (or poor metabolic health more generally) and depression have a reciprocal relationship: the presence of either condition increases the risk of subsequently developing the other[16,17]. In future, to better understand the possible role of altered cognition and motivational tendency in this bi-directional relationship, one could conduct a longitudinal study measuring motivational changes in effort-based decision-making over time and assess how this relates to metabolic health and risk for diabetes. Additionally, studying changes in effort-based decision-making as T2D patients undergo treatment could also offer valuable insights to the causal relationship between T2D and motivation. Our results indicate this is a promising area of study.

Conducting our study online allowed us to access a much larger and more diverse participant population than we could have obtained in person. However, a corollary of this study setup is that we are unable to obtain physiological measures from our participants, limiting us in our ability to infer underlying biological mechanisms. A laboratory experiment taking quantitative measures of metabolic health, such as insulin sensitivity, would be a useful complement to our current

study. Based on the results presented here, we would predict participants with poorer insulin sensitivity to show a corresponding shift in motivational tendency.

In summary, we have identified a cognitive signature of diabetes, reduced motivational tendency, which may play a part in maintaining or worsening disease progression. The reduced motivational tendency in T2D may result from central insulin insensitivity affecting the dopaminergic reward and motivation pathways. This may be part of a broader mechanism of energy regulation, underlying the comorbidity between metabolic disease and mental illness.

Methods and materials

Experimental design

The objective of this study was to investigate whether diabetes and risk of diabetes were associated with changes in effort-based decision-making, and additionally, whether these changes were altered by ongoing treatment with the GLP-1 agonist semaglutide. The design we employed was a between-subjects online experiment, measuring effort-based decision-making with a gamified online task (see below) combined with computational models of decision-making, and additional self-report questionnaires.

Pre-registration and open data

The pre-registration for this study can be found at https://osf.io/7kmf5. The study was approved by the University of Cambridge Psychology Research Ethics Committee (PRE.2022.078), and all participants provided informed consent. The analysis code and data are openly available at https://github.com/smehrhof/semaglutide-study. The effort-expenditure task code is available at https://github.com/smehrhof/effort-study.

Recruitment and data acquisition

All data was collected on Prolific [18] in the United States and the United Kingdom. All participants provided informed consent through an online form, complying with the University of Cambridge Human Biology Research Ethics Committee procedures for online studies.

Study groups with type-2 diabetes

To identify participants meeting criteria for the study groups of patients with T2D, we implemented a screening procedure. The screening included demographic variables (age, gender, ethnicity, socio-economic status (SES), income, and English proficiency), general health (neurological conditions, mental health/neurodevelopmental conditions, chronic disease, and daily medication), current GLP-1 agonist treatment status, metabolic-related health (current height and weight for BMI calculation, current desire for weight-loss and weight-loss interventions, other current treatments for diabetes), and physical activity using the International Physical Activity Questionnaire – short form (IPAQ)[19]. Prolific pre-screeners were used to restrict recruitment to participants with T2D.

Participants reporting to be on GLP-1 agonist treatment with weekly injections of semaglutide (i.e., *Ozempic* or *Wegovy*) for at least 4 weeks fulfilled criteria to be included in the *T2D, on semaglutide* group and were invited to the main testing. Subjects indicating they are not currently (nor have ever been) on any form

of GLP-1 agonist treatment were matched to the *T2D, on semaglutide* group by age, gender, BMI, and IPAQ. The matched subset was then invited to the main testing.

Study groups of healthy controls

To obtain healthy control (HC) groups of subjects without diabetes, we used a pre-existing general population dataset derived from a previous study, which implemented a largely overlapping study procedure[12]. Participants without diabetes and off any GLP-1 agonist treatment were identified as possible HC. For the first HC group (*HC, BMI matched*), we further restricted inclusion to participants with a score below 12 on the Finnish Type-2 Diabetes Risk Score questionnaire (FINDRISC)[20], indicating slight metabolic risk. *HC, BMI matched* subjects were matched to the *T2D, on semaglutide* group by age, gender, IPAQ, and BMI. For the second HC group (*HC, low-BMI)*, inclusion was restricted to participants with a score below 7 on the FINDRISC (low metabolic risk), and a BMI of 18.5 – 25. *HC, low-BMI* subjects were matched to the *T2D, on semaglutide* group by age, gender, and IPAQ.

Study procedure

The main testing session consisted of three parts: a brief assessment of changes in medication (including diabetes medication and other daily medication) since the screening, an effort-based decision-making task, and a battery of self-report questionnaires.

279 ***Effort-based decision-making task***

280 We adopted a previously described effort-expenditure task[12] to assess effort-
281 based decision-making online. The task starts with a calibration phase in which
282 subjects are prompted to click as fast as they can for ten seconds, repeated across
283 three trials. The average of trials two and three are used as a reference for effort
284 calibration. To familiarize subjects with their individual effort levels, a practice trial
285 of each effort level must be completed. The main task consists of 64 trials, split into
286 four blocks. In each trial, participants decide whether to accept or reject a challenge
287 associated with effort and reward (points won throughout the game). When
288 accepting a challenge, the associated effort level must be completed to obtain the
289 reward. If a challenge is rejected, subjects wait and receive one point. The
290 presentation of effort-reward combinations is made semi-adaptively by randomly
291 interleaved staircases: when a challenge is accepted, the next offer is adjusted by
292 increasing effort or decreasing reward, while the opposite is implemented after
293 challenges are rejected.

294 ***Self-report questionnaires***

295 Subjects completed self-report questionnaires, presented in randomized
296 order. We assessed psychiatric variables using the Snaith-Hamilton Anhedonia
297 Rating Scale (SHAPS)[21] and the Apathy Evaluation Scale (AES)[22]. To target circadian
298 rhythm, we included the Morningness-Eveningness Questionnaire (MEQ)[23] and the
299 Munich Chronotype Questionnaire (MCTQ)[24]. Additionally, we assessed diabetes
300 risk using the FINDRISC[20].

301 ***Compliance checks and exclusion criteria***

302 Participants were excluded at the time of screening when reporting a severe
303 neurological disorder or English proficiency below B2 (i.e., good command/working
304 knowledge).

305 No subjects were excluded following our pre-registered task-based exclusion
306 criteria. Four attention check questions were included in the questionnaires to assess
307 questionnaire adherence. This included two easy questions (e.g., "Please answer
308 'Not at all'") and two harder questions (e.g., "When I am thirsty, spoiled milk is my
309 drink of choice", expected answer: *Definitively false*). Participants were excluded
310 when failing at least one easy question, or both harder questions (n = 3).

311 Further, we excluded participants reporting conflicting data regarding their
312 health or medication status between the screening and the main study session (n =
313 19), as well as subjects reporting anthropometric data resulting in a BMI more than
314 two standard deviations above the median (n = 5). Taken together, a total of n = 13
315 participants on semaglutide and n = 12 participants off semaglutide were excluded
316 from analyses.

317 ***Within-subject study***

318 A subset of participants in the *T2D, on semaglutide* group were randomized to
319 be tested on different days of their treatment schedule: one day after their weekly
320 injection of semaglutide or one day before (i.e., six days after their last injection).
321 Subjects were then invited to participate in a second study session, to be tested at the
322 respective other day in their treatment schedule (i.e., subjects tested one day before

injections in the first session were tested one day after their injection). A total of n = 25 participants completed two study sessions.

Analyses

Computational modelling of the effort expenditure task

Prior to computational modelling, we used model-agnostic analyses to ensure our task elicited effects of effort discounting across groups. We investigated main effects of effort- and reward-levels and their interaction, using a mixed-effects analysis of variances (ANOVA) of repeated measures.

For model-based analyses, we considered a model space of six models, all of which are variations on a standard economic decision-theory model. In brief, rewards (R) are discounted by effort (E), weighted by sensitivity parameters (β_R and β_E for reward and effort sensitivity), forming the subjective value (SV) of an action:

$$SV = (\beta_R \cdot R) - (\beta_E \cdot E) \quad (1)$$

The SV is transformed to an acceptance probability by a softmax function:

$$p(accept) = \frac{1}{1 + e^{-(\alpha + SV)}} \quad (2)$$

with $p(accept)$ for the predicted acceptance probability and α for the intercept representing motivational tendency. Models differed from each other in their implemented discounting function (parabolic or linear) and the in- or exclusion of free parameters reward sensitivity (weighting of the reward magnitude during decision making) and motivational tendency (decision tendency towards exerting effort), resulting in a model space of six models (parabolic/linear x with/without

reward sensitivity x with/without motivational tendency). See[12] for a validation study of the model space, including parameter recovery and posterior predictive checks and https://github.com/smehrhof/effort-study/tree/main/code/stan for the model code.

We took a hierarchical Bayesian approach to model fitting, using weakly informative Gaussian priors for all free parameters, at both the group- and individual-level. Models were fitted in the R Stan interface cmdstanr, using Markov-Chain Monte Carlo (MCMC) sampling (6000 iterations by four chains, 2000 warm-up samples). The performance of our six models was compared based on out-of-sample predictive accuracy using the leave-one-out cross-validation information criterion (LOOIC; lower values are better) and expected log predictive density (ELPD; higher values are better).

Group comparisons

Group comparisons between our four study groups were conducted using one-way ANOVAs, with group status predicting computational parameter estimates and questionnaire scores. To follow up on significant effects, multiple comparisons were conducted.

Within-subject analyses

We tested the effect of time since the last semaglutide injection on computational parameters using paired sample t-tests, comparing testing sessions one and six days after the last semaglutide injection.

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