Metabolic Tracking Using Heart Rate:

A Model for Energy Balance and Glycemic Control

White Paper Version 1.0

The METFIT PhD study, University of Iceland

Authors

Agnar Steinarsson[1,2], Gréta Jakobsdóttir[1], Jón Hlöðver Friðriksson[2] and Erlingur Jóhannsson[1].

Affiliations

1) Faculty of Health Promotion, Sport and Leisure Studies, University of Iceland.

2) Driftline – Applied Physiology and Metabolic Analytics, Reykjavik, Iceland.

Date

August 2025

Correspondence: agnar@driftlineanalytics.com
www.driftline.io

Abstract

Background: Accurate assessment of energy balance is essential for understanding and managing metabolic health, especially amid aging populations and rising obesity rates. Current methods for estimating energy expenditure (EE) and intake (EI), such as wearables and dietary self-reports, suffer from significant measurement errors. This study introduces **metabolic tracking (MT)**, a heart rate–based system for estimating EE and EI by analyzing postprandial heart rate kinetics and the thermic effect of food (TEF). The system derives individualized metabolic indices for EE prediction, leverages TEF for EI estimation, and defines a novel metabolic fitness index (MFI) for assessing metabolic health. The study aimed to test the validity of the MT concept and the accuracy of its metabolic estimations.

Methods: Eight healthy adults, tested for fasting metabolic biomarkers, participated in a controlled exercise trial for EE estimation and a feeding trial for EI estimation, consuming a large carbohydrate-rich meal and resting for 7 hours postprandial. EE and blood glucose were measured and TEF quantified to estimate EI and calculate MFI from heart rate dynamics.

Results: MT model validation showed strong agreement between measured and predicted values, with a mean absolute error of 0.25% for EE (range: –2.6% to +2.6%) and –1.6% for EI (range: –0.7% to –9.8%). Postprandial resting heart rate, blood glucose, body temperature, substrate oxidation, and resting metabolic rates peaked within one hour postprandial and followed a structured wave-like pattern, typically resolving within seven hours. MFI correlated strongly with glycemic responses and fasting metabolic biomarkers.

Conclusion: This study demonstrates the validity of metabolic tracking as a non-invasive method for metabolic assessment and monitoring. EE was accurately predicted across rest, exercise, and recovery, while EI estimates based on postprandial heart rate aligned closely with measured values. The Metabolic Fitness Index (MFI) showed strong correlations with glycemic and metabolic biomarkers, supporting its potential as a proxy for metabolic health.

Keywords: energy expenditure, heart rate, thermic effect of feeding, metabolic flexibility, glycemic control, wearable technology.

Introduction

Population ageing and rising obesity rates present critical global health challenges, particularly in Europe and the United States. Over 20% of the EU population is currently aged 65 or older, a figure projected to exceed 30% by the end of the century (Eurostat, 2023). At the same time, obesity prevalence has risen dramatically, affecting approximately two billion individuals worldwide (WHO, 2024), with rates among elderly Europeans nearing 70% in some countries (WHO Europe, 2023). These demographic shifts amplify the burden of chronic diseases such as cardiovascular disease, diabetes, and sarcopenia, increasing healthcare costs and reducing quality of life (Cesari et al., 2022; Chooi et al., 2019).

Human energetics is defined by the balance between energy intake (EI) and energy expenditure (EE), essential for maintaining metabolic health and body weight. Total energy expenditure (TEE) includes resting metabolic rate (RMR), physical activity-related expenditure (PEE), and the thermic effect of food (TEF). Disruptions in this balance contribute to obesity, malnutrition, and metabolic disease. As Hall and Guo (2017) emphasize, even modest but sustained mismatches between EI and EE can lead to significant changes in body weight over time, driven by complex physiological adaptations in appetite, metabolism, and energy efficiency. Understanding the dynamics of energy balance is therefore critical for both the prevention and treatment of metabolic disorders.

The thermic effect of food (TEF), or diet-induced thermogenesis, represents the postprandial increase in energy expenditure required for digestion, absorption, and nutrient metabolism. It is generally estimated to account for about 10% of total energy expenditure (TEE) (Westerterp, 2004), though considerable variability exists. TEF is influenced by numerous factors, including meal size, macronutrient composition, circadian timing, sleep, age, sex, insulin sensitivity, thyroid function, and recent physical activity. Early studies consistently reported the highest thermogenic response from protein, followed by carbohydrate and fat (Westerterp, 2004), but more recent reviews reveal substantial heterogeneity in macronutrient-specific responses, reflecting differences in study design, measurement windows, and participant characteristics (Calcagno et al., 2019; Tzeravini et al., 2024). This complexity underscores the need for standardized protocols and individualized approaches in TEF research.

Metabolic health depends on effective regulation of glycemic control and insulin sensitivity, both of which are essential for maintaining energy balance and preventing metabolic disease. Impaired glucose regulation is a hallmark of both obesity and aging and is closely linked to reductions in postprandial thermogenesis. The thermic effect of food (TEF), particularly after carbohydrate-rich meals, is partly mediated by brown adipose tissue (BAT) activation, which depends on intact sympathetic nervous system (SNS) signaling (Aita et al., 2022). Obesity is associated with reduced SNS activity and insulin resistance — both of which suppress TEF (de Jonge & Bray, 1997). Similarly, aging is associated with blunted TEF responses, likely due to decreased lean mass, reduced SNS responsiveness, and impaired metabolic flexibility (Tzeravini et al., 2024).

Metabolic flexibility — defined as the ability to switch efficiently between fat and carbohydrate oxidation — is impaired by mitochondrial dysfunction. Reduced oxidative capacity, impaired fatty acid oxidation, and elevated reactive oxygen species contribute to insulin resistance in muscle, liver, and adipose tissue (Kim et al., 2008; Sergi et al., 2019; Kelley et al., 2002; Galgani & Ravussin, 2008; San-Millán & Brooks, 2018). Cardiorespiratory fitness acts as a powerful modulator of insulin sensitivity and glycemic control, independent of adiposity. Higher fitness levels are associated with enhanced glucose uptake, reduced hepatic glucose output, and greater substrate switching capacity (Solomon et al., 2015; Bruce et al., 2000; Goodpaster et al., 2003). These findings underscore the importance of mitochondrial efficiency and aerobic fitness in preserving postprandial glucose regulation and overall metabolic resilience.

Smart wearables now offer continuous tracking of EE, but accuracy is limited—particularly for mixed, non-rhythmic, or post-exercise activities. Most devices rely on accelerometry and population-level heart rate models. O'Driscoll et al. (2020) reported mean absolute errors ranging from 13% to over 30%, with performance declining during resistance or free-living activities. Uphill et al. (2024) confirmed that EE estimation errors are highest during strength training and recovery and noted that most devices fail to account for post-exercise oxygen consumption (EPOC), a known contributor to EE.

To address these gaps, the present study introduces metabolic tracking — the indirect estimation of EE and EI from postprandial heart rate. This is theoretically feasible given the established links between heart rate and energy expenditure, and between TEF and meal size. Animal studies (Brosh et al., 2007) have successfully modeled EE or predicted feed intake using heart rate. In humans, postprandial heart rate responses (Aita et al., 2022) have not yet been used to infer EI.

The present study introduces **metabolic tracking (MT)**, a heart rate–based system for estimating EE and EI by analyzing postprandial heart rate kinetics and the thermic effect of food (TEF). The MT system derives individualized metabolic indices for EE prediction, leverages TEF for EI estimation, and defines a novel metabolic fitness index (MFI) for assessing metabolic health. The study aimed to test the validity of the MT concept and the accuracy of the metabolic estimations. by addressing three specific questions:

a) Can EE during rest, exercise, and recovery be accurately predicted from heart rate data?
b) Can EI be accurately predicted from the thermogenic heart rate response?
c) Does MFI correlate with biomarkers for metabolic health and glycemic control?

Methods

Participants and biomarker testing

Eight adults (4 women, 4 men; age range 31–60 years) with varying body compositions and physical activity levels participated in the study. Exclusion criteria included chronic disease, pregnancy, lactation, or recent significant weight change. Blood pressure medication (two participants) and hormonal supplementation (one participant) were permitted. Body size was indexed using allometric BMI (weight in kg divided by height in m to the power of 2.5), which better accounts for physiological scaling and avoids the height-related bias inherent in conventional BMI (Heymsfield et al., 2016). Participants arrived at the Sameind Medical Laboratory, Ármúli 32 in Reykjavík, in a fasting state for blood sample collection. Parameters measured were blood glucose, blood lipids (total cholesterol, HDL, LDL, triglycerides), insulin, cortisol, estrogen, testosterone, and thyroid-stimulating hormone (TSH). The study was approved by the Icelandic Bioethics Committee (VSN-24-123).

Feeding Trial

The study used a crossover design with two trials: a feeding trial and an activity trial. The feeding trial was conducted at the University of Iceland's School of Education Home Economics kitchen. Participants fasted for at least 10 hours before the trial and after arrival rested for 30 minutes before baseline measurements. Each participant was fitted with a heart rate monitor (H10, Polar Electro Oy, Kempele, Finland) and underwent a 15-minute indirect calorimetry session using a portable metabolic system (K5, COSMED, Rome, Italy). Following this baseline assessment, measurements were taken hourly over an 8-hour period. Capillary blood glucose was measured using a wireless glucose meter (Gluco+, iHealth Labs, Mountain View, CA, USA), and forehead temperature was assessed using an infrared thermometer (Thermo, Withings, Issy-les-Moulineaux, France).

Participants consumed a standardized pasta meal (500–1200 g; Knorr tomato pasta), with macronutrient content averaging 79% carbohydrates, 12% protein, and 8% fat. Participants remained within the building, engaging only in light activity, such as office work. Meals were weighed pre- and post-consumption and individual EI ranged between 432–1077 kcal. The estimated glycemic load (GL) ranged from 1.19–2.75 g/kg body weight, based on standard metabolic assumptions (95% carbohydrates, 50% protein, 10% fat converted to glucose). A standardized Glycemic Response Index (GRI) to a meal was defined and calculated using the following formula: $GRI = G_{max} / GL$, where G_{max} is the peak glucose level (mg/dL), GL is the standardized glycemic load (carb intake adjusted for body weight, g/kg).

The single meal cardiovascular (HR) and metabolic (EE) responses, measured at hourly intervals, were plotted and fitted with fourth-order polynomial curves (Figure 1). Response magnitude and duration were quantified as the area under the curve (AUC), yielding total HR (HR_{total}) and EE (EE_{total}) responses. From these, individual parameters were derived:

- Food Factor (FF) = HR_{total} / EI
- Thermic Factor (TF) = HR_{total} / EE_{total}
- Thermic Effect of Food (TEF) = EE_{total} / EI

The quantification of energy expenditure (EE) from indirect calorimetry used the adjusted de V. Weir formula (1949): EE (kcal/day) = $5.67 \cdot VO_2 + 1.6 \cdot VCO_2 - 2.17 \cdot UN$), where VO_2 is oxygen uptake (mL/min), VCO_2 is carbon dioxide output (mL/min), and UN is urinal nitrogen excretion (g/day). The COSMED K5 uses a default UN value of 12 g/day, and this was adopted for the study.

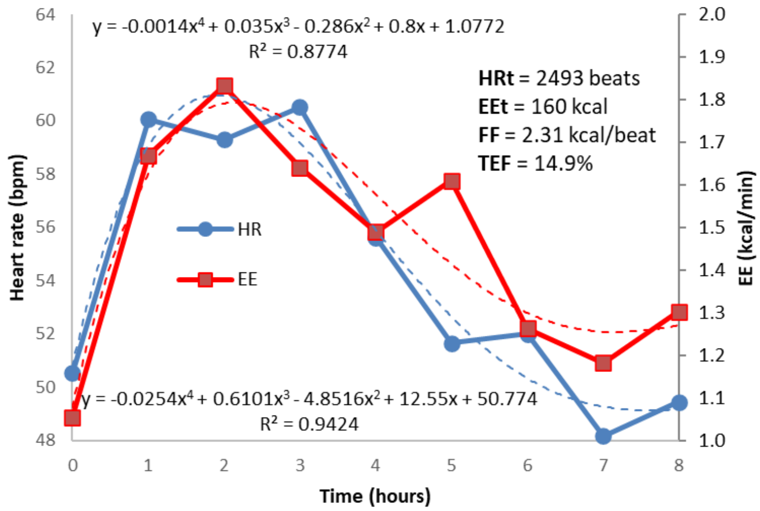

Figure 1. *Example quantification of the single-meal metabolic responses from one participant.* Heart rate and EE were measured at hourly intervals during the feeding trial and fitted using fourth-order polynomials (displayed with coefficients of determination (R^2)). The summed metabolic EE (EE_t) and heart rate (HR_t) responses are indicated, together with the food factor (FF) (see below) and TEF value.

Activity trial

The activity trial was conducted at the University of Iceland Sports Science Lab. Participants performed metabolic testing through a submaximal treadmill exercise including walking and running. The protocol began with five minutes of seated rest, followed by 15–18 minutes of walking and 3–21 minutes of running, ending with five minutes of seated recovery. Walking started at 3 kph, increasing by 1 kph every three minutes. All participants completed at least one running increment. Metabolic data were collected using a metabolic cart (Vyntus CPX, Vyaire Medical, Mettawa, IL, USA), and heart rate was recorded using chest strap and armband sensors (H10, Polar Electro Oy, Kempele, Finland; Rhythm+, Scosche Industries, Oxnard, CA, USA). Ventilatory thresholds (VT1 and VT2) were obtained as curve breakpoints from the CPX gas exchange diagrams (Vyaire Medical, 2019). For detailed representation and individual data analytics, see Supplementary Figures S1-3.

Metabolic Tracking

Metabolic tracking (MT) is a novel heart rate–based system for estimating EE and EI by analyzing postprandial heart rate kinetics and the thermic effect of food (TEF). The system subdivides total heartbeats into base beats (BB), activity beats (AB), and food beats (FB). The subclasses are converted to energy via metabolic coefficients, i.e. Base Factor (BF), Food Factor (FF) and Activity Factor (AF), all in units of beats per kcal. The food beats are identified with a **thermogenic food wave**, capturing the gradual rise and exponential decay of TEF and thus isolating the postprandial food-related heart rate response. Overlapping waves are resolved using a maximum filter to isolate the net postprandial signal. Total energy intake (EI) and expenditure (EE) are estimated as:

- EI = Food Beats (FB) / Food Factor (FF)
- EE = Basal (BE) + Activity (AE) + Food Expenditure (FE)

This approach enables real-time, non-invasive tracking of metabolic responses using heart rate alone. For detailed algorithmic representation, model parameters and individual data analytics, see Supplementary Figures S4-5.

The Metabolic Fitness Index (MFI) was defined as a dimensionless proxy for cardiometabolic efficiency, integrating metabolic economy, cardiovascular performance, and body composition. It was calculated as:

$$MFI = (((V2 \times 1000))/(60 \times (P2 - RHR)\ ))/(HRmax \times BMIa \times 6)$$

where V2 is the speed at the second ventilatory threshold, VT2 (km/h), P2 is heart rate at VT2, RHR is resting heart rate, HRmax is maximum heart rate, and BMIa is the allometric BMI. The denominator includes a scaling constant (6) to constrain MFI to a 0–5 range. Higher MFI values indicate superior metabolic fitness.

Analytics and Statistics

Data analyses were conducted using Jamovi (Version 2.6.44, The Jamovi Project, Sydney, Australia; https://www.jamovi.org). Predictions were validated against calorimetry using correlation analysis, regression, and Bland-Altman plots. Significance was set at $p < 0.05$.

Results

Activity Trial

The heart rate (HR) responses in the activity trial followed an exponential increase during walking and shifted to a linear pattern during running, as exemplified in Supplementary Figure S1. Table 1 summarizes the results of the activity trial, revealing wide variability in cardiorespiratory performance among participants. Ventilatory threshold speeds (V2) ranged from 7.0 to 18.6 kph, reflecting the inclusion of both sedentary individuals and trained athletes.

Table 1. *The summarized results from the activity trials.*

Peak running speed (V_p), lactate threshold markers (P2 and V2), runfactor (RF), transition speed (V_t), activity factor (AF_b), peak respiratory exchange ratio (RER_p), peak oxygen uptake ($VO2_p$) and running economy (RE), for participants (no. 1-8) in the activity trials.

No.	V_p (kph)	P2 (bpm)	V2 (kph)	RF (slope)	V_t (kph)	AF_b (b/kcal)	RER_p (ratio)	$VO2_p$ (ml/kg/min)	RE (ml/kg/km)
1	8	153	7.0	7.44	6.59	5.57	0.86	25.4	199.2
2	11	141	9.0	8.01	8.06	4.94	1.03	31.3	191.0
3	14	180	11.8	7.73	8.01	5.30	1.14	45.7	207.8
4	11	157	8.0	9.62	7.70	10.66	1.06	32.9	190.2
5	15	170	18.6	7.29	8.03	7.70	1.15	66.3	174.7
6	15	163	17.2	5.72	7.84	4.71	1.22	60.6	202.6
7	13	140	10.0	6.98	7.90	6.87	1.12	41.4	212.1
8	9	176	8.0	10.74	7.09	8.92	1.06	30.1	188.7

EE was calculated using the MT model (EE_{MT}) and compared to measured CPET values (EE_{CPET}) and speed-predicted estimates (EE_{SPEED}). The running activity factor (AF_b) ranged from 4.7 to 10.7 beats/kcal, indicating substantial inter-individual differences in heart rate-EE relationships. Oxygen cost per km (RE) followed a U-shaped curve during walking and stabilized near 200 mL O2/kg/km during running, consistent with individual fitness levels.

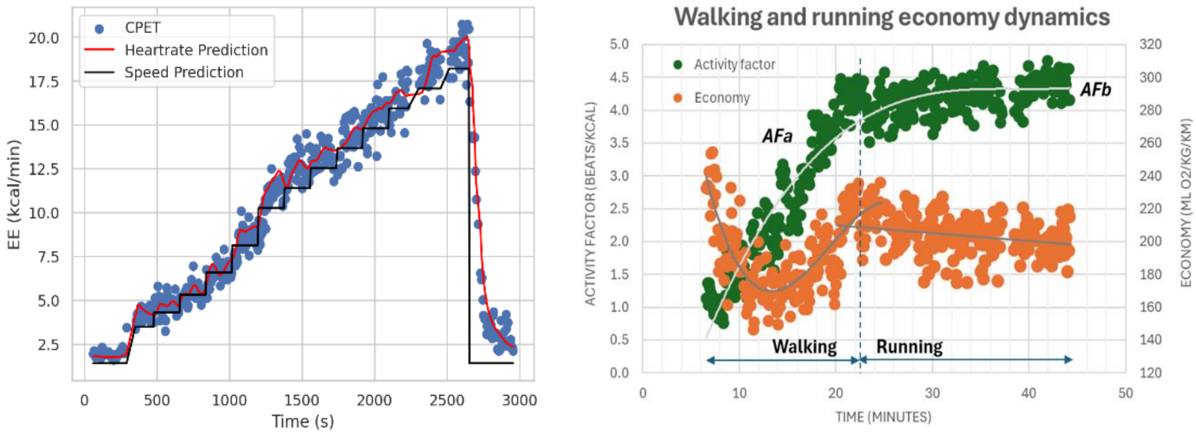

Figure 2. *Comparison of measured and predicted energy expenditure.*

Energy expenditure (EE) over time for different subjects during activity trials, comparing EE_{HR} (red line) and EE_{SPEED} (black line) with measured EE_{CPET} (blue dots). The last panel shows the activity factor (AF_a and AF_b) and running economy (RE) over time for one subject during the activity trial.

Figure 2 shows that the deviation in measured and predicted total EE was less than 2.6% for all the 8 participants and averaged only 0.25%. The speed-predicted EE, on the other hand, deviated from measured by 6.5% on average, mainly because of the poor fit during recovery (Table 2).

Table 2. *Comparisons of predicted and measured energy expenditure during the activity trials.*

Cardiorespiratory fitness parameters, including maximum heart rate (HR_{max}), max speed (V_{max}), and base heart rate (P_{zero}), and deviations (Dev.) between predicted (EE_{MT} and EE_{SP}) and measured (EE_{CPET}) total energy expenditure (EE) for all the 8 participants in the activity trials.

No.	HR_{max} (bpm)	V_{max} (kph)	P_{zero} (bpm)	EE_{CPET} (kcal)	EE_{MT} (kcal)	Dev. (%)	EE_{SP} (kcal)	Dev. (%)
1	189	14.0	95	140.5	137.9	-1.9	124.2	-11.6
2	175	17.2	73	264.0	266.6	1.0	273.2	3.5
3	211	23.6	90	449.7	453.2	0.8	386.2	-14.1
4	194	18.2	64	150.8	150.2	-0.4	142.1	5.8
5	190	31.1	43	336.6	336.4	-0.1	344.0	2.2
6	184	30.8	66	485.2	497.9	2.6	443.4	-8.6
7	164	20.3	70	258.6	256.3	-0.9	222.1	-14.1
8	199	17.5	73	141.5	142.7	0.9	122.9	-13.1

Feeding Trial

Postprandial physiological responses exhibited sharp increases in HR, EE, VO2, VCO2, glucose, and body temperature within the first hour after meal intake, peaking around 45 minutes, followed by a gradual return to baseline within 6–8 hours (Figure 3). Fat oxidation dropped post-meal, while carbohydrate oxidation spiked transiently, reflecting substrate switching.

Metabolic responses were modeled using fourth-order polynomial fits to derive total HR and EE responses. The duration of the feeding response (d) was strongly correlated with the energy content of the meal and ranged between about 5.5 – 9.0 hours for different subjects (Table 3).

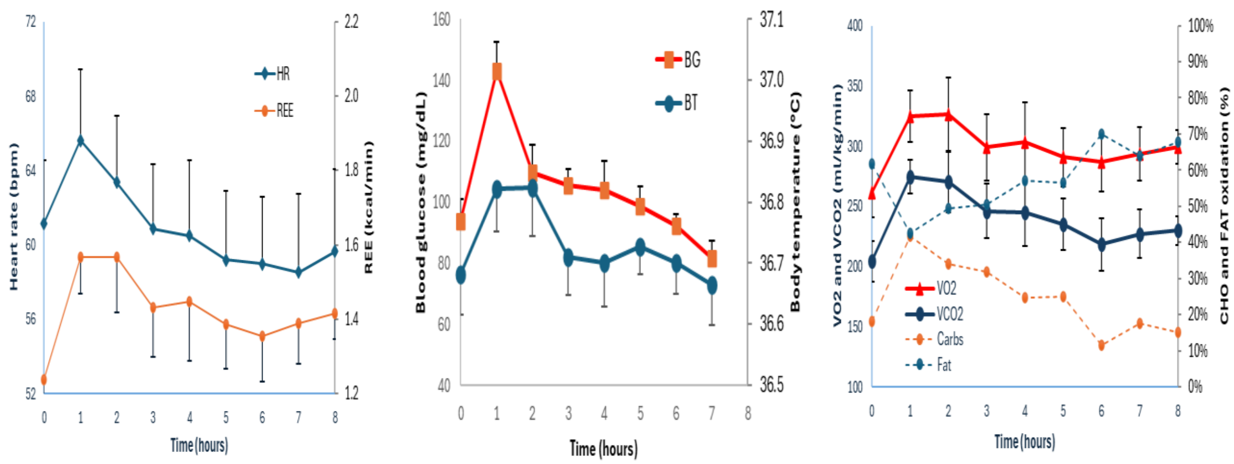

Figure 3. *Average post-prandial physiological responses (±SE) during 7-8 hours after meal intake.* (A) Resting heart rate (HR) and Resting energy expenditure (REE). (B) Blood glucose levels (BG) and Body temperature (BT). (C) Oxygen uptake (VO2) and Carbon dioxide output (VCO2) (whole lines). Carb oxidation (CHO) and Fat oxidation (FAT) (dashed lines).

The quantified food-induced metabolic responses (Sum) varied greatly, with HR_{total} ranging between 1176 – 2787 heartbeats (food beats), and EE_{total} ranging between 54 – 160 kcal (thermic calories). The measured food factor (FF) also showed great inter-individual variability, ranging between 1.50 – 4.72 beats per ingested kcal. The thermic effect of food (TEF) averaged 12.6%, ranging from 10.9% to 14.8%. This TEF value was set as a default in the metabolic model.

Table 3. *The post-prandial metabolic response measured with indirect calorimetry.*

Polynomial equation terms (a-e) from Figure 1 with coefficients of determination (R^2), response duration (d) and total response (Sum) in heart rate (HR, beats) and energy expenditure (EE, kcal). Thermic factor (TF), food factor (FF) and thermic effect of food (TEF) for all the 8 subjects in the feeding trial.

No.		a	b	c	d	e	R^2	d	Sum	TF	FF	TEF
1	HR	-0.0557	0.8245	-4.286	8.483	67.456	0.83	6.0	2001	26.6	3.95	0.148
	EE	-0.0127	0.1607	-0.657	0.893	1.169	0.69	6.0	75			
2	HR	-0.0521	0.9550	-5.901	12.410	60.611	0.87	8.0	2600	30.2	3.44	0.114
	EE	-0.0018	0.0316	-0.196	0.433	1.414	0.59	8.0	86			
3	HR	-0.0459	0.9048	-5.983	13.189	66.514	0.67	8.5	2787	23.9	2.92	0.122
	EE	-0.0022	0.0379	-0.236	0.554	1.535	0.67	8.5	117			
4	HR	-0.4200	5.2460	-21.624	28.727	59.388	0.99	5.5	2040	37.6	4.72	0.125
	EE	-0.0100	0.1153	-0.458	0.643	0.699	0.60	5.5	54			
5	HR	-0.0461	0.8325	-4.757	8.280	36.928	0.81	7.5	1176	12.1	1.50	0.124
	EE	-0.0011	0.0163	-0.089	0.169	1.088	0.62	7.5	98			
6	HR	-0.0254	0.6101	-4.852	12.550	50.774	0.94	9.0	2493	15.6	2.31	0.149
	EE	-0.0014	0.0350	-0.286	0.800	1.077	0.88	9.0	160			
7	HR	-0.1175	1.7732	-8.613	13.364	52.158	0.81	6.5	1395	22.1	2.39	0.109
	EE	-0.0022	0.0350	-0.177	0.305	0.945	0.55	6.5	63			
8	HR	-0.1130	1.9809	-11.090	19.668	70.907	0.97	6.0	1984	28.6	3.29	0.115
	EE	0.0021	-0.0174	-0.028	0.312	0.883	0.93	6.0	69			

Metabolic Tracking Validation

The MT model fitted multiple thermogenic food waves to postprandial heart rate data and allocated heartbeats accordingly into base, activity, and food-related components to estimate EE (Figure 4). There was a large variation in metabolic parameters among the study participants, with BF ranging from 40 – 75 beats/kcal and FF from 1.77 – 4.50 beats/kcal. Modeled EI closely aligned with measured intake, with a mean deviation of –1.6% (range –0.7% to –9.8%), as shown in Figure 5. This supports the validity of the food factor–based estimation approach under controlled conditions (Table 4).

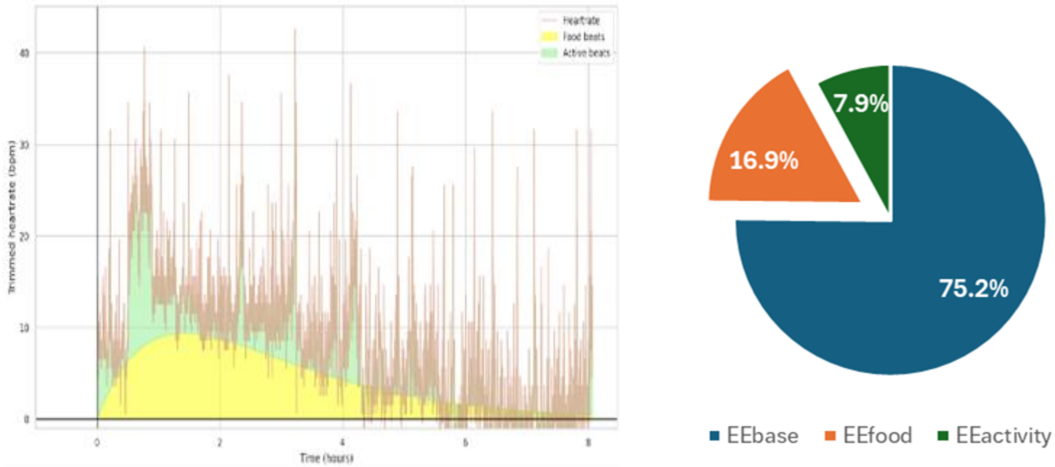

Figure 4. *Example thermogenic EE estimation from the post-prandial heart rate response.*
A) Thermogenic food wave fitting to allocate food beats (yellow area), activity beats (green area) and base beats (not shown). B) Estimated relative EE components based on heartbeat allocation.

Table 4. *Energy expenditure and energy intake calculations in the single meal feeding study.*
Base beats (BB), activity beats (AB), and food beats (FB) correspond to heart rate components attributed to base- (BE), active- (AE), and food energy expenditure (FE), respectively, summing up to total energy expenditure (EE). Basal heart rates (BHR), basal metabolic rates (BMR), metabolic factors (BF and FF), and FF test values (FF_b) are listed. Measured (EI) and calculated (EI_b) energy intake with deviations.

	BB	FB	AB	BE	FE	AE	EE	BHR	BMR	BF	FF	FF_b	EI	EI_b	Dev
1	33,281	2,276	2,273	547	63	50	660	69.2	1.14	60.8	4.50	4.37	506	521	2.9
2	28,166	2,350	1,712	697	95	57	848	59.6	1.48	40.4	3.11	3.13	756	751	-0.7
3	28,273	2,758	2,549	700	119	84	903	60.4	1.50	40.4	2.89	3.05	955	904	-5.3
4	26,291	1,807	2,660	350	54	47	451	56.6	0.75	75.1	4.18	3.93	432	460	6.4
5	16,476	1,390	1,781	414	98	60	572	34.8	0.87	39.8	1.77	1.83	785	760	-3.2
6	23,914	2,119	1,890	599	135	63	797	49.4	1.24	39.9	1.97	1.90	1077	1115	3.6
7	22,583	1,522	2,169	480	73	62	615	49.0	1.04	47.0	2.61	2.80	583	544	-6.8
8	23,590	2,389	2,581	394	75	57	527	67.4	1.13	59.9	3.96	4.39	603	544	-9.8

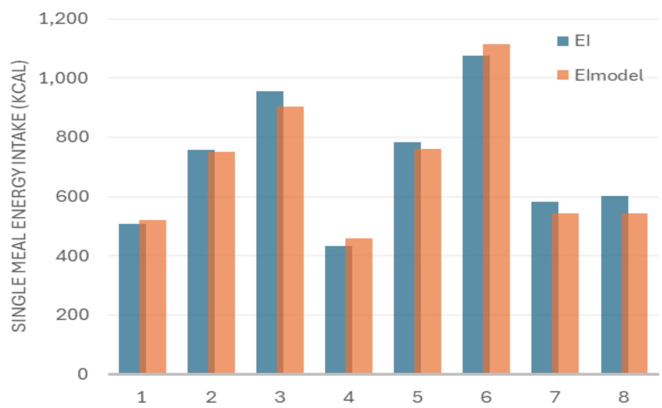

Figure 5. *Comparison of measured and modeled energy intake in the feeding trial.*
Measured energy intake (EI) from food consumption (blue bars) was compared to energy intake estimated by the metabolic model (orange bars).

Blood profiles and glycemic control

Fasting blood biomarkers revealed inter-individual variability in glucose, insulin, lipids, thyroid hormone, cortisol, and sex hormones (Table 5). Participants with elevated fasting triglycerides, glucose, and insulin also showed higher postprandial glucose responses and lower MFI scores.

Table 5. *Fasting blood sample analysis from participants in the feeding study.*
Fasting blood biomarkers measured for the eight participants in the feeding study: glucose (Glu.), insulin (Ins.), cholesterol (Chol.), high-density lipoproteins (HDL), low-density lipoproteins (LDL), triglycerides (Trigl.), thyroid-stimulating hormone (TSH), estradiol (Estra.), testosterone (Testo.), and cortisol (Cort.). Gender (Gen) is reported for reference. Bold values are beyond the clinic's normative range.

Part	Gluc.	Ins.	Chol.	HDL	LDL	Trigl.	TSH	Estra.	Testo.	Cort.
Nr	mmol/l	mU/L	mmol/l	mmol/l	mmol/l	mmol/l	mU/l	pmol/L	nmol/l	nmol/l
1	4.5	3.8	4.3	1.2	2.7	0.9	3.1	67	25	516
2	4.9	2.0	3.4	1.8	1.4	0.6	2.6	92	19	309
3	4.8	2.2	5.7	1.6	3.8	0.6	1.9	51	23	303
4	5.9	**17.9**	4.1	1.1	2.2	**1.5**	2.4	110	11	463
5	5.6	**10.3**	**6.8**	1.8	**4.5**	1.0	2.9	10	0.05	516
6	4.8	5.1	4.2	1.7	2.2	0.6	3.3	**476**	1.0	320
7	4.9	4.2	4.6	2.1	2.3	0.5	2.3	223	0.8	411
8	5.0	2.1	5.2	2.1	2.7	0.7	1.9	**1534**	1.3	396

The Glycemic Response Index (GRI), calculated as peak glucose relative to glycemic load, correlated strongly with the Metabolic Fitness Index (MFI), suggesting that MFI could serve as a non-invasive proxy for glycemic control and metabolic flexibility. Figure 6 illustrates these relationships, highlighting the inverse association between GRI and cardiorespiratory fitness.

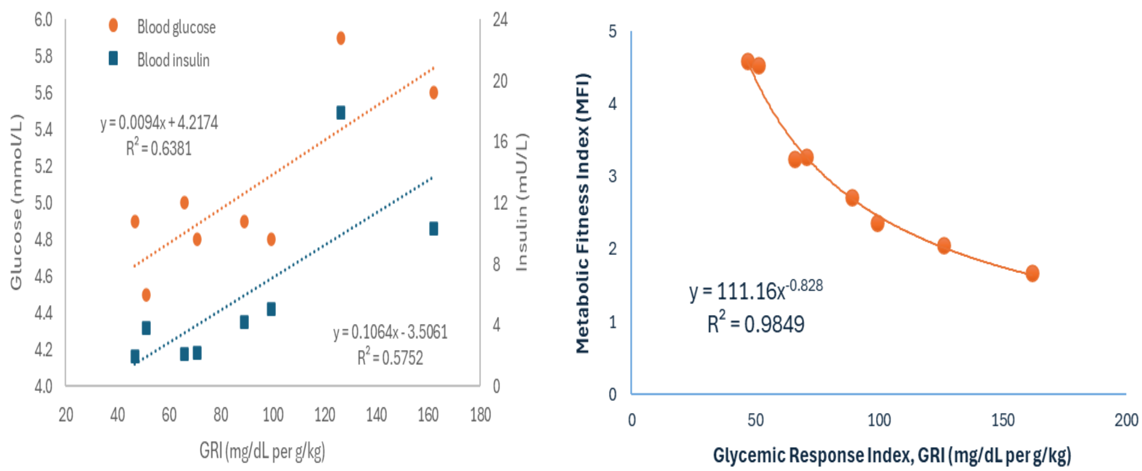

Figure 6. *Relation of glycemic response index and blood biomarkers to cardiorespiratory fitness.* (Left) The relationship between fasting blood glucose (red circles) and fasting insulin (blue squares) with the glycemic response index (GRI). (Right) The power-based relationship between GRI and MFI for the eight subjects in the feeding study. The regression equations are shown on the graphs.

Fasting blood glucose (GLU) and insulin levels (INS) were shown to be strongly intercorrelated ($R^2 = 0.79$, $p<0.01$). GLU was also correlated with BMI ($R^2 = 0.63$, $p<0.01$), peak post-prandial glucose ($R^2 = 0.49$, $p<0.01$) and fasting triglycerides ($R^2 = 0.60$, $p<0.01$). INS was also correlated with BMI ($R^2 = 0.67$, $p<0.01$), peak post-prandial glucose ($R^2 = 0.58$, $p<0.01$) and fasting triglycerides ($R^2 = 0.83$, $p<0.01$).

Overall, the results indicate that heart rate–based metabolic modeling can reliably track energy intake, energy expenditure, and glycemic control dynamics, offering a non-invasive method for metabolic health monitoring. The robust power-based relationship between GRI and MFI suggests that MFI could serve as an indirect indicator or proxy for glycemic control and metabolic flexibility.

Discussion

Study Design and Participant Considerations

This pilot study was constrained by practical limitations, including participant availability and equipment. A novel interspersed calorimetry design allowed simultaneous testing of four individuals, maximizing efficiency. The sample included a balanced gender mix and a broad range of ages, fitness levels, and body compositions. Participants on blood pressure medications were not excluded, supporting broader generalizability. While the sample size was modest, the high number of repeated measurements per participant adds robustness.

Validation of the Driftline Metabolic Model

Findings confirm the feasibility of estimating energy intake (EI) and expenditure (EE) from heart rate dynamics. The Driftline Metabolic Model showed strong agreement with calorimetry-based measurements (EE deviation: 0.25%; EI deviation: -1.6%). Accurate modeling relied on proper alignment of basal HR, food wave fitting, and individualized food factor (FF) values. The model excelled during walking and recovery phases — conditions that often challenge traditional EE estimation. Static exercise modes like cycling were not tested, suggesting a direction for future research.

Activity Trials and Metabolic Flexibility

Activity trials revealed exercise thresholds and efficiency metrics such as economical speed (ES: 5.0 kph) and energetically optimal transition speed (EOTS: 7.3 kph), consistent with prior studies. Elevated resting HR and REE may reflect pre-test anxiety or residual food effects. One participant displayed a blunted RER at high intensity (0.86), indicating metabolic inflexibility. This may be linked to impaired mitochondrial function and substrate switching, supporting the integration of metabolic flexibility into fitness assessments.

Postprandial Metabolic Responses

Postprandial measurements revealed wave-like HR and metabolic fluctuations that scaled with meal size. Most responses returned to baseline within 8 hours, with extended duration for the largest meal. A 4th-order polynomial model effectively captured these dynamics. Despite non-continuous sampling and incidental movement, synchronized trends among HR, REE, and other metrics support the model's validity. Factors such as ambient conditions, anxiety, or incidental activity likely contributed to REE variability.

Future Directions and Applications

The Driftline model segments heartbeats into basal, activity, and food components to infer individualized metabolic parameters. While promising, the model's response to diverse meal types (e.g., high-protein, high-fat) remains unexplored. Most TEF studies last only 2–3 hours, whereas our design extended to 8 hours, allowing a broader evaluation. However, each participant consumed only one standardized meal, limiting within-subject analysis of meal size effects. Future studies should assess both composition and size.

Glycemic Response and the Metabolic Fitness Index

The Glycemic Response Index (GRI) was introduced as a normalized measure of postprandial glycemic response. The Metabolic Fitness Index (MFI)—a dimensionless metric based on heartbeats per meter walked, scaled by height$^{2.5}$ and HRmax—correlated strongly with GRI. Higher MFI scores reflected better glycemic control and metabolic flexibility. MFI was also inversely associated with fat percentage and fasting metabolic risk markers. As a non-invasive proxy for glycemic control, MFI could enhance fitness screening, wearable tracking, and exercise prescription targeting metabolic health.

References

1. Abe, D., Fukuoka, Y., & Horiuchi, M. (2017). Muscle activities during walking and running at energetically optimal transition speed under normobaric hypoxia on gradient slopes. *PloS one*, *12*(3), e0173816. https://doi.org/10.1371/journal.pone.0173816
2. Aita, S., Matsushita, M., Yoneshiro, T., Hatano, T., Kameya, T., Ohkubo, I., & Saito, M. (2022). Brown fat-associated postprandial thermogenesis in humans: Different effects of isocaloric meals rich in carbohydrate, fat, and protein. *Frontiers in nutrition*, *9*, 1040444. https://doi.org/10.3389/fnut.2022.1040444
3. Brosh, A. (2007). Heart rate measurements as an index of energy expenditure and energy balance in ruminants. Livestock Science, 110(1–2), 79–91. https://doi.org/10.1016/j.livsci.2006.03.018
4. Bruce, C. R., Anderson, M. J., Carey, A. L., Newman, D. G., Bonen, A., Kriketos, A. D., & Hawley, J. A. (2000). Muscle oxidative capacity is a better predictor of insulin sensitivity than lipid status. The Journal of Clinical Endocrinology & Metabolism, 85(11), 4094–4097. https://doi.org/10.1210/jcem.85.11.6984
5. Burke, E. (2019). (Manufacturer's Brochure) K5: Wearable metabolic system for both laboratory and field testing. COSMED. Retrieved from COSMED resources.
6. Calcagno, M., Kahleova, H., Alwarith, J., Burgess, N. N., Flores, R. A., Busta, M. L., & Barnard, N. D. (2019). The Thermic Effect of Food: A Review. Journal of the American College of Nutrition, 38(6), 547–551. https://doi.org/10.1080/07315724.2018.1552544
7. Cesari, M., Landi, F., Vellas, B., Bernabei, R., & Marzetti, E. (2022). Sarcopenia and frailty: From theoretical concepts to clinical impact. Frontiers in Medicine, 9, 841019. https://doi.org/10.3389/fmed.2022.841019
8. Chooi, Y. C., Ding, C., & Magkos, F. (2019). The epidemiology of obesity. Metabolism, 92, 6–10. https://doi.org/10.1016/j.metabol.2018.09.005
9. de Jonge, L., & Bray, G. A. (1997). The thermic effect of food and obesity: A critical review. Obesity Research, 5(6), 622–631. https://doi.org/10.1002/j.1550-8528.1997.tb00692.x
10. Eurostat. (2023). Ageing Europe – Looking at the lives of older people in the EU. Publications Office of the European Union. https://ec.europa.eu/eurostat

11. Galgani, J. E., & Ravussin, E. (2008). Energy metabolism, fuel selection and body weight regulation. International Journal of Obesity, 32(S7), S109–S119. https://doi.org/10.1038/ijo.2008.246
12. Goodpaster, B. H., Katsiaras, A., & Kelley, D. E. (2003). Enhanced fat oxidation through physical activity is associated with improvements in insulin sensitivity in obesity. Diabetes, 52(9), 2191–2197. https://doi.org/10.2337/diabetes.52.9.2191
13. Hall, K. D., & Guo, J. (2017). Obesity energetics: body weight regulation and the effects of diet composition. Gastroenterology, 152(7), 1718–1727.e3. https://doi.org/10.1053/j.gastro.2017.01.052
14. Heymsfield, S. B., Peterson, C. M., Thomas, D. M., Heo, M., & Schuna, J. M. (2016). Why are we fat? Obesity beyond the energy balance equation. Obesity Reviews, 17(2), 90–95. https://doi.org/10.1111/obr.12340
15. Kelley, D. E., He, J., Menshikova, E. V., & Ritov, V. B. (2002). Dysfunction of mitochondria in human skeletal muscle in type 2 diabetes. Diabetes, 51(10), 2944–2950. https://doi.org/10.2337/diabetes.51.10.2944
16. Kim, J. A., Wei, Y., & Sowers, J. R. (2008). Role of mitochondrial dysfunction in insulin resistance. Circulation research, 102(4), 401–414. https://doi.org/10.1161/CIRCRESAHA.107.165472
17. O'Driscoll, R., Turicchi, J., Beaulieu, K., Scott, S., Matu, J., Deighton, K., & Finlayson, G. (2020). How well do activity monitors estimate energy expenditure? A systematic review and meta-analysis of the validity of current technologies. British Journal of Sports Medicine, 54(6), 332–340. https://doi.org/10.1136/bjsports-2018-099643
18. San-Millán, I., & Brooks, G. A. (2018). Assessment of Metabolic Flexibility by Means of Measuring Blood Lactate, Fat, and Carbohydrate Oxidation Responses to Exercise in Professional Endurance Athletes and Less-Fit Individuals. Sports medicine (Auckland, N.Z.), 48(2), 467–479. https://doi.org/10.1007/s40279-017-0751-x
19. Sergi, D., Naumovski, N., Heilbronn, L. K., Abeywardena, M., O'Callaghan, N., Lionetti, L., & Luscombe-Marsh, N. (2019). Mitochondrial (Dys)function and Insulin Resistance: From Pathophysiological Molecular Mechanisms to the Impact of Diet. *Frontiers in physiology*, *10*, 532. https://doi.org/10.3389/fphys.2019.00532

20. Solomon, T. P., Malin, S. K., Karstoft, K., Knudsen, S. H., Haus, J. M., Laye, M. J., & Kirwan, J. P. (2015). Association between cardiorespiratory fitness and the determinants of glycemic control across the entire glucose tolerance continuum. *Diabetes care*, *38*(5), 921–929. https://doi.org/10.2337/dc14-2813
21. The jamovi project (2022). jamovi. (Version 2.3) [Computer Software]. Retrieved from https://www.jamovi.org.
22. Tzeravini, E., Anastasios, T., Alexander, K., Nikolaos, T., & Nikolaos, K. (2024). Diet induced thermogenesis, older and newer data with emphasis on obesity and diabetes mellitus - A narrative review. *Metabolism open*, *22*, 100291. https://doi.org/10.1016/j.metop.2024.100291
23. Uphill, M., Majumdar, S., Barnes, A., Mahadevan, V., Broom, D., & Byrne, H. (2024). The accuracy of wearable devices for estimating energy expenditure across exercise modalities and intensities. European Journal of Sport Science, 24(4), 614–625. https://doi.org/10.1080/17461391.2023.2261500
24. Vyaire Medical. (2019). Special Edition Cardiopulmonary Exercise Testing. Eschenbacher, H. (ed). CareFusion Germany 234 GmbH. https://www.vyaire.com/sites/us/files/2019-04/Special-Edition-CPET_EN_Web.pdf
25. Weir, J. B. de V. (1949). New methods for calculating metabolic rate with special reference to protein metabolism. The Journal of Physiology, 109(1–2), 1–9. https://doi.org/10.1113/jphysiol.1949.sp004363
26. Westerterp, K. R. (2004). Diet induced thermogenesis. Nutrition & Metabolism, 1(1), 5. https://doi.org/10.1186/1743-7075-1-5
27. World Health Organization – Europe. (2023). European regional obesity report 2022. https://www.who.int/europe/publications/i/item/9789289057738
28. World Health Organization. (2024). Obesity and overweight. https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight