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Deceleration Capacity of the Heart Rate: Measurement Characteristics and Biological Determinants

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Abstract

Purpose

Deceleration capacity is becoming a more widely used predictor for health outcomes and cardiac autonomic dysfunction, but its measurement characteristics and biological determinants remain insufficiently understood. This study examined these factors in a large population-based sample (n = 1291) from the Netherlands Twin Register.

Methods

Participants wore an physiological monitoring device for 24 hours, and deceleration capacity was computed through phase-rectified signal averaging. To test whether short recordings might suffice, deceleration capacity estimates derived from 5-minute segments were compared with estimates obtained from full 24-hour ambulatory recordings. Temporal stability was examined in a subsample of participants measured again roughly three years later, and twin modeling was used to determine how much of the individual variance was explained by environmental and genetic factors.

Results

The results indicated that short-term deceleration capacity derived from 5-minute segments can deviate substantially from those derived from the full 24-hour recording, and that these deviations vary systematically across the day. It showed moderate to strong temporal stability across two measurement occasions roughly three years apart (r = .74, ICC(A,1) = .74). In addition, deceleration capacity was negatively associated with age (β = -0.04, R^2 = .14) and BMI (β = -0.04, R^2 = .03), and was higher in males than females (M = 4.70 vs M = 4.12). We further found substantial heritability of deceleration capacity (h^2 = .62).

Conclusions

Together, these findings establish that 24-hour DC reflects a stable biological trait, which is in line with its prognostic potential for health outcomes.

1 | Introduction

Deceleration capacity (DC) is a cardiac measure that quantifies the slowing down of heart rate (HR) between successive heart beats. It expresses, in milliseconds, how much the time interval between adjacent heart beats (RR interval) typically lengthens around a spontaneous deceleration. It is computed through phase-rectified signal averaging (PRSA), which effectively aligns and averages the change in RR intervals at points where an RR interval is longer than the preceding interval. The beat-to-beat

prolongation of RR intervals, or beat-to-beat deceleration of HR, is thought to be primarily mediated by parasympathetic (vagal) influences on the sinoatrial node of the heart [1, 2]. Therefore, DC has been proposed as a relatively specific marker for cardiac vagal control in comparison with conventional heart rate variability (HRV) metrics [2–4].

The concept of DC was originally developed to improve risk stratification after myocardial infarction [1]. DC was found to have higher prognostic value in predicting mortality than left-ventricular ejection fraction and conventional HRV metrics [1]. Subsequent studies have provided additional evidence that DC has prognostic value for predicting mortality and cardiovascular health outcomes [2, 4–11]. Its clinical relevance has been demonstrated across a range of cardiovascular health outcomes, such as non-ischemic dilated cardiomyopathy [5], stable angina pectoris [12], and aortic stenosis [4]. Clinical applications have also extended beyond cardiovascular health outcomes, such as Parkinson's disease [13], renal disease [14], and vasovagal syncope [11, 15].

With DC becoming a more widely used marker for health outcomes and cardiac autonomic dysfunction, a better understanding of its measurement characteristics is needed. Most of the above studies derived DC from 24 hour recordings in daily life, but some studies have suggested that shorter recordings (even five minutes) can be used without losing prognostic power [7, 10]. This would have large practical utility for clinical risk prediction. However, estimates derived from such short recordings may be particularly sensitive to the time of day. Given the strong circadian effects on other HRV metrics (e.g., Burgess et al., 1997; Guo & Stein, 2002; Malpas & Purdie, 1990), these may be present in DC as well. Failure to account for recording time could introduce systematic or residual confounding. As a first aim, this study investigates whether DC estimates derived from 5-minute segments are comparable to estimates obtained from full 24-hour ambulatory recordings. We will also establish the pattern and extent of circadian variation in the 5-minute DC values.

As a second aim, we will investigate the temporal stability of DC. The temporal stability of DC has rarely been investigated. Most studies obtained DC only once and either used it to predict future health outcomes, or to discriminate between groups (e.g., patients and healthy controls). One study obtained DC up to 12 times within individuals, but did not explicitly report the temporal stability across repeated measurements [12]. It is therefore still unclear to what extent DC reflects a stable phenotype.

Similarly, relatively little is known about the biological determinants of DC. Do we need to account for the impact of demographic and anthropometric traits when using DC for risk stratification? In particular, age, sex, and body composition could be confounders of the association between DC and health outcomes. The third aim, therefore, is to examine the associations between DC and age, sex, and BMI. If these associations are strong, these individual characteristics should be taken into account in prediction models as is now routinely done for HRV measures. For HRV measures, associations with stable individual characteristics (e.g., age, sex, BMI) are well-established [19–21], but these associations are currently underexplored for DC.

The prognostic value of DC could either reflect an innate vulnerability in autonomic regulation or some environmental exposure impacting on the autonomic nervous system, like chronic stressor exposure [22, 23]. To date, the relative contributions of genetic and environmental factors to DC are largely unknown. The fourth aim is to examine the relative contribution of genetic and environmental factors to DC. Twin and family studies can provide insight into the extent to which differences in DC between individuals are attributable to genetic or environmental influences. They have shown that HR and HRV metrics have a substantial heritable component [24–30]. Parallel evidence for heritability of DC is absent, and environmental factors might be more important. The only study of DC to include twin pairs (Huang et al., 2024) controlled their analyses for confounding through shared genetics, but did not report heritability.

The present study will address these aims in a population of 1291 individuals from the Netherlands Twin Register. Specifically, we examine (1) the impact of recording duration and circadian variation on DC, (2) the temporal stability of DC, (3) the association of DC with stable individual characteristics, and (4) the relative contribution of genetic and environmental factors to DC. By characterizing the measurement characteristics and biological determinants, this study aims to strengthen the utility of DC for disease prediction.

2 | Methods

2.1 | Participants

The study sample comprised 1366 participants from the Netherlands Twin Register (NTR), a longitudinal twin cohort maintained by the department of Biological Psychology at Vrije Universiteit Amsterdam [31]. The NTR collects genetic samples and longitudinal questionnaire data on personality, lifestyle, and health. Additionally, detailed biological and psychological measures are actively collected for specific projects. The present study used data collected between 1998 and 2012 from NTR projects that combined the genetic samples from biobank data with ambulatory physiological recordings [24, 32].

These studies included healthy twins aged 14 to 80 years and their non-twin siblings, provided the age of the non-twin siblings was within three years of the twins. Only families for whom genetic samples were available for at least two family members were selected to participate. The selected participants were then screened for medical conditions that can influence the cardiovascular system or autonomic nervous system. Individuals with known cardiovascular disease or other relevant conditions were excluded (e.g., ischemic heart disease, thyroid disease, hypertension, diabetic neuropathy). Additional exclusion criteria were pregnancy, heart transplantation, or having a pacemaker. All study protocols were approved by the VUmc Medical Ethics Committee, and all participants provided written informed consent.

2.2 | Procedure

Participants who were willing to participate started the procedure by filling in questionnaires about sleep quality, life events, and personality traits at home. The research team then visited the participants at

home to conduct further interviews (about health, sleep quality, medication use among others), to measure anthropometrics, and to set up equipment.

Participants were instructed to wear equipment that recorded physiological and accelerometer data continuously for 24 hours. They were explicitly asked to maintain their typical daily routines, with the only exception being vigorous physical activity. They further received detailed instructions on identifying and resolving possible technical issues, including how to reattach loose electrodes. Telephone assistance was available during typical waking hours.

2.3 | Materials

Participants wore a Vrije Universiteit Ambulatory Monitoring System (VU-AMS) device to collect electrocardiogram (ECG), impedance cardiogram (ICG), and accelerometer signals with a 1000 Hz sampling frequency [33]. Participants carried the device in a dedicated carrying bag secured by a belt on the left hip. Two different versions of the device were used across studies (version 4.6 and 5fs). ECG and ICG signals were obtained using seven electrodes in both devices. One combined ECG and ICG electrode was placed on the sternum, between the medial ends of the clavicles. A second ECG electrode was placed near the cardiac apex over the left ninth rib. A second ICG electrode was positioned right underneath the xiphoid process. Two electrodes supplying the ICG current were attached to the back along the spinal column, one at the level of the fourth cervical vertebra and the other between the eighth and ninth thoracic vertebrae.

2.4 | Data processing

Preprocessing of the ECG signal was carried out in the dedicated analysis software for VU-AMS recordings (VU-DAMS for Windows, Vrije Universiteit, Amsterdam, the Netherlands). The R-peaks were algorithmically detected, and beats with an interbeat interval (IBI) deviating from the local mean and variability were flagged as artifacts and excluded. This was double-checked by visual inspection of the detected R-peaks in combination with the raw ECG signal.

The IBI time series were exported from VU-DAMS and read into Python (version 3.9, Python Software Foundation, Wilmington, Delaware, USA). DC was calculated from these IBI time series using a custom phase-rectified signal averaging (PRSA) function [34]. IBIs marked as interpolated were treated as missing, and analysis windows that contained more than 20% missing IBIs were excluded from analysis. Deceleration anchors were identified by classifying which IBI was longer than the preceding IBI. In line with Bauer et al. (2006), successive IBI prolongations of more than 5% were excluded.

For all deceleration anchors, a segment containing the 30 IBIs preceding the anchor and the following 30 IBIs were extracted. These segments were aligned at the anchor, and the aligned datapoints of all segments were averaged. This is the PRSA curve. From this PRSA curve, DC was computed using a wavelet scale of two, meaning that two measurements on each side of the anchor were included. Specifically, the two values directly preceding the anchor were subtracted from the sum of the two values at and directly after the anchor, and then divided by four. This led to the following formula: “DC =

(X(0) + X(1) – X(-1) – X(-2)) / 4” [1]. Since DC is directly calculated from IBIs, it is also expressed in milliseconds (ms).

2.5 | Statistical analysis

2.5.1 | Descriptives

The number of participants excluded at each data processing stage was reported to make clear how the final study sample was obtained. Individual characteristics of the final study sample were summarized using the mean and standard deviation (SD) for age and BMI, and counts for biological sex. Recording duration and data quality were described as the number of recording hours and as percentage missingness in the IBI time series data. We further reported elaborate descriptive statistics for DC: mean, SD, minimum, 25th percentile, median, 75th percentile, and maximum.

A correlation table was reported to characterize the relationship between DC and conventional time-domain HRV metrics. The correlation table included DC, acceleration capacity (AC), standard deviation of normal-to-normal intervals (SDNN), and root-mean-square of successive differences (RMSSD). The computation of AC is comparable to DC, the main difference is that it focuses on acceleration of HR instead of deceleration. Specifically, the anchors were identified whenever an RR-intervals shortened in comparison with the previous RR-interval. SDNN was computed as the standard deviation of all RR-intervals, and RMSSD was computed as the square root of the mean of the squared differences between successive RR-intervals.

2.5.2 | Measurement Characteristics

The first aim was to examine the impact of recording duration and circadian variation on DC. Although DC is typically derived from 24-hour ambulatory recordings, some studies have estimated DC using shorter recordings (e.g., 5 minutes). The present study systematically compared DC estimates derived from complete 24-hour recordings with those obtained from 5-minute segments. The full recordings were divided into non-overlapping 5-minute segments (00:00–00:05, 00:05 – 00:10, et cetera). DC was derived for both the full recording and these 5-minute segments. For each segment, ΔDC was defined as the difference between the 5-minute segment estimates and the corresponding 24-hour estimate.

To quantify the agreement between DC derived from the full recording and DC derived from 5-minute segments, we computed the bias, mean absolute error (MAE), and root-mean-square error (RMSE) for every participant. Bias was computed as the mean difference, MAE as the mean absolute difference, and RMSE as the square root of the squared differences. For these three metrics, we reported the mean and SD across participants.

The circadian variation of the 5-minute estimates of DC was visualized by plotting these estimates against the time of day. First, we visualized the deviation of the difference between 5-minute segment estimates of DC and the corresponding 24-hour estimate (ΔDC). The ΔDC was standardized by dividing it by the participant-specific SD across all 5-minute estimates, and visualized as the average and 95%

confidence interval per 5-minute segment across all participants. Values above zero therefore indicate that 5-minute DC was on average higher than the corresponding 24-hour DC, whereas values below zero indicate that it was on average lower. For risk stratification, the relevant question is not only how closely a 5-minute measurement approximates the 24-hour value in absolute terms, but also whether it preserves the rank order of individuals. Therefore, we computed and visualized the Pearson's correlation coefficient and 95% confidence intervals between the 5-minute estimate and 24-hour estimate over participants for each 5-minute segment.

The second aim was to examine the temporal stability of 24-hour DC across several years. Fifty-six participants completed a second day of ambulatory monitoring using the same procedure and equipment approximately three years after the initial assessment. The mean (and SD) time between two measurement occasions was reported. The temporal stability across measurement occasions was examined using both the Pearson correlation coefficient ($r_{t1,t2}$) and intraclass correlation coefficient (ICC). We extracted the random-effect single-measure ICC (ICC(A,1)) and its 95% confidence interval from the Pingouin package (version 0.5.5) in Python.

2.5.3 | Biological Determinants

The third aim was to examine the association of DC with stable demographic and anthropometric characteristics. We reported the unadjusted associations of DC with individual characteristics first, but adjusted effects were accounted for in the genetic model below. Simple linear regression with unstandardized variables was used to examine the association of DC with age and BMI. This was performed using the Statsmodel package (version 0.13.2) in Python. An independent t-test was used to examine the difference in DC between males and females. This was performed using the SciPy package (version 1.11.2) in Python. The significance level was set conservatively to adjust for multiple testing ($\alpha = .01$).

The fourth aim was to examine the relative contribution of genetic and environmental factors to DC. For genetic modeling a total of 225 complete monozygotic (MZ) twin pairs, 131 dizygotic (DZ) twin pairs, 300 twin–sibling (non-twin) pairs, and 29 sibling - sibling pairs were available. This allowed the observed variance in DC to be decomposed into four possible sources of variance: variance due to additive (A) genetic effects, nonadditive (D) genetic effects, common environment (C) shared by family members, and nonshared or unique (E) environment [35]. Our sample was not powered to detect qualitative or quantitative sex differences and we restricted analyses to two zygosity groups. In the biometrical model, twin-sibling and sibling-sibling pairs were treated similarly as DZ twin pairs, in that they share half of their additive genetic variance, a quarter of their non-additive genetic variance, and have a fully shared family environment. Because a priori power for estimates of D was very low, we modeled an ACE model only. This was further justified by the observed pattern of twin and sibling correlations.

To answer the question to what extent A, C, and E factors contribute to the variance in DC, biometrical genetic models were fitted to the observed data using the structural equation modeling program OpenMx [36]. First, fully saturated models were fitted to estimate means and variances freely for both

sexes. Then, we tested for birth order or zygosity effects on means and variances by constraining these to be equal for first and second born twins and for DZ and MZ twins, and tested whether these more constrained models led to a significant worse fit to the data. Next, we tested for heterogeneity of means and variances of twin versus non-twin siblings. The resulting most parsimonious saturated model indicated to what extent we could limit the specification of the ACE variance decomposition models. To account for the potential effects on age, sex and BMI (see above), these models specifically regressed the effects of sex, age and BMI on DC before ACE variance decomposition.

3 | Results

3.1 | Descriptives

In total, 72 participants who completed the ambulatory procedure were excluded from statistical analyses for the following reasons: insufficient recording duration or data quality (less than 12 hours of valid data; n = 68), missing basic demographics (n = 4), and withdrawal of consent for data reusage (n = 3).

The final study sample consisted of 1291 participants (808F, 483M). The mean age was 33.6 years (SD = 9.7), and the mean BMI was 24.0 kg/m² (SD = 4.0). The mean recording duration was 23.2 hours (SD = 2.0), with a mean missingness of 2.1% in the IBI time series data (SD = 4.7). The descriptives for DC can be found in Table 1.

Table 1

Descriptive statistics for deceleration capacity (DC). The table reports the count, mean, standard deviation (SD), minimum (Min), 25th percentile (25%), median (50%), 75th percentile (75%), and maximum (Max). The correlation table (Table 2) shows a strong negative association between DC and AC. Furthermore, DC shows modest positive correlations with SDNN and RMSSD, whereas AC shows modest negative correlations with SDNN and moderate negative correlation with RMSSD. The association with common time-domain HRV metrics is thus slightly stronger for AC than for DC.

	Count	Mean	SD	Min	25%	50%	75%	Max
DC	1291	8.68	1.78	1.71	7.55	8.66	9.83	14.22

Table 2
Correlation table including deceleration capacity (DC), acceleration capacity (AC), standard deviation of normal-to-normal intervals (SDNN), and root-mean-square of successive differences (RMSSD).

DC	DC	AC	SDNN	RMSSD
	-			
AC	-0.94	-		
SDNN	0.40	-0.48	-	
RMSSD	0.40	-0.60	0.66	-

3.2 | Measurement Characteristics

3.2.1 | Recording Duration and Circadian Variation

The mean bias of the 5-minute DC estimates in comparison with the corresponding 24-hour estimate was 0.34 ms (SD = 0.29), the MAE was 1.95 ms (SD = 0.52), and the RMSE was 2.50 ms (SD = 0.68). The distribution of bias, MAE, and RMSE across participants can be found in Fig. 1. Although the mean bias suggested little systematic over- or underestimation of DC in shorter segments, the larger MAE and RMSE indicate that short-term DC can substantially deviate from the corresponding 24-hour estimates.

The standardized ΔDC trajectory showed a clear circadian rhythm (see Fig. 1). Five-minute DC estimates were generally higher than the corresponding 24-hour estimate during the night, and lower than the corresponding 24-hour estimate during the daytime. Two smaller increases were visible halfway during the morning and afternoon. This suggests that short-term DC estimates vary systematically across the day, explaining the large MAE and RMSE for the 5-minute DC estimates. The correlation coefficient across individuals also showed a circadian rhythm (see Fig. 1). The correlation was higher during the daytime than during the night, and remained fairly stable between .60 and .70 throughout the day. This correlation coefficients suggests moderate rank order agreement of 5-minute estimates and the 24-hour estimate of DC.

3.2.2 | Temporal Stability

Fifty-six individuals completed the retest study. The mean time between the two measurement occasions was 3.20 years (SD = 0.61). Figure 1 shows a scatterplot with DC at both measurement occasions. DC showed a significant correlation between two measurement occasions ($r_{t1,t2}$ = .75, p < .001), and moderate absolute agreement (ICC(A,1) = .74, 95% CI [.60, .84], p < .001). This suggests moderate to strong long-term temporal stability of DC.

3.3 | Biological Determinants

3.3.1 | Association with Individual Characteristics

Figure 2 shows the association of DC with individual characteristics. DC was negatively associated with age (β = -0.07, R^2 = .14, F(1,1289) = 206.58, p < .001), and with BMI (β = -0.08, R^2 = .04, F(1,1272) = 47.40, p < .001). In addition, DC was significantly higher in males (M = 9.34, SD = 1.83) than in females (M = 8.28, SD = 1.63; t(1289) = 10.82, p<.001). Age explained substantially more variance in DC than BMI did, suggesting that age may be an important characteristic to consider when using DC for risk stratification.

3.3.2 | Heritability

The age- and sex-adjusted correlations for were .61 (95% CI [.52, .69]) for MZ twin pairs, .41 (95% CI [.26, .55]) for DZ twin pairs, and .17 (95% CI [.17, .38]) for twin-sibling pairs. This indicates greater similarity among MZ twins than DZ twins or twin-sibling pairs. Saturated models showed that the assumptions to fit an ACE model were all met and the ACE model provided a good fit (see Supplementary Table S1), although the C component could be dropped without a significant reduction in model fit. In the full ACE model, additive genetic factors (A) accounted for .62 (95% CI [.39, .86]) of the variance, shared environmental factors (C) accounted for .02 (95% CI [− .20, .21]), and unique environmental factors (E) accounted for .36 (95% CI [.30, .44]). The restricted AE model had heritability estimate for DC of .62. These results suggest a large contribution of genetic factors with unique environmental factors accounting for the remaining individual differences in DC.

4 | Discussion

This study examined the measurement characteristics and biological determinants of DC in a large sample of 1291 individuals from the Netherlands Twin Register. We focused specifically on the impact of recording duration and circadian variation of DC, and its long-term temporal stability, association with age, sex, and BMI, and its heritability. The results indicated that short-term DC derived from 5-minute segments can deviate substantially from DC derived from the full 24-hour recording, and that these deviations vary systematically across the day. 24-hour DC showed moderate to strong temporal stability across two measurement occasions roughly three years apart. In addition, 24-hour DC was negatively associated with age and BMI, and was higher in males than females. Lastly, 24-hour DC showed substantial heritability with 62% of individual differences explained by genetic variance.

4.1 | Measurement Characteristics

Previous studies have suggested that DC can be derived from recordings as short as five minutes without losing prognostic power [7, 10]. The first aim of this study was to systematically compare DC estimates derived from complete 24-hour recordings with those obtained from 5-minute segments. The results indicated that there was no consistent over- or underestimation of short-term DC, but that it can still deviate substantially from the corresponding 24-hour DC estimate. The deviation showed a circadian pattern, with short-term DC generally being higher than the corresponding 24-hour estimate during the

night and lower during the daytime. This is consistent with the well-established circadian variation in other HRV metrics [16–18].

This suggests that short-term DC estimates are not interchangeable with 24-hour DC estimates. The higher measurement error and moderately preserved rank order agreement of short-term DC could reduce its prognostic power for predicting adverse health outcomes. Short-term estimates of DC may still be useful for risk stratification, but the present study highlights the importance of standardizing them for timing of their measurement.

The 24-hour DC showed significant test-retest correlation and moderate absolute agreement across two measurement occasions roughly three years apart. The significant test-retest correlation suggests that differences in DC between individuals have good long-term temporal stability. Individuals with relatively high DC tend to still have a relatively high DC three years later. This supports the use of DC for risk stratification across individuals. The absolute agreement was moderate, but this could also reflect actual changes in DC (for instance due to ageing or changes in lifestyle or health status). It is important to note that the temporal stability analyses were based on a relatively small subsample of 56 individuals. Future studies should therefore further examine the utility of DC to monitor physiological changes over time within individuals.

4.2 | Biological Determinants

We further examined the biological determinants of individual differences in 24-hour DC, with a particular focus on its association with stable individual characteristics (i.e., age, sex, BMI) and heritability. The analysis on individual characteristics indicated that DC was negatively associated with age and BMI, and that there was a significant difference in DC between males and females. These associations are consistent with previously reported associations between age and HRV [37–39], age and cardiac vagal control [20, 21], BMI and HRV [19, 40], and biological sex and HRV [41].

The explained variance of 14% suggested that age is a particularly important individual characteristic to consider when using DC for risk stratification or any other between-person comparison. Our results show that DC should be corrected for age and sex rather than using a single reference value of DC below 2.5 ms, as age and sex may introduce confounding on disease outcomes. Additional correction for BMI is more tentative as we cannot rule out that BMI is mediating changes in DC and not a real confounder. Adjusting for BMI may then inadvertently also remove meaningful variance in DC.

In a previous study with the same dataset, we showed that various HRV metrics have heritability between 48% and 57% [32]. Twin studies in other independent samples further provided evidence for heritability of resting levels HRV metrics with estimates around 50%-55% [28, 30, 42] or stress levels as assessed in a laboratory [43]. In the current study, age-, sex-, and BMI-adjusted correlations were substantially higher in MZ twins than DZ twins. In keeping, genetic modeling revealed substantial heritability of 24-hour DC, with additive genetic factors accounting for 62% of its variance. This is, to our knowledge, the first evidence for heritability of DC. Many of the disease outcomes predicted by DC are

also substantially heritable. It is tempting to hypothesize that a part of this heritability is mediated by genetic variants influencing DC. However, solid confirmation of this hypothesis would require longitudinal prediction of disease outcomes in genetically informative designs.

4.3 | Strengths and Limitations

An important strength of this study is its large population-based sample drawn from the Netherlands Twin Register. All participants completed a full day of physiological monitoring in daily life, and provided genetic samples as part of a biobank study. The dataset also included repeated measurements roughly three years later for a subsample of 56 participants. The combination of 24-hour physiological recordings, repeated measurements, and genetic samples for twin pairs created a unique dataset to examine understudied measurement characteristics and biological determinants of DC.

Several limitations should be considered. First, we did not account for the large variety of factors that may influence cardiac measures in daily life. The unrestricted nature of ambulatory physiological recordings means that many factors can influence cardiac measures, such as physical activity, posture, respiration, and food intake. This is well-established for HR and HRV, and therefore likely generalizes to DC. These influences are currently also not accounted for in 24-hour DC estimates. It therefore remains unclear to what extent differences in daily routines (e.g., being physically active or resting, working or having a day off) contribute to variation in DC. This may be particularly relevant for short-term DC estimates, variation in DC may reflect differences in what participants were doing at (or right before) the time of measurement rather than stable differences in cardiac vagal control. Future studies could combine physiological monitoring equipment with passive sensing or fine-grained ecological momentary assessment to investigate the impact of these confounding factors.

A related limitation is that the analysis of circadian variation of DC was primarily descriptive. The observed pattern across time of day should not be interpreted as exclusively reflecting the endogenous circadian rhythm. The abovementioned factors can also vary systematically across the day (e.g., no physical activity or postural changes throughout the night), contributing independently to the observed circadian rhythm in DC. The term “circadian variation” was liberally used here to describe the 24-hour pattern, but controlled laboratory studies (e.g., constant routine) would be needed to fully understand the endogenous circadian rhythm of DC.

5 | Conclusion

DC is increasingly used as a marker for adverse health outcomes and cardiac autonomic dysfunction, which requires it to be a stable individual trait with clear biological roots. We here provide support for this in showing moderate-to-strong temporal stability of 24-hour DC across two measurement occasions roughly three years apart and substantial genetic contribution to the individual differences in DC. Short-term DC derived from 5-minute segments is not the preferred measurement method in comparison with 24-hour estimates, and adjustment for demographic and anthropometric characteristics (e.g., age, sex, or BMI) may be required.

Declarations

Conflict of Interest

Sjors R.B. van de Ven: No conflict of interest.

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Author Contributions

Sjors R.B. van de Ven: Data curation, Formal analysis, Methodology, Visualization, Writing – original draft. **Martin J. Gevonden**: Conceptualization, Supervision, Investigation, Methodology, Writing – review & editing. **Matthijs L. Noordzij**: Conceptualization, Methodology, Writing – review & editing. **Eco J.C. de Geus**: Conceptualization, Funding acquisition, Investigation, Methodology, Writing – review & editing

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Figures

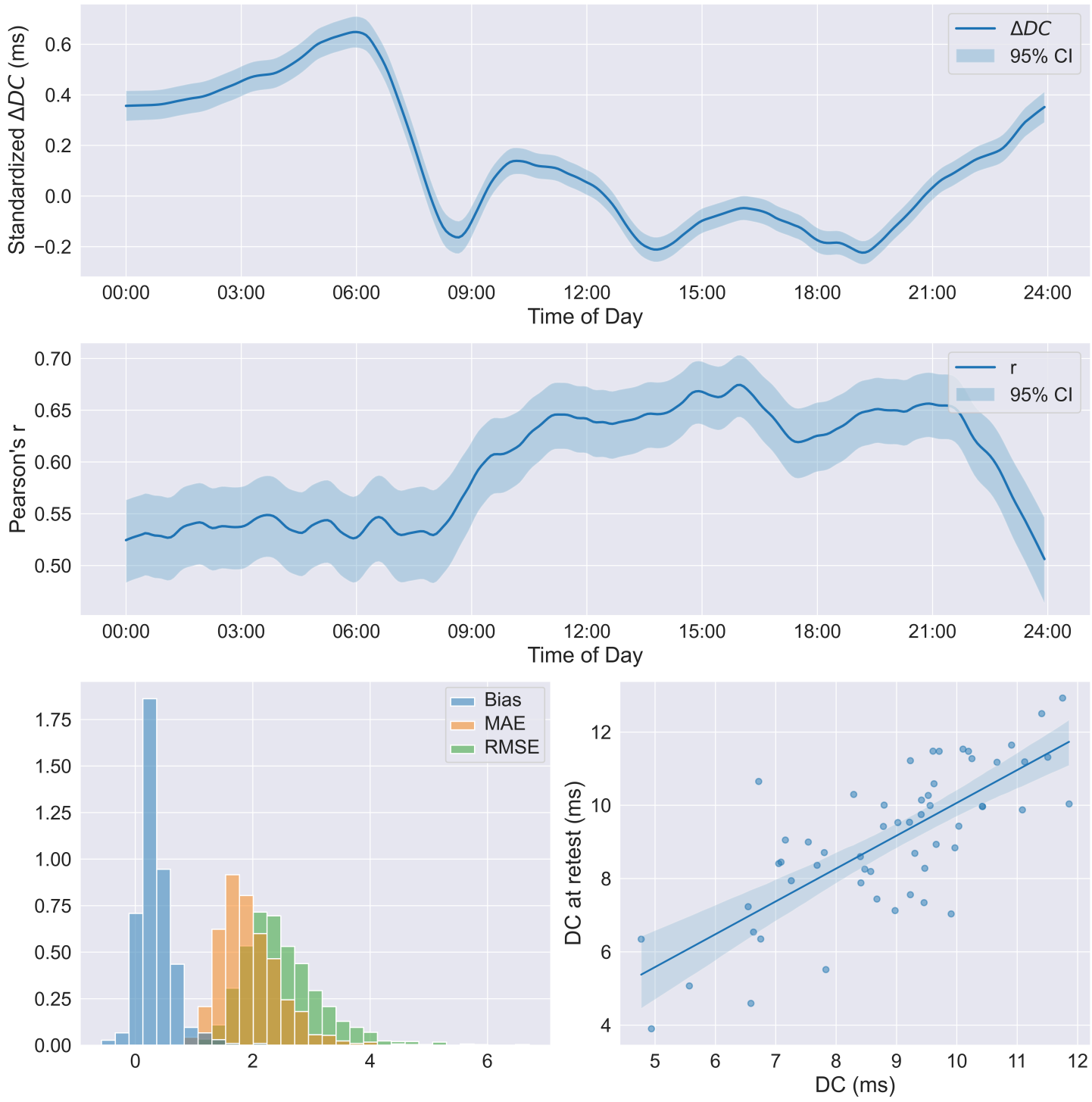


Figure 1

Circadian variation of deceleration capacity (DC), and its association with individual characteristics. The top panel shows the mean circadian variation of standardized ΔDC across all participants. For each participant, ΔDC was computed by subtracting the DC derived from the full 24-hour recording from each 5-minute DC estimate. These differences were then divided by the participant-specific standard deviation across all 5-minute estimates. Values above zero therefore indicate that 5-minute DC was higher than the corresponding 24-hour DC, whereas values below zero indicate that it was lower. The middle panel shows

the Pearson's correlation coefficients between every 5-minute segment and the 24-hour estimate of DC across participants. The bottom panels show the distribution of the agreement of the 5-minute DC estimates in comparison with the corresponding 24-hour estimate (bias, mean absolute error, and root-mean-square error) across participants on the left, and the temporal stability of the 24-hour DC estimates across two measurement occasions on the right.

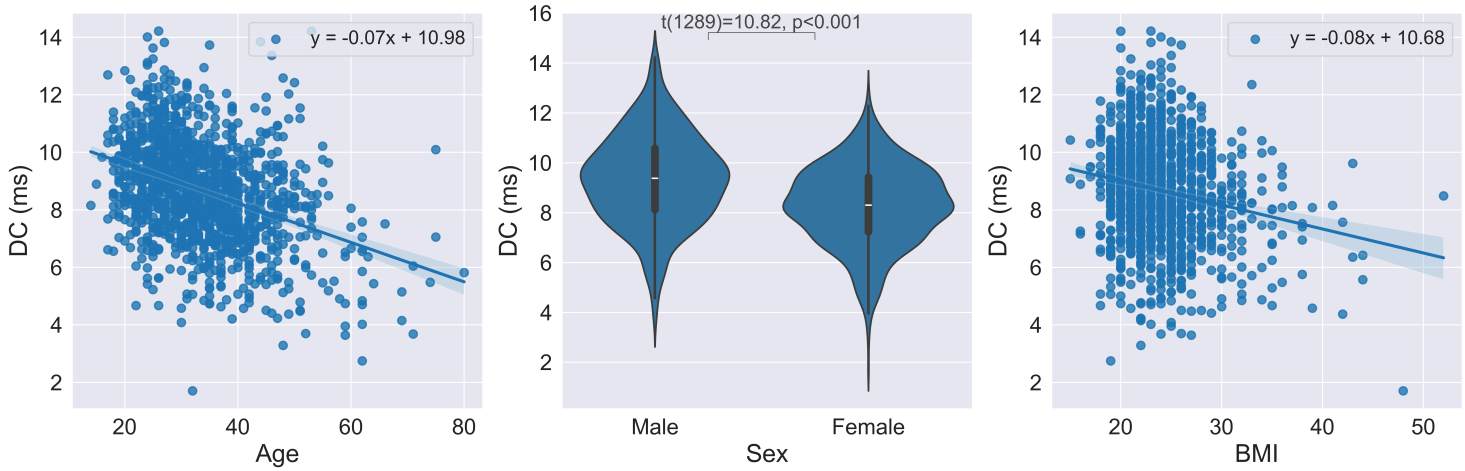


Figure 2

The association between deceleration capacity (DC) and individual characteristics. From left to right represent the individual characteristics age, biological sex, and body mass index (BMI).

Supplementary Files

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