Heart rate variability with photoplethysmography in 8 million individuals: a cross-sectional study

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Summary

Background Heart rate variability, or the variation in the time interval between consecutive heart beats, is a non-invasive dynamic metric of the autonomic nervous system and an independent risk factor for cardiovascular death. Consumer wrist-worn tracking devices using photoplethysmography, such as Fitbit, now provide the unique potential of continuously measuring surrogates of sympathetic and parasympathetic nervous system activity through the analysis of interbeat intervals. We aimed to leverage wrist-worn trackers to derive and describe diverse measures of cardiac autonomic function among Fitbit device users.

Methods In this cross-sectional study, we collected interbeat interval data that are sent to a central database from Fitbit devices during a randomly selected 24 h period. Age, sex, body-mass index, and steps per day in the 90 days preceding the measurement were extracted. Interbeat interval data were cleaned and heart rate variability features were computed. We analysed heart rate variability metrics across the time (measured via the root mean square of successive RR interval differences [RMSSD] and SD of the RR interval [SDRR]), frequency (measured by high-frequency and low-frequency power), and graphical (measured by Poincare plots) domains. We considered 5 min windows for the time and frequency domain metrics and 60 min measurements for graphical domain metrics. Data from participants were analysed to establish the correlation between heart rate variability metrics and age, sex, time of day, and physical activity. We also determined benchmarks for heart rate variability (HRV) metrics among the users.

Findings We included data from 8 203 261 Fitbit users, collected on Sept 1, 2018. HRV metrics decrease with age, and parasympathetic function declines faster than sympathetic function. We observe a strong diurnal variation in the heart rate variability. SDRR, low-frequency power, and Poincare S_2 show a significant variation with sex, whereas such a difference is not seen with RMSSD, high-frequency power, and Poincare S_1. For males, when measured from 0600 h to 0700 h, the mean low-frequency power decreased by a factor of 66·5% and high-frequency power decreased by a factor of 82·0% from the age of 20 years to 60 years. For females, the equivalent factors were 69·3% and 80·9%, respectively. Comparing low-frequency power between males and females at the ages of 40–41 years, measured from 0600 h to 0700 h, we found excess power in males, with a Cohen's *d* effect size of 0·33. For high-frequency power, the equivalent effect size was –0·04. Increased daily physical activity, across age and sex, was highly correlated with improvement in diverse measures of heart rate variability in a dose-dependent manner. We provide benchmark tables for RMSSD, SDRR, high and low frequency powers, and Poincare S_1 and S_2, separately for different ages and sex and computed at two times of the day.

Interpretation Diverse metrics of cardiac autonomic health can be derived from wrist-worn trackers. Empirical distributions of heart rate variability can potentially be used as a framework for individual-level interpretation. Increased physical activity might yield improvement in heart rate variability and requires prospective trials for confirmation.

Funding Fitbit.

Introduction

Heart rate variability (HRV) refers to the variation in time between successive heart beats and represents a non-invasive index of the autonomic nervous system. Because the autonomic nervous system regulates heart rate during sinus rhythm, HRV summarises complex non-linear cardiovascular accommodative responses, which are dictated by the parasympathetic and sympathetic nervous systems, to dynamic physiological variations.

Although HRV is significantly affected by sex and ageing,[1] reduced compensatory response (ie, low HRV) is independently predictive of first fatal and non-fatal cardiovascular disease events in the general population.[2–6] Robust data also link low HRV with adverse outcomes and mortality after sustaining a cardiovascular event, such as a myocardial infarction.[7,8] β blockers and exercise therapy reduce risks of cardiovascular events among individuals with coronary artery disease and congestive heart failure, and enhancement of HRV is believed to be a mechanism for improved prognosis.[9–11] Thus, a less adaptive autonomic nervous system is predictive of first and recurrent cardiovascular events, and restoration

Research in context

Evidence before this study

We searched PubMed, Google, and Google Scholar for research articles published in English up to Jan 1, 2020, using common search terms including "HRV and aging", "HRV and exercise", "PPG and ECG", and "HRV and wearable devices". Articles were also retrieved through searching the citations of known literature. Heart rate variability (HRV) is a non-invasive probe of the autonomic nervous system and can independently measure the working of the sympathetic and parasympathetic branches. It is well accepted that consistently low HRV is predictive of adverse cardiovascular events, whereas transient dips might be indicative of stress or illness. Due to its diagnostic value, standards for HRV have been set by the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Both parasympathetic and sympathetic HRV measures are known to decrease with age. There is also a marked circadian pattern, with HRV measures peaking in the early morning and reaching their lowest values in the late afternoon.

Added value of this study

We present HRV analyses on the largest dataset (to our knowledge) to date, by several orders of magnitude. We computed HRV measures in the time, frequency, and graphical domains, and expanded on the existing literature in several ways. We found that both parasympathetic and sympathetic HRV measures decrease with age most rapidly from the age of 20 years to 40 years, and more slowly after that, with the difference between sexes only significant for sympathetic HRV measures. We provided scaling relationships, which allow for rapid estimates of HRV for a given age, sex, and time of day, and provide benchmark tables for the estimation of the well known HRV metrics. We showed that exercise affects HRV in a dose-dependent manner (ie, an increase in activity increases HRV). It is estimated that male (female) participants in the age range of 20–24 years could increase their high-frequency power by 1 ms^2 by taking an additional 32 (30) steps per day.

Implications of all the available evidence

The widespread availability of heart rate-enabled, wrist-worn devices such as trackers and smart watches have made HRV accessible to millions of people. Our work could potentially make it possible for millions of consumers to compare their HRV to others in their age range and sex, and make meaningful inferences regarding their overall cardiovascular health, daily fitness, and stress level. Changes in HRV accompanying an exercise programme could provide useful feedback to consumers and serve as a motivational tool.

of homoeostatic capacity might reduce the risk of such events.

A position statement from professional societies points out a general disconnect between HRV as a research tool and practical clinical use.[12] Among the barriers for clinical use are assessments in relatively small selected cohorts, requirement for continuous electrocardiogram (ECG) monitoring, and substantial variation by age, sex, and time of day.

In recent years, the widespread availability of heart rate tracking devices has led to considerable interest in use of HRV as a potential clinical tool. Commercial wrist-worn tracking devices measure interbeat intervals (IBIs) through photoplethysmography (PPG) at a single point of contact. PPG devices use multiple wavelengths of light to illuminate the skin and photodiodes to measure the reflected light, thereby inferring changes in blood volume by measuring changes in light absorption.[13–15]

A review of HRV computed by various devices found that although multi-lead ambulatory ECG devices have served as the gold standard, several alternative devices, mainly based on single-lead ECG and PPG, are more convenient and practical for measuring HRV parameters.[16] A comparison of RR intervals (ie, the time between successive heart beats) from a wrist-based PPG device and an ECG device showed that PPG data from wrist devices were accurate enough for both HRV analysis and to differentiate between sinus rhythm and atrial fibrillation cases.[17]

Standards for HRV were set by the European Society of Cardiology and the North American Society of Pacing and Electrophysiology.[18] Benchmark HRV values from 24 h measurements have been published.[19] HRV can be measured in many ways: in the time domain, the frequency domain, or using graphical and non-linear techniques.[20] It is well known that HRV declines with age, although the decline can be ameliorated by healthy habits, such as staying active and meditation practices. Umetani and colleagues' study of the decline in time domain HRV metrics with age, from a dataset of 240 healthy individuals, found that the root mean square of successive RR interval differences (RMSSD) declines more rapidly than the SD of the RR interval (SDRR; called the SD of the NN intervals [SDNN] index).[1] This study also found that for an age of younger than 30 years, HRV in females was lower on average than in males for all time domain metrics, with no sex differences for ages older than 50 years. Findings of other studies that discuss HRV and ageing are consistent with those of Umetani and colleagues.[21–25] A study of the diurnal variation of HRV metrics over a 24 h period involving eight healthy male participants found that HRV metrics vary throughout the day, reaching peak values in the early morning hours.[26] Several authors have discussed the effect of physical activity on HRV, and studies have shown beneficial results.[27–30] According to a study by one group,[31] the correlation between the number of steps measured by Fitbit devices compared with steps

counted by researchers is variable during walking and jogging, but this variation is within 7%.

We aimed to characterise HRV metrics to understand variations by age, sex, time of day, and physical activity level, and to provide benchmarks for HRV metrics among users of Fitbit, a wrist-worn tracking device that uses PPG. Additionally, we aimed to confirm the existing literature findings with a much larger and more diverse population, to establish the correlation between HRV and exercise, and to more carefully examine how the circadian rhythm is affected.

Methods

Data collection and cleaning

In this cross-sectional study, over the course of a randomly selected 24 h, we collected data from individuals using Fitbit devices, which send data to a central database when users sync their devices. The data were anonymised, and the analysis was consistent with Fitbit's terms and conditions and privacy policy statements. Users were not specifically notified of monitoring beyond those statements. We extracted age, sex, body-mass index (BMI; although it was not included in analyses), and steps per day over a 90-day period preceding the measurement. The output of a PPG device is the IBI tachogram—ie, the time between peaks of blood volume. The IBI data were cleaned before HRV features were extracted (for a comparison between HRV derived from ECG and HRV derived from PPG, see the appendix p 1). More details regarding the cleaning process and the data can be found in the appendix (p 2).

Measures of HRV

Time domain metrics include RMSSD and SDRR, frequency domain metrics include high-frequency power and low-frequency power, and graphical domain metrics include Poincare S_1 and S_2. Time domain metrics are computationally easy to calculate and do not require contiguous data. The commonly studied HRV metrics are described in the appendix (p 4). Frequency domain calculations can be computationally expensive and require the data to be contiguous and evenly sampled, but have the benefit of separating the data into short-range fluctuations and long-range fluctuations. One way of interpreting these fluctuations is to associate short-range fluctuations with the working of the parasympathetic nervous system and long-range fluctuations with both the sympathetic and parasympathetic nervous systems.[20] Short-range fluctuations are associated with the high-frequency band, whereas long-range fluctuations are associated with the low-frequency band.

Poincare plots are scatter plots obtained by plotting the IBI at time index i against the succeeding IBI—ie, at time index $i+1$ (more accurately, these are called first order lag-1 Poincare plots). The Poincare plots will contain a high density of points scattered close to the 45 line. This scatter is a measure of variability. The Poincare plot resembles a tapered ellipse since larger IBI allow for more variability. The SD of points along the major axis (S_2) is a measure of long-term variability, whereas the SD along the minor axis (S_1) is a measure of short-term variability.

Time windows of 5 min (short term) and 24 h (long term) are commonly considered in the literature.[18] We considered 5 min windows for the time domain and frequency domain metrics. For graphical domain metrics, we considered 60 min measurements. For the benchmark tables, we first calculated the median of all 5 min window measurements within a 1 h interval for each user. We then computed the mean and median of these values over users.

See Online for appendix

To assess how HRV metrics change with physical activity, we used daily steps from accelerometry as a proxy for physical activity. We analysed the HRV of all participants (measured between 0600 h and 0700 h) grouped by the mean number of steps taken per day (steps per day is averaged over a 90-day period preceding the measurement). To estimate the impact of physical activity, we modelled high-frequency and low-frequency power variation by a linear fit: HRV power=C + steps/σ, where σ is the number of steps necessary to increase the power by 1 ms² on average and C is a constant for a particular HRV metric, age, and sex.

Data coverage

To investigate data coverage, we did an analysis on a randomly selected subset of 40 000 individuals. With the HRV computed every 5 min, the maximum number of HRV samples in a 1 h window was 12, and the maximum number of samples in a 24 h period was 288. In practice, data coverage will be lower due to factors such as motion artifacts and noise. Averaged over individuals, the mean number of samples was 78·2 (SD 39·9) in a 24 h period. In the 1 h period between 0600 h and 0700 h, the mean number of samples was 6·3 (SD 5·2). In the period between 1800 h and 1900 h, the mean was 0·8 (SD 2·0) samples. These two time periods were selected to assess HRV metrics since they are 12 h apart and correspond approximately to the minimum and maximum circadian

	Females			Males		
	Anytime*	0600 h to 0700 h	1800 h to 1900 h	Anytime*	0600 h to 0700 h	1800 h to 1900 h
Age <20 years	168 149	118 130	12 395	68 685	44 783	4523
Age 20–29 years	894 713	597 940	83 544	339 921	217 395	31 958
Age 30–39 years	1 234 770	810 926	114 020	581 550	359 815	53 944
Age 40–49 years	1 162 165	748 297	130 952	592 782	358 646	64 492
Age 50–59 years	1 058 589	650 693	133 752	569 948	333 366	72 356
Age 60–69 years	688 173	416 732	96 092	405 650	230 920	56 096
Age ≥70 years	248 632	150 446	35 858	189 534	105 670	27 266

The amount of data depends on the time of day since we lose data during the daytime probably due to motion artifacts. *Refers to data measured at any time during the day, when participants are sedentary or asleep.

Table 1: **Number of individuals included in the study, by age and sex**

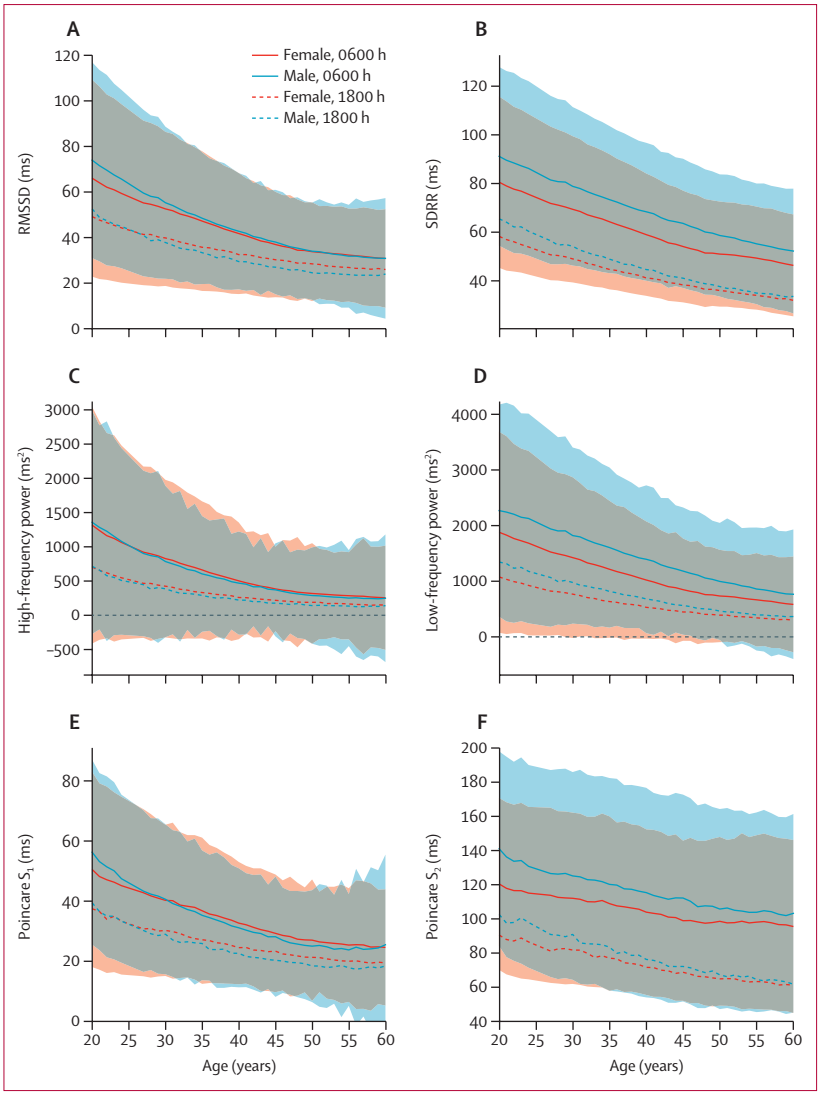

Figure 1: **Effect of age on HRV**
The shaded contours are 1 SD ranges for measurements taken from 0600 h to 0700 h. HRV=heart rate variability. RMSSD=root mean square of successive RR interval differences. RR interval=time between successive heart beats. SDRR=SD of the RR interval.

rhythm.[26] We considered the presence of motion artifacts during the daytime to be the most probable cause for lower coverage during daytime.

Data analysis

We did *t* tests and Cohen's *d* effect size computations, as well as Pearson *r* correlation calculations using the standard Python libraries numpy (version 1.18.5) and scipy (version 1.5.3).

Roles of the funding source

The funder of the study provided financial support towards collection and analysis of the data, but had no role in study design, data interpretation, or writing of the report. AN, AP, and HE-F had access to the raw data. The corresponding author had full access to all the data in the study and had the final responsibility for the decision to submit the paper for publication.

Results

We collected data from 10 424 196 individuals using Fitbit devices over the course of a randomly selected 24 h, on Sept 1, 2018, and data from 8 203 261 individuals were included. Data from 2 220 935 individuals were excluded because they were insufficient to compute HRV metrics. 74 countries were represented with more than 1000 individuals each. The most common countries represented in the dataset were the USA (48%), the UK (11%), and Canada (5%), with all other countries contributing less than 5% each.

We were able to obtain more data during the night and the early morning hours than during daytime hours. Table 1 shows details of our data by age and sex.

Mean age was 46·49 years (SD 14·32) for females and 48·95 years (14·37) for males. Mean BMI was 27·69 kg/m² (SD 6·50) for females and 28·17 kg/m² (5·25) for males. To ensure that the data were representative of the user base, we computed the same demographics on two other reference dates and found that the largest differences in mean ages were 0·26 years for females and 0·51 years for males (data not shown). For BMI, the largest differences in the mean were 0·22 kg/m² for females and 0·16 kg/m² for males. When measured between 0600 h and 0700 h, users who had no data (yet wore their devices) took a mean 10 732 steps (SD 5794) per day, while those who had data took a mean 9210 steps (SD 4931) per day.

Data collected were from several Fitbit device models, but primarily from Charge 2, Alta HR, Blaze, Versa, and Ionic. The HRV metrics computed from data from these various devices were in agreement. As an example, when comparing the RMSSD measured between 0600 h and 0700 h for women in the age range of 40–41 years, the mean was 41·10 ms (SD 20·75) for Charge 2, 38·11 ms (20·23) for Alta HR, 38·96 ms (20·19) for Blaze, 39·54 ms (21·02) for Versa, and 42·68 ms (21·68) for Ionic. The largest difference corresponded to a Cohen's *d* effect size of 0·22. As such, data from all devices were pooled.

HRV declined from the ages of 20 years to 60 years, with a decrease in all HRV metrics (figure 1). Comparing the SDRR in men and women, the effect size averaged over ages was 0·32 when measured at 0600 h and 0·16 when measured at 1800 h. At all ages, the SDRR was higher in men than in women (p<0·0001). The effect size was smaller when the RMSSD was compared between men and women. For individuals aged 20–21 years, the RMSSD was higher in men than in women, with an effect size of 0·18 when measured at 0600 h, remaining higher in men aged younger than 46 years than in women of the same ages (p<0·0001). When measured at 1800 h, the RMSSD was also higher in men aged 20–21 years, with an effect size of 0·10, but became higher in women than in men for ages older than 30 years (p<0·0001). The effect

size decreased to 0 at the age of 50 years when measured at 0600 h and at the age of 25 years when measured at 1800 h, and thereafter became negative, indicating that in older participants women have a higher RMSSD than men. Results were similar for the high-frequency and low-frequency powers and also for Poincare S_1 and S_2 (although there was no consistent difference between males and females with high-frequency power). When measured from 0600 h to 0700 h, the mean low-frequency power decreased by 66·5% for males and 69·3% for females and high-frequency power decreased by 82·0% for males and 80·9% for females from the age of 20 years to 60 years (appendix p 10). Comparing low-frequency power between males and females at the ages of 40–41 years, measured from 0600 h to 0700 h, we found excess power in males, with a Cohen's *d* effect size of 0·33. For high-frequency power, the equivalent effect size was –0·04, implying that no consistent difference was found in high-frequency power between males and females at this age. From a dataset of randomly selected 10 000 values measured at 0600 h, the Pearson correlation was 0·898 between RMSSD and high-frequency power, 0·916 between RMSSD and Poincare S_1, 0·828 between SDRR and low-frequency power, and 0·617 between SDRR and Poincare S_2.

The RMSSD, the SDRR, high-frequency and low-frequency power, and Poincare S_1 and S_2 varied as a function of the time of day (figure 2). The SDRR, low-frequency power, and Poincare S_2 showed a change in phase with increase in age: users aged 60–61 years had an earlier peak in the daily cycle for the long-range measures than did users aged 20–21 years. All HRV metrics peaked early in the day (between 0500 h and 0800 h) and reached a minimum in the late evening (1900 h and 2000 h). High-frequency power at 0600 h to 0700 h was compared with high-frequency power at 1800 h to 1900 h among females aged 20–21 years, females aged 60–61 years, males aged 20–21 years, and males aged 60–61 years. The effect size was 0·37 among female users aged 20–21 years and 0·15 among female users aged 60–61 years (p<0·0001). For male users, the effect size was 0·41 for users of age 20–21 years and 0·13 for those aged 60–61 years (p<0·0001). Considering the modulation of low-frequency power, we found an effect size of 0·46 for female users of age 20–21 years and 0·35 for those aged 60–61 years (p<0·0001; too small to be meaningful). For male users, the effect size was 0·50 for those aged 20–21 years and 0·37 for those aged 60–61 years (p<0·0001; too small to be meaningful).

Finally, we investigated the correlation between HRV and exercise. A strong correlation was seen between physical activity and high-frequency and low-frequency power (figure 3; similar conclusions hold true for other metrics; data not shown). Among users aged 20–24 years, and comparing users who take on average 5000–6000 steps per day with users who take between 12 000–13 000 steps per day, the high-frequency power

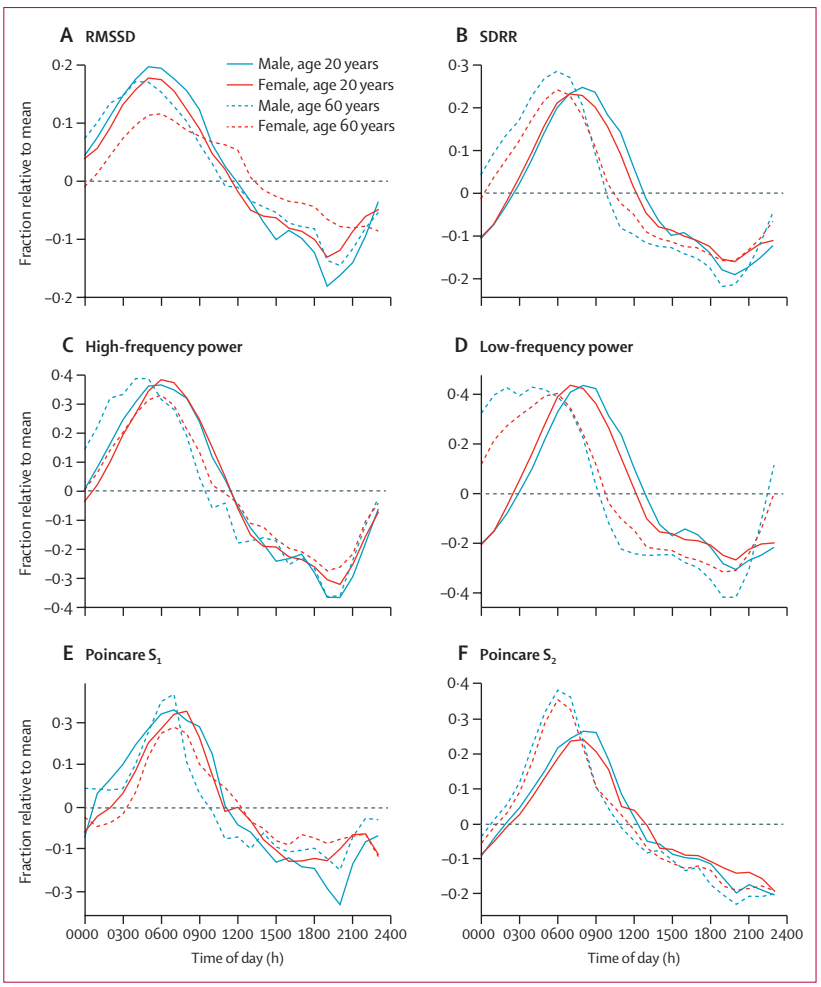

Figure 2: **Daily variation of HRV features**
Mean and SE of the mean are shown. IQRs for the median values are presented in the appendix (pp 9–12). Table 1 summarises the number of participants whose data was used in these aggregates. HRV=heart rate variability. RMSSD=root mean square of successive RR interval differences. RR interval=time between successive heart beats. SDRR=SD of the RR interval.

for the more active cohort was 21·2% greater for males and 22·9% greater for females. For the low-frequency power, the equivalent ratios were 13·6% for males and 25·2% for females. In the same analysis for users aged 50–54 years, the increase in high-frequency power was 9·4% for males and 9·3% for females. For the low-frequency power, the equivalent ratios were 21·0% for males and 25·4% for females. The values of Pearson correlation coefficient (*r*), C, and σ are listed in table 2. Considering high-frequency power, and comparing users who took on average 5000 steps per day with users who averaged 10 000 steps per day, we found an effect size of 0·12 for both female (p<0·0001) and male (p<0·0001) users in the age range of 20–25 years. The effect size for the change in low-frequency power was 0·18 for female individuals and 0·14 for male users. For older individuals in the age range of 50–55 years, we found an effect size of 0·02 for increase in

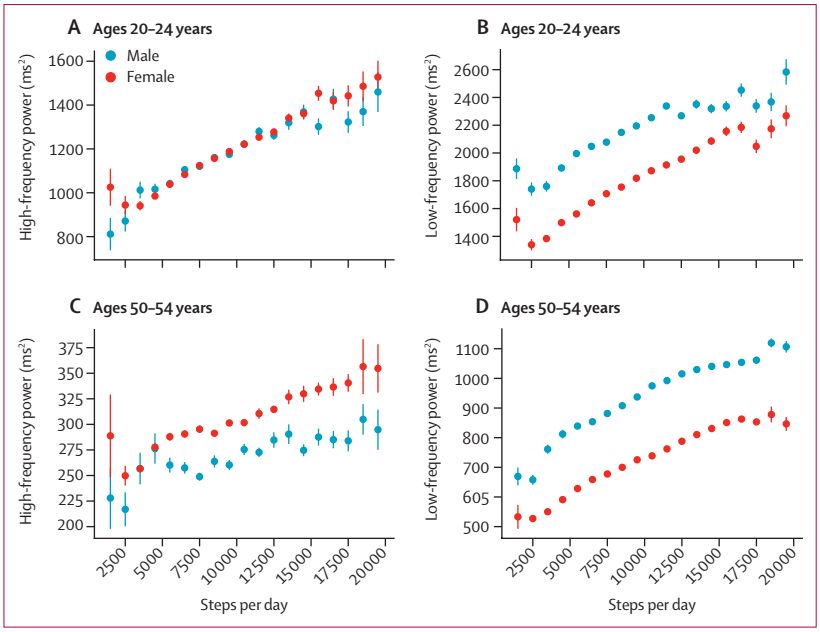

Figure 3: **Effect of physical activity on HRV**
Error bars are SE of the mean. HRV=heart rate variability. RMSSD=root mean square of successive RR interval differences. RR interval=time between successive heart beats. SDRR=SD of the RR interval.

	Metric	*r*	C, ms²	σ, steps
Age 20–24 years				
Male	High-frequency power	0·961	865	32
Female	High-frequency power	0·985	873	30
Male	Low-frequency power	0·946	1758	25
Female	Low-frequency power	0·973	1314	20
Age 50–54 years				
Male	High-frequency power	0·859	234	295
Female	High-frequency power	0·954	254	195
Male	Low-frequency power	0·969	683	42
Female	Low-frequency power	0·979	510	48

HRV=heart rate variability.

Table 2: **Effect of activity on HRV power**

high-frequency power for both female and male users. HRV was correlated with steps in female users ($p<0·0001$ for all cases) and male users ($p=0·0198$ for high-frequency power among users aged 50–54 years, $p<0·0001$ otherwise) in both age groups. The effect size for the increase in low-frequency power was 0·14 for females and 0·13 for males ($p<0·0001$). The benchmark values of RMSSD, SDRR, high and low frequency powers, and Poincare S_1 and S_2 computed for different ages, sex, and time of day are presented in the appendix (pp 9–12).

Discussion

In this study, we extracted HRV measurements from more than 8 million individuals using Fitbit devices, taken over a period of 24 h. With age, RMSSD and high-frequency power decline faster than SDRR and low-frequency power. Low-frequency power varies significantly between sexes. Last, we observe a dose-dependent association between physical activity and HRV metrics, with an increase in physical activity associated with increased HRV. Our results might have important implications for the remote monitoring of human health given the widespread availability of wrist-worn trackers.

First, our method allows for the continuous monitoring of autonomic and cardiovascular responses throughout life's experiences. Consistent with previous small studies, all HRV metrics decrease with age.[1,21–25] The RMSSD, high-frequency power, and Poincare S_1 decrease with age faster than the SDRR, low-frequency power, and Poincare S_2. This finding suggests a more rapid decline of parasympathetic function with increasing age than of sympathetic activity. Previous work implies that the slope of decline in parasympathetic HRV metrics is inversely related to longevity.[25] Our results are consistent with the findings of Umetani and colleagues,[1] especially for measurements taken during the daytime (our SDRR corresponds to their SDNN index). We also presented results showing the diurnal variation of HRV metrics. The variation is substantial, and hence it is advisable for people to interpret HRV measurements at the same time of day. With the help of this very large dataset, we have been able to possibly show a difference between HRV metrics with respect to the phase variation of the daily modulation with age.

Second, given previous associations of HRV metrics with cardiovascular disease event risk, an individual's HRV metrics complement existing strategies for assessing cardiovascular disease risk. Despite previous supportive epidemiological studies, HRV is rarely considered in clinical practice, partly because of the requirement for bulky equipment. As such, the normal distributions of HRV in the general population have largely been poorly defined. Previous work has shown the association of HRV, measured over limited timeframes, with incident cardiovascular disease events and cardiovascular disease in selected individuals. We now present the feasibility of estimating HRV metrics across different ages. Further prospective analyses incorporating longitudinal HRV metrics are necessary to evaluate how HRV measures could be used for risk prediction.

Third, given the correlation between physical steps and HRV, increasing physical activity might optimise HRV metrics. Although other studies have evaluated the association between physical activity and HRV, they were typically limited to small selected populations,[27,28] to participants in a narrow age range,[29] or relied on self-reported physical activity level. Due to the size of our dataset and method of measurement, we are able to examine the correlation between physical activity (quantified by daily steps from accelerometry) and HRV with greater precision, across a broad age range. The correlation

is larger for younger people, especially for high-frequency power (a metric of parasympathetic activity). The linear fit models suggest that people in the age range of 20–24 years might increase their high-frequency power by 1 ms² with every approximately 30 additional steps. By contrast, individuals in the age range of 50–54 years need about 200 (female) to 300 (male) additional steps for each 1 ms² increase in high-frequency power. As a result, older individuals can improve their low-frequency power (both sympathetic and parasympathetic activity) more than high-frequency power, with physical activity. These findings imply that parasympathetic function might be more challenging to restore with physical activity versus sympathetic function. We emphasise that the connection between physical activity and increased HRV is merely evidence of correlation, not causation.

Possible limitations of this work include the absence of cardiovascular outcomes and data on comorbidities. Additionally, we were unable to ascertain prevalent cardiovascular conditions. The short-range HRV metrics such as RMSSD and high-frequency power are sensitive to erratic heart beats, which we have not been able to compensate for. Age, sex, and BMI were self-reported. We also note that the population of Fitbit users is probably not representative in the sense that not all ethnicities and income brackets are equally represented.

Since we only recorded PPG data when there was no movement, it is possible that people who are sedentary were over-represented when computing daytime HRV. There is a potential for bias if a large proportion of data is non-continuous, but this potential bias is mitigated due to our coverage requirement. When measured between 0600 h and 0700 h, users with data took comparatively fewer mean steps per day than users who had no data. Therefore, there is a bias that slightly over-represents less active people, but this does not diminish the value of the results.

HRV metrics were obtained from different Fitbit devices, which might have introduced a small error (we estimated the largest possible effect size to be only 0·22). Another limitation of the present study is that the data are purely transverse—ie, we obtained data from a lot of users, but over a short period of time. We also note that the reported time and frequency domain HRV metrics were computed over 5 min, and should not be compared with metrics computed over 24 h (the SDRR in particular can be larger with larger time windows). Note that the Poincare calculations are over 60 min.

Although HRV metrics have been previously correlated with cardiovascular health and mortality, our technical advance in the analysis of wearable data at large scale and descriptions of the data now permit its potential use for health promotion through tens of millions of currently available wrist-worn commercial trackers. Future longitudinal studies can demonstrate how change in health behaviours such as physical activity, diet, sleep, stress, and weight management can affect HRV. Prospective randomised controlled trials are necessary to demonstrate effective ways to use HRV metrics to improve health.

Contributors

AN was responsible for data analysis, software development, and manuscript preparation. HE-F contributed to the scientific analysis, critical aspects of manuscript writing, and revisions. AP provided valuable expertise regarding device specifics and signal processing. PN contributed scientific expertise, manuscript preparation, and revisions.

Declaration of interests

AN, AP, and HE-F are employees of Fitbit. PN is an unpaid (by Fitbit) scientific collaborator, and reports grants from Amgen, Apple, and Boston Scientific, personal fees from Novartis and Vertex, and consulting income from Apple and Blackstone Life Sciences, all unrelated to the current work.

Data sharing

The data for the HRV benchmarks is provided in the appendix, and can be used freely for research and educational purposes. We are unable to share the raw PPG data, or the raw ECG data because it would violate the terms and conditions under which Fitbit acquired the data. We are also unable to share the software since it was developed as part of the intellectual property of Fitbit. Fitbit's privacy policy does not permit us to share raw heart rate data with scientists unaffiliated with Fitbit.

Acknowledgments

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References

1 Umetani K, Singer DH, McCraty R, Atkinson M. Twenty-four hour time domain heart rate variability and heart rate: relations to age and gender over nine decades. *J Am Coll Cardiol* 1998; **31:** 593–601.

2 Hillebrand S, Gast KB, de Mutsert R, et al. Heart rate variability and first cardiovascular event in populations without known cardiovascular disease: meta-analysis and dose-response meta-regression. *Europace* 2013; **15:** 742–49.

3 Tsuji H, Venditti FJ Jr, Manders ES, et al. Reduced heart rate variability and mortality risk in an elderly cohort. The Framingham Heart Study. *Circulation* 1994; **90:** 878–83.

4 Dekker JM, Crow RS, Folsom AR, et al. Low heart rate variability in a 2-minute rhythm strip predicts risk of coronary heart disease and mortality from several causes: the ARIC Study. Atherosclerosis risk in communities. *Circulation* 2000; **102:** 1239–44.

5 Patel VN, Pierce BR, Bodapati RK, Brown DL, Ives DG, Stein PK. Association of Holter-derived heart rate variability parameters with the development of congestive heart failure in the cardiovascular health study. *JACC Heart Fail* 2017; **5:** 423–31.

6 Stein PK, Barzilay JI, Chaves PH, et al. Novel measures of heart rate variability predict cardiovascular mortality in older adults independent of traditional cardiovascular risk factors: the Cardiovascular Health Study (CHS). *J Cardiovasc Electrophysiol* 2008; **19:** 1169–74.

7 Carney RM, Blumenthal JA, Freedland KE, et al. Low heart rate variability and the effect of depression on post-myocardial infarction mortality. *Arch Intern Med* 2005; **165:** 1486–91.

8 La Rovere MT, Pinna GD, Hohnloser SH, et al. Baroreflex sensitivity and heart rate variability in the identification of patients at risk for life-threatening arrhythmias: implications for clinical trials. *Circulation* 2001; **103:** 2072–77.

9 Niemelä MJ, Airaksinen KE, Huikuri HV. Effect of beta-blockade on heart rate variability in patients with coronary artery disease. *J Am Coll Cardiol* 1994; **23:** 1370–77.

10 Malfatto G, Facchini M, Sala L, Branzi G, Bragato R, Leonetti G. Effects of cardiac rehabilitation and beta-blocker therapy on heart rate variability after first acute myocardial infarction. *Am J Cardiol* 1998; **81:** 834–40.

11 Sandrone G, Mortara A, Torzillo D, La Rovere MT, Malliani A, Lombardi F. Effects of beta blockers (atenolol or metoprolol) on heart rate variability after acute myocardial infarction. *Am J Cardiol* 1994; **74:** 340–45.

12 Sassi R, Cerutti S, Lombardi F, et al. Advances in heart rate variability signal analysis: joint position statement by the e-Cardiology ESC Working Group and the European Heart Rhythm Association co-endorsed by the Asia Pacific Heart Rhythm Society. *Europace* 2015; **17:** 1341–53.

13 Allen J. Photoplethysmography and its application in clinical physiological measurement. *Physiol Meas* 2007; **28:** R1–39.

14 Elgendi M. On the analysis of fingertip photoplethysmogram signals. *Curr Cardiol Rev* 2012; **8:** 14–25.

15 Alian AA, Shelley KH. Photoplethysmography. *Best Pract Res Clin Anaesthesiol* 2014; **28:** 395–406.

16 Singh N, Moneghetti KJ, Christle JW, Hadley D, Plews D, Froelicher V. Heart rate variability: an old metric with new meaning in the era of using mhealth technologies for health and exercise training guidance. Part one: physiology and methods. *Arrhythm Electrophysiol Rev* 2018; **7:** 193–98.

17 Tarniceriu A, et al. Detection of beat-to-beat intervals from wrist photo-plethysmography in patients with sinus rhythm and atrial fibrillation after surgery. 2018 IEEE EMBS International Conference on Biomedical & Health Informatics (BHI); Las Vegas, NV; March 4–7, 2018: 133–36.

18 Electrophysiology TFES. Heart rate variability: standards of measurement, physiological interpretation and clinical use. *Circulation* 1996; **93:** 1043–65.

19 Sammito S, Böckelmann I. Reference values for time- and frequency-domain heart rate variability measures. *Heart Rhythm* 2016; **13:** 1309–16.

20 Shaffer F, Ginsberg JP. An overview of heart rate variability metrics and norms. *Front Public Health* 2017; **5:** 258.

21 Nunan D, Sandercock GR, Brodie DA. A quantitative systematic review of normal values for short-term heart rate variability in healthy adults. *Pacing Clin Electrophysiol* 2010; **33:** 1407–17.

22 Voss A, Schroeder R, Heitmann A, Peters A, Perz S. Short-term heart rate variability—influence of gender and age in healthy subjects. *PLoS One* 2015; **10:** e0118308.

23 Irurzun IM, Defeo MM, Garavaglia L, Mailland T, Mola EE. Scaling behaviour in the heart rate variability characteristics with age. *arXiv* 2018; 1810.04269 (preprint).

24 Reardon M, Malik M. Changes in heart rate variability with age. *Pacing Clin Electrophysiol* 1996; **19:** 1863–66.

25 Zulfiqar U, Jurivich DA, Gao W, Singer DH. Relation of high heart rate variability to healthy longevity. *Am J Cardiol* 2010; **105:** 1181–85.

26 Vandewalle G, Middleton B, Rajaratnam SM, et al. Robust circadian rhythm in heart rate and its variability: influence of exogenous melatonin and photoperiod. *J Sleep Res* 2007; **16:** 148–55.

27 Farah BQ, Andrade-Lima A, Germano-Soares AH, et al. Physical activity and heart rate variability in adolescents with abdominal obesity. *Pediatr Cardiol* 2018; **39:** 466–72.

28 May R, McBerty V, Zaky A, Gianotti M. Vigorous physical activity predicts higher heart rate variability among younger adults. *J Physiol Anthropol* 2017; **36:** 24.

29 Rennie KL, Hemingway H, Kumari M, Brunner E, Malik M, Marmot M. Effects of moderate and vigorous physical activity on heart rate variability in a British study of civil servants. *Am J Epidemiol* 2003; **158:** 135–43.

30 Aubert A, Ramaekers D, Cuche Y, Lysens R, Ector H, Van de Werf F. Effect of long term physical training on heart rate variability. *Comput Cardiol* 1996; **22:** 17–20.

31 Nelson MB, Kaminsky LA, Dickin DC, Montoye AH. Validity of consumer-based physical activity monitors for specific activity types. *Med Sci Sports Exerc* 2016; **48:** 1619–28.