ORIGINAL RESEARCH

Heart Rate Recovery as an Assessment of Cardiorespiratory Fitness in Young Adults

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ABSTRACT

Background: Cardiorespiratory fitness, typically measured as peak oxygen uptake ($\dot{V}o_{2peak}$) during maximal graded exercise testing (GXT_{max}), is a predictor of morbidity, mortality, and cardiovascular disease. However, measuring $\dot{V}o_{2peak}$ is costly and inconvenient and thus not widely used in clinical settings. Alternatively, postexercise heart rate recovery (HRRec), which is an index of vagal reactivation, is a valuable assessment of $\dot{V}o_{2peak}$ in older adults and athletes. However, the validity of HRRec as a clinical indicator of cardiorespiratory fitness in young, sedentary adults, who are a rapidly growing population at risk for developing obesity and cardiovascular disease, has not been fully elucidated.

Methods: We investigated the association between cardiorespiratory fitness, measured by $\dot{V}o_{2peak}$ (mL·kg^{-1}·min^{-1}), and HRRec measures after a GXT_{max} in 61 young (25.2 ± 6.1 years), sedentary adults (40 females) using 3 methods. We examined the relationship between $\dot{V}o_{2peak}$ and absolute (b·min^{-1}) and relative (%) HRRec measures at 1, 2, and 3 min post GXT_{max}, as well as a measure of the slow component HRRec (HRRec 1 min minus HRR 2 min), using Pearson's correlation analysis.

Results: $\dot{V}o_{2peak}$ (36.5 ± 7.9 mL·kg^{-1}·min^{-1}) was not significantly correlated with absolute HRRec at 1 min (r = 0.18), 2 mins (r = 0.04), or 3 min (r = 0.01). We also found no significant correlations between $\dot{V}o_{2peak}$ and relative HRRec at 1 min (r = 0.09), 2 min (r = −0.06), or 3 min (r = −0.10). Lastly, we found no correlation between the measure of the slow component HRRec and $\dot{V}o_{2peak}$ (r = −0.14).

Conclusion: Our results indicate that HRRec measures are not a valid indicator of cardiorespiratory fitness in young, sedentary adults. *J Clin Exerc Physiol*. 2022;11(2):44–53.

Keywords: sedentary lifestyle, physical activity, cardiovascular health, graded exercise test

INTRODUCTION

Accurate assessments of cardiovascular health and fitness are important for predicting at-risk individuals and for developing interventional therapeutic strategies (1). Accumulating evidence has established that clinical assessments of cardiorespiratory fitness improve risk stratification for adverse health outcomes and are a powerful tool for patient management (2–4). The American Heart Association released a statement indicating that cardiorespiratory fitness should be considered a clinical vital sign and should be assessed regularly in the clinic along with other preventative assessments (4). However, direct measures of cardiorespiratory fitness rely on

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determining peak oxygen uptake ($\dot{V}o_{2Peak}$) during a graded exercise test conducted in a laboratory setting. Because measuring $\dot{V}o_{2Peak}$ during a graded exercise test can be costly and inconvenient, it is often more practical to estimate cardiorespiratory fitness using simple, noninvasive measures that are easily collected during exercise. One such measure is heart rate recovery (HRRec) after exercise testing, which is an index of vagal reactivation and is a strong predictor of morbidity and mortality in older adults (5–9).

HRRec is used in some clinical settings as a measure of autonomic dysfunction to identify high-risk cardiovascular disease patients. However, it is not typically used as a marker of cardiorespiratory fitness. For example, HRRec is predictive of long-term outcomes and survival in patients with coronary artery disease and congestive heart failure who have known autonomic nervous system dysfunction (7,8). The HRRec is also an independent risk factor for development of metabolic diseases, suggesting it is an informative marker for at-risk individuals (10,11). Evidence suggests that HRRec is a valid method to assess cardiorespiratory fitness. Cross-sectional studies report that physically active individuals have improved HRRec compared with their sedentary counterparts (12–15). Likewise, both $\dot{V}o_{2peak}$ and HRRec improve after an exercise regimen in longitudinal studies (16–18). In addition, HRRec and $\dot{V}o_{2peak}$ are highly associated in studies that include older adults, athletes, and physically active individuals (19–23). Together authors of these studies suggest that HRRec is a valid marker of cardiorespiratory fitness. However, authors of one study examined the association between cardiorespiratory fitness and HRRec in young, healthy, sedentary females and found no association between $\dot{V}o_{2peak}$ and submaximal HRRec (24). Therefore, despite evidence in other populations, the use of HRRec as an accurate assessment of cardiorespiratory fitness in young and sedentary but otherwise healthy (nonsmoking, nonhypertensive, nondiabetic) adults is unclear.

The purpose of this study was to investigate the validity of using HRRec as an estimate of cardiorespiratory fitness in young, sedentary adults by determining if a significant association exists between HRRec and $\dot{V}o_{2peak}$ during a maximal graded exercise test (GXT_{max}). It is well known that sedentary behavior is associated with cardiovascular disease risk factors (25). In the US, the amount of time young adults spent in sedentary behaviors increased by 12% from 2007 to 2016 (26). Thus, young adults are an emerging at-risk population, and valid clinical measures of cardiorespiratory fitness in this group are needed.

METHODS

The study was reviewed and approved by the University of Kentucky Office of Research Integrity Medical Institutional Review Board (16-0789-F6A), and participants provided written informed consent before inclusion in the study. Participants between the ages of 18 and 45 years were recruited for a previously reported intervention study (27). Each participant completed a Physical Activity Readiness-Questionnaire and Health History Form and were excluded if he or she had existing contraindications to the GXT_{max} as specified in the American College of Sports Medicine Guidelines for Exercise Testing and Prescription (28).

Anthropometric and body composition measures, including standing height, body mass, circumference measurements, and a total-body dual-energy x-ray absorptiometry (DXA) scan were performed. Participants were measured in lightweight clothing containing no metal and without shoes. Standing height was determined to the nearest 0.1 cm from a wall-fixed meter stick (Pittsburgh, Pennsylvania) with the participants' hands positioned on the hips during a maximal inhalation. Body mass was determined to the nearest 0.1 kg using a calibrated electric scale (Escali Corp., Burnsville, Minnesota). Circumference measurements (waist, abdominal, and hip) were taken in triplicate using a fiberglass anthropometric tape (Creative Health Products BMS-8) in accordance with the guidelines established by the Airlie Conference Proceedings (29). The mean of the 3 measures was used for analysis. Body composition was measured using total body DXA scans performed using a GE Lunar iDXA bone densitometer (Lunar Inc., Madison, Wisconsin). All female participants had negative urine pregnancy tests, which were taken immediately before DXA scanning. A single trained investigator completed and analyzed all scans using the Lunar software, Version 14.10. Total body DXA absolute fat-free mass (kg), mineral-free lean mass (kg), and absolute (kg) and relative (% of body mass) fat masses were determined for each participant.

GXT_{max} tests were completed using an indirect calorimetry testing system (Vmax Encore, Vyaire Medical, Yorba Linda, California) with an integrated electrocardiogram (ECG; 60 Hz sampling rate; Cardiosoft v6.51, GE Healthcare, Chicago, Illinois) and a treadmill ergometer. Before the exercise test, baseline heart rate (HR) and blood pressure (BP) were measured while the participant stood on the treadmill. During the test and recovery period, continuous cardiovascular measurements (HR and ECG) were monitored. During the continuous, progressive (speed and grade) tests, oxygen consumption ($\dot{V}o_2$) was measured breath by breath and averaged over 1-min intervals. The GXT_{max} tests were performed using an incremental treadmill protocol, with 2-min workload stages, developed for a previous study (30). Authors of prior studies have shown that similar $\dot{V}o_{2peak}$ and maximal HR are achieved regardless of treadmill protocol (ramp versus incremental) (31,32). The initial stage of the test began with a walking speed of 3.2 mph (5.1 kph) and 0% grade. The test progressed with a 0.4 mph (0.6 kph) increase in speed and 2% increase in grade with each subsequent stage. During the final minute of each stage, BP (by manual auscultation) and ratings of perceived exertion (RPE; using the original 6–20 Borg Scale (33)) were recorded. HR was recorded in the last 10 s of each stage. The test was terminated for all participants at volitional fatigue (3–4.5 s for treadmill to fully stop). Verbal encouragement was given throughout the test. After completing the GXT_{max}, 5 min of passive recovery data (HR and BP) were taken while the participants remained standing on the treadmill.

Achievement of $\dot{V}o_{2peak}$ (mL·kg^{-1}·min^{-1}) was defined as a participant's ability to obtain a minimum of 2 of the following criteria: respiratory exchange ratio ≥1.1 (determined by 1-min averaging), RPE ≥ 17, and/or age-predicted maximal HR achieved or exceeded (34). The highest $\dot{V}o_2$ value observed during GXT was used for analysis.

Our primary analysis included HRRec data at 1, 2, and 3 min postexercise. Absolute HRRec (b·min^{-1}) was defined as the HR at 1, 2, and 3 min postexercise subtracted from the maximal HR achieved during the graded exercise test. Relative HRRec was defined as absolute HRRec divided by maximal HR, multiplied by 100. Also, the difference between 1- and 2-min HRRec was calculated as an index of the slow component of the postexercise HRRec (35).

STATISTICAL ANALYSIS

Data were analyzed using SPSS (Version 26, IBM, Armonk, NY). Descriptive data are presented as means ± SD. Independent sample *t* tests were conducted to assess differences in measured variables between males and females. Pearson's correlation coefficients were used to assess associations between $\dot{V}o_{2peak}$ and relative and absolute HRRec as well as measures of body composition. Correlation analyses were stratified to assess potential sex differences. Since obesity could influence the analysis, a secondary sensitivity analysis was performed to determine if adiposity influenced the findings. Pearson's partial correlation coefficients were used to assess associations between $\dot{V}o_{2peak}$ and relative and absolute HRRec in participants while controlling for body fat percentage. A one-way repeated measures analysis of variance (ANOVA) was performed to determine if relative and absolute HRRec differed between 1, 2, and 3 min postexercise. Significance was ascribed at $P < 0.05$.

RESULTS

All participants reported good health, including no known cardiovascular disease or hypertension, were medication free (except contraceptives), and did not participate in a structured exercise regimen at the time of the study or participate in greater than 2 h moderate-vigorous physical activity each week. Eleven of the 72 participants were excluded because they did not achieve $\dot{V}o_{2peak}$ (details below). The remaining 61 participants included in the analytic dataset (40 females) had varying adiposities (body mass index (BMI): 16.6–37.0 kg·min^{-2}; body fat percentage: 12.4–51.7%). Males had significantly greater height, body mass, and waist circumference than females (Table 1).

During the GXT, all participants in the analytic dataset, except 3 females, were above an absolute HRRec of 18 b·min^{-1}, a cutoff value for abnormal 1-min HRRec observed in a previous study (Supplemental Table 1; https://dx.doi.org/10.6084/m9.figshare.14691099) (36). $\dot{V}o_{2peak}$ was not associated with absolute HRRec at 1, 2, or 3 min when males and females were examined separately or when the entire cohort of participants was combined (Table 2; Figure 1). Absolute HRRec was also not significantly associated with $\dot{V}o_{2peak}$ at 1, 2, or 3 min when controlling for body fat percentage. Increased absolute HRRec at 3 min was associated with an increased waist circumference for the total study group only. However, absolute HRRec at 1, 2, and 3 min were not significantly associated with age, BMI, or other anthropometric measures for males, females, or the total study group (Table 2). Males had a significantly greater $\dot{V}o_{2peak}$ than females (Table 3). The difference between absolute HRRec at 1 and 2 min was also not associated with $\dot{V}o_{2peak}$ in males, females, or the total study group.

Age ($r = -0.28$; $P = 0.03$), BMI ($r = -0.47$; $P < 0.01$), and waist ($r = -0.27$; $P = 0.04$), abdominal ($r = -0.43$;

TABLE 1. Participant characteristics and anthropometric measures.

Variable	Male (N = 21), Mean ± SD (range)	Female (N = 40), Mean ± SD (range)	Total Group (N = 61), Mean ± SD (range)
Age (y)	24.0 ± 4.4 (18.0–31.0)	25.9 ± 6.7 (18.0–45.0)	25.2 ± 6.1 (18.0–45.0)
Height (cm)	176.0 ± 6.4* (161.7–188.5)	164.0 ± 6.3 (152.0–183.2)	168.1 ± 8.5 (152.0–188.5)
Body mass (kg)	78.7 ± 16.7* (61.8–127.7)	67.8 ± 14.7 (42.9–107.6)	71.6 ± 16.2 (42.9–127.7)
BMI (kg·m^{-2})	25.3 ± 4.5 (19.6–35.9)	25.2 ± 5.4 (16.6–37.0)	25.3 ± 5.1 (16.6–37.0)
Anthropometric			
Abdominal circumference (cm)	89.3 ± 11.3 (75.3–110.3)	85.7 ± 13.2 (63.2–123.6)	87.0 ± 12.6 (63.2–123.6)
Waist circumference (cm)	83.8 ± 9.9* (71.4–104.4)	76.3 ± 11.3 (58.3–107.2)	78.9 ± 11.3 (58.3–107.2)
Hip circumference (cm)	102.0 ± 9.7 (89.2–126.8)	102.6 ± 11.4 (84.5–134.0)	102.4 ± 10.8 (84.5–134.0)
Body composition			
Body fat (%)	27.1 ± 7.1* (12.4–39.8)	35.5 ± 8.6 (21.4–51.7)	32.6 ± 9.0 (12.4–51.7)
Fat mass (kg)	21.5 ± 8.7 (7.4–36.1)	24.4 ± 10.8 (9.1–55.1)	23.4 ± 10.1 (7.4–55.1)
Fat-free mass (kg)	55.5 ± 10.1* (43.5–90.2)	41.8 ± 6.1 (31.3–53.7)	46.5 ± 10.1 (31.3–90.2)
Mineral-free lean mass (kg)	52.7 ± 9.5* (41.4–85.3)	39.3 ± 5.8 (29.4–50.5)	44.0 ± 9.6 (29.4–85.3)

*Sex differences $P < 0.05$

TABLE 2. Pearson correlation coefficients among absolute HRRec, $\dot{V}o_{2peak}$, and anthropometric and body composition measures.

Variable	1-min Absolute HRRec			2-min Absolute HRRec			3-min Absolute HRRec		
	Male (N = 21)	Female (N = 40)	Total (N = 61)	Male (N = 21)	Female (N = 40)	Total (N = 61)	Male (N = 21)	Female (N = 40)	Total (N = 61)
Age	0.27	0.08	0.09	0.21	0.11	0.11	0.34	0.21	0.18
BMI	−0.19	0.15	0.05	−0.03	0.27	0.16	0.09	0.27	0.21
Cardiorespiratory fitness									
$\dot{V}o_{2peak}$	0.15	−0.03	0.18	−0.08	−0.07	0.04	−0.19	−0.04	0.01
Anthropometric									
Abdominal circum	−0.15	0.15	0.09	0.08	0.19	0.17	0.24	0.21	0.25
Waist circum	−0.20	0.18	0.14	0.03	0.24	0.20	0.19	0.23	0.26*
Hip circum	0.01	0.07	0.05	0.21	0.18	0.18	0.26	0.19	0.22
Body composition									
Body fat	−0.08	0.05	−0.10	0.26	0.14	0.09	0.33	0.21	0.16
Fat mass	−0.05	0.14	0.05	0.21	0.22	0.18	0.31	0.24	0.25
Fat-free mass	0.03	0.24	0.26*	−0.03	0.28	0.19	0.06	0.13	0.19
Mineral-free lean mass	0.02	0.19	0.24	−0.03	0.25	0.18	0.06	0.10	0.18

Circum = circumference; HRRec = heart rate recovery; $\dot{V}o_{2peak}$ = peak oxygen uptake
*Significant correlation $P < 0.05$.

$P < 0.01$), and hip circumferences ($r = -0.46$; $P < 0.01$) were each negatively associated with $\dot{V}o_{2peak}$ for the total study group. Males, compared with females, had higher HR at peak exercise as well as higher systolic and diastolic BP at baseline, peak exercise, and during recovery (Table 3).

Body composition varied in the cohort (body fat percentage range: 12.4–51.7%). Increased absolute HRRec at 1 min was associated with increased fat-free mass in the total study group only. However, fat mass, body fat percentage, and mineral-free lean mass were not associated with absolute HRRec in males, females, or the total study group (Table 2). Increased body fat percentage and fat mass were associated with a reduced $\dot{V}o_{2peak}$ in males and females separately as well as the total study group. Also, increased mineral-free lean mass and fat-free mass were associated with an increased $\dot{V}o_{2peak}$ for the total study group only (Figure 2). Males had greater fat-free mass and mineral-free lean mass than females (Table 1). Additionally, females had greater body fat percentage than males (Table 1).

Since peak HR varied within the cohort (range: 157–210 $b \cdot min^{-1}$), we also examined the relationship between $\dot{V}o_{2peak}$ and relative HRRec, which accounts for variability in peak HR. Relative HRRec at 1, 2, or 3 min were not significantly associated with measures of $\dot{V}o_{2peak}$ when males and females were examined separately or when the entire cohort of participants was combined (Figure 1, Table 4). Relative HRRec was also not significantly associated with $\dot{V}o_{2peak}$ at 1, 2, or 3 min when controlling for body fat percentage. Greater relative HRRec at 3 min was associated with increasing age and increasing fat mass for the total study group only. However, relative HRRec measures were not associated with BMI or any other anthropometric and body composition measure for males, females, or the total study group (Table 4).

DISCUSSION

HRRec is recognized as a prognostic measure and predictor of mortality in older adults (5,6,9). HRRec and $\dot{V}o_{2peak}$ are used to inform clinical practices in older adults because they are associated with health and longevity (2,3,9). Furthermore, authors of numerous studies have shown that HRRec and $\dot{V}o_{2peak}$ are greater in physically active than sedentary participants and after completing various exercise regimens, suggesting an important physiological relationship between HRRec and cardiorespiratory fitness (14,16,17,37). However, the utility of HRRec as an indicator of cardiorespiratory fitness may vary by population and has not been well studied in young, sedentary adults.

Consistent with this study, Tonello et al. (24) also examined the association between cardiorespiratory fitness and HRRec in young (mean age: 34.5 years) adults not participating in structured exercise and found no association between $\dot{V}o_{2peak}$ and 1-, 2-, 3-, and 5-min HRRec. While we and Tonello et al. (24) both studied young sedentary adults, the 2 studies used somewhat different methods. Our study included both sexes and determined $\dot{V}o_{2peak}$ and HRRec from treadmill exercise testing, while Tonello et al. (24) studied only females and used a cycle ergometer. Maximal oxygen uptake is lower when measured by cycle ergometer than a treadmill (38,39). Tonello et al. (24) used a submaximal test to measure HRRec (that induced a HR at 86% age-predicted max), while our participants performed a GXT_{max} to

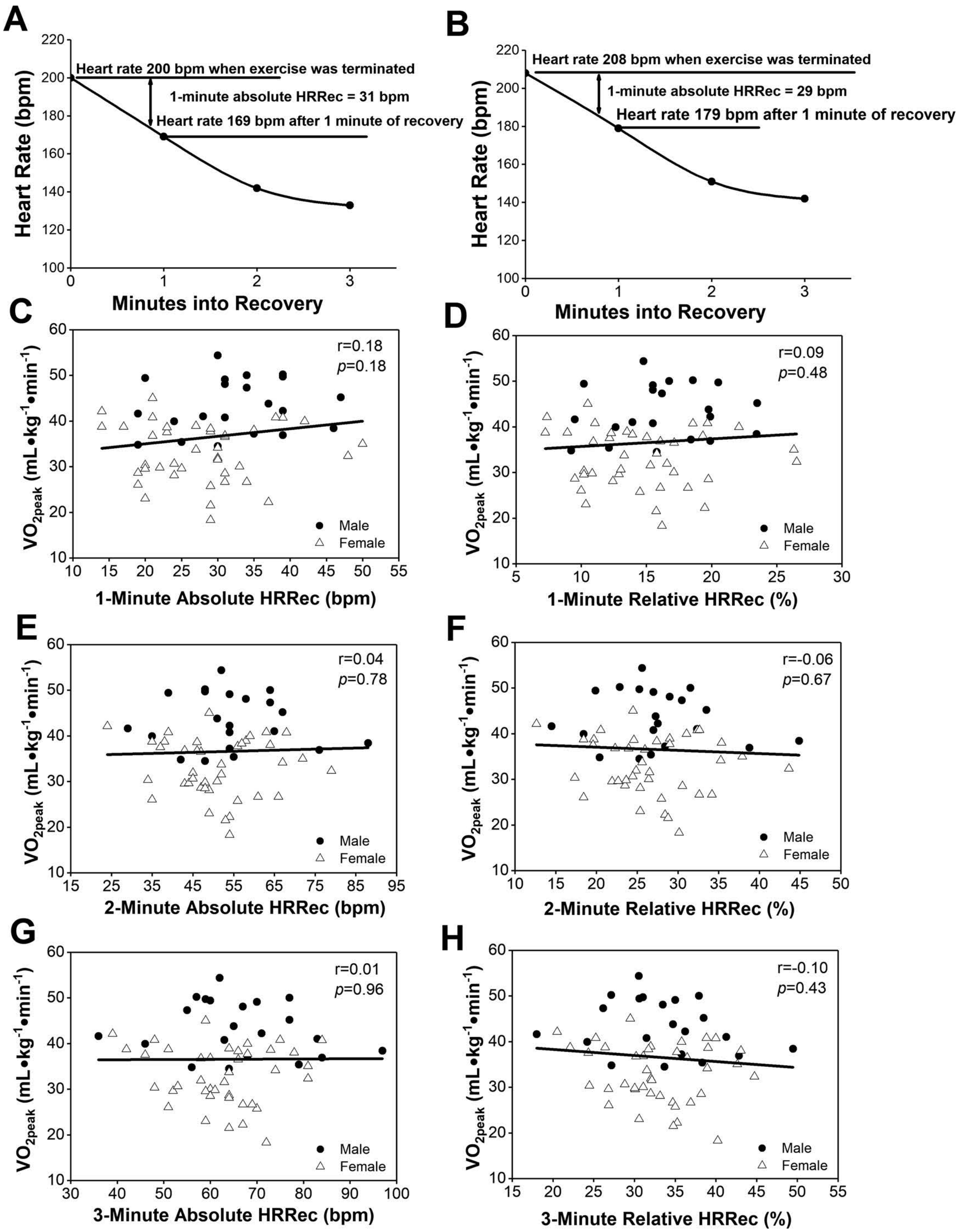

FIGURE 1. Absolute and relative heart rate recovery (HRRec) are not associated with $\dot{V}o_{2peak}$. Representative exercise HRRec data from (A) male and (B) female participants. Pearson correlations were used to compare $\dot{V}o_{2peak}$ from the GXT_{max} with measures of (C), (E), and (G) absolute and (D), (F), and (H) relative HRRec at 1, 2, and 3 min after exercise termination.

volitional fatigue for determination of HRRec. Thus, despite distinct methodological differences, the fact that our 2 studies had similar results is strong evidence that HRRec may not be a valid indicator of cardiorespiratory fitness in young, sedentary adults.

In this study, we examined HRRec at 1, 2, and 3 min, as these are the measures that have been associated with cardiorespiratory fitness in other populations (19,23,40). HRRec after exercise is orchestrated by both the parasympathetic and sympathetic branches of the autonomic nervous system (41). Parasympathetic reactivation is predominately responsible for the decrease in HR immediately after exercise, while sympathetic withdrawal occurs more gradually (41,42). For this reason, authors of previous studies have considered HRRec at 1 and 2 min an indicator of vagal reactivation (5,6). In our study of young sedentary adults, HRRec

TABLE 3. $\dot{V}o_{2peak}$, HR, and BP responses to maximal graded exercise test.

Variable	Male (N = 21), Mean ± SD (range)[a]	Female (N = 40), Mean ± SD (range)[a]	Total Group (N = 61), Mean ± SD (range)[a]
Cardiorespiratory fitness			
$\dot{V}o_{2peak}$ (mL·kg^{-1}·min^{-1})	43.3 ± 6.0* (34.5–54.4)	33.0 ± 6.4 (18.3–45.0)	36.5 ± 7.9 (18.3-54.4)
HR (b·min^{-1})			
Baseline HR	83.3 ± 13.8 (63.0–122.0)	88.8 ± 10.9 (62.0–109.0)	86.9 ± 12.1 (62.0-122.0)
Peak HR	198.6 ± 6.6* (187.0–210.0)	191.3 ± 9.1 (157.0–210.0)	193.8 ± 9.0 (157.0-210.0)
Absolute HRRec (b·min^{-1})			
1 min	32.2 ± 8.0 (19.0–47.0)A	27.9 ± 8.3 (14.0–50.0)A	29.4 ± 8.4 (14.0–50.0)A
2 min	54.5 ± 13.4 (29.0–88.0)A	50.8 ± 11.4 (24.0–79.0)A	52.1 ± 12.1 (24.0–88.0)A
3 min	66.5 ± 13.7 (36.0–97.0)A	62.7 ± 10.5 (39.0–84.0)A	64.0 ± 11.8 (36.0–97.0)A
Relative HRRec (%)			
1 min	16.3 ± 4.1 (9.2–23.5)B	14.7 ± 4.6 (7.3–26.5)B	15.2 ± 4.5 (7.3–26.5)B
2 min	27.5 ± 6.8 (14.5–44.9)B	26.6 ± 6.1 (12.6–43.7)B	26.9 ± 6.3 (12.6–44.9)B
3 min	33.5 ± 7.0 (18.0–49.5)B	32.8 ± 5.8 (20.5–44.8)B	33.1 ± 6.2 (18.0–49.5)B
Baseline BP (mmHg)			
Systolic	118.1 ± 6.0* (106.0–126.0)	112.5 ± 7.4 (90.0–130.0)	114.4 ± 7.4 (90.0–130.0)
Diastolic	76.6 ± 4.2* (68.0–82.0)	74.0 ± 4.5 (62.0–82.0)	74.9 ± 4.6 (62.0–82.0)
Peak BP (mmHg)			
Systolic	189.6 ± 13.7* (160.0–220.0)	164.7 ± 16.1 (140.0–224.0)	173.2 ± 19.4 (140.0–224.0)
Diastolic	88.2 ± 2.0* (86.0–94.0)	85.5 ± 1.3 (82.0–90.0)	86.4 ± 2.0 (82.0–94.0)
Recovery BP (mmHg)			
1-min systolic	163.7 ± 18.6* (142.0–216.0)	145.4 ± 15.1 (118.0–198.0)	151.7 ± 18.4 (118.0–216.0)
1-min diastolic	85.5 ± 2.0* (84.0–90.0)	83.4 ± 1.9 (78.0–86.0)	84.1 ± 2.2 (78.0–90.0)
3-min systolic	140.9 ± 15.6* (108.0–188.0)	129.4 ± 10.8 (102.0–164.0)	133.3 ± 13.7 (102.0–188.0)
3-min diastolic	83.0 ± 2.2* (78.0–88.0)	79.5 ± 3.9 (64.0–84.0)	80.7 ± 3.8 (64.0–88.0)

BP = blood pressure; HR = heart rate; HRRec = heart rate recovery
[a]Like capital letters indicate significant differences based on repeated measures analysis of variance
*Sex differences $P < 0.05$

at 1, 2, and 3 min was not significantly associated with cardiorespiratory fitness in our total analytic dataset or when stratified by sex. Thus, vagal reactivation may not be a reliable indicator of cardiorespiratory fitness in this population.

Our study cohort spanned a large range of adiposity, and 47% of participants were overweight or obese (BMI ≥ 25 kg·min^{-2}). Previous studies found that increased obesity is associated with reduced 1-min HRRec (43,44). In contrast, our data revealed that an increased waist circumference was associated with a greater absolute 3-min HRRec, and increased fat mass was associated with a greater relative 3-min HRRec. However, this unexpected finding may be due to the difference in physiological significance of the 3-min HRRec measure compared with the 1 min (i.e., sympathetic withdrawal versus parasympathetic reactivation). Also, our previous data showed that HRRec after a GXT_{max} was similar in healthy-weight and obese children, indicating that young individuals with poor body composition can have normal vagal reactivation after exercise (45).

Physical activity status may be another factor influencing the relationship between $\dot{V}o_{2peak}$ and HRRec. Authors of studies in young adults, which included both physically active and sedentary participants, reported a significant association between $\dot{V}o_{2peak}$ and HRRec (22,23,40). Authors of studies have also found that subjectively and objectively measured physical activity were associated with HRRec (23,24). Although our subjects did not participate in structured exercise, incidental activity may have influenced HRRec, as shown by Tonello et al. (24). In fact, fat-free mass, which is affected by sedentary behavior (46), was associated with 1-min HRRec in our cohort. Age may also be an important factor since a significant association between $\dot{V}o_{2peak}$ and HRRec after a maximal treadmill test was observed in older adults with congestive heart failure (19). Thus, sedentary behavior and young age appear to be important contributing factors when determining if HRRec is a valid indicator of cardiorespiratory fitness.

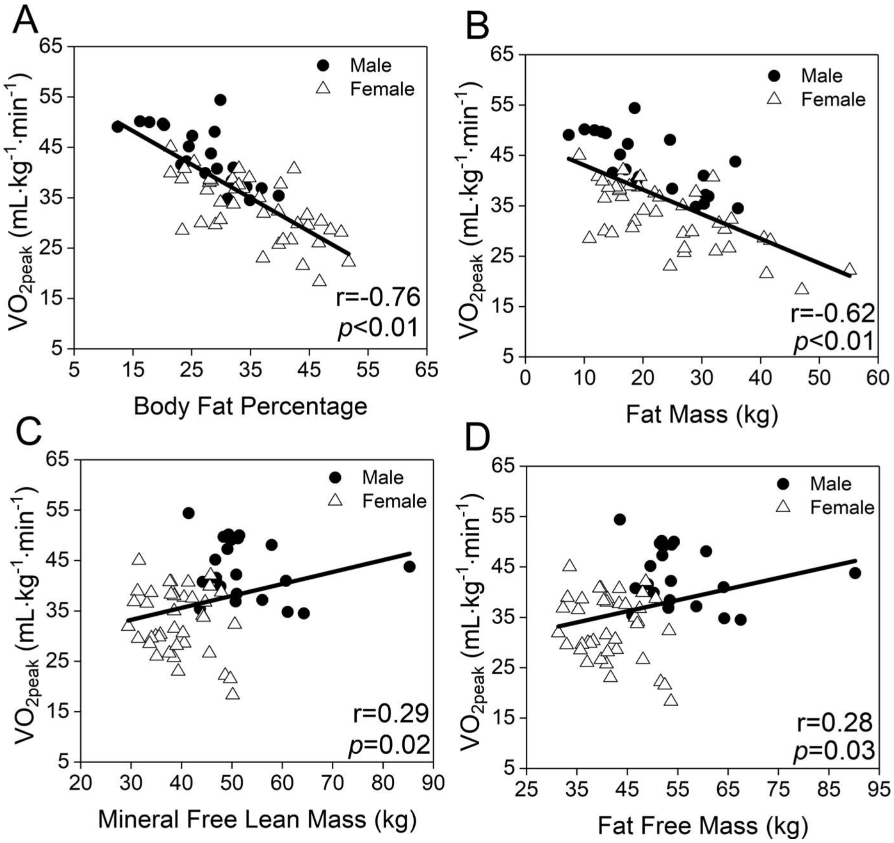

FIGURE 2. Absolute and relative measures of body composition are associated with $\dot{V}o_{2peak}$. Pearson correlation was used to compare $\dot{V}o_{2peak}$ from the GXT_{max} with (A) body fat percentage, (B) fat mass, (C) mineral-free lean mass, and (D) fat-free mass.

TABLE 4. Pearson correlation coefficients among relative HRRec, $\dot{V}o_{2peak}$, and anthropometric and body composition measures.

	1-min Relative HRRec			**2-min Relative HRRec**			**3-min Relative HRRec**		
	Male (N = 21)	**Female (N = 40)**	**Total (N = 61)**	**Male (N = 21)**	**Female (N = 40)**	**Total (N = 61)**	**Male (N = 21)**	**Female (N = 40)**	**Total (N = 61)**
Age	0.31	0.12	0.13	0.25	0.16	0.17	0.39	0.23	0.25*
BMI	−0.12	0.12	0.06	0.03	0.24	0.17	0.16	0.24	0.21
Cardiorespiratory fitness									
$\dot{V}o_{2peak}$	0.11	−0.07	0.09	−0.12	−0.13	−0.06	−0.23	−0.15	−0.10
Anthropometric									
Abdominal circum	−0.09	0.13	0.09	0.13	0.18	0.17	0.29	0.21	0.24
Waist circum	−0.13	0.16	0.12	0.09	0.23	0.19	0.25	0.22	0.23
Hip circum	0.08	0.05	0.05	0.26	0.16	0.19	0.32	0.19	0.23
Body composition									
Body fat	−0.04	0.01	−0.08	0.29	0.10	0.11	0.35	0.16	0.17
Fat mass	0.01	0.12	0.06	0.26	0.19	0.20	0.36	0.23	0.25*
Fat-free mass	0.09	0.24	0.24	0.04	0.30	0.17	0.13	0.19	0.16
Mineral-free lean mass	0.09	0.19	0.22	0.03	0.26	0.15	0.13	0.15	0.14

BMI = body mass index; Circum = circumference; HRRec = heart rate recovery; $\dot{V}o_{2peak}$ = peak oxygen uptake

We also found that $\dot{V}O_{2peak}$ was associated with body fat and fat-free body composition measures. Although $\dot{V}O_{2peak}$ is expressed relative to body mass, the composition of body mass varies. In agreement with our findings, previous researchers have reported that greater body fat percentage was associated with reduced $\dot{V}O_{2peak}$ and greater fat-free mass was associated with increased $\dot{V}O_{2peak}$ (47,48).

Some limitations of our study existed. First, our HRRec measures were collected during passive recovery while participants remained standing. Authors of other studies collected either active recovery measures or passive recovery measures while participants were seated or lying down (6,23,36). However, immediately moving participants to a seated or supine position after a maximal exercise test can be difficult in practice. Since our goal was to inform clinical utility, we collected measures while participants remained standing. Second, our sample size was small with varying adiposities. However, since we designed this study to inform on clinical utility of HRRec in the general population, our inclusion criteria included young, relatively healthy, and sedentary individuals, and no exclusion criteria regarding obesity status were implemented. Third, we did not control for dietary supplements that may have been consumed during the study. Fourth, we did not control for the phase of the menstrual cycle when the GXT_{max} was performed for female participants. It is possible that phase of the menstrual cycle may influence maximal oxygen uptake (49).

Since the clinical utility of HRRec was first introduced in the late 1990s, many authors have investigated HRRec as a measure of cardiovascular health and fitness in a variety of populations (5,20,23,24). HRRec has been shown to be a useful diagnostic and prognostic tool for coronary artery disease, heart failure, and mortality in older adults, including healthy participants and heart failure patients (5–7). However, few studies have been performed in young, sedentary adults, which is a rapidly expanding and at-risk population. Valid measures of cardiorespiratory fitness are needed to identify at-risk individuals at a young age and develop interventional therapeutic strategies.

CONCLUSION

The prevalence of cardiovascular disease among US adults is a staggering 49% of the population (50). We found that HRRec measures were not significantly associated with $\dot{V}O_{2peak}$ in a sample of young, sedentary adults. While HRRec measures have been used as a clinical indicator of health and morbidity in other populations, they are not a reliable indicator of cardiorespiratory fitness in sedentary young adults.

REFERENCES

1. Benjamin EJ, Muntner P, Alonso A, Bittencourt MS, Callaway CW, Carson AP, Chamberlain AM, Chang AR, Cheng S, Das SR, Delling FN, Djousse L, Elkind MSV, Ferguson JF, Fornage M, Jordan LC, Khan SS, Kissela BM, Knutson KL, Kwan TW, Lackland DT, Lewis TT, Lichtman JH, Longenecker CT, Loop MS, Lutsey PL, Martin SS, Matsushita K, Moran AE, Mussolino ME, O'Flaherty M, Pandey A, Perak AM, Rosamond WD, Roth GA, Sampson UKA, Satou GM, Schroeder EB, Shah SH, Spartano NL, Stokes A, Tirschwell DL, Tsao CW, Turakhia MP, VanWagner LB, Wilkins JT, Wong SS, Virani SS, American Heart Association Council on E, Prevention Statistics C, Stroke Statistics S. Heart disease and stroke statistics—2019 update: a report from the American Heart Association. Circulation. 2019;139(10):e56–528.
2. Holtermann A, Marott JL, Gyntelberg F, Sogaard K, Mortensen OS, Prescott E, Schnohr P. Self-reported cardiorespiratory fitness: prediction and classification of risk of cardiovascular disease mortality and longevity—a prospective investigation in the Copenhagen City Heart Study. J Am Heart Assoc. 2015;4(1):e001495.
3. Clausen JSR, Marott JL, Holtermann A, Gyntelberg F, Jensen MT. Midlife cardiorespiratory fitness and the long-term risk of mortality: 46 years of follow-up. J Am Coll Cardiol. 2018;72(9):987–95.
4. Ross R, Blair SN, Arena R, Church TS, Despres JP, Franklin BA, Haskell WL, Kaminsky LA, Levine BD, Lavie CJ, Myers J, Niebauer J, Sallis R, Sawada SS, Sui X, Wisloff U, American Heart Association Physical Activity Committee of the Council on L, Cardiometabolic H, Council on Clinical C, Council on E, Prevention, Council on C, Stroke N, Council on Functional G, Translational B, Stroke C. Importance of assessing cardiorespiratory fitness in clinical practice: a case for fitness as a clinical vital sign: a scientific statement from the American Heart Association. Circulation. 2016;134(24):e653–99.
5. Cole CR, Blackstone EH, Pashkow FJ, Snader CE, Lauer MS. Heart-rate recovery immediately after exercise as a predictor of mortality. N Engl J Med. 1999;341(18):1351–7.
6. Cole CR, Foody JM, Blackstone EH, Lauer MS. Heart rate recovery after submaximal exercise testing as a predictor of mortality in a cardiovascularly healthy cohort. Ann Intern Med. 2000;132(7):552–5.
7. Tang YD, Dewland TA, Wencker D, Katz SD. Post-exercise heart rate recovery independently predicts mortality risk in patients with chronic heart failure. J Card Fail. 2009; 15(10):850–5.
8. Lipinski MJ, Vetrovec GW, Froelicher VF. Importance of the first two minutes of heart rate recovery after exercise treadmill testing in predicting mortality and the presence of coronary artery disease in men. Am J Cardiol. 2004;93(4):445–9.
9. Qiu S, Cai X, Sun Z, Li L, Zuegel M, Steinacker JM, Schumann U. Heart rate recovery and risk of cardiovascular events and all-cause mortality: a meta-analysis of prospective cohort studies. J Am Heart Assoc. 2017;6(5):e005505.
10. Yu TY, Hong WJ, Jin SM, Hur KY, Jee JH, Bae JC, Kim JH, Lee MK. Delayed heart rate recovery after exercise predicts development of metabolic syndrome: a retrospective cohort study. J Diabetes Investig. 2021;13(1):167–76.
11. Jae SY, Bunsawat K, Kunutsor SK, Yoon ES, Kim HJ, Kang M, Choi YH, Franklin BA. Relation of exercise heart rate recovery to predict cardiometabolic syndrome in men. Am J Cardiol. 2019;123(4):582–7.
12. Darr KC, Bassett DR, Morgan BJ, Thomas DP. Effects of age and training status on heart rate recovery after peak exercise. Am J Physiol. 1988;254(2 Pt 2):H340–3.

13. Dixon EM, Kamath MV, McCartney N, Fallen EL. Neural regulation of heart rate variability in endurance athletes and sedentary controls. Cardiovasc Res. 1992;26(7):713–9.
14. Du N, Bai S, Oguri K, Kato Y, Matsumoto I, Kawase H, Matsuoka T. Heart rate recovery after exercise and neural regulation of heart rate variability in 30–40 year old female marathon runners. J Sports Sci Med. 2005;4(1):9–17.
15. Barak OF, Ovcin ZB, Jakovljevic DG, Lozanov-Crvenkovic Z, Brodie DA, Grujic NG. Heart rate recovery after submaximal exercise in four different recovery protocols in male athletes and non-athletes. J Sports Sci Med. 2011; 10(2):369–75.
16. Sugawara J, Murakami H, Maeda S, Kuno S, Matsuda M. Change in post-exercise vagal reactivation with exercise training and detraining in young men. Eur J Appl Physiol. 2001;85(3–4):259–63.
17. Giallauria F, Del Forno D, Pilerci F, De Lorenzo A, Manakos A, Lucci R, Vigorito C. Improvement of heart rate recovery after exercise training in older people. J Am Geriatr Soc. 2005;53(11):2037–8.
18. Hochsmann C, Dorling JL, Apolzan JW, Johannsen NM, Hsia DS, Church TS, Martin CK. Effect of different doses of supervised aerobic exercise on heart rate recovery in inactive adults who are overweight or obese: results from E-MECHANIC. Eur J Appl Physiol. 2019;119(9):2095–103.
19. Hirsh DS, Vittorio TJ, Barbarash SL, Hudaihed A, Tseng CH, Arwady A, Goldsmith RL, Jorde UP. Association of heart rate recovery and maximum oxygen consumption in patients with chronic congestive heart failure. J Heart Lung Transplant. 2006;25(8):942–5.
20. Watson AM, Brickson SL, Prawda ER, Sanfilippo JL. Short-term heart rate recovery is related to aerobic fitness in elite intermittent sport athletes. J Strength Cond Res. 2017; 31(4):1055–61.
21. Mongin D, Chabert C, Courvoisier DS, Garcia-Romero J, Alvero-Cruz JR. Heart rate recovery to assess fitness: comparison of different calculation methods in a large cross-sectional study. Res Sports Med. 2021:1–14. doi:10.1080/15438627.2021.1954513
22. Facioli TP, Philbois SV, Gastaldi AC, Almeida DS, Maida KD, Rodrigues JAL, Sanchez-Delgado JC, Souza HCD. Study of heart rate recovery and cardiovascular autonomic modulation in healthy participants after submaximal exercise. Sci Rep. 2021;11(1):3620.
23. Fan LM, Collins A, Geng L, Li JM. Impact of unhealthy lifestyle on cardiorespiratory fitness and heart rate recovery of medical science students. BMC Public Health. 2020;20:1012.
24. Tonello L, Reichert FF, Oliveira-Silva I, Del Rosso S, Leicht AS, Boullosa DA. Correlates of heart rate measures with incidental physical activity and cardiorespiratory fitness in overweight female workers. Front Physiol. 2015;6:405.
25. Park JH, Joh HK, Lee GS, Je SJ, Cho SH, Kim SJ, Oh SW, Kwon HT. Association between sedentary time and cardiovascular risk factors in Korean adults. Korean J Fam Med. 2018;39(1):29–36.
26. Du Y, Liu B, Sun Y, Snetselaar LG, Wallace RB, Bao W. Trends in adherence to the physical activity guidelines for Americans for aerobic activity and time spent on sedentary behavior among US adults, 2007 to 2016. JAMA Netw Open. 2019;2(7):e197597.
27. Thomas JM, Kern PA, Bush HM, McQuerry KJ, Black WS, Clasey JL, Pendergast JS. Circadian rhythm phase shifts caused by timed exercise vary with chronotype. JCI Insight. 2020;5(3):e134270.
28. Pescatello LS, Arena R, Riebe D, Thompson PD. ACSM's guidelines for exercise testing and prescription. Ninth ed. Philadelphia, PA: Wolters Kluwer; 2014.
29. Lohman TG, Roche AF, Martorell R. Anthropometric standardization reference manual. Champaign, IL: Human Kinetics Books; 1988.
30. Tranel HR, Schroder EA, England J, Black WS, Bush H, Hughes ME, Esser KA, Clasey JL. Physical activity, and not fat mass is a primary predictor of circadian parameters in young men. Chronobiol Int. 2015;32(6):832–41.
31. Davies B, Daggett A, Jakeman P, Mulhall J. Maximum oxygen uptake utilising different treadmill protocols. Br J Sports Med. 1984;18(2):74–9.
32. Myers J, Buchanan N, Walsh D, Kraemer M, McAuley P, Hamilton-Wessler M, Froelicher VF. Comparison of the ramp versus standard exercise protocols. J Am Coll Cardiol. 1991;17(6):1334–42.
33. Borg G. Perceived exertion as an indicator of somatic stress. Scand J Rehabil Med. 1970;2(2):92–8.
34. Tanaka H, Monahan KD, Seals DR. Age-predicted maximal heart rate revisited. J Am Coll Cardiol. 2001;37(1):153–6.
35. Boullosa DA, Tuimil JL, Leicht AS, Crespo-Salgado JJ. Parasympathetic modulation and running performance in distance runners. J Strength Cond Res. 2009;23(2):626–31.
36. Watanabe J, Thamilarasan M, Blackstone EH, Thomas JD, Lauer MS. Heart rate recovery immediately after treadmill exercise and left ventricular systolic dysfunction as predictors of mortality: the case of stress echocardiography. Circulation. 2001;104(16):1911–6.
37. Haroonrashid M. Hattiwale SHH, Salim A. Dhundasi, Kusal K. Das. Recovery heart rate response in sedentary and physically active young healthy adults of Bijapur, Karnataka, India. Basic Sci Med. 2012;1(5):30–3.
38. Keren G, Magazanik A, Epstein Y. A comparison of various methods for the determination of $\dot{V}o_{2peak}$. Eur J Appl Physiol Occup Physiol. 1980;45(2–3):117–24.
39. Maeder MT, Ammann P, Rickli H, Brunner-La Rocca HP. Impact of the exercise mode on heart rate recovery after maximal exercise. Eur J Appl Physiol. 2009;105(2):247–55.
40. Vicente-Campos D, Martin Lopez A, Nunez MJ, Lopez Chicharro J. Heart rate recovery normality data recorded in response to a maximal exercise test in physically active men. Eur J Appl Physiol. 2014;114(6):1123–8.
41. Pierpont GL, Voth EJ. Assessing autonomic function by analysis of heart rate recovery from exercise in healthy subjects. Am J Cardiol. 2004;94(1):64–8.
42. Kannankeril PJ, Le FK, Kadish AH, Goldberger JJ. Parasympathetic effects on heart rate recovery after exercise. J Investig Med. 2004;52(6):394–401.
43. Dimkpa U, Oji JO. Association of heart rate recovery after exercise with indices of obesity in healthy, non-obese adults. Eur J Appl Physiol. 2010;108(4):695–9.
44. Barbosa Lins TC, Valente LM, Sobral Filho DC, Barbosa e Silva O. Relation between heart rate recovery after exercise testing and body mass index. Rev Port Cardiol. 2015;34(1): 27–33.
45. Easley EA, Black WS, Bailey AL, Lennie TA, Sims WJ, Clasey JL. Recovery responses to maximal exercise in healthy-weight children and children with obesity. Res Q Exerc Sport. 2018;89(1):38–46.

46. Kyle UG, Morabia A, Schutz Y, Pichard C. Sedentarism affects body fat mass index and fat-free mass index in adults aged 18 to 98 years. Nutrition. 2004;20(3):255–60.
47. Maciejczyk M, Wiecek M, Szymura J, Szygula Z, Wiecha S, Cempla J. The influence of increased body fat or lean body mass on aerobic performance. PLoS One. 2014;9(4):e95797.
48. Mondal H, Mishra SP. Effect of BMI, body fat percentage and fat free mass on maximal oxygen consumption in healthy young adults. J Clin Diagn Res. 2017;11(6):CC17–20.
49. Godbole G, Joshi AR, Vaidya SM. Effect of female sex hormones on cardiorespiratory parameters. J Family Med Prim Care. 2016;5(4):822–4.
50. Virani SS, Alonso A, Aparicio HJ, Benjamin EJ, Bittencourt MS, Callaway CW, Carson AP, Chamberlain AM, Cheng S, Delling FN, Elkind MSV, Evenson KR, Ferguson JF, Gupta DK, Khan SS, Kissela BM, Knutson KL, Lee CD, Lewis TT, Liu J, Loop MS, Lutsey PL, Ma J, Mackey J, Martin SS, Matchar DB, Mussolino ME, Navaneethan SD, Perak AM, Roth GA, Samad Z, Satou GM, Schroeder EB, Shah SH, Shay CM, Stokes A, VanWagner LB, Wang NY, Tsao CW, American Heart Association Council on E, Prevention Statistics C, Stroke Statistics S. Heart disease and stroke statistics–2021 update: a report from the American Heart Association. Circulation. 2021;143(8):e254–743.