

BRIEF REPORT

Balancing Exercise Benefits Against Heartbeat Consumption in Elite Cyclists



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The cardiovascular system demonstrates remarkable efficacy in responding to the increased metabolic demand during exercise. However, uncertainty persists regarding the potential health implications of more extreme volumes of intense exercise training.¹ Colloquially, the assertion regarding the potential harms of exercise has been trivialized by the statement that we have a fixed number of heartbeats over a lifetime and that exercise might deplete this. This is based on the observation that, across mammalian species, life span is predetermined by the basic energetics of living cells, heart rate serves as a marker of metabolic rate, and that life expectancy is inversely proportional to the heart rate.²

This concept resonates with the well-established fact that an increased resting heart rate is associated with cardiovascular risk.³ However, regular

exercise also reduces the resting heart rate long-term, likely offsetting a transient exertional heart rate increase.³ Thus, we are left with balancing the equation between increased heart rates throughout exercise and the lower resting heart rates observed in habitual exercisers.

As an exploratory analysis, we aimed to investigate the relationship between exercise training and an average 24-hour heart rate. We examined the average heart rate on 24h-Holter monitoring in 109 athletes and 38 healthy controls using the full Australian cohort of the Prospective Athletic Heart (Pro@Heart) study, which has been described in full previously.⁴ Although the Pro@Heart study includes both Belgian and Australian participants, only the Australian cohort was included in this analysis due to the use of a different Holter monitoring device in Belgium and the unavailability of some required data. The study protocol was approved by the Human Research Ethics Committee at each recruiting site. All participants were encouraged to perform their normal activities and exercise training while wearing the Holter monitor which recorded heart rhythm, rate, and an accelerometer-derived estimate of activity time. The athletes were slightly younger than controls (median 19 [17-22] vs 21 [19-22] years; $P = 0.001$), sex ratio was similar (69.6% male vs

What is the clinical question being addressed?

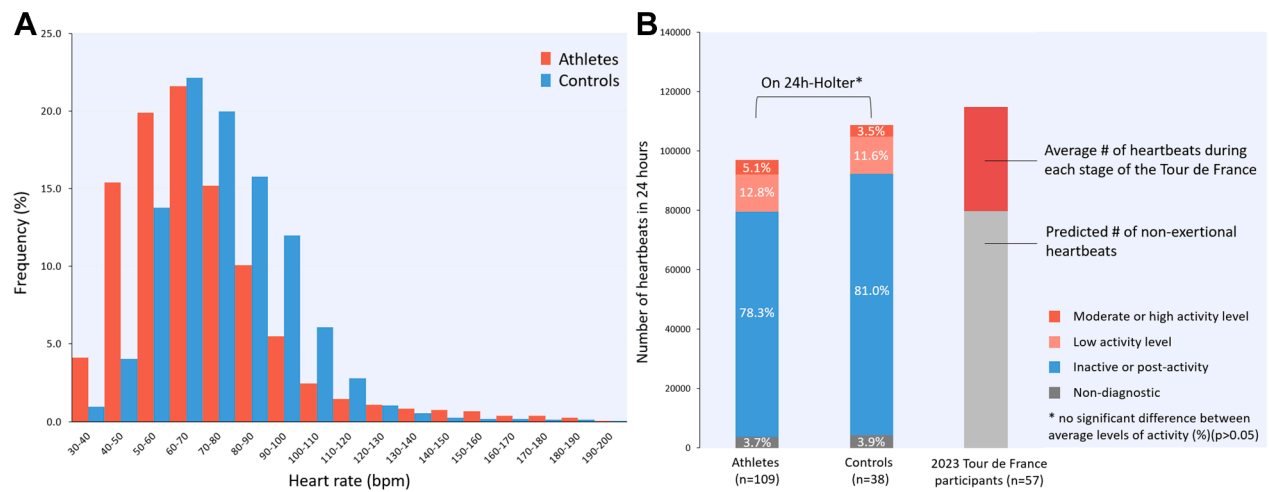
What is the relationship between lower resting heart rates and exertional increases in athletes?

What is the main finding?

The concept of "heartbeat consumption" is introduced as a straightforward metric that may offer insights on training load and the possible adverse effects of intense exercise.

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the [Author Center](#).

FIGURE 1 Relation Between Exercise and Heart Rate Variation

(A) Heart rate variation on 24h-Holter monitoring in athletes (n = 109) and healthy controls (n = 38) and (B) their number of heartbeats over 24 hours compared to participants of the 2023 Tour de France and Tour de France Femmes.

68.4%; $P = 0.888$), and despite the Holter device having been worn during exercise, accuracy of the recordings was excellent in both groups (1.1% [0.0%-3.6%] and 1.6% [0.6%-2.8%] nondiagnostic data, respectively; $P = 0.123$).

The average heart rate was lower in the athletes (68 ± 11 vs 76 ± 8 beats/min; $P < 0.001$). This difference was observed across sexes (within-group differences by sex; $P = 0.305$ in athletes and $P = 0.951$ in controls), although no formal interaction test was performed. The discrepancy in average heart rates was mostly attributable to the lower resting heart rates in athletes, illustrated by a leftward shift in the heart rate histograms, and by the low percentage of heart rates 100 bpm and over, compared to the total distribution (7.6% [2.7%-12.2%] for the athletes and 9.4% [3.9%-13.9%] for the controls; $P = 0.115$) (Figure 1A). This is not surprising as, for instance, 2 hours of exercise represents only 8% of the total time of the day. The potential benefit of a reduced average heart rate is evident, considering that for the athletes in our study, it results in 11,520 or 10.6% fewer heartbeats over 24 hours!

More intense or prolonged exercise may influence the total daily heart rate exposure differently, potentially offsetting the resting bradycardia in athletes. To explore this further, we examined the exercise data of 22 male and 35 female professional cyclists that was publicly shared on Strava during the 2023 Tour de France and Tour de France Femmes.⁵ Average heart rates during each stage, excluding time

trials, were multiplied by the minutes of exercise, to calculate the number of heartbeats which were spent during the race. On average $35,177 \pm 6,024$ heartbeats were spent during each stage. Interestingly, no differences were observed between male and female cyclists ($P = 0.097$) because the women's stages, although generally shorter in duration, were completed at higher heart rates (234 ± 39 min vs 273 ± 33 min; $P < 0.001$ and 148 ± 9 beats/min vs 127 ± 14 beats/min in females and males, respectively; $P < 0.001$). To estimate cumulative daily heartbeats, we combined these exertional data with average 24-hour heart rates from the Pro@Heart study and the time outside of racing to approximate nonexercise heartbeats. These calculations suggest that the heart rate burden during a professional stage race far exceeds the potential reduction in total heartbeats associated with resting bradycardia (Figure 1B).

Therefore, it appears to be true that exercise has the potential to increase the average heart rate, but only when the effort is extreme and prolonged. Our findings should be interpreted with caution, with the heart rate analysis in Tour de France participants, in particular, intended to be hypothesis-generating rather than conclusive. Nevertheless, the interrogation of the relationship between exercise and the heart rate in elite athletes supports the premise that metabolic energetics can be optimized by regular exercise training, but may also be compromised by high bouts of intense exercise. A higher vagal tone

may represent a protective mechanism linking the lower resting heart rate in athletes to more efficient metabolic function. This “heartbeat consumption” model may provide a novel insight into the U-shaped curve of exercise benefits that has been proposed to explain the protection and exacerbation of some arrhythmic and atherosclerotic associations with the athlete’s heart.¹ Although preliminary, due to the limited sample size and absence of adjustment for confounding variables such as age, fitness level, or heart rate recovery metrics, our findings merit further investigations into the relationship between heartbeat consumption and cardiovascular health and longevity. In particular, sex-specific differences in exercise-induced heart rate changes deserve more attention, especially because women generally have longer life expectancies.³

The simple yet effective metric of heartbeat consumption could help define the optimal exercise dose, adapt to an individual’s fitness level, and even serve as a signal of overtraining. Considering the ubiquitous use of smartwatches enabling constant

heart rate acquisition, this would seem to be a health metric worthy of further evaluation.

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