

Effects of Exercise on Heart Rate Variability: Inferences from Meta-Analysis

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ABSTRACT

SANDERCOCK, G. R. H., P. D. BROMLEY, and D. A. BRODIE. Effects of Exercise on Heart Rate Variability: Inferences from Meta-Analysis. *Med. Sci. Sports Exerc.*, Vol. 37, No. 3, pp. 433–439, 2005. **Introduction:** Chronic exercise training produces a resting bradycardia that is thought to be due partly to enhanced vagal modulation. **Purpose:** The aim of the present study was to determine the effects of exercise training on heart rate and measures of heart rate variability associated with vagal cardiac modulation and to quantify the relationship between changes in these measures. **Methods:** A random effects model of effect size (d) for change in high frequency (HF) power and RR interval was calculated. Within-group heterogeneity was assessed using the Q statistic. Where heterogeneous effects were found, subgroup analyses were performed using the between-group Q statistic. **Results:** A meta-analysis of 13 studies measuring HF ($N = 322$ cases) produced an overall effect size of $d = 0.48$ (C.I. 0.26–0.70, $P = 0.00003$). Twelve studies (298 cases) reported a change in RR interval with an overall effect size of $d = 0.75$ (C.I. 0.51–0.96, $P < 0.00001$). Effect sizes for RR interval data were significantly heterogeneous. Subgroup analysis revealed significantly smaller responses of RR interval to training in older subjects ($P < 0.1$). Effect sizes for change in HF were homogenous, although a trend toward an attenuated response to training was exhibited in older subjects ($P > 0.10$). Linear, quadratic, and cubic fits all revealed weak ($P > 0.05$) relationships between effect sizes for change in HF and RR interval. **Discussion:** Exercise training results in significant increases in RR interval and HF power. These changes are influenced by study population age. The smaller effect size for HF and weak relationship between HF and RR interval suggest factors additional to increased vagal modulation are responsible for training bradycardia. **Key Words:** TRAINING, AUTONOMIC CONTROL, FREQUENCY DOMAIN, BRADYCARDIA

Time and frequency domain analyses of heart rate variability (HRV) provide a noninvasive method to evaluate the autonomic regulation of heart rate. Low levels of HRV are related to risk of sudden cardiac death and are associated with numerous other cardiac events such as: heart disease, heart failure, diabetes, hypertension, asymptomatic left ventricular dysfunction, and myocardial infarction. In these disease states, as well as in the general population, increased sympathetic drive is associated with arrhythmia formation and sudden death (36). Conversely, interventions that reduce sympathetic activity and/or increase parasympathetic activity have been shown to protect against lethal arrhythmias (21).

Exercise has been proposed as a possible antiarrhythmic intervention in humans (3). Experimental data in the dog suggest that exercise eliminates the incidence of ventricular fibrillation in previously susceptible animals via enhanced baroreceptor control and vagal modulation (18), represented by increased HRV after endurance exercise.

Cross-sectional differences between athletes and controls support the notion of increased parasympathetic and/or decreased sympathetic drive in endurance-trained individuals. Specifically, the resting bradycardia observed in endurance-trained athletes is commonly accompanied by augmented markers of cardiac vagal modulation (28,30). Longitudinal data are less consistent, and a recent review (2) suggested that further, controlled, studies using larger cohorts should be carried out to clarify the effect of exercise in HRV.

The aim of this article is to present the results of a meta-analysis on the effects of exercise on HRV. The main objective of this meta-analysis was to determine whether aerobic exercise interventions in healthy subjects cause significant increases in HRV concomitant to resting bradycardia.

METHODS

Search strategy. The databases PubMed and Ovid were searched using the terms “heart rate variability” and

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“exercise” “activity” “athlete(s).” Further searches using the terms: “bradycardia” and “autonomic control” in conjunction with the previous search terms were also performed. The bibliographies of articles obtained were searched manually to obtain further studies not identified electronically.

Criteria. Only English language studies involving healthy persons at least 18 yr old were included. An aerobic exercise intervention of at least 4-wk duration was required. The use of a control group was originally an inclusion criterion but was later revoked due to the small number of studies meeting this criterion. However, where a control group was present, the use of randomized group allocation remained a criterion. All studies were required to have measured high-frequency spectral power (HF). In studies meeting the above criteria, changes in RR interval (or heart rate) were then sought.

Review process. The searches led to the identification of 68 potential studies for inclusion in the analyses. The terms used provided many irrelevant studies due to the common use of the terms in combination. Studies were assessed against the inclusion criteria, and 48 were selected for further consideration.

Studies reporting high-frequency spectral power (HF) were included in the analysis only if they had been derived from either autoregressive, Fourier type transformation or reported as the harmonic component from coarse graining spectral analysis (CGSA). Fast Fourier transformation and autoregressive modeling produce very similar estimates of HF power when measured in the supine position (13). Where CGSA has been compared with other spectral measures, lower values for HF values have been reported (37). The reliability of spectral HRV methods appears to be similar and resting measures of HRV can be highly reproducible under certain conditions (31). It was required that all HF data be presented in raw (ms^2) or transformed units. Normalized units, ratios, and percentages of total power were excluded from the analysis. Studies were only included if they provided assurance of correct processing of the RR interval data. Specifically, it was required that the authors had made a clear statement ensuring that manual or automated checking of the data for aberrant beats had been made. Only short-term data recordings made in the supine position were included. It was not deemed necessary that respiratory rate had been controlled during short-term data collection. Where ambulatory measures were used only the full 24-h data were included. For both data collection methods, it was required that data acquisition was carried out at least 24 h after the last bout of exercise training. It was deemed necessary that ambulatory recordings were made on a nonexercise day.

Application of the above criteria led to the exclusion of a further 30 studies. Of the 18 remaining entrants to the analysis of change in HRV due to exercise, it was found that HF data were recorded but not presented in one study. HF data were presented in unsuitable units in a further study. Data were presented in a form that did not allow calculation of the standard difference (d) in three further studies. All studies made explicit statements assuring that automated

checks for aberrant beats had been made. All 24-h data had also been visually inspected. Therefore, 13 studies including a total of 20 trials were entered into the meta-analysis. Two trials from one study were consequently rejected due to very small sample sizes.

Data concerning change in RR interval from all of the above studies was included in a separate meta-analysis except in three cases. Of the 13 studies entered into the first analysis, one study did not report RR data. In two further studies, RR interval data were not reported in a format that facilitated the calculation of effect size.

Statistical methods. To analyze differences in HF and RR, the standardized effect size was calculated. The pooled standard deviation was calculated from the pre- and posttest standard deviations of the experimental group. Previous data (16) suggest HRV data do not demonstrate regression toward the mean after exercise intervention, therefore allowing this choice of standard deviations to be used. This method may overestimate d slightly, because it does not take into account the correlation between the two sets of scores. It was chosen because HRV is a dynamic measure that commonly shows some random variation from test to retest. This serves to reduce estimates of effect size. Therefore, the use of this liberal method of effect size estimation was deemed to be advantageous. This also allowed inclusion of a large number of studies into the analysis where no control group was used.

The overall effect for the pooled standardized differences was calculated using a random effects model for RR interval and HF. All values were expressed as means $\pm$ 95% confidence intervals (95% C.I) and a value of $P < 0.05$ was assumed to show statistical significance.

Tests for heterogeneity. The within-group heterogeneity was assessed by calculation of the Q statistic for all data in the analysis. Briefly, the squared distance of each study from the combined effect is calculated. Each value is weighted, with a greater weight given to more precise studies. The standard error (SE) is based on the inverse variance method and all calculations are based on log values. The $Q/(k - 1)$ statistic was also calculated; in this statistic, Q is the chi-squared value in a test for heterogeneity, and k is the number of studies in the meta-analysis. This has the particular feature by which a value greater than one may be used to provoke further investigation even if the statistic is non-significant. Such a facility is helpful as it means the test is not solely dependent on sample size.

To examine potential sources of heterogeneity, subgroup analysis of several potential moderator variables was undertaken. The possibility of generating spurious findings from subgroup analyses is well documented (17). Therefore, only a small number of subgroup analyses were undertaken. The subgroups themselves were determined *a priori* and were based on causal mechanisms, magnitude of effects, and statistical significance. In further accordance with current recommendations (17), all subgroup analyses were, when appropriate, physiologically justified.

Statistically, heterogeneity was assessed using the between-groups value of the Q statistic. This is analogous to the use of a one-way ANOVA. All analysis was carried out

on values from a fixed effects model, and log values were used in all cases. Due to the low differential power of the Q statistic a value of ($P < 0.1$) was assumed to show significant between-group heterogeneity.

Selection of moderator variables. The potential moderator variables identified in this analysis were classified as either subject characteristics, training intervention characteristics, or HRV methodological characteristics. The first subgroup analysis based on subject characteristics concerned subject sex. Trials including males, females, and mixed groups are all present in the literature. Physiological justification for this subgrouping lies in the known differences in HRV measures between the sexes (22).

The second subgroup analysis concerned subject age. Subjects were divided into three groups defined as young (mean age < 30 yr), middle aged (mean age 30–60 yr), or old (mean age > 65 yr). This analysis was justified physiologically as differences in HRV are evident between these age groups (22). It is also known that other physiological measures such as cardiopulmonary adaptations to training are affected by age (5).

The final subject characteristic used to define subgroups was previous training status. It is common for subjects with a relatively high value for a given physiological characteristic to show less adaptation in response to training than those with lower initial values, and *vice versa*. This phenomenon is known as the “law of initial values.” There is some evidence to suggest an opposite direction of effect for certain characteristics of HRV (15). There are also data to suggest that high levels of HRV may prevent further increases in very fit subjects due to physiological factors such as acetylcholine saturation of the SA node (11,14).

The only analysis conducted on the training intervention subgroup concerned duration of intervention. This subgroup comprised studies of ≤ 12 wk duration and those of > 12 wk. This point of division was derived from the literature, as 12 wk seemed to be an upper limit to the shorter interventions used. Above this point studies varied from 14 wk to 2 yr in duration. This was justified by the occurrence of such a wide range of intervention durations in the literature. Many physiological variables show differences in magnitude of adaptation dependent on duration of stimulus. It was felt, logically, that studies with an 8-wk duration could not be compared fairly with those of 1 yr.

Additional subgroup analyses based on exercise intensity and duration were not carried out due to the relatively homogeneity of these variables between studies. For instance, intensity typically only ranged between 60–80% of a given maximum and duration only from 40 to 60 min. Additionally, these measures were often varied within studies due to exercise progression.

The last subgroup classification was HRV methodology and contained subgroup analysis between short-term and 24-h ECG recordings.

RESULTS

Training-induced changes in RR interval. Figure 1 shows the results of the meta-analysis for changes in RR

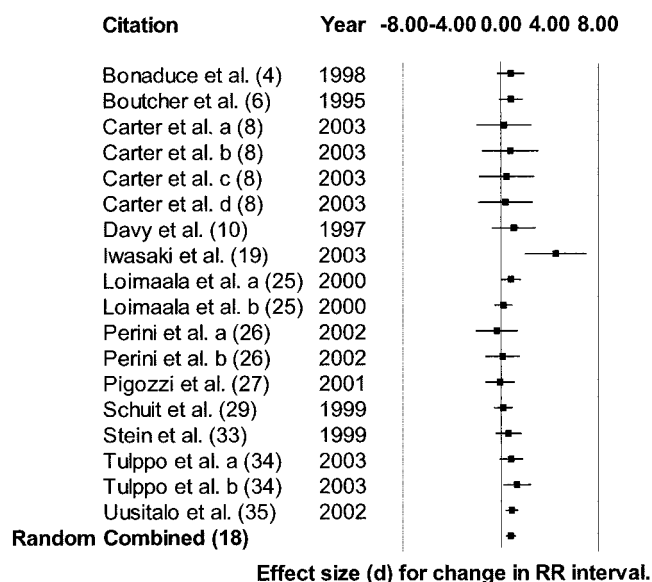


FIGURE 1—Meta-analysis of studies showing change in RR interval due to training. Within study groups—Carter et al. (8): a, middle-aged males; b, young females; c, middle-aged males; d, middle-aged females. Liomaala et al. (25): a, low-intensity training; b, high-intensity training. Perini et al. (26): a, male; b, female. Tulppo et al. (34): a, low volume of training; b, high volume of training.

interval due to exercise training. Eighteen groups were drawn from 12 studies, giving a total of 298 cases. The overall effect size was $d = 0.75$ (C.I. 0.51–0.96, $P < 0.00001$). There was, however, significant heterogeneity between estimates ($Q = 50.99$, $P = 0.00003$). On this basis, subgroup analyses were performed.

In the analysis of HF power (Fig. 2), a total of 20 group comparisons from 13 studies were entered into the final analysis. A total of 322 cases were analyzed, and the overall effect size was $d = 0.48$ (C.I. 0.26–0.70, $P = 0.00003$). The effect sizes for differences in HF were found to show statistical homogeneity ($Q = 19.23$, $P = 0.44$). This was confirmed by calculating $Q/(k - 1) = 1.01$. This value was a borderline value for showing heterogeneity (1.00); therefore, subgroup analyses were performed.

Effect of moderator variables. The moderator variable subgroup analysis for RR interval (Table 1) revealed significant between-group heterogeneity for study subgroups based on subject age and data collection method ($P < 0.10$). Although not statistically significant ($P > 0.10$), there was evidence of some between-group heterogeneity for subject sex, previous activity level, and length of exercise intervention ($Q/(k - 1) > 1$).

Subgroup analysis revealed no statistically significant between-group heterogeneity for HF (Table 2). However, between subgroups based on age, older subjects clearly showed smaller overall effect sizes than either young or middle-aged groups.

Relationships between effect sizes for HRV variables. In addition to assessing linear relationships between RR interval change effect sizes and HRV effect sizes, a variety of fits were applied to the data to assess this relationship more fully. By a process of serial curve estimation

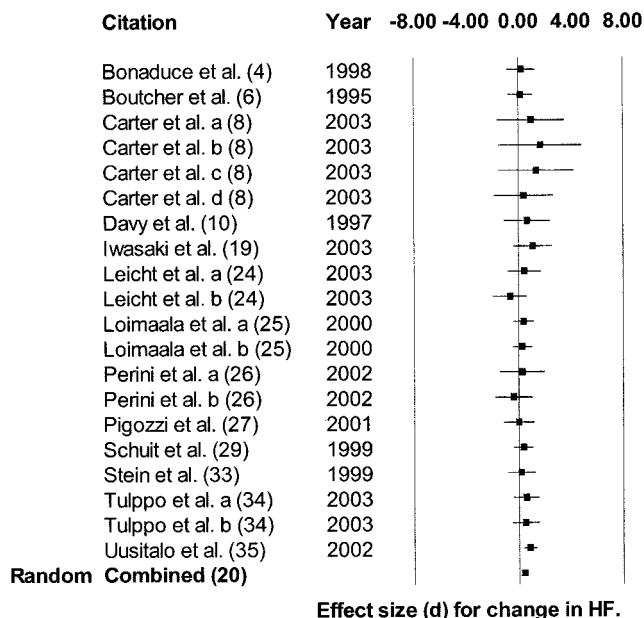


FIGURE 2—Meta-analysis of studies showing change in HF interval due to training. Within study groups—Carter et al. (8): a, middle-aged males; b, young females; c, middle-aged males; d, middle-aged females. Leicht et al. (24): a, middle aged; b, young. Liomaala et al. (25): a, low-intensity training; b, high-intensity training. Perini et al. (26): a, male; b, female. Tulppo et al. (34): a, low volume of training; b, high volume of training.

it was found that a linear relationship was not the best method by which to describe the relationship between RR interval and HF effect sizes (Table 3). The weak linear relationship between effect size for change in RR and HF was improved by using a cubic fit. Neither of these fits achieved statistical significance.

DISCUSSION

The results of the present meta-analysis showed a significant effect of exercise training on resting RR interval and HF power. This supports current theory that aerobic exercise training can alter neuroregulatory control over the heart. Furthermore, these data support the notion that bradycardia after exercise training is partially associated with increased

vagal modulation (12,32) and that this vagal influence may manifest itself as increases in measures of HF power (18).

Additionally, the effect sizes of trials reporting changes in HF due to training were homogenous. Therefore, the evidence provided by the random effects model used with these data may be interpreted with confidence and generalized to populations not included within this analysis. The effects of exercise on RR interval were significantly heterogeneous; thus, subgroup analysis of these data was required. Identical subgroup analyses were also applied to the HF data for comparison.

Effects of moderator variables on RR interval and HF. The effects of training on RR interval and HF differed significantly between age groups. Old and middle-aged subjects showed small and moderate effect sizes, respectively, which were common in magnitude for both RR and HF. This may demonstrate a reduced trainability of the heart and associated neural input with age. Data from studies in the present analysis both support and refute this notion. Carter et al. (8) found increases in HF to be significant in younger subjects but not middle-aged counterparts despite both groups being subjected to a similar training stimulus. Conversely, Leicht et al. (24) found decreased HF in young subjects posttraining, but increases in older counterparts. The latter change (from 1518 ± 702 to 1827 ± 630 ms²) was not significant, due probably to the small ($N = 12$) sample size. A possible reason for the discrepant results in young subjects is the vastly differing levels of baseline HF in these two studies. Leicht et al. (24) found extremely high levels of HF power in their mixed-sex group (3940 ± 704 ms²), whereas Carter et al. (9) found much lower values for females (417.5 ± 303.7 ms²) and males (307.0 ± 407.6 ms²). The very high values in the former study may indicate chronically high levels of vagal activity in this group. Additionally, physical training may have lead to further increased vagal modulation and even AV node saturation. This in turn, may reduce the variability of heart rate at high frequencies and actually lead to reduced HF power (11,14). This phenomenon has been highlighted as a weakness in the application of HRV to certain populations (14).

Discrepancies between these studies (8,24) are difficult to explain as, with the exception of age and sex, these papers give little comparable information about the subjects used.

TABLE 1. Effects of moderator variables on effect size for change in RR interval due to training.

Effect Name	Categories	Effect Size: Mean (95% C.I.)	N Total	P	Between-Group Q	P	Q/(k - 1)	Within-Group Q	P
Sex	Female	0.33 (0.28 to 0.95)	43	0.27	1.20	0.27	1.20	2.11	0.71
	Male	0.70 (0.43 to 0.98)	201	<0.01				6.14	0.70
	Combined	0.64 (0.39 to 0.89)	244	<0.01					
Age	Young	1.32 (0.28 to 0.76)	51	0.144	8.85	0.01	4.42	36.83	<0.001
	Middle aged	0.75 (0.46 to 0.10)	186	<0.001				4.01	0.83
	Old	0.19 (-0.32 to 0.71)	61	0.453				1.29	0.73
	Combined	0.74 (0.51 to 0.96)	298	0.003					
Activity level	Active	0.44 (-0.12 to 0.99)	52	0.123	1.10	0.29	1.10	1.8	0.88
	Sedentary	0.80 (0.55 to 1.05)	246	0.011				46.8	<0.001
	Combined	0.74 (0.51 to 0.96)	298	0.003					
Intervention duration	Long duration	0.83 (0.54 to 1.10)	181	<0.001	1.14	0.28	1.14	42.77	<0.001
	Short duration	0.58 (0.21 to 0.95)	117	0.002				7.06	0.72
	Combined	0.74 (0.51 to 0.96)	298	0.001					
HRV recording	24-h	0.57 (0.18 to 0.95)	106	0.004	2.82	0.09	2.82	2.01	0.57
	Short-term	0.82 (0.53 to 1.10)	192	<0.001				45.6	<0.001
	Combined	0.70 (0.51 to 0.96)	298	0.004					

TABLE 2. Effects of moderator variables on effect size for change in HF due to training.

Moderator variable	Subgroup	Effect Size Mean $\pm$ (95% C.I.)	N Total	P	Between-Group Q	P	Q/(k - 1)	Within-Group Q	P
Sex	Female	0.50 (-0.12 to 1.12)	43	0.168	0.014	0.90	0.007	7.74	0.11
	Male	0.54 (0.26 to 0.82)	201	<0.001				5.53	0.80
	Combined	0.54 (0.28 to 0.78)	287	<0.001					
Age	Young	0.38 (-0.13 to 0.88)	63	0.123	2.06	0.36	1.03	10.9	0.06
	Middle aged	0.61 (0.32 to 0.90)	186	<0.001				5.29	0.72
	Old	0.23 (-0.24 to 0.70)	73	0.333				1.44	0.83
	Combined	0.48 (0.26 to 0.70)	322	<0.001					
Activity level	Active	0.70 (0.15 to 1.26)	52	0.025	0.81	0.37	0.40	7.26	0.20
	Sedentary	0.43 (0.19 to 0.67)	270	0.002				11.0	0.60
	Combined	0.48 (0.26 to 0.70)	322	0.001					
Intervention duration	Long duration	0.46 (0.18 to 0.73)	205	0.001	0.05	0.82	0.02	8.60	0.37
	Short duration	0.51 (0.14 to 0.88)	117	0.006				10.55	0.39
	Combined	0.48 (0.26 to 0.70)	322	<0.001					
HRV recording	24-h	0.34 (-0.04 to 0.72)	106	0.08	0.72	0.40	0.36	0.93	0.97
	Short-term	0.54 (0.27 to 0.81)	216	<0.001				17.6	0.17
	Combined	0.48 (0.26 to 0.70)	322	<0.001					

Particularly no details regarding subjects' training status are given in either paper. Leicht et al. (24) merely state that subjects had not exercised regularly for at least 3 months, and Carter et al. (9) give no subject information. The reasons for these discrepancies in baseline values and training response therefore remain unknown.

In the present study, the largest increase in RR interval was found in the youngest subjects. This was, however, accompanied by a much smaller and statistically nonsignificant increase in HF, suggesting that factors other than increased parasympathetic activity are responsible for the observed bradycardia.

The current meta-analysis showed effect size for change in RR interval was significantly greater in long (>12 wk) interventions than in shorter (<12 wk) studies despite almost identical effect sizes for change in HF. In longer studies, effect size for change in RR interval was, therefore, much greater than that for HF. Similarly, Iwasaki et al. (19) found a much larger effect for increased RR interval than for HF in a group of previously sedentary, young subjects who undertook a year-long training program. Interestingly, there was a large increase in HF early in the training (3 months) that later regressed toward initial values, whereas RR interval tended to increase more uniformly. This may suggest that higher vagal modulation is responsible for initial increases in RR interval but that other factors, such as changes in heart geometry may be responsible for further adaptation.

Evidence of cardiac adaptation due to training, independent of altered autonomic regulation, has been provided in high level athletes (4). Bonaduce et al. (4) found increased RR interval, left ventricular mass, dimensions, and aerobic capacity in elite athletes, whereas HRV remained unchanged. This may help to explain why, in the present data, there were no differences in effect size for HF due to

intervention duration, whereas reported increases in RR interval were greater in longer interventions.

Of methodological interest is the moderator variable HRV recording. It is recommended that HRV measures may be extracted from two broad categories of ECG measurement protocol: ambulatory 24-h recordings and resting measures of 2–7 min (7). Few data are available on which of these types of measurement are the more sensitive. In the present study, however, the effect size for change in RR interval was significantly greater when calculated from short-term recordings than from 24-h data. Similarly, effect size for HF was greater when short-term analysis was used. In fact, HF only showed a statistically significant effect ($d = 0.54$, $P < 0.001$) when extracted from short term ECG recording as opposed to 24-h monitoring ($d = 0.34$, $P = 0.08$).

Due to this strong methodological influence on effect size, it would have been useful to conduct a further subgroup analysis for all variables recorded by each method separately. However, this was deemed to be impracticable due to small group numbers within the subgroups, especially the 24-h group ($N = 4$ studies, $N = 71$ cases).

Problems with subgroups analysis. Despite large differences in mean effect size for subgroup analyses, few statistically significant findings were evident. One reason for this was the large within-group heterogeneity of effect sizes that remained in subgroups. The reasons for such heterogeneity may be fourfold. First, the relatively small number of trials and cases in the overall analysis naturally translates into small numbers in certain subgroups. This in turn means that small disparities in direction and magnitude of effects give rise to large confidence intervals for a given effect size and, therefore, seem heterogenous.

Second, it may be that the highly varied nature of the empirical methods employed to study this phenomenon are responsible. In compiling this analysis, a large number of studies were reviewed and rejected. Yet, a number of studies are included in this analysis that perhaps would not be entered if it were possible to gain sufficient case numbers using stricter criteria. For example, many studies do not include a control group. This is a commonly used criterion

TABLE 3. Effects of different fits on percentage variance explained between measures.

Relationship	Regression	R-squared	P
RR vs HF	Linear	0.119	0.191
RR vs HF	Quadratic	0.174	0.289
RR vs HF	Cubic	0.177	0.489

for inclusion in meta-analyses in other areas, but in the present situation, the number of included cases would be too small to facilitate any meaningful analysis. A second possible criterion would be the use of controlled respiration in studies using short-term data collection. It is known that oscillations in the HF band are closely linked to respiration. Respiration may also impact bradycardia. Only three of the present short-term studies controlled respiratory rate (6,9,10). Of the remaining four, one measured respiratory rate pre- and posttraining and found no difference (26), and three studies exerted no monitoring or control (16,24,35). As logical arguments for use of both spontaneous and controlled breathing exist (24), both types of studies were included in the present analysis. However, the effects of these different techniques were not assessed for the reasons discussed below.

A further reason for the existence of few statistically significant effects may be that the moderator variables responsible for the differences in effect sizes between groups were not identified. Evidence for this lies within the large within-group heterogeneity that was commonly preserved after subgroup analysis. However, in meta-analysis, a balance must be met between the number of subgroup analyses made and finding the reasons for spreads of values. The subgroups analyzed in the present study were numerous in comparison with other analyses. They were, however, made on the basis of physiological justification in line with current recommendations (17).

The final reason for the heterogeneity of results is that this may be a feature of HRV measures themselves. Studies measuring changes in HRV commonly show very disparate effect sizes. For instance, the data of al-Ani et al. (1) met the criteria for inclusion in this analysis but after initial entry were removed due to their disparate nature. In this study, all subjects were exposed to a similar training stimulus. Baseline values for HRV measures often show large between-group variation. It is therefore unsurprising that large differences in effect sizes for change in such variables are commonplace.

Correlations between effect sizes. Although change in RR interval was positively related to HF, this association was only weak, explaining less than 12% of the variance between measures (Table 3). The effect of different fits on the relationship between HF and RR was similar but smaller. Again a cubic model gave a better description of this relationship but managed to explain only a small additional amount (from 11.9 to 17.7%) of common variance.

It seems, therefore, that changes in HF show a smaller overall effect size than changes in RR interval. This is evidence that mechanisms other than increased vagal modulation are responsible for the resting bradycardia observed after a period of physical training.

One such change may be reduced sympathetic modulation, which was not measured here. The reason for this is that within the literature there is poor agreement concerning the use of a single, HRV measure to represent sympathetic nervous modulation. Sympathetic modulation can be measured effectively using muscle sympathetic nerve activity. However, this technique is both expensive and invasive.

Blood pressure variability is growing as an accepted measure of sympathetic modulation, and the combination of this noninvasive method with HRV shows good potential (23).

Other nonneural mechanisms that may influence resting HR include alterations in the mechanical strain put on the pacemaker cells due to cardiac hypertrophy and metabolic alterations in pacemaker cells and neurotransmitter sensitivity (20). It is thought that such adaptations are the result of prolonged exercise training and would typically be brought about by the relatively short exercise interventions used in the studies included here. This proposition is again supported by the differential reactivity of RR interval and HF to varying training regimen durations.

Study limitations. A possible limitation of the study is the use of HF power itself as a marker of cardiac vagal modulation. Some researchers (34) believe the use of this measure to be flawed as its increase is related to increased RR interval length. Proponents of this view commonly employ the coefficient of component variance in addition to HF power in milliseconds² to control for increased RR interval. However, at present, too few studies exist that utilize this measure to facilitate any meta-analysis.

Despite the use of the most commonly employed vagal HRV measure in the current literature, a relatively small number of studies were entered into the analysis, providing a small overall number of cases. This was due mainly to the small number of published studies in this area but also due to the frequent use of small sample sizes. Finally, the inability to perform a full meta-regression on the data weakens the conclusions that can be made concerning the factors responsible for the range of effect sizes shown in the literature. This is not so much a shortcoming of the present study but of the literature cited, which commonly fails to give the necessary information to facilitate such an analysis.

CONCLUSIONS

Analysis of change in RR interval demonstrated that training induces a resting bradycardia accompanied by increased cardiac vagal modulation in healthy individuals. By this mechanism, exercise training may be able to exert an antiarrhythmic effect.

The change in HF power in response to training was both positive and homogenous. Subgroup analyses revealed subject age, sex, and previous level of physical activity to influence the RR interval response to exercise training. A greater effect size was evident in longer duration studies for RR interval but not HF. Where short-term ECG recordings were made to measure RR interval and HF, greater effect sizes were evident than when 24-h recordings were used. The small number of studies using the latter methodology means caution should be employed before any generalizations being made regarding the superiority of one technique over the other.

Recommendations. The large, positive effect of exercise on RR interval and HF shown here mean future simple studies of changes in these variables due to exercise may be redundant. In healthy populations, research should,

instead, focus on randomized controlled trials to examine the effects of possible moderator variables such as those identified here. Additionally, research should focus on populations that typically display low levels of HRV such as postmyocardial infarction patients, diabetics, and the elderly to see whether exercise can facilitate increase or at least attenuate decreases in vagal modulation.

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To facilitate further meta-analyses, authors should report nonsignificant findings and report all HRV measures even if they are not tested statistically. Editors of journals should be encouraged to accept such findings for publication. Where HRV data are published, this should be done in a manner that facilitates their inclusion in meta-analytical studies (27,29,33).