Resting Heart Rate and the Association of Physical Fitness With Carotid Artery Stiffness

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BACKGROUND
Physical fitness is known to influence arterial stiffness. Resting heart rate is reduced by exercise and positively associated with arterial stiffness. This study aimed to investigate the role of resting heart rate in the relationship of physical fitness with arterial stiffness.

METHODS
Subjects were 2,328 young adults from the Childhood Determinants of Adult Health study. Cardiorespiratory fitness was estimated as physical work capacity at a heart rate of 170 bpm. Muscular strength was estimated by hand-grip (both sides), shoulder (pull and push), and leg strength. Arterial stiffness was measured using carotid ultrasound.

RESULTS
Arterial stiffness was negatively associated with cardiorespiratory fitness (men $P < 0.001$; women $P = 0.002$), and positively associated with muscular strength in women ($P = 0.002$) but not in men. Resting heart rate was positively associated with arterial stiffness ($P < 0.001$ both men and women). Adjustment for resting heart rate reduced the inverse association of arterial stiffness with cardiorespiratory fitness by 93.7% (men) and 67.6% (women) but substantially increased the positive association of arterial stiffness with muscular strength among women and revealed a positive association of arterial stiffness with muscular strength among men. These findings were independent of body size, blood pressure, biochemical markers, socioeconomic status, smoking, and alcohol consumption.

CONCLUSIONS
Our findings attribute a key intermediary role for resting heart rate in the relationship between fitness and arterial stiffness, whereby higher cardiorespiratory fitness may reduce arterial stiffness mainly through resting heart rate, and higher muscular strength might have deleterious effects on arterial stiffness that are partially offset by lower resting heart rate.

Keywords: blood pressure; carotid; elasticity; fitness; heart rate; hypertension; strength.

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Stiffening of the large arteries (carotid and aorta) independently predicts future cardiovascular events and all-cause mortality.[1–3] Fully understanding the determinants of arterial stiffness may lead to methods for reducing cardiovascular risk. Currently, there are no pharmacological agents targeted at reducing arterial stiffness. Improvements in lifestyles, including regular exercise to increase cardiorespiratory fitness (CRF), have been shown to be associated with reduced arterial stiffness through a variety of mechanisms that remain unproven.[4] Strength training, however, has been reported to increase arterial stiffness.[5,6]

Elevated resting heart rate (RHR) is an independent cardiovascular risk factor[7] that is positively associated with arterial stiffness.[8,9] High RHR, possibly due to sympathetic hyperactivity, may directly increase arterial stiffness through greater cyclic mechanical shear stress on the arterial wall.[10] Regular exercise to improve CRF reduces RHR,[11] and this could be a pathway by which arterial stiffness is reduced. In contrast, the relationship between arterial stiffness and strength training, which increases muscular strength and may also reduce RHR, remains controversial. Contrary to evidence from intervention studies showing an acute[12,13] or chronic[5,6] increase in arterial stiffness induced by strength training, some studies have shown no change[14,15] or a decrease in arterial stiffness.[16] Despite the uncertain implication for arterial stiffness, strength training is recommended to improve general health.[17,18]

The aim of this study was to investigate the associations of CRF and muscular strength with arterial stiffness in young adults and to determine whether RHR had an intermediary role in any associations.

METHODS

Study population

The Childhood Determinants of Adult Health (CDAH) study collected baseline data in 1985 on a nationally representative sample of 8,498 Australian schoolchildren aged

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7–15 years.[19] In this study, the analyses were restricted to 2,328 nonpregnant subjects (49.4% male) who attended clinics in the first follow-up during the period of 2004–2006.[20] The CDAH study was approved by the local ethics committee.

Cardiorespiratory fitness and muscular strength

CRF was estimated as physical work capacity (PWC) at a heart rate of 170 bpm (PWC_{170}). PWC_{170} was measured using a bicycle ergometer (Monark Exercise AB, Vansbro, Sweden) pedaled at 60 rpm.[21] Because the absolute workload achieved is a function of muscle mass,[22] CRF was calculated as PWC_{170} adjusted for lean body mass to create an index uncorrelated with lean body mass (see Supplementary Methods for more details).

Five measures of strength (left and right grip, shoulder push and pull, and leg strength) were measured using appropriate dynamometers (Smedley's Dynamometer, TTM, Tokyo, Japan). Detailed procedure is reported in the Supplementary Methods. Principal components analysis was used to estimate the first principal component of the five measures of strength for men and women separately.[23] The first principal component was then adjusted for body weight to create an index uncorrelated with weight. This was the indicator of muscular strength used in this study.

Blood pressure and arterial stiffness

B-mode ultrasound studies of arterial stiffness were performed using a portable Acuson Cypress (Siemens Medical Solutions USA, Mountainview, CA) platform with a 7.0 MHz linear-array transducer by a single technician,[24] following standardized protocols.[25] Intima-media thickness and end-systolic and end-diastolic diameters (with intrclass correlations of repeated measurements $r = 0.99$) were measured from the posterior wall of the left common carotid artery.[20] Brachial systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured supine during the ultrasound study.[20] Carotid distensibility (CD), the inverse of stiffness, was measured as:

$$CD = ([D_{sbp} - D_{dbp}]/D_{dbp})/(SBP - DBP)$$

where D_{sbp} is the end-systolic diameter, D_{dbp} is the end-diastolic diameter. CD is defined as the ability of the arterial wall to expand and contract passively with the changes in pressure.

Covariables

RHR was measured using an Omron HEM907 Digital Automatic Blood Pressure Monitor (Omron Corporation, Kyoto, Japan) after a 5-minute rest. A mean of 3 readings was used. RHR was also measured by electrocardiogram during the ultrasound procedure; analyses using this value of RHR provided the same results. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. Mean arterial pressure (MAP) was calculated (MAP = ⅓SBP + ⅔DBP). Socioeconomic status, smoking, alcohol consumption, and data on resistance-type activities (including work-related vigorous activities and strength training) were obtained by questionnaire.[20] Total minutes per week of leisure-time physical activity was obtained using the International Physical Activity Questionnaire.[26] High-density lipoprotein cholesterol, low-density lipoprotein cholesterol, insulin, triglycerides, and glucose were measured using 12-hour overnight fasting blood samples.[20]

Statistical analyses

Muscular strength and CRF were standardized z scores for men and women separately. Values reported in Table 2 were calculated by applying Pearson's correlation coefficients to the ranks of the variables. For other analyses, right-skewed outcomes were transformed before analysis. The regression estimates reported are in original units of the outcomes for 1 SD increase in the study factor.[27,28] Direct and indirect associations (through RHR) of CRF and muscular strength with arterial stiffness were estimated by structural equation model analysis. Three levels of participation in resistance-type activities were defined as reporting no activities at all, either reporting strength training or occupational exposure to vigorous activities, or reporting both. For analyses, these ordered levels were graded by consecutive integer scores. Of the 2,328 participants, 28 participants (1.2%) were on antihypertensive medication, 2 participants (0.08%) had had a heart attack, and 3 participants (0.13%) had had a stroke. Excluding these participants from analyses did not change our findings (not shown). The fit of all final models were assessed as being adequate.

RESULTS

Table 1 displays participants' characteristics. Male participants had higher values of PWC_{170} and measures of strength and greater body sizes (weight, height, BMI, waist circumference, and lean body mass) but smaller skinfolds than females. Males also had lower mean values of CD, reflecting stiffer carotid arteries. Other than having lower RHR than women, men generally had higher values of cardiovascular risk factors.

Table 2 shows correlations of CRF and muscular strength with CD that were almost unchanged after mutual adjustment. CRF was positively correlated with CD, but this correlation was almost eliminated after adjusting for RHR (men $r = 0.01$; women $r = 0.04$). Leisure-time physical activity was correlated (all $P < 0.001$) with both CRF (men $r = 0.30$; women $r = 0.33$) and RHR (men $r = -0.20$; women $r = -0.19$). Men and women with strength training had lower CD on average than those without ($P = 0.05$). The correlation between muscular strength and CD was much weaker among women who undertook strength training than women who did not ($P = 0.02$). Muscular strength was not correlated with CD before adjusting for RHR among men and among women with strength training ($r = -0.01$) but was negatively correlated with CD after adjusting for RHR (men $r = -0.06$; women with strength training $r = -0.06$). The correlation of muscular

Table 1. Participants' characteristics

	Men		Women		
	Mean	(SD)	Mean	(SD)	*P* values[a]
Age, y	31.6	(2.6)	31.3	(2.6)	0.044
PWC_{170}, watts	193.7	(45.1)	127.9	(30.5)	<0.001
Right grip, kg	48.8	(7.7)	29.5	(5.2)	<0.001
Left grip, kg	46.9	(7.7)	27.9	(5.1)	<0.001
Shoulder push, kg	49.1	(13.0)	25.6	(7.6)	<0.001
Shoulder pull, kg	40.0	(13.0)	20.9	(7.1)	<0.001
Leg strength, kg	167.9	(38.9)	89.6	(28.2)	<0.001
Weight, kg	83.5	(13.9)	65.8	(12.9)	<0.001
Height, cm	179.5	(6.8)	165.6	(6.3)	<0.001
Body mass index, kg/m^2	25.9	(3.9)	23.9	(4.2)	<0.001
Waist circumference, cm	88.0	(9.8)	75.7	(9.6)	<0.001
Sum of four skinfolds, mm	61.7	(25.9)	73.4	(30.8)	<0.001
Lean body mass, kg	63.6	(7.7)	43.9	(6.1)	<0.001
Resting heart rate, bpm	68.3	(9.9)	73.2	(9.7)	<0.001
Systolic pressure, mm Hg	124.7	(10.7)	110.5	(10.1)	<0.001
Diastolic pressure, mm Hg	74.5	(8.9)	69.8	(8.6)	<0.001
Mean arterial pressure, mm Hg	91.3	(8.7)	83.4	(8.5)	<0.001
Insulin, mU/L	6.3	(4.0)	6.0	(3.4)	0.001
HDL-cholesterol, mmol/L	1.26	(0.25)	1.51	(0.33)	<0.001
LDL-cholesterol, mmol/L	3.04	(0.83)	2.74	(0.74)	<0.001
Triglycerides, mmol/L	1.03	(0.65)	0.81	(0.42)	<0.001
Carotid distensibility, %/10 mm Hg	1.94	(0.64)	2.35	(0.80)	<0.001
Stiffness index	5.29	(1.82)	4.83	(1.68)	<0.001
Young's elastic modulus, mmHg.mm	293.3	(111.5)	230.3	(87.4)	<0.001

Abbreviations: HDL, high-density lipoprotein; LDL, low-density lipoprotein; PWC_{170}, physical work capacity at a heart rate of 170 bpm.
[a]*P* values for the difference between men and women.

strength with participation in resistance-type activities (see Methods) was stronger for men ($r = 0.19$; $P < 0.001$) than for women ($r = 0.06$; $P = 0.05$). Among participants undertaking strength training, total time spent on training was more strongly correlated with muscular strength among men ($r = 0.16$; $P = 0.02$) than among women ($r = 0.08$; $P = 0.24$).

RHR was positively associated with arterial stiffness (Supplementary Figure S1) and was negatively associated with the difference between D_{sbp} and D_{dbp} (men $r = -0.29$; women $r = -0.25$; both $P < 0.001$) but was not associated with carotid intima-media thickness (men $r = -0.04$, $P = 0.22$; women $r < 0.01$, $P = 0.88$). Adjustment for blood pressure did not change the association of RHR with either CD or the difference between D_{sbp} and D_{dbp} (not shown).

We next examined which other factors might be associated with CRF and muscular strength to better understand the possible causal pathways in the associations of CRF and muscular strength with arterial stiffness. Supplementary Table S1 presents age-adjusted associations of CRF and muscular strength with RHR and with MAP, BMI, insulin, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and triglycerides, which are known to be associated with arterial stiffness. Among these factors, RHR was most strongly correlated with CRF (men $r = -0.36$; women $r = -0.30$) and muscular strength (men $r = -0.14$; women $r = -0.15$). The associations of CRF and muscular strength with RHR were slightly reduced after mutual adjustment (not shown).

Table 3 shows the estimated change in CD with a 1 SD increase of CRF and muscular strength after adjustment for relevant factors. For muscular strength, the regression coefficients were changed slightly to moderately by controlling for BMI or insulin, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and triglycerides, but the largest changes occurred on adjustment for RHR, revealing a substantially stronger association. The direct and indirect effects (through RHR) of 1 SD increase in muscular strength on CD were −0.03% per 10 mm Hg (−0.07, −0.00) and 0.03% per 10 mm Hg (0.02, 0.05) for men and −0.10% per 10 mm Hg (−0.16, −0.05) and 0.03% per 10 mm Hg (0.01, 0.04) for

Table 2. Rank correlations of cardiorespiratory fitness and muscular strength with carotid distensibility.

Fitness measures	Men	Women
Cardiorespiratory fitness[a]	0.121***	0.103**
Muscular strength[b]	−0.002	−0.108**
Partial correlations[c]		
Cardiorespiratory fitness	0.128***	0.119***
Muscular strength	−0.020	−0.112**

**$P < 0.01$; ***$P < 0.001$.

[a]Sex-specific z scores of cardiorespiratory fitness, which was physical work capacity at a heart rate of 170 bpm adjusted for lean body mass.

[b]The first principal component of 5 measures of strength that was then adjusted for body weight and expressed as a sex-specific z score.

[c]Correlations of cardiorespiratory fitness with carotid distensibility adjusting for muscular strength and of muscular strength with carotid distensibility adjusting for cardiorespiratory fitness.

women. For CRF, adjustment for RHR reduced the association with CD by 93.7% (men) and 67.6% (women). The direct and indirect effects (through RHR) of 1 SD increase in CRF on CD were 0.01% per 10 mm Hg (−0.03, 0.05) and 0.07% per 10 mm Hg (0.05, 0.09) for men and 0.01% per 10 mm Hg (−0.05, 0.06) and 0.05% per 10 mm Hg (0.03, 0.07) for women. Additional adjustment for MAP, BMI, or blood biomarkers after adjustment for RHR slightly changed the coefficients produced by adjustment for RHR. There was no interaction of RHR with blood pressure and other factors (not shown). Further adjustment for socioeconomic status, smoking, and alcohol consumption did not alter these findings (not shown).

Although these cross-sectional data are silent about attribution of causation, Figure 1 shows hypothesised pathways through which CRF and muscular strength could affect arterial stiffness. An increase in CRF might reduce arterial stiffness mainly through reducing RHR. There are 2 possible pathways through which an increase in muscular strength may affect arterial stiffness. First, muscular strength may influence

Table 3. Regression of carotid distensibility on cardiorespiratory fitness and muscular strength with adjustment for resting heart rate and other relevant factors

	Men			Women		
Fitness measures	β	(95% CI)[a]	R^2	β	(95% CI)[a]	R^2
Cardiorespiratory fitness						
Unadjusted	7.7	(3.4–12.0)	0.03	6.5	(1.1–12.0)	0.02
Adjusted for						
Age	7.9	(3.6–12.2)	0.03	7.4	(2.0–12.8)	0.03
Age, MAP	6.4	(2.1–10.8)	0.04	6.3	(1.0–11.7)	0.05
Age, BMI	6.7	(2.3–11.0)	0.04	5.8	(0.4–11.3)	0.04
Age, biomarkers[b]	6.4	(1.9–10.8)	0.04	5.4	(−0.1 to 11.0)	0.04
Age, RHR	0.5	(−3.8 to 4.8)	0.12	2.4	(−3.1 to 7.9)	0.10
Age, RHR, MAP	0.3	(−4.0 to 4.6)	0.12	2.2	(−3.3 to 7.7)	0.10
Age, RHR, BMI	−0.4	(−4.8 to 3.9)	0.12	0.8	(−4.8 to 6.4)	0.10
Age, RHR, biomarkers[b]	−0.5	(−4.9 to 3.9)	0.12	0.4	(−5.3 to 6.1)	0.09
Muscular strength						
Unadjusted	−0.8	(−5.2 to 3.5)	0.03	−8.2	(−13.4 to −3.1)	0.04
Adjusted for						
Age	−0.4	(−4.8 to 3.9)	0.03	−8.4	(−13.5 to −3.2)	0.05
Age, MAP	−0.6	(−4.9 to 3.7)	0.05	−8.9	(−14.0 to −3.9)	0.06
Age, BMI	−1.2	(−5.9 to 3.1)	0.04	−9.0	(−14.1 to −3.9)	0.05
Age, biomarkers[b]	−1.5	(−5.9 to 2.9)	0.04	−9.0	(−14.1 to −3.9)	0.06
Age, RHR	−3.6	(−7.7 to 0.5)	0.13	−11.1	(−16.1 to −6.2)	0.12
Age, RHR, MAP	−3.5	(−7.6 to 0.6)	0.13	−11.2	(−16.1 to −6.2)	0.13
Age, RHR, BMI	−4.1	(−8.2 to −0.0)	0.14	−11.6	(−16.5 to −6.6)	0.12
Age, RHR, biomarkers[b]	−4.5	(−8.6 to −0.3)	0.14	−11.4	(−16.4 to −6.4)	0.12

Abbreviations: BMI, body mass index; CI, confidence interval; MAP, mean arterial pressure; RHR, resting heart rate.

[a]Regression coefficient (95% confidence interval) multiplied by 100 for better display.

[b]Including adjustment for insulin, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and triglycerides.

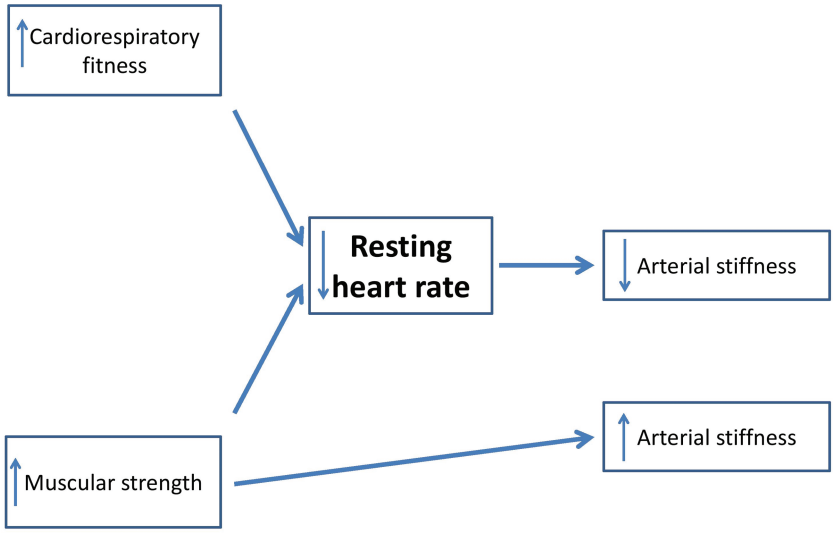

Figure 1. The pathways illustrate postulated direct and indirect effects of cardiorespiratory fitness and muscular strength on arterial stiffness.

arterial stiffness directly, possibly more so for women. Second, muscular strength may exert its influence indirectly by reducing RHR. If these pathways exist as postulated, our results suggest they are roughly counterbalanced for men.

DISCUSSION

We found that arterial stiffness was negatively associated with CRF but positively associated with muscular strength. Greater CRF and muscular strength were independently associated with lower RHR and, thereby, lower arterial stiffness. Although the negative association of CRF with arterial stiffness appeared to be mediated by RHR, adjustment for RHR revealed a stronger positive association of muscular strength with arterial stiffness because the offsetting beneficial effects by lower RHR were removed. Adjustment for BMI, MAP, or blood biomarkers did not change these findings. These results suggest that lower RHR is a key intermediary factor in the beneficial effect of CRF on arterial stiffness and that the deleterious effect of greater muscular strength on arterial stiffness is partially offset by its indirect effect by lower RHR. If correct, these pathways enhance understanding of the mechanisms involved and clarify the controversial findings on strength training and cardiovascular health.

Women in this age range had lower carotid stiffness than men. This is consistent with previous findings.[25,29] Although the relationship of CRF with arterial stiffness was similar for men and women, there were differences for muscular strength. Arterial stiffness was more strongly associated with muscular strength for women than for men, and for men an association was revealed only after adjusting for RHR. The different ways in which men and women acquire muscular strength may play a part in this. Muscular strength was greater for men and was associated with time spent on strength training and occupational exposure to vigorous activities. This suggests men are more likely to acquire muscular strength from participation in resistance-type activities. This is the mechanism proposed by which acquisition of muscular strength leads to increased arterial stiffness.[5] Results from subgroup analyses for women with strength training support this inference. The relationship between muscular strength and arterial stiffness was weaker for women with strength training than those without and more like that of the men. Again, as with the men, adjusting for RHR strengthened the relationship markedly.

The intermediary role of RHR may help explain previous inconsistent findings about strength training and arterial stiffness. Our findings are inconsistent with those from Fahs *et al.*,[30] that showed an inverse association of muscular strength with arterial stiffness among 79 men. Whereas we adjusted our measures of strength for body weight to create an index uncorrelated with weight as recommended,[31] Fahs *et al.*,[30] divided muscular strength by body weight. Cole *et al.*,[31] indicated that this may produce a spurious association. Using our data, we noted that the effect of dividing muscular strength by body weight was to reverse the sign of the correlations with CD, changing them from negative to positive. A spurious association arising in this way will be reduced by adjusting for weight, and, consistent with this, adjusting for body weight or BMI eliminated the negative association in our data (not shown).

For CRF, its inverse association with arterial stiffness was mostly eliminated by controlling for RHR. This suggests RHR mediates the relationship between CRF and arterial stiffness. It might be argued that RHR may be a marker of CRF, so adjusting for RHR would eliminate any association of CRF with any outcomes. However, this was not true in our study. Findings from intervention studies suggest RHR to be an outcome of endurance training to improve CRF.[11] In our data, RHR could not be considered as a marker of CRF because the correlation between them was only weak to moderate. Furthermore, the results of adjusting for CRF and RHR are very different. For example, the association of muscular strength with arterial stiffness was substantially increased

after adjusting for RHR (Table 3) but was slightly changed after adjusting for CRF (Table 2). Also suggesting they are associated with outcomes by different pathways, adjustment for RHR eliminated the association of CRF with arterial stiffness but only weakly to moderately reduced the associations of CRF with other cardiovascular risk factors such as blood pressure, metabolic syndrome, or fatness (not shown).

Our findings show physical fitness is associated with arterial stiffness even among young and generally-healthy adults with low levels of arterial stiffness, which is consistent with associations between physical activity and arterial stiffness among children and young adults reported in other studies.[32,33] Because the association of CRF with arterial stiffness was independent of muscular strength and the association of muscular strength was independent of CRF, the effects on arterial stiffness for those who undertake both endurance and strength training may be the net effect from the 2 types of training. Although the direct detrimental effect of high muscular strength due to training on arterial stiffness may be partially offset by its indirect beneficial effect by RHR, individuals who do strength training might also benefit from endurance training to further minimize the adverse effects on arterial stiffness from strength training, as previously suggested.[34]

Although not completely understood, high RHR may increase mechanical load on the arterial wall and expose it to greater pressure and shear stress by shortening the diastolic period. This might lead to greater arterial wall stiffness, possibly by promoting vascular smooth muscle cell growth and collagen deposition.[10,35] Changes in heart rate can change the computed value of CD. However, distinction needs to be drawn between the acute vs. chronic exposure to changes in heart rate and the corresponding response in CD. In the acute setting,[36] the increase in blood pressure that accompanies the rise in heart rate will result in a functional increase in arterial stiffness. That is, there is temporary recruitment of collagenous fibres in the arterial wall to "stiffen" the vessel against increased distending pressure.[37] On the other hand, chronic exposure to high RHR and blood pressure results in arterial wall remodelling and a structural increase in stiffness. Our study measured heart rate at rest, and the estimated relationship with arterial stiffness is likely to represent a chronic effect. The associations of RHR with either CD or carotid diameters remained unchanged after adjusting for blood pressure, which suggests that the relationship between RHR and CD was not due to an acute change in pressure. We therefore believe that the relationship between RHR and arterial stiffness observed in our study was due to structural changes in the arterial wall and not from artefact related to computation of CD.

It is well known that blood pressure can influence arterial stiffness and independently predicts mortality.[38] Thus, any studies of arterial stiffness predicting mortality should account for blood pressure.[3] RHR independently predicts mortality[7] and is positively associated with arterial stiffness[8,9] but is generally not considered in studies of arterial stiffness. These data together suggest that future studies of arterial stiffness predicting mortality need to take account of RHR.

This study used a large nationally representative sample of young Australians on whom standardized measurements were made of an extensive range of study factors. The few other studies of muscular strength and arterial stiffness have either used small samples or compared normal, sedentary controls with high-intensity trained athletes. A limitation of our study was the use of brachial, instead of carotid, pulse pressure to calculate CD. This is likely to have resulted in underestimation of the association between RHR and CD because the fittest people (with higher CD and lower RHR) would also be more likely to have the greatest systolic and pulse pressure amplification (higher brachial compared with central systolic and pulse pressure). The higher estimated values for SBP may therefore underestimate the calculated CD. The cross-sectional design limits the causal inference that can be drawn about the relationship of RHR with arterial stiffness. However, given evidence that endurance training to improve CRF reduces arterial stiffness,[4] we discount the possibility of reverse causation in our data (where greater arterial stiffness leading to higher RHR) because adjusting the association between CRF (an antecedent of arterial stiffness) and RHR by arterial stiffness had only the most minor impact in our data (not shown). This is consistent with recent findings that CD and aortic distensibility decreased with increasing RHR.[9,39] Because our study included data on young adults only, we cannot generalize our findings to an older population.

In conclusion, our findings attribute a key intermediary role for RHR in the relationship between physical fitness and arterial stiffness. Higher CRF may reduce arterial stiffness by reducing RHR, and although higher muscular strength is associated with greater arterial stiffness, the association is partially offset by reduced RHR. An indirect association of muscular strength with arterial stiffness by RHR would help reconcile the inconsistent evidence on the response of arterial stiffness to strength training and provide some support for the inclusion of strength training in recommendations and guidelines for exercise to improve general health.

SUPPLEMENTARY MATERIAL

Supplementary materials are available at *American Journal of Hypertension* (http://ajh.oxfordjournals.org).

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DISCLOSURE

The authors declared no conflict of interest.

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