

The association between muscular power from childhood to adulthood and adult measures of glucose homeostasis

Short Running Title: Muscular power and adult glucose homeostasis

Brooklyn J. Fraser, BBiotechMedRes(Hons) <sup>1</sup>

Leigh Blizzard, PhD <sup>1</sup>

Michael D. Schmidt, PhD <sup>2</sup>

Terence Dwyer, MD, MPH <sup>1, 3</sup>

Alison J. Venn, PhD <sup>1</sup>

Costan G. Magnussen, PhD <sup>1, 4\*</sup>

<sup>1</sup> *Menzies Institute for Medical Research, University of Tasmania, Hobart, Tasmania, Australia.*

<sup>2</sup> *Department of Kinesiology, University of Georgia, Athens, USA.*

<sup>3</sup> *George Institute for Global Health, Oxford Martin School and Nuffield Department of Obstetrics & Gynaecology, Oxford University, Oxford, UK.*

<sup>4</sup> *Research Centre of Applied and Preventive Cardiovascular Medicine, University of Turku, Turku, Finland.*

Correspondence to Costan G Magnussen, Menzies Institute for Medical Research, University of Tasmania, Private Bag 23, Hobart 7001, Tasmania, Australia. Phone: +61 3 6226 7700. Fax: +61 3 6226 7704. E-mail: [cmagnuss@utas.edu.au](mailto:cmagnuss@utas.edu.au)

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## **Abstract**

This study aimed to assess whether the longitudinal association between childhood muscular fitness and adult measures of glucose homeostasis persist despite changes in muscular fitness across the life course. This prospective longitudinal study included 586 participants who had their muscular power (standing long jump distance), cardiorespiratory fitness (CRF) and waist circumference measured as children (aged 9, 12, 15 years) and again 20 years later as adults. In adulthood, these participants also provided a fasting blood sample which was tested for glucose and insulin. Glucose homeostasis measures including insulin resistance (HOMA2-IR) and beta cell function (HOMA2- $\beta$ ) were estimated. Child and adult muscular power levels were separated into thirds and tracking groups (persistently low, decreasing, persistently moderate, increasing, persistently high) were created. Sex-stratified multivariable linear regression models were used to examine the association between muscular power tracking groups and adult measures of glucose homeostasis. Compared with males with persistently high muscular power, males with increasing and persistently low muscular power had higher fasting insulin (increasing:  $\beta=1.12$  mU/L,  $p=0.04$ ; persistently low:  $\beta=2.12$  mU/L,  $p=0.001$ ) and HOMA2- $\beta$  (increasing:  $\beta=8.50\%$ ,  $p=0.03$ ; persistently low:  $\beta=11.27\%$ ,  $p=0.01$ ) independent of CRF and males with persistently low muscular power had greater fasting insulin ( $\beta=1.22$  mU/L,  $p=0.02$ ) and HOMA2-IR ( $\beta=0.14$ ,  $p=0.02$ ) independent of waist circumference. Non-significant associations were present for females. For males, maintaining persistently high muscular power between childhood and adulthood could lead to a healthier adult glucose homeostasis profile.

**Keywords:** Muscle Fitness, Muscle power, Epidemiology, Cohort

## **Introduction**

The long-term health benefits of childhood muscular fitness are becoming increasingly recognised<sup>1</sup>. Of interest, is whether childhood muscular fitness is associated with adult cardiovascular and metabolic disease, collectively referred to as cardiometabolic health outcomes. Childhood muscular fitness is inversely associated with metabolic syndrome risk<sup>2</sup> and impaired glucose homeostasis in adulthood<sup>3,4</sup>. In adults, muscular fitness is inversely associated with type 2 diabetes<sup>5,6</sup>. Better muscular fitness could reflect greater physical activity levels and lower adiposity, or increased participation in resistance training which could be stimulating key proteins in the insulin signalling pathway<sup>7</sup>. Jumping performance, one measure of muscular power assessed by a standing long jump, is a reliable and valid measure of overall muscular fitness as it demonstrates very good test-retest reliability and good construct validity<sup>8-11</sup>. The standing long jump is commonly used to assess muscular fitness in field-based settings. Whether the association between childhood muscular power and cardiometabolic health outcomes persist despite changes in muscular power into adulthood is unknown. Understanding these associations could provide insight into whether strategies aimed at improving cardiometabolic health should target child or adult muscular power levels, or whether muscular power at both life stages is important. Using data from the Childhood Determinants of Adult Health (CDAH) Study, we aimed to examine the association between muscular power from childhood to adulthood and measures of glucose homeostasis in adulthood.

## **Materials and Methods**

### *Participants*

The Australian Schools Health and Fitness Survey (ASHFS) was conducted in 1985, where health, anthropometric and physical fitness data, including jumping performance, were collected on a nationally representative sample of 8,498 Australian school children. Children aged 9, 12, and 15 years received additional testing including a sub-maximal cardiorespiratory fitness (CRF) test. A total of 2,615 children provided jumping performance, CRF and anthropometric data at baseline. From 2004-06, as part of the Childhood Determinants of Adult Health Study, participants had their health and fitness, including jumping performance, retested and a fasting blood sample taken. Non-pregnant

participants with child and adult measures of jumping performance, CRF and waist circumference, and adult measures of glucose homeostasis were included in analyses (n=586). A flow chart of participation is represented in Figure S1. The State Directors General of Education approved the ASHFS. Participant consent was obtained from a parent and assent obtained from the child. The Southern Tasmania Health and Medical Human Research Ethics Committee approved the follow-up study with participants providing written informed consent.

### *Muscular power*

A standing long jump test was used to measure muscular power in both childhood and adulthood. The standing long jump test required a two-footed take-off and landing. Before take-off, participants were encouraged to bend their knees and during the jump, encouraged to swing their arms to aid forward momentum. The farthest distance (to the closest 0.1 cm) from two attempts was used in analyses. To remove the influence of differences in body mass on jumping performance, measures of muscular power not attributable to body mass were created by regressing standing long jump distance on body mass and using the residuals<sup>2,12</sup>. Muscular power was then standardised for age and sex. Child and adult muscular power levels were categorised into thirds and “persistently low” (lowest third in both childhood and adulthood), “decreasing” (moved from highest or middle third in childhood to a lower third in adulthood), “persistently moderate” (remained in middle third in both childhood and adulthood), “increasing” (moved from bottom or middle third in childhood to a higher third in adulthood) and “persistently high” (remained in highest third in both childhood and adulthood) muscular power groups were created.

### *Clinical measurements*

Waist circumference was measured to the nearest 0.1 cm using a constant tension tape at the level of the umbilicus in childhood and at the narrowest point between the lower costal border and iliac crest in adulthood. Childhood body mass was measured using regularly calibrated scales to the nearest 0.5 kg and adult body mass was measured using Heine scales (Heine, Dover, NH) to the nearest 0.1 kg. Height was measured to the closest 0.1 cm using a KaWe height tape (KaWe Kirchner & Wilhelm,

Aspeg, Germany) in childhood and a Leicester height measure (Invicta, Leicester, UK) in adulthood. Body mass index (BMI) was calculated as body mass (kg) divided by height (m) squared. Using age-specific regression estimates<sup>13</sup>, body density and fat percentage were calculated using the log of the sum of skinfolds (triceps, biceps, subscapular, and suprailiac) measured to the nearest 0.1 mm using Holtain calipers (Holtain, Crymych, UK) in childhood and to the nearest 0.5 mm using Slim Guide Calipers in adulthood. The Siri formula was used to calculate body fat from body density<sup>14</sup> and fat-free mass was estimated as the difference between total body mass and fat mass. CRF was measured as physical work capacity at a heart rate of 170 beats per minute (PWC<sub>170</sub>). To perform this test, children used a Monark 818E bicycle ergometer (Monark Exercise AB, Vansbro, Sweden) and adults used a Monark 828E bicycle ergometer (Monark Exercise AB, Vansbro, Sweden). The sub-maximal PWC<sub>170</sub> test incorporated three successive 3-minute workloads (childhood) or three successive 4-minute workloads (adulthood) that increased resistance stepwise. In the final minute of each workload, heart rate and watts were recorded and these data were plotted and extrapolated to provide PWC<sub>170</sub>. Given the absolute work load achieved in this test is a function of muscle mass, measures of PWC<sub>170</sub> not attributable to fat-free mass were created by regressing PWC<sub>170</sub> on fat-free mass and using the residuals<sup>12</sup>. Child and adult measures of waist circumference and PWC<sub>170</sub> were age- and sex-standardised.

#### *Adult measures of glucose homeostasis*

In adulthood, participants provided a blood sample after fasting for 12 hours. Serum glucose concentrations were measured enzymatically<sup>15</sup> and fasting insulin was measured using a microparticle enzyme immunoassay kit (AxSYM; Abbot Laboratories, Abbot Park, IL) or an electrochemiluminescence immunoassay (Elecsys Modular Analytics E170; Roche Diagnostics, Mannheim, Switzerland) with interassay standardisation<sup>16</sup>. The homeostatic model assessment (HOMA2) calculator (version 2.2.3 available from <http://www.dtu.ox.ac.uk/homacalculator>) estimated insulin resistance (HOMA2-IR) and beta cell function (HOMA2- $\beta$ ) using fasting glucose and fasting insulin values<sup>17-19</sup>. Fasting glucose levels outside of 3.5-25 mmol/L and fasting insulin levels outside of 2.88-57.60 mU/L (20-400 pmol/L) were classified as being outside the range of

realistic fasting values accepted by the calculator<sup>18</sup>. Eligible participants with levels outside these ranges were excluded from HOMA2 calculations (n=50).

### *Statistical analyses*

All statistical analyses were performed using Stata (Version 15.0, StataCorp, College Station, Texas).

### *Demographics*

Participant characteristics are sex-stratified and presented as mean (standard deviation, SD) for normally distributed data or mean (SD) following the appropriate transformation for skewed variables (fasting insulin and HOMA2-IR).

### *Muscular power from childhood to adulthood and adult measures of glucose homeostasis*

Multivariable linear regression models examined the association between muscular power from childhood to adulthood and adult glucose homeostasis measures, independent of age, length of follow-up (Model 1) and childhood and adulthood CRF (Model 2) and waist circumference (Model 3) z-scores. A significant sex interaction was present, therefore analyses were sex-stratified. The persistently high muscular power group was used as the reference category. Linear regression models followed an approach by Seamen et al.<sup>20</sup>, where inverse probability weighting, with multiple imputation of incomplete baseline data, was used to account for missing data at follow-up.

## **Results**

### *Demographics*

Participant characteristics are sex-stratified and presented in Table 1. Length of follow-up ranged from 18.7 to 21.0 years, with a mean (SD) of 19.9 (0.6) years. In both childhood and adulthood, males had greater muscular power, body mass, waist circumference and CRF, compared with females. In adulthood, females had lower fasting glucose and insulin than males.

### *Muscular power from childhood to adulthood and adult measures of glucose homeostasis*

For both males (Table 2) and females (Table 3), muscular power from childhood to adulthood was not associated with fasting glucose. Males with increasing and persistently low muscular power had higher fasting insulin and HOMA2- $\beta$  independent of CRF and males with persistently low muscular power had greater fasting insulin and HOMA2-IR independent of waist circumference, compared with males with persistently high muscular power. Although not statistically significant, similar trends were present for males with persistently moderate and decreasing muscular power. For females, upon adjustment for CRF, effect estimates attenuated, and the direction of effect often reversed.

## **Discussion**

Our findings suggest that males able to maintain high muscular power from childhood to adulthood could have a healthier adult glucose homeostasis profile. Although the difference in effect by sex was not expected, our findings for males were in line with existent literature where the association between low muscular fitness and less favourable glucose homeostasis profiles spans childhood to adulthood<sup>3,4</sup>, and adults with low muscular fitness have an increased risk of type 2 diabetes<sup>5,6</sup>. These associations could reflect higher muscular power resulting from increased participation in resistance training improving insulin sensitivity by stimulating key proteins in the insulin signalling pathway<sup>7</sup>. Our observation that males who increased or decreased their muscular power across the life course had a more adverse glucose homeostasis profile compared with those who maintained high levels also highlight the importance, at least for males, of having high muscular power in childhood and maintaining these high levels into adulthood.

In this study, male sex and deficits of muscular power emerging in childhood were associated with elevated insulin and other indicators of impaired glucose homeostasis among young adults. The association between male sex and future impaired glucose homeostasis is supported by findings from a previous review in which it was highlighted that for a given BMI, males have greater insulin resistance and lower insulin sensitivity compared with females<sup>21</sup>. Sex-differences in insulin sensitivity, explained by males having greater visceral and hepatic adipose tissue in combination with not having the protective effect offered by heightened estrogen levels<sup>21</sup>, could explain our observed



sex-differences between muscular power tracking groups and measures of glucose homeostasis. Our results suggest that male sex and persistent deficits in muscular power are on the same causal pathway. That is, differences in measures of glucose homeostasis between muscular power tracking groups were greater for males compared with females, where those with the most unfavourable glucose homeostasis profile in early adult years were males with persistently low muscular power. Furthermore, the observed sex difference could be a result of our handling of relative changes in muscular power. It is possible that the relatively high muscular power group for adult females was not high enough in an absolute sense to improve glucose homeostasis.

Contemporary Australian children have lower levels of muscular power compared with their counterparts from 30-years ago<sup>22,23</sup>. A potential consequence of the secular decline in childhood muscular power levels is that a lower proportion of today's children will have "high" muscular power and therefore, are less likely to be part of the favourable persistently high muscular power group. This could mean more of today's children are at risk of developing an adverse glucose homeostasis profile in the future. There is a need to promote the importance of childhood muscular fitness and its associated health benefits. Our findings reinforce recent updates to physical activity guidelines which now emphasise the importance of performing both aerobic and muscle strengthening activities in childhood, as well as in adulthood<sup>24</sup>.

We observed stronger associations with outcomes that included insulin rather than glucose. This could be explained by the natural history of type 2 diabetes<sup>25</sup>. Increases in insulin resistance and insulin secretion occur during the early stages of disease progression, with fasting glucose levels becoming elevated and type 2 diabetes developing after beta cells fail and insulin deficiency occurs<sup>25</sup>. Fasting blood samples were collected when participants were young adults (aged 26–36 years) and potentially in the early stages of type 2 diabetes progression when compensatory mechanisms were keeping fasting glucose levels relatively normal. We expect the association between muscular power tracking groups and glucose-based outcomes to become stronger if these analyses were repeated using data from future follow-ups, where participants are older, and the disease has progressed.

In the CDAH Study, we previously found that a decline in CRF between childhood and adulthood was more strongly associated with obesity and insulin resistance in adulthood than low levels of childhood CRF<sup>16</sup>. Furthermore, those with persistently high CRF and increasing CRF were less likely to have insulin resistance in adulthood than the persistently low and decreasing CRF groups. We concluded that high adult, but not child, CRF was the more important factor associated with low risk of insulin resistance in adulthood<sup>16</sup>. Our findings for muscular power in males oppose those for CRF, suggesting that both child and adult levels of muscular power are important.

This study had limitations. Our muscular power tracking groups were created based on a single measurement of muscular power in childhood and adulthood and are cohort specific. Further, these muscular power tracking groups were created based on relative changes in muscular power. This approach meant a group of participants were classified as having “low muscular power” at both time points. The ideal approach would have been to use thresholds of muscular power known to reduce health risks, but Australian based criterion-referenced health-related fitness thresholds known to associate with adverse health outcomes in adulthood have not been defined. Future research is required to identify muscular fitness (strength, power, endurance) thresholds associated with adverse health outcomes in later life. Another potential limitation is bias due to differential loss to follow-up. However, we reduced the likelihood of bias by using a statistical approach that takes account of missingness and restores representativeness by rebalancing the study sample to reflect the baseline population<sup>20</sup>. Study strengths include the long follow-up (~20 years) of a national sample and the use of a standardised measure of muscular power, the standing long jump, at both time-points that has demonstrated good construct validity<sup>8,9</sup> and very good test-retest reliability<sup>10,11</sup>.

Our findings reinforce recent updates to physical activity guidelines by highlighting the importance of children and adults performing both aerobic and muscle strengthening activities<sup>24</sup> and suggest activities aimed at specifically improving muscular power could benefit future health. As the health benefits of muscular fitness are becoming increasingly recognised for both sexes and extend beyond

improved cardiometabolic health, both males and females are encouraged to engage in activities aimed at making their muscles stronger and more powerful. Findings from this study further highlight the importance of muscular power in both childhood and adulthood, particularly for males. As persistently high muscular power was associated with the most favourable glucose homeostasis profile in adulthood, implementing strategies aimed at increasing muscular power in childhood and maintaining these behaviours into adulthood could benefit future glucose homeostasis.

### **Perspective**

Our findings highlight the importance of both child and adult muscular power levels for future glucose homeostasis, indicators of risk for type 2 diabetes mellitus. Given contemporary children have lower muscular power compared with the children in this study<sup>22,23</sup>, our findings suggest a lower proportion of today's children will have "high" muscular power and a greater proportion could potentially be at risk of developing an adverse glucose homeostasis profile in later life. These findings suggest implementing strategies aimed at increasing muscular power in childhood and maintaining these behaviours into adulthood could provide most benefit. The findings reinforce recent updates to physical activity guidelines by highlighting the importance of children and adults performing activities aimed at improving the strength and power of their muscles in addition to aerobic exercise<sup>24</sup>.

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Table 1. Characteristics of participants.

		Male	Female
	n	Mean (SD)	Mean (SD)
Childhood			
Age, y	586	11.9 (2.5)	11.9 (2.5)
Standing long jump, cm	586	161.2 (31.7)	143.6 (23.9)
Standing long jump not attributable to body mass, cm	586	161.1 (31.4)	143.4 (23.9)
Age- and sex-standardised standing long jump not attributable to body mass	586	0.12 (1.01)	0.11 (0.99)
Body mass, kg	586	44.2 (13.9)	42.1 (11.8)
Body mass index, kg/m <sup>2</sup>	586	18.4 (2.7)	18.5 (2.8)
Waist circumference, cm	586	66.0 (8.1)	62.9 (7.4)
PWC <sub>170</sub> , watts	586	109.6 (43.8)	79.0 (27.6)
PWC <sub>170</sub> not attributable to fat-free mass, watts	586	109.3 (40.0)	79.6 (26.5)
Adulthood			
Age, y	586	31.9 (2.5)	31.7 (2.6)
Standing long jump, cm	586	188.4 (25.0)	133.8 (27.5)
Standing long jump not attributable to body mass, cm	586	188.6 (23.9)	134.5 (24.0)
Age- and sex-standardised standing long jump not attributable to body mass	586	0.03 (0.98)	−0.06 (1.03)
Body mass, kg	586	85.8 (13.7)	68.9 (14.7)
Body mass index, kg/m <sup>2</sup>	586	26.4 (3.9)	24.9 (5.2)
Waist circumference, cm	586	89.7 (9.5)	78.0 (11.3)
PWC <sub>170</sub> , watts	586	198.0 (43.8)	132.3 (32.5)
PWC <sub>170</sub> not attributable to fat-free mass, watts	586	197.6 (38.7)	131.8 (30.5)
Fasting glucose, mmol/L	586	5.20 (0.42)	4.85 (0.40)
Fasting insulin, mU/L	585	6.25 (5.01)	5.87 (3.38)
HOMA2-IR	536	0.83 (0.51)	0.79 (0.34)
HOMA2-β, %	536	84.58 (29.07)	93.03 (30.03)

Abbreviations: SD, standard deviation; PWC<sub>170</sub>, physical work capacity at 170 beats per minute; HOMA2-IR, homeostasis model assessment 2-insulin resistance; HOMA2- $\beta$ , homeostasis model assessment 2-beta cell function.

Table 2. Association between muscular power from childhood to adulthood and adult glucose homeostasis measures for males.

Muscular power from childhood to adulthood	Model 1*				Model 2†			Model 3‡		
	n	$\beta$	SE	p-value	$\beta$	SE	p-value	$\beta$	SE	p-value
Fasting glucose, mmol/L										
Persistently high	66	0	REF	REF	0	REF	REF	0	REF	REF
Increasing	64	-0.03	0.07	0.71	-0.03	0.07	0.70	-0.05	0.07	0.49
Persistently moderate	31	0.07	0.09	0.38	0.07	0.08	0.38	0.06	0.08	0.51
Decreasing	86	0.01	0.07	0.94	0.01	0.07	0.85	-0.00	0.07	0.99
Persistently low	58	0.09	0.08	0.24	0.10	0.08	0.21	0.08	0.08	0.31
Fasting insulin, mU/L										
Persistently high	66	0	REF	REF	0	REF	REF	0	REF	REF
Increasing	64	1.17	0.57	0.04	1.12	0.53	0.04	0.37	0.44	0.40
Persistently moderate	31	1.41	0.72	0.05	1.39	0.70	0.05	0.81	0.59	0.17
Decreasing	86	0.94	0.58	0.11	0.77	0.57	0.18	0.35	0.47	0.46
Persistently low	58	2.40	0.64	<0.001	2.12	0.61	0.001	1.22	0.53	0.02
HOMA2-IR										
Persistently high	57	0	REF	REF	0	REF	REF	0	REF	REF
Increasing	60	0.11	0.06	0.08	0.11	0.06	0.08	0.04	0.05	0.41
Persistently moderate	30	0.10	0.08	0.22	0.10	0.08	0.20	0.05	0.07	0.49
Decreasing	79	0.08	0.06	0.22	0.07	0.06	0.26	0.04	0.05	0.47
Persistently low	55	0.23	0.07	0.002	0.21	0.07	0.003	0.14	0.06	0.02
HOMA2- $\beta$ , %										
Persistently high	57	0	REF	REF	0	REF	REF	0	REF	REF
Increasing	60	8.70	4.10	0.03	8.50	3.99	0.03	4.75	2.96	0.11
Persistently moderate	30	5.80	5.11	0.26	6.18	4.98	0.22	2.90	4.29	0.50
Decreasing	79	6.39	3.74	0.09	5.82	3.72	0.12	3.58	3.05	0.24
Persistently low	55	12.34	4.70	0.009	11.27	4.55	0.01	6.67	3.68	0.07

\* Model 1 is adjusted for childhood age and length of follow-up; † Model 2 is adjusted for Model 1 covariates and childhood and adulthood cardiorespiratory fitness age and sex specific z-scores; ‡ Model 3 is adjusted for Model 2 covariates and childhood and adulthood waist circumference age and sex specific z-scores. Abbreviations:  $\beta$ , beta coefficient; HOMA2-IR, homeostatic model assessment 2-insulin resistance; HOMA2- $\beta$ , homeostatic model assessment 2-beta cell function; SE, standard error.

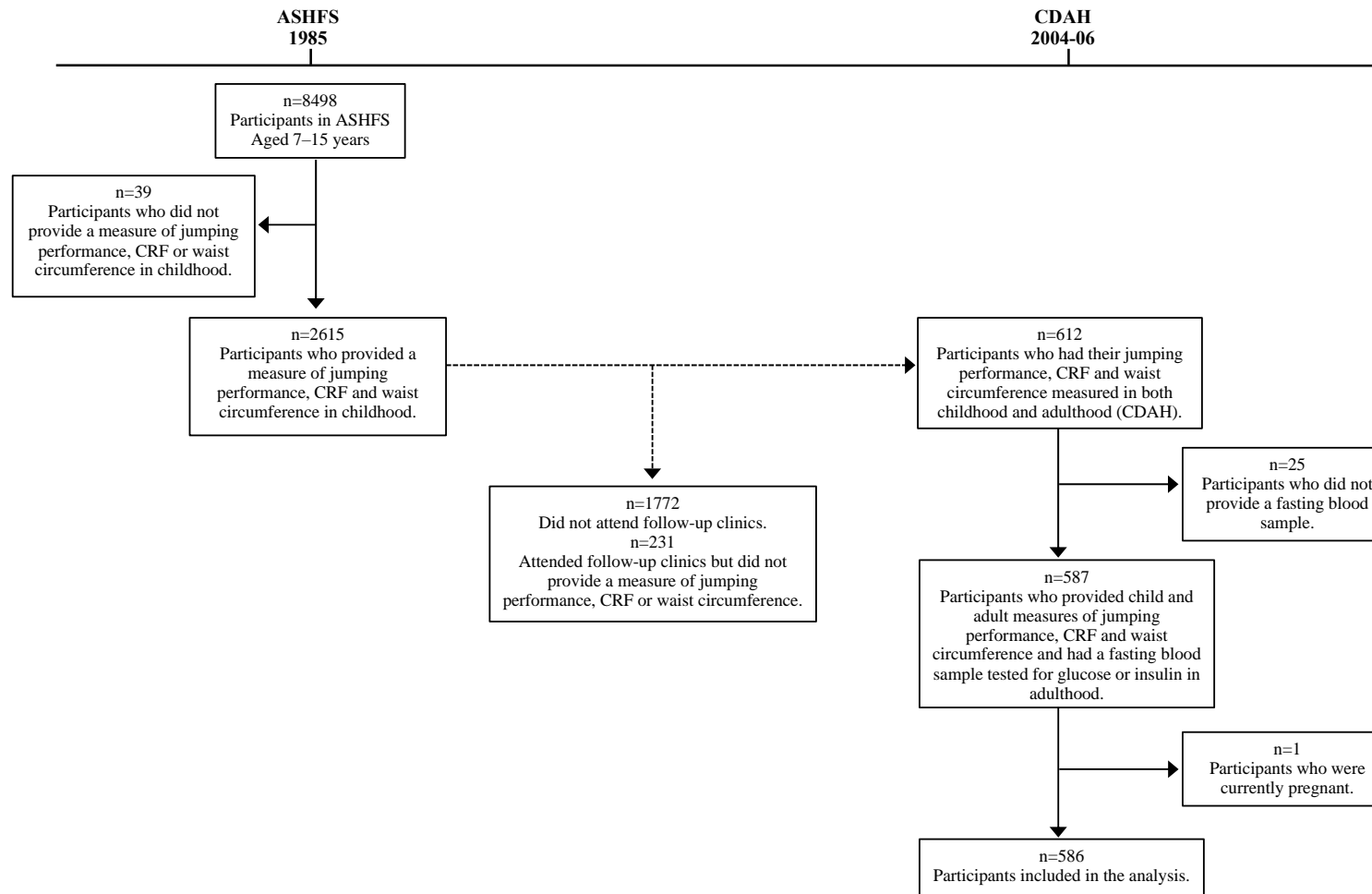
Table 3. Association between muscular power from childhood to adulthood and adult glucose homeostasis measures for females.

Muscular power from childhood to adulthood	Model 1*				Model 2†			Model 3‡		
	n	$\beta$	SE	p-value	$\beta$	SE	p-value	$\beta$	SE	p-value
Fasting glucose, mmol/L										
Persistently high	40	0	REF	REF	0	REF	REF	0	REF	REF
Increasing	57	-0.02	0.09	0.83	0.00	0.09	0.98	-0.03	0.09	0.70
Persistently moderate	40	-0.09	0.08	0.31	-0.05	0.09	0.56	-0.06	0.09	0.46
Decreasing	98	-0.04	0.09	0.64	-0.01	0.09	0.91	-0.05	0.09	0.58
Persistently low	46	-0.10	0.10	0.29	-0.06	0.09	0.52	-0.11	0.09	0.24
Fasting insulin, mU/L										
Persistently high	39	0	REF	REF	0	REF	REF	0	REF	REF
Increasing	57	0.79	0.85	0.35	0.53	0.80	0.51	-0.10	0.66	0.88
Persistently moderate	40	-0.55	0.57	0.35	-0.92	0.63	0.15	-1.19	0.68	0.08
Decreasing	98	1.12	0.59	0.06	0.41	0.65	0.53	-0.28	0.66	0.67
Persistently low	46	0.69	0.61	0.26	-0.28	0.67	0.68	-1.12	0.71	0.12
HOMA2-IR										
Persistently high	35	0	REF	REF	0	REF	REF	0	REF	REF
Increasing	49	0.10	0.09	0.25	0.08	0.08	0.32	0.00	0.08	0.95
Persistently moderate	34	-0.00	0.06	0.99	-0.03	0.07	0.69	-0.07	0.08	0.36
Decreasing	94	0.12	0.06	0.06	0.06	0.07	0.33	-0.01	0.07	0.92
Persistently low	43	0.06	0.07	0.40	-0.02	0.07	0.82	-0.10	0.08	0.19
HOMA2- $\beta$ , %										
Persistently high	35	0	REF	REF	0	REF	REF	0	REF	REF
Increasing	49	8.48	5.83	0.15	6.44	5.59	0.25	1.88	5.05	0.71
Persistently moderate	34	4.12	5.11	0.42	0.39	5.08	0.94	-1.75	5.56	0.75
Decreasing	94	12.05	4.52	0.008	6.16	4.53	0.17	2.19	4.69	0.64
Persistently low	43	8.32	5.48	0.13	0.57	5.34	0.92	-3.92	5.65	0.49

\* Model 1 is adjusted for childhood age and length of follow-up; † Model 2 is adjusted for Model 1 covariates and childhood and adulthood cardiorespiratory fitness age and sex specific z-scores; ‡ Model 3 is adjusted for Model 2 covariates and childhood and adulthood waist circumference age and sex specific z-scores. Abbreviations:  $\beta$ , beta coefficient; HOMA2-IR, homeostatic model assessment 2-insulin resistance; HOMA2- $\beta$ , homeostatic model assessment 2-beta cell function; SE, standard error.



## Online-only supplement



**Figure S1.** Flow chart of participation. Abbreviations: ASHFS, Australian Schools Health and Fitness Survey; CRF, cardiorespiratory fitness; CDAH, Childhood Determinants of Adult Health.