ANALYTIC REVIEW

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Associations of Objectively Measured Vigorous Physical Activity With Body Composition, Cardiorespiratory Fitness, and Cardiometabolic Health in Youth: A Review

Abstract: Background. *Increasing evidence suggests that vigorous physical activity (VPA) in youth may yield greater health benefits than moderate (MPA) or moderate-to-vigorous physical activity (MVPA). The purpose of this review was to assess the relationship between PA intensity and body composition, cardiorespiratory fitness (CRF), and cardiometabolic (CM) biomarkers in youth.* Methods. *We conducted a systematic review of observational studies examining PA intensity and selected health outcomes in youth aged 6 to 18 years. Forty-five articles were selected for final review.* Results. *VPA was more strongly associated with reduced body fat and central adiposity compared with MPA and/or MVPA. Additionally, VPA was more strongly associated with increased CRF when compared with lower intensities. Findings were inconclusive between all PA intensity levels and CM biomarkers, and several significant relationships observed for VPA were attenuated when controlling for CRF.* Conclusions. *A potential VPA dose is identified as yielding favorable health benefits in adiposity and fitness. While CM biomarkers were not consistently associated with PA intensity level, the literature suggests VPA may yield health benefits above those received from MPA for reduced adiposity and improved CRF. This review highlights the need for longitudinal observational and experimental studies to determine optimal VPA dose for CM health in youth.*

Keywords: vigorous physical activity; intensity; youth; biomarkers

> Accumulating evidence suggests that VPA [vigorous physical activity] may be more beneficial than moderate PA (MPA) for several health outcomes among youth.

Among youth, substantial evidence has documented that physical activity (PA) is associated with several favorable health outcomes, including body composition, fitness, and

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an optimal cardiometabolic (CM) profile.[1,2] On the weight of this evidence, the US Department of Health and Human Services (USDHHS) developed PA guidelines, which recommend that youth engage in 60 minutes of moderate-to-vigorous PA (MVPA) daily.[3] Evidence from several observational studies supports that 60 minutes of MVPA per day is a sufficient dose to elicit reductions in obesity, increases in fitness, and improvements in CM biomarkers indicative of chronic disease.[4-7] However, only one-fourth of youth report achieving this 60-minute MVPA recommendation,[8] with objective assessments indicating considerably fewer.[9]

Accumulating evidence suggests that VPA may be more beneficial than moderate PA (MPA) for several health outcomes among youth.[10-12] This has been observed in adult populations, where a PA intensity dose-response relationship has been demonstrated for favorable health outcomes.[13-15] As a result, PA recommendations for adults include a specific dose for VPA.[9] However, among youth no specific dose for VPA exists in the current PA guidelines.[3]

While previous reviews have assessed the influence of PA intensity on health outcomes among youth, many included self-reported measures of PA.[11,12] Considering the ubiquitous use and superiority of objectively measured PA tools and more recently published studies, an updated review of studies evaluating the association between PA intensity and health outcomes among youth is warranted. Thus, the purpose of this review is to summarize and evaluate the scientific literature on studies assessing the impact of PA intensity on adiposity, cardiorespiratory fitness (CRF) and CM biomarkers in the youth population. Our hypothesis is that the studies in this review will demonstrate that VPA is a stronger predictor of adiposity, CRF, and CM biomarkers compared with lesser PA intensities. As such, the aim of this study is to provide an evidence base from which VPA-specific recommendations may be developed for youth.

Methods

Four electronic research databases were searched (Web of Science, PubMed-Medline, CINAHL Complete, and Physical Education Index) to identify peer-reviewed articles assessing the effect of PA intensity on health outcomes in youth. Search terms were categorized into 4 main domains: activity, age group, mode of measurement, and health outcome. The following keywords were used in varying combinations: activity (eg, "physical activity" OR "exercise" OR "sport" OR "dance") AND youth (eg, "child" OR "adolescent" OR "teen") AND objective monitoring (eg, "accelerometer" OR "heart rate monitor") AND health outcomes (eg, "BMI" OR "adiposity" OR "fitness" OR "performance" OR "blood pressure" OR "cholesterol" OR "triglycerides" OR "glucose" OR "insulin"). Age group was limited to youth aged 6 to 18 years. There were no restrictions for publication date. In order to be eligible for analysis the studies must have satisfied the following inclusion criteria:

1. Observational study design (eg, cohort or cross-sectional)
2. Utilized objective measures of PA (eg, accelerometers or heart rate monitors)
3. Published in peer-reviewed journals in the English language

Intervention studies were not included, as the focus of this review is on the benefits of PA incorporated into daily activity as opposed to the effects of acute changes in PA over a limited duration. In addition, all studies must have independently evaluated and reported the relationship between VPA and health outcomes of interest. For example, studies that assessed MVPA but did not evaluate VPA separately were excluded. Moreover, accelerometer validation studies as well as those assessing PA as an outcome variable were excluded. Studies drawing from the same cohort were included if they reported different outcomes.

Data Extraction

Data were extracted by the primary author (MG) from all selected articles, including sample size, participant demographics (age, sex, race/ethnicity, country), and the identified cohort when applicable. Information on PA assessment methods was also gathered, which included details regarding the assessment tool (accelerometer model) and PA intensity cut-points (eg, accelerometer counts). Statistical results describing the association between VPA and selected health outcomes were extracted to examine the magnitude and directionality of the relationship. For studies that also assessed the relationship between MPA and/or MVPA for the selected health outcomes, similar statistical information was extracted for comparison purposes.

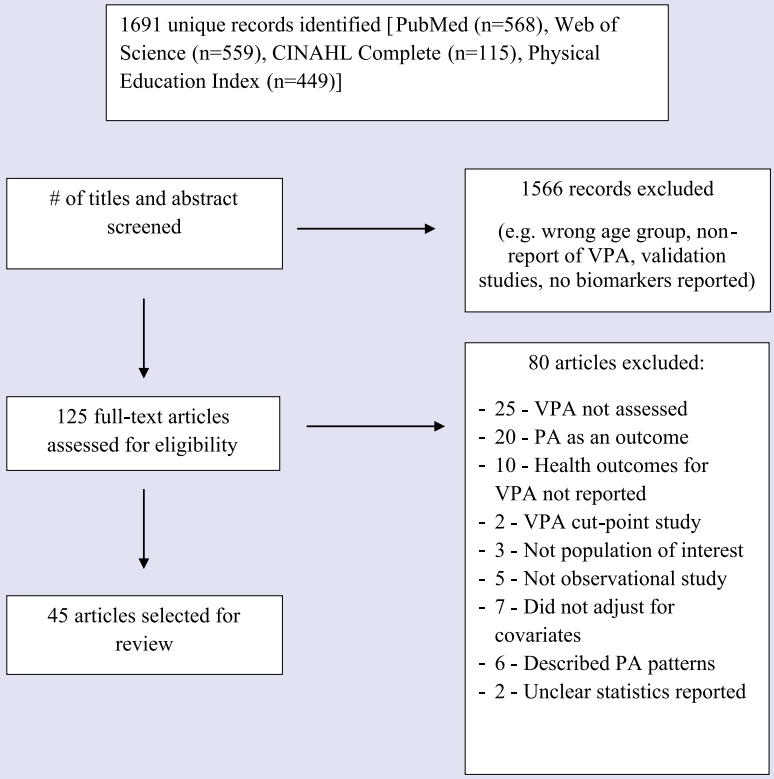
Two additional reviewers (SM and CB) interpreted the statistical data to determine the independent associations of PA intensities (MPA, MVPA, VPA) with health outcomes. In the case where partial correlations and regression data were both reported, results from regression models were chosen to represent the relationship between PA intensity and the health outcome of interest. Studies only reporting unadjusted bivariate associations were removed from the review, as established covariates were not adjusted for, to reduce bias in the findings.

Results

A flowchart describing the study selection process is depicted in Figure 1. Initial searches for all databases yielded a total of 1691 records. Titles and abstracts were examined, and of these, 1566 were excluded yielding 125 articles eligible for the initial review. After a full-text review, 46 articles were eliminated for failing to meet inclusion criteria such as it did not assess VPA (54%; n = 25) or did not assess health outcomes of interest (22%, n = 10), resulting in 79 articles for further review. Of these, an additional 34 articles were removed for reasons including assessment of PA intensity as an outcome variable (59%; n = 20) or failing to adjust for covariates

Figure 1.

Flowchart of study selection and ascertainment processes.



(20.6%; n = 7), resulting in 45 articles eligible for the final review.

Demographics

Participant demographics are presented in Table 1. Geographical locations varied across the studies with 7 conducted in the United States,[16-22] 4 in Canada,[23-26] and 33 in Europe.[27-57] One study was conducted internationally[7] and the final study did not specify location.[58] Sample sizes varied considerably ranging from 36 to 6539 participants.[7,38] A majority of articles (~96%) used a sample that included children and adolescents of both sexes and the remaining studies consisted of female-only[16] or male-only[57] samples. Several individual studies were derived from numerous large-scale studies, including the European Youth Heart Study (EYHS; n = 6)[31,32,41,42,44,46] and Healthy Lifestyle in Europe by Nutrition in Adolescence (HELENA; n = 6).[33,35,36,39,40,54]

Outcome Variables

Because of varied methods of statistical reporting, results were categorized according to the directionality and statistical significance of the relationships for comparison within each intensity level. Magnitudes of the associations are listed in Table 2, which includes all reported relationships regardless of statistical significance. Outcomes are presented separately by sex, age, PA measurement modality, and/or analytical method employed within a study. A representation of proportional directionality of associations between PA and all health outcomes are summarized in Tables 3-5.

Body Composition

Body Mass Index. Nineteen studies examined the link between PA intensity and body mass index (BMI).* Eleven of the 19 associations tested indicated that VPA was significantly, negatively correlated with weight status, and the magnitude was greater than the relationship between MPA or MVPA and BMI for both sexes.† Two studies[45,57] demonstrated that VPA was more strongly associated with BMI only among boys,[45,57] with MVPA being a stronger predictor among only girls.[45]

The remaining 8 associations from 7 studies did not demonstrate a significant relationship between PA and weight status, regardless of intensity.[22,27,32,36,38,53,55] Despite these nonsignificant findings, there were several noticeable trends between PA intensity and weight status. Three of these 8 associations found VPA to be a stronger negative predictor of weight status,[27,32,53] whereas 4 reported MPA to be either more strongly related to weight status than VPA or of equal magnitude with VPA.[36,38,55] Mixed results were found for the remaining study as there were no consistent trends observed.[22]

Body Fat. Seventeen studies addressed PA intensity and its connection with percent body fat (%BF).‡ Of these studies, 13 (~76%) demonstrated that VPA was negatively associated with %BF and at a greater magnitude than the relationship between MPA or MVPA and %BF.§ One of the 17 studies[17] found that MPA was more strongly associated with %BF than VPA while another study[31] found MPA and VPA similarly associated in magnitude. For 1 report[25] that employed various analytical techniques (eg, partial correlation, logistic and mean regression), each technique observed a different PA intensity more strongly and negatively predicting percent body fat. The remaining study found a nonsignificant relationship between PA intensity and percent body fat.[22]

Two studies[38,47] assessed the relationship between fat mass index

*References 7, 16, 21, 22, 24-27, 32, 35, 36, 38, 42, 45, 47, 53, 56, 57, 59.

†References 7, 16, 21, 24-26, 35, 42, 47, 56.

‡References 17, 18, 22, 23, 25, 28, 31, 32, 35, 36, 40, 46, 48, 50, 52, 53, 55.

§References 18, 23, 28, 32, 35, 36, 40, 46, 48, 50, 52, 53, 55.

Table 1.

Sample Demographics From Articles Reporting the Relationship Between Moderate Physical Activity, Moderate-to-Vigorous Physical Activity, and Vigorous Physical Activity and Measures of Body Composition, Cardiorespiratory Fitness, and Cardiometabolic Biomarkers in Youth.

Authors (Year)	N	Females (n)	Males (n)	Average Age (Years)	Race/ Ethnicity	Average Weight Status	Location
Aires et al (2010)[26]	111	62	49	14.5	NR	All: 22.24 kg/m^2 %OWOB: Males: 38.8% Females: 27.4%	Portugal (1 school)
Alhassan and Robinson (2008)[15]	208	208	0	9.4	African American	All: 20.7 kg/m^2	United States (RCT subsample)
Blaes et al (2011)[27]	187	101	86	9.1	European	Males: 17.6 kg/m^2 Females: 17.1 kg/m^2	France (16 elementary school classes)
Butte et al (2007)[16]	897	456	441	NW: Males: 10.7 Females: 10.7 OW: Males: 11.1 Females: 10.9	Hispanic	%OW: Males: 56% Females: 49.6%	United States—VIVA la Familia Study
Carson et al (2014)[25]	315	187	128	12.2	NR	All: 18.5 kg/m^2 Males: 18.7 kg/m^2 Females: 18.4 kg/m^2	Canada—Healthy Hearts Prospective Cohort Study of PA and CM Health in Youth
Chaput et al (2012)[22]	550	251	299	9.6	European Canadian	%OW: Males: 20% Females: 17% %OB: Males: 22% Females: 23%	Canada, Quebec—QUALITY study

(continued)

Table 1. (continued)

Authors (Year)	N	Females (n)	Males (n)	Average Age (Years)	Race/ Ethnicity	Average Weight Status	Location
Danielsen et al (2011)[28]	86	38	48	11.0	European	NW: BMI_{mean}: 17.12kg/m^2 OB: BMI_{mean}: 28.41kg/m^2	Norway—Treatment program for families with obese children
Dencker et al (2010)[48]	468	222	246	6.7	European	Males: 15.9 kg/m^2 Females: 16.0 kg/m^2	Denmark—Copenhagen School Child Intervention Study subsample (46 preschool classes in 18 schools)
Dencker et al (2008)[59]	225	101	124	Males: 9.8 Females: 9.8	European	Males: 17.4 kg/m^2 Females: 17.5 kg/m^2	Sweden (4 schools, grades 3-4)
Dencker et al (2008)[49]	158	76	82	Males: 9.9 Females: 9.8	European	All: 17.4 kg/m^2	Sweden (4 schools, grades 3-4)
Dencker et al (2007)[50]	226	101	125	Males: 9.9 Females: 9.7	European	All: 17.4 kg/m^2	Sweden (4 schools, grades 3-4)
Dencker et al (2006)[51]	248	108	140	Males: 9.9 Females: 9.7	European	All: 17.4 kg/m^2	Sweden (4 schools, grades 3-4)
Denton et al (2013)[29]	1292	654	638	9.7	European	All: 17.2 kg/m^2	Denmark, Estonia, Portugal, Norway—EYHS subsample
Dubose et al (2015)[57]	72	61%	39%	9.5	European American: 60%	All: BMI_{mean}: 22.1 kg/m^2 %OW: 16% %OB: 45%	United States
Ekelund et al (2007)[30]	9-10 y: 1008 15-16 y: 738	9-10 y: 504 15-16 y: 404	9-10 y: 504 15-16 y: 334	9-10; 15-16	European	9-10 y: Males: 17.4 kg/m^2 Females: 17.2 kg/m^2 15-16 y: Males: 20.8 kg/m^2 Females: 20.8 kg/m^2	Denmark, Estonia, Portugal—EYHS subsample

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Table 1. (continued)

Authors (Year)	N	Females (n)	Males (n)	Average Age (Years)	Race/ Ethnicity	Average Weight Status	Location
Ekelund et al (2004)[31]	409	224	185	11	European	%OW: Males: 18.3% Females: 18.8% %OB: Males: 3.2% Females: 4.9%	England (Northwest; 8 schools)
España-Romero et al (2010)[32]	254	132	122	12.5-17.5	European	NR	Spain—HELENA study subsample
Fairclough et al (2012)[55]	409	224	185	11	European	%OW: Males: 18.3% Females: 18.8% %OB Males: 3.2% Females: 4.9%	England (Northwest; 8 schools)
Gaya et al (2009)[33]	163	97	66	13.91-14.02	European	NR	Portugal
Gutin et al (2005)[17]	421	225	196	16.2	European American (48.9%) African American (51.1%)	All: 23.1 kg/m^2	United States (high schools in Augusta, GA)
Hay et al (2012)[23]	605	357	248	12.1	Canadian	OWOB: All: 26%	Canada—Healthy Hearts Prospective Cohort Study of PA and CM Health in Youth Study subsample
Jiménez-Pavón et al (2013)[34]	2200	1184	1016	14.7	European	All: 21.1 kg/m^2	Europe—HELENA Study sample

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Table 1. (continued)

Authors (Year)	N	Females (n)	Males (n)	Average Age (Years)	Race/ Ethnicity	Average Weight Status	Location
Jiménez-Pavón et al (2013)[36]	2025	987	1038	7.6	European	%BF All: 16.9 kg/m^2 Males: 16.9 kg/m^2 Females: 16.9 kg/m^2	Europe—IDEFICS study sample
Jiménez-Pavón et al (2013)[35]	1053	554	499	14.9	European	All: 21.4 kg/m^2	Europe—HELENA Study sample
Katzmarzyk et al (2015)[7]	6539	3554	2985	10.4		Males: 18.4 kg/m^2 Females: 18.4 kg/m^2 %OB: All: 12.4% (5.2% - 24.6%) Males: 15.5% Females: 9.9%	Australia, Brazil, Canada, China, Colombia, Finland, India, Kenya, Portugal, South Africa, United Kingdom, United States—ISCOLE Study
Kennedy et al (2012)[37]	36	16	20	6.7	European	Males: 0.27 (BMI *z*-score) Females: 0.40 (BMI *z*-score)	Scotland—SGA follow-up study
Lätt et al (2015)[56]	136	0	136	11.9 (baseline)	European	20.4 kg/m^2 (baseline) 21.5 kg/m^2 (2-y follow-up)	Estonia
Mark and Janssen (2011)[18]	1165	557	608	12.9	All %NHW 26.1% %NHB 34.1% %Hispanic 36.6% %Other 3.3%	%BF All: 29.1%	United States—NHANES 2003-2004
Martínez-Gómez et al (2010)[52]	192	94	98	Males: 14.7 Females: 15.0	European	Males: 21.8 kg/m^2 Females: 21.7 kg/m^2	Spain—AFINOS Study subsample
Martínez-Gómez et al (2010)[53]	1808	964	844	CRF UH: 14.7 H: 14.6	European	CRF UH: 22.5 kg/m^2 H: 20.2 kg/m^2	Europe—HELENA Study sample
Martínez-Gómez et al (2009)[54]	202	103	99	Males: 14.7 Females: 14.9	European	Males: 22.2 kg/m^2 Females: 21.8 kg/m^2	Spain—AFINOS Study subsample

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Table 1. (continued)

Authors (Year)	N	Females (n)	Males (n)	Average Age (Years)	Race/ Ethnicity	Average Weight Status	Location
Moliner-Urdiales et al (2010)[38]	363	180	183	Males: 14.7 Females: 14.8	European	All: 21.2 kg/m^2	Spain—HELENA Study subsample
Moliner-Urdiales et al (2009)[39]	365	182	183	14.8	European	All: 21.2 kg/m^2	Spain—HELENA Study subsample
Moore et al. (2013)[19]	285	160	125	Middle school	%NHW 30% %AA 49% %Other 21%	All %NW 52% %OWOB 48%	United States (3 middle schools in North Carolina)
Ortega et al (2010)[40]	9 y: 557 15 y: 518	9 y: 288 15 y: 280	9 y: 269 15 y: 238	9.5 15.6	European	9 y males: 17.2 kg/m^2 9 y females: 17.3 kg/m^2 15 y males: 20.7 kg/m^2 15 y females: 21.2 kg/m^2	Sweden—EYHS subsample
Ortega et al (2007)[41]	9 y: 557 15 y: 517	9 y: 288 15 y: 279	9 y: 269 15 y: 238	9.5 15.6	European	9 y males: 17.2 kg/m^2 9 y females: 17.3 kg/m^2 15 y males: 20.7 kg/m^2 15 y females: 21.2 kg/m^2	Sweden—EYHS
Patrick et al (2004)[20]	878	471	407	12.7	A/PI: 3.4% AA: 6.6% NA: 0.7% Hispanic: 13.1% NHW: 57.1% Other: 18.3%	All: 23.6 kg/m^2 %OW: 18.1% %OB: 27.6%	United States—PACE+ study subsample (RCT 1 y intervention study in California)
Radtke et al (2013)[42]	52	28	24	14.5	European	All: 19.8 kg/m^2	Switzerland (1 elementary school)
Rizzo et al (2008)[43]	613	352	261	15.5	European	All BMI: 20.5 kg/m^2 %NW 90% %OW 9% %OB 1%	Sweden, Estonia—EYHS subsample

(continued)

Table 1. (continued)

Authors (Year)	N	Females (n)	Males (n)	Average Age (Years)	Race/ Ethnicity	Average Weight Status	Location
Rowlands et al (2006)[44]	76	38	38	Males: 9.1 Females: 9.0	European	Males: 18.0 kg/m^2 Females: 17.5 kg/m^2	Wales (8 schools)
Ruiz et al (2006)[45]	780	401	379	9.5	European	All: 16.9 kg/m^2	Sweden, Estonia—EYHS subsample
Steele et al (2009)[46]	1862	1042	820	Males: 10.2 Females: 10.3	European Non-white: Males: 3.8% Females: 4%	Males: 17.9 kg/m^2 %OW: 15.9% %OB 3.9% Females: 18.4 kg/m^2 %OW: 19.4% %OB 6.0%	United Kingdom—Norfolk–SPEEDY study (92 rural and urban primary schools)
Tanha et al (2011)[47]	223	100	123	9.8	European	Males: 17.4 kg/m^2 Females: 17.5 kg/m^2	Sweden (4 schools, grades 3-4)
Treuth et al (2005)[21(p200)]	229	130	99	ES: Males: 9.3 Females: 9.2 MS: Males: 12.3 Females: 11.8 HS: Males: 15.9 Females: 15.4	%White: Males: 71% Females: 74% %AA: Males: 29% Females: 24%	ES: Males: 19.98 kg/m^2 Females: 19.81 kg/m^2 MS: Males: 21.79 kg/m^2 Females: 23.02 kg/m^2 HS: Males: 22.59 kg/m^2 Females: 24.23 kg/m^2	United States (1 elementary school and 1 combined middle/high school in Maryland)
Wittmeier et al (2008)[24]	251	121	130	10	NR	All: 18.5 kg/m^2 %OWOB: 29.5%	Canada (4 schools, grades 3-5)

Abbreviations: y, year; N, population size; n, sample size; NR, not reported; OW, overweight; OB, obese; RCT, randomized control trial; NW, normal weight; BMI, body mass index; PA, physical activity; CM, cardiometabolic; %BF, percent body fat; SGA, small for gestational age; NHW, non-Hispanic white; NHB, non-Hispanic black; CRF, cardiorespiratory fitness; UH, unhealthy; H, healthy; A/PA, Asian/Pacific Islander; AA, African American; NA, Native American; ES, elementary school; MS, middle school; HS, high school.
Cohorts: QUALITY, Quebec Adiposity and Lifestyle Investigation in Youth; EYHS, European Youth Heart Study; HELENA, Healthy Lifestyle in Europe by Nutrition in Adolescence; IDEFICS, Identification and prevention of Dietary- and lifestyle-induced health EFfects In Children and infantS; AFINOS, La Actividad Fi´sica como Agente Preventivo del Desarrollo de Sobrepeso, Obesidad, Alergias, Infecciones y Factores de Riesgo Cardiovascular en Adolescentes: Physical Activity as a Preventive Agent of the Development of Overweight, Obesity, Infections, Allergies and Factors of Cardiovascular Risk in Adolescents; PACE+, Patient-Centered Assessment and Counseling for Exercise Plus Nutrition Project; SPEEDY, Sport, Physical Activity and Eating Behaviour, Environmental Determinants in Young People;

Table 2.

Reported Relationships Between Moderate Physical Activity, Moderate-to-Vigorous Physical Activity, and Vigorous Physical Activity and Measures of Body Composition, Cardiorespiratory Fitness, and Cardiometabolic Biomarkers in Youth.

Author (Year)	Sample Size and Characteristics	BMI/Body Composition			CRF			CM Biomarkers		
		MPA	MVPA	VPA	MPA	MVPA	VPA	MPA	MVPA	VPA
Aires (2010)	n = 111 Age: 11-18 y Sex: Both	Logistic regression OR: 1.01 (0.98-1.05)	Logistic regression OR: 1.00 (0.98-1.02)	Logistic regression OR: 0.63 (0.33-1.21)	—	—	—	—	—	—
Alhassan (2008)	n = 261 Age: 8-10 y Sex: Females Race/Ethnicity: AA	Partial correlation BMI: N/R	Partial correlation BMI: $r_s = 0.18$	Partial correlation BMI: $r_s = -0.28$***	—	—	—	Partial correlation Insulin: $r_s = -0.23$***	Partial correlation SBP: −0.04 (ns) TC: −0.01(ns) LDL: 0.007 (ns) HDL: −0.01 (ns) TG: −0.04 (ns) Insulin: $r_s = -0.21$** Glucose: −0.05 (ns)	Partial correlation Insulin: −0.13 (ns)
Blaes (2011)	n = 187 Age: 6-12 y Sex: Both	Multivariate stepwise regression[a] Males: %BF β = 0.02 (ns) (min of VPA) Females: %BF: N/R because correlations were nonsignificant	—	Multivariate stepwise regression[a] Males: %BF: β = −0.29* (min of VPA) β = −0.05** (bout of VPA ≤15 sec) Females: %BF: N/R because correlations were nonsignificant	—	—	—	—	—	—

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Table 2. (continued)

Author (Year)	Sample Size and Characteristics	BMI/Body Composition			CRF			CM Biomarkers		
		MPA	MVPA	VPA	MPA	MVPA	VPA	MPA	MVPA	VPA
Butte (2007) VIVA LA FAMILIA Study	n = 897 Age: 4-19 y Sex: Both Race/Ethnicity: Hispanic	Partial correlation %BF: $r = -0.07$* WC: Model 1: $r = -0.05$ (ns) Model 2: $r = -0.01$ (ns) GEE models[a] %BF $\beta = -0.13$*	Partial correlation 5-minute bout accumulated minutes %BF: $r = -0.10$** WC: Model 1: $r = -0.07$* Model 2: $r = -0.03$ (ns) GEE models[a] %BF: $\beta = -0.001$** Partial correlation 10-minute bout accumulated minutes %BF: $r = -0.10$** WC: Model 1: $r = -0.06$ (ns) Model 2: $r = -0.01$ (ns) GEE models %BF: $\beta = -0.001$**	Partial correlation %BF: $r = -0.03$ (ns) WC: Model 1: $r = -0.02$ (ns) Model 2: $r = -0.01$ (ns)	Partial correlation VO_2peak: Model 1: $r = 0.09$* Model 2: $r = 0.06$ (ns)	Partial correlation 5-minute bout accumulated minutes VO_2peak: Model 1: $r = 0.16$*** Model 2: $r = 0.12$** GEE models[a] %BF: $\beta = 0.02$** Partial correlation 10-minute bout accumulated minutes VO_2peak: Model 1: $r = 0.16$*** Model 2: $r = 0.12$** GEE models[a] %BF: $\beta = 0.03$*	Partial correlation VO_2peak: Model 1: $r = 0.11$** Model 2: $r = 0.10$**	Partial correlation Insulin: Model 1: $r = -0.07$* Model 2: $r = -0.05$ (ns)	Partial correlation 5-minute bout accumulated minutes Insulin: Model 1: $r = -0.08$ (ns) Model 2: $r = -0.06$ (ns) GEE models[a] $\beta = -0.02$** Partial correlation 10-minute bout accumulated minutes Insulin: Model 1: $r = -0.05$ (ns) Model 2: $r = -0.03$ (ns) GEE models[a] $\beta = -0.04$(ns) Ordinal logistic regression: MetS: OR = 0.94*	Partial correlation Insulin: Model 1: $r = -0.03$(ns) Model 2: $r = -0.03$ (ns)
Carson (2014)	n = 315 Age = 9.15 y Sex: Both	Regression BMI z-score: Q1: reference Q2: $\beta = -0.001$ (ns) Q3: $\beta = 0.05$ (ns) Q4: $\beta = -0.04$ (ns) WC[b]: Q1: reference Q2: $\beta = 0.02$ (ns) Q3: $\beta = 0.002$ (ns) Q4: $\beta = -0.02$ (ns)	—	Regression BMI z-score: Q1: reference Q2: $\beta = -0.13$* Q3: $\beta = -0.15$* Q4: $\beta = -0.10$ (ns) WC[b]: Q1: reference Q2: $\beta = -0.02$* Q3: $\beta = -0.03$* Q4: $\beta = -0.02$ (ns)	Regression VO_2max: Q1: reference Q2: $\beta = -0.30$ (ns) Q3: $\beta = 0.04$ (ns) Q4: $\beta = 0.43$ (ns)	—	Regression VO_2max: Q1: reference Q2: $\beta = 0.81$ (ns) Q3: $\beta = 1.39$ (ns) Q4: $\beta = 1.99$*	Regression SBP: Q1: reference Q2: $\beta = -1.32$ (ns) Q3: $\beta = 1.64$ (ns) Q4: $\beta = -0.90$ (ns)	—	Regression SBP: Q1: reference Q2: $\beta = -1.06$ (ns) Q3: $\beta = -1.63$ (ns) Q4: $\beta = -2.41$ (ns)

(continued)

Table 2. (continued)

Author (Year)	Sample Size and Characteristics	BMI/Body Composition			CRF			CM Biomarkers		
		MPA	MVPA	VPA	MPA	MVPA	VPA	MPA	MVPA	VPA
Chaput (2012) QUALITY Study	n = 550 Age: 8- 10 y Sex: Both Race/Ethnicity: Caucasian	Hierarchichal regression MPA min/d[a]: %BF: Model 1: β = –0.12*** Model 2: β = –0.11*** Model 3: β= –0.06 (ns) Waist/height ratio: Model 1: β = –0.11*** Model 2: β = –0.10*** Model 3: β = –0.10**	Hierarchichal regression MVPA min/d[a]: %BF: Model 1: β = –0.09*** Model 2: β = –0.08*** Model 3: β = –0.05* Waist/height ratio: Model 1: β = –0.08*** Model 2: β = –0.08*** Model 3: β = –0.07***	Hierarchichal regression VPA min/d[a]: %BF: Model 1: β = –0.20*** Model 2: β = –0.19*** Model 3: β = –0.11** Waist/height ratio: Model 1: β = –0.19*** Model 2: β = –0.18*** Model 3: β = –0.16***	—	—	—	—	—	—
Danielsen (2011)	n = 86 Age: 7-13 y Sex: Both	—	—	—	—	—	—	—	—	Hierarchical regression[a] HbA1c: β = 0.21 (ns) IR: β = –0.20 (ns) TC: β = 0.10 (ns) LDL: β = 0.02 (ns) HDL: β = 0.17 (ns) TG: β = –0.19 (ns)
Dencker (2010) Copenhagen School Child Intervention Study	n = 468 Age: 6-7 y Sex: Both	—	—	—	Partial correlation VO_2peak: Males: r = 0.28* Females: r = 0.14* Regression[a] VO_2peak: Males: β = 0.28*** Females: β = 0.15*	Partial correlation VO_2peak: Males: r = 0.27* Females: r = 0.12 Regression[a] VO_2peak: Males: β = 0.26*** Females: N/R (ns)	Partial correlation VO_2peak: Males: r = 0.16* Females: r = 0.07 Regression[a] VO_2peak: Males: β = 0.14* Females: N/R (ns)	—	—	—

(continued)

Table 2. (continued)

Author (Year)	Sample Size and Characteristics	BMI/Body Composition			CRF			CM Biomarkers		
		MPA	MVPA	VPA	MPA	MVPA	VPA	MPA	MVPA	VPA
Dencker (2008)	n = 225 Age: 8-11 y Sex: Both	Partial correlation %BF[b]: $r = -0.09$ (ns) Abdominal fat: $r = -0.07$ (ns) BF distribution: $r = -0.05$ (ns)	Partial correlation %BF[b]: $r = -0.22$* Abdominal fat: $r = -0.18$* BF distribution: $r = -0.12$ (ns)	Partial correlation %BF[b]: $r = -0.40$* Abdominal fat: $r = -0.35$* BF distribution: $r = -0.22$*	Partial correlation VO_2peak: $r = 0.14$*	Partial correlation VO_{2peak}: $r = 0.25$*	Partial correlation VO_2peak: $r = 0.38$*			
Dencker (2008)	n =172 Age: 8-11 y Sex: Both	—	—	Regression[a] TBF: $\beta = -0.011$*** %BF: $\beta = -0.008$*** AFM: $\beta = -0.011$*** AFM/TBF: $\beta = -0.0005$*	—	—	—	—	—	—
Dencker (2007)	n = 248 Age: 8-11 y Sex: Both	—	—	—	—	—	Regression[a] abs VO_2peak: $\beta = 0.09$* VO_2peak/kg of body mass: $\beta = 0.10$* VO_2peak/LBM: $\beta = 0.13$*	—	—	—
Dencker (2006)	n = 248 Age: 8-11 y Sex: Both	N/R (ns)	—	Regression %BF: $r^2 = 0.21$	—	—	—	—	—	—
Denton (2013) HAPPY Study	n = 135 Age:10 -14 y Sex: Both	—	—	—	Regression All: N/R (ns) Males: N/R (ns) Females: $\beta = 0.27$*	—	Regression All: N/R (ns) Males: $\beta = -0.50$* Females: N/R (ns)	—	—	—

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Table 2. (continued)

Author (Year)	Sample Size and Characteristics	BMI/Body Composition			CRF			CM Biomarkers		
		MPA	MVPA	VPA	MPA	MVPA	VPA	MPA	MVPA	VPA
Dubose (2015)	n = 72 Age: 7-11 y Sex: Both	Univariate correlation WC: −0.14 (ns) Regression WC: β = −0.039 (ns)	Univariate correlation WC: −0.15 (ns) Regression WC: β = −0.399 (ns)	Univariate correlation WC: −0.21 (ns) Regression WC: β = −0.333 (ns)	—	—	—	Univariate correlation MAP: −0.19 (ns) HDL: 0.21 (ns) TG: −0.05 (ns) Glucose: 0.05 (ns) MetS score: −0.18 (ns) Regression SBP: β = 0.018 (ns) DBP: β = −0.054* MAP: β = −0.029 (ns) HDL: β = 0.057 (ns) TG: β = −0.175 (ns) Glucose: β = 0.012 (ns) MetS score: β = −0.014 (ns)	Univariate correlation MAP: −0.19 (ns) HDL: 0.17 (ns) TG: −0.03 (ns) Glucose: 0.04 (ns) MetS score: −0.16 (ns) Regression SBP: β = 0.010 (ns) DBP: β = −0.045* MAP: β = −0.027 (ns) HDL: β = 0.042 (ns) TG: β = −0.138 (ns) Glucose: β = 0.012 (ns) MetS score: β = −0.012 (ns)	Univariate correlation MAP: −0.11 (ns) HDL: −0.15 (ns) TG: 0.11 (ns) Glucose: 0.04 (ns) MetS score: −0.01 (ns) Regression SBP: β = −0.213 (ns) DBP: β = −0.008* MAP: β = −0.076 (ns) HDL: β = −0.276 (ns) TG: β = 0.417 (ns) Glucose: β = 0.003 (ns) MetS score: β = 0.0013 (ns)
Ekelund (2007) EYHS	n = 1709 Age: 9-10 y and 15-16 y Sex: Both	Partial correlation WC: *r* = −0.06* Skinfolds: *r* = −0.06** Regression WC: β = 0.006 (ns)	—	Partial correlation WC: *r* = −0.11*** Skinfolds: *r* = −0.06** Regression WC: β = −0.01 (ns)	Partial correlation CRF: *r* = 0.08***	—	Partial correlation CRF: *r* = 0.13***	Partial correlation SBP: *r* = −0.10*** DBP: *r* = −0.08** Glucose: *r* = −0.08** HDL: *r* = 0.2 (ns) TG: *r* = −0.04 (ns) Insulin: *r* = −0.07**	—	Partial correlation SBP: *r* = −0.06* DBP: *r* = −0.08** Glucose: *r* = −0.08** HDL: *r* = −0.01(ns) TG: *r* = −0.04 (ns) Insulin: *r* = −0.07** Regression

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Table 2. (continued)

Author (Year)	Sample Size and Characteristics	BMI/Body Composition			CRF			CM Biomarkers		
		MPA	MVPA	VPA	MPA	MVPA	VPA	MPA	MVPA	VPA
								Regression DBP: β = −0.12*** SBP: β = −0.16*** Glucose: β = −0.11*** HDL: β = 0.02 (ns) TG: β = −0.05* Insulin: β = -0.08** MetS risk score: β = −0.06***		DBP: β = −0.07** SBP: β = −0.07** Glucose: β = −0.10** HDL: β = −0.004 (ns) TG: β = −0.03 (ns) Insulin: β = −0.08** MetS risk score: β = −0.05***
Ekelund (2004) EYHS	n = 1292 Age: 9-10 y Sex: Both	—	Regression BMI: N/R (ns) %BF[a]: β = −0.0019*	Regression BMI: (ns) %BF[a]: β = −0.0034*	—	—	—	—	—	—
España-Romera (2010) HELENA Study	n = 254 Age: 12.5-17.5 y Sex: Both	Regression Low CRF: WC: β = −0.01 (ns) Waist/height ratio: β = 0.01 (ns) High CRF: WC: β = 0.01 (ns) Waist/height ratio: β = 0.02 (ns)	Regression Low CRF: WC: β = −0.07 (ns) Waist/height ratio: β = −0.08 (ns) High CRF: WC: β = −0.06 (ns) Waist/height ratio: β = −0.07 (ns)	Regression Low CRF: WC: β = −0.18* Waist/height ratio: β = −0.20* High CRF: WC: β = −0.15* Waist/height ratio: β = −0.17*	—	—	—	—	—	—
Fairclough (2012)	n = 409 Age::10-11 y Sex: Both	Binomial logistic regression Males: OR_{NW} = 0.94 (95% CI: 0.89-1.0) Females: OR_{NW} = 0.96 (95% CI: 0.90-1.02)	—	Binomial logistic regression Males: OR_{NW} = 1.13 (95% CI: 1.03-1.23) Females: OR_{NW} = 1.13 (95% CI: 1.02-1.25)	—	—	—	—	—	—

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Table 2. (continued)

Author (Year)	Sample Size and Characteristics	BMI/Body Composition			CRF			CM Biomarkers		
		MPA	MVPA	VPA	MPA	MVPA	VPA	MPA	MVPA	VPA
Gaya (2009)	n = 163 Age: 11-17 y Sex: Both	—	—	—	—	—	—	Regression[a] SBP: β = −0.25* DBP: (ns)	—	Regression SBP: (ns) DBP: (ns)
Gutin (2005)	n = 421 Age: 16 y Sex: Both	Regression %BF[c]: β = −0.64 (ns)	Regression %BF[c]: β = −0.16 (ns)	Regression %BF[c]: β = −4.19**	Regression CRF: β = 4.80**	Regression CRF: β = 5.63**	Regression CRF: β = 6.17**	—	—	—
Hay (2012) 2008 Healthy Hearts Prospective Cohort Study of PA and CM Health in Youth	n = 605 Age: 9- 17 y Sex: Both	Regression[a] WC: β = 0.003 (ns) BMI *z*-score: β = 0.04 (ns)	—	Regression[a] WC: β = −0.11 (ns) BMI *z*-score: β = −0.15** Multiple logistic regression Highest VPA to low tertile: OR_{owob}: 0.43 (95% CI: 0.27-0.68)	Regression[a] VO_2max: β = 0.02 (ns)	—	Regression[a] VO_2max: β = 0.17**	Regression[a] SBP: β = 0.04 (ns)	—	Regression SBP: β = −0.19** Highest VPA to low tertile: %high SBP: OR = 0.36 (95% CI: 0.19-0.66)
Jiménez-Pavón (2013)	n = 2200 Age: 14.7 (1.2) y Sex: Both	Regression Males: BMI: β = −0.02 (ns) Skinfolds: β = −0.04 (ns) %BF: β = −0.05 (ns) WC: β = −0.05 (ns) FFM: β = −0.04 (ns) FFM (BIA): β = −0.04 (ns) Females: BMI: β = −0.01 (ns) Skinfolds: β = −0.04 (ns) %BF: β = −0.04 (ns) WC: β = −0.03 (ns) FFM: β = 0.03 (ns) FFM (BIA): β = 0.01 (ns)	Regression Males: BMI: β = −0.11** Skinfolds: β = −0.16*** %BF: β = −0.17*** WC: β = −0.11*** FFM: β = 0.03 (ns) FFM (BIA): β = 0.02 (ns) Females: BMI: β = −0.064* Skinfolds: β = −0.115*** %BF: β = −0.110*** WC: β = −0.092** FFM: β = 0.05 (ns) FFM (BIA): β = 0.03 (ns)	Regression Males: BMI: β = −0.20*** Skinfolds: β = −0.26*** %BF: β = −0.26*** WC: β = −0.18*** FFM: β = 0.10** FFM (BIA): β = 0.06* Females: BMI: β = −0.142*** Skinfolds: β = −0.178*** %BF: β = −0.176*** WC: β = −0.146*** FFM: β = 0.08** FFM (BIA): β = 0.06*	—	—	—	—	—	—

(continued)

Table 2. (continued)

Author (Year)	Sample Size and Characteristics	BMI/Body Composition			CRF			CM Biomarkers		
		MPA	MVPA	VPA	MPA	MVPA	VPA	MPA	MVPA	VPA
Jiménez-Pavón (2013)	n = 2025 Age: 6-9 y Sex: Both	—	—	—	—	—	—	Logistic regression for CVD risk score (only in 6- 9-y-olds) Males: Q1: $OR_{CVDscore}$ = 5.4 (95% CI: 2.05-14.20) Q2: OR = 2.1 (95% CI: 0.76-5.58) Q3: OR = 2.0 (95% CI: 0.75-5.31) Q4: OR = 1.2 (95% CI: 0.42-3.20) Q5: ref (highest total PA) Females: Q1: OR = 3.7 (95% CI: 1.21-11.29) Q2: OR = 4.5 (95% CI: 1.69-11.77) Q3: OR = 5.3 (95% CI: 2.12-13.42) Q4: OR = 1.9 (95% CI: 0.66-5.38) Q5: ref (highest total PA)	Logistic regression for CVD risk score (only in 6-9-y olds) Males: Q1: OR = 4.4 (95% CI: 95% CI: 1.62-11.71) Q2: OR = 2.38 (95% CI: 0.91-6.23) Q3: OR = 2.06 (95% CI: 0.78-5.48) Q4: OR = 1.29 (95% CI: 0.47-3.56) Q5: ref (highest total PA) Females: Q1: OR = 6.0 (95% CI: 1.86-19.05) Q2: OR = 7.1 (95% CI: 2.47-20.15) Q3: OR= 5.23 (95% CI: 1.81-15.13) Q4: OR = 3.5 (95% CI: 1.17-10.26) Q5: ref (highest total PA)	Logistic regression for CVD risk score (only in 6- 9-y olds) Males: Q1: OR = 2.7 (95% CI: 1.06-6.80) Q2: OR = 1.7 (95% CI: 0.66-4.16) Q3: OR = 1.2 (95% CI: 0.44-2.99) Q4: OR = 0.73 (95% CI: 0.26-2.04) Q5: ref (highest total PA) Females: Q1: OR: 5.9 (95% CI: 2.20-15.76) Q2: OR = 2.9 (95% CI: 1.02-7.96) Q3: OR = 1.9 (95% CI: 0.64-5.51) Q4: OR = 1.8 (95% CI: 0.63-5.36) Q5: ref (highest total PA)

(continued)

Table 2. (continued)

Author (Year)	Sample Size and Characteristics	BMI/Body Composition			CRF			CM Biomarkers		
		MPA	MVPA	VPA	MPA	MVPA	VPA	MPA	MVPA	VPA
Jiménez-Pavón (2013) HELENA Study	n = 1053 Age: 12.5-17.5 y Sex: Both	Partial correlations Males: BMI: $r = -0.05$ (ns) WC: $r = 0.03$ (ns) TBF: $r = -0.01$ (ns) Females: BMI: $r = -0.01$ (ns) WC: $r = -0.07$ (ns) TBF: $r = -0.12$**	Partial correlations Males: BMI: $r = -0.04$ (ns) WC: $r = 0.02$ (ns) TBF: $r = -0.10$ (ns) Females: BMI: $r = -0.10$ (ns) WC: $r = -0.12$* TBF: $r = -0.20$**	Partial correlations Males: BMI: $r = -0.02$ (ns) WC: $r = -0.04$ (ns) TBF: $r = -0.19$** Females: BMI: $r = -0.19$** WC: $r = -0.17$** TBF: $r = -0.26$**	Partial correlations Males: CRF: 0.13** Females: CRF: −0.03 (ns)	Partial correlations Males: CRF: 0.17** Females: CRF: 0.06 (ns)	Partial correlations Males: CRF: 0.20** Females: CRF: 0.19**	Partial correlations Males: Insulin: $r = 0.05$ (ns) INS: $r = -0.04$ IR: $r = 0.06$ (ns) Glucose: $r = 0.01$ (ns) Females: Insulin: $r = -0.09$ (ns) INS: $r = 0.11$** IR: $r = 0.09$ (ns) Glucose: $r = -0.07$ Regression Model 3: Males: Insulin: $\beta = 0.07$ (ns) INS: $\beta = -0.06$ (ns) IR: $\beta = 0.08$ (ns) Stratified by CRF: N/R (ns) Females: Insulin: $\beta = -0.08$ (ns) INS: $\beta = 0.08$ (ns) IR: $\beta = -0.08$ (ns) Stratified by CRF: Low CRF Insulin: $\beta = -0.2$* INS: $\beta = 0.24$** IR: $\beta = -0.20$* Middle to high CRF Insulin: $\beta = -0.07$ (ns) INS: $\beta = 0.07$ (ns) IR: $\beta = -0.08$ (ns)	Partial correlations Males: Insulin: $r = 0.04$ (ns) INS: $r = 0.05$ (ns) IR: $r = -0.03$ (ns) Glucose: $r = 0.02$ (ns) Females: Insulin: $r = -0.14$* INS: $r = 0.15$** IR: $r = -0.12$* Glucose: $r = -0.04$ (ns) Regression Model 3: Males: Insulin: $\beta = -0.02$ (ns) INS: $\beta = 0.02$ (ns) IR: $\beta = -0.02$ (ns) Stratified by CRF: N/R (ns) Females: Insulin: β= -0.06 (ns) INS: $\beta = 0.06$ (ns) IR: $\beta = -0.07$ (ns) Stratified by CRF: Low CRF Insulin: $\beta = -0.22$* INS: $\beta = 0.26$** IR: $\beta = -0.21$* Middle to high CRF Insulin: $\beta = -0.08$ (ns) INS: $\beta = 0.08$ (ns) IR: $\beta = -0.08$ (ns)	Partial correlations Males: Insulin: $r = 0.12$* INS: $r = 0.13$** IR: $r = -0.18$* Glucose: $r = -0.04$ (ns) Females: Insulin: $r = 0.15$** INS: $r = 0.16$** IR: $r = -0.13$* Glucose: $r = -0.01$ (ns) Regression Model 3: Males: Insulin: $\beta = -0.11$ (ns) INS: $\beta = 0.11$ (ns) IR: $\beta = -0.11$ (ns) Stratified by CRF: N/R (ns) Females: Insulin: $\beta = -0.001$ (ns) INS: $\beta = -0.002$ (ns) IR: $\beta = -0.003$ (ns) Stratified by CRF: Low CRF Insulin: $\beta = -0.17$* INS: $\beta = 0.21$* IR: $\beta = -0.16$* Middle to high CRF Insulin: $\beta = -0.05$ (ns) INS: $\beta = 0.04$ (ns) IR: $\beta = -0.04$ (ns)

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Table 2. (continued)

Author (Year)	Sample Size and Characteristics	BMI/Body Composition			CRF			CM Biomarkers		
		MPA	MVPA	VPA	MPA	MVPA	VPA	MPA	MVPA	VPA
Katzmarzyk (2015)	n = 6539 Age: 9-11 y Sex: Both	—	Odds ratio Obesity: 0.49* ROC analysis Obesity thresholds: All: 55 min/d* Boys: 65 min/d* Girls: 49 min/d*	Odds ratio Obesity: 0.41* ROC analysis Obesity thresholds: All: 14 min/d* Boys: 20 min/d* Girls: 11 min/d*	—	—	—	—	—	—
Kennedy (2012)	n = 36 Age: 6.7 y Sex: Both	Partial correlations Minutes /day: BMI *z*-score: *r* = 0.06 (ns) FMI *z*-score: *r* = −0.28 (ns) LMI *z*-score: *r* = 0.40* %Time in MPA: N/R	Partial correlations Minutes/day: N/R %Time in MVPA: BMI *z*-score: *r* = −0.07 (ns) FMI *z*-score: *r* = −0.26 (ns) LMI *z*-score: *r* = 0.35*	Partial correlations Minutes/day: BMI *z*-score: *r* = 0.06 (ns) FMI *z*-score: *r* = −0.02 (ns) LMI *z*-score: *r* = 0.18 (ns) %Time in VPA: N/R	—	—	—	—	—	—
Lätt (2015)	n = 136 Age: 10-12 y at baseline Sex: Males	Partial correlation BMI: *r* = −0.152 (ns) ROC analysis NW vs OW: 46 min/d (ns) NW vs OB: 46 min/d (ns)	Partial correlation BMI: *r* = −0.260** ROC analysis NW vs OW: 59 min/d* NW vs OB: 55 min/d**	Partial correlation BMI: *r* = −0.317*** ROC analysis NW vs OW: 14 min/d** NW vs OB: 10 min/d***	—	—	—	—	—	—
Mark (2011) 2003-2004 NHANES	n = 1165 Age: 8-17 y Sex: Both	—	Regression Model 3: Trunk fat %tile (Q1 reference)[a]: Low activity Q2: β = −6.97 (ns) Q3: β = −9.70 (ns) Q4 (low): β = −16.18 (ns) Q4 (high): β = −22.57* P_{trend} = .029	Regression Model 3: Trunk fat %tile (Q1 reference)[a]: Low activity Q2: β = −4.34 (ns) Q3: β = −6.57 (ns) Q4 (low): β = −6.92 (ns) Q4 (high): β = −6.28 (ns) P_{trend} = .41	—	—	—	—	—	—

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Table 2. (continued)

Author (Year)	Sample Size and Characteristics	BMI/Body Composition			CRF			CM Biomarkers		
		MPA	MVPA	VPA	MPA	MVPA	VPA	MPA	MVPA	VPA
Martínez-Gómez (2010) AFINOS Study	n = 192 Age: 13-17 y Sex: Both	Partial correlations BMI: $r = -0.004$ (ns) SF: $r = 0.07$ (ns) WC: $r = 0.05$ (ns)	Partial correlations BMI: $r = -0.01$ (ns) SF: $r = -0.01$ (ns) WC: $r = 0.03$ (ns)	Partial correlations BMI: $r = -0.11$ (ns) SF: $r = -0.27$*** WC: $r = -0.08$ (ns)	Partial correlation CRF: $r = 0.08$ (ns)	Partial correlation CRF: $r = 0.25$***	Partial correlation CRF: $r = 0.48$***	Partial correlations Glucose: $r = 0.04$ (ns) Insulin: $r = 0.04$ (ns) IR: $r = 0.04$ (ns)	Partial correlations Glucose: $r = 0.01$ (ns) Insulin: $r = 0.02$ (ns) IR: $r = 0.02$ (ns)	Partial correlations Glucose: $r = -0.04$ (ns) Insulin: $r = -0.04$ (ns) IR: $r = -0.05$ (ns)
Martínez-Gómez (2010) HELENA Study	n =1808 Age: 12.5-17.5 y Sex: Both	—	—	—	Logistic regression All: $OR_{healthy\ CRF} = 1.71$*** (95% CI: 1.39-2.12) Males: $OR_{healthy\ CRF} = 1.81$*** (95% CI: 1.31-2.49) Females: $OR_{healthy\ CRF} = 1.68$*** (95% CI: 1.28-2.21)	Logistic regression All: $OR_{healthy\ CRF} = 1.96$*** (95% CI: 1.58-2.43) Males: $OR_{healthy\ CRF} = 2.13$*** (95% CI: 1.54-2.94) Females: $OR_{healthy\ CRF} = 1.90$*** (95% CI: 1.45-2.51)	Logistic regression All: $OR_{healthy\ CRF} = 2.20$*** (95% CI: 1.74-2.71) Males: $OR_{healthy\ CRF} = 2.30$*** (95% CI: 1.64-3.14) Females: $OR_{healthy\ CRF} = 2.32$*** (95% CI: 1.75-3.06)	—	—	—
Martínez-Gómez (2009) AFINOS Study	n = 202 Age: 13-17 y Sex: Both	Partial correlations BMI: $r = 0.02$ (ns) %BF: $r = -0.01$ (ns) WC: $r = 0.03$ (ns)	Partial correlations BMI: $r = 0.02$ (ns) %BF: $r = -0.08$ (ns) WC: $r = 0.01$ (ns)	Partial correlations BMI: $r = -0.03$ (ns) %BF: $r = -0.29$*** WC: $r = -0.06$ (ns)	Partial correlation CRF: N/R (ns)	Partial correlation CRF: $r = 0.280$***	Partial correlation CRF: $r = 0.484$***	Partial correlations SBP: $r = 0.03$ (ns) DBP: $r = 0.05$ (ns) TC: $r = 0.06$ (ns) HDL: $r = 0.08$ (ns) TG: $r = -0.05$ (ns) Glucose: $r = -0.01$ (ns) Insulin: $r = -0.02$ (ns)	Partial correlations SBP: $r = 0.06$ (ns) DBP: $r = -0.05$ (ns) TC: $r = 0.08$ (ns) HDL: $r = 0.11$ (ns) TG: $r = -0.11$ (ns) Glucose: $r = -0.02$ (ns) Insulin: −0.03 (ns) Regression MetS: CCCHS: $\beta = -0.08$ (ns) ACLS: $\beta = -0.09$ (ns) EYHS: $\beta = -0.11$ (ns)	Partial correlations SBP: $r = 0.04$ (ns) DBP: $r = -0.05$ (ns) TC: $r = 0.05$ (ns) HDL: $r = 0.18$* TG: $r = -0.19$** Glucose: $r = -0.06$ (ns) Insulin: $r = -0.09$ (ns) Regression MetS:

(continued)

Table 2. (continued)

Author (Year)	Sample Size and Characteristics	BMI/Body Composition			CRF			CM Biomarkers		
		MPA	MVPA	VPA	MPA	MVPA	VPA	MPA	MVPA	VPA
								Regression MetS: CCCHS: β = −0.04 (ns) ACLS: β = −0.08 (ns) EYHS: β = −0.05 (ns)		CCCHS: β = −0.19* ACLS: β = −0.16* EYHS: β = −0.24* MetS (additionally controlled for fitness): CCCHS: β = −0.03 (ns) ACLS: β = −0.03 (ns) EYHS: β = −0.09 (ns)
Moliner-Urdiales (2010) HELENA Study	n = 363 Age: 12.5-17.5 y Sex: Both	Regression[a] FFM (DXA) [a] Males: β = 0.11 (ns) Females: β = −0.01 (ns)	Regression[a] FFM(DXA)[a] Males: β = 0.08 (ns) Females: β = 0.02 (ns)	Regression[a] FFM(DXA)[a] Males: β = 0.04 (ns) Females: β = 0.05 (ns)	—	—	—	—	—	—
Moliner-Urdiales (2009) HELENA Study	n = 365 Age: 12.5-17.5 y Sex: Both	Regression[a] TBF(DXA): β = −0.10 (ns) TBF(BodPod): β = −0.06 (ns) SF: β = −0.09 (ns) WC: β = −0.03 (ns)	Regression[a] TBF(DXA): β = −0.20*** TBF(BodPod): β = −0.14** SF: β = −0.19*** WC: β = −0.10 (ns)	Regression[a] TBF(DXA): β = −0.26*** TBF(BodPod): β = −0.22*** SF: β = −0.24*** WC: β = −0.17**	—	—	—	—	—	—
Moore (2013)	n = 285 Age: Middle school Sex: Both	—	—	—	Regression Model 4 CRF: β = −0.03 (ns)	—	Regression Model 4 CRF: β = −0.22*	—	—	—
Ortega (2010) EYHS	n = 1075 Age: 9-10 y and 15-16 y Sex: Both	Regression All: β = 0.13** Low CRF: β = −0.002 (ns) High CRF: β = 0.27***	Regression All: β = 0.10* Low CRF: β = −0.10 (ns) High CRF: β = 0.23**	Regression All: β = 0.01 (ns) Low CRF: β = −0.09 (ns) High CRF: β = 0.10*	—	—	—	—	—	—

(continued)

Table 2. (continued)

Author (Year)	Sample Size and Characteristics	BMI/Body Composition			CRF			CM Biomarkers		
		MPA	MVPA	VPA	MPA	MVPA	VPA	MPA	MVPA	VPA
Ortega (2007) EYHS	n = 1074 Age: 9.5 (0.3) y and 15.6 (0.4) y Sex: Both	Logistic regression Overweight: Ref = High PA Low PA: OR_{OW} = 2.0 (95% CI: 0.9-4.4) Middle PA: OR_{OW} = 1.7 (95% CI: 0.7-3.7) High WC: Ref = High PA Low PA: OR_{Hi_WC} = 1.3 (95% CI: 0.7-2.4) Middle PA: OR_{Hi_WC} = 1.1 (95% CI: 0.6-2.0)	Logistic regression Overweight: Ref = High PA Low PA: OR_{OW} = 2.1 (95% CI: 0.9-4.3) Middle PA: OR_{OW} = 1.0 (95% CI: 0.5-2.3) High WC: Ref = High PA Low PA: OR_{Hi_WC} = 1.3 (95% CI: 0.6-2.8) Middle to high PA level: OR_{Hi_WC} = 0.6 (95% CI: 0.3-1.5)	Logistic regression Overweight: Ref = High PA Low to high PA level: OR_{OW} = 4.1*** (95% CI: 1.8-9.5) Middle to high PA level: OR_{OW} = 1.9 (95% CI: 0.8-4.7) High WC: Ref = High PA Low PA: OR_{Hi_WC} = 2.1* (95% CI: 1.1-3.9) Middle PA: OR_{Hi_WC} = 1.4 (95% CI: 0.8-2.7)	—	—	—	—	—	—
Patrick (2004) PACE+ Study	n = 878 Age: 11-15 y Sex: Both	Logistic regression Males: N/R (ns) Females: N/R (ns)	—	Logistic regression Males: $OR_{at\ risk\ plus\ OW}$ = 0.92*** (95% CI: 0.89-0.95) Females: $OR_{at\ risk\ plus\ OW}$ = 0.93** (95% CI: 0.89-0.97)	—	—	—	—	—	—
Radtke (2013)	n = 52 Age: 14.5 (0.7) y Sex: Both	—	—	—	—	Regression Maximum power output: β = 0.01 (ns) $MVPA_{low\ vs\ high}$ = P > .05 VO_2peak: β = 0.06 (ns) $MVPA_{low\_tertile\ vs\ high\_tertile}$ = P > .05	Regression Maximum power output: β = 0.25* $VPA_{low\ vs\ high}$: P = .01 VO_2peak: β = 0.12 (ns) $VPA_{low\_tertile\ vs\ high\_tertile}$ = P > .05	—	—	—

(continued)

Table 2. (continued)

Author (Year)	Sample Size and Characteristics	BMI/Body Composition			CRF			CM Biomarkers		
		MPA	MVPA	VPA	MPA	MVPA	VPA	MPA	MVPA	VPA
Rizzo (2008) EYHS	n = 613 Age: 15.5 y Sex: Both	—	—	—	—	—	—	Regression Glucose: β = −0.09* Insulin: β = −0.11** IR: β = −0.11** $MPA_{low\_tertile\ vs\ high\_tertile} = P = .02$ $MPA_{low\ vs.\ middle} = P > .05$	Regression Glucose: β = −0.12** Insulin: β = −0.14*** IR: β = −0.14***	Regression Glucose: β = −0.15*** Insulin: β = −0.16*** IR: β = −0.16*** $VPA_{low\_tertile\ vs\ high\_tertile} = P = .02$ $VPA_{low\ vs\ middle} = P = .05$
Rowlands (2006)	n = 76 Age: 8-11 y Sex: Both	—	Partial correlation Males: $MVPA_{low\_tertile\ vs\ upper\ two\ tertiles} = P > .05$ Females: $MVPA_{low\_tertile\ vs\ high\_tertiles} = P < .05$	Partial correlation Males: $r = -0.22$ (ns) $VPA_{low\_tertile\ vs\ upper\ two\ tertiles} = P < .05$ Females: $r = -0.28$ (ns) $VPA_{low\_tertile\ vs\ upper\ two\ tertiles} = P > .05$	—	—	—	—	—	—
Ruiz (2006) EYHS	n = 780 Age: 9-10 y Sex: Both	Regression %BF: β = 0.018 (ns)	Regression %BF: β = −0.011 (ns)	Regression %BF: β = −0.081* Time spent in VPA $VPA_{2nd\_quintile\ vs\ 5th\_quintile} = P < .001$	Regression CRF: β = 0.087**	Regression CRF: β = 0.108**	Regression CRF: β = 0.124*** Time spent in VPA $VPA_{1st\_quintile\ vs.\ 5th\_quintile} = P < .001$ $VPA_{2nd\_quintile\ vs\ 4th\_quintile} = P = .018$	—	—	—

(continued)

Table 2. (continued)

Author (Year)	Sample Size and Characteristics	BMI/Body Composition			CRF			CM Biomarkers		
		MPA	MVPA	VPA	MPA	MVPA	VPA	MPA	MVPA	VPA
Steele (2009) SPEEDY Study	n = 1862 Age: 9-10 y Sex: Both	Regression WC:[a] Model 1: β = −0.048** Model 2: β = −0.039* Model 3: β = −0.034 (ns) FMI[b]: Model 1: β = −0.002** Model 2: β = −0.002* Model 3: β = −0.002 (ns) BMI: Model 1: β = −0.003 (ns) Model 2: β = −0.001 (ns) Model 3: β = −0.0004 (ns) Standardized coefficients: WC: β = −0.49(ns) FMI: β = −0.022(ns) BMI: β = −0.006(ns)	Regression WC:[a] Model 1: β = −0.053*** Model 2: β = −0.042*** Model 3: β = −0.044*** FMI[b]: Model 1: β = −0.003*** Model 2: β = −0.002*** Model 3: β = −0.002*** BMI: Model 1: β = −0.005*** Model 2: β = −0.00** Model 3: β = −0.003** Standardized coefficients: WC: β = −1.09*** FMI: β = −0.059*** BMI: β = −0.094** Time spent in MVPA Odds of OWOB OR_{OWOB} $MVPA_{\text{least active quartile vs. most active quintile}} = P_{trend} < .01$	Regression WC: [a] Model 1: β = −0.12*** Model 2: β = −0.098*** Model 3: β = −0.10*** FMI[b]: Model 1: β = −0.006*** Model 2: β = −0.005*** Model 3: β = −0.06*** BMI: Model 1: β = −0.013*** Model 2: β = −0.01*** Model 3: β = −0.01*** Standardized coefficients: WC: β = −1.32*** FMI: β = −0.075*** BMI: β = −0.15*** Time spent in VPA WC and FMI $VPA_{\text{least active quartile vs. most active quintile}} = P_{trend} < .001$	—	—	—	—	—	—

(continued)

Table 2. (continued)

Author (Year)	Sample Size and Characteristics	BMI/Body Composition			CRF			CM Biomarkers		
		MPA	MVPA	VPA	MPA	MVPA	VPA	MPA	MVPA	VPA
Tanha (2011)	n = 223 Age: 7.9-11.1 y Sex: Both	—	GLM-derived partial correlation %BF: $r = -0.32$*** AFM: $r = -0.29$*** AFM/TBF: $r = -0.21$**	GLM-derived partial correlation %BF: $r = -0.38$*** AFM: $r = -0.34$*** AFM/TBF: $r = -0.23$***	—	GLM-derived partial correlation VO_2peak: $r = 0.32$***	GLM-derived partial correlation VO_2peak: $r = 0.28$***	—	GLM-derived partial correlation SBP: $r = -0.10$ (ns) DBP: $r = -0.12$ (ns) MAP: $r = -0.13$ (ns) CVD risk score: Males: $r = -0.29$** Females: $r = -0.28$**	GLM-derived partial correlation SBP: $r = -0.14$* DBP: $r = -0.08$ (ns) MAP: $r = -0.12$ (ns) CVD risk score: Males: $r = -0.33$*** Females: $r = -0.32$**
Treuth (2005)	n = 229 Age: 7-19 y Sex: Both	Partial correlation Males: ES: BMI: $r_s = 0.14$ (ns) TBF: $r_s = -0.05$ (ns) %BF: $r_s = -0.02$ (ns) MS: BMI: $r_s = 0.02$ (ns) TBF: $r_s = -0.07$ (ns) %BF: $r_s = -0.04$ (ns) HS: BMI: $r_s = 0.01$ (ns) TBF: $r_s = 0.02$ (ns) %BF: $r_s = -0.01$ (ns) Females: ES: BMI: $r_s = 0.14$ (ns) TBF: $r_s = -0.07$ (ns) %BF: $r_s = -0.04$ (ns) MS: BMI: $r_s = 0.10$ (ns) TBF: $r_s = -0.07$ (ns) %BF: $r_s = -0.08$ (ns) HS:	—	Partial correlation Males: ES: BMI: $r_s = 0.12$ (ns) TBF: $r_s = 0.01$ (ns) %BF: $r_s = 0.01$ (ns) MS: BMI: $r_s = -0.06$ (ns) TBF: $r_s = -0.17$ (ns) %BF: $r_s = -0.11$ (ns) HS: BMI: $r_s = -0.21$ (ns) TBF: $r_s = -0.16$ (ns) %BF: $r_s = -0.17$ (ns) Females: ES: BMI: $r_s = 0.20$ (ns) TBF: $r_s = 0.01$ (ns) %BF: $r_s = 0.04$ (ns) MS: BMI: $r_s = 0.30$* TBF: $r_s = 0.21$ (ns) %BF: $r_s = 0.18$ (ns)	—	—	—	—	—	—

(continued)

Table 2. (continued)

Author (Year)	Sample Size and Characteristics	BMI/Body Composition			CRF			CM Biomarkers		
		MPA	MVPA	VPA	MPA	MVPA	VPA	MPA	MVPA	VPA
		BMI: r_s = 0.04 (ns) TBF: r_s = –0.13 (ns) %BF: r_s = –0.14 (ns)		HS: BMI: r_s = 0.02 (ns) TBF: r_s = –0.11 (ns) %BF: r_s = –0.12 (ns)						
Wittmeier (2008)	n = 251 Age: 8-10 y Sex: Both	Partial correlation BMI: r = –0.076 (ns) %BF: r = –0.113 (ns) Time spent in MPA (Q1-Q4) %BF $Q1_{(<15min)}$ vs Q3 and $Q4_{(>30-45min)}$: P = .009** BMI $Q1_{(<15min)}$ vs $Q4_{(>45min)}$: P = .016* Regression[a] N/R (ns) Logistic regression BMI OW ≤45 min vs >45 $min_{(ref)}$: OR_{BMI_OW} = 1.52 (ns) ≤30 min vs 45 $min_{(ref)}$: OR_{BMI_OW} = 1.49 (ns) ≤15 min vs 45 $min_{(ref)}$: OR_{BMI_OW} = 2.04 (ns) %BF (≥20%) ≤45 min vs >45 $min_{(ref)}$: OR_{OW} = 3.15***	Partial correlation BMI: r = –0.123 (ns) %BF: r = –0.143* Logistic regression ≤60 min vs >60 $min_{(ref)}$: OR_{BMI_OW} = 1.96 (ns) ≤30 min vs 60 $min_{(ref)}$: OR_{BMI_OW} = 2.08 (ns) %BF (≥20%) ≤60 min vs >60 $min_{(ref)}$: OR_{OW} = 3.36*** ≤30 min vs 60 $min_{(ref)}$: Logistic regression ≤60 min vs >60 $min_{(ref)}$: OR_{BMI_OW} = 1.96 (ns) ≤30 min vs 60 $min_{(ref)}$: OR_{BMI_OW} = 2.08 (ns)	Partial correlation BMI: r = –0.197** %BF: r = –0.173** Time spent in VPA (Q1-Q4) %BF $Q1_{(<5min)}$ vs Q3 and $Q4_{(>10-15min)}$: P = .002** BMI $Q1_{(<5min)}$ vs Q4 $_{(>15min)}$: P = .011* Regression[a] BMI: β = –0.21*** %BF: β = –0.17** Logistic regression ≤15 min vs >15 $min_{(ref)}$: OR_{BMI_OW} = 4.45** ≤10 min vs >15 $min_{(ref)}$: OR_{BMI_OW} = 4.63** ≤5 min vs >15 $min_{(ref)}$: OR_{BMI_OW} = 5.21** %BF (≥20%) ≤15 min vs >15 $min_{(ref)}$: OR_{OW} = 3.23** ≤10 min vs >15 $min_{(ref)}$:					

(continued)

Table 2. (continued)

Author (Year)	Sample Size and Characteristics	BMI/Body Composition			CRF			CM Biomarkers		
		MPA	MVPA	VPA	MPA	MVPA	VPA	MPA	MVPA	VPA
		≤30 min vs 45 $min_{(ref)}$: OR_{OW} = 3.51*** ≤15 min vs 45 $min_{(ref)}$: OR_{OW} = 4.20*** %BF (≥25%) ≤45 min vs >45 $min_{(ref)}$: OR_{OW} = 1.97 (ns) ≤30 min vs 45 $min_{(ref)}$: OR_{OW} = 2.16* ≤15 min vs 45 $min_{(ref)}$: OR_{OW} = 3.04**	%BF (≥20%) ≤60 min vs >60 $min_{(ref)}$: OR_{OW} = 3.36*** ≤30 min vs 60 $min_{(ref)}$: OR_{OW} = 4.15*** %BF (≥25%) ≤60 min vs >60 $min_{(ref)}$: OR_{OW} = 2.71* ≤30 min vs 60 $min_{(ref)}$: OR_{OW} = 3.03**	OR_{OW} = 3.26** ≤5 min vs >15 $min_{(ref)}$: OR_{OW} = 4.03*** %BF (≥25%) ≤15 min vs >15 $min_{(ref)}$: OR_{OW} = 2.23 (ns) ≤10 min vs >15 $min_{(ref)}$: OR_{OW} = 2.58* ≤5 min vs >15 $min_{(ref)}$: OR_{OW} = 2.90*	—	—	—	—	—	—

Abbreviations: PA, physical activity; BMI, body mass index; CRF, cardiorespiratory fitness; CM, cardiometabolic; y, year; MPA, moderate physical activity; MVPA, moderate to vigorous physical activity; VPA, vigorous physical activity; n, sample size; OR, odds ratio; AA, African-American; N/R, not reported; r, Pearson partial correlation; SBP, systolic blood pressure; ns, nonsignificant; TC, total cholesterol; LDL, low-density lipoprotein; HDL, high-density lipoprotein; TG, triglycerides; %BF, body fat percentage; β, standardized regression coefficient; min, minute; sec, second; WC, waist circumference; GEE, generalized estimating equation; VO2peak, peak volume of oxygen consumption; MetS, metabolic syndrome; min/d, minutes per day; HbA1c, glycated hemoglobin; IR, insulin resistance; TBF, total body fat; AFM, abdominal fat mass; abs, absolute; kg, kilogram; LBM, lean body mass; DBP, diastolic blood pressure; ow, overweight; ob, obese; VO_2max, maximum volume of oxygen consumption; FFM, fat-free mass; BIA, bioelectrical impedance analysis; CVD, cardiovascular disease; Q, quartile; TBF, total body fat; INS, insulin sensitivity; FMI, fat mass index; LMI, lean mass index; DXA, dual energy x-ray absorptiometry; SF, subcutaneous fat; MAP, mean arterial pressure; ROC, receiver operating characteristic.

Cohorts: GEMS, Stanford Girls Health Enrichment Multisite Studies; QUALITY, Quebec Adiposity and Lifestyle Investigation in Youth; HAPPY, Health and Physical Activity Promotion in Youth; EYHS, European Youth Heart Study; HELENA; Healthy Lifestyle in Europe by Nutrition in Adolescence; NHANES, National Health and Nutrition Examination Survey; AFINOS, La Actividad Fı´sica como Agente Preventivo del Desarrollo de Sobrepeso, Obesidad, Alergias, Infecciones y Factores de Riesgo Cardiovascular en Adolescentes: Physical Activity as a Preventive Agent of the Development of Overweight, Obesity, Infections, Allergies and Factors of Cardiovascular Risk in Adolescents; ACLS, Aerobic Center Longitudinal Study; PACE+, Patient-Centered Assessment and Counseling for Exercise Plus Nutrition Project; SPEEDY, Sport, Physical Activity and Eating Behaviour, Environmental Determinants in Young People

[a]Standardization not reported.

[b]Log-transformed.

[c]Unstandardized.

*$p < 0.05$.

**$p < 0.01$.

***$p < 0.001$.

Table 3.

Summary of Articles Reporting Beneficial, Not Beneficial, or No Association Between Physical Activity Intensity and Body Mass Index/Body Composition Outcomes in Youth.

	Beneficial	Not Beneficial	No Association
Body mass index			
MPA		1/14 (7.1%)[55]	13/14 (92.9%)[21,23-26,34,35,37,41,46,52,54-56]
MVPA	5/13 (38.5%)[7,15,34,46,56]		8/13 (61.5%)[24,26,31,35,37,41,52,54]
VPA	12/19 (63.2%)[7,15,20,23-25,34,35,41,46,55,56]	1/19 (5.3%)[21]	8/19 (42.1%)[21,26,31,35,37,44,52,54]
Fat mass index			
MPA	2/2 (100.0%)[37,46]		
MVPA	2/2 (100.0%)[37,46]		
VPA	2/2 (100.0%)[37,46]		
Total body fat			
MPA	2/5 (40.0%)[18,35]		3/5 (60.0%)[21,35,39]
MVPA	2/2 (100.0%)[35,39]		1/2 (50.0%)[35]
VPA	3/4 (75.0%)[18,35,39]		1/4 (25.0%)[21]
Percent body fat			
MPA	2/11 (18.2%)[16,22]		9/11 (81.8%)[17,21,24,27,30,34,45,49,54]
MVPA	7/10 (70.0%)[16,22,24,34,47,49,55]		3/10 (30.0%)[17,45,54]
VPA	12/15 (80.0%)[17,22,24,27,31,34,45,47,49,51,54,59]		3/15 (20.0%)[16,21,30]
Waist circumference			
MPA	1/13 (7.7%)[46]	1/13 (7.7%)[40]	12/13 (92.3%)[16,23,25,30,32,34,35,39,40,52,54,57]
MVPA	3/10 (30.0%)[34,35,46]	1/10 (10.0%)[40]	8/10 (80.0%)[16,32,35,39,40,52,54,57]
VPA	8/13 (61.5%)[23,25,32,34,35,39,40,46]	1/13 (7.7%)[40]	6/13 (46.2%)[16,30,35,52,54,57]
Waist/height ratio			
MPA	1/2 (50.0%)[22]		1/2 (50.0%)[32]
MVPA	1/2 (50.0%)[22]		1/2 (50.0%)[32]
VPA	2/2 (100.0%)[22,32]		
Subcutaneous fat			
MPA			2/2 (100.0%)[39,52]
MVPA	1/2 (50.0%)[39]		1/2 (50.0%)[52]
VPA	2/2 (100.0%)[39,52]		

(continued)

Table 3. (continued)

	Beneficial	Not Beneficial	No Association
Abdominal fat mass			
MPA			
MVPA	1/1 (100.0%)[47]		
VPA	1/1 (100.0%)[47]		
Fat-free mass			
MPA			1/1 (100.0%)[38]
MVPA			1/1 (100.0%)[38]
VPA			1/1 (100.0%)[38]
Lean mass index			
MPA	1/1 (100.0%)[37]		
MVPA	1/1 (100.0%)[37]		
VPA			

Abbreviations: MPA, moderate physical activity; MVPA, moderate to vigorous physical activity; VPA, vigorous physical activity.

(FMI) and PA intensity. One study found significant, negative connections between the 2 variables for both VPA and MVPA, with a strong association reported with VPA.[47] Although the other study reported nonsignificant findings, VPA was more strongly associated with FMI than MPA or MVPA.[38]

Central Adiposity. Seventeen studies assessed the relationship between PA intensity and a measure of central adiposity[||] with 2 studies addressing 2 separate central adiposity measures,[33,50] resulting in a total of 17 associations. Eleven of the 17 associations were statistically significant,[¶] of which 10 demonstrated that VPA was a stronger negative predictor for central adiposity compared with MPA or MVPA.[#] Of these, one study[36] found this relationship only among females. The remaining association reported MVPA as a stronger negative predictor compared to VPA.[19]

Six of the 17 associations were nonsignificant for all PA intensities with central adiposity.[17,24,31,53,55,58] Albeit nonsignificant, 4 of these 6 relationships reported a stronger link between VPA and central adiposity compared with MPA or MVPA.[24,31,53,55] The remaining studies found MVPA to be a stronger negative predictor of central adiposity compared with MPA or VPA.[17,58]

Ortega et al[41] showed that when the association between PA intensity and central adiposity was stratified by CRF level, findings were contradictory. Among youth with a high CRF level, MPA and MVPA yielded significant positive associations with central adiposity, but this relationship was reversed and nonsignificant for those with low CRF levels.[41]

Age and Sex. Eight articles presented information on sex differences in the relationship between PA intensity and markers of body composition, which yielded mixed findings.[21,22,28,35,36,39,45,56] For youth younger than 12 years, Blaes et al[28] indicated that a significant, inverse correlation was observed between VPA and %BF for males, but not females. In contrast, Rowlands et al[45] reported females exhibiting a stronger relationship between VPA and %BF than males, although nonsignificant. Still, Fairclough et al[56] showed equal odds of normal weight status from VPA engagement for both sexes.

Similar evidence was presented for youth older than 12 years. Two articles reported stronger correlations observed between VPA and body composition markers for males than females.[35,39] Significant inverse relationships between VPA and body composition markers was observed for females but not males in another study,[36] and the last article reported similar findings irrespective of sex.[21] Interestingly, Treuth et al[22] found that while all non-significant, elementary and middle school-aged females exhibited stronger relationships

[||]References 17, 19, 23, 24, 26, 31, 33, 35, 36, 40, 41, 47, 48, 50, 53, 55, 58, 60.
[¶]References 17, 19, 23, 26, 33, 35, 40, 47, 48, 50.
#References 23, 26, 33, 35, 36, 41, 47, 48, 50.

Table 4.

Summary of Articles Reporting Beneficial, Not Beneficial, or No Association Between Physical Activity Intensity and Cardiorespiratory Fitness Outcomes in Youth.

	Beneficial	Not Beneficial	No Association
Cardiorespiratory fitness			
MPA	5/7 (71.4%)[17,29,30,45,53]		3/7 (42.9%)[29,52,54]
MVPA	5/5 (100.0%)[17,45,52-54]		
VPA	6/7 (85.7%)[17,30,45,52-54]	1/7 (14.3%)[29]	1/7 (14.3%)[29]
VO_2peak			
MPA	3/3 (100.0%)[16,48,49]		1/3 (33.3%)[16]
MVPA	4/5 (80.0%)[16,47-49]		2/5 (40.0%)[42,48]
VPA	5/6 (83.3%)[16,47-50]		2/6 (33.3%)[42,48]
VO_2max			
MPA			2/2 (100.0%)[23,25]
MVPA			
VPA	2/2 (100.0%)[23,25]		
Wmax			
MPA			
MVPA			1/1 (100.0%)[42]
VPA	1/1 (100.0%)[42]		
Heart rate			
MPA		1/1 (100.0%)[19]	
MVPA			
VPA		1/1 (100.0%)[19]	

Abbreviations: VO_2peak, peak volume of oxygen; VO_2max, maximal volume of oxygen; Wmax, maximal power output; MPA, moderate physical activity; MVPA, moderate to vigorous physical activity; VPA, vigorous physical activity.

between VPA and markers of body composition than males.[22] However, this observation was switched in high school–age youth.

Cardiorespiratory Fitness

Sixteen studies assessed the link between PA intensity and CRF.** Fifteen of the 16 studies reported a significant, direct relationship between PA intensity and CRF with the remaining study demonstrating mixed findings.[30] Ten articles demonstrated that VPA was more strongly associated with CRF compared with MPA or MVPA.†† Three of the 15 studies found MPA or MVPA to be more strongly and positively associated with CRF compared with VPA.[17,48,49] Interestingly, 2 studies found VPA to be a significant negative predictor of CRF.[20,30]

Age and Sex. In youth aged 6 to 7 years, Dencker et al[49] found that MPA was significantly related to CRF for both males and females, but only males retained this significance as intensity increased. Similarly, age at 12 (±2) years showed a significant direct correlation between MPA and CRF in females only, while a significant inverse relationship

**References 17, 18, 20, 24, 26, 30, 31, 36, 43, 46, 48-51, 53-55.

††18, 24, 26, 31, 36, 43, 46, 53-55, 60.

Table 5.

Summary of Articles Reporting Beneficial, Not Beneficial, or No Association Between Physical Activity Intensity and Cardiometabolic Biomarker Outcomes in Youth.

	Beneficial	Not Beneficial	No Association
CVD/MetS risk score			
MPA	2/4 (50.0%)[30,36]		2/4 (50.0%)[54,57]
MVPA	3/5 (60.0%)[16,36,47]		2/5 (40.0%)[54,57]
VPA	4/5 (80.0%)[30,36,47,54]		1/5 (20.0%)[57]
Systolic BP			
MPA	2/6 (33.3%)[30,33]		4/6 (66.7%)[23,25,54,57]
MVPA			4/4 (100%)[15,47,54,57]
VPA	3/7 (42.9%)[23,30,47]		4/7 (57.1%)[25,33,54,57]
Diastolic BP			
MPA	2/4 (50.0%)[30,57]		2/4 (50.0%)[33,54]
MVPA	1/3 (33.3%)[57]		2/3 (66.7%)[47,54]
VPA	2/5 (40.0%)[30,57]		3/5 (60.0%)[33,47,54]
Total cholesterol			
MPA			1/1 (100.0%)[54]
MVPA			2/2 (100.0%)[15,54]
VPA			2/2 (100.0%)[28,54]
HDL-cholesterol			
MPA			3/3 (100.0%)[30,54,57]
MVPA			3/3 (100.0%)[15,54,57]
VPA	1/4 (25.0%)[54]		3/4 (75.0%)[28,30,57]
LDL-cholesterol			
MPA			
MVPA			1/1 (100.0%)[15]
VPA			1/1 (100.0%)[28]
Triglycerides			
MPA	1/3 (33.3%)[30]		2/3 (66.7%)[54,57]
MVPA			3/3 (100.0%)[15,54,57]
VPA	1/4 (25.0%)[54]		3/4 (75.0%) [28,30,57]
Glucose			
MPA	2/6 (33.3%)[30,43]		4/6 (66.7%)[35,52,54,57]
MVPA	1/6 (16.7%)[43]		5/6 (83.3%)[15,35,52,54,57]
VPA	2/6 (33.3%)[30,43]		4/6 (66.7%)[35,52,54,57]

(continued)

Table 5. (continued)

	Beneficial	Not Beneficial	No Association
Insulin			
MPA	5/8 (62.5%)[15,16,30,36,43]		4/8 (50.0%)[16,35,52,54]
MVPA	3/7 (42.9%)[15,36,43]		4/7 (57.1%)[16,35,52,54]
VPA	2/7 (28.6%)[30,36]		5/7 (71.4%)[15,16,35,52,54]
Insulin resistance			
MPA	2/4 (50.0%)[36,43]		2/4 (50.0%)[35,52]
MVPA	2/4 (50.0%)[36,43]		2/4 (50.0%)[35,52]
VPA	2/5 (40.0%)[36,43]		3/5 (60.0%)[28,35,52]

Abbreviations: CVD, cardiovascular disease; MetS, metabolic syndrome; MPA, moderate physical activity; MVPA, moderate to vigorous physical activity; VPA, vigorous physical activity; BP, blood pressure; HDL, high-density lipoprotein; LDL, low-density lipoprotein.

existed between VPA and CRF in males only.[30] For youth older than 12 years, strength of relationship between VPA and CRF was similar for both sexes. However, males displayed stronger relationships at lower PA intensities.[36,54]

Cardiometabolic Biomarkers and Disease Risk Scores

Cardiovascular Biomarkers. Seven articles evaluated the association between varying levels of PA intensity and cardiovascular biomarkers (systolic blood pressure [SBP; n = 7],[24,26,31,34,48,55,58] diastolic blood pressure [DBP; n = 5],[31,34,48,55,58] and mean arterial pressure [MAP; n = 1]).[48] Overall, findings from the 7 studies did not suggest that any PA intensity was more strongly related to cardiovascular markers. Among studies assessing SBP as an outcome, 2 studies[24,48] reported that VPA was a significant, negative predictor of SBP of a greater magnitude than MPA and/or MVPA, while 2 other studies[31,34] reported that MPA was significantly and more strongly, negatively associated with SBP than VPA and/or MVPA. The remaining studies reported a nonsignificant correlation between PA at all intensities and SBP.[26,55,58]

Of the 5 studies evaluating DBP as an outcome, 2 reported a significant result, one of which[31] indicated MPA as a stronger negative predictor of DBP than VPA and/or MVPA, and the other[58] having significant negative associations for all PA intensities. The 3 remaining articles yielded nonsignificant observations between DBP and PA intensity, with 1 study[48] finding MVPA to be more strongly and inversely related with DBP than VPA, while another[55] finding the association to be equivalent across MPA, MVPA, and VPA. In the final study, it could not be determined which PA intensity potentially resulted in a stronger influence on DBP as only the significance (*P* value) of the association was reported.[34] The 2 studies examining the link between PA intensity and MAP found both to be nonsignificant with relatively equal magnitudes of association.[48,58]

Metabolic Biomarkers. Nine studies assessed the relationship between PA intensity and metabolic biomarkers (total cholesterol [TC; n = 3],[16,29,55] high-density lipoprotein cholesterol [HDL-C; n = 5],[16,29,31,55,58] low-density lipoprotein cholesterol [LDL-C; n = 2],[16,29] triglycerides [TGs; n = 5],[16,29,31,55,58] glucose [n = 6],[16,31,44,53,55,58] insulin [n = 7],[16,17,31,36,44,53,55] insulin resistance [IR; n = 4],[29,36,44,53] and insulin sensitivity [INS; n = 1]).[36] Of the 5 studies evaluating lipids (TC, HDL-C, LDL-C, TG) as an outcome,[16,29,31,55,58] 2 studies assessed the influence of a single PA intensity and found nonsignificant associations for both MVPA[16] and VPA.[29] Among the 3 remaining studies, 1 study found that VPA was significantly directly associated with HDL-C and significantly inversely related to TGs, and more strongly linked with these biomarkers than MPA and/or MVPA.[55] The second study found a significant inverse correlation between MPA and TGs, greater in magnitude when compared to MPA and/or VPA.[31] The last study reported nonsignificant associations among all PA intensities.[58]

Of the 6 articles that assessed the relationship between PA intensity and glucose,[16,31,36,44,55,58] one study assessed the influence of a single PA intensity and found a non-significant association for MVPA.[16] Two of the 5 remaining studies demonstrated that PA intensity was significantly and negatively associated with fasting glucose levels,[31,44] with 1 study[44] finding a stronger inverse correlation for VPA, and the other[31] observing a stronger connection between MPA and glucose levels. The remaining 3 studies reported nonsignificant relationships between PA intensity and glucose.[53,55,58] Of these nonsignificant

findings, 1 study[55] observed VPA to have a stronger negative association with glucose levels compared with MPA and MVPA, and the others had conflicting findings.[53,58]

Seven studies measured insulin[16,17,31,36,44,53,55] as an outcome with only 3 observing a significant and negative association between PA intensity and insulin.[16,31,44] Of these significant findings, 1 article[44] reported that VPA was inversely and more strongly related to insulin than MPA or MVPA, while the others showed a stronger relationship for insulin and MPA[16] or displayed similar findings between MPA and VPA.[31] Two of the 7 studies observed a nonsignificant association between PA intensity and insulin, with 1 study[55] demonstrating VPA as a stronger negative predictor of insulin whereas the other[17] found MPA to have a stronger relationship.[17] The remaining 2 studies reported mixed findings between PA intensity and insulin: one found nonsignificant direct correlations for MPA and MVPA whereas VPA demonstrated a nonsignificant inverse correlation,[53] and the other exhibited conflicting results between sexes when stratified by CRF.[36] Among females with low CRF, MVPA exerted a significant and stronger negative influence on insulin levels compared with MPA and VPA. This relationship in females became nonsignificant with increased CRF for all PA intensity levels. Conversely, for males, regardless of the level of CRF, PA intensity was not significantly associated with insulin levels.[36]

Four studies assessed the link between PA intensity and IR[29,36,44,53] with 1 study assessing the influence of a single PA intensity and found a nonsignificant relationship with VPA.[29] Of the remaining 3 articles, only 1 significant association was reported, which indicated that VPA was a stronger negative predictor of IR compared with MPA and MVPA.[44] Another found conflicting results between the sexes when stratified by CRF.[36] Among females with low CRF, MVPA displayed a significant and stronger inverse association with IR compared with MPA and VPA; however, higher levels of CRF yielded nonsignificant findings for all PA intensities. Among males, nonsignificant relationships were found for all PA intensities and IR.[36] In the remaining report,[53] the influence of PA intensity on IR was conflicting as VPA demonstrated an inverse relationship with IR, whereas a positive relationship was found for MPA and MVPA.

Only 1 study examined the link between PA intensity and INS and found that the results differed by sex when stratified by CRF level.[36] The findings of this study suggested that MVPA significantly and positively influenced INS compared with MPA and VPA only among females with low CRF. This relationship was nonsignificant among females with moderate to high CRF. No association existed among males between any PA intensity and INS regardless of CRF level.

Cardiovascular Disease and Metabolic Syndrome Risk Scores. Five articles evaluated the association between PA intensity and cardiovascular disease (CVD)[37,48] and/or metabolic syndrome (MetS)[31,55,58] risk score, of which all but one demonstrated significant relationships.[58] Two studies found VPA was more strongly inversely associated with disease risk score than MPA and/or MVPA.[48,55] However, the correlations with MPA and MVPA with MetS risk score were nonsignificant for one of these studies.[55] In this report, the magnitude of association between VPA and MetS risk score was substantially attenuated and became nonsignificant after further adjustment for CRF.[55] Another article found that across all quintiles of PA (highest PA level as referent), MVPA demonstrated a significant negative and stronger influence on CVD risk score compared with MPA and VPA, however, only occurring among females.[37] In males, MPA was a significant negative predictor of CVD risk score compared with MVPA and VPA, although only among those with the lowest level of PA.[37] The remaining study showed both MPA and VPA to be significantly and negatively associated with MetS risk score, with impact appearing to be similar in magnitude.[31]

Age and Sex. Three articles reported outcomes for PA intensity and CM biomarkers by sex.[36,37,48] Tanha et al[48] showed that males and females, aged 7.9 to 11.1 years, had relatively similar, significant correlations between VPA and CVD risk score, as well as MVPA and CVD risk score. The 2 other articles, both by Jiménez-Pavón et al,[36,37] found mixed results. In youth aged 6 to 9 years, females had higher CVD risk scores than males across all PA intensities.[37] While CVD risk for both sexes generally declined with increasing PA quintiles, it increased with increasing MPA in females. Additionally, the odds ratio retained significance for females as PA quintiles increased, whereas males lost significance.[37]

In the second report concerning youth older than 12 years, Jiménez-Pavón et al[36] noted that partial correlations yielded stronger significant findings for females regarding the connection between VPA and insulin and INS. However, males had a stronger, significant correlation between VPA and IR, and a stronger nonsignificant correlation between VPA and glucose.[36] When intensity decreased, these relationships for males became nonsignificant, but females retained significance, although strength was reduced but still stronger than respective results for males. When reporting regression coefficients, stronger nonsignificant associations for females were demonstrated concerning MPA and MVPA versus insulin, INS, and IR, with males showing a stronger nonsignificant relationship between VPA and these biomarkers.[36]

Discussion

The purpose of this review was to systematically examine the current literature on the associations between PA intensity and body composition, CRF, and CM biomarkers in youth. Major findings of this study were (1) VPA was more strongly negatively associated with body composition, and more strongly positively associated with CRF compared with MPA or MVPA, (2) no

specific PA intensity appeared to consistently and more strongly associate with CM biomarkers, and (3) among the majority of studies demonstrating that VPA had a stronger relationship with adiposity and CRF outcomes, participants often engaged in at least 10 minutes of VPA, potentially suggesting that 10 minutes of VPA may be a sufficient dose for youth.

In a previous review of PA intensity and health outcomes related to adiposity and fitness, which examined 17 observational studies published between 1999 and 2009, authors noted that a shorter duration of VPA yielded a stronger association with body fat than a longer duration of MPA.[11] A more recent review remarked that every minute of VPA yields the same improvements in body composition as 2 or 3 minutes of MPA.[12] In our expanded and updated review, among the studies that found VPA significantly related to BMI, body composition, and central adiposity compared with other PA intensities, a majority reported participants engaging in an average of at least 10 minutes of VPA.[#] Considering that the "dose" of VPA was relatively modest in many of these studies, as well as the sporadic nature of PA in youth with brief intermittent bouts of VPA,[61] it is conceivable that VPA may be more attainable simply because a health-improving dose is less time-consuming to attain. Therefore, it could be suggested that VPA should be promoted independent of MPA as a behavior. Additionally, Martinez-Gomez et al[62] and Katzmarzyk et al[7] conducted receiver operating characteristic analyses on activity dose and identified that 17 and 20 minutes of VPA in boys and 9 and 11 minutes of VPA in girls, respectively, mitigated the risk of being overfat and/or obese, highlighting possible sex differences in VPA recommendations.[7,62]

In line with previous studies, we found that VPA was consistently more strongly associated with CRF compared to MPA and/or MVPA. This finding has also been demonstrated among exercise training studies with intensities greater than 70% of age-predicted maximum heart rate (vigorous intensity) eliciting significant changes in CRF.[3] A prior review by Ortega et al[63] on fitness and health outcomes in youth discussed the importance of VPA to increase CRF for favorable changes in adiposity and CM profile. Additionally, among adults substantial evidence indicates that there is a dose-response relationship between PA intensity and CRF, with the greatest improvement in CRF found among very high intensities (90% to 100% VO_2max).[64] Importantly, CRF has been demonstrated to be a strong predictor of morbidity[65,66] and mortality[67-69] among adults, and some evidence indicates that CRF tracks modestly from childhood to adolescence to adulthood.[70-72] As a result, it is reasonable to suggest that increasing CRF among youth should be a public health priority; as such, including a dose of PA that may elicit improvements in CRF (ie, VPA) in the PA guidelines is warranted. Among the studies in this review that found VPA to be more strongly associated with CRF, a majority described youth engaging in at least 10 minutes of VPA (range 12-46 minutes), suggesting this dose of VPA may be a good starting point for youth.[36,43,46,53-55,60]

In contrast to the previous findings of this review, associations between PA intensity and CM biomarkers were equivocal. We did not find a PA intensity (MPA, MVPA, or VPA) that was strongly associated with any of the CM biomarkers consistently when compared with other intensities. VPA did not display a strong relationship with MetS/CVD risk score in youth, and in fact became nonsignificant after controlling for CRF.[55] This finding was echoed for insulin and IR, with higher levels of CRF negating any significant influence of VPA on these CM biomarkers.[36] It is possible that the conflicting findings in our review may be due to the inherent healthiness of children and adolescents. The review by Owens et al[12] suggested that VPA was associated with a more favorable blood lipid profile and glucose-insulin profile. Conversely, inconclusiveness of findings between PA intensity and CM biomarkers was demonstrated in a previous review[6] that examined BP, plasma lipid and lipoproteins, and MetS. However, authors noted that favorable changes in blood lipids and lipoproteins post-exercise intervention were more likely to occur in high-risk participants, indicating that the initial health status of the study population may greatly affect the observed association.[6]

In addition, Gutin and Owens[10] in their review of both observational and intervention studies identified fitness and fatness as mediating factors of the relationship between PA and CM biomarkers among youth, noting that PA interventions conducted among overweight and obese youth had a beneficial effect on several CM biomarkers. Conversely, the evidence was limited for PA interventions targeting nonobese youth.[10] However, there is evidence demonstrating the emergence of obesity-related complications in early childhood that includes the premature manifestation of CM risk factors.[73-76] Ferreira et al[77] noted in their longitudinal study following 450 participants from adolescence to young adulthood that increases in body fat, declines in CRF, and a greater shift from time spent in VPA to light-to-moderate intensity PA were characteristic of MetS at the age of 36 years. As such, the findings of our review provide additional support for weight control and improving CRF in youth as a preventive measure for future CM disease.

There are several strengths to this review that warrant attention. Few reviews in the past have solely focused on objective PA measurement, which has now become more standardized in PA assessment. We also included CM biomarkers in addition to body composition and CRF, which are often not collectively examined despite their connections. Additionally, this is the first review to identify a possible VPA dose for youth. As with any study, there are also limitations that are worth mentioning. We chose not to examine intervention studies as done in prior reviews,[10-12] narrowing our focus to

[#]References 7, 21, 23, 28, 35, 36, 39, 40, 42, 46-48, 50, 52, 55-57.

observational studies. Even though not limited to objective measurement tools, we felt these previous reviews provided sufficient support for the changes that high-intensity structured exercise could make over the course of an intervention, and chose instead to focus on larger observational studies that gathered data on VPA when incorporated in normal daily activity. Nonetheless, this review highlights the importance of further conducting PA interventions focused on intensity in youth for future review.

An additional limitation in the observational studies we reviewed also involves variation in method of PA measurement. All articles used accelerometry to measure PA intensity, but accelerometer model, length of wear, and VPA cutpoints varied widely. The Actigraph GT1M model was primarily used (33.3%; n = 15)[§§] or one of its predecessors (ActiGraph 7164, MTI 7164, or CSA 7164; 43.9%; n = 18),[‖‖] but even when using the same model, VPA cutpoints applied were inconsistent (eg, 3000 to 4012 counts/min; Actigraph GT1M).[20,34] Considering that youth primarily engage in very short periods of VPA at a time, a 60-second or even a 10-second epoch may not adequately differentiate VPA as it is combined with periods of rest or light PA.[61,78]

In conclusion, while there are certainly benefits to MPA, as evidenced in this review, VPA was a stronger predictor of improved body composition, particularly for reduced body fat and central adiposity, and increased fitness compared with MPA and/or MVPA. These patterns were observed even when findings were nonsignificant. From our results, it appears that a dose of 10 minutes of VPA/day may be sufficient to elicit favorable changes in these health outcomes, with possible differences for male and female youth. However, these suggestions need to be confirmed with rigorously randomized controlled exercise training studies. AJLM

[§§]References 20, 33-37, 39, 40, 47, 53-58.
[‖‖]References 16, 18, 21, 23, 27, 28, 32, 41-44, 46, 48-52, 60.

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