Applied Physiology, Nutrition, and Metabolism
Physiologie appliquée, nutrition et métabolisme

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Journal:	*Applied Physiology, Nutrition, and Metabolism*
Manuscript ID	apnm-2016-0441.R3
Manuscript Type:	Article
Date Submitted by the Author:	01-Nov-2016
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Keyword:	Inactivity, Intensity, Behaviour, Genetics, kinesiology < biomechanics

Genetic Characterization of Physical Activity Behaviours in University Students Enrolled in Kinesiology Degree Programs

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Abstract

Studies of physical activity behaviours have increasingly shown the importance of heritable factors such as genetic variation. Non-synonymous polymorphisms of alpha-actinin 3 (*ACTN3*) and the β-adrenergic receptors 1 and 3 (*ADRB*) have been previously associated with exercise capacity and cardiometabolic health. We thus hypothesized that these polymorphisms are also related to physical activity behaviors in young adults. To test this hypothesis we examined relationships between *ACTN3* (R577X), *ARDB1* (Arg389Gly) and *ADRB3* (Trp64Arg), and physical activity behaviors in university students. We stratified for student enrollment in kinesiology degree programs compared to non-majors as we previously found this to be a predictor of physical activity. We did not identify novel associations between physical activity and *ACTN3*. However, the minor alleles of *ADRB1* and *ADRB3* were significantly underrepresented in kinesiology students compared to non-majors. Furthermore, carriers of the *ADRB1* minor allele reported reduced participation in moderate physical activity and increased afternoon fatigue compared to ancestral allele homozygotes. Together, these findings suggest that the heritability of physical activity behaviours in young adults may be linked to non-synonymous polymorphisms within β-adrenergic receptors.

Key Words: Inactivity, Intensity, Behaviour, Genetics, Kinesiology

Introduction

Inactivity and sedentary behaviors contribute to ~5.3 million deaths annually (Katzmarzyk and Janssen 2004; Lee et al. 2015; Ogden et al. 2014; Rao et al. 2015). A significant proportion of these deaths are due to complications from type 2 diabetes (T2D) and the insulin resistance syndrome that precedes it (Cowie et al. 2006). Regular physical activity is the most effective intervention for the prevention of insulin resistance and T2D (Fiuza-Luces et al. 2013). However, significant variability exists in both the effectiveness of and adherence to prescribed physical activity for health and fitness (de Geus et al. 2014; Houmard et al. 2004; Johnson et al. 2004). Since some of this variability can be attributed to polymorphisms within exercise-related genes, a precision medicine approach is warranted to manage the burden of inactivity-related disease (Bouchard et al. 2011; Bray et al. 2016; de Geus et al. 2014; Lee et al. 2015).

Previous research from our laboratory has identified significant effects of genetics (Deschamps et al. 2015), educational setting and social/geographical factors (Many et al. 2016) on parameters of cardiometabolic fitness (Myslicki et al. 2014) and physical activity behaviors in healthy young university students. For instance, we have recently shown that the common 577X stop-codon mutation within the gene encoding sarcomeric alpha-actinin 3 (*ACTN3*) is associated with lower cardiovascular fitness (peak VO_2), increased body fat and an atherogenic metabolite profile relative to carriers of the R577 ancestral allele (Deschamps et al. 2015). These findings support a large body of published research linking the *ACTN3* "sports gene" to exercise capacity in both elite and amateur athletes (Chan et al. 2008; Clarkson et al. 2005; Delmonico et al. 2007; Eynon et al. 2014; MacArthur and North 2004; Mills et al. 2001; Norman et al. 2014). In this same

cohort we determined that students enrolled in accredited kinesiology degree programs in Canada and the United States display improved parameters of insulin sensitivity and increased physical activity levels relative to non-kinesiology majors (Many et al. 2016). Given the significant heritability of physical activity behaviors in young adults (~84%), our kinesiology cohort represents a novel population of convenience for investigating the relationship between genetics and physical activity (de Geus et al. 2014; Nedovic et al. 2016).

The β-adrenergic receptors encoded by the *ADRB* genes, are catecholamine-sensitive, G-coupled transmembrane proteins that are expressed in variety of tissues, including heart, skeletal and smooth muscle, adipose and the brain (Burguete-Garcia et al. 2014; Clement et al. 1995; Numajiri et al. 2012; Snyder et al. 2006; Wagoner et al. 2002; Walston et al. 1995; Widen et al. 1995). Like *ACTN3* (Deschamps and Hittel 2016), non-synonymous (amino acid changing) variants of the β-adrenergic receptors genes *ARDB1* (Arg389Gly) and *ADRB3* (Trp64Arg) have been associated with both insulin resistance and athletic performance (Burguete-Garcia et al. 2014; Clement et al. 1995; Walston et al. 1995; Widen et al. 1995). Although the mechanisms are not well established as *ACTN3*, *ADRB* receptors in the brain have been shown to modulate anxiolytic (anxiety reducing) behaviours (Stemmelin et al. 2008) and thus may influence the perception of exercise as a means to reduce anxiety (Pedersen and Saltin 2015).

To test this hypothesis we examined *ACTN3*, *ADRB1* and *ADRB3* gene variants in relation to physical activity behaviors in a population of healthy young university students. Findings from this study may be of importance for identifying genetic markers

associated with physical activity that can be used for targeting physical activity interventions in at risk individuals.

Methods and Materials

Participants

All participants were a part of the Assessing Inherited Markers of Metabolic Syndrome in the Young (AIMMY) study described previously (Deschamps et al. 2015; Karlos et al. 2013; Klein et al. 2014; Many et al. 2016). University students were enrolled at three recruiting sites: (UC) University of Calgary (n=197, Calgary, Canada), (ECU) East Carolina University (n=91, Greenville, NC) and (UM) University of Massachusetts (n=207, Amherst, MA). This study was approved by the Conjoint Health Research Ethics Board at the University of Calgary (Ethics ID: E23521) and is registered under the clinicaltrials.gov identifier NCT00966407. Written informed consent for genetic and all other testing was obtained from all subjects before participation and was conducted under the provisions of the Declaration of Helsinki. Subjects were: (1) between the ages of 18 and 35 years; (2) had completed puberty; and (3) willing and able to provide informed consent. At all sites, recruitment occurred on-campus using posters, information on campus-wide monitors, brief classroom sessions and the university's website. All eligible, consenting participants were considered to be healthy at the time of enrolment. Health was defined as an absence of: (1) evidence of clinically relevant systemic disease associated with metabolic disorders ; (2) chronic use of glucocorticoid or appetite suppressants; (3) the use of drugs that alter glucose metabolism or other medications known to alter blood levels being tested in this study; (4) previous diagnosis or treatment

for any hematologic-oncologic disorder; (5) history or current treatment for an eating disorder; (6) current treatment for weight loss; (7) history of bariatric surgery; (8) history of neurosurgical procedure.

Clinical Blood Measures

Blood samples were collected in de-identified tubes after an 8–12 hour, overnight fast. Blood for lipoprotein assays (LDL-C, High-Density Lipoprotein Cholesterol (HDL-C), Total Cholesterol (TC), and Triglycerides (TG)) as well as insulin, glucose and HbA1c was collected using serum stopper tubes containing a clot activator and a silicon gel separator. After collection, samples were spun at 3000 rpm for 10 minute and stored at 2–8°C until being transported to Calgary Lab Services (Calgary, AB) or Quest Diagnostics (Madison, NJ) for analysis as described previously (Deschamps et al. 2015; Karlos et al. 2013; Many et al. 2016).

Genotyping

Genomic DNA for genetic analysis was isolated from peripheral blood as described previously. Blood samples were collected in tubes containing an ethylene diamine tetra-ascetic acid (EDTA) anticoagulant and were stored at 2–8°C for a maximum of one week before being sent to the Children's National Medical Centre (CNMC) in Washington, DC without subject identification. The *ACTN3* R577X (rs1815739), *ADRB1* Arg389Gly (rs1801253) and *ADRB3* Trp64Arg single nucleotide polymorphisms (SNPs) (rs4994) were identified using TaqMan allele discrimination assay (Deschamps et al. 2015).

Fitness Assessment

Seated resting blood pressure and heart rate measures were taken 3 times over 2 separate visits using an automated monitor cuff. Grip strength was assessed using an Almedic 100kg hand grip dynamometer (Almedic, Montreal, QC, Canada). Body mass index (BMI) was calculated by dividing the subjects height in meters by their weight in kg^2. Percent body fat (%BF) and bone mineral density (BMD) was measured using a dual-energy x-ray absorptiometry scan (DXA) (Hologic QDR 4500A scanner, Hologic Inc, Walthan, MA.). VO_2peak was assessed using the Bruce treadmill protocol as an indicator of cardiovascular fitness. Oxygen consumption was assessed with a Hans Rudolph nonbreathing 2-way valve mouthpiece and a ParvoMedics TrueOne 2400 metabolic cart (ParvoMedics, Sandy, UT) (Shah 2013).

Questionnaires

Family history, ethnicity, diet and physical activity levels were recorded by self-report using secure online questionnaires and an iPad as described previously (Many et al. 2016). Self-reported physical activity was assessed via a 12-month Paffenbarger physical activity survey from which, weekly energy expenditure was calculated from the time and energy (metabolic equivalents [METs]) spent participating in leisure and non-leisure physical activities (Simpson et al. 2015). Further, subjects were asked to divide the time spent engaging in light, moderate, and vigorous-intensity physical activity as well as sitting and exercise intensity over a typical 24-hour weekday and weekend (Many et al. 2016). To assess exercise intensity specifically we used the Borg Category Ratio 0-10 (Borg CR-10) rate of perceived exertion (RPE) scale where 0 means "rest" and 10 means "maximal exertion" (Irving et al. 2006). The Epworth Sleepiness Scale (ESS) was

used to assess daytime sleepiness (Johns 1991). Finally, students at the University of Calgary and East Carolina University recruitment sites (n=288) were asked to report their college major (kinesiology vs non-kinesiology major) as described previously (Many et al. 2016).

Statistical Analysis

Unless otherwise stated, all statistical analyses were performed using SPSS Statistics, version 20 (IBM). All data are presented as mean ± SEM. To test for homogeneity of *ACTN3*, *ADRB1* and *ADRB3* genotype frequencies between kinesiology students and non-majors χ^2 values were estimated using genotype numbers as described previously (MacArthur and North 2004).

We used a dominant genetic model for all genotype associations. Age and sex were used as covariates except for when sexes were being compared. Analysis of covariance (ANCOVA) with the Sidak method for post-hoc multiple comparisons adjustment was used for post-hoc multiple comparisons adjustment of *P* values (Lee et al. 2015). All resulting adjusted means are shown as transformed values as those are the numbers used for statistical models.

Results

AIMMY Subject Characteristics

Of the 288 participants enrolled in AIMMY from the UC and ECU sites, 150 (52%) subjects were female and 138 (48%) were male with a mean age of 22.4 ± 2.8 (age range 18-35, Table 1). Among AIMMY participants, 223 (77.4%) self-identified as Caucasian, 27 (9.4%) as Asian, 19 (6.6%) as African American, 11 (3.8%) as Other or

not-specified and 8 (2.8%) as Hispanic or Latino. Kinesiology (KNES) majors exhibited significantly lower total body fat, lower fasting insulin levels, HOMA-IR and a higher VO_2peak (Table 1) compared to non-majors (NON). KNES majors also reported significantly higher physical activity levels compared to NON-majors and self-reported "regular" exercise to be at a higher intensity as captured by the Borg CR-10 scale (Table 2). Additional physical activity and dietary data from this cohort have been published previously (Deschamps et al. 2015; Many et al. 2016).

For population verification and further genetic analyses, an additional 207 University of Massachusetts students without faculty information (KNES vs NON) were added to the original AIMMY cohort creating a combined cohort of 495 individuals, 229 (46.3%) Female, 266 (53.7%) Male).

Genetic Analysis

As a means of ensuring genotyping accuracy we determined that the *ACTN3* rs1815739 Minor Allele Frequency (MAF) was in Hardy-Weinberg Equilibrium (HWE) in both KNES majors (MAF =37, p=0.99) and NON-majors (MAF=45, p=0.60) in our combined cohort. Further, there were no significant differences in the distribution of *ACTN3* genotypes (P=0.423) (Figure 1), nor were there any additional associations with cardiometabolic or fitness traits other than those reported previously by our lab (Peak VO_2, Systolic & Diastolic Blood Pressure and % Body Fat) (Deschamps et al. 2015).

Similarly, we determined that *ADRB1* rs1801253 (MAF = 32%, p=0.17) and *ADRB3* rs4994 (MAF = 7.4%, p=0.31) loci were also in HWE in the combined AIMMY cohort. However a Pearson's χ^2 test identified significant differences in the distribution of *ADRB1* CC, CG and GG genotypes (P=0.004) and *ADRB3* AA, AG and GG genotypes

(*P*=0.018) between kinesiology students and non-major sub-populations (Figure 1). In the case of *ADRB1*, 32% (32) kinesiology students carried one or more copies of the minor G/Arg389Gly allele compared to 52% (98) non-majors. For ADRB3, 7% (7) of kinesiology students carried one or more copies of the minor G/Trp64Arg allele compared to 14% (26) non-majors. Taken together these findings inspired a deeper investigation of the relationship between *ADRB* genotypes, cardiometabolic and fitness characteristics of our AIMMY cohort (Tables 1 and 2) that may underlie differences in the population structure of kinesiology students (Many et al. 2016). The observed MAFs were within the range of population genetics data from the 1000 genomes (Kuehn 2012) and NIH Exome Sequencing Project cohorts (Auer et al. 2012) for *ACTN3* (37.2 %) *ARDB1* (30.0%) and *ARDB3* (11.5%).

In the original (n=288, Table 3A), verification (n=207, Table 3B) and combined (n=495, Table 3C) AIMMY cohorts, *ADRB1* CG/GG individuals reported significantly fewer calories expended during moderate-type physical activity. Whereas *ADRB1* CG/GG genotypes in the original cohort reported increased "afternoon fatigue" (*P*=0.04) compared to CC homozygotes (Table 3A) these differences persisted below the threshold for statistical significance in the verification (p=0.06, Table 3B) and combined (p=0.057, Table 3C) AIMMY cohorts. *ADRB1* CG/GG individuals also exhibited significantly lower peak VO_2 in our combined AIMMY cohort (Table 3C) that was not apparent in the smaller cohorts. Taken together, these findings indicate a role for *ARDB1* genotype in voluntary exercise that supports previous associations with exercise capacity (Wagoner et al. 2002). Because the mean values for both *ADRB1* heterozygotes (CG) and

homozygotes (GG) were similar and because of the relatively low numbers of risk allele homozygotes we used a dominant genetic model to calculate associations.

In our original cohort, individuals with *ADRB3* AG/GG genotypes scored significantly lower on the Borg CR10 RPE scale when asked to assess their "normal" intensity level during exercise (Table 3A). Although these differences were not significant in the verification cohort (p=0.07, Table 3B) they were apparent in female subjects in the combined larger cohort (p=0.05, n=229). We also identified significant associations between *ADRB3* genotype, fasting triglyceride levels and HOMA-IR in female subjects only wherein possession of 1 or more copy of the G allele was associated with higher triglycerides and HOMA-IR (Table 3C). These findings are consistent with the role of beta-adrenergic receptors in the regulation of lipolysis and thermogenesis in white and brown adipose tissue(Chang et al. 2012; Ueta et al. 2012). These findings also support previous research linking the *ADRB3* Trp64Arg polymorphism with increased susceptibility to insulin resistance and type 2 diabetes (Sakane et al. 2016) (Fujisawa et al. 1996).

Finally, the associations of *ADRB3* genotypes with the perception of exercise intensity and *ADRB1* with afternoon fatigue were only significant in NON-majors (n=198) (data not shown). Although these associations may be due to reduced statistical power in the smaller KNES cohort (n=99), they may also indicate that educational setting/environment can override genotype-associated physical activity behaviors.

Discussion

We have discovered that common polymorphisms of the β-adrenergic receptors 1 and 3 are significantly underrepresented in a population of healthy young university

students enrolled in kinesiology degree programs. Carriers of the *ADRB1* G allele expended on average, 776 fewer kilocalories per week participating in moderate-intensity physical activities, experienced greater afternoon fatigue and exhibited lower cardiovascular fitness (peak VO_2) compared to homozygous carriers of the ancestral C allele. Similarly, individuals with one or more copy of the *ADRB3* G allele reported lower exertion levels during regular exercise, higher fasting triglycerides and higher HOMA-IR values (in females) indicating lower sensitivity to insulin. Three of these characteristics (Moderate Sports, HOMA-IR and Peak VO_2) were also different in comparing kinesiology with non-major cohorts (Tables 1 and 2) suggesting that β-adrenergic receptor genetics play a role in determining the population structure of our kinesiology cohort. By way of contrast, *ACTN3* R577X alleles were equally distributed between kinesiology students and non-majors despite the many associations of this well-characterized "exercise performance" gene with cardiometabolic and muscle strength characteristics (Deschamps et al. 2015). Taken together, these convergent lines of experimental evidence indicate that *ARDB1* and *ADRB3* polymorphisms influence voluntary participation in physical activity. As with many energy conserving or "thrifty" genes, this may explain their association with obesity and insulin resistance in multiple ethnic groups (Burguete-Garcia et al. 2014; Deschamps and Hittel 2016; Kim et al. 2010; Sakane et al. 2016; Takenaka et al. 2012; Wagoner et al. 2002; Widen et al. 1995).

Associations between physical activity behaviours and adrenergic receptor genotypes underscores the influence of genetics on behaviour (de Geus et al. 2014; Lee et al. 2015; Perusse et al. 1989; Stubbe et al. 2006). For instance, researchers from the Social Science Genetics Association Consortium (SSGAC) have recently identified a

connection between the genetic components of cognition and years of formal education (Kovas and Malykh 2016). Therefore, given the unique focus on exercise and athletics encapsulated by the modern kinesiology student, it follows that individuals with an innately higher drive towards exercise would be drawn to such programs (Many et al. 2016).

Despite their primary roles in the sympathetic control of cardiac output and lipolysis, β1 and β3-adrenergic receptors are also localized to neuronal synapses in the basolateral amygdala where they modulate anxiolytic (anxiety reducing) and other behaviours (Stemmelin et al. 2008). Indeed, recent published research supports a role for the *ADRB1* Arg389Gly polymorphism in both the perception of pain (Wei et al. 2015) and the psychological trait of persistence as assessed by the temperament and character inventory (Numajiri et al. 2012). In addition, β3-adrenergic receptor agonists such as SR58611A (amibegron) have proven to be an effective treatment strategy for anxiety and depressive disorders (Stemmelin et al. 2008). Because both *ADRB1* Arg389Gly and *ADRB3* Trp64Arg polymorphisms decrease agonist mediated coupling of activated receptors to adenylate cyclase activity (Fujisawa et al. 1996), it is conceivable that these polymorphisms may alter the perception and/or enjoyment of habitual exercise. Given the poorly understood role of the adrenergic system in the brain our findings provide a plausible experimental framework for testing the effect adrenergic receptor polymorphisms on the perception of exercise intensity and fatigue.

The effect of β-adrenergic receptor function on cardiovascular physiology has also been well studied (Kim et al. 2010; Snyder et al. 2006; Twentyman et al. 1981; Wagoner et al. 2002). As we observed in our AIMMY cohort, heart failure patients

homozygous for the *ADRB1* 389Gly polymorphism had significantly lower peak VO_2 compared with those with one or more copy of the Arg389 receptor (Wagoner et al. 2002). Similarly, associations of the *ADRB3* Trp64Arg polymorphism with elite endurance performance in Spanish cyclists and Korean volleyball players suggests a role for this gene in regulating the cardiovascular response to exercise (Santiago et al. 2011). As such, it is also possible that the higher levels of physical activity associated with ancestral *ADRB1* and *ADRB3* genotypes may attributable to a more efficient cardiovascular and metabolic response to exercise. As such, increased cardiometabolic efficiency may influence perception of exercise difficulty and thus influence participation (Oliver 2012).

Freshman university students are an ideal population to target education about lifelong fitness habits that will shape future health (Many et al. 2016). This is particularly true for female carriers of *ADRB3* Trp64Arg polymorphism in our AIMMY cohort who exhibit elevated triglycerides and HOMA-IR at a relatively young age (Table 3B). Furthermore, *ADRB3* Trp64Arg has been designated as a risk allele based on research linking it to weight gain and an early risk for developing cardiovascular disease in women (Clement et al. 1995; Kumar et al. 2014). On the other hand, high fitness levels in carriers of the *ADRB3* Trp64Arg polymorphism have been shown to eliminate the increased risk of atherosclerosis associated with this common variant (Iemitsu et al. 2014). This is consistent with our observation that associations between *ADRB3* and RPE and *ADRB1* with fatigue were only significant in non-majors compared to kinesiology students. This suggests that possession of one or more *ADRB* risk loci is not deterministic in regards to cardiometabolic risk and lends strength to our argument that physical

activity may be the mechanistic bridge between polymorphisms in the beta-adrenergic receptor and the development of obesity and insulin resistance. These findings may indicate that an accredited kinesiology curriculum, which includes courses in exercise physiology, motor learning and sports psychology can potentially overcome genotype-associated limitations on physical activity.

Whereas once we held a dichotomous view of nature (genes) vs nurture (environment), a more nuanced model is emerging wherein gene-environment interactions affect our perceptions and choice of educational setting (Okbay et al. 2016). Although significant limitations of this study include the realtively small sample size and post-hoc nature of our analysis, it is of future interest to our laboratory to examine the genomic characteristics of a significantly larger cohort of kinesiology students (Nedovic et al. 2016). Apart from these findings, there are many fascinating research directions that may be explored involving the application of our research. For instance, individuals who are polymorphic for *ADRB1* and *3* risk alleles may need extra assistance (personal trainer or feedback from a wearable fitness monitor or app) to adhere to prescribed exercise program, not because they are lazy or unmotivated, rather that exercise is perceived as harder for them. In addition, exercise behavior and aptitude genes could be used in Mendelian randomization studies proxies for physical activity to better discern the effects of environmental and social factors on cardiometabolic health (Burgess and Harshfield 2016). Findings from this study are also of importance for identifying and removing barriers to physical activity given the strong association of sedentary behavior with all-cause morbidity (Deforche et al. 2015; Friedenreich et al. 2006).

Conflict of Interest Statement

The authors report no conflicts on interest associated with this manuscript.

Acknowledgements

DSH and CLD were funded by philanthropic donations from the Eresman Family and BURNCO Foundations, Jay and Karen Westman. DSH and JS are individually funded by a Discovery Grant from the Natural Sciences and Engineering Research Counsel of Canada (NSERC). GMM was funded by a NIH postdoctoral training grant T32HD071866 as well as the Clark Charitable Foundation, Inc. This study is funded in part by the Children's National Medical Center General Clinical Research Center (5-M01-RR-020359-02), the DC-Baltimore Research Center on Child Health Disparities (5P20MD00165), The Dairy Research Institute, NICHD/NINDS 5R24HD050846-08: NCMRR-DC Core Molecular and Functional Outcome Measures in Rehabilitation Medicine. The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

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Table 1. ANCOVA comparisons of original AIMMY cohort characteristics between kinesiology (KNES) and non-kinesiology majors (NON). Data are mean values +/- SEM. Significant *P* values ≤0.05 are shaded and NS indicates non-significant differences.

Subject Characteristics	KNES Majors n=99	Non-Majors n=189	*P*-Value
Age (y)	22.09 ± 0.34	22.24 ± 0.33	NS
Height (cm)	171.93 ± 0.95	172.31 ± 0.76	NS
Weight (kg)	70.85 ± 0.95	72.45 ± 0.76	NS
BMI	23.31 ± 0.29	23.71 ± 0.31	NS
% Body Fat	19.20 ± 0.94	23.83 ± 0.92	0.002
Hip (cm)	98.00 ± 0.74	99.40 ± 0.77	NS
Triglycerides (mg/dL)	80.31 ± 4.03	85.51 ± 2.98	NS
Cholesterol (mg/dL)	158.61 ± 3.41	162.18 ± 2.45	NS
HDL (mg/dL)	62.07 ± 2.13	60.37 ± 1.23	NS
LDL (mg/dL)	83.41 ± 2.96	84.63 ± 1.75	NS
Glucose (mg/dL)	80.56 ± 0.68	83.37 ± 0.47	NS
Insulin (uIU/ml)	5.34 ± 0.30	6.56 ± 0.28	0.007
% HbA1c	5.48 ± 0.03	5.48 ± 0.02	NS
HOMA-IR	1.08 ± 0.07	1.32 ± 0.07	0.013
CRP (mg/L)	1.19 ± 0.24	2.16 ± 0.58	NS
Grip Strength (kg)	45.11 ± 1.31	42.51 ± 1.15	NS
BP-Systolic	114.04 ± 0.24	112.56 ± 0.24	NS
BP-Diastolic	67.67 ± 0.24	68.98 ± 0.24	NS
VO_2 (ml/kg/min)	48.26 ± 0.24	46.21 ± 0.24	0.026

All models were adjusted for recruitment location, age and sex using a one-way analysis of covariance (ANCOVA). The Sidak method was used post-hoc to account for multiple comparisons. BMI = body mass index; HDL= high-density lipoprotein cholesterol; LDL = low-density lipoprotein cholesterol; HOMA-IR = homeostatic model assessment for insulin resistance.

Table 2. ANCOVA comparisons of original AIMMY cohort physical activity survey data between kinesiology (KNES) and non-kinesiology majors (NON). Data are mean values +/- SEM. Significant *P* values ≤0.05 are shaded and NS indicates non-significant differences.

Selected Physical Activity Participation and Perception Scores	KNES-Majors n=99	NON-Majors n=189	*P*-Value
Walking (kcal/week)	1067.46 ± 116.21	844.07 ± 54.49	NS
Stair Climbing (kcal/week)	398.91 ± 62.81	299.52 ± 24.99	NS
Combined (kcal/week)	1466.37 ± 145.06	1143.59 ± 63.84	NS
Light Sports (kcal/week)	391.06 ± 59.98	370.01 ± 78.97	NS
Moderate Sports (kcal/week)	4043.86 ± 483	2873.77 ± 287	0.029
Vigorous Sports (kcal/week)	5070.25 ± 865	2582.02 ± 242	0.0004
Total Sports (kcal/week)	9505.17 ± 1192	5854.85 ± 442	0.0004
Total Physical Activity (kcal/week)	10971.54 ± 1271	6998.44 ± 454	0.0005
Sitting Time (hours/week)	42.03 ± 1.99	44.76 ± 1.34	NS
Borg CR-10 RPE (0-10)	6.90 ± 0.16	6.30 ± 0.13	0.004
Epworth Afternoon Fatigue (1-3)	1.96 ± 0.10	1.99 ± 0.07	NS

All models were adjusted for recruitment location, age and sex using a one-way analysis of covariance (ANCOVA). The Sidak method was used post-hoc to account for multiple comparisons. RPE = Rate perceived exertion.

Table 3. Significant associations of *ADRB1* and *ADRB3* genotypes with subject characteristics and physical activity patterns in our original (A) verification (B) and combined AIMMY cohorts. Shown are numbers for each genotype (N), adjusted mean values +/- SEM, and genotype *P* values.

A.

SNP	Cohort	Phenotype	*P*-Value	N; Adjusted Mean ± SEM
ADRB1 (rs1801253)	AIMMY (n=288)	Moderate Sports	0.040	CC (158; 3314.2 ± 42.4) CG/GG (130; 2141.3 ± 69.7)
ADRB3 (rs4994)	AIMMY (n=288)	Borg CR10	0.008	AA (255; 6.6 ± 0.1) AG/GG (33; 5.8 ± 0.3)
ADRB1 (rs1801253)	AIMMY (n=288)	Epworth Fatigue	0.050	CC (158; 1.9 ± 0.1) CG/GG (130; 2.2± 0.1)

B.

SNP	Cohort	Phenotype	*P*-Value	N; Adjusted Mean ± SEM
ADRB1 (rs1801253)	AIMMY (n=207)	Moderate Sports	0.020	CC (79; 3910.9 ± 22.3) CG/GG (128; 3161.6 ± 39.1)
ADRB3 (rs4994)	AIMMY (n=207)	Borg CR10	0.070	AA (158; 6.3± 0.1) AG/GG (49; 5.8 ± 0.1)
ADRB1 (rs1801253)	AIMMY (n=207)	Epworth Fatigue	0.060	CC (79; 1.8 ± 0.1) CG/GG (128; 2.0± 0.1)

C.

SNP	Cohort	Phenotype	*P*-Value	N; Adjusted Mean ± SEM
ADRB1 (rs1801253)	AIMMY (n=495)	Moderate Sports	0.030	CC (237; 3512.8 ± 49.7) CG/GG (258; 2736.8 ± 42.4)
ADRB3 (rs4994)	Females (n=229)	Borg CR10	0.050	AA (188; 6.4 ± 0.1) AG/GG (41; 5.8 ± 0.1)
ADRB1 (rs1801253)	AIMMY (n=495)	Epworth Fatigue	0.057	CC (237; 1.9 ± 0.1) CG/GG (258; 2.1± 0.1)
ADRB1 (rs1801253)	AIMMY (n=495)	VO_2 max	0.040	CC (237; 43.1 ± 0.9) CG/GG (258; 39.8 ± 1.3)
ADRB3 (rs4994)	Females (n=229)	Triglycerides	0.011	AA (188; 85.2 ± 2.9) AG/GG (41;101.5 ± 8.9)
ADRB3 (rs4994)	Females (n=229)	HOMA-IR	0.047	AA (188; 1.2 ± 0.1) AG/GG (41; 1.5 ± 0.2)

We used a dominant genetic model for genotype association with age and sex as possible covariates with the exception of B, where sexes were compared. All comparisons were made with 1-way analysis of covariance (ANCOVA) with the Sidak method for post-hoc multiple comparisons adjustment.

Figure 1. Genotype distributions of ACTN3, ADRB1 and ADRB 3 gene variants in kinesiology majors (KNES) and non-majors (NON) from our AIMMY cohort of healthy young college students.

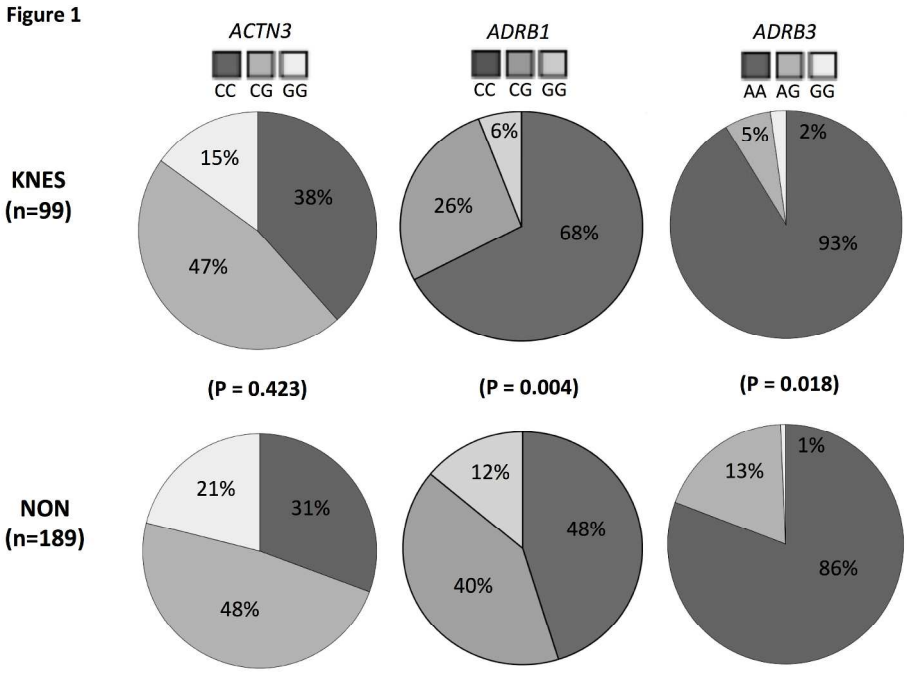

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Figure 1
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