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**Original Article** 

# Prospective associations between sedentary time, sleep duration and adiposity in adolescents



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#### ABSTRACT

*Objective:* The objective of this study was to investigate whether objectively measured sedentary time and sleep duration are associated with changes in adiposity from mid- to late adolescence.

*Methods:* Students (n = 504, 42% boys) were recruited from schools in Cambridgeshire, UK. At baseline (mean age  $15.0 \pm 0.3$  years), sedentary time was objectively measured by  $\geq 3$  days of combined heart rate and movement sensing. Concurrently, sleep duration was measured by combined sensing in conjunction with self-reported bed times. Fat mass index (FMI; kg/m<sup>2</sup>) was estimated at baseline and follow-up ( $17.5 \pm 0.3$  years) by anthropometry and bioelectrical impedance. FMI change ( $\Delta$ FMI) was calculated by subtracting the baseline from follow-up values. Linear regression models adjusted for basic demographics, moderate-to-vigorous physical activity (MVPA), and depressive symptoms were used to investigate associations of sedentary time and sleep duration (mutually adjusted for one another) with  $\Delta$ FMI.

*Results:* FMI increased by 0.5 and 0.6 kg/m<sup>2</sup> in boys and girls, respectively, but there was no association between sedentary time and  $\Delta$ FMI in either gender ( $p \ge 0.087$ ), and no association between sleep duration and  $\Delta$ FMI in girls ( $p \ge 0.61$ ). In boys, each additional hour of baseline sleep significantly reduced the  $\Delta$ FMI by 0.13 kg/m<sup>2</sup> (p = 0.049), but there was little evidence for this association after adjusting for MVPA and depressive symptoms (p = 0.15).

*Conclusions:* Sedentary time may not determine changes in adiposity from mid- to late adolescence, nor may sleep duration in girls. However, sleep length may be inversely associated with adiposity gain in boys, depending on whether the relationship is confounded or mediated by MVPA and depression.

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#### 1. Introduction

Prolonged sitting is highly prevalent in modern society [1], and it is composed of numerous diverse behaviours many of which (eg, seated reading, writing, and screen viewing) but not all (seated cycling or rowing) contribute to the total time spent sedentary. There is some evidence that a secular trend of increasing sedentary time

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has coincided with continuing emergence of the obesity epidemic [2,3], leading to the conjecture that a causal relationship may exist. However, most aetiological investigations of sedentary time and obesity have measured TV-viewing duration only, which is an imperfect and unrepresentative proxy of the total time spent sedentary [4,5]. More studies are needed to investigate the total sedentary time, also measured more precisely by objective instead of self-report methods, and its association with obesity in youth, particularly as doubts have arisen as to whether an association exists for total sedentary time that is independent of physical activity [6].

Like sedentary time, sleep is a state of rest that involves immobile posture and low energy expenditure. However, it is further characterised by reversible complete or partial loss of consciousness and responsiveness to external stimuli. Adequate sleep is essential for normal growth, development and functioning in youth [7–9]. It is therefore concerning that sleep durations have declined over the 20th century by >1 h/night [10]. Adolescents are

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Abbreviations: FM, fat mass; FFM, fat-free mass; FFMI, fat-free mass index; FMI, fat mass index; METs, metabolic equivalents of thermogenesis; MVPA, moderate-to-vigorous physical activity; PAEE, physical activity energy expenditure; SES, socio-economic status;  $\Delta$ FFMI, change in fat-free mass index;  $\Delta$ FMI, change in fat mass index.

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currently failing to reach sleep targets [9], and the prevalence estimates for adolescent fatigue have increased since the mid-1980s [11]. Speculatively, short sleep durations may be implicated in the aetiology of obesity. Supporting this theory, laboratory studies in adults have shown that restricted sleep deregulates endocrine secretions, causing decreased leptin and increased ghrelin, which may predispose to obesity through increased energy intake [12]. Physical activity, sedentary time, and depression have also been proposed as mediating factors [13]. However, the strength of evidence directly linking sleep length to obesity in adolescence is currently limited. The literature is dominated by cross-sectional studies, measuring sleep duration with non-validated questions and utilising body mass index (BMI) as an outcome [14,15].

The purpose of this prospective study was to incorporate objective measurements of total sedentary time and sleep duration, to investigate whether they are independently and oppositely associated with changes in body fatness from mid- to late adolescence.

#### 2. Subjects and methods

#### 2.1. Study design

Participants were from schools registered in the ROOTS study, an observational cohort study based in Cambridge, UK [16]. Schools (n = 18) were recruited from a geographical perimeter surrounding the city and extending to surrounding villages. Students within schools were eligible to take part if they were aged 14 years. At the start of the study (wave 0), 1238 students gave written informed consent to participate, with 1203 students (45% boys) eventually attending for data collection. Details about basic demographics (date of birth, gender, ethnicity and postcode to indicate socio-economic status, SES) and pubertal development were self-reported, and anthropometrics and body composition were also measured. Six months later (wave 1, from here on referred to as baseline), 930 students (43% boys) were seen for a second measurement of anthropometry and body composition, and at this time habitual activity monitoring was performed. Approximately 2.5 years later (wave 2, here referred to as follow-up), 844 students (44% boys) attended for the third and final measurements of anthropometry and body composition. All stages of the ROOTS project were approved by the Cambridge Local Research Ethics Committee (reference number 03/302) and were conducted in agreement with the Declaration of Helsinki guidance.

#### 2.2. Anthropometry and body composition

Detailed physical measurements were made by trained personnel at all waves using identical procedures and instrumentation. Height was measured to the nearest 0.1 cm (Leicester Height Metre; Invicta Plastics, Leicester, UK) whilst barefoot, and weight was measured to the nearest 0.1 kg in light clothing (Tanita TBF-300 MA, Tanita, Tokyo, Japan) using standard procedures. BMI (kg/m<sup>2</sup>) was calculated and body tissue impedance ( $\Omega$ ) was measured by bioimpedance (Tanita TBF-300 MA). Subsequently, child-specific equations [17] were used to derive multiple estimates of fat mass (FM, kg) and fat-free mass (FFM, kg) by utilising the data on height, weight, BMI, and body tissue impedance (values from equations predicting total body water were converted to FFM and FM using ageand sex-specific data on the hydration of lean tissue [18]). All permutations were pooled alongside body composition measured by the Tanita TBF-300 MA to produce aggregated measures of FM and FFM [19], which were expressed relative to height-squared (fat mass index (FMI),  $kg/m^2$ ) and height raised to the power of 2.5 (fatfree mass index (FFMI), kg/m<sup>2.5</sup>), respectively [20].

#### 2.3. Sedentary time, sleep duration, and physical activity

Habitual activity was measured objectively by combined heart rate and movement sensing (Actiheart, CamNtech Ltd, Papworth, UK) at wave 1. A detailed description of the monitoring protocol can be found elsewhere [17]. In brief, following a graded sub-maximal step test to establish individual calibration of heart rate, the sensor was initialised to record data every 30 s and was worn by participants without interruption for up to four consecutive days, including a weekend. Data from participants who had worn the sensor for at least 32 h on weekdays and 16 h on weekend days were considered useable, with a further proviso that these hours were distributed across the 24-h period (thereby providing the equivalent of  $\geq$ 3 days and nights of activity monitoring).

Information collected during the free-living period were preprocessed [21] and an activity intensity (J/min/kg) estimate made by a branched equation framework [22]. Upon summarising the data, potential bias caused by non-wear periods (segments of nonphysiological data) was minimised [23] and the resulting timeseries data were collapsed into physical activity energy expenditure (PAEE, kJ/kg/day) and time spent in different intensity categories. Standard metabolic equivalents of thermogenesis (METs) were used to establish the time spent sedentary ( $\leq$ 1.5 METs) and in moderateto-vigorous physical activity (MVPA, > 4.0 METs). To assist separation of sleep and sedentary time, participants were asked to report the times that they usually went to bed and got up on school days and weekend days separately, as defined in the Sleep Habits Survey for Adolescents, which has been validated against sleep diary and actigraphy [24]. This self-reported information was overlaid on the habitual activity data to provide a region of interest within which to identify objective markers of sleep onset (considered the beginning of prolonged minimal movement accompanied by a decline in heart rate) and termination (movement initiation together with an abrupt increase in heart rate); the self-reported sleep data were subsequently adjusted commensurate with these objective sleep indicators by reviewer visual inspection [17]. A single researcher reviewed all activity plots whilst blinded to all other participant characteristics, and every occurrence of time spent  $\leq$ 1.5 METs was allocated either a sleep (=1) or awake (=0, ie, sedentary time) score depending on whether the behaviour fell within or outside of a designated sleep phase.

#### 2.4. Other covariates

Home postcodes were used to create an area-level SES indicator that was collapsed from five original groups [25] to three categories: low (hard-pressed and moderate means), middle (comfortably off), and high (urban prosperity and wealthy achievers).

Puberty status (dichotomised as pre- or post-pubertal) was determined by features including self-reported menarcheal status, selfreported Tanner stages and objective levels of salivary testosterone. Girls who were post menarche at wave 0 were considered pubertal, as were pre-menarcheal girls reporting advanced signs of puberty (pubic hair or breast development  $\geq$ 3 of the Tanner scale) [26]. All remaining girls were defined as pre-pubertal. Boys who reported pubic hair coverage and genital development  $\geq$ 4 of the Tanner scale were classified as post-pubertal, whereas boys reporting stages  $\leq$ 2 on both axes were regarded as pre-pubertal. All remaining boys were categorised according to salivary testosterone levels, with those whose level was  $\geq$ 25th percentile of the distribution from the pubertal group being labelled post-pubertal.

Students at wave 0 completed the Mood and Feelings Questionnaire (MFQ), a 33-item self-report scale eliciting information about depressive symptoms occurring in the previous fortnight. The MFQ has been validated as a screening tool for clinical unipolar depression in adolescents [27]; higher summed MFQ scores are indicative Download English Version:

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