# Temporal relationships among physical activity levels and stationary time in preschool-aged children and adolescents: An investigation of the ActivityStat hypothesis

by

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## ABSTRACT

The ActivityStat hypothesis suggests that movement can be internally regulated by a biological control center. Specifically, humans maintain their total physical activity (PA) at a constant level by triggering behavioural and/or energy compensatory responses. However, there is considerable debate about the possibility of a compensation mechanism. Thus, the original intent of this dissertation was to test the hypothesis in an intervention involving adults living with liver disease. For **Study 1**, a scoping review was conducted addressing the following objectives: (1) to assess whether individuals living with liver disease display behavioural (i.e., PA, sedentary-related behaviour, and sleep) and/or energy (i.e., total energy expenditure [EE], basal EE, resting EE, and activity EE) compensation throughout the day and/or days; and (2) to examine whether a prescribed PA intervention triggers compensatory responses. Findings revealed insufficient evidence to support the ActivityStat hypothesis throughout the day and/or across days in people living with liver disease. Additionally, findings related to the effects of a PA intervention on behavioural and energy responses do not support the hypothesis. Due to interruptions related to the COVID-19 pandemic, I switched the focus of my dissertation to children and adolescents. These studies investigated the ActivityStat hypothesis by examining the presence of continuous-time multivariate relationships and the timeframe during which the multivariate relationships existed between light PA (LPA), moderate-to-vigorous PA (MVPA), and stationary time (ST). In **Study 2**, a secondary analysis was performed on data from the Parent-Child Movement Behaviours and Pre-School Children's Development project involving preschool-aged children (3 to 5 years old) in Edmonton (Canada). Participants wore accelerometers to assess PA levels (i.e., LPA and MVPA) and ST. Though positive continuous-time relationships were found within LPA, MVPA, and ST, no relationships were observed between the behaviours after accounting

for all other dynamic relationships (e.g., within-behavioural) examined. The timeframe at which LPA, MVPA, and ST predicted their later behaviours was about 0.5 days later. In conclusion, findings do not support the behavioural compensation component of ActivityStat hypothesis in preschool-aged children when simultaneously taking into account all dynamic relationships among LPA, MVPA and ST. In **Study 3,** a secondary analysis was performed on data from the Built Environment and Active Transport to School (BEATS) study involving adolescents (13 to 18 years old) in Dunedin (New Zealand). Participants wore accelerometers to assess PA levels and ST. Increases in LPA, MVPA, and ST were positively associated with their later behaviours until 1.7 to 2.5 days. A cross-behavioural reciprocal and negative relationship between LPA and ST was demonstrated 0.4 days later. A positive relationship between ST and MVPA was observed until about 0.4 day later. Though behavioural compensation was not observed for LPA and MVPA, evidence of compensation was noted for ST. Thus, findings from this study support evidence of the hypothesis in adolescents. Overall, this dissertation provides novel insights on examining the continuous-time multivariate relationships among PA levels and ST to investigate the ActivityStat hypothesis in different populations.

# PREFACE

This dissertation includes six chapters describing the results from three studies examining the ActivityStat hypothesis in different populations. Chapter 1 presents a general introduction of my research topic and study objectives. Chapter 2 discusses the rationale of the studies, the knowledge gaps, and the purpose of the dissertation.

Chapter 3 (Study 1) reviews the presence of compensatory mechanisms throughout the day and/or days, and in response to a prescribed physical activity intervention in individuals living with liver disease. I was responsible for conceptualizing the review, creating a codebook, conducting data extraction, data analysis and synthesis, and writing the manuscript. A. P. McCurdy, Y.-B. Kim, C. Lindeman, and A. J. Mangan completed documents screening and reviewing, systematic data extraction; and critically revised the manuscript. A. Sivak acquired the data. D. Mager assisted on the development of data extraction protocol and provided scholastic view. J. C. Spence was the supervisory author and was involved with concept formation and manuscript composition. All authors read and approved the final manuscript.

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Chapter 4 (Study 2) and Chapter 5 (Study 3) report on analyses using secondary data from the Parent-Child Movement Behaviours and Pre-School Children's Development project and Built Environment and Active Transport to School (BEATS) study, respectively. These studies investigated the presence and the timeframe of multivariate relationships among physical

activity levels and stationary time in preschool-aged children and adolescents, in continuous time. Both studies attained ethics approval from the University of Alberta Research Ethics Board (Study 2, Pro000117335, and Study 3, Pro00119474). For both Study 2 and 3, I was responsible for conceptualizing the studies, preparing the datasets, conducting statistical analyses, and writing the manuscripts. In Study 2, G. R. Ruissen was responsible for assisting on data analysis and interpretation, writing review and editing. N. Kuzik and V. Carson are the main investigators of Parent-Child Movement Behaviours and Pre-School Children's Development project, they were responsible for its conception, recruitment, and data collection. They also provided scholastic views of Study 2. J. C. Spence was the supervisory author and was involved with concept formation and manuscript composition. In Study 3, G. R. Ruissen was responsible for assisting on data analysis and interpretation, writing review and editing. S. Mandic and E. G. Bengoechea are the main investigators of the BEATS study, they were responsible for its conception, recruitment, and data collection. They also provided scholastic views of Study 3. J. C. Spence was an investigator and the supervisory author and was involved with concept formation and manuscript composition. All authors read and approved the final manuscript.

Chapter 6 presents a general discussion and conclusion for the dissertation.

# DEDICATION

To Paulo Andriola and my 2-year old son Noah Lamboglia Andriola.

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# TABLE OF CONTENTS

# LIST OF TABLES

# LIST OF FIGURES

# GLOSSARY

*Children and adolescents*: Individuals aged 5-17 years (World Health Organization [WHO], 2020).

*Correlates*: A variable that is associated with or predicts an outcome of interest (e.g., physical activity; Bauman et al., 2002).

*Determinant*: A causal factor that changes an outcome of interest (e.g., physical activity; Bauman et al., 2002; Atkin et al., 2016).

*Energy expenditure*: The amount of energy the body uses to maintain its functions (Ainsworth et al., 2011).

*Movement behaviour*: A pattern of activities (i.e., sleep, sedentary behaviour, physical activity) in a 24-hour period (Tremblay et al., 2016).

*Physical activity*: Physical movements involving energy expenditure (Ainsworth et al., 2011).

*Sedentary behaviour*: A sitting or reclining position and low energy expenditure (Tremblay et al., 2017).

*Stationary behaviour*: Any waking behaviour with no ambulation (Tremblay et al., 2017).

*Stationary time*: Time spent in stationary behaviours (Tremblay et al., 2017).

*The ActivityStat hypothesis*: The idea that individuals who increase or decrease physical activity to a certain level may experience compensatory responses (i.e., energy, behavioural) to maintain a stable energy expenditure over time (Gomersall et al., 2013).

*Young children or preschool-aged children:* Children aged under 5 years old (WHO, 2019).

## ACRONOMYNS

BCI = bayesian credibility intervals

BEATS = Built Environment and Active Transport to School

CNS = central nervous system

CT-SEM = continuous-time structural equation models

EE = energy expenditure

IPAQ = International Physical Activity Questionnaire

LPA = light physical activity

MB = movement behaviour

METs = metabolic equivalents

MVPA = moderate-vigorous physical activity

NAFLD = non-alcoholic fatty liver disease

NEAT = non-exercise activity thermogenesis

$N_{eff}$ = effective sample size

PA = physical activity

PRISMA-ScR = Preferred Reporting Items for Systematic reviews and Meta-Analyses extension for Scoping Reviews

SB = sedentary behaviour

SD = standard deviation

ST = stationary time

WHO = World Health Organization

YPAP = Youth Physical Activity Promotion model

# CHAPTER 1: Introduction

## General introduction

The ActivityStat hypothesis supports the idea that humans have biological mechanisms that regulates how much individuals engage in physical activity (Gomersall et al., 2013). It is hypothesized that an internal biological control center is activated when total energy expenditure reaches its set point triggering a cascade of compensatory mechanisms to return the system back to its steady state (Gomersall et al., 2013; Pontzer, 2018; Rowland 1998). Specifically, an increase or decrease in physical activity (PA) greater than a certain set point at one time activates energy and/or behavioural compensatory responses at another to maintain total energy expenditure stable overtime. The ActivityStat hypothesis is supported by the constrained total energy expenditure model, which suggests that the compensation phenomenon is a physiological mechanism that keeps energy requirements in check while prioritizing and allocating energy for reproductive fitness and survival (Pontzer, 2015; Pontzer, 2018).

To investigate the ActivityStat, studies have examined regular temporal variation in spontaneous PA within- and/or between-days, and/or in response to PA programs or interventions (Beck et al., 2022; Swelam et al., 2022). For instance, children and adolescents (7 to 18 years old) who had an active morning commute subsequently walked less throughout the day (Tan et al., 2018). Further, an increase in 10 minutes of moderate-vigorous intensity PA (MVPA) on one day resulted in a reduction of 9.3 minutes of MVPA and 16.8 minutes of light PA (LPA) the following day in children 8 to 11 years old (Ridgers et al., 2018). Similarly, adolescents demonstrated compensatory behaviours by reducing MVPA and increasing sedentary time after a moderate-vigorous intensity exercise session (Paravidino et al., 2017).

Even though some evidence exists in support of the ActivityStat hypothesis, limited research has been done on this topic in clinical populations, preschool-aged children and adolescents. Further, studies in this area have made use of traditional longitudinal multilevel models that present important limitations when investigating the ActivityStat hypothesis. Specifically, these traditional methods can only test univariate models (Ruissen et al., 2022) and are limited in providing a complete picture of how compensation may unfold over time. Therefore, the use of a novel approach known as the continuous-time structural equation modeling (CT-SEM) may comprehensively examine and discuss further insights regarding the ActivityStat hypothesis lacking in the present literature.

**Purpose**

The initial purpose of this dissertation was to examine the ActivityStat hypothesis in individuals living with liver disease. However, due to COVID-19-related restrictions in accessing clinical populations and patient groups, I switched the focus of my dissertation to preschool-aged children and adolescents and conducted secondary analysis of existing data.

**Objectives**

The specific objectives of this research were:

**Objective 1:** Conduct a systematic review to assess whether individuals living with liver disease display behavioural and/or energy compensation within the day and/or between days, and to examine whether a prescribed PA intervention triggers compensatory responses.

**Objective 2:** Examine the presence and the timeframe of the multivariate relationships among PA levels and stationary time (ST) in preschool-aged children using CT-SEM.

**Objective 3:** Investigate the presence and the timeframe of the multivariate relationships among PA levels and ST in adolescents using Bayesian CT-SEM.

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# CHAPTER 2: Literature Review

Humans are born to move. Being physically active throughout the lifespan is associated with life expectancy and well-being (Anokye, Trueman, & Green, 2012; Marker, Steele & Noser, 2018; Reimers, Knapp, & Reimers, 2012). Through movement and play, children explore and learn about the world which promotes healthy child development (Cliff et al., 2016). Adolescents should also engage in physical activity (PA) regularly to promote positive effects on health and to prevent noncommunicable diseases (Cliff et al., 2016). Prolonged sedentary behaviour (SB), on the contrary, are recommended to be minimized due to negative health consequences (e.g., metabolic syndrome, cardiovascular diseases; Carson et al., 2016).

PA is defined as any physical movement involving energy expenditure (EE) (i.e., the amount of energy a body uses to support daily functions) higher than 1.5 metabolic equivalents (METs) divided into light PA, moderate PA, and vigorous PA (Ainsworth et al., 2011; Tremblay et al., 2017). In children and adolescents, PA includes activities such as active leisure time/play, school physical education, active transportation, and organized sports. SB is defined as a combination of two different factors: metabolic rate (i.e., level of EE) and behavioural topography (posture) (Marshall & Merchant, 2013). Thus, SB refers to waking activities done at low EE (i.e., $\leq$ 1.5 METs) while sitting, reclined, or in a lying posture (Tremblay et al., 2017). "Sedentary time" refers to the time spent in SB (Tremblay et al., 2017). "Stationary behaviour", another term, which is becoming more commonly used by movement behaviour researchers, refers to non-ambulatory waking behaviours unrestricted by body posture and EE. Stationary time (ST) refers to the time spent in stationary behaviours (Tremblay et al., 2017). This term allows researchers to study SB with devices that detect motion and are limited to reliably estimate body posture (Edwardson et al., 2016).

This chapter will summarize the PA and SB public health guidelines and present evidence of prevalence meeting PA and SB guidelines, discuss measurements and correlates of PA and SB, and examine the literature investigating the ActivityStat hypothesis in preschool-aged children and youth.

**Guidelines and Prevalence of Physical Activity and Sedentary Behaviour**

To experience the health benefits of physical activities, public health guidelines recommend preschool-aged children (3 to 4 years) accumulate 180 minutes of PA daily, including at least 60 minutes of moderate-to-vigorous PA (World Health Organization [WHO], 2019). Additionally, preschool-aged children should limit SB to no more than one hour, including sedentary screen time (WHO, 2019). Non-screen SB (e.g., storytelling with a caregiver, singing, drawing), in contrast, is encouraged as it shows beneficial associations with health and cognitive development (Carson et al., 2016; Horowitz-Kraus & Hutton, 2018; Sweetser et al., 2012; WHO, 2019). More recently, sleep has been incorporated into the guidelines reflecting emerging research and evidence of the important relationship with PA and SB. The guidelines recommend a range of 10-13h of sleep in preschoolers (WHO, 2019).

Children and adolescents (5 to 17 years) should be physically active throughout the day and achieve an average of 60 minutes of moderate-to-vigorous intensity PA each day (WHO, 2020). Additionally, it is strongly recommended that youth engage in vigorous-intensity aerobic activities, strengthen muscle and bone at least 3 days a week. To reduce the impact of SB on health outcomes, time spent being sedentary should be limited, especially recreational screen time (WHO, 2020). Even though many countries (e.g., Australia, Canada, New Zealand) already integrated the sleep component into the guidelines (Tremblay et al., 2016; Okely et al., 2022;

Oliver et al., 2022), this behaviour has yet to be incorporated into the WHO guidelines for this age group.

The prevalence of individuals meeting the guidelines globally is still insufficient (Guthold et al., 2018; Mclaughlin et al., 2020). A systematic review that examined the associations between meeting guidelines and multiple health indicators showed that between only 5% and 24% of preschoolers met all three behaviours (i.e., sleep, SB, and PA) (Rollo, Antsygina, & Tremblay, 2020). A more recent study reveals that there is still 8.81% of that population not meeting any of the three recommendations, representing 1 in 5 young people (Tapia-Serrano et al., 2022).

A global study reveals that children and adolescents have insufficient levels of PA, girls engage in lower levels of PA in comparison to boys, and PA level decreases with age (Aubert et al., 2021). According to the Global Matrix initiative, led by the Active Healthy Kids Global Alliance aiming to encourage PA in children and youth worldwide, among 49 countries, 75% of countries have an average grade of D or F for overall PA (Aubert et al., 2018). Slovenia is leading the ranking with overall PA graded as A-. Countries such as England and Hong Kong graded C-. Australia, Brazil, Canada, Germany, and The United States have their grade ranging from D- to D+. China and South Korean's overall PA were classified in one of the least successful countries with an average grade of F. Overall, a total of 81% and 83.7% of children and adolescents are not meeting PA guidelines (Guthold et al., 2020), and are spending four to 12 hours per day being sedentary (Pate et al., 2011) worldwide.

**Measurements of Physical Activity and Sedentary Behaviour**

There are a variety of methods available to assess PA and SB. Questionnaires and self-reports are considered indirect measures of these behaviours. The Child Physical Activity

Questionnaire (CPAQ, Corder et al., 2009) and Physical Activity Questionnaire for Older Children (PAQ-C) and Adolescents (PAQ-A) (Kowalski, Crocker & Donen, 2004) are examples of frequently employed questionnaires. Pedometers, accelerometers, inclinometers, and heart rate monitors are direct measures of PA and SB (Bakker et al., 2020; Loprinzi & Cardinal, 2011; Sylvia et al., 2014). For the purpose of this dissertation, the accelerometry method will be described in further detail.

**Accelerometry method.** Accelerometers have been used extensively in research to measure the magnitude of the body's acceleration; the latest version (i.e., triaxial accelerometer) assesses acceleration along three different axes (i.e., vertical, horizontal, and perpendicular) by providing "activity counts" per unit of time (Sylvia et al., 2014). Studies have shown that accelerometers are reliable devices to estimate the level of PA and/or PA energy expenditure when compared to the gold standard method (i.e., indirect calorimetry), and they are also useful tools to assess PA under free-living conditions (Aadland & Ylvisåker, 2015; Lynch et al., 2019).

The Actigraph, Actical, GENEActiv, and SenseWear are the most common accelerometer brands able to measure PA and SB (Doherty et al., 2017; Rosenberg et al., 2016). In the population of preschool-aged children and youth specifically, the majority of accelerometer studies have been conducted using the Actigraph (Cliff, Reilly, & Okely, 2009; Migueles et al., 2017). Because acceleration and force of gravity are both being measured, Actigraph devices can directly measure body movement and estimate body posture (ActiGraph Support Center, 2017).

For data collection protocols and processing criteria, decisions about device placement, sampling frequency, epoch length, wear-time criteria, and PA and ST cut-points have to be made in order to set up the device and process the data (Miguelis et al., 2017). The literature on

accelerometers has shown that these devices can be placed in many locations on the human body (e.g., arm, wrist, waist, thigh, ankle). Waist and wrist placements are the most common, and have been widely adopted in the literature (Miguelis et al., 2017). Participants are asked to wear accelerometers for several days, usually at least seven consecutives days, including weekdays and weekends. The sampling frequency is the frequency at which the sensor records acceleration data. It can range from 30 Hz to 100 Hz, with a higher sampling rate requiring more memory and power supply when conducting prolonged measurements (Miguelis et al., 2017). Epoch length relates to a specific time window in which counts are accumulated, and the lower epoch has been shown to be more suitable for young children since it captures very short bouts of movement (Altenburg et al., 2021). Criteria for wear-time validation should be established to flag non-wear periods. Conventionally, wear-time greater than ten hours per day is considered compliant and sufficient to distinguish wear-time from non-wear time in preschoolers and youth (Bingham, et al., 2016; Choi et al., 2011). The cut-points are important to classify the different intensity levels of PA and ST. Furthermore, the algorithm used to estimate different PA and ST patterns should be age-specific when conducting the data collection and processing criteria (Miguelis et al., 2017). Therefore, researchers should make these decisions based on validation/calibration studies. Specifically, cut-points developed by Pate (Pate et al., 2006) and Evenson (Evenson et al., 2008) are commonly used in preschool-aged children and youth. PA and ST can be determined based on accelerometer counts measurement (i.e., counts per minute) and/or by estimating the EE in METs (Carr & Mahar, 2012; Crouter, Churilla, & Bassett, 2006; Tremblay et al., 2017).

The advantage of using an accelerometer is that it measures PA and ST objectively, and it estimates the duration and the intensity of the activity in a free-living environment. On the other

hand, it has some limitations. It is costly and, does not specify the context of measurement (e.g., leisure, occupational, or transportation activities); moreover, the use of these devices in water-based activities has not been assessed (Skender et al., 2016) and the use of cut points to classify activity intensities across individuals presents limitations (Migueles et al., 2019). Lastly, even though the ActiGraph accelerometers worn on the waist are capable of postural classification during periods of inactivity (ActiGraph Support Center, 2017), these devices have poor accuracy for detecting static accelerations (e.g., standing posture; Chen et al., 2012; Edwardson et al., 2016).

## Correlates of Physical Activity and Sedentary Behaviour

Promoting PA and encouraging people to reduce SB have been a challenge for public health. Consistent with ecological models (Welk, 1999; Spence & Lee, 2003), physical and psychological capacity, physical and social environments, and motivation all impact how individuals engage in different levels of PA and in SB.

**Correlates of physical activity in children and adolescents.** Understanding the factors that influence individuals to engage in PA is important so that researchers and practitioners can better design and improve programs to reduce inactivity. Ecological models of health behaviour propose that various nested levels of intra-personal and extra-personal factors determine individuals' behaviour (Sallis & Fisher, 2008; Spence & Lee, 2003). Specifically, the ecological model of PA (EMPA) suggests that biological and psychological factors and environmental settings influence PA (Spence & Lee, 2003). More specifically, the youth physical activity promotion (YPAP) model endorses the use of ecological models to understand the factors that influence youth to be physically active and proposes a conceptual framework to promote PA in

this population (Welk, 1999). Thus, the biological, psychological, and environmental correlates of PA will be discussed in the context of children and adolescents.

Evidence shows that biological processes can influence PA (Lee et al., 2016; Rowland, 1988). For instance, 78% of PA and 72% of the activity associated EE in daily life are explained by genetic factors (Frank et al., 2005; Joosen et al., 2005). It has been hypothesized that variations in how individuals move within and between days may be explained by a biological set point that controls the amount of activity in which an individual engages to maintain an energy steady state (Gomersall et al., 2013). For instance, oscillations between high and low activity were observed in children while they were playing (Wade, Ellis, & Bohrer, 1973). More recently, an increase in moderate-vigorous PA (MVPA) in one day was associated with reducing the time engaged in that activity the following day, suggesting behavioural compensation (Ridgers et al., 2018). Additionally, biological factors may explain gender differences in the PA of children and adolescents and the decline in PA with aging. It has been demonstrated that girls are less active than boys (Aubert et al., 2021; Lee, Carson, & Spence, 2017), which may be due to an energy-conserving mechanism in response to growth and maturation in girls (Goran et al., 1998). However, investigations on how biological factors may influence the ways children and adolescents move around are still scarce (Beck et al., 2022; Thorburn & Proietto, 2000).

Regarding psychological influences, reviews of correlates have demonstrated that high perceived competence and high levels of self-efficacy are positively associated with PA in children and adolescents, respectively (Cortis et al., 2017; Martins et al., 2021). Likewise, physical self-concept (i.e., self-perception of strength and endurance) is strongly correlated with habitual PA (Schmidt et al., 2019). On the contrary, perceived barriers and depression were

negatively correlated with PA in children and adolescents, respectively (Sallis, Prochaska, & Taylor, 2000).

Physical ecology and physical topography can also influence PA. In a systematic review with meta-analysis, weather conditions such as rainfall, temperature, wind speed, sunlight, and humidity have been shown to be associated with PA in children and adolescents (Zheng et al., 2021). The majority of the studies reported negative associations between rainfall, wind speed, and humidity with overall PA. On the contrary, linear positive associations were found between higher temperatures and longer sunlight with overall PA and MVPA. Regarding the effects of air pollution on movement behaviour, a recent scoping review suggests that ambient air pollution may negatively impact an individual's PA (Kim et al., 2021). Additionally, studies conducted in China show that a decline in overall ambient air quality and an increase in particulate matter (i.e., $PM_{2.5}$) are associated with reduced outdoor leisure-time and active commuting (i.e., walking, running, and biking) (An et al., 2019). Measures to restrict outdoor activities to avoid exposure to ambient air pollution may negatively contribute to children and adolescents achieving the recommended levels of PA. For instance, less time spent outdoors is correlated with less participation in PA (Gray et al., 2015; Larouche et al., 2016). Furthermore, during COVID-19 pandemic and subsequent home confinement orders and restrictions for using outdoor spaces, it was found that children and youth were less active and showed a reduction in PA and participation in sports (Moore et al., 2020).

The built environment, neighborhood, and school features can also impact how children and adolescents engage in PA. Less traffic exposure, safety street/road features (i.e., pedestrian infrastructure for walking and cycling, shorter distances to facilities, and greater walkability), and proximity to greenspaces, parks, and recreational facilities encourage PA participation and

active travel (An et al., 2019; Nordbø et al. 2020). On the contrary, the number of roads to cross, traffic density/speed, and local conditions (i.e., crime, area deprivation) are negatively correlated with PA participation (Davison & Lawson, 2006). Lastly, active learning environment schools increase children's PA when compared with traditional school environments (Lanningham-Foster et al., 2008). In that study, Lanningham-Foster found the physical design of the school and the school's philosophy and values had to be restructured to encourage children to be more active throughout the day. As for the social environment, the PA of parents and friends can influence children's PA. One study demonstrated that an increase of 1,000 steps in parents' PA is associated with their children taking 260 additional steps (Stearns et al., 2016). Further, individuals' PA levels are influenced by their friends. Individuals who have friends with a high PA level can be influenced positively (Sawka et al., 2013).

**Correlates of sedentary behaviour in children and adolescents.** Researchers have extensively investigated SB related to screen-based activities, especially TV viewing, in the past (Salmon et al., 2011; Stamatakis et al., 2019). Recent scholarly consensus indicates that SB now has a consistent and universal terminology and should be investigated independently from PA (Katzmarzyk, 2010; Hamilton et al., 2008; Sedentary Behaviour Research Network, 2012). Due to these reasons, this area of study is relatively new and, therefore, theoretical approaches to understanding the influences on SB are limited (Spence, Rhodes, & Carson, 2017). Owen and colleagues (2011) propose a behaviour-specific ecological model of SB for adults adapted from a PA model. This model will be discussed here to describe the general factors that influence SB in children and adolescents. In this model, the multilayered influences on SB are described within different behaviour settings (i.e., household, leisure-time, transport, and occupation). In children and adolescents, more specifically, SB can occur at home, in transportation, at school, or in

childcare (Salmon et al., 2011), which may be influenced by the interaction between intra-personal and extra-personal factors.

At the individual level, biological factors can influence how children and adolescents engage in SB throughout the day (Lee, Carson, & Spence, 2017). The hypothesized existence of a biological set point that controls the amount of activity individuals engage in to maintain an energy steady state may explain why individuals increase SB after engaging in high intensity levels of PA. For instance, adolescents who engage in moderate and vigorous exercise showed an increase in sedentary time in the days following that exercise (Paravidino, Mediano, & Sichieri, 2017). In this case, it could be speculated that the exercise session promoted a sudden increase in the adolescents' PA levels which was greater than activity set points triggering a behaviour compensation on the following days. Additionally, it has been found that SB increases with age; adolescents spent significantly more time being sedentary compared to children (Van Sluijs et al., 2010). Further, poor weight status, higher waist circumference, and being male (among adolescents) are factors that correlate positively with SB in children and adolescents (LeBlanc et al., 2015a; LeBlanc et al., 2015b; Van der Horst et al., 2007). Regarding the psychological factors that influence SB in children and adolescents, it has been suggested that feelings of depression have a positive association with SB. On the contrary, self-esteem is negatively correlated with SB (Temmel & Rhodes, 2013).

Lastly, regarding extra-personal influences, having a television or computer in the child's or adolescent's bedroom is associated with higher SB (LeBlanc et al., 2015a; Temmel & Rhodes, 2013). Additionally, the number of cars in the household and the distance from home to school have been positively correlated with being driven to school (Wen et al., 2008) and, consequently, with more time spent sitting. Social environmental correlates positively with children's screen-

based SB including parents' model of travel to work, parents' attitudes to their child walking to school, and time spent by parents watching television, playing videogames, and surfing the internet (Wen et al., 2008; Temmel & Rhodes, 2013).

## The ActivityStat Hypothesis

**The concept.** It has been suggested that biological factors may modulate how much individuals move around. The ActivityStat is a hypothesis that supports the idea that humans have intrinsic mechanisms that regulate how much they engage in PA (Gomersall et al., 2013). This phenomenon was discussed decades ago by Rowland (1998), and it is now receiving more attention by the research community. It is hypothesized that an internal biological control center is activated when total EE reaches its set point triggering a cascade of compensatory mechanisms to return the system back to its steady state (Gomersall et al., 2013; King et al., 2007; Pontzer, 2015; Pontzer, 2017; Pontzer, 2018; Rowland, 1998). Specifically, an increase or decrease in EE beyond a certain set point activates behavioural and/or energy compensatory responses to maintain total EE stable, in homeostasis. This evolutionary mechanism keeps energy requirements in check while prioritizing and allocating energy to reproductive fitness and survival (Pontzer, 2015; Pontzer, 2018).

Homeostasis is an essential physiological mechanism present in every system of the body. According to Modell and colleagues (2015, p. 264), it is "the maintenance of a relatively stable internal environment by an organism in the face of a changing external environment and varying internal activity using negative feedback mechanism to minimize an error signal." An example of this mechanism can be found in the regulation of the core body temperature. More specifically, thermosensors detect changes in the external environment, and an "error detector" distinguishes any variation in the normal range of body temperature (i.e., 37°C) to the set point

value. The hypothalamus, which is the "controller" of this system, receives the information from the "error detector" and activates a negative feedback loop. Blood vessels, sweat glands (skin), and skeletal muscles, which are the "effectors," are activated to return the body temperature to normal values by altering peripheral resistance, heat gain/loss, sweat secretion rate, or shivering (Modell et al., 2015).

For the ActivityStat, researchers have discussed how the body regulates the amount of PA in which individuals engage and how this impacts EE. To understand this phenomenon, it is important to highlight the components involved. The regulated variable is a component that is maintained at a constant through an external perturbation of the system (Cabanac, 2001). In the ActivityStat context, total EE is hypothesized to be the variable that is regulated (Pontzer, 2015). Sensors are responsible for monitoring the system (Cabanac, 2001). The sensor of the ActivityStat may be the Central Nervous System (CNS), more specifically the hypothalamus, which plays an important role in the regulation of EE (Gomersall et al., 2013; Pontzer, 2015). The controlled variables are those that help the system return the regulated variable back to its set point (Cabanac, 2001). In that case, the literature has shown that behaviour and/or other energy conservation mechanisms vary to maintain a relatively stable EE (King et al., 2007; Gomersall et al., 2013). Figure 2.1 illustrates patterns of response in homeostatic systems and will be used to describe how the ActivityStat mechanism operates. For instance, a sudden increase in EE due to an external perturbation of the system (e.g., engaging in vigorous PA) greater than its set point activates the CNS. Consequently, regulatory behaviour and/or energy-related mechanisms are then activated to return EE back to its target state, within its set point (Gomersall et al., 2013). Similarly, because the ActivityStat is hypothesized to be symmetrical, long periods of inactivity may stimulate bodily movement to increase EE.

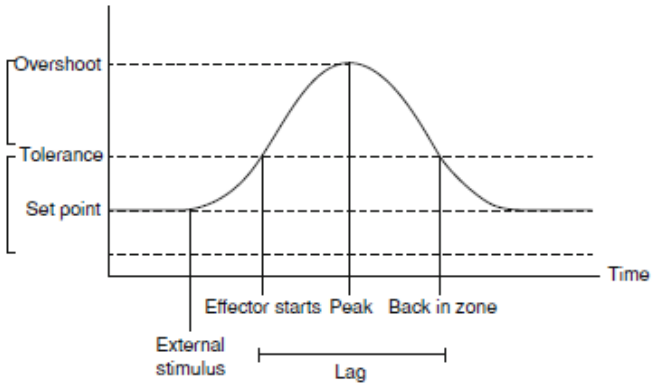


*Figure 2.1* Representation of a homeostatic mechanism response pattern

Source: Gomersall et al (2013).

The ActivityStat hypothesis is supported by the Constrained Total Energy Expenditure model proposed by Hermann Pontzer (2015a, 2018), which suggests that total EE is stable homeostatically and does not increase linearly with PA. That means "increasing PA has a limited effect on total EE and instead leads to a reduction in non-PA metabolic activity [i.e., on organ systems other than the musculoskeletal] to keep total EE near its set point" (Pontzer, 2015, p. 111). Figure 2.2 illustrates how the compensation mechanism is hypothesized in the context of the Constrained model and how it is related to individuals' lifestyle and health (Pontzer, 2018). Overall, as daily PA increases, other components of daily EE are reduced to maintain total EE stability. The body prioritizes expending energy from the nonessential expenditure components (e.g., inflammation, stress hormone levels, immune function, reproductive hormone levels). That demonstrates how PA has a positive impact on individuals' health. For instance, for those who have a more active lifestyle, the body may supress nonessential expenditures and thereby reduce the risk of a range of chronic diseases (e.g., cardiovascular disease, type 2 diabetes, cancer). In extreme situations, the body uses energy from essential expenditure which would negatively impact an individual's health. For example, elite athletes who engage in high intensity and

volumes of PA may have high a risk of infection, and may show the presence of amenorrhea and irregular menstrual cycling in females (Hackney, 2013; Melin et al., 2015). The curve for all-cause morbidity and mortality, consequently, is presented in a U-shaped format with PA. Thus, individuals who are sedentary experience a much higher risk of developing chronic diseases compared to those who are active.

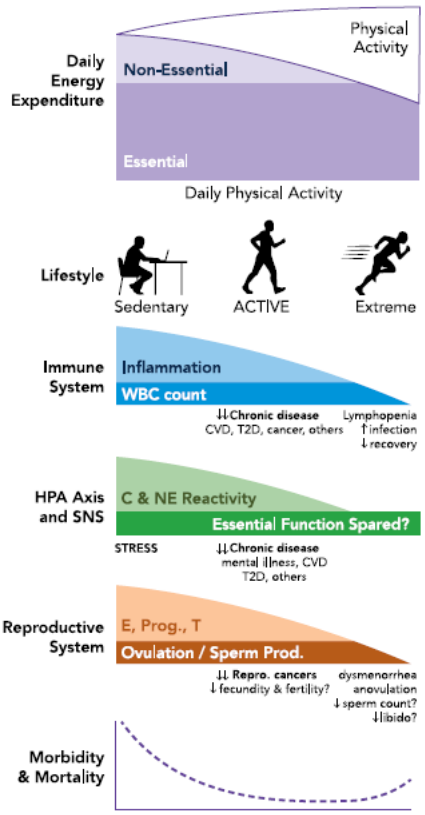


*Figure 2.2* Compensation mechanism in the context of the Constrained model

Source: Pontzer (2018).

**Behavioural and energy compensation.** The cascade of events that helps maintain the TEE in check can be classified as behavioural and/or energy-related (King et al., 2007; Pontzer,

2018; Rowland, 1998). Behavioural compensatory responses occur when PA is increased or decreased beyond a certain set point in one domain whilst compensatory change occurs in other domains (Gomersall et al., 2013; Rowland, 1998). For instance, an increase in MVPA in traveling to work (e.g., cycling) may increase sedentary behaviour (e.g., sitting) and/or decrease light PA (LPA, e.g., standing) during work-related activities. A classic example of behavioural compensation was illustrated in a study by Mansoubi et al. (2016). After the introduction of sit-to-stand workstations, office workers reduced sitting time and increased standing, stepping, and LPA during working hours. However, compensatory responses were observed in nonworking hours in which standing time (from 198±69 min/d to 136±50 min/d), stepping (from 71±31 min/d to 40±17 min/d) and LPA (from 96±29 min/d to 72±23 min/d) decreased after three months. Likewise, Nooijen et al (2018) also showed compensatory responses to changes in occupational activity levels on structured PA. Participants who had sedentary occupations and transitioned to active working hours were more likely to compensate with decreasing structured PA levels (OR= 1.22, 95% CI= 1.02–1.47). The opposite is also true for those workers who changed from active to sedentary types of occupations, who showed higher odds of increasing structured PA (OR = 1.21, 95% CI = 0.97–1.52) (Nooijen et al., 2018). The authors suggest that compensatory behaviours are more likely to occur in other domains because changes in work-related PA levels involve individuals performing these activities for many hours a day.

According to King and colleagues (2007), behavioural compensatory responses can be triggered by volitional and/or automatic mechanisms. The former relates to psychological processes in which individuals' intentions to be inactive can be triggered by compensatory health beliefs, fear of overexertion, deficient motivation, and perceived time barriers (Gray et al., 2018; King et al., 2007). For instance, elderly participants in a study by Gray et al. (2018) stated that

the risk of getting injured was a reason why they decreased spontaneous PA, "[…] there is only a certain amount your body can take, you don't want to overdo it as well and get injured that would be counterproductive" (p. 215). In children, feelings of boredom can influence how they engage in activity regardless of their previous movement behaviour. For instance, a child stated, "I don't know, it's just kind of boring being inside and just sitting down" (Swelam et al., 2023, p. 6). As for automatic mechanisms not under an individual's control, compensatory behaviour can be triggered by physiological reasons due to "a drive to be inactive" (Gray et al., 2018; King et al., 2007). In this scenario, a reduction in spontaneous PA can be caused by exercise-induced fatigue as a result of substrate depletion and/or delayed onset of muscle soreness (King et al., 2007). As an example, individuals explained subsequent compensatory behaviour due to being tired, saying that "I would usually [get] out walking in the morning, but for me the exercise, made it more difficult, I think because I was tired. . . I remember getting back and wanting to sleep" (Gray et al., 2018, p. 214). Potential physiological mechanisms for compensation have also been noted by children and their parents (Swelam et al., 2023). A child said, "I'm not really sure because sometimes I run too much and my legs start hurting" (p. 7), which was confirmed by his parent who said "… maybe he's probably a bit tired from the running around or his muscles are sore" (p. 7). However, more studies should be conducted to further understand how psychological and automatic factors impact compensation.

The energy-related compensatory mechanism is an automatic physiological response that is biologically determined. It tends to be a continuous process that is not under an individual's control, and is an evolutionary-based protective mechanism (Freese et al., 2018; King et al., 2007). Energy compensation occurs when an increase in PA expenditure leads to reductions in other expenditures; firstly, in non-essential EE components and, subsequently, in essential EE

components (Pontzer, 2018). Remarkable studies in this area have been published that examine the PA of Hadza hunter-gatherers. Though originally, these people expend more calories than Western people because of their more active lifestyle (i.e., hunting and gathering), Pontzer and colleagues (2012) found that their total EE did not differ among the populations studied. The Hadza individuals had greater daily walking distances not correlated with total energy expended. The energy they expended during walking accounted for only 6.7% and 11% of the total EE in females and males, respectively (Pontzer et al., 2012). Thus, Hadza individuals may compensate in other systems (e.g., reproduction, immune, digestive, musculoskeletal) to maintain a steady EE (Pontzer et al., 2015). Other studies investigated energy compensation in response to a structured PA program among adults. For instance, after an eight-week endurance PA program, no change was observed in total EE but a 62% reduction occurred in non-exercise EE (from 571 ± 386 to 340 ± 452 and kcal/day) (Goran & Poehlman, 1992). Similarly, Colley et al. (2010) found that non-exercise EE declined 22% after a walking group intervention. These results may suggest that the human body was compensating due to the high energy cost of structured PA programs.

**Evidence in children and adolescents.** Although findings from systematic reviews are inconclusive and do not support evidence of the ActivityStat Hypothesis (Beck, Engel & Reimers, 2022; Gomersall et al., 2013; Washburn et al., 2014; Lamboglia et al., 2022; Nigg et al., 2022; Silva et al., 2018; Swelam et al., 2022), some empirical studies involving children and adolescents show compensatory behaviours both within-days and between-days, and in response to prescribed PA interventions.

Compensatory within-day mechanisms were observed in a sample of children aged 7 to 18 years old. Specifically, those children who had an active morning commute mode walked less

throughout the day due to compensatory behaviour (Tan et al., 2018). Ridgers et al. (2015) observed children aged 8 to 11 years old during periods within weekend days, and found that every ten minutes of sitting during a period of the day was correlated with a reduction of 2.9 minutes sitting and an increase of 3.7 and 3.0 minutes standing and stepping, respectively, in the following period of the day.

Studies have demonstrated day-to-day PA and SB compensatory effects in children and adolescents. Findings published recently by Nigg and colleagues (2022) partially agree with the ActivityStat hypothesis. They found that two more hours of SB in any given day is associated with an increase of five minutes of MVPA the next day in youth between 6 to 17 years old. A study conducted by Ridgers and colleagues (2014) showed that for every additional ten minutes of MVPA engaged in on one day, LPA declined by 25 minutes on the following day in a sample of children (8 to 11 years). Likewise, an additional ten minutes of stepping on any given day was associated with fewer minutes of stepping (8.8 minutes) and standing (15 minutes) the next day (Ridgers et al., 2015). In addition, time spent sitting on one day was correlated with less time sitting and more time standing and stepping on another day (Ridgers et al., 2015). A more recent study reported similar findings in the same population in which an increase in ten minutes of MVPA one day resulted in a reduction of 9.3 minutes of MVPA and 16.8 minutes of LPA the next day (Ridgers et al., 2018). In young children from 4 to 9 years old, Wilkin et al (2006) noted that strong associations were found between weekday and weekend day activity, and daily activity from one year to another. Compensatory PA response was also observed when in-school and out-of-school activities were compared. Even though children who are driven to school showed 16.1% less PA before school and within the hour after school, the total weekly PA between those who were driven and those who walked did not differ. This means that PA in

those who were driven to school was compensated (91%) in another period of the day. In addition, findings from this study show that PA in children is independent of their environment, thus reinforcing the hypothesis of a biological regulation of PA (Wilkin et al., 2006).

A study examining compensatory effects of sedentary-related behaviour in healthy children and adolescents compared to those with cystic fibrosis also confirmed the above findings (Mackintosh, Ridgers, & McNarry, 2019). Specifically, a ten minute increase in sedentary time on one day was followed by a decrease of 3.1 minutes the next day in healthy youth. A compensatory mechanism was not confirmed in youth with cystic fibrosis (Mackintosh, Ridgers, & McNarry, 2019). Researchers speculate that the PA body regulation in the clinical population may display a different pattern from that of their healthy counterparts (Eisenmann & Wickel, 2009; Rowland, 1998).

Compensation effects have also been observed in response to prescribed PA interventions. A study of the influence of a single bout of structured PA was conducted to investigate the impact of that session on spontaneous activity in adolescents (11 to 13 years old) (Paravidino, Mediano, & Sichieri, 2017). It was found that the structured PA triggered an increase in the percentage of time spent in ST after the moderate ($\Delta$= +6.08%) and vigorous ($\Delta$= +4.30%) sessions compared to control conditions ($\Delta$= +1.52). Additionally, a significant decline in spontaneous moderate (after moderate condition, $\Delta = -5.43$, and after vigorous conditions, $\Delta = -4.44$, compared to the control condition, $\Delta = -1.77$) and vigorous (after vigorous condition, $\Delta = -1.45$, compared to moderate, $\Delta = -0.42$, and control conditions, $\Delta = -0.08$) intensity PA was observed. A study by Thivel and colleagues (2014) agreed with the above findings. It was observed that 12 to 15 year-old adolescents showed a compensatory response to an acute, high

intensity, structured PA session. Specifically, participants reduced nonexercise activity EE as a compensatory effect to offset the impact of structured PA on the total EE.

Even though there is some evidence of ActivityStat occurring in within-days and between-days (Mackintosh, Ridgers, & McNarry, 2019; Nigg et al., 2022; Ridgers et al., 2014; Ridgers et al., 2015; Ridgers et al., 2018; Tan et al., 2018; Wilkin et al., 2006) and in acute response to a prescribed PA intervention (Paravidino, Mediano, & Sichieri, 2017; Thivel et al., 2014), compensatory mechanisms are unlikely to occur within a short timescale (i.e., hours, within-day, and from day-to-day) (Gomersall et al., 2013). Instead, compensation is hypothesized to occur over a longer period (i.e., weeks or months). However, the lack of empirical research limits discussion of this topic.

**Gaps in the Literature and Link to Dissertation**

My dissertation addresses several current gaps in the literature investigating the ActivityStat. First, few studies have examined this phenomenon in patients with chronic diseases and, to my knowledge, no reviews have investigated compensatory responses in individuals living with liver disease. Additionally, compensatory mechanisms in clinical populations may display a different pattern from healthy populations (Eisenmann & Wickel, 2009; Rowland, 1998), thus the investigation into how this mechanism occurs is critically important to the advance of knowledge in this area. Second, though some evidence of the ActivityStat hypothesis exists in the youth population (Wilkin et al., 2006; Mackintosh, Ridgers, & McNarry 2019; Ridgers et al., 2014; Ridgers et al., 2015; Ridgers et al., 2018), the study of this phenomenon in specific age groups is critical as PA and SB show different patterns in preschool-aged children, children, and adolescents. Specifically, total EE increases throughout childhood and adolescence

due to metabolic demands of growth and development (Pontzer et al., 2021). Thus, the EE set point may vary with aging, which would impact when compensation occurs.

Additionally, the compensatory mechanisms of PA and SB in preschool-aged children and adolescents have been understudied, suggesting a new avenue of research. Third, the literature lacks empirical studies investigating the timeframe in which any supposed compensatory behaviours occur, thus, suggesting a research gap to be addressed in future studies. Lastly, to date, studies investigating compensatory mechanisms have mainly used traditional longitudinal multilevel models (e.g., hierarchical linear models, mixed models). Specifically, these traditional methods can only test univariate models (Ruissen et al., 2022), analyzing the relationship among the constructs in one direction or the other, disregarding the multivariate dependence among PA levels and SB. Further, because these traditional methods employ the discrete-time approach, findings from these studies are bound to a specific discrete time interval (Ruissen et al., 2022; Voelkle et al., 2018). Therefore, novel statistical techniques should be employed to study the ActivityStat hypothesis, as the traditional methods present important limitations (Schuurman et al., 2016) and findings from these studies should be interpreted with caution.

**Purpose and Data Sources**

The **initial purpose** of my dissertation research was to examine the ActivityStat hypothesis in patients living with liver disease. Thus, in **Study 1**, a scoping review was conducted to assess whether individuals living with liver disease display behavioural and/or energy compensation within the day and/or between days, and to examine whether a prescribed PA intervention triggers compensatory responses. However, due to COVID-19, my plans linked to that project had to be cancelled. For that reason, secondary data analyses were conducted to

investigate the same phenomenon in a different population. The **general purpose** of my dissertation was to examine the ActivityStat hypothesis in preschool-aged children (3 to 5 years old) and adolescents (13 to 18 years old). **Study 2** examined the presence and the timeframe of the multivariate relationships among PA levels and ST in preschool-aged children using Bayesian continuous-time structural equation models (CT-SEM). This study uses data from the Parent-Child Movement Behaviours and Pre-School Children's Development project (Kuzik et al., 2020) to examine the associations of parental movement behaviours and parent-child proximity with preschool-aged children's movement behaviours in Edmonton and surrounding areas, Canada. **Study 3** investigated the presence and the timeframe of the multivariate relationships among PA levels and ST in adolescents using Bayesian CT-SEM. This study uses a large dataset of adolescents attending schools in Dunedin, New Zealand, who are involved with the Built Environment and Active Transport to School (BEATS) Study (Mandic et al., 2015).

**Significance of the Research**

These studies provide insights into how individuals living with liver disease, preschool-aged children, and adolescents engage in activity levels throughout the days by investigating the ActivityStat hypothesis. This knowledge is important for the development and refinement of theory and can be used to inform future PA interventions.

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# CHAPTER 3: Study 1

## Investigation of movement-related behaviors and energy compensation in people living with liver disease: A scoping review

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## Abstract

The importance of integrated movement behaviors (MB, i.e., physical activity [PA], sedentary behaviour, and sleep) and their interdependence for health has been recently discussed in the literature. The proposition that the amount of time spent in any one of these behaviors may impact the amount of time spent in another is supported by the ActivityStat hypothesis. The aim of this review is to (1) to assess whether individuals with liver disease display MB and/or energy (i.e., total energy expenditure [EE], basal EE, resting EE, and activity EE) compensation throughout the day and/or days; and (2) to examine whether a prescribed PA intervention triggers compensatory responses. Documents were included if they focused on people living with liver disease; analyzed MB and/or EE components; were data-based; and were published in English. Fifteen documents were included in the final synthesis and 57 discrete findings were analysed. The one finding that addressed research objective 1 showed no compensatory response. As for research objective 2, most of the findings suggest no compensation effects in response to a PA intervention. There is insufficient evidence to support the ActivityStat hypothesis in people living with liver disease. Further research should be conducted to test this hypothesis using standardized methodological procedures.

## Introduction

The liver is one of the largest human organs and it is essential for digesting food and removing toxic substances from the body. Liver damage can be caused by genetic factors (e.g., Wilson's disease), viral infection (e.g., Hepatitis), alcohol abuse or excessive liver fat (e.g., Non-alcoholic fatty liver disease [NAFLD]). Repeated damage over time can lead to scarring (cirrhosis) and liver failure (Canadian Liver Foundation, 2013). The most common liver disease (i.e., NAFLD) has a global prevalence rate of 25% (Cotter & Rinella, 2020) and approximately two million people die annually due to a damaged liver condition (Younossi & Henry, 2016; Asrani et al., 2019). Despite posing a considerable challenge for public health (Younossi, 2019), liver disease tends to be disregarded in comparison to other chronic diseases (Marcellin & Kutalla, 2018). Individuals with chronic liver disease face physical deconditioning, sarcopenia, physical frailty, and impaired muscle health resulting in functional deterioration during advanced stages and increased risk of mortality (Lai et al., 2014; Montano-Loza, 2014; Tandon et al., 2016; Duarte-Rojo et al., 2018).

Physical activity (PA) is recommended for people living with liver disease as it can ameliorate functional degeneration (Tandon et al., 2018; Kwak & Kim, 2018). With the aim of fostering health for people of all ages, the World Health Organization recently released guidelines outlining evidence-based recommendations for PA and sedentary behavior (Bull et al., 2020). Youth (aged from 5 to 17 years old) should engage in at least 60 minutes of moderate-vigorous physical activity (MVPA) daily and limit sedentary behavior. For adults (aged 18 to 64 years), the guidelines recommend 150-300 minutes of MVPA, 75-150 minutes of vigorous PA, or a combination of both throughout the week. They should also reduce or substitute sedentary behavior with PA of any type of intensity (i.e., light, moderate, or vigorous). Older adults (aged

65 or older) should meet the same recommendations stated for adults including activities to heighten functional capacity (i.e., balance and strength training) to prevent falls. Studies in individuals living with liver disease have shown that incremental increases in incidental PA and structured exercise improve overall physical fitness, such as cardiovascular endurance, muscle strength and endurance, and balance (Román et al., 2014; Debette-Gratien et al., 2015). Enhanced physical fitness can improve independence in activities of daily living and quality of life in liver patients (Duarte-Rojo et al., 2018). Furthermore, a reduction in sedentary-related behavior (i.e., sitting, reclining or lying posture) is associated with a less liver fat (Bowden et al., 2019) and a decrease in insulin resistance (Sabinicz et al., 2016) among people living with liver disease. However, given reduced muscle mass and fatigue in people living with liver disease (Swain, 2006), meeting the guidelines can be difficult. Specifically, people living with NAFLD are more physically inactive than their healthy counterparts and those diagnosed with cirrhosis spend 76% of their waking hours sedentary (Hallsworth et al., 2015; Dunn et al., 2016).

Considering that the health benefits of a specific movement behavior (MB) in isolation may be misleading, a composition of MBs (i.e., PA of all intensities, sedentary behavior, and sleep) should be considered as it is associated with better overall health in people of all ages (Carson et al., 2017; Janssen et al., 2020; Kuzik et al., 2020). An integrated approach to MBs acknowledges their interdependence, meaning that a change (an increase or decrease) in the amount of time spent in any one of these behaviors may impact the amount of time spent in another. This mechanism, otherwise known as a compensatory response, is proposed by the ActivityStat hypothesis (Gomersall et al., 2013). It suggests that our bodies have a biological control center that regulates PA (Rowland, 1998). An increase or decrease in PA greater than a certain set point activates behavioral and/or energy compensatory responses (King et al., 2007;

Pontzer, 2018). For instance, a substantial increase in PA, especially at the vigorous level, may trigger a compensatory behavioral response resulting in reduced PA and/or increased sedentary-related behavior at another time (Mansoubi et al., 2016). As for energy compensation, in general, humans seek efficiencies in energy expenditure (EE) and regulate how they move around daily to that end (Pontzer, 2015). Therefore, an increase in activity EE greater than a specific set point may automatically activate a reduction in resting EE.

Although systematic reviews reveal that 46% to 71% of the articles analyzed showed no behavioral or energy compensatory responses (Gomersall et al., 2013; Washburn et al., 2014; Silva et al., 2018) several studies have demonstrated compensatory mechanisms in different populations. As an example, for every additional 10 minutes of MVPA engaged in on one day, light PA declined by 25 minutes on the following day in a sample of children (Ridgers et al., 2014). A more recent study reported similar findings in the same population in which an increase in 10 minutes of MVPA one day resulted in a reduction of 9.3 minutes of MVPA and 16.8 minutes of light PA (LPA) the next day (Ridgers et al., 2018). Adolescent boys classified as overweight also displayed compensatory behaviors after a single moderate-vigorous intensity exercise session, reducing MVPA and increasing sedentary time during the following six days (Paravidino et al., 2017). Regarding energy compensation, sedentary men living with overweight and obesity experienced a reduction in non-exercise activity thermogenesis (NEAT) in response to exercise (Herrmann et al., 2015). In Goran and Poehlman's study (1992), older individuals exposed to an endurance training program showed an average reduction in NEAT of 62%.

Compensatory responses have been extensively explored in healthy populations and adults living with overweight and obesity (Gomersall et al., 2013; Washburn et al., 2014; Silva et al., 2018). However, few studies examined this phenomenon in patients with chronic diseases

and, to our knowledge, no reviews have investigated compensatory responses in individuals living with liver disease. Therefore, we conducted a scoping review addressing the following objectives: (1) to assess whether individuals living with liver disease display behavioral (i.e., physical activity, sedentary-related behavior, and sleep) and/or energy (i.e., total EE, basal EE, resting EE, and activity EE) compensation throughout the day and/or days; and (2) to examine whether a prescribed PA intervention triggers compensatory responses.

## Methods

A scoping review was chosen as it offers an appropriate methodology for identifying gaps in the literature and is relevant for addressing research topics with limited or emerging evidence (Arksey & O'Malley, 2005). The procedures for this review were guided by established recommendations for scoping reviews (Arksey & O'Malley, 2005; Levac, Colquhoun, & O'Brien, 2010), and the Preferred Reporting Items for Systematic reviews and Meta-Analyses extension for Scoping Reviews (PRISMA-ScR) (Tricco et al., 2018). The protocol of this review was not registered in the International Prospective Register of Systematic Reviews (PROSPERO) because the registry does not currently accept scoping reviews, literature reviews, or mapping reviews.

Studies were included if they: (1) focused on people living with liver disease; (2) analyzed non-exercise physical activity, exercise, sedentary time, sedentary behavior, body posture, sleep time, total EE, basal EE, resting EE, or activity EE; (3) were data-based (quantitative or qualitative); and (4) were published in English. Both published and grey literature (e.g., dissertations) were eligible for inclusion and no restrictions were placed on the age of the participants or study design.

The search was conducted by an expert librarian (AS) and member of the research team in CINAHL, Cochrane, EMBASE, MEDLINE (Ovid), Pubmed, Scopus, SPORTDiscus, and Web of Science, using terms related to the key concepts 'liver disease', 'movement behaviours', and 'energy expenditure'. The search was completed on 11 February 2020. Dissertations & Theses Global and OCLC PapersFirst were also used to identify dissertations, theses, and conference papers/abstracts. Additional searching was conducted through other sources (e.g., Google Scholar) and by screening the reference lists of included documents (see complete search strategy in Appendix A).

The study selection was performed as follows. First, five reviewers screened titles and abstracts for eligibility. All documents were independently screened by two reviewers; the lead author reviewed all cases and the other four reviewers each screened 25%. Second, four reviewers independently assessed the remaining full-text articles for eligibility. During this process, all discrepancies were resolved through discussions, which allowed the opportunity to address challenges and uncertainties that arose during study selection review. The interrater reliability was moderate for title/abstract (Cohen's $\kappa = 0.57$) and full-text (Cohen's $\kappa = 0.46$) screening.

Data extraction was conducted using a codebook consisting of the following categories: characteristics of the document (i.e., author, year of study, and geographic location of the research); characteristics of the study (i.e., publication status, publication type, and design); characteristics of sample (i.e., sample size, age, sex, liver condition, and body mass index); characteristics of the methods for research objective one (i.e., group comparison, outcomes measured, and method used to assess MB and/or EE) and research objective two (i.e., PA intervention characteristics, diet intervention characteristics, intervention length, outcomes

measured, and method used to assess MB and/or EE); the effects of behavior and energy outcomes measured (i.e., overall PA, sedentary time or behavior, sleep, total EE, basal EE, resting EE, and activity EE); purpose; and findings. Compensation in MB or EE was considered to have occurred when sedentary time, sedentary behavior or sleep increased, or when overall PA, total EE, resting EE, and activity EE decreased throughout the day and/or days, or after prescribed PA. Two reviewers extracted data from the studies and subsequently met to compare the results and resolve discrepancies. A senior member of the research team was consulted in case of unresolved discrepancies. The interrater reliability ranged from moderate to perfect agreement (Cohen's $\kappa = 0.6$) for the document level coding and almost perfect agreement for the findings level (Cohen's $\kappa = 0.84$).

Data were coded and frequency counts were calculated for each categorical variable. The main findings were recorded as text and subjected to content analysis (Hsieh & Shannon, 2005). Specifically, the primary meaning of the text was categorized according to the differing purpose stated (i.e., overall PA, sedentary time or behavior, sleep, total EE, basal EE, resting EE, or activity EE). The direction of change (i.e., decrease, increase, or no change) for each MB and EE components were coded and the corresponding frequency was calculated. The coding and analyses were conducted from July to August 2020.

## Results

In accordance with PRISMA-ScR guidelines, Figure 3.1 presents a flow chart of the search and selection process. The initial search of databases (n = 3,907) and additional sources (n = 16) resulted in 3,923 potential includes. After removal of duplicates, 2,703 documents were considered for title and abstract screening; a further 2,417 documents did not satisfy the

inclusion criteria and were subsequently excluded. The remaining 286 documents were subjected to full-text review, which resulted in 15 documents and 57 discrete findings being included in the final analysis.

**Description of studies**

As detailed in Table 3.1, most of the documents were published (93.3%) in peer reviewed journals (93.3%), originated from Europe (40%), conducted interventions (80%), and focused on adult patients (60%) living with NAFLD (66.7%) and obesity (73.4%).

**Description of findings**

Of the total findings, 2% ($n_{findings}$ = 1) addressed research objective 1 and 98% ($n_{findings}$ = 56) were related to research objective 2. For research objective 1, the one finding compared MB (e.g., overall PA) in adolescents with NAFLD during days of the week and days of the weekend. No significant difference was observed between the number of steps taken on weekdays and weekends (Gibson et al., 2015). As for research objective 2, Table 3.2 shows a summary of the interventions and outcomes measured. Studies employed incidental PA or exercise interventions, ranging from one to 12 months, most of which included a dietary component. The majority of findings (76.8%; $n_{findings}$ = 43) were related to MBs responses and 22.2% ($n_{findings}$ = 13) to EE responses (Table 3.3).

Overall PA (88.3%; $n_{findings}$ = 38) and activity EE (69.3%; $n_{findings}$ = 9) were the outcomes predominantly investigated for MB and EE, respectively. In relation to the total, most findings within research objective 2 (94.6%, $n_{findings}$ = 53) suggest no compensation effects in response to a PA intervention (Table 3.3). For instance, “the average number of daily steps [...] was greater

after 3 months as compared with that at enrollment (5792 ± 2631 steps vs 4803 ± 3396 steps, p = 0.038)" (Hiraoka et al., 2017), whereas "the numbers of sedentary patients (those performing <2500 steps/day) decreased in the home-based physical activity program group (33% vs. 17%)" (Chen et al., 2020). Similarly, for EE "all our patients already had a basal minimal or null physical activity (METs ranging between 1.2 to 1.8) which, after a 3-month multidisciplinary intervention, increased by +9.5% [expressed both in number of daily steps (10,554 ± 4942 vs. 11,560 ± 4212), and in METs (1.50 ± 0.27 vs. 1.58 ± 0.25)] to a statistically non-significant extent" (Scaglioni et al., 2013). A compensation effect was identified in only 2.3% (i.e., increase in sleep, $n_{findings} = 1$) and 15.4% (i.e., decrease in total EE and resting EE, $n_{findings} = 2$) of the MB and EE findings (Table 3.3). For instance, "mid-day rest/naps (siesta) differed significantly in the Mediterranean lifestyle group (baseline, n = 13, 61.9%; after 6 months, n = 15, 71.4%) compared with the control group (baseline, n = 8, 38.1%; after 6 months, n = 7, 33.3%, after adjusting for baseline values" (Katsagoni et al., 2018) and total and resting EE decreased significantly, after a lifestyle intervention in participants with NAFLD (Rachakonda et al., 2019).

## Discussion

This scoping review summarizes evidence on movement-related behavior and energy compensation in people living with liver disease. We asked two questions: (1) whether individuals with a liver condition show MB and/or energy compensation throughout the day and/or across days, and (2) whether compensation occurs in response to a prescribed PA intervention. The reviewed documents mainly examined PA and diet interventions for adults living with NAFLD. It is not surprising that most of the documents investigated people living with NAFLD given the high prevalence of this condition. The global number of NAFLD cases

has increased from 391.2 million (8.2%) in 1990 to 882.1 million (10.9%) in 2017 (Ge X et al., 2020). Furthermore, the participants in the studies were predominantly living with overweight and obesity which reflects the pathogenic association between excessive body mass index (BMI) and NAFLD (Mitra & Chowdhury, 2020).

Regarding our first objective, almost no research examined whether MB and/or energy compensation occurs throughout the day or across days in people living with liver disease. Only one finding addressed research objective 1 and it showed no compensatory movement patterns in a sample of children with NAFLD (Gibson et al., 2015). Studies done in healthy children, on the contrary, have shown day-to-day variation in MBs demonstrating an interdependence among them (Kuzik et al., 2020). For instance, an independent increase in light PA and/or moderate PA on one day is associated with a decrease in those behaviors on the following day (Ridgers et al., 2014; Ridgers et al., 2018). In 2019, Mackintosh and colleagues (2019) first addressed day-to-day MB variability in children with cystic fibrosis and found a compensatory effect only for sedentary-related behavior. Specifically, a 10-minute increase in sedentary time on one day was followed by a decrease of 3.1 minutes the next day. Researchers speculate that compensatory mechanisms in clinical population may display a different pattern from healthy populations (Rowland, 1998; Eisenmann & Wickel, 2009). Further, it is presumed that these compensation effects may take longer to emerge in clinical populations and that biological, psychological, and physical environment-related factors play an important role in shaping individuals' PA and EE set points (Eisenmann & Wickel, 2009). However, further research is needed to understand the MB and EE patterns across days and the factors that determine compensation effects in individuals living with liver disease.

With respect to research objective 2, this review found that MB and EE findings were not consistent with the ActivityStat hypothesis (Gomersall et al., 2013). Most of the findings were related to MB specifically ($n_{findings}$=43), which was expected given the diversity of direct and indirect measurements available to assess MB. The impact of the interventions on overall PA would be considered positive from a health perspective in that they indicate an increase ($n_{findings}$=25) or no change ($n_{findings}$=13) in PA behavior (i.e., no compensation). In this case, we speculate that the compensation was less likely to occur due to already low baseline PA levels. As this population is characterized with lower PA levels compared to healthy counterparts (Hallsworth et al., 2015), the intervention may not be sufficient to raise incidental PA. Nevertheless, the lack of compensation effect is somewhat surprising considering that individuals living with liver disease are more likely than healthier individuals to display PA-induced fatigue due to psychophysical reasons inherent to their clinical condition (Newton et al., 2008). The feeling or perception of fatigue is likely to change the behavior of this clinical population by reducing levels of PA, increasing sedentary-related behaviors, and causing excessive sleep (Newton et al., 2008; Hallsworth et al., 2015). As an automatic physiological response, PA interventions promote changes in the biochemical equilibrium of the muscle cells and the metabolites produced (Ament & Verkerke, 2009). During this process, the oxidative stress caused by the depletion of the muscle cells ATP leads to dysregulation of the skeletal muscle pathways that may generate feelings of fatigue. However, in clinical populations, the process of re-stablishing body homeostasis after physical exertion can take longer to occur (Newton et al., 2008), which may trigger a compensation effect. For instance, healthy elderly individuals who showed compensation in response to a structured PA program stated that exercise made them tired and sore (Gray et al., 2018). As for the psychological aspect, PA

interventions could evoke compensatory beliefs in which the adoption of a healthy behavior (e.g., exercise, physical activity) at one point is rewarded by a subsequent unhealthy behavior (e.g., high calorie energy intake, sedentary behavior) at another, or vice versa (Rabia, Knäuper, & Miquelon, 2006). This is categorized as a volitional response by which compensatory responses can be triggered due to individuals' intentions to be inactive (King et al., 2007); for instance, rewarding themselves with reduced PA levels due to a previous bout or period of increased vigorous PA. In Gray and colleagues' study (2018), people who engaged in PA interventions believed that they had earned a rest after exercising. Likewise, these compensatory beliefs can also be identified prior to an exercise session or programme after which individuals may engage in more sedentary behavior (Gray et al., 2018). In this review, no qualitative evidence was found considering the psychological or belief aspects of compensation which that should be addressed in future studies.

Regarding the impact of a PA intervention on EE addressed in research objective 2 ($n_{findings}$=13), few findings support the notion that individuals with liver disease compensate. Other reviews document that energy compensation is more likely to occur after significant body weight loss (Silva et al., 2018). This seems to be an automatic physiological response (outside an individuals' control), which could be a protective mechanism with an evolutionary basis (King et al., 2007; Freese et al., 2018). Among the studies in our review, it may be that intensity of the interventions was not sufficient to trigger compensation via weight loss (i.e., EE did not surpass its set point).

Despite the fact that this review did not support the ActivityStat hypothesis among individuals with liver disease involved in PA programs, several studies confirm MB and/or energy compensation in other populations, particularly with respect to incidental PA and NEAT

(Meijer, Westerterp, & Verstappen, 2000; Martin et al., 2011; Di Blasio et al., 2012; Schutz et al., 2014; Herrmann et al., 2015). However, a recent review identified a high percentage of findings ($n_{findings}$=29, 56.9%) demonstrating no change in MB and EE in healthy individuals involved in dietary and/or PA interventions (Silva et al., 2018). This is similar to our review, in which 34.8% ($n_{findings}$=15) and 53.8% ($n_{findings}$=7) of MB and EE findings indicated no change in the outcomes studied. These null findings may be related to the limitations within the studies themselves, which have implications for future research. Specifically, studies should use standardized procedures regarding how compensation is determined, when to assess MB and EE in a PA intervention program (i.e., timeframe), the design of the PA interventions, the methods used to measure MB and EE and the analysis of all their components (i.e., PA of all intensities, sedentary behavior or time, sleep, total EE, basal EE, resting EE, and activity EE). Additionally, as ActivityStat set points may be flexible and dynamic depending on the age, season, or PA level, intra-individual variability in the outcomes studied play an important role (Gomersall et al., 2013). Thus, researchers should consider the pattern of composition of an individual's MB and EE trajectory when analyzing the data.

## Limitations

This review has some limitations that should be acknowledged, most of which were related to the nature of the included studies. First, conclusions regarding day-to-day variation in MB and potential compensation effects could not be achieved due to scant information in the literature. Second, the documents assessed MB and EE using a diversity of measurement techniques (e.g., direct and indirect measurements) limiting the comparison of the outcomes measured. Additionally, given the high cost of trustworthy EE methods (Ndahimana & Kim,

2017), most of the studies used indirect measurements to assess the EE components which may raise concerns about the validity of the results. Third, this review included documents that investigated people living with liver disease diagnosed with varying disease severity, across all ages, and utilized a diversity of intervention designs (i.e., intervention length, duration, intensity, and mode of the PA session). These factors can impact whether a compensation effect may occur or be detected (Eisenmann & Wickel, 2009; Gomersall et al., 2013; Silva et al., 2018).

## Conclusion

Insufficient evidence exists to make any firm conclusions regarding whether individuals living with liver diseases show compensatory responses to PA throughout the day and/or across days. Findings related to the effects of a PA intervention on MB and EE responses do not support the ActivityStat hypothesis (Gomersall et al., 2013). However, further studies should be conducted to better answer our research objectives as methodological differences of the studies included in our review may be masking compensatory mechanisms.

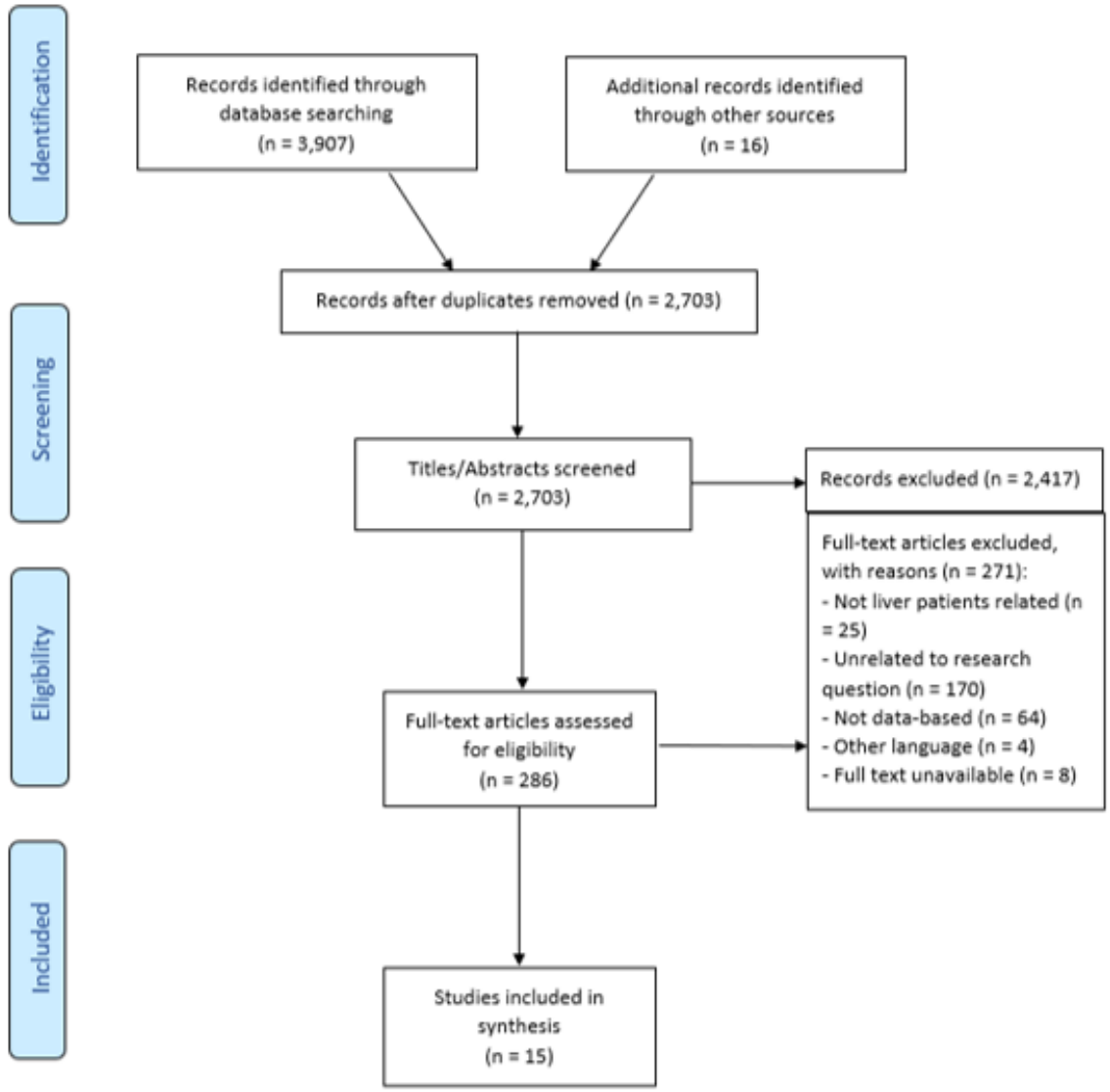


*Figure 3.1* PRISMA flowchart of study selection

*Table 3.1* Summary of reviewed documents and sample characteristics (n = 15)

| Variable | | $n_{studies}$ | % |
|---|---|---|---|
| Publication status | | | |
| | Published | 14 | 93.3 |
| | Unpublished | 1 | 6.7 |
| Publication type | | | |
| | Peer reviewed article | 14 | 93.3 |
| | Thesis | 1 | 6.7 |
| Geographic location | | | |
| | Europe | 6 | 40 |
| | North America | 3 | 20 |
| | Asia | 3 | 20 |
| | Oceania | 3 | 20 |
| Study design | | | |
| | Non-randomized trial | 5 | 33.3 |
| | Randomized trial | 7 | 46.7 |
| | Other | 3 | 20.0 |
| Age group | | | |
| | Adults (18-64 years) | 9 | 60.0 |
| | Mixed | 6 | 40.0 |
| Sex of participants* | | | |
| | Male | 2 | 13.3 |
| | Female | 1 | 6.7 |
| | Mixed | 11 | 73.3 |
| Liver condition | | | |
| | NAFLD | 10 | 66.7 |
| | Liver cirrhosis | 3 | 20.0 |
| | Hepatitis C | 1 | 6.7 |
| | Mixed | 1 | 6.7 |
| Body mass index | | | |
| | Normal | 2 | 13.3 |
| | Overweight | 2 | 13.3 |
| | Obese | 11 | 73.4 |

*One study did not provide the sex of the participants.

*Table 3.2* Summary of intervention and outcome measure

| Study | Sample | PA intervention | Diet intervention | Length | MB outcome measure | Energy outcome measure |
|---|---|---|---|---|---|---|
| Chen et al., 2020 | Exercise arm - n = 9, Control arm - n = 8 | Walking. Daily step logs achieved in the preceding 2 weeks is analyzed, and goals are set for the upcoming 2-week period with targeted weekly to biweekly increments of 500 steps/day. | Yes | 12 weeks | Overall PA and sedentary behavior | NA |
| Hallsworth et al., 2011 | Standard care group - n = 8, Exercise group - n = 11 | Resistance exercise during three times per week on non-consecutive days for 45 and 60 minutes each session. 50% 1 RM progressing to at least 70% 1 RM. | No | 8 weeks | Overall PA | NA |
| Hiraoka et al., 2017 | Control group - n = 21, Mediterranean diet group - n = 21, Mediterranean lifestyle group - n = 21 | Walking exercise (additional 2000 steps/day prescribed based on the baseline average daily steps by each participant) | Yes | 3 months | Overall PA | NA |
| Katsagoni et al., 2018 | Control group - n = 21, Mediterranean diet group - n = 21, Mediterranean lifestyle group - n = 21 | Walking, running, dancing, tennis, etc. Aimed at gradual increase in steps, with the ultimate goal of 10,000 steps/d. Duration of each session at least 30 minutes. Moderate-vigorous intensity PA. | Yes | 6 months | Overall PA, sedentary time, and sleep | Activity EE |

| | | | | | | |
|---|---|---|---|---|---|---|
| Montesi et al., 2014 | CBT - n = 44, PA Group - n = 22 | Patients were stimulated to increase their preferred leisure-time physical activities 3 h/wk. Habitual physical activity workload > 20 METs/h per week (corresponding to 3 h/wk moderate-intense PA). | Yes | 3-4 months | Overall PA | NA |
| Nishida et al., 2017 | N = 6 | Bench step exercises for 140 min of exercise per week. An exercise intensity that corresponded to their Anaerobic threshold. | Yes | 12 months | Overall PA | Activity EE |
| Oh et al., 2015 | IM group (MVPA < 150 minutes/week) - n = 40, IIM group (MVPA 150-250 minutes/week) - n = 42, IIIM group (MVPA ≥ 150 minutes/week) - n = 87 | Aerobic exercise, walking and/or light jogging. 3 days/week during 90 min per session. Targeted to the Borg scale ranging from 11 (light) to 13 (fairly hard) - moderate to vigorous intensity. | Yes | 12 weeks | Overall PA | Activity EE |

| | | | | | | |
|---|---|---|---|---|---|---|
| Pattullo et al., 2013 | n = 16 | Each participant was recommended a PA target of an increment in activity of at least 3000 steps per day above the average daily baseline activity, aiming for an absolute level of over 10 000 steps per day through a gradual increase in both incidental and intentional PA. | Yes | 12 weeks | Overall PA | Activity EE |
| Rachakonda, DeLany, Kershaw, & Behari, 2019 | No NAFLD - n = 62, NAFLD - n = 52 | Brisk walking, 5 days/week. Duration of the session up to 60 minutes/day. | Yes | 6 months | Overall PA | Total EE, resting EE, activity EE |
| Rødgaard-Hansen et al., 2017 | Control group - n=29, Low-intensity group - n=35, Moderate-intensity group - n=29, Moderate-intensity group + maintenance - n=33 | Walking was the primary modality of exercise recommended. Program was individually tailored regarding type, amount, frequency and intensity. Duration tailored at least 150 min/week for general health and to target more than 200 min/week for weight loss. | Yes | 3 months | Overall PA | NA |
| Sargeant, 2019 | n = 9 | Supervised sprint interval exercise training (cycling). Three exercise sessions per week for 35 to 45 minutes each session. Vigorous-intensity exercise. | No | 6 weeks | Overall PA and sedentary time | NA |

| | | | | | | |
|---|---|---|---|---|---|---|
| Scaglioni et al., 2013 | n = 12 | Multidisciplinary non-pharmacological treatment based on personalized diet, physical activity and behavior therapy. | Yes | 3 months | Overall PA | Activity EE |
| St. George et al., 2009a | Low-intensity group (Group A) - 38, Moderate-intensity group (Group B) - 38, Moderate-intensity group (Group C) - 38, Control group (Group D) - 38 | Participants were encouraged to increase both planned and incidental PA for at least 150 min/week for general health and >= 200 min/week for weight loss. Low to moderate intensity PA. | Yes | 4 to 10 weeks | Overall PA | NA |
| St. George et al., 2009b | Low-intensity group (Group A) - n = 38, Moderate-intensity group (Group B) - n = 33, Moderate-intensity group (Group C) - n = 36, Control group (Group D) - n = 34 | Participants were encouraged to increase both planned and incidental PA. Walking was the primary modality encouraged. Frequency varied depending on baseline level of activity, medical history, and the personal preferences of each patient. Duration should be at least 150 min/week for general health and more than 200 min/week for weight loss. Moderate-intensity PA. | No | 3 months | Overall PA | NA |

PA = Physical activity, EE = Energy expenditure, NAFLD - Non-alcoholic fatty liver disease, CBT = Cognitive-behavioral treatment, MET = Metabolic equivalent of tasks, RM = Repetition maximum.

*Table 3.3* Movement behaviour and energy expenditure compensatory mechanisms in response to a PA intervention (nfindings = 56)

| Variable | | | $n_{findings}$ | % of total findings | % within group |
|---|---|---|---|---|---|
| MB response | Impact on overall PA | Increase | 25 | 44.5 | 58.2 |
| | | No change | 13 | 23.2 | 30.2 |
| | Impact on sedentary time or behavior | Decrease | 2 | 3.6 | 4.7 |
| | | No change | 1 | 1.8 | 2.3 |
| | Impact on sleep | Increase | 1 | 1.8 | 2.3 |
| | | No change | 1 | 1.8 | 2.3 |
| | Total | | 43 | 76.8 | |
| EE response | Impact on total EE | Increase | 1 | 1.8 | 7.7 |
| | | Decrease | 1 | 1.8 | 7.7 |
| | | No change | 1 | 1.8 | 7.7 |
| | Impact on resting EE | Decrease | 1 | 1.8 | 7.7 |
| | Impact on activity EE | Increase | 3 | 5.4 | 23.1 |
| | | No change | 6 | 10.7 | 46.1 |
| | Total | | 13 | 23.2 | |

PA = Physical activity; MB = Movement behavior; EE = Energy expenditure.

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# CHAPTER 4: Study 2

Continuous-time modeling of the multivariate relationships between physical activity levels and stationary time in preschool-aged children: An investigation of the ActivityStat hypothesis

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## Abstract

The ActivityStat hypothesis proposes that an increase or decrease in physical activity (PA) greater than a certain set point activates behavioural and/or energy compensatory responses to maintain a stable level of total energy expenditure. Few studies have tested this hypothesis in children and even fewer have focused on young children. Therefore, the purpose of this study was to investigate the ActivityStat hypothesis by examining the presence and timeframe of the relationships among PA levels and stationary time (ST) in preschool-aged children. A secondary analysis was performed on repeated measurement data (i.e., day-to-day activity) involving 98 preschool-aged children (age: 4.5 ± 0.7 years) in Edmonton, Canada. Participants were asked to wear an ActiGraph wGT3X-BT on the waist for 7 consecutive days to assess PA levels (i.e., light PA [LPA], and moderate-to-vigorous PA[MVPA]) and ST. Bayesian continuous-time structural equation modeling (CT-SEM) was used to examine the relationship between behaviours over time and the timeframe during which these relationships occur. Each behaviour (i.e., LPA, MVPA, and ST) positively and meaningfully predicted itself at a later time. These relationships persisted up to 0.5 days later, at which point past behaviour no longer meaningfully predicted future behaviour. In contrast, no relationships were observed between the three behaviours. This is the first study to investigate the ActivityStat hypothesis using Bayesian CT-SEM in preschool-aged children. When simultaneously taking into account all dynamic relationships suggested by the ActivityStat, the findings fail to support the hypothesis.

## Introduction

To experience the health benefits of physical activity (PA), public health guidelines recommend preschool-aged children (under 5 years old) accumulate 180 minutes of PA daily, including at least 60 minutes of moderate-to-vigorous PA (MVPA), and limit time spent sedentary (World Health Organization [WHO], 2019). However, a majority of children have insufficient levels of PA and spend long hours in sedentary-related behaviours globally (Guthold et al., 2018). To date, efforts to produce sustained changes in health-enhancing PA and sedentary behaviour (SB) remain a formidable challenge (Altenburg, Kist-van Holthe, & Chinapaw, 2016). Thus, new perspectives are needed to understand the mechanisms underpinning children's activity patterns. Biological factors, for instance, may also influence how individuals engage in PA but investigation in this area is scarce (Beck, Engel, & Reimers, 2022; Gomersall et al., 2013; Swelam et al., 2022).

The ActivityStat hypothesis proposes that humans have biological mechanisms that regulate the extent to which individuals engage in PA (Gomersall et al., 2013). It is hypothesized that an internal biological control center (i.e., hypothalamus) is activated when total energy expenditure reaches its set point triggering a cascade of compensatory mechanisms to return the system back to its steady state (Gomersall et al., 2013; Pontzer, 2018; Rowland, 1998). Specifically, an increase or decrease in PA greater than a certain set point activates behavioural and/or energy compensatory responses to maintain a stable level of total energy expenditure. This evolutionary trait keeps energy requirements in check while prioritizing and allocating energy to reproductive fitness and survival (Pontzer, 2018). In studies involving children, both within- and between-days have been examined for evidence of behavioural compensation. For instance, within-day activity compensation was observed in children who had an active morning

commute and then walked less throughout the day (Tan et al., 2018). As for between-days compensatory effects, Ridgers et al. (2018) showed that for every additional 10 minutes of MVPA on one day resulted in a reduction of 9.3 minutes of MVPA and 16.8 minutes of LPA the following day in children 8 to 11 years of age. Likewise, associations between sitting and PA both within- and between-days show that children may compensate for increased sitting, standing, and stepping (Ridgers et al., 2015). However, other empirical studies and systematic reviews do not support compensatory mechanisms in this population (Beck, Engel, & Reimers, 2022; Gomersall et al., 2013; Nigg et al., 2022). Thus, the ActivityStat presents an intriguing area for further study. Additionally, preschool-aged children are underrepresented in studies examining compensatory mechanisms of PA and SB, suggesting a new avenue of research.

A lack or inadequate alignment between theory (i.e., ActivityStat), methods, and statistical modelling may explain inconsistent findings in the literature (Collins, 2006). Studies investigating the ActivityStat hypothesis have mainly used traditional longitudinal multilevel models (e.g., hierarchical linear models, mixed models) (Nigg et al., 2022; Ridgers et al., 2015; Ridgers et al., 2018). These include the analysis of static mechanisms using univariate approaches focusing on the relationship among the constructs in one direction or the other. For example, to understand how a change in individuals' MVPA at one point results in changes in SB at a subsequent point in time, separate analyses examining the relationship of MVPA on SB, and the relationship of SB on MVPA are typically conducted. However, without accounting for autoregressive effects (i.e., the relationship within the same variable over time) of all study variables and the reciprocal cross-lagged effect (i.e., the relationship between variables over time) within the same analysis, the observed lagged effects will be artificially inflated

(Schuurman et al., 2016). Therefore, findings for the ActivityStat hypothesis that are based on traditional longitudinal multilevel models should be interpreted with caution.

Instead, dynamic modelling methods are more appropriate for examining the ActivityStat hypothesis for several reasons. Specifically, such modelling methods: 1) account for multivariate reciprocal relationships between the constructs studied; 2) allow the analysis of variables that act both as predictor and outcome variables; and 3) include testing of a system as a whole, with all relevant constructs in one model (Ruissen et al., 2022). For instance, continuous-time structural equation models (CT-SEM) treat dynamic processes as a function of the continuous variable of time. Thus, findings can be generalized to other timeframes providing a complete picture of how a phenomenon unfolds over time (Driver & Voelkle, 2018a, 2018b). Additionally, a novel advantage of CT-SEM is it can help answer research questions regarding the temporal specificity (i.e., timeframe) of when the behavioural compensation may occur (e.g., acute effect, between days, between multiple days).

Therefore, the current study aims to investigate the ActivityStat hypothesis in preschool-aged children by using CT-SEM to (1) examine the presence of continuous-time multivariate relationships between PA levels (operationalized by LPA and MVPA) and SB (as represented by stationary time [ST]) (Tremblay et al., 2017), and (2) explore the temporal specificity underlying the multivariate relationships between PA levels and ST.

## Methods

### Participants and procedure

This study involved a secondary analysis of data derived from the Parent-Child Movement Behaviours and Pre-School Children's Development project (Kuzik, 2020). The

original cross-sectional study recruited children, aged 3-5 years, and their families in Edmonton, Canada and surrounding areas through a program that aims to teach fundamental sport skills through play. A total of 131 children initially agreed to participate and data collection occurred from July to November 2018. For both the primary and secondary analysis, approval was granted by the University of Alberta research ethics board (Study ID Pro00117335 and Pro00117335, respectively).

**Measures**

LPA, MVPA, and ST were measured with ActiGraph wGT3X-BT accelerometers. Because this accelerometer is limited in reliably estimating body posture (Edwardson et al., 2016), the recommendation is to use ST to represent sedentary-related behaviors (Tremblay et al., 2017). Specifically, ST refers to the time spent in "any waking behavior done while lying, reclining, sitting, or standing, with no ambulation, irrespective of energy expenditure" (Tremblay et al., 2017). Participants were instructed to wear the accelerometer on an elastic belt on their right hip for 24 hours a day over 7 days, except during water-based activities. The devices were programmed at 30 Hz for sampling frequency and the data were downloaded in 15-second epochs for both normal filter files and low frequency extension filter files. The normal filtered files were used to categorize children's LPA (26-419 counts/15 seconds), MVPA (≥420 counts/15 seconds), and ST (≤25 counts/15 seconds). Days with valid wear time of at least 10 hr/day (i.e., 600 minutes) and a minimum of 3 days represented a valid and reliable estimate for PA and ST via accelerometry (Bingham et al., 2016), and non-wear time was removed (≥20 minutes consecutive 0 counts (Cliff, Reilly, & Okely, 2009). Minutes per day for LPA, MVPA, and ST were then included in the analysis. Though sleep is part of the activity spectrum

(Tremblay et al., 2010), and should be considered when testing the ActivityStat hypothesis, its inclusion in the current analysis was not possible due to model complexity in CT-SEM. Regardless, the focus on PA and ST allows for comparisons with previous studies on the topic (Nigg et al., 2022; Ridgers et al., 2015; Ridgers et al., 2018; Tan et al., 2018).

**Statistical analysis**

Accelerometry data were structured in a stacked format (i.e., long format) and a time index variable was created to identify each time-point (i.e., day, $t = 0$ to 6). Data were standardized, and grand-mean centered to facilitate model convergence. Bayesian CT-SEM uses distinct modeling allowing for more flexibility compared to traditional longitudinal multilevel models (Driver & Voelkle, 2018a). For example, Bayesian estimation methods are robust to moderate violations of the normality assumption (Hecht & Zitzmann, 2021a). Regardless, the shape of the distributions for the outcomes of interest were visually inspected and all appeared normal. Due to the use of a continuous-time framework for this study, missing data were considered missing at random. Specifically, it was interpreted as reflecting unequal time intervals between measurements (Oud & Voelkle, 2014). Further, Bayesian CT-SEM does not impose any restriction on sample size and length of time series (i.e., minimum number of days or other time period/s) (Driver & Voelkle, 2018a). The models were fit using the ctsem package (Driver, Oud, & Voelkle, 2017), which interfaces to Stan (2023) in the R 4.2.1 environment (RCore, 2021). We computed a CT-SEM model examining the multivariate dynamic system with reciprocal relationships between LPA, MVPA, and ST (Appendix B).

As recommended for Bayesian models, the default burn-in (50% of the chain [i.e., one sequence of randomly sampled values]), the default aggregation statistic (mean of the chain), and

the default priors were used (Driver & Voelkle, 2018a). The model (i.e., LPA, MVPA, ST) was run using a NUTS (No U-Turn sampler) with four chains and 20,000 iterations per chain (Gelman et al., 2013). For the model convergence and statistic precision, the potential scale reduction factor $\hat{R}$ and effective sample size ($N_{eff}$) were reported (Gelman et al., 2013). A fully random effects model was first constructed, however, it showed problems with model fit. A random intercept only model was also performed presenting a better fit with an adequate model convergence and precision. The inclusion of the random intercept in the model allows variation in the intercept only resulting in similar parameters estimated in the model, thus accommodating stable individual differences, but not individual differences in the dynamic relationships. As the ActivityStat hypothesis proposes that these compensatory mechanisms occur within-individuals (Gomersall et al., 2013), marked differences in these compensatory dynamics between individuals were outside the scope of this study. Thus, the latter findings are presented here. Further details about the complete code and the fully random effects model output illustrating the pattern of findings can be seen in Appendices C and D.

The auto- and cross-effects parameters are of primary interest for answering the main research question and refer to the continuous-time parameters presented in the drift matrix. The auto-effects reflect the stability (or persistence) of the relationship within a behaviour over time. The cross-effects describe the reciprocal effects of one variable on the other. The autoregressive and cross-lagged effect terms refer to the discrete-time parameters. From these parameters, the time interval at which dynamic processes reach their peak effects and their maximum and minimum discrete-time coefficient time intervals were examined. For interpreting the results, the parameter's posterior mean in relation to its posterior standard deviation (SD) and posterior 95% Bayesian credibility intervals (BCI) were examined. Meaningful results are interpreted when

zero does not fall within the lower (2.5%) or upper (97.5%) limits of the BCI parameter. Evidence of continuous-time temporal relationship is indicated by meaningful auto- or cross-effect relationships between PA levels and ST. Finally, evidence of temporal specificity is indicated by the timeframe at which the autoregressive or the cross-lagged effects between PA levels and ST reach their peak effects with behaviours aggregated at a day level.

## Results

A total of 98 children met the minimum wear-time criteria with an average of 772 min per day (SD = 66.65, range 623.36 to 937.91) and 46% (n = 45) presented 7 days of valid accelerometer data. Boys represented 69.4% (n = 68) of the sample and the average age was 4.5 ± 0.7 years. The children engaged in an average 303.18 ± 49.09 minutes of LPA, 108.72 ± 40.29 minutes of MVPA, and 360 ±72.59 minutes of ST per day.

The Bayesian estimation required approximately 20 RAM usage and 15.8 hours of runtime for the LPA, MVPA, and ST model on an Intel i9-10900T (4.60 GHz Turbo) CPU of a 64-bit Windows OS, with 64 GB RAM. All parameters had a minimum of 2270.19 effective samples and a Rhat ($\hat{R}$) of 1.0, indicating adequate model convergence and precision. Based on the prediction model by Hecht and Zitzmann (2021a, 2021b) using $N = 98$, $T = 7$ (total number of time points), and standardized peak effect set to 0.01 (small effect), our post hoc analysis suggests we had a sufficient sample size to reliably estimate the continuous-time cross-lagged dynamics between LPA, MVPA, and ST (estimated "power" for standardized peak cross-lagged effect = 1.00).

Table 4.1 displays the posterior population means, standard deviations, and 95% Bayesian credibility intervals for the $T_0$ mean parameters, continuous-time intercept (i.e., b), and

$T_0$ variance between LPA, MVPA, and ST. The $T_0$ mean indicates the initial state (i.e., starting point) estimate for the outcomes. It shows to what extent the participants' initial states tend to be higher or lower than their later states (Driver & Voelkle, 2018a, 2018b). A negative estimate indicates that the initial process was lower than future states, whereas a positive estimate indicates that the initial state was higher. For LPA, MVPA, and ST, no meaningful increase or decrease was observed in the overall level over time, with the 95% BCI encompassing 0 (LPA, M = 0.08, SD = 0.1, 95% BCI [-0.12, 0.27]; MVPA, M = 0.16, SD = 0.11, 95% BCI [-0.05, 0.38]; ST, M = -0.18, SD = 0.1, 95% BCI [-0.37, 0.02]). This means that LPA, MVPA, and ST fluctuated around a stationary, average level, during the observation period (i.e., 7 days). The continuous-time intercept represents the average process means for each outcome studied. Because they were grand-mean centered and standardized, they are not relevant for interpretation. The $T_0$ variance, which refers to the asymptotic within-person covariance, indicated that all processes studied have similar variation over time (LPA, M = 0.98, SD = 0.07, 95% BCI [0.85, 1.13]; MVPA, M = 1.07, SD = 0.08, 95% BCI [0.93, 1.24]; ST, M = 0.98, SD = 0.07, 95% BCI [0.85, 1.13]).

The direct instantaneous ($\Delta t \rightarrow 0$) temporal relationship a variable has with its own rate of change (i.e., auto-effects) and between two distinct variables (i.e., cross-effect) are shown in Table 4.1 These are called drift parameters and both the auto- and cross-effects are of particular interest in this study. For the auto-effects, the closer the estimates are to zero, the longer the changes persisted over time (Driver & Voelkle, 2018a, 2018b). A negative estimate reveals a diminishing auto-effect over time (returning to baseline) (Driver & Voelkle, 2018a, 2018b). On the contrary, a positive estimate represents an explosive process (distancing away from the baseline over time). As shown in Table 4.1 and Figure 4.1, the auto-effects for LPA (M = -3.13,

SD = 2, 95% BCI [-9.04, -1.24]), MVPA (M = -6.11, SD = 2.79, 95% BCI [-12.23, -1.95]), and ST (M = -3.41, SD = 1.96, 95% BCI [-9.1, -1.54]) demonstrated some degree of persistence, with all the auto-effects parameters excluding zero. LPA, MVPA, and ST were predictors of themselves at a later point in time. For the cross-effects, a negative estimate indicates that an increase in the level of one process predicts a decrease in the level of the other process. A positive cross-effect, in contrast, indicates that an increase in the level of one process predicts an increase in the other process (Driver & Voelkle, 2018a, 2018b). As demonstrated in Table 4.1 and Figure 4.2, the cross-effect parameters reveal that increases in the levels of LPA, MVPA, and ST do not predict any subsequent change in the levels of the other constructs over time.

The diffusion parameters allow further interpretation of the temporal relationship between the processes (see Table 4.1). These parameters account for fluctuations in dynamic processes of interest over time that cannot be accounted by the deterministic model elements (i.e., $T_0$ mean, drift matrix parameters). The on-diagonal elements of the diffusion matrix are variances that quantify the extent to which the variables in a dynamic system are influenced by unmodelled exogenous inputs (Driver & Voelkle, 2018a, 2018b). The findings indicate that all latent processes (i.e., LPA, MVPA, ST) were influenced by random (i.e., unpredictable) fluctuations over time. More specifically, LPA and ST processes have similar levels of variation from exogenous inputs (LPA, M = 1.96, SD = 0.53, 95% BCI [1.39, 3.45]; ST, M = 1.92, SD = 0.51, 95% BCI [1.3, 3.32]). As for MVPA, on the contrary, there is relatively more variation in this behaviour over time due to unmodeled factors (M = 2.62, SD = 0.61, 95% BCI [1.62, 3.84]). The off-diagonal elements of the diffusion matrix represent covariances that quantify the extent to which the stochastic variation between two latent processes share common causes (Driver & Voelkle, 2018a, 2018b). The findings show that there is meaningful negative covariation

between ST and MVPA (M = -0.32, SD = 0.1, 95% BCI [-0.56, -0.15]). Furthermore, the 95% BCI for LPA and both MVPA and ST included zero, indicating that variation in LPA is largely independent from the exogenous inputs that influence MVPA (M = 0.15, SD = 0.09, 95% BCI [-0.02, 0.32]) and ST (M = -0.01, SD = 0.09, 95% BCI [-0.19, 0.17]) latent processes. Thus, other factors may determine LPA in this sample.

As illustrated in Figure 4.1, the autoregressive effects (i.e., the discrete-time parameters presented in the plots) for children's LPA, MVPA and ST appear to be predictive of their later behaviours until about 0.5 days later. For example, after accounting for all other dynamic relationships included in the multivariate dynamic model, a child's MVPA at one moment in time was predictive of their subsequent MVPA until about 0.5 days hence. This pattern was also consistent across LPA and ST. Further, the autoregressive coefficients are positive (see Figure 4.1), demonstrating persistence in processes. In contrast, a negative autoregressive effect illustrates an oscillating compensatory process (Schuurman et al., 2016), which was not detected in this study. Further, although no meaningful discrete-time cross-lagged effects were observed over time, Figure 4.2 suggests the temporal dependencies among the behaviours were largest at 0.25 days between measurement occasions after accounting for all other dynamic relationships included in the model.

The between-person relationships among LPA, MVPA, and ST are presented in Table 4.1. No meaningful relationship was found between the behaviours suggesting that the constructs were independent from each other.

## Discussion

The primary aim of this study was to examine the presence of continuous-time multivariate relationships between PA levels and ST in preschool-aged children. Based on the ActivityStat hypothesis, compensatory mechanisms would be indicated by meaningful negative within-behavior relationships for LPA, MVPA or ST (Gomersall et al., 2013). As for the cross-behavioral relationships, ActivityStat is operating when meaningful negative associations are observed between MVPA and LPA, and meaningful positive associations are observed between LPA and ST or between MVPA and ST (Gomersall et al., 2013). Findings for the primary research question show that children's level of LPA, MVPA, and ST on a given day positively predict LPA, MVPA, and ST the following day, respectively. However, the cross-effects parameters indicated no relationship among these behaviours. Thus, this study does not support the ActivityStat in preschool-aged children.

Absent of compensation, some studies have demonstrated similar patterns to what was found in the present study where an increase in PA in a specific context stimulated children between 8 to 10 years of age to engage in more PA at another time (Cheung, 2019; Pesola et al., 2018; Smith et al., 2012). Similarly, Nigg et al. (2022) report that youth between 6 to 17 years of age do not compensate when MVPA increases. In contrast, a systematic review of 12 studies in preschool-aged children (3 to 6 years old) found evidence of compensation in 42% of the cases (Beck, Engel, & Reimers, 2022), but the studies were not designed explicitly to test for the ActivityStat hypothesis. This fact may have impacted why compensation was not observed (Swelam et al., 2022). Regardless, investigation of compensatory mechanisms in young children is still understudied and all studies supporting and refuting the ActivityStat hypothesis in this population have employed traditional multilevel models (Nigg et al., 2022; Ridgers et al., 2018;

Wilkin et al., 2006), disregarding the temporal multivariate dependence among LPA, MVPA, and ST. Therefore, more studies designed to test the hypothesis are needed in the population of preschool-aged children. Additionally, the use of dynamic modelling methods is critical for understanding such complex systems in future research.

Further interpretation of the temporal relationship between the processes studied show that LPA, MVPA, and ST appear to be influenced by other factors not included in the analysis. For instance, sleep may influence PA and SB in preschool-aged children (WHO, 2019). This behaviour should be incorporated as a component of the movement behaviour continuum (i.e., sleep, SB, PA; Tremblay et al., 2010). Poor sleep may increase sedentary-related playtime activities in children while decreasing their MVPA levels (Lin et al., 2018). In contrast, variability in LPA appears to have unique causes independent of those that explain variability in MVPA and ST. For example, young children who have siblings may still engage in LPA regardless of restrictions for using outdoor spaces (Ostermeier et al., 2022). Due to the complexity of the factors that impact movement-related behaviours, investigating the ActivityStat is a challenge. Future studies should focus on compensatory mechanisms in young children examining simultaneously movement-related behaviours directly and parents' perceptions of activity compensation.

The secondary aim of this study was to explore the temporal specificity underlying the multivariate relationships between PA levels and ST. All behaviour autoregressive components were predictive of themselves until about 0.5 days later. This implies that preschool-aged children who move more at a specific time point positively predict PA (i.e., LPA and MVPA) until about 0.5 days later. The same pattern is observed for ST. As for the cross-lagged effects, a relationship among LPA, MVPA, and ST appeared to occur about 0.25 days later. For instance,

preschool-aged children who engage in MVPA at a specific time point may positively predict LPA until about 0.25 days later. However, the cross-behavioural relationships among the latent processes were not meaningful and were mostly in the opposite direction of what would have been anticipated by the ActivityStat hypothesis (Gomersall et al., 2013). Thus, no compensation effects were observed at days greater than or equal to one. According to Gomersall et al. (2013), the timeframe for compensation is unlikely to occur within a short timescale (i.e., within- and between-days). However, cross-sectional studies testing the hypothesis in children demonstrated evidence of behavioural compensation within- and between-days refuting the argument for longer-term observations (Ridgers et al., 2015; Ridgers et al., 2018; Wilkin et al., 2006). These studies focused on short-time relationships among PA levels and SB and employed traditional longitudinal analyses. Recent systematic reviews highlight the importance of examining the timeframe for compensation as it impacts conclusions of whether compensation occurs (Beck, Engel, & Reimers, 2022; Swelam et al., 2022). Therefore, because the timeframe for behavioural compensation is unknown, future studies should consider a wider range of timescales, as effects can be aggregated to longer time intervals if required. This potentially has practical importance for designing PA programs impacting on the design, frequency of measurements, and duration of the intervention (Beck, Engel, & Reimers, 2022; Gomersall et al., 2013; Swelam et al., 2022).

This study has some limitations that should be considered when interpreting the results. First, because CT-SEM is computationally intensive (it needs a processor with as many cores as chains, i.e., 4 cores), the dynamic measurement parameters were not included in the final model. Though, this parameter accounts for measurement error in each individual behaviour, it would require a few months for the model to run. Instead, a final model without this parameter still showed adequate model convergence and precision. Additionally, because CT-SEM can

accommodate two to three dynamic processes at most, the examination of a complex and multivariate system is computationally burdensome. Therefore, the inclusion of sleep in this study was not feasible. Secondly, the non-experimental design has low internal validity and constrains our ability to make causal claims. Because habitual activity patterns may show temporal variations in PA and ST, these can be misinterpreted as compensatory mechanisms. Additionally, the examination of behavioral compensation in preschool-aged children may be more challenging due to the characteristics of the population. For this age-group, it is difficult to conclude whether variations in PA are due to biological mechanisms, or a response influenced by external factors (e.g., parents, preschool or daycare schedule). Thirdly, SB was represented as ST because the ActiGraph wGT3X-BT detects motion and is limited in reliably detecting the posture component of SB (Edwardson et al., 2016; Tremblay et al., 2017). Lastly, because compensation could occur via energy expenditure mechanisms that were not necessarily reflected in behavioural adaptations (Gomersall et al., 2013; Pontzer, 2018; Rowland, 1998), the lack of energy expenditure measures in this study limited the ability to comprehensively test the hypothesis. Nevertheless, a number of strengths should be highlighted: the use of the novel insights provided by Bayesian CT-SEM to examine the ActivityStat hypothesis, employing device-assessed measurement of PA and ST over several days, and the examination of this hypothesis in preschool-aged children.

## Conclusion

This is the first study to investigate the ActivityStat hypothesis using Bayesian CT-SEM in preschool-aged children. Though we found positive continuous-time relationships within LPA, MVPA, and ST, no relationships were observed between the behaviours. Overall, the findings do

not support the ActivityStat hypothesis in preschool-aged children when simultaneously taking into account all dynamic relationships among LPA, MVPA and ST. One potential explanation is that our participants did not exceed their individual activity set point as a result of engaging in PA. Therefore, compensatory behaviours were not initiated (Gomersall et al., 2013). Future research should employ dynamic modelling analyses in experimental designs and attempt to measure both energy expenditure and movement behaviours. Absent of evidence for compensation effects, PA programs involving young children should continue follow current guidelines for promoting health movement behaviour.

*Table 4.1* Means of estimated population distributions of the relationship between LPA, MVPA, and ST.

| Parameter | | Est. | SD | BCI [2.5%, 97.5%] | | Rhat | $N_{eff}$ |
|---|---|---|---|---|---|---|---|
| $T_0$ Mean | LPA | 0.08 | 0.1 | -0.12 | 0.27 | 1 | 56627.31 |
| | MVPA | 0.16 | 0.11 | -0.05 | 0.38 | 1 | 50793.65 |
| | ST | -0.18 | 0.1 | -0.37 | 0.02 | 1 | 50109.75 |
| Continuous-Time Intercept | LPA | -0.04 | 0.25 | -0.6 | 0.42 | 1 | 14581.17 |
| | MVPA | -0.2 | 0.49 | -1.31 | 0.73 | 1 | 20603.15 |
| | ST | 0.1 | 0.28 | -0.39 | 0.73 | 1 | 14621.88 |
| $T_0$ Variance | LPA | 0.98 | 0.07 | 0.85 | 1.13 | 1 | 61489.69 |
| | MVPA | 1.07 | 0.08 | 0.93 | 1.24 | 1 | 53878.21 |
| | ST | 0.98 | 0.07 | 0.85 | 1.13 | 1 | 55909.85 |
| Auto-effects parameters | LPA | -3.13 | 2 | -9.04 | -1.24 | 1 | 4717.66 |
| | MVPA | -6.11 | 2.79 | -12.23 | -1.95 | 1 | 8155.83 |
| | ST | -3.41 | 1.96 | -9.1 | -1.54 | 1 | 5405.83 |
| Cross-effects parameters | LPA_MVPA | -0.02 | 0.82 | -1.57 | 1.68 | 1 | 2270.19 |
| | LPA_ST | -0.68 | 0.68 | -2.1 | 0.64 | 1 | 4439.61 |
| | MVPA_LPA | 0.39 | 0.86 | -1.38 | 2.06 | 1 | 25240.28 |
| | MVPA_ST | 0.12 | 0.9 | -1.73 | 1.82 | 1 | 11317.45 |
| | ST_LPA | -0.47 | 0.66 | -1.88 | 0.79 | 1 | 23068.58 |
| | ST_MVPA | -0.29 | 0.84 | -1.99 | 1.34 | 1 | 29190.89 |
| Diffusion parameters | LPA | 1.96 | 0.53 | 1.39 | 3.45 | 1 | 4370.99 |
| | MVPA | 2.62 | 0.61 | 1.62 | 3.84 | 1 | 8328.92 |
| | ST | 1.92 | 0.51 | 1.3 | 3.32 | 1 | 5415.59 |
| | MVPA_LPA | 0.15 | 0.09 | -0.02 | 0.32 | 1 | 8912.24 |
| | ST_LPA | -0.01 | 0.09 | -0.19 | 0.17 | 1 | 4292.36 |
| | ST_MVPA | -0.32 | 0.1 | -0.56 | -0.15 | 1 | 11144.99 |
| Between-subject | MVPA_LPA | 0.25 | 0.38 | -0.57 | 0.84 | 1 | 9339 |
| | ST_LPA | -0.37 | 0.35 | -0.87 | 0.45 | 1 | 39710 |
| | ST_MVPA | -0.51 | 0.29 | -0.89 | 0.23 | 1 | 39676 |

Continuous-time intercept = b coefficient; Est. = mean of the chain; BCI = Bayesian credibility interval; Rhat ($\hat{R}$) = potential scale reduction factor; $N_{eff}$ = effective sample size; LPA = light physical activity; MVPA = moderate-vigorous physical activity; ST = stationary time.

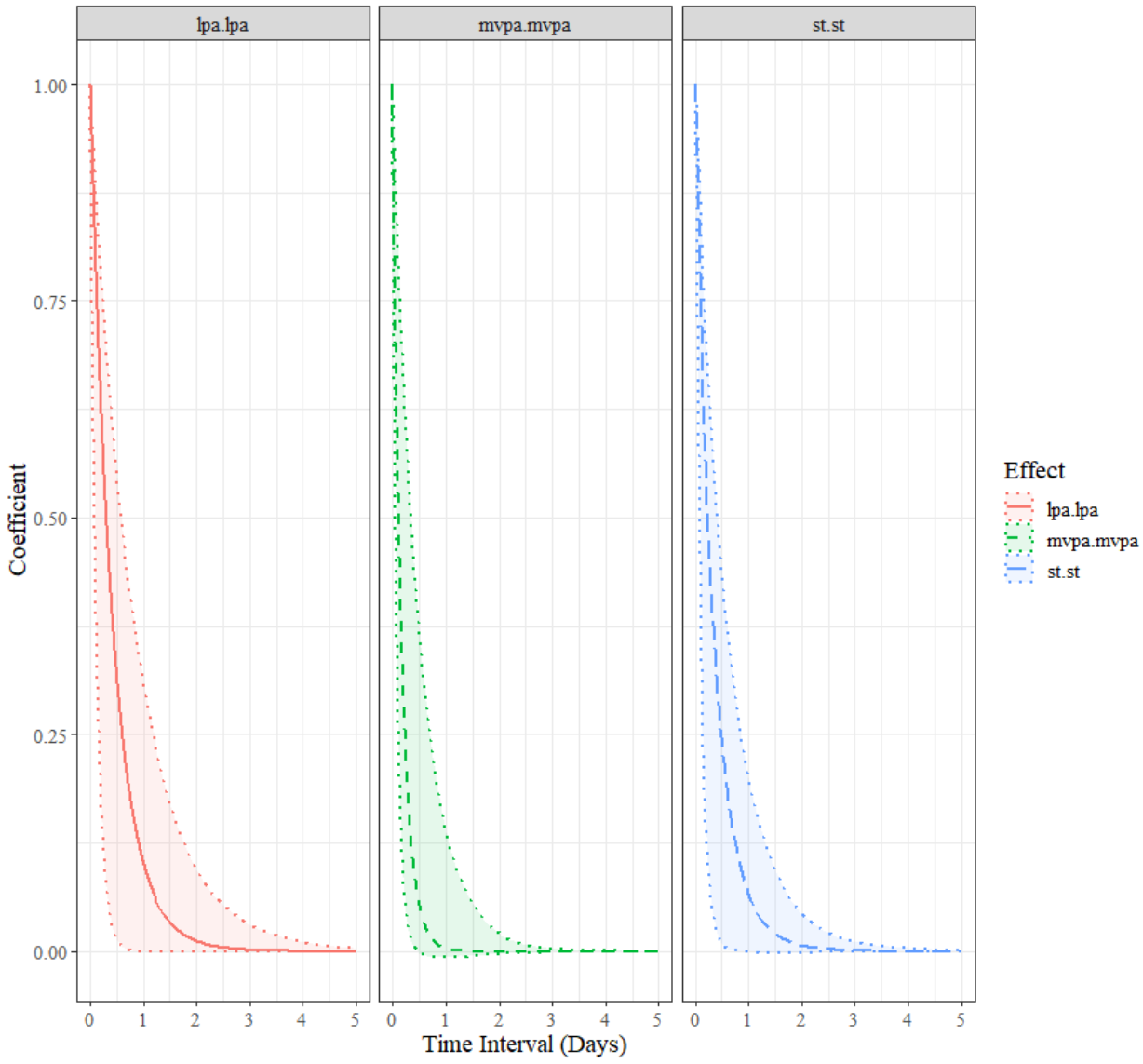


*Figure 4.1* Standardized discrete-time autoregressive effects.

LPA = light physical activity; MVPA = moderate-vigorous physical activity; ST = stationary time.

*Note*. This figure shows that all autoregressive coefficients are above zero, demonstrating positive relationships within the movement behaviours. The largest timeframe of these relationships become apparent when the 95% Bayesian credibility is approaching zero. Thus, the largest timeframe is 0.5 for all autoregressive relationships.

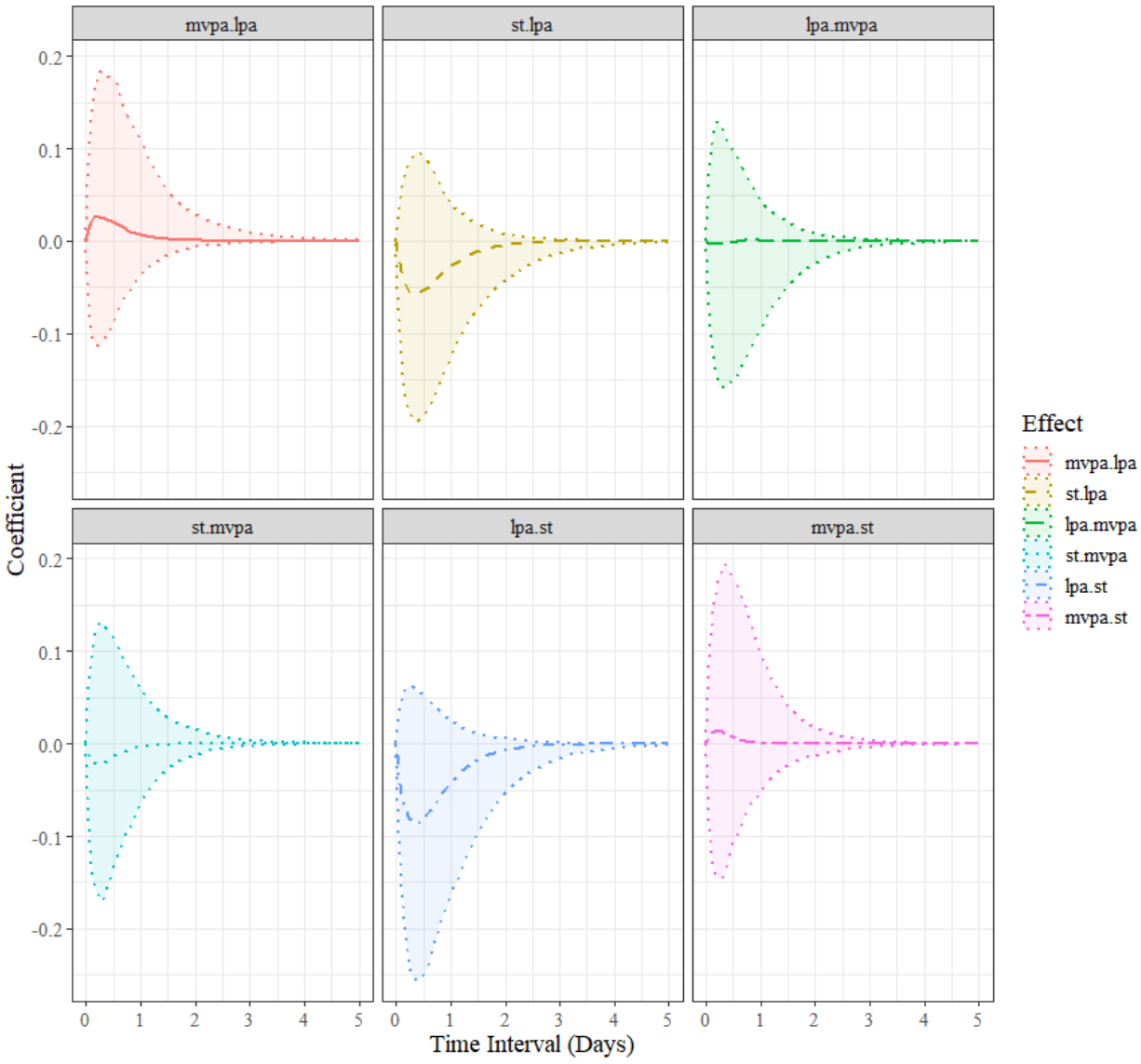


*Figure 4.2* Standardized discrete-time cross-lagged effects.

LPA = light physical activity; MVPA = moderate-vigorous physical activity; ST = stationary time.

*Note*. This figure shows that all the cross-behavioural relationships include zero in all time intervals demonstrating no meaningful associations over time.

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# CHAPTER 5: Study 3

## Temporal relationships in the movement behaviour of adolescents: Evidence of the ActivityStat hypothesis for stationary time

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**Abstract**

The ActivityStat hypothesis suggests that movement is a biological necessity and that it can be internally regulated. Specifically, humans maintain their total physical activity (PA) at a constant level by triggering behavioural and/or energy compensatory responses. However, there is considerable debate about the possibility of PA compensation. Thus, the purpose of this study is to examine the presence and the timeframe of the temporal relationships among PA levels and stationary time (ST) in adolescents. A secondary analysis was performed on data from a non-experimental study involving 356 adolescents (age: 14.7 ± 1.4 years) in Dunedin (New Zealand). Participants were asked to wear an accelerometer on the waist for several consecutive days to assess PA levels [i.e., light PA (LPA), and moderate-to-vigorous PA (MVPA)] and ST. Bayesian continuous-time structural equation modeling (CT-SEM) was employed to examine the relationships between LPA, MVPA, and ST and the timeframe during which these relationships occur. Increases in LPA, MVPA, and ST were positively associated with their later behaviours until 1.7 to 2.5 days hence. A cross-behavioural reciprocal and negative relationship between LPA and ST was demonstrated 0.4 days later. A positive relationship between ST and MVPA was observed until about 0.4 days later. This is the first study to investigate the ActivityStat hypothesis using Bayesian hierarchical CT-SEM in adolescents. Though behavioural compensation was not observed for LPA and MVPA, it was noted for ST. Thus, the findings support evidence of the ActivityStat hypothesis in adolescents.

## Introduction

Public health guidelines recommend individuals be physically active throughout the day and avoid excessive sedentary time (World Health Organization, 2018). However, most adolescents do not meet such guidelines (Guthold et al., 2020). A variety of factors influence how much individuals engage in physical activity (PA; Spence & Lee, 2003). Though biological processes, for instance, may regulate PA (Gomersall et al., 2013; Rowland, 1998), limited research has been done on this topic.

The ActivityStat hypothesis proposes that a biological control center is activated when total energy expenditure reaches its set point triggering a cascade of compensatory mechanisms to return the system back to its steady state (Gomersall et al., 2013; King et al., 2007; Pontzer, 2015; Pontzer, 2017; Pontzer, 2018; Rowland, 1998). Specifically, an increase or decrease in PA greater than a certain set point activates behavioural and/or energy compensatory responses to maintain stable total energy expenditure levels. For instance, a sudden increase in vigorous PA may increase energy expenditure greater than then set point, initiating compensatory events such as an increase in sedentary behaviour (SB) and/or a decrease in nonessential energy expenditure (e.g., inflammation, stress hormone levels, immune function, reproductive hormone levels). Thus, returning energy expenditure levels to its target state, within its set point. The ActivityStat hypothesis is supported by the constrained total energy expenditure model which suggest that the compensation phenomenon is a physiologically mechanism that keeps energy requirements in check while prioritizing and allocating energy for reproductive fitness and survival (Pontzer, 2015; Pontzer, 2018).

In studies involving children and adolescents, behavioural compensation has been examined both within- and between-days. For example, within-day activity compensation was

found in youth (7 to 18 years of age), engaging in an active morning commute who then walked less throughout the day (Tan et al., 2018). As for between-days compensatory effects, a 10-minute increase in sedentary time on one day was followed by a decrease of 3.1 minutes the next day in children and adolescents (Mackintosh, Ridgers & McNarry 2019). Additionally, two hours more of SB in any given day was associated with an increase of 5 minutes of moderate to vigorous PA (MVPA) the next day in youth between 6 to 17 years of age (Nigg et al., 2022). However, findings from other empirical studies and systematic reviews are inconclusive or do not support evidence of such compensatory behaviour (Bagget et al., 2010; Beck et al., 2022; Beck, Engel & Reimers, 2022; Costigan et al., 2018; Gomersall et al.; 2013; Goodman, Mackett, & Paskins, 2011; Lamboglia et al., 2022; Long et al., 2013; Silva et al., 2018; Swelam et al., 2022; Washburn et al., 2014).

The ActivityStat hypothesis has been tested mainly using traditional longitudinal multilevel models (e.g., hierarchical linear models, mixed models) (Dale, Corbin & Dale, 2000; Mackintosh, Ridgers, & McNarry 2019; Nigg et al 2022; Ridgers et al., 2014; Ridgers et al., 2015; Ridgers et al., 2018). While multilevel analyses are well suited for measuring growth and change over time, they are not suited for examining the dynamic compensatory processes suggested by the ActivityStat hypothesis. Further, these traditional methods can only test univariate models (Ruissen et al., 2022). Specifically, the relationship among predictor and outcome can be only examined in one direction or the other (e.g., one model for MVPA predicting SB and a separate model for SB predicting MVPA). Therefore, because univariate approaches do not account for other dynamic relationships, coefficients from this model can be biased. Thus, findings for the ActivityStat hypothesis that are based on traditional longitudinal multilevel models should be interpreted with caution.

Instead, dynamic modelling provides a novel approach to examine complex processes and overcome the limitations of traditional longitudinal multilevel models (Ruissen et al., 2022). Specifically, continuous-time structural equation modelling (CT-SEM) allows for the study of relationships among processes over time continuously, and for meaningful interpretation of the findings. Another innovative advantage is the possibility of answering research questions regarding the temporal specificity (i.e., timeframe) of when the relationships among processes occur. In traditional models, can only make inferences about the compensatory relationships at one point in time (e. g., one day). These findings can not generalize to other timeframes, and thus provide an incomplete picture of how behavioural compensation unfolds over time. To best of our knowledge, CT-SEM has yet to be employed to investigate the ActivityStat hypothesis.

Therefore, the current study investigated the ActivityStat hypothesis in adolescents by using CT-SEM to (1) examine the presence of continuous-time multivariate relationships between PA levels (i.e., light PA [LPA], MVPA) and SB (as represented by stationary time [ST]; Tremblay et al., 2017), and to (2) explore the temporal specificity underlying the multivariate relationships between PA levels and ST. In line with the ActivityStat hypothesis (Gomersall et al., 2013; Rowland, 1998), compensation is determined when: a) a negative within movement behaviour (i.e., LPA, MVPA, ST) relationship exists; b) positive reciprocal relationships are observed between PA levels (i.e., LPA, MVPA) and ST; or c) a negative reciprocal relationship exists between LPA and MVPA.

## Methods

### Participants and procedures

This study is a secondary analysis of data derived from the Built Environment and Active Transport to School (BEATS) study (Mandic et al., 2015). The original project focused on active transport to school in adolescents. Specifically, its purpose was to (1) understand adolescents' and their parents' choice of transport mode to school; (2) examine the interaction between the transport choices, built environment, PA levels, and weight status in adolescents; and (3) identify policies that promote or limit active transport to school in adolescents. Participants included students in years 9 to 13 (between 13 to 18 years of age) attending schools in Dunedin, New Zealand. A total of 1780 adolescents were recruited in 2014 and 2015 and 433 were asked to wear an accelerometer. Further details about the recruitment procedure are reported elsewhere (Mandic et al., 2015; Mandic et al., 2016). The BEATS project received ethics clearance by the University of Otago Research Ethics Board (Study ID: 13/203). For the secondary analysis, ethics approval was granted by the University of Alberta research ethics board (Study ID, Pro00119474).

**Measures**

PA and ST were measured with ActiGraph WGT3XPlus accelerometers. Participants were instructed to wear the accelerometer on an elastic belt on their right hip for at least 12 hours, except during water-based activities. Data was collected for up to 8 days and downloaded in 10-second epoch (Migueles et al., 2017). LPA, MVPA, and ST (i.e., no movement during waking hours; Tremblay et al., 2017) were the outcomes of interest for this study. Low frequency filtered files were used to categorize adolescents' LPA, MVPA, and ST using Evenson cut-points (Evenson et al., 2008). A minimum of 3 days with at least 10 hr/day (i.e., 600 minutes) represented a valid and reliable estimate for PA and ST (Corder et al., 2008; Rich et al., 2013).

**Statistical analysis**

Bayesian hierarchical CT-SEM were fit using the ctsem package (Driver et al., 2017), which interfaces to Stan (Stan Development Team, 2023) in the R 4.2.1 environment (R Core Team, 2021). We computed a CT-SEM model examining the multivariate dynamic system with reciprocal relationships between LPA, MVPA, and ST (Appendix B). Previous attempts to study complex systems accounting for within- and between-person variability using maximum likelihood (ML) approaches were computationally intractable. In contrast, Bayesian estimation (i.e., Markov Chain Monte-Carlo) offers improved computational/estimation methods for those situations in which ML has difficulty (Muthén & Asparouhov, 2012). CT-SEM uses distinct modeling that provide more flexibility and does not require strict assumptions that are often violated in traditional longitudinal multilevel models (Driver & Voelkle, 2018a)

Students who did not meet the accelerometer wear time criteria (n = 40) and boarders (i.e., students not living with their family; n = 37) were excluded from the analysis. Data were structured in a stacked format (i.e., long format) and a time index variable was created to identify each time-point (i.e., day, $t$ = 0 to 7). The data were standardized and grand-mean centered to facilitate model convergence. Though Bayesian estimation methods are robust to moderate violations of the normality assumption (Hecht & Zitzmann, 2021a), the shape of the distributions for the outcomes of interest were visually inspected, and all approximated normal symmetric distribution. Due to the use of a continuous-time framework for this study, missing data was considered missing at random. Specifically, it was interpreted as reflecting unequal time intervals between measurements, which implies that the number of observed time points is infinite (Oud & Voelkle, 2014).

As recommended for Bayesian models, the default burn-in (50% of then chain [i.e., one sequence of randomly sampled values]), the default aggregation statistic (mean of the chain), and the default priors (Driver & Voelkle, 2018a) were used. The model (i.e., LPA, MVPA, ST model) was run using a NUTS (No U-Turn sampler) with four chains and 20,000 iterations per chain (Gelman et al., 2013). For the model convergence and statistic precision, the potential scale reduction factor $\hat{R}$ and effective sample size ($N_{eff}$) were reported (Gelman et al., 2013; Zitzmann & Hecht, 2019). A fully random effects model was first performed, however, problems occurred with model fit. A random intercept only model presented a better fit with an adequate model convergence and precision. The inclusion of the random intercept allows for person-specific differences in the average levels of LPA, MVPA, and ST, but fixes the compensatory processes between movement behaviours to be the same across participants. As the ActivityStat hypothesis proposes that these compensatory mechanisms occur within-individuals' response (Gomersall et al., 2013), marked differences in these compensatory dynamics between individuals were outside the scope of this study. Additionally, the measurement model was removed from the analysis as it was not converging well. That means only the observed scores was modelled. Thus, findings are derived from the random intercept model without the measurement model. Further details about the complete code and the fully random effects model output illustrating the pattern of results can be seen in Appendices C and E.

The auto- and cross-effects parameters examined the direct instantaneous ($\Delta t \rightarrow 0$) temporal relationships and refer to the continuous-time parameters. The auto-effects reflect the stability (or persistence) of the relationship within a behaviour over time. The cross-effects describe the reciprocal effects between two distinct behaviours. The autoregressive and cross-lagged effect terms refer to the discrete-time parameters. From these parameters, the effect of

dynamic processes was examined throughout all time intervals. For interpreting the results, the parameter's posterior mean in relation to its posterior standard deviation (SD) and posterior 95% Bayesian credibility intervals (BCI) were examined. Meaningful results are apparent when zero does not fall within the lower (2.5%) or upper (97.5%) limits of the BCI for both the continuous-time and discrete-time parameters. To examine the size of the cross-lagged effects between the behaviours, the guidelines proposed by Orth and colleagues (2022) were followed. The time interval where the cross-lagged dynamics reached their peak effects and the discrete-time coefficients at those maximum or minimum time intervals was determined. Evidence of continuous-time temporal relationships is indicated by meaningful auto- or cross-effect relationships between PA levels and ST. As for evidence of temporal specificity, it is indicated by the timeframe at which the autoregressive or the cross-lagged effects between PA levels and ST reach their peak effects with behaviours aggregated at a day level. In the analysis, time-varying factors are statistically accounted for based on the stochastic element of our model (i.e., diffusion matrix). Time-invariant differences (i.e., age, gender) were accounted for through a random intercept (Hamaker, Kuiper, & Grasman, 2015; Mund, Johnson, & Nestler, 2021).

## Results

The Bayesian estimation needed approximately 20 RAM usage and 27 hours of runtime for the LPA, MVPA, and ST model, on an Intel(R) Xeon(R) Gold 6134 (3.20GHz and 3.19GHz processors) CPU of a 64-bit operating system and x64-based processor, with 128 GB RAM. All parameters had a minimum of 1549.56 effective samples and a $\hat{R}$ (Rhat) of 1.0, indicating adequate model convergence and precision. Based on the prediction model by Hecht and Zitzmann (2021a, 2021b) using $N = 356$, $T = 8$ ($t = 0$ to 7), and standardized peak effect set to

0.01 (small effect), our *post hoc* analysis suggests we had a sufficient sample size to reliably estimate the continuous-time cross-lagged dynamics between LPA, MVPA, and ST (estimated "power" for standardized peak cross-lagged effect = 1.00).

A total of 356 adolescents met the minimum wear-time criteria with an average of 827.81 min per day (SD = 106.64, range 601.83 to 1439). Girls represented 65.2% of the sample and average age was 14.7 ± 1.4 years. Adolescents engaged in an average of 192.60 ± 62.42 minutes of LPA, 55.90 ± 32.88 minutes of MVPA, and 576.03 ± 115.03 minutes of ST per day.

Table 5.1 displays a descriptive analysis of how each behaviour alter over time. The $T_0$ mean quantifies the extent to which their initial levels of behaviour (i.e., LPA, MVPA, ST at t = 0) are higher or lower than the subsequent states (i.e., t = 1 to 7; Driver & Voelkle, 2018a, 2018b). A negative estimate indicates that the initial process was lower than future states, whereas a positive estimate indicates that the initial state was higher. For LPA, MVPA, and ST, no meaningful increase or decrease was observed in the overall level over time, with the 95% BCI encompassing zero (LPA, M = 0.03, SD = 0.06, 95% BCI [-0.09, 0.14]; MVPA, M = 0.01, SD = 0.06, 95% BCI [-0.11, 0.12]; ST, M = -0.07, SD = 0.05, 95% BCI [-0.18, 0.03]). This means that LPA, MVPA, and ST fluctuated around a stationary, average level, during the observation period. The continuous-time intercept displays the average process means for each outcome. Because they were grand-mean centered and standardized to facilitate model convergence, they are not relevant for interpretation. The $T_0$ variance, which refers to the asymptotic within-person covariance, indicates that all processes studied have similar variation over time (LPA, M = 1.05, SD = 0.04, 95% BCI [0.97, 1.14]; MVPA, M = 0.99, SD = 0.04, 95% BCI [0.92, 1.07]; ST, M = 0.92, SD = 0.04, 95% BCI [0.85, 0.99]).

To examine the within-person effects of the continuous-time, multivariate dynamics between PA levels and ST, Table 5.2 shows the direct instantaneous temporal relationship a variable had with its own rate of change (i.e., auto-effects) and between two distinct variables (i.e., cross-effect). For the auto-effects, the closer the estimates are to zero, the longer the impulse response persisted over time (Driver & Voelkle, 2018a, 2018b). A negative estimate reveals a diminishing auto-effect over time (returning to baseline; Driver & Voelkle 2018b). On the contrary, a positive estimate represents an explosive process (distancing away from the baseline over time). As shown in Table 5.2 and Figure 5.1, the auto-effects for LPA (M = -2.46, SD = 0.46, 95% BCI [-3.56, -1.76]), MVPA (M = -2.28, SD = 0.41, 95% BCI [-3.26, -1.72]), and ST (M = -2.73, SD = 0.58, 95% BCI [-4.16, -1.92]) demonstrated some degree of persistence, with all parameters excluding zero. Thus, illustrating that LPA, MVPA, and ST were predictors of themselves at a later point in time after accounting for all other dynamic relationships included in the model. Figure 5.1 displays the direction of these relationships showing that all coefficients are positive over all time intervals, demonstrating persistence in processes. In contrast, negative coefficients illustrate an oscillating process (Schuurman et al., 2016) which were not apparent in this study. For the autoregressive effects (i.e., the discrete-time parameters), illustrated in Figure 5.1, adolescents' LPA, MVPA, and ST appear to be predictive of their later behaviours until about 1.7 to 2.5 days hence, after accounting for all other dynamic relationships included in the multivariate dynamic model. For instance, a change in adolescent's LPA at one moment in time was predictive of their subsequent LPA until about 2.5 days after the change in LPA. As for the MVPA and ST autoregressive effects, the timeframe in which a behaviour predicts its later behaviour appears to be about 1.7 and 2 days, respectively. Therefore, LPA is relatively more persistent over time compared to MVPA and ST.

For the cross-effects, a negative estimate indicates that an increase in the rate of change of one process predicts a decrease in the rate of change of the other process. A positive cross-effect, in contrast, indicates that an increase in the level of one process predicts an increase in the other process (Driver & Voelkle, 2018a, 2018b). As demonstrated in Table 5.2 and Figure 5.2, the relationship in the levels of ST and LPA is negative, after accounting for all other dynamic relationships included in the model. Specifically, an increase in the rate of change of ST at one time point predicted a subsequent decrease in the rate of change of LPA at a later point (ST_LPA, M = -1.25, SD = 0.5, 95% BCI [-2.4, -0.45]). The influence of ST on subsequent LPA was relatively large with cross-lagged effect sizes corresponding to 0.18 (Figure 5.2). The discrete-time cross-lagged effects between ST and LPA show the largest temporal dependence at 0.4 days between measurement occasions. As for the relationship between LPA and ST, both Table 5.2 and Figure 5.2 show a meaningful negative dynamic relationship (LPA_ST, M = -0.61, SD = 0.36, 95% BCI [-1.44, -0.01]). Thus, an increase in the rate of change of LPA at one time point predicted a subsequent decrease in the rate of change of ST at a later point, after accounting for all other dynamic relationships included in the model. The cross-lagged effect size of this relationship is 0.08, which corresponds to a medium effect. The effects of LPA on ST appears largest at 0.4 days between measurement occasions. The direct instantaneous temporal relationship between ST and MVPA reveals no meaningful relationship (ST_MVPA, M = 0.61, SD = 0.36, 95% BCI [-0.03, 1.39]). However, Figure 5.2 shows a positive meaningful relationship with the 95% BCI not encompassing zero in some time intervals. Thus, an increase in the rate of change of ST at one time point predicted a subsequent increase in the rate of change of MVPA at a later point, after accounting for all other dynamic relationships included in the model. The influence of ST on subsequent MVPA was medium with the cross-lagged effect sizes

corresponding to 0.09. The temporal dependency between ST and MVPA was largest at 0.4 days between measurement occasions. As for the remaining dynamic relationships (i.e., LPA_MVPA, MVPA_LPA, and MVPA_ST), an increase in the levels of one process did not predict any subsequent change in the levels of the other constructs over time. However, the temporal dependencies between these movement behaviours were largest approximately 0.5 days between measurement occasions.

The diffusion parameters in Table 5.2 present further information regarding the temporal relationship between the processes. These parameters account for fluctuations in dynamic processes of interest over time that cannot be accounted by the deterministic model elements (i.e., $T_0$ mean, drift matrix parameters). The on-diagonal elements of the diffusion matrix are variances that quantify the extent to which the variables in a dynamic system are influenced by unmodelled exogenous inputs (Driver & Voelkle, 2018a, 2018b). The findings indicate that all latent processes (i.e., LPA, MVPA, ST) were influenced by random (i.e., unpredictable) fluctuations over time. More specifically, LPA, MVPA, and ST have similar levels of variation from exogenous inputs (LPA, M = 1.58, SD = 0.12, 95% BCI [1.39, 1.87]; MVPA, M = 1.8, SD = 0.14, 95% BCI [1.59, 2.13]; ST, M = 1.8, SD = 0.16, 95% BCI [1.56, 2.2]). The off-diagonal elements of the diffusion matrix represent covariances that quantify the extent to which the stochastic variation between two observed processes share common causes (Driver & Voelkle, 2018a, 2018b). The results of the model show that meaningful positive covariation exists between MVPA and LPA (M = 0.2, SD = 0.04, 95% BCI [0.12, 0.3]). In contrast, ST and MVPA present a meaningful negative covariance (M = -0.13, SD = 0.05, 95% BCI [-0.22, -0.03]). These relationships suggest that the unmodeled, time-varying factors that influence MVPA also influence LPA and ST. Surprisingly, however, the 95% BCI for ST and

LPA include zero suggesting that the unmodeled factors that cause fluctuations in LPA were not shared with ST (M = 0.03, SD = 0.05, 95% BCI [-0.06, 0.14]).

The between-person relationships among LPA, MVPA, and ST are presented in Table 5.3. The results show that MVPA and LPA were dependent on each other. Specifically, individuals' average daily MVPA was positively related to average LPA. No meaningful relationship was found between individuals' levels of ST and either of LPA or MVPA.

## Discussion

The primary aim of this study was to examine the presence of continuous-time multivariate relationships between PA levels and ST in adolescents. We found that adolescents' level of LPA, MVPA, and ST on a given day positively predicted LPA, MVPA, or ST later in time, respectively. For the cross-behavioural relationships, levels of LPA and ST on a given day negatively predicted ST and LPA later in time, respectively. Additionally, levels of ST on a given day positively predict MVPA later in time. Though behavioural compensation was not observed for LPA and MVPA, it was noted for ST. Thus, the findings support evidence of the ActivityStat hypothesis in adolescents.

The biological basis of PA regulation can occur symmetrically due to its homeostatic nature (Gomersall et al., 2013; Rowland, 1998). Specifically, an increase or decrease in total energy expenditure beyond the biological setpoint would activate compensatory mechanisms that return the system back to its stable state. Evidence of compensation for LPA and MVPA was not observed in our study, instead, findings suggest activity synergy (i.e., engaging in a behaviour may increase activity at another time), similar to previous research (Goodman, Mackett, & Paskins, 2011; Long et al., 2013). Even though the literature has mostly shown behavioural

compensation in adolescents after engaging in sudden levels of PA, we found evidence of compensation for ST. Likewise, Nigg and colleagues (2022) reported displacement of sedentary-related behaviour by PA. Specifically, children and adolescents who spent two more hours than usual in sedentary-related behaviours increased MVPA by five minutes the next day. However, evidence supporting behavioural displacement in youth is limited (Beck, Engel, & Reimers, 2022). Furthermore, our results show a negative reciprocal relationship between LPA and ST, thus suggesting that, within a specific period of time (e.g., 24 hrs), these behaviours are mutually exclusive (Pedišić, Dumuid, & Olds, 2017). This means that changing the amount of time engaged in one behaviour leads to changes in the allotted time of one or more other behaviours.

Regardless, studies testing the ActivityStat hypothesis in adolescents show conflicting results (Bagget et al., 2010; Goodman, Mackett, & Paskins, 2011; Long et al., 2013; Nigg et al., 2022; Paravidino et al., 2017; Tan et al., 2018; Thivel et al., 2013). It is possible that some of this variation in effect is due to the intensity of the stimulus (e.g., duration of MVPA). Specifically, in observational studies that do not support the ActivityStat hypothesis, individuals may not be exceeding their internal activity set point (Gomersall et al., 2013). Considering that activity volume is critically important to trigger any supposed compensatory mechanism, future studies should examine this hypothesis in experimental designs.

Additionally, studies refuting (Bagget et al., 2010; Goodman, Mackett, & Paskins, 2011; Long et al., 2013) and supporting (Nigg et al., 2022; Paravidino et al., 2017; Tan et al., 2018; Thivel et al., 2013) with ActivityStat in the population of adolescents present a major limitation regarding the statistical modelling employed. The use of repeated measures ANOVA and traditional regression models are mainly performed to study this phenomenon, disregarding the temporal multivariate dependence among LPA, MVPA, and ST. Dynamic modelling methods, in

contrast, would allow the study of complex systems accounting for all multivariate relationships among the processes included in the model. The use of CT-SEM, specifically, would include the analysis of those processes continuously over time (Ruissen et al., 2022). Therefore, future studies investigating the ActivityStat hypothesis should employ this novel method.

An additional purpose of this study was to explore the temporal specificity underlying the multivariate relationships between PA levels and ST. Specifically, adolescents who moved more at a specific time positively predicted LPA and MVPA until about 2.5 and 1.5 days later, respectively. Additionally, adolescents who moved less at a specific time positively predicted ST until about 2 days hence. As for the discrete-time cross-lagged effects, adolescents who move less than usual at a specific time point negatively predicted LPA until 0.4 days hence. The same pattern was observed for the effects of ST on LPA. As for the relationship between ST and MVPA, adolescents who move less than usual at one time point positively predicted PA levels at another point until 0.4 days later. Though most of the findings are in the opposite direction of what would have been anticipated by the ActivityStat hypothesis, the later indicates evidence of compensation occurring in a short-term. According to Gomersall and colleagues (2013), the compensation mechanisms are unlikely to occur within a short timescale (i.e., within- and between-days). Furthermore, because compensation can be triggered by behavioural and/or energy-related mechanisms, the timeframe may vary by mechanism. Specifically, behavioural compensatory mechanisms may be initiated sooner than those for energy conservation. To date, the timeframe during which behavioural compensation occurs has been investigated in a short time interval for studies supporting (Nigg et al., 2022; Paravidino et al., 2017; Tan et al., 2018; Thivel et al., 2013) and refuting (Bagget et al., 2010; Goodman et al., 2011; Long et al., 2013) the ActivityStat hypothesis in adolescents. In addition, findings from these studies are bound to a

specific discrete time interval (Ruissen et al., 2022; Voelkle et al., 2018). The use of a continuous-time framework, in contrast, allows the dynamic processes studied existing continuously making inferences about the dynamic processed studied from the observed and non-observed time measurement intervals. Therefore, future studies testing the ActivityStat should include timeframes of multiple durations to determine if and when compensation may occur. This potentially has practical importance for designing PA programs impacting on the design, frequency of measurements, and duration of the intervention (Gomersall et al., 2013).

Further interpretation of the temporal relationship, displayed in the diffusion parameters, between the processes shows that LPA, MVPA, and ST are influenced by other factors not included in the analysis. For instance, emerging investigations indicate that sleep influence PA and SB (Kim et al., 2020; Master et al., 2019; Tremblay et al., 2016), as it is a component of the movement behaviour intensity continuum (Tremblay et al., 2010; Tremblay et al., 2016). The present findings also suggest that positive variation in the levels of LPA and MVPA shares common causes. For instance, favorable weather conditions increase levels of LPA and MVPA simultaneously in adolescents (Zheng et al., 2021). Findings from the present study also indicate a negative variation in the levels of MVPA and ST that share common causes. The impact of day of the week on how much adolescents move may explain this finding. PA and sedentary time tend to be lower on weekends due to a shorter waking period (Burchartz et al., 2022). Unexpectedly, we found that LPA and ST do not share common causes, thus, challenging the temporal relationship between LPA and ST demonstrated in this study. Specifically, posture allocation may present a common cause for determining LPA and ST, as transitioning between these behaviours may only require a change in posture (Tremblay et al. 2017). The unexpected finding may be due to the removal of the dynamic measurement model from the analysis which

accounts for measurement error in each individual movement behaviour. Even though device-assessed measurements are a reliable tool to assess activity levels (Westerterp, 2009), distinguishing body posture (e.g., sitting vs standing) in ActiGraph WGT3XPlus accelerometers present critical limitations (Edwardson et al., 2016). Future work should continue accounting for unmeasured, time-varying factors, through stochastic models, or considering other time-varying factors that exert influence in supposed compensation mechanisms (e.g., age, weekday vs weekend, season).

The main strength of this study is the Bayesian CT-SEM, allowing the use of longitudinal multiple observations from the same participant to examine multivariate relationships among different behaviours employing a continuous-time approach (Driver & Voelkle, 2018a; Voelkle et al., 2018). The relatively large study sample of adolescents living in Dunedin and the device-assessed measurement of activity levels are also strengths of this study. However, several limitations should be acknowledged. First, the non-experimental design is a limitation considering that the activity stimulus must be sufficient to trigger any possible compensatory mechanisms. Second, the inclusion of the dynamic measurement model was not feasible, which may have impacted the diffusion parameter (i.e., ST_LPA). However, it did not affect patterns of the auto and cross-effects. Third, SB was represented as ST because the Actigraph device is limited in assessing posture, which is a key component for determining SB status (Tremblay et al., 2017b). Lastly, sleep behaviour was not assessed.

## Conclusion

This study provides novel insights on examining the continuous-time multivariate relationships among PA levels and ST to investigate the ActivityStat hypothesis in adolescents.

Our findings suggest positive continuous-time relationships within LPA, MVPA, and ST. The timeframe during which these relationships occur is about 1.7 to 2.5 days hence. Further, a cross-behavioural reciprocal and negative relationship was noted between LPA and ST, occurring about 0.4 days later. Additionally, a positive relationship existed between ST and MVPA, occurring about 0.4 days later. Though behavioural compensation was not observed for LPA and MVPA, it was noted for ST. Thus, the findings support evidence of the ActivityStat hypothesis in adolescents.

*Table 5.1* Means, standard deviations, and posterior credibility intervals for the T0 means, continuous-time intercept, and manifest variance for the levels of LPA, MVPA, and ST

| Parameter | | Est. | SD | BCI [2.5%, 97.5%] | | Rhat | $N_{eff}$ |
|---|---|---|---|---|---|---|---|
| $T_0$Mean | LPA | 0.03 | 0.06 | -0.09 | 0.14 | 1 | 37008.56 |
| | MVPA | 0.01 | 0.06 | -0.11 | 0.12 | 1 | 41825.85 |
| | ST | -0.07 | 0.05 | -0.18 | 0.03 | 1 | 34532.53 |
| Continuous-Time Intercept | LPA | -0.03 | 0.09 | -0.22 | 0.16 | 1 | 9219.06 |
| | MVPA | 0.00 | 0.09 | -0.18 | 0.18 | 1 | 12379.64 |
| | ST | -0.01 | 0.1 | -0.21 | 0.18 | 1 | 13297.55 |
| $T_0$Variance | LPA | 1.05 | 0.04 | 0.97 | 1.14 | 1 | 38674.68 |
| | MVPA | 0.99 | 0.04 | 0.92 | 1.07 | 1 | 45662.25 |
| | ST | 0.92 | 0.04 | 0.85 | 0.99 | 1 | 36438.16 |

Continuous-time intercept = b coefficient; Est. = mean of the chain; BCI = Bayesian credibility interval; Rhat ($\hat{R}$) = potential scale reduction factor; $N_{eff}$ = effective sample size; LPA = light physical activity; MVPA = moderate-vigorous physical activity; ST = stationary time.

*Table 5.2* Posterior means, standard deviations, and credibility intervals for the means of estimated population distributions of the relationship between LPA, MVPA, and ST

| Parameter | | Est. | SD | BCI [2.5%, 97.5%] | | Rhat | $N_{eff}$ |
|---|---|---|---|---|---|---|---|
| Auto-effects parameters | LPA | -2.46 | 0.46 | -3.56 | -1.76 | 1 | 3357.9 |
| | MVPA | -2.28 | 0.41 | -3.26 | -1.72 | 1 | 2808.23 |
| | ST | -2.73 | 0.58 | -4.16 | -1.92 | 1 | 3310.66 |
| Cross-effects parameters | LPA_MVPA | -0.17 | 0.3 | -0.81 | 0.38 | 1 | 1798.11 |
| | LPA_ST | -0.61 | 0.36 | -1.44 | -0.01 | 1 | 1549.56 |
| | MVPA_LPA | -0.53 | 0.41 | -1.4 | 0.22 | 1 | 4143.17 |
| | MVPA_ST | -0.42 | 0.38 | -1.2 | 0.31 | 1 | 1696.1 |
| | ST_LPA | -1.25 | 0.5 | -2.4 | -0.45 | 1 | 3829.75 |
| | ST_MVPA | 0.61 | 0.36 | -0.03 | 1.39 | 1 | 4442.98 |
| Diffusion parameters | LPA | 1.58 | 0.12 | 1.39 | 1.87 | 1 | 5244.41 |
| | MVPA | 1.8 | 0.14 | 1.59 | 2.13 | 1 | 2921.94 |
| | ST | 1.8 | 0.16 | 1.56 | 2.2 | 1 | 3385.86 |
| | MVPA_LPA | 0.2 | 0.04 | 0.12 | 0.3 | 1 | 2508.5 |
| | ST_LPA | 0.03 | 0.05 | -0.06 | 0.14 | 1 | 2084.46 |
| | ST_MVPA | -0.13 | 0.05 | -0.22 | -0.03 | 1 | 2414.82 |

Est. = mean of the chain; BCI = Bayesian credibility interval; Rhat ($\hat{R}$) = potential scale reduction factor; $N_{eff}$ = effective sample size; LPA = light physical activity; MVPA = moderate-vigorous physical activity; ST = stationary time.

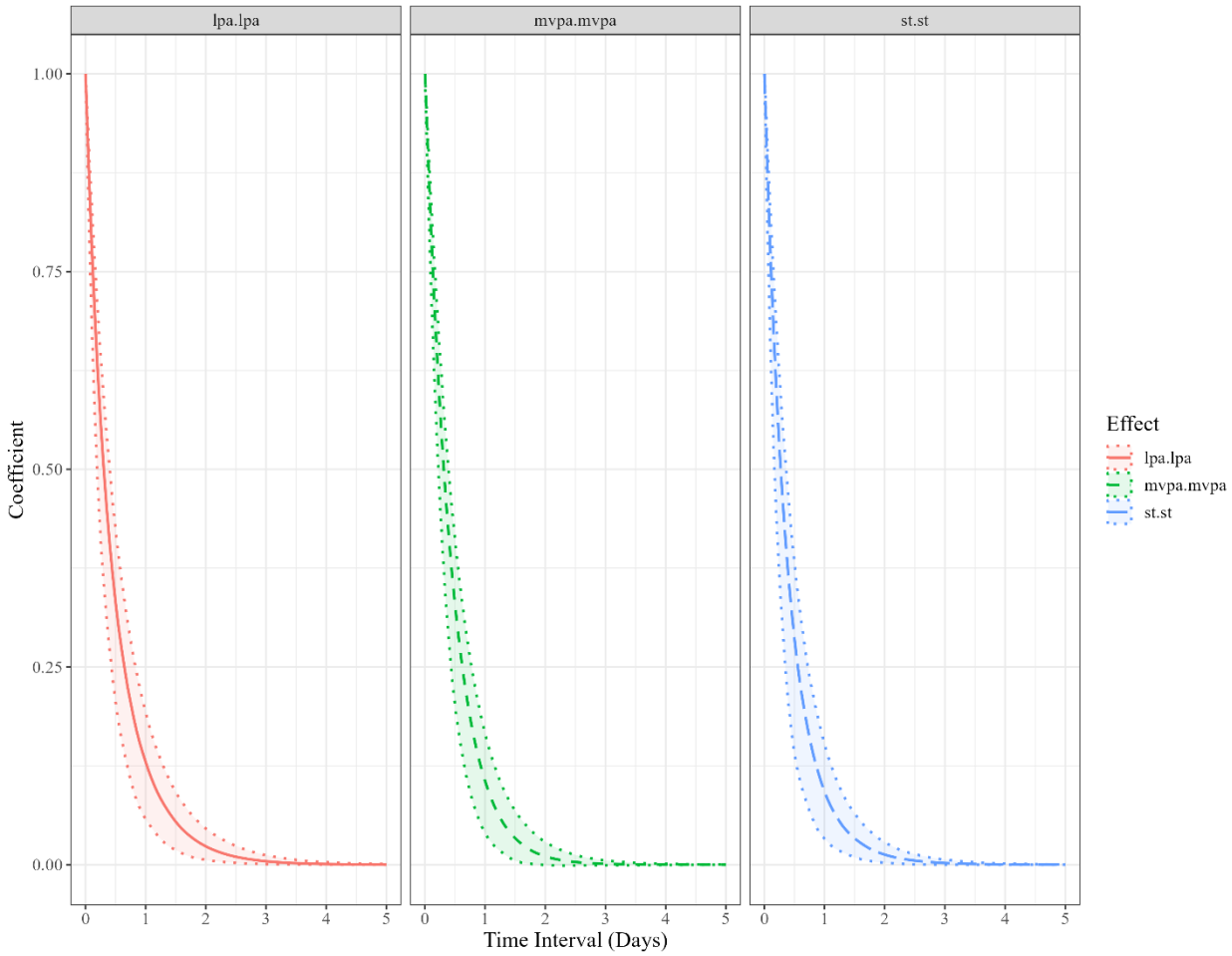


*Figure 5.1* Posterior mean and 95% credibility intervals for the standardized discrete-time autoregressive effects.

LPA = light physical activity; MVPA = moderate-vigorous physical activity; ST = stationary time.

*Note*. This figure shows that all autoregressive coefficients are above zero, demonstrating positive relationships within movement behaviours. The largest timeframe of these relationships become apparent when the 95% Bayesian credibility is approaching zero. Thus, the LPA_LPA, MVPA_MVPA, and ST_ST largest timeframe are 2.5, 1.7, and 2 days, respectively.

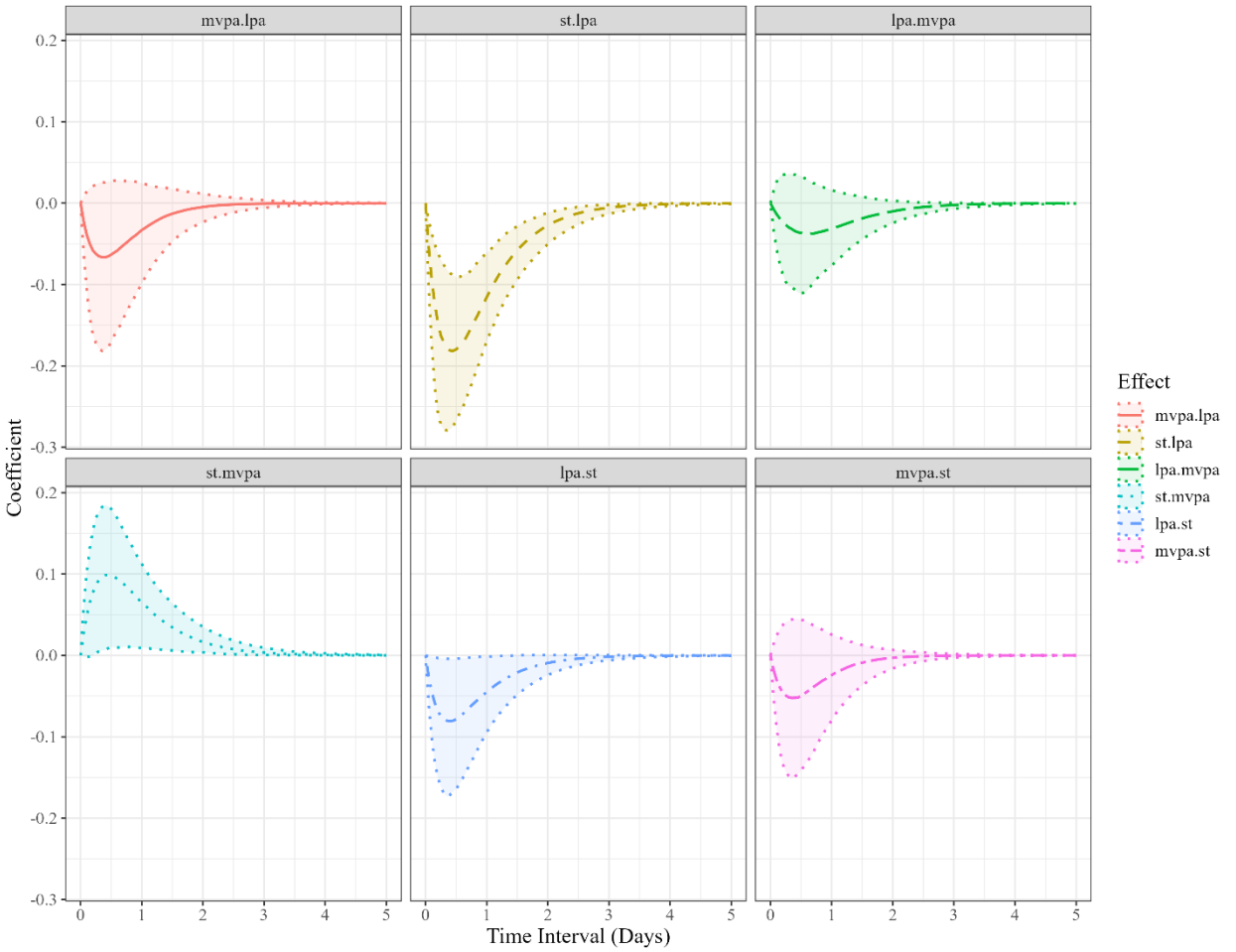


*Figure 5.2* Posterior mean and 95% credibility intervals for the standardized discrete-time cross-lagged effects

LPA = light physical activity; MVPA = moderate-vigorous physical activity; ST = stationary time.

*Note*. This figure shows that the cross-behavioural relationships among ST_LPA, ST_MVPA, LPA_ST do not include zero in some time intervals, demonstrating meaningful negative relationships between ST_LPA and LPA_ST, and meaningful positive relationships between ST_MVPA. The largest timeframe of these relationships become apparent at about 0.4 days. The remaining cross-behavioural relationships demonstrated no meaningful associations over time.

*Table 5.3* Posterior means, standard deviations, and credibility intervals for the between-person means of estimated population distributions of the relationship between LPA, MVPA, and ST

| | b_LPA | b_MVPA | b_ST |
|---|---|---|---|
| b_LPA | - | - | - |
| b_MVPA | 0.51 (0.17)<br>[0.14, 0.78] | - | - |
| b_ST | 0.0 (0.23)<br>[-0.43, 0.45] | -0.32 (0.21)<br>[-0.68, 0.14] | - |

b = continuous-time intercept; LPA = light physical activity; MVPA = moderate-vigorous physical activity; ST = stationary time. Rhat ($\hat{R}$) = 1.00; $N_{eff}$ = 38881 to 40257.

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# CHAPTER 6: General Discussion

## Overview

The overall goal of this dissertation is to systematically advance the field of a possible biological determinant of physical activity (PA) known as the ActivityStat hypothesis (Rowland, 1998; Gomersall et al., 2013). The idea of an internal biological determinant of activity in humans is not new; however, it is now receiving more attention from the research community. Among the objectives of this dissertation are to address some research gaps previously identified for the ActivityStat hypothesis including: 1) the population studied; 2) the application of novel statistical modelling; 3) examining relationships among behaviours; and 4) the timeframe in which these relationships occur. To address the overall goal and specific objectives, a scoping review was performed to investigate this hypothesis in individuals living with liver disease. Secondary analyses were conducted to examine the compensatory mechanisms in two sample groups: preschool-aged children and adolescents. This chapter will summarize key findings of the three studies, outline overarching strengths and limitations of the research, and identify key implications for future research.

## Summary of findings

Studying the ActivityStat hypothesis in unique populations is the first step in advancing research, as this phenomenon has been mainly explored in healthy populations and adults living with overweight and obesity (Beck, Engel & Reimers, 2022; Gomersall et al., 2013; Silva et al., 2018; Swelam et al., 2022; Washburn et al., 2014). The original intent of my dissertation was to test the hypothesis in an intervention involving adults living with liver disease. Thus, for Study 1, a scoping review was conducted addressing the following objectives: (1) to assess whether individuals living with liver disease display behavioural (i.e., PA, sedentary behaviour [SB], and

sleep) and/or energy (i.e., total energy expenditure [EE], basal EE, resting EE, and activity EE) compensation throughout the day and/or days; and (2) to examine whether a prescribed PA intervention would trigger compensatory responses. Findings revealed insufficient evidence to support the ActivityStat hypothesis throughout the day and/or days in people living with liver disease. Additionally, findings related to the effects of a PA intervention on behavioural and energy responses did not support the ActivityStat hypothesis in this population.

Due to interruptions related to the COVID-19 pandemic, I switched the focus of my dissertation to children and adolescents. The ActivityStat has been understudied in these populations. Also, existing studies testing this hypothesis mainly used traditional longitudinal multilevel models (e.g., hierarchical linear models, mixed models) (Dale, Corbin & Dale, 2000; Mackintosh, Ridgers, & McNarry 2019; Nigg et al 2022; Ridgers et al., 2014; Ridgers et al., 2015; Ridgers et al., 2018), disregarding the temporal multivariate dependence among PA levels (i.e., light PA [LPA], moderate-vigorous PA [MVPA]) and other movement behaviours (i.e., stationary time [ST]). In order to account for these multivariate relationships, a novel approach to understanding complex processes that impact PA levels and ST is employed in Studies 2 and 3. Specifically, these studies investigated the ActivityStat hypothesis by examining the presence of continuous-time multivariate relationships between LPA, MVPA and ST in a population of preschool-aged children and adolescents. Additionally, we explored the timeframe during which the multivariate relationships existed between LPA, MVPA, and ST. Though we found positive continuous-time relationships within LPA, MVPA, and ST, both in preschool-aged children and adolescents, relationships between the behaviours were observed only in adolescents after accounting for all other dynamic relationships examined. In one case, a cross-behavioural reciprocal and negative relationship was demonstrated between LPA and ST in adolescents. In

addition, a positive cross-behavioural relationship was observed between ST and MVPA. Among the children, the timeframe at which LPA, MVPA, and ST predicted their later behaviours was about 0.5 days hence. As for adolescents, within-behaviour relationships predicted their behaviours about 1.7 to 2.5 days later. All cross-behaviour relationships (i.e., ST on LPA, LPA on ST, and ST on MVPA) occurred about 0.4 days later. Though behavioural compensation was not indicated for most of the findings, the positive relationship of ST with MVPA suggest evidence for the ActivityStat hypothesis in adolescents.

**Strengths and limitations**

The specific strengths of each study are discussed in detail in Chapters 3-5. However, some overlap was seen across the studies. One of the major strengths of this dissertation is that it examines an intriguing phenomenon that has received limited attention. Studies examining the ActivityStat have mostly focused on adults (Beck, Engel & Reimers, 2022; Gomersall et al., 2013; Silva et al., 2018; Swelam et al., 2022; Washburn et al., 2014), whereas an investigation of this hypothesis in children, adolescents, and individuals living with chronic conditions is scarce. Thus, discussions about compensatory mechanisms in populations in which this phenomenon is not fully understood were conducted in all studies in this dissertation. Another distinct strength of this dissertation is the use of novel statistical modelling in Studies 2 and 3. Specifically, Bayesian continuous-time structural equation modelling (CT-SEM) allows the examination of complex and dynamic systems using dense sequences of repeated measurements to describe changes and understand the mechanisms underlying change (Ruissen et al., 2022). Additionally, the use of CT-SEM allows for both within- and between-person analyses (Ruissen et al., 2022), which are critically important for detecting compensation. Because compensatory mechanisms are hypothesised to occur at the intraindividual level (Gomersal et al., 2013), focusing solely on

between-person comparisons will perpetuate the "ecological fallacy." Specifically, false assumptions can be made when inference is made about an individual based on aggregated group-level data. CT-SEM also makes it possible to explore the timeframe of any supposed compensation discussing novel insights to the study of the ActivityStat hypothesis. Lastly, even though Bayesian estimation methods are particularly well-suited for studies with small samples (Depaoli et al., 2017; Smid et al., 2020), the sample sizes in Studies 2 and 3 were relatively large in comparison to previous work (e.g., Mackintosh, Ridgers, McNarry, 2019; Ridgers et al., 2014; Ridgers et al., 2018).

The specific limitations of each study are discussed in detail in Chapters 3-5. However, important limitations are addressed here. First, in Study 1, the included articles for the scoping review focused on individuals with distinct liver disease severity, across all ages, employed distinct PA and SB measurement techniques (e.g., direct and indirect measurements), and conducted PA interventions with distinct designs (e.g., intervention length, duration, intensity, and mode of the PA session) impacting whether a compensation effect would occur or be detected (Eisenmann & Wickel, 2009; Gomersall et al., 2013; Silva et al., 2018). Second, while the use of accelerometery data, in Studies 2 and 3, allowed for reliable, valid, and feasible measurement of motion, SB was operationalised as ST. Third, the absence of sleep to study the dynamic multivariate relationships among LPA, MVPA, and ST was another limitation. Fourth, because compensation could occur via EE mechanisms that were not necessary reflected in behavioural adaptations (Gomersall et al., 2013; King et al., 2007; Pontzer, 2015; Pontzer, 2017; Pontzer, 2018; Rowland, 1998), the lack of EE measures in these studies limited our ability to comprehensively test the hypothesis. Lastly, testing the ActivityStat hypothesis in observational

studies present a limitation. Because habitual activity patterns may show temporal variations in PA and ST, these can be misinterpreted as compensatory mechanisms.

**Implications and future directions**

The first implication of this research is that the ActivityStat hypothesis was not supported in most of the findings. Specifically, we found no evidence for behavioural compensatory mechanisms in individuals living with liver disease or pre-school aged children. One potential explanation is that our participants did not exceed their individual activity set point as a result of engaging in PA. Thus, compensatory behaviours were not initiated (Gomersall et al., 2013). Future studies should consider testing the hypothesis in experimental studies with different doses of PA to trigger any supposed compensation. Additionally, compliance with the PA program should be monitored during the intervention and subsequently reported in the study. If the ActivityStat is found to exist in individuals living with chronic diseases or pre-school aged children, programs that aim to increase PA need to consider that compensatory mechanisms may occur across the movement behaviour intensity continuum (i.e., sleep, SB, LPA, and MVPA; Tremblay et al., 2010), depending on the volume of the stimulus. Strategies to minimize an increase in sedentary-related behaviours as a response to a PA intervention could consider varying the intensity of the PA program. Ultimately, even though these compensation effects are a natural and a biological response (Gomersall et al., 2013; Rowland, 1998), experimental research is needed to investigate the impact of compensation on health indicators and whether different types of compensation affect health.

In contrast, a second implication is that the ActivityStat hypothesis was demonstrated in adolescents. Though behavioural compensation was not observed for PA (i.e., LPA, or MVPA), it was indicated for ST. In that case long periods of inactivity may stimulate bodily movement to

increase EE. Because the ActivityStat is a homeostatic and symmetrical system by nature (Gomersall et al., 2013), an increase or decrease of the regulated variable (i.e., EE) beyond the set point zone triggers a "correcting response" in the opposite direction to counterbalance the deviation and return the system back to its stable levels (Cabanac, 2001). While testing the ActivityStat hypothesis by restricting activity is unusual in the literature, evidence of compensation for SB has been demonstrated in non-experimental conditions in a sample of youth (Nigg et al., 2022).

Thirdly, the timeframe during which studies investigate the ActivityStat may be a critical factor. In our study, adolescents demonstrated behavioural compensation for ST 0.4 days later in time after a more than usual increase in ST. Because we employed a CT approach to understand the multivariate relationships between PA levels and ST, processes are analyzed continuously over time. Specifically, dynamic processes are studied from the observed and non-observed time measurement intervals allowing for a more comprehensive understanding about the role of time (Voelkle et al., 2018). However, compensation is unlikely to occur within a short term, rather, it is hypothesised to require weeks or months (Gomersall et al., 2013). Regardless of whether research supported the hypothesis (Mackintosh, Ridgers, & McNarry, 2019; Nigg et al., 2022; Paravidino et al., 2017; Ridgers et al 2014; Ridgers et al., 2015; Ridgers et al., 2018; Tan et al., 2018; Thivel et al., 2014; Wilkin et al., 2006) or refuted it (Cheung, 2019; Bagget et al., 2010; Goodman et al., 2011; Long et al., 2013; Nigg et al., 2022; Pesola et al., 2018; Smith et al., 2012), a majority of studies have employed a short-time interval in their investigation of behavioural compensation in preschool-aged children and adolescents. Furthermore, studies have applied traditional longitudinal analyses limiting the timeframe at which processes are analyzed (Voelkle et al., 2018). Future studies should investigate timeframes of multiple durations to

determine if and when compensation occurs. This potentially has practical importance for designing PA programs, as such information can impact the design, frequency of measurements, and duration of the intervention (Gomersall et al., 2013).

Another implication for future research is the choice of appropriate statistical modelling techniques for investigating behavioural compensation. Bayesian CT-SEM was chosen because it allows for a reasonable alignment with theory (i.e., ActivityStat) and methods. Specifically, the hypothesis suggests compensatory mechanisms among interconnected behaviours (i.e., PA and SB) (Gomersal et al., 2013), and available data to investigate the hypothesis contain dense sequences of repeated measurements. Because CT-SEM allows us to address all the nuances of the ActivityStat and employ intensive longitudinal data to examine associations among processes over time, this statistical modeling presents a potential tool for future studies to investigate compensatory mechanisms. Additionally, CT-SEM accounts for multivariate relationships among the constructs studied, which presents a major advantage when examining complex and dynamic systems (Ruissen et al., 2022). CT-SEM also facilitates the examination of relationships among processes over time, continuously allowing for meaningful interpretation of the finding (Ruissen et al., 2022). Therefore, to overcome limitations of traditional longitudinal multilevel modelling, future studies investigating the ActivityStat hypothesis should consider employing this novel method. Even though CT-SEM presents some challenges such as the running time required for the computer processing, and the need for strategies to reduce complexity (e.g., inclusion of the random intercept only), the advantages are numerous.

A recent review suggests using compositional data analysis (CoDA) as an alternative to examining compensatory mechanisms (Swelam et al., 2022). CoDA is a statistical technique that analyzes compositional constructs (i.e., variables that are co-dependent), which are part of a

whole (Pawlowsky-Glahn & Egozcue, 2006). In the context of movement-related behaviour research, CoDA allows the analysis and interpretation of various behaviours altogether as they form a composition within a fixed time of 24 h per day (Dumuid et al., 2020; Pedišić, 2014). Specifically, changing the amount of time engaged in one behaviour leads to changes in the allotted time of one or more other behaviours. For instance, an increase in 60 minutes of SB per day must simultaneously reduce PA and/or sleep by 60 minutes in the same day. However, employing CoDA to examine the ActivityStat hypothesis may present some limitations. The major issue has to do with the role of time. Because CoDA is restricted to data occurring in 24-h blocks (Chastin et al., 2015; Pedišić, Dumuid, & Olds, 2017), interpretating findings is tied to a specific time interval. Since there is no consensus thus far regarding when the ActivityStat may occur (e.g., within hours, between days), restricting the interpretation of the findings to a specific period may mask any compensation occurring outside of this interval. Therefore, even though CoDA offers the potential to examine compensation among compositional constructs, it may not be an alternative to test the hypothesis.

Lastly, because compensation may occur through behavioural or energy conservation mechanisms, all constructs that constitute these elements should be assessed when examining the ActivityStat hypothesis. As for the behavioural components, PA levels (i.e., LPA and MVPA) and SB, instead of measuring ST (i.e., motionless regardless of posture), should be examined. Additionally, sleep should be considered when examining the hypothesis. Sleep is a component of the movement behaviour intensity continuum (i.e., sleep, SB, LPA, and MVPA; Tremblay et al., 2010), is associated with health outcomes in youth (Colley et al., 2012; Saunders et al., 2016), and accounts for a large proportion of a 24-h period. Therefore, measurement of posture (e.g., inclinometer) and sleep (e.g., accelerometer) along with the measurement of PA should be

considered in future studies. As for the energy components, total EE, basal or resting EE, and activity EE should be assessed (Gomersall et al., 2013; Pontzer, 2015). This may help to establish a more comprehensive understanding of the hypothesis.

**Challenges experienced**

The greatest challenge I experienced in conducting my dissertation research was the outbreak of COVID-19. My initial plan was to: (1) conduct a scoping review examining evidence for the ActivityStat in individuals living with liver disease; (2) and then to examine the same question in a PA intervention in which we were to investigate whether these individuals experienced activity compensation within- and between-days in the baseline phase; and to (3) compare whether activity compensation occurs during and after the PA program. The scoping review was started in 2019 and only published in 2022. As for the PA intervention that was planned to start in 2019, it was delayed for 24 months because individuals living with liver disease (i.e., with the presence of cirrhosis) were considered high risk if exposed to the virus. Therefore, along with my supervisor and supervisory committee, the decision was made to change the focus of my dissertation to secondary analyses of datasets that included children and adolescents.

The second challenge in conducting my dissertation research had to do with my statistical model. Bayesian CT-SEM is an advanced, complex, and computationally demanding statistical model that requires a high-performance computer. Due to these characteristics, run time for this model can be problematic. Finding a high-performance computer was the first difficult task. To run Bayesian CT-SEM, a computer processor with as many cores as chains (i.e., 4 core) is recommended. For Study 2, the first few models were run on my personal desktop. However, the full model analysis, including the random intercept and slope, required more processing power

than my computer offered. Thus, I sought assistance from Dr. Geri Ruissen who had a computer with sufficient power to run the model. For Study 3, I was able to run all the analysis by myself using remote access to a high-performance computer available at the Digital Scholarship Centre at the University of Alberta. The final challenge was adjusting the analysis runtime with my PhD timeline. Because CT-SEM is very demanding, the runtime took a few days to a few weeks to complete. Also, I had some problems with the computer along the way (e.g., crashing), creating even more delays in the data analysis.

**Conclusion**

There is insufficient evidence of compensation mechanisms within-days and between-days in individuals living with liver disease. Additionally, there is no evidence to support ActivityStat in response to a PA intervention in this population. In children, ActivityStat was not observed. Specifically, the levels of LPA, MVPA, and ST on a given day were found to have positive predictive relationships with the levels of LPA, MVPA, and ST at another time, and these relationships persisted until about 0.5 days later. Though behavioural compensation was not observed for LPA and MVPA in our sample of adolescents, it was demonstrated for ST; thus, supporting the ActivityStat hypothesis. Specifically, previous LPA, MVPA, and ST at one time predicted a positive relationship of themselves at a subsequent time until about 1.7 to 2.5 days hence. Additionally, levels of LPA and ST on a given day negatively predicted ST and LPA at another time, respectively. These relationships persisted until about 0.4 days hence. Lastly, levels of ST among adolescents at a given time predicted an increase in levels of MVPA at another and this relationship persisted until about 0.4 days later. Overall, to continue progressing investigations in the area of the ActivityStat hypothesis, experimental studies are necessary to properly examine the presence of compensatory mechanisms among movement behaviours and

the potential timeframe in which compensation may occur. This knowledge can have practical importance for designing future PA programs.

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## Appendix A: Search Strategy for Scoping Review

MEDLINE

1. exp Exercise/ or Athletes/ or exp Exercise Movement Techniques/ or exp Exercise Therapy/ or exp Sports/ or Motor Activity/ or Physical Exertion/
2. (exercise or physical* activ* or physical* inactiv* or sedentary or running or plyometric* or yoga or tai chi or weight training or resistance training or swim* or sport* or athlet* or walk or walking or mvpa or ltpa or stretching or aerobic capacity or fitness).ti,kf.
3. exercise.ab. /freq=2 or (weight* adj2 lift*).ti,kf. or ((muscle or muscular or strength*) adj2 conditioning).ti,kf
4. 1 or 2 or 3
5. sedentary lifestyle*.mp. or exp Sedentary Behavior/
6. exp Sleep/ or sleep time.mp.
7. exp Posture/ or body posture.mp.
8. motor activity/
9. exp "Activities of Daily Living"/
10. locomotion/
11. (non?exercise physical activ* or nepa).mp.
12. energy metabolism/ or basal metabolism/
13. exp Thermogenesis/ or non-exercise activity thermogenesis.mp.
14. resting metabolic rate.mp.
15. 5 or 6 or 7 or 8 or 9 or 10 or 11 or 12 or 13 or 14
16. liver disease*.mp. or exp Liver Diseases/
17. Exp fatty liver/ or exp liver cirrhosis/
18. Exp Jaundice, neonatal/ or Hepatolenticular Degeneration/
19. Liver diseases, alcoholic/ or exp neoplasms, liver/
20. Exp Cholestasis, Intrahepatic/ or exp liver neoplasms/
21. Exp hepatitis/ or exp bile duct diseases/ or exp Hyperbilirubinemia, Hereditary/
22. Exp Cholangitis/ or Glycogen Storage Disease Type I/ or Adenoma, Liver Cell/ or Focal Nodular Hyperplasia/ or Galactosemias/ or Hemochromatosis/
23. (Steatohepatitis or fatty liver or cirrhosis or hepatitis or intrahepatic Cholestasis or Clinical Features of Lysosomal Acid Lipase Deficiency or liver cyst* or alagille syndrome or Nonalcoholic steatohepatitis or liver disease* or Alpha 1 antitrypsin deficiency).ti,ab.
24. (Primary biliary cholangitis or Primary schlerosing cholangitis or Progressive familial intrahepatic cholestasis or pfic or Glycogen Storage Disease Type I or wilson disease or Crigler najjar syndrome or gilbert disease or gilbert syndrome or Galactosemia*).ti,ab.
25. (Biliary atresia or bile duct disease* or Cholestasis or Cholangitis or Choledochal or Hemochromatosis or Hepatic encephalopathy or Hepatorenal syndrome).ti,ab.
26. (Cyst* adj3 liver).ti,ab. Or ((cancer or neoplasm*) adj3 liver).ti,ab. Or ((neonatal or newborn) adj3 jaundice).ti,ab. Or ((liver tumor*) adj4 benign).ti,ab. Or (bile duct adj3 (cancer* or neoplasm*)).ti,ab.
27. or/16-26
28. 4 and 15 and 27
29. exp animal/ not (exp animal/ and human/)
30. 22 not 23

EMBASE

1. exp Exercise/ or exp Athlete/ or exp kinesiotherapy/
2. (exercise or physical* activ* or physical* inactiv* or sedentary or running or plyometric* or yoga or tai chi or weight training or resistance training or swim* or sport* or athlet* or walk or walking or mvpa or ltpa or stretching or aerobic capacity or fitness).mp.
3. exercise.ab. /freq=2 or (weight* adj2 lift*).ti,ab. or ((muscle or muscular or strength*) adj2 conditioning).mp.
4. 1 or 2 or 3
5. exp sedentary lifestyle/
6. exp Sleep/ or sleep time.mp.
7. exp body position/
8. motor activity/
9. exp daily life activity/
10. locomotion/
11. (non?exercise physical activ* or nepa).mp.
12. exp energy metabolism/ or exp basal metabolic rate/
13. exp Thermogenesis/ or non-exercise activity thermogenesis.mp.
14. exp resting metabolic rate/
15. 5 or 6 or 7 or 8 or 9 or 10 or 11 or 12 or 13 or 14
16. exp liver disease/
17. exp fatty liver/ or exp liver cirrhosis/
18. exp newborn jaundice/
19. exp alcohol liver disease/ or exp liver tumor/
20. exp Cholestasis, Intrahepatic/ or exp liver neoplasms/ or exp Wilson disease/
21. exp hepatitis/ or exp bile duct diseases/ or exp Hyperbilirubinemia/
22. exp Cholangitis/ or exp primary schlerosing cholangitis/ or exp Glycogen Storage Disease Type I/ or exp liver Adenoma/ or exp Nodular Hyperplasia/ or exp Galactosemia/ or exp Hemochromatosis/
23. (Steatohepatitis or fatty liver or cirrhosis or hepatitis or intrahepatic Cholestasis or Clinical Features of Lysosomal Acid Lipase Deficiency or liver cyst* or alagille syndrome or Nonalcoholic steatohepatitis or liver disease* or Alpha 1 antitrypsin deficiency).ti,ab.
24. (Primary biliary cholangitis or Primary schlerosing cholangitis or Progressive familial intrahepatic cholestasis or pfic or Glycogen Storage Disease Type I or wilson disease or Crigler najjar syndrome or gilbert disease or gilbert syndrome or Galactosemia*).ti,ab.
25. (Biliary atresia or bile duct disease* or Cholestasis or Cholangitis or Choledochal orHemochromatosis or Hepatic encephalopathy or Hepatorenal syndrome).ti,ab.
26. ((Cyst* adj3 liver) or ((cancer or neoplasm*) adj3 liver) or ((neonatal or newborn) adj3 jaundice) or (liver tumor* adj4 benign) or (bile duct adj3 (cancer* or neoplasm*))).ti,ab.
27. or/16-26
28. 4 and 15 and 27
29. exp animal/ not (exp animal/ and human/)
30. 28 not 29

CINAHL

See: SPORTdiscus search

1. SU Exercise or SU exercise therapy or SU Sports or SU physical activity or SU physical fitness
2. exercise or physical* activ* or physical* inactiv* or sedentary or running or plyometric* or yoga or tai chi or weight training or resistance training or swim* or sport* or athlet* or walk or walking or mvpa or ltpa or stretching or aerobic capacity or fitness
3. weight* n2 lift* OR ( (muscle or muscular or strength*) n2 conditioning )
4. S1 OR S2 or S3
5. SU sedentary lifestyles or SU sedentary beahvior OR SU sedentary people or SU sleep or SU posture or SU activities of daily living)
6. ( physical* n3 activ* or nepa or thermogenesis or metabolic rate ) OR ( energy metabolism or thermogenesis )
7. S5 or s6
8. ( SU liver diseases+ OR liver disease* ) OR TI ( (Steatohepatitis or fatty liver or cirrhosis or hepatitis or intrahepatic Cholestasis or Clinical Features of Lysosomal Acid Lipase Deficiency or liver cyst* or alagille syndrome or Nonalcoholic steatohepatitis or liver disease* or Alpha 1 antitrypsin deficiency) ) OR AB ( (Steatohepatitis or fatty liver or cirrhosis or hepatitis or intrahepatic Cholestasis or Clinical Features of Lysosomal Acid Lipase Deficiency or liver cyst* or alagille syndrom ...
9. TI ( (Primary biliary cholangitis or Primary schlerosing cholangitis or Progressive familial intrahepatic cholestasis or pfic or Glycogen Storage Disease Type I or wilson disease or Crigler najjar syndrome or gilbert disease or gilbert syndrome or Galactosemia*) ) OR AB ( (Primary biliary cholangitis or Primary schlerosing cholangitis or Progressive familial intrahepatic cholestasis or pfic or Glycogen Storage Disease Type I or wilson disease or Crigler najjar syndrome or gilbert disease or gilb ...
10. TI ( Biliary atresia or bile duct disease* or Cholestasis or Cholangitis or Choledochal or Hemochromatosis or Hepatic encephalopathy or Hepatorenal syndrome ) OR AB ( Biliary atresia or bile duct disease* or Cholestasis or Cholangitis or Choledochal or Hemochromatosis or Hepatic encephalopathy or Hepatorenal syndrome ) OR SU ( Biliary atresia or bile duct disease* or Cholestasis or Cholangitis or Choledochal orHemochromatosis or Hepatic encephalopathy or Hepatorenal syndrome )
11. (SU Liver Diseases)
12. (SU Liver cancer) or (su CIRRHOSIS of the liver)
13. (SU Glycogen Storage Disease)
14. (SU "body temperature regulation") or thermogenesis
15. (su Basal Metabolism)
16. (SU Energy Metabolism)
17. "non-exercise activity thermogenesis"
18. S5 or s6 ...or s16
19. S7 and s4 and 218

PUBMED

Search ((((((((((((((((liver disease*[tw] OR "Liver Diseases"[mesh]))) OR (("fatty liver"[mesh] OR "liver cirrhosis"[mesh]))) OR (("Jaundice, neonatal"[mesh] OR "Hepatolenticular Degeneration"[mesh:noexp]))) OR (("Liver diseases, alcoholic"[mesh:noexp] OR "neoplasms, liver"[mesh]))) OR (("Cholestasis, Intrahepatic"[mesh] OR "liver neoplasms"[mesh]))) OR (("hepatitis"[mesh] OR "bile duct diseases"[mesh] OR "Hyperbilirubinemia, Hereditary"[mesh]))) OR (("Cholangitis"[mesh] OR "Glycogen Storage Disease Type I"[mesh:noexp] OR "Adenoma, Liver Cell"[mesh:noexp] OR "Focal Nodular Hyperplasia"[mesh:noexp] OR "Galactosemias"[mesh:noexp] OR "Hemochromatosis"[mesh:noexp]))) OR ((("Steatohepatitis"[tiab] OR "fatty liver"[tiab] OR "cirrhosis"[tiab] OR "hepatitis"[tiab] OR "intrahepatic Cholestasis"[tiab] OR "Clinical Features of Lysosomal Acid Lipase Deficiency"[tiab] OR liver cyst*[tiab] OR "alagille syndrome"[tiab] OR "Nonalcoholic steatohepatitis"[tiab] OR liver disease*[tiab] OR "Alpha 1 antitrypsin deficiency"[tiab]))) OR ((("Primary biliary cholangitis"[tiab] OR "Primary schlerosing cholangitis"[tiab] OR "Progressive familial intrahepatic cholestasis"[tiab] OR "pfic"[tiab] OR "Glycogen Storage Disease Type I"[tiab] OR "wilson disease"[tiab] OR "Crigler najjar syndrome"[tiab] OR "gilbert disease"[tiab] OR "gilbert syndrome"[tiab] OR Galactosemia*[tiab]))) OR ((("Biliary atresia"[tiab] OR bile duct disease*[tiab] OR "Cholestasis"[tiab] OR "Cholangitis"[tiab] OR "Choledochal or" Hemochromatosis[tiab] OR "Hepatic encephalopathy"[tiab] OR "Hepatorenal syndrome"[tiab]))) OR (((Cyst*[tiab] AND "liver"[tiab]) OR (("cancer"[tiab] OR neoplasm*[tiab]) AND "liver"[tiab]) OR (("neonatal"[tiab] OR "newborn"[tiab]) AND "jaundice"[tiab]) OR ((liver tumor*[tiab]) AND "benign"[tiab]) OR ("bile duct"[tiab] AND (cancer*[tiab] OR neoplasm*[tiab]))))) AND ((((((((((sedentary lifestyle*[tw] OR "Sedentary Behavior"[mesh]))) OR (("Sleep"[mesh] OR "sleep time"[tw]))) OR (("Posture"[mesh] OR "body posture"[tw]))) OR "motor activity"[mesh:noexp]) OR "Activities of Daily Living"[mesh]) OR "locomotion"[mesh:noexp]) OR (((non?exercise physical activ*[tw] OR "nepa"[tw]))) OR (("energy metabolism"[mesh:noexp] OR "basal metabolism"[mesh:noexp]))) OR (("Thermogenesis"[mesh] OR "non-exercise activity thermogenesis"[tw]))) OR "resting metabolic rate"[tw])) AND (((((("Exercise"[mesh] OR "Athletes"[mesh:noexp] OR "Exercise Movement Techniques"[mesh] OR "Exercise Therapy"[mesh] OR "Sports"[mesh] OR "Motor Activity"[mesh:noexp] OR "Physical Exertion"[mesh:noexp]))) OR ((((("exercise"[ti] OR "exercise"[ot]) OR (physical* activ*[ti] OR physical* activ*[ot]) OR (physical* inactiv*[ti] OR physical* inactiv*[ot]) OR ("sedentary"[ti] OR "sedentary"[ot]) OR ("running"[ti] OR "running"[ot]) OR (plyometric*[ti] OR plyometric*[ot]) OR ("yoga"[ti] OR "yoga"[ot]) OR ("tai chi"[ti] OR "tai chi"[ot]) OR ("weight training"[ti] OR "weight training"[ot]) OR ("resistance training"[ti] OR "resistance training"[ot]) OR (swim*[ti] OR swim*[ot]) OR (sport*[ti] OR sport*[ot]) OR (athlet*[ti] OR athlet*[ot]) OR ("walk"[ti] OR "walk"[ot]) OR ("walking"[ti] OR "walking"[ot]) OR ("mvpa"[ti] OR "mvpa"[ot]) OR ("ltpa"[ti] OR "ltpa"[ot]) OR ("stretching"[ti] OR "stretching"[ot]) OR ("aerobic capacity"[ti] OR "aerobic capacity"[ot]) OR ((weightlifting[ti] OR weightlifting[ot])))) OR (((weight-lifting[ti] OR weight-lifting[ot]) OR (weight lifting[ti] OR weight lifting[ot])) OR (((“muscle strengthening”[ti] OR "muscle strengthening"[ot]) OR ("muscular conditioning"[ti] OR "muscular conditioning"[ot]) OR (“strength training”[ti] OR “strength training”[ot])) OR ("strength conditioning"[ti] OR "strength conditioning"[ot]) OR ("muscular conditioning"[ti] OR "muscular conditioning"[ot])))))) NOT ((((animal[Title] OR animals[Title] OR canine*[Title] OR dog[Title] OR dogs[Title] OR feline[Title] OR hamster*[Title] OR lamb[Title] OR

lambs[Title] OR mice[Title] OR monkey[Title] OR monkeys[Title] OR mouse[Title] OR murine[Title] OR pigs[Title] OR pig[Title])) OR (piglet*[Title] OR porcine[Title] OR primate*[Title] OR rabbit*[Title] OR rats[Title] OR rat[Title] OR rodent*[Title] OR sheep*[Title])) NOT (human*[Title] OR patient*[Title]))

SCOPUS
exercise or physical* activ* or physical* inactiv* or sedentary or running or plyometric* or yoga or tai chi or weight training or resistance training or swim* or sport* or athlet* or walk or walking or mvpa or ltpa or stretching or aerobic capacity or fitness or weightlift*

and

physical* activ* or nepa or thermogenesis or metabol*

and

Biliary atresia or bile duct disease* or Cholestasis or Cholangitis or Choledochal or Hemochromatosis or Hepatic encephalopathy or Hepatorenal syndrome or Primary biliary cholangitis or Primary schlerosing cholangitis or Progressive familial intrahepatic cholestasis or pfic or Glycogen Storage Disease Type I or wilson disease or Crigler najjar syndrome or gilbert disease or gilbert syndrome or Galactosemia* or Steatohepatitis or fatty liver or cirrhosis or hepatitis or intrahepatic Cholestasis or Clinical Features of Lysosomal Acid Lipase Deficiency or liver cyst* or alagille syndrome or Nonalcoholic steatohepatitis or liver disease* or Alpha 1 antitrypsin deficiency or liver cancer* or liver tumor or neonatal jaundice or bile duct cancer*

Web of Science
exercise or physical* activ* or physical* inactiv* or sedentary or running or plyometric* or yoga or tai chi or weight training or resistance training or swim* or sport* or athlet* or walk or walking or mvpa or ltpa or stretching or aerobic capacity or fitness or weightlift*

and

physical* activ* or nepa or thermogenesis or metabol*

and

Biliary atresia or bile duct disease* or Cholestasis or Cholangitis or Choledochal or Hemochromatosis or Hepatic encephalopathy or Hepatorenal syndrome or Primary biliary cholangitis or Primary schlerosing cholangitis or Progressive familial intrahepatic cholestasis or pfic or Glycogen Storage Disease Type I or wilson disease or Crigler najjar syndrome or gilbert disease or gilbert syndrome or Galactosemia* or Steatohepatitis or fatty liver or cirrhosis or hepatitis or intrahepatic Cholestasis or Clinical Features of Lysosomal Acid Lipase Deficiency or liver cyst* or alagille syndrome or Nonalcoholic steatohepatitis or liver disease* or Alpha 1 antitrypsin deficiency or liver cancer* or liver tumor or neonatal jaundice or bile duct cancer*

Cochrane

1. exp Exercise/ or Athletes/ or exp Exercise Movement Techniques/ or exp Exercise Therapy/ or exp Sports/ or Motor Activity/ or Physical Exertion/
2. (exercise or physical* activ* or physical* inactiv* or sedentary or running or plyometric* or yoga or tai chi or weight training or resistance training or swim* or sport* or athlet* or walk or walking or mvpa or ltpa or stretching or aerobic capacity or fitness).ti,kf.
3. exercise.ab. /freq=2 or (weight* adj2 lift*).ti,kf. or ((muscle or muscular or strength*) adj2 conditioning).ti,kf
4. 1 or 2 or 3
5. sedentary lifestyle*.mp. or exp Sedentary Behavior/
6. exp Sleep/ or sleep time.mp.
7. exp Posture/ or body posture.mp.
8. motor activity/
9. exp "Activities of Daily Living"/
10. locomotion/
11. (non?exercise physical activ* or nepa).mp.
12. energy metabolism/ or basal metabolism/
13. exp Thermogenesis/ or non-exercise activity thermogenesis.mp.
14. resting metabolic rate.mp.
15. 5 or 6 or 7 or 8 or 9 or 10 or 11 or 12 or 13 or 14
16. liver disease*.mp. or exp Liver Diseases/
17. Exp fatty liver/ or exp liver cirrhosis/
18. Exp Jaundice, neonatal/ or Hepatolenticular Degeneration/
19. Liver diseases, alcoholic/ or exp neoplasms, liver/
20. Exp Cholestasis, Intrahepatic/ or exp liver neoplasms/
21. Exp hepatitis/ or exp bile duct diseases/ or exp Hyperbilirubinemia, Hereditary/
22. Exp Cholangitis/ or Glycogen Storage Disease Type I/ or Adenoma, Liver Cell/ or Focal Nodular Hyperplasia/ or Galactosemias/ or Hemochromatosis/
23. (Steatohepatitis or fatty liver or cirrhosis or hepatitis or intrahepatic Cholestasis or Clinical Features of Lysosomal Acid Lipase Deficiency or liver cyst* or alagille syndrome or Nonalcoholic steatohepatitis or liver disease* or Alpha 1 antitrypsin deficiency).ti,ab.
24. (Primary biliary cholangitis or Primary schlerosing cholangitis or Progressive familial intrahepatic cholestasis or pfic or Glycogen Storage Disease Type I or wilson disease or Crigler najjar syndrome or gilbert disease or gilbert syndrome or Galactosemia*).ti,ab.
25. (Biliary atresia or bile duct disease* or Cholestasis or Cholangitis or Choledochal or Hemochromatosis or Hepatic encephalopathy or Hepatorenal syndrome).ti,ab.
26. (Cyst* adj3 liver).ti,ab. Or ((cancer or neoplasm*) adj3 liver).ti,ab. Or ((neonatal or newborn) adj3 jaundice).ti,ab. Or ((liver tumor*) adj4 benign).ti,ab. Or (bile duct adj3 (cancer* or neoplasm*)).ti,ab.
27. or/16-26
28. 4 and 15 and 27
29. exp animal/ not (exp animal/ and human/)
30. 22 not 23

Grey Literature Database

exercise or physical* activ* or physical* inactiv* or sedentary or running or plyometric* or yoga or tai chi or weight training or resistance training or swim* or sport* or athlet* or walk or walking or mvpa or ltpa or stretching or aerobic capacity or fitness or weightlift*

and

physical* activ* or nepa or thermogenesis or metabol*

and

Biliary atresia or bile duct disease* or Cholestasis or Cholangitis or Choledochal or Hemochromatosis or Hepatic encephalopathy or Hepatorenal syndrome or Primary biliary cholangitis or Primary schlerosing cholangitis or Progressive familial intrahepatic cholestasis or pfic or Glycogen Storage Disease Type I or wilson disease or Crigler najjar syndrome or gilbert disease or gilbert syndrome or Galactosemia* or Steatohepatitis or fatty liver or cirrhosis or hepatitis or intrahepatic Cholestasis or Clinical Features of Lysosomal Acid Lipase Deficiency or liver cyst* or alagille syndrome or Nonalcoholic steatohepatitis or liver disease* or Alpha 1 antitrypsin deficiency or liver cancer* or liver tumor or neonatal jaundice or bile duct cancer*

# Appendix B: Model Lattex for Study 2 and 3

Subject parameter distribution:

$$\underbrace{\begin{bmatrix} \text{b\_lpa}_i \\ \text{b\_mvpa}_i \\ \text{b\_st}_i \end{bmatrix}}_{\boldsymbol{\phi}(i)} \sim \text{tform}\left\{ \text{N}\left( \begin{bmatrix} \text{raw\_b\_lpa} \\ \text{raw\_b\_mvpa} \\ \text{raw\_b\_st} \end{bmatrix}, \begin{bmatrix} \text{rawPCov\_1\_1} & \text{rawPCov\_2\_1} & \text{rawPCov\_3\_1} \\ \text{rawPCov\_2\_1} & \text{rawPCov\_2\_2} & \text{rawPCov\_3\_2} \\ \text{rawPCov\_3\_1} & \text{rawPCov\_3\_2} & \text{rawPCov\_3\_3} \end{bmatrix} \right) \right\}$$

Initial latent state:

$$\underbrace{\begin{bmatrix} \text{lpa} \\ \text{mvpa} \\ \text{st} \end{bmatrix}}_{\boldsymbol{\eta}(t_0)} (t_0) \sim \text{N}\left( \underbrace{\begin{bmatrix} \text{T0m\_lpa} \\ \text{T0m\_mvpa} \\ \text{T0m\_st} \end{bmatrix}}_{\text{T0MEANS}}, \underbrace{\textit{covsdcor}\left\{ \begin{bmatrix} \text{T0var\_lpa} & 0 & 0 \\ \text{T0var\_mvpa\_lpa} & \text{T0var\_mvpa} & 0 \\ \text{T0var\_st\_lpa} & \text{T0var\_st\_mvpa} & \text{T0var\_st} \end{bmatrix} \right\}}_{\underbrace{\mathbf{Q}^*_{t0}}_{\text{T0VAR}}} \right)$$

Deterministic change:

$$\underbrace{\text{d}\begin{bmatrix} \text{lpa} \\ \text{mvpa} \\ \text{st} \end{bmatrix}(t)}_{\text{d}\boldsymbol{\eta}(t)} = \left( \underbrace{\begin{bmatrix} \text{a\_lpa} & \text{a\_lpamvpa} & \text{a\_lpast} \\ \text{a\_mvpalpa} & \text{a\_mvpa} & \text{a\_mvpast} \\ \text{a\_stlpa} & \text{a\_stmvpa} & \text{a\_st} \end{bmatrix}}_{\underbrace{\mathbf{A}}_{\text{DRIFT}}} \underbrace{\begin{bmatrix} \text{lpa} \\ \text{mvpa} \\ \text{st} \end{bmatrix}(t)}_{\boldsymbol{\eta}(t)} + \underbrace{\begin{bmatrix} \text{b\_lpa} \\ \text{b\_mvpa} \\ \text{b\_st} \end{bmatrix}}_{\underbrace{\mathbf{b}}_{\text{CINT}}} \right) \text{d}t \; +$$

Random change:

$$\underbrace{\textit{cholsdcor}\left\{ \begin{bmatrix} \text{sd\_lpa} & 0 & 0 \\ \text{cor\_mvpalpa} & \text{sd\_mvpa} & 0 \\ \text{cor\_stlpa} & \text{cor\_stmvpa} & \text{sd\_st} \end{bmatrix} \right\}}_{\underbrace{\mathbf{G}}_{\text{DIFFUSION}}} \underbrace{\text{d}\begin{bmatrix} W_1 \\ W_2 \\ W_3 \end{bmatrix}(t)}_{\text{d}\mathbf{W}(t)}$$

Observations:

$$\underbrace{\begin{bmatrix} \text{lpa\_cs} \\ \text{mvpa\_cs} \\ \text{st\_cs} \end{bmatrix}(t)}_{\mathbf{Y}(t)} = \underbrace{\begin{bmatrix} 1 & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 1 \end{bmatrix}}_{\underbrace{\boldsymbol{\Lambda}}_{\text{LAMBDA}}} \underbrace{\begin{bmatrix} \text{lpa} \\ \text{mvpa} \\ \text{st} \end{bmatrix}(t)}_{\boldsymbol{\eta}(t)} + \underbrace{\begin{bmatrix} 0 \\ 0 \\ 0 \end{bmatrix}}_{\underbrace{\boldsymbol{\tau}}_{\text{MANIFESTMEANS}}} + \underbrace{\begin{bmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}}_{\underbrace{\boldsymbol{\Theta}}_{\text{MANIFESTVAR}}} \underbrace{\begin{bmatrix} \epsilon_1 \\ \epsilon_2 \\ \epsilon_3 \end{bmatrix}(t)}_{\boldsymbol{\epsilon}(t)}$$

Latent noise per time step: $\Delta\left[W_{j\in[1,3]}\right](t-u) \sim \text{N}(0, t-u)$

Observation noise: $\left[\epsilon_{j\in[1,3]}\right](t) \sim \text{N}(0,1)$

*cholsdcor* converts lower tri matrix of std dev and unconstrained correlation to Cholesky factor covariance.
*covsdcor* = transposed cross product of cholsdcor, to give covariance.
See Driver & Voelkle (2018) p11.

## Appendix C: Code for Study 2 and 3

```
## Actistat Code

# load/install packages
library(magrittr)
library(psych)
library(ctsem)
library(rstan)
library(tidyverse)
library(ggplot2)

# Set working directory to source file location
# Click "Session" -> "Set Working Directory" -> "To Source File Location"

# import SPSS file
library(haven)
daytoday_data <- read_sav("~/Carminda/Project1/daytoday_data.sav")
View(daytoday_data)

# Step 1: set up data

# creating subsetting to variable of interest
daytoday_datasub <- daytoday_data [ ,c("id","time","lpa_min","mvpa_min","st_min")]

# per Driver et al recommendations, grand mean-center and scale variables
csdaytoday_datasub <- daytoday_datasub %>%
mutate(lpa_cs = scale(daytoday_datasub$lpa_min, center = TRUE, scale = TRUE))%>%
mutate(mvpa_cs = scale(daytoday_datasub$mvpa_min, center = TRUE, scale =
TRUE))%>%
mutate(st_cs = scale(daytoday_datasub$st_min, center = TRUE, scale = TRUE))

csdaytoday_datasub <- csdaytoday_datasub [ ,c("id","time","lpa_cs","mvpa_cs","st_cs")]

# descriptive statistics for within-person means
sumlpa_min <- daytoday_datasub %>% group_by(id) %>%
summarise(sum = sum(lpa_min, na.rm = TRUE)/2)

summvpa_min <- daytoday_datasub %>% group_by(id) %>%
summarise(sum = sum(mvpa_min, na.rm = TRUE)/2)

sumst_min <- daytoday_datasub %>% group_by(id) %>%
summarise(sum = sum(st_min, na.rm = TRUE)/2)

describe(sumlpa_min$sum)
multi.hist(sumlpa_min$sum)
```

```
describe(summvpa_min$sum)
multi.hist(summvpa_min$sum)

describe(sumst_min$sum)
multi.hist(sumst_min$sum)

# Step 2: set up the model

# abreviations:
# lpa ... light physical activity
# mvpa ... moderate-vigorous physical activity
# st ... stationary time
# lpamvpa ... effect of lpa on mvpa
# lpast ... effect of lpa on st
# mvpalpa ... effect of mvpa on lpa
# mvpast ... effect of mvpa on st
# stlpa ... effect of st on lpa
# stmvpa ... effect of st on mvpa

# drift matrix 3x3 with endogenous variables (lpa, mvpa, and st)
A <- matrix( c("a_lpa","a_mvpalpa","a_stlpa",
            "a_lpamvpa","a_mvpa","a_stmvpa",
            "a_lpast","a_mvpast","a_st"), nrow = 3, ncol = 3)

# diffusion covariancE matrix
Q.sdcor <- matrix( c("sd_lpa","cor_mvpalpa","cor_stlpa",0,"sd_mvpa","cor_stmvpa",
            0, 0, "sd_st"), nrow = 3, ncol = 3)

# continuous-time intercepts
b <- matrix( c("b_lpa","b_mvpa","b_st"), nrow = 3, ncol = 1)

# set all intercepts of manifest variables to 0, because estimating cint
manifestmeans <- matrix( 0, nrow = 3, ncol = 1)

# loading matrix
Lambda <- diag(3)

# set up model
actimod <- ctModel( n.latent = 3,
            latentNames = c("lpa", "mvpa", "st"),
            n.manifest = 3,
            manifestNames = c("lpa_cs","mvpa_cs","st_cs"),
            DRIFT = A,
            DIFFUSION = Q.sdcor,
            CINT = b,
```

```
                LAMBDA = Lambda,
                MANIFESTMEANS = manifestmeans,
                MANIFESTVAR = matrix( 0, nrow = 3, ncol = 3),
                type = "stanct" )


        # set parameters that vary over persons
        # set all parameters non-varying
        actimod$pars$indvarying <- FALSE

        # set continuous-time intercept varying
        actimod$pars$indvarying[ actimod$pars$matrix %in% c('CINT') ] <- TRUE

        # view model Latex
        ctModelLatex(actimod, compile = TRUE)

        # Step 3: run the model

        # start time
        start2 <- Sys.time()

        # run the continuous-time model (Bayesian estimation)
        options(mc.cores = parallel::detectCores())
        rstan_options(auto_write = TRUE)


        actifit <- ctStanFit (datalong = csdaytoday_datasub,
                ctstanmodel = actimod,
                iter = 20000,
                optimize = FALSE,
                intoverstates = TRUE,
                algorithm = "NUTS",
                nopriors = FALSE,
                chains = 4,
                cores = 4,
                control = list(max_treedepth=12),
                fit = TRUE,
                plot = TRUE)

        # run time
        print(runtime2 <- Sys.time() - start2)

        # view results
        actisum <- summary(actifit, digits = 2, parmatrices = TRUE) #renamed to clarify
summary object
```

```
#plot(actifit, types = c('intervals', 'regression'), wait = FALSE) # commented out for custom plots below.

# summary of the findings
print(actisum[c("popsd","popmeans","rawpopcorr","popNote")] )

## Save fit object for later - to avoid re-running model

saveRDS(actifit, "actistat.rds")

#Custom plot options

require(ggplot2) #overrides ctsem setting for plots

#Plot Auto-effects
plotauto = ctStanDiscretePars(actifit, times = seq(from = 0, to = 5, by = 0.05), observational=FALSE,
                              nsamples = 500, plot=TRUE,indices='AR', facets = 'Effect') #AR plots auto effects

plotauto$layers[[2]]$aes_params <- NULL #removes the useless linetype setting on the central line
plotauto + aes(linetype=Effect, size=Effect) +
  labs (x= "Time Interval (Days)", title = NULL) +
  theme(text=element_text(family="serif", size = 14)) +
  scale_linetype_manual(name = "Effect", values = c("solid", "dashed","longdash")) +
  scale_size_manual(name = "Effect", values = c(0.8,0.8,0.8))

ggsave( #creates a separate file in your directory folder that is a high quality image
  "ctsemauto.png",
  width = 11,
  height = 8.5,
  units = "in",
  dpi = "retina"
)

#Plot cross-effects
plotcross = ctStanDiscretePars(actifit, times = seq(from = 0, to = 5, by = 0.05), observational=FALSE,
                               nsamples = 500, plot=TRUE,indices='CR', facets = 'Effect') # CR plots cross-effects

plotcross$layers[[2]]$aes_params <- NULL #removes the useless linetype setting on the central line
plotcross + aes(linetype=Effect, size=Effect) +
  labs (x= "Time Interval (Days)", title = NULL) +
```

```
  theme(text=element_text(family="serif", size = 14)) +
  scale_linetype_manual(name = "Effect", values = c("solid",
"dashed","longdash","dotted", "dotdash", "twodash")) +
  scale_size_manual(name = "Effect", values = c(0.8,0.8,0.8,0.8,0.8,0.8))

ggsave( #creates a separate file in your directory folder that is a high quality image
  "ctsemcross.png",
  width = 11,
  height = 8.5,
  units = "in",
  dpi = "retina"
)
```

## Appendix D: Fully Random Effect Model Output for Study 2

```
$popsd
         mean   sd 2.5%  50% 97.5%   n_eff Rhat
TOm_lpa  0.71 0.11 0.52 0.70  0.96 1535.60 1.01
TOm_mvpa 0.99 0.10 0.79 1.00  1.19 1445.07 1.00
TOm_stat 0.82 0.12 0.61 0.82  1.04  499.40 1.01
b_lpa    2.24 1.37 0.60 1.86  5.82 4642.83 1.00
b_mvpa   3.46 1.44 1.36 3.21  6.68  274.41 1.02
b_stat   2.56 1.47 0.83 2.12  6.30 6623.48 1.00

$popmeans
              mean   sd   2.5%   50% 97.5%    n_eff Rhat
TOm_lpa       0.07 0.10  -0.12  0.07  0.26 18355.79 1.00
TOm_mvpa      0.16 0.11  -0.05  0.15  0.36 10984.83 1.00
TOm_stat     -0.17 0.10  -0.36 -0.17  0.02 12591.30 1.00
a_lpa        -4.31 2.27 -10.03 -3.85 -1.35  7335.34 1.00
a_lpamvpa     0.58 0.92  -1.25  0.59  2.43  4832.68 1.00
a_lpastat    -0.76 0.85  -2.43 -0.81  0.99  5425.07 1.00
a_mvpalpa     0.17 0.92  -1.65  0.15  1.98 12243.46 1.00
a_mvpa       -5.68 2.20 -10.65 -5.46 -2.16  8277.05 1.00
a_mvpastat   -0.23 0.98  -2.14 -0.23  1.64   198.43 1.02
a_statlpa    -0.35 0.88  -2.14 -0.33  1.30   810.31 1.01
a_statmvpa   -0.64 0.91  -2.41 -0.65  1.15   777.59 1.01
a_stat       -4.57 2.27 -10.13 -3.95 -1.64  7354.69 1.00
sd_lpa        1.14 0.54   0.22  1.08  2.35  1997.89 1.01
cor_mvpalpa   0.34 0.21  -0.03  0.32  0.77  3369.35 1.00
sd_mvpa       2.19 0.56   1.21  2.15  3.31   633.99 1.01
cor_statlpa  -0.19 0.26  -0.72 -0.17  0.30  1590.83 1.00
cor_statmvpa -0.55 0.17  -0.86 -0.55 -0.25   303.90 1.02
sd_stat       1.61 0.55   0.75  1.54  2.94  2194.81 1.00
mvarlpa_cs    0.63 0.11   0.30  0.66  0.76  1047.47 1.00
mvarmvpa_cs   0.32 0.16   0.05  0.33  0.59   280.83 1.03
mvarstat_cs   0.45 0.17   0.03  0.51  0.65    77.88 1.06
b_lpa        -0.04 0.32  -0.77 -0.02  0.58 15495.10 1.00
b_mvpa       -0.22 0.43  -1.19 -0.20  0.58 16297.07 1.00
b_stat        0.13 0.35  -0.52  0.11  0.96 16395.74 1.00

$rawpopcorr
                    mean   sd  2.5%   50% 97.5% n_eff Rhat     z
TOm_mvpa__TOm_lpa   0.57 0.11  0.32  0.58  0.77 80281    1  5.05
TOm_stat__TOm_lpa  -0.58 0.14 -0.83 -0.59 -0.30 79438    1 -4.32
b_lpa__TOm_lpa      0.65 0.20  0.15  0.70  0.91 73026    1  3.29
```

```
b_mvpa__TOm_lpa     0.47 0.19  0.04  0.49  0.78 79966    1  2.44
b_stat__TOm_lpa    -0.58 0.20 -0.85 -0.62 -0.06 73832    1 -2.84
TOm_stat__TOm_mvpa -0.64 0.09 -0.81 -0.64 -0.45  3521    1 -6.81
b_lpa__TOm_mvpa     0.04 0.25 -0.47  0.04  0.50 80440    1  0.16
b_mvpa__TOm_mvpa    0.67 0.09  0.47  0.67  0.83 76912    1  7.35
b_stat__TOm_mvpa   -0.34 0.19 -0.66 -0.36  0.12 79815    1 -1.77
b_lpa__TOm_stat    -0.21 0.23 -0.61 -0.23  0.32 79475    1 -0.90
b_mvpa__TOm_stat   -0.31 0.16 -0.60 -0.32  0.05 77880    1 -1.86
b_stat__TOm_stat    0.63 0.13  0.35  0.64  0.84 73584    1  4.98
b_mvpa__b_lpa       0.10 0.33 -0.57  0.13  0.68 76352    1  0.29
b_stat__b_lpa      -0.32 0.32 -0.80 -0.38  0.40 38543    1 -1.01
b_stat__b_mvpa     -0.41 0.26 -0.78 -0.47  0.25 79511    1 -1.54

$popNote
[1] "popmeans are reported as specified in ctModel -- covariance related
matrices are in sd / unconstrained correlation form -- see $parmatrices for
simpler interpretations!"
```

## Appendix E: Fully Random Effect Model Output for Study 3

$popsd

| | mean | sd | 2.5% | 50% | 97.5% | n_eff | Rhat |
|---|---|---|---|---|---|---|---|
| TOm_lpa | 0.98 | 0.07 | 0.83 | 0.98 | 1.11 | 211.26 | 1.02 |
| TOm_mvpa | 0.92 | 0.08 | 0.75 | 0.93 | 1.05 | 342.77 | 1.01 |
| TOm_stat | 0.63 | 0.07 | 0.51 | 0.63 | 0.78 | 2321.28 | 1.00 |
| b_lpa | 2.01 | 0.76 | 1.08 | 1.82 | 4.02 | 6032.81 | 1.00 |
| b_mvpa | 1.34 | 0.32 | 0.83 | 1.31 | 2.06 | 1481.58 | 1.00 |
| b_stat | 0.86 | 0.32 | 0.44 | 0.79 | 1.65 | 5159.18 | 1.00 |

$popmeans

| | mean | sd | 2.5% | 50% | 97.5% | n_eff | Rhat |
|---|---|---|---|---|---|---|---|
| TOm_lpa | 0.04 | 0.06 | -0.08 | 0.04 | 0.15 | 10577.00 | 1.00 |
| TOm_mvpa | -0.01 | 0.06 | -0.12 | -0.01 | 0.10 | 9511.97 | 1.00 |
| TOm_stat | -0.11 | 0.05 | -0.21 | -0.11 | -0.01 | 12978.92 | 1.00 |
| a_lpa | -3.33 | 1.25 | -6.58 | -3.04 | -1.72 | 5272.07 | 1.00 |
| a_lpamvpa | -0.07 | 0.49 | -0.99 | -0.10 | 1.01 | 3456.10 | 1.00 |
| a_lpastat | -1.16 | 0.64 | -2.55 | -1.10 | -0.08 | 3966.49 | 1.00 |
| a_mvpalpa | -0.38 | 0.53 | -1.36 | -0.40 | 0.79 | 6439.82 | 1.00 |
| a_mvpa | -2.20 | 0.56 | -3.38 | -2.16 | -1.25 | 1157.89 | 1.00 |
| a_mvpastat | -0.18 | 0.49 | -1.15 | -0.18 | 0.79 | 3418.24 | 1.00 |
| a_statlpa | -0.53 | 0.53 | -1.80 | -0.45 | 0.31 | 3730.79 | 1.00 |
| a_statmvpa | 0.62 | 0.26 | 0.22 | 0.58 | 1.23 | 3607.94 | 1.00 |
| a_stat | -1.22 | 0.63 | -2.77 | -1.11 | -0.33 | 3278.08 | 1.00 |
| sd_lpa | 1.45 | 0.33 | 0.86 | 1.45 | 2.16 | 222.47 | 1.02 |
| cor_mvpalpa | 0.21 | 0.09 | 0.03 | 0.21 | 0.39 | 738.61 | 1.01 |
| sd_mvpa | 1.58 | 0.29 | 0.97 | 1.62 | 2.06 | 282.52 | 1.01 |
| cor_statlpa | -0.26 | 0.19 | -0.70 | -0.23 | 0.03 | 925.60 | 1.00 |
| cor_statmvpa | -0.47 | 0.15 | -0.79 | -0.46 | -0.21 | 2241.46 | 1.00 |
| sd_stat | 0.70 | 0.18 | 0.46 | 0.67 | 1.18 | 1851.35 | 1.01 |
| mvarlpa_cs | 0.35 | 0.15 | 0.04 | 0.37 | 0.58 | 83.54 | 1.04 |
| mvarmvpa_cs | 0.33 | 0.17 | 0.05 | 0.33 | 0.63 | 160.20 | 1.02 |
| mvarstat_cs | 0.66 | 0.05 | 0.53 | 0.67 | 0.72 | 1278.70 | 1.01 |
| b_lpa | -0.03 | 0.13 | -0.30 | -0.03 | 0.22 | 16016.16 | 1.00 |

```
b_mvpa        0.01 0.09 -0.17  0.01  0.18 20965.00 1.00
b_stat        0.01 0.06 -0.12  0.01  0.11 20580.84 1.00


$rawpopcorr
                     mean   sd  2.5%   50% 97.5% n_eff Rhat      z
TOm_mvpa__TOm_lpa    0.36 0.07  0.24  0.36  0.51 21012 1.01   5.24
TOm_stat__TOm_lpa   -0.81 0.06 -0.91 -0.82 -0.67 63694 1.00 -13.47
b_lpa__TOm_lpa       0.69 0.07  0.54  0.69  0.82  3557 1.01   9.63
b_mvpa__TOm_lpa      0.39 0.15  0.06  0.40  0.64 80076 1.00   2.65
b_stat__TOm_lpa     -0.27 0.21 -0.65 -0.28  0.17 75194 1.00  -1.28
TOm_stat__TOm_mvpa -0.53 0.08 -0.69 -0.53 -0.37 79411 1.00  -6.52
b_lpa__TOm_mvpa      0.16 0.10 -0.04  0.16  0.34 79222 1.00   1.60
b_mvpa__TOm_mvpa     0.50 0.07  0.36  0.50  0.65 75809 1.00   6.99
b_stat__TOm_mvpa    -0.32 0.12 -0.55 -0.32 -0.08 76462 1.00  -2.70
b_lpa__TOm_stat     -0.41 0.12 -0.63 -0.42 -0.17 79335 1.00  -3.53
b_mvpa__TOm_stat    -0.41 0.13 -0.64 -0.42 -0.14 78631 1.00  -3.21
b_stat__TOm_stat     0.52 0.15  0.20  0.54  0.77 64805 1.00   3.56
b_mvpa__b_lpa        0.42 0.22 -0.12  0.46  0.76 80202 1.00   1.89
b_stat__b_lpa       -0.01 0.31 -0.62  0.00  0.54 67842 1.00  -0.05
b_stat__b_mvpa      -0.57 0.20 -0.88 -0.60 -0.09 79686 1.00  -2.85


$popNote
[1] "popmeans are reported as specified in ctModel -- covariance related
matrices are in sd / unconstrained correlation form -- see $parmatrices for
simpler interpretations!"
```