NEUROBEHAVIOURAL PREDICTORS OF MOBILITY DURING DISTRACTED WALKING IN HEALTHY ADULTS

L. MAUREEN KRELOVE

A THESIS SUBMITTED TO THE FACULTY OF GRADUATE STUDIES IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF

'MASTER OF SCIENCE'

GRADUATE PROGRAM IN KINESIOLOGY & HEALTH SCIENCE

YORK UNIVERSITY

TORONTO, ONTARIO

MAY 2026

Abstract

Brain activity and balance strategies change throughout adulthood such that older adults have increased fall risk and rely on executive resources to maintain balance. Falls have significant negative outcomes for older adults, however, fall risk begins to increase during mid-life. The specific timing of the age-related change in neurobehavioural markers of mobility have not been established. This work aimed to determine the age at which brain activity and gait speed during complex walking deviated from young-adult patterns and identify sociodemographic factors that influenced this relationship. Piecewise linear regression on data points from 113 participants indicated that neurobehavioural outcomes became more similar to older adults by the mid-Fifties. Two-step cluster analysis showed that age and education predicted neurobehavioural mobility outcomes. Mediation analyses revealed that brain activity compensated for age-related mobility decline. Together, these findings suggest that neurobehavioural changes in mobility begin in mid-life and that sociodemographic factors can predict mobility outcomes.

Dedication

This thesis is dedicated to everyone who participated in this study, many of whom were taking part in research for the first time. Thank you for your trust and contribution, I aim for these findings to one day make their way back to you.

Acknowledgements

This work has taken many twists, turns, and forms that I would not have known how to navigate without the continued guidance of the best mentor I could have asked for. Dr. Mochizuki has shaped me into the researcher I am as I complete this work. Thank you for never suggesting that any idea was too big, too challenging, or too ambitious, and instead teaching me how to channel my ideas into practical, meaningful possibilities. Your mentorship has profoundly shaped this work and has built the foundation of my development as a scientist. Through the countless hours spent in the lab discussing results and interpretations and all of the conferences, talks, opportunities, thank you for encouraging my curiosity.

I would like to thank my supervisory committee, Dr. Lauren Sergio and Dr. Will Gage, for their thoughtful feedback throughout this project. I am very appreciative for the support of my labmates – especially Norman, Marcel, and Kailey – for their assistance in data collections and around the lab. I am grateful to have gone through this process alongside my dearest friend, Liza, whose encouragement and perspective provided balance throughout our mutual Master's experience. And, to my little sister, who is in every one of my talks, papers, and posters (see Figure 2.1), and is always the first person to listen to the pilot run of every presentation.

The scope of this project would not have been possible without the community partners who went out of their way to make this scientific vision into reality. I am most appreciative towards the fantastic staff at Reconnect Health Services – Frank and Colleen - and the YMCA of Greater Toronto – Beth and Jessica (Braley and Markham centres). And, most of all, to my participants, especially those who had never been part of a study before. Their trust and openness made this work possible. From now until the end of my career, I aim for my research results to have the capacity to be meaningful for those who were part of it.

Table of Contents

List of Tables

List of Figures

List of Abbreviations

ABC	Activities Specific Balance-Confidence Scale
ADLs	Activties of Daily Living
AR	Augmented Reality
AICc	Small Sample Corrected Akaike Information Criterion
BIC	Bayesian Information Criterion
CC 1	Community Centre 1
CC 2	Community Centre 2
CRUNCH	Compensation-Realted Utilization of Neural Circuits Hypothesis
CV	Cross-Validation
DE	Direct Effect
dlPFC	Dorsolateral Prefrontal Cortex
DT	Dual-Task
EDF	Estimated Degrees of Freedom
EF	Executive Function
fNIRS	Functional Near-Infrared Spectroscopy
GAM	Generalized Additive Model
HAROLD	Hemispheric Asymmetry Reduction in Older Adults
HHb	Deoxygenated Hemoglobin
IE	Indirect Effect
LOWESS	Locally Weighted Scatterplot Smoothing
MA	Middle-Aged Adults (Age 35-64)
MSE	Mean Squared Error

M/S	Metres-per-second
O_2Hb	Oxygenated Hemoglobin
OA	Older Adults (Age 65+)
PASA	Posterior to Anterior Shift in Aging
PCA	Principle Component Analysis
PFC	Prefrontal Cortex
PM	Proportion Mediated
SES	Socioeconomic Status
ST	Single Task
STAC	Scaffolding Theory of Aging and Cognition
STAC-r	Revised Scaffolding Theory of Aging and Cognition
TE	Total Effect
TSC	Two-Step Cluster Analysis
W	study condition “walk”
WD	study condition “walk + distraction”
WO	study condition “walk + obstacles”
WOD	study condition “walk + obstacles + distraction”
YA	Young Adults (Age 18-34)
µM	Micromolar

List of Equations

2-1 Change-point Regression:

$$hinge(age; \tau) = max(0, age - \tau)$$

2-2 Hinge Model:

$$y = \beta_0 + \beta_1 \cdot age + \beta_2 \cdot hinge(age; \tau) + \varepsilon$$

2-3 Hinge Model Before change-point:

$$y = \beta_0 + \beta_1 \cdot age + \varepsilon$$

2-4 Hinge Model After Change-Point:

$$y = \beta_0 + \beta_0 \cdot age + \beta_2(age - \tau) + \varepsilon$$

3-1 Mediator Model:

$$M = \alpha_0 + \alpha_1 X + \varepsilon_M$$

3-2 Outcome Model:

$$Y = \beta_0 + \beta_1 X + f(M) + \varepsilon_Y$$

3-3 Total Effect:

$$E[Y(X_1, M(X_1)) - Y(X_0, M(X_0))]$$

3-4 Natural Direct Effect

$$E\left[Y\left(X_1, M(X_1)\right) - Y\left(X_0, M(X_0)\right)\right]$$

3-5 Natural Indirect Effect

$$E[Y(X_1, M(X_1)) - Y(X_1, M(X_0))]$$

B-1 Akaike Information Criterion (AICc)

$$AICc = AIC + \frac{2k(k+1)}{n-k-1}$$

E-1 Generalized Additive Model

$$Y = \beta_0 + \beta_1 X + f(M) + \varepsilon_Y$$

Preface

This thesis explores neurobehavioural markers of mobility during complex walking across adulthood with a focus on prefrontal cortex activity and gait performance. Through integration of data-driven nonlinear modelling, mediation analyses, and sociodemographic investigations the present work aimed to identify early indicators of mobility decline from a neurobehavioural perspective. This preface outlines the relationship between each chapter and provides a roadmap for each component's contribution to the overall objectives of this thesis.

This work is structured as a manuscript-based thesis consisting of multiple interrelated chapters. Each Data Chapter is written as an independent manuscript addressing a distinct objective and research question while contributing to the overarching aim of identifying neurobehavioural markers of mobility across adulthood. The work builds sequentially with findings from earlier chapters informing subsequent analyses and all chapters draw from the same dataset and conceptual framework. Participants, variables, and analytical approaches are intentionally interconnected to build a cohesive understanding of neurobehavioural correlates of mobility capacity. The thesis is structured as follows:

Chapter 1 – General Introduction

Chapter 1 provides an overview of dual-task walking and brain activity focussed on mobility decline throughout adulthood. This chapter outlines theoretical frameworks related to compensatory neural recruitment and mobility capacity with age to introduce the conceptual model, objectives, and primary research questions that guide the thesis.

Chapter 2 – Data Chapter 1

Chapter 2 is the first Data Chapter of this thesis, a manuscript entitled "Prefrontal Cortex Activity and Gait Velocity Through Adulthood: Shifts in Neurobehavioural Markers of Mobility Begin in Mid-Life", which examined the entire dataset of participants aged 18-79 to identify when there is deviation from young-adult levels of neural activity and gait velocity during complex dual-tasks. This chapter establishes change-point ages in mid-life using data-driven analyses.

Chapter 3 – Data Chapter 2

Chapter 3 is the second Data Chapter of this thesis, a manuscript entitled "Neurobehavioural and Sociodemographic Predictors Of Mobility in Adults 50-79 Years of Age", which investigated participants aged 50-79 for relationships between the brain activity and gait velocity. Cluster and mediation analyses explored the relationship between sociodemographic variables, prefrontal cortex activity, and gait velocity in a mobility context.

Chapter 4 – General Discussion

Chapter 4 integrates findings from both Data Chapters and discusses practical implications for the detection of mobility decline based on changes in neural activity related to mobility. The conceptual model is re-visited and updated based on study results and recommendations are made for future research directions.

Together, these chapters provide a comprehensive examination of neurobehavioural correlates of mobility capacity throughout adulthood. By integrating neural, behavioural, and sociodemographic factors, this thesis advances the fundamental understanding of the aging brain and identifies when intervention can be maximized to promote independent, active living.

The candidate, L. Maureen Krelove, was responsible for study design, data collection, all analyses, and manuscript/thesis preparation. Supervisory committee members provided guidance on study design, interpretation, and thesis/manuscript approval for defence.

Chapter 1 General Introduction

The Canadian population is aging. It is estimated that 25% of Canadians will be over age 65 by 2040.[1] Falls are the leading cause of injury in seniors and fall-related hospitalizations cost the Canadian healthcare system ~$2 billion annually.[2] Approximately 20-30% of older adults (OA; age 65+) experience at least one fall per year. Fall events in OA are linked to a subsequent downward cycle of health that results in reduced mobility and strength, increasing needs for healthcare support.[2] Independent living and interaction with the community is of high importance to many aging adults, making the maintenance of functional mobility of critical importance for healthy aging.[3]

1.1 Introduction

Effective fall prevention begins with identification of individuals at high risk. The most significant fall predictor is a history of falls, making first fall prevention a priority.[4,5] Most fall prevention guidelines focus on OA, aiming to reduce falls in those most negatively impacted. However, fall occurrence sharply increases during mid-life, starting at age 35-40.[4] Middle-aged adults (MA; age 35-64) accumulate risk factors for mobility decline and falls, including reduced physical activity and increased medication use, and experience an array of age-related physiological changes that alter their stability.[6] As young adults (YA: age 18-34) transition to mid-life, the most common fall circumstance changes from sport/exercise related to a result of ambulation during activities of daily living (ADLs).[7] Between age 45-55, the risk of a fall requiring hospitalization increases and continues to rise into older adulthood.[6,7] In terms of functional activity, difficulty performing ADLs and functional impairment affect ~15% of MA and continues to increase annually.[5]

Given the significant increase in risk factors for mobility decline and rise in fall occurrence in MA, it is of scientific and public health priority to focus on fall prevention before the age of 65.[4,8] Mid-life may also be ideal for intervention to mitigate risk factors as MA have a higher capacity to improve outcomes due to greater physical resilience.[5–8] Thus, intervention during mid-life has a greater potential to prevent mobility loss and fall occurrence than solely focussing on OA or those or have already fallen. However, for this early prevention to be effective there first must be characterization of mobility changes at physiological and behavioural levels throughout adulthood. Characterizing the progression of neurobehavioural changes allows for the identification of individuals who would benefit from intervention as early as possible to promote wellbeing and independence throughout the lifespan.[5,9,10]

1.1.1 Dual-tasking, Attention, and Aging

Daily life often involves doing two things at once, such as walking and talking. Usually, maintaining balance during a motor task, like walking, is often done with ease and can be paired with a secondary cognitive task, such as having a conversation or using a mobile device. However, this simultaneous execution of two tasks, or "dual-tasking" (DT), is known to be distracting and divides attention.[11,12] For YA, cognitive-motor DT is often successfully performed as balance is maintained using brain systems that require minimal active attention.[11,13,14] The resulting flexibility in attention allocation allows for direction towards the cognitive task and its successful completion.[11,13,15] DT becomes increasingly difficult with age due to functional and structural brain changes that result in diminished cognitive resources, impaired attentional capacity, and difficulty shifting attention.[14,16,17] Altered neural capacity manifests as an increase in already limited attentional resources being directed towards stability, this "posture-first" approach often reduces cognitive task performance.[11,14,18]

Regardless of prioritization, the division of attention during DT creates a DT "cost" wherein performance of one or both tasks is reduced due to the division of attention between them. DT cost highlights the active, attention demanding nature of balance and gait and OA often experience higher cost than YA. Frameworks of DT cost include the multiple resource model, which suggests that two tasks will interfere with one another if they require the same resources.[12] Expanding on this, the capacity sharing model suggests that two processes requiring limited parallel processing steps divide resources between tasks to reduce overall capacity and result in impaired performance of at least one task.[19]

In everyday environments, tasks must be completed while also processing environmental stimuli, such as when walking down the street and having a conversation at the same time. The age-related change in attention allocation during DT indicates that YA and OA process balance-distractor conflicts differently. In OA, motor-task prioritization and the overall reduction in attentional capacity significantly impact neural resource availability to additionally process the environment. Consequently, OA often experience non-hazardous daily life falls due to environmental inattention.[20–22]

1.1.2 The Prefrontal Cortex & Age-Related Changes

The prefrontal cortex (PFC) has a central role in directing executive function (EF), including the allocation of attention between tasks.[16,17,22,23] EF refers to the higher cognitive processes involved in non-routine, goal-directed behaviours and is broadly made up of working memory and attention.[12] Working memory is the limited capacity temporary storage system able to manipulate current information.[12] Attention underlies all executive functioning. It acts as a spotlight to select goal-relevant visuospatial stimuli in the environment and perform multiple

tasks simultaneously.[12] Specifically, the dorsolateral PFC (dlPFC) is highly involved in selective attention allocation, especially during balance tasks, as well as in working memory.[12,16,18,22,24]

There is a consistently observed increase in overall PFC activity in OA.[11,16,17,25] This upregulation is particularly prominent during DT, in line with the "posture-first" prioritization. The increase in frontal activity is often associated with reduced cognitive and behavioural performance, suggesting that balance control is more attention demanding in OA and preventing instability requires a higher level of attention.[11,22] Functional neuroimaging studies using functional near-infrared spectroscopy (fNIRS) have shown increased dlPFC activity in adults over age 50 during DT compared to YA.[16,22] fNIRS measures the concentration of oxygenated (O_2Hb) and deoxygenated (HHb) hemoglobin using near-infrared light to indirectly measure regional cortical activity. Emitting optodes release light in the near-infrared spectrum that uniquely interact with the chromophore of interest, the amount of light measured by receivers are used to calculate concentration values and infer activity at the cortical surface.

Studies examining dlPFC chromophore concentrations found that increased oxygenation is associated with higher attentional demand, reduced balance performance, less accurate cognitive performance in OA, and has a positive correlation with task difficulty.[12,16,18,21,22,26] As attentional demands of a walking task increase, such as when avoiding obstacles or remembering words, dlPFC oxygenation tends to increase to a greater extent in OA than YA, and additional increases are seen in OA at high risk of falling.[27–29] However, findings are inconsistent on the direction of PFC activity changes with age. Some work has found reduced O_2Hb in OA relative to YA in gait and balance tasks, potentially reflecting reduced processing capacity, and other work has shown no change in activity between YA and OA.[22,26,30] Further, the age at which PFC activity increases during locomotion is task dependent, with the youngest age of upregulation

during DT identified at age 30-35.[31] Taken alongside the other physiological, behavioural, and lifestyle related factors that change in MA, there is a not a clear understanding of when during adulthood that the onset of neurophysiological changes in attention allocation and EF take place. Focussed investigation into MA develops the understanding of the when and how neurobehavioural changes in attentional priority manifest and provides an opportunity to identify risk to intervene when benefit can be maximized.

To this end, the dlPFC has shown to mediate the effect of balance on DT performance wherein increased activity is associated with worse balance, gait, and cognitive performance.[21] Given the established changes in PFC activity during aging and its role in DT, there has been investigation into the predictive capacity of PFC activity from a mobility perspective. Subclinical alterations in EF during healthy aging are linked to diminished gait performance.[29,32] PFC activity thus may be a preclinical marker of future gait impairment in high functioning OA. Alongside the inconsistent directionality of PFC activity changes with age, it is reasonable to suggest that the relationship between PFC activity and age is not as simple as a linear upregulation. Factors beyond chronological age may contribute to PFC activity changes during aging and influence the extent to which age-related neural alterations influence behaviour. For example, education and physical activity can alter when structural and functional neural changes influence behavioural outcomes and can prolong the timing of functional decline.[33–35] Thus, it is of importance to identify the timing of changes in both PFC and gait outcomes and what factors can influence this neurobehavioural relationship.

1.1.3 Cognitive Mechanisms of Aging

The age-related change in PFC activity may reflect a compensatory mechanism or a reflection of neural inefficiency in response to altered brain structure and function. The structural changes include reduced cortical specialization and consequent impairment in processing efficiency that leads to increased requirement for executive control.[14,16] Dedifferentiation, the loss of specialized processing regions, results in broad bilateral recruitment of cortical regions. This is a major structural and functional change with age; YA show task-selective activation patterns, whereas OA have less distinct regional activations for the same task.[33] Another critical structural change is the reduction in cortical gray matter volume, particularly in the frontal lobes.[33] Indeed, during DT walking there is an established structure-function relationship wherein increased PFC oxygenation is associated with reduced gray matter volume.[36]

Structural changes in brain anatomy result in functional neural network alterations. A primary functional compensation in OA is the posterior-anterior shift in aging (PASA), which summarizes the consistent neuroimaging findings of reduced activation of posterior brain regions and upregulation of frontal regions with age.[25] Along similar lines, the hemispheric asymmetry reduction in older adults (HAROLD) model states that PFC activity in older adults becomes more bilateral, reflecting compensation or dedifferentiation.[37] Further, the compensation-related utilization of neural circuits hypothesis (CRUNCH) postulates that older adults show PFC upregulation at submaximal levels of task demand to preserve motor performance at the cost of approaching their cognitive resource ceiling at a faster rate.[38,39] This upregulation can be sufficient to maintain performance, in alignment with the compensatory reallocation hypothesis, which states that increased PFC activity with increasing task demands allow for task performance to be maintained at YA levels.[29] However, as task demands increase, such as in DT,

limited cognitive resources are depleted quickly and performance is reduced.[39,40] The amount of age-related change in frontal activity can also be related to life experience, as postulated by the revised scaffolding theory of aging and cognition (STAC-r) wherein "neural scaffolding" supports sustained EF throughout the lifespan.[38] STAC-r also provides a neural basis for the theory of cognitive reserve, which suggests that individual differences in life experiences (such as education) can impact cognitive processes and allows some individuals to more effectively compensate for age-related brain changes through higher reserve.[41,42] These functional neural adaptations are well established in OA and have been shown to start during middle-age years in rodent models,[43] but very little work has investigated when these structural changes begin in humans.

It should be noted that the PFC hemodynamic response alone is not sufficient to draw conclusions about functional DT patterns as behavioural outcomes heavily influence the interpretations of PFC activity changes. Successful compensation should refer to only upregulations that benefits cognitive/behavioural performance.[33,37] Attempted or unsuccessful compensation refers to scenarios where neural upregulation is not associated with or result in maintenance or increased performance, possibly reflecting inefficiency.[33,37]

1.1.4 Functional Mobility, Gait, and Falls

The second most important fall predictor, after fall history, is impaired or abnormal balance/gait.[8] Standing balance is essential for independence and completion of ADLs. There is an inverse relationship between age and balance ability, where older ages tend to result in reduced stability.[44] At the population level, change in balance ability, physical function, and strength occurs between 40-60 years of age.[4] Further, balance measures show age-related decline continuously throughout mid-life, and do not abruptly appear in OA.[45] These changes are not

considered age-related until age 50, and earlier decline is associated with inactive lifestyle and other risk-factors for mobility decline.[8] It remains unclear when during the middle-age transition to older adulthood that shifts in attention, task prioritization, and resulting gait performance changes occur.

Gait is often considered an "automatic" process that does not require attention. If this were the case, however, there would not be a DT cost on gait. There is consistent evidence across the literature of diminished gait performance during DT. This includes decreased velocity, step length, and cadence, as well as increased gait variability, clearly highlighting the attentional requirement of gait.[16,46,47] In YA, gait control is *primarily* controlled by subcortical circuitry, however as cognitive load increases, such as during DT, the requirement for executive resources also increases. This heightened task demand results in involvement of the executive locomotor pathway to facilitate gait, even in YA.[31] The dlPFC, as part of the frontoparietal circuit, is part of the executive locomotor pathway.[12] The general aging process leads to increased involvement of the executive locomotor pathway even without high task demand.[12,31,40] For OA, the transition from subcortical control of gait is needed to counter age-related sensory, motor, and cognitive reductions.[40] Eventually, the upregulation of frontal circuitry becomes insufficient to counter reduced attentional capacity. Prior to this, YA-level performance can be maintained by increasing frontal activity, but after a certain, unknown point, this compensation becomes unsuccessful.[29,31]

Gait velocity is an established predictor and indicator of functional balance in older adults. It is a common mobility measure in aging populations due to its sensitivity to changes in cognition and brain structure.[48–50] Reduced gait velocity may reflect damaged locomotor systems and increased attentional demands of walking, suggesting that walking speed is a comprehensive summary measure of mobility and longevity in aging adults.[48,51,52] Faster gait speeds are

positively associated with longer lifespan and velocities of at least 1.0 metres-per-second (m/s) predict overall longevity in OA.[51] In line with this observation, walking speed is considered a "sixth vital sign" and functional mobility indicator, and is an established predictor of falls and general health status in aging populations.[51,52] Given the aging population and prevalence of falls among the elderly, identification of risk for early prevention is essential. In Ontario, the recommended cross walking timing for safe and successful crossing for the general population is 1.0 m/s, the City of Toronto has implemented 1.0 m/s crossings in update to the prior requirement of 1.2 m/s to accommodate a wider range of pedestrians.[53,54] Taken together, the scientific literature and urban design both suggest that a gait speed of at least 1.0 m/s is critical for community ambulation and maintenance of safe mobility with age.

1.2 Summary & Rationale

Middle-aged adults are an understudied population in mobility research with unidentified neurobehavioural mechanisms related to changing DT and mobility capacities. This fundamental knowledge gap requires investigation to inform understanding of how the brain changes attention allocation throughout adulthood and its impact on gait and mobility. The establishment of methods capable of identifying individuals at risk of mobility decline before their first fall is critical to reducing fall-related injury and mobility-related disability in late adulthood.

The neurophysiological and behavioural markers of altered DT capacity have been extensively established in OA. OA prioritize stability at the expense of cognitive task performance, rely on executive resources, have a higher risk for instability, and experience more falls than younger adults. Overall, OA have a reduced DT capacity and higher DT cost that negatively influences their mobility. However, the timing of the transition in these neurobehavioural correlates of altered DT capacity from mid-life to older adulthood is unclear,

and it is unknown how these relationships are influenced by non-biological factors. This is of particular focus as many risk-factors for falls and mobility decline, and falls themselves, begin increasing during mid-life.

The conceptual framework related to this line if inquiry is presented in a graphical model, below (**Figure 1.1**). The primary research objective of each Data Chapter are labelled as 1 and 2, respectively, in this figure. In the centre, YA chronologically transition to OA, Data Chapter 1 focused on identifying the age at which neurobehavioural changes begin, as indicated by label 1. Specifically, the neurobehavioural measures of interest are the changes in motor behaviour and attention-related neural activity, as indicated by the boxes following label 1. PFC activity, which usually upregulates with age to allow for increased EF and attention in compensation for reduced subcortical motor control, was measured using fNIRS. Motor behaviour was quantified using gait velocity, which usually decreases with age and is an established predictor and indicator of functional and general health status in aging adults. Given that age-related changes in brain structure and function influence mobility, the second Data Chapter assessed if PFC activity mediates gait velocity across tasks, indicated by label 2.

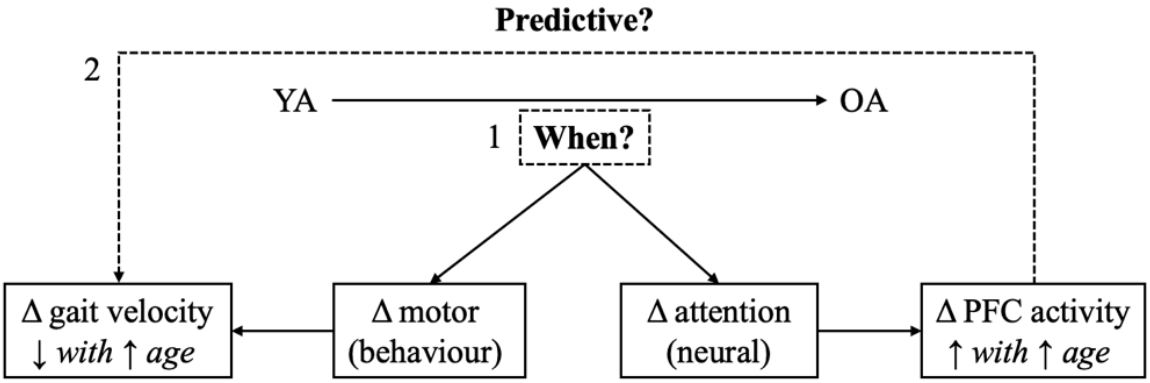


Figure 1.1. Conceptual model outlining the framework and scope of this thesis. Solid lines represent relationships establishing the initial framework of this work. Dashed lines indicate research questions and areas of investigation. Data Chapter 1 and 2 objectives are labelled as 1 and 2, respectively.

1.3 Objectives & Hypotheses

Given the established neural and behavioural changes that occur between young-adulthood at age 18-34 and older-adulthood at age 65, the ***overall objective*** of this work was to determine if, and at what age, neurobehavioural aspects of DT capacity diverge from YA levels and identify predictors of the neurobehavioural relationship. This thesis is comprised of 2 aims: 1) to determine at what age neurobehavioural correlates of dual-task capacity deviate from YA levels; and 2) identify if PFC activity can predict behavioural outcomes and identify any sociodemographic factors that influence this neurobehavioural relationship.

The first Data Chapter of this thesis, entitled "Prefrontal Cortex Activity and Gait Velocity Through Adulthood: Shifts in Neurobehavioural Markers of Mobility Begin in Mid-Life", investigated the relationship between chronological age and the neurobehavioural correlates of DT capacity in healthy adults age 18-79. The specific research question was: at what age do neurobehavioural correlates of mobility diverge from YA levels? It was expected that PFC activity, as measured by dlPFC oxygenation and gait velocity, would diverge from YA patterns in the middle age range of 50-64. This would be evidenced by a decrease in gait velocity and increase in PFC activity with age, and change-point ages during mid-life across walking conditions.

The second Data Chapter, containing the manuscript entitled "Neurobehavioural and Sociodemographic Predictors Of Mobility in Adults 50-79 Years of Age", involved an exploratory analysis of the sociodemographic factors that influence gait behaviour outcomes and assessed whether this relationship is mediated by PFC activity. The specific research question

was: does PFC activity mediate the relationship between sociodemographic variables and mobility outcomes? Identification of sociodemographic variables that predict gait velocity was paired with assessment of whether PFC activity mediates the relationship between these factors and gait velocity. The focus of this analysis on adults over age 50 allows for outcomes to address participants who are likely to already be experiencing neurobehavioural change in DT capacity, and will assist in broadly characterizing those with successful/unsuccessful PFC activity compensations who may be at higher risk for future mobility decline.

Chapter 2 Prefrontal Cortex Activity and Gait Velocity Through Adulthood: Shifts in Neurobehavioural Markers of Mobility Begin in Mid-Life

2.1 Abstract

Introduction: There are established age-related changes in neural function, motor output, and cognitive capacity. These factors directly relate to mobility in aging adults, and prior work has focus on the neurobehavioural differences between young (YA) and older (OA) adulthood. However, physiological alterations occur throughout the adult lifespan, and middle-aged adults (MA) experience the onset of physiological and functional decline, as well as an increase in fall occurrence during daily tasks. Despite this, MA remain an understudied population in mobility research. **Methods:** Using a previously validated complex gait dual-task involving word-recall and virtual obstacle avoidance, prefrontal cortex activity and gait velocity were measured using fNIRS and a pressure sensitive mat across four conditions: walk, walk + distraction, walk + obstacles, and walk + distraction + obstacles. **Results:** Locally weighted scatterplot smoothing was applied to neurobehavioural data from 113 healthy participants aged 18-79 and a piecewise regression revealed that there is a significant deviation from YA patterns of neural activity and behaviour in the mid-fifties across dual-tasks. **Conclusions:** This work, for the first time, shows that frontal brain activity, dual-task performance, and gait velocity deviate from YA patterns during mid-life. This suggests that middle-age may be a critical point to identify and intervene to prevent future falls and mobility decline.

2.2 Introduction

By 2040, 25% of the Canadian population will be over age 65.[1] Falls are the leading cause of injury in Canadian older adults (OA) and fall-related injuries stress individuals, families, and the healthcare system.[2] OA who experience a fall become vulnerable to subsequent deterioration in strength, independence, and mobility which ultimately increases risk of another fall.[2,55] Fall prevention is thus a high priority in the face of an aging population and interventions usually focus on OA who are severely negatively impacted by falls. However, the age-related changes in balance and gait that contribute to increasing fall risk accumulate throughout adulthood and fall occurrence sharply increases during mid-life, starting at 35-40 years of age.[4,56] Middle-aged adults (MA; age 35-64) progressively accumulate factors that place them at a higher risk of mobility decline and falls later in life.[5,6,57]

The most prominent predictor of a fall is fall history.[4,5] As young adults (YA; age 18-34) transition to mid-life, the most common circumstance of a fall changes from sport/exercise related to a result of ambulation during activities of daily living (ADLs).[7] Fall etiology is complex and occurs across parallel body and brain systems. Mid-life sees the onset of age-related metabolic and biological changes, reduction in nerve conduction speeds, and the start of decline in musculoskeletal health and strength.[4,8,58,59] For example, the loss of lean muscle mass begins in the 30s and accelerates from 40-60 years of age such that 3-8% of body muscle is lost per decade.[60–63] This has a direct effect on stability, balance, and gait; impairment in these systems are the second strongest predictor for a fall.[5,8,64] Between age 45-55, the risk of a fall requiring hospitalization increases and continues to rise into older adulthood. Fall prevalence increases from ~9% at age 43 to ~21% by age 54.[6,7,56] Indeed, early signs of emerging physical disability can be clear in MA and physical decline can begin 8-12 years prior to the first

fall.[56,58,64,65] Thus, mid-life is a critical yet understudied period where many subtle physiological changes begin to accumulate that contribute to fall risk and motor decline.[4–6,56,58]

Many daily life activities involve performing two simultaneous tasks, such as walking while talking. This "dual-task" (DT) is known to be distracting, requiring the division of attention between cognitive and motor tasks.[12,66] Behavioural performance metrics will often indicate slightly decreased stability or reduced locomotion during distraction in all age groups.[11] This DT "cost" evidences that fundamental motor tasks, like standing and walking, require some level of active attention.[14] With increasing age, DT cost increases and secondary task performance declines.[11,14] This age-effect has been related to attentional prioritization. For YA, motor tasks, including gait, are primarily controlled through subconcious, subcortical circuity.[11,14,18] Thus, the secondary cognitive task can be prioritized by attentional systems and completed with minor cost to either task.[11,13,14] On the other hand, DT becomes increasingly difficult for OA due to functional and structural brain changes that result in reduced cognitive resources, impaired attentional capacity, and difficulty shifting attention.[14,16,17] These changes manifest as an increase in demand on limited attentional resources, where a "posture-first" approach is taken wherein more active attention is directed to stability and cognitive task performance diminishes.[11,14,18]

To counter the age-related changes in sensory, motor, and cognitive systems, OA increase recruitment of executive resources.[12,31,40] There is a transition from subcortical control of walking to increased involvement of the executive locomotor pathway during even single-task walking in OA.[12,31,40] Gait velocity is an established fall predictor and general health indicator.[52,67,68] Self-selected comfortable gait velocities of <1.0 metres-per-second (m/s) in adults over 65 are associated with falls and adverse health events, and suggest need for fall-risk

intervention.[69,70] Gait velocities of 0.8 m/s and slower are a risk factor for hospitalization, cognitive decline, and negative health outcomes.[52,68] A DT walking task can reveal otherwise undetected subtle changes in gait reflective of altered attention prioritization before senior years.[27,71,72] DT walking thus can both indicate and predict fall risk and mobility, and provide insight to the role of attention in maintaining mobility and health.[72]

The prefrontal cortex (PFC) is a critical region for attention allocation. The PFC has a central role in executive function (EF), the higher cognitive processes involved in non-routine, goal-directed behaviours.[12] Attention is the neural basis of EF, acting as a "spotlight" for stimuli in the environment.[12] The dorsolateral PFC (dlPFC) is highly involved in attention allocation, especially during balance tasks.[12,16,18,22,24] There is a robust association between EF, gait, and balance.[59,73] Impaired EF predicts accelerated decline of lower extremity functioning in OA, and is the strongest cognitive correlate of functional status.[74,75] During DT, PFC activity increases and is associated with increased attention to maintain task performance that is further increased in OA.[16,17,23,76] .[12,16,22,77] This upregulation is especially clear during DT, in line with a "posture-first" prioritization, and is associated with reduced cognitive and behavioural performance. Neuroimaging studies using functional near-infrared spectroscopy (fNIRS) have shown increased dlPFC activation in adults over age 50 during DT compared to YA.[11,16,16,17,22,25] fNIRS provides non-invasive and portable indirect measurement of cortical activity by assessing concentration of oxygenated (O_2Hb) and deoxygenated (HHb) hemoglobin. Studies examining dlPFC oxygenation have found that increased O_2Hb is associated with higher attentional demand, reduced balance performance, less accurate cognitive performance, and has a positive correlation with task difficulty.[12,16,18,21,22,26]

Taken together, fall risk and prevalence begin to increase during mid-life, and evidence suggests that the changes in neural physiology, structure, and function that influence behavioural outcomes may begin during mid-life as YA transition to OA. The overall objective of this work is to determine if, and at what age, neurobehavioural correlates of DT capacity diverge from YA levels in healthy adults. The primary research question is: at what age do the neurobehavioural correlates of mobility and DT capacity diverge from YA levels? Based on established differences in YA and OA and the onset of neurobehavioural changes throughout mid-life, it is expected that PFC activity, measured by dlPFC oxygenation, and gait velocity will deviate from YA patterns somewhere in the mid-life range of 35-64 years of age.

2.3 Methodology

2.3.1 Participants

One-hundred and thirteen healthy adults were recruited from the York University community in Toronto, Canada, as well as from two Greater Toronto Area community centres. To be included in this study, participants had to be aged 18-79 with no stroke history, neurological diagnoses, concussion diagnoses and/or symptoms in the previous year, and no musculoskeletal injuries or surgeries in the previous year that could impact balance or gait. Written informed consent was obtained from all participants prior to participation; this study was approved by the Research Ethics Board at York University (certificate #e2023-198).

2.3.2 Equipment

fNIRS continually measured blood oxygen concentration using a portable and non-invasive 8-channel continuous wave unit (Octamon, Artinis Medical Systems, Elst, The Netherlands).

Penetrating wavelengths in the near-infrared spectrum were emitted at 760nm and 850nm and receivers measured at returning wavelengths at 50Hz. Emitting optodes and receivers were 30mm apart with a tissue penetration depth of 15mm. Chromophore concentrations (μM) were collected in real-time using OxySoft v3.2.72.

Gait data were collected using a 14x3 foot pressure sensitive mat (ZENO^{TM} Walkway Gait Analysis System, ProtoKinetics, Havertown, PA, USA). Spatiotemporal gait metrics were collected at 120Hz and analyzed using the ProtoKinetics Movement Analysis Software v5.08C1i6. Virtual obstacles were projected into the participant's view using an augmented reality (AR) headset (MagicLeap 2, MagicLeap, Plantation, FL, USA) that was be worn comfortably with the fNIRS (**Figure 2.1 A**).[78] Custom code was written to project three-dimensional objects onto the AR lenses (Unity Technologies, San Francisco, CA, USA). In the AR scene, there was full pass-through so participants could see the entire room and themselves, such as when using a regular pair of glasses. The headset projected a fence, shrub, and two orange pilons onto the gait mat (**Figure 2.1 B**). The obstacles were oriented such that there was enough room to navigate between them while remaining on the mat and encouraged avoidance behaviours to the right and left while walking forward (**Figure 2.1 C**). Real time head position in three directions was also collected by the AR headset but is not applied in the present analyses.

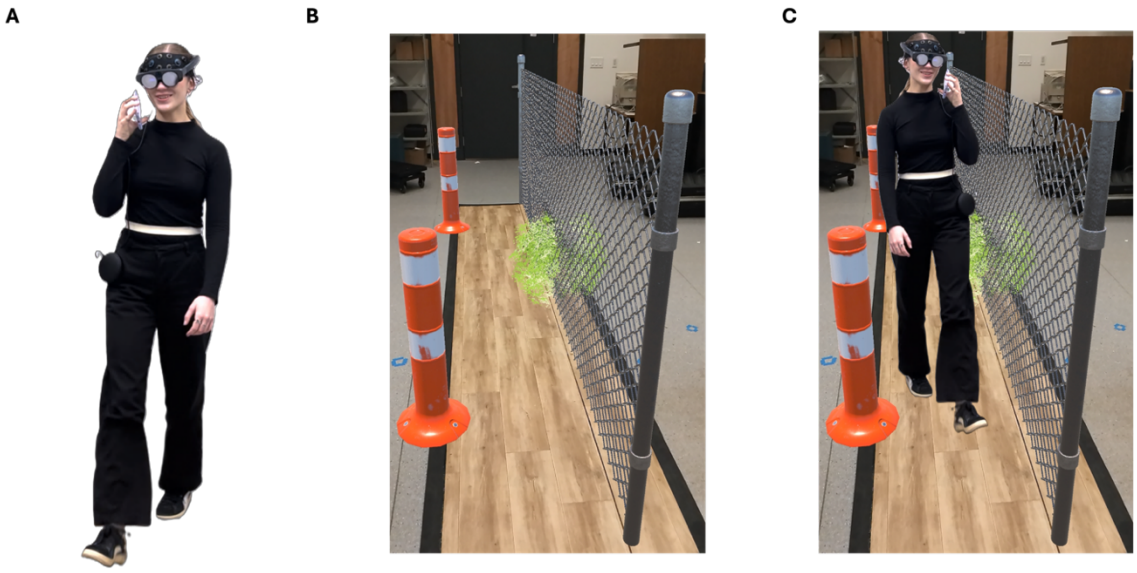


Figure 2.1. A. Participant wearing all equipment for the study and completing a distracted task. **B.** Virtual obstacles as viewed through the AR on the gait mat. **C.** Example of participant on gait mat during the obstacle avoidance and distraction condition (WOD).

2.3.3 Experimental Design

The experimental paradigm was separated into walking and rest conditions. All walking tasks were ~45 seconds in length at a self-selected comfortable pace on the gait mat. The specific task condition varied with the addition of virtual, AR-projected obstacles and/or word-recall distraction. Participants wore the AR headset and fNIRS for the duration of the study, viewing the room either as-is through the headset or with the addition of virtual, three-dimensional daily life obstacles on the gait mat (**Figure 2.1 B, C**). For the baseline condition, participants sat in a chair for one minute while counting by one from one. Simple counting reduced mind-wandering to produce a consistent baseline between participants without being challenging enough to engage PFC activity.[16] This one-minute rest task was repeated between walking conditions for a total of four seated baselines. **Figure 2.2** shows the general study procedure.

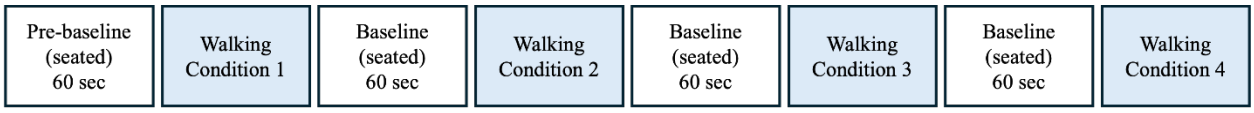


Figure 2.2. General temporal study procedure.

During obstacle avoidance conditions, participants were instructed to avoid the obstacles to the best of their ability without walking off of the gait mat. To increase cognitive challenge, distracted conditions required participants to facilitate a mimicked phone call. An audio recording of the experimenter providing a five-world list was played from their smartphone device while the participant held the phone up to their ear (**Figure 2.1 B, C**). To ensure consistency of neural activity, the participant held the phone to their ear for the duration of the condition. Words were randomly selected from the Toronto Word Pool using custom R code for near immediate recall at ~10-15 seconds after the end of the recorded word list.[79,80] A different word list was used for each distracted condition. Taken together, this study involves four walking conditions: walk (W); walk + distraction (WD); walk + obstacles (WO); and walk + obstacles + distraction (WOD).

The length of walking conditions varied between participants based on their self-selected gait velocity. This resulted in a varied number of times a participant needed to walk up and down the gait mat to obtain sufficient footfalls for accurate analysis (>15 footfalls). In general, each walking condition was one minute at maximum with approximately 30 footfalls captured per condition. Participants started their walk two metres before stepping on the mat and continued to walk 2 metres after the mat before turning to complete the next “pass”. The added off-mat distance allowed for steady state agit to be quantified until sufficient footfalls were obtained

while acceleration and deacceleration took place off the mat. Overall, the data collection duration per participant was approximately 10 minutes.

2.3.4 Data Analysis

2.3.4.1 Neurophysiology

fNIRS analysis was performed in alignment with guidelines by Pinti et al. (2018) and filtered using an adapted pipeline from Holtzer et al. (2011).[30,81,82] During data collection, optimal scalp-optode coupling was confirmed visually by the presence of the heartbeat at ~1Hz.[82] After collection, data were visually inspected for large motion artefacts, which were minimal but removed if present. Raw light intensities were converted to MATLAB script using open access code from Artinis Medical Systems (oxysoft2matlab) to convert .oxy4 files to .snirf format.

fNIRS processing software Homer3 was used to filter all signals.[83] First, optical density data were low-pass filtered at 0.1Hz to remove most physiological noise, such as blood pressure and Mayer waves.[81,84] The low pass filter alone is not sufficient to remove very-low frequencies (<0.01Hz) corresponding to vascular regulation and instrument noise.[82] A principal component analysis (PCA) was applied to address non-hemodynamic response signals that overlap with the signal of interest at 0.01-0.09 Hz including vascular motion and respiration.[30] A PCA was selected as a short source-detector separation was not possible with the hardware, which would have allowed for collection of local physiological noise and its regression out of the signal. Filtered optical density was converted to concentration using the Modified Beer-Lambert law.[81] Concentration data were averaged across all eight channels and separated based on whether it was from a walking or rest condition, and, if relevant, which walking condition it was. Using Spike2 v10 (Cambridge Electronic Design, Cambridge, UK), each DT walking condition (WD,

WO, WOD) was compared to single-task walking (W) with the "modulus" function to calculate of the absolute area-under-the-curve. Final data were expressed as a dual-task:single-task ratio of the absolute area-under-the-curve for O_2Hb concentration. This work investigates only the O_2Hb signal due to its high contrast-to-noise ratio, robust indication of regional changes in cerebral blood flow, and sensitivity in accordance with locomotor activities.[85–88]

2.3.4.2 Gait

Gait analysis was performed using the ProtoKinetic Movement Analysis Software. Gait data were visually inspected for incomplete footsteps, and any identified were removed (usually 0-3 steps) manually such that only complete footsteps in which the entire footfall was on the gait mat were used to compute gait metrics. The analysis software then outputted spatiotemporal and pressure-based gait metrics.

For the purposes of this study, gait velocity was selected as the primary measure of interest. Gait velocity is a common outcome measure to assess the effect of cognitive load and aging on mobility, as well as an established indicator and predictor of motor function, balance, falls, and general health outcomes in aging adults.[52,68] Across DT gait literature, the effect of a cognitive task is predominantly shown through changes in gait speed; gait velocity is an established measure to sensitively assess and monitor functional state and general health across the lifespan and is considered a "sixth vital sign".[52] Functional gait thresholds at 1.0 m/s as a "crosswalk" threshold and at 0.8 m/s as a general health indicator were used to assess participant performance across tasks and infer current and future functional capacity.[52]

2.3.4.3 Data Processing

To derive the age at which changes neural and gait measures began to deviate from YA levels, scatterplot smoothing was applied to PFC activity ratio and gait velocity data points. Data were smoothed using locally weighted scatterplot smoothing (LOWESS), a nonparametric and data-driven approach to data processing.[89,90] LOWESS was run with a 0.8 spanning factor for a moderate-strong smoothing of the data (~80% of the data were used in each local regression) and no outlier down-weighting iterations. These parameters were determined as optimal through k-fold cross-validation at $k = 10$.[89–92] The output was a smoothed curve to visualize the relationship between age and PFC activity or gait velocity across conditions. **Appendix A** details LOWESS theory, equations, and the cross-validation selection of the spanning parameter and robustness iterations.

2.3.5 Statistical Analysis

All analyses were run separately for each walking condition and outcome measure (PFC activity and gait velocity). Sex-differences were assessed using two-tailed independent samples t-tests in SPSSv29 (IBM, New York, USA). Descriptive statistics were used to characterize the study participants. All other statistical analyses were run in custom R code using the segmented package ('segmented' v 2.2-1) and significance set to $p \leq 0.05$.[93] To estimate a single interpretable age at which each outcome measure across conditions shows a change in the linear trend, which identifies the onset of deviation from YA patterns, a continuous piecewise (or hinge) linear regression with one breakpoint was run wherein;[94,95]

The change-point at age τ is defined as:

$$hinge(age; \tau) = max(0, age - \tau) \quad 2\text{-}1$$

Equation 2-1 was used to create a new predictor. A standard linear model was then fit as per Equation 2-2 to create the hinge model.

$$y = \beta_0 + \beta_1 \cdot age + \beta_2 \cdot hinge(age; \tau) + \varepsilon \quad 2\text{-}2$$

The hinge is zero before τ and increases linearly after. So, for ages less than or equal to τ, the hinge is zero and the model becomes:

$$y = \beta_0 + \beta_1 \cdot age + \varepsilon \quad 2\text{-}3$$

Such that the pre-change slope before τ is β_1.
For ages greater than τ, the hinge equals age – τ and the model becomes:

$$y = \beta_0 + \beta_0 \cdot age + \beta_2(age - \tau) + \varepsilon \quad 2\text{-}4$$

Such that the post-change slope after τ is $(\beta_1 + \beta_2)$. The hinge coefficient, β_2, tests $(\beta_2 - \beta_0)$. So, if $\beta_2 < 0$, there is further decline after τ; if $\beta_2 > 0$, slope is more positive after τ and decline slows after τ. The corresponding p-value at the hinge determines if there is significant slope change at τ and two-slope modelling is supported in the same, singular line.[94]

Candidate τ were identified from the full x-range (age 18-79) and a grid search was used to identify the ideal change-point using a hinge regression. Grid density was chosen to provide sub-year resolution at N = 200.[96] All candidate τ were estimated and linear model fit using small-sample corrected Akaike Information Criterion (AICc). The final change-point was identified by the value that minimized AICc.[95] Full AICc profiles and fitted hinge plots were saved for diagnostic evaluation of change-point stability. **Appendix B** details the hinge regression pipeline, all candidate change-points, and respective AICc values for the regression on LOWESS smoothed data points.

2.4 Results

2.4.1 Participant Demographics

In total, 113 participants aged 18-79 were included in the study. Participant demographic information can be found in **Table 2.1**. For descriptive purposes only, participants were separated into age groups as such: YA as age 18-34, MA as age 35-64; and OA as age 65-79.[5,58,64,97] This age grouping of participants is presented to describe demographic information in these typical age groups, the data were not analyzed in these groups outside of demographics.

Table 2.1. Participant demographics.

	YA	MA	OA	All
Group size	26 (14F)	40 (25F)	47 (35F)	113 (74F)
Age (years)	23.4±4.2	53.2±7.7	71.7±4.1	54.0±19.5
Height (cm)	171.0±8.9	166.8±9.9	161.5±10.1	165.5±10.4
Weight (kg)	71.7±14.8	75.1±16.1	68.21±16.5	71.4±16.1
Highest level of education				
Post-graduate	9	12	6	27
Undergraduate/college	4	24	23	51
Highschool	14	2	16	32
Public school	0	1	2	3
Recent Fall History (≤1 year)	4	6	6	16
Activities-specific balance confidence	98 (81,100)	98 (59,100)	94 (56,100)	98 (56,100)
High confidence (>80%)	27	37	36	100
Medium confidence (50-80%)	0	2	11	13
Physical activity (5x per week, 30min)	19	18	35	72
Daily prescribed medication	2	11	30	43
Medical History				
CNS – previous concussion	5	1	0	6
CNS – mental health/psychiatric	1	3	2	6
CNS – seizure history	0	0	1	1
Vision (acuity/cataracts)	4	5	18	27
Vestibular/hearing	1	6	12	19
Musculoskeletal	3	15	27	45

Group size, level of education, falls in the past year, physical activity level, daily prescribed medications, and diagnoses are expressed as counts. Age, height, and weight are presented as mean ± standard deviation. Activities specific balance confidence is expressed as median (range). F = female.

2.4.2 Dual-Task Performance

DT performance was recorded based on the number of total and correct responses and corresponding error rate (error rate = number of errors / number of responses x 100%) as shown in **Table 2.2**. The assessment of DT cost via error rate accounted for the fact that the number of total and correct responses may inversely relate to one another (i.e. more correct responses but fewer total responses) and more accurately reflect DT performance than a simple error count alone.[26,78] In MA, median total and correct responses decreased by a single response relative to YA. The reduction from perfect or near-perfect recall in YA to a lower median total and correct response in MA suggests increased dual-task costs. Further, the range expands to include zero and one total and correct response, further supporting that in MA there is altered DT capacity than YA.

Table 2.2. Word recall performance during DT

	YA	MA	OA	ALL
Median Responses				
WD	5 (3,5)	4 (1,5)	3 (1,5)	4 (1,5)
WOD	5 (3,5)	4 (0,5)	3 (0,5)	4 (0,5)
Median Correct Responses				
WD	4.5 (3,5)	4 (1,5)	3 (0,5)	4 (0,5)
WOD	5 (2,5)	4 (0,5)	3 (0,5)	4 (0,5)
Error Rate				
WD	11%±20%	10%±15%	16%±30%	12%±24%
WOD	11%±24%	15%±19%	12%±20%	13%±20%

Total and correct responses expressed as median (range) and mean ± standard deviation for error rate.

2.4.3 Neurophysiology

PFC activity as a ratio of O_2Hb concentration for DT:ST are shown as scatterplots in **Figure 2.3**. Across participants aged 18-79, there was a general increase in PFC oxygenation ratio with age. Oxygenation ratios below 1 suggest that DT required less PFC activity than ST walking, which may reflect reduced task engagement with increasing challenge or the use of non-PFC areas to handle increasing task demands.[26] There was no sex difference in PFC activity across conditions.

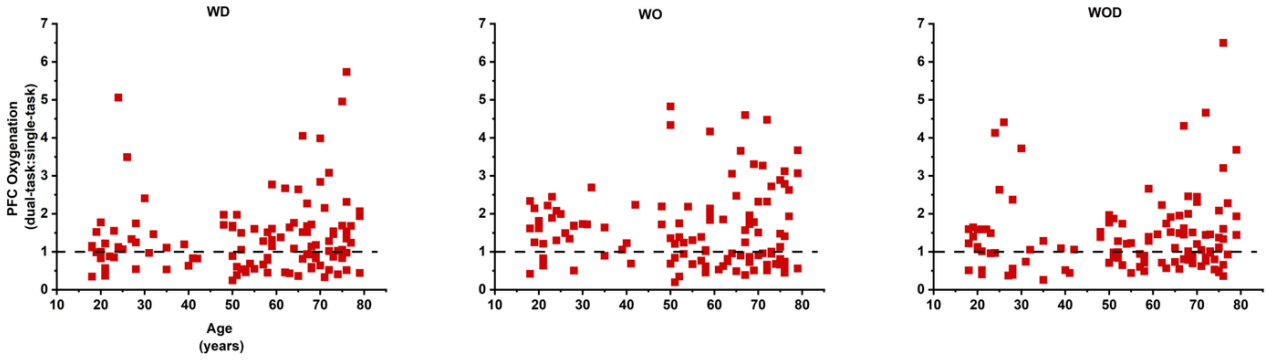


Figure 2.3. PFC oxygenation shown as a ratio of O_2Hb concentration for DT:ST per condition across participants aged 18-79. Baseline PFC activity for ST has a value of 1 (dashed black horizontal line).

2.4.4 Gait Velocity

Gait velocity (m/s) for each participant are shown as scatterplots for each condition (**Figure 2.4**). There was a general decrease in gait velocity with increasing age, as well as overall decreases in velocity with increasing task difficulty. Further, after age 50 there was an increase in the proportion of participants below the 1.0 m/s crosswalk threshold across conditions. During DT, there was an increase in individuals over 50 who fall below the 0.8 m/s general health

indicator, especially during the most complex WOD condition. Non-height normalized values were presented as functional mobility is the focus of this work; in daily situations, such as crossing at a crosswalk, there is not a consideration of height.

There were significant sex differences across conditions, as expected for a predominantly female participant pool. In general, females tend to walk slower than males due to anthropometric differences, such as height. **Appendix C** further details and discusses these sex differences.

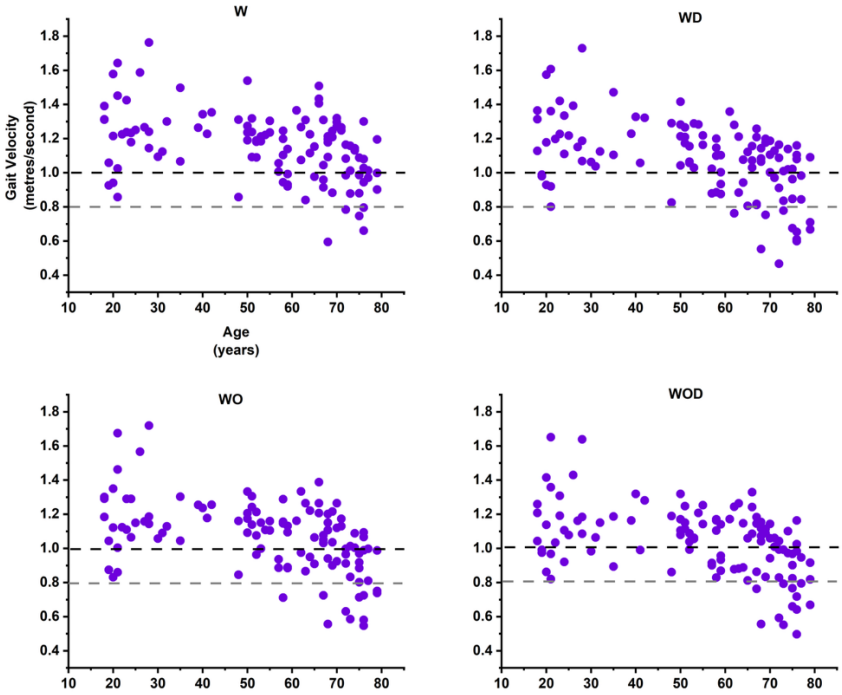


Figure 2.4. Gait velocity per condition across participants aged 18-79. Dashed black line at 1.0 m/s indicates the functional "crosswalk" threshold, dashed gray line at m/s indicates the general health threshold.

2.4.5 Piecewise Regression

Across tasks, the change-point ages, representing a significant change in linearity, were in the Fifties for both PFC activity and gait velocity. **Figure 2.5** and **Figure 2.6** show the change-points on LOWESS smoothed curves for each measure and condition.

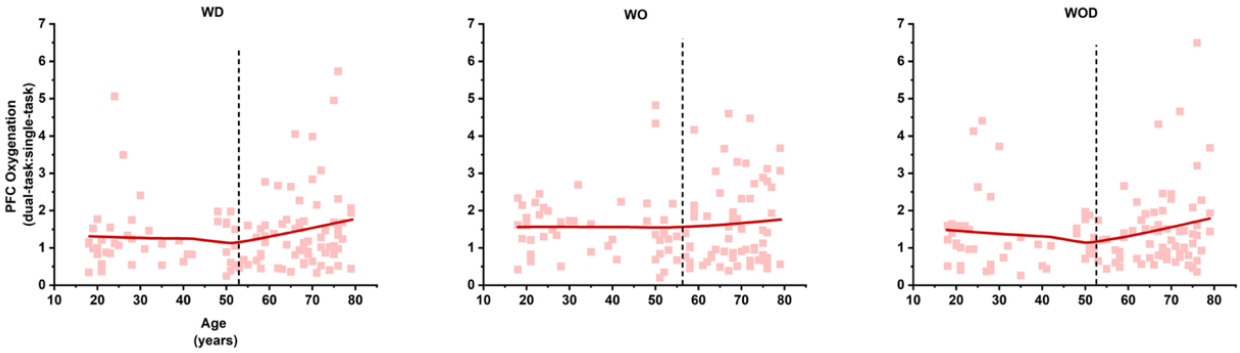


Figure 2.5. PFC oxygenation ratio plotted against age. LOWESS smoothed curve shown per condition, overlayed on raw data points. Change-point age for each condition indicated by dashed horizontal lines.

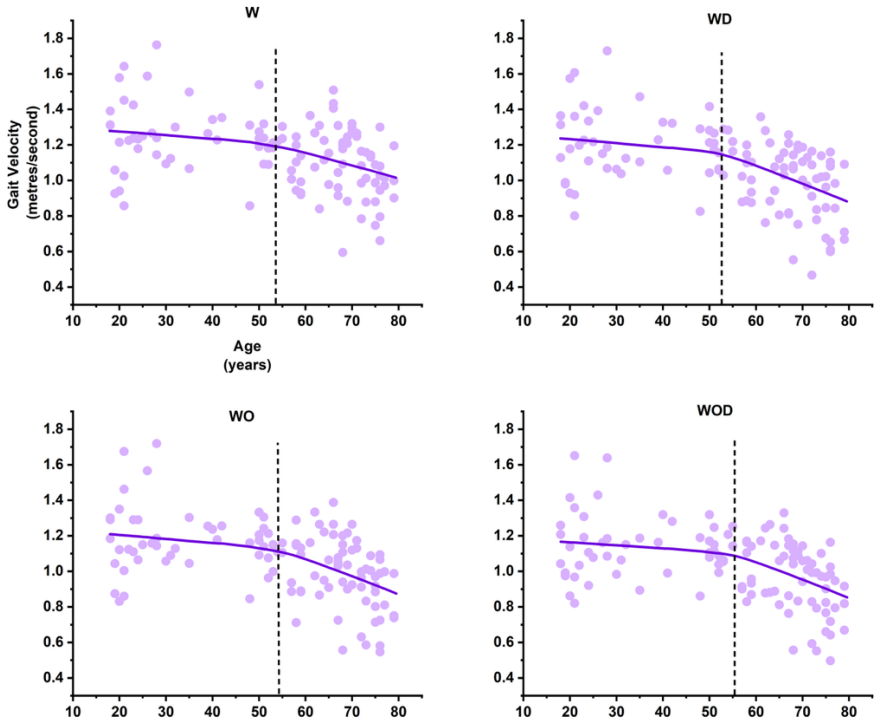


Figure 2.6. Gait velocity plotted against age. LOWESS smoothed curve shown per condition, overlayed on raw data points. Change-point age for each condition indicated by dashed horizontal lines.

Hinge model and piecewise regression outcomes are summarized in **Table 2.3** for the change-point slopes. Given that the regression was used to identify a significant change in linearity, outcomes are reported in terms of slope on either side of the ideal τ. Traditional regression outcomes and further detail on the intercept and hinge model and regression outputs on both smoothed and unsmoothed data can be found in **Appendix D.**

Across conditions and measures, ideal τ ranged from 52-55 years of age for gait velocity and 52-56 for PFC activity. These outcomes indicate that the most error is minimized by τ at these x-values, and suggest that the significant deviation (all $p < 0.001$) from YA levels of neuro-behavioural outcomes across tasks occurs during the fifties. The addition of the hinge coefficient, $\beta1+\beta2$, creates negative values for gait velocity and positive for PFC activity. This indicates that gait velocity declines after the change-point, and PFC activity increases after the change-point.

Table 2.3. Hinge model and piecewise regression outputs.

	τ	Est. β_1	Est. $\beta_1+\beta_2$	SE β_1	SE $\beta_1+\beta_2$	T-stat β_1	T-stat $\beta_1+\beta_2$	df	p	CI β_1	CI $\beta_1+\beta_2$
Gait											
W	53.56	-0.24	-0.71	1.87E-03	2.69E-03	-126.18	-263.64	110	**<0.001**	[-0.24, -0.23]	[-0.71, -0.70]
WD	52.64	-0.25	-1.01	1.97E-03	2.64E-03	-126.43	-382.87	110	**<0.001**	[-0.25, -0.25]	[-1.02, -1.01]
WO	54.79	-0.26	-0.98	2.22E-03	3.51E-03	-117.31	-278.48	110	**<0.001**	[-0.27, -0.26]	[-0.99, -0.97]
WOD	55.40	-0.20	-1.01	1.81E-03	2.99E-03	-110.36	-335.71	110	**<0.001**	[-0.20, -0.20]	[-1.01, -0.99]
PFC											
WD	52.95	-0.005	0.02	1.05E-04	1.44E-04	-48.86	163.17	110	**<0.001**	[-5.33E-04, -4.91E-03]	[0.023, 0.024]
WO	56.32	-0.001	0.01	4.54E-05	8.06E-05	-8.53	110.95	110	**<0.001**	[-4.77E-04,-2.97E-04]	[8.78E-03, 9.10E-03]
WOD	52.64	-0.009	0.02	1.21E-04	1.62E-04	-83.83	147.96	110	**<0.001**	[-1.10E-02, -9.87E-03]	[2.37E-02, 2.43E-02]

SE abbreviates standard error, the t-statistic (T), degrees of freedom (df), and 95% confidence interval of the slope estimates before (β_1) and after the change-point ($\beta_1+\beta_2$). The df and p- values are the same for both slope values.

2.5 Discussion

The aim of this study was to identify when neurobehavioural correlates of DT capacity deviate from YA levels of PFC activity, gait velocity, and task performance in healthy adults aged 18-79. It was expected that these neurobehavioural deviations would begin in mid-life sometime between age 35 and 64. Using non-parametric data driven modelling, the hypothesis was supported. We identified significant change-point ages in PFC activity and gait velocity in between ages 52-56, suggesting that the neural control of locomotion begin to shift towards OA patterns in mid-life. These novel outcomes support that altered attention prioritization during complex tasks occur earlier in adulthood than previously reported. This is the first mobility-based evidence for dynamic cortical engagement and executive resource utilization across the adult lifespan that result in negatively altered gait behaviour and cognitive performance.

2.5.1 Increased Executive Recruitment in Mid-Life

Across tasks, PFC oxygenation deviated from YA levels in the Fifties (**Figure 2.5**). Altered PFC activity, specifically in O_2Hb concentration, with age is often observed during DT and has a positive association with increasing age and task demands.[16,22,29,46,47,76] Increased O_2Hb concentration during DT walking is related to reduced gait and/or cognitive performance.[16,22,46] Other work has found that resting state functional networks have inflection points for connectivity in the third and fourth decade of life.[98] The trajectories of age-related differences in functional neural network connectivity across adulthood have been shown to be a non-linear, negative quadratic.[98] This inverted U-shape shows that functional network decline begins around age 40 and accelerates with increasing age.[98] The identified PFC change-point ages between age 52-56 are slightly older, which may reflect the low participant density in the Forties. Regardless, based on these results, the Fifties are a critical change-point where increased EF is required to

complete complex tasks. This work also did not identify a parametric relationship between age and PFC activity, but rather a significant change in linearity in the fifties followed by a gradual increase in PFC activity through to age 79 (**Table 2.3**). The increase in PFC recruitment in the Fifties supports a non-linear aging process whereby increasing EF reliance begins in mid-life and, alongside the overlap in behavioural change-points, suggests increased executive locomotor control in MA.

Executive reliance for successful locomotion is in line with several theories of cognitive aging. Frontal recruitment and the loss of task-specific lateralization are theoretically described through the hemispheric asymmetry reductions in old age (HAROLD) model and the posterior to anterior shift in aging (PASA).[25,99] Both of these theories support the age-related increase in PFC activity.[16,22,26] There is an established reduction in hemispheric lateralization and de-differentiation of specialized cortical regions with age such that the maintenance of subsequent behavioural performance requires more executive resources and broader cortical recruitment.[37,38,98,100] Functional hypotheses suggest that OA recruit more frontal/executive resources relative to compensate for functional deficits in other regions.[98,101,102] The compensation-related utilization of neural circuits hypothesis (CRUNCH) postulates that OA recruit higher levels of neural resources than YA at the same level of submaximal task demands to preserve motor performance.[101,103] CRUNCH aligns with the results of this work; there is increased executive recruitment in the Fifties during DT in an attempt to maintain gait velocity and cognitive task performance. EF and attention are limited cognitive resources with a declining executive resource "ceiling" with age.[12,101] Chen et al. (2022) found that as walking task difficulty increased, OA changed from right sided PFC activation to bilateral recruitment, the left PFC resources compensated for the right during DT obstacle avoidance.[16] Efficiency in the

recruitment and use of neural resources is another theoretical model in aging.[98,104] The scaffolding theory of aging and cognition (STAC) suggests that recruitment of additional neural resources and establishment of neural "scaffolds" preserves cognitive and executive functions with age.[105] Under STAC, dedifferentiation and compensation are two related processes wherein dedifferentiation and resulting alteration of functional networks compensate for the change in the efficacy of neural scaffolds with age.[103,105,106]

2.5.2 Mid-life Mobility Markers

Locomotion is one of the most complex and integrative behaviours of the human nervous system, requiring coordination across executive, sensory, and motor systems. In particular, gait velocity is a sensitive indicator of overall neural and physiological health in aging adults.[48,52,68] In the present study, the significant change-point in gait velocity across conditions from 52-55 years of age suggests that that gait behaviour cannot be maintained at YA levels by the mid-fifties. This change point age is lower than identified in previous work, where it has been found that motor performance can be maintained at YA levels until the Sixties and it is not until the Seventies that gait automaticity declines, with the most pronounced decrease during DT happening between ages 70-85.[31,107] The earlier behavioural change-point age in this work is likely due to the type of individual data-point analyses conducted. Age-groups were not the focus of this work but rather these results were from data-driven approaches on individual data points, which may be more sensitive to slight deviations in behavioural outcomes.

The nearly identical change-points for PFC activity and gait velocity in mid-life suggest that suggest that PFC upregulation may represent an immediate attempt to compensate for physiological changes that result in gait velocity reductions. If the compensation were consistently successful, the change-point age for gait velocity would be later in life than that for

PFC activity, as the increased use of executive resources would be able to maintain gait velocity at YA levels until later in life. Instead, this work shows diminishing subcortical locomotor control in the Fifties and re-organization of functional brain networks, reflecting adaptive and active neuroplasticity.[7,98] The unsuccessful compensation may not necessarily be a marker of decline, but rather a possible reflection of the onset of compensatory strategies through alterations in neural control strategies. Indeed, PFC activity alone may be an inefficient adjustment to changing brain structure, function, and overall physiology. At a structural level, the start of cortical anatomical alterations in mid-life may initiate dynamic engagement of neural networks in a compensatory manner for a changing brain. For example, the loss of cortical gray and white matter and reduced nerve conduction velocity begins in the Forties and Fifties.[98,108,109] There is evidence of wide-spread brain atrophy, especially in areas for sensorimotor processing, that may further exacerbate DT cost in aging adults while walking.[110] Taken together, this study suggests that PFC upregulation in mid-life is less about compensation for decline but rather a dynamic process of functional re-organization in a changing brain and body.

2.5.3 Attention Priority Shifts in Mid-life

The deviation of neurobehavioural outcomes in the Fifties suggests that MA are shifting towards a "posture-first" attentional priority. DT word recall performance in this study decreased in MA relative to YA, and performance was similar between WD and WOD (**Table 2.2**). WOD, however, has a wider range of correct and total responses, going from three to five responses in YA to zero to five in MA. This decrease in response range heavily suggests that attentional priority during a complex task can elicit altered attentional prioritization in MA that is not seen in YA but is maintained into OA. Conceptual framing of DT cost aligns with many of the neurocognitive theories discussed. The central capacity sharing model suggests that interference

between cognitive and motor tasks occurs because of a single, limited-capacity parallel processor that divides its resources between tasks (ex. executive/attentional resources).[12,19] Since both tasks increase PFC and EF demands, there is a lower capacity for each individual task and performance of at least one will be impaired. Along similar lines, the multiple resource model suggests that two tasks will interfere with one another if they require common limited resources.[12,111] The bottleneck theory of DT interference suggests that performance on one or both tasks results from serial processing wherein two tasks need the same processing network or when processing networks overlap.[12] These processors can only act on one task at a time, leading to a processing "bottleneck" and a delay or impairment of one or both tasks. In aging adults, the motor and cognitive task both require increased PFC activity and EF resources to try to maintain performance at YA levels during DT. These findings are in line with PASA and HAROLD, while deficits in task performance on one or both tasks are in line with the capacity sharing model.

Based on gait velocity and DT performance, MA may be allocating more executive resources towards the motor task. However, this redirection of attention is not entirely successful, as gait velocity is also beginning to decrease as PFC activity increases based on post-change point slopes (**Table 2.3**). The increase in PFC activity without maintenance of YA levels of word recall performance suggests inflexible attention and reduced EF. Cognitive domains, like EF, have shown to peak in the third decade of life and gradually decline.[98,112] Further, OA with poorer EF have a higher fall risk.[113] Adapting behaviour under changing environmental demands is essential for daily life and effective locomotion, relying on the ability to flexibly allocate attention.[114] The reduction in DT performance in MA suggests inflexible attention prioritized to stability more similar to EF in OA than YA.[98] This work presents novel evidence that DT

strategies and attention priority shift to OA-like patterns of attention allocation and prioritization in the Fifties.

2.5.4 Functional Mobility throughout Adulthood

In healthy middle-aged adults, reductions in self-selected normal walking speed (such as condition W) reflects poor physical, reduced cognitive function, and an accelerated rate of biological aging.[51,115] While OA often have more severe consequences of a fall, it is in mid-life that first fall occurrence increases and risk factors accumulate.[5,58] MA may be experiencing the beginning of age-related changes in attention, brain structures, and neural network processing, as discussed, but are ultimately more capable to participate in and respond to early intervention.

In this work, there was a general task-related decline in gait velocity, wherein participants of all ages tend to walk slower with increasing task difficulty. For those who are younger than 65 and show gait speeds below 1.0 m/s in any task, there may be a need to investigate balance. For YA, this may be due to ease of task challenge and disengagement. In MA, this may provide identification of individuals who are at a higher risk of future mobility decline or falls, particularly during DT. These individuals may be those who are at a higher risk of a first fall in mid-life, and would benefit from early fall prevention intervention.[70] For those who show gait velocities of 0.8 m/s or slower, there is further concern for future impairment beyond only gait and balance.[52,68,70,116] In W, only participants over 65 slowed their gait below this “general health” threshold. With the addition of more complex gait in DT, participants in later mid-life show gait speeds at and below 0.8 m/s, highlighting the additional sensitivity of DT to identify potential gait impairment.[117]

2.5.6 Limitations & Future Directions

This study is not without limitations. In terms of neurophysiology, hardware limitations meant that only the PFC activity could be measured and signals could have originated from areas outside of only the dlPFC. Further, there was no screening of participants' cognitive status, such as through the Mini-Mental State Exam (MMSE) or Montreal Cognitive Assessment (MOCA).[118,119] The only measure of EF in this study was cognitive performance on the dual-task. Finally, this study was cross-sectional and not longitudinal. A longitudinal assessment is required to confirm when neurobehavioural changes in DT capacity occur within a person, in this dataset the cohort vs age-related effects cannot be disentangled. Future work should undertake a longitudinal approach or apply a mid-life focus to existing longitudinal aging datasets to disentangle cohort and age effects, as well as allow for within-person assessment of deviation from YA levels of PFC activity, DT performance, and gait velocity.

2.5.7 Conclusions

For the first time, this study shows that neurobehavioural correlates of mobility deviate from YA levels in mid-life. Using nonparametric and data-driven regressions on PFC activity and gait velocity during a complex gait DT, significant change-point ages in the 50s across conditions were identified. Previous work has shown that fall-risk and occurrence rise during mid-life, which is supported by these results from a neurobehavioural perspective. This work suggests that PFC upregulation is an immediate response to age-related motor decline and that attention priority changes during mid-life. The results of this work indicate that focussed fall prevention efforts are needed in mid-life, and that middle-age is a crucial point for intervention to maintain mobility throughout the lifespan and empower active, healthy aging.

Chapter 3 Neurobehavioural and Sociodemographic Predictors of Mobility in Adults 50-79 Years of Age

3.1 Abstract

Introduction: Maintaining mobility with age requires engagement of cognitive, executive, and motor systems. Neurobehavioural outcomes related to mobility may be influenced by sociodemographic factors beyond biological aging. It is unknown how sociodemographic variables relate to neural and behavioural correlates of mobility in aging adults. **Methods:** Adults aged 50-79 participated in a complex cognitive-motor dual-task either in the laboratory or at community centres. Prefrontal cortex activity (PFC) was measured using functional near-infrared spectroscopy and a pressure sensitive mat recorded gait velocity during single-task walk, walk + obstacles, walk + distraction, and walk + obstacles + distraction. **Results:** Data from 79 participants show that community-based participants exhibited significantly slower gait velocity compared to laboratory-based participants. Two-step cluster analysis shows that age, location of participation, and level of education determine participant groupings. Non-linear mediation analyses demonstrated that age and level of education predict gait velocity across tasks but these relationships are not mediated by the PFC. **Conclusions:** PFC activity may be a parallel compensatory mechanism for age-related cognitive and behavioural performance influenced by sociodemographic factors driven by compensatory scaffolding and cognitive reserve. The relationship between neural activity, mobility, and sociodemographic factors requires further investigation to address mobility outcomes across the aging population.

3.2 Introduction

Maintaining independent mobility is a priority for aging individuals and healthcare systems in Canada. Cognitive, executive, and motor systems are essential for successful mobility in daily life, especially when task or environmental demands are challenging.[48,75] Daily life often requires managing multiple tasks at once, like walking while talking. This "dual-tasking" (DT) divides attentional resources and creates a need to prioritize attention between tasks. This prioritization of attention resources can result in reduced cognitive task performance and environmental inattention that can lead to instability.[11,120]

The prioritization of attention changes with age, wherein young adults (YA; age 18-35) prioritize successful secondary cognitive task completion and older adults (OA; age 65+) prioritize stability, even at the cost of cognitive task performance.[11,120] This shift in attention allocation is related to age-related changes in neural and body systems, including reduced cortical specialization, increased executive demand, reduced muscular strength, and impaired sensory processing.[39,61] Regions in the frontal lobes, namely the prefrontal cortex (PFC), direct executive functions (EF), the higher-order cognitive processes required for goal-directed behaviour.[16,23] Attention underlies all EF, acting as a spotlight on environmental stimuli for further processing.[59] EFs are also the strongest cognitive correlates of functional mobility status in aging adults with a robust association to gait and balance.[58,59,73,74] Importantly, these changes start in the mid-life years (mid-50s; Chapter 2), making middle-age a critical group to include when examining changes in DT capacity, PFC activity, and gait velocity.[8,26,31,121]

During DT, PFC upregulation often reflects heightened executive and attentional demands of locomotion and attempt to maintain task performance at YA levels, particularly in aging adults.[22,23,46] Upregulation of the dorsolateral PFC (dlPFC) measured using functional

near-infrared spectroscopy (fNIRS), has been associated with higher attentional demand, poorer balance and cognitive performance, and has a positive correlation with task difficulty.[16,22,23,77,122] In this way, upregulation of the PFC is often considered a compensatory response to age-related changes in brain structure and function. This compensatory mechanism, however, is only successful until task demands exceed resource capacity and DT "cost" increases. This resource ceiling is reached earlier for OA performing the same task as YA, who also show increased DT costs reflecting the interplay of gait and cognition.[39,120,123,124]

The maintenance of effective gait reflects a complex interplay of clinical and lifestyle factors. Specifically, gait velocity is an established predictor and indicator of falls, health status, and longevity in aging adults.[12,52,68,125,126] DT gait paradigms can more sensitively probe for challenges in maintaining gait velocity by increasing cognitive load and can reveal subclinical levels of mobility or cognitive impairment.[66,72] Thus, there is an established neurobehavioural relationship between PFC activity and gait velocity. However, biological aging is not the only contributor to altered DT capacity and gait velocity. Age and biological sex have shown to account for gait velocity variance, mobility outcomes, and neural structure and function with age. For example, being of female sex, older age, lower socioeconomic status (SES), and lower health status have shown to predict slower gait velocity.[127] SES is usually measured by education, occupation, employment status, income, and wealth,[128] and is broadly considered to be a general health indicator.[129,130] In both men and women, lower education level and lower median household income are risk factors for mobility decline.[131,132] Factors that positively influence neurobehavioural factors, like higher levels of education, can provide enhanced capacity to maintain behaviours with age-related changes in the brain and body.[42] Level of education is a strong predictor of life-long cognitive performance, wherein post-secondary education or higher

is considered neuroprotective due to positive effects on EF and memory systems.[58,133,134] Longer periods of schooling are also associated with greater resources to compensate for age- and disease- related changes in neural structure and function.[135] For example, higher levels of education support EF and memory likely through strengthening the mediating connections between the dlPFC and hippocampus.[136] Lower SES is related to smaller dlPFC volume and poorer EF such that the dlPFC mediates the relationship between low SES and decline cognitive performance.[137]

Overall, it has been shown that sociodemographic factors can influence neural substrate but this has not been directly explored in a DT and mobility context. Taken together, there is an interconnection between gait velocity, PFC activity, and sociodemographic factors in a mobility context. However, it is unclear which sociodemographic factors influence neurobehavioural outcomes and how they relate to PFC activity and gait velocity in a mobility context. The objective of the present work is to identify the sociodemographic factors that are most important in differentiating individuals and assess if the dlPFC mediates the relationship between sociodemographic predictors and resultant gait velocity across complex gait dual-tasks. The primary research question is thus: what are the sociodemographic and lifestyle predictors of gait velocity in adults age 50+, and is this relationship mediated by PFC activity?

3.3 Methodology

3.3.1 Participants

Given that the low-to-mid Fifties were identified as the change-point for neurobehavioural outcomes in Data Chapter 1, the 79 participants between ages 50-79 were

included in the present analysis. Participants were recruited from the York University community and from two Greater Toronto Area community centres. Participants in this analysis had to be between ages 50-79 with no history of stroke, neurological diagnoses, concussion and/or concussion symptoms in the past year, and no recent musculoskeletal injuries or surgeries. Written informed consent was obtained from all participants prior to participation; this study was approved by the Research Ethics Board at York University (certificate #e2023-198).

3.3.2 Equipment & Experimental Design

The full experimental paradigm and its validation are detailed at length in Data Chapter 1 and Krelove et al., (2025).[78] Briefly, an 8-channel fNIRS system (Octamon, Artinis Medical Systems, Elst, The Netherlands) continually measured blood-oxygen concentration at 50Hz using emitted wavelengths of 760 and 850nm in OxySoft v3.2.72. Gait data were collected using a 14x3 foot pressure sensitive mat (ZENO™ Walkway Gait Analysis System, ProtoKinetics, Havertown, PA, USA) at 120Hz using ProtoKinetics Movement Analysis Software v5.08C1i6. Virtual obstacles were integrated using custom code (Unity Technologies, San Francisco, CA, USA) via an augmented reality (AR) headset (MagicLeap 2, MagicLeap, Plantation, FL, USA).

The experimental paradigm was separated into walking and rest conditions. All conditions involved walking on the gait mat at a self-selected pace with and without AR-projected obstacles and/or word-recall (**Figure 2.1**). While walking, extra steps were taken on and off the mat for acceleration and deacceleration between passes. Validated virtual obstacles created "obstacle" conditions, wherein participants were instructed to avoid the obstacles while remaining on the mat to the best of their ability.[78] To increase cognitive load in "distraction" tasks, participants completed a mimicked phone call involving an audio recording of the experimenter providing a 5-word list that they were instructed to listen to, remember, and recall

after 10-15 seconds as they walked. Words were randomly selected from the Toronto Word Pool using custom R code.[79,80] A different word list was used for each distracted condition, and randomly alternated between nine different word lists across participants. Taken together, this study involves four walking conditions: walk (W); walk + distraction (WD); walk + obstacles (WO); and walk + obstacles + distraction (WOD).

Participants were familiarized with the study procedures and equipment, and began the study by sitting in a chair for one minute and asked to count out loud by one from one. This baseline task was repeated between each walking condition to allow for physical rest and for the PFC hemodynamic response to return to baseline. Overall, the duration of data collection was approximately 10-15 minutes (**Figure 2.2**).

3.3.3 Sociodemographic & Lifestyle Questionnaires

Age, sex, level of education, physical activity level, balance confidence, dual-task performance, medical history, and location of participation were collected as part of the general personal information forms. Balance confidence was based on self-report through the Activities-Specific Balance Confidence Scale (ABC).[138] Physical activity was based on self-report for at least five activities a week of at least thirty minutes each, in line with American College of Sports Medicine recommendations.[139] Medical history included clinical diagnoses of the central nervous system and visual, vestibular, musculoskeletal, and other body systems. Participants were asked if they took any daily prescribed medications. Participants' pain rating was also collected, but not part of the present analysis. Median household income was determined based on Canadian Census data from 2021 for the postal code of each location of data collection.[140] Household income for each individual participant was not collected. For the data collected in the

laboratory, participants did not necessarily have their households in the postal region of the university campus. **Appendix H** contains these data collection forms.

3.3.4 Data Analysis

3.3.4.1 Neurophysiology

The same analysis pipeline as Data Chapter 1 and Krelove et al. 2025 was applied here.[78] Briefly, raw light intensity data were converted to MATLAB script using open access code from Artinis Medical Systems (oxysoft2matlab) and fNIRS processing toolbox Homer3 was used for signal processing.[83] Optical density data were low-pass filtered at 0.1Hz and a principal component analysis (PCA) applied, then data were converted to concentration (uM) using the Modified Beer-Lambert Law.[30,81,82,84] Concentration was averaged across all eight channels and condition separated. Using Spike2 v10 (Cambridge Electronic Design, Cambridge, UK) each dual-task condition (WD, WO, WOD) was compared to single-task walking (ST) using the modulus function to calculate the absolute area-under-the-curve. Final PFC data was expressed as the O_2Hb modulus ratio for DT:ST walking.

3.3.4.2 Gait Velocity

Gait analysis was performed in the ProtoKinetic Movement Analysis Software. Incomplete footsteps were removed based on visual analysis, and spatiotemporal outcomes computed by the software. Gait velocity was the primary metric of interest due to functional and predictive capacity in aging adults.[52,141,142] As per Data Chapter 1, velocity thresholds at the functional “crosswalk” threshold at 1.0 m/s and general health threshold at 0.8 m/s were used to visually categorize distributions of gait data.

3.3.5 Statistical Analysis

All analyses were run separately for each walking condition and outcome measure (O_2Hb ratio and gait velocity). Descriptive statistics, two-tailed independent samples t-tests, and two-step cluster analyses were run in SPSSv29 (IBM, New York, USA). Mediation analyses were run in custom R code using the 'mediation' and 'medflex' packages with significance set to $p < 0.05$.[143–146] Two-tailed independent t-tests were used to identify any differences in PFC ratio and gait velocity between locations of participation.

3.3.5.1 Two-Step Cluster Analysis

Two-step cluster (TSC) analyses were used for systemic identification of the most important sociodemographic factors in grouping participants. The TSC algorithm takes "two-steps" to categorize data into natural clusters.[147,148] First, a log-likelihood distance measure from the mean is used to assess similarity of participants and group those with similar values into temporary clusters. In step two, the pre-clusters are merged hierarchically to evaluate each possible cluster solution using the Bayesian Information Criterion (BIC). This second step selected the number of clusters that optimally balances model fit and simplicity via the lowest BIC value. Outcomes of TSC reflect natural groupings of the data as there was no preset number of cluster outputs. TSC outcomes are evaluated based on the silhouette coefficient, a summary measure of the cluster cohesion and separation.[149] Coefficients above 0.5 indicate good cluster solution, wherein elements within a single cluster are similar to one another (cohesion) and the different clusters are dissimilar (separated).[148] For each variable, the silhouette is the difference between the smallest average between cluster distance and the average within cluster distance, divided by the larger of the two distances. In a good solution, the within distances are small and

the between cluster distances are large to result in a silhouette measure approaching and close to one.

TSC was applied in this work to 1) determine if participants from different locations cluster together in terms of neurobehavioural outcomes, and 2) determine the most important sociodemographic predictors in this sample. In TSC 1, PFC ratios and gait velocity data were treated as continuous variables in separate analyses per condition and measure. The output was used to identify the number of natural clusters for each measure (PFC ratio or gait velocity) per condition, as well as each participants' cluster membership. For TSC 2, all sociodemographic data were added to the algorithm as either categorical or continuous variables. The data were: age; sex; balance confidence (ABC score); location of participation (on or off campus), physical activity level (yes or no to 30 minutes 5 times/week); medication use (one or more of any prescribed medication taken daily); DT performance (error rate, number of errors/number of responses x 100%); level of education (categorical from public school to post-graduate education); and fall history in the past year (yes or no). It did not change predictor importance outcomes if location of participation was binary on or off campus or separated into the three sites of data collection – community-based participants clustered together in the same groupings.

3.3.5.2 Causal Mediation Analysis

To investigate if PFC activity mediated the relationship between sociodemographic predictors of gait velocity, causal mediation analyses with a counterfactual framework were used. Separate mediation models were estimated for each walking condition independently with gait velocity as the outcome/dependent variable (Y), PFC ratio as the mediator (M), and the strongest cluster predictors as independent variables/predictors (X). Three mediation analyses were run per condition, for a total of 12 analyses per outcome measure and 24 overall mediations.

The mediation pipeline allows for estimation of whether the condition-specific PFC activity mediates the association between a sociodemographic variable and gait velocity; i.e. does the sociodemographic variable influence gait velocity through PFC activity, or is the relationship is direct (Figure 3.1). Mediation analysis uses three regression pathways to decompose the total effect of X onto Y into a direct effect (DE) not operating through M and an indirect effect (IE) that operates through M. This outcome is the cross-product of two paths, the direct of X on M (**Figure 3.1** path a) and the effect of M on Y, controlling for X (**Figure 3.1** path b). Overall, the three steps of the mediation pathway, also shown in **Figure 3.1**, were: Step 1 assessed the total effect, does X predict Y overall, accounting for the mediator?; Step 2 assessed the mediator model, does X predict M? (path a); and finally Step 3 is the outcome model, does M predict Y when controlling for X (path b). Results from Data Chapter 1 suggested that the relationship between PFC activity and gait velocity has a nonlinear trend, as supported by the identification of non-linear change-point ages. Thus, a non-linear form is applied for the mediator outcome relationship in Step 3/path b using a generalized additive model (GAM), detailed in length in **Appendix E**.

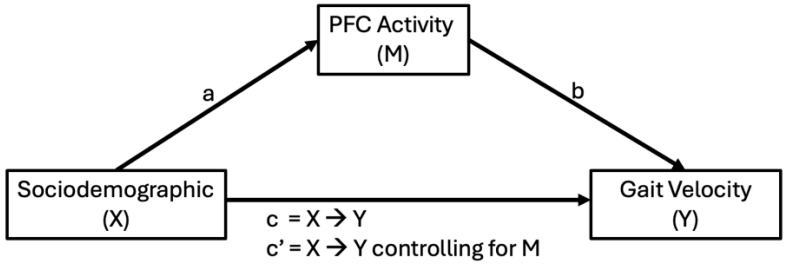


Figure 3.1. Mediation analysis pathway.

Using the outcomes of each regression model, causal mediation effects are estimated. The total effect (TE) was derived from Step 1 to estimate the total effect of X on Y. The indirect effect (IE) was estimated from Step 2 and 3 to estimate the proportion of the TE that occurs through the mediator. In general, the IE assesses the product of derivates of the GAM smoothed functions across each sociodemographic variable (X) range to create sociodemographic varying IE that are averaged across the sample distribution.[150] Statistical significance of the IE was assessed using non-parametric bootstrap resampling (B = 10,000 resamples). For each bootstrap replicate, both mediator and outcome models were re-fit and causal effects recomputed to account for uncertainty in model estimation and nonlinear predictions.[143,144,150,151] The direct effect (DE) is estimated from Step 3 to assess how much of the TE is not through the mediator. The DE and TE are linear models as they are adequately described in linear terms and unnecessary model complexity is avoided while satisfying the assumptions of causal mediation analysis.[144,150,151] Percentile-based 95% confidence intervals were obtained from the empirical bootstrap distributions.[145] Two-sided p-values were calculated as twice the smaller of the proportions of bootstrap estimates above or below zero.[152,153]

Mediator models were estimated using linear regression in the form:

$$M = \alpha_0 + \alpha_1 X + \varepsilon_M \qquad 3\text{-}1$$

And outcome models were estimated as:

$$Y = \beta_0 + \beta_1 X + f(M) + \varepsilon_Y \qquad 3\text{-}2$$

Where $f(M)$ represents a smooth function of PFC activity estimated using penalized regression splines with restricted maximum likelihood (REML) smoothing parameter selection.[151,153] This formulation allowed for nonlinear relationships between PFC activity and gait velocity.

Causal effects were defined using counterfactual outcomes with the potential outcomes framework of causal mediation analysis.[150,154] This approach estimates how outcomes would differ under hypothetical scenarios in which exposure is varied while the mediator is held constant or allowed to vary naturally.[155] Specifically, the IE were defined as the difference between predicted outcomes when X predicts M compared to when the mediator is fixed at the level corresponding to the controlled X condition. DE were defined as the differences in outcomes when X changes while holding M constant. This counterfactual framework allows for the estimation of mediation effects in nonlinear regression models and provides a causal interpretation of IE and DE.[150,154,155]

For continuous predictors, contrasts were defined as the 2.5th and 97.5th percentiles of the observed distribution consistent with counterfactual mediation approaches for continuous exposures.[150,154] This approach allowed for interpretation of mediation effects across meaningful differences in X values while accommodating for nonlinear relationships between M and Y.[150]

For categorical predictors (education and location), contrasts were defined using meaningful category comparisons.[154,156,157] Education level was restricted to levels 2–4 due to sparse representation at level 1, as is recommended for categories with low representation to prevent unstable parameter estimates and unreliable bootstrap interference, and contrasts were defined between level 2 (control) and level 4 (treatment).[158,159] Location of participation was defined at three levels to represent the three locations of data collection and defined between

level 1 (laboratory) and level 3 (CC 2). Contrasts were defined between levels to capture meaningful differences across levels of education and location. Using predicted mediator and outcome values, causal outcomes were computed under counterfactual conditions, wherein $X_0$and X_1denote the control and treatment levels of the predictor variable,[150,154] respectively:

Total Effect:

$$E[Y(X_1, M(X_1)) - Y(X_0, M(X_0))] \quad 3\text{-}3$$

Natural Direct Effect

$$E[Y(X_1, M(X_1)) - Y(X_0, M(X_0))] \quad 3\text{-}4$$

Natural Indirect Effect

$$E[Y(X_1, M(X_1)) - Y(X_1, M(X_0))] \quad 3\text{-}5$$

These effects represent the expected change in gait velocity attributable to changes in X acting through both direct and mediated pathways (TE), only non-mediated pathways (DE), and only mediated pathways through PFC activity (IE).[144,145,160,161]

Thus, the revised mediation steps are as follows in the present analysis.

1. Estimate the effect of each predictor value (X) on gait velocity (Y) using linear regression. Estimates the overall association between X and Y prior to introducing the mediator to answer the question: does the sociodemographic variable (X) predict gait velocity (Y) (Path c)?

2. Estimate the association between each predictor value (X) and PFC activity (M) using linear regression. Represented the extent to which X is associated with changes in M to answer the question: does the sociodemographic variable (X) predict PFC activity (M) (Path a)?
3. Estimate the nonlinear relationship between PFC activity (M) and gait velocity (Y) using GAM fitting and a spline smoothed function of M to answer the question: does M predict Y nonlinearly? A significant non-linear relationship would suggest a mediation effect, IE (Path b, c').
4. Assess the significance of the IE using counterfactual bootstrap estimation of the mediation effects.

3.4 Results

3.4.1 Participant Demographics

Seventy-nine participants aged 50-79 were collected from three locations in the Greater Toronto area. Thirty-nine participants attended data collection sessions in the on-campus laboratory and forty from two off-campus community centers. Community Center 1 (CC 1) had twenty-seven participants and Community Center 2 (CC 2) had thirteen participants. Participant demographics for all participants are presented in **Table 3.1**. For descriptive purposes only, demographics are shown for all three locations and total study population.

Table 3.1. Participant demographics and dual-task performance in the laboratory and each community centre.

	LAB	CC 1	CC 2	ALL
Group size	39 (22F)	27 (26F)	13 (9F)	79 (58F)
Age (years)	60.3 ± 8.7	71.2 ± 5.0	69.2 ± 6.8	65.5 ± 8.8
Height (cm)	167.0 ± 8.4	157.9 ± 8.5	160.8 ± 9.9	162.9 ± 9.6
Weight (kg)	73.7 ± 15.8	67.9 ± 15.3	62.8 ± 14.6	69.9 ± 15.8
Fall history (≤1 year)	7	2	2	11
Activities specific balance confidence	98 (59,100)	88 (56, 100)	96 (62,100)	97 (56,100)
High (>80%)	38	18	10	66
Medium (50-80%)	1	9	3	13
Physical activity (30min 5x/wk)	19	15	13	47
≥1 daily prescribed medication	13	17	10	40
Highest level of education				
Post-graduate	13	0	2	15
Post-secondary	26	12	5	43
Secondary	0	12	6	19
Elementary	0	3	0	3
Medical history				
CNS – concussion	2	0	0	2
CNS – psychiatric	2	2	0	4
CNS – seizure	0	1	0	1
Vision (acuity/cataracts)	5	11	7	23
Vestibular/hearing	7	8	3	18
Musculoskeletal	17	14	9	40
Median household income*	$62,600	$59,300	$94,000	$72,000
Dual-task performance				
Number of responses				
WD	4 (1,5)	2 (1,5)	3 (1,5)	3 (1,5)
WOD	4 (2,5)	3 (0,4)	3 (2,4)	4 (0,5)
Error rate				
WD	11% ± 20%	13% ± 25%	20% ± 40%	13% ± 25%
WOD	14% ± 19%	15% ± 20%	11% ± 22%	13% ± 20%

*LAB = laboratory-based participants, CC = community center 1 and 2. Age, height, weight presented as mean ± standard deviation. Fall history, physical activity, education, medication use, and medical history are expressed as counts. ABC score, number of DT responses, and location-centred household income expressed as median (range). *Median household income based on Canadian census data from 2021 for the location-centered postal code, may not reflect participants' median household income.*

3.4.2 Location-Based Differences & Environmental Familiarity

There was not an a priori hypothesis made for a difference between community and laboratory-based participants, as all participants were within the same age range and met the same study inclusion criteria. PFC activity showed no significant difference between locations of participation (**Figure 3.2 A**). However, contrary to expectations, gait velocity showed a significant difference ($p < 0.01$) across conditions between locations, wherein community-based participants showed a slower velocity (**Figure 3.2 B**). Further, in all but single-task walking (W), average gait velocity for community-based participants were below the 1.0 m/s crosswalk threshold. This may have been due to environmental differences and participant familiarity between locations, or indicate a location difference in sociodemographic drivers of motor performance.

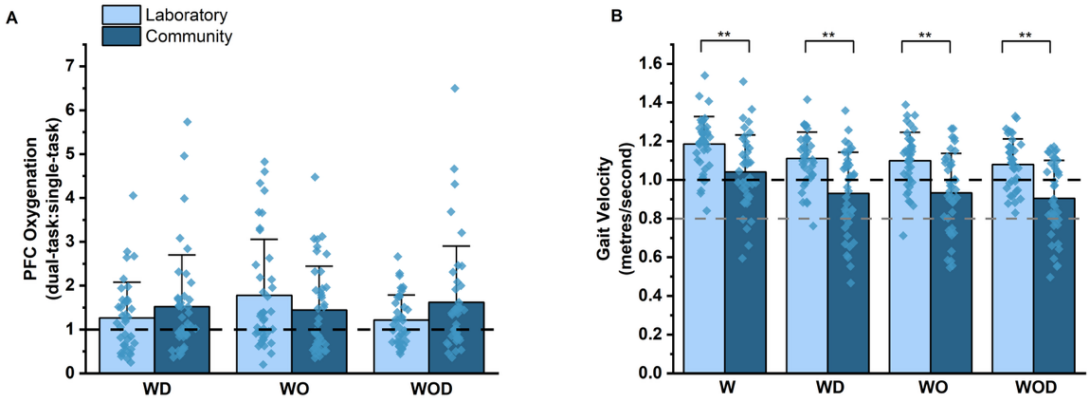


Figure 3.2. Combined bar + scatter plots per condition for laboratory-based (light blue) and community-based (dark blue) participants. **A.** PFC activity expressed as a dual-task:single-task ratio. Dashed horizontal line at 1 indicates level of activity during single task walk (condition W). **B.** Gait velocity (metres/second) across tasks. Dashed black line at 1.0 m/s indicates the functional "crosswalk" threshold, gray dashed line at 0.8 m/s indicates the general health threshold. ** = $p < 0.001$.

To assess if there was an influence of environmental familiarity on neurobehavioural outcomes, TSC 1 was applied. If participants from across locations did not cluster together in terms of PFC activity or gait velocity, then environmental influence may drive the observed differences, or lack thereof. Laboratory-based participants were often visiting for the first time, whereas community-based participants were usually familiar with the environment (i.e. they were regular attendees of activities at the community center). TSC 1 shows that participants from all locations cluster together in terms of PFC activity and gait velocity (**Figure 3.3**), evidenced by circles (laboratory-based participants) and triangles (community-based participants) in the same clusters. Cluster 3 for both PFC activity and gait velocity mainly or entirely consist of community-based participants (**Figure 3.3**), however all other clusters are mixed between locations. Overall, TSC 1 results suggest that environmental familiarity does not drive the differences in gait velocity between locations of participation, nor the similarities in PFC activity (**Figure 3.2**), and suggest that other factors elicit these neurobehavioural differences.

Further, TSC 1 showed that PFC activity identified an outlier group cluster, a cluster with O_2Hb ratio = 1 (i.e. PFC activity during single-task walking), and a group with an upregulation of PFC activity positioned between the other two clusters (**Table 3.2**, **Figure 3.3 A**). All clusters were of good quality (silhouette coefficients > 0.5; **Table 3.2**). The largest cluster for WD and WOD conditions contains those with a PFC ratio ~1, suggesting that most participants were not upregulating PFC activity from ST levels during distraction. However, WO has only two natural clusters, with most participants showing PFC ratio > 1, suggesting that the PFC is upregulated more during obstacle navigation than word-recall, and that the cognitive task was prioritized over obstacle avoidance in WOD. Gait velocity clusters aligned to the functional and general health thresholds at 1.0 m/s and 0.8 m/s with good cluster quality (**Table 3.3**, **Figure 3.3 B**). Across

tasks, the largest cluster was around 1.0 m/s, suggesting that most participants maintained gait velocity. However, the number of participants in clusters below 1.0 m/s and 0.8 m/s increased with task challenge, indicating that more individuals over age 50 fall below functional thresholds with increasing task complexity.

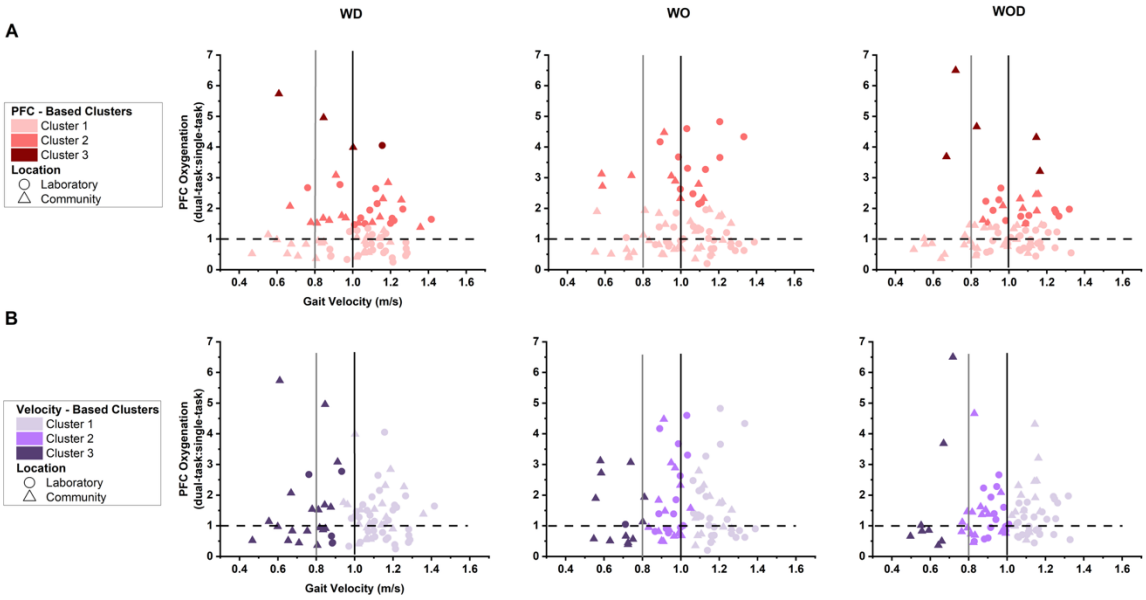


Figure 3.3. Two-step cluster membership for each for each participant per condition for **A.** PFC activity and **B.** gait velocity. Symbol shape indicates location of participation; symbol colour indicates cluster membership. Dashed line at the PFC oxygenation value of 1 indicates single-task PFC activity. Solid black line at 1.0 m/s identifies the functional crosswalk threshold, solid gray line at 0.8 m/s identifies the general health threshold.

Table 3.2. Cluster outcomes and distributions per condition and average PFC activity.

CONDITION	SILHOUETTE COEFFICIENT	TWO-STEP CLUSTER	N	AVERAGE [O_2HB] RATIO
WD	**0.7**	**1**	46	0.8 ± 0.4
		2	29	1.9 ± 0.5
		3	4	4.7 ± 1.0
WO	**0.8**	**1**	21	1.0 ± 0.5
		2	-	-
		3	58	3.2 ± 0.8
WOD	**0.7**	**1**	53	0.9 ± 0.3
		2	21	1.9 ± 0.3
		3	5	4.5 ± 1.3

Table 3.3. Cluster outcomes and distributions per condition and average gait velocity per cluster.

CONDITION	SILHOUETTE COEFFICIENT	TWO-STEP CLUSTER	N	AVERAGE GAIT VELOCITY (M/S)
WD	**0.7**	**1**	55	1.1 ± 0.1
		2	-	-
		3	24	0.7 ± 0.1
WO	**0.7**	**1**	38	1.2 ± 0.01
		2	28	0.9 ± 0.06
		3	13	0.7 ± 0.1
WOD	**0.7**	**1**	41	1.1 ± 0.01
		2	30	0.9 ± 0.01
		3	8	0.6 ± 0.01

3.4.3 Cluster Predictors

TSC 2 revealed the most significant predictors of sociodemographic clusters were location of participation, age, and level of education, each with a predictor importance of at least 50% (**Figure 3.4**). The TSC algorithm identified two natural clusters differentiated by location of participation (**Table 3.4**). Cluster 1 included mostly those who participated in the laboratory, were in the older middle-age range, and had at least post-secondary education. Participants in Cluster 2 participated mostly in the community, were on average older adults, and had levels of education ranging from elementary school to post-secondary. The silhouette coefficient was 0.4, showing fair cluster cohesion and separation.[148,149]

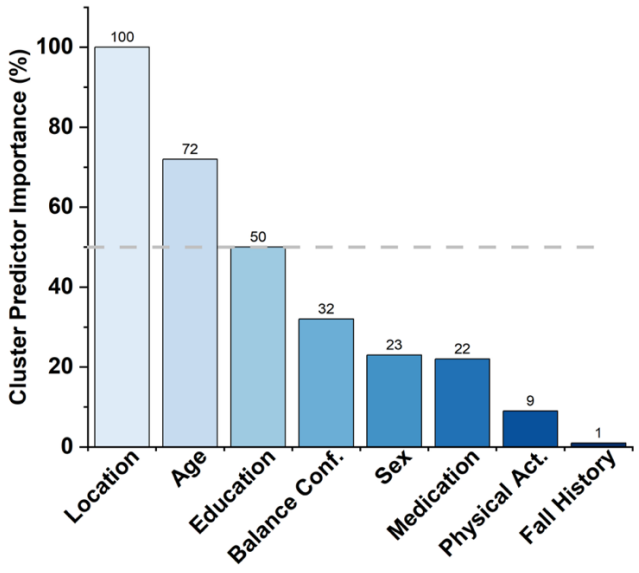


Figure 3.4. Bar plot of sociodemographic cluster predictor importance from 0-100% for each variable.

Table 3.4. Cluster outcomes and distributions per variable with at least 50% cluster predictor importance.

CLUSTER	LOCATION		AGE	LEVEL OF EDUCATION			
	Lab	Community		Elementary	Secondary	Post-Secondary	Post-Graduate
1	95% (37)	5% (2)	59.6 ± 7.9	0%	0%	56% (24)	100% (15)
2	5% (2)	95% (38)	71.2 ± 4.9	100% (3)	100% (18)	44% (19)	0%

Location and level of education are shown as a frequency (%) and number of participants per sociodemographic predictor variable in either Cluster 1 or 2. Age is shown as average ± standard deviation per cluster.

Given that the differences between community- and laboratory- based participants were not due to environmental familiarity, age-group differences, or study inclusion criteria, sociodemographic factors were explored in TSC 2. **Figure 3.5** shows cluster membership based on the three most important sociodemographic cluster predictors. Examination of cluster distributions revealed that the two community-based participants in Cluster 1 were the only two from the community with post-graduate education. The two laboratory-based participants in

Cluster 2 were ~15 years older than the average age of Cluster 1, at ages 73 and 76, and were otherwise both post-secondary educated, active, and of high balance-confidence.

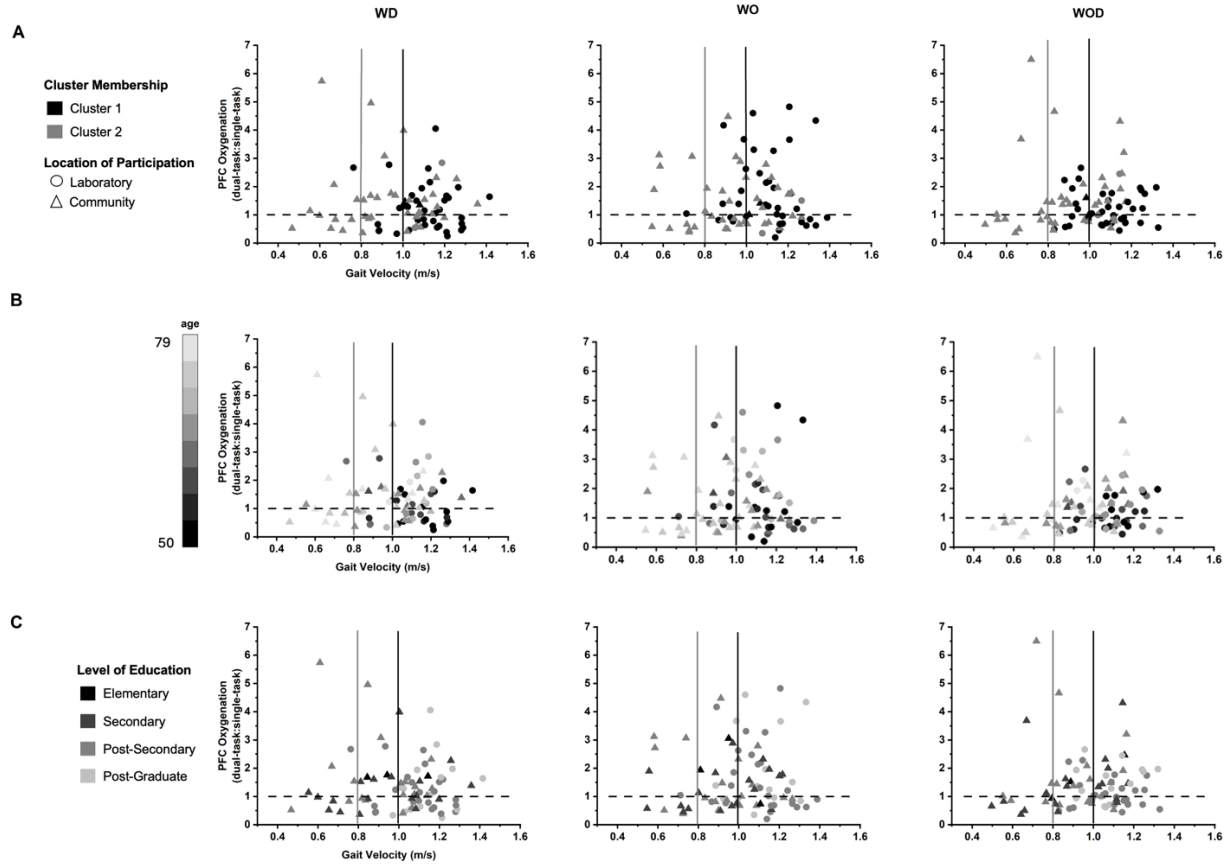


Figure 3.5. Two-step cluster analysis output for location of **A.** participation, **B.** age, and **C.** level of education. Axes show PFC Oxygenation and Gait velocity. Symbol colour describes each demographic factor, symbol shape indicates location of participation (triangles = community-based, circles = laboratory-based). Horizontal lines at 1.0 m/s and 0.8 m/s indicate gait thresholds and dashed horizontal line at 1.0 shows ST level of PFC activity.

3.4.4 Causal Mediation

Mediation analyses were designed to evaluate if age, location of participation, or level of education predict gait velocity and assessed if this relationship is mediated by PFC activity. **Table 3.5** shows regression model fits for the total effect (Step 1; S1), mediator model (Step 2; S2), and outcome model (Step 3, shown as GAM and smooth (SMTH) terms). Since Step 3

employs a non-linear analysis, the GAM R^2 reflects the variance explained by the model, the smooth term reports the estimated degrees of freedom (EDF), F-statistic, and associated p-value describing the nonlinear effect of the mediator modelled using a smoothing function.[151] **Figure 3.6** shows GAM outcomes for each condition and X value, displaying the neurobehavioural relationships. Visual representation of the linear relationships for Steps 1 and 2 can be found in **Appendix F.** Bootstrap resampling distribution plots are found in **Appendix G**.

Table 3.5. Regression model fits for Steps 1-3 per condition and X value.

	X	S1 R^2	S1 F	S1 P	S2 R^2	S2 F	S2 P	GAM R^2	SMTH EDF	SMTH F	SMTH P
WD	Location	0.24	11.89	**<0.001**	0.02	0.68	0.51	0.24	2.03	1.41	0.24
	Age	0.24	24.59	**<0.001**	0.06	4.66	**0.03**	0.27	2.18	1.93	0.13
	Lvl Ed	0.13	5.41	**0.006**	0.01	0.20	0.81	0.16	2.15	2.10	0.11
WO	Location	0.23	11.13	**<0.001**	0.02	0.83	0.44	0.20	1.00	0.17	0.68
	Age	0.23	23.03	**<0.001**	0.01	0.38	0.54	0.21	1.00	0.27	0.61
	Lvl Ed	0.09	3.88	**0.03**	0.04	1.46	0.24	0.06	1.00	0.06	0.80
WOD	Location	0.24	11.85	**<0.001**	0.07	2.64	0.07	0.28	2.21	2.68	0.06
	Age	0.25	26.26	**<0.01**	0.05	4.03	**0.04**	0.31	2.34	3.16	**0.03**
	Lvl Ed	0.13	5.34	**0.01**	0.01	0.08	0.92	0.15	2.16	1.87	0.16

*S1 is the total effect model, S2 is the mediator model, and S3 is shown as the GAM and smooth terms. Values in **bold** font represent statistically significant models ($p \leq 0.05$).*

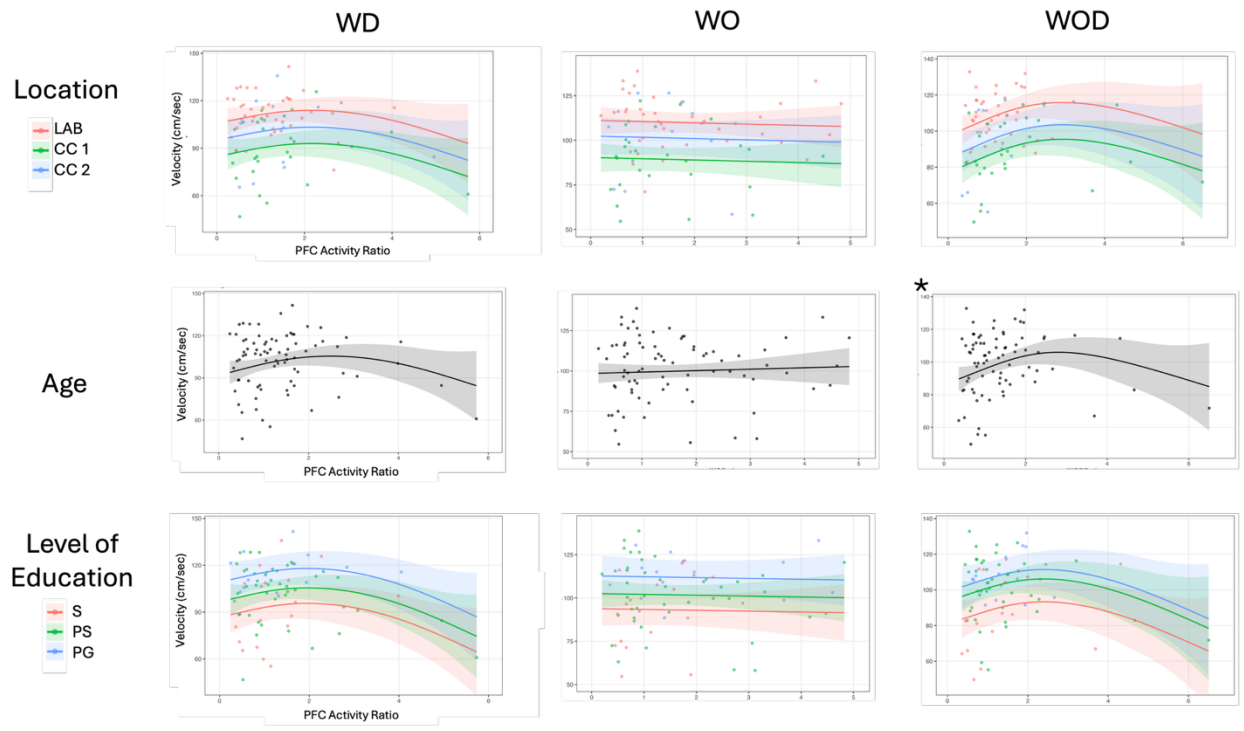


Figure 3.6. Nonlinear GAM-smoothed PFC activity plotted against gait velocity for each condition (column) and sociodemographic variable (row) used as independent variables in mediation. Smoothed fit of PFC activity is plotted against gait velocity with 95% CI range overlayed. * denotes significant nonlinear relationship between PFC activity and gait velocity ($p \leq 0.05$). Level of Education legend: S = secondary; PS = post-secondary; PG = post-graduate.

3.4.4.1 Regression Models

Across sociodemographic (X) values, there was a significant linear relationship with gait velocity in all conditions (**Table 3.5**, Step 1). Thus, location of participation, age, and level of education predict gait velocity in all conditions. The mediator model (**Table 3.5**, Step 2) was only significant for Age in WD and WOD, indicating that age only predicted PFC activity during distraction. The outcome model, which assesses if the direct effect acts through the mediator (PFC activity) is only significant for WOD when controlling for age (**Table 3.5**, Step 3). This suggests that the only significant nonlinear relationship between GAM-smoothed PFC activity and gait velocity was for the most challenging, real-world condition. In this outcome model, age explains 31% of the variance in gait velocity (**Table 3.5**, Step 3 SMTH). All other outcome

models were not significantly significant, but the smooth term EDF values >1 suggest nonlinearity between PFC activity and gait velocity across conditions and independent variables (**Table 3.5**, Step 3 EDF).

3.4.4.2 Mediation Effects

There were significant TE for age and level of education in one or more DT conditions, indicating that not all the important sociodemographic cluster predictor variables also predict gait velocity (**Table 3.6**, TE). Only level of education significantly predicted velocity across all conditions, wherein lower levels of education were associated with slower gait velocity. Being the only variable with significant prediction of behaviour in all conditions suggests that level of education may influence multiple neural mechanisms that support effective and efficient executive and cognitive resources across adulthood. Age predicted gait velocity during obstacle avoidance conditions (WO and WOD) such that increasing age was associated with slower gait velocity. This outcome makes intuitive sense and matches with the change-point ages identified in Data Chapter 1 (**Table 2.3**). Location of participation is a borderline significant predictor of gait velocity in only the most challenging condition, WOD ($p = 0.07$). The results of other analyses show that location of participation was a ~100% cluster predictor in TSC and a significantly slower gait velocity across conditions for community-based participants, suggesting that location as a sociodemographic summary variable is descriptive rather than predictive.

The DE for age and level of education remained significant after accounting for the mediator, PFC activity, during all conditions, suggesting that the sociodemographic differences in gait velocity are independent of the PFC (**Table 3.6**, DE). The IE indicate limited evidence for mediation by the PFC on the relationship between age and gait velocity in WOD (**Table 3.6**, IE $p = 0.05$); however, because the 95% CI includes zero, this effect must be interpreted with

caution. This emerging significant mediation may indicate that the mediator is related to changes in gait velocity rather than a mediator of the age-effect. Indeed, the Step 3 regression model for WOD age was significant (**Table 3.5**), however, indicating that the mediator is significantly nonlinearly related to gait velocity but is not a full mediation. Thus, PFC activity is likely related to gait velocity in WOD when controlling for age but does not transmit or mediate the significant age-related DE. Taken together, mediation analysis suggest that sociodemographic factors predict gait velocity without mediation by the PFC.

Table 3.6. Mediation effects across conditions and sociodemographic X-values.

X	IE	95% CI	P	DE	95% CI	P	TE	95% CI	P	PM
WD										
Location	1.00	[-2.49,5.79]	0.64	-10.63	[-23.87-2.39]	0.10	-9.62	[-23.36,-3.06]	0.13	-0.10
Age	2.28	[-0.68,6.89]	0.14	-17.15	[-23.33, -10.69]	**<0.001**	-14.87	[-21.16, -8.27]	<0.001	-0.15
Lvl Ed	0.42	[-3.26,4.28]	0.84	22.4	[9.87,35.07]	**<0.001**	22.82	[9.42,36.18]	**0.001**	-0.02
WO										
Location	0.26	[-2.09,4.60]	0.77	-8.81	[-21.79,4.05]	0.20	-8.54	[-21.36,4.68]	0.23	-0.03
Age	0.12	[-1.67,1.63]	0.88	-16.17	[-21.57,-10.40]	**<0.001**	-16.05	[-21.79,-10.26]	**<0.001**	-0.01
Lvl Ed	-0.34	[-10.00,3.85]	0.82	18.9	[6.63,30.94]	**0.001**	18.57	[4.04,30.12]	**0.02**	-0.02
WOD										
Location	0.68	[-3.82,5.76]	0.75	-12.34	[-24.53,-1.11]	**0.03**	-11.66	[-25.5,0.67]	0.07	-0.06
Age	3.27	[-0.01,8.77]	**0.05**	-16.68	[-21.65,-11.52]	**<0.001**	-13.41	[-19.13, -7.10]	**<0.001**	-0.24
Lvl ed	0.29	[-3.09,4.77]	0.82	18.10	[7.26,29.28]	**<0.01**	18.39	[7.07,30.69]	**<0.01**	0.02

*IE is the indirect effect, DE is the direct effect, TE is the total effect. PM = proportion mediated (IE/TE), negative PM values suggest inconsistent mediation. Values in **bold** font represent statistically significant models ($p \leq 0.05$).*

3.5 Discussion

This work aimed to identify the relationship between sociodemographic factors, PFC activity, and gait velocity during complex gait DT in adults aged 50-79. Data were collected in either the laboratory or community and there was an unexpected difference in gait velocity between locations of participation separate from environmental influence. To investigate the potential drivers of this difference, two-step cluster analyses of sociodemographic variables

revealed that that location of participation, age, and level of education were the most important cluster predictors. Mediation analyses identified predictive relationships between age and level of education for gait velocity, and that this relationship was not mediated by the PFC. This work highlighted that sociodemographic factors may influence the extent to which the PFC can compensate for decline in locomotor control in older adulthood.

3.5.1 Slow gait velocity in community-based participants

It was expected that neurobehavioural outcomes would be homogenous across participants, as there were consistent inclusion criteria and participant age-ranges between locations. However, there was a significant difference in gait velocity between locations of participation not driven by environmental familiarity. Sociodemographic analyses in TSC 2 showed that location of participation differentiated clusters and directed the proceeding interpretations. Community-based participants were significantly slower walkers than laboratory-based participants across obstacle navigation and distraction conditions (**Figure 3.2 B**). Community-based participants also showed average gait velocities below the functional crosswalk threshold of 1.0 m/s in all conditions and made up the majority of those below the general health indicator of 0.8 m/s (**Figure 3.2 B**). This result is not uncommon, as many older pedestrians are unable to successfully use a crosswalk due to the average walking speed in OA being below that required to cross at a metropolitan crosswalk.[127] Over 80% of males and females over age 65 walk too slowly to navigate a crosswalk safely, showing average gait speeds of 0.9m/s in males and 0.8 in females.[127] Thus, these results are in line with the literature and suggest overall that urban infrastructure may not match the needs of aging adults.[53]

Examining the neurobehavioural correlates of DT from a cognitive perspective shows the community-based participants' range of DT responses was wider and included zero in WOD

without reaching the maximum of five responses (**Table 3.1**). This suggests that community-based, but not laboratory-based, participants prioritized gait during DT which, alongside slow gait velocities, indicates potentially ineffective neural compensations for locomotor decline and limited EF resources. Indeed, location of participation was the only cluster predictor with >50% importance in TSC 2 but did not predict gait velocity in mediation analyses (**Table 3.5** and **Table 3.6**). This result suggests that location of participation was descriptive of participant's current mobility status rather a predictor of future mobility behaviour. Potentially, this is due to the older age of the community-based participants and already slower gait velocity, indicating that predicted locomotor decline may not have been as large as that for the younger laboratory-based participants. Location of participation may be a summary variable for a collection of sociodemographic differences between locations and possibly be a proxy measure for SES factors not collected in this study.

3.5.2 Sociodemographic Cluster Predictors

Location of participation, age, and level of education were the strongest cluster predictors with at least 50% importance each (**Figure 3.4**). Location, as discussed, may be an informative summary descriptor capable of indicating several key SES variables that were not collected in this study including race, ethnicity, household income, and current/prior occupation. In this work, a higher level of education predicted faster gait velocity and increasing age predicted slower gait velocity. Having less than post-secondary education is an independent predictor of mobility-related disability, slower gait velocity, and poorer functional capacity.[134,162–165] Education, which is the most impactful when obtained during early adulthood,[125] may modify the neural regions and networks critical to maintaining mobility with age by increasing cognitive capacities and compensation potential.[163,166,167] SES and sociodemographic differences can

further influence neurocognitive aging trajectories throughout adulthood and disparities in SES become the most prevalent in health and mobility outcomes during middle- and early-old age.[168]

Interestingly, balance confidence (ABC score), physical activity, and fall history were not cluster predictors despite being established predictors of PFC activity and gait velocity in mobility contexts.[169–173] This may be due to the generally high balance confidence in this sample (**Table 3.1**), and suggests those with moderate ABC scores, mostly community-based participants, did not differ in neurobehavioural measures to create a lower balance confidence cluster in TSC 1 or 2. Further, the ABC Scale is influenced by ceiling effects and self-report biases, potentially limiting its efficacy in independently mobile adults.[138] Along similar lines, fall history was not an important cluster predictor despite being the number one predictor for future falls, which may be attributed to overall low fall occurrence across the sample. Other predictors of falls, such as medication use and medical history, were also not identified as significant cluster predictors. Medication type was unspecific in this analysis, and results may have been more insightful and further investigation into type of medications, such as those for cardiac conditions, may provide further insights. Sex did not predict gait velocity, despite females often showing slower gait velocity and worse mobility outcomes with age. The predominantly female sample likely accounts for the absence of an effect of sex.[7,64,134,163,170]

3.5.3 PFC Activity is Compensatory, not Mediary

Unexpectedly, PFC activity did not mediate the relationship between sociodemographic factors and gait velocity. The emerging significant IE of age in WOD (**Table 3.6**) suggests that upregulation of the PFC was related to the decline in gait velocity, potentially as a compensatory mechanism, but the age-effect was not through PFC mediation. Further, the negative proportions mediated (**Table 3.6**, **PM**) suggest that the mediation pathway (if it was significant) would be in

the opposite direction to the DE. This would suggest, for example, that increasing age is associated with slower gait velocity, so an increase in PFC activity indicates faster gait velocity. PFC activity during complex gait DT may thus reflect compensatory use of executive locomotor control.[12,23,47,77]

However, PFC activity did not increase or otherwise "respond" to low behavioural performance in community-based participants. Higher PFC activity in community-based participants would have reflected increased executive resources and attention to maintain gait performance in attempted compensation. The observed lack of neurophysiological differences despite slower gait velocity may suggest that PFC upregulation is not the only neural response to altered attentional priority and reduced behavioural performance. Across the literature, there is not a consistently observed upregulation of the PFC with age in a balance control context, there is evidence for increased, decreased, and no change in PFC activity in OA.[30,47,77,174] From this perspective, the lack of difference in PFC activity between locations of participation is reasonable, as participants were in the same age grouping and free from pathology. Unmeasured components of the motor control network, such as the parietal cortex, may have a larger role than the PFC in compensating, driving, or mediating relationships related to the observed decline in gait velocity in community-based participants.[175,176] On the other hand, the absence of PFC engagement in response to gait velocity decline may indicate that non-executive regions are unable to effectively support DT behaviour.[42,177] In either scenario, the low PFC engagement and slow gait velocity may suggest either under-recruitment of the PFC or ineffective neural scaffolding that resulted in inability to engage executive resources and support behavioural performance under complex task demands.[30,178]

3.5.4 Sociodemographic Factors Influence Executive Compensation

The theory of cognitive reserve provides grounding for the mismatch observed between brain aging/pathology and clinical symptoms.[41] Life experiences such as education, occupation, and leisure activities tend to strengthen reserve and delay decline in EF.[33,179–181] Higher cognitive reserve results in more efficient, flexible, and higher-capacity cognitive networks better able to cope with age related changes.[179] Based on TSC 2, community-based participants were older and less educated than the laboratory-based adults, both non-modifiable factors associated with lower cognitive reserve, reduced EF, and lower cognitive capacity.[179,180] It is possible that the observed results were due to lower cognitive reserve in community-based participants compared to laboratory-based participants. The higher education level of laboratory-based participants may have resulted in the higher DT performance and maintained gait velocity indicative of effective and efficient use of PFC resources. Conversely, the lower level of education in community-based participants may have resulted in fewer cognitive resources available to support behavioural maintenance during DT. Indeed, slower gait velocities have shown to predict future mobility impairment only in those with low cognitive reserve, suggesting that higher reserve can compensate for slower walking ability and is protective against mobility impairment.[182]

Cognitive reserve has shown to moderate the change in PFC activity measured by fNIRS during DT such that higher cognitive reserve is associated with smaller increases in PFC oxygenation in DT walking.[34] This suggests that higher cognitive reserve is associated with less PFC upregulation and executive reliance with age. However, the absence of PFC upregulation in community-based adults despite impaired behavioural performance may suggest that cognitive reserve is influenced by the efficacy of neural networks.

Cognitive reserve offers a non-PFC mechanism for the effects of sociodemographic factors on gait velocity, supported by the revised scaffolding theory of aging and cognition (STAC-r), which postulates functional neural correlates of reserve.[42,179] The original STAC attributed differences in cognitive function with age to a range of adverse biological and neurophysiological factors and their interaction with protective factors, representing the brain as a dynamically adaptive structure.[103] Resultant cognitive functioning is a cumulative outcome of negative and positive processes, referred to as "compensatory scaffolding", that attempts to counteract the adverse effects of neural and functional decline, such as through increased PFC activation.[42] Thus, the extent to which the PFC is able to compensate for impaired subcortical locomotor control using executive resources relies on the extent and efficiency of neural scaffolding that supports cognitive reserve.

The consideration of the impact that non-biological factors have on neural structure and cognitive function (i.e. the 'r'evised version of STAC) highlights the importance of life experiences on brain aging and behavioural outcomes.[42] High levels of education (post-secondary or higher), physical activity, and other executive resource-promoting experiences may directly preserve brain function and increase capacity for compensatory scaffolding.[42,179] Higher levels of education increase dlPFC connectivity with memory centres, such as the hippocampus, providing a potential mechanism for retained EF with age through greater cognitive reserve and compensation capacity.[136] This suggests, under STAC-r, that sociodemographic factors influence the ability to build cognitive reserve through compensatory scaffolding. Further, the lower cognitive reserve in community-based participants may explain their lower DT performance and inability to maintain gait velocity without PFC mediation or upregulation. The ineffective neural compensation in community-based participants may be a result of less efficient neural networks,

reduced scaffolding, and overall lower cognitive reserve. Thus, sociodemographic diversity across the study sample that predict behavioural outcomes supports that non-biological components have a role in the extent of PFC and executive compensations to maintain mobility throughout adulthood.

3.5.5 Limitations

This study is not without limitations. First, the sociodemographic information collected were incomplete to make assessments of SES. Race, ethnicity, current/prior occupation, and other fundamental SES variables were not obtained and limit the conclusions that can be made. Similarly, medication history was not thoroughly examined, and conditions such as hypertension, diabetes, or arthritis have shown to predict and alter gait velocity and mobility outcomes.[58,59,137,183] This sample was also predominantly female and with little to no history of falls. Future studies in the community should focus on equal biological sex recruitment and expand to those with recent fall histories. Finally, the neural signals were limited to only the PFC due to hardware limitations. It is assumed that the majority of signals are from the dlPFC; however, this region is not a specific anatomical structure but rather a functional region, so signals may be from more global PFC regions than solely the dorsolateral aspect.[137]

3.5.6 Conclusions

This work showed that in healthy adults age 50-79, neurobehavioural and mobility outcomes differs between those able to access laboratories and those who participate in the community. Further, age, level of education, and location of participation predict dual-task gait velocities through pathways not mediated by the PFC. Rather, the extent to which the PFC can compensate for age-related mobility changes using executive resources is influenced by sociodemographic factors that alter cognitive reserve. Thus, these results show that the PFC is

part of a compensatory parallel process to the functional neural correlates of mobility decline with age. Taken together, this study highlights the importance of including sociodemographic factors on neurobehavioural correlates of mobility in aging adults.

Chapter 4 General Discussion

This thesis aimed to identify the age at which neurobehavioural outcomes during complex walking tasks first deviate from YA levels and to probe the sociodemographic factors that can influence neurobehavioural correlates of mobility. An experiment involving complex gait DT (virtual obstacle avoidance and word-recall) involving 113 individuals age 18-79 was designed to determine the window during which neurobehavioural correlates of DT change from YA levels in reflection of altered attentional prioritization. This work also assessed whether this change is influenced by sociodemographic factors. fNIRS was used to measure PFC activity as an indicator of executive resource allocation and attention, and a pressure sensitive walking mat was used to capture spatiotemporal gait metrics with a focus on gait velocity.

The first manuscript, "Prefrontal Cortex Activity and Gait Velocity Through Adulthood: Shifts in Neurobehavioural Markers of Mobility Begin in Mid-Life" (Data Chapter 1), showed that PFC activity and gait velocity significantly deviate from young-adult patterns at ages 52-56 across conditions, suggesting that changes in attention priority during DT, cognition, and mobility begin during mid-life. These results indicate that increased PFC activity is an immediate compensation for alterations in gait velocity. The second manuscript, "Neurobehavioural and Sociodemographic Predictors Of Mobility in Adults 50-79 Years of Age" (Data Chapter 2), showed that location of participation, age, and level of education are the most important sociodemographic descriptors for the subset of participants aged 50-79, and that these factors significantly predict gait velocity without mediation by the PFC. The efficacy of executive compensation may be partly determined by the influence of sociodemographic factors on the extent of neural scaffolding and cognitive reserve that can be established. Taken together, these findings suggest that changes in the neurobehavioural correlates of DT capacity in relation to

mobility begin during mid-life, and that these mobility changes can be influenced and predicted by sociodemographic factors.

4.1 Neurobehavioural Prediction of Mobility

The combination of findings that altered neurobehavioural DT capacity begins in the 50s and that age predicts gait velocity without mediation by the PFC supports that increased age is associated with behavioural decline, increased executive reliance, and altered attentional priority. Theoretically, upregulation of the PFC in compensation for age-related locomotor and executive changes aims to increase the use of executive resources to maintain behavioural performance.[23,31] However, the functional interpretation of this change in PFC activity is based on corresponding behavioural outcomes.[33] In this work, PFC upregulation is considered a successful compensation when behavioural performance is improved or maintained ($\geq$ 1.0 m/s), but is an attempted, unsuccessful compensation when behavioural performance declines (<1.0 m/s).[33] PFC activity and the corresponding gait velocities in the most complex condition, WOD, can be used to create two axes to assess participants' functional mobility status. Using the PFC single-task ratio threshold of 1.0 and the functional gait velocity threshold at 1.0 m/s, quadrants are developed to categorize participants into theoretical functional mobility domains based on PFC activity and gait velocities (**Figure 4.1**).

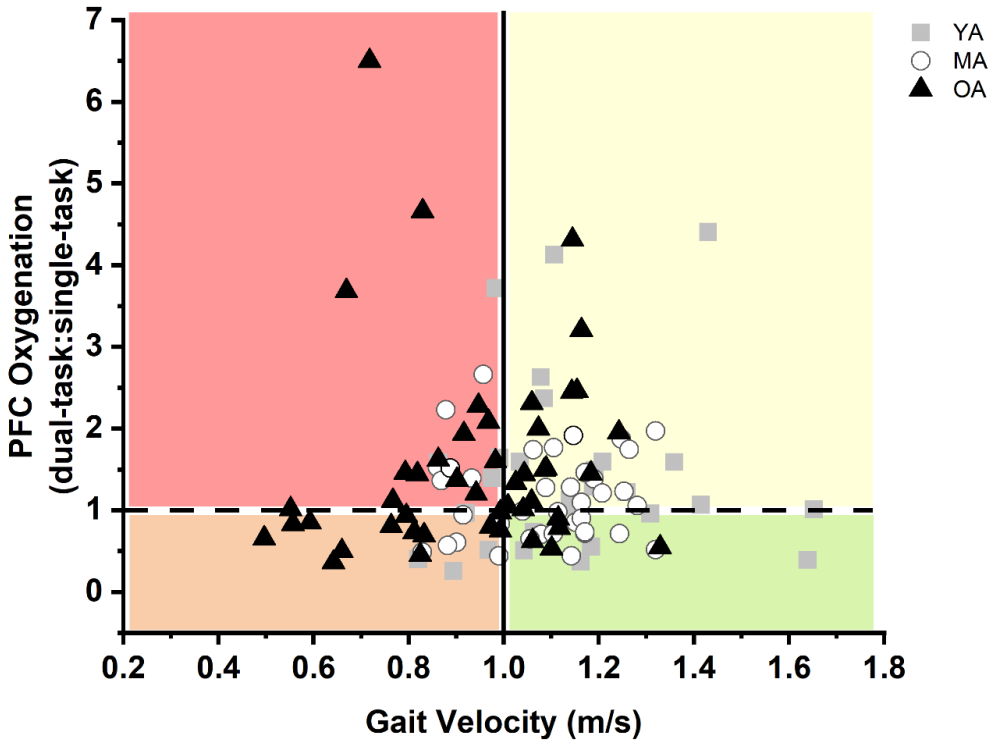


Figure 4.1. Functional neurobehavioural framework to identify mobility capacity. Functional gait threshold indicated at 1.0 m/s (solid black line), PFC recruitment threshold at a PFC oxygenation ratio of 1.0 (dashed black line). YA age group shown as gray squares, MA as white circles, and OA as black triangles.

Interpretations of associations between quadrants and functional mobility domains are hypothesized as follows. The green quadrant (lower right) identifies individuals who are maintaining mobility without PFC upregulation during DT. In this quadrant, gait velocity is maintained at or above 1.0 m/s and PFC activity is at or below single-task levels, suggesting no executive upregulation is required to maintain gait velocity. This suggests optimal neural efficiency and reflects automatic control of locomotion and low EF reliance.[66,184,185]

In the yellow quadrant (top right), gait velocity is maintained above 1.0 m/s with an upregulation in PFC activity above single-task level, suggesting an increased requirement for EF

and compensatory recruitment of the PFC. This is a successful compensation, as gait performance is maintained.[33] The additional cortical and executive resources allow for behavioural performance to be maintained in the face of physiological and task related challenges, aligned with frameworks of age-related compensation, such as CRUNCH and PASA, and increased reliance on bilateral frontal control for locomotion.[25,37,101,186] Conversely, this increase in PFC activity above single-task levels may be the optimal response to DT, and the green (lower right) quadrant instead reflects a lack of task engagement leading to the same or lower PFC activity as single-tasks. However, this is an unlikely explanation when considering PFC upregulation as adaptive compensation, and increased PFC oxygenation during dual-tasks has shown to uniquely predict falls in high functioning adults.[38,101,186,187]

The orange (lower left) quadrant contains those who may be exhibiting cautious mobility behaviour. In this quadrant, participants are not maintaining gait velocity, but are also not upregulating the PFC. This slow velocity without PFC increase suggests use of alternate compensatory pathways, and the PFC remains as an additional, not yet used, compensation. Thus, this quadrant may contain individuals who are not yet at an immediate risk of mobility decline, as PFC compensatory pathways have not yet been recruited. To this point, most of the orange quadrant are participants in MA and OA age-groups, reinforcing that these are individuals who may be cautious about their stability. Alternatively, this lack of PFC "response" to slow gait velocity may indicate less EF resources available to direct attention, possibly due to low cognitive reserve and inefficient scaffolding.[38,42] Low PFC activation and impaired gait may reflect under-recruitment and suggest a lack of ability to engage with the necessary neural resources to support locomotion under cognitive load resulting in a limited capacity for

compensatory upregulation.[30,178] These individuals may benefit from mobility and fall prevention intervention to improve gait velocity under cognitive load.[7,188]

Finally, the red (upper left) quadrant may identify those with low functional mobility. The slow gait velocity with high PFC activity suggests attempted, maladaptive compensation, wherein the increase in EF and attention cannot assist in maintaining motor output and reflects neural inefficiency.[29,31] The compensatory PFC recruitment is insufficient to offset the decline in DT and mobility capacity, a common pattern in OA and individuals with low cognitive reserve during DT walking.[34,47] These individuals may be at risk of mobility decline and falls, intervention is necessary to prevent immediate falls and attempt to prevent further decline.[52] Both the slow velocity quadrants (red and orange) contain the majority of participants over age 50 (MA and OA), and the red quadrant predominantly includes participants over age 65 (OA only). This distribution of age supports that the orange quadrant contains those who still have capacity to improve behaviour through executive compensation compared to those in the red quadrant who require intervention to prevent a fall or progressive decline.

Taken together, this proposed neurobehavioural mobility framework highlights that changes in DT capacity across the adult lifespan reflects not only differences in gait performance, but also heterogeneity in the neural strategies used to support locomotion and daily-life mobility. Similar behavioural outcomes can arise from distinct neural states, such as maintained functional gait velocity arising from efficient locomotor control or compensatory PFC recruitment, and slow gait velocity can emerge from inefficient compensatory activity or insufficient engagement of executive resources. This highlights that neural and behavioural outcomes must be interpreted together, and neurobehavioural relationships are valuable for characterizing functional mobility status.

By situating participants within this framework, there is a basis to identify early deviations from optimal neuorbehavioural responses to DT, distinguish adaptive and maladaptive PFC compensations, and improve early detection of future mobility-related decline. These approaches are essential during mid-life, where subtle shifts can be recognized before overt behavioural impairment manifests, offering a window for early detection and intervention. From the perspective of an aging population who prioritize remaining in their communities, there needs to be consideration of capacities of older adults. This includes crosswalking timing for safe and successful crossing, such as the adjustment to 1.0 m/s in the City of Toronto as an update to the prior requirement of 1.2 m/s to accommodate a wider range of pedestrian functional capacities.[53,54] However, the results of this work and of others suggest that this reduction to 1.0 m/s may still be insufficient to accommodate community ambulation in an aging population. Future work should focus on the validation of functional neurobehavioural relationships, such as the ones proposed here, for intervention recommendation and focus efforts specifically on mid-life intervention and incorporate results into societal and community-based considerations for an aging population.

4.2 Revisiting the Conceptual Model

The functional neurobehavioural framework presented here is not developed purely from physiological changes, nor is the change in DT capacity and mobility with age uniform across individuals. These neurobehavioural relationships, as supported by Data Chapter 2, are impacted by sociodemographic factors that influence cognitive reserve, neural scaffolding, and the availability of executive resources.[34,41,182] Increasing age and level of education strongly predict gait velocity in this work and directly influence cognitive reserve, and might also play a role in when neurobehavioural changes in DT capacity change from YA patterns. Thus, the range of

mid-life inflection points for PFC activity and velocity from Data Chapter 1 may reflect participant heterogeneity in terms of cognitive reserve capacities as they manifest at a neural and behavioural level. Given these results, the original conceptual model presented in the Chapter 1 (**Figure 4.2 A**) has been revised to include the mid-life deviation in neurobehavioural relationships from YA levels (**Figure 4.2 B**, # 1) and the impact of sociodemographic factors on cognitive reserve and how this influences neurobehavioural relationships (**Figure 4.2 B**, # 2).

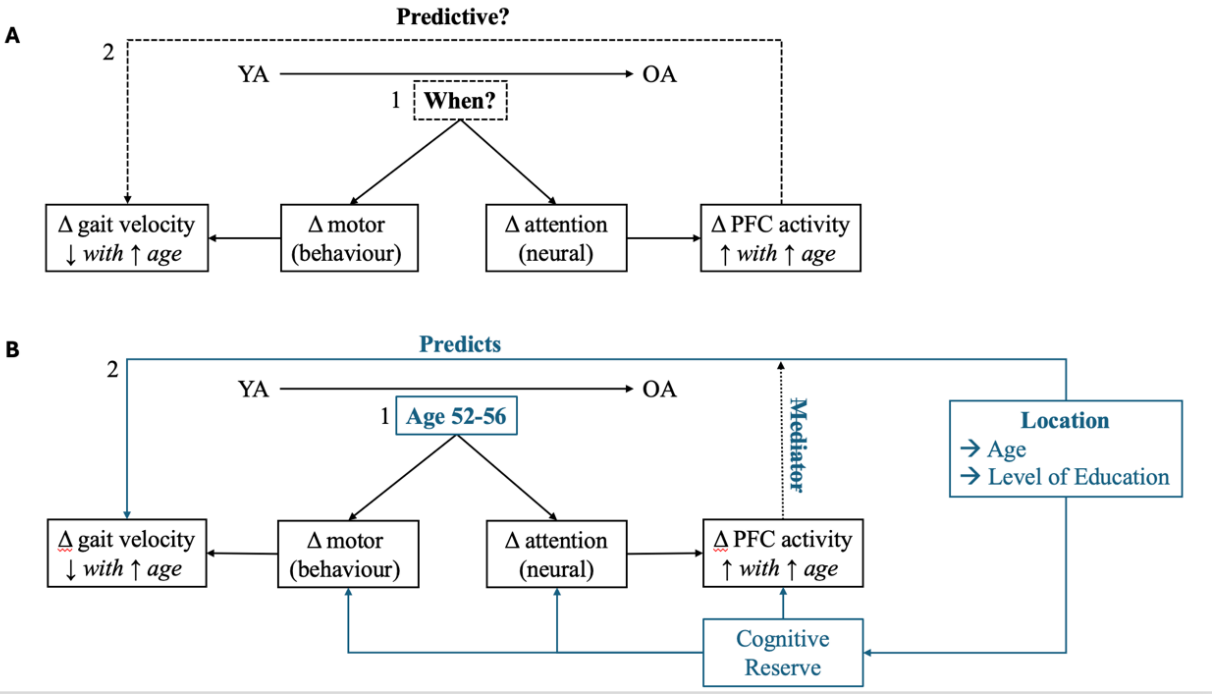


Figure 4.2. A. The conceptual model used to frame this thesis. Solid lines represent established relationships used to construct the initial framework, dashed lines indicate relationships that were investigated in this thesis. Box 1 and 2 represent the main research questions of each Data Chapter. **B**. The revised conceptual model incorporating the findings from this thesis. Solid black lines represent relationships that persist from the original model. Solid blue lines indicate revisions to the initial model, boxes 1 and 2 show the primary result from each Data Chapter.

Probe # 1 in **Figure 4.2 A** (original model) and **B** (revised model) highlight the mid-life inflection points identified in Data Chapter 1. In mid-life there is significant deviation from

linear, YA patterns of PFC activity and gait velocity. At the inflection point, PFC activity begins to significantly increase and gait velocity decreases, suggesting the onset of altered DT capacity, attention prioritization switch for a "posture-first" approach, and emergence of a variety of neural engagement strategies in the face of physiological aging.[11,189] Probe #2 identifies the significant linear regression result between age, level of education, and location of participation and gait velocity, showing that these sociodemographic variables predict behaviour.

The inclusion of sociodemographic factors is the most impactful change to the conceptual model. In the initial model, sociodemographic factors were not highlighted as drivers or predictors of neurobehavioural outcomes in a DT context and instead focused on the predictive or mediatory capacity of the PFC within mediation analyses. Results of mediation analyses (**Table 3.5** and **Table 3.6**) showed that across conditions, sociodemographic variables identified by TSC 2 as important cluster predictors (**Figure 3.5**, **Table 3.4**) strongly predict gait velocity without evidence for mediation by the PFC. Further, TSC 2 showed that location of participation had ~100% predictor importance (**Figure 3.4**) but did not predict gait velocity in mediation analyses, thus location of participation was considered a descriptive summary variable that may indicate a cumulative sociodemographic difference between locations. Primarily, this difference is such that community-based participants were of an older age and lower level of education than laboratory-based participants. Thus, the revised model shows age and level of education as sub-points of location of participation on the right side of the revised figure.

In terms of the predictive capacity of the PFC for gait behaviour, results of mediation analyses (**Table 3.5** and **Table 3.6**) suggest that PFC is an immediate compensatory region that varies in its efficacy across individuals, not a predictor of gait velocity. There was only an emerging mediation effect of the PFC in one condition for one sociodemographic variable, and

PFC activity did not show a significant non-linear relationship to gait velocity in most mediation analyses. Thus, it appears that the PFC is a parallel compensatory process that varies in its efficacy across individuals. The variance in the utility of increasing executive resources through PFC upregulation during DT may be driven, in part, by the impact of sociodemographic factors on neurobehavioural relationships through their contribution to cognitive reserve and neural scaffolding.[41,42,165,182] In the revised model, this result is summarized by the arrow from sociodemographic variables to cognitive reserve. Each sociodemographic factor has shown to influence the establishment of reserve throughout adulthood.[33,41,129,190] Neural scaffolding functionally supports reserve and can influence the extent to which attention and executive function can compensate for motor and cognitive changes with age.[38,42] The additional components of the revised model (blue) show that the PFC contributes to an immediate compensatory process that begins when gait velocity declines in mid-life and does not mediate the predictive relationship between sociodemographic factors and gait velocity. Taken together, the deviation from YA levels of neurobehavioural correlates of DT capacity that occur in the Fifties can be influenced by sociodemographic factors that contribute to cognitive reserve and partially determine the extent to which executive resources can compensate for motor decline.

4.3 Final Conclusions

This thesis aimed to identify the window during adulthood when neurobehavioural correlates of DT capacity diverge from YA levels and identify sociodemographic predictors of this neurobehavioural relationship. The outcomes from this work show, for the first time, that changes in attention, executive function, and mobility begin in mid-life and that dynamic cortical engagement and resource utilization related to mobility occurs throughout adulthood. Further, this neurobehavioural change is influenced by sociodemographic factors through their

relationship with cognitive reserve, reinforcing that mobility outcomes are heterogenous across the population and not only dependent on age. Overall, outcomes of this work recommend that fall prevention and mobility support efforts start for adults in their 50s and that these interventions should be sufficiently flexible for individuals at different levels of cognitive reserve. This shift away from only providing older adults with balance, gait, and mobility intervention with an understanding of different the neural recruitment approaches allows for more effective fall prevention and encourages lifelong mobility. In summary, this work aimed to identify “tomorrow” problems throughout adulthood and provide a fundamental neurobehavioural foundation to address them today.

References

1. Canada, P. H. A. of. (2024, February 20). *Surveillance report on falls among older adults in Canada*. https://www.canada.ca/en/public-health/services/publications/healthy-living/surveillance-report-falls-older-adults-canada.html

2. Canada, P. H. A. of. (2014, April 10). *Seniors' Falls in Canada: Second Report*. https://www.canada.ca/en/public-health/services/health-promotion/aging-seniors/publications/publications-general-public/seniors-falls-canada-second-report.html

3. Löfqvist, C., Granbom, M., Himmelsbach, I., Iwarsson, S., Oswald, F., & Haak, M. (2013). Voices on Relocation and Aging in Place in Very Old Age—A Complex and Ambivalent Matter. *The Gerontologist*, *53*(6), 919–927. https://doi.org/10.1093/geront/gnt034

4. Peeters, G., Schoor, N. M. van, Cooper, R., Tooth, L., & Kenny, R. A. (2018). Should prevention of falls start earlier? Co-ordinated analyses of harmonised data on falls in middle-aged adults across four population-based cohort studies. *PLOS ONE*, *13*(8), e0201989. https://doi.org/10.1371/journal.pone.0201989

5. Brown, R. T., & Covinsky, K. E. (2020). Moving prevention of functional impairment upstream: Is middle age an ideal time for intervention? *Women's Midlife Health*, *6*(1), 4. https://doi.org/10.1186/s40695-020-00054-z

6. Talbot, L. A., Musiol, R. J., Witham, E. K., & Metter, E. J. (2005). Falls in young, middle-aged and older community dwelling adults: Perceived cause, environmental factors and injury. *BMC Public Health*, *5*, 86. https://doi.org/10.1186/1471-2458-5-86

7. Harmon, K. J., Hakenewerth, A. M., Waller, A. E., Ising, A., & Tintinalli, J. E. (2019). Begin risk assessment for falls in women at 45, not 65. *Injury Prevention*, *25*(3), 184–186. https://doi.org/10.1136/injuryprev-2018-042875

8. Adams, M., Gordt-Oesterwind, K., Bongartz, M., Zimmermann, S., Seide, S., Braun, V., & Schwenk, M. (2023). Effects of Physical Activity Interventions on Strength, Balance and Falls in Middle-Aged Adults: A Systematic Review and Meta-Analysis. *Sports Medicine - Open*, *9*(1), 61. https://doi.org/10.1186/s40798-023-00606-3

9. Kuh, D., Karunananthan, S., Bergman, H., & Cooper, R. (2014). A life-course approach to healthy ageing: Maintaining physical capability. *The Proceedings of the Nutrition Society*, *73*(2), 237–248. https://doi.org/10.1017/S0029665113003923

10. Kuh, D. & New Dynamics of Ageing (NDA) Preparatory Network. (2007). A life course approach to healthy aging, frailty, and capability. *The Journals of Gerontology. Series A, Biological Sciences and Medical Sciences*, *62*(7), 717–721. https://doi.org/10.1093/gerona/62.7.717

11. Woollacott, M., & Shumway-Cook, A. (2002). Attention and the control of posture and gait: A review of an emerging area of research. *Gait & Posture*, *16*(1), 1–14. https://doi.org/10.1016/s0966-6362(01)00156-4

12. Bayot, M., Dujardin, K., Tard, C., Defebvre, L., Bonnet, C. T., Allart, E., & Delval, A. (2018). The interaction between cognition and motor control: A theoretical framework for dual-task interference effects on posture, gait initiation, gait and turning. *Neurophysiologie Clinique = Clinical Neurophysiology*, *48*(6), 361–375. https://doi.org/10.1016/j.neucli.2018.10.003

13. Bernard-Demanze, L., Dumitrescu, M., Jimeno, P., Borel, L., & Lacour, M. (2009). Age-related changes in posture control are differentially affected by postural and cognitive task complexity. *Current Aging Science*, *2*(2), 139–149.

14. Shumway-Cook, A., Woollacott, M., Kerns, K. A., & Baldwin, M. (1997). The Effects of Two Types of Cognitive Tasks on Postural Stability in Older Adults With and Without a History of Falls. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, *52A*(4), M232–M240. https://doi.org/10.1093/gerona/52A.4.M232

15. Shumway-Cook, A., & Woollacott, M. (2000). Attentional demands and postural control: The effect of sensory context. *The Journals of Gerontology. Series A, Biological Sciences and Medical Sciences*, *55*(1), M10-16. https://doi.org/10.1093/gerona/55.1.m10

16. Chen, Y., Cao, Z., Mao, M., Sun, W., Song, Q., & Mao, D. (2022). Increased cortical activation and enhanced functional connectivity in the prefrontal cortex ensure dynamic postural balance during dual-task obstacle negotiation in the older adults: A fNIRS study. *Brain and Cognition*, *163*, 105904. https://doi.org/10.1016/j.bandc.2022.105904

17. Seidler, R. D., Bernard, J. A., Burutolu, T. B., Fling, B. W., Gordon, M. T., Gwin, J. T., Kwak, Y., & Lipps, D. B. (2010). Motor control and aging: Links to age-related brain structural, functional, and biochemical effects. *Neuroscience & Biobehavioral Reviews, Special Section: Dopaminergic Modulation of Lifespan Cognition*, *34*(5), 721–733. https://doi.org/10.1016/j.neubiorev.2009.10.005

18. Yu, S.-H., & Huang, C.-Y. (2017). Improving posture-motor dual-task with a supraposture-focus strategy in young and elderly adults. *PLOS ONE*, *12*(2), e0170687. https://doi.org/10.1371/journal.pone.0170687

19. Tombu, M., & Jolicœur, P. (2003). A central capacity sharing model of dual-task performance. *Journal of Experimental Psychology: Human Perception and Performance*, *29*(1), 3–18. https://doi.org/10.1037/0096-1523.29.1.3

20. Halliday, D. W. R., Mulligan, B. P., Garrett, D. D., Schmidt, S., Hundza, S. R., Garcia-Barrera, M. A., Stawski, R. S., & MacDonald, S. W. S. (2018). Mean and variability in functional brain activations differentially predict executive function in older adults: An investigation employing functional near-infrared spectroscopy. *Neurophotonics*, *5*(1), 011013. https://doi.org/10.1117/1.NPh.5.1.011013

21. Lehmann, N., Kuhn, Y.-A., Keller, M., Aye, N., Herold, F., Draganski, B., Taube, W., & Taubert, M. (2022). Brain Activation During Active Balancing and Its Behavioral Relevance in Younger and Older Adults: A Functional Near-Infrared Spectroscopy (fNIRS) Study. *Frontiers in Aging Neuroscience*, *14*, 828474. https://doi.org/10.3389/fnagi.2022.828474

22. Marusic, U., Taube, W., Morrison, S. A., Biasutti, L., Grassi, B., De Pauw, K., Meeusen, R., Pisot, R., & Ruffieux, J. (2019). Aging effects on prefrontal cortex oxygenation in a posture-cognition dual-task: An fNIRS pilot study. *European Review of Aging and Physical Activity*, *16*, 2. https://doi.org/10.1186/s11556-018-0209-7

23. Baek, C. Y., Kim, H. D., Yoo, D. Y., Kang, K. Y., & Lee, J. W. (2023). Change in activity patterns in the prefrontal cortex in different phases during the dual-task walking in older adults. *Journal of NeuroEngineering and Rehabilitation*, *20*(1), 86. https://doi.org/10.1186/s12984-023-01211-x

24. Yogev-Seligmann, G., Giladi, N., Gruendlinger, L., & Hausdorff, J. M. (2013). The contribution of postural control and bilateral coordination to the impact of dual tasking on gait. *Experimental Brain Research*, *226*(1), 81–93. https://doi.org/10.1007/s00221-013-3412-9

25. Davis, S. W., Dennis, N. A., Daselaar, S. M., Fleck, M. S., & Cabeza, R. (2008). Qué PASA? The Posterior-Anterior Shift in Aging. *Cerebral Cortex (New York, N.Y. : 1991)*, *18*(5), 1201–1209. https://doi.org/10.1093/cercor/bhm155

26. Krelove, L. M., & Mochizuki, G. (2025). The effect of distraction and feedback on prefrontal cortex activity and balance in middle-aged adults: A functional near-infrared spectroscopy pilot study. *Neuroscience*, *568*, 333–342. https://doi.org/10.1016/j.neuroscience.2025.01.026

27. Rosso, A. L., Cenciarini, M., Sparto, P. J., Loughlin, P. J., Furman, J. M., & Huppert, T. J. (2017). Neuroimaging of an attention demanding dual-task during dynamic postural control. *Gait & Posture*, *57*, 193–198. https://doi.org/10.1016/j.gaitpost.2017.06.013

28. Pelicioni, P. H. S., Lord, S. R., Sturnieks, D. L., Halmy, B., & Menant, J. C. (2021). Cognitive and Motor Cortical Activity During Cognitively Demanding Stepping Tasks in Older People at Low and High Risk of Falling. *Frontiers in Medicine*, *8*. https://doi.org/10.3389/fmed.2021.554231

29. Verghese, J., Wang, C., Ayers, E., Izzetoglu, M., & Holtzer, R. (2017). Brain activation in high-functioning older adults and falls: Prospective cohort study. *Neurology*, *88*(2), 191–197. https://doi.org/10.1212/WNL.0000000000003421

30. Holtzer, R., Mahoney, J. R., Izzetoglu, M., Izzetoglu, K., Onaral, B., & Verghese, J. (2011). fNIRS study of walking and walking while talking in young and old individuals. *The Journals of Gerontology. Series A, Biological Sciences and Medical Sciences*, *66*(8), 879–887. https://doi.org/10.1093/gerona/glr068

31. Nóbrega-Sousa, P., Gobbi, L. T. B., Orcioli-Silva, D., Conceição, N. R. da, Beretta, V. S., & Vitório, R. (2020). Prefrontal Cortex Activity During Walking: Effects of Aging and Associations With Gait and Executive Function. *Neurorehabilitation and Neural Repair*, *34*(10), 915–924. https://doi.org/10.1177/1545968320953824

32. Licence, S., Smith, R., McGuigan, M. P., & Earnest, C. P. (2015). Gait Pattern Alterations during Walking, Texting and Walking and Texting during Cognitively Distractive Tasks while Negotiating Common Pedestrian Obstacles. *PloS One*, *10*(7), e0133281. https://doi.org/10.1371/journal.pone.0133281

33. Grady, C. (2012). The cognitive neuroscience of ageing. *Nature Reviews Neuroscience*, *13*(7), 491–505. https://doi.org/10.1038/nrn3256

34. Holtzer, R., Ross, D., O'Brien, C., Izzetoglu, M., & Wagshul, M. E. (2022). Cognitive Reserve Moderates the Efficiency of Prefrontal Cortex Activation Patterns of Gait in Older Adults. *The Journals of Gerontology: Series A*, *77*(9), 1836–1844. https://doi.org/10.1093/gerona/glab288

35. Holtzer, R., Choi, J., Motl, R. W., Foley, F. W., Wagshul, M. E., Hernandez, M. E., & Izzetoglu, M. (2024). Brain control of dual-task walking can be improved in aging and neurological disease. *GeroScience*, *46*(3), 3169–3184. https://doi.org/10.1007/s11357-023-01054-3

36. Wagshul, M. E., Nayak, S., Foley, F. W., Motl, R. W., Hernandez, M. E., Izzetoglu, M., & Holtzer, R. (2025). Grey matter atrophy patterns of mobility shared across older adults with and without multiple sclerosis. *Journal of Neurology*, *272*(10), 656. https://doi.org/10.1007/s00415-025-13373-w

37. Cabeza, R. (2002). Hemispheric asymmetry reduction in older adults: The HAROLD model. *Psychology and Aging*, *17*(1), 85–100. https://doi.org/10.1037//0882-7974.17.1.85

38. Park, D. C., & Reuter-Lorenz, P. (2009). The Adaptive Brain: Aging and Neurocognitive Scaffolding. *Annual Review of Psychology*, *60*, 173–196. https://doi.org/10.1146/annurev.psych.59.103006.093656

39. Reuter-Lorenz, P. A., & Cappell, K. A. (2008). Neurocognitive Aging and the Compensation Hypothesis. *Current Directions in Psychological Science*, *17*(3), 177–182. https://doi.org/10.1111/j.1467-8721.2008.00570.x

40. Wu, Y., Dong, Y., Tang, Y., Wang, W., Bo, Y., & Zhang, C. (2023). Relationship between motor performance and cortical activity of older neurological disorder patients with dyskinesia using fNIRS: A systematic review. *Frontiers in Physiology*, *14*, 1153469. https://doi.org/10.3389/fphys.2023.1153469

41. Stern, Y. (2009). Cognitive reserve. *Neuropsychologia*, *47*(10), 2015–2028. https://doi.org/10.1016/j.neuropsychologia.2009.03.004

42. Reuter-Lorenz, P. A., & Park, D. C. (2014). How Does it STAC Up? Revisiting the Scaffolding Theory of Aging and Cognition. *Neuropsychology Review*, *24*(3), 355–370. https://doi.org/10.1007/s11065-014-9270-9

43. Chong, H. R., Ranjbar-Slamloo, Y., Ho, M. Z. H., Ouyang, X., & Kamigaki, T. (2023). Functional alterations of the prefrontal circuit underlying cognitive aging in mice. *Nature Communications*, *14*, 7254. https://doi.org/10.1038/s41467-023-43142-0

44. Weirich, G., Bemben, D. A., & Bemben, M. G. (2010). Predictors of Balance in Young, Middle-Aged, and Late Middle-Aged Women. *Journal of Geriatric Physical Therapy*, *33*(3), 110. https://doi.org/10.1097/JPT.0b013e3181eda25b

45. Hill, M. W., Duncan, M. J., & Price, M. J. (2020). The emergence of age-related deterioration in dynamic, but not quiet standing balance abilities among healthy middle-aged adults. *Experimental Gerontology*, *140*, 111076. https://doi.org/10.1016/j.exger.2020.111076

46. Mirelman, A., Maidan, I., Bernad-Elazari, H., Nieuwhof, F., Reelick, M., Giladi, N., & Hausdorff, J. M. (2014). Increased frontal brain activation during walking while dual tasking: An fNIRS study in healthy young adults. *Journal of NeuroEngineering and Rehabilitation*, *11*(1), 85. https://doi.org/10.1186/1743-0003-11-85

47. Mirelman, A., Maidan, I., Bernad-Elazari, H., Shustack, S., Giladi, N., & Hausdorff, J. M. (2017). Effects of aging on prefrontal brain activation during challenging walking conditions. *Brain and Cognition*, *115*, 41–46. https://doi.org/10.1016/j.bandc.2017.04.002

48. Beck Jepsen, D., Robinson, K., Ogliari, G., Montero-Odasso, M., Kamkar, N., Ryg, J., Freiberger, E., & Masud, T. (2022). Predicting falls in older adults: An umbrella review of instruments assessing gait, balance, and functional mobility. *BMC Geriatrics*, *22*(1), 615. https://doi.org/10.1186/s12877-022-03271-5

49. Varela-Vásquez, L. A., Minobes-Molina, E., & Jerez-Roig, J. (2020). Dual-task exercises in older adults: A structured review of current literature. *Journal of Frailty, Sarcopenia and Falls*, *5*(2), 31–37. https://doi.org/10.22540/JFSF-05-031

50. Demnitz, N., Zsoldos, E., Mahmood, A., Mackay, C. E., Kivimäki, M., Singh-Manoux, A., Dawes, H., Johansen-Berg, H., Ebmeier, K. P., & Sexton, C. E. (2017). Associations between Mobility, Cognition, and Brain Structure in Healthy Older Adults. *Frontiers in Aging Neuroscience*, *9*. https://doi.org/10.3389/fnagi.2017.00155

51. Studenski, S., Perera, S., Patel, K., Rosano, C., Faulkner, K., Inzitari, M., Brach, J., Chandler, J., Cawthon, P., Connor, E. B., Nevitt, M., Visser, M., Kritchevsky, S., Badinelli, S., Harris, T., Newman, A. B., Cauley, J., Ferrucci, L., & Guralnik, J. (2011). Gait Speed and Survival in Older Adults. *JAMA*, *305*(1), 50–58. https://doi.org/10.1001/jama.2010.1923

52. Middleton, A., Fritz, S. L., & Lusardi, M. (2015). Walking speed: The functional vital sign. *Journal of Aging and Physical Activity*, *23*(2), 314–322. https://doi.org/10.1123/japa.2013-0236

53. Toronto, C. of. (2017, November 17). Pedestrian Countdown Signals. *City of Toronto*. (Toronto, Ontario, Canada). https://www.toronto.ca/services-payments/streets-parking-transportation/traffic-management/traffic-signals-street-signs/types-of-traffic-signals/pedestrian-countdown-timers/

54. *Pedestrian Crossing Treatments*. (2016). Ministry of Transportation.

55. Deandrea, S., Lucenteforte, E., Bravi, F., Foschi, R., La Vecchia, C., & Negri, E. (2010). Risk Factors for Falls in Community-dwelling Older People: A Systematic Review and Meta-analysis. *Epidemiology*, *21*(5), 658. https://doi.org/10.1097/EDE.0b013e3181e89905

56. Blodgett, J. M., Hardy, R., Davis, D., Peeters, G., Kuh, D., & Cooper, R. (2022). One-Legged Balance Performance and Fall Risk in Mid and Later Life: Longitudinal Evidence From a British Birth Cohort. *American Journal of Preventive Medicine*, *63*(6), 997–1006. https://doi.org/10.1016/j.amepre.2022.07.002

57. Ärzteblatt, D. Ä. G., Redaktion Deutsches. (n.d.). *Physical Performance in Middle Age and Old Age (19.11.2010)*. Deutsches Ärzteblatt. Retrieved December 9, 2025, from https://www.aerzteblatt.de/int/archive/article?id=79256

58. Leibel, D. K., Williams, M. R., Katzel, L. I., Evans, M. K., Zonderman, A. B., & Waldstein, S. R. (2020). Relations of Executive Function and Physical Performance in Middle Adulthood: A Prospective Investigation in African American and White Adults. *The Journals of Gerontology: Series B*, *75*(6), e56–e68. https://doi.org/10.1093/geronb/gbaa012

59. Watson, N. L., Rosano, C., Boudreau, R. M., Simonsick, E. M., Ferrucci, L., Sutton-Tyrrell, K., Hardy, S. E., Atkinson, H. H., Yaffe, K., Satterfield, S., Harris, T. B., Newman, A. B., & for the Health ABC Study. (2010). Executive Function, Memory, and Gait Speed Decline in Well-Functioning Older Adults. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, *65A*(10), 1093–1100. https://doi.org/10.1093/gerona/glq111

60. Volpi, E., Nazemi, R., & Fujita, S. (2004). Muscle tissue changes with aging. *Current Opinion in Clinical Nutrition and Metabolic Care*, *7*(4), 405–410. https://doi.org/10.1097/01.mco.0000134362.76653.b2

61. Larsson, L., Degens, H., Li, M., Salviati, L., Lee, Y. il, Thompson, W., Kirkland, J. L., & Sandri, M. (2019). Sarcopenia: Aging-Related Loss of Muscle Mass and Function. *Physiological Reviews*, *99*(1), 427–511. https://doi.org/10.1152/physrev.00061.2017

62. Park, W. T., Shon, O.-J., & Kim, G. B. (2023). Multidisciplinary approach to sarcopenia: A narrative review. *Journal of Yeungnam Medical Science*, *40*(4), 352–363. https://doi.org/10.12701/jyms.2023.00724

63. Wilkinson, D. J., Piasecki, M., & Atherton, P. J. (2018). The age-related loss of skeletal muscle mass and function: Measurement and physiology of muscle fibre atrophy and muscle fibre loss in humans. *Ageing Research Reviews*, *47*, 123–132. https://doi.org/10.1016/j.arr.2018.07.005

64. Peeters, G. M. E. E. (Geeske), Jones, M., Byles, J., & Dobson, A. J. (2015). Long-term Consequences of Noninjurious and Injurious Falls on Well-being in Older Women. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, *70*(12), 1519–1525. https://doi.org/10.1093/gerona/glv102

65. Rosso, A. L., Metti, A. L., Faulkner, K., Brach, J. S., Studenski, S. A., Redfern, M., & Rosano, C. (2019). Associations of Usual Pace and Complex Task Gait Speeds With Incident Mobility Disability. *Journal of the American Geriatrics Society*, *67*(10), 2072–2076. https://doi.org/10.1111/jgs.16049

66. Hausdorff, J. M., Schweiger, A., Herman, T., Yogev-Seligmann, G., & Giladi, N. (2008). Dual-Task Decrements in Gait: Contributing Factors Among Healthy Older Adults. *The Journals of Gerontology: Series A*, *63*(12), 1335–1343. https://doi.org/10.1093/gerona/63.12.1335

67. Andrews, A. W., Chinworth, S. A., Bourassa, M., Garvin, M., Benton, D., & Tanner, S. (2010). Update on distance and velocity requirements for community ambulation. *Journal of Geriatric Physical Therapy (2001)*, *33*(3), 128–134.

68. Montero-Odasso, M., Schapira, M., Soriano, E. R., Varela, M., Kaplan, R., Camera, L. A., & Mayorga, L. M. (2005). Gait Velocity as a Single Predictor of Adverse Events in Healthy Seniors Aged 75 Years and Older. *The Journals of Gerontology: Series A*, *60*(10), 1304–1309. https://doi.org/10.1093/gerona/60.10.1304

69. Mantel, A., Trapuzzano, A., Chizmar, S., Haffke, L., & Dawson, N. (2019). An Investigation of the Predictors of Comfortable and Fast Gait Speed in Community-Dwelling Older Adults. *Journal of Geriatric Physical Therapy*, *42*(4), E62. https://doi.org/10.1519/JPT.0000000000000216

70. Klima, D. W., & Hood, E. (2020). Gait and Balance Assessment of Older Adults. *Current Geriatrics Reports*, *9*(3), 154–162. https://doi.org/10.1007/s13670-020-00327-5

71. Minta, K., Colombo, G., Taylor, W. R., & Schinazi, V. R. (2023). Differences in fall-related characteristics across cognitive disorders. *Frontiers in Aging Neuroscience*, *15*, 1171306. https://doi.org/10.3389/fnagi.2023.1171306

72. Menant, J. C., Schoene, D., Sarofim, M., & Lord, S. R. (2014). Single and dual task tests of gait speed are equivalent in the prediction of falls in older people: A systematic review and meta-analysis. *Ageing Research Reviews*, *16*, 83–104. https://doi.org/10.1016/j.arr.2014.06.001

73. Muir-Hunter, S. W., & Wittwer, J. E. (2016). Dual-task testing to predict falls in community-dwelling older adults: A systematic review. *Physiotherapy*, *102*(1), 29–40. https://doi.org/10.1016/j.physio.2015.04.011

74. Royall, D. R., Lauterbach, E. C., Kaufer, D., Malloy, P., Coburn, K. L., & Black, K. J. (2007). The Cognitive Correlates of Functional Status: A Review From the Committee on Research of the American Neuropsychiatric Association. *The Journal of Neuropsychiatry and Clinical Neurosciences*, *19*(3), 249–265. https://doi.org/10.1176/jnp.2007.19.3.249

75. Gothe, N. P., Fanning, J., Awick, E., Chung, D., Wójcicki, T. R., Olson, E. A., Mullen, S. P., Voss, M., Erickson, K. I., Kramer, A. F., & McAuley, E. (2014). Executive Function Processes Predict Mobility Outcomes in Older Adults. *Journal of the American Geriatrics Society*, *62*(2), 285–290. https://doi.org/10.1111/jgs.12654

76. Pelicioni, P. H. S., Tijsma, M., Lord, S. R., & Menant, J. (2019). Prefrontal cortical activation measured by fNIRS during walking: Effects of age, disease and secondary task. *PeerJ*, *7*, e6833. https://doi.org/10.7717/peerj.6833

77. Beurskens, R., Helmich, I., Rein, R., & Bock, O. (2014). Age-related changes in prefrontal activity during walking in dual-task situations: A fNIRS study. *International Journal of Psychophysiology*, *92*(3), 122–128. https://doi.org/10.1016/j.ijpsycho.2014.03.005

78. Krelove, L. M., Machula, A., Sergio, L. E., & Mochizuki, G. (2025). Prefrontal cortex activity during virtual obstacle avoidance and distracted walking: A methodological proof of concept using augmented reality and functional near-infrared spectroscopy. *PLOS One*, *20*(10), e0333622. https://doi.org/10.1371/journal.pone.0333622

79. Friendly, M., Franklin, P. E., Hoffman, D., & Rubin, D. C. (1982). The Toronto Word Pool: Norms for imagery, concreteness, orthographic variables, and grammatical usage for 1,080 words. *Behavior Research Methods & Instrumentation*, *14*(4), 375–399. https://doi.org/10.3758/BF03203275

80. Watkins, M. J., Neath, I., & Sechler, E. S. (1989). Recency Effect in Recall of a Word List When an Immediate Memory Task Is Performed after Each Word Presentation. *The American Journal of Psychology*, *102*(2), 265–270. https://doi.org/10.2307/1422957

81. Pinti, P., Tachtsidis, I., Hamilton, A., Hirsch, J., Aichelburg, C., Gilbert, S., & Burgess, P. W. (2020). The present and future use of functional near-infrared spectroscopy (fNIRS) for cognitive neuroscience. *Annals of the New York Academy of Sciences*, *1464*(1), 5–29. https://doi.org/10.1111/nyas.13948

82. Yücel, M. A., Lühmann, A. v, Scholkmann, F., Gervain, J., Dan, I., Ayaz, H., Boas, D., Cooper, R. J., Culver, J., Elwell, C. E., Eggebrecht, A., Franceschini, M. A., Grova, C., Homae, F., Lesage, F., Obrig, H., Tachtsidis, I., Tak, S., Tong, Y., … Wolf, M. (2021). Best practices for fNIRS publications. *Neurophotonics*, *8*(1), 012101. https://doi.org/10.1117/1.NPh.8.1.012101

83. Huppert, T., Diamond, S., Franceschini, M. A., & Boas, D. (2009). HomER: A review of time-series analysis methods for near-infrared spectroscopy of the brain. *Applied Optics*, *48*, D280-98. https://doi.org/10.1364/AO.48.00D280

84. Dans, P. W., Foglia, S. D., & Nelson, A. J. (2021). Data Processing in Functional Near-Infrared Spectroscopy (fNIRS) Motor Control Research. *Brain Sciences*, *11*(5), Article 5. https://doi.org/10.3390/brainsci11050606

85. Strangman, G., Culver, J. P., Thompson, J. H., & Boas, D. A. (2002). A Quantitative Comparison of Simultaneous BOLD fMRI and NIRS Recordings during Functional Brain Activation. *NeuroImage*, *17*(2), 719–731. https://doi.org/10.1006/nimg.2002.1227

86. Bonilauri, A., Sangiuliano Intra, F., Baselli, G., & Baglio, F. (2022). Assessment of fNIRS Signal Processing Pipelines: Towards Clinical Applications. *Applied Sciences*, *12*(1), Article 1. https://doi.org/10.3390/app12010316

87. Suzuki, M., Miyai, I., Ono, T., Oda, I., Konishi, I., Kochiyama, T., & Kubota, K. (2004). Prefrontal and premotor cortices are involved in adapting walking and running speed on the treadmill: An optical imaging study. *NeuroImage*, *23*(3), 1020–1026. https://doi.org/10.1016/j.neuroimage.2004.07.002

88. Hoshi, Y., Kobayashi, N., & Tamura, M. (2001). Interpretation of near-infrared spectroscopy signals: A study with a newly developed perfused rat brain model. *Journal of Applied Physiology*, *90*(5), 1657–1662. https://doi.org/10.1152/jappl.2001.90.5.1657

89. Cleveland, W. S. (1979). Robust Locally Weighted Regression and Smoothing Scatterplots. *Journal of the American Statistical Association*, *74*(368), 829–836. https://doi.org/10.2307/2286407

90. Cleveland, W. S., & Devlin, S. J. (1988). Locally Weighted Regression: An Approach to Regression Analysis by Local Fitting. *Journal of the American Statistical Association*, *83*(403), 596–610. https://doi.org/10.2307/2289282

91. Stone, M. (1974). Cross-Validatory Choice and Assessment of Statistical Predictions. *Journal of the Royal Statistical Society: Series B (Methodological)*, *36*(2), 111–133. https://doi.org/10.1111/j.2517-6161.1974.tb00994.x

92. Clark, R. M. (1980). Calibration, Cross-Validation and Carbon-14. II. *Journal of the Royal Statistical Society. Series A (General)*, *143*(2), 177–194. https://doi.org/10.2307/2981989

93. Muggeo, V. M. R. (n.d.). *segmented: An R Package to Fit Regression Models with Broken-Line Relationships*.

94. Das, R., Banerjee, M., Nan, B., & Zheng, H. (2016). Fast estimation of regression parameters in a broken-stick model for longitudinal data. *Journal of the American Statistical Association*, *111*(515), 1132–1143. https://doi.org/10.1080/01621459.2015.1073154

95. Hurvich, C. M., & Tsai, C.-L. (1989). Regression and Time Series Model Selection in Small Samples. *Biometrika*, *76*(2), 297–307. https://doi.org/10.2307/2336663

96. Buuren, S. van. (2023). Broken Stick Model for Irregular Longitudinal Data. *Journal of Statistical Software*, *106*, 1–51. https://doi.org/10.18637/jss.v106.i07

97. Shinan-Altman, S., & Werner, P. (2019). Subjective Age and Its Correlates Among Middle-Aged and Older Adults. *The International Journal of Aging and Human Development*, *88*(1), 3–21. https://doi.org/10.1177/0091415017752941

98. Deery, H. A., Di Paolo, R., Moran, C., Egan, G. F., & Jamadar, S. D. (2023). The older adult brain is less modular, more integrated, and less efficient at rest: A systematic review of large-scale resting-state functional brain networks in aging. *Psychophysiology*, *60*(1), e14159. https://doi.org/10.1111/psyp.14159

99. Cabeza, R. (2002). Hemispheric asymmetry reduction in older adults: The HAROLD model. *Psychology and Aging*, *17*(1), 85–100. https://doi.org/10.1037/0882-7974.17.1.85

100. Agcaoglu, O., Miller, R., Mayer, A. R., Hugdahl, K., & Calhoun, V. D. (2015). Lateralization of resting state networks and relationship to age and gender. *NeuroImage*, *104*, 310–325. https://doi.org/10.1016/j.neuroimage.2014.09.001

101. Reuter-Lorenz, P. A., & Cappell, K. A. (2008). Neurocognitive Aging and the Compensation Hypothesis. *Current Directions in Psychological Science*, *17*(3), 177–182. https://doi.org/10.1111/j.1467-8721.2008.00570.x

102. Cabeza, R., Albert, M., Belleville, S., Craik, F. I. M., Duarte, A., Grady, C. L., Lindenberger, U., Nyberg, L., Park, D. C., Reuter-Lorenz, P. A., Rugg, M. D., Steffener, J., & Rajah, M. N. (2018). Maintenance, reserve and compensation: The cognitive neuroscience of healthy ageing. *Nature Reviews Neuroscience*, *19*(11), 701–710. https://doi.org/10.1038/s41583-018-0068-2

103. Park, D. C., & Reuter-Lorenz, P. (2009). The Adaptive Brain: Aging and Neurocognitive Scaffolding. *Annual Review of Psychology*, *60*, 173–196. https://doi.org/10.1146/annurev.psych.59.103006.093656

104. Anderson, M. L., & Finlay, B. L. (2014). Allocating structure to function: The strong links between neuroplasticity and natural selection. *Frontiers in Human Neuroscience*, *7*. https://doi.org/10.3389/fnhum.2013.00918

105. Reuter-Lorenz, P. A., & Park, D. C. (2014). How Does it STAC Up? Revisiting the Scaffolding Theory of Aging and Cognition. *Neuropsychology Review*, *24*(3), 355–370. https://doi.org/10.1007/s11065-014-9270-9

106. Naik, S., Banerjee, A., Bapi, R. S., Deco, G., & Roy, D. (2017). Metastability in Senescence. *Trends in Cognitive Sciences*, *21*(7), 509–521. https://doi.org/10.1016/j.tics.2017.04.007

107. Autenrieth, C. S., Karrasch, S., Heier, M., Gorzelniak, L., Ladwig, K.-H., Peters, A., & Döring, A. (2013). Decline in gait performance detected by an electronic walkway system in 907 older adults of the population-based KORA-Age study. *Gerontology*, *59*(2), 165–173. https://doi.org/10.1159/000342206

108. Raz, N., Gunning, F. M., Head, D., Dupuis, J. H., McQuain, J., Briggs, S. D., Loken, W. J., Thornton, A. E., & Acker, J. D. (1997). Selective aging of the human cerebral cortex observed in vivo: Differential vulnerability of the prefrontal gray matter. *Cerebral Cortex (New York, N.Y.: 1991)*, *7*(3), 268–282. https://doi.org/10.1093/cercor/7.3.268

109. Gong, Z., Faulkner, M. E., Akhonda, M. A. B. S., Guo, A., Bae, J., Laporte, J. P., Church, S., D'Agostino, J., Bergeron, J., Bergeron, C. M., Ferrucci, L., & Bouhrara, M. (2024). White matter integrity and motor function: A link between cerebral myelination and longitudinal changes in gait speed in aging. *GeroScience*, *47*(2), 1441–1454. https://doi.org/10.1007/s11357-024-01392-w

110. Hupfeld, K. E., Geraghty, J. M., McGregor, H. R., Hass, C. J., Pasternak, O., & Seidler, R. D. (2022). Differential Relationships Between Brain Structure and Dual Task Walking in Young and Older Adults. *Frontiers in Aging Neuroscience*, *14*, 809281. https://doi.org/10.3389/fnagi.2022.809281

111. Wickens, C. D. (2008). Multiple resources and mental workload. *Human Factors*, *50*(3), 449–455. https://doi.org/10.1518/001872008X288394

112. Harada, C. N., Natelson Love, M. C., & Triebel, K. L. (2013). Normal Cognitive Aging. *Clinics in Geriatric Medicine, Mild Cognitive Impairment*, *29*(4), 737–752. https://doi.org/10.1016/j.cger.2013.07.002

113. Herman, T., Mirelman, A., Giladi, N., Schweiger, A., & Hausdorff, J. M. (2010). Executive Control Deficits as a Prodrome to Falls in Healthy Older Adults: A Prospective Study Linking Thinking, Walking, and Falling. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, *65A*(10), 1086–1092. https://doi.org/10.1093/gerona/glq077

114. Fettrow, T., Hupfeld, K., Hass, C., Pasternak, O., & Seidler, R. (2023). Neural correlates of gait adaptation in younger and older adults. *Scientific Reports*, *13*(1), 3842. https://doi.org/10.1038/s41598-023-30766-x

115. Studenski, S. (2019). Gait Speed Reveals Clues to Lifelong Health. *JAMA Network Open*, *2*(10), e1913112. https://doi.org/10.1001/jamanetworkopen.2019.13112

116. Chui, K., Hood, E., & Klima, D. (2012). Meaningful Change in Walking Speed. *Topics in Geriatric Rehabilitation*, *28*(2), 97. https://doi.org/10.1097/TGR.0b013e3182510195

117. Deshpande, N., Metter, E. J., Guralnik, J., & Ferrucci, L. (2013). Can Failure on Adaptive Locomotor Tasks Independently Predict Incident Mobility Disability? *American Journal of Physical Medicine & Rehabilitation / Association of Academic Physiatrists*, *92*(8), 704–709. https://doi.org/10.1097/PHM.0b013e31827d634e

118. Nasreddine, Z. S., Phillips, N. A., Bédirian, V., Charbonneau, S., Whitehead, V., Collin, I., Cummings, J. L., & Chertkow, H. (2005). The Montreal Cognitive Assessment, MoCA: A Brief Screening Tool For Mild Cognitive Impairment. *Journal of the American Geriatrics Society*, *53*(4), 695–699. https://doi.org/10.1111/j.1532-5415.2005.53221.x

119. Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). "Mini-mental state": A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research*, *12*(3), 189–198. https://doi.org/10.1016/0022-3956(75)90026-6

120. Shumway-Cook, A., Woollacott, M., Kerns, K. A., & Baldwin, M. (1997). The effects of two types of cognitive tasks on postural stability in older adults with and without a history of falls. *The Journals of Gerontology. Series A, Biological Sciences and Medical Sciences*, *52*(4), M232-240. https://doi.org/10.1093/gerona/52a.4.m232

121. Hill, M. W., Duncan, M. J., & Price, M. J. (2020). The emergence of age-related deterioration in dynamic, but not quiet standing balance abilities among healthy middle-aged adults. *Experimental Gerontology*, *140*, 111076. https://doi.org/10.1016/j.exger.2020.111076

122. Koren, Y., Parmet, Y., & Bar-Haim, S. (2019). Treading on the unknown increases prefrontal activity: A pilot fNIRS study. *Gait & Posture*, *69*, 96–100. https://doi.org/10.1016/j.gaitpost.2019.01.026

123. Kahya, M., Moon, S., Ranchet, M., Vukas, R. R., Lyons, K. E., Pahwa, R., Akinwuntan, A., & Devos, H. (2019). Brain activity during dual task gait and balance in aging and age-related neurodegenerative conditions: A systematic review. *Experimental Gerontology*, *128*, 110756. https://doi.org/10.1016/j.exger.2019.110756

124. Li, K. Z. H., Bherer, L., Mirelman, A., Maidan, I., & Hausdorff, J. M. (2018). Cognitive Involvement in Balance, Gait and Dual-Tasking in Aging: A Focused Review From a Neuroscience of Aging Perspective. *Frontiers in Neurology*, *9*. https://doi.org/10.3389/fneur.2018.00913

125. Figgins, E., Choi, Y.-H., Speechley, M., & Montero-Odasso, M. (2021). Associations Between Potentially Modifiable and Nonmodifiable Risk Factors and Gait Speed in Middle- and Older-Aged Adults: Results From the Canadian Longitudinal Study on Aging. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, *76*(10), e253–e263. https://doi.org/10.1093/gerona/glab008

126. Middleton, A., Fulk, G. D., Herter, T. M., Beets, M. W., Donley, J., & Fritz, S. L. (2016). Self-Selected and Maximal Walking Speeds Provide Greater Insight Into Fall Status Than Walking Speed Reserve Among Community-Dwelling Older Adults. *American Journal of Physical Medicine & Rehabilitation*, *95*(7), 475–482. https://doi.org/10.1097/PHM.0000000000000488

127. Asher, L., Aresu, M., Falaschetti, E., & Mindell, J. (2012). Most older pedestrians are unable to cross the road in time: A cross-sectional study. *Age and Ageing*, *41*(5), 690–694. https://doi.org/10.1093/ageing/afs076

128. Pampel, F. C., Krueger, P. M., & Denney, J. T. (2010). Socioeconomic Disparities in Health Behaviors. *Annual Review of Sociology*, *36*(Volume 36, 2010), 349–370. https://doi.org/10.1146/annurev.soc.012809.102529

129. Campanholo, K. R., Boa, I. N. F., Hodroj, F. C. da S. A., Guerra, G. R. B., Miotto, E. C., & Lucia, M. C. S. de. (2017). Impact of sociodemographic variables on executive functions. *Dementia & Neuropsychologia*, *11*, 62–68. https://doi.org/10.1590/1980-57642016dn11-010010

130. Rej, S., Begley, A., Gildengers, A., Dew, M. A., Reynolds, C. F., & Butters, M. A. (2015). Psychosocial Risk Factors for Cognitive Decline in Late-Life Depression: Findings from the MTLD-III Study. *Canadian Geriatrics Journal*, *18*(2), 43–50. https://doi.org/10.5770/cgj.18.134

131. Dury, S., De Roeck, E., Duppen, D., Fret, B., Hoeyberghs, L., Lambotte, D., Van der Elst, M., van der Vorst, A., Schols, J., Kempen, G., Rixt Zijlstra, G. A., De Lepeleire, J., Schoenmakers, B., Kardol, T., De Witte, N., Verté, D., De Donder, L., De Deyn, P. P., Engelborghs, S., … Dierckx, E. (2017). Identifying frailty risk profiles of home-dwelling older people: Focus on sociodemographic and socioeconomic characteristics. *Aging & Mental Health*, *21*(10), 1031–1039. https://doi.org/10.1080/13607863.2016.1193120

132. Melzer, D., & Parahyba, M. I. (2004). Socio-demographic correlates of mobility disability in older Brazilians: Results of the first national survey. *Age and Ageing*, *33*(3), 253–259. https://doi.org/10.1093/ageing/afh075

133. Ross, C. E., & Wu, C.-L. (1996). Education, Age, and the Cumulative Advantage in Health. *Journal of Health and Social Behavior*, *37*(1), 104–120. https://doi.org/10.2307/2137234

134. Kline, P. W., Shaikh, F. D., Tennant, J. E., Hamel, R., & Zukowski, L. A. (2024). Global Cognition, Gender, and Level of Education Predict Dual-Task Gait Speed Variability Metrics in Older Adults. *Gerontology*, *70*(7), 724–732. https://doi.org/10.1159/000537720

135. Campos-Magdaleno, M., Pereiro, A., Navarro-Pardo, E., Juncos-Rabadán, O., & Facal, D. (2022). Dual-task performance in old adults: Cognitive, functional, psychosocial and socio-demographic variables. *Aging Clinical and Experimental Research*, *34*(4), 827–835. https://doi.org/10.1007/s40520-021-02002-x

136. Xu, S., Sun, Q., Li, M., Luo, J., Cai, G., Chen, R., Zhang, L., & Liu, J. (2021). Hippocampal resting-state functional connectivity with the mPFC and DLPFC moderates and mediates the association between education level and memory function in subjective cognitive decline. *Brain Science Advances*, *7*(2), 124–140. https://doi.org/10.26599/BSA.2021.9050013

137. Shaked, D., Katzel, L. I., Seliger, S. L., Gullapalli, R. P., Davatzikos, C., Erus, G., Evans, M. K., Zonderman, A. B., & Waldstein, S. R. (2018). Dorsolateral prefrontal cortex volume as a mediator between socioeconomic status and executive function. *Neuropsychology*, *32*(8), 985–995. https://doi.org/10.1037/neu0000484

138. Powell, L. E., & Myers, A. M. (1995). The Activities-specific Balance Confidence (ABC) Scale. *The Journals of Gerontology: Series A*, *50A*(1), M28–M34. https://doi.org/10.1093/gerona/50A.1.M28

139. Garber, C. E., Blissmer, B., Deschenes, M. R., Franklin, B. A., Lamonte, M. J., Lee, I.-M., Nieman, D. C., & Swain, D. P. (2011). Quantity and Quality of Exercise for Developing and Maintaining Cardiorespiratory, Musculoskeletal, and Neuromotor Fitness in Apparently Healthy Adults: Guidance for Prescribing Exercise. *Medicine & Science in Sports & Exercise*, *43*(7), 1334. https://doi.org/10.1249/MSS.0b013e318213fefb

140. *Median Income Explorer*. (n.d.). Retrieved March 16, 2026, from https://censusmapper.ca/maps/3348?index=3#16/43.7755/-79.5047

141. Montero-Odasso, M., Verghese, J., Beauchet, O., & Hausdorff, J. M. (2012). Gait and Cognition: A Complementary Approach to Understanding Brain Function and the Risk of Falling. *Journal of the American Geriatrics Society*, *60*(11), 2127–2136. https://doi.org/10.1111/j.1532-5415.2012.04209.x

142. Montufar, J., Arango, J., Porter, M., & Nakagawa, S. (2007). Pedestrians' Normal Walking Speed and Speed When Crossing a Street. *Transportation Research Record*, *2002*(1), 90–97. https://doi.org/10.3141/2002-12

143. Steen, J., Loeys, T., Moerkerke, B., & Vansteelandt, S. (2017). medflex: An R Package for Flexible Mediation Analysis using Natural Effect Models. *Journal of Statistical Software*, *76*, 1–46. https://doi.org/10.18637/jss.v076.i11

144. MacKinnon, D. P., Fairchild, A. J., & Fritz, M. S. (2007). Mediation Analysis. *Annual Review of Psychology*, *58*, 593. https://doi.org/10.1146/annurev.psych.58.110405.085542

145. Preacher, K. J., & Hayes, A. F. (2004). SPSS and SAS procedures for estimating indirect effects in simple mediation models. *Behavior Research Methods, Instruments, & Computers: A Journal of the Psychonomic Society, Inc*, *36*(4), 717–731. https://doi.org/10.3758/bf03206553

146. Imai, K., Keele, L., Tingley, D., & Yamamoto, T. (2010). Causal Mediation Analysis Using R. In H. D. Vinod (Ed.), *Advances in Social Science Research Using R* (Vol. 196, pp. 129–154). Springer New York. https://doi.org/10.1007/978-1-4419-1764-5_8

147. Molenaar, E. A. L. M., Barten, D.-J. J., de Hoop, A. M. S., Bleijenberg, N., de Wit, N. J., & Veenhof, C. (2022). Cluster analysis of functional independence in community-dwelling older people. *BMC Geriatrics*, *22*(1), 996. https://doi.org/10.1186/s12877-022-03684-2

148. Norusis, M. (2011, February 25). *IBM SPSS Statistics 19 Advanced Statistical Procedures Companion*. https://www.semanticscholar.org/paper/IBM-SPSS-Statistics-19-Advanced-Statistical-Norusis/27e0b26cd5a0bcf009e9ffe7d2c8e8934a74944f

149. Chuang, H.-Y., & Chou, W. (2025). SilhouetteScoreinR: Beyond traditional network layouts by leveraging local cohesion and nearest neighbor separation. *MethodsX*, *15*, 103622. https://doi.org/10.1016/j.mex.2025.103622

150. Imai, K., Keele, L., & Tingley, D. (2010). A general approach to causal mediation analysis. *Psychological Methods*, *15*(4), 309–334. https://doi.org/10.1037/a0020761

151. Wood, S. N. (2017). *Generalized Additive Models: An Introduction with R, Second Edition* (2nd ed.). Chapman and Hall/CRC. https://doi.org/10.1201/9781315370279

152. Efron, B., & Tibshirani, R. (1998). *An introduction to the bootstrap* (Nachdr.). Chapman & Hall.

153. Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods*, *40*(3), 879–891. https://doi.org/10.3758/BRM.40.3.879

154. VanderWeele, T. J. (2016). Explanation in causal inference: Developments in mediation and interaction. *International Journal of Epidemiology*, *45*(6), 1904–1908. https://doi.org/10.1093/ije/dyw277

155. *Interpretation and identification of causal mediation.* (n.d.). Retrieved March 29, 2026, from https://psycnet.apa.org/record/2014-21922-001

156. Iacobucci, D. (2012). Mediation analysis and categorical variables: The final frontier. *Journal of Consumer Psychology*, *22*(4), 582–594. https://doi.org/10.1016/j.jcps.2012.03.006

157. Hayes, A. F. (2013). *Introduction to mediation, moderation, and conditional process analysis: A regression-based approach* (pp. xvii, 507). The Guilford Press.

158. Gelman, A., & Hill, J. (2006, December 17). *Data Analysis Using Regression and Multilevel/Hierarchical Models*. Cambridge University Press. Cambridge Aspire Website. https://doi.org/10.1017/CBO9780511790942

159. *Regression Modeling Strategies: With Applications to Linear Models, Logistic and Ordinal Regression, and Survival Analysis | Springer Nature Link.* (n.d.). Retrieved March 29, 2026, from https://link.springer.com/book/10.1007/978-3-319-19425-7

160. Baron, R. M., & Kenny, D. A. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology: Interpersonal Relations and Group Processes*, *51*(6), 1173–1182. https://doi.org/10.1037/0022-3514.51.6.1173

161. MacKinnon, D. P. (2008). *Introduction to statistical mediation analysis* (pp. x, 477). Taylor & Francis Group/Lawrence Erlbaum Associates.

162. Quiben, M. U., & Hazuda, H. P. (2015). Factors Contributing to 50-ft Walking Speed and Observed Ethnic Differences in Older Community-Dwelling Mexican Americans and European Americans. *Physical Therapy*, *95*(6), 871–883. https://doi.org/10.2522/ptj.20140152

163. Gregory, P. C., Szanton, S. L., Xue, Q.-L., Tian, J., Thorpe, R. J., & Fried, L. P. (2011). Education Predicts Incidence of Preclinical Mobility Disability in Initially High-Functioning Older Women. The Women's Health and Aging Study II. *The Journals of Gerontology: Series A*, *66A*(5), 577–581. https://doi.org/10.1093/gerona/glr023

164. Sulander, T., Martelin, T., Sainio, P., Rahkonen, O., Nissinen, A., & Uutela, A. (2006). Trends and educational disparities in functional capacity among people aged 65–84 years. *International Journal of Epidemiology*, *35*(5), 1255–1261. https://doi.org/10.1093/ije/dyl183

165. Sainio, P., Martelin, T., Koskinen, S., & Heliövaara, M. (2007). Educational differences in mobility: The contribution of physical workload, obesity, smoking and chronic conditions. *Journal of Epidemiology & Community Health*, *61*(5), 401–408. https://doi.org/10.1136/jech.2006.048306

166. Kyrönlahti, S. M., Stenholm, S., Raitanen, J., Neupane, S., Koskinen, S., & Tiainen, K. (2021). Educational Differences in Decline in Maximum Gait Speed in Older Adults Over an 11-Year Follow-up. *The Journals of Gerontology: Series A*, *76*(4), 703–709. https://doi.org/10.1093/gerona/glaa196

167. Koster, A., Penninx, B. W. J. H., Bosma, H., Kempen, G. I. J. M., Harris, T. B., Newman, A. B., Rooks, R. N., Rubin, S. M., Simonsick, E. M., van Eijk, J. T. M., & Kritchevsky, S. B. (2005). Is There a Biomedical Explanation for Socioeconomic Differences in Incident Mobility Limitation? *The Journals of Gerontology: Series A*, *60*(8), 1022–1027. https://doi.org/10.1093/gerona/60.8.1022

168. House, J. S., Lantz, P. M., & Herd, P. (2005). Continuity and Change in the Social Stratification of Aging and Health Over the Life Course: Evidence From a Nationally Representative Longitudinal Study From 1986 to 2001/2002 (Americans' Changing Lives Study). *The Journals of Gerontology: Series B*, *60*(Special_Issue_2), S15–S26. https://doi.org/10.1093/geronb/60.Special_Issue_2.S15

169. Barha, C. K., Best, J. R., Rosano, C., Yaffe, K., Catov, J. M., & Liu-Ambrose, T. (2020). Sex-Specific Relationship Between Long-Term Maintenance of Physical Activity and Cognition in the Health ABC Study: Potential Role of Hippocampal and Dorsolateral Prefrontal Cortex Volume. *The Journals of Gerontology: Series A*, *75*(4), 764–770. https://doi.org/10.1093/gerona/glz093

170. Liu-Ambrose, T., Katarynych, L. A., Ashe, M. C., Nagamatsu, L. S., & Hsu, C. L. (2009). Dual-Task Gait Performance Among Community-Dwelling Senior Women: The Role of Balance Confidence and Executive Functions. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, *64A*(9), 975–982. https://doi.org/10.1093/gerona/glp063

171. Lajoie, Y., & Gallagher, S. P. (2004). Predicting falls within the elderly community: Comparison of postural sway, reaction time, the Berg balance scale and the Activities-specific Balance Confidence (ABC) scale for comparing fallers and non-fallers. *Archives of Gerontology and Geriatrics*, *38*(1), 11–26. https://doi.org/10.1016/s0167-4943(03)00082-7

172. Tiernan, C., Schwarz, D. J., & Goldberg, A. (2024). Associations of Usual and Fast Gait Speed With Physical Performance and Balance Confidence in Community-Dwelling Older Adults: Implications for Assessment. *Journal of Geriatric Physical Therapy*, *47*(4), 192. https://doi.org/10.1519/JPT.0000000000000397

173. Tsang, C. S. L., Lam, F. M. H., Leung, J. C. S., & Kwok, T. C. Y. (2023). Balance Confidence Modulates the Association of Gait Speed With Falls in Older Fallers: A Prospective Cohort Study. *Journal of the American Medical Directors Association*, *24*(12), 2002–2008. https://doi.org/10.1016/j.jamda.2023.05.025

174. Tagliabue, C. F., & Mazza, V. (2021). What Can Neural Activity Tell Us About Cognitive Resources in Aging? *Frontiers in Psychology*, *12*, 753423. https://doi.org/10.3389/fpsyg.2021.753423

175. Angel, L., Fay, S., Bouazzaoui, B., & Isingrini, M. (2011). Two Hemispheres for Better Memory in Old Age: Role of Executive Functioning. *Journal of Cognitive Neuroscience*, *23*(12), 3767–3777. https://doi.org/10.1162/jocn_a_00104

176. Huang, C.-M., Polk, T. A., Goh, J. O., & Park, D. C. (2012). Both left and right posterior parietal activations contribute to compensatory processes in normal aging. *Neuropsychologia*, *50*(1), 55–66. https://doi.org/10.1016/j.neuropsychologia.2011.10.022

177. Causse, M., Chua, Z., Peysakhovich, V., Del Campo, N., & Matton, N. (2017). Mental workload and neural efficiency quantified in the prefrontal cortex using fNIRS. *Scientific Reports*, *7*(1), 5222. https://doi.org/10.1038/s41598-017-05378-x

178. Montero-Odasso, M., Verghese, J., Beauchet, O., & Hausdorff, J. M. (2012). Gait and Cognition: A Complementary Approach to Understanding Brain Function and the Risk of Falling. *Journal of the American Geriatrics Society*, *60*(11), 2127–2136. https://doi.org/10.1111/j.1532-5415.2012.04209.x

179. Stern, Y. (2009). Cognitive reserve. *Neuropsychologia*, *47*(10), 2015–2028. https://doi.org/10.1016/j.neuropsychologia.2009.03.004

180. Steffener, J., Barulli, D., Habeck, C., O'Shea, D., Razlighi, Q., & Stern, Y. (2014). The Role of Education and Verbal Abilities in Altering the Effect of Age-Related Gray Matter Differences on Cognition. *PLOS ONE*, *9*(3), e91196. https://doi.org/10.1371/journal.pone.0091196

181. Giogkaraki, E., Michaelides, M. P., & Constantinidou, F. (2013). The role of cognitive reserve in cognitive aging: Results from the neurocognitive study on aging. *Journal of Clinical and Experimental Neuropsychology*, *35*(10), 1024–1035. https://doi.org/10.1080/13803395.2013.847906

182. O'Brien, C., & Holtzer, R. (2021). Cognitive Reserve Moderates Associations Between Walking Performance Under Single- and Dual-Task Conditions and Incident Mobility Impairment in Older Adults. *The Journals of Gerontology: Series A*, *76*(10), e314–e320. https://doi.org/10.1093/gerona/glab178

183. Shaked, D., Millman, Z. B., Moody, D. L. B., Rosenberger, W. F., Shao, H., Katzel, L. I., Davatzikos, C., Gullapalli, R. P., Seliger, S. L., Erus, G., Evans, M. K., Zonderman, A. B., & Waldstein, S. R. (2019). Sociodemographic disparities in corticolimbic structures. *PLOS ONE*, *14*(5), e0216338. https://doi.org/10.1371/journal.pone.0216338

184. Liu, S., Rosso, A. L., Baillargeon, E. M., Weinstein, A. M., Rosano, C., & Torres-Oviedo, G. (2023). Novel attentional gait index reveals a cognitive ability-related decline in gait automaticity during dual-task walking. *Frontiers in Aging Neuroscience*, *15*, 1283376. https://doi.org/10.3389/fnagi.2023.1283376

185. Webber, S. C., Porter, M. M., & Menec, V. H. (2010). Mobility in Older Adults: A Comprehensive Framework. *The Gerontologist*, *50*(4), 443–450. https://doi.org/10.1093/geront/gnq013

186. Cabeza, R., Albert, M., Belleville, S., Craik, F. I. M., Duarte, A., Grady, C. L., Lindenberger, U., Nyberg, L., Park, D. C., Reuter-Lorenz, P. A., Rugg, M. D., Steffener, J., & Rajah, M. N. (2018). Maintenance, reserve and compensation: The cognitive neuroscience of healthy ageing. *Nature Reviews Neuroscience*, *19*(11), 701–710. https://doi.org/10.1038/s41583-018-0068-2

187. Verghese, J., Wang, C., Ayers, E., Izzetoglu, M., & Holtzer, R. (2017). Brain activation in high-functioning older adults and falls. *Neurology*, *88*(2), 191–197. https://doi.org/10.1212/WNL.0000000000003421

188. Colón-Emeric, C. S., McDermott, C. L., Lee, D. S., & Berry, S. D. (2024). Risk Assessment and Prevention of Falls in Older Community-Dwelling Adults: A Review. *JAMA*, *331*(16), 1397–1406. https://doi.org/10.1001/jama.2024.1416

189. Shumway-Cook, A., Woollacott, M., Kerns, K. A., & Baldwin, M. (1997). The effects of two types of cognitive tasks on postural stability in older adults with and without a history of falls. *The Journals of Gerontology. Series A, Biological Sciences and Medical Sciences*, *52*(4), M232-240. https://doi.org/10.1093/gerona/52a.4.m232

190. Thorpe, R. J., Jr, Koster, A., Kritchevsky, S. B., Newman, A. B., Harris, T., Ayonayon, H. N., Perry, S., Rooks, R. N., Simonsick, E. M., & for the Health, A., and Body Composition Study. (2011). Race, Socioeconomic Resources, and Late-Life Mobility and Decline: Findings From the Health, Aging, and Body Composition Study. *The Journals of Gerontology: Series A*, *66A*(10), 1114–1123. https://doi.org/10.1093/gerona/glr102

Appendices

Appendix A: LOWESS Theory, Parameter Selection & Optimization

Locally weighted scatterplot smoothing (LOWESS) is a nonparametric approach to scatterplot smoothing.[89,90] For a scatterplot with data points (x_i, y_i), $i = 1 \ldots n$, a fitted value, x_k, is determined using weighted least squares.[89,90] The weighting for a data point (x_i, y_i), is large if x_i is close to x_k and small if it is not. An optional additional robustness procedure guards against distortions in smoothing from outlying datapoints.[89] This smoothing procedure, based on Cleveland (1979), involves a weighting function W, wherein for each x_i, weights $w_k(x_i)$ are defined for all x_k, $k = 1 \ldots n$ using W.[89] The fitted value, $\hat{y}_i$, at each x_i is the fitted value of a polynomial fitted to the data using weighted least squares with weights $w_k(x_i)$. Each (x_i, y_i) has a different weight based on the size of the residual of $y_i - \hat{y}_i$. Large residuals have small weights and small residuals have large weights. The assumption of smoothness allows for points in the neighborhood of (x_i, y_i) to be used in forming $\hat{y}_i$. For a weight function W(x), which decreases for increasing x, the weights $w_k(x_i)$ decrease and distance of x_k from x_i increase. Thus, datapoints with x close to x_i have a large role in determining $\hat{y}_i$ and far away points have a lesser weight.

There are four values in the LOWESS algorithm that must be specified. First, the order of the polynomial being locally fit to each point on the scatterplot must be selected. In most cases, a first order polynomial, $d = 1$, provides adequate smoothing.[89] Second, the weight function must be selected, most often a tricube function will provide optimal smoothing.[89,90] Third, the number of robustness iterations of the algorithm must be specified, if robustness against outlying datapoints is needed, two iterations are often sufficient to prevent smoothing distortions.[89]

Finally, the smoothing parameter, f, must be optimized. Increases in f often increase the smoothness of points. Altering the spanning parameter determines the neighborhood of

inferential points. Increasing f increases the neighborhood, and often also increases smoothness. When selecting the spanning parameter, the goal is to pick a value as large as possible to minimize variance in smoothed points without distorting the data pattern. Usually, factors between 0.2-0.8 are accepted, with a starting value of 0.5 when the pattern within the data is not clear.[89] The selection of an appropriate spanning parameter may be through the minimization of error, especially when smoothed data will be used for prediction analyses.[90] Cross-validation (CV) is a popular selection to identify the best smoothing parameter value.[90,91] This value involves the division of data into two subsamples. One subsample will act as a predictor and its performance assessed based on predictions against the other subsample. The division of sample size n into a "construction/training" subsample (n-1) and a "validation/test" subsample (1) in all (n) possible ways. The mean squared error (MSE) of the CV (CV-MSE) is often used as the criterion for the selection of the appropriate spanning parameter, wherein the best choice spanning parameter minimizes the CV-MSE.[92]

In the analyses applied in this work, d and W were used as per Cleveland (1979) recommendations.[89,90] No robustness iterations were used as CV was not meaningfully improved as prediction error was not consistently minimized with iterations based on MSE, especially at higher f values, and therefore there was no benefit of outlier robustness iterations. To optimize the spanning parameter and the degree of smoothing applied across datapoints, values from 0.6-0.8 were assessed via 10-fold CV for each PFC and gait outcome separately per condition. LOWESS is fitted to k-1 (9) training/construction subsamples and prediction for the held-out test/validation subsample is generated from the training subsamples via interpolation. MSE was calculated on the held-out test/validation subsample predictions and averaged across all 10 repetitions of this analysis to estimate sample prediction error for each condition per measure.

This procedure ensures that test observations are not influencing the smoothing, as smoothing is only applied to the training subgroup and parameter optimization remains unbiased.

Candidate f values of 0.7, 0.75, and 0.8 produced nearly identical CV-MSE (**Table A.1** for gait velocity and **Table A.2** for PFC activity). As per Cleveland (1979), the largest smoothing parameter that does not distort the data should be selected and, as per Clark (1980), the spanning parameter should minimize MSE.[89,92] Thus, this analysis applied f = 0.8 across measures and all conditions. No outlier robustness reweighting was applied. During spanning parameter selection, all candidate f values were run with iterations of 0 and 1.

Table A.1: Cross-validation outcomes for gait velocity across conditions for f = 0 and 1 and at 0 and 1 robustness iteration (iter). Final value chosen is shown in bold.

Condition	f	Iter	MSE Mean	MSE SD	MSE SE
W	0.7	0	364.007	152.979	48.376
	0.7	1	361.635	145.646	46.057
	0.75	0	364.276	152.045	48.081
	0.75	1	362.264	145.394	45.978
	0.8	**0**	**363.249**	**151.143**	**47.796**
	0.8	1	361.413	144.914	45.826
WD	0.7	0	388.686	138.313	43.738
	0.7	1	390.597	137.916	43.613
	0.75	0	388.262	137.223	43.394
	0.75	1	390.617	137.220	43.393
	0.8	**0**	**387.163**	**137.072**	**43.346**
	0.8	1	389.577	137.285	43.413
WO	0.7	0	357.235	155.139	49.059
	0.7	1	353.462	143.079	45.246
	0.75	0	360.972	154.904	48.985
	0.75	1	357.588	143.501	45.379
	0.8	**0**	**360.789**	**153.847**	**48.651**
	0.8	1	357.759	143.390	45.344
WOD	0.7	0	331.396	122.352	38.691
	0.7	1	331.109	118.107	37.349
	0.75	0	333.467	120.190	38.007
	0.75	1	333.240	116.056	36.700
	0.8	**0**	**332.939**	**118.870**	**37.590**
	0.8	1	332.868	115.066	36.387

MSE is the mean squared error. SD is standard deviation.

Table A.2: Cross-validation outcomes for PFC activity across conditions for f = 0 and 1 and at 0 and 1 robustness iteration (iter). Final value chosen is shown in bold.

Condition	f	iter	MSE Mean	MSE (SD)	MSE SE
WD	0.7	0	0.964	0.671	0.212
	0.7	1	0.972	0.730	0.231
	0.75	0	0.958	0.665	0.210
	0.75	1	0.967	0.726	0.230
	0.8	**0**	**0.957**	**0.665**	**0.210**
	0.8	1	0.966	0.727	0.230
WO	0.7	0	1.047	0.758	0.240
	0.7	1	1.055	0.841	0.266
	0.75	0	1.035	0.751	0.237
	0.75	1	1.042	0.833	0.263
	0.8	**0**	**1.032**	**0.751**	**0.238**
	0.8	1	1.038	0.834	0.264
WOD	0.7	0	1.002	0.806	0.255
	0.7	1	1.024	0.843	0.267
	0.75	0	0.997	0.806	0.255
	0.75	1	1.021	0.846	0.267
	0.8	**0**	**0.995**	**0.807**	**0.255**
	0.8	1	1.020	0.848	0.268

MSE is the mean squared error. SD is standard deviation.

Appendix B: Piecewise Regression Grid Search & Error Minimization

In the grid search with a resolution of 200 in a search width of approximately 60 years (participants were aged 18-79), there were 121-241 candidate τ. This provides 0.25-0.5 candidates per year across the search interval. Evaluating a predefined set of candidate parameter values in this way and selecting the optimal value from the candidates (ie a grid search) is common in machine learning practices and is appropriate for a change-point analysis to evaluate candidates and select the best τ based on likelihoods, such as AICc.[94–96] The grid resolution at 200 allows τ to be assessed in 0.3 year increments across the age range, which is a reasonable search range for a piecewise modelling as greater a range of change points can create a smoother curve to select the optimal candidate change-point.[93]

For each candidate τ identified in the grid search, a piecewise regression model was fit and the corrected Akaike Information Criterion (AICc) was computed as:

$$AICc = AIC + \frac{2k(k+1)}{n-k-1} \qquad \text{B-1}$$

where k is the number of parameters (=3) and n is sample size. AICc was chosen instead of AIC or BIC to reduce small-sample bias in information-criterion model selection.

In the output, τ is the selected change-point age and there is a reported model R^2, pre- and post- change slopes, and hinge-term p-values recorded. Full AICc profiles and fitted hinge plots were saved for diagnostic evaluation of change-point stability.

Appendix C: Sex Differences in Gait Velocity

There were significant two-sided independent t-tests for all gait velocity conditions such that female participants walked slower than male participants, as shown in **Figure C.1** (W $p = 0.003$, WD $p < 0.001$, WO $p = 0.002$, WOD $p = 0.005$). These anthropometric and fundamental biomechanical differences between males and females are consistent in the literature. Females tend to be shorter in height and have a smaller leg length and tend to increase step cadence to increase velocity. On the other hand, males tend to be taller with longer leg length and will take larger steps (i.e. increase step length) in order to increase velocity.

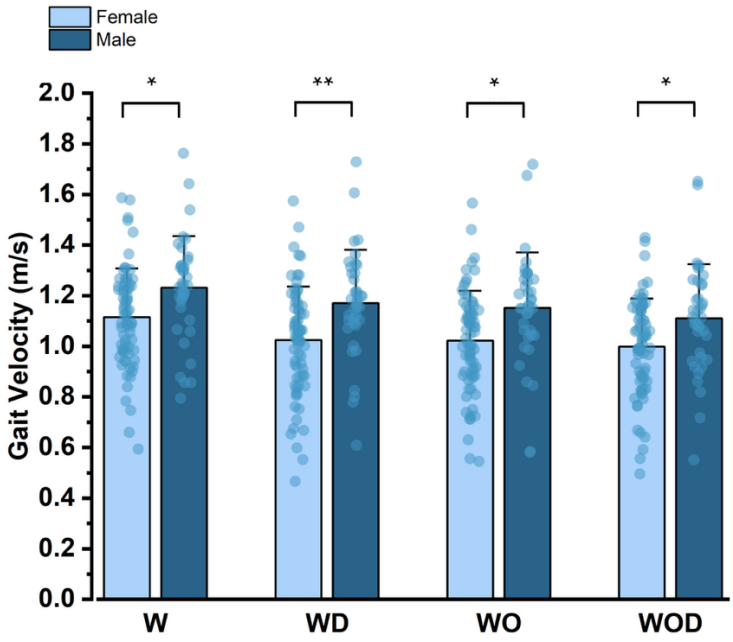


Figure C.1. Bar graph with overlayed participant data points showing gait velocity across conditions for female (light blue) and male (dark blue) participants. * $p \leq 0.05$, ** $p < 0.01$.

Appendix D: Hinge Model & Piecewise Regression Outputs

Since piecewise regressions and hinge models were run on LOWESS smoothed values, interpretation of regression outcomes must be done carefully. The fitted model is used to approximate a smooth curve with two straight lines, so the hinge model will explain almost all remaining variance in the R^2 value. In this case, an R^2 value of 0.99 does not mean that age explains 99% of the variability in brain activity/gait, but rather that two straight lines explain 99% of the LOWESS curve. Thus, the piecewise linear approximation fits a smooth trend, not about biological effect strength. For these LOWESS-based regressions, R^2 is not interpretable as effect size, and the p-values are not about inferentially, but summarize shape and slope changes.

Regressions were run on LOWESS data to identify meaningful and significant change in linearity of neural and behavioural outcomes across the 18-79 age range. The hinge regression run on raw data, due to its dispersion, was particularly biased to the edges of the data. This is common for segmented regression frameworks and produced statistical artefact change-points that reflected error minimization but not true biological change (**Table D.1**). Thus, hinge regression on the LOWESS smoothed data points allowed for the identification of a true change in biological patterns. **Tables D1-4** shows the difference in hinge regression outputs when run on LOWESS vs RAW values. The LOWESS smoothing removes most of the variance within raw values, based on the increase in R^2 values. The RAW hinge regression outcomes are informative for inference and effect magnitudes, wherein the breakpoint can be in interpreted with caution due to prevalence of edge bias, the pre- and post- change-point slope, the hinge p-value, and the change in AICc vs linear modelling. The LOWESS hinge outputs are a trend-level breakpoint estimate with more descriptive slopes for participant patterns, but the R^2 and p-values here do not relate to statistical outputs of the actual sample.

Table D.1. Nonparametric hinge regression outputs identifying the change-points (t) and model fit statistics for both LOWESS smoothed and RAW data across neurobehavioural outcomes per condition.

		Input Data Type	*τ*	*df (residual)*	*sigma*	*RMSE*	*R^2*	*Adj. R^2*	*F*	*df1*	*df2*	*p*	*AICc*
Gait Velocity	W	LOWESS	53.558	110	0.199	0.196	0.999	0.999	101803.390	2	110	<0.001	-39.066
		RAW	70.111	110	18.347	18.102	0.199	0.184	13.641	2	110	<0.001	983.405
	WD	LOWESS	52.638	110	0.203	0.201	0.999	1.000	179887.097	2	110	<0.001	-34.047
		RAW	43.136	110	18.785	18.534	0.292	0.279	22.686	2	110	<0.001	988.726
	WO	LOWESS	54.784	110	0.247	0.243	0.999	0.999	104889.742	2	110	<0.001	9.644
		RAW	66.126	110	18.142	17.900	0.292	0.279	22.714	2	110	<0.001	980.863
	WOD	LOWESS	55.397	110	0.205	0.202	0.999	1.000	135331.638	2	110	<0.001	-32.580
		RAW	66.126	110	17.431	17.198	0.286	0.273	22.023	2	110	<0.001	971.820
PFC Ratio	WD	LOWESS	52.945	110	0.011	0.011	0.996	0.996	15475.973	2	110	<0.001	-694.143
		RAW	53.251	110	0.973	0.960	0.042	0.024	2.382	2	110	0.097	319.718
	WO	LOWESS	56.317	110	0.005	0.005	0.994	0.994	8864.373	2	110	<0.001	-858.936
		RAW	75.935	110	1.007	0.994	0.025	0.008	1.430	2	110	0.243	327.492
	WOD	LOWESS	52.638	110	0.012	0.012	0.995	0.995	10992.085	2	110	<0.001	-664.775
		RAW	74.402	110	0.994	0.981	0.048	0.031	2.762	2	110	0.066	324.534

Model fit statistics: residual degrees of freedom (df), residual standard deviation, root mean square error (RMSE), the R^2, F-statistic (df1 and df2), model p-value, and correct Akaike Information Criterion (AICc). Models were fit separately for each measure and condition.

Table D.2. Hinge regression coefficient estimates for LOWESS smoothed and RAW data across neurobehavioural measures and conditions.

Measure	*Condition*	*Data Type*	*τ*	*Intercept*						
				Estimate	**SE**	**t statistic**	**df**	**P**	**CI low**	**CI high**
Gait Velocity	W	LOWESS	53.558	131.917	0.075	1751.149	110	<0.001	131.767	132.067
		RAW	70.111	133.460	5.523	24.165	110	<0.001	122.515	144.405
	WD	LOWESS	52.638	127.455	0.078	1630.244	110	<0.001	127.300	127.610
		RAW	43.136	116.775	9.462	12.341	110	<0.001	98.023	135.527
	WO	LOWESS	54.784	125.792	0.092	1372.969	110	<0.001	125.611	125.974
		RAW	66.126	126.302	5.723	22.067	110	<0.001	114.959	137.644
	WOD	LOWESS	55.397	119.939	0.075	1593.117	110	<0.001	119.790	120.089
		RAW	66.126	121.071	5.499	22.017	110	<0.001	110.173	131.968
PFC Ratio	WD	LOWESS	52.945	1.392	0.004	332.241	110	<0.001	1.384	1.401
		RAW	53.251	1.443	0.370	3.896	110	<0.001	0.709	2.176
	WO	LOWESS	56.317	1.555	0.002	811.805	110	<0.001	1.551	1.559
		RAW	75.935	1.520	0.284	5.351	110	<0.001	0.957	2.083
	WOD	LOWESS	52.638	1.656	0.005	345.133	110	<0.001	1.646	1.665
		RAW	74.402	1.360	0.286	4.750	110	<0.001	0.792	1.927

Regression parameters include the intercept, pre- and post- break point slope, hinge term coefficients with corresponding standard error (SE), test statistics, and confidence intervals. Models were fit separately for each measure and condition

Table D.3. Estimated change-points from hinge regression of each measure per condition on LOWESS smoothed and RAW data separately.

Measure	*Condition*	*Data Type*	*τ*	*Change-point age (τ)*						
				Estimate	**SE**	**t statistic**	**df**	**P**	**CI low**	**CI high**
Gait Velocity	W	LOWESS	53.558	-0.235	0.002	-126.182	110	<0.001	-0.239	-0.232
		RAW	70.111	-0.291	0.106	-2.740	110	0.007	-0.501	-0.080
	WD	LOWESS	52.638	-0.249	0.002	-126.432	110	<0.001	-0.252	-0.245
		RAW	43.136	0.184	0.293	0.627	110	0.532	-0.397	0.765
	WO	LOWESS	54.784	-0.261	0.002	-117.305	110	<0.001	-0.265	-0.257
		RAW	66.126	-0.281	0.116	-2.416	110	0.017	-0.511	-0.051
	WOD	LOWESS	55.397	-0.200	0.002	-110.355	110	<0.001	-0.203	-0.196
		RAW	66.126	-0.237	0.112	-2.122	110	0.036	-0.458	-0.016
PFC Ratio	WD	LOWESS	52.945	-0.005	0.000	-48.857	110	<0.001	-0.005	-0.005
		RAW	53.251	-0.007	0.009	-0.756	110	0.451	-0.025	0.011
	WO	LOWESS	56.317	0.000	0.000	-8.528	110	<0.001	0.000	0.000
		RAW	75.935	0.001	0.005	0.146	110	0.885	-0.009	0.011
	WOD	LOWESS	52.638	-0.010	0.000	-83.828	110	<0.001	-0.010	-0.010
		RAW	74.402	0.000	0.005	-0.091	110	0.928	-0.011	0.010

For each measure, condition, and data-type, the estimated change-point is reported along with its standard error (SE), t-statistic, degrees of freedom (df), p-value, and the 95% CI range. These values indicate the statistical significance and precision of the estimated age at which neurobehavioural changes occur.

Table D.4. Hinge term parameter estimates from piecewise linear regression models to identify change-point age across conditions and measures. The hinge term represents the change in slope following the change-point, quantifying the difference between pre- and post- change-point slopes.

Measure	*Condition*	*Data Type*	*τ*	*Hinge*						
				Estimate	**SE**	**t statistic**	**df**	**P**	**CI low**	**CI high**
Gait Velocity	W	LOWESS	53.558	-0.473	0.004	-115.089	110	<0.001	-0.481	-0.465
		RAW	70.111	-2.002	0.903	-2.217	110	0.029	-3.791	-0.213
	WD	LOWESS	52.638	-0.764	0.004	-183.779	110	<0.001	-0.772	-0.756
		RAW	43.136	-1.222	0.457	-2.672	110	0.009	-2.129	-0.316
	WO	LOWESS	54.784	-0.717	0.005	-138.360	110	<0.001	-0.727	-0.706
		RAW	66.126	-1.885	0.597	-3.158	110	0.002	-3.068	-0.702
	WOD	LOWESS	55.397	-0.805	0.004	-185.488	110	<0.001	-0.814	-0.797
		RAW	66.126	-1.920	0.573	-3.349	110	0.001	-3.057	-0.784
PFC Ratio	WD	LOWESS	52.945	0.029	0.000	127.587	110	<0.001	0.028	0.029
		RAW	53.251	0.034	0.020	1.695	110	0.093	-0.006	0.074
	WO	LOWESS	56.317	0.009	0.000	81.883	110	<0.001	0.009	0.010
		RAW	75.935	0.307	0.193	1.591	110	0.114	-0.075	0.688
	WOD	LOWESS	52.638	0.034	0.000	133.758	110	<0.001	0.034	0.035
		RAW	74.402	0.257	0.117	2.202	110	0.030	0.026	0.488

For each measure, condition, and data type (LOWESS and RAW), the analysis was run separately and the hinge coefficient is reported alongside its standard error (SE), t-statistic, degrees of freedom (df), p-value, and 95% confidence interval (CI). These estimates indicate whether the rate of change in the outcome significantly differs before and after the change point.

Appendix E: Generalized Additive Model & Counterfactual Frameworks

Given the evidence for a nonlinear relationship between PFC activity and gait velocity in Data Chapter 1, the use of a GAM in the mediator-outcome relationship within mediation analysis allows for non-linear mediation while maintaining a linear, parametric effect of X on Y.[151] The predictor was retained as a linear term to preserve interpretability of counterfactual contrasts. functional relevance of PFC activity to gait performance was evaluated via the significance of the smooth term $f(M)$ in the outcome model. For each mediation run, the estimated degrees of freedom (EDF) and p-value associated with the smooth term were extracted and reported. A significant smooth term was interpreted as evidence of a nonlinear association between PFC activity and gait velocity, conditional on the predictor.

The shape of the GAM requires estimating the smoothed relationship between PFC activity and gait velocity after accounting for age, so all other variables/predictors must be held constant, usually at the sample median. This is referred to as the "hold value" and allows visualization of the partial effect prediction from the GAM. The predicted values are based on age and the measure of interest via smoothed model s(M). To produce a clean curve with PFC activity on the x-axis age must be held constant while the PFC activity ratio value is altered. Age is held at the "hold age" for all predictions and then the ratio is applied across the age range to predict velocity at each ratio. Thus, the shape of the GAM is determined by ratio and velocity while accounting for age, and the vertical position of the curve depends on the hold value but the shape of the curve from s(PFC) remains the same.

To allow for nonlinear brain–behaviour relationships, gait velocity was modeled as a function of the predictor and a smooth function of the mediator using a generalized additive model:

$$Y = \beta_0 + \beta_1 X + f(M) + \varepsilon_Y \quad \text{E-1}$$

where $f(M)$denotes a penalized regression spline. Models were estimated using restricted maximum likelihood (REML) smoothing parameter selection. The basis dimension of the smooth term was fixed at $k = 5$, limiting the maximum complexity of the nonlinear function while allowing sufficient flexibility to capture compensatory or threshold-like relationships between PFC activity and gait performance.

Since the M-Y relationship is modelled non-linearly via GAM, direct and indirect effects cannot be using traditional approaches to regression coefficients. Instead, mediation effects were estimated using a counterfactual framework with nonparametric bootstrap resampling (B = 10,000) to general empirical distributions of TE, DE, and IE. Bootstrapping estimates the sampling disruption of the IE and DE by repeatedly resampling subjects, refitting both models, and recalculating counterfactual contrasts. Resampling was set at 10,000 to ensure stable CI boundaries and p-values.

Across 3 conditions and 4 X variables, 12 mediations were modelled with 120,000 GAM fits. Counterfactual design was required as the same participant cannot be observed both old and young, at different levels of education, or at different simultaneous levels of PFC activity, for example. The counterfactual design allows the mediation to be defined as: what Y would have been if X were different than observed with and without a fixed M to give potential counterfactual outcomes under the Neyman causal framework.[150,154]

This analysis contains four counterfactual quantifies using control (Xo) and treatment (X_1) values. Mediation needs to compare two levels of X, so when x is continuous the control value represents the 25th percentile and treatment the 75th percentile. So, effects are interpreted as expected changes in gait when moving from a relatively low to a relatively high value of X. For a categorical variable, control is the lowest category, and treat is the highest category. Within the causal mediation, M(X) and Y(x,m) are defined for each participant representing the mediator of X is set to x and the outcome if X is set to x and the mediator is fixed to m, respectively. If $X_0$and X_1denote control and treatment values of the predictor, $M(x)$, the mediator value predicted under exposure x, and $Y(x, m)$, wherein the outcome predicted under exposure x with mediator fixed at m. Three counterfactual outcomes were computed per participant:

- $Y(X_1, M(X_1))$
- $Y(X_1, M(X_0))$
- $Y(X_0, M(X_0))$

Bootstrap interference used for uncertainty quantification via nonparametric case resampling with replacement within each run (condition x X). Each bootstrap replicate resamples, refits lm(M~X) and refits the GAM to recompute IE, DE, TE, and PM via counterfactual predictions. For each bootstrap sample, mediator values were predicted under both control and treat, these predicted values are then inserted into the outcome model to generate counterfactual predictions. Effects were computed by averaging individual level differences across participants.

Appendix F: Mediation Regression Model

The linear regression model outcomes for Step 1 and Step 2 are shown per condition for level of education (**Figure F.1**), location of participation (**Figure F.2**), and age (**Figure F.3**).

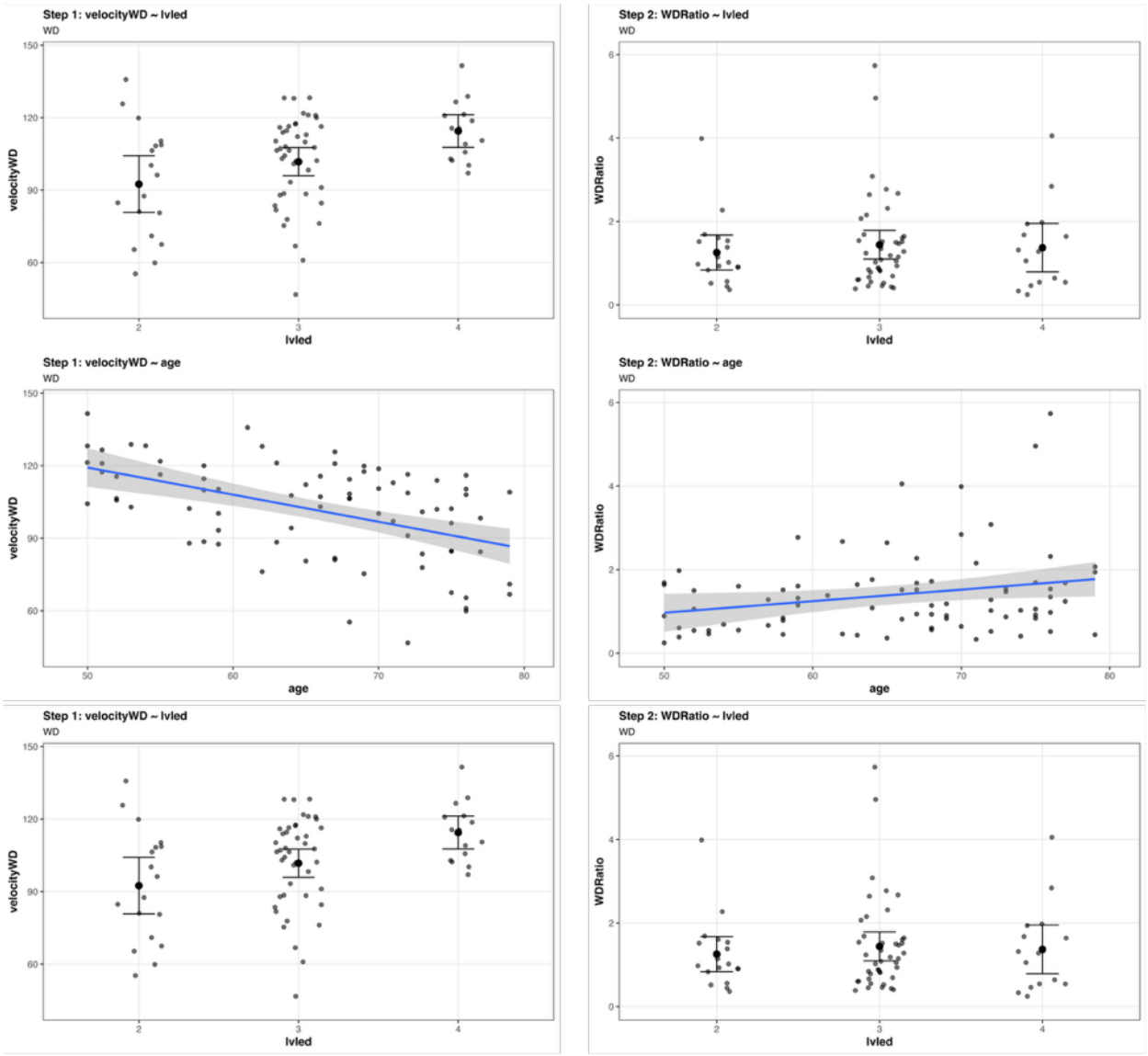


Figure F.1. Step 1 (left) and Step 2 (right) for location of participation (top), age (middle), and level of education (bottom) in the WD condition.

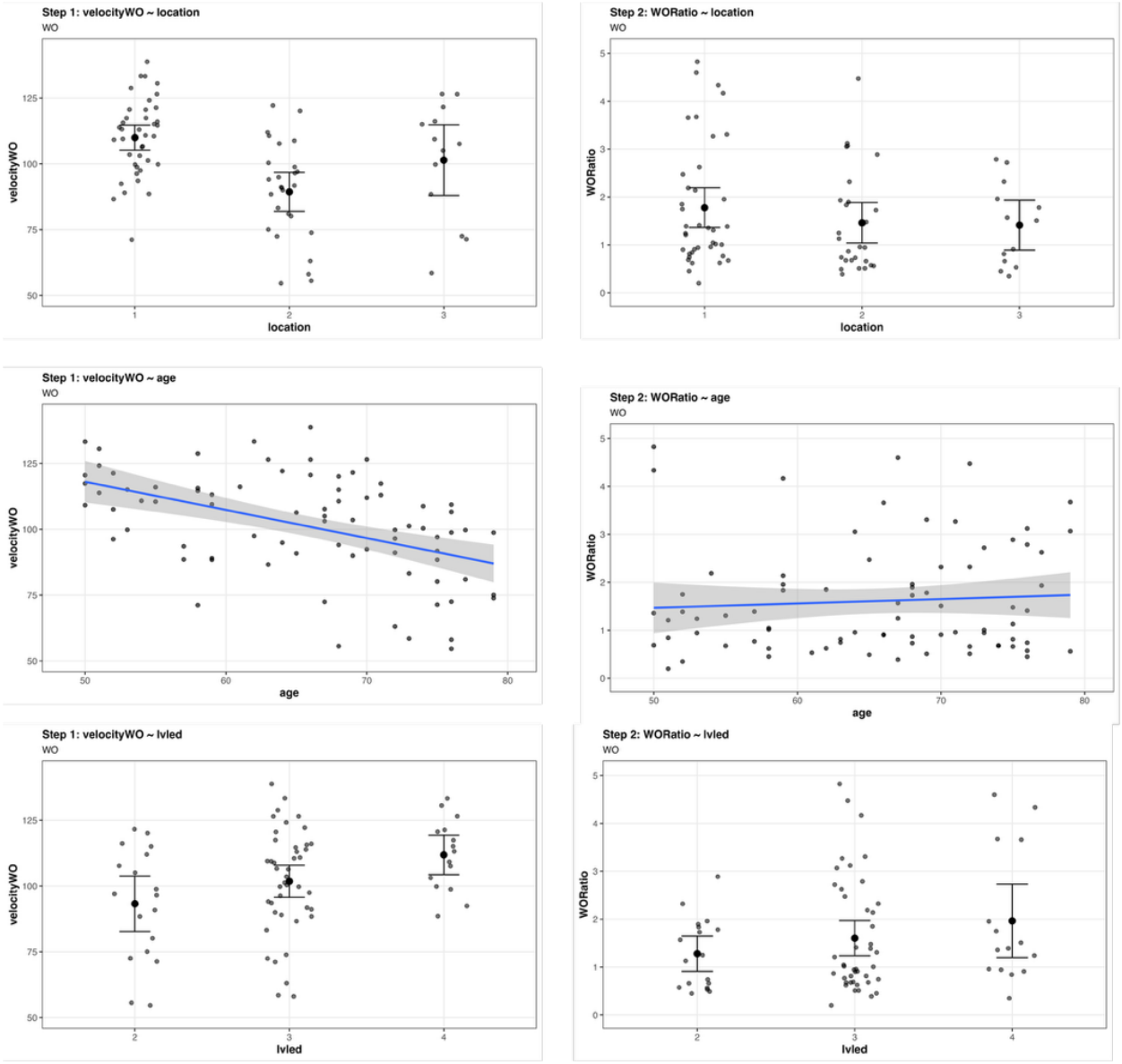


Figure F.2. Step 1 (left) and Step 2 (right) for location of participation (top), age (middle), and level of education (bottom) in the WO condition.

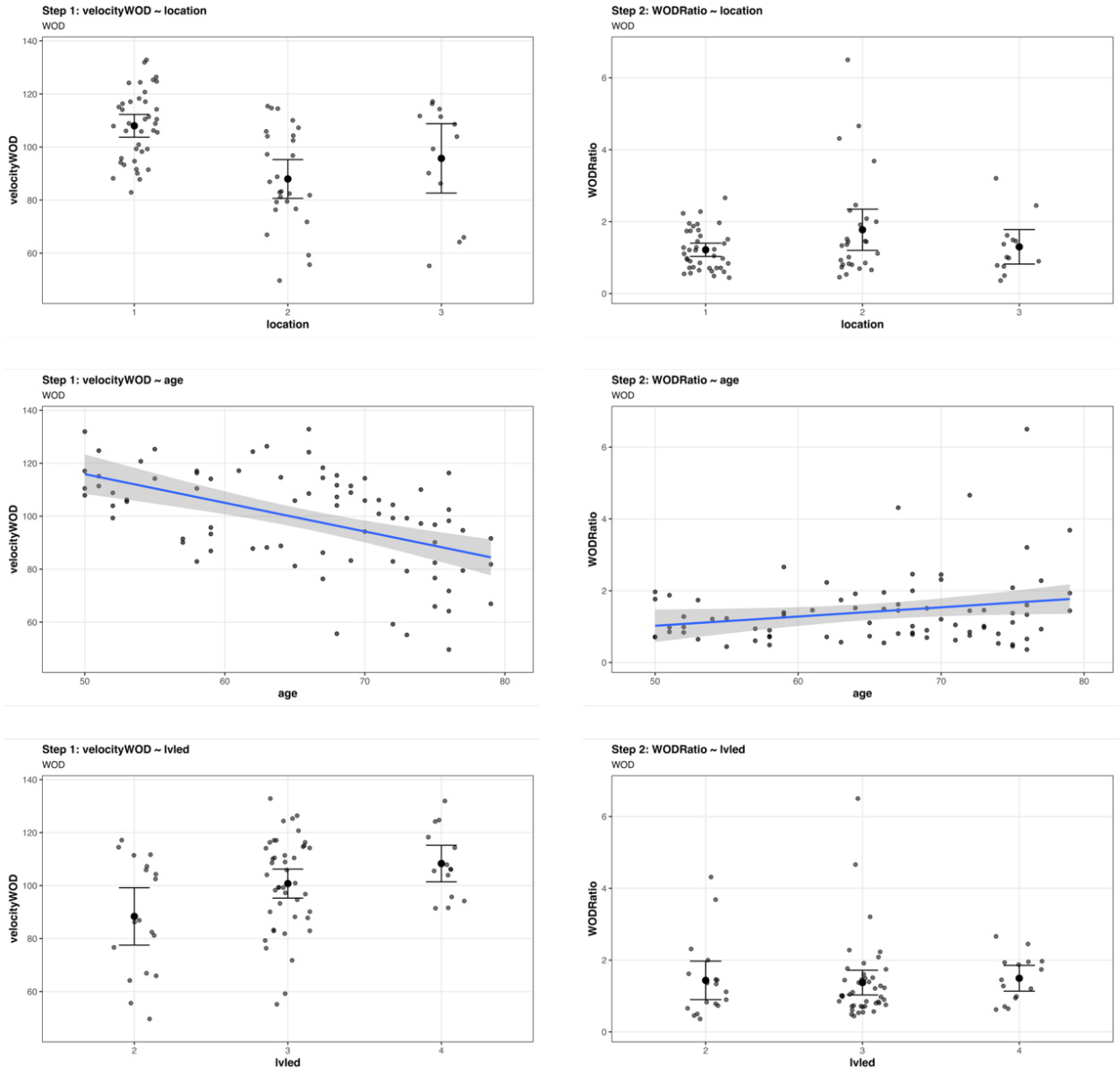


Figure F.3. Step 1 (left) and Step 2 (right) for location of participation (top), age (middle), and level of education (bottom) in the WOD condition.

Appendix G: Mediation Counterfactual Bootstrap Distribution

Bootstrap resampling with 10,000 resamples was used in mediation analyses to calculate the DE, TE, and IE values. Bootstrap density plots per condition and sociodemographic predictor are shown in **Figure G.1**.

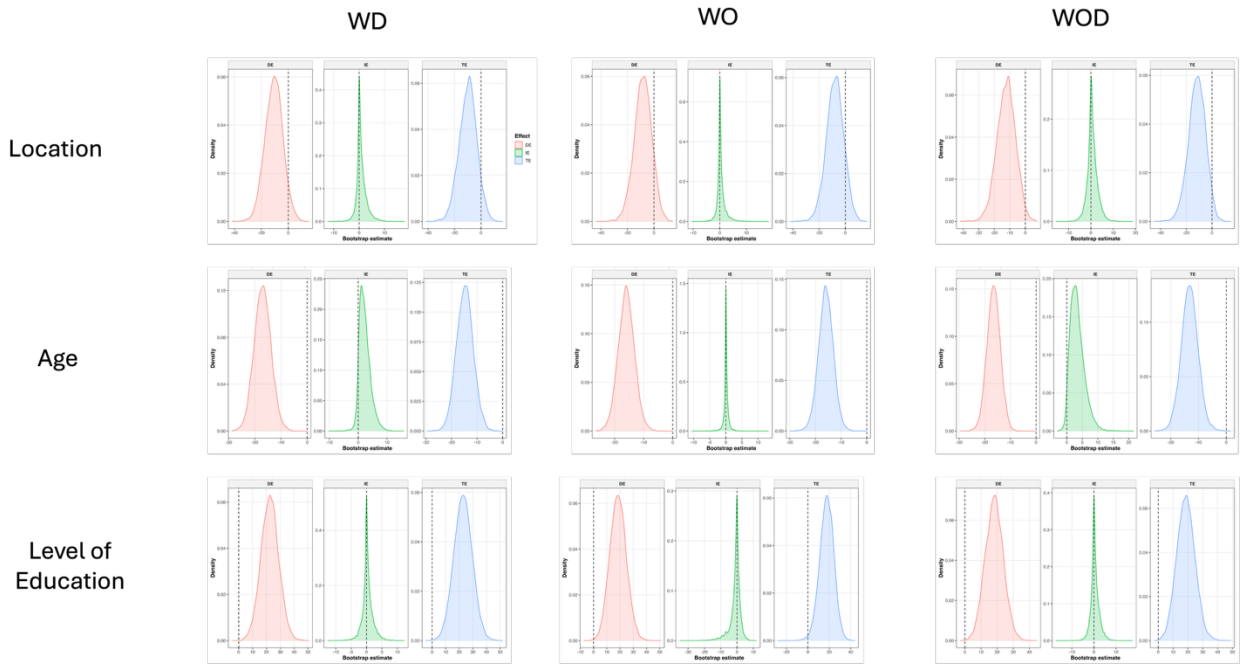


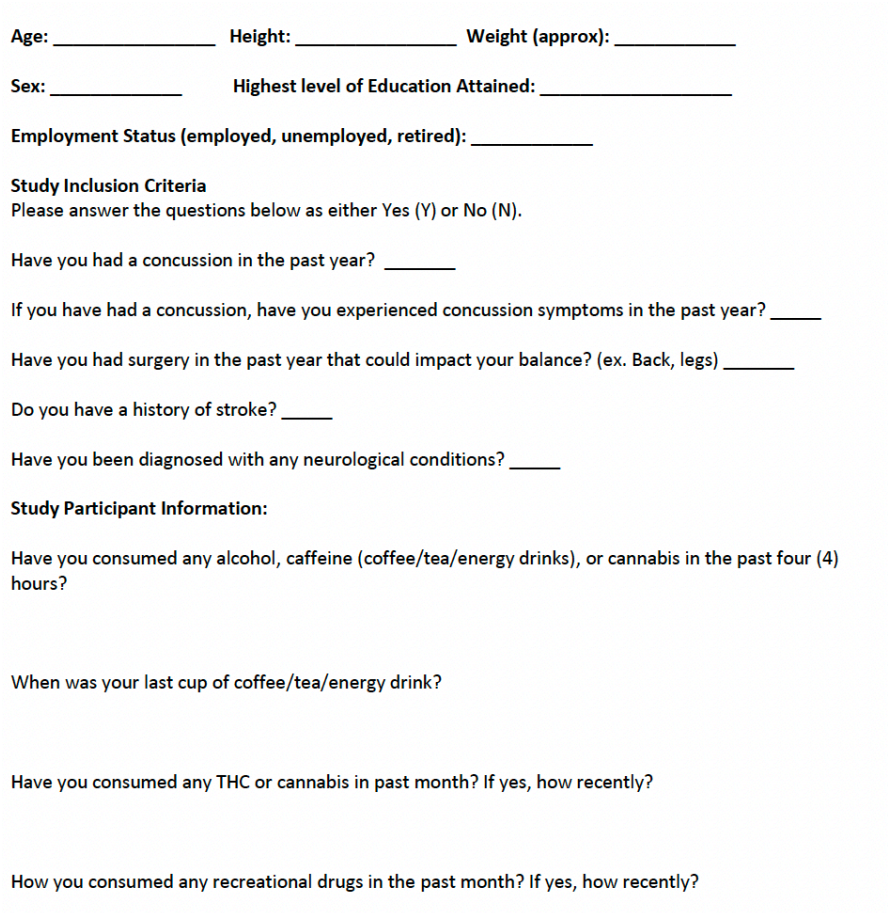
Figure G.1. Bootstrap resampling distribution density plots for each condition and X-value in mediation analyses.

Appendix H: Sociodemographic & Lifestyle Forms

This set of Appendices displays participant questionnaires.

H.1 Sociodemographic Information & Medical History

The following forms were used to collect sociodemographic and medical history forms that comprise **Table 2.1** and **Table 3.1**, and provided information for subsequent analyses. **Figure H.1.1** shows sociodemographic/study inclusion criteria. **Figure H.2.1** shows the medical history form, and **Figure H.3.1** presents the ABC scale.



Age: _____ Height: _____ Weight (approx): _____

Sex: _____ Highest level of Education Attained: _____

Employment Status (employed, unemployed, retired): _____

Study Inclusion Criteria
Please answer the questions below as either Yes (Y) or No (N).

Have you had a concussion in the past year? _____

If you have had a concussion, have you experienced concussion symptoms in the past year? _____

Have you had surgery in the past year that could impact your balance? (ex. Back, legs) _____

Do you have a history of stroke? _____

Have you been diagnosed with any neurological conditions? _____

Study Participant Information:

Have you consumed any alcohol, caffeine (coffee/tea/energy drinks), or cannabis in the past four (4) hours?

When was your last cup of coffee/tea/energy drink?

Have you consumed any THC or cannabis in past month? If yes, how recently?

How you consumed any recreational drugs in the past month? If yes, how recently?

Figure H.1.1. Study inclusion criteria and demographic questionnaire.

H.2 Fall History

In the <u>past year</u>, have you had any fall including a slip or trip in which you lost your balance and landed on the floor or ground or lower level?

Yes [ ] No [ ]

If you have had <u>no falls</u> please skip to the next form, otherwise please continue.

If yes, how many times has this happened?

Once [ ] Twice [ ] Three or more [ ]

1. Where have you fallen?

<u>Inside:</u>

On the one ground level	Yes [ ]	No [ ]
Getting out of bed	Yes [ ]	No [ ]
Getting out of a chair	Yes [ ]	No [ ]
Using the shower/bath	Yes [ ]	No [ ]
Using the toilet	Yes [ ]	No [ ]
Walking up or down stairs	Yes [ ]	No [ ]

<u>Home entrances or in the garden:</u>

Walking up or down a step/stairs	Yes [ ]	No [ ]
On the one ground level (e.g. pathway)	Yes [ ]	No [ ]
In the garden	Yes [ ]	No [ ]

<u>Away from home:</u>

On a footpath	Yes [ ]	No [ ]
On a kerb/gutter	Yes [ ]	No [ ]
In a public building	Yes [ ]	No [ ]
Getting out of a vehicle	Yes [ ]	No [ ]
In another person's home	Yes [ ]	No [ ]

Other (please describe):

2. How did you fall?

I tripped	[ ]
I slipped	[ ]
I lost my balance	[ ]
My legs gave way	[ ]
I felt faint	[ ]
I felt giddy/dizzy	[ ]
I am not sure	[ ]

3. As a result of this fall or falls did you suffer any injuries?

Yes [ ] No [ ]

If yes, what type of injuries did you suffer?

Bruises	[ ]
Cuts/grazes	[ ]
Broken wrist	[ ]
Broken hip	[ ]
Broken ribs	[ ]
Back pain	[ ]

Other (please specify):

Figure H.2.1. Fall history form.

H.3 Activities Specific Balance Confidence Scale

The Activities-specific Balance Confidence (ABC) Scale*

Instructions to Participants: For each of the following activities, please indicate your level of confidence in doing the activity without losing your balance or becoming unsteady from choosing one of the percentage points on the scale from 0% to 100% If you do not currently do the activity in question, try and imagine how confident you would be if you had to do the activity. If you normally use a walking aid to do the activity or hold onto someone, rate your confidence as if you were using these supports.

0%	10	20	30	40	50	60	70	80	90	100%
No Confidence										Completely Confident

How confident are you that you will not lose your balance or become unsteady when you…

1. …walk around the house? _____%
2. …walk up or down stairs? _____%
3. …bend over and pick up a slipper from the front of a closet floor? _____%
4. …reach for a small can off a shelf at eye level? _____%
5. …stand on your tip toes and reach for something above your head? _____%
6. …stand on a chair and reach for something? _____%
7. …sweep the floor? _____%
8. …walk outside the house to a car parked in the driveway? _____%
9. …get into or out of a car? _____%
10. …walk across a parking lot to the mall? _____%
11. …walk up or down a ramp? _____%
12. …walk in a crowded mall where people rapidly walk past you? _____%
13. …are bumped into by people as you walk through the mall? _____%
14. …step onto or off of an escalator while you are holding onto a railing? _____%
15. …step onto or off an escalator while holding onto parcels such that you cannot hold onto the railing? _____%
16. …walk outside on icy sidewalks? _____%

*Powell LE & Myers AM. The Activities-specific Balance Confidence (ABC) Scale. Journal of Gerontology Med Sci 1995; 50(1):M28-34.

Total ABC Score: _____

Scoring: _____ / 16 = _____% of self confidence
Total ABC Score

Figure H.3.1. Activities-Specific Balance Confidence (ABC) Scale questionnaire.