Abstract

This study compared cognitive-motor integration between young individuals with a concussion history to participants with Alzheimer’s disease risk. Participants made finger movements on a touchscreen from a central to peripheral target. We observed performance impairments in both groups relative to neurologically healthy control groups, even when age was accounted for. Interestingly, the pattern of performance impairments was distinct between the two clinical groups. A discriminant analysis differentiated individuals based on task performance with a history of concussion from those at Alzheimer’s disease risk with 91% accuracy. This suggests that the brain networks responsible for cognitive-motor integration are differently affected by concussion and Alzheimer’s disease risk. More generally, cognitive-motor integration provides a sensitive means to monitor Alzheimer’s disease-related functional decline, and to detect long-term functional impairment that can distinguish mild traumatic brain injury from dementia risk.

Keywords: Mild Cognitive Impairment; Mild Traumatic Brain Injury; Alzheimer’s Disease Risk; Movement Control
Introduction

The ability to integrate rules into action allows one to perform sophisticated tasks, and increasingly is involved with everyday life. When one’s movements are decoupled from the object being controlled, the brain must perform additional, ‘non-standard’, visuomotor transformations beyond what is required for direct object interaction, where the spatial locations of the guiding sensory information and required motor action are the same. Examples of such behaviour include the use of a computer mouse, operation of a vehicle, or control of remote aerial or land-based machinery. Elucidating the control networks in the healthy brain involved in controlling rule-based movement performance, also known as cognitive-motor integration (CMI), has been the topic of increasing research [1-12]. These studies have begun to characterize the differences in both behaviour and underlying neural control mechanisms when one requires the integration of rules to guide action.

In addition to the study of brain and behaviour in the healthy brain, one can also study the control of movement through an examination of behaviour in those affected by different neuropathological conditions. To this end, a number of recent studies have observed impaired cognitive-motor integration in adults either in the early stages of dementia [13,14] or at a heightened risk for developing the disease. Similarly, adults with Parkinson’s disease, in addition to their well-known basic movement impairments, displayed impaired performance in an ‘anti-tapping’ task where both the eye and hand had to move to a location opposite to the presented target. Another situation is concussion, where it is increasingly recognized that neural effects may linger long after signs and symptoms (assessed using current standard measures) have resolved [16,17]. While this is an acquired rather than a neurodegenerative form of brain dysfunction, it has also been shown that cognitive-motor integration performance is impaired in both asymptomatic young adults [18-20]. As with the dementia-related studies, these participants displayed intact basic motor and cognitive performance when these domains were tested in isolation.

A fundamental question that arises from these findings is the relationship between the impairments seen in different clinical groups. It is known that some of the characteristics of AD-related neuropathology are shared with concussion history. For example, research has shown that head trauma can trigger βA4 deposition in the cerebral cortex within days [21,22]. β-Amyloid is the main component of protein plaques which are thought to be the fundamental cause of AD, and the inheritance of APOE- ε4 is a well-known genetic risk factor for developing AD. Apolipoproteins enhance the breakdown of beta amyloid, APOE- ε4 isoform is not very effective at this task, leading to amyloid plaque accumulation in the brain [23]. demonstrated that the effect of head injury on the risk of AD was greater in subjects having one or two APOE- ε4 alleles, in comparison with those lacking APOE-ε4, and that magnitude of this AD risk was proportional to the severity of the concussion. It is also well documented that concussion itself is a risk factor for dementia development [24-27]. On a functional level, however, are these different modes of brain dysfunction affecting behavioural abilities in the same way? The answer to this question would have both theoretical and clinical relevance, in terms of understanding the basic neural control of rule-based motor behaviour, and improving the clinical utility of diagnostic and functional ability tracking tools.

In the present study, we compare the pattern of cognitive-motor integration impairment between young adults with a history of concussion, and older adults at-risk for the development of Alzheimer’s disease in order to seek insight into how rule-based visuomotor control is affected by mild dysfunction. We hypothesize, based on research relating previous head trauma to dementia risk, that the observed impairments will be similar, even when age-related performance differences are accounted for.

Materials and Methods

Participants

This study involved an analysis of data collected from 88 participants as part of two studies on cognitive-motor integration [27,28]. Data were examined from four groups...
Conjecture youth with a history of concussion, age-matched youth with no history of concussion, adults at risk for Alzheimer’s disease, and age-matched adults at low risk for Alzheimer’s disease. Exclusion criteria included vision or upper-limb impairments, any medical condition that would hinder task performance (e.g., severe arthritis), any neurological or psychiatric illnesses (e.g., schizophrenia, depression, alcoholism, epilepsy, Parkinson’s disease), and any history of stroke or severe head injury, and a Montreal Cognitive Assessment (MoCA) score greater than 27. Additionally, inclusion criteria for the history of concussion group included being ‘asymptomatic’ in accordance with current return-to-play protocol guidelines, a Sport Concussion Assessment Tool cognitive assessment (SCAT3-SAC) score greater than 25/30, and participating in their team sport normally. Older adults were recruited in through collaborations with the local retirement associations and local gerontology clinics. Younger adults were recruited in through collaborations with the local retirement associations and local gerontology clinics.

Table 1: Descriptive statistics of participants

<table>
<thead>
<tr>
<th></th>
<th>Concussion</th>
<th>Youth</th>
<th>Older adults</th>
<th>AD risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>22</td>
<td>22</td>
<td>22</td>
<td>22</td>
</tr>
<tr>
<td>MCI</td>
<td>8</td>
<td></td>
<td>79.7 ± 7.7</td>
<td></td>
</tr>
<tr>
<td>DH</td>
<td>14</td>
<td></td>
<td>60.5 ± 5.9</td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>13.2 ± 1.4</td>
<td>11.8 ± 1.6</td>
<td>64.3 ± 9.8</td>
<td>67.5 ± 11.4</td>
</tr>
<tr>
<td>Range (years)</td>
<td>11 – 16</td>
<td>9 – 15</td>
<td>54 – 84</td>
<td>53 – 91</td>
</tr>
<tr>
<td>Sex (% female)</td>
<td>59%</td>
<td>59%</td>
<td>50%</td>
<td>73%</td>
</tr>
<tr>
<td>Handedness (% right)</td>
<td>91%</td>
<td>91%</td>
<td>91%</td>
<td>91%</td>
</tr>
<tr>
<td>MoCA score</td>
<td>†</td>
<td>†</td>
<td>27.6 (1.6)</td>
<td>25.8 (4.3)</td>
</tr>
</tbody>
</table>

MCI = mild cognitive impairment; DH = familial dementia history; †: SCAT3-SAC subscore > 25; *: p > 0.05 (between young and old groups, but not within age groups).
recruited through collaborations with local youth athletic leagues (soccer, lacrosse, hockey). Informed consent was obtained from all participants, and the experimental protocol was approved by the Human Participants Review Subcommittee and ethics review board of York University.

Procedure

An Acer Iconia 6120 dual touch screen tablet was used for the testing of subjects in a vertical (V) and horizontal rotated (HR) condition (Figure 1a). Participants were seated and used a glove to touch the screen to ensure fluid movements. In the condition V, a standard mapping task was performed in which the spatial location of the target and required movement were in the same plane on the screen. In the nonstandard mapping HR condition, the target’s spatial location was dissociated from the plane of the required movement. Participants were asked to look at the presented target on the vertical screen while moving their finger to the target in the opposite direction in the horizontal plane. Therefore, for the horizontal rotated condition, the spatial positions of gaze and the hand were decoupled.

One practice run (maximum 2 trials per target) was conducted before each of the conditions to ensure adequate comprehension by the participants. 20 trials were conducted for both the standard and nonstandard conditions for a total of 40 trials per subject. Subjects were instructed to use their index finger of their dominant hand to touch the screen and then move as quickly and accurately as possible from the home central circle to the peripheral target. Targets were located 75 mm the left, right, below, or above the home central target. The target’s position and size (20 mm in diameter) were held constant for all the trials. Participants touched the home central target directly or indirectly with the horizontal tablet depending on the condition. The home target changed colour from green to yellow indicating contact with the home target had occurred. After holding the central target for 4000 ms a red peripheral target appeared, coinciding with the disappearance of the central target. This represented the “go signal” for the subject to look at the visual target and slide their finger along the screen to move the cursor to the peripheral target. After the target was acquired and held for 500 ms, the target disappeared and the trial ended. The subsequent trial began with the presentation of the central target after an inter-trial interval of 2000 ms.

Figure 1: A) Diagram of the experimental design for the standard vertical (V) and non-standard horizontal rotated (HR) conditions. For all trials, the visual stimuli were presented on the vertical screen. The starting point of the home target is represented as the light grey eye, finger, and cursor and the dark grey eye, finger, and cursor denote the movement of the eye and finger for the specific condition. The dark circle represents the cursor feedback during the motion and the grey open circle shows the home central position prior to initiation of movement. Grey open circle shows the home central position prior to initiation of movement. B) Individual examples of movement trajectories in the standard direct interaction and in the non-standard decoupled cognitive-motor integration condition for both experimental groups form one young participant with concussion-history, and one participant with AD risk.

Data Processing

Information about finger position, timing, and errors was collected from the touch screens and converted into MATLAB readable format using a custom C++ applica-
tion. Trials were excluded from further analysis if the finger left the home target prematurely (<4000 ms), reaction time was too short (<150 ms) or too long (>1800 ms), or movement time was too long (>10000 ms). In the horizontal rotated condition, trials were also eliminated in which the first ballistic movement moved the cursor in the direction away from the visual target. The processed data were then analyzed for precision, accuracy, and timing measures using a custom computer analysis program.

Dependent Measures

In this study, six dependent variables of interest were analyzed: reaction time, movement time, ballistic path length, full path length, absolute error, and variable error. The movement onsets and ballistic movement offsets (the initial movement prior to path corrections) were scored at 10% peak velocity. Reaction time was defined as the time when the home target disappeared at movement onset. Movement time was the difference between movement onset and offset, thus representing the initial ballistic movement without making corrections. The ballistic movement time provided information about the initial feedforward movement control command (initial execution) for our different groups, rather than the on-line corrective feedback control commands. The latter control behaviour was captured in our full path length measure. Path length was defined as the total distance travelled, measured as the resultant of the x and y finger locations in mm. Path length was calculated as both the full path length (FPL, when the finger reached 10% peak velocity within the target) as well as the ballistic path length (BPL, initial movement offset). Note that in the situation where the initial movement brought the finger into the final target, these path length variables would be equivalent. Absolute error (sometimes referred to as non-directional constant error), representing the accuracy of the ballistic movement, was calculated as the absolute value of the difference between the average movement endpoints and the target position (\( \sum x/n, \sum y/n \)). Variable error (\( \sigma^2 \)) reflected the precision of the movement, and was calculated as the difference between individual endpoints and the mean endpoint of the ballistic movement.

Data analysis

The participants’ performance was compared as a function of their change from the standard condition V. For each dependent variable, the value for the V condition was subtracted from the HR condition (Delta HR – V, “Delta”). Importantly, the Delta values for the AD risk group and concussion-history group were normalized as fractions of the mean Delta values of the older control group and youth control group respectively. This allowed for a comparison of AD risk and concussion-history groups independent from age. A one-way ANOVA was conducted to explore significant group differences. For each dependent variable, a Shapiro-Wilk’s test was performed to ensure a normal distribution of data. A Mauchly’s sphericity test was conducted and a Greenhouse-Geisser correction was applied in the event of a violation of the sphericity assumption. The significance value used was \( \alpha = 0.05 \). Data analysis was done using the SPSS (IBM Inc.) computer program.

Discriminant analysis

A stepwise discriminant analysis was performed comparing the concussion and the AD risk groups in order to examine whether the task could successfully differentiate between concussion and AD risk participants based on performance. Dependent variables from the HR and V tasks were used in the analysis which demonstrated high predictive potential based on the ANOVA results.

Results

Table 1 provides detailed demographic information on the 88 participants comprising the four groups examined in the current study. The four groups were similar in terms of handedness. Age ranges were also similar between the younger healthy control and concussion history groups, and the older healthy controls and dementia risk groups. The only notable difference was a larger percentage of females in the older dementia risk group relative to the older healthy control group, reflecting current global demographic trends.
Relative to age matched controls, the concussion-history group and AD risk participants both had impaired behavioural performance when cognitive-motor integration was required. For reaction time, movement time, and precision these performance decrements were similar. In contrast to our hypothesis, however, there were differences between the clinical groups in terms of the pattern of impairment observed, differences found in Full and Ballistic pathlength, and accuracy. An example of the behavioural performance for one participant with an increased dementia risk and one participant with a history of concussion is shown in Figure 1b. Note that the basic, standard point-to-point movements done for the vertical condition where individuals simply slide their finger from one point to another are fairly straight, and not different between participants. However, both participants showed greater trajectory deviations for the movements requiring cognitive-motor integration whereby the hand was moving in a different plane of motion than the viewed targets, and visual feedback was reversed between the motion of the hand and the cursor reflecting this motion. Note the particular difficulty in maintaining a smooth trajectory to the target demonstrated by the participant with increased dementia risk. This behaviour was observed more generally, as described below and summarized in Figures 2 to 4.

### Performance timing

Across all conditions and all groups, timing measures increased with increasing task complexity, as expected. With respect to the two clinical groups, we observed longer reaction times and movement times in the AD risk participants compared to the concussion-history group (Table 2, Figure 2). However, when the age of the two groups was accounted for, there were no significant differences in these two timing variables as a function of task complexity. Specifically, the changes in performance timing measures between the HR and V conditions (Delta) were calculated and normalized relative to the controls. There were no significant differences between the concussion-history and AD risk groups in MT Delta (F(1, 43) = 3.14, p>0.05) and RT Delta (F(1, 43) = 3.14, p>0.05) as a fraction of their age-matched control groups.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group</th>
<th>Condition</th>
<th>Group x Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>MT</td>
<td>F(4,87) = 16.51 n.s.</td>
<td>F(1,87) = 191.31***</td>
<td>F(4,87) = 7.33***</td>
</tr>
<tr>
<td>RT</td>
<td>F(4,87) = 21.65***</td>
<td>F(1,87) = 176.70***</td>
<td>F(4,87) = 11.10***</td>
</tr>
<tr>
<td>TMT</td>
<td>F(4,87) = 12.34***</td>
<td>F(1,87) = 206.77***</td>
<td>F(4,87) = 7.04***</td>
</tr>
<tr>
<td>CMT</td>
<td>F(4,87) = 8.55***</td>
<td>F(1,87) = 130.01***</td>
<td>F(4,87) = 7.21***</td>
</tr>
<tr>
<td>Ballistic PL</td>
<td>F(4,87) = 11.46***</td>
<td>F(1,87) = 10.53**</td>
<td>F(4,87) = 14.35***</td>
</tr>
<tr>
<td>Full PL</td>
<td>F(4,87) = 6.75***</td>
<td>F(1,87) = 77.72***</td>
<td>F(4,87) = 4.58**</td>
</tr>
<tr>
<td>VE</td>
<td>F(4,87) = 4.62**</td>
<td>F(1,87) = 86.72***</td>
<td>F(4,87) = 11.64***</td>
</tr>
</tbody>
</table>

n.s. = not significant; * p < 0.05; ** p < 0.01; *** p < 0.001.

### Table 2: Statistical results of the repeated measures mixed ANOVA of group and condition for all dependent variables.

Abbreviations: RT = Reaction time, MT = Movement time, TMT = Total movement time, CMT = Corrective movement time, AE = Absolute error (accuracy), VE = Variable error (precision), PL = Path length; V = Vertical, HR = Horizontal Rotated

Figure 2: A) Movement time and reaction time data for participants in all groups (Alzheimer’s disease risk, concussion-history, youth control, old control) for the standard vertical condition (V) and the non-standard horizontal dissociated condition (HR). Error bars: standard error of the mean.

Path length

Ballistic path length decreased for all groups with the more difficult non-standard HR condition (Figure 3). That is, participants’ initial movements tended to stop short of the final target when the finger was moving in a different plane and was guided by reversed visual feedback. We observed a significantly shorter Ballistic path length in the AD risk group compared to the concussion-history group (Table 2) once the age difference was accounted for (BPL Delta $F_{1,43} = 10.42$, $p<0.001$, Figure 3b, right panel). Across all groups, Full path length was longer for the more complex condition, reflecting the additional distance travelled by the finger in correcting initial movements. Similar to that found for BPL, the difference in cognitive-motor integration performance between these two groups was also observed, reflected in a significantly longer Full path length in the AD risk group (FPL Delta, $F_{1,43} = 5.39$, $p<0.05$, Figure 3b, left panel).

Figure 3: A) Full path length and ballistic path length data for participants in all groups (Alzheimer’s disease risk, concussion history, youth control, old control) for the standard vertical condition (V) and the non-standard horizontal dissociated condition (HR). B) Delta Path length data for the AD risk and concussion-history groups of the vertical condition subtracted from the horizontal rotated condition and normalized as a fraction of the age matched controls. Error bars represent standard error of the mean.

Endpoint Analysis

Endpoint accuracy and precision decreased for all groups as task difficulty increased with the HR condition (Table 2, Figure 4a). However, a greater decline in accuracy was observed for the AD risk group compared to the concussion-history participants (Table 2, AE Delta $F_{1,43} = 14.10$, $p<0.001$, Figure 4b, left panel). In contrast, there were no significant differences between clinical groups in precision (VE Delta $F_{1,43} = 1.23$, $p>0.05$, Figure 4b, right panel).
Figure 4: A) Absolute error (accuracy) and variable error (precision) data for participants in all groups (Alzheimer’s disease risk, concussion history, youth control, old control) for the standard vertical condition (V) and the non-standard horizontal dissociated condition (HR). B) Delta Error data for the AD risk and concussion-history groups of the vertical condition subtracted from the horizontal rotated condition and normalized as a fraction of the age matched controls. 0.001; n.s. = Not significant. Error bars represent standard error of the mean.

**Discriminant analysis**

The discriminant analysis showed a strong separation between the AD risk and concussion history groups (Figure 5). Predictors included in the discriminant function were MT, RT, ballistic path length, full path length, AE, and VE. Using these variables, the outcome of the discriminant analyses showed that the behavioural test involving vision-action dissociation could distinguish between concussion-history and AD-risk participants with an accuracy of 91% (Table 3).

**Discussion**

The main goal of the present study was to compare cognitive-motor integration performance between youth with a history of concussion versus older adults with an elevated AD risk. Previous research has found differences in cognitive-motor integration performance between healthy participants and both adults at risk for [29-31], and youth and young adults with concussion history [32-34]. Here we hypothesized that AD risk participants would exhibit similar deficits to those with a history of concussion in their cognitive-motor integration capacities, once age-related changes were accounted for. This hypothesis was not fully support-

### Table 3: Classification of results of stepwise discriminant analysis

<table>
<thead>
<tr>
<th></th>
<th>Predicted Group Membership</th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Group</td>
<td>Concussion</td>
<td>AD risk</td>
<td>Total</td>
</tr>
<tr>
<td><strong>Original</strong></td>
<td>Count</td>
<td>19</td>
<td>3</td>
<td>22</td>
</tr>
<tr>
<td></td>
<td>Concussion</td>
<td>1</td>
<td>21</td>
<td>22</td>
</tr>
<tr>
<td></td>
<td>AD risk</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>%</td>
<td>86.4</td>
<td>13.6</td>
<td>100.0</td>
</tr>
<tr>
<td></td>
<td>Concussion</td>
<td>4.5</td>
<td>95.5</td>
<td>100.0</td>
</tr>
<tr>
<td><strong>Cross validated</strong></td>
<td>Count</td>
<td>18</td>
<td>4</td>
<td>22</td>
</tr>
<tr>
<td></td>
<td>Concussion</td>
<td>2</td>
<td>20</td>
<td>22</td>
</tr>
<tr>
<td></td>
<td>AD risk</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>%</td>
<td>81.8</td>
<td>18.2</td>
<td>100.0</td>
</tr>
<tr>
<td></td>
<td>Concussion</td>
<td>9.1</td>
<td>90.9</td>
<td>100.0</td>
</tr>
<tr>
<td></td>
<td>AD risk</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

While some of our dependent variables were affected in the same way between our two clinical groups, contrary to expectations we observed that on half of our variables our two groups differed in the extent of their impairment. Once age was accounted for, our observed performance differences were able to predictively discriminate our two clinical groups with an accuracy of 91%. These data extend our previous work by characterizing the ways in which complex skill, requiring the integration of brain network activity processing both cognition and action, are differentially affected by different types of mild brain dysfunction.

**Timing versus trajectory measures: insight into differential brain mechanisms underlying performance deficits**

We found similar reaction time increases relative to age-matched healthy controls in our two clinical groups. This observation suggests that the brain regions responsible for movement planning are similarly affected by dementia risk and concussion history. One area known to be heavily involved in movement preparation is the premotor cortex [35]. Previous work using brain anatomical and functional imaging of individuals diagnosed with MCI have revealed cortical alterations in the bilateral frontal cortices in the premotor area [36-39]. Research on concussed individuals demonstrated similar functional changes in the premotor cortex. In particular, increased activation of the pre-frontal cortex, dorsolateral prefrontal cortex, and cerebellum was observed in concussed subjects when performing specific actions [40-42]. Such widespread alterations in movement control likely reflects a compensatory mechanism, although whether the compensation is due to premotor cortex damage itself or the ability of other regions to communicate with premotor cortex is an open question requiring further study.

We also observed that both concussed and dementia-risk individuals displayed similar increases in movement time, once the well-known age-related psychomotor slowing [43] was taken into account. Thus, in terms of timing for both movement planning and movement execution, the control mechanisms appear to be similarly affected by both neurological conditions that were studied here. However, it is known that neural mechanisms for temporal and spatial aspects of movement control are to some extent independent [44]. Here, we observed that spatial aspects of movement trajectory formation did differ between our two clinical groups, reflected in our finding that both path length and accuracy were longer/worse in the AD risk group. Our observed differences in visuomotor impairment between clinical groups suggest that the mechanism underlying

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**Figure 5:** Stepwise discriminant analysis results displaying canonical discriminant functions for A) concussion history group B) dementia risk group. Scores are a weighted contribution of dependent variables which were to maximize discriminability between groups. StDev: standard deviation.
their impairment is not the same. This does not dismiss the concept that repetitive concussion leads to an increased AD risk. Indeed, there is a growing body of evidence that the underlying mechanism for a wide range of neurological conditions may be neuroinflammation caused by repeated head trauma, and its pathological effect on neural tissue at the cellular level [45,46]. Rather, it may be that there are differences in either specific brain regions affected by the two neuropathological conditions, or differences in the integrity of the fibre tracts allowing communication between different brain regions controlling CMI (or both).

Brain regions intimately involved in the control of visually-guided behaviour include parietal, premotor and primary motor cortices, and the cerebellum and basal ganglia subcortically [48-50]. Neuroimaging studies have found reduced white matter integrity in fibre tracts connecting frontal-parietal regions, hippocampal-cortical, and interhemispheric regions in adults with AD and MCI. Recently, the level of reduced integrity has been related to cognitive-motor integration performance in adults at risk for dementia [51]. Further, individuals diagnosed with MCI show structural alterations in bilateral inferior and superior parietal cortices. Parietal cortical atrophy was observed in patients with early AD. Neuroimaging work in youth and adults with concussion history has also demonstrated altered fronto-parietal white matter tract diffusion characteristics [52-57] and augmented compensatory activation in frontal, parietal, and cerebellar regions [58]. However, different paradigms and group characteristics from these studies make it difficult to conclude exactly how the various neuropathologies are differentially affecting brain function. Behaviourally, our observation that our AD risk group had significantly longer full path lengths in the CMI task relative to our concussion group suggests that on-line feedback control – a process controlled by the cerebellum and its interactions with the movement network mentioned above, may be more impaired in this group. Our observed greater decline in accuracy in the AD risk group compared to the concussion-history group may reflect a greater disruption to somatosensory processing between the parietal lobe and other regions of the brain, necessary for the integration of visual and spatial stimuli to produce a motor program [59]. Future work directly comparing the integrity of brain structure and function in AD risk to that of concussion history, accounting for age differences, will allow for better characterization of the neural mechanisms underlying impaired behavioural performance in these clinical situations.

Limitations

Several limitations in the study may have reduced the generalizability of the results. Firstly, a relatively small sample size was used in the experiment (n = 22 in each group) reducing the power of statistical analyses. Heterogeneity in the AD risk subjects, which consisted of MCI and familial dementia history participants, may have influenced the results. In addition, there was a greater percentage of females in the AD risk group (73%) compared to the concussion-history group (59%) and controls (50%). Further, the majority of our concussed participants had only one concussion, thus we could not analyse the effect of multiple concussion on behaviour. It may be that behavioural features of complex skill performance in these two groups begin to merge in the face of repeated head injury.

Conclusions and Implications

Neurological deficits associated with both concussion and Alzheimer’s disease compromise the ability to integrate visual information in order to plan and execute movement. However, a discriminant analysis distinguished individuals with a history of concussion from those at increased Alzheimer’s disease risk with 91% accuracy based on the distinct ways in which these two groups were impaired in their performance. On a practical level, such behavioural differences may assist the clinician in distinguishing functional activity impairments associated with neurodegeneration risk from those associated with head injury history, in situations where it is not clear what the source of the impairment is. On a fundamental level, the observed behavioural distinctions between clinical groups suggests that the neural mechanisms responsible for the control of rule-based motor behaviour are differentially affected by neurodegenerative versus impact-related neurological history.
Acknowledgements

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