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# **The Effect of Type 1 Diabetes on Executive Function in Young Adults**

A thesis  
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## **Abstract**

Type 1 diabetes mellitus (T1DM) is a chronic and potentially life-threatening condition affecting the production of insulin, with rapidly increasing incidence worldwide. The disease impacts on nearly all domains of life, including the physical, social, neurological and psychological. Adequate management of the disease requires careful monitoring and self-care to maintain glycaemic control. Executive function (EF) refers to a cluster of top-down cognitive processes engaged in the planning and completion of goal-directed behaviour allowing an individual to plan their actions, contemplate novel challenges, resist temptation and focus attention while avoiding distracting stimuli. Young adulthood represents an important developmental stage during which robust EF is essential. Previously limited research has explored the impact of T1DM on EF specifically within a young adult population. The current study recruited a sample of young adults between the ages of 16 and 24 with T1DM (n=14) and an age and gender matched control group (n=14) of non-diabetic individuals. Groups were assessed on measures of EF while maintaining blood glucose levels within a euglycaemic range. The central aims of the study were to compare and contrast the diabetic and control group EF performance, and to explore the relationship between diabetic related variables and measures of EF. It was found that the diabetic group committed significantly more errors than the control group on a test of set-shifting ability. It was also found that the relationship between performance-based tests of EF and a rating scale measure of EF was unique to each group. HbA1c, a measure of longer term glycaemic control, was found to be

significantly related to a rating scale measure of EF but not performance-based measures. It was also found that mean blood glucose level during assessment was significantly related to performance on measures of working memory. Despite a limited sample size, the findings suggest that in a young adult population there is some evidence that T1DM is related to impairments in certain areas of EF — including set-shifting, inhibition and the higher-level construct of problem-solving ability. Future research could extend these findings by specifically exploring the relationship between trail-making tasks and rating scales of EF, examining the role of hot EF in diabetes self-care, and determining the sensitivity of specific EF to subtle changes in blood glucose within a euglycaemic range.

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## List of Abbreviations

BGL/s	Blood glucose level/s
BMI	Body mass index
BRI	Behavioral Regulation Index
BRIEF-SR	<i>Behavior Rating Inventory of Executive Function - Self-Report</i>
CWI	Color-word inhibition
CWIS	Color-word inhibition switching
CWIT	<i>Color-Word Interference Test</i>
DKA	Diabetic ketoacidosis
D-KEFS	<i>Delis-Kaplan Executive Function System</i>
EF/s	Executive function/s
FSIQ	Full scale IQ
GEC	Global Executive Composite
GMLT	<i>Groton Maze Learning Test</i>
GMLTDR	<i>Groton Maze Learning Test - Delayed Recall</i>
HADS	<i>Hospital Anxiety and Depression Scale</i>
HbA1c	Glycated haemoglobin
MI	Metacognition Index
MMFFT	<i>Modified Matching Familiar Figures Test</i>
NLS	Number-letter sequencing
OBT	<i>One-Back Test</i>
OGTT	Oral glucose tolerance test

PFC	Prefrontal cortex
SS	Scaled score
SST	<i>Set-Shifting Test</i>
T1DM	Type 1 diabetes mellitus
T2DM	Type 2 diabetes mellitus
TBT	<i>Two-Back Test</i>
TCT	<i>Timed Chase Test</i>
TMT	<i>Trail Making Test</i>
WASI-II	<i>Wechsler Abbreviated Scale of Intelligence – Second Edition</i>
WCST	<i>Wisconsin Card Sorting Test</i>
WM	Working memory
WRDS	Waikato Regional Diabetes Service

## **Introduction**

### **Diabetes**

Diabetes mellitus is a chronic disease which impairs the normal function of insulin with the potential for numerous negative health outcomes for those affected. In 1980, global estimates of individuals diagnosed with the disease were 108 million, rising to 422 million in 2014 (NCD Risk Factor Collaboration). This equates to nearly a doubling in prevalence from 4.7% to 8.5% (age standardized). Economically, diabetes presents a significant and increasing burden on public health (Zhang et al., 2010), with estimates of total global healthcare expenditure of 376 billion USD in 2010, representing 12% of overall health expenditure. This value is projected to rise to 490 billion USD by 2030. Demographic shifts have also occurred, with prevalence increasing at a greater rate in low and middle-income countries in comparison to high-income countries (World Health Organisation, 2016).

In New Zealand overall prevalence resides at close to 7%, and is more common in males (8%) than females (6%). Self-reported prevalence is almost double in Maori compared to non-Maori (Ministry of Health, 2016b). With respect to all health loss causes in New Zealand, diabetes has advanced most in rank between 1990 and 2013—from 16th to 7th among males and from 22nd to 12th among females (Ministry of Health, 2016a).

All forms of diabetes share a common factor in that they affect the body's capacity to produce or use insulin. Insulin is a hormone produced by the  $\beta$ -cells of the pancreas responsible for maintaining blood glucose levels (BGL) within a narrow range despite the intake of large amounts of food. When food or more specifically

carbohydrate is eaten, the secretion of insulin promotes the storage of glucose as glycogen into the liver or muscle structure where it can be used for energy (Atkinson, Eisenbarth, & Michels, 2014). Insulin also encourages triglyceride (body fat) storage, exerting influence on lipid production in the liver, adipose tissue, and fat cells (DeFronzo, Ferrannini, Zimmet, & Alberti, 2015). Accordingly, adequate production of insulin and the body's ability to use insulin effectively is essential for maintaining blood glucose homeostasis.

Diabetes is most commonly divided into two disease subtypes: Type 1 (T1DM) and Type 2 (T2DM; Egan & Dinneen, 2014). T2DM is considerably more common, constituting around 90-95% of all diabetes cases. T2DM generally arises when the tissues of the body develop resistance to the effects of insulin, often in combination with some impairment to insulin production. Risk factors for developing T2DM include: increasing age, obesity and a lack of physical exercise. Improvements in the latter two factors can significantly ameliorate the effects of the disease process, and generally the administration of exogenous insulin is not required.

In T1DM, or insulin dependent diabetes, there is an autoimmune initiated deterioration of  $\beta$ -cells in the islets of the pancreas which causes a progressive decline in the body's ability to produce insulin (Thrower & Bingley, 2014). Subsequently the pancreas may produce little or no endogenous insulin, leaving BGL unregulated. The autoimmune damage to the  $\beta$  cells is unable to be reversed, meaning the disease process is without cure. In response, the affected individual will generally require on-going, regular monitoring of BGLs and administration of exogenous insulin to support and maintain healthy levels of blood glucose. Failure to do so can lead to severe consequences including ketoacidosis, coma and death (DeFronzo et al.,

2015).

T1DM, while only accounting for 5-10% of overall diabetes prevalence, constitutes close to 80% of paediatric diabetes diagnoses and is slightly more common in males (Borschuck, Everhart, & Everhart, 2015). The peak age of diagnosis is around 5-7 years of age or early adolescence, however increasing disease prevalence also appears to be accompanied by increasingly younger age of diagnosis (van Belle, Coppieters, & von Herrath, 2011). Given the more typical early age of diagnosis, T1DM is also sometimes referred to as juvenile-onset diabetes.

The exact epidemiology of T1DM is unclear, with most current hypotheses suggesting some form of gene-environment interaction (Vehik & Dabelea, 2011). Proposed risk factors include: genetic vulnerability, diet, body-size, viruses, and geographic location. There is increasing evidence to suggest that both maternal diet during pregnancy and diet in the early years of life may play a significant role in the likelihood of developing islet autoimmunity, a common precursor to T1DM (Norris, 2010).

At onset, symptoms of T1DM may include increased thirst, tiredness, excessive urination, weight loss, excessive hunger and mood disruption. Diagnosis is typically informed by a BGL measurement, either in the fasting state or following an oral glucose tolerance test (OGTT). A diabetic individual will usually present with a fasting BGL of greater than 7mmol/L, or greater than 11.1mmol/L following an OGTT (Diabetes New Zealand, 2016). More recently it has been recommended that HbA1c, a measure of glycated haemoglobin may be a better diagnostic measure. This reflects the individual's glycaemic control over the past two to three months, providing a more comprehensive picture of blood glucose regulation over time. An

HbA1c level greater than 48mmol/mol is the recommended diagnostic cut-off (International Expert Committee, 2009).

For the individual affected by T1DM, proper management of BGL requires considerable monitoring, organisation, planning and commitment. Proper disease management will usually involve multiple self-administered daily injections of a basal (long acting) insulin, as well as bolus (rapid acting) insulin before or after meals to lower BGL to within a healthy range (Diabetes New Zealand, 2016). While more recent technological advances have seen increasing use of insulin pumps and continuous glucose monitoring — this carries additional cost, is not suited to all individuals, and is not without potential for complication (van Dijk et al., 2012).

Diet and exercise plays an important role in BGL regulation and overall diabetes management (American Diabetes Association, 2007). The inclusion of fruits, vegetables, whole-grains, legumes and low fat milk is recommended. To ensure the accurate provision of postprandial (after meal) bolus insulin it becomes important to monitor the carbohydrate content of each meal (Neupane & Evans, 2014). This may mean it is easier for the individual to have routine meals with known carbohydrate content. In contrast, exercise and physical activity may serve to deplete glucose stores, effectively lowering BGL. This may require the consumption of additional carbohydrate to counter any glucose depletion and avoid hypoglycaemia (low BGL). Maintaining good health and functioning is a complex exercise in counter-balancing both the intake and expenditure of energy (glucose), which will ultimately determine longer-term health outcomes.

Chronic poor management of BGL significantly increases the risk of several severe health concerns associated with diabetes including retinopathy (eye damage),

nephropathy (kidney damage), neuropathy (nerve damage), and cardiovascular disease (Atkinson, Eisenbarth, & Michels, 2014). These complications are primarily a result of long-term elevated BGL (hyperglycaemia), in which cells are bathed in an excess concentration of glucose. Certain cells such as the capillary endothelial cells in the retina, mesangial cells in the renal glomerulus, and neurons and Schwann cells of peripheral nerves show limited capacity to self-regulate internal glucose concentrations and thus are more vulnerable to damage (Brownlee, 2005). As might be expected, the development of diabetes related complications has been found to significantly reduce quality of life and increase the risk of developing psychiatric symptoms such as depression (Alan M. Jacobson et al., 2013; van Steenbergen-Weijenburg et al., 2011).

Severe and extended periods of hyperglycaemia can also lead to diabetic ketoacidosis (DKA), an acute and life threatening condition that is considered one of the most serious and common complications of T1DM (Perilli, Saraceni, Daniels, & Ahmad, 2013). During DKA, excess blood glucose and increased levels of counterregulatory hormones cause an overproduction and underutilisation of ketones in the body. Unregulated ketones can encourage metabolic acidosis, a lowering of pH in the bodily fluids (Kitabchi, Umpierrez, Miles, & Fisher, 2009). DKA can occur within a matter of hours, and without intervention can lead to coma and death. Recurrent episodes of DKA have been found to increase risk of mortality, particularly among young adults who are socially disadvantaged (Gibb, Teoh, Graham, & Lockman, 2016).

While the use of exogenous insulin is essential to regulate BGL, a potential side-effect of this intervention is excessively lowering blood glucose concentrations,

causing hypoglycaemia (Heller & Chow, 2014). Hypoglycaemia can also occur due to missed meals or excess exercise without carbohydrate to compensate. An acute episode of hypoglycaemia can present as sweating, tremor, palpitations, confusion and drowsiness. As BGLs continue to decline, coma, brain damage and even death can occur. With disease duration the likelihood of a severe hypoglycaemic episode increases as the body's physiological defence mechanisms become decreasingly sensitive to lower concentrations of blood glucose, meaning an individual may be less likely to become aware of the signs of a hypoglycaemia (Snoek, Tibor & Rondags, 2013). Even low level hypoglycaemic episodes often show an effect on the individual's mood, presenting as agitation, irritability and rapid mood fluctuation.

**Diabetes and cognition.** Given that glucose is the primary source of fuel for the brain (Heikkilä et al., 2010) it is perhaps unsurprising that T1DM is associated with poorer cognitive performance across several domains. These include intelligence, attention, processing speed and cognitive flexibility in both children (Gaudieri, Chen, Greer, & Holmes, 2008) and adults (Brands, Biessels, de Haan, Kappelle, & Kessels, 2005). Gaudieri et al. (2008) conducted a meta-analysis of 15 studies of paediatric cognitive function between the years of 1985 and 2008 to examine the relationship between T1DM and cognition.

The study included a total of 2144 children, 1393 of which had a diagnosis of T1DM. Salient inclusion criteria were: a diagnosis before age 18, a measure of cognition administered, and the presence of a control group (at least age-matched). Average age of diabetes onset was 6.6 years (range 4.13 -11.18) and average disease duration was 5.23 years (range 0.5-8.9).

It was found that the diabetic participants performed more poorly than the controls on nearly all cognitive measures including: overall cognition, intelligence (fluid and crystallised), attention and executive function, speed of information processing, motor speed, visual-motor integration and academic achievement. Groups did not differ significantly on measures of learning and memory.

A meta-analysis by Brands et al. (2005) examined 33 studies between 1980 and 2004 looking at cognitive function in adults with T1DM. Salient inclusion criteria for studies included: participants over 18 years of age, the presence of a control group, and the use of reliable neuropsychological measures administered at a normal (euglycaemic) blood glucose level. Compared with non-diabetic control participants, the T1DM group demonstrated significantly poorer performance in overall cognition, intelligence (fluid and crystallized), speed of information processing, psychomotor efficiency, visual and sustained attention, mental flexibility, and visual perception. The authors surmised that the primary deficits in T1DM appear centred around reductions in both processing speed and cognitive flexibility; a pattern of cognitive dysfunction that appears to be similar for both children and adults (McCrimmon, Ryan, & Frier, 2012).

It has been posited that mild cognitive dysfunction may occur as a result of impaired neurotransmitter function, potentially arising as a cumulative result of poor glycaemic control, or glycaemic fluctuations between periods of hypo- and hyperglycaemia. (Northam et al., 1998; Rustad et al., 2013).

Another theory suggests that rather than a cumulative effect, it is potentially the timing of glycaemic events that is most salient, for example during critical developmental periods or periods of central nervous system vulnerability (Schwartz,

Axelrad, & Anderson, 2014). Both glucose demand and brain volume rapidly increase from the early months of life to the age of six; by age 10 glucose usage of the brain is twice that of the adult brain. Throughout adolescence significant synaptic pruning occurs alongside increasing myelination (Arbelaez, Semenkovich, & Hershey, 2013). Given that the onset of T1DM often co-occurs with these structural brain changes, and the brain's reliance on a constant supply of glucose, it follows that the effect of a significant glycaemic event may be detrimental during this period.

There is evidence that disease duration appears to be positively correlated with cognitive impairment, with progressive impairments in motor speed, visual and verbal memory, executive function and visuospatial performance in some cases being observed within 1-3 years of diagnosis (Desrocher & Rovet, 2004). Structurally, it has been found that within type 1 diabetics, chronic hyperglycaemia has been associated with reduction in whole brain grey matter, while episodes of severe hypoglycaemia are associated with reduction in parietal/occipital white matter (Perantic et al., 2011). Both structural and functional changes to the brain have been observed in older type 1 diabetics independent of cardiovascular risk factors and diabetic complications (Hughes et al., 2013), suggesting the disease may exert an independent neurodegenerative effect.

A series of studies which specifically demonstrated cognitive decline over time were conducted by Northam et al. (1998). The initial study compared the cognitive performance of 129 children (age range 3-14 years) three months after receiving a diagnosis of T1DM against a control group matched on age and sex. At baseline testing it was found that the neuropsychological profile and general intelligence of those with T1DM was not different to that of the control group.

The groups were retested two years later, at which point the diabetic group were found to perform significantly poorer in domains of visuospatial ability, vocabulary, processing speed and learning. Those within the diabetic group exhibited less positive changes to their full-scale IQ score over the two years than the control group, particularly if developing diabetes prior to age five (early onset).

In a follow-up study Northam et al. (2001) compared children from their original study six years post-diagnosis, on measures of intelligence and neuropsychological functioning (n=90, T1DM and n=84, control). Children with T1DM showed significantly lower verbal and full-scale IQ, and reduced attention, processing speed, and executive skills when compared to the control group. Attention, processing speed and executive skills were most affected in children who had developed diabetes prior to the age of four.

However, not all studies have demonstrated an impact of T1DM on cognitive ability. Alan M. Jacobson et al. (2007) found limited evidence of cognitive decline in a large sample (1144) of individuals (mean age =27 at entry) with T1DM that were followed over a course of 18 years, despite approximately 40% of the sample experiencing episodes of severe hypoglycaemia. The study tested the sample group at baseline and at 18 years on measures of problem solving, learning, memory, attention, visuospatial ability, motor speed and psychomotor and mental efficiency. It should however be noted that only a small proportion of individuals in this study experienced early onset of the disease.

To summarise, diabetes mellitus, specifically T1DM, is a chronic and potentially life-threatening condition with rapidly increasing incidence worldwide. The disease impacts on nearly all domains of life, including the physical, social,

neurological and psychological. Adequate management of the disease requires careful monitoring and adjustment to maintain a balance between hypo- and hyperglycaemic states. Failure to do so can have acute and serious effects, while even minor deviations over time may result in longer term impairment. Several studies suggest cognitive impairment is a common correlate of T1DM; however it is unclear whether this is purely a function of poorly managed BGL, disease pathology, or a combination of both. The current study now focusses on a specific area of cognition, that of executive function, and its relationship to T1DM.

### **Executive Function**

Executive function or functions (EF or EFs) refers to a cluster of top-down cognitive processes engaged in the planning and completion of goal-directed behaviour (Diamond, 2013). According to Lezak (1982), EFs “are at the heart of all socially useful, personally enhancing, constructive, and creative activities” (p.281). EFs allow an individual to plan their actions, contemplate novel challenges, resist temptation and focus attention while avoiding distracting stimuli.

The executive processes that contribute to EFs are associated with a complex range of interrelated neural networks within the prefrontal cortex (PFC) (Stuss & Knight, 2002). However the function of the PFC itself is interrelated with nearly every other region of the brain, meaning structural damage outside of the PFC may still result in some executive dysfunction. It also appears that reliance on the PFC may change over time, with EFs in elderly individuals drawing on a broader range of brain regions in comparison to young adults (Spreng, Wojtowicz, & Grady, 2010). While PFC integrity is important for stable and robust EFs, it is not the only region upon which EF relies.

A number of neuropsychological models of EF have been proposed to date, however no model has received universal acceptance. Some early models conceptualised EFs as existing within a unitary control system such as the “central executive” (Baddeley, 1986) or the “supervisory attentional system” (Norman & Shallice, 1986). However these models generally experienced difficulty accounting for phenomena such as the poor correlation between measures of executive processes and the absence of global executive “dysfunction” (Anderson, 2008).

More recent research suggests EFs constitute multiple separate yet interrelated cognitive control processes (Baddeley, 1998; Sylvester et al., 2003). These processes are ultimately responsible for the control and organisation of cognitions, behaviour and emotions. Broadly speaking, all conceptual models suggest some or all of the following components as central to EF: attention, inhibition, initiation of activity, working memory, cognitive flexibility, planning and problem solving (Anderson, 2008).

Miyake et al. (2000) and Lehto, Juujärvi, Kooistra, and Pulkkinen (2003) suggest there are potentially three core EFs: inhibition (of automatic responses), updating and monitoring of information in working memory, and mental set-shifting or cognitive flexibility. These serve to provide the foundation for more complex higher order EFs including reasoning, problem-solving and planning (Diamond, 2013).

Inhibition refers to both behavioural inhibition; self-control or avoiding responding impulsively, and also cognitive inhibition or interference control recruited when selective attention is required, allowing for sustained and focussed attention. The “updating” and monitoring of working memory (WM) refers to the sorting of

incoming information by relevance to a required task, then revision and replacement of old redundant information held in WM with new relevant information. In short; it is the dynamic and active manipulation of information held in WM. Cognitive flexibility or “shifting” refers to the ability to configure mental resources to switch between various cognitive tasks (Monsell, 2003). This requires intentional discrimination between procedural schemas or task sets relative to a desired or dominant goal.

A further distinction is the classification of EFs into “hot” or “cool” executive processes (Zelazo & Muller, 2010). Cool executive processes refer to those that are purely cognitive, recruited when approaching abstract or decontextualized problems; effortful attention and prepotent response inhibition are examples of these. Hot processes are those associated with emotional or affective regulation and decision making, for example in tasks requiring delayed gratification (Kim, Nordling, Yoon, Boldt, & Kochanska, 2013).

**Executive function and development.** EF is essential to almost every aspect of life including mental and physical health, academic performance, career success, and social ability (Diamond, 2013). As EFs are typically associated with several areas of the PFC, they develop throughout childhood and adolescence, reaching maturation in early adulthood (Rossi et al., 2013). Generally, EFs exhibit progressive linear development until around 22 years of age, although some particular EFs may peak before others (Taylor, Barker, Heavey, & McHale, 2013). The progressive development of EFs is believed to be a result of increasing myelination of axons in the PFC which continues through adolescence, in particular cortico-cortical and

cortico-limbic neural circuits. This allows for a significantly improved speed of signal transmission between neurons (Choudhury, Charman, & Blakemore, 2008). EFs are observed to decline in later life, associated with volumetric shrinkage of the PFC (Raz, Gunning-Dixon, Head, Dupuis, & Acker, 1998).

**Executive function and the young adult.** Adolescence and early adult life is a period of particular vulnerability to impaired decision making, with the potential for life-long negative impacts. Individuals in this developmental period are more likely to engage in substance abuse, risky sexual behaviour and breaking of societal norms (Crone, Bullens, van der Plas, Kijlkuit, & Zelazo, 2008). The decision to engage in these behaviours is often made in spite of explicit knowledge of potential negative consequences. This increase in risk-taking behaviour is believed to be partly a result of rapid sub-cortical development, which exceeds the concurrent cortical capacity for regulation and inhibition (Romer et al., 2011). Put simply, the adolescent is likely to experience strong emotional drives without the required capacity for cognitive regulation of these emotions.

There is a general pattern of reduced risk-taking behaviour as the young person matures into early adulthood, correlating with normal cortical development, but in which good EF appears to play a significant mediating role (Brand & Schiebener, 2013). In fact, EF appears to mediate risk-taking and facilitate decision making throughout the lifespan, in both children (Lahat et al., 2012) and older adults (Menon, Jahn, Mauer, & O'Bryant, 2013). Schiebener, García-Arias, García-Villamizar, Cabanyes-Truffino, and Brand (2015) suggest that on average, children and adolescents make the most high-risk decisions, with potential for the most severe

consequences, with middle adulthood representing the most stable decision making period.

A study conducted in New Zealand examined risk-taking behaviour and EF in a community sample of young people between the ages of 13 and 22 (Pharo, Sim, Graham, Gross, & Hayne, 2011). The authors found that in this sample, poorer results on neuropsychological tests of EF correlated with increased real-world risk taking behaviours. Specifically, deficits in the area of inhibitory control may make adolescents and young adults more vulnerable to putting themselves at risk.

EF, while a construct that is not always consistently defined, is shown to constitute an incredibly important range of cognitive functions that allow an individual to plan, strategize, inhibit, problem-solve and maintain control over their behaviour. In many ways they separate the adult mind from that of the child, representing the higher order cognitive functions that enable one to independently function in the world. These functions develop rapidly during the course of adolescence and into young adulthood and appear most stable in middle-adulthood, before declining in old age. The role of well-developed EFs is particularly salient for the young adult, as this represents a developmental period in which there is a greater likelihood of risk-taking behaviour. Any factor that threatens to impair EF therefore places the young adult at greater risk, and warrants careful examination.

**Diabetes and executive function.** As discussed, there is evidence T1DM may exert a significant impact on several domains of cognition, even after a relatively short disease duration. Of relevance to the current study is the specific impact of T1DM on EF, with a particular focus on the young adult.

A study by Rovet and Alvarez (1997) explored intelligence and components of EF in a group of 103 children and adolescents with T1DM (age range 9.3-18.3 years) and compared their performance to a control group approximately matched on age and gender. Each group shared an approximately even gender split. On average participants in the diabetic group had been diagnosed for 6.8 years (range 2-14 years). The authors chose to focus on areas of EF relating specifically to attention, and included the following relevant measures: *Stroop Test*, *Modified Matching Familiar Figures Test* (MMFFT), *Trail Making Test*, *Visual Search Test* and the *Wisconsin Card Sorting Test* (WSCT). Transient glucose levels were not regulated prior to assessment; BGL was however monitored at five intervals throughout the testing process.

The authors divided the attentional process under examination into subcomponents: Focus, Select, Inhibit, Suppress, Shift and Sustain. The diabetic group only differed significantly from the control group on 'Select', which was derived from mean error scores on the MMFT. This task requires the participant to select the correct match to a presented card (target) from a range of response alternatives. The authors concluded that the result was primarily a function of the performance of those participants who had been diagnosed prior to the age of six; however length of diagnosis was not related to any aspect of attention. This suggests early-onset diabetes may exert a more significant impact on EF than disease duration.

The authors found that a history of hypoglycaemic seizures was associated with poorer performance on the Select, Focus and Inhibit attentional components. They also found higher BGL during testing was associated with a more impulsive responding style on a computer based vigilance test. Interestingly length of diagnosis

or HbA1c level was not found to correlate with any aspect of attention/EF.

The findings of Rovet and Alvarez (1997) have a several potential limitations. The test on which a between-group difference was observed, the MMFT, was initially developed as a measure of impulsivity vs reflectiveness, suggesting the more impulsive individual would be less careful and more likely make more errors in their selection (Egeland & Weinberg, 1976). Although the authors did not find a correlation between transient or acute BGLs and errors on the MMFT, fluctuations in transient BGL has been found to affect performance on other tests of impulsivity and attention (Topitsch, Schober, Wurst, & Kryspin-Exner, 1998).

There is also some debate as to whether the MMFT is more of an ability test — reflecting intelligence more so than any specific cognitive component (Weijers, Wiesbeck, & Böning, 2001). While the groups did not differ significantly on overall IQ, intelligence was not controlled for in their analysis. IQ has been shown to correlate with some measures of EF (Friedman et al., 2006), suggesting controlling for this variable is important when assessing EF.

A more recent study by Ly, Anderson, McNamara, Davis, and Jones (2011), compared the performance of a group of 33 young people (mean age 19.3 years) with early-onset T1DM, to a matched control group (n=34, mean age 19.5) on measures of memory, intelligence, EF and mood. The diabetic participant sample represented a subset of an original study group (n=84) that had been monitored for approximately 16 years, since time of diagnosis. The authors therefore had access to reliable data on HbA1c at diagnosis and a history of glycaemic events, including seizures, coma and microvascular complications.

The primary measure of EF used was the Wisconsin Card Sorting Test

(WCST), a measure of set-shifting ability, concept formation and problem solving in response to feedback. BGLs were maintained between 4-15 mmol/L throughout testing. The authors found no difference between the groups on measures of IQ, memory or reported emotional difficulties. On the WCST, the diabetic group subjects completed fewer categories ( $p=.022$ ) and committed more perseverative errors ( $p=.002$ ) than the control group. The authors concluded that the presence of diabetes did not appear to impact on an individual's memory, general intelligence or emotional difficulties however did potentially result in minor deterioration in EF. The authors also found the presence of early severe hypoglycaemia events were associated with poorer EF in the diabetic group, however this group subset was relatively small ( $n=6$ ).

As the WCST is believed to assess a range of executive domains, it considered a good overall measure of general EF (Mitrushina, 2005). However a general measure is not likely to be as informative as a more comprehensive test battery, particularly for a construct as diverse as EF.

The two studies described above provide some insight into the potential relationship between EF and T1DM in the young adult. However both share limitations in the scope of EF they explore; the former focused on attentional components with limited exploration of set-shifting/cognitive flexibility or updating of working memory, and the latter using only one more general measure of EF. The former study also examined EF in a primarily adolescent rather than young adult age range, and did not attempt to control BGL during assessment. Given the varied timeline of EF development and impact of acute changes in BGL on cognition, this represents a potential limitation to the generalisability of these findings.

## **Summary and Research Aims**

Several topics central to the current study have been discussed including: T1DM pathology, impact and management; the effect of diabetes on cognition, and specifically its effect on EF; EF and development, and the importance of EF for the young adult. The young adult with T1DM as such provides a complex intersection of a developmental stage and a disease process. Developmentally, this is a life period where risk-taking is more likely, and the consequences of these risks are potentially serious and long-lasting. Young adults are often for the first time trying to navigate many new and complex life tasks including relationships, employment, learning to drive, managing finances, and developing a stable identity.

T1DM potentially impacts upon this developmental stage by its effects on cognition and specifically on EF, a set of complex higher-order cognitive processes particularly essential to helping the young-adult safely navigate the world around them, implicated in inhibition, planning, working memory and cognitive flexibility. For the young adult with diabetes, there is an additional burden of the many tasks of disease management, all of which require effective monitoring, planning and behavioural regulation. Poor disease management at best dysregulates one's energy, mood and cognition, and at worst can be fatal. Accordingly, the young-adult with T1DM represents a particularly vulnerable population, who may be at increased risk of impairments in EF, which may have serious outcomes for both disease management and effective and safe functioning in the world.

The majority of previous studies have explored cognition more broadly within the type 1 diabetic population, many of which including some measures of EF within

their scope. However, few studies have chosen to focus specifically on EF, or specifically examine a young adult population. There is also adequate research that has shown the acute effects of altered BGLs on EF, but the literature is inconclusive regarding whether T1DM by its very presence exerts an influence on EF or if poor diabetes management and/or glycaemic events during sensitive stages of development must occur before any significant effect is observed.

The current study intends to explore some of these gaps in the literature by specifically examining EF in a population of young-adults diagnosed with T1DM.

The two central research aims are:

- To examine any differences between a sample of young adults with and without T1DM in the area of EF
- To examine the relationship between diabetic related variables including HbA1c, time since diagnosis and age of diagnosis, and EF in a sample of young adults with T1DM

## **Method**

The current study used data gathered for a larger parent project looking at the effects of blood glucose levels on driving behaviour and executive functioning in young people with T1DM. Additional data was collected from a non-diabetic control group for comparison purposes. Ethics approval for the parent project was obtained from the University of Waikato' School of Psychology and the Health and Disability Ethics Committee, reference number 14:80 and Ref 14/CEN/181.

The current study adhered to the parent project protocol, did not involve the diabetes service and did not require manipulation or testing of blood glucose therefore ethics approval was granted under the existing ethics application. This was approved by the School of Psychology Research and the Ethics Committee, University of Waikato.

### **Participants**

**Diabetic group.** Contact details for potential participants for this group were extracted from the Waikato Regional Diabetes Database. All individuals were located within the Waikato region, and used the Hamilton clinic as their primary point of contact with the Diabetes Service. Inclusion criteria were age (16-24 years) and a diagnosis of T1DM. N=99 individuals were found to meet the inclusion criteria, the large majority of which were invited to participate in the study. Following contact initially by mail and then by phone, individuals wishing to participate were required to meet additional inclusion criteria which were: a diagnosis of T1DM of greater than 6 months; a full or restricted drivers licence; and driving of a vehicle at least four

times per week. Exclusion criteria were: pregnancy; regular use of illicit substances; an episode of DKA in the last 48 hours or an episode of documented severe hypoglycaemia requiring assistance in the previous 24 hours.

**DKA and diagnosis of diabetes.** DKA is a serious condition arising from decreased circulating insulin, insulin resistance and increases in counterregulatory hormones, usually lasting around 48 hours (Jefferies, 2008). Pathophysiology typically involves an increase in ketone production, severe hypoglycaemia and dehydration. Individuals with T1DM are at increased risk of DKA, and it is the leading cause of diabetes-related mortality amongst children and adolescents.

In New Zealand, diagnosis of diabetes requires a fasting blood glucose level of  $\geq 7$  mmol/L, or a blood glucose of  $\geq 11.1$  mmol/L following an oral glucose tolerance test (OGTT) or random glucose test (non-fasting) (Diabetes New Zealand, 2016). Diagnosis is typically precipitated by an individual becoming symptomatic (polyuria, excessive thirst, weight loss, lethargy) and presenting to their health practitioner, or an episode of DKA requiring medical intervention.

**Control group.** Participants for this group were primarily recruited through flyers located on the University of Waikato campus, a University internet noticeboard and a database of individuals previously consenting to research at the University of Waikato (see Appendix A). Contact details for the researcher were provided and interested individuals were asked to make contact directly via phone or email. For this group, a diagnosis of diabetes (all types) became part of the exclusion criteria, while all other criteria were maintained as per the diabetic group. Attempts were made to recruit participants for the control group matched to the diabetic group on

age and gender.

**Sample characteristics.** The sample characteristics for both diabetic and control groups can be found in Table 1. Total sample size was 28 participants, the majority which were male ( $n=20$ ), and identified as New Zealand European. Mean age at assessment was similar for both groups with the control group being on average 1.3 years older ( $t(26) = -1.89, p=.07$ ).

The large majority of diabetic participants had been diagnosed after the age of five, with mean age of diagnosis being 11.3 years, and a mean time since diagnosis of eight years. Eight of the participants had experienced at least one episode of DKA. The mean body mass index (BMI) for the diabetic group is considered to be within the normal range (Ministry of Health, 2009). HbA1c, a measure of longer term glycaemic control varied markedly, ranging from 47 to 130 mmol/mol. The mean value for the group of 80.4mmol/mol is considered very high, with a suggested healthy value of 53 mmol/mol (Diabetes New Zealand, 2016).

Groups were relatively well matched on mean estimated IQ ( $t(26) = -.56, p =.58$ ) as well as current reported symptoms of anxiety and depression ( $t(26) = -.21, p =.84$ , and  $t(26) = -1.75, p =.09$ , respectively) as measured by the Hospital Anxiety and Depression Scale (HADS). The mean HADS scores for both anxiety and depression for each group is considered to be in the normal range (Snaith & Zigmond, 1994).

Table 1

*Characteristics of the Study Sample*

	Group			
	Diabetic (n=14)		Control (n=14)	
Participant characteristics				
Male, n (%)	10	(71.4)	10	(71.4)
Age (years), <i>M</i> (min/max)	19.3	(17-23)	20.6	(18-23)
Ethnicity, n (%) <sup>a</sup>				
New Zealand European	14	(100)	13	(93)
Maori	1	(7)	2	(14)
Other	1	(7)	4	(28)
Diabetic related variables				
BMI, <i>M</i> (min/max)	23.1	(18-29)	NA	NA
HbA1c (mmol/mol), <i>M</i> ( <i>SD</i> )	80.4	26.7	NA	NA
Age at diagnosis (years), <i>M</i> ( <i>SD</i> )	11.3	(4.2)	NA	NA
Time diagnosed (years), <i>M</i> ( <i>SD</i> )	8.0	(5.1)	NA	NA
Early onset (<5 years) n (%)	2	(14.3)	NA	NA
Previous episode of DKA (participants), n (%)	8	(57)	NA	NA
IQ and mood variables, <i>M</i> ( <i>SD</i> )				
Estimated IQ	114	(10)	116	(10)
HADS Anxiety	6.1	(3.3)	6.4	(4.1)
HADS Depression	2.1	(2.1)	3.6	(2.4)

*Note:* <sup>a</sup> Participants were able to select more than one ethnicity. NA = Not applicable/available. BMI = body mass index. HADS= Hospital Anxiety and Depression Scale, scores range from 0 to 21 with higher score indicating greater level of distress.

## **Measures**

A range of psychometric measures were used for both groups which are described in detail below. These included performance-based tests and questionnaires. Measures were chosen in an attempt to cover a range of components of EF, employing both verbal and nonverbal tasks. We also obtained a number of biomedical measures from the diabetic group. All participants in the study provided demographic and general medical information via a questionnaire, including any current or historical medical issues and hospital admissions (see Appendix C).

### **Executive Functioning**

Cogstate (<http://www.cogstate.com>) is a computerized test battery providing reliable and valid computerised touch screen assessment of cognitive function including psychomotor performance, attention, memory, and executive functioning. Cogstate offers a number of individual tests, which can be put together to form a test battery. All tests are designed for repeated administration, are brief to administer, and show minimal learning effects. A computerised test format was deemed appropriate given that the study population would likely be familiar with media devices. All Cogstate tests were administered on a touch-screen tablet, with the participant using a stylus and letter keys to complete the tasks. A summary of the six tests administered can be found in Table 2 with a description provided below. Each test is preceded by a short trial run to familiarise the participant with the task requirements and format.

Table 2  
*Summary of Cogstate Tests Used in the Current Study, in Order of Presentation from Top to Bottom*

Test	Outcome Measure	Scoring and Interpretation	Cognitive Domain
<i>Timed Chase Test</i>	Moves per second	Speed; higher =better performance	Visual motor
<i>Groton Maze Learning Test</i>	Total errors	Accuracy; lower=better performance	Executive function
<i>One Back Test</i>	Log10 milliseconds	Mean reaction times for correct responses; lower score = better performance	Working memory
<i>Two Back Test</i>	Arcsine proportion correct	Accuracy; higher score=better performance	Working memory
<i>Set-Shifting Test</i>	Total errors	Accuracy; lower=better performance	Executive function
<i>Groton Maze Learning Test (Delayed Recall)</i>	Total errors	Accuracy; lower=better performance	Memory

***Timed Chase Test*** (TCT). This test assesses a person’s visual motor function and familiarises the participant with the Groton Maze environment. Using the touch screen participants “chase” a target on a grid as quickly and accurately as they can over the course of a single 30 second trial.

***Groton Maze Learning Test*** (GMLT). This test uses a maze learning paradigm to assess executive function and spatial problem solving. Using a grid, participants attempt to make their way through a hidden maze, to get from the identified start to finish receiving trial and error feedback. Once they have completed the maze, they repeat the maze trying to recall the pathway they have previously completed. Five consecutive trials are administered over a single session.

***One Back Test*** (OBT). This test assesses working memory and attention.

Participants are shown a card and participants have to indicate if the card shown is the same as the previous card. Primary outcome measure is the log<sub>10</sub> transformed reaction time for correct responses, indicating speed of performance. A lower score indicates a better (faster) performance.

***Two Back Test*** (TBT). This test also assesses working memory and attention.

During this test the participant is shown a card on a screen and has to decide if the card is the same as the card that was shown two cards ago. Primary outcome measure is the arcsine proportion of correct responses indicating accuracy of performance. A higher score indicates a better (more accurate) performance.

***Set-Shifting Test*** (SST). This test assesses executive functioning and spatial problem solving. Using the computer screen, a card is presented (colour or number) and the participant has to guess whether the card is the “target” card, and receive auditory feedback as to whether they have guessed correctly. A series of cards are then presented and the target stimulus changes dimension, forcing the participant to relearn the new target. Multiple shifts are made through the assessment. Primary outcome measure is total number of errors made over five rounds indicating accuracy of performance. A lower score indicates a better performance.

***Groton Maze Learning Test - Delayed Recall*** (GMLTDR). For this test the participant is provided with a single trial in which they are required to recall the hidden maze they learned in the earlier GMLT.

*Delis Kaplan Executive Function System* (D-KEFS; Delis, Kaplan, & Kramer, 2001). The D-KEFS is a battery consisting of nine subtests designed to assess various components of executive functioning. The measure was normed on a representative sample from the U.S population, of healthy individuals ranging in age from 8-89 (N=1750). There is research support for the general validity and reliability (internal consistency and test-retest) of the D-KEFS (Delis, Kramer, Kaplan, & Holdnack, 2004). Each subtest is presented in an interactive game-style format, and were administered by the examiner. Similar to the core components of EF suggested by Miyake et al. (2000), a factor analysis of the D-KEFS by Litzman and Markon (2010) identified three factors: conceptual flexibility, monitoring and inhibition. Two D-KEFS subtests were administered as detailed below, both most strongly loading on the inhibition factor.

Both subtests are timed tasks with primary outcome measures being completion time (seconds) and errors (n). Completion times are converted to scaled scores ( $M=10$ ;  $SD=3$ ).

*Colour-Word Interference Test* (CWIT). This measure consisting of four conditions is primarily intended to assess the capacity for one to inhibit an automatic verbal response when required to name a discrepant coloured patch. The first two conditions provide a baseline measure of time to name coloured patches and time to read colour name words (for example “yellow”) respectively. In the third condition, the participant is asked to inhibit reading the colour word names while naming the colour. In the fourth and final condition the participant is asked to switch between naming the colour and reading the word. This measures both inhibition and cognitive flexibility. For each condition, completion time in seconds is used to generate an age-

corrected scaled score ( $M=10$ ;  $SD=3$ ). The current study examined data from condition three and four: colour-word inhibition (CWI) and colour-word inhibition switching (CWIS) respectively.

**Trail Making Test (TMT).** This test contains five conditions which assess visual scanning and attention, flexibility of thinking and working memory. The primary EF task used for the current study was condition four, the number-letter switching (NLS) task which is a measure of cognitive flexibility or set-shifting ability. The remaining four conditions help to provide normative data for cognitive processes that contribute to the switching task. They consist of tasks of visual scanning, number sequencing, letter sequencing and motor speed. The completion time for each condition (seconds) is converted to an age-corrected scaled score ( $M=10$ ;  $SD=3$ ).

### **General Cognitive Ability**

**Wechsler Abbreviated Scale of Intelligence – Second Edition (WASI-II;** Weschler, 2011). This measure is a validated four subtest measure of cognitive ability and has been widely used in clinical and research settings. The WASI-II standardisation sample was drawn from a nonclinical, national, stratified sample of the U.S population aged 6 – 90 ( $N=2300$ ). To reduce test administration time, only two subtests were administered: Matrix Reasoning and Vocabulary. These subtests were selected in accordance with WASI-II protocol as a valid abbreviated version of the test. Each subtest generates a T-score ( $M=50$ ,  $SD=10$ ), which can be converted to a scaled score ( $M=10$ ,  $SD=3$ ) and contributes to a composite or Full Scale-IQ (FSIQ) score ( $M=100$ ,  $SD=15$ ). This assessment was used to provide an estimate of IQ for

each participant as there is evidence that intelligence may correlate with some components of EF (Ardila, Pineda, & Rosselli, 2000). This ensured any potential influence of IQ could be considered when examining EF.

***Matrix Reasoning.*** This subtest consists of 30 picture items which each require the participant to correctly select the required part or symbol to complete an incomplete matrix or series. This subtest is intended to reflect several areas of cognition including: fluid intelligence, broad visual intelligence, classification and spatial ability, knowledge of part-whole relationships, simultaneous processing and perceptual organisation.

***Vocabulary.*** This subtest consists of 28 word and 3 picture items. The participant is required to provide a definition for the word item or to name the picture item. Word items are presented both visually and orally. This test is intended to measure word knowledge and verbal concept formation.

### **Self-Report Measures**

***Behavior Rating Inventory of Executive Function - Self-Report*** (BRIEF-SR; Guy, Isquith, & Gioia, 2004). This is a self-administered rating scale measure of executive functioning, designed for young people aged 11 – 18 years. The measure has 80 items and assesses a young person's ability to complete tasks which target several domains of executive functioning, including organising, planning and attention. The participant indicates how frequently specific behaviours are a problem on a three-point Likert Scale; 1=never, 2=sometimes, 3=often. Table 3 provides an overview of the measure's composition.

Eight clinical scales contribute to two index scores and an overall composite

score. Responses to the BRIEF–SR are converted into T-scores ( $M = 50$ ,  $SD = 10$ ), with higher scores indicative of poorer EF. Generally T-scores above 65 are considered clinically significant, however scores between 60-64 may be considered “mildly elevated”. In the standardization sample, measurements of internal consistency ranged from .73–.96, and test–retest correlations from .59–.89.

Table 3  
*Composition of the Behavioral Regulation Index of Executive Function-Self Report (BRIEF-SR)*

	Composite	Clinical Scale	Ability measured	
Global Executive Composite (GEC)		Inhibit	<i>Inhibit impulsive responses</i>	
	Behavioural Regulation Index (BRI)	Shift	<i>Adjust to changes in routine or task demands</i>	
		Emotional Control	<i>Modulate emotions</i>	
		Monitor	<i>Monitor own behaviour</i>	
	Metacognition Index (MI)		Working Memory	<i>Sustain working memory</i>
			Plan/Organise	<i>Plan and organise problem solving approaches</i>
			Organisation of materials	<i>Organise environment and materials</i>
		Task Completion	<i>Finish tasks, for example homework</i>	

## **Other Measures**

*Hospital Anxiety and Depression Scale.* (HADS; Zigmond & Snaith, 1983).

This is a 14 item brief screening instrument for anxiety and depression. Screening for anxiety and depression was important as it may affect participant's performance on the cognitive and neuropsychological tests. The HADS has good validity and reliability and has been used extensively in other research studies, with both clinical and non-clinical populations (Kjærgaard, Arfwedson Wang, Waterloo, & Jorde, 2014). Good internal consistency has been demonstrated with a Cronbach's  $\alpha = .82$  and  $.77$  for anxiety and depression scales respectively (Crawford, Henry, Crombie, & Taylor, 2001). Scores for anxiety and depression each range from 0 to 21 and are classified as follows: normal 0–7, mild 8–10, moderate 11–14, and severe 15–21. Each item is given a score between 0-3 based on symptom frequency, with 3 being the highest.

## **Biomedical – Diabetic Group Only**

**HbA1c.** This is a measure of glycated haemoglobin which indicates average plasma glucose concentration over the past four to six weeks. A Siemens Healthcare Diagnostic DCA analyser was used to assess HbA1c from a blood sample obtained via a finger prick. This device is checked for quality assurance by the Waikato Hospital Laboratory, an accredited laboratory service, once per month. The DCA analyser automatically provides all calculations, measures automatically and was quality controlled every 24hrs by a trained user. Unit of measurement is mmol/mol.

**Blood glucose.** A Stat Strip Glucose Meter was used for all blood glucose measurements, taken via a finger prick blood sample of at least 1.2 microliters. This device is checked for quality assurance by the Waikato Hospital Laboratory, an accredited laboratory service, once per month. A trained user ensures quality control every 24hrs. Unit of measurement is mmol/L.

**Body mass index (BMI).** This provides a general measure of tissue mass by comparing an individual's height against their weight. Height and weight measurements were taken by a diabetes nurse using clinic measures based at the Diabetes Service. Units of measurement were metres and kilograms respectively. BMI was calculated using the following formula: weight in kilograms/height in metres squared. Unit of measurement is kg/m<sup>2</sup>.

**Medical information.** Additional medical information was collected by the research nurse directly from the participant's medical record. This included: age of diagnosis, length of diagnosis, insulin delivery device and management regimen, hospitalisations for diabetes-related issues, additional medical diagnoses, and diabetes-related complications.

## **Design**

Some data was obtained from a parent study in which the diabetic participants were assessed under euglycaemic and hyperglycaemic conditions. The current study focuses solely on data collected in the euglycaemic condition as a basis for comparison with the control group. Any information about the hyperglycaemic data collection is included only for clarification of research design. Table 4 outlines the assessments and order of administration. All diabetic group data collected for the

current study was obtained in the euglycaemic condition, aside from the BRIEF-SR.

Table 4

*Summary of Measures and Order of Administration, From Top to Bottom*

Session	Measures Assessed	
Condition	Diabetic Group	Control Group
Euglycaemic	Biomedical measures <sup>a</sup>	
	BRIEF-SR <sup>b</sup>	BRIEF-SR
	HADS	HADS
	WASI-II	WASI-II
	D-KEFS	D-KEFS
	Cogstate	Cogstate

<sup>a</sup> Blood glucose levels were also measured every hour and at conclusion of assessment

<sup>b</sup> BRIEF-SR was administered to the diabetic group in the hyperglycaemic condition

**Diabetic Group.** For the diabetic group a within-group cross over design was used, each participant taking part in two assessments spaced approximately 2 weeks apart, one in the euglycaemic condition and one in the hyperglycaemic condition. The euglycaemic condition was specified as a BGL between 4.0mmol/L – 9.4mmol/L.

In the first research session participants were assigned to either of the two conditions. To minimise order effects, participants were alternately assigned to a condition depending on recruitment order. This process operated independently for both males and females, meaning both the first female and first male participants started in the euglycaemic condition while the second participant of each gender started in the hyperglycaemic condition, and so on. The remaining condition was completed in the participant’s second research session.

**Control group.** The control group received a single administration of the same psychometric measures as the diabetic group. Biomedical information was not collected for this group. In a group of healthy adult volunteers Freckmann et al. (2007) recorded peak postprandial BGL of up to 9.3mmol/L occurring around 50 minutes after a main meal. This suggests that at peak, the BGL of a healthy individual would more than likely still be in a range comparable with the euglycaemic condition of the diabetic group. However, to reduce the likelihood of any transient effect of elevated BGL, participants were advised to arrange a test session time approximately 1-2hrs after their last main meal and to avoid the intake of high carbohydrate snacks or beverages within this window.

### **Procedure**

All research sessions for the diabetic group were held at the Waikato Regional Diabetes Service (WRDS) as a diabetic nurse was required for the monitoring of BGL during testing. The non-diabetic test sessions were held on the University of Waikato campus. Research session time was approximately 1.5 to 2.5hrs per session for participants from both groups. Session length variability was contributed to by the speed of task administration and completion, as well as blood glucose manipulation for the diabetic group.

**Diabetic group.** Eligible individuals were mailed information about and invited to participate in the study. The information informed potential participants they would be contacted in future by a research assistant and were provided contact details if they desired to make contact before then. As an additional recruitment measure, study information flyers were posted in the WRDS clinic and clinic nurses

were encouraged to discuss the study with any eligible patients presenting at the service.

Letters were followed-up within approximately two weeks of posting via a phone-call or text message by the research assistant. Individuals were asked if they had received the letter, and if they had any questions about the study. An appointment was made for a mutually convenient time for those who wished to participate.

**Research sessions.** All research sessions were conducted prior to 12pm on weekdays, usually starting around 9am. In an effort to minimise BGL related down-time, participants were contacted the night before their session to inform them of the condition they would be assigned to. Participants assigned to the euglycaemic condition were advised to take their insulin as per usual.

On arrival to the diabetes clinic, participants provided written consent to participate in the study prior to a research nurse obtaining biomedical data. Following the collection of biomedical information, the research nurse reported as to whether the participant's BGL was in the required range for the assessment to begin. If BGL was found to be outside of the target range, the research nurse administered either insulin to reduce BGL or a high carbohydrate drink (Ensure Plus) to raise BGL. The required dose of either insulin or glucose was calculated by the diabetes nurse. BGL was then retested at approximately 15-30 minute intervals until within the required euglycaemic range, at which point the research session was able to proceed.

Prior to administration of psychometric measures, participants completed the demographic and background information questionnaire. During administration of psychometrics measures, BGL was monitored each hour. If it was found that BGL

had deviated outside of the euglycaemic range, testing was paused and BGL adjusted to within range before testing continued. Participants were informed that a diabetes nurse was available if at any time they should feel unwell during testing. Participants were also told they could take a break at any time during testing if required, however all participants completed the sessions without a break.

Following completion of all assessments, participants BGL was tested for a final time and (if required) adjusted to within the euglycaemic range before the participant left the clinic. Participants were thanked for their time, asked if they had any questions, and were given a \$20 gift voucher as a gesture of appreciation.

**Control group.** Participants typically volunteered for the study via email, and were subsequently provided with further information and a copy of the consent form (see Appendix B). Participants were offered the opportunity to ask any questions about the study prior to consenting to participate and were advised they could withdraw at any stage without penalty. Written consent to participate was provided by each participant either by email or at the commencement of assessment. For those wishing to participate, a mutually agreeable time to meet was arranged. At the conclusion of testing, participants were offered their choice of university course credit (2%) or a \$20 gift voucher as a gesture of appreciation for their time.

**Research sessions.** As control group participants were only tested on a single occasion, all relevant measures were completed in one session. The majority of research sessions were conducted on the University of Waikato campus, in a small research room. While not practical to measure BGL for this group, participants were advised to refrain from high carbohydrate snacks or beverages directly prior to the

testing session. One participant in the control group reported a history of traumatic brain injury and dyslexia and subsequently their results were excluded from analysis and another participant was recruited in their place.

### **Statistical Analysis**

Quantitative analysis of data was completed using SPSS software version 23. An alpha level of .05 (two-tailed) was used for all analyses. Before analysis, the distribution of the data was examined and non-parametric tests used where appropriate.

First the characteristics of the participants in the diabetic and control group were compared using independent samples *t*-tests (see Table 1, page 23). To compare the performance of the two groups on the cognitive and neuropsychological assessments, a series of between group ANOVAs were conducted. This was intended to compare diabetic to control group performance across the measures of EF. For the BRIEF only the three main indices were compared — the BRI, MI and GEC (see page 44). For the performance-based measures a subsequent series of ANCOVA analyses were performed using the WASI-II FSIQ as a covariate (see Table 5, page 39).

For the BRIEF, where clinical cut-off scores were available (i.e., T-score over 65), between group comparisons were made based on the number of participants over the cut-off on any of the three main indices. Due to low expected frequencies, a Fisher's Exact Test was used for this comparison (see Table 6, page 45). Next, to explore the relationship between the performance-based measures of EF and the BRIEF, Spearman's correlations were performed. For this analysis only the three

main indices of the BRIEF were used (see Table 7, page 47).

Additional analyses focussed solely on the diabetic group. Spearman's correlations were conducted to examine the relationship between performance on all measures of EF and three diabetic related variables — HbA1c, age of diagnosis and time since diagnosis (see Table 8, page 49). Finally, Spearman's correlations were then also performed to explore the relationship between average BGL over the course of assessment, and scores on the both the subscales and indices of the BRIEF (see Table 9, page 51).

## Results

The two primary aims of the current study were to explore differences between the diabetic and control group on the administered measures of EF, and to examine the relationship between diabetic related variables and EF in the diabetic sample.

### Performance-Based Measures of EF.

Table 5

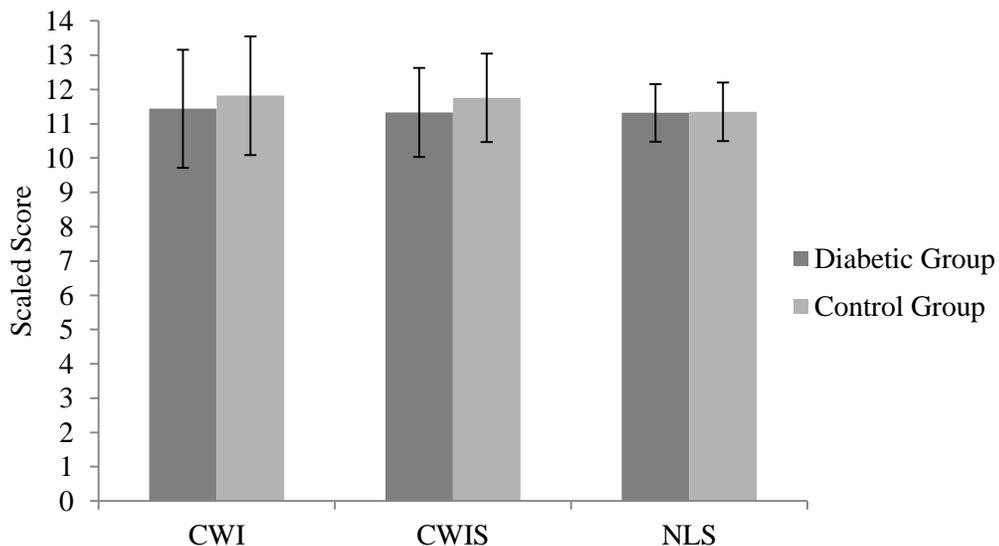
*Summary of Between-Groups Comparison of Scores on Performance-Based Measures of EF*

Measure	Group				ANCOVA		
	Diabetic (n=14)		Control (n=14)		<i>F</i> (1,26)	<i>p</i>	$\eta_p^2$
	<i>Mean<sup>a</sup></i>	<i>(95% CI)</i>	<i>Mean<sup>a</sup></i>	<i>(95% CI)</i>			
D-KEFS (SS)							
CWI	11.44	(9.71-13.16)	11.82	(10.09-13.55)	.10	.75	<.01
CWIS	11.33	(10.4-12.63)	11.76	(10.46-13.05)	.93	.34	.04
NLS	11.32	(10.48-12.16)	11.35	(10.51-12.20)	.25	.62	.01
Cogstate							
TCT	1.74	(1.60-1.88)	1.65	(1.51-1.80)	.25	.62	.01
GMLT	40.66	(31.05-50.26)	53.19	(43.58-62.79)	.30	.60	.01
GMLTDR	3.61	(1.99-5.23)	5.05	(3.49-6.61)	.04	.84	<.01
OBT	1.14	(.97-1.31)	1.32	(1.15-1.49)	1.27	.27	.05
TBT	1.18	(1.08-1.29)	1.22	(1.12-1.32)	2.39	.14	.09
SST	21.59	(17.02-26.17)	13.80	(9.22-18.37)	8.12	.01*	.25

*Note:* <sup>a</sup> WASI-II full scale IQ was included as a covariate - adjusted means are presented.  
 \*significant at  $p < .05$  level. CWI=Color-word inhibition; CWIS; Color-word inhibition switching; NLS=number-letter sequencing; TCT=*Timed Chase Test* (moves per second); GMLT = *Groton Maze Learning Test* (errors); GMLTDR; *Groton Maze Learning Test - Delayed Recall* (errors); OBT= *One Back Test* (reaction time); TBT=*Two Back Test* (accuracy); SST =*Set-Shifting Test* (errors). SS=scaled score; ANCOVA=Analysis of covariance; EF=Executive function.

A summary of between-groups analyses on performance-based measures of EF are presented in Table 5. For reference, sample characteristics of the study groups are previously presented in Table 1 (page 23). Generally, all D-KEFS scores of the control group were on average higher (better) than the diabetic group, while on the Cogstate the diabetic group performed better than the control group on four of the six tests.

A between-group series of ANOVA analyses found the diabetic and control group did not differ significantly on most measures (due to the relatedness of the dependent variables and comparatively small sample size, a MANOVA analysis was deemed inappropriate). As there is evidence to suggest IQ is related to performance-based measures of EF (Friedman et al., 2006), an additional series of between-groups ANCOVAs using WASI-II full scale IQ as a covariate were performed — all tables and figures for the performance-based measures show adjusted means.



*Figure 1.* Between-group comparisons of mean scaled score on D-KEFS subtest conditions including 95% confidence intervals, adjusted for IQ. Higher score indicates better performance. CWI= colour-word inhibition; CWIS= colour-word inhibition switching; NLS= number letter sequencing.

Figure 1 provides illustrative comparison between groups on the D-KEFS tests of executive function. While not significantly different (Table 5), the control group's scaled scores were on average higher than that of the diabetic group, suggestive of a better performance. On the NLS task, the groups' mean performance was virtually identical.

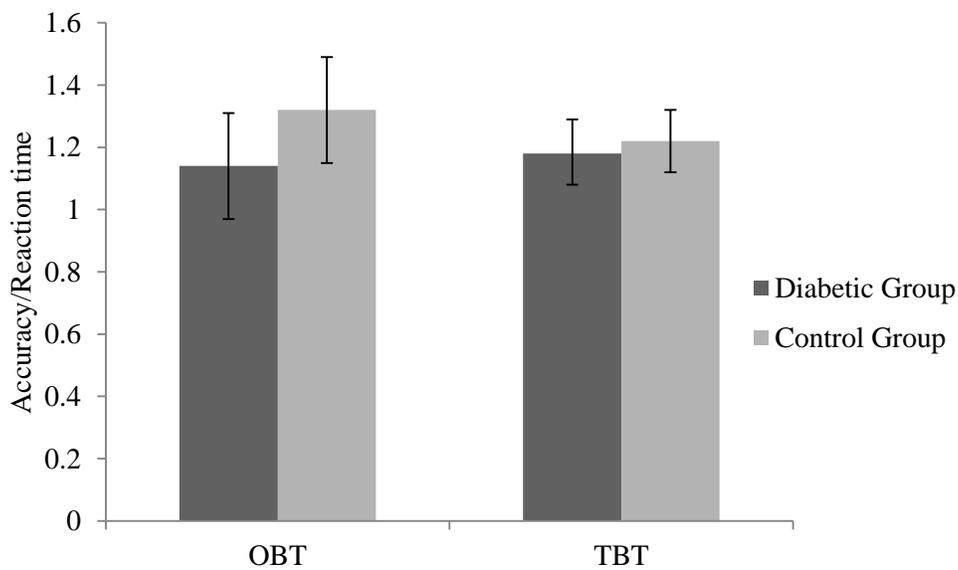
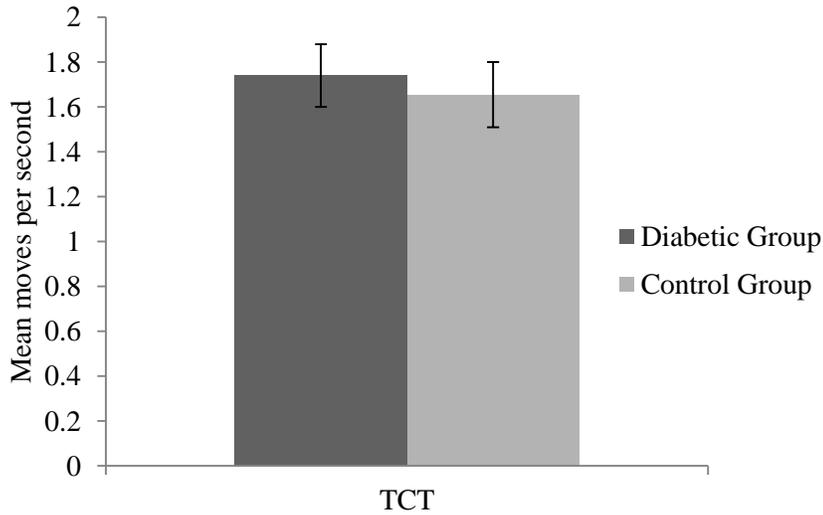


Figure 2. Between group comparisons of mean scores on the Cogstate OBT and TBT including 95% confidence intervals, adjusted for IQ. OBT = *One-Back Test*, higher score indicates slower performance; TBT = *Two-Back Test*, higher score indicates more accurate performance.

Figure 2 provides comparison of mean scores between groups on the Cogstate OBT and TBT, which measure speed and accuracy of working memory respectively. While not significantly different (Table 5), on the OBT the diabetic group were on average faster at responding than the control group, while on the TBT the diabetic group were less accurate in their selections than the control group. This suggests the

diabetic group demonstrated a faster reaction time, but with decreased accuracy in comparison to the control group, who were slower but more accurate in their responding.



*Figure 3.* Between-group comparisons of mean moves per second on the Cogstate TCT including 95% confidence intervals, adjusted for IQ. TCT = *Timed Chase Test*, higher score indicates faster performance.

Figure 3 shows the difference in speed measured in moves per second between groups on the Cogstate TCT. While not significantly different (Table 5), the diabetic group on average were marginally faster at performing this task.

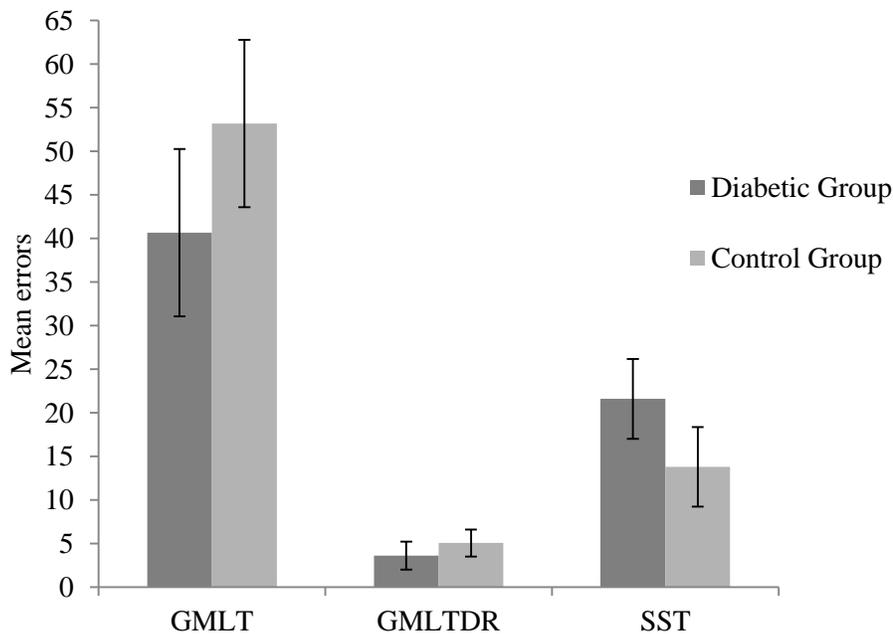


Figure 4. Between group comparisons of mean number of errors committed on Cogstate GMLT, GMLTDR and SST including 95% confidence intervals, adjusted for IQ. Lower score indicates better performance. GMLT= Groton Maze Learning Test; GMLTDR= Groton Maze Learning Test - Delayed Recall; SST = Set-Shifting Test.

Figure 4 illustrates between-group comparison of mean errors on the Cogstate GMLT, GMLTDR and the SST. On both Groton Maze tests the control group was found on average to commit more errors than the diabetic group. Initial ANOVA analyses (without controlling for IQ) found the between-group differences in GMLT mean scores to be significant,  $F(1,26) = 4.24$ ,  $p = .05$ ,  $\eta_p^2 = .14$ . Following inclusion of the covariate (WASI-II FSIQ), the difference between groups on this test failed to reach significance (Table 5). This finding suggests that variations in IQ are likely to account for the between-group differences observed on the GMLT.

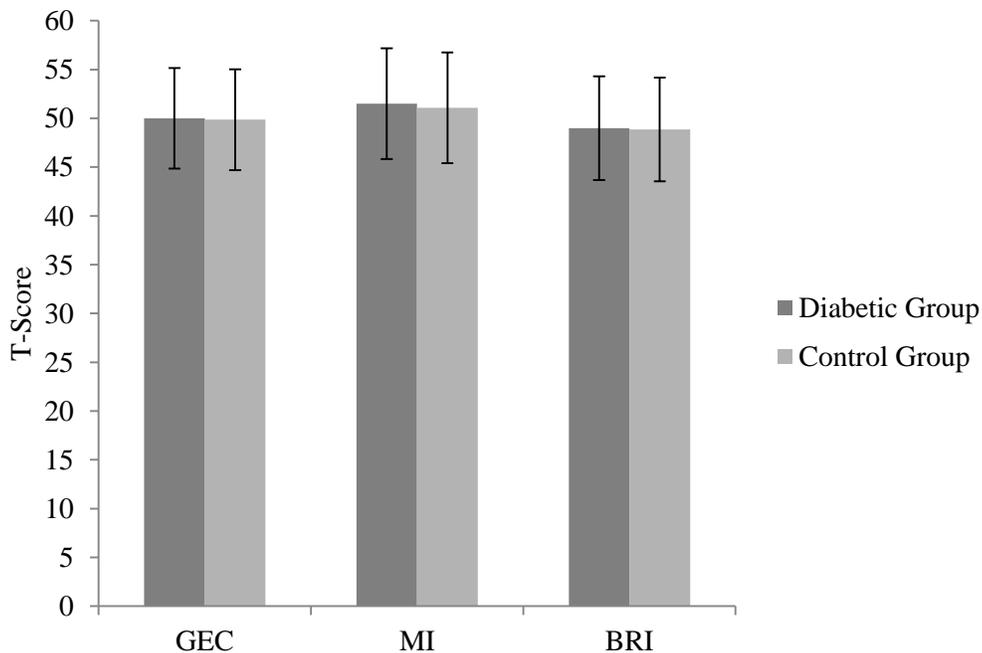
For the SST, initial ANOVA analysis found the control group committed on average significantly less errors than the diabetic group,  $F(1,26) = 5.51$ ,  $p = .03$ ,  $\eta_p^2 =$

.18. After inclusion of the covariate this result remained significant (Table 5). The differences observed on the SST however do not appear related to variations in IQ and therefore may suggest that the diabetic group experienced greater difficulty than the control group with set-shifting ability.

### **Self-Report Measure of EF.**

*The Behavioral Rating Scale of Executive Function Self-Report (BRIEF-SR)*

was used to provide a subjective measure of EF. The three primary BRIEF index scores examined for comparison were: Behavioral Regulation Index (BRI), Metacognition Index (MI) and Global Executive Composite (GEC). Figure 5 illustrates a comparison of each group's average performance on the three indices. Groups were remarkably similar in their performance on this measure, with the control group scoring marginally better on each index.



*Figure 5.* Between group comparisons of mean scores on BRIEF Global Executive Composite (GEC), Metacognition Index (MI) and Behavioural Regulation Index (BRI) including 95% confidence intervals. Higher score indicates greater impairment.

To compare the groups' performance, a series of between-group ANOVAs were completed. Given that the BRIEF index scores were unlikely to be significantly related to IQ (Hocking, Reeve, & Porter, 2015), no covariate was included in analysis. None of the differences were found to be significant — BRI  $F(1,26) = .002$ ,  $p = .97$ ,  $\eta_p^2 = <.01$ , MI  $F(1,26) = .010$ ,  $p = .91$ ,  $\eta_p^2 = <.01$ , and GEC  $F(1,26) = .002$ ,  $p = .97$ ,  $\eta_p^2 = <.01$ .

Table 6

*Between-Groups Comparison of Number of Participants with BRIEF Index T-scores Exceeding Clinical Cut-Off*

Index	Group				P	Cramer's V
	Diabetic		Control			
	n	(% of Diabetic)	n	(% of Control)		
BRI	1	(7.1)	1	(7.1)	1.00	.000
MI	2	(14.3)	1	(7.1)	1.00	.115
GEC	1	(7.1)	0	(0)	1.00	.192

*Note:* BRIEF = Behavioural Rating Inventory of Executive Function; BRI = Behavioural Regulation Index; MI = Metacognition Index; GEC = Global Executive Composite. FET = Fishers Exact Test – two sided.

To further examine each group's performance on the BRIEF, a comparison was made between the groups based on the number of participants whose T-score exceeded the clinical cut-off of 65 for the three primary indices (Table 6). For all indices a total of six participants were found to have scores exceeding the clinical cut-off, with the diabetic group having twice the number of individuals (n=4) compared with the control group (n=2). Given the small total number of participants over the cut-off, a Fishers Exact Test (two-sided) was performed. The number of individuals exceeding the cut-off on the BRI, MI or GEC was not found to be significantly related to group type ( $p = 1.00$  for each index). This suggests that individuals in the

diabetic group were no more likely than those on the control group to score over the cut-off on the indices of the BRIEF, however the very small sample size suggests this result should be interpreted with caution.

### **Summary**

Between-groups analyses of results on the performance-based measures of EF found few significant differences. In general the control group performed better on the tasks of the D-KEFS while diabetic group performed better on the majority of Cogstate tests. After inclusion of IQ as a covariate, a significant difference was observed between groups on the Cogstate SST on which the diabetic group committed a greater average number of errors than the control group, potentially indicative of an impairment in set-shifting ability. On the self-report measure of EF, the BRIEF-SR, no significant differences were observed between groups on any of the three primary indices —BRI, MI or GEC. While the diabetic group had more participants with index T-scores over the clinical cut-off of 65, it was found that this was not significantly related to group type.

### **Relationships between Performance-Based and Subjective Measures of Executive Function**

Correlations were conducted for the diabetic and control group separately to examine the relation between scores on the performance-based tests of EF and the three indices of the BRIEF (Table 7). Generally, stronger relationships between the BRIEF and performance-based measures were observed for the diabetic group in comparison to the control group. For many of the measures, directionality of the relationship was different within each group.

While the majority of these relationships were not significant, this was an

unexpected pattern. For example, the Cogstate GMLT and GMLTDR errors were positively related to the BRI and MI T-score for the diabetic group however were negatively related to these same indices for the control group. This suggests greater impairment as measured by the BRIEF may be associated with more errors in learning and recall of the maze task for the diabetic but not control group.

Table 7  
*Correlations Between BRIEF Index T-scores and Performance-Based Measures of EF for Diabetic and Control Groups*

Measure	Group					
	Diabetic <i>n</i> =14			Control <i>n</i> =14		
	BRI <i>r</i> <sub>s</sub>	MI <i>r</i> <sub>s</sub>	GEC <i>r</i> <sub>s</sub>	BRI <i>r</i> <sub>s</sub>	MI <i>r</i> <sub>s</sub>	GEC <i>r</i> <sub>s</sub>
CWI (SS)	-.3	-.29	-.32	.17	-.03	.15
CWIS (SS)	-.16	-.42	-.46	.51	.19	.43
NLS (SS)	-.53	-.82**	-.73**	.15	-.31	-.04
TCT (mps)	-.36	-.09	-.38	.17	.10	.28
GMLT (errors)	.22	.29	.35	-.20	-.19	-.14
GMLTDR (errors)	.39	.42	.35	.00	-.15	.05
OBT (reaction time)	-.4	-.63*	-.63*	-.24	-.42	-.58*
TBT (accuracy)	-.35	-.20	-.29	-.18	.33	.19
SST (errors)	.01	.56*	.36	-.61*	-.57*	-.54*

*Note:* \**p*<.05, \*\**p*<.01 (2-tailed). CWI=color-word inhibition; CWIS; color-word inhibition switching; NLS=number-letter sequencing; TCT=*Timed Chase Test*; GMLT= *Groton Maze Learning Test*; GMLTDR; *Groton Maze Learning Test - Delayed Recall*; OBT= *One-Back Test*; TB=*Two-Back Test*; SST=*Set-Shifting Test*. SS=scaled score; mps=moves per second; BRI= Behavioral Regulation Index; MI = Metacognition Index; GEC = Global Executive Composite; BRIEF = Behavioral Rating Inventory of Executive Function.

**Significant correlations.** A Spearman's rank-order correlation found that for the diabetic group, BRIEF MI T-score was negatively related to D-KEFS NLS scaled score and Cogstate OBT reaction time, while positively related to Cogstate SST errors. This result suggests that for the diabetic group, an increasing score on the MI (greater level of impairment) is associated with a worse performance on the NLS task, reduced speed on the OBT and a greater number of SST errors. Similarly to the MI, the GEC was also negatively related to NLS scaled score, and the OBT.

For the control group BRIEF BRI, MI and GEC T-score all significantly correlated with Cogstate SST errors. However converse to the diabetic group, this result suggests a higher BRIEF index score is associated with fewer Cogstate SST errors. The only other significant relationship observed was between GEC score and the OBT. Similar to the diabetic group, this suggests an increased GEC score is associated with reduced speed (increased reaction time) on the OBT.

### **Relationship between Diabetic Related Variables and EF**

The second primary aim of the current study was to examine the relationship between diabetic related variables and EF performance. This was intended to support the between-group comparisons by providing additional information about specific factors which may be affecting EF for the young adult with T1DM.

Spearman's correlations were performed between three diabetic specific variables (HbA1c, age of diagnosis and time since diagnosis) and scores obtained on the performance-based measures of EF as well as the scales and indices of the BRIEF. No significant correlations were observed between any of the objective measures of EF and HbA1c, age of diagnosis or time since diagnosis. A summary of

the correlations between the BRIEF and diabetic related variables are presented in Table 8 (details of the composition of the BRIEF can be found in Table 3, page 30).

Table 8  
*Correlations Between HbA1c, Age of Diagnosis, Time Since Diagnosis and T-scores on the BRIEF for the Diabetic Group (n=14)*

Index/Scale	HbA1c	Age of Diagnosis	Time since Diagnosis
	$r_s$	$r_s$	$r_s$
Behavioural Regulation Index	.79*	-.27	.30
Inhibit	.65*	.11	-.03
Shift	.49	-.28	.18
Emotional Control	.40	-.36	.39
Monitor	.71**	-.52	.53
Metacognition Index	.31	.35	-.28
Working Memory	.30	.12	.04
Plan/organise	.26	.42	-.39
Organisation of materials	.46	.12	-.04
Task completion	.10	.26	-.24
Global Executive Composite	.52	.12	-.05

*Note:* \* $p < .05$ , \*\* $p < .01$  (2-tailed). BRIEF=Behavior Rating Inventory of Executive Function

Generally a participant's age of diagnosis negatively correlated with the BRI and most of its contributing subscales, while positively correlating with the subscales of the MI. In contrast, time since diagnosis demonstrated predominantly negative correlations with the MI and the subscales and positive correlations with the BRI and its scales. This suggests that an earlier the age of diagnosis and longer disease duration is associated with greater likelihood of impairment in the area of behavioural regulation, but lesser likelihood of difficulty in the area of metacognition

— namely planning, organisation and task completion. It should however be noted that this was just a general trend and no significant relationships were observed.

In general HbA1c was found to have relatively large (considering the small sample size) and exclusively positive correlations with all BRIEF subscales and indices. It was found that participants' HbA1c was significantly related to scores on the BRI ( $r_s=.79$ ,  $p<.001$ ), the 'Inhibit' scale ( $r_s=.65$ ,  $p=.012$ ) and the 'Monitor' scale ( $r_s=.71$ ,  $p=.004$ ). This suggests that an increase in HbA1c, which typically indicates poorer long-term glycaemic control, is associated with increasing impairment in the ability to inhibit impulsive responses and monitor one's behaviour. No significant correlations were observed between HbA1c and the MI or its' contributing subscales.

**Within session factors and EF performance.** An important part of the study design was to ensure diabetic participants were assessed while in a euglycaemic state to limit any potential influence of transient BGLs on EF. While HbA1c reflects longer term glycaemic control, current BGL reflects a potential acute effect. To examine whether the minor variations in BGL while in the euglycaemic condition influenced participant's EF performance, a Spearman's correlation was performed comparing mean BGL with performance-based measures of EF which can be observed in Table 9. Correlations between the BRIEF scores and mean BGL were not calculated as this measure was not completed in the euglycaemic condition.

Generally participant's D-KEFS scores were positively related to mean BGL suggesting higher average blood glucose improved performance on these tasks. Higher mean BGL was also related to improved performance on the majority of the Cogstate tests.

There was a significant positive relationship between mean BGL and participants' scores on both the OBT and TBT of the Cogstate which relate to speed and accuracy of working memory respectively. This suggests that a participant with a higher average BGL during the assessment was more likely to have a slower reaction time on the OBT, but be more accurate in their decisions on the TBT.

Table 9  
*Correlations Between Mean Blood Glucose Level During Assessment and Scores on Performance-Based Measures of EF for the Diabetic Group (n=14)*

	Mean BGL
	$r_s$
D-KEFS (SS)	
CWI	.24
CWIS	.22
NLS	.50
Cogstate	
TCT (mps)	.51
GMLT (errors)	-.20
GMLTDR (errors)	-.26
OBT (reaction time)	.59*
TBT (accuracy)	.54*
SST (errors)	-.39

*Note:* \* $p < .05$ , \*\* $p < .01$  (2-tailed). CWI=color-word inhibition; CWIS; color-word inhibition switching; NLS=number-letter sequencing; TCT=Timed Chase Test; GMLT = Groton Maze Learning Test; GMLTDR= Groton Maze Learning Test - Delayed Recall; OBT= One-Back Test; TBT=Two-Back Test; SST=Set-Shifting Test. SS=scaled score; mps=moves per second; BGL=Blood glucose level (mmol/L).

## Summary of Results

Between-groups analyses of results on the performance-based measures of EF found few significant differences between the groups. Generally the control group performed better on the tasks of the D-KEFS, while on the Cogstate the diabetic group performed better on the majority of tests. A significant difference was observed between groups on the Cogstate SST on which the diabetic group on average committed more errors than the control group, which may indicate impairment in set-shifting ability. For the BRIEF, group's performance was remarkably similar with no significant differences observed between groups on any of the three primary indices—BRI, MI or GEC.

Correlational analysis between the performance-based measures of EF and the BRIEF found patterns unique to each group. While the majority of relationships were not significant, generally stronger correlations were observed between measures for the diabetic group. Within the diabetic group a higher (greater impairment) BRIEF MI score was significantly and at least moderately associated with a worse performance on the D-KEFS NLS task, reduced speed on the Cogstate OBT and a greater number of errors on the SST. Similar significant relationships were also found between the BRIEF GEC and the NLS and the OBT.

For the control group BRI, MI and GEC were significantly and moderately related to Cogstate SST errors, however converse to the diabetic group a higher BRIEF index score was associated with less as opposed to more errors. Similar to the diabetic group, a moderate and significant relationship was observed between GEC score and the Cogstate OBT suggesting increasing impairment as measured by the BRIEF was associated with reduced speed of working memory.

Further analysis focussed solely on the diabetic group to examine the relationship between diabetic related variables and measures of EF. Several significant correlations were observed for the BRIEF, but none were found for performance-based measures of EF. Most notable was the positive relationship between HbA1c and the scores on the BRI index, and the ‘inhibit’ and ‘monitor’ subscales, suggesting poor glycaemic management is potentially associated with impairments in the ability to inhibit impulsive responses and monitor one’s behaviour. It was also found that participants’ mean BGL during assessment was significantly related to performance on the OBT and TBT of the Cogstate which measure speed and accuracy of working memory respectively. This meant a higher mean BGL was associated with reduced speed on the OBT but increased accuracy on the TBT.

## Discussion

This study investigated the effect of T1DM on EF in a sample of young adults, using an age and gender matched control group for comparison. This was achieved by recruiting both diabetic and non-diabetic participants and administering subjective and performance-based measures of EF to both groups. Given that diabetes is primarily a disease of insulin and glucose dysregulation, attempts were made to keep the BGL of the diabetic group within a normal or euglycaemic range for the duration of testing.

The study had two primary aims: to identify any differences on measures of EF between a diabetic and non-diabetic sample of young adults, and to examine any relationship between diabetic related variables and scores on measures of EF. There has been limited previous research specifically looking at the effect of T1DM on EF in this age range. Young adulthood represents a unique developmental period in which healthy and robust executive functioning is essential to navigate many significant new risks and responsibilities, and which may influence one's future health, educational and vocational outcomes.

The primary analysis of the study was to compare each group's scores on the measures of EF. For the performance-based measures, WASI-II full scale IQ was included as a covariate to help reduce the effect of IQ. It was found that the groups did not differ significantly on most of the administered measures. The exception to this was the Cogstate SST, on which the diabetic group performed more poorly, demonstrated by a greater average number of errors. This result is consistent with the study by Ly et al. (2011), in which it was found a sample of young adults with T1DM

committed more perseverative errors on the *Wisconsin Card Sorting Test* (WCST), than a healthy matched control group. The WCST is similar to the SST in that they both require the participant to learn and apply a rule in response to feedback.

Interestingly, the diabetic sample used by Ly et al. (2011) all experienced early-onset diabetes, with a mean age of diagnosis of 3.3 years, and mean disease duration of 16 years. Given that disease duration has been associated with increasing impairments in EF (Desrocher & Rovet, 2004), it would follow that a young adult diagnosed early in life may be more likely to show impairment in EF than an equivalent peer with a more recent diagnosis.

In the current study, mean age of diagnosis was 11.3 years with only two participants receiving a diagnosis before the age of five. This suggests that the group differences observed in set-shifting ability may be independent of disease duration, and may represent a unique neurocognitive impairment associated with disease pathology. This assertion is tentatively supported by the lack of any significant relationship between disease duration and diabetic participant scores on the SST.

The D-KEFs NLS task is also considered a measure of set-shifting, requiring the participant to switch between alternating stimuli to complete a sequence. Like the WCST, the NLS task is believed to activate similar cortical structures — the dorsolateral and medial PFC (Riehemann et al., 2001; Zakzanis, Mraz, & Graham, 2005). However, unlike the SST, the groups did not differ significantly on this task. Several possible explanations could account for this.

It is possible that the set-shifting test of the Cogstate and the NLS task are not equivalent measures of set-shifting ability. On face value, the former is a computerised task while NLS is pen and paper based. The NLS requires the

individual to alternate between different stimuli under a fixed-rule condition —that is they must alternate between number and letter in ascending order, while the Cogstate test requires a change of response dependent on schedules of reinforcement —that is the participant’s correct response produces a new card, or the card remains and an error-sound signals that they must determine the new target card via trial and error.

Latzman and Markon (2010) make an interesting assertion in their factor analysis of the D-KEFS structure, finding that the NLS task loaded most strongly on the ‘inhibition’ factor. That is the individual must inhibit the prepotent response of completing the respective category in order (numbers or letters) to accurately complete the task. They must also be able to recall numbers and letters from long-term memory, while keeping track of where they are in the sequence, drawing on working memory ability (SÁNchez-Cubillo et al., 2009) and rely on visual-motor ability. Considering Miyake et al. (2000) proposed core EFs: shifting, inhibition and updating of working memory — the NLS could be considered a test of all these domains.

The primary outcome measure of the NLS is completion time, which is converted to a scaled score, while the Cogstate SST measures errors. The extent to which NLS errors affect completion time is dependent on the examinee noticing the error or the examiner providing a prompt to make a correction. Given that the study sample rarely committed errors on the NLS task, it is possible that task requirements other than ‘shifting’ exerted greater influence on completion time; or that it is a least difficult to quantify how an individual’s set-shifting ability specifically contributed to their NLS score.

As the Cogstate task does not appear to place similar demands on working

memory or inhibition, it could be posited that it is a more accurate reflection of set-shifting ability. It is possible that if the NLS task was more complex, or if the study population was less functional (a lower mean IQ for example) that between-group differences would have been observed on this task also.

It should be noted that an important limitation to findings on the Cogstate SST was the difficulty observed in some participant's comprehension of test requirements. A number of participants reported they did not understand the instructions of the task, even after repeated explanation. It is possible that some participants were responding at random, rather than in accordance with the task instructions. This potential inconsistency is undesirable, however it should be considered that both groups received the same instructions, and difficulty understanding the task was not only a diabetic group anomaly, which may have exerted some balancing effect.

While there were no other between-group scores that were significantly different, a general trend was that the control group on average performed better on the tests of the D-KEFS, while the measures of the Cogstate provided mixed results. Interestingly the diabetic group performed better on the Cogstate maze learning and delayed-recall tests.

The GMLT is broadly categorised as a general measure of EF; the participant is required to find the same hidden pathway over a number of trials in response to trial and error feedback while following the rules of the maze. The primary outcome measure is total number of errors. Delayed-recall of this pathway is considered primarily an assessment of spatial memory, and is also measured by total errors. It would be difficult to explain why the diabetic group would be better at these tasks, and more than likely the trend is attributable to sample variation. It is however worth

noting that in the meta-analysis examining diabetes and T1DM by Brands et al. (2005), it was found that the domains related to learning and memory showed relatively small impairment in individuals with T1DM, and did not differ significantly from non-diabetic controls. It is also relevant that in the absence of poor glycaemic control, any potential cognitive decline associated with T1DM may be modest even over long disease duration (Jacobson et al., 2011). Given the relative youth and high level of functioning of the current study sample, it is especially unlikely that between-group differences would be observed on cognitive domains not highly sensitive to diabetes.

On the Cogstate OBT and TBT, which are considered primarily tasks of working memory, the diabetic group on average appeared to respond faster (OBT) but with decreased accuracy (TBT). This may be indicative of a more impulsive response style which also could reflect poorer inhibitory processes. As previously mentioned, while not significantly different, the control group on average performed better than the diabetic group on the measures of the DKEFS, all of which place demands on inhibitory control. This may suggest that there was greater impairment in inhibition for the diabetic group.

Jasinska et al. (2012) found that impulsivity and poor inhibitory control were associated with increases in unhealthy eating in young adults — a pertinent finding given the demands placed on diabetic individuals to monitor and modulate their glycaemic intake. Poor inhibitory control is also associated with decreased academic performance (Garner, 2009), which is also relevant given that many of the study demographic are still enrolled in education.

Interestingly, inhibitory control appears to have limited specific research in diabetes literature, aside from the specific influence of variation in acute blood glucose levels (e.g., Graveling, Deary, & Frier, 2013). A study by Rovet and Alvarez (1997) which focussed on attentional processes, found no significant difference between type 1 diabetic children and adolescents and a matched control group on a computer based task of inhibition. However a subset of the diabetic group who had experienced a history of at least one seizure following hypoglycaemia, performed significantly worse than the control group on the same task.

Ishizawa, Kumano, Sato, Sakura, and Iwamoto (2010) found impaired response inhibition compared to healthy controls in a sample of middle-aged type two diabetics as assessed by a Go/NoGo task of inhibition. It is however unclear if this pattern would be the same in young adults with T1DM (Kodl & Seaquist, 2008) and the inhibitory components assessed by the Go/NoGo and those of the D-KEFS (specifically the CWIT) may have limited commonality (Morooka et al., 2012). A more thorough investigation into the specific components of inhibition potentially affected by T1DM could be a useful avenue for further research.

Given the BRIEF is a subjective, questionnaire-format assessment, it was considered useful to examine how this correlated with the D-KEFS and Cogstate to compare and contrast a behavioural rating scale with performance-based measures for each group. The most salient of the observed relationships are discussed below. In general the results were quite varied, with some inconsistent and unusual differences between groups which likely reflected the small study sample.

An interesting finding was the significant correlation between D-KEFS NLS task and the BRIEF MI and GEC, which shared a strong negative relationship in the

diabetic group but was more weakly and not significantly in the control group. This suggests for the diabetic group, poorer performance on the NLS task (set-shifting) was related to increasing impairments in overall executive function as measured by the BRIEF. This finding is unusual in that a number of studies have generally found little correlation between the BRIEF and performance- based measures of EF in other (non-diabetic) clinical populations (e.g., V. A. Anderson, Anderson, Northam, Jacobs, & Mikiewicz, 2002; McAuley, Chen, Goos, Schachar, & Crosbie, 2010; Vriezen & Pigott, 2002).

Toplak, West, and Stanovich (2013) suggest that in general “performance-based measures of executive function provide important information regarding efficiency of processing, but ratings of executive function tell us more about success in rational goal pursuit” (p.138). This statement is supported by McAuley et al. (2010) who found that the BRIEF correlated well with reported behavioural disruption and impairment but not any performance-based measure of EF — suggesting the true value of the BRIEF may in part lie in its’ ecological validity.

While the NLS is considered a measure of set-shifting ability, the MI primarily purports to measure one’s ability to plan, organise and complete tasks. Given that a central cognitive skill associated with set-shifting is problem solving (Suchy, 2009), which is also essential for good diabetes management (Glasgow, Toobert, Barrera, & Strycker, 2004) — it is possible that in the current diabetic population, impairments on the NLS and MI may both be related to problem-solving ability. This may in turn facilitate poor diabetes management and increase the likelihood of future impairment.

A study by Mitchell and Miller (2008) found that in a sample of community-

dwelling older adults, the NLS task was the best predictor of functional status among four D-KEFS tests employed. While this is a considerably different population, it suggests that there is a precedent for relatedness of the NLS and a more functionally relevant measure such as the BRIEF.

It should be noted that as previously discussed, the NLS task is likely not a ‘process-pure’ assessment of set-shifting ability. However the Cogstate SST also shared significant yet opposing relationships with the MI for each group. This was again unusual but further supports the notion that increased impairments in set-shifting may be related to functional impairments as measured by the BRIEF in a diabetic sample.

In summary, within a diabetic population a rating scale such as the BRIEF may hold more ecological validity than a performance-based measure, but may also provide information about underlying executive processes. The NLS task or similar trail-making tests may correlate with behavioural or functional outcomes as a result of impairments in problem-solving ability. Further research with a larger study population may be useful to understand this relationship further.

Analysis solely focussed on the diabetic group data provided some insight into other diabetic specific variables related to EF. Most notable was the relationship between HbA1C, a measure of longer term (2-3 months) glycaemic control and the BRIEF, namely the BRI, and the ‘Inhibit’ and ‘Monitor’ subscales. Broadly speaking, the BRI purports to measure attentional, inhibitory and emotional components of EF employed when modulating behaviour. That this relationship exists is unsurprising as one likely needs considerable behavioural control to effectively engage in the self-care required to manage diabetes, and is not a unique finding (e.g., Bagner, Williams,

Geffken, Silverstein, & Storch, 2007; McNally, Rohan, Pendley, Delamater, & Drotar, 2010). It is however unclear the causal direction, and if elevated BRI scores would be reduced if adequate glycaemic control was restored.

Interestingly, there was no significant relationship between any performance-based measure and HbA1C, however this is consistent with the findings of Suchy et al. (2016) who also used measures of the D-KEFS in a sample of late adolescents with T1DM. This may again reflect differences in the processes being measured.

Another possibility is that broadly speaking the BRIEF, in particular the BRI, is more of a measure of so-called ‘hot’ EFs. That is those EFs related to “stimuli, decisions, and outcomes that are motivationally salient” (Prencipe et al., 2011, p. 622). It is possible that these hot executive processes are more closely related to and influenced by longer term glycaemic control than the ‘cool’ or abstract problem-solving EFs of the performance-based measures. Hot executive processes are suggested to be employed in affective decision-making, delayed gratification, and impulse control and have been found to exhibit a relationship with eating style and weight gain (Groppe & Elsner, 2015).

These findings may hold relevance for diabetes self-care by tentatively suggesting that hot EFs may be particularly vulnerable to poor glycaemic control as evidenced by elevated HbA1C, which in turn can impact patterns of eating and affect-based decision making. Alternatively, impairments in hot EFs may lead to poorer diabetes management and thus higher HbA1c. This is consistent with research by Stupiansky, Hanna, Slaven, Weaver, and Fortenberry (2013) who found that poor impulse-control was associated with poor diabetes management. Given that hot EFs may exhibit a longer developmental time frame than cool EFs (Prencipe et al., 2011),

younger adolescents may be at greater risk of poorly managing their diabetes. The current findings also support the assertion by McNally et al. (2010) that measures of EF may have some additional utility in diabetes management. Future research may benefit from focussing on a potential mediational role of hot executive processes in the context of diabetes self-care.

While not significant, a general trend was that the younger a participant was diagnosed the higher (greater impairment) they scored on the BRI while an older diagnosis was associated with a higher MI score. As might be expected, disease duration showed the inverse of this relationship, negatively correlating with the MI and positively correlating with the BRI.

As disease duration, like HbA1c, is also associated with greater impairment on the BRI, this relationship may allude to hot executive process involvement also — possibly hot EFs are more prone to the longer term effects of diabetes. However, other variables such as the unique psychosocial pressures on a child with T1DM, and the complex relationship between diabetes management and parenting practice (Young, Lord, Patel, Gruhn, & Jaser, 2014) suggests that this association may be much more complex.

Given that the MI purports to measure those components of EF primarily relating to planning, organisation and task completion, it may follow that a longer history of managing diabetes means an individual has developed more aptitude in these areas. However like the relationship with the BRI, it is likely that a simplistic explanation is insufficient.

A final area of interest to the current study was exploring relationships between mean BGL and participants' scores on all measures. A participant's mean

BGL was derived from the multiple readings taken throughout the assessment process. Given the attempts to maintain blood glucose within a euglycaemic range it was surprising to see that minor variations in acute blood glucose demonstrated a significant relationship with the Cogstate OBT and TBT. These tasks assert to measure working memory processes, namely speed and accuracy respectively.

It has been well documented that acute BGL is related to cognitive performance in several domains (e.g., Cox, Kovatchev, Gonder-Frederick, & Summers, 2005; Graveling et al., 2013), however the current findings suggest that certain areas of EF may be particularly sensitive. A recent study by Hawkins, Gunstad, Calvo, and Spitznagel (2016), found that even among a sample of healthy young adults tested within a ‘normal’ BGL range, higher blood glucose was associated with poorer inhibitory control. While the Cogstate tests in discussion are more related to working memory than inhibition, a larger question remains as to the sensitivity of EF in general to subtle changes in acute blood glucose levels—and to what extent this may have also exerted influence on the control group performances.

Interestingly, the Cogstate findings suggest that increasing mean BGL was associated with slower but more accurate working memory processes. This is potentially indicative of a speed-accuracy trade-off which is also associated with later disease onset (Desrocher & Rovet, 2004). Another contributing factor could be an attentional lag, which is a residual impairment in attention observed by Rovet and Alvarez (1997) that occurs when a diabetic individual returns to a euglycaemic state. This may mean for those individuals presenting at the test session with a high BGL (and likely recording a higher mean BGL), a residual impairment in attention may have encouraged a more cautious response style.

These findings hold several implications for future research. Firstly that minor variations in BGL may influence EF, even in healthy controls. It may be useful to collect blood glucose data from any control group, and to control for BGL in analysis. It would also be preferable to ensure adequate time in a euglycaemic state for any diabetic individual prior to assessment to allow for any latency in post-hyperglycaemic cognitive function.

### **Study Limitations and Considerations**

The current study experienced a number of limitations, the most central of which are discussed below. Likely the most salient limitation was the small sample size which made meaningful interpretation of data difficult and conclusions tentative at best. This was primarily a reflection of the difficulty in recruiting a young adult clinical population. Potential participants were often difficult to contact, would fail to return contact and several failed to attend scheduled research appointments. Many of the study population reported difficulty finding time to attend appointments due to other commitments or a lack of suitable transport. This was partly a function of a lack of flexibility in the testing schedule and testing location. All research appointments were held at the WRDS, before 12pm, and only on certain weekdays as required for ethical approval. It is also possible given the relatively long time commitment per session (up to four hours), there was insufficient incentive for young people to attend.

The difficulty in recruitment also suggests that it is likely that the study group were not a representative sample, meaning the selection process favoured those who were willing and had resources to invest (i.e., time, money, transportation). It is possible this was evidenced in the relatively high estimated IQ of the sample, suggesting a greater than average level of functioning. While the control group were

relatively well matched on IQ, it would have been interesting to include a more diverse and potentially more representative diabetic sample. It is also of note that a disproportionate number of diabetic participants were male. It is unclear why this was the case given that T1DM is only slightly more common in males. It is possible that if a larger sample had been recruited there would have been a more even gender balance.

Future studies may benefit from streamlining the assessment procedure, providing a more flexible testing schedule (e.g., after hours, weekends) and potentially using other platforms for communication (e.g., social media) to connect with a young adult population and encourage more equal gender participation.

Given the functional level of the study population, it is unclear whether all the measures used in the current study possessed suitable sensitivity to minor variations in EF. In particular the measures of the D-KEFS, which are pen and paper based and require accurate timing by the examiner, allow some room for testing discrepancy. Given that most young adults are completing the measures relatively fast and with few errors (compared for example to older adult population), small variations in completion time may hold greater significance — and any examiner errors more influential. A study of the D-KEFS by Keifer and Tranel (2013) also raised concerns about the sensitivity of the D-KEFS in detecting subtle executive dysfunction, suggesting the measure has a low floor and high ceiling with insufficient items of medium difficulty.

By comparison the Cogstate records results automatically placing less reliance on the examiner, and has been found to correlate well with more traditional neuropsychological tests (Maruff et al., 2009). Given the likelihood that many young

adults are frequently engaged with media devices, future research may benefit from continuing to find ways to use computerised platforms for test administration with this population.

A final and pertinent methodological consideration is the difficulty of consistently defining EF. According to Baggetta and Alexander (2016), a meta-analysis yielded the conclusion that EF is one of the most widely cited yet confusing cognitive constructs. This fact became more apparent throughout the duration of the current study, and suggests a more narrow focus potentially looking at a specific component of EF may have aided in maintaining conceptual clarity.

### **Summary**

The current study sought to better understand the relationship between T1DM and EF in a sample of young adults. Few differences were observed between the diabetic and control group on the measures of EF used — only on the Cogstate SST did the control group significantly outperform the diabetic group, however this result was brought into question by difficulties in task comprehension. In general however the control group performed better on the majority of tasks of EF than the diabetic group — a larger sample size would be required to explore this trend more definitively.

Along with set-shifting, there was some evidence the diabetic group may also have more difficulty with inhibitory control, impulsivity and problem-solving ability which may have implications for patterns of eating, risk-taking behaviour and overall diabetes management.

In examining the diabetic group it was found that subtle changes in BGL, within the euglycaemic range, may have an impact on certain EFs, which may suggest that

tighter glycaemic control is required for both diabetic and control groups when trying to isolate the effects of T1DM on EF. It was also proposed that hot executive processes may interact with HbA1c and play a part in diabetes management.

While not significant, disease duration was associated with poorer BRI scores on the BRIEF, but a better MI score. It was posited that this relationship may be subject to a complex range of psychosocial influences. It was found that the BRIEF shared a significant relationship with both HbA1c and several performance-based measures of EF, suggesting that the BRIEF may have useful ecological validity in a diabetic population, and be of value in diabetes management.

While a number of limitations made it hard to draw definitive conclusions, the current study findings tentatively suggests that in a young adult population there is some evidence that the presence of T1DM is related to impairments in certain areas of EF — including set-shifting, inhibition and the higher-level construct of problem-solving ability. There is also some evidence that in this population the diabetic-related variable HbA1c shares a relationship with EF as measured by the BRIEF, but not with performance-based measures of EF. Future research suggestions for this population included: specifically examining the EF of inhibition; further examination of the relationship between the NLS task (trail-making) and the BRIEF; the role of hot EF in diabetes self-care, and the sensitivity of specific EF to subtle changes in BGL within a euglycaemic range.

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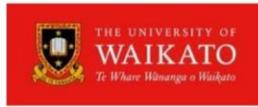
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## Appendices

## Appendix A: Control Group Recruitment Flyer



# Research participants required!

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Effects of Blood Glucose Levels on Driving Behaviour and Executive  
Functioning in Young People with Type 1 Diabetes

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As part of my Masters project I am looking to recruit willing participants to complete a number of assessments related to driving behaviours and executive function. This is an exciting opportunity to be part of some valuable research. At the conclusion of assessment participants will be offered 2% course credit (eligible courses include: PSYC208, 229, 317 and 319) or a \$20 gift voucher.

Suitable candidates will:

- Hold a full or restricted driving licence
- Drive regularly (>4 times per week)
- Be between the ages of 16-24
- Be non-diabetic (i.e. do not have diabetes)

What will you need to do:

- Attend one research session on campus for approximately 2 hrs
- Complete some questionnaires, assessments and some computer based driving tasks

For more info please contact Matt with details shown below.

*This project is being supervised by Dr Nicola Starkey and Dr Robert Isler, and has been approved by the Research and Ethics Committee (Ref 14:80)*

Matt Reid 027 634 8546  
earplayproductions@hotmail.com

## Appendix B: Participant Information and Consent Forms

### Participant Information Sheet

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**Project Title:** Effects of Blood Glucose Levels on Driving Behaviour and Executive Functioning in Young People with Type 1 Diabetes

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**Researcher:** Matthew Reid **Supervisors:** Dr Nicola Starkey, Dr Robert Isler

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I would like to invite you to participate in this project looking at the effects of blood glucose levels on driving behaviour in young people with Type 1 Diabetes. For this particular research sample we are looking to gather data from a non-diabetic population to use as a control group for comparison with data collected from a larger parent study. This project is being conducted as part of my Master of Social Sciences at the University of Waikato.

This Participant Information Sheet will help you decide if you'd like to take part. We will go through this information with you and answer any questions you may have. You do not have to decide today whether or not you will participate in this study. Before you decide you may want to talk about the study with other people, such as family, whānau, friends. Feel free to do this.

If you agree to take part in this study, you will be asked to sign the Consent Form. You will be given a copy of both the Participant Information Sheet and the Consent Form to keep.

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#### WHAT WILL I HAVE TO DO?

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This project will involve you coming to the University of Waikato on a single occasion. You will complete some questionnaires and carryout some game-like virtual driving exercises. You will also complete some psychological tests that examine your thinking, problem-solving, and reasoning skills. Your appointment time will take approximately one and a half hours.

As we are trying to minimise any effect blood glucose fluctuations may have on your performance, we will try to arrange your appointment to occur approximately two hours after your last main meal, and request you refrain from high carbohydrate (sugar) snacks prior to assessment.

As recognition of your time and participation you will receive a \$20 voucher at the end of assessment, or if eligible, 2% course credit. Eligible papers for course credits include PSYC208, PSYC229, PSYC317 and PSCY319.

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#### WHAT ARE THE POSSIBLE BENEFITS AND RISKS OF THIS PROJECT?

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There are no anticipated risks associated with this research project. You are welcome to take a short break any time during testing if required.

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#### WHAT WOULD EXCLUDE ME FROM PARTICIPATING IN THIS PROJECT?

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Exclusion criteria for this project include:

1. Being pregnant
2. Regular use of illicit substances

This is to ensure that the results on the tests and exercises are not influenced by other factors.

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**WHAT ARE MY RIGHTS?**

Whether or not you take part is your choice. If you don't want to take part, you don't have to give a reason. If you do want to take part now, but change your mind later, you can pull out of the study at any time. The information that is collected from you will still be part of the research.

The information collected will be stored confidentially and securely. The data will be stored on the University of Waikato computer system. It will be accessed by a password by the researcher collecting the data. The data will be held indefinitely and may be used in future research.

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**WHAT HAPPENS AFTER THE STUDY?**

At the end of the study, a summary sheet with the general research findings will be sent to you directly.

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**WHAT IF SOMETHING GOES WRONG?**

If you were injured in this study, which is unlikely, you would be eligible for compensation from ACC just as you would be if you were injured in an accident at work or at home. You will have to lodge a claim with ACC, which may take some time to assess. If your claim is accepted, you will receive funding to assist in your recovery.

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**WHO CAN I CONTACT?**

If you have any questions, concerns or complaints about the study at any stage, you can contact:

**Dr Nicola J. Starkey**

Associate Professor in Psychology  
University of Waikato School of Psychology  
Private Bag 3105, University of Waikato, Hamilton  
07 838 4466 ext. 6472      nstarkey@waikato.ac.nz

*This project is being supervised by Dr Nicola Starkey and Dr Robert Isler, and has been approved by the Research and Ethics Committee (Ref 14:80)*

**CONSENT FORM**

**Project Title:** Effects of Blood Glucose Levels on Driving Behaviour and Executive Functioning in Young People with Type 1 Diabetes

Please complete the following checklist. Tick (✓) the appropriate box for each point.	YES	NO
1. I have read the Participant Information Sheet (or it has been read to me) and I understand it.		
2. I have been given sufficient time to consider whether or not to participate in this study		
3. I am satisfied with the answers I have been given regarding the study and I have a copy of this consent form and information sheet		
4. I understand that taking part in this study is voluntary (my choice) and that I may withdraw from the study at any time without penalty		
5. I have the right to decline to participate in any part of the research activity		
6. I know who to contact if I have any questions about the study in general.		
7. I understand my response to the Hazard Perception task will be audio recorded.		
8. I understand that my participation in this study is confidential and that no material, which could identify me personally, will be used in any reports on this study.		

**Declaration by participant:**

I agree to participate in this research project and I understand that I may withdraw at any time. If I have any concerns about this project, I may contact the convener of the Psychology Research and Ethics Committee (Dr James McEwan, Tel: 07 838 4466 ext 8295, email: [jmcewan@waikato.ac.nz](mailto:jmcewan@waikato.ac.nz))

Participant's name (Please print): \_\_\_\_\_

Signature: \_\_\_\_\_ Date: \_\_\_\_\_

**Declaration by member of research team:**

I have given a verbal explanation of the research project to the participant, and have answered the participant's questions about it. I believe that the participant understands the study and has given informed consent to participate.

Researcher's name (Please print): \_\_\_\_\_

Signature: \_\_\_\_\_ Date: \_\_\_\_\_

## Appendix C: Medical and General Background Information Forms

RegNo       PPT initials     DOB:

### BGL to be checked every hour during the assessment

E.1.2	Date of assessment (dd/mm/20yy)	

#### 1. General background information

E1.1	Who do you live with? (tick one only)	Living with family Living with others Living with partner Alone
	What is your work situation? (tick one only)	Student Full time paid work Part time paid work Unemployed or redundant Beneficiary Other
	If other, please specify (Text)	
	If a student what year at school or study are you in?	
	How do you rate yourself academically compared to your classmates	Above Average Average Below Average
	If employed, what is your occupation (Text)	
	What is the main lifetime occupation of the main income earner? (Text)	
	What is your current relationship status? (tick one only)	Single Partner
	How many hours per week do you spend doing physical exercise?	
	How many hours per week do you spend on screen time?	

RegNo       PPT initials       DOB:

**2B. Medical Issues – Non-Diabetes Related**

For the first assessment: Now I am going to ask you about other medical conditions. Has a doctor or medical person **ever** told you that you have any of the following:  
(if any are present, get confirmation details from medical files)

E2.11	Asthma/Respiratory Problems	Present	Not present
E2.12	Vision problems requiring glasses/contact lenses	Present	Not present
E2.13	Hearing problems	Present	Not present
E2.14	Chronic Allergies	Present	Not present
E2.15	Developmental Disability/Handicap	Present	Not present
E2.16	Learning disability	Present	Not present
E2.17	Head Injury	Present	Not present
E2.18	Loss of Consciousness	Present	Not present
E2.19	Concussion	Present	Not present
E2.20	Migraines	Present	Not present
E2.21	Thyroid Problems	Present	Not present
E2.22	High Blood Pressure	Present	Not present
E2.23	High Cholesterol	Present	Not present
E2.24	Irregular pulse (arrhythmia), fibrillation	Present	Not present
E2.25	Genetic Conditions	Present	Not present
E2.26	If yes, specify		
E2.27	Attention Deficit Hyperactivity Disorder	Present	Not present
E2.28	Sexually transmitted disease	Present	Not present
E2.29	Epilepsy/Seizures	Present	Not present
E2.30	Chronic fatigue syndrome, fibromyalgia syndrome, irritable bowel syndrome or similar	Present	Not present
E2.31	Any mental health difficulty (psychiatric illness such as depression, anxiety disorder, schizophrenia, paranoia)	Present	Not present
E2.32	If present, what diagnosis and what health professional (free text)		
E2.33	Is there anything else you have been diagnosed with?	Yes	No
E2.34	If yes, specify (Text)		

RegNo       PPT initials     DOB:

E2.35	How often have you seen your GP in the last 6 months for non-diabetes related issues		
E2.36	What was the primary issue you saw your GP for (free text)		
E2.37	Have you ever been admitted to a hospital other than Waikato Hospital for a non-diabetes-related issue	Yes	No
E2.38	If yes, specify		
E2.39	Have you ever been admitted to a Waikato based Hospital for a non-diabetes-related issue	Yes	No
E2.40	If yes, check medical files		

RegNo       PPT initials       DOB:

### 3. Health

Q #		
E.3.1	Which of these best describes your smoking status? (tick one only)	<p><b>Never smoked</b></p> <p><b>Ex-smoker;</b> smoked cigarettes, ready-made or roll your own; cigars, cigarillos or pipe more than once per day for at least one year</p> <p><b>Current smoker;</b> currently smokes cigarettes, ready-made/ roll your own, cigars, cigarillos or pipe more than once per day for at least one year</p>
E.3.2	Have you ever consumed any type of alcohol? If no, go to question 4	Yes No
E.3.3	If yes, did you <b>ever</b> drink alcohol at least once a month? If no, go to question 4	Yes No
E.3.4	If yes, which of the following best describes how often (tick one only)	Four or more times a day Two or three times a day Once a day Every 2 days Every 3 or 4 days Every 5 or 6 days Once a week Every 10 days Once a fortnight Once a month
E.3.5	Average number of standard drinks consumed on each occasion (2 digits) <i>(for example a standard drink is a small can of beer (330 ml), a small glass of wine or a single measure of spirits, equivalent to 10g of alcohol)</i>	

### 4. Current Medication

Confirm from Medical Records, use list of medications provided.

E.4.4	Have you been prescribed medication for sleep, depression, mood or other psychiatric or psychological illness (psychotropic medication) If yes, continue.	Yes No
E.4.5	What are the psychotropic medications taken in the LAST 7 DAYS (tick all that apply)	Antipsychotic/neuroleptic Anxiolytic Antidepressants Hypnotic

RegNo       PPT initials     DOB:

E.4.6 What other medications have you taken in the last 7 days?  
 (Ask to see that participant's medication bottles/packets to record this information if necessary)

No	Name	Dose	Unit	Dispensing date	Amount of medication (e.g. number of tablets taken each time medicine is taken)	Frequency
1						
2						
3						
4						
5						

