Executive Functioning Outcomes in 7 Year Old Survivors of Bronchopulmonary

Dysplasia

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Declaration

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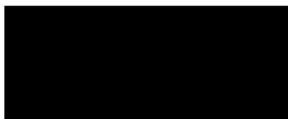
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Abstract

Preterm birth (<37 weeks gestational age) is facing an upward trend, with increased survival rates. Some of these infants are at risk of developing severe medical conditions in the neonatal period, one of the most common being Bronchopulmonary Dysplasia (BPD). To date, the literature suggests that preterm birth is associated with cognitive delay (namely, lower IQ) and that BPD may exacerbate cognitive dysfunction. Less is known about specific cognitive abilities such as executive functions. The role that BPD plays in the developmental trajectory of the preterm infant is surprisingly unknown. Determining how BPD maps onto executive functioning at school age is extremely important in identifying clear weaknesses and designing appropriate interventions for this population. This literature review provides an overview of preterm birth, and the health effects and neurodevelopmental outcomes associated with preterm birth.

Health Effects of Preterm Birth and Neurodevelopmental Outcomes: A Literature Review

Overview

Approximately 15 million preterm births (<37 weeks gestational age) occur globally each year, and of these, more than one million do not survive the initial stages of life due to arising complications (Bickle Graz, Tolsa & Fischer Fumeaux, 2015; World Health Organization [WHO], 2016). A large proportion of infant mortality is confined to poorer nations where prevalence rates of preterm birth climb as high as 18% (i.e., Sub-Saharan Africa), compared to <10% in developed countries such as Australia (WHO, 2016). A recent report states that 8.6% (>26,000) of infants born in 2014 in Australia were preterm (Australian Institute of Health and Welfare [AIHW], 2016). While the mortality rate is extremely low, the rate of preterm birth is stable, if not increasing, given advancements in medicine and neonatal care. This also means that the most at risk preterm infants are surviving, resulting in more cases of serious conditions associated with preterm birth.

With this in mind, the focus for researchers and clinicians lies on infant morbidity, in particular, the relationship between preterm birth and neurodevelopmental functioning. All preterm infants have an elevated risk of poorer outcomes, with medical complications increasing this vulnerability. General intelligence, or an intelligence quotient (IQ) is the most ubiquitously studied outcome; however, there is emerging interest in uncovering the more specific cognitive profile of these infants. This is relevant because preterm infants are vulnerable to complications at birth (i.e., breathing difficulties) yet exactly how these initial physical health experiences map onto specific cognitive impairment during early childhood remains unresolved. The potentially rising number of preterm births in Australia means it is important to establish the neurodevelopmental sequelae associated with preterm birth. Moreover, the added affect that medical conditions have on these outcomes, in particular cognitive functioning, will help to characterise the outlook of Australia's preterm population.

Preterm Birth

Entry into the world for the preterm infant is more complex than those who are term born. The first few days, weeks, and sometimes months can consist of long hospital stays, invasive procedures, and subsequently a disruption in attachment with the primary caregiver (Anderson & Doyle, 2006). Crucial stages of fetal growth are often incomplete, causing the infant to be born with underdeveloped organs (McCormick, Litt, Smith & Zupancic, 2010). Thus, preterm infants usually require special care in the neonatal intensive care unit, are more likely to be re-hospitalised after initial discharge, and are at higher risk of experiencing health problems beyond the newborn period, even in the absence of severe disabilities (Vinall et al., 2014).

In Australia, the largest proportion of preterm infants fall within the moderate to late preterm bracket (32 to 37 weeks gestational age) and the normal birth weight range (2,500 to 4,499 grams) (AIHW, 2016). Since the survival rates of the very (28 to 32 weeks gestational age) and extremely (23 to 28 weeks gestational age) preterm infants have dramatically improved, research has focused on these groups, as they constitute a higher neurodevelopmental risk (Anderson, 2014). Being born too early and being born too small cannot be thought of interchangeably (a preterm infant can be born at the expected weight for their gestational age, just as a term infant can be born underweight), however, infants who experience overlap between these two situations, have the highest risks of mortality and morbidity. Historically, within the literature, preterm birth was defined by birth weight, however gestational age is now considered a more accurate proxy of preterm birth. Therefore, older studies are limited due to this outdated definition.

It has been consistently reported in the literature that gestational age and general intelligence (IQ) share a dose-response relationship (Kerr-Wilson, Mackay, Smith & Pell, 2011). There are a multitude of factors that may contribute to the neurodevelopmental

problems experienced among preterm children that are likely to represent a "complex relationship between cognitive function, biological, and environmental factors, and clinical events during and after the perinatal period of a very preterm birth" (Linsell, Malouf, Morris, Kurinczuk & Marlow, 2015, p. e2).

Physical Health Complications of Preterm Birth

Physical complications seen in the initial stages of the preterm infant's life are in most cases a reflection of developmental immaturity. The brain and lungs are two major organs that are compromised with preterm birth, representing a significant public health issue. The preterm brain is not fully formed and continues to develop after birth, outside of its natural environment, the womb. Similarly, the lungs normally continue developing during the third trimester of pregnancy, therefore preterm infants are born with lungs that often cannot function sustainably on their own. Vital stages of brain and lung development are disrupted, such as synaptic pruning in the brain and surfactant production in the lungs, both of which can result in brain injury and have a detrimental effect on infant development. For example, one of the causes of Cerebral Palsy, is damage to cerebral white matter, one of the most common forms of brain injury present in preterm infants (Khwaja & Volpe, 2008). Moreover, breathing difficulties due to inadequate oxygenation (also called hypoxia) can lead to respiratory disease (Raman, Georgieff, & Rao, 2006). Organ immaturity coupled with exposure to intensive procedures can lead to sustained lung injury. For example, the high levels of pressure generated by mechanical ventilation can have inflammatory side effects (Carvalho, Silveria, & Procianoy, 2013). Hypoxia often leads to other health problems including excessive sleepiness, growth failure, and brain injury (Raman et al., 2006) and may therefore be a risk factor for neurocognitive deficits among children with a history of neonatal illness.

Other health threats among the preterm population include impairments to the nervous system (i.e., sensory), postnatal growth (i.e., growth difficulties), and sepsis (i.e., infection) (McCormick et al., 2010). Preterm infants are also at risk of experiencing problems with temperature control (i.e., hypothermia and low blood sugar levels), the gastrointestinal and digestive systems (i.e., necrotizing enterocolitis), blood conditions (i.e., anemia and jaundice), and metabolism (i.e., hypoglycemia). In addition to health problems in infancy, research also shows that preterm infants are more susceptible to chronic health diseases in adolescence and adulthood, such as heart disease, hypertension, Type 2 diabetes, and asthma (Doyle & Anderson, 2010). It is evident that serious health complications can arise from preterm birth and that they may be at least, a partial explanation for poorer neurodevelopmental outcome at later stages (Nosarti et al., 2008; Woodward, Clark, Pritchard, Anderson, & Inder, 2011).

Bronchopulmonary Dysplasia (BPD)

As aforementioned, the respiratory system is compromised in preterm infants as a result of unformed lungs. The likelihood of insufficient oxygen supply is great and the infant is prone to ongoing complications. Some preterm infants have few air sacs (alveoli) and those that are present tend to be unable to function normally, therefore the infant needs assistance breathing and with other vital lung functions. In some cases, particularly infants with lower gestational age and birth weight, the lungs require protracted periods of assisted help, during which Respiratory Distress Syndrome is likely to develop into a more severe condition called Bronchopulmonary Dysplasia (BPD). BPD affects a significant number of preterm infants, with prevalence rates being as high as 50% in some preterm populations, such as those with very low birth weights (Bhandari & Bhandari, 2007; Raman et al., 2006). BPD was termed in 1967 by Northway, Rosan, and Potter to describe lung injury in preterm infants, typically characterised by severe lung damage (scarring), fibrosis, and insult to alveolar development

(Jobe, 2011; Philip, 2012). This form of classical BPD, also referenced in the context of the "pre-surfactant" era, more often than not resulted in death of the infant who was diagnosed (Philip, 2012). In 1990, exogenous surfactant, a protein substance that aids in the restoration of lung function, was made commercially available, and was responsible for eradicating the most severe forms of BPD (Zhang, Sun, Xue, & Wang, 2013). However, the chronicity of BPD became omnipresent as the survival of preterm infants and ability to provide optimal care and support, with or without associated complications, increased. The "new" BPD is considered a disorder in lung development and in most countries a diagnosis is based on continued oxygen treatment at 36 weeks gestational age with mild, moderate, and severe criteria (contingent on the infant's dependence on oxygen treatment) (Chess, D'Angio, Pryhuber, & Maniscalco, 2006).

The definition of BPD continues to evolve in part due to the development of newer treatment techniques. While the dependence on mechanical ventilators and prolonged oxygen exposure reduces acute risk (and death), such procedures can be detrimental in themselves (i.e., inflammation or scarring resulting from high levels of pressure) (Chess et al., 2006), the effects of which have been studied (i.e., Carvalho et al., 2013). Furthermore, infants with BPD are at increased risk of infection, and problems with other bodily systems (i.e., digesting food). Moreover, studies investigating children with histories of pre-surfactant BPD (Doyle et al., 2006; Northway, Rosan, & Potter, 1967) and those examining new BPD populations (Korhonen, Laitinen, Hyodymaa, & Tammela, 2004) have both found lower levels of airflow than matched controls, highlighting that despite increased survival rates and improved care of these infants, the ongoing respiratory burden remains, even amongst the most modern BPD population. The research suggests that there may be adverse effects of BPD in the long term, such as difficulty in school, lower levels of intelligence, and executive dysfunction (Anderson & Doyle, 2006).

Neurodevelopment of the Preterm Infant

The cognitive, motor, behavioral, and academic outcomes of preterm birth have been well documented throughout the literature where it has been argued that "prematurity has a pervasive effect on all neurodevelopmental domains" (Linsell et al., 2015, p. e2). The likelihood of complications occurring as a result of preterm birth increases with decreased gestational age and can lead to severe neurodevelopmental disability (i.e., Cerebral Palsy) (Nosarti et al., 2008). Other types of brain injury among the preterm baby include white matter abnormalities; magnetic resonance imaging studies have reported that preterm children have decreased cerebral volume in childhood as well as lower levels of volume of cortical grey and white matter, the basal ganglia, and the cerebellum than in age-matched term controls (Ment & Vohr, 2008; Woodward et al., 2011). Insult to the brain, stemming from neonatal medical conditions, can have a detrimental effect on long term neurodevelopment.

Preterm birth can undermine general cognitive ability, including memory and learning, language, visual and perceptual skills, motor development, attention, behavioural problems, and sensory abilities (Anderson, 2014). Such deficits can extend to problems in academic and educational functioning, social performance, and execution of everyday activities. The latter can particularly be affected by problems with more specific skillsets such as executive functioning (EF) abilities. Moreover, preterm birth has been linked with higher rates of psychological disorders, specifically anxiety and depression (Hack et al., 2009; Johnson & Marlow, 2011), and severe neurodevelopmental disorders including but not limited to Attention Deficit Hyperactivity Disorder and Autism Spectrum Disorder (Johnson et al., 2010). Evidently, preterm infants are at risk of a vast array of developmental difficulties that in some circumstances may be fixed throughout the lifespan.

Cognitive Development

Cognitive delay, measured as IQ, among preterm children has been widely reported as significantly lower than that of term peers revealing a relationship between decreased gestational age and lower IQ (Anderson, 2014; Bhutta, Cleves, Casey, Cradock, & Anand, 2002; Kerr-Wilson et al., 2011). An earlier systematic review analysed the results from 15 case-control studies, including 1556 preterm and 1720 term children, and found that the weighted mean difference between cognitive scores was 10.9 points in favour of the controls (Bhutta et al., 2002). Anderson (2014) interpreted this score in terms of IQ, reporting that the preterm population had an IQ of 0.7 standard deviations (SD) lower than their term peers. To address the issue of whether older studies are relevant today's preterm population, a more recent systematic review was conducted confirming earlier results (Kerr-Wilson et al., 2011). The results of 27 studies, (3504 preterm and 3540 term children), revealed a mean difference of 11.9 points (Kerr-Wilson et al., 2011); the preterm children acquired IQ measures that were 0.8 SD below term peers (Anderson, 2014). Associations between IQ and gestational age, but not with year of birth, were reported demonstrating that children of more recent generations did not exhibit greater or improved IQ scores than those of earlier generations (Bhutta et al., 2002; Kerr-Wilson et al., 2011).

Moreover, preterm birth may have consequences for visual-perceptual deficits. An older study reported that preterm children, performed significantly poorer than controls on tasks measuring perceptual motor abilities, had nearly double the amount of abnormal stereopsis (perception of depth, also referred to as binocular vision) compared with controls, and demonstrated deficits in accuracy of spatial attention (Torrioli et al., 2000). More recently, Molloy et al. (2013) found that extremely low birth weight/extremely preterm adolescents had worse visual acuity, stereopsis and visual perception than controls. Preterm children have also exhibited more difficulties than term children in various memory domains,

particularly working memory (Hutchinson, De Luca, Doyle, Roberts, & Anderson, 2013).

There is also evidence of language delay in children born preterm, showing deficits in

phonological awareness, semantics, grammar, discourse, and pragmatics (Barre, Morgan,

Doyle, & Anderson, 2011; Reidy et al., 2013).

Executive Functioning

To date, the majority of studies within this field of research have focused on outcome

measures of general cognitive abilities, namely IQ. Therefore, less is known about specific

cognitive outcomes of preterm school-aged children. Studies of general cognitive

development have highlighted the likely predictive role of executive dysfunction at early ages

(Pozzetti et al., 2014). Naturally, the idea of exploring early executive skills became

increasingly popular and the impact of prematurity and associated medical complications on

executive skills such as working memory, response delay, and planning was stressed

(Pozzetti et al., 2014).

Executive functioning (EF) refers to a set of 'higher-order' cognitive processes that

are essentially responsible for controlling thoughts and behavior. Executive functions include

processes such as inhibitory control, attentional control, working memory, and cognitive

flexibility and planning. They are necessary in facilitating goal-directed behavior with

principal elements of planning and organisation, initiation of activity, and self-regulation

(Anderson & Doyle, 2004). Among the most studied with reference to preterm children are

working memory and attention, which are abilities considered "of great importance for

successful school attainments" (Böhm, Smedler, & Forssberg, 2004, p. 1368). Take, for

example, Böhm, Smedler and Forssberg (2004) who assessed 182 preterm children

(birthweight < 1500g) and 125 controls aged 5.5 years on various executive functions. Full-

term controls surpassed the preterm children on all executive functions including impulse

control, working memory and mental speed after controlling for IQ (Böhm et al., 2004). A

systematic review by Mulder, Pitchford, Hagger, and Marlow (2009) confirmed that executive functions, in particular attention processes, are an area of significant weakness in preterm children. Employing a large representative sample, Anderson et al. (2011) examined the construct of executive attention among extremely preterm and extremely low birth weight children aged 8 years. The extremely preterm/extremely low birth weight group had significantly increased impairments in selected, sustained, shifting, and divided attention when compared to the full term/normal birth weight group (Anderson et al., 2011). These results confirmed an earlier study by Luciana, Lindeke, Georgieff, Mills, and Nelson (1999) who found that preterm 7-9 year olds performed 25% more memory errors on a spatial working memory task, as well as poorer planning, pattern recognition, and spatial memory abilities than children in the control group. Preterm children at 2 years corrected age have showed greater difficulty on EF tasks than controls (Pozzetti et al., 2014) and other studies have demonstrated significant impairment in cognitive flexibility (Aarnoudse-Moens, Weisglas-Kuperus, van Goudoever, & Oosterlaan, 2009). Preterm children have also demonstrated unique difficulties in working memory (Böhm et al., 2004; Luciana et al., 1999) and executive control of attention (Anderson & Doyle, 2004; Bayless & Stevenson, 2007) compared to term children.

The majority of the reviewed studies on EF abilities excluded children with severe neonatal complications, highlighting a clear gap in the literature, as it would seem plausible to suggest that preterm infants with a history of severe neonatal medical conditions would be at even higher risk of developing EF deficits than those without. Thus, the importance of understanding the relationship between prematurity, medical conditions in the neonatal period and executive functioning is apparent

Behavioural Development

Unsurprisingly, there is also evidence suggesting that behavioral and socio-emotional problems are elevated among children who were born preterm (Gray, O'Callaghan, & Poulsen, 2008). However, findings regarding the implications of preterm birth on these functional domains have been inconsistent, in part due to varying ages of assessment (Hornman, de Winter, Kerstjens, Bos, & Reijneveld, 2016). In a meta-analysis of nine studies, attention problems followed by internalising behaviors were pronounced in very preterm and very low birth weight children compared to term-born controls (Aarnoudse-Moens et al., 2009). Teacher and parent ratings of attention problems were 0.43 to 0.59 SD higher for the very preterm and very low birth weight children than the controls and teachers also reported significantly more internalising problems (i.e., anxiety or depression) for the preterm children, however no differences were found for externalising behaviors, contradicting previous research (i.e., Bhutta et al., 2002). In an earlier meta-analysis by Bhutta et al. (2002), majority of the studies included (69%) reported higher rates of externalising behaviors among the preterm group, however, Aarnoudse-Moens et al. (2009) report that the authors may have grouped attention problems with externalising behavior, therefore rendering the two sets of findings incomparable. Another earlier study reported significantly more internalising problems, poorer adaptive skills, and higher levels of attention problems, hyperactivity, somatic complaints, and atypical behaviors in the very preterm and extremely low birth weight groups compared to term-born controls (Anderson, Doyle, & Victorian Infant Collaborative Infant Group, 2003). Rates of attention deficit hyperactivity disorder are higher among preterm children as well as symptoms consistent with anxiety and depression, and general classroom functioning (Bhutta et al., 2002).

Academic Development

Preterm birth may also be a pre-cursor for poorer academic achievement and educational outcomes. More preterm children than term counterparts repeat year levels at school, attend special education classes, and exhibit deficits in mathematics, spelling, reading, writing, and language skills within the classroom (Anderson, 2014). Wong et al. (2014) observed 111 extremely preterm or extremely low birth weight children and 110 normal birth weight controls in their regular kindergarten classrooms. The extremely preterm/extremely low birth weight group required more undivided teacher attention and exhibited more "off-task" behavior than the controls (Wong et al., 2014). Other research has confirmed these findings describing severe deficits in key academic achievement areas, especially mathematics and spelling (Aarnoudse-Moens et al., 2009).

Overall, it is well known that preterm birth places the infant at higher risk of neurodevelopmental difficulties in all domains that will likely be experienced during childhood, and perhaps even continue into adolescence and adulthood. However, in light of some of the findings, longitudinal research is needed to facilitate a more comprehensive understanding of the extent to which preterm children will face such difficulties throughout their lifespan. As well as this, in order to create clinically meaningful outcomes, research is needed that focuses on "determining the profile of cognitive vulnerabilities…as this knowledge will influence the focus of surveillance programs and the content of intervention strategies" (Anderson, 2014, p. 5).

<center>**BPD and Neurodevelopmental Outcomes**</center>

BPD and Cognitive Development

Although preterm birth per se delays development in many areas of functioning, BPD has been posed as an additional risk factor for cognitive difficulties (Anderson & Doyle, 2006). The physical complications associated with lung immaturity can interact with brain

abnormalities often detected in preterm infants. For example, the preterm brain is vulnerable

to processes such as neuronal migration, synaptogenesis pruning, and cortical connectivity

being threatened as they are completed in the second and third trimester of pregnancy

(Raman et al., 2006). Hypoxia (inadequate oxygenation), a bi-product of BPD, can severely

disrupt these processes and therefore BPD may be a pathway leading to neurological

morbidity (Raman et al., 2006). In particular, studies have shown that preterm infants with

BPD have poorer developmental outcomes, lower IQ's, deficits in gross and fine motor skills,

language and speech delay, academic underachievement, impairments in attention and

memory, and executive functioning compared to preterm children without BPD (Anderson &

Doyle, 2006; Newman, Debastos, Batton, & Raz, 2011; Short et al., 2003). An earlier study

(aligned with the old BPD definition and treatment protocol) by Farel, Hooper, Teplin,

Henry, and Kraybill (1998) demonstrated that chronic lung disease (defined as oxygen

dependence at 30 weeks) may relate to neuropsychological dysfunction among very low birth

weight children at 7 years corrected age. Mean difference scores favoured the control group,

however they failed to reach statistical significance, albeit that 59% of the children with a

history of chronic lung disease fell at least one or two SD below normal expectations,

compared to 32% of children without a history of chronic lung disease. This gives rise to the

argument that the added complication of BPD would only further increase the disparity in

scores.

More recently, Gray, O'Callaghan, and Rogers (2004) found that preterm school age

children with a history of BPD demonstrated impaired psychoeducational performance,

specifically in the areas of language and reading abilities. Similarly, Short et al. (2003)

revealed that BPD and duration of oxygen supplements in addition to being born very low

birth weight have effects on cognitive development and academic performance above and

beyond that of being born very low birth weight in isolation, demonstrating the added

complexity of BPD during this critical time. However, there have also been inconsistent

results arising from some studies including Böhm and Katz-Salamon (2003) who investigated

cognitive development and visual-motor skills at 5.5 years of age with preterm and term

children and found no group differences on cognitive outcomes except preterm's with very

severe chronic lung disease (defined by frequent episodes of hypoxia). The authors suggest

that this is due to advances in neonatal care since their population was born in the 1990's.

There is a relatively small pool of research directly assessing specific cognitive

outcomes (i.e., attention, memory and learning, executive skills) among children with a

history of BPD (Anderson & Doyle, 2006). Anderson and Doyle are two prominent

researchers in the field and in 2006 conducted a review of neurodevelopmental outcomes of

preterm infants with BPD. Specifically, their paper noted attention, visual-motor, memory,

executive skills, and academic performance may be more compromised among children with

BPD than those without (Anderson & Doyle, 2006). However, it is evident that that long-

term outcome studies are needed to confirm previous findings (Anderson & Doyle, 2006).

Comparatively, Short et al. (2003) conducted a landmark study examining the effects of BPD

and very low birth weight on cognitive and academic achievement among children aged 8

years. The BPD group demonstrated weaknesses compared to very low birth weight children

on the full-scale IQ's, as well as verbal and performance skills, and they performed

significantly more poorly on a range of academic outcomes including reading,

comprehension, and calculation (Short et al., 2003). Children with BPD represented

significant differences on majority of educational outcomes; the highest group differences

were detected on ADHD diagnosis, special education, speech-language services,

occupational therapy, and physical therapy requirements (Short et al., 2003).

Given the emerging research on preterm children with a history of BPD and cognitive

development, it is reasonable to argue that BPD plays a role, whether it be big or small. As

researchers have noted, particularly Anderson and Doyle (2006), more investigation is needed to gauge the independent effect of BPD on specific cognitive functions, rather than general cognitive ability. In providing more insight about specific cognitive abilities, health professionals would be better positioned to predict those who are at risk for specific poorer outcomes and therefore appropriately target interventions. Additionally, generating more research on the potential environmental and socio-demographic factors that influence these outcomes in children with a history of BPD would shed light on the mechanisms that may lead to neuropsychological impairment at later ages.

BPD and Executive Functioning

Very few studies have examined the relationship between BPD and EF at any age. Taylor, Minich, Bangert, Filipek, and Hack,'s (2004) analysis of adolescents born with low birth weights, revealed specific impairments in visual-motor skills, spatial memory, and executive function, compared to term born controls. A particularly important finding was that a longer period of oxygen requirement was predictive of worse outcomes on many of the tests, including executive functions (such as, shifting, problem solving, and spatial working memory) at a significant level (Taylor et al., 2004). These findings lend strong support to the hypothesis that children with a diagnosis of BPD are at elevated risk of executive dysfunction. More recently, Gough et al. (2015) examined preterm adult survivors of BPD and their term peers, and found that significantly more BPD adults showed deficits in EF domains, namely problem solving, awareness of behavior, and organisation of their environment. This is the most recent (and perhaps first) published study highlighting the ongoing long-term effect of BPD on EF (Gough et al., 2015). However, the research by Taylor et al. (2004) and Gough et al. (2015) is limited as samples were from the pre-surfactant era (BPD was based on the old definition and treatment) , yet it is evident that even

with changes in the condition and its treatment, BPD still seems to have some effect on neurodevelopmental outcomes.

Potharst et al. (2013) investigated perinatal risk factors for dysfunctions in neurocognitive domains, including EF, in preterm and term-born children. Results suggested that BPD was the only significant medical predictor of performance on the neurocognitive domains, including working memory, explaining approximately 4-12% of the variance in scores (Potharst et al., 2013). BPD was also a risk factor for focused and sustained attention (Potharst et al., 2013), a finding also reported by Short et al. (2003) who discovered a significant difference between children with a history of BPD (8-years old) and both very low birth weight children and term children on reading recognition and comprehension. Short et al. (2003) also found that an increased number of very low birth weight children with a history of BPD received special education services, and demonstrated more difficulties in several cognitive domains, including perceptual and gross motor skills, reading, math's, spatial memory and attention, however the latter was only significant in comparison to term infants.

Despite the link between preterm birth and EF becoming clearer, the frequency, nature, and severity of executive dysfunction in preterm children with a history of BPD is for the most part, unknown. If preterm children demonstrate more deficits in cognitive domains such as memory, learning and attention, then it seems plausible to suggest, that executive dysfunction may be more present in these individuals too and even more so in preterm children who are survivors of BPD.

Conclusion

General Limitations

The research on preterm infants with BPD and how this might predict executive dysfunction at school is sparse. Conversely, the literature has focused more broadly on

preterm birth and cognitive development during childhood, adolescence, and even adulthood. Overall, findings indicate a positive relationship between younger and smaller infants and decreased IQ. Little is known about predictors other than gestational age and birth weight, such as BPD. Moreover, specific cognitive functions such as those of the executive type are less studied.

The reviewed body of the literature encompasses some important limitations that are critical when interpreting findings. As with most other fields of enquiry, some of these shortcomings are possible reasons to doubt the plausibility of results and the scope, within which they can be considered (beyond that individual study itself). Firstly, many studies, particularly those examining effects on adolescents and adults, are representative of the pre-surfactant era, which causes significant issues in terms of validity of the results and comparability to studies using populations that were born after this change (Anderson et al., 2011). Increased survival rates have seen a surge in the number of studies that are able to examine extremely preterm infants and those with the most severe forms of BPD (Newman et al., 2011). Survivors of BPD born today are likely to differ from those born before the 1990s (who are now in adulthood) because treatment was different, and therefore depending on the age of the sample, findings should take this into consideration (Gough et al., 2015). Additionally, the limited availability and use of standardised measures to assess specific cognitive functions (i.e., EF) should be taken into consideration. Some studies have used tests that may not have been specific to EF abilities, tapping into other cognitive functions or more 'general' abilities (Luu, Ment, Allan, Schneider, & Vohr. 2011; Mulder at el., 2009).

Conclusion and recommendations for future research

The purpose of this paper was to review the health effects and neurodevelopmental sequelae associated with preterm birth with a focus on BPD and EF. This is significant because preterm infants are at higher risk than term infants of physical complications, which

can lead to deficits in cognitive and behavioral development. While the mechanisms that predict these vulnerabilities remain less clear, BPD has been posed as an added predictor of cognitive impairment. It is clear from the research reviewed that being born preterm may alter EF, and that having the added burden of BPD during the neonatal period, may further exacerbate this deficit. However, presently there is little evidence to suggest the latter, although the research is promising. This field of inquiry is very important as Australia and the rest of the developed world face a continual increase in the numbers of preterm births, thus the need to understand the sequelae of this population and the effects of common neonatal conditions like BPD on later neurodevelopment is vital. The relationship between preterm birth and BPD, with cognitive development, warrants further investigation, specifically in terms of more critical cognitive functions, for instance EF. Future research could focus on the effect of BPD on EF at school age, which would provide more insight on the potential long-term effects of this condition.

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Research Report

Executive Functioning Outcomes in 7 Year Old Survivors of Bronchopulmonary Dysplasia

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Executive Functioning Outcomes in 7 Year Old Survivors of Bronchopulmonary Dysplasia

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Note: The student is aware of the 30 page limit for the *Health Psychology* journal, however in order to meet the university requirements of 5,000 words, the report is 34 pages. The paper will be reduced in size to meet journal guidelines prior to submission.

Abstract

Objectives. Preterm birth is associated with ongoing developmental issues. Severe medical conditions arising from preterm birth such as Bronchopulmonary Dysplasia (BPD) may further exacerbate these difficulties. The aim of this study was to explore the relationship between BPD and executive functioning outcomes in children aged 7 years with and without a history of BPD.

Methods. A cohort study was conducted utilising existing data from a previously published randomized controlled trial and follow-up study. In a large representative sample ($n = 473$) of children aged 7 years who were born preterm (< 33 weeks gestational age) t-tests of independence were computed to detect group differences between children with a history of BPD ($n = 93$) and children with no history of BPD ($n = 380$) on four executive functioning measures and multiple regression analyses to reveal amount of variance predicted by BPD on these scores.

Results. Children with a history of BPD scored more poorly on the majority of executive functioning outcomes, however when socio-demographic and perinatal variables were adjusted for these differences did not remain, with the exception of one subtest on the BRIEF ($p = .015$). BPD did not make any significant contributions to executive functioning outcomes after known predictors were controlled for.

Conclusions. These findings broaden the knowledge around BPD as a contributor to executive functioning skills at school age. Clinicians can use this information to appropriately target interventions. Future research should focus on non-medical factors that may contribute to cognitive deficits at school age.

Keywords: bronchopulmonary dysplasia, executive function, preterm birth, development, cognitive

Executive Functioning Outcomes in 7 Year Old Survivors of Bronchopulmonary Dysplasia

In Australia in 2014, 8.6% of babies (>26,000), were born preterm (<37 weeks gestation) (Australian Institute of Health and Welfare, 2016). Globally, approximately 15 million preterm births occur each year, resulting in 1 million infant deaths, most of which are confined to developing nations (World Health Organization, 2016). In Australia and other developed countries, advancements in medical practices and neonatal care have dramatically increased survival rates with even the most at risk babies going on to live relatively healthy lives. However, this means that the rate of preterm birth continues to increase, as does the prevalence of many neonatal diseases and conditions. Thus, the effect of preterm birth experiences and complications on the developmental trajectory of the infant is obviously important.

The beginning of life can be a challenging time for the preterm infant, commonly characterised by long periods of hospitalisation, exposure to invasive procedures, and separation from the primary caregiver. The health of those born earlier and/or smaller is specifically threatened with research indicating that preterm infants are at increased risk of developing severe medical conditions, neurodevelopmental disorders (i.e., Cerebral Palsy), and experiencing ongoing cognitive, behavioural, and academic problems (Anderson, 2014; Aarnoudse-Moens, Weisglas-Kuperus, van Goudoever, & Oosterlaan, 2009). The literature strongly suggests that cognitive delay, commonly measured as an intelligent quotient (IQ), is lower among preterm children compared to their term peers revealing a dose-response relationship between decreased gestational age and lower IQ (Anderson, 2014; Bhutta, Cleves, Casey, Cradock, & Anand, 2002; Kerr-Wilson, Mackay, Smith, & Pell, 2011). Reports have indicated that preterm children have IQ scores up to 0.8 standard deviations (SD) lower than their term-born counterparts (Anderson, 2014; Kerr-Wilson et al., 2011).

This has remained consistent over time even in the face of improved neonatal care

(Anderson, 2014).

Preterm birth disrupts normal organ development that usually occurs in the third

trimester of pregnancy with the lungs and brain being particularly affected. Consequently,

preterm infants are at greater risk of developing respiratory disease and in being exposed to

long periods of high levels of oxygen ventilation may develop a severe condition called

Bronchopulmonary Dysplasia (BPD). Given the complex and changing nature of BPD over

time, it is a somewhat poorly understood condition. Nonetheless, BPD may be related to

cognitive impairment as it can interfere with other bodily functions, and in particular, brain

processes (i.e., neuronal migration). Hypoxia (inadequate oxygenation) is a bi-product of

BPD that can inhibit important brain functioning and cause abnormalities (Raman, Georgieff,

& Rao, 2006). Therefore, BPD may be a pathway leading to neurological morbidity.

BPD is more common among those who are born extremely preterm or born with

extremely low birth weight and it has been posed as an additional risk factor for cognitive

delay above and beyond the preterm infant who doesn't develop BPD (Anderson & Doyle,

2006). The "pre-surfactant" BPD (present in larger preterm infants with high oxygen needs),

referring to BPD as it was originally characterised by severe scarring, inflammation, and

fibrosis often resulting in death of the infant, is somewhat redundant nowadays (since the

introduction of exogenous surfactant and various other improved medical techniques). Older

studies (i.e., O'Shea et al., 1996; Katz-Salamon, Gerner, Jonsson, & Lagercrantz, 2000)

found associations between BPD and poorer cognitive outcomes, however the old

classification of BPD means that the findings are deemed no longer relevant as they do not

apply to current children with a history of BPD. More recent studies, using samples based on

the new classification of BPD (a disorder of the lungs as opposed to direct injury to the lungs

and typically present in very premature infants with modest oxygen needs) have produced

mixed results with some revealing clear significant deficits (Gray, O'Callaghan, & Rogers, 2004; Short et al., 2003), others demonstrating impairments only among the most severe BPD participants (Böhm & Katz-Salamon, 2003), and others reporting little or no differences when compared to preterm children with no BPD (Gray, O'Callaghan, & Poulsen, 2008; Newman, DeBastos, Batton, & Raz, 2011). Methodological limitations (such as the use of unstandardised tests) and differences across studies (i.e., exclusion of neonates with disabilities or only including mild cases of BPD) are likely to have contributed to the variability in findings.

With most of the interest thus far being on general cognitive outcomes, research is sparse on specific abilities in relation to children who were born preterm. Executive functioning (EF) refers to a set of higher order cognitive processes responsible for planning, organisation, working memory, time management, and flexible thinking. These skills are vital for the execution of everyday behaviours; for example, strong working memory skills allow a child to store information and use it to complete certain tasks. Executive dysfunction is a weakness in one or more areas of EF (i.e., neurocognitive and/or behavioural) and such disruptions can implicate social functioning, school performance, and behavioural and emotional stability. Previous literature indicates that executive dysfunction is more prevalent in preterm children, particularly in the areas of executive attention (Anderson & Doyle, 2004; Anderson et al., 2011; Bayless & Stevenson, 2007), working memory (Böhm, Smedler, & Forssberg, 2004; Luciana, Lindeke, Georgieff, Mills, & Nelson, 1999), and even cognitive flexibility (Aarnoudse-Moens et al., 2009) compared to term controls. However, again, findings have been mixed and the vast differences in age at assessment, and measures used has been identified as an issue (Aarnoudse-Moens at el., 2009).

Even less research has focused on specific abilities in relation to children with a history of BPD, however they may be at elevated risk of executive dysfunction. Potharst et al.

(2013) reported BPD as a significant medical risk factor for working memory as well as focused and sustained attention. Short et al. (2003) reported that very low birth weight children with a history of BPD were more likely to receive special education services and demonstrated more difficulty in several cognitive domains including spatial memory and attention, however interestingly this difference was only apparent in comparison to term controls, not very low birth weight children without BPD. Longer periods on oxygen treatment (mechanically administered oxygen is a common treatment for BPD) has been found to be a predictor of poorer EF outcomes such as shifting, problem solving and spatial working memory, among preterm adolescents (Taylor, Minich, Bangert, Filipek, & Hack, 2004). More recently, Gough et al. (2015) reported significant deficits on tests of problem solving, awareness of behaviour and organisation of their environment among young adult survivors of BPD compared to controls. This study highlighted the potential long-term effect of BPD. Little else has been published investigating BPD as a predictor of EF abilities and overall, evidence of predictive factors of specific abilities at school age is sparse.

To date, no studies have explored whether impairments in EF exist in childhood survivors of the new BPD compared to those born preterm without BPD. Notably, the majority of the studies mentioned above utilised samples from the pre surfactant era, and so findings need to be interpreted with caution. Using data from a previously published large randomised controlled trial at initial study (Makrides et al., 2009) and at follow up (Collins et al., 2015), a cohort study was conducted to investigate the relationship between BPD in preterm infants and EF at school age (7 years corrected age).The aim of this study was to undertake such an evaluation in children who were born preterm and developed BPD comparing them to children who were born preterm who didn't develop BPD.

The present study had two hypotheses; (1) that the children with a history of BPD would have greater impairment in EF (across all tests) than the children without a history of

BPD and (2) that BPD would be a stronger predictor for variances in EF scores than other sociodemographic (i.e., maternal education) and perinatal (i.e., gestational age) variables.

Method

This is a cohort study of infants who participated in a randomised controlled trial and were followed up at 7-years corrected age.

Initial Trial

The original trial, has been previously published (Makrides et al., 2009). The randomised controlled trial consisted of preterm infants (<33 weeks gestation) recruited from five Australian Tertiary Hospitals who were randomised to receive "high Docosahexaenoic Acid" (intervention) or "standard Docosahexaenoic Acid" (control) to determine the effect of Docosahexaenoic Acid on developmental outcomes at 18 months and 7 years. Infants who had a major congenital or chromosomal abnormality, were from a multiple birth in which not all births were considered eligible, were enrolled in other trials of fatty acid supplementation, or if fish oil was contraindicated in the lactating mother were excluded from the study. Baseline demographic and clinical characteristics were gathered including maternal and paternal data (i.e., education level, smoker status), and health outcomes including the onset of neonatal complications and diseases (i.e., BPD) at trial entry. Labor and birth details (i.e., birth weight) were obtained from medical records. BPD was defined as infants who required oxygen treatment at 36 weeks corrected age. Analyses revealed negligible effects of high-dose Docosahexaenoic Acid intervention and development at 18 months or 7 years corrected age (Collins et al., 2015; Makrides et al., 2009) so the groups will be analysed as a cohort here.

Follow-up at 7 Years Corrected Age

The results of the intervention at 7 years corrected age have been previously published (Collins et al., 2015). The parent/guardian of each infant enrolled in the original

trial was approached to take part in the 7-year follow-up study. Of the original 657

participants, 626 were eligible to participate in the follow-up (i.e., they had not withdrawn or

died) and 604 consented to participate at 7 years. Each participant was administered a

psychological test, conducted within 3 months of the 7-year corrected age date. Aside from

general intelligence, outcomes that were assessed and are related to the current study

included attention, memory and learning, EF, and behavior.

Participants

Only children who participated in the 7 year follow-up study were included in the

present study. Only the first born of families with multiple children were included in the

study. This decision was based on a chi-square test of independence which indicated no

significant association between birth order and BPD χ^2 (3, $n = 653$) = 4.32, $p = .229$. Missing

data were excluded pairwise from the analysis. Note that several children who were assessed

at follow-up did not complete every subtest; therefore, several cases had missing data for

various subtests. A case was only deleted if they had no data for *all* of the outcome variables,

that is, children who had data for one subtest but not another, remained in the data set. The

number of missing data for each subtest is presented in each table. Infants were divided into

two categories; those who were diagnosed with BPD ($n = 93$) and the control group who did

not receive a diagnosis of BPD ($n = 380$). This resulted in a total sample of 473 infants.

Measures

Executive Functioning

The Behaviour Rating Inventory of Executive Function (BRIEF [Gioia, Isquith, Guy,

& Kenworthy, 2000]) is a standardised 86-item parent report measure. The tool yields eight

clinical scales relating to domains of EF. These include: inhibit, shift, emotional control,

initiate, working memory, plan/organise, organisation of materials, and monitor. The former

three subdomains result in an additional composite labelled the Behavioural Regulation Index

(BRI) and the latter five, the Metacognition Index (MI). The BRI and MI are combined to achieve an overall Global Executive Composite (GEC) score.

The Test of Everyday Attention for Children (TEA-Ch [Manly, Robertson, Anderson, & Nimmo-Smith, 1999]) is a standardised test comprising of nine subtests. This study used four of the nine subtests; (1) Sky Search (Number Correct), (2) Score (Number Correct), (3) Creature Counting (Number Correct), and (4) Sky Search Dual Task (Composite Score), which measured selective attention, sustained attention, shifting attention/attentional control, and divided attention. The TEA-Ch provides scores that allow for comparison across each attentional domain.

The Rey Complex Figure Test (Meyers & Meyers, 1995) is a standardised test that measures visuospatial ability and visual memory. Only the Copy Raw condition was analysed in this study as it has been shown to specifically highlight executive dysfunction (Weber, Riccio, & Cohen, 2013).

The Fruit Stroop test (Archibald & Kerns, 1999) is a non-reading Stroop task and is a modified version of the Fruit Distraction Task (Santostefano, 1988). The test consists of four pages of stimuli consisting of common fruits and colours. This test predominantly measures inhibition as children are presented with incongruent stimuli. The Interference score was used in the current study.

Statistical Analyses

Data were analysed using SPSS Version 24 (SPSS Inc., Chicago, IL). Differences between groups for continuous variables were analysed by independent samples *t*-tests, whereas dichotomous variables were compared using *chi*-squared analysis. Group differences in EF outcomes were re-analysed with adjustment for covariates by computing general linear regression. Pearson's correlation coefficients were calculated for the relationship between various socio-demographic and perinatal variables to exclude issues of collinearity. Effect

sizes in terms of Cohen's *d* are provided. Cohen's guidelines were followed to indicate the

strength of effect sizes, with 0.20, 0.50, and 0.80 referring to small, medium, and large effect

sizes, respectively (Cohen, 1992).

Hierarchical multiple regression analyses were conducted to test the impact of socio-

demographic and perinatal variables on the EF outcomes. The socio-demographic and

perinatal predictor variables gestational age, surfactant required, maternal smoker status

during pregnancy, maternal highest level of education, and maternal age were entered into the

first block, and BPD was entered into the second (and last) block to examine its impact over

and above background data. For all analyses, the threshold for significance was $p < .05$.

Results

Multiple births could not be assumed to be independent therefore second and third

born infants were removed from the data set ($n = 115$). All children who were lost to follow-

up at 7 years' corrected age or did not have adequate data for analysis were also removed

from the data set ($n = 54$). Analyses were conducted on EF outcomes of 473 children at 7

years' corrected age who were born preterm.

Descriptive Statistics

The characteristics (including perinatal factors) of the total sample and the BPD and

non-BPD groups are presented in Table 1. In the current cohort study, 93 children born

preterm with a history of BPD and 380 children born preterm with no history of BPD were

included. Gender and race did not differ significantly between the groups nor did maternal

factors such as maternal age at trial entry, maternal smoking status during pregnancy, and

maternal highest level of completed education.

The BPD group had significantly lower birth weight and gestational age than the non-

BPD group ($p = < .001$). Similarly, those in the BPD group were more likely to be given

surfactant in the neonatal period ($p = < .001$). BPD participants had significantly lower full

scale IQ scores than non-BPD participants ($p = < .001$).

Table 1

Perinatal and Demographic Characteristics of the BPD and Non-BPD Groups

Characteristic	Total sample (*n* = 473)	BPD (*n* = 93)	Non-BPD (*n* = 380)
Male gender	251 (53.1%)	51 (54.8%)	200 (52.6%)
Caucasian race	435 (92.0%)	85 (95.7%)	346 (91.1%)
Gestational age***	29.18	26.95	29.72
Mean Birth Weight (g)***	1327.78	978.42	1413.29
High-dose DHA	240 (50.7%)	41 (44.1%)	199 (52.4%)
Maternal age at trial entry	30.02	29.29	30.20
Maternal education (Year 12 or higher)[a]	336 (71.2%)	70 (75.3%)	266 (70.2%)
Maternal smoker (during pregnancy)	121 (25.6%)	20 (21.5%)	101 (26.6%)
Antenatal steroids	416 (87.9%)	85 (91.4%)	331 (87.1%)
Surfactant***	215 (45.5%)	75 (80.6%)	140 (36.8%)
Full Scale IQ***[a]	98.78	95.06	99.69

Note. Continuous variables are reported as a mean and categorical variables are reported in frequencies and percentages. BPD = Bronchopulmonary Dysplasia; DHA = Docosahexaenoic Acid; IQ = Intelligent Quotient; Surfactant = A complex mixture of molecules to support air breathing.

[a] *n* = 1 missing data.

* P <.05. ** p <.01. *** p <.001.

Executive functioning

Table 2 lists the means, unadjusted, and adjusted mean differences, p-values, and effect sizes for all EF tests within the BPD and non-BPD group.

Unadjusted mean scores were significantly different for the following subtests; Sky Search (p = <.001), Creature Counting (p = .008), Sky Search Dual Task (p = .018), Copy Raw (p = .001), Metacognition Index (p = .021), Behavioural Regulation Index (p = .015), and Global Executive Composite (p = .016). The remaining two subtests, Score, and the Interference score, did not reach significance. The BPD group had mean scores on the TEA-Ch that were indicative of poorer attention skills (<8), whilst the non-BPD group scores did not. Both the BPD and non-BPD groups scored below the clinical cutoff (<64) for executive dysfunction on the Global Executive Composite score. Additionally, neither group demonstrated negative scores on the Interference score from the Fruit Stroop test.

General linear regression models accounted for five confounding variables; (1) gestational age, (2) surfactant required, (3) maternal age, (4) maternal smoker status during pregnancy, and (5) maternal highest education status. After adjustment, the significant differences did not remain. The Behavioural Regulation Index from the BRIEF was the only score that children with a history of BPD scored significantly worse than children without BPD after adjusting for covariates (p = .029). Several subtests came close to reaching significance, for example the Copy Raw (p = .053) and Creature Counting (p = .060) conditions.

Small effect sizes were found for all of the EF outcomes (Cohen, 1992). The strongest small effect was found for the Behavioural Recognition Index score, which also retained significance (d = 0.28, p = .029). Of the remaining nine subtests, six demonstrated small negative effect sizes ranging from -0.11 to -0.28, and three produced small positive effect sizes ranging from 0.08 to 0.28.

Given that results did not maintain statistical significance after adjusting for covariates, the first hypothesis was rejected.

Table 2

Group Means and SD's of the BPD and non-BPD Groups on Executive Functioning Outcomes

Outcome	BPD mean (SD) (*n* = 93)	Non-BPD mean (*n* = 380)	Unadjusted mean difference (95% CI)	Unadjusted *p* value	Adjusted mean difference (95% CI)	Adjusted *p* value	Cohen's *d*
TEA-Ch							
Sky Search [a]	7.79 (3.61)	9.35 (3.60)	-1.56 (-2.40, -0.73)	.000***	-.82 (-1.76, 0.12)	.087	-0.22
Score [b]	7.49 (3.74)	8.08 (3.54)	-.60 (-1.44, 0.25)	.166	-.47 (-1.41, 0.48)	.333	-0.13
Creature Counting [c]	7.07 (3.15)	8.26 (3.50)	-1.19 (-2.07, -0.32)	.008**	-.97 (-1.99, 0.04)	.060	-0.28
Sky Search Dual Task [d]	3.70 (2.80)	4.50 (2.75)	-.80 (-1.47, -0.14)	.018*	-.352 (-1.10, 0.39)	.354	-0.12
Rey Complex Figure							
Copy Raw[e]	14.67 (7.14)	17.36 (7.05)	-2.69 (-4.32, -1.06)	.001***	-1.78 (-3.60, 0.03)	.053	-0.24
BRIEF							
MI	57.10 (12.87)	53.83 (11.99)	3.27 (0.49, 6.05)	.021*	1.37 (-1.74, 4.49)	.388	0.11
BRI	55.82 (13.97)	51.94 (11.58)	3.88 (0.76, 6.99)	.015*	3.47 (0.36, 6.59)	.029*	0.28
GEC	57.32 (13.47)	53.59 (11.89)	3.73 (0.69, 6.76)	.016*	2.29 (-0.84, 5.43)	.151	0.18

Outcome	BPD mean (SD) (*n* = 93)	Non-BPD mean (*n* = 380)	Unadjusted mean difference (95% CI)	Unadjusted *p* value	Adjusted mean difference (95% CI)	Adjusted *p* value	Cohen's *d*
Fruit Stroop							
Interference	1.09 (6.07)	0.95 (5.64)	0.13 (-1.21, 1.47)	.847	0.46 (-1.07, 1.99)	.554	0.08

Note. SD = standard deviation; BPD = Bronchopulmonary Dysplasia; CI = confidence interval; Cohen's *d* = measure of effect size; TEA-Ch = Test of Everyday Attention for Children; BRIEF = The Behaviour Rating Inventory of Executive Function; MI = Metacognition Index; BRI = Behavioural Regulation Index; GEC = Global Executive Composite;

[a] *n* = 10 missing data, [b] *n* = 15 missing data, [c] *n* = 73 missing data, [d] *n* = 33 missing data, [e] *n* = 13 missing data, [f] *n* = 4 missing data, [g] *n* = 16 missing data, [h] *n* = 5 missing data, [I] *n* = 4 missing data, [j] *n* = 4 missing data, [k] *n* = 5 missing data, [l] *n* = 5 missing data, [m] *n* = 8 missing data, [n] *n* = 5 missing data, [o] *n* = 5 missing data, [p] *n* = 6 missing data, [q] *n* = 5 missing data, [r] *n* = 6 missing data.

* P <.05. ** p <.01. *** p <.001.

To address the second hypothesis, that BPD would be a stronger predictor of variance in EF scores than other socio-demographic and perinatal variables, hierarchical multiple regression analyses were computed. Prior to conducting the analyses, the relevant assumptions of this statistical analysis were tested. The sample size of $N = 473$ well exceeds the recommendations regarding sample size per independent variable as per Tabachnick & Fidell (2001). Collinearity was tested using Pearson's r, revealing a high correlation between gestational age and birth weight ($r = 0.81$, $p = < .001$). Therefore, birth weight was excluded from the regression model. The assumption of singularity was thus met and no issues of multicollinearity were identified as assessed by tolerance and variance inflation factors (Pallant, 2013). Residual and scatter plots indicated the assumptions of normality, linearity, and homoscedasticity were met.

A two stage hierarchical multiple regression was used to assess the ability of oxygen required at 36 weeks gestation (BPD) to predict scores on various measures of EF, after controlling for five independent variables, (1) gestational age, (2) surfactant required, (3) maternal age, (4) maternal smoker status during pregnancy, and (5) maternal highest education status.

Table 3 illustrates variable entry at the two steps; beta values and squared semi-partial correlation between predictor variables and EF subtest scores. Demographic variables explained 4.1% ($p = < .01$) of the variance in the Behavioural Recognition Index score at Step 1. The addition of BPD group in Step 2 did not improve the model, explaining an additional 0.7% of the variance in these scores. The best predictor in Step 2 of this model was maternal age ($\beta = .165$, $p = < .001$). Demographic variables explained 5.4% of the variance in the Metacognition Index score at Step 1 and the addition of BPD group in Step 2 did not explain any additional variance. The most significant predictor in Step 2 of the Metacognition Index score was gestational age ($\beta = -.180$, $p = < .001$). Similarly, demographic variables

explained 5.3% of the variance in the Global Executive Composite score at Step 1, with BPD

adding .2% in step 2, an insignificant change. The most significant predictor for the Global

Executive Composite scores in Step 2 was maternal age (β = -.161, p = < .001).

Table 4 illustrates hierarchical multiple regression analyses with the remaining EF

tests. Demographic variables explained 6.1% (p = < .001) of the variance in Sky Search

scores and when BPD group was entered in Step 2 it accounted for an additional .5%

variance, which is insignificant. The largest predictor of scores on this test was gestational

age (β = .196, p = < .001). Demographic variables explained 4.6% (p = < .001) of the

variance in the Sky Search Dual Task scores and when BPD group was entered in Step 2 it

accounted for an additional .1% of the variance, an insignificant change. The most significant

predictors were gestational age (β = .180, p = < .01) and maternal smoking status (β = .099, p

= < .05). Demographic variables explained 5.9% (p = < .001) of the variance in the Copy

Raw scores whilst BPD group when added in Step 2 only added .6% of a variance, which is

insignificant. The largest predictor was gestational age (β = .194, p = < .001). The Score,

Creature Counting, and Interference subtests revealed no significant variances and no

changes when BPD was added in Step 2. However, it is worth noting that maternal age was

the largest predictor for the Score subtest (β = .074). Surfactant required was the largest

predictor for on the Interference score (β = .044) and BPD was the largest predictor on

Creature Counting scores (β = .110), however the change in variance was insignificant.

Given that BPD did not predict any significant amount of variance over and above the

other socio-demographic and perinatal variances, the second hypothesis was rejected.

Table 3

Regression Model of Socio-demographic and Perinatal Variables Predictive of Composite

Measures of Executive Functioning

	Step 1	Step 2
Variable/Predictor	β	β
BRIEF: BRI		
Gestational age, weeks	-.103	-.066
Surfactant required	.022	.037
Maternal smoker	.023	.022
Maternal education	-.025	-.028
Maternal age	-.171***	-.165***
BPD		-.098
Change in R^2	.041**	.007
BRIEF: MI		
Gestational age, weeks	-.188***	-.180**
Surfactant required	.023	.027
Maternal smoker	.016	.015
Maternal education	-.004	-.004
Maternal age	-.143**	-.142**
BPD		-.022
Change in R^2	.054***	.000
BRIEF: GEC		
Gestational age, weeks	-.163**	-.143*
Surfactant required	.022	.031
Maternal smoker	.019	.018

	Step 1	Step 2
Variable/Predictor	β	β
Maternal education	-.013	-.014
Maternal age	-.164***	-.161***
BPD		-.054
Change in R^2	.053***	.002

Note. BPD = Bronchopulmonary Dysplasia; BRIEF = Behaviour Rating Inventory of

Executive Functioning; BRI = Behavioural Recognition Index; MI = Metacognition Index;

GEC = Global Executive Composite; β = standardised regression coefficients.

*p <.05; **p <.01; *** p <.001

Table 4

Regression Model of Socio-demographic and Perinatal Variables Predictive of Executive

Functioning Outcomes

	Step 1	Step 2
Variable/Predictor	β	β
TEA-Ch: Sky Search		
Gestational age, weeks	.228***	.196***
Surfactant required	.007	-.006
Maternal smoker	.091	.093
Maternal education	.037	.039
Maternal age	-.005	-.010
BPD		.085
Change in *R²*	.061***	.005
TEA-Ch: Score		
Gestational age, weeks	.056	.040
Surfactant required	.017	.010
Maternal smoker	.044	.045
Maternal education	.047	.048
Maternal age	.077	.074
BPD		.040
Change in *R²*	.019	.001
TEA-Ch: Creature Counting		
Gestational age, weeks	.040	-.002
Surfactant required	.082	.064
Maternal smoker	.003	.005

	Step 1	Step 2
Variable/Predictor	β	β
Maternal education	.049	.052
Maternal age	.048	.042
BPD		.110
Change in R^2	.018	.009

TEA-Ch: Sky Search Dual Task

	Step 1	Step 2
Gestational age, weeks	.197***	.180**
Surfactant required	-.020	-.027
Maternal smoker	.101*	.102*
Maternal education	.046	.047
Maternal age	-.007	-.010
BPD		.044
Change in R^2	.046***	.001

Rey Complex Figure: Copy Raw

	Step 1	Step 2
Gestational age, weeks	.228***	.194***
Surfactant required	-.003	-.018
Maternal smoker	.055	.057
Maternal education	.081	.083
Maternal age	-.082	-.087
BPD		.090
Change in R^2	.059***	.006

Fruit Stroop: Interference Score

	Step 1	Step 2
Gestational age, weeks	.015	.029
Surfactant required	.038	.044

Variable/Predictor	Step 1 β	Step 2 β
Maternal smoker	.025	.025
Maternal education	.028	.027
Maternal age	-.009	-.007
BPD		-.036
Change in R^2	.004	.001

Note. TEA-Ch = Test of Everyday Attention for Children; BPD = Bronchopulmonary

Dysplasia; β = standardised regression coefficients.

*p < .05. **p < .01. *** p < .001

Discussion

This study aimed to explore the relationship between BPD among preterm infants (born <33 weeks gestational age) and EF outcomes at 7 years corrected age. In accordance with the reviewed literature, BPD was defined as requiring oxygen treatment at 36 weeks gestational age. A comprehensive range of EF abilities were explored via the outcome measures. Results indicated that on all but one subtest of the BRIEF (Behavioural Recognition Index), infants with a history of BPD did not perform significantly worse on EF outcomes after controlling for confounding variables. In addition to this, BPD was not a stronger predictor of EF outcomes than other socio-demographic and perinatal factors. Nonetheless, in light of the stable, if not rising number of preterm births in Australia, and survival of infants who develop severe medical conditions in the neonatal period (such as BPD), the findings of the current study have important implications for ongoing research, and the care of infants and children with a history of BPD.

To date, it has been proposed that children with a history of BPD may be more susceptible to ongoing cognitive deficits than their preterm counterparts who do not have a history of BPD (Anderson & Doyle, 2006). EF skillsets in particular are important for everyday functioning, however the impact that BPD has, if any, on EF at school age has been largely neglected throughout the literature. Thus, our understanding of the frequency, nature, and severity of executive dysfunction in this population is limited. This study examined a large cohort of children with a history of BPD and those with no history of BPD as controls. It was found that children with a history of BPD demonstrated lower scores on the majority of EF tests with small effect sizes. However, almost all of these differences did not remain significant when adjusting for socio-demographic variables. Further analyses revealed that BPD was not necessarily a stronger predictor than other socio-demographic and perinatal variables on EF outcomes.

The results of the current study lent little evidence to support the hypotheses. Furthermore, it is difficult to compare the current findings as this is the first study of its kind, however, when considering the existing literature, both contrasts and consistencies can be drawn. The current findings revealed no group differences on eight out of nine EF tests following adjustment, similar to the results of a recent systematic review (Newman et al., 2011). Therefore, it can be said that overall, children with BPD did not appear to perform significantly worse on EF outcomes than their non-BPD peers. This is somewhat consistent with Gough et al. (2015) who also failed to find differences between BPD and non-BPD adolescents on the Global Executive Composite score, and the Metacognition Index (from the BRIEF) when adjusting for SES and educational achievement. Interestingly, Gough et al. (2015) did not find a significant difference on the Behavioural Regulation Index (four out of five scales), whereas this was the only subtest that reached significance in the current study. Scores on the Behavioural Regulation Index were only evident when the BPD group was compared to the term born group. Note that this study sample consisted of adolescents, not school aged children. Anderson and Doyle's (2004) findings showed that extremely low birth weight/very preterm (BPD was not a focus) infants scored lower than term controls, making more errors on EF measures such as the Rey Complex Figure, yet only one scale on the Behavioural Recognition Index (shift) contained elevated scores for the BPD group. Notably, none of the scores constituted executive dysfunction, still being in the average range. Short et al. (2003) also only found significant differences between the BPD group and the term controls on measures of spatial memory and attention, however not when compared to very low birth weight infants alone. This lends evidence to the suggestion that neonatal illness may not be a predetermining factor for EF deficits over and above preterm birth alone (Taylor et al., 2004). In the present study, on the majority of the EF outcomes, neither group

scored above clinical cut-off ranges, which is inconsistent with some of the previous findings (i.e., Gough et al., 2015).

While our results demonstrate that BPD does play a small role in predicting EF scores, it accounts for a very small amount of variance, with other non-medical factors predicting more variance. Interestingly, Aarnoudse-Moens et al. (2009) reported that the degree of neonatal illness (including chronic lung disease defined by the same criteria as BPD) did not contribute to EF difficulties. Instead, maternal education and gestational age were predictive of executive dysfunction (Aarnoudse-Moens et al., 2009), similar to other previous studies (i.e., Gough et al., 2015) as well as the present study in that maternal age, gestational age, and maternal smoking status were larger predictors of scores on EF outcomes than BPD. Other studies have revealed that higher levels of neonatal illness and longer periods on oxygen treatment are associated with poorer aspects of EF such as working memory, and focused and sustained attention (Luciana et al., 1999; Potharst et al., 2013; Taylor et al., 2004). Taylor et al. (2004) found that longer periods on oxygen treatment (used as a marker for chronic lung disease) predicted poorer outcomes after controlling for birth weight. Although it cannot be conclusively stated that BPD is not a significant predictor of EF outcomes, the results of the current study extend support for the indication that factors other than neonatal illness contribute more to EF difficulties. As Newman et al. (2011) state, "other biological risk factors that tend to precede, follow, or co-occur with, BPD, also may serve to compromise developmental outcome" (p. 667). Many have noted the complex pattern of development after birth, giving rise to the notion that gestational age, gender, type and severity of brain injury, and environmental circumstances (i.e., SES), are all likely interplay and influence all spectrums of a child's development. This combined with the new face of BPD, may be a sufficient reason for it to not significantly contribute to specific

cognitive deficits like executive dysfunction (Mulder, Pitchford, Hagger, & Marlow, 2009; Newman at el., 2011; Raman, Georgieff, & Rao, 2006).

The current findings have considerable weight in regard to informing clinical practice. They highlight the independent role of BPD as a predictor of EF outcomes, which no other previous study has done to date. However, the significance level of the results has relatively limited clinical relevance. The effect sizes produced in this study are far more meaningful and while only weak, they are deemed an important finding given that preterm birth and associated physical illnesses such as BPD are prevalent in modern society. Given the size of the current study's sample and the considerable efforts made to adjust for potentially confounding variables, it is believed that the statistical power of the study was sufficient to detect small effect sizes and was therefore not undermined in any way.

In terms of clinical implications, the insight that this paper provides is important for thinking about the interventions and approaches required for children at school age with a history of BPD, but also the care of the infant after initial discharge as it is likely that environmental and social factors that come about during this period may have a significant effect on the future development of the infant. How these infants are cared for at home (perhaps while still receiving support for BPD), the availability of social supports etc. may be extremely important in determining appropriate methods of intervention and care moving forward. Moreover, this study also highlights the need for careful follow up of children who have a history of BPD, specifically in light of weak effect sizes. While the developmental challenges may not be visible at a clinical level, recognition of their difficulties in comparison to peers without a history of BPD will aid in enhancing their competence and ability to flourish at the same rate as their peers.

Strengths and Limitations of the Present Study

This study, to the best of the author's knowledge, is the first to directly examine the relationship between BPD and EF outcomes at school age, with the particular sample characteristics it employed. Therefore, it makes a significant contribution the field of health psychology and has important future research and practical implications, some of which were discussed above.

A considerable strength of this study is the sample size ($n = 473$), particularly given that no previous study in this area has employed one of this magnitude. This increases the generalisability of the findings, capturing a more realistic picture of the population. Additionally, the original randomised controlled trial from which the data was taken from was extensively representative of Australian mothers (i.e., mothers were recruited from a number of hospitals at different locations). With modern medical advancements, survivors of BPD born today differ from those born in the pre-surfactant era, which is where majority of studies to date (i.e., Gough et al., 2015) have used samples. This study included participants from the modern BPD population. Furthermore, this study controlled for a number of confounding variables. Previous studies have failed to document methods of adjustment or provide an explanation why certain variables were accounted for in analyses. While there is no universal approach, it is believed that this comprehensive inclusion of controlling for confounding variables adds weight to the reliability and validity of our findings and brings in to question the findings of studies that have not done so.

Our study removed second and third born infants completely from the data set, which may limit generalisability as these infants, may be inherently different to singletons and first-born infants (Hibbs et al., 2010). Ideally, non-independence of the data would be accounted for in future studies via appropriate statistical analyses. Our cohort did not differentiate between mild, moderate, and severe forms of BPD and given the increase in survival rates of

those with severe forms of the disease, this may have further clarified findings. This study

also did not take into account other medical conditions or diseases (i.e., sepsis, severe

retinopathy) that may have been a result of or co-occurred with BPD and therefore

contributed to compromising development.

Conclusions

Overall, the results of this study show that preterm children with a history of BPD do

not necessarily exhibit more impairment in EF than preterm children without a history of

BPD and that BPD is not a significant predictor of EF outcomes over other established

predictors. Nonetheless, this study highlights the large number of other factors that may be

contributing to specific cognitive deficits in preterm children with a history of BPD that are

most likely present simultaneously when BPD emerges in the neonatal period or take affect

later in early childhood. Studies need to focus on non-medical predictors of later cognitive

deficits such as environmental, social, and family factors to best determine where to intervene

with these at risk infants to prevent ongoing difficulties at school age. These areas represent a

more productive research focus at this point in light of the current study's findings.

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Appendix

Health Psychology Author Guidelines

Prior to submission, please carefully read and follow the submission guidelines detailed

below. Manuscripts that do not conform to the submission guidelines may be returned

without review.

Submission

The main emphasis of *Health Psychology*® is on original research in health psychology.

Systematic reviews (including meta-analyses) and narrative reviews are also considered for

publication. Editorials, commentaries, scientific statements, and tutorials are by invitation

only. Submissions are welcomed from authors in psychology and other health-related

disciplines.

Submit manuscripts electronically (.rtf, PDF, or .doc) to

Kenneth E. Freedland, PhD, Editor-in-Chief

Professor of Psychiatry and Psychology

Washington University School of Medicine

St. Louis, Missouri, USA

Email

Keep a copy of the manuscript to guard against loss. Do not submit manuscripts via mail or

email.

In recognition of the reality that institutional spam filters may capture files from the APA and

the Journals Back Office, please take the following steps to facilitate communication with our

editorial office:

- Provide an alternative email address which we can use to contact you in the event of

    technical difficulties with email communication using your primary address;

- Add "apa.org" to your list of "safe" addresses and consider asking your IT administrators to add it to their "white list;" and

- Contact Lindsay MacMurray if you do not receive confirmation of your submission within three business days or an editorial decision letter within three months.

General correspondence may be directed to the Editor's Office.

Information About Submissions

The page limit for research manuscripts, reviews, and meta-analyses is 30 pages. The page limit is inclusive of **all** parts of the manuscript, including the cover page, abstract, text, references, tables and figures.

Authors may request consideration of longer papers, in advance of submission, when there is clear justification for additional length (e.g., the paper reports on two or more studies or has an unusual or complex methodology). If possible, excess material should be placed in an online supplement rather than in the manuscript.

Brief reports are acceptable for innovative work that may be premature for publication as a full research report because of small sample size, novel methodologies, etc. Brief reports should be designated as such and should not exceed a total of 12 pages, inclusive of **all** parts of the manuscript, including the cover page, abstract, text, references, tables and figures.

All manuscripts should be double-spaced, with margins of at least 1 inch on all sides and a standard font (e.g., Times New Roman) of 12 points.

On the submission portal you will be asked to provide contact information for three individuals who are qualified to serve as unbiased reviewers for your paper. These people must have published peer reviewed work in a relevant field. They must be without any real or perceived conflict of interest with you and your co-authors. They cannot be at the same institution as any author, cannot be a co-author on any recent publications, and must not be a former or current trainee, advisor or mentor, etc.

Submissions that exceed the page limits will be returned to the author for shortening prior to the initiation of peer review.

Submission Letter

The cover letter should indicate that the authors have read and followed the *Health Psychology* Instructions for Authors. It should also include a statement indicating that the paper has been seen and approved by all authors. The cover letter should describe how the paper advances research in health psychology, referring to the journal mission to assure that the submission fits with the scope of papers published in *Health Psychology*.

The full mailing address, telephone, fax, and email address for the corresponding author should be included in the cover letter and title page, along with the names and affiliations of all co-authors.

The cover letter must confirm that the manuscript has not been published, is not currently submitted elsewhere, and that it does not contain data that is currently submitted or published elsewhere.

When a manuscript contains data that is part of a larger study, authors should describe the larger study and provide references for other study papers. Authors must be prepared to provide copies of related manuscripts when requested as part of the editorial review process. Authors should clarify the relationship between their paper, including detailed specification of the overlap in participants, measures, and analysis, and others from the study. The value-added scientific contribution of their study must be clearly stated in the cover letter.

Authors of brief reports should indicate in the cover letter that the full report is not under consideration for publication elsewhere and similarly address potential overlap with other papers.

Manuscripts

The manuscript title should be accurate, fully explanatory, and no longer than 12 words. The title should reflect the content and population studied, and it should not be in the form of an assertion or conclusion. If the paper reports a randomized clinical trial, this should be indicated in the title. The title of brief reports should start with the words "Brief Report". The title page should include the names of all authors and their affiliations at the time the research was done.

All research manuscripts must include a structured abstract containing a maximum of 250 words with the following sections:

- Objective (brief statement of the purpose of the study);

- Methods (summary of the participants, design, measures, procedure);

- Results (primary findings); and

- Conclusions (specific statement of the implications of the data).

Papers such as invited commentaries, for which a structured abstract would be inappropriate, should include an unstructured abstract containing a maximum of 250 words.

Please supply up to five keywords or brief phrases after the abstract. We recommend that you choose medical subject headings (MeSH) and/or psychological index terms for your keywords. The National Library of Medicine offers a free, searchable MeSH database for PubMed

. Also, APA publishes the *Thesaurus of Psychological Index Terms* for our family of databases.

The Introduction should not exceed 3–4 pages in length. The paper should be referenced appropriately but excessive citations should be avoided.

All research involving human participants must describe oversight of the research process by the relevant Institutional Review Boards, along with the name(s) of the approving

institution(s), or an explanation of why no approval was needed. Consent and assent procedures should be described briefly in the Methods section.

All statistical tests should include an effect size with confidence intervals whenever possible.

First person language ("I", "we") should be avoided. Terminology should be sensitive to the individual who has a disease or disability. The journal endorses the concept of "people first, not their disability." Terminology should reflect the "person with a disability" (e.g., children with diabetes, persons with HIV infection, families of people with cancer) rather than the condition as an adjective (e.g., diabetic children, HIV patients, cancer families). Nonsexist language should be used.

It is important to highlight the significance and novel contribution of original work. Replications and extensions of previous studies are welcome, but the rationale and discussion should give due weight to the main purpose of the study (i.e., to confirm, disconfirm, or extend previous research), and it should not give excessive weight to minor innovations or superficially novel features.

Health Psychology publishes a variety of types of papers and work across the entire spectrum of translational research. The translational implications of the research should be discussed but not overstated. Programmatic research is especially welcome. If the study is integral to an ongoing, well-focused program of research, the study's relationship to previous and planned work in the research program should be described.

Qualitative Research

Research papers that utilize qualitative methods should follow the general instructions to authors for style and format. We ask that authors of qualitative papers review the additional guidance below to assure that papers meet the following criteria utilized by *Health Psychology*.

The introduction should make a compelling case for the significance of the study and clearly identify whether it is a stand-alone study or if it fits into a larger research project. For example, qualitative manuscripts may inform the development of a survey, use small-incident samples, or establish feasibility. The specific qualitative paradigm should be specified (e.g., grounded theory, qualitative descriptive approach, interpretive phenomenology) with a rationale as to why it was selected to address the research question.

At the same time, authors are encouraged to avoid methodological tutorials and cite appropriate references for the methodology. Describe your sampling frame clearly and how the sample was selected, justifying the type and size of your sample using appropriate language for qualitative studies.

While many qualitative studies may not use a conceptual model, if you have done so, explain how the model may have shaped the design, data collection, analysis and interpretation.

Explain carefully how you insured rigor in your study e.g., data analysis protocols (including how coders were trained), audit procedures, and demonstration of data saturation. Describe the data analysis and how it relates to your overall approach or paradigm. Present rich and compelling results with data that have been analyzed and interpreted appropriately for your method (e.g., discourse analytic results would be presented differently than those of a grounded theory).

The paper should convey how this research fills an important gap in the science and promises to change the way we approach future studies.

Scale Development

Empirical papers reporting the development of new instruments related to health psychology should follow the general guidelines for style and format of this journal. Authors should make a convincing case for the need and rationale for the new instrument, particularly with respect to new and innovative constructs. Included in this rationale should be the theoretical

foundation on which their new instrument rests along with presentation of other, related scales currently in use. The instrument should be evaluated in the population(s) for which it is intended. If it is intended for use across a variety of populations and/or settings, evidence of its generalizability should be provided. Studies of instruments that are of limited clinical or research utility may be better suited for subspecialty journals.

Health Psychology will also consider studies of existing instruments that were developed in one population but that are now being validated and applied, with or without modification, in a different population that fits within the journal's scope. For example, a measure that was originally developed for otherwise medically well psychiatric patients may be evaluated in patients with cancer or heart disease, if there is a cogent rationale for this work. The journal welcomes relevant health-related applications and evaluations of measures produced by major initiatives of the National Institutes of Health, such as PROMIS, NeuroQoL, ASCQ-ME, and the NIH Toolbox. Authors should clearly articulate the specifics of the study design and of the analytical techniques used. There should be strong consistency among the purpose statements, methods, and the manner in which findings are presented.

Some studies incorporate mixed-methods designs. The specifics of these designs should be presented in sufficient but not excessive detail. Attention should be given to the nature of the items, the basis for their creation, and the rationale for the response options.

The underlying theoretical structure of the approach should be evident, for example, whether one is premising a study on classical or modern theory (IRT, Rasch) techniques. The characteristics of the research will be in part dictated by the nature of the scale. For instance, large, nationally-normed tests may have a much different make-up than that of small, more narrowly-defined measures. Research involving both types of instruments will be considered.

Finally, all instrument development papers should convey how the literature base will be strengthened with the addition of the particular instrument along with a clear and convincing case for the clinical relevance of the information that it provides.

Letters to the Editor

Health Psychology will, at the discretion of the Editor-in-Chief, publish Letters to the Editor on the journal website. However, eligible authors are urged to consider posting a reader comment in PubMed Commons instead of, or in addition to, submitting a Letter to the Editor. Further information about PubMed Commons is available at www.ncbi.nlm.nih.gov/pubmedcommons.

Letters should be prepared in direct response to articles published in the journal, should include a reference to the published paper, and should be sent to the Editorial Manuscript Coordinator, Lindsay MacMurray within 60 days of the date when the relevant article is published in hard copy.

The text of the letter, excluding the title, references and author(s) name, title, affiliation and email, may not exceed 400 words. There should be no more than five references.

In a separate cover letter, the author should indicate that the submission is a Letter to the Editor for consideration of posting on the *Health Psychology* website and provide the full citation of the original article to which the letter refers. The cover letter should also indicate if the letter writer(s) have any conflicts of interest related to the article or correspondence. Letters will not be a forum for ongoing dialogue.

Review Policy

Health Psychology has revised its peer review policies and now provides single-blinded rather than double-blinded reviews. In other words, the reviewers are anonymous but the authors are not. The title page of all submitted manuscripts should include the names of all

authors and their affiliations at the time the research was done. Identifying information should **not** be masked on the title page or anywhere else in the manuscript.

Clinical Trials

Overview

In line with current publication standards, *Health Psychology* has implemented several requirements for randomized controlled trial (RCT) reports. These include 1) trial registration, 2) protocol submission, and 3) adherence to reporting guidelines.

Trial Registration

Health Psychology will publish reports of RCTs only if they have been duly registered at ClinicalTrials.gov or at another recognized, publicly accessible registry. A complete list of acceptable trial registries can be found via the WHO International Clinical Trials Registry Platform.

If recruitment commences on or after January 1, 2018, the trial must be registered prospectively (i.e., before recruitment begins). If recruitment commenced before January 1, 2018, the trial must be registered prior to submission of the manuscript, even if the registration is retrospective (i.e., filed after recruitment began). Trial registrations must include all elements of each primary and secondary outcome, including the times at which each outcome will be measured and analyzed. The name of the trial registry and the registration number should be listed below the abstract. All differences between (a) the reported methods and outcomes and (b) the registered methods and outcomes must be described and explained in the manuscript.

Protocol Submission

The complete trial protocol, including the entire a priori statistical analysis plan, should be readily available to readers of manuscripts reporting the results of clinical trials, both for primary outcome papers as well as for ones limited to secondary, exploratory, or *post*

hoc outcome analyses. Few reports include the complete RCT protocol in the Methods section. We therefore advise authors to upload the complete protocol with their manuscript, along with a <u>Standard Protocol Items: Recommendations for Intervention Trials (SPIRIT) checklist</u>.

Both published and unpublished protocols are acceptable. Published protocols should be cited in the submitted manuscript. Previously unpublished protocols will be published as an online-only supplement if the trial report manuscript is accepted.

Adherence to Reporting Guidelines

All RCT reports must be accompanied by a completed CONSORT checklist. CONSORT extension checklists should be used when appropriate. The manuscript itself should include a CONSORT flow diagram. The CONSORT guidelines, checklists, and flow diagram templates are available at <u>The EQUATOR Network</u>.

Reporting Guidelines for Other Types of Studies

Reporting guidelines have been developed for many other types of studies besides clinical trials. For example, there are guidelines for reporting observational studies, meta-analyses, diagnostic and prognostic studies, and qualitative research. If a reporting guideline exists for the type of study that is being submitted to *Health Psychology*, the report must be accompanied by the associated checklist. If there is an associated flow diagram, it must be included as a figure in the manuscript. Reporting guidelines, checklists, and flow diagrams for many different types of studies are available at <u>www.equator-network.org</u>.

Manuscript Preparation

Prepare manuscripts according to the <u>*Publication Manual of the American Psychological Association* (6th edition)</u>

. Double-space all copy. Other formatting instructions, as well as instructions on preparing tables, figures, references, metrics, and abstracts, appear in the *Manual*. Additional guidance on APA Style is available on the APA Style website.

Review APA's Checklist for Manuscript Submission before submitting your article. Manuscripts may be copyedited for bias-free language (see Chapter 3 of the *Publication Manual*).

Below are additional instructions regarding the preparation of display equations, computer code, and tables.

Display Equations

We strongly encourage you to use MathType (third-party software) or Equation Editor 3.0 (built into pre-2007 versions of Word) to construct your equations, rather than the equation support that is built into Word 2007 and Word 2010. Equations composed with the built-in Word 2007/Word 2010 equation support are converted to low-resolution graphics when they enter the production process and must be rekeyed by the typesetter, which may introduce errors.

To construct your equations with MathType or Equation Editor 3.0:

- Go to the Text section of the Insert tab and select Object.

- Select MathType or Equation Editor 3.0 in the drop-down menu.

If you have an equation that has already been produced using Microsoft Word 2007 or 2010 and you have access to the full version of MathType 6.5 or later, you can convert this equation to MathType by clicking on MathType Insert Equation. Copy the equation from Microsoft Word and paste it into the MathType box. Verify that your equation is correct, click File, and then click Update. Your equation has now been inserted into your Word file as a MathType Equation.

Use Equation Editor 3.0 or MathType only for equations or for formulas that cannot be produced as Word text using the Times or Symbol font.

Computer Code

Because altering computer code in any way (e.g., indents, line spacing, line breaks, page breaks) during the typesetting process could alter its meaning, we treat computer code differently from the rest of your article in our production process. To that end, we request separate files for computer code.

In Online Supplemental Material

We request that runnable source code be included as supplemental material to the article. For more information, visit Supplementing Your Article With Online Material.

In the Text of the Article

If you would like to include code in the text of your published manuscript, please submit a separate file with your code exactly as you want it to appear, using Courier New font with a type size of 8 points. We will make an image of each segment of code in your article that exceeds 40 characters in length. (Shorter snippets of code that appear in text will be typeset in Courier New and run in with the rest of the text.) If an appendix contains a mix of code and explanatory text, please submit a file that contains the entire appendix, with the code keyed in 8-point Courier New.

Tables

Use Word's Insert Table function when you create tables. Using spaces or tabs in your table will create problems when the table is typeset and may result in errors.

Submitting Supplemental Materials

APA can place supplemental materials online, available via the published article in the PsycARTICLES® database. Please see Supplementing Your Article With Online Material for more details.

References

List references in alphabetical order. Each listed reference should be cited in text, and each

text citation should be listed in the References section.

Examples of basic reference formats:

- **Journal Article:**

  Hughes, G., Desantis, A., & Waszak, F. (2013). Mechanisms of intentional binding

  and sensory attenuation: The role of temporal prediction, temporal control, identity

  prediction, and motor prediction. *Psychological Bulletin, 139,* 133–151.

  http://dx.doi.org/10.1037/a0028566

- **Authored Book:**

  Rogers, T. T., & McClelland, J. L. (2004). *Semantic cognition: A parallel distributed*

  processing approach. Cambridge, MA: MIT Press.

- **Chapter in an Edited Book:**

  Gill, M. J., & Sypher, B. D. (2009). Workplace incivility and organizational trust. In

  P. Lutgen-Sandvik & B. D. Sypher (Eds.), *Destructive organizational*

  communication: Processes, consequences, and constructive ways of organizing (pp.

  53–73). New York, NY: Taylor & Francis.

Figures

Graphics files are welcome if supplied as Tiff or EPS files. Multipanel figures (i.e., figures

with parts labeled a, b, c, d, etc.) should be assembled into one file.

The minimum line weight for line art is 0.5 point for optimal printing.

For more information about acceptable resolutions, fonts, sizing, and other figure

issues, please see the general guidelines.

When possible, please place symbol legends below the figure instead of to the side.

APA offers authors the option to publish their figures online in color without the costs associated with print publication of color figures.

The same caption will appear on both the online (color) and print (black and white) versions. To ensure that the figure can be understood in both formats, authors should add alternative wording (e.g., "the red (dark gray) bars represent") as needed.

For authors who prefer their figures to be published in color both in print and online, original color figures can be printed in color at the editor's and publisher's discretion provided the author agrees to pay:

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- An additional $600 for the second figure

- An additional $450 for each subsequent figure

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Ethical Principles

It is a violation of APA Ethical Principles to publish "as original data, data that have been previously published" (Standard 8.13).

In addition, APA Ethical Principles specify that "after research results are published, psychologists do not withhold the data on which their conclusions are based from other competent professionals who seek to verify the substantive claims through reanalysis and who intend to use such data only for that purpose, provided that the confidentiality of the participants can be protected and unless legal rights concerning proprietary data preclude their release" (Standard 8.14).

APA expects authors to adhere to these standards. Specifically, APA expects authors to have their data available throughout the editorial review process and for at least 5 years after the date of publication.

Authors are required to state in writing that they have complied with APA ethical standards in the treatment of their sample, human or animal, or to describe the details of treatment.

- Download Certification of Compliance With APA Ethical Principles Form (PDF, 26KB)

The APA Ethics Office provides the full <u>Ethical Principles of Psychologists and Code of Conduct</u> electronically on its website in HTML, PDF, and Word format. You may also request a copy by <u>emailing</u> or calling the APA Ethics Office (202-336-5930). You may also read "Ethical Principles," December 1992, *American Psychologist*, Vol. 47, pp. 1597–1611.