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Lifestyle factors and Alzheimer's disease in people with Down syndrome

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Background: Lifestyle has previously been associated with the onset of Alzheimer's disease (AD) in the typically developing population, but research investigating this association in Down syndrome (DS) is limited.

Method: Adults with DS and AD (*n* = 27) were compared to adults with DS without AD (*n* = 30) on physical activity, diet, weight, where participants currently lived, where participants had lived for the majority of their lives, educational attainment, occupational attainment and cognitive activity.

Results: There was a significant difference between samples on where participants currently lived, with the majority of the clinical sample living in institutionalised settings and the majority of the control sample living in independent/supported living settings. This may reflect a tendency to move people once they start to deteriorate which, if correct, is contrary to clinical recommendations that people with AD should be supported to 'die in place'.

Conclusions: Further research into the way in which lifestyle factors, particularly living environment, could contribute to the increased risk of AD in adults with DS is required. This may support interventions aimed at preventing or delaying the onset of the disease.

Keywords: Intellectual disabilities, Dementia, Alzheimer's disease, Down syndrome, Lifestyle

Introduction

Alzheimer's Disease in People with Down Syndrome

The life expectancy of individuals with Down syndrome (DS) has significantly increased over the past few decades, with the average life expectancy of people with DS increasing from 12 years of age in 1949 (Penrose, 1949) to 60 years of age in 2002 (Bittles and Glasson, 2004). The increasing numbers of individuals with DS living into middle and old age (Torr *et al*., 2010) creates the potential for people with DS to live much fuller lives, yet also brings with it the increased risk of developing age-related illnesses, particularly dementia (Jordens *et al*., 1997).

In particular, as they age, people with DS have an increased risk of developing the characteristic neuropathological manifestations of Alzheimer disease (AD), including an excessive build-up of amyloid plaques and neurofibrillary tangles (Burger and Vogel, 1973; Holland and Oliver, 1995; Kolb and Whishaw, 2003; Wisniewki *et al*., 1985). This has been attributed to the triplication and overexpression of the gene encoding for the amyloid precursor protein located on chromosome 21 (Rumble *et al*., 1989). In many individuals with DS, the neuropathological manifestations of AD are evident by the age of 40, even though individuals may appear to be clinically asymptomatic. Individuals with DS are thus at risk of developing AD some 10-15 years earlier than those in the typically developing population (Evenhuis, 1990; Lai and Williams, 1989), although even with this increased risk not all will inevitably develop the disease. A recent meta-analysis also suggests, in

conjunction with findings in the typically developing population, that individuals with DS harboring the apolipoprotein E4 (APOE4) allele may have an increased risk of developing early onset AD and early mortality compared to those without this protein present (Rohn et al., 2014); therefore increasing the genetic risk of AD in some DS individuals further.

Due to the level of learning disabilities present in individuals with DS, it is often difficult to identify the symptoms associated with AD. This difficulty can be mitigated if an extensive baseline of skills is recorded, ideally during early adulthood, to better ensure that any changes in cognitive and social functioning later in life can be detected (Jethwa and Cassidy, 2010; McBrien *et al*., 2005; British Psychological Society and Royal College of Psychiatrists, 2015). Early detection of symptoms has the potential to lead to more accurate diagnoses of dementia, and to better enable services and carers to provide the support required by dementia patients.

Lifestyle Factors and Alzheimer's Disease in the Typically Developing Population

A number of lifestyle factors have been associated with the onset of AD in the typically developing population. These include: physical activity, diet, weight, educational attainment, occupational attainment and cognitive activity.

Physical activity.

A mounting body of evidence suggests that regular physical activity maintains cognitive performance and reduces the chances of developing of AD (Podewils *et al*., 2005). Neurological explanations have suggested that this is, in part, due to the increased blood flow to the brain during physical exercise,

which has been proven to reduce several of the cardiovascular risk factors associated with cognitive impairment and dementia. These include hypertension and diabetes (Fillet *et al*., 2008; Ott *et al*., 1999).

Research with physically capable elderly men has suggested that men who walk on a regular basis have better cognitive function and are less likely to develop dementia, compared to those who do not (Abbott *et al*., 2004). Additionally, engaging in physical exercise at least twice a week during midlife has been suggested to reduce the likelihood of developing, or delay the onset of, dementia in late-life, especially among genetically susceptible individuals (Rovio *et al*., 2005).

Diet.

Some data have linked specific dietary patterns to a lowered chance of developing AD. The Mediterranean diet in particular, which is characterised by high consumption of fruits, vegetables, whole grains, olive oil and low consumption of red meat and saturated fat, has been strongly related to a lowered chance of developing AD and slower cognitive decline (Scarmeas *et al*., 2006) as well as other forms of dementia (Barberger-Gateau *et al*., 2007). For example, a prospective cohort study of 1880 older people without dementia found that those who adhered to a Mediterranean diet and engaged in regular physical exercise had up to a 40% lowered chance of developing AD, compared to those neither adhering to the diet nor participating in physical activity (Scarmeas *et al*., 2009).

Weight.

A lifestyle factor believed to significantly increase the likelihood of elderly women developing AD is being overweight (Gustafson *et al.*, 2003). Over an 18 year longitudinal study, Gustafson and her colleagues found that women who developed dementia between the ages of 79 and 88 years had a higher average body mass index (BMI) compared to women without dementia. It was also found that women who developed AD between the ages of 70 and 79 years were more overweight than women without dementia, and that for every 1.0 increase in BMI at the age of 70 years, the chances of developing AD increased by 36%. It should be noted that this study did not find these associations in men. Nevertheless, further research conducted by Whitmer *et al.* (2005) found that obesity in middle age increases the likelihood of developing AD and vascular dementia, in both males and females, independently of co-morbid conditions. The results of their study demonstrated that obese people (those with a BMI ≥ 30) had a 74% increased chance of developing dementia, and overweight people (those with a BMI 25.0 – 29.9) had a 35% increased chance of developing dementia compared with those of normal weight (those with a BMI of 18.6 -24.9).

Educational and occupational attainment.

In a longitudinal cohort study of 593 non-demented individuals, aged 60 years and above, Stern *et al.* (1994) demonstrated that individuals with either low educational or low lifetime occupational attainment had a greater chance of developing AD, compared to those with a higher level of educational or occupational attainment. Those individuals with both low educational and occupational attainment were found to have the greatest chance of developing AD. Later research, by Brayne *et al*. (2010), also provided

evidence of an association between higher levels of education and a decreased likelihood of developing dementia in 872 brain donors, of which 56% had dementia at death. They found that before death those individuals who spent more years in education displayed less of the clinical features of dementia compared to those who spent fewer years in education.

Furthermore, an additional association has been found between higher education levels and factors that reduce the build-up of extracellular Aβ plaques (Bennett *et al*., 2005), which are believed to be one of the most common causes of AD (Wilson *et al*., 2007).

Cognitive activity.

During the past two decades, many large prospective studies conducted within the typically developing population have highlighted an association between regular engagement in cognitive activity and a reduced likelihood of developing AD (Hultsch *et al*., 1999; Verghese *et al*., 2003; Wang *et al*., 2002; Wilson *et al*., 2002). More recently, a longitudinal clinical-pathologic study by Wilson *et al*. (2007) found that those individuals who reported being cognitively active, i.e. those that regularly engaged in activities that required them to seek or process information such as reading a newspaper, were 2.6 times less likely to develop AD than those who reported being cognitively inactive. This finding suggests that regular engagement in mentally stimulating activity during old age may prevent, or at least delay, the onset of AD.

Rationale and Hypotheses

There is reasonable evidence to suggest that several lifestyle factors are associated with the onset of AD in the typically developing population. This evidence has also contributed to our understanding of the way in which certain lifestyle changes could potentially protect or provide resilience against dementia related pathology. However, there are significant gaps in our understanding of the way in which these lifestyle factors might contribute to the increased risk of AD in adults with DS, which is typically only attributed to genetic factors (Folstein and Folstein, 1997). It may be that the brain pathology associated with DS overrides the impact of any significant lifestyle factors whatsoever. However, given the mounting evidence of associations between lifestyle factors and AD in the typically developing population, and the increasing life expectancy of individuals with DS, it seems important to investigate these associations in the DS population. This may support interventions aimed at preventing or delaying the onset of the disease and possibly improving overall quality of life.

An investigation of the contribution of lifestyle factors to the onset of AD in people with DS was conducted by Temple *et al*. (2001). This study compared 17 individuals with DS and AD, with 18 individuals with DS and without AD, on the following lifestyle factors: education, employment, recreational activities, years in an institution and overall level of cognitive functioning. The results did not reveal any significant direct associations between any of the lifestyle factors and the onset of AD; however it was found that overall level of cognitive functioning was associated with all of the lifestyle factors under investigation. Specifically, a higher level of cognitive functioning was

associated with fewer cases of AD. These findings led the researchers to conclude that environmental interventions aimed at improving level of cognitive functioning might be useful in deferring the onset of AD. Although no direct associations were found, the researchers noted that the influence of lifestyle factors on the onset of AD in the DS population should not be ruled out. They suggested that because this study was conducted with a small sample, the power of the analysis was reduced for detecting differences between groups.

The work of Temple *et al*. (2001) represents an important start in our understanding of the influence that lifestyle factors may have on the onset of AD in people with DS. The present study aims to build on the findings of Temple and colleagues by comparing people with DS, with and without a diagnosis of AD, on the following lifestyle factors: physical activity, diet, weight, educational attainment, where participants currently live, where participants had lived for the majority of their lives, occupational attainment, and cognitive activity. It was hypothesised that there would be differences between the two groups on these factors and that individuals with DS and a higher level of cognitive functioning (those with mild to moderate learning disabilities) would be less likely to develop AD than individuals with DS and a lower level of cognitive functioning (those with severe to profound learning disabilities).

Method

Participants

The existing data of 169 adults with DS, which had been collected over a 10 year period for the purpose of a prospective dementia screening programme led by the Plymouth Community Learning Disabilities Team (PCLDT), were reviewed for the identification of potential participants. This programme was set up to increase the chances of identifying early onset AD in individuals with DS living in Plymouth.

In 2001, this programme identified all adults (aged 18+) with DS living in Plymouth, many of which were already known to the PCLDT, and offered them a baseline neuropsychological dementia assessment. Following baseline testing, all individuals aged 40 to 50 were assessed biannually and then annually after the age of 50. None of the participants were deemed to have the capacity to consent to enter into the screening programme, so in each case the participant's involvement in the screening programme was carefully appraised in light of consultation with people deemed to know the participant best (e.g. their next of kin, or closest carer), and re-appraised throughout their involvement in the study (e.g. by paying close attention to levels of engagement, balancing costs/benefits, etc). These procedures were reviewed and approved by the National Health Service Local Research Ethics Committee and by the School of Psychology Ethics Committee, University of Plymouth.

The data of both living and deceased participants were reviewed for the purpose of the current study. These data included information gained from the

dementia assessments completed with participants over the past 10 years, and included detailed information about the person's history. Consequently, there was a minimum of 10 years worth of dementia assessments for each participant, the number of which depended on the age of the participant. For example, if the participant was aged 40 at baseline then they should have had six dementia assessments in total (one at baseline and then one every two years for 10 years). From the available data, participants were excluded if: i) they had a diagnosis of dementia other than AD; ii) they did not have a diagnosis of any dementia type, but dementia related concerns had previously been raised. The latter exclusion criterion was implemented as a way of best ensuring that the control group contained participants who did not have any clinical signs or symptoms of AD.

Following the inclusion and exclusion process, 59 participants were identified as eligible for the current study. Two of these participants were excluded from the sample due to data unavailability. Twenty seven of the participants (19 males and 8 females) had a diagnosis of AD and formed the clinical sample. Thirty of the participants (19 males and 11 females) did not have a diagnosis of AD and formed the control sample. Participants varied in terms of age (*age range at analyses = 46-78 years*; mean age = 57.5 years) and pre-morbid level of learning disabilities. Table 1 provides an overview of the age ranges of participants in the clinical and the control group, and the range of learning disabilities experienced by all of the participants.

Insert Table 1: Age ranges of the participants in the clinical (*n* = 27) and control (*n* = 30) groups, and p*re-morbid level of learning disabilities for all participants (N = 57)*

Materials

The information gained from each of the participants' routine dementia assessments was thoroughly explored to locate data related to the following lifestyle factors: physical activity, diet, weight, where participants currently lived, where participants had lived for the majority of their lives, educational attainment, occupational attainment, and cognitive activity. This information was found in each of the participant's independent, confidential files held with the PCLDT, and/or on the programme's confidential database.

Participants' pre-morbid levels of learning disabilities, previously assessed by the British Picture Vocabulary Scale – Second Edition (BPVS II; Dunn et al., 1997), were used to determine each of the participants' level of cognitive functioning. The BPVS II is a test of receptive vocabulary ability which can be also be used to determine an individuals' level of learning disability, using criteria proposed by the ICD-10 diagnostic criteria (WHO, 2015). The BPVS forms part of the dementia assessment battery, and was completed with each of the participants at their baseline dementia assessment.

Procedure

Data collection was conducted exclusively on PCLDT premises and all confidential information remained within the PCLDT according to organisational policies. Data were numerically coded according to the criteria described under 'Coding of Lifestyle Factors' below and collated onto an anonymised SPSS database for statistical analysis. The investigation of the principal hypothesis implemented a case control study design, which involved

comparing each of the lifestyle factors under investigation between the clinical sample and the control sample, to identify any differences between people with DS, with and without a diagnosis of AD.

Additional analyses were conducted to investigate the secondary hypotheses, which examined whether level of cognitive functioning was significantly associated with the onset of AD, and whether cognitive functioning itself was directly associated with any of the lifestyle factors under investigation. Data for some of the lifestyle factors were unavailable from some of the participants and were therefore coded as missing for all statistical analysis.

Coding of Lifestyle Factors

Physical activity, diet, weight, education and cognitive activity were coded on an ordinal scale:

Physical Activity was coded in line with research conducted by Laurin *et al*. (2001) who investigated the association between physical activity and the risk of cognitive impairment and dementia in the typically developing population. This included identifying the frequency and intensity of physical activity undertaken by each participant, and then assigning them with one of the following codes: 1 = High level of physical activity (participants who engaged in physical activity three or more times per week at an intensity greater than walking); 2 = Moderate level of physical activity (participants who engaged in physical activity three or more times per week at an intensity equal to walking); 3 = Low level of physical activity (participants who engaged in all other combinations of frequency and intensity of physical activity).

Diet was coded according to the following: 1 = Good diet (participants deemed to have a balanced and healthy diet, including frequent consumption of fruit and vegetables); 2 = Poor diet (participants deemed to have an unbalanced and unhealthy diet, including regular intake of foods high in fat).

Weight was coded according to the following: 1 = Normal weight (participants with a BMI 18.6 - 24.9); 2 = Overweight (participants with a BMI 25.0 - 29.9); 3 = Obese (participants with a BMI ≥ 30).

Educational attainment was coded in line with research conducted by Brayne *et al.* (2010). This involved recording the number of years each participant had spent in formal education: 1 = 12+ years spent in formal education; 2 = 8-11 years spent in formal education; 3 = 4-7 years spent in formal education; 4 = 0-3 years spent in formal education; 5 = Evidence of formal education, but number of years unknown.

Cognitive activity was coded according to the following: 1 = High level of cognitive activity (participants who engaged in activities that required concentration on a daily basis at an intensity deemed equal to or greater than that required for reading and writing), 2 = Moderate level of cognitive activity (participants who engaged in activities that required concentration on a weekly basis at an intensity deemed less than that required for reading and writing), 3 = Low level of cognitive activity (participants who engaged in all other combinations of frequency and intensity of cognitive activity).

Where participants lived, where participants had lived for the majority of their lives and occupational attainment were coded on a nominal scale:

Living environment was coded in two ways. Firstly, it was coded according to *where participants lived* (or where they were living when they died for deceased participants): 1 = Independent Living/Supported Living (participants living with relatives or in supported living accommodation); 2 = Institutionalised Living (participants living in residential accommodation). Secondly, it was coded according to *where participants had lived for the majority of their lives*: 1 = Independent Living/Supported Living (participants who have lived the majority of their lives with relatives or in supported living accommodation); 2 = Institutionalised Living (participants who have lived the majority of their lives in residential accommodation).

Occupational attainment was coded according to the following: 1 = Evidence of occupational attainment (participants who have had paid or voluntary work experience and/or attended a day placement); 2 = No evidence of occupational attainment (participants who have had no paid or voluntary work experience and/or never attended a day placement).

Results

Non-parametric tests were used to test for differences between the clinical sample and control sample on all the lifestyle factors under investigation. The use of these tests was due to significant amounts of missing lifestyle data which resulted in a non-normal distribution.

Differences between the samples on all of the ordinal variables, including: physical activity, diet, weight, education and cognitive activity were tested

using a Mann Whitney U test. Descriptive statistics for these data are shown in Table 2. The Mann Whitney U test did not reveal any statistically significant differences between the clinical sample and the control sample on physical activity (U = 339, p = .61), diet (U = 152, p = .43), weight (U = 169.5, p = .23) or cognitive activity (U = 316, p = .72). However, the test approached significance for education (U = 87, p = .09) suggesting that the clinical sample may have spent fewer years in education. This could be validated by a further study with a larger sample of participants.

Insert Table 2: *Mean, standard deviation and median for each ordinal lifestyle factor code attributed to participants in the clinical (n = 27) and control (n = 30) samples*

A Pearson's Chi-Square test was conducted to test for differences between the clinical sample and the control sample on all of the nominal variables, including: where participants currently lived, where participants had lived for the majority of their lives and occupational attainment. The observed frequencies of these data are shown in Table 3. The Pearson's Chi-Square test revealed a statistically significant difference between the samples on where participants currently lived, X^2 (1, n = 56) = 10.85, p = .001. Specifically, the majority of individuals in the clinical sample were living in institutionalised living environments, whereas the majority of individuals in the control sample were living in independent/supported living environments. No differences were found between the samples on where participants had lived for the majority of their lives, X^2 (1, n = 46) = .69, p = .41. The majority of participants, across both conditions, had spent most of their lives living in

independent/supported living environments. Also, no difference was found between the conditions on occupational attainment, with X^2 (1, *n* = 55) = .36, *p* = .85. The majority of participants, across both conditions, had gained some degree of work experience throughout their lives.

Insert Table 3: *Observed frequencies of where participants in the clinical (n = 27) and control (n = 30) samples currently live, had lived for the majority of their lives, and of the work experience they have gained*

Insert Table 4: *Frequency distribution of the level of learning disabilities in the clinical (n = 27) and control (n = 30) samples*

Subsequent analyses were carried out to investigate whether individuals with a higher level of cognitive functioning (those with mild to moderate learning disabilities) were less likely to develop AD than those with a lower level of cognitive functioning (those with severe to profound learning disabilities). A frequency distribution of the pre-morbid levels of learning disabilities experienced by participants in both samples revealed no difference between the two samples on levels of learning disabilities. The majority of individuals in both the clinical sample and the control sample had a lower level of cognitive functioning. This distribution is demonstrated in Table 4. Confirming this impression, a Mann Whitney U test did not reveal a statistically significant difference between the two samples on levels of learning disabilities (*U* = 122, *p* = .17).

In order to determine whether level of cognitive functioning itself was associated with the lifestyle factors, the relationships between pre-morbid level of learning disability and each ordinal lifestyle factor were examined using scatter plots and Kendall tau b correlation coefficients (see Table 5). There were no statistically significant relationships between level of learning disabilities and: physical activity, r (33) = -.16, p = .33; diet, r (22) = -.18, p = .35; weight, r (19) = -.02, p = .91; education, r (22) = .28, p = .11. A statistically significant relationship was revealed between level of learning disabilities and cognitive activity, r (33) = .40, p = .008. This suggests that the higher the level of cognitive activity engaged in by participants, the higher their level of cognitive functioning. Regression was considered but ruled out due to only one significant correlation.

Insert Table 5: *Kendall tau b correlation coefficients of the relationships between pre-morbid level of learning disability and physical activity, diet, weight, education and cognitive activity*

Discussion

Overview of Findings

The present study principally hypothesised that there would be differences between people with DS, with and without a diagnosis of AD on: physical activity; diet; weight; where participants currently lived; where participants had lived for the majority of their lives; educational and occupational attainment; cognitive activity. For the majority of the lifestyle factors investigated, no differences were found between samples. Nevertheless, there was a difference between the samples on where participants currently lived. Even though there were no differences between samples on where participants had lived for the majority of their lives, we found that participants with AD were more likely to *currently* live in institutionalised settings. This could be explained by a tendency for individuals with DS and AD to be moved into institutionalised environments as they start to deteriorate, due to their increasingly higher care needs than those individuals with DS without AD. We will return to this finding in 'Recommendations for Clinical Practice' below.

Additionally, the present study hypothesised that individuals with DS and a higher level of cognitive functioning (i.e. those with mild to moderate pre-morbid levels of learning disabilities) would be less likely to develop AD than individuals with DS and a lower level of cognitive functioning (those with severe to profound pre-morbid levels of learning disabilities). Contrary to this prediction, we found no significant differences between the two samples on level of cognitive functioning. This is counter to the findings of Temple *et al*. (2011). Closer inspection of our data suggests that this finding may reflect the

fact that the vast majority of our participants in both the clinical sample (*n* = 15) and the control sample (*n* = 14) had severe to profound pre-morbid levels of learning disabilities, and only a small minority in the clinical sample (*n* = 1) and the control sample (*n* = 7) had mild to moderate pre-morbid levels of learning disabilities. Moreover, the pre-morbid levels of learning disabilities for some of the participants' (*n* = 20) were missing and therefore unavailable for analysis. It is likely that many of these participants had missing data because the extent of their learning disabilities prevented them from engaging with the BPVS II (i.e. they also had severe to profound learning disabilities), although it is difficult to be sure of this without further analysis. Larger numbers of participants in the mild to moderate learning disabilities range would be needed to explore these findings further.

Further analyses, using scatter plots and Kendall tau b correlations, revealed a significant association between the cognitive functioning of participants (i.e. their level of learning disabilities) and levels of cognitive activity, such that more able participants were reported to participate in higher levels of cognitive activity (i.e. they engaged in more mentally stimulating exercises on more of a regular basis). No significant correlations were found between the cognitive functioning of participants and physical activity, diet, weight, or educational attainment. Temple *et al*. (2011) identified an association between level of cognitive functioning and levels of education, so it is possible that the lack of a statistically significant result in the present study was due to missing data on this lifestyle factor.

The finding of a relationship between cognitive functioning and cognitive activity is not surprising, and supports previous research conducted within the

typically developing population. Salthouse (2006) demonstrated that individuals with cognitively demanding occupations, such as college professors, pilots, and physicians, maintain higher cognitive functioning with ageing than individuals with less cognitively demanding occupations. Similarly, research has indicated that older adults who regularly engage in leisure activities which involve working memory and reasoning skills (e.g. chess, bridge, crosswords), maintain higher cognitive functioning than adults of the same age, who do not engage in these cognitively stimulating activities (Mireles and Charness, 2002). The direction of cause and effect between level of learning disabilities and cognitive activity cannot be established from the results of the present study; further research is required.

The interpretations of the present findings should be taken with caution, and with consideration of two key limitations. Firstly, the present study was limited by missing data, which was often as result of information (e.g. weight, diet) not being recorded in clinical files. These missing data reduced the representativeness of our samples, resulted in a non-normal distribution of the lifestyle data and the use of non-parametric tests, and limited our ability to make inferences about the DS population. It is possible that lifestyle factors are poorly understood in this population because they are rarely recorded as a matter of course. Alternatively, it may be that the brain pathology associated with DS overrides any significant impact of lifestyle factors. Secondly, the lifestyle data collected in the present study were obtained from archived files which only held information about each of the participants since they were first known to the PCLDT and may not have been representative of the participants' complete lifestyle history.

Recommendations for Clinical Practice

The availability of lifestyle information for individuals with DS appears to contrast with the information available for individuals within the typically developing population. This may represent a health inequality for people with learning disabilities, particularly for people with DS. Research suggests that individuals with learning disabilities access health care services less often than the typical population, and moreover, that a number of barriers exist in accessing these services (e.g. scarcity of appropriate services for individuals with learning disabilities, physical and informational barriers to access, inexperienced healthcare staff, and increasingly stringent eligibility criteria for accessing social care services: see Emerson *et al*., 2012). These barriers may mean that key lifestyle information is not being recorded. It is also possible that lower expectations regarding the lifestyles of people with learning disabilities, and particularly DS, mean that professionals do not routinely offer interventions around issues such as diet or weight (see Hamilton *et al*., 2007). Finally, the missing data on lifestyle factors may simply reflect the fact that our knowledge of the impact of environmental factors on the expression of genetic risk factors for AD in the DS population is extremely limited compared to that in the typically developing population, and that research efforts in this area have been scant up to now. Prospective dementia screening programmes for people with Down's syndrome are one way that data on lifestyle factors could be collected routinely in order to further efforts in this area. The development of a national or international protocol to guide service providers in collecting this type of data would be particularly

beneficial. [1]The findings of the current study suggest that people with DS and AD are more likely to currently live in institutionalised settings, which may reflect a tendency to move people once they start to deteriorate. More evidence is needed to support this explanation, but if correct, these findings are contrary to recommendations that people with AD should be supported to stay in their own homes where possible ("Remaining Independent", 2013), and particularly to recommendations from clinicians expert in DS and AD that advocate for the person staying in their own home where possible and 'dying in place' (Dodd, 2012). With an ageing population, it feels important for Community Learning Disabilities Teams to be advising support providers on how they may 'future proof' their provision, such that individuals with DS can remain in their familiar homes should they start to develop dementia. There is an important role here in offering consultation to providers, and in conducting clinically based research on the impact of placement moves for people with AD and DS.

Directions for Future Research

The limitations of the present study indicate the need for longitudinal research to be carried out investigating the effect of lifestyle factors on the onset of AD in people with DS. Future investigations might consider using a longitudinal cohort study design, comparing the lifestyles of individuals with DS with individuals in the typically developing population. Where prospective dementia screening programmes exist for people with DS, clinicians and researchers should consider recording lifestyle factors as routine. It may also

[1] This was a recommendation made by an anonymous reviewer.

be possible to collect these data as part of the annual health checks offered to all people with learning disabilities in the UK.

There may also be a role for clinically based research on the impact of placement moves for people with AD and DS, and the factors that enable families and providers to adjust to a person's changing needs so that they are able to remain at home for as long as possible.

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	Clinical				Control		
Lifestyle Factor	***n***	***M***	***SD***	***Mdn***	***n***	***M***	***SD***
Physical Activity	25	2.72	.46	3	29	2.66	.48
Diet	18	1.56	.51	2	20	1.40	.50
Weight	17	1.53	.72	1	16	1.81	.66
Education	11	4	1.34	4	25	2.92	1.66
Cognitive Activity	23	2.39	.78	3	29	2.34	.72

	Currently Live		Lived Majority of Lives		Work Hist	
	Independent/ Supported Living	Diet	Independent/ Supported Living	Education	Work Experience	Le Lea Dis
Physical Activity	1.00	.13	.24	.18	.23	-
Diet	.13	1.00	.01	-.02	-.15	-
Weight	-.24	.01	1.00	-.19	-.21	-
Education	.18	-.02	-.19	1.00	.35	
Cognitive Activity	.23	-.15	-.21	.35	1.00	
Level of Learning Disability	-.16	-.18	-.02	.28	.40	1

	Level of Learning Disability	Clinical	Co
High Level of Cognitive Functioning	Mild	1	
	Moderate	0	
Low Level of Cognitive Functioning	Severe	6	
	Profound	9	
Missing	----	11	

Table 1: *Age ranges of the participants in the clinical (n = 27) and control (n = 30) groups, and pre-morbid level of learning disabilities for all participants (N = 57)*

Age Groups			**Level of Learning Disability (Receptive Vocabulary Age Equivalent)**	
	Clinical (*n* = 27)	**Control (*n* = 30)**		**Clinical & Control (*N* = 57)**
40-50 years	2	4	Mild (9.00 - < 12.00 years)	3
50-60 years	11	18	Moderate (6.00 - < 9.00 years)	5
60-70 years	13	6	Severe (3.00 - < 6.00 years)	12
70-80 years	1	2	Profound	17
80+ years	0	0	Unknown	20

Table 2: *Mean, standard deviation and median for each ordinal lifestyle factor code attributed to participants in the clinical (n = 27) and control (n = 30) samples*

Table 3: Observed frequencies of where participants in the clinical (n = 27) and control (n = 30) samples currently live, had lived for the majority of their lives, and of the work experience they have gained

Table 4: *Frequency distribution of the level of learning disabilities in the clinical (n = 27) and control (n = 30) samples*

Table 5: *Kendall tau b correlation coefficients of the relationships between pre-morbid level of learning disability and physical activity, diet, weight, education and cognitive activity*