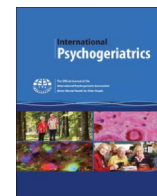




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Original Research Article

Strength together: Risk and protective factors for dementia and cognitive impairment in Aboriginal and Torres Strait Islander peoples

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ABSTRACT

Objectives: Dementia prevalence is higher among Aboriginal and Torres Strait Islander peoples than in the non-Indigenous population, however, reported risk factors vary across studies. This study aimed to elucidate factors associated with dementia and Cognitive Impairment Not Dementia (CIND) in a harmonised dataset of Aboriginal and Torres Strait Islander participants.

Design: Univariable and multivariable logistic regression was used to assess cross-sectional factors associated with dementia and/or CIND diagnoses (N = 898). Multinomial logistic regression was applied in a longitudinal subsample (n = 354) to account for the competing risk of death when examining incident dementia/CIND.

Results: Of 898 participants, 13% had a diagnosis of dementia and 15% had CIND. Cross-sectional risk factors included age, lower educational attainment, stroke, head injury with loss of consciousness, epilepsy, no obesity and antidepressant use. Hearing impairment, urinary incontinence, mobility impairment and difficulties with certain activities of daily living were associated with a greater risk of incident dementia/CIND.

Conclusions: This study identified several factors associated with dementia and CIND among Aboriginal and Torres Strait Islander peoples. Findings from this observational study may support clinical awareness and inform future research; however, causal relationships cannot be inferred. Further longitudinal studies are needed to clarify these associations.

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Background

Dementia is a global public health challenge projected to affect 152 million people worldwide by 2050, driven by population ageing [1]. There is an urgent need to reduce dementia incidence, delay its onset and enhance care and support for affected individuals, families, and communities. The 2024 report of the Lancet Commission on dementia prevention, intervention, and care proposed that at the population level, up to 45% of dementia may be preventable through targeting 14 modifiable risk factors over the life-course [2]. These include improving educational attainment, reducing certain lifestyle and environmental exposures (smoking, excessive alcohol consumption, physical inactivity, social isolation and air pollution) and management of key health conditions (hearing impairment, hypertension, obesity, diabetes, depression, traumatic brain injury, high cholesterol and vision loss) [2].

In Australia, dementia prevalence is three to five times higher among Aboriginal and Torres Strait Islander peoples compared to non-Indigenous Australians. The estimated population attributable fraction (PAF) for dementia based on 11 modifiable risk factors for Aboriginal and Torres Strait Islander peoples is 44.9% and higher than for people of European (36.4%) or Asian ancestry (33.6%) [3]. Similarly, the dementia PAF based on these modifiable risk factors was higher for First Nations people compared to the non-Indigenous population in Canada and New Zealand [4,5]. The similar patterns of prevalence of risk factors in these developed countries with parallel histories of colonialism, dispossession, trauma and socioeconomic adversity, highlight that many of the modifiable risk factors for dementia coalesce around social inequalities that disproportionately affect First Nations people.

Older age, head injury and stroke are well established risk factors associated with all-cause dementia in different Aboriginal and Torres Strait Islander communities around Australia [6–8]. In two longitudinal studies of urban and regional communities in New South Wales and remote communities in the Kimberley region of Western Australia, older age and stroke remained as risk factors for incident dementia or cognitive impairment after adjustment [9,10]. While many factors are associated with dementia in individual studies, few are significant across studies limiting guidance for population-level interventions. Moreover, vascular risk factors such as diabetes, hypertension and cardiac disease were not associated with dementia in any of the cohorts, possibly due to the ubiquity of these conditions, non-linear risk relationships, premature mortality or insufficient statistical power.

Alzheimer's disease (AD) was the main dementia subtype diagnosis for participants in existing cohort studies [11–13]. However, Alzheimer's dementia in older adults frequently involves mixed pathologies, with vascular risk factors playing an increasing role in disease phenotype and progression [14,15]. The contributions of vascular and metabolic risk factors beyond stroke were not readily apparent in studies of Aboriginal and Torres Strait Islander peoples to date and warrant further delineation.

Greater understanding of common and unique determinants of cognitive health and ageing will assist in the promotion of healthy ageing, and the development of risk reduction and prevention strategies that are culture and context appropriate. The primary aim of this study was to elucidate cross-sectional and longitudinal risk and protective factors associated with dementia and Cognitive Impairment Not Dementia (CIND) in a harmonised dataset involving Aboriginal and Torres Strait Islander participants. It was expected that data harmonisation across three landmark cohort studies would offer greater insights and strengthen knowledge to guide brain health promotion and dementia reduction strategies for Aboriginal and Torres Strait Islander peoples.

Methods

The cross-sectional harmonised dataset

The Kimberley Healthy Adults Project (KHAP), Koori Growing Old Well Study (KGOWS) and the Torres Strait Dementia Prevalence Study

(TSDPS) were population ageing studies involving Aboriginal and Torres Strait Islander participants and local community-controlled service partners from different parts of Australia. These studies used structured interview protocols to collect information on demographic, medical, cultural and psychosocial factors from participants and family carers where available. The cross-sectional dataset was created through harmonisation of data from wave 2 (W2) of the KHAP, and wave 1 (W1) of the KGOWS and the TPDS (see Appendix A). The data from W2 of the KHAP was chosen for harmonisation as collected variables aligned more closely with data collected in the KGOWS and TSDPS.

Risk and protective variables associated with dementia chosen for exploration were also well established in the literature and available in all three cohorts. Variables of interest were recoded to be consistent across datasets prior to harmonisation into a single final dataset. Data harmonisation utilised the best available information from patient self-reports and corroboration from family reports, medications and primary care software-derived medical history and medications where available and appropriate. Harmonised variables across the three cohorts encompassed demographics (age, sex, residential location, education); health conditions (vision and hearing impairment, stroke, diabetes, hypertension, hyperlipidaemia, obesity, cardiac and kidney disease, urinary incontinence, mobility issues, falls, head injury with loss of consciousness [LOC], epilepsy, depression); lifestyle factors (alcohol use, smoking); and functional abilities (ADLs: dressing, cooking, cleaning). Questions related to alcohol use were assigned scores based on the short-form of the Alcohol Use Disorders Identification Test (AUDIT-C) [16] and allowed KHAP and TSDPS cohort participants' past and current drinking patterns to be classified as 'no alcohol', 'low-risk alcohol use' or 'high-risk alcohol use' for comparison with the KGOWS cohort. Medication lists available for the participants were coded using the World Health Organization's Anatomical Therapeutic Chemical Classification to allow for sub-analyses of outcomes in relation to medication categories of interest such as anticholinergics, analgesics, and anti-psychotics [17]. Polypharmacy was defined as use of five or more medications. Full details of variables of interest in the harmonised dataset are provided in the published protocol paper [18].

The longitudinal harmonised dataset

Longitudinal follow-up data was available for participants in the KHAP (Wave 2: 2011–2013) and KGOWS (Wave 2: 2016–2018) studies but not for the TSDPS at the time of this study (see Appendix B). This smaller longitudinal dataset differed in the granularity and categorisation of demographic, health, lifestyle, and functional variables and omitted several key distinctions captured in the more comprehensive cross-sectional dataset. For example, educational attainment was limited to a binary classification of "some schooling" versus "no schooling," omitting finer distinctions such as primary, secondary, or further education. Medication data were unavailable in KHAP Wave 1, preventing the inclusion of specific medication classes to supplement health-related variables. Functional status was assessed via core ADLs (feeding, dressing, toileting, and cleaning) but was simplified to a binary indicator of "having difficulty" versus "no difficulty," and did not include instrumental activities of daily living such as cooking. The recoding of variables and data harmonisation processes are detailed elsewhere [18] and in Appendices C and D.

Outcome measures

Consensus diagnoses assigned to participants in the individual cohort studies were based on data obtained through comprehensive medical assessment performed by a specialist physician with expertise in dementia assessment. Consensus diagnoses included normal cognition, dementia (all cause) and CIND or Mild Cognitive Impairment (MCI). The CIND and MCI groups demonstrated a decrement in

cognitive functioning that met DSM-IV-TR criteria for a cognitive or amnesic disorder but without significant loss in ADLs and not attributable to a psychiatric illness or intellectual disability. In the harmonised dataset, MCI diagnoses in the KGOWS cohort were reclassified as CIND based on DSM IV-TR diagnoses in line with the other cohorts for analyses. Participants given a consensus diagnosis of dementia were further categorised as Alzheimer's type, vascular dementia, dementia not otherwise specified (NOS) and dementia due to other cause based on the DSM IV-TR. Access to neuroimaging to support clinical diagnoses and subtyping of dementia was limited, especially in remote areas.

Ethics and governance

Ethics approval for the sharing of de-identified data to facilitate the harmonisation of the three cohort studies was obtained from the Aboriginal Health and Medical Research Council (1362/18), the Western Australian Aboriginal Health Ethics Committee (WAAHEC 858), the University of Melbourne Human Research Ethics Committee (12140), the Far North Queensland Human Research Ethics Committee (FNQHREC/2020/QCH/63123–1432), and James Cook University Human Research Ethics Committee (H9079). Prior to data sharing, a Memorandum of Understanding and a Statistical Analysis Plan were registered on the Open Science Framework platform (<https://osf.io/jv6sx/>). This research was conducted under the oversight and guidance of the Indigenous Reference Group from the Let's CHAT (Community Health Approaches To) Dementia in Aboriginal and Torres Strait Islander Communities project and the Community Reference Group from the On TRACK (Teaching, Research, and Community Knowledge) research programs, as outlined in the study protocol [19].

Data analysis

Data were analysed using the Stata statistical package (StataCorp, College Station, Texas), version 17. Descriptive statistics were used to describe the harmonised cross-sectional dataset (KHAP W2, KGOWS W1 and TSPDS, $N = 898$) and combined longitudinal cohort (KHAP W1/W2 and KGOWS-I/II, $N = 354$). Univariable and multivariable logistic regression was used to assess the associations between individual variables of interest and outcomes of dementia and/or CIND. Univariate analyses were conducted as an exploratory step to identify candidate variables for inclusion in multivariable models. Subgroup analyses explored whether being on medication for diabetes, hypertension, or hyperlipidaemia influenced associations with dementia or CIND outcomes. Results were reported as odds ratios (OR) with 95% confidence intervals (95% CI). Multivariable binary logistic regression was used to adjust for the effect of confounding variables of age, sex, education and study cohort. Variables that were significant in univariable analyses were entered into a multivariable model. Participants with missing covariate data were excluded and multivariable logistic regression was conducted using complete-case analysis ($n = 731$). In longitudinal analyses, multinomial logistic regression was used to account for the competing risk of death and estimate associations between variables of interest in three groups: 1) those who neither developed cognitive impairment nor dementia nor died; 2) those who developed dementia from normal or CIND states or progressed from normal to CIND during the study (incident CIND/dementia) and 3) those who died between baseline and follow-up. Results were reported as Relative Risk Ratios (RRR) with 95% CI. The level of statistical significance was set at $p < 0.05$.

Results

Demographics of the cross-sectional harmonised cohort

The harmonised cross-sectional dataset comprised 898 participants, with mean age of 64.4 (SD 9.8) years, with 60% female. One hundred

and thirteen participants (13%) had a consensus diagnosis of dementia and 244 (27%) were diagnosed with dementia or CIND. Over 62% resided in remote communities compared to 21% in regional and 15% from urban locations. Nearly half of the participants had some secondary schooling with 23% going on to further education. Vascular comorbidities including diabetes (54%), hypertension (65%), hyperlipidaemia (44%) and obesity (47%) were common among participants. Polypharmacy was present for half of the cohort with the most common prescribed medication classes being antihypertensives (70.2%), lipid lowering (52%), diabetic (43%) and antithrombotic medications (50%). Approximately one-third (35%) of the cohort identified as current alcohol consumers and 26% as current smokers. The proportion of participants who were classified as current high-risk alcohol consumers was lower than those deemed to have been high-risk consumers in the past (13% versus 36%). Around 22% of participants described a history of depression or current depressive symptoms but only 8% were on an antidepressant. Demographic and clinical characteristics of the harmonised study cohort by consensus diagnoses are provided in Table 1.

With the exception of obesity, the proportion of participants with missing data was low for potential risk and protective factors of interest, ranging from 0% to 6.7% (Table 1). AD was the most common dementia subtype assigned based on the DSM IV classification and accounted for 38% of dementia diagnoses with vascular dementia accounting for 16% of dementia diagnoses (see Table 2).

Cross-sectional factors associated with dementia and/or CIND

In the cross-sectional harmonised dataset, factors associated with an increased likelihood of dementia included older age, hearing impairment, history of stroke, diabetes, head injury with LOC, epilepsy, and the use of antidepressant, antipsychotic, and analgesic medications. Additionally, comorbid conditions such as mobility impairment, urinary incontinence, and limitations in ADLs requiring assistance with dressing, cooking, or cleaning were also associated with higher odds of dementia. Conversely, higher educational attainment beyond primary schooling, alcohol consumption, and obesity were associated with a lower likelihood of dementia in univariable analyses.

In multivariable analyses, age, education, history of stroke, head injury with LOC, epilepsy, and antidepressant medication use remained significantly associated with dementia outcomes, after excluding comorbid conditions such as mobility impairment, urinary incontinence and functional impairments (see Table 3).

Diabetes was associated with a greater odds of dementia in univariable analysis (OR 1.62; 95% CI: 1.06–2.48). However, its statistical significance was attenuated after adjusting for age and in multivariable analyses. In subgroup analyses, participants with diabetes who were not on treatment had a greater odds of dementia (OR = 2.07; 95% CI: 1.17–3.67) than those on medications for diabetes (OR = 1.46; 95% CI: 0.92–2.32) when compared to individuals without diabetes. After adjusting for age, individuals with diabetes on medication had lower odds of dementia (OR = 1.35; 95% CI: 0.83–2.21) compared to those with untreated diabetes (OR = 1.90; 95% CI: 1.01–3.56), relative to individuals without diabetes.

Subgroup analyses revealed a two-fold increased odds of non-AD compared to AD associated with stroke (OR = 4.56; 95% CI: 2.50–8.29 versus OR = 1.91; 95% CI: 0.91–4.02) and diabetes (OR = 2.45; 95% CI: 1.28–4.70 versus OR = 1.28; 95% CI: 0.67–2.45). In the non-AD group, individuals with untreated diabetes had a higher likelihood of dementia (OR = 4.32; 95% CI: 2.02–9.26) compared to those without diabetes, whereas the association was weaker among those receiving treatment (OR = 1.82; 95% CI: 0.89–3.72). Comorbid conditions including urinary incontinence, mobility impairment, falls and ADL impairments were strongly associated with non-AD dementia compared to AD dementia (see Appendix E).

When CIND was added to dementia outcomes, renal disease (OR = 1.45; 95% CI: 1.05–2.00), polypharmacy (OR = 1.48; 95% CI:

Table 1

Baseline characteristics of the harmonised cross-sectional dataset.

	Total (N = 898)	By diagnosis		
		Normal (N = 654)	CIND (N = 131)	Dementia (N = 113)
Age (years)	64.4 (9.8)	62.1 (8.9)	68.2 (8.9)	73.4 (9.1)
Female sex	541 (60.2%)	399 (61.0%)	80 (61.1%)	62 (54.9%)
Male sex	357 (39.8%)	255 (39.0%)	51 (38.9%)	51 (45.1%)
Locality <i>Urban</i>	140 (15.6%)	106 (16.2%)	15 (11.5%)	19 (16.8%)
<i>Regional</i>	196 (21.8%)	147 (22.5%)	23 (17.6%)	26 (23.0%)
<i>Remote</i>	562 (62.6%)	401 (61.3%)	93 (71.0%)	68 (60.2%)
Education ²⁸ <i>No formal schooling</i>	68 (7.8%)	37 (5.7%)	11 (8.8%)	20 (20.8%)
<i>Primary schooling</i>	187 (21.5%)	111 (17.1%)	46 (36.8%)	30 (31.2%)
<i>Secondary schooling</i>	410 (47.1%)	330 (50.8%)	42 (33.6%)	38 (39.6%)
<i>Further education</i>	205 (23.6%)	171 (26.3%)	26 (20.8%)	8 (8.3%)
Vision impairment ³⁹	237 (27.6%)	175 (27.3%)	35 (28.5%)	27 (28.4%)
Hearing impairment ²²	166 (18.9%)	112 (17.3%)	26 (20.3%)	28 (27.5%)
Stroke ²¹	138 (15.7%)	76 (11.8%)	24 (18.5%)	38 (36.9%)
Diabetes ¹⁹ <i>no medication</i>	127 (14.4%)	85 (13.2%)	20 (15.4%)	22 (21.2%)
<i>on medication</i>	349 (39.7%)	254 (39.4%)	50 (38.5%)	45 (43.3%)
Hypertension ²⁵ <i>no medication</i>	79 (9.0%)	61 (9.5%)	12 (9.2%)	6 (5.9%)
<i>on medication</i>	485 (55.6%)	351 (54.7%)	78 (60.0%)	56 (55.4%)
Hyperlipidaemia ⁴ <i>no medication</i>	63 (7.4%)	49 (7.8%)	10 (7.8%)	4 (4.1%)
<i>on medication</i>	315 (36.9%)	222 (35.3%)	52 (40.6%)	41 (42.3%)
Obesity ⁶⁴	389 (46.6%)	314 (51.1%)	44 (35.5%)	31 (32.6%)
Cardiac disease ¹⁹	290 (33.0%)	208 (32.2%)	49 (37.7%)	33 (31.7%)
Renal disease ²²	245 (28.0%)	167 (25.9%)	43 (33.1%)	35 (34.3%)
Urinary incontinence ³⁸	223 (25.9%)	139 (22.0%)	35 (28.2%)	49 (47.1%)
Mobility impairment ³⁴	319 (36.9%)	195 (30.7%)	60 (48.4%)	64 (61.5%)
Falls ⁴⁹ <i>1 fall</i>	152 (17.9%)	106 (16.6%)	28 (23.0%)	18 (20.0%)
<i>2 or more falls</i>	94 (11.1%)	64 (10.0%)	20 (16.4%)	10 (11.1%)
Head injury with LOC ⁴¹	247 (28.8%)	168 (26.4%)	40 (32.5%)	39 (40.2%)
Epilepsy ¹³	43 (4.9%)	21 (3.3%)	10 (7.6%)	12 (11.0%)
Depression ¹⁴ <i>no medication</i>	117 (13.2%)	100 (15.6%)	9 (6.9%)	8 (7.3%)
<i>on medication</i>	73 (8.3%)	44 (6.8%)	10 (7.6%)	19 (17.3%)
Polypharmacy ⁶⁰	423 (50.5%)	288 (47.8%)	73 (56.2%)	62 (59.0%)
Medications ⁶⁰ <i>antidepressants</i>	73 (8.7%)	44 (7.3%)	10 (7.7%)	19 (18.1%)
<i>anticholinergics</i>	28 (3.3%)	19 (3.2%)	3 (2.3%)	6 (5.7%)
<i>analgesia</i>	251 (30.0%)	166 (27.5%)	42 (32.3%)	43 (41.0%)
<i>opioid</i>	49 (5.8%)	35 (5.8%)	6 (4.6%)	8 (7.6%)
<i>antipsychotic</i>	18 (2.1%)	10 (1.7%)	1 (0.8%)	7 (6.7%)
<i>benzodiazepines and other sedatives</i>	25 (3.0%)	17 (2.8%)	2 (1.5%)	6 (5.7%)
<i>antithrombotics</i>	419 (50.0%)	287 (47.6%)	73 (56.2%)	59 (56.2%)
<i>antihypertensives</i>	588 (70.2%)	415 (68.8%)	99 (76.2%)	74 (70.5%)
<i>lipid lowering medications</i>	438 (52.3%)	313 (51.9%)	71 (54.6%)	54 (51.4%)
<i>diabetic medications</i>	360 (43.0%)	258 (42.8%)	54 (41.5%)	48 (45.7%)
<i>thyroid medications</i>	49 (5.8%)	33 (5.5%)	8 (6.2%)	8 (7.6%)
Alcohol status ⁶⁰ <i>No alcohol</i>	220 (25.4%)	138 (21.8%)	42 (32.8%)	40 (38.1%)
<i>Ex-alcohol</i>	340 (39.3%)	243 (38.4%)	52 (40.6%)	45 (42.9%)
<i>Current alcohol</i>	306 (35.3%)	252 (39.8%)	34 (26.6%)	20 (19.0%)
Current alcohol ²⁸ <i>Abstinent</i>	565 (65.0%)	385 (60.5%)	94 (73.4%)	86 (81.1%)
<i>Low risk</i>	194 (22.3%)	155 (24.4%)	27 (21.1%)	12 (11.3%)
<i>High risk</i>	110 (12.7%)	95 (15.0%)	7 (5.5%)	8 (7.5%)
Past alcohol ⁴² <i>Abstinent</i>	265 (31.1%)	179 (28.6%)	45 (35.4%)	41 (39.8%)
<i>Low risk</i>	281 (32.9%)	212 (33.9%)	46 (36.2%)	23 (22.8%)
<i>High risk</i>	307 (36.0%)	234 (37.4%)	36 (28.3%)	37 (36.6%)
Smoking status ⁴⁷ <i>Never smoked</i>	261 (30.7%)	189 (30.0%)	36 (28.8%)	36 (37.5%)
<i>Ex-smoker</i>	366 (43.0%)	262 (41.6%)	65 (52.0%)	39 (40.6%)
<i>Current smoker</i>	224 (26.3%)	179 (28.4%)	24 (19.2%)	21 (21.9%)
Cooking ³⁷ <i>Independent</i>	656 (76.2%)	544 (85.9%)	92 (74.2%)	20 (19.2%)
<i>Assistance</i>	205 (23.8%)	89 (14.1%)	32 (25.8%)	84 (80.8%)
Cleaning ³⁶ <i>Independent</i>	556 (64.5%)	469 (73.9%)	71 (57.3%)	16 (15.5%)
<i>Assistance</i>	306 (35.5%)	166 (26.1%)	53 (42.7%)	87 (84.5%)
Dressing ³³ <i>Independent</i>	796 (92.0%)	625 (98.4%)	118 (95.2%)	53 (50.0%)
<i>Assistance</i>	69 (8.0%)	10 (1.6%)	6 (4.8%)	53 (50.0%)
Assistance required in any ADL ³²	330 (38.1%)	177 (27.8%)	59 (47.6%)	94 (88.7%)

Notes: ADL = activities of daily living; LOC = loss of consciousness; SD = standard deviation.

Superscript numerals denote number of participants with missing data.

Continuous variables are reported with their mean and standard deviation and categorical variables as number and percentages.

1.09–2.00), antithrombotic medications (OR = 1.41; 95% CI: 1.04–1.91), and falls (OR = 1.61; 95% CI: 1.00–2.59) were also significantly associated with greater odds of dementia and CIND in univariable analyses, as described in Table 4.

The longitudinal cohort

The harmonised longitudinal dataset (KHAP and KGOWS) comprised 354 study participants (239 with normal cognition, 52 with

Table 2
Dementia subtypes based on DSM-IV criteria.

Dementia subtype based on the DSM-IV	Participants	Percent (%)
Dementia of Alzheimer's type	43	38
Vascular dementia	18	16
Dementia NOS	34	30
Dementia due to other causes	18	16
	113	100

Notes: DSM-IV = Diagnostic and Statistical Manual, fourth edition; NOS = not otherwise specified

CIND, 63 with dementia) and 177 who had died at wave 2. In assessed participants, those with dementia were significantly older, with a mean age of 68.2 ± 6.9 years, compared to 61.4 ± 9.5 years for those without dementia. Gender distribution indicated a higher proportion of females in the non-dementia group (61.5%) compared to the incident dementia group (52.8%). Additionally, a greater percentage of individuals with incident dementia resided in regional areas (35.8%) compared to those without dementia (24.9%). While most participants reported some schooling, 26.4% of the incident dementia group had no formal education, compared to 18.6% in the non-dementia group. The mean total KICA score for the cohort was $36.9 (\pm 3.4)$, with a mean score of $35.4 (\pm 4.4)$ in the incident dementia group versus $37.1 (\pm 3.1)$ in those who did not develop dementia. The baseline characteristics of participants who were followed up longitudinally are provided in Table 5.

Factors associated with incident dementia or incident dementia/CIND

After excluding those with dementia at baseline, 53 participants developed incident dementia, 86 participants had incident dementia/CIND, and 121 died prior to any known CIND or dementia. In unadjusted analyses, factors associated with incident dementia included age (OR = 1.08; 95% CI: 1.05–1.12), hearing impairment (OR = 3.40; 95% CI: 1.75–6.60), and requiring assistance with cleaning (OR = 2.43; 95% CI: 1.29–4.58). Residing in a remote rather than urban location was associated with a lower odds of incident dementia (OR = 0.42; 95% CI: 0.20–0.88), whereas there was no difference between urban and regional settings. When the outcomes included individuals with CIND, the same factors remained significant except for remote residence; impairment in any ADL was also significant (see Table 6).

Multinomial logistic regression analyses accounting for the competing risk of death identified increasing age (RRR = 1.09; 95% CI: 1.06–1.12) and hearing impairment (RRR = 2.96; 95% CI: 1.58–5.54) as significant risk factors for incident dementia/CIND. Educational attainment was protective, with individuals reporting some schooling exhibiting a reduced risk of incident dementia/CIND (RRR = 0.46; 95% CI: 0.25–0.83). Functional difficulties significantly correlated with incident dementia/CIND, including urinary incontinence (RRR = 1.67; 95% CI: 0.84–3.32), mobility impairment (RRR = 1.76; 95% CI: 1.05–2.96), and any difficulty with ADLs (RRR = 2.32; 95% CI: 1.39–3.86), particularly with cleaning (RRR = 2.88; 95% CI: 1.57–5.29), as described in Table 7.

Discussion

Our harmonised cross-sectional analysis identified several factors associated with increased odds of dementia as shown in Fig. 1. In fully adjusted cross-sectional models that excluded comorbid conditions (such as mobility impairment, urinary incontinence, and functional impairments in dressing, cleaning, and cooking), older age, stroke, head injury with LOC, epilepsy, and antidepressant use remained significantly associated with increased dementia risk. Education and obesity continued to be associated with lower odds of dementia. While

midlife obesity is widely recognised as a risk factor for dementia, the observed inverse association between obesity and dementia risk in this study may reflect reverse causality, wherein weight loss and frailty are more prevalent among individuals with cognitive impairment [20,21].

The relationship between alcohol consumption and dementia risk was complex. In univariable analyses, alcohol use was associated with lower odds of dementia. However, after adjusting for age and other covariates, high-risk alcohol consumption showed a non-significant trend toward increased dementia risk, while low-risk alcohol use was associated with lower odds of dementia. These findings are consistent with existing literature highlighting the effects of alcohol consumption on cognitive health and dementia risk [2].

Diabetes, cardiac disease, kidney disease, hyperlipidaemia and hypertension were associated with increased odds of all-cause dementia, albeit without reaching statistical significance. Diabetes was associated with a 62% increase in the odds of dementia, with a 61% difference between those on treatment compared to those untreated. This relationship between diabetes and treatment was amplified in the non-AD group. Thirty percent of dementia diagnoses were unable to be categorised further (dementia NOS) and are likely to include those with mixed pathologies.

HbA1c data was insufficient for harmonisation preventing differentiation between participants with well controlled diabetes and those whose condition is not optimised. We also analysed diabetic treatments as a whole and cannot comment on the potential benefits of certain types of medications. However, the lower odds of dementia in those on treatment compared to those not on treatment, even accounting for individuals not requiring medication, suggest that treating diabetes reduces the risk of dementia, particularly for those with vascular or mixed aetiologies. This aligns with the wider literature where medication treatment has been linked with slower cognitive decline and lower dementia risk in older people with diabetes [22].

Multimorbidity among Aboriginal Australians contributes to a higher risk of suboptimal prescribing, including polypharmacy, and is associated with increased medication-related complications and adverse health outcomes [23,24]. In our study, polypharmacy was highly prevalent (50%) and within the range of 43–53% reported in cohort studies and analyses of primary care prescribing patterns in older Aboriginal adults [24–27].

After adjusting for age, the use of antidepressants, antipsychotics, benzodiazepines, and other sedatives was associated with increased odds of dementia. Antidepressant use, in particular, was linked to a five-fold increase in dementia risk, which may reflect either the pharmacological effects of these medications or the severity of underlying depressive symptoms. Although anticholinergic burden (ACB) was not directly assessed in our dataset, previous analyses of the KGOWS cohort reported that 12% of participants had a significant ACB score (≥ 3). Mate et al. identified tricyclic antidepressants and other anticholinergic medications as contributors to negative health outcomes, including cognitive impairment, depression and hospitalisation within the previous 12 months [25]. However, one third of participants with a significant ACB score accumulated this burden from the additive effects of multiple medications. These findings underscore the importance of improving prescribing practices for Aboriginal people as they age, to reduce the risk of cognitive impairment, dementia, and other adverse outcomes associated with high-risk medications, or the cumulative burden of multiple medications.

Age, hearing impairment and functional impairments in cleaning were associated with higher risk of incident dementia in the longitudinal dataset. While an association between male sex and increased dementia risk was previously reported in both the KHAP and KGOWS cohorts, this trend did not reach statistical significance in our harmonised dataset. Biological female sex has traditionally been associated with a higher risk of dementia in many epidemiological studies however, a recent systematic review on dementia incidence and prevalence stratified by sex found no difference in incidence between men and

Table 3

Cross-sectional factors associated with dementia.

	Unadjusted	Age adjusted	Multivariable analyses*	
Factor	OR (95% CI)	OR (95% CI)	OR (95% CI)	p value
Age (per year)	1.12 (1.1–1.15)	-	1.14 (1.10–1.19)	< 0.001
Sex (male)	1.29 (0.87–1.92)	1.41 (0.92–2.18)		
Location <i>Urban</i>	REF			
<i>Regional</i>	0.97 (0.52–1.84)	0.98 (0.50–1.92)		
<i>Remote</i>	0.88 (0.51–1.51)	0.89 (0.50–1.61)		
Education <i>No schooling</i>	REF			
<i>Primary schooling</i>	0.46 (0.24–0.88)	0.51 (0.25–1.03)	0.34 (0.14–0.81)	0.041
<i>Secondary schooling</i>	0.25 (0.13–0.46)	0.53 (0.27–1.04)	0.43 (0.19–0.95)	
<i>Further education</i>	0.1 (0.04–0.23)	0.25 (0.10–0.63)	0.25 (0.085–0.75)	
Vision impairment	1.05 (0.65–1.68)	0.92 (0.55–1.54)		
Hearing impairment	1.74 (1.09–2.80)	1.09 (0.64–1.84)		
Stroke	3.94 (2.51–6.19)	4.34 (2.63–7.14)	3.06 (1.61–5.83)	0.001
Diabetes	1.62 (1.06–2.48)	1.49 (0.94–2.35)		
Diabetes, no medication	2.07 (1.17–3.67)	1.90 (1.01–3.56)		
Diabetes, on medication	1.46 (0.92–2.32)	1.35 (0.83–2.21)		
Hypertension	0.86 (0.56–1.31)	0.64 (0.40–1.03)		
Hypertension, no medication	0.57 (0.23–1.40)	0.50 (0.19–1.30)		
Hypertension, on medication	0.90 (0.58–1.40)	0.67 (0.41–1.08)		
Hyperlipidaemia	1.1 (0.72–1.68)	1.05 (0.66–1.65)		
Hyperlipidaemia, no medication	0.55 (0.19–1.58)	0.46 (0.15–1.39)		
Hyperlipidaemia, on medication	1.22 (0.79–1.89)	1.19 (0.74–1.90)		
Obesity	0.52 (0.33–0.81)	0.64 (0.40–1.04)	0.54 (0.29–0.98)	0.041
Cardiac disease	0.94 (0.60–1.45)	0.73 (0.45–1.17)		
Renal disease	1.4 (0.91–2.17)	1.14 (0.70–1.84)		
Urinary incontinence	2.98 (1.96–4.54)	2.87 (1.81–4.55)	-	
Mobility impairment	3.17 (2.08–4.84)	2.27 (1.44–3.59)	-	
Falls <i>No falls</i>	REF			
<i>1 fall</i>	1.17 (0.67–2.05)	1.03 (0.56–1.89)		
<i>2 or more falls</i>	1.04 (0.51–2.11)	1.05 (0.50–2.20)		
Head injury with LOC	1.78 (1.15–2.76)	2.48 (1.53–4.02)	2.43 (1.31–4.51)	0.005
Epilepsy	2.97 (1.48–5.98)	4.71 (2.17–10.25)	3.84 (1.22–12.12)	0.022
Depression	1.22 (0.76–1.95)	2.20 (1.30–3.74)		
Depression, no medication	0.54 (0.25–1.15)	1.06 (0.48–2.38)		
Depression, on medication	2.59 (1.46–4.58)	4.12 (2.16–7.85)		
Polypharmacy	1.49 (0.98–2.25)	1.22 (0.78–1.91)		
Medication <i>antidepressant</i>	2.78 (1.57–4.91)	4.14 (2.19–7.86)	5.25 (2.38–11.58)	< 0.001
<i>anticholinergic</i>	1.96 (0.78–4.95)	2.39 (0.86–6.64)		
<i>analgesia</i>	1.75 (1.15–2.67)	1.55 (0.98–2.45)		
<i>opioid</i>	1.39 (0.63–3.06)	1.62 (0.69–3.84)		
<i>antipsychotic</i>	4.69 (1.78–12.38)	6.24 (1.98–19.73)		
<i>benzodiazepines and other sedatives</i>	2.28 (0.89–5.84)	3.14 (1.16–8.50)		
<i>antithrombotic</i>	1.33 (0.88–2.01)	0.94 (0.60–1.48)		
<i>antihypertensive</i>	1.02 (0.65–1.59)	0.70 (0.43–1.15)		
<i>lipid lowering medication</i>	0.96 (0.64–1.45)	1.00 (0.64–1.55)		
<i>diabetic medication</i>	1.14 (0.75–1.71)	1.08 (0.70–1.69)		
<i>thyroid medication</i>	1.39 (0.63–3.06)	1.06 (0.45–2.50)		
Alcohol status <i>No alcohol</i>	REF			
<i>Ex-alcohol</i>	0.69 (0.43–1.09)	1.05 (0.63–1.76)		
<i>Current alcohol</i>	0.31 (0.18–0.56)	0.76 (0.40–1.43)		
Current alcohol <i>No alcohol</i>	REF			
<i>Low risk</i>	0.37 (0.20–0.69)	0.58 (0.30–1.12)		
<i>High risk</i>	0.44 (0.21–0.93)	1.35 (0.59–3.08)		
Past alcohol <i>No alcohol</i>	REF			
<i>Low risk</i>	0.49 (0.28–0.84)	0.76 (0.41–1.41)		
<i>High risk</i>	0.75 (0.46–1.21)	1.41 (0.84–2.39)		
Smoking status <i>Never smoked</i>	REF			
<i>Ex-smoker</i>	0.75 (0.46–1.21)	1.01 (0.59–1.73)		
<i>Current smoker</i>	0.65 (0.37–1.14)	1.41 (0.74–2.68)		
Dressing <i>Independent</i>	REF			
<i>Assistance</i>	46.44 (24.86–86.73)	35.97 (18.26–70.85)	-	
Cooking <i>Independent</i>	REF			
<i>Assistance</i>	22.08 (13.06–37.32)	15.89 (9.20–27.44)	-	
Cleaning <i>Independent</i>	REF			
<i>Assistance</i>	13.41 (7.69–23.37)	8.36 (4.70–14.88)	-	
Requiring assistance in any ADL	17.39 (9.35–32.34)	11.09 (5.86–20.99)		

Note: Multivariable analyses were adjusted for statistically significant variables in univariable analyses but excluded comorbid syndromes often associated with dementia (urinary incontinence, mobility impairment, ADL impairments).

Sample size reflects the analytic cohort included in the multivariable model (n = 731).

women for any dementia (AD and VaD) [28]. Meta-regression analyses from the same review suggest that observed sex differences in dementia prevalence are largely attributable to differences in life expectancy and

gendered life experiences, particularly those influencing educational attainment and potentially contributing to downstream socioeconomic sequelae [28]. However, further research is required to more clearly

Table 4

Cross-sectional factors associated with dementia and CIND.

	Unadjusted	Age adjusted	Multivariable analyses*	
Factor	<i>OR (95% CI)</i>	<i>OR (95% CI)</i>	<i>OR (95% CI)</i>	<i>p value</i>
Age (per year)	1.11 (1.09–1.13)	-	1.09 (1.07–1.12)	< 0.001
Sex (male)	1.12 (0.83–1.52)	1.18 (0.85–1.63)		
Locality <i>Urban</i>	REF			
<i>Regional</i>	1.04 (0.63–1.72)	1.04 (0.61–1.76)		
<i>Remote</i>	1.25 (0.82–1.92)	1.57 (0.99–2.47)		
Education <i>No schooling</i>	REF			
<i>Primary schooling</i>	0.82 (0.47–1.43)	0.95 (0.53–1.73)	0.97 (0.51–1.86)	0.02
<i>Secondary schooling</i>	0.29 (0.17–0.49)	0.53 (0.30–0.95)	0.49 (0.26–0.93)	
<i>Further education</i>	0.24 (0.13–0.43)	0.54 (0.28–1.05)	0.64 (0.31–1.30)	
Vision impairment	1.06 (0.75–1.49)	1.01 (0.69–1.46)		
Hearing impairment	1.46 (1.01–2.11)	0.99 (0.66–1.48)		
Stroke	2.71 (1.86–3.95)	2.66 (1.77–4.00)	2.35 (1.45–3.83)	0.001
Diabetes	1.27 (0.94–1.73)	1.17 (0.84–1.62)		
Diabetes, no medication	1.56 (1.01–2.41)	1.51 (0.93–2.45)		
Diabetes, on medication	1.18 (0.85–1.64)	1.07 (0.75–1.52)		
Hypertension	1.07 (0.78–1.47)	0.85 (0.60–1.21)		
Hypertension, no medication	0.86 (0.48–1.54)	0.79 (0.42–1.48)		
Hypertension, on medication	1.11 (0.80–1.54)	0.86 (0.60–1.23)		
Hyperlipidaemia	1.20 (0.88–1.63)	1.11 (0.80–1.55)		
Hyperlipidaemia, no medication	0.87 (0.46–1.63)	0.74 (0.37–1.45)		
Hyperlipidaemia, on medication	1.27 (0.92–1.75)	1.20 (0.85–1.70)		
Obesity	0.50 (0.36–0.69)	0.54 (0.38–0.76)	0.53 (0.36–0.78)	0.001
Cardiac disease	1.13 (0.83–1.55)	0.90 (0.64–1.27)		
Renal disease	1.45 (1.05–2.00)	1.30 (0.91–1.86)		
Urinary incontinence	2.07 (1.49–2.87)	2.03 (1.41–2.91)		
Mobility impairment	2.70 (1.98–3.68)	2.19 (1.57–3.07)		
Falls <i>no falls</i>	REF			
<i>1 fall</i>	1.49 (1.00–2.21)	1.47 (0.95–2.26)		
<i>2 or more falls</i>	1.61 (1.00–2.59)	1.71 (1.02–2.86)		
Head injury with LOC	1.56 (1.13–2.17)	1.97 (1.37–2.81)	1.96 (1.31–2.93)	0.001
Epilepsy	3.00 (1.62–5.56)	4.13 (2.13–8.01)	3.56 (1.59–7.95)	
Depression	0.82 (0.56–1.18)	1.18 (0.79–1.77)		
Depression, no medication	0.44 (0.25–0.75)	0.68 (0.38–1.20)		
Depression, on medication	1.69 (1.03–2.77)	2.16 (1.26–3.70)		
Polypharmacy	1.48 (1.09–2.00)	1.25 (0.90–1.73)		
Medications <i>antidepressant</i>	1.79 (1.09–2.93)	2.19 (1.28–3.73)	1.95 (1.05–3.61)	0.03
<i>anticholinergic</i>	1.22 (0.55–2.75)	1.33 (0.55–3.18)		
<i>analgesia</i>	1.49 (1.08–2.06)	1.36 (0.96–1.93)		
<i>opioid</i>	1.03 (0.54–1.95)	1.12 (0.56–2.25)		
<i>antipsychotic</i>	2.09 (0.81–5.36)	2.37 (0.81–6.96)		
<i>benzodiazepines and other sedatives</i>	1.21 (0.52–2.85)	1.36 (0.56–3.33)		
<i>antithrombotic</i>	1.41 (1.04–1.91)	1.06 (0.76–1.48)		
<i>antihypertensive</i>	1.26 (0.90–1.77)	0.95 (0.66–1.38)		
<i>lipid lowering medication</i>	1.05 (0.78–1.42)	1.04 (0.75–1.45)		
<i>diabetic medication</i>	1.03 (0.76–1.39)	0.94 (0.68–1.31)		
<i>thyroid medication</i>	1.26 (0.68–2.34)	1.00 (0.51–1.96)		
Alcohol status <i>No alcohol</i>	REF			
<i>Ex-alcohol</i>	0.67 (0.47–0.96)	0.92 (0.61–1.36)		
<i>Current alcohol</i>	0.36 (0.24–0.54)	0.68 (0.44–1.07)		
Current alcohol <i>No alcohol</i>	REF			
<i>Low risk</i>	0.54 (0.36–0.80)	0.73 (0.48–1.10)		
<i>High risk</i>	0.34 (0.19–0.60)	0.76 (0.41–1.40)		
Past alcohol <i>No alcohol</i>	REF			
<i>Low risk</i>	0.54 (0.35–0.83)	0.88 (0.57–1.37)		
<i>High risk</i>	0.74 (0.53–1.04)	1.09 (0.74–1.61)		
Smoking status <i>Never smoked</i>	REF			
<i>Ex-smoker</i>	1.04 (0.73–1.48)	1.30 (0.88–1.92)		
<i>Current smoker</i>	0.66 (0.43–1.01)	1.12 (0.70–1.80)		
Dressing <i>Independent</i>	REF			
<i>Assistance</i>	14.16 (6.39–31.38)	9.70 (4.23–22.22)		
<i>Dependent</i>	51.17 (12.07–216.94)	34.86 (7.88–154.17)		
Cooking <i>Independent</i>	REF			
<i>Assistance</i>	1.57 (0.94–2.63)	1.27 (0.74–2.19)		
<i>Dependent</i>	25.10 (14.56–43.24)	17.13 (9.70–30.27)		
Cleaning <i>Independent</i>	REF			
<i>Assistance</i>	2.13 (1.43–3.17)	1.45 (0.95–2.20)		
<i>Dependent</i>	12.97 (8.30–20.26)	8.55 (5.30–13.80)		
Any ADL <i>Independent</i>	REF			
<i>Assistance</i>	2.19 (1.46–3.28)	1.54 (1.00–2.36)		
<i>Dependent</i>	14.98 (9.69–23.14)	10.10 (6.35–16.04)		

Note: Multivariable analyses were adjusted for statistically significant variables in univariable analyses but excluded comorbid syndromes often associated with dementia (urinary incontinence, mobility impairment, ADL impairments).

Sample size reflects the analytic cohort included in the multivariable model (n = 729).

Table 5

Baseline characteristics of the harmonised longitudinal dataset.

	Total W2 participants (N = 354)	No incident dementia at W2 (n = 301)	Incident dementia at W2 (n = 53)
Age	62.4 (9.5)	61.4 (9.5)	68.2 (6.9)
Female sex	213 (60.2%)	185 (61.5%)	28 (52.8%)
Urban locality	71 (20.1%)	56 (18.6%)	15 (28.3%)
Regional locality	94 (26.6%)	75 (24.9%)	19 (35.8%)
Remote locality	189 (53.4%)	170 (56.5%)	19 (35.8%)
Some schooling	284 (80.2%)	245 (81.4%)	39 (73.6%)
Vision impairment ⁷	136 (39.3%)	118 (39.9%)	18 (36.0%)
Hearing impairment ⁷	60 (17.3%)	42 (14.2%)	18 (36.0%)
Stroke ⁸	62 (18.0%)	53 (18.0%)	9 (18.0%)
Diabetes ¹¹	157 (45.6%)	134 (45.6%)	23 (46.0%)
Hypertension ¹⁷	196 (57.6%)	166 (57.0%)	30 (61.2%)
Heart disease ⁹	101 (29.4%)	85 (28.9%)	16 (32.0%)
Renal disease ¹¹	56 (16.3%)	47 (16.0%)	9 (17.6%)
Depression ⁸	38 (11.0%)	32 (10.8%)	6 (12.0%)
Head injury with LOC ⁷	159 (45.8%)	138 (46.6%)	21 (41.2%)
Polypharmacy ⁷	94 (27.0%)	80 (26.8%)	14 (28.0%)
Never alcohol ⁸	93 (26.8%)	76 (25.7%)	17 (33.3%)
Ex-alcohol	128 (36.9%)	110 (37.2%)	18 (35.3%)
Current alcohol	126 (36.3%)	110 (37.2%)	16 (31.4%)
Current alcohol low risk	66 (19.0%)	55 (18.5%)	11 (21.6%)
Current alcohol high risk	60 (17.2%)	55 (18.5%)	5 (9.8%)
Past alcohol low risk ⁸	63 (18.2%)	56 (18.9%)	7 (13.7%)
Past alcohol high risk	151 (43.5%)	128 (43.2%)	23 (45.1%)
Never smoker ¹¹	132 (38.4%)	108 (36.9%)	24 (47.1%)
Ex-smoker	112 (32.6%)	93 (31.7%)	19 (37.3%)
Current smoker	100 (29.1%)	92 (31.4%)	8 (15.7%)
Urinary incontinence ⁸	57 (16.4%)	51 (17.2%)	6 (11.8%)
Mobility impairment ⁷	119 (34.2%)	97 (32.7%)	22 (43.1%)
Fall ⁷	88 (25.4%)	73 (24.7%)	15 (29.4%)
ADL feeding difficulty ¹⁴	2 (0.6%)	2 (0.7%)	0 (0.0%)
ADL dressing ¹²	20 (5.8%)	18 (6.1%)	2 (3.8%)
ADL toileting ¹⁶	6 (1.8%)	6 (2.1%)	0 (0.0%)
ADL cleaning ¹⁴	75 (21.8%)	56 (19.2%)	19 (36.5%)

Notes: ADL = activities of daily living; CI = confidence interval; LOC = loss of consciousness; SD = standard deviations.

Superscript numerals denote number of participants with missing data.

Continuous variables are reported with their mean and standard deviation and categorical variables as number and percentages.

delineate this relationship, with careful consideration of the differential life-course experiences of men and women.

Hearing impairment is a well-recognised and important potentially modifiable risk factor for dementia that is particularly relevant to Aboriginal and Torres Strait Islander communities [29,30]. While hearing impairment was not statistically significant in adjusted cross-sectional analyses, it was associated with dementia in unadjusted models and remained independently associated with incident dementia and CIND in longitudinal analyses. The prevalence of otitis media is reported to range from 50% in urban areas to as high as 91% in some remote Northern Territory communities, with peak incidence occurring within the first 12 months of life [31,32]. When left untreated or inadequately managed, otitis media can progress to chronic suppurative otitis media and tympanic membrane perforation, leading to long-term conductive hearing loss [33].

In this study, nearly one in five participants self-reported hearing loss, consistent with the 2018–2019 National Aboriginal and Torres Strait Islander Health Survey, which found a 28% prevalence among those aged 55 years and older [34]. However, self-reported hearing impairment often underestimates the prevalence of objectively measured hearing loss in this population by approximately half [34]. This early-life, preventable condition can negatively impact cognitive and language development, potentially influencing long-term outcomes across the life course, including educational attainment, employment, and socioeconomic circumstances [35,36]. Addressing hearing impairment across the life span represents an important strategy for reducing dementia risk in Aboriginal and Torres Strait Islander communities.

Table 6

Factors associated with incident CIND and/or dementia.

	Incident Dementia	Incident Dementia/CIND
	OR (95% CI)	OR (95% CI)
Age (per year)	1.08 (1.05–1.12)	1.07 (1.04–1.10)
Male sex	1.42 (0.79–2.56)	1.19 (0.73–1.95)
Urban locality	REF	
Regional locality	0.95 (0.44–2.02)	0.94 (0.47–1.89)
Remote locality	0.42 (0.20–0.88)	0.81 (0.43–1.51)
Some schooling	0.64 (0.32–1.25)	0.54 (0.30–0.94)
Vision impairment ⁷	0.85 (0.46–1.59)	1.46 (0.89–2.41)
Hearing impairment ⁷	3.40 (1.75–6.60)	2.39 (1.32–4.33)
Stroke ⁸	1.00 (0.46–2.19)	1.03 (0.54–1.96)
Diabetes ¹¹	1.02 (0.56–1.86)	0.75 (0.45–1.24)
Hypertension ¹⁷	1.19 (0.64–2.21)	1.22 (0.73–2.03)
Heart disease ⁹	1.16 (0.61–2.21)	1.48 (0.87–2.51)
Renal disease ¹¹	1.12 (0.51–2.46)	1.21 (0.63–2.32)
Depression ⁸	1.12 (0.44–2.84)	0.99 (0.45–2.20)
Head injury with LOC ⁷	0.80 (0.44–1.46)	1.06 (0.65–1.74)
Polypharmacy ⁷	1.06 (0.54–2.07)	0.77 (0.43–1.37)
Alcohol status ⁸ <i>abstinent</i>	REF	
<i>ex-alcohol</i>	0.73 (0.35–1.01)	0.68 (0.37–1.25)
<i>current alcohol</i>	0.65 (0.31–1.37)	0.60 (0.33–1.12)
Current alcohol ⁸ <i>abstinent</i>	REF	
<i>low risk</i>	1.07 (0.51–2.24)	0.78 (0.40–1.51)
<i>high risk</i>	0.49 (0.18–1.30)	0.72 (0.36–1.46)
Past alcohol ⁸ <i>abstinent</i>	REF	
<i>low risk</i>	0.67 (0.27–1.66)	0.57 (0.27–1.21)
<i>high risk</i>	0.96 (0.50–1.82)	0.84 (0.49–1.44)
Smoking ¹¹ <i>never</i>	REF	
<i>ex-smoker</i>	0.92 (0.47–1.78)	0.77 (0.43–1.38)
<i>current smoker</i>	0.39 (0.17–0.91)	0.71 (0.38–1.31)
Urinary incontinence ⁸	0.64 (0.26–1.58)	1.17 (0.61–2.23)
Mobility impairment ⁷	1.56 (0.85–2.86)	1.57 (0.95–2.61)
Fall ⁷	1.27 (0.66–2.45)	1.48 (0.86–2.55)
ADL feeding difficulty ¹⁴	1	
ADL dressing ¹²	0.61 (0.14–2.72)	0.79 (0.26–2.44)
ADL toileting ¹⁶	1	
ADL cleaning ¹⁴	2.43 (1.29–4.58)	1.86 (1.06–3.26)
Any ADL impairment	1.70 (0.93–3.09)	1.80 (1.09–2.96)

Notes: ADL = activities of daily living; CI = confidence interval; LOC = loss of consciousness; OR = odds ratio; RRR = relative risk ratio.

In our longitudinal analyses, having some formal schooling was associated with reduced risk of incident dementia or CIND, though not dementia alone. This may reflect that conventional measures of education attainment may insufficiently capture cognitive reserve and resilience within this population [10]. In previously published work for the KGOWS, when history of unskilled work was added to models with education, it remained significant for an increased risk of incident MCI/dementia but reduced the original effect of education [10]. Similarly, in a large longitudinal study of octogenarians in New Zealand, some secondary education was found to be protective and associated with better annual cognitive function among non-Māori participants, but not among Māori [37]. The authors suggested that bilingualism, present in 52% of the Māori cohort, may act as a potential modifier. Likewise, Suchy-Dacey et al. (2022) examined the relationship between educational attainment and cognitive as well as brain-imaging outcomes in First Nations people of North America, finding only modest correlations between years of formal education and cognitive test scores [38]. These findings suggest that conventional measures of education primarily capture formal or Western schooling, whereas cognitive reserve may also be influenced by additional dimensions including bilingualism, culturally embedded knowledge, and competencies acquired through informal or skilled work experience.

Longitudinal Australian studies have reported lower AD risk in outer regional and communities compared to major cities, and aligns with international evidence from the UK [39–41], France [42] and Spain [43]. The Lancet Commission may partly attribute this elevated

Table 7

Unadjusted association of variables of interest with incident dementia/CIND in multinomial logistic regression analyses.

	Incident Dementia/CIND (n = 86)	Death preceding any dementia or CIND (n = 121)
	RRR (95% CI)	RRR (95% CI)
Age (per year)	1.09 (1.06–1.12)	1.07 (1.04–1.09)
Male Sex	1.31 (0.79–2.17)	1.96 (1.25–3.05)
Location <i>urban</i>	REF	
<i>regional</i>	1.00 (0.49–2.04)	1.33 (0.64–2.77)
<i>remote</i>	0.78 (0.41–1.47)	1.67 (0.89–3.16)
Education <i>no schooling</i>		
<i>some schooling</i>	0.46 (0.25–0.83)	0.39 (0.23–0.65)
Vision impairment	1.47 (0.88–2.44)	1.46 (0.93–2.28)
Hearing impairment	2.96 (1.58–5.54)	1.92 (1.05–3.50)
Stroke	1.25 (0.64–2.44)	1.41 (0.80–2.51)
Diabetes	0.73 (0.44–1.23)	1.04 (0.66–1.62)
Hypertension	1.17 (0.70–1.97)	1.24 (0.78–1.96)
Heart disease	1.55 (0.91–2.67)	1.52 (0.94–2.45)
Renal disease	1.17 (0.60–2.26)	1.35 (0.76–2.38)
Depression	1.02 (0.46–2.29)	1.39 (0.72–2.70)
Head injury	1.19 (0.72–1.97)	1.02 (0.65–1.60)
Polypharmacy	0.87 (0.48–1.57)	1.75 (1.08–2.82)
Alcohol status <i>abstinent</i>	REF	
<i>ex-alcohol</i>	0.57 (0.31–1.07)	1.17 (0.65–2.10)
<i>current alcohol</i>	0.49 (0.26–0.92)	0.84 (0.46–1.55)
Current alcohol <i>abstinent</i>	REF	
<i>low risk</i>	0.71 (0.36–1.39)	0.63 (0.34–1.17)
<i>high risk</i>	0.65 (0.32–1.32)	0.91 (0.51–1.61)
Past alcohol <i>abstinent</i>	REF	
<i>low risk</i>	0.51 (0.24–1.08)	0.89 (0.47–1.68)
<i>high risk</i>	0.80 (0.46–1.39)	1.19 (0.72–1.97)
Smoking <i>never</i>	REF	
<i>ex-smoker</i>	0.74 (0.41–1.35)	0.85 (0.49–1.46)
<i>current smoker</i>	0.64 (0.34–1.19)	0.92 (0.54–1.58)
Urinary incontinence	1.67 (0.84–3.32)	2.14 (1.18–3.86)
Mobility impairment	1.76 (1.05–2.96)	3.06 (1.93–4.85)
Fall	1.61 (0.92–2.81)	1.66 (1.01–2.72)
ADL feeding difficulty	1	
ADL dressing difficulty	1.62 (0.46–5.68)	4.95 (1.98–12.41)
ADL toileting difficulty	1	
ADL cleaning difficulty	2.88 (1.57–5.29)	3.28 (1.91–5.65)
Any ADL difficulty	2.32 (1.39–3.86)	2.69 (1.70–4.24)

Notes: ADL = activities of daily living; CIND = cognitive impairment, not dementia; RRR = relative risk ratio. *Neither CIND, dementia, nor death was used as the reference outcome.

dementia risk in urban areas to higher levels of air pollution, particularly fine particulate matter ($\leq 2.5 \mu\text{m}$ and $\leq 10 \mu\text{m}$ in diameter) [2]. However, most studies linking air pollution to incident dementia have been conducted in regions with moderate to high air pollution levels. In contrast, a large cohort study of older men in an Australian urban setting with low ambient air pollution found no association between pollution exposure and dementia incidence [44]. Research is needed to explore other environmental contributors such as noise, access to green space, and pesticide exposure that may underlie the observed differences in dementia risk by geographic location [45]. In this study the association between remote residence and lower risk of dementia was not significantly associated with incident dementia/CIND after accounting for the competing risk of death.

Strengths and limitations

While acknowledging the importance of diversity and locally driven solutions, data harmonisation enables the integration of individual participant data from multiple studies, offering broader perspectives and understanding of complex diseases.

This study is observational, comprising cross-sectional analyses and correlational longitudinal follow-up. As such, causal relationships between identified risk factors and cognitive outcomes cannot be inferred. Associations may be influenced by unmeasured variables that affect both exposure and outcome or residual confounding, despite multivariable adjustment.

This study is limited to variables available across all cohorts. Notably, information on physical activity – a significant potentially modifiable risk factor in Aboriginal and Torres Strait Islander populations – was absent [3]. Data on social determinants of well-being, resilience constructs, and genotyping were inconsistently available, limiting our ability to expand on findings from KGOWS, which identified associations between childhood trauma, social isolation, unskilled work history, and APOE $\epsilon 4$ with dementia risk [7,10]. Potentially protective cultural determinants were also unable to be assessed.

Given the large number of variables examined in univariate analyses, there is an increased risk of Type I error due to multiple comparisons. Although the proportion of missing data for individual variables was low, complete-case analysis resulted in a reduced analytic sample. This approach may introduce bias if data are not missing completely at random (MCAR).

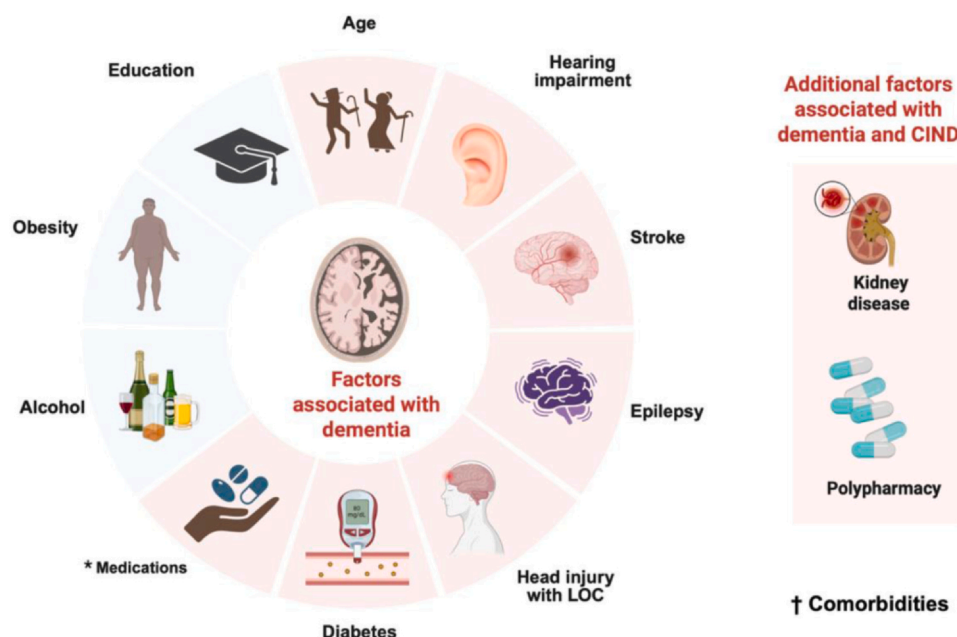


Fig. 1. Factors associated with dementia and/or CIND. Notes: CIND = cognitive impairment, not dementia; LOC = loss of consciousness. Factors highlighted in red indicate increased odds of dementia; blue indicates decreased odds (based on univariable analyses). *Medication classes significantly associated with dementia include antidepressants, analgesics, and antipsychotics. †Comorbidities significant in univariable analyses: mobility impairment, urinary incontinence, and ADL impairments (dressing, cooking, cleaning). Renal disease, polypharmacy, and antithrombotics were associated with both dementia and CIND outcomes. Created with BioRender.com, based on a template by Liv Nordestgaard and Samara Ona.

Some risk factor data may represent late-life rather than midlife exposures, potentially affecting their relevance in dementia prevention strategies. The retrospective nature of risk factor data collection also introduces the possibility of recall bias. To mitigate this limitation, we reference the most relevant literature to contextualise our findings.

Conclusion

This study reinforced important risk factors for dementia for Aboriginal and Torres Strait Islander peoples but was limited in the factors examined. Future research would benefit from standardised protocols that capture both globally recognised dementia risk and protective factors, as well as cultural determinants uniquely relevant to Aboriginal and Torres Strait Islander communities. Longitudinal studies with integration of comprehensive biomedical, psychosocial, and lifestyle data with physical, biomarker, and imaging measures, which follow cohorts from midlife over a longer period of time are needed.

CRediT authorship contribution statement

Robert Cumming: Writing – review & editing, Methodology, Conceptualization. **Kylie Radford:** Writing – review & editing, Writing – original draft, Resources, Methodology, Data curation, Conceptualization. **Louise Lavrencic:** Writing – review & editing, Data curation, Conceptualization. **Leon Flicker:** Writing – review & editing, Writing – original draft, Resources, Methodology, Conceptualization. **Bridgette J. McNamara:** Writing – review & editing, Supervision, Methodology, Conceptualization. **Rachel Quigley:** Writing – review & editing, Methodology, Data curation, Conceptualization. **Jo-anne Hughson:** Writing – review & editing, Project administration, Conceptualization. **Sarah Russell:** Writing – review & editing, Methodology, Data curation, Conceptualization. **Edward Strivens:** Writing – review & editing, Methodology, Conceptualization. **Dina LoGiudice:** Writing – review & editing, Supervision, Methodology, Funding acquisition, Data curation, Conceptualization. **Betty Sagigi:** Writing – review & editing, Resources. **Alison Timbery:** Writing – review & editing, Data curation. **Zoë Hyde:** Writing – review & editing, Supervision, Resources, Methodology, Formal analysis, Data curation, Conceptualization. **Adrienne Withall:** Writing – review & editing, Methodology. **Huong Xuan Thi Nguyen:** Writing – review & editing, Writing – original draft, Visualization, Methodology, Investigation, Funding acquisition, Conceptualization. **Kim Delbaere:** Writing – review & editing, Methodology. **Roslyn Malay:** Writing – review & editing, Resources. **Brian Draper:** Writing – review & editing, Methodology. **Kate Smith:** Writing – review & editing, Resources, Data curation. **Terence Donovan:** Data curation. **Rosie Watson:** Writing – review & editing, Supervision, Methodology, Conceptualization.

Consent Statement

Consent was not necessary.

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Conflicts

The authors report no disclosures relevant to the manuscript. This study has received ethics approval from the Aboriginal Health and Medical Research Council (AHMRC), Western Australian Aboriginal Health Ethics Committee (WAAHEC), University of Melbourne and Far

North Queensland and James Cook University Human Research Ethics Committees. Informed consent was obtained from all subjects and/or their legal guardians in the original research. This research was carried out in accordance with the key principles for ethical conduct in research with Aboriginal and Torres Strait Islander peoples based on NHMRC and AHMRC guidelines.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at [doi:10.1016/j.inpsyc.2026.100206](https://doi.org/10.1016/j.inpsyc.2026.100206).

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