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Original Research

Incident dementia risk by frailty status and living arrangements in older adults

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ABSTRACT

Background: Frailty and social isolation are independently associated with dementia risk. This study examined whether frailty predicts dementia differently for older adults living alone versus with others.
Methods: Participants were 967 community-dwelling, dementia-free older adults from the Sydney Memory and Ageing Study (2005–2020). Frailty was assessed using an adapted Fried Frailty Phenotype (robust, pre-frail, frail). Dementia was diagnosed via biennial neuropsychological testing and clinical consensus. Cox regressions analysed associations between baseline frailty, living arrangements (alone vs. with others), and incident dementia over 12 years.
Result: Frail individuals had 1.74 times higher dementia risk than robust individuals (HR = 1.74, 95% CI: 1.16–2.60, $p = .007$). Living alone was not independently associated with dementia (HR = 1.03, $p = .825$). The interaction between frailty and living arrangements was non-significant (HR = 1.23, $p = 0.600$). Exploratory stratified analyses showed frailty predicted dementia among those living alone (HR = 1.92, 95% CI: 1.06–3.51, $p = .033$) but not those living with others (HR = 1.42, $p = 0.224$).
Conclusion: Frailty is a significant predictor of dementia risk in older adults. While the formal interaction test was non-significant, exploratory findings suggest frail individuals living alone may be particularly vulnerable, warranting further investigation and potential targeting for combined physical and social interventions, pending replication.

1. Introduction

The global population is ageing, driving a rise in dementia and subsequent social and health challenges. Over 55 million people live with dementia currently, projected to increase to 140 million by 2050 [1]. As limited disease modifying pharmacological therapy is available, primary prevention and early detection are critical. Attention has turned toward modifiable risk factors. Frailty is one such factor, with meta-analyses demonstrating that physical frailty increases dementia risk by 50–60% and that preventing frailty could reduce dementia incidence by 14.2% [2,3]. Similarly, social isolation has been associated with a 30% increased dementia risk in population-based studies [4].

Frailty is a clinical syndrome characterised by declining physiological reserve, associated with age related changes. It results in an increased vulnerability to stressors and a higher risk of adverse health outcomes such as falls, disability, cognitive impairment and mortality [2,5]. Frailty exists on a continuum with pre-frailty being a reversible intermediate state between healthy ageing and frailty. There are several tools commonly used to assess frailty across various domains including physical, psychological and social. One tool is the Fried Frailty Phenotype (FP) [6], which captures physical frailty, meaning it is not confounded by the inclusion of social and psychological domains of frailty. The FP defines frailty as a distinct clinical entity based on five domains: 1) unintentional weight loss, 2) self-reported exhaustion, 3) weakness, 4) slow walking speed, and 5) low physical activity.

Frailty is associated with worse cognitive performance and clinical diagnoses of dementia cross-sectionally [5] and steeper cognitive decline and increased dementia risk longitudinally [2]. Whilst this relationship may be bidirectional, most current literature examines frailty as a predictor of cognitive decline rather than the reverse [7]. A

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better understanding of these associations is warranted to disentangle the relationship between frailty and cognitive decline.

Social isolation accounts for 5% of the population attributable risk of dementia worldwide [8]. Social isolation (a lack of integration in a social environment) and loneliness (the feeling of being alone) are distinct concepts that are weakly correlated, and independently associated with cognitive decline [9,10]. Living alone is often used as a proxy measure for social isolation [11,12], and has been linked to physical and psychosocial vulnerabilities that lead to a higher prevalence of chronic diseases, including dementia [13]. It is estimated that 8.9% of incident dementia cases in those aged 65 and above are associated with living alone [11].

There is increasing evidence that socially isolated individuals and those living alone have a higher risk of developing frailty and other adverse health outcomes [13,14]. Further, living alone is more frequent in those with co-existent frailty and cognitive decline and social connection may reduce the risk of dementia [15]. However, longitudinal studies examining the potential mitigating effect of living with others on cognitive decline in frail older individuals are sparse.

Social bridging and social bonding hypotheses have been proposed as potential mechanisms driving the association between frailty and adverse health outcomes in older adults [16]. Social bridging suggests that exposure to novel and diverse social stimuli could be cognitively enriching, whilst social bonding suggests that close and supportive connections could reduce the harmful effects of stress on the brain. Individuals living alone may lack both.

Whilst it is well established that both frailty and living alone are associated with dementia risk, the effect of living alone on the longitudinal association between frailty and dementia remains unknown. To our knowledge, no studies have investigated whether living with others affects the risk of incident dementia in robust, pre-frail and frail individuals over time.

This study aimed to examine the associations between baseline frailty status and living arrangements with incident dementia over 12 years in a well-characterised, sample of older Australians, and whether the associations between frailty and dementia were similar in individuals living alone compared to those living with others. We hypothesised that living alone and frailty status would be associated with increased dementia risk, and that this association would be stronger for frail individuals living alone.

2. Methods

2.1. Participants

The data for this study were drawn from the Sydney Memory and Ageing Study (MAS), a longitudinal cohort study conducted from 2005 to 2020 [17]. The baseline sample consisted of 1037 community-dwelling, dementia-free older adults aged between 70 and 90 years, recruited from two electorates in Eastern Sydney. Baseline and follow-up assessments ("Waves") were conducted biennially, and included a detailed neuropsychological test battery, medical examination, and lifestyle questionnaires. For the current analysis, 70 participants were excluded due to missing baseline frailty data, resulting in a final sample of 967. See Supplementary Fig. 1 for study flow diagram. The study was approved by the Human Research Ethics Committee of UNSW Sydney (HC: 05,037, 09,382, 14,327, 190,962).

2.2. Predictor variables

2.2.1. Frailty

To utilise the MAS dataset, some minor adaptations were made to the FP [6]; which was originally coded dichotomously across five categories: 1) unintentional weight loss, 2) self-reported exhaustion, 3) weakness, 4) slow walking speed, and 5) low physical activity [6]. Unintentional weight loss was approximated using Body Mass Index (BMI) $\leq$18.5 due to unavailability of weight change data at baseline; exhaustion was determined by one question from the Geriatric Depression Scale ("Do you feel full of energy?") [18]; weakness was defined by the slowest 20% to complete 5 repetitions of transitioning from sitting to standing; slow walking speed was defined as the slowest 20% to complete a 6-metre walk; and low physical activity was defined as the least 20% of minutes per week spent on physical activity, weighted by intensity (see Supplementary Table 1). Scores ranged from 0 to 5, and participants were assigned a status of robust (FP score = 0), pre-frail (FP score 1 or 2), or frail (FP score $\geq$ 3).

2.2.2. Living arrangements

Baseline living arrangements were measured using two questions: 1) what type of accommodation participants lived in and 2) how many other people they reported living with. A binary variable was created from these data (see Supplementary Table 2), grouping participants into those who lived alone (0) or lived with others (1).

2.3. Outcome variable

Dementia diagnoses were made at each wave by a consensus panel comprised of neuropsychiatrists, neuropsychologists and old age psychiatrists according to the DSM-IV criteria [19]. Criteria were met if: 1) the participant was impaired in the domain of memory and one other cognitive domain, 2) their IADLs were impaired (i.e., Bayer-ADL score $\geq$ 3), and 3) they did not have a delirium or other medical conditions which could account for cognitive or functional impairments.

2.4. Covariates

Covariates considered for inclusion in the final analyses were sex, age, years of education, marital status (never married, married/de facto), non-English speaking background (NESB) status, Mini-Mental State Exam (MMSE; [20]) scores, apolipoprotein E4 (*APOE4)* carrier status, as well as other health variables shown to be associated with dementia: diabetes (present/not present), hypertension (present/not present), alcohol consumption (categorised based on frequency), smoking status (current, past or never), hearing aid use, depression symptoms (Geriatric Depression Scale; [18]) anxiety symptoms (Goldberg Anxiety Scale; [21]) and personality traits (NEO Five-Factor Inventory; [22]). See Supplementary Table 3.

2.5. Statistical analysis

Descriptive statistics were used to characterise baseline demographics, lifestyle and clinical variables across the robust, pre-frail and frail groups.

Cox regression analyses examined: (1) the main effect of frailty status on dementia risk, (2) the main effect of living arrangements, and (3) whether the frailty-dementia association differed by living arrangement. To further explore the interaction, we conducted follow-up stratified analyses by splitting the data according to living arrangements (alone vs. with others) and examining the association between frailty status and incident dementia within each group.

A time-to-event variable was created for incident dementia over 12 years. The association between baseline frailty status and dementia incidence was examined using log rank testing [18] and Kaplan-Meier (KM) curves. Cox Proportional Hazards Survival Regressions [18] were used to calculate the hazard ratios (HR) with 95% confidence interval (CI) for incident dementia over 12-years, with robust participants as the reference group. A forwards stepwise selection method, with a cut off $p \leq .100$, was used to determine covariates that would be included. The variables that met these criteria were sex, age, NESB status, MMSE scores, *APOE4* carrier status and smoking status.

Finally, to explore social support mechanisms underlying the relationships between living arrangements, physical frailty, and dementia

risk, post-hoc exploratory analyses were conducted using two baseline social variables: availability of a confidant (yes/no) and frequency of face-to-face contact (dichotomized to <5 vs. ≥5 contacts per month). These variables were examined descriptively across frailty strata and entered into exploratory Cox Proportional Hazards Models.

3. Results

3.1. Demographics

Baseline descriptive characteristics by FP status are presented in Table 1. Regarding social support mechanisms, availability of a confidant exhibited zero variance, with 100% of participants reporting having someone to confide in. Conversely, objective social contact significantly varied by physical health status; the proportion of individuals reporting low face-to-face social contact (<5 visits per month) increased progressively from 9.4% in the robust group to 14.0% in the pre-frail group, and 24.0% in the frail group ($p<.001$). Post-hoc analysis confirmed that the 70 excluded participants did not differ significantly from the analytic sample on key baseline characteristics, including age ($p = .800$), sex ($p = .920$), MMSE scores ($p = .600$), or living arrangements ($p = .309$); these data are presented in Supplementary Table 8.

3.2. Incident dementia and frailty status

An adjusted log rank test compared survival probabilities between robust, pre-frail and frail individuals. The survival distributions were statistically significantly different ($\chi^2 = 29.25$, $p<0.001$). Fig. 1 displays the Kaplan-Meier survival curves comparing cumulative survival probabilities over the 12-year study between frailty phenotypes. Censored data points represent participants who were lost to follow up, died or were dementia free at the end of the study.

Next, we ran a series of Cox survival analyses, controlling for all covariates, to examine the association between frailty status and dementia ($\chi^2 = 140.70$, $df = 2$, $p<.001$). Frail individuals had a 74% greater risk of incident dementia (HR = 1.74, 95%CI: 1.16 – 2.60, $p = .007$) compared to robust individuals. There was no significant difference in risk of incident dementia between pre-frail and robust individuals (HR = 0.93, 95%CI: 0.7 – 1.23, $p = .601$). These data are presented in Table 2.

3.3. Incident dementia and living arrangements

In the fully adjusted model that examined the association between living arrangements and dementia no significant associations were identified (HR = 1.03, 95%CI: 0.79–1.35, $p = .825$). These results are presented in Supplementary Table 4.

3.4. Interaction between frailty status and living arrangement

To test whether the association between frailty status and dementia risk differed by living arrangement, we examined the frailty × living arrangements interaction term in a Cox Regression Model adjusting for all selected covariates. The interaction term was not statistically significant (frail × living alone: HR = 1.23, 95% CI: 0.57–2.66, $p = .60$), indicating that the association between frailty and dementia risk did not significantly differ between those living alone and those living with others. These results are presented in Supplementary Table 5.

3.5. Exploratory stratified analyses by living arrangement

Given the potential clinical importance of understanding frailty-dementia associations across different living arrangements, we conducted exploratory stratified analyses examining the association between frailty status and incident dementia separately for those living alone versus with others. Whilst these exploratory stratified analyses suggested that frailty predicted dementia risk for those living alone but not for those living with others, the formal interaction term was not statistically significant ($p = .600$), this difference between groups should be interpreted with caution.

Among participants living alone, the overall association between frailty status and incident dementia over 12-year follow-up was not significant but suggestive of an association ($\chi^2 = 5.34$, $df = 2$, $p = .069$).

Individuals who lived alone and were classified as frail had a 92%

Table 1
Baseline variables as a function of frailty status.

Wave 1	Robust (n = 290)	Pre-frail (n = 537)	Frail (n = 140)	Total (n = 967)	p-value
Covariates *N (%)*					
Sex (female)	159 (54.8)	290 (54.0)	84 (60.0)	533 (55.1)	.400
Age *M (SD)*	**76.80 (4.35)**	**78.58 (4.84)**	**80.61 (4.56)**	**78.34 (4.81)**	**<.001**
Education years *M (SD)*	11.93 (3.44)	11.53 (3.49)	10.89 (3.15)	11.56 (3.44)	.006
Non-English speaking background status	47 (16.2)	86 (16.0)	19 (13.6)	152 (15.7)	.800
Marital status (married/de-facto)	**140 (48)**	**224 (42)**	**40 (29)**	**404 (42)**	**<.001**
APOE4 carrier status	65 (22.8)	117 (22.7)	31 (24.2)	213 (22.9)	>.900
MMSE *M (SD)*	28.73 (1.29)	28.74 (1.35)	28.56 (1.37)	28.71 (1.33)	.300
Diabetes	**99 (37.1)**	**196 (37.8)**	**78 (56.1)**	**373 (40.4)**	**<.001**
Hypertension	**233 (80.6)**	**468 (87.3)**	**131 (93.6)**	**832 (86.2)**	**<.001**
Smoking status					.400
Never	144 (50.2)	235 (44.0)	59 (42.1)	438 (45.6)	
Past	135 (47.1)	278 (52.1)	75 (53.6)	488 (50.9)	
Current	8 (2.8)	21 (3.9)	6 (4.3)	35 (3.6)	
Alcohol consumption frequency					.110
Not in the last year	28 (9.7)	70 (13.0)	21 (15.0)	119 (12.3)	
Monthly or less	44 (15.2)	79 (14.7)	31 (22.1)	154 (15.9)	
2–4 times a month	41 (14.1)	94 (17.5)	25 (17.9)	160 (16.5)	
2–3 times a week	42 (14.5)	70 (13.0)	16 (11.4)	128 (13.2)	
4–6 times a week	46 (15.9)	79 (14.7)	10 (7.1)	135 (14.0)	
Daily	89 (30.7)	145 (27.0)	37 (26.4)	271 (28.0)	
Uses hearing aid	46 (18.4)	115 (23.4)	38 (28.8)	199 (22.8)	.063
Geriatric Depression Scale *M (SD)*	**1.00 (1.24)**	**2.72 (2.08)**	**3.36 (2.28)**	**2.29 (2.09)**	**<.001**
Goldberg Anxiety Scale *M (SD)*	**0.78 (1.53)**	**1.25 (1.96)**	**1.25 (1.93)**	**1.11 (1.84)**	**<.001**
Neuroticism *M (SD)*	**13.25 (6.69)**	**16.27 (6.89)**	**17.01 (6.75)**	**15.43 (6.96)**	**<.001**
Openness *M (SD)*	**27.34 (6.06)**	**26.57 (6.11)**	**24.77 (5.08)**	**26.57 (6.02)**	**<.001**
Conscientiousness *M (SD)*	**35.47 (5.26)**	**33.58 (5.95)**	**31.83 (5.98)**	**33.93 (5.94)**	**<.001**
Available confidant	281 (100.0)	514 (100.0)	133 (100.0)	928 (100.0)	
<5 face-to-face contacts per month	**27 (9.4)**	**74 (14.1)**	**33 (24.1)**	**134 (14.1)**	**<.001**
Predictor Variables					
Living arrangements					.400
Living with others	162 (56.3)	279 (52.1)	70 (50.7)	511 (53.2)	
Living alone	126 (43.8)	256 (47.9)	68 (49.3)	450 (46.8)	

Note. M = mean; SD = standard deviation; APOE4 = apolipoprotein E4; MMSE = mini-mental state exam.

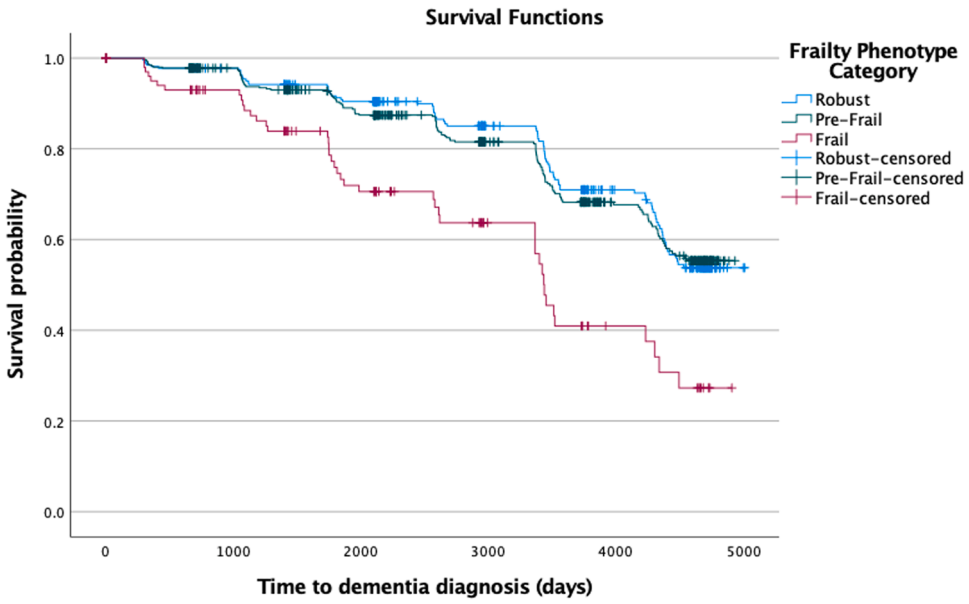

Fig. 1. Kaplan-Meier curves for incident dementia by frailty status.

Table 2
Adjusted Cox regression analysis examining the association between baseline frailty status and incident dementia.

								95% CI for *HR*	
		B	*SE*	Wald	*df*	*p*-value	*HR*	Lower	Upper
FP	Robust			**11.32**	**2**	**.033**			
	Pre-frail	-.08	.14	.27	1	.601	.93	.70	1.23
	Frail	**.55**	**.20**	**7.21**	**1**	**.007**	**1.74**	**1.16**	**2.60**
Sex		-.22	.13	2.77	1	.096	.81	.62	1.04
Age		**.11**	**.01**	**61.94**	**1**	**<.001**	**1.12**	**1.09**	**1.15**
NESB status		**.44**	**.16**	**7.15**	**1**	**.007**	**1.55**	**1.12**	**2.13**
APOE4 carrier status		**.56**	**.14**	**16.71**	**1**	**<.001**	**1.75**	**1.34**	**2.28**
MMSE		**-.15**	**.04**	**11.09**	**1**	**<.001**	**.86**	**.78**	**.94**
Smoking status	Never			5.36	2	.068			
	Past	-.06	.13	.17	1	.681	.95	.73	1.23
	Current	**.65**	**.31**	**4.58**	**1**	**.032**	**1.92**	**1.06**	**3.49**

Note. FP = Fried Frailty Phenotype; NESB = non-English speaking background; APOE4 = apolipoprotein E4; MMSE = mini-mental state exam.

greater risk of incident dementia (HR = 1.92, 95% CI: 1.06–3.51, p = .033) compared to robust individuals living alone. Pre-frail individuals living alone did not differ significantly in risk for incident dementia from robust individuals (HR = 1.08, 95% CI: 0.70–1.66, p = .724). These data are presented in Table 3; however, it should be noted that the overall association between frailty status and dementia in the living alone group was only suggestive (p = .069), and the significant HR for the frail subgroup (p = .033) should be interpreted cautiously given the risk of type 1 error in exploratory stratified analyses.

Conversely, for participants living with others, the overall

Table 3
Adjusted Cox regression analysis of the association between frailty status and incident dementia among participants living alone.

								95% CI for HR	
		B	*SE*	Wald	*df*	*p*-value	HR	Lower	Upper
Living alone									
	Sex	**-.50**	**.21**	**5.64**	**1**	**.018**	**.61**	**.40**	**.92**
	Age	**.12**	**.02**	**26.77**	**1**	**<.001**	**1.12**	**1.07**	**1.17**
	NESB status	**.48**	**.24**	**4.21**	**1**	**.040**	**1.62**	**1.02**	**2.56**
	APOE4 carrier status	**.70**	**.20**	**12.29**	**1**	**<.001**	**2.02**	**1.36**	**2.99**
	MMSE	-.15	.08	3.86	1	.050	.86	.74	1.00
Smoking status	Never			1.96	2	.376			
	Past	-.18	.20	.83	1	.361	.84	.57	1.23
	Current	.37	.43	.73	1	.392	1.45	.62	3.39
FP	Robust			5.34	2	.069			
	Pre-frail	.08	.22	.13	1	.724	1.08	.70	1.66
	Frail	**.65**	**.31**	**4.55**	**1**	**.033**	**1.92**	**1.06**	**3.51**

Note. NESB = non-English speaking background; APOE4 = apolipoprotein E4; MMSE = mini-mental state exam; FP = Fried Frailty Phenotype.

association between frailty status and incident dementia was not significant ($\chi^2 = 3.86$, $df = 2$, $p = .145$). Compared to robust individuals, the risk of incident dementia was not significantly different for frail participants (HR = 1.42, 95%CI: 0.81–2.52, $p = .224$) nor pre-frail participants (HR = 0.84, 95%CI: 0.57–1.23, $p = .367$), suggesting that living with others may mitigate the association between frailty and dementia risk. These results are presented in Table 4.

3.6. Sensitivity analyses

To examine whether broader aspects of social connection influenced the observed associations between frailty and incident dementia, post-hoc exploratory Cox Regression Models were conducted using frequency of face-to-face social contact. When examined independently alongside covariates, low face-to-face contact (<5 per month) was significantly associated with an increased risk of incident dementia. However, when the physical frailty phenotype was added to the same multivariable model, the effect of low face-to-face contact was attenuated and lost statistical significance, indicating substantial shared variance between increasing frailty and declining face-to-face social contact. An additional model incorporating a frailty and social contact interaction term did not identify a statistically significant interaction effect, suggesting limited evidence that social contact modified the association between frailty and dementia risk. These data are presented in Supplementary Tables 9–11.

4. Discussion

This study found that frailty is associated with incident dementia and the association is most evident among those living alone. To our knowledge, this is one of the first studies to examine the longitudinal relationships between frailty status, dementia and living arrangements over an extended time.

Our findings are consistent with current literature documenting the associations between frailty and cognitive decline both cross-sectionally [5] and longitudinally [2]. Despite pre-frailty being a precursor to frailty, pre-frail participants in this study did not have a significantly increased risk of incident dementia compared to robust participants, which is in line with some studies. For example, one study found that pre-frailty was not significantly associated with a higher risk of cognitive decline on a brief screening assessment over two years [23]. In contrast, more recent findings from the English Longitudinal Study of Ageing found pre-frail individuals had a 1.51 times increased risk of developing dementia compared to robust individuals [24] and a UK Biobank study found that pre-frailty contributed to a higher number of cases of dementia than frailty [25]. However, these cohorts were younger, suggesting the relationship between pre-frailty and dementia may change with age.

While we hypothesised that living alone would be associated with a greater risk of incident dementia, this was surprisingly not the case in the full sample, despite much of the literature suggesting that living alone predicts an increased risk of dementia [11,26]. However, one study demonstrated that living alone at baseline was not associated with increased risk of dementia and was inversely associated with risk of incident dementia in females [12]. This suggests that associations between living alone and dementia may be dependent on other factors such as age, sex, quantity or quality of social connections, or personality. When examining the association between frailty status and dementia risk in participants who lived alone versus with others, we found that frail individuals living alone were 92.3% more likely to develop dementia compared to robust individuals living alone. Frailty was not associated with an increased dementia risk among those living with others.

The formal interaction between frailty status and living arrangement was not statistically significant ($p = .600$), indicating that living arrangement did not significantly moderate the frailty-dementia association at the population level. Stratified analyses revealed that frailty predicted dementia risk for those living alone but not for those living with others, the observed pattern should be interpreted cautiously given the non-significant interaction term and exploratory nature of these analyses.

Post-hoc analyses help explain why no formal interaction between living alone and frailty was observed. Objective social isolation (less than 5 face-to-face contacts a month) tracked closely with increasing frailty, and the independent association between low contact and dementia was attenuated once frailty entered the model. As physical decline restricts social mobility and engagement, these two risk factors share substantial variance, therefore their combined impact is likely overlapping rather than multiplicative. No significant frailty × contact interaction was observed, providing limited evidence that social connectedness modified the frailty–dementia association in this cohort.

One plausible explanation involves competing mechanisms. The literature supports that social bridging (exposure to diverse social stimuli) and social bonding (formation of close relationships) are cognitively protective, underpinned by cognitive reserve and stress buffering hypotheses [11,27]. An alternative mechanism is that individuals experiencing early cognitive decline preferentially live with others for support. This could mask protective social effects, resulting in the null association between living arrangements and dementia risk.

In the living-alone group, frailty was associated with higher dementia risk, suggesting that without co-resident support, physical vulnerability may translate into cognitive risk. Longitudinal analyses tracking changes in living arrangements against cognitive trajectory would help disentangle these mechanisms. The association between

Table 4
Adjusted Cox regression analysis of the association between frailty status and incident dementia among participants living with others.

								95% CI for HR	
		B	*SE*	Wald	*df*	*p*-value	HR	Lower	Upper
Living with others									
	Sex	-.01	.19	.00	1	.976	.99	.69	1.44
	Age	**.12**	**.02**	**37.91**	**1**	**<.001**	**1.12**	**1.08**	**1.16**
	NESB status	.43	.23	3.48	1	.062	1.54	.98	2.43
	APOE4 carrier status	**.45**	**.19**	**5.53**	**1**	**.019**	**1.57**	**1.08**	**2.29**
	MMSE	**-.15**	**.06**	**6.58**	**1**	**.010**	**.86**	**.77**	**.97**
Smoking status	Never			5.36	2	.069			
	Past	.08	.19	.17	1	.676	1.08	.75	1.56
	Current	**1.01**	**.44**	**5.35**	**1**	**.021**	**2.75**	**1.17**	**6.48**
FP	Robust			3.86	2	.145			
	Pre-frail	-.18	.20	.81	1	.367	.84	.57	1.23
	Frail	.35	.29	1.48	1	.224	1.42	.81	2.51

Note. NESB = non-English speaking background; APOE4 = apolipoprotein E4; MMSE = mini-mental state exam; FP = Fried Frailty Phenotype.

living alone and frailty may be bidirectional [28]; cross-lagged analyses between living alone and frailty would help identify directionality of causal relationships. In this study, living arrangement was used as a baseline indicator of social context, at the time frailty was assessed. However, living arrangements may change over time due to widowhood, transition to residential aged care, or moving in with relatives or caregivers. Longitudinal analyses that model changes in living arrangements across time would help clarify how social context modifies the relationship between frailty and dementia risk.

This study has strengths including a large, well-characterised sample of community dwelling older adults who were followed over 12 years with extensive demographic and clinical information. This longitudinal follow-up is important given the progressive nature of dementia and its long pre-clinical stage [29]. The consensus diagnoses, which used comprehensive neuropsychological tests, is another strength as many studies rely on brief cognitive screening tools. The breadth of medical data allowed multiple modifiable risk factors to be controlled for, important given that frailty and dementia are multifactorial conditions [8].

The current study had some limitations. The baseline cohort was relatively homogenous, from two electorates in Eastern Sydney with high socioeconomic status. Thus, most participants (97.8%) were Caucasian, highly educated, and spoke fluent English, limiting generalisability. The study may have been subject to sampling bias as most participants had a knowledgeable informant, meaning the most socially isolated populations would not have been captured.

The FP was selected to avoid confounding between physical and social domains of frailty, given the study's focus on living arrangements as a social variable. However, frailty is a multidimensional construct encompassing physical, psychological and social domains, and a purely physical measure may not capture the interplay between these domains. Future research using a multidimensional frailty index may provide a more nuanced understanding of how physical and social vulnerability interact to predict dementia risk. Additionally, we used BMI $\leq$18.5 as a proxy for unintentional weight loss which may not accurately capture frailty, as a low, stable BMI may not reflect the same physiological decline as rapid unintentional loss, thus future studies should utilise weight change data at baseline. We also used living alone as a proxy for social isolation. Whilst often employed in the literature, this measure may be reductive, as some older adults may not experience social isolation when living alone, instead seeking connections from friends and family outside the home [12]. Additionally, living arrangements were assessed at baseline and may have changed over the 12-year follow-up period. Changes in living arrangements could influence both frailty status and dementia risk. As such, living arrangement should be interpreted as a baseline contextual factor rather than a time-varying exposure. Future studies should examine how changes in living arrangements affect the relationship between frailty and dementia. Finally, while 70 participants were excluded due to missing baseline frailty data, a missing data analysis showed no significant differences between excluded and included participants across key variables, suggesting their exclusion is unlikely to have introduced bias.

The findings of this study provide evidence that frailty is associated with increased risk of dementia and older adults living alone may be particularly vulnerable. Living alone is an easily identifiable, objective measure at both the individual and population level [7]. Therefore, future research should target pre-frail and frail older individuals who live alone and potentially investigate the efficacy of social interventions.

5. Conclusion

Frailty is predictive of incident dementia longitudinally in older adults. Living alone was not significantly associated with incident dementia in the full sample, and the interaction between frailty status and living arrangements was not statistically significant. However, exploratory stratified analyses revealed that frailty was significantly associated with dementia risk among participants living alone but not those living with others. While these patterns suggest that living with others may attenuate the frailty-dementia association, the non-significant interaction indicates that larger studies are needed to confirm whether living arrangements moderate this relationship. Nevertheless, identifying frail individuals who live alone is an important target in dementia prevention efforts.

Ethical approval statement and patient consent statement

The study was approved by the Human Research Ethics Committee of the University of New South Wales (HC: 05,037, 09,382, 14,327, 190,962). Written informed consent was obtained from all participants and informants.

Data statement

The terms of consent for research participation stipulate that an individual's data can only be shared outside of the Memory and Ageing Study investigators group if the group has reviewed and approved the proposed secondary use of the data. This consent applies regardless of whether data have been de-identified. Access is mediated via a standardised request process managed by the CHeBA Research Bank, who can be contacted at ChebaData@unsw.edu.au.

Declaration of generative AI use

We declare no use of generative AI and AI-assisted technologies in scientific writing and in Fig.s, images and artwork.

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CRediT authorship contribution statement

Kate McEwen: Writing – review & editing, Writing – original draft, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Suraj Samtani:** Writing – review & editing, Supervision, Methodology, Conceptualization. **Rory Chen:** Writing – review & editing, Supervision, Methodology, Formal analysis, Data curation, Conceptualization. **John D. Crawford:** Writing – review & editing, Formal analysis. **Perminder S. Sachdev:** Writing – review & editing, Project administration, Funding acquisition. **Henry Brodaty:** Writing – review & editing, Project administration, Funding acquisition. **Katya Numbers:** Writing – review & editing, Supervision, Investigation, Formal analysis, Data curation, Conceptualization.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests:

Henry Brodaty and Perminder S Sachdev reports a relationship with Biogen that includes: board membership and consulting or advisory. Henry Brodaty reports a relationship with Eisai Australia Pty Ltd that includes: board membership and consulting or advisory. Henry Brodaty and Perminder S Sachdev reports a relationship with Eli Lilly and Company that includes: board membership and consulting or advisory. Henry Brodaty reports a relationship with Medicines Australia that includes: board membership and consulting or advisory. Henry Brodaty and Perminder S Sachdev reports a relationship with Roche that includes: board membership and consulting or advisory. Henry Brodaty reports a relationship with Skin2Neuron that includes: board

membership and consulting or advisory. Henry Brodaty reports a relationship with Cranbrook Care Pty Ltd that includes: board membership and consulting or advisory. Perminder S Sachdev reports a relationship with Novo Nordisk Inc that includes: board membership and consulting or advisory. Kate McEwen has patent Creative Commons Attribution CC-BY license licensed to UNSW Intellectual Property Policy. If there are other authors, they 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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Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.tjfa.2026.100185.

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