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RESEARCH ARTICLE

Incidence of dementia and mild cognitive impairment in two Indigenous Bolivian cohorts

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Abstract

INTRODUCTION: We studied dementia incidence and risk factors in Indigenous Tsimane and Moseten Amerindian populations in Amazonian Bolivia with largely subsistence lifestyles.

METHODS: A median of 4.7 years after baseline dementia assessment, 730 participants (52% male; mean age 67.3 years) were re-visited at two follow-up phases to determine new cases among non-demented at baseline. Clinical evaluation was adapted to culture, language, and illiteracy.

RESULTS: There were 22 incident dementia cases (7.40/1000 person years; 95% confidence interval = 4.87–11.24). Prevalence rates remained < 2%. Mortality among dementia cases at baseline was significantly greater versus cognitively normal participants. In exploratory analyses, apolipoprotein E ε4 genotype, thoracic aortic calcium, and plasma neurofilament light chain were associated with incident dementia; other risk factors, amyloid, and phosphorylated tau were non-significant.

DISCUSSION: In these populations, dementia prevalence is strikingly low, reflecting modestly low incidence and shortened life expectancy after diagnosis. Next steps include delineating vascular contributions versus Alzheimer's disease pathology in dementia cases.

KEYWORDS
dementia, incidence, Indigenous, Moseten, Tsimane

Highlights

- Incidence and especially prevalence of dementia are low in Indigenous Bolivians.
- Dementia cases have shortened life expectancy, contributing to lower prevalence.
- Apolipoprotein E ε4 is a risk factor for incident dementia.
- A subsistence lifestyle with low vascular risk factors may be protective.
- Results may shed light on the evolution of dementia and its risk factors.

1 | BACKGROUND

Dementia is a global health concern, with unequal distribution across populations.[1] Compared to European or US studies, the reported prevalence of dementia and cognitive impairment in Indigenous groups is generally higher, particularly for those living in urban or peri-urban conditions.[2–4] In contrast, several other studies found comparatively low prevalence of dementia or of cognitive impairment in rural, low-literacy, low-acculturation, Indigenous populations.[5–7] Incidence rates for dementia for Indigenous populations have received little research attention. The few reports, mostly from Australia, suggest high incidence compared to non-Indigenous.[8,9]

Some high-income countries (HICs) have recently seen reduced incidence of dementia, attributed to population-level improvements in vascular risk factors and education.[10] Conversely, low- and middle-income countries (LMICs) anticipate, or already report, rising dementia rates, reflecting population aging combined with increased lifestyle-related risk factors such as midlife obesity.[1]

We previously reported low dementia prevalence in the Tsimane and Moseten Amerindian populations in Amazonian Bolivia (Phase 1).[5] We have now followed the cohort for two further assessment phases (Phase 2 and Phase 3) to assess incident dementia. Because prevalence reflects both incidence and survival, these follow-up assessments are needed to measure both incidence and survival after onset. Identifying incident cases also allows for testing risk factors for dementia, especially those distinctive to the Indigenous context. In particular, in the Tsimane and Moseten populations, vascular risk factors tend to be low and likely reflective of a high-fiber, low-sugar, low–saturated fat diet and high levels of physical activity.[11] However, there is high pathogen exposure, low availability of antibiotics, and nearly no vaccination.[12] Tuberculosis and infection from intestinal helminths remain widespread.[13]

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In our initial assessment of prevalence, our clinical impression was that dementia rarely presented as typical, amnesia-prominent, Alzheimer's disease (AD)—the most common etiology in HIC and European populations. Computed tomography (CT) scans, available for some cases, often showed marked medial calcification in the internal carotid, vertebral, and lenticulostriate arteries, with greater calcification volumes in those with dementia or mild cognitive impairment (MCI).[14] Less prominent were patterns of atrophy consistent with AD.[5] We now complement those observations with preliminary analyses of plasma biomarkers, newly available for a subset of individuals, enabling examination of amyloid beta (Aβ), phosphorylated tau (p-tau), and neurodegeneration (ATN) levels in these participants.[15]

This report aims to: (1) determine incidence of dementia and MCI in the same Tsimane and Moseten populations ≈ 5 years after their baseline assessment; (2) calculate prevalence at Phases 2 and 3; and (3) identify possible risk or protective exposures, biomarker correlates, patterns of mortality, or other factors to help characterize dementia and explain rates in these populations. We hypothesize lower incidence relative to European or US rates; however, we predict that shorter survival among those diagnosed with dementia will contribute to the observed low prevalence rate. We expect Phase 3 prevalence will be similar to Phase 2 prevalence. We predict that cardiometabolic indicators, lifestyle factors, and inflammation will be associated with the likelihood of developing dementia. We also test whether apolipoprotein E (*APOE*) genotypes predict incident dementia and whether ATN biomarkers are significantly associated with dementia status. We predict lower prevalence of ATN profiles that are prototypically associated with AD.

RESEARCH IN CONTEXT

1. **Systematic review**: We searched PubMed for reports in English of incidence and prevalence rates of dementia and mild cognitive impairment in Indigenous or aboriginal populations or in low-income countries, as well as articles referenced in those publications that were not already identified. Incidence of dementia for low-income countries, especially for rural subsistence or Indigenous populations, is understudied.
2. **Interpretation**: In the Indigenous populations that we studied, overall incidence of dementia for age ≥ 60 was low. Moreover, survival once dementia developed was reduced. As prevalence rates depend on both incidence and survival with disease, findings help elucidate observed low prevalence.
3. **Future directions**: Older age and the apolipoprotein E ε4 allele were the strongest risk factors for incident dementia. Differences in presentation and risk factors for dementia in traditional subsistence populations may point to the evolution of the disorder and to potentially modifiable risk factors that emerge with greater acculturation.

2 | METHODS

2.1 | Study design, setting, and participants

Participants are part of the Tsimane Health and Life History Project (THLHP), which has worked with the Indigenous Tsimane since 2002 and Moseten since 2015.[16] Data collection includes medical evaluations; census updates; demographic surveys; cognitive testing; and assessments of diet, physical activities, and economic productivity roughly every 2 years. Chest and brain CT scans were collected between 2015 and 2018 for consenting individuals.[16] Clinical dementia assessments were added in 2017.[5]

All individuals aged ≥ 60 years when a community was visited were invited to participate, along with a stratified random sample of those 40 to 59. Only those ≥ 60 are included here. Participants were from 89 Tsimane and 16 Moseten villages, representing 83% of communities. Omitted villages were not visited because they are outside the jurisdiction of the main Indigenous political units with which there are research agreements. We have no evidence that omitted villages were more or less acculturated or differed in any other systematic way. Participation among those ≥ 60 was 97% for Phase 2 and 91% for Phase 3. Assessments take place in the community. Phase 1 data were collected between July 2017 and September 2019, as has been described;[5] Phase 2 data, between December 2020 and September 2022; Phase 3 data, between February 2023 and October 2024. The surveillance window for person-years at risk was the date the individual was first seen through the last date seen (or date of death).

2.2 | Data sources

Dementia assessments—including clinical and informant interviews, and a brief neurological exam—were conducted by the same Bolivian physician (R.Q.G.) in all three phases. Tsimane were interviewed in the Tsimane language with a translator; Moseten, in Spanish. Cognitive testing was conducted by a bilingual Tsimane research assistant (J.B.C.). Both have > 10 years' experience with participants in THLHP. Training in these assessments and quality control included extensive in-person and video supervision (M.G.; H.C.C.). Figure 1 illustrates data collection. Development of the assessment tools was iterative, with pilot testing and refinement before Phase 1 and after each phase. All protocols and references are available at https://dornsife.usc.edu/cesr/scrap-lab_tsimane/.

2.2.1 | Mental status screening

The clinical interview began with self-reported evaluation of vision, hearing, memory, and reasoning, including subjective concerns about

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FIGURE 1 Data collection in the field. Testing a Tsimane participant at her home. Table and stools are brought to the site by the interviewers. Photo by Margaret Gatz.

memory and thinking. Next, we administered the Modified Mini-Mental State Examination (3MS),[17] which we further adapted for illiteracy and innumeracy. For literate individuals in the THLHP, we used the Spanish 3MS already used in US Latino populations. For the other THLHP participants, for items requiring literacy or skills acquired in the course of schooling, such as use of a pencil, we substituted items in the same domain. For example, instead of asking the participant to read "CLOSE YOUR EYES" and then obey the instruction, we substituted the tester saying, "Do what I do" and then closing his eyes. Instead of copying pentagons, we substituted asking participants to manipulate frames to copy a design. Other items requiring adaptation involved information to which some participants had never been exposed; for example, their date of birth or the name of the country and state in which they lived. In addition, scores were prorated when visual impairment made specific tasks infeasible. All interviews were filmed.

2.2.2 | Informant interview

A family member completed an informant interview, covering changes in memory and thinking, judgment, finding one's way, mood, daily activities, and personal care. The first part incorporates questions from the Kimberley Indigenous Cognitive Assessment (KICA),[18] focused mostly on memory. The second part adds more questions about memory and questions from the Blessed Dementia Scale that capture how cognitive changes may interfere with everyday function as well as changes in personality.[19] Together the interview covered all categories comprising the Clinical Dementia Rating Scale.[20] Open-ended fields were included for impressions, notes, and contextual details. If a participant died before their follow-up visit, a visit was made to the relative and the informant interview was completed with respect to changes in the participant's cognition, function, and medical condition between the last visit and their death.

2.2.3 | Neurological examination

The neurological exam included assessment of extrapyramidal, pyramidal, and focal neurological signs, resting tremor, masked face, bradykinesia, rigidity/cogwheeling, gait with en bloc turning, and deep tendon reflexes. Bolivian physicians received training at a neurology clinic in the United States (University of Southern California) in addition to video and in-person supervision in the field.

2.2.4 | Cognitive test battery

Cognitive measures included processing speed, memory, fluency, and visuospatial ability. Cognitive testing began in 2002 with a battery derived in part from the Mexican Health and Aging Study:[21,22] digits forward, including a spatial version in which participants tapped squares in a display; immediate and delayed verbal recall; category fluency; and visual scan. At Phase 1, we added the matchstick design test from the Indianapolis–Ibadan Dementia Project study[23] and simple reaction time. We also replaced the spatial digit span with a more traditional Corsi blocks task that more directly tests spatial span

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without the distraction of numbers being written on the blocks. Because too many participants could not see the symbols in visual scan, we replaced visual scan with colored dot span in which the participant crosses out the yellow dots rather than small black and white symbols. Digits backward, even counting backward from 5 to 1, could not be comprehended by most participants. Finally, the Wechsler Adult Intelligence Scale Block Design test, which had been administered to a small sample of Tsimane in 2013, was added to the battery, with the instructions to skip if the participant was incapable of matchstick design. Particularly for those with any formal education, Block Design extends the range of difficulty in the visuoconstruction domain, and has been shown to correlate with matchstick design.[23] The tester also indicates when a test is not administered, why it was not done, whether he saw visual impairment or hearing problems as interfering with test performance, and whether the participant appeared to understand the instructions for all tests.

Population-specific norms for those ≥ 60 were derived from a dementia-free sample using prior waves of cognitive assessment.[24] Standard scores were obtained by age, sex, and literacy. For use in diagnosis, an individual's cognitive performance on every test at each phase was classified as better than average (> 1 standard deviation [SD] above average), average (± 1 SD), low (−1 SD), borderline (−1.5 SD), or impaired (≤ 2 SD below average).

2.3 | Outcomes

The primary outcome was diagnosis of clinical syndrome: normal cognition, MCI, or dementia, the latter two following Diagnostic and Statistical Manual of Mental Disorders Fifth Edition (DSM-5) criteria for major and mild neurocognitive disorder.[25] Diagnosis drew on clinical and informant interviews, neurological exam, and longitudinal cognitive scores. The distinction between MCI and dementia relied on whether the 3MS suggested impairment, whether cognitive scores showed a pattern of decline and were borderline or impaired across domains, whether cognitive difficulties interfered with ability to function independently based on the informant interview descriptions and scores, and whether cognitive and functional decline could be accounted for by physical or sensory impairments. The distinction between normal cognition and MCI took into account self-reported difficulties with memory and thinking, a pattern of decline on cognitive tests but without multiple domains with scores below −1.5 SD, and continued engagement in daily activities.

Two experienced Bolivian physicians (D.E.R.; R.Q.G.) and a US-based neurologist and clinical psychologist (H.C.C.; M.G.) made diagnoses at independent case conferences. Teams noted any etiological hypotheses and—for MCI—cognitive domains affected. Disagreements were resolved through consensus. Diagnoses at each phase were made blind to the final consensus diagnosis at the prior phase. Across the three phases, agreement between the two independent diagnostic teams was moderate to substantial (polychoric correlation = 0.91–0.95; weighted kappa = 0.66–0.70), improving slightly over successive phases. The most common disagreement was between normal cognition and MCI. This diagnostic determination necessitated deciding whether individuals had likely been low in ability their entire lives, whether low scores represented a decline, and whether sensory loss could account for the decline. Diagnostic decisions were complicated by widespread uncorrected vision and hearing problems that could account for some of the performance issues. How these sensory impairments were weighed by the two teams led to disagreements.

2.4 | Other variables

A population variable indicated whether the participant was from a Tsimane or Moseten community. Tsimane communities are almost entirely ethnically Tsimane.[26] Moseten are linguistically and culturally similar, but communities are more ethnically diverse.[16] Sex is physician reported. Because many participants do not know their birth date, age was determined through demographic interviews, genealogical reconstruction, sequencing of household and community members, reproductive histories, mission baptism records, and anchoring to dated historical events.[27] Whether a participant had died and their date of death was obtained from family or others in the community during visits by the census team or dementia assessment team to a community.

Risk factors collected at the closest assessment period prior to baseline dementia assessment were used to predict subsequent dementia. See Table S1 in supporting information for exact time between risk factor and visit. A set of risk factors was identified to reflect both traditional risk factors and our prior knowledge of this population, that is, vascular risk, inflammation, and Indigenous lifestyle, as well as to minimize missing data. These risk factors included illiteracy, *APOE* genotype, blood pressure, lipids, fasting glucose, thoracic aorta calcium (TAC), physically active lifestyle, smoking, and selected inflammatory biomarkers.

Generally illiteracy corresponds to no formal schooling.

APOE genotyping derived from two single nucleotide polymorphisms, rs429358 and rs7412, using the TaqMan Allelic Discrimination system (Thermo Fisher Scientific).

Blood pressure, lipids, and fasting glucose were collected at the biennial medical visits just prior to Phase 1. Midlife values are not available for a sufficient proportion of the sample.

Total TAC, including thoracic and aortic arch calcium, was chosen to indicate atherosclerosis and risk of stroke.[28] TAC was scored from CT scans collected in 2015 through 2018 using Agatston methodology. Analyses used both log TAC and TAC dichotomized at 500, following suggested cutoffs.[29]

Physically active lifestyle was operationalized by daily step count 7:00 am to 7:00 pm measured using wrist-worn ActiGraph GT3X accelerometers measured over an average of 4.4 days.

Inflammatory biomarkers included C-reactive protein (CRP) as an indicator of systemic inflammation, interleukin-5 (IL5) as an indicator of parasitic infections and eosinophilic activity; interleukin-6 (IL6) and interleukin-10 (IL10), both focused on inflammation; and vascular endothelial growth factor (VEGF), which may reflect vascular

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dysfunction affecting the brain. Cytokines were measured in serum samples from intravenous blood draws collected in the morning between 6:00 am and 7:00 am after an overnight fast and before any strenuous physical activity. Serum was allowed to clot, then immediately centrifuged, aliquoted, and frozen in liquid nitrogen. Samples were shipped on dry ice to Arizona State University, where they were stored at −80°C until analysis using the Q-Plex Human Cytokine HS 15-Plex (Quansys) multiplex enzyme-linked immunosorbent assay kit on the first freeze–thaw cycle.

For plasma-based biomarkers, fasting morning blood was drawn from as many participants as possible aged 60+ between 2020 and 2024. For dementia cases, we used the sample from the date most proximate to when dementia was diagnosed; otherwise, we used the sample closest to the most recent date of assessment. In effect, the bulk of samples used for analysis were from 2023 and 2024. Specimens were centrifuged and plasma was stored in liquid nitrogen after aliquoting. Specimens were transferred on dry ice to the United States, where they were stored at −80°C prior to analysis. A central nervous system (CNS) brain biomarker panel was analyzed at the University of Wisconsin–Madison Alzheimer's Disease Research Center Biomarker Lab using Alamar's NULISAseq CNS panel (Alamar), measuring Aβ40, Aβ42, p-tau 181, p-tau 217, p-tau 231, glial fibrillary acidic protein (GFAP), and neurofilament light chain (NfL).[30] This was done in a single batch.

2.5 | Statistical methods

Those in the sampling frame who did and who did not participate in the dementia assessment at Phase 2 and at Phase 3 were compared to check for non-participation biases. Differences were tested with chi-squared test for male, population, and literacy, with a Wilcoxon rank sum test for age groups, and with a *t* test for continuous age.

Incidence of dementia was calculated among participants in follow-up Phases 2 and 3 with a Phase 1 baseline clinical diagnosis of normal cognition or MCI plus participants in Phase 3 entering the sample at Phase 2 with a Phase 2 baseline clinical diagnosis of normal cognition or MCI (n = 730). Incidence rates exclude those with dementia at a prior phase. Incidence of MCI was calculated among those with normal cognition at their baseline phase (n = 680). Person-time denominators were calculated from date of the individual's baseline assessment to date of their dementia (or MCI) diagnosis or, for those not diagnosed with dementia, their final follow-up assessment or date of death (if prior to Phase 3). Dementia incidence rates with 95% confidence intervals (CIs) were calculated in the overall sample of non-demented persons (i.e., MCI or normal cognition) aged ≥ 60 at their baseline. Age-specific incidence rates were calculated in 5-year age groups based on age at their incident diagnosis or last phase.

Associations of dementia incidence with demographics, *APOE* genotype, blood pressure, glucose, lipids, total TAC, daily step count, and measures of inflammation were assessed using Fine and Gray semi-parametric proportional hazards regression models to account for deaths occurring before dementia onset,[31] as the primary interest was in assessing factors that influence the probability of developing dementia, or cumulative dementia risk, over follow-up as an assessment of public health burden. Secondarily, semi-parametric Cox proportional hazards regression was used to estimate cause-specific hazard ratios (HRs) to identify possible factors for the etiology of dementia in this indigenous population. Results are summarized as age-adjusted HRs with 95% CIs and age-adjusted subhazard ratios (SHRs) with 95% CIs for competing risk models. SHRs and HRs for continuous variables are reported per SD. All analyses were adjusted for baseline age, sex, and population. Each model included individuals with complete data for a given risk factor (age, sex, and population were available for all participants); given the small numbers of incident cases, imputations of missing data were not used. In sensitivity analyses, stabilized inverse probability weighting was used, with probability of completing the risk factor assessment estimated from logistic models based on age, population, and sex. Proportional hazards assumptions were tested using correlations of Schoenfeld residuals and interactions of independent variables with follow-up time. False discovery rate (FDR) adjustment for the tested risk factors used the Benjamini–Hochberg method.

Plasma biomarkers were analyzed using a Firth penalized logistic regression, due to the small number of dementia cases, with dementia status (dementia cases vs. normal controls) as the binary dependent variable, controlling for population (Tsimane vs. Moseten), sex, and age and year at which the blood sample was collected. For applying the ATN framework, we used only those cognitively normal at the last assessment to calculate age-residualized biomarker values. In the absence of a clinical standard, we created ATN profiles for each dementia case, taking any score beyond the 75th percentile as a positive score for a given marker.

Dementia and MCI prevalence were estimated by phase for each population. Denominators are number assessed during the phase. The numerator is number of cases diagnosed with dementia (or MCI) when visited during that phase. As each village is visited approximately every 2 years, this approach accounts for the fact that point prevalence measured at the end of the phase is biased by deaths and by our inability to know what has happened to each participant between their assessment and the end of the phase. For each phase, each individual's age is age when visited for assessment, required to be ≥ 60 years. To compare Tsimane and Moseten, age-standardized prevalence estimates with confidence limits were computed, using the combined population age distribution.

Mortality rates over assessment Phases 1 through 3 were compared among diagnostic groups. HRs for mortality were estimated using proportional hazards regression, modeling diagnostic group at Phase 1 and Phase 2 as a time-varying covariate and adjusting for age, sex, and population. Mortality rates by diagnostic group were also computed at each of Phase 1 and Phase 2. The person-time denominator was calculated from Phase 1 or 2 assessment date to date of death or date last assessed in cognitive follow-up. Mortality rates after a diagnosis of incident dementia in Phase 2 were similarly calculated.

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TABLE 1 Demographic characteristics of Phase 2 and Phase 3 samples, including total sampling frame and those not participating at each phase.

	PHASE 2			PHASE 3		
	Total sampling frame±	Total with consensus clinical diagnosis	Total not diagnosed^	Total sampling frame±	Total with consensus clinical diagnosis	Total not diagnosed^
N	790	770	20	860	779	81
Population						
Tsimane	539 (68.2%)	527 (68.4%)	12 (60.0%)	586 (68.1%)	532 (68.3%)	54 (66.7%)
Moseten	251 (31.8%)	243 (31.6%)	8 (40.0%)	274 (31.9%)	247 (31.7%)	27 (33.3%)
Sex						
Men	413 (52.3%)	402 (52.2%)	11 (55.0%)	457 (53.1%)	409 (52.5%)	48 (59.3%)
Women	377 (47.7%)	368 (47.8%)	9 (45.0%)	403 (46.9%)	370 (47.5%)	33 (40.7%)
Literacy						
Illiterate	583 (73.8%)	568 (73.8%)	15 (75.0%)	609 (71.0%)	549 (70.5%)	60 (76.0%)
Literate	207 (26.2%)	202 (26.2%)	5 (25.0%)	259 (29.0%)	230 (29.5%)	19 (24.0%)
Age group						
60–64	245 (31.1%)	238 (30.9%)	7 (35.0%)	271 (31.5%)	248 (31.8%)	23 (28.4%)
65–69	199 (25.2%)	192 (24.9%)	7 (35.0%)	229 (26.6%)	206 (26.4%)	23 (28.4%)
70–74	170 (21.3%)	168 (21.8%)	2 (10.0%)	170 (19.8%)	156 (20.0%)	14 (17.3%)
75–79	107 (13.5%)	103 (13.4%)	4 (20.0%)	109 (12.7%)	100 (12.8%)	9 (11.1%)
80–84	41 (5.2%)	41 (5.3%)	0 (0.0%)	47 (5.5%)	42 (5.4%)	5 (6.2%)
85+	28 (3.5%)	28 (3.6%)	0 (0.0%)	34 (4.0%)	27 (3.5%)	7 (8.6%)
Mean (SD) age	69.32 (7.02)	69.35 (7.05)	68.15 (5.68)	69.25 (7.22)	69.13 (7.12)	70.41 (8.08)

Notes: For continuous variables, table shows mean (standard deviation). For categorical variables, table shows frequency (percent). ±The total sampling frame for Phase 2 includes everyone in the Phase 1 sampling frame who had not (a) moved out of the communities in the study or (b) died prior to the Phase 1 assessment, plus everyone in the communities in the study who had become age 60 between Phase 1 and Phase 2. The total sampling frame for Phase 3 includes everyone in the Phase 2 sampling frame who had not (a) moved out of the communities in the study or (b) died prior to the Phase 2 assessment, plus everyone in the communities in the study who had become age 60 between Phase 2 and Phase 3. ^The total not diagnosed includes those communities that were inaccessible, refusals, or those unable to evaluate due to physical condition. Literacy is unavailable for 2 people in the Phase 2 sampling frame.
Abbreviation: SD, standard deviation.

3 | RESULTS

3.1 | Participants

Table 1 shows demographic characteristics of both participants and those in the sampling frame but unable to be assessed. There were no significant differences in population, sex, literacy, and age between those able versus unable to be assessed (Tables S2 and S3 in supporting information). The sampling frame for Phase 1 included all participants in THLHP aged ≥ 60 and alive. For Phase 2, the sampling frame included all persons visited at Phase 1 (including a relative if deceased), those who could not be visited during Phase 1 (e.g., temporarily away, community inaccessible due to flooding), and all who became age ≥ 60 since Phase 1. For Phase 3, the sampling frame included all persons previously visited unless deceased before Phase 2 (including a relative if deceased after their Phase 2 visit), those who could not be visited previously but still believed to be living in an included community, and all who became age ≥ 60 since Phase 2. Figures S1 and S2 in supporting information provide sample flow for Phase 2 and Phase 3 and indicate which participants were included in prevalence samples. Table 2 shows the incidence sample.

Of 108 participants diagnosed as normal cognition or MCI at their last visit and who died before their next scheduled visit, we were able to make a *post mortem* visit to a relative for 98 (91%), although 2 participants had insufficient information to form a *post mortem* diagnosis. *Post mortem* diagnoses were based on all prior assessments plus the new information from the informant. Of the 96 receiving a *post mortem* diagnosis, 7 were evaluated as having become more impaired and were given a *post mortem* diagnosis reflecting that progression. No significant differences were found between those without a *post mortem* informant visit and those with a *post mortem* visit with respect to sex or population (Table S4 in supporting information). Those with MCI were significantly less likely to receive a *post mortem* visit (4 people missed, 25%) than those with NC diagnoses (6 people missed, 7%) and accordingly *post mortem* visits were more likely to be missed in people who were older. The result could be some under-ascertainment.

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TABLE 2 Demographic characteristics of incidence sample, by incident group.

	Incident dementia	Non-dementia cases	Incident MCI	Non-MCI cases
N	22	708	31	649
Population				
Tsimane	15 (68.2%)	488 (68.9%)	21 (67.7%)	448 (69.0%)
Moseten	7 (31.8%)	220 (31.1%)	10 (32.3%)	201 (31.0%)
Sex				
Men	13 (59.1%)	366 (51.7%)	16 (51.6%)	341 (52.5%)
Women	9 (40.9%)	342 (48.3%)	15 (48.4%)	308 (48.4%)
Age at end of follow-up				
60-64	0	133 (18.8%)	1 (3.2%)	132 (20.3%)
65-69	3 (13.6%)	218 (30.8%)	3 (9.7%)	212 (32.7%)
70-74	6 (27.3%)	174 (24.6%)	10 (32.3%)	153 (23.6%)
75-79	8 (36.4%)	114 (16.1%)	7 (22.6%)	97 (14.9%)
80-84	1 (4.5%)	42 (5.9%)	6 (19.3%)	33 (5.1%)
85-89	4 (18.2%)	21 (3.0%)	4 (12.9%)	17 (2.6%)
90+	0	6 (0.8%)	0	5 (0.8%)
Mean (SD) age	77.2 (6.5)	71.2 (6.5)	76.8 (6.5)	70.7 (6.3)
Tsimane				
N	15	488	21	448
Sex				
Men	8 (53.3%)	251 (51.4%)	10 (47.6%)	236 (52.7%)
Women	7 (46.7%)	237 (48.6%)	11 (52.4%)	212 (47.3%)
Mean (SD) age	77.0 (5.5)	71.3 (6.6)	76.5 (6.4)	70.8 (6.4)
Moseten				
N	7	220	10	201
Sex				
Men	5 (71.4%)	115 (52.3%)	6 (60.0%)	105 (52.2%)
Women	2 (28.6%)	105 (47.7%)	4 (40.0%)	96 (47.8%)
Mean (SD) age	77.6 (8.7)	71.1 (6.2)	77.5 (7.1)	70.7 (6.2)

Notes: Total dementia sample ($N = 730$) excludes 7 individuals with dementia in Phase 1 and 1 individual whose baseline was Phase 2 and was identified as having dementia. Those categorized as non-cases include those diagnosed as normal cognition or MCI in all phases (including normal cognition/normal cognition, MCI/MCI, MCI/normal cognition, normal cognition/MCI transitions). For analyses of MCI incidence (total sample = 680), those with baseline MCI or dementia were excluded and non-cases included only normal cognition.
Abbreviation: MCI, mild cognitive impairment; SD, standard deviation.

3.2 | Prevalence rates

Phase 2 and Phase 3 dementia prevalence estimates were based on those alive when visited during the phase. Within each population, crude prevalence of dementia at age 60+ was similar in Phases 2 and 3. In Tsimane, prevalence was 2.09% (95% CI = 1.01%–3.81%) in Phase 2 and 2.00% (95% CI = 0.97%–3.65% in Phase 3. In Moseten, prevalence was 1.80% (95% CI = 0.49%–4.55%) in Phase 2 and 1.70% (95% CI = 0.47%–4.30%) in Phase 3. Table 3 shows prevalence for all three phases, with standardized rates adjusting for population age structures. Point estimates at both follow-up Phases 2 and 3 were slightly higher than Phase 1 but the difference was within the confidence intervals.

Prevalence of MCI at Phase 2 in Tsimane was 3.97% (95% CI = 2.41%–6.14%), and in Moseten, 3.15% (95% CI = 1.28%–6.39%). Prevalence of MCI at Phase 3 was 3.21% (95% CI = 1.84%–5.15%) in Tsimane, and 3.40% (95% CI = 1.48%–6.60%) in Moseten. These rates are lower than Phase 1 MCI prevalence: 7.7% (95% CI = 5.2%–10.3%) and 9.8% (95% CI = 4.9%–14.6%), respectively. The lower rates of MCI at Phases 2 and 3 reflect the additional longitudinal data available with the additional phases, permitting better discrimination between low average cognitive ability or temporary fluctuations and decline in

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TABLE 3 Prevalence of dementia at three phases.

	Phase 1 Prevalence (total dementia cases/total assessed)	Phase 2 Prevalence (total dementia cases/total assessed)	Phase 3 Prevalence (total dementia cases/total assessed)
All	7/592 = 1.18% (95% CI = 0.48%, 2.42%)	14/700 = 2.00% (95% CI = 1.10%, 3.33%)	14/734 = 1.91% (95% CI = 1.05%, 3.18%)
Tsimane			
crude	5/418 = 1.20% (95% CI = 0.39%, 2.77%)	10/478 = 2.09% (95% CI = 1.01%, 3.81%)	10/499 = 2.00% (95% CI = 0.97%, 3.65%)
standardized	1.14% (95% CI = 0.14%, 2.14%)	1.87% (95% CI = 0.70%, 3.04%)	2.10% (95% CI = 0.80%, 3.40%)
Moseten			
crude	2/174 = 1.15% (95% CI = 0.14%, 4.09%)	4/222 = 1.80% (95% CI = 0.49%, 4.55%)	4/235 = 1.70% (95% CI = 0.47%, 4.30%)
standardized	1.30% (95% CI = 0%, 3.10%)	1.55% (95% CI = 0.03%, 3.08%)	1.62% (95% CI = 0.02%, 3.22%)

Note: Numerator is number of cases alive when visited during the phase. Denominator is total number assessed alive when visited during the phase. Exact binomial 95% confidence intervals (CI) are shown. Standardized prevalence is based on the combined Tsimane and Moseten age structure at each phase. Excluded from prevalence calculations are those deceased before their scheduled visit, where only a relative was interviewed.

TABLE 4 Incidence of dementia (per 1000 person-years), by population and by population and age group.

Age at Incidence Group	Tsimane		Moseten	
	Cases/person-years	Rate (95% CI)	Cases/person-years	Rate (95% CI)
60–64	0 / 203.00		0 / 87.16	
65–69	1 / 675.79	1.48 (0.218–10.50)	2 / 327.99	6.10 (1.53–24.38)
70–74	5 / 593.52	8.42 (3.51–20.24)	1 / 243.47	4.11 (0.58–29.16)
75–79	7 / 345.15	20.28 (9.67–42.54)	1 / 175.56	5.70 (0.80–40.44)
80–84	0 / 121.34		1 / 61.14	16.36 (2.30–116.11)
85–89	2 / 78.45	25.49 (6.38–101.94)	2 / 32.84	60.91 (15.23–243.55)
>90	0 / 20.59		0/ 5.80	
Total (aged 60+)	15 / 2037.85	7.36 (4.44–12.21)	7 / 933.96	7.49 (3.57–15.72)

Note: Incidence rates and 95% confidence intervals (CI) per 1000 person years. Age group at incidence of dementia.

cognition. As a result, some individuals diagnosed as MCI at Phase 1 were subsequently reclassified as normal cognition but are included in the Phase 1 prevalence.

3.3 | Incidence rates and correlates

For calculating incidence, we retained all individuals diagnosed in Phase 1 or Phase 2 and followed up in one or both subsequent phases, including those deceased after their immediately prior visit but whose informant was interviewed after the participant's death. Demographic characteristics are in Table 2. Median follow-up was 4.73 years (range 0.08–7.05). Times of < 1 year reflect participants who died shortly after their baseline assessment. Overall incidence for age 60+ was 7.36 cases/1000 person-years (95% CI = 4.44–12.21) for Tsimane and 7.49 cases/1000 person-years for Moseten (95% CI = 3.57–15.72; see Table 4).

Twenty-two incident dementia cases were identified; 8 diagnosed as MCI at baseline, and 14 as normal cognition. Rates were higher for Moseten than Tsimane, and for men (8.71 [95% CI = 5.06–15.00]) than for women (6.09 [95% CI = 3.17–11.70]), although not significantly. Two cases were diagnosed *post mortem*, and seven more died before the end of the last phase when they were evaluated in person. Two of the cases were literate; few had any formal schooling (HR [95% CI] = 0.24 [0.05–1.22]; SHR [95% CI] = 0.27 [0.06–1.18]). As context, ≈ 84% of all Tsimane and 40% of all Moseten aged 60+ are illiterate. Five incident cases were suggestive of AD or displayed amnestic profiles. Two had suffered head injuries but also showed progressive cognitive impairment prior to injury. Two had Parkinson's disease or significant tremor. Hypertension or stroke was mentioned in three cases. Five had significant depression. Multi-morbidity including visual impairment, hearing impairment, and/or impaired mobility affected more than half. Need for food was noted in five cases.

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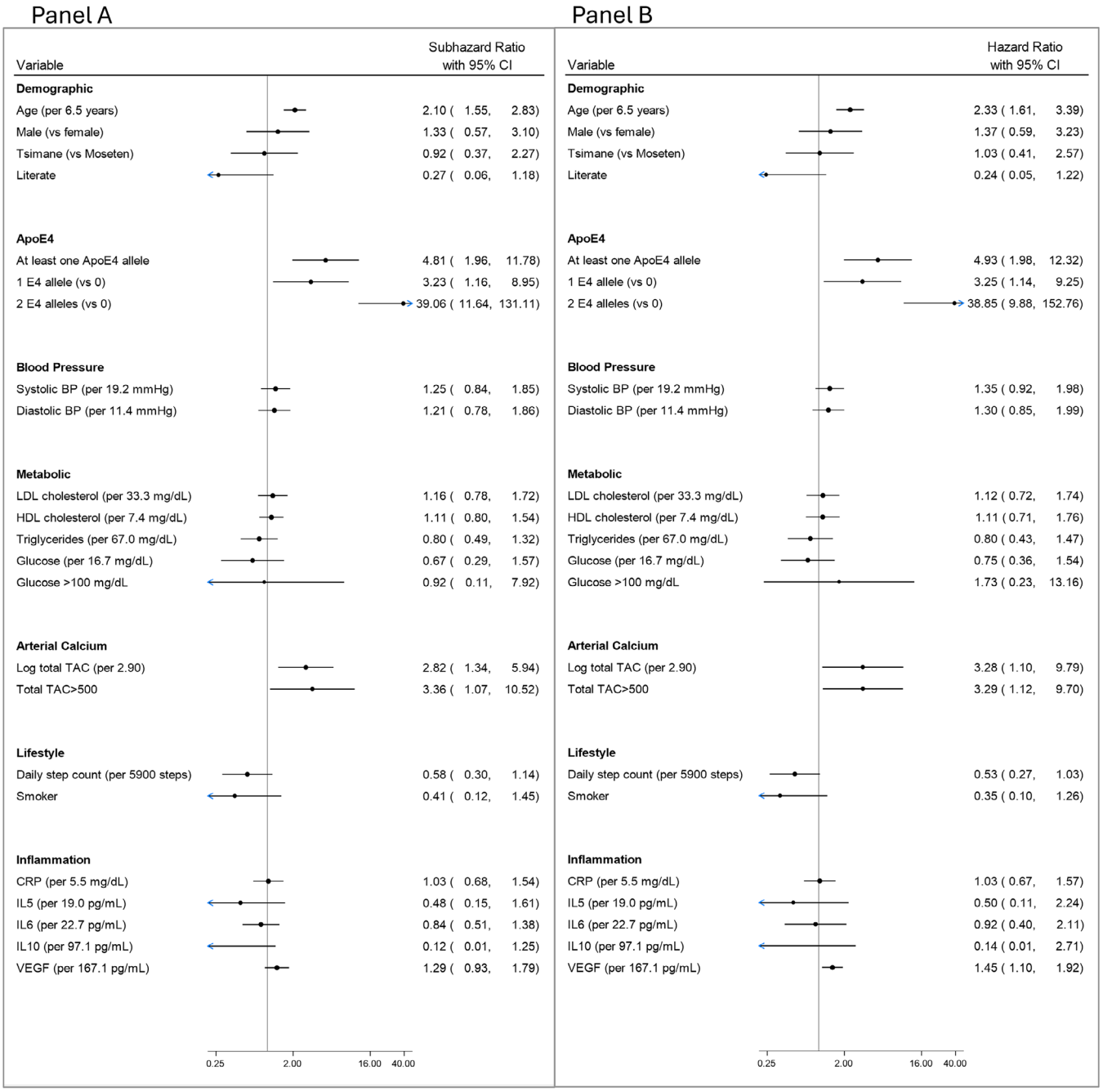

FIGURE 2 Risk factors for incident dementia: A, Subhazard ratios (SHRs), taking into account competing risks due to mortality. SHRs are expressed per SD of continuous variables. B, Baseline age-, sex-, and population-adjusted hazard ratios (HRs). SHRs are expressed per SD of continuous variables. All models analyze each variable separately, adjusted for age, sex, and population. Total TAC = thoracic artery plus aortic arch calcium. *APOE*, apolipoprotein E; BP, blood pressure; CRP, C-reactive protein; HDL, high density lipoprotein; IL, interleukin; LDL, low density lipoprotein; VEGF, vascular endothelial growth factor.

Figure 2 shows predictors of dementia risk as age-, sex-, and population-adjusted relative HRs for each risk factor, calculated as competing risk SHRs to consider competing risks due to mortality. Table S5 in supporting information shows corresponding detailed data. Nineteen of the 22 cases were genotyped, of whom 10 (52.6%) had at least one *APOE* ε4 allele, compared to 104 of 520 non-cases (20.0%; HR [95% CI], FDR q value = 4.93 [1.98–12.32], q = 0.006; SHR [95% CI] = 4.81 [1.96–11.78], q = 0.006). VEGF was associated with elevated dementia risk (16 cases, 543 non-cases; HR [95% CI], FDR q value = 1.45 [1.10–1.95] per SD, q = 0.06; SHR [95% CI] = 1.29 [0.93–1.79] per SD, q = 0.29).

Among 19 cases, 73.4% had total TAC scores > 500 versus 34.6% of 587 non-cases (HR [95% CI], FDR q value = 3.29 [1.12–9.70], q = 0.11; SHR [95% CI] = 3.36 [1.07–10.52], q = 0.17). Hypertension, lipids, and glucose assessed prior to baseline cognitive assessment were not associated with incident dementia. *APOE*

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ε4 and total TAC but not VEGF remained significant after FDR adjustment.

Those who had data for each risk factor were compared on age, sex, population, and baseline diagnosis to those for whom data were missing (Table S6 in supporting information). Moseten were more likely to be missing glucose, lipids, and accelerometry than were Tsimane. Older participants were more likely to be missing accelerometry than younger participants, with the oldest age group also more likely to be missing glucose and blood pressure. SHR and HR estimates were not appreciably altered in sensitivity analyses that used stabilized inverse probability weights that modeled probability of having risk factor measure completed as a function of age, sex, and population.

There were 31 incident MCI cases, for an overall rate of 11.11/1000 person-years in Tsimane (95% CI = 7.24–17.04) and 11.64/1000 person-years in Moseten (95% CI = 6.26–21.64; Table S7 in supporting information). Risk factor profiles for MCI tended to be between scores for those with dementia and cognitively normal participants (Table S8 in supporting information). Elevated risk for MCI was associated with *APOE* ε4 genotype, higher literacy, elevated glucose, total TAC, and lower steps per day. Only *APOE* ε4 was significant after adjusting for FDR. SHR and HR estimates were in general not appreciably altered in sensitivity analyses that used stabilized inverse probability weights; estimates for *APOE* ε4 were reduced (HR [95% CI] = 2.94 [1.10–7.83]; SHR [95% CI] = 2.67 [0.95–7.53) and estimates for daily step count were magnified (HR [95% CI] = 0.47 [0.30–0.73]; SHR [95% CI] = 0.50 [0.33–0.77). Still, these risk factor results should be treated as exploratory given the instability of MCI classifications.

3.4 | Plasma based biomarkers

Analyses included 573 participants who were cognitively normal across all three phases; 17 incident MCI cases, and 11 incident dementia cases who had a plasma sample corresponding to the visit. Comparing the dementia cases to cognitively normal controls with Firth logistic regression, none of the Aβ or p-tau markers were statistically significant, and only NfL survived FDR. NfL was associated with higher odds of dementia, OR = 2.81 (95% CI 1.69–4.68), $P < 0.0001$. NfL was also associated with higher odds of MCI, OR = 1.87 (95% CI 1.10–3.19), $P = 0.0214$, although not significant after FDR.

Tests for non-participation bias showed a significant difference by population with Tsimane dementia cases less likely than Moseten dementia cases to have a plasma sample. This result reflects that nine Tsimane cases had died before they could be visited for a plasma sample. A sensitivity analysis showed that NfL was associated with higher odds of dementia in both Tsimane, OR = 5.10 (1.90–13.68, $P = 0.0012$, and Moseten, OR = 1.94 (1.04–3.62), $P = 0.0374$.

Using the 75th percentile categorization, of the eleven dementia cases, one showed no positive biomarker, one was A−/T−/N+, one was A+/T−/N−, four were A+/T−/N+, three were A−/T+/N+, and one was A+/T+/N+. More than half the incident dementia cases had elevated NfL. Given the small *N* with available data and arbitrary cutoffs, the results of these analyses are not strongly consistent with a prototypical AD profile, although there is evidence of neurodegeneration. See Table S9 in supporting information. Importantly, these data are preliminary and available only for a subset of participants.

3.5 | Mortality

Among 149 deaths in 758 individuals, mortality hazards after Phase 1 and 2 assessments were compared among diagnostic groups. Age-, sex-, and population-adjusted HRs (95% CI) were 6.14 (3.32–11.35) for dementia compared to normal cognition, and 2.70 (1.66–4.36) for MCI compared to normal cognition.

Median follow-up time from date of Phase 1 visit among 586 persons aged ≥ 60 was 5.01 person-years (range 0.04 to 7.66). By the end of Phase 3, 100% of dementia cases had died; 40% of MCI cases; and 21% of those diagnosed with normal cognition, chi2[2] = 32.18, $P < 0.001$.

Median follow-up time available for Phase 2 was 2.07 years (range 0.02 to 4.25). Therefore, we calculated 2-year survival rates for Phase 1 and Phase 2. Deaths per 1000 person-years for dementia cases were strikingly higher than rates for MCI or normal cognition at both phases, with a 10 times higher rate of deaths within 2 years of assessment among dementia cases than those with normal cognition (Table 5).

We were able to find descriptive notes about cause of death for 100 deaths, generally reflecting what the THLHP physician learned when he went to visit and found that the person had died. When there were notes, average age at death for those with dementia and MCI was 80; for normal cognition, 72. Notes were coded by a physician (H.C.C.) blind to the person's dementia status. Across all diagnostic groups, the chief contributors to death that stood out were (1) treatable infections both chronic (e.g., pulmonary tuberculosis) and acute (e.g., after ruptured gall bladder or bowel obstruction); (2) renal failure frequently attributed to benign prostatic hypertrophy but also noted in individuals with Type 2 diabetes mellitus or hypertension; (3) less frequently but still notable, accidents (e.g., fall resulting in head trauma). In 30% of dementia cases, the death notes mentioned stroke; but in only 15% of MCI cases, and almost none of those with normal cognition.

4 | DISCUSSION

In this population-based study of Indigenous Tsimane and Moseten Amerindians of Amazonian Bolivia, we replicated our prior observation of a very low prevalence of dementia, as measured by an in-person clinical workup and consensus clinical diagnosis with DSM-5 criteria. After 4.7 years of follow-up, total dementia incidence for Tsimane and Moseten, aggregated across age ≥ 60, was modestly lower than most published studies, chiefly in Europe and the United States.[32–36] However, age-specific dementia incidence rates for Tsimane and Moseten are within the same range as these comparison studies, especially before age 80, albeit with large CIs due to low numbers of cases (Figure 3). The population pyramid for Tsimane and Moseten is triangular, unlike the rectangular pyramids in Europe and the United States.

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TABLE 5 Two-year mortality rates (per 1000 person-years), for dementia, MCI, and normal cognition for Phase 1 and Phase 2.

A. Two-year mortality rates for all cases in Phase 1 and in Phase 2				
	Phase 1		Phase 2	
Diagnostic group	Deaths/person-years	Rate (95% CI)	Deaths/person-years	Rate (95% CI)
Normal cognition	29 / 1030.15	28.15 (19.56–40.51)	37 / 1194.10	30.99 (22.45–42.77)
MCI	6 / 93.21	64.37 (28.92–143.28)	10 / 35.17	284.32 (152.98–528.42)
Dementia	3 / 11.42	262.77 (84.75–814.74)	8 / 12.90	620.32 (310.22–1240.39)

B. Two-year mortality rates for incident cases in Phase 2		
	Phase 2 incidence sample	
Diagnostic group	Deaths/person-years	Rate (95% CI)
Normal cognition	34 / 934.82	36.37 (25.99–50.90)
MCI	8 / 30.72	260.39 (130.22–520.68)
Dementia	5 / 8.23	607.74 (252.96–1460.11)

Abbreviations: CI, confidence interval; MCI, mild cognitive impairment.

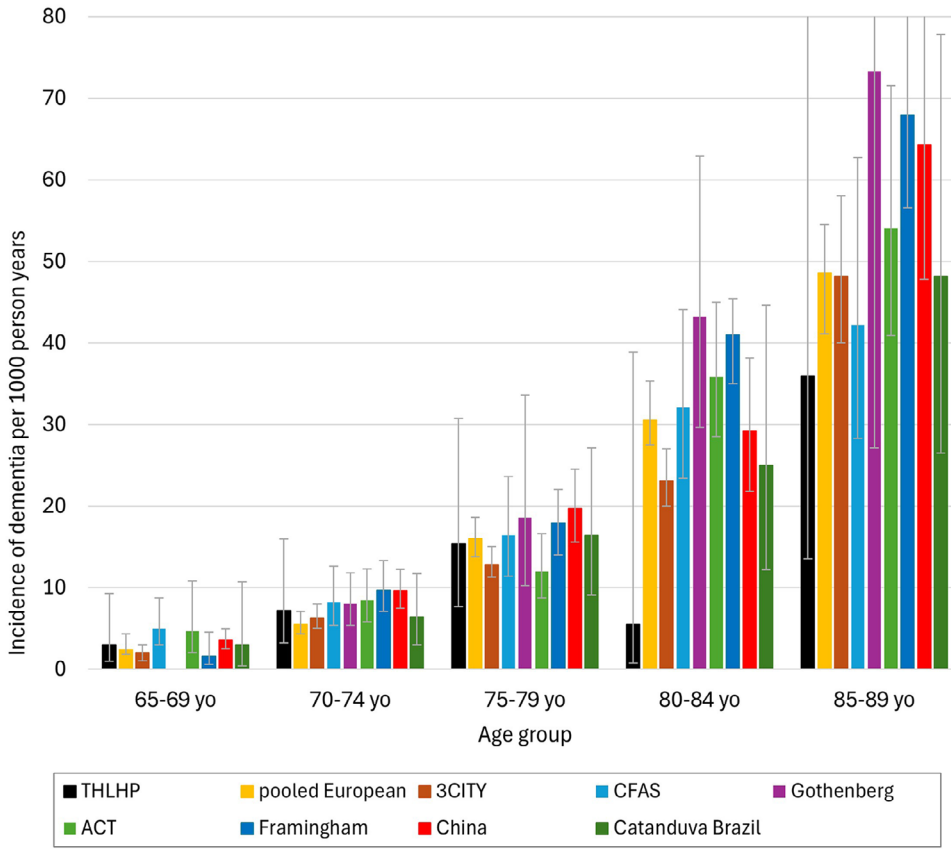

FIGURE 3 Comparative incidence rates for dementia by age group for THLHP (Tsimane Health and Life History Project, including both Tsimane and Moseten) and available comparison studies. Systematic reviews and meta-analyses were prioritized, with additional non-duplicative studies in which diagnoses were based on clinical workups and where comparable incidence rates by 1000 person-years for 5-year age bands were available. Pooled European includes studies from Finland, Sweden, Denmark, Netherlands, United Kingdom, France, and Spain.[32] Pooled European does not include any of the individual European studies in the chart. Individual European studies include: 3CITY (France), CFAS (Cognitive Function and Ageing Studies; United Kingdom), Gothenberg studies (Sweden).[33] US studies include ACT (Adult Changes in Thought)[34] and Framingham Heart Study.[33] Other studies include an analysis of four regions in China[35] and a project conducted in a town in São Paulo state, Brazil.[36] For the study in China, the age category labeled 85 to 89 includes age 85+. There are no available data for the age category 65 to 69 for the Gothenberg studies

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Aggregated incidence for age 60+ is higher in countries in which the population is older overall.

Compared to the few published incidence for other Indigenous groups, Tsimane and Moseten rates are two to nearly three times lower (Figure S3 in supporting information).[8,9]

For incident MCI, comparison studies (Figure S4, Panel A in supporting information) are largely from Europe or the United States and generally show higher incidence than Tsimane or Moseten. Similar to patterns for dementia, Australian Aboriginal studies show considerably higher MCI incidence (Figure S4, Panel B).[8,9] In our results, lower prevalence of MCI at Phases 2 and 3 than Phase 1 may in part reflect expected fluctuation, or reclassification in those who did not show progression in impairment.[37]

Prevalence of dementia remains low compared to other populations, with European and US rates three to four times greater. Compared to other Indigenous or rural illiterate groups and reports from LMIC, we see a bifurcation: some show more dementia than HICs; others, less.[2,4,38] High rates occur in forcibly settled Australian aboriginals, in whom hypertension and stroke are dementia risk factors related to lifestyle changes associated with greater integration into the dominant culture. Higher rates are seen in the Melanau of Malaysia, in whom Western dietary habits such as soda intake and metabolic syndrome are noted.[39] Higher rates of cognitive impairment (dementia and MCI) were reported in American Indians compared to other US studies.[40] Both higher rates of cognitive impairment and younger age at onset were seen in a Brazilian Indigenous community near Manaus where loss of territories and adoption of Western habits were noted.[41] In contrast, Indigenous populations living traditional lifestyles show low rates, evidenced by rural Indian subsistence farmers assessed in 1991 to 1999,[42] rural populations in the Brazilian Amazonian region,[6] and recent work with Peruvian Quechua.[7]

In Tsimane and Moseten, low prevalence combined with similar incidence by age group compared to other studies suggests that part of the explanation for low prevalence is short survival among cases. While sample sizes preclude formally decomposing prevalence, mortality analyses help explain the difference: Dementia cases have shortened life expectancy, helping to account for observed prevalence, with selective mortality and possible ascertainment bias also to consider.

Although survival time after diagnosis with dementia varies across studies, compared to the United States, survival with dementia appears two to three times shorter for Tsimane and Moseten.[43,44] Caution is needed as age at onset in prevalent cases in Tsimane and Moseten is uncertain—we can only report age at diagnosis. Still, our field observations offer some context for these results and highlight living with dementia in a society in which lifestyle and environment are more like the human ancestral pre-industrial past. Everyone contributes to family food acquisition. High fertility means many children to feed.[26] Food insecurity is widespread.[45] If dementia prevents a person from contributing to food production, it can become a burden on the family, with the older person's decreasing nutritional status contributing to death. We noticed that dementia often co-occurs with other health conditions, for example, impaired vision or mobility, worsening inability to contribute. Minimal medical care means many treatable conditions receive little remediation. Thus, we speculate that multi-morbidities, including dementia, as well as insufficient family capacity to provide care may contribute to shortened survival.

A subsistence lifestyle may plausibly be protective against dementia, with low exposure to usual modifiable risk factors for dementia such as obesity, hypertension, diabetes, and physical inactivity. Tsimane and Moseten generally show low prevalence of these risks, and we did not find support for higher scores predicting which individuals would develop dementia. There was some indication that physical activity might be protective, although reverse causation cannot be ruled out.

Contrary to hypothesis, we found little evidence that inflammatory cytokines serve as risk factors. Only VEGF, which has pro-inflammatory effects and may contribute to neuroinflammation, was predictive of incident dementia (and did not remain significant on FDR adjustment)—and may be related to higher TAC. We have previously reported that age-related chronic inflammation is low, especially among Tsimane.[46] Those authors further suggested that the immune system response developed from parasitic exposure may contribute to lowering age-related inflammation.[46]

APOE ε4 was a significant predictor of incident dementia and incident MCI. In a review of diverse Indigenous populations, including both prevalence and incidence studies, half the studies found *APOE ε4* to be a significant risk factor for either AD or all-cause dementia, while half did not.[47]

Low education is a highly cited risk factor for dementia. However, among Tsimane and Moseten, there is little variability in years of schooling, and even those reporting 1 to 2 years of school are largely illiterate. We found weak evidence that illiteracy predicted dementia; however, literacy was higher in MCI cases than in those with normal cognition. Access to schooling is also increasing, with illiteracy less frequent among those later born.

Literacy clearly affects how participants approach cognitive tests. We adapted assessments for illiteracy to avoid conflating inability to read or spell or unfamiliarity with writing implements with cognitive impairment and standardized test scores for age, sex, and literacy. Most Tsimane, even those able to count from 1 to 5, had little understanding of numbers; for example, a participant might believe that a 200 boliviano note was worth less than two 100 boliviano notes.

Our impression remains that the majority of dementia cases in Tsimane and Moseten are not prototypical AD, initially based on clinical judgment of their pattern of deficits and on the intracranial arterial calcification that tends to be more prominent in cases.[14] The neurological exam elicits symptoms that may be vascular parkinsonism. Elevated total TAC is indicative of systemic vascular disease and stroke risk. A weak signal that VEGF is elevated is compatible with a compensatory response to reduced cerebral blood flow. Preliminary analysis of plasma-based brain biomarkers finds strong indication of neurodegeneration and neuroinflammation measured by NfL and GFAP, which are non-specific but could reflect vascular brain injury. Remarkably, there was little support for ATN profiles characteristic of AD. Neither Aβ nor p-tau profiles were significantly associated with incident MCI or dementia. Finally, other studies of Indigenous populations have noted that AD formed a smaller part of the total dementia than in European

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and US studies.[3] This picture nonetheless remains speculative as we continue to follow the participants.

Limitations of the study include the small number of incident cases and hence imprecise estimates, assessment challenges, possible misclassification especially for MCI, inclusion with incident dementia of cases with baseline MCI, possible *post mortem* under-ascertainment, multiple testing, missingness for risk factors, absence of measurements of midlife risk factors, and possible influence of selective survival on incidence estimates and biomarker interpretation. Accordingly, results must be taken with caution. Differential diagnosis is challenging, given reliance on what can be done in these remote villages, although for the next phase, new and higher quality CT imaging and additional plasma-based AD biomarker data will be available. Sensitivity analyses using inverse probability weighting to account for missing risk factor data are also limited by the small event count, potentially contributing to unstable estimation of weights and extreme weights; use of stabilized weights and a simple weight model yielded very stable weights (mean 1.00, range 0.97–1.05).

We do not believe that the low rate of dementia reflects failure to locate cases. All villages within the main Indigenous political units are in the sampling frame, a biennial census is conducted, and everyone aged 60+ is included. At Phases 2 and 3, of those in the sampling frame, visits by the assessment team were made to 98.2% and 92.3%, respectively (Figures S1 and S2). Refusals were < 1%. Many incidence studies do not attempt to determine dementia status *post mortem*; we were able to locate an informant for 90.7% of those deceased. Longitudinal cognitive testing provided cognitive trajectories. Even considering possible underdiagnosis from *post mortem* informant interviews, incidence would only modestly increase.

In the future, dementia rates in Tsimane and Moseten may increase with greater cell phone coverage broadening exposure to Westernized lifestyles, and motorized canoes and motorcycles leading to greater market access, less healthy commercialized diets, and reduced physical activity. At the same time, improved public health and medical care may extend survival among those who do not develop dementia as well as for those who do.

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CONFLICT OF INTEREST STATEMENT

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CONSENT STATEMENT

The study was approved by the ethics committee of the Faculty of Medicine, Universidad Mayor de San Simon, Cochabamba, Bolivia and the institutional review board of the University of California, Santa Barbara. The Tsimane and Moseten governments, village leaders, and participants approved all protocols. Informed consent was obtained in the participant's native language.

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