may be driven by cognitive rather than motoric status at baseline, supporting MCR as a cognitive pre-dementia syndrome.

P2-610 CIGARETTE SMOKING AND RISK OF DEMENTIA IN A KENTUCKY COHORT: A COMPETING RISK ANALYSIS

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Background: The role of smoking in the genesis of dementia is not well understood. We re-examined this issue using a longitudinally followed cohort of elderly participants living in a high tobacco producing area. **Methods:** This study is based on 531 subjects enrolled in the Biologically Resilient Adults in Neurological Studies (BRAiNS) at the University of Kentucky's Alzheimer's Disease Center. At baseline, all subjects were cognitively intact. The dependent variable was the time on study until a diagnosis of dementia, which was determined using a consensus review. Smoking status at baseline (none, former smoker, current smoker) was assessed as a predictor for incident dementia using two models. (1) A Cox proportional hazards model, adjusted for baseline covariates: age, low education, sex, diabetes, myocardial infarction, high blood pressure, *APOE ε*4 carrier status, family history of dementia, and use of hormone replacement therapy. (2) The Fine-Gray proportional hazards sub-distribution model included the same adjustment variables; additionally, the Fine-Gray model accounts for the competing risk of death without dementia. **Results:** Mean age at enrollment was 73.2±7.4 years; participants were majority female (63.1%) and highly educated (mean 16.0±2.4 years). Smoking prevalence was high in respect to general population estimates: 49 (9.2%) participants declared current smoking status (median pack years 47.2) and 231 (43.5%) declared former smoking status (median pack years 24.5). The cohort was followed for an average of 11.5 years, during which time 111 (20.9%) were diagnosed with dementia, while 242 (45.6%) died without dementia. The Cox model showed that former smokers had an adjusted hazard rate (HR) of 1.66 (P=0.017) and current smokers had HR=0.99 (P=0.98). The Fine-Gray model, however, showed an adjusted HR=1.26 (P=0.26) for former and HR=0.61 (P=0.61) for current smokers indicating no increased dementia risk associated with smoking. In contrast, current smoking was a strong risk for death without dementia (adjusted HR=2.07, P=0.003). **Conclusions:** Once adjusted for the competing risk of death without dementia, smoking was not associated with incident dementia in our cohort.

P2-611 GEOGRAPHIC INEQUALITIES IN RATE OF MEMORY DECLINE AMONG OLDER ADULTS IN THE UNITED STATES

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Background: Stark geographic inequalities in health and life expectancy divide the United States (U.S.), with residents of the rural south experiencing some of the poorest health outcomes. Life-course epidemiology suggests childhood place of residence may be particularly relevant for late-life health, but few prior studies have linked geographic place of residence in childhood to late-life cognitive outcomes. We evaluated whether childhood residence in the rural southern U.S. was associated with late-life memory decline among U.S.-born participants of a nationally-representative study of adults age 50 years and older. **Methods:** Health and Retirement Study participants (n=12,806) were interviewed biennially from 1998-2014. Rural southern childhood residence (yes/no) was based on self-reported rurality (rural or non-rural) and state of childhood residence. Composite memory (z-scored) was assessed using immediate and delayed word list recall or the Informant Questionnaire for Cognitive Decline. We used linear mixed effects models (with age in years as the timescale and linear splines with a knot at age 70 to account for nonlinearities) to estimate the effect of rural southern childhood residence (yes/no) on rate of memory decline, accounting for practice effects, adjusting for sex, race, birth year, and parental educational attainment, and applying sampling weights. **Results:** Mean age in 1998 was 64.5 years (range 50-102); 17.9% of the sample reported rural southern childhood residence. Participants were followed for a mean of 12.7 years. Average annual rate of change in memory after age 70 among unexposed (non-rural and non-southern childhood residence) participants was -0.755 (95% confidence interval: -0.796, -0.714). On average, participants with rural southern childhood residence experienced approximately 10% faster rates of memory decline compared with others (for estimated effect of rural southern childhood residence on annual rate of change after age 70, beta = -0.072; 95% confidence interval: -0.101, -0.043) (Figure 1). **Conclusions:** Childhood residence in the rural south may influence late-life memory decline through multiple pathways, including educational experience, labor market opportunities, and cardiometabolic health. Future studies are needed to investigate the lifecourse pathways driving this inequality, the extent to which geographic inequalities contribute to racial inequalities, and potential population-level interventions to promote equity in cognitive aging.

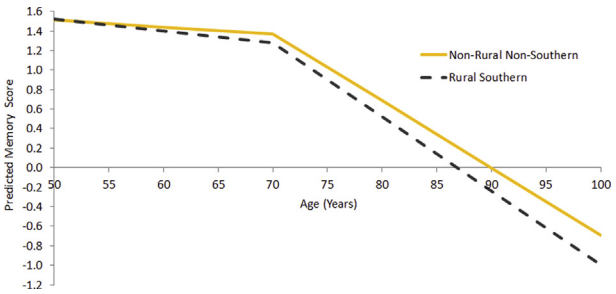

Figure 1. Predicted memory trajectories by rural southern childhood residence (yes/no) from adjusted linear mixed effects models. Shown for a non-Latino white woman born in 1930 whose mother and father had <8 years of education.

P2-612 THE IMPACT OF NUTRIENT PATTERNS ON COGNITIVE DECLINE AMONG SWEDISH OLDER ADULTS: A POPULATION-BASED LONGITUDINAL STUDY

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Background: The impact of nutrient patterns on cognitive decline remains unclear. We thought to identify major nutrient patterns and to explore their association with cognitive decline change over time among the Swedish older adults. **Methods:** In a population-based cohort, 2250 cognitively healthy people aged ≥60 years were identified at baseline (2001-2004), and followed-up to 9 years. Global cognitive function was tested with the Mini-Mental State Examination (MMSE) at baseline and follow-ups. Nutrient intake was assessed using a 98-semi-quantitative food frequency questionnaire (SFFQ) at baseline, and nutrient patterns were derived using principal components analysis based on 30 nutrients. Mixed-effects linear regression models were used to determine their association with change in cognitive function taking into account potential confounders. **Results:** Four major patterns (the plant-, animal-, and milk-derived nutrients and animal/plants-derived fats) were identified. In multiple-adjusted mixed effect model, the higher intake of plant- (β=0.081, P=0.002) and animal-derived nutrients (β=0.098, P<0.001) patterns was significantly associated with slower decline in MMSE score, whereas the milk nutrients pattern was associated with a faster cognitive decline (β=-0.064, P=0.014). No significant association with animal/plants fats

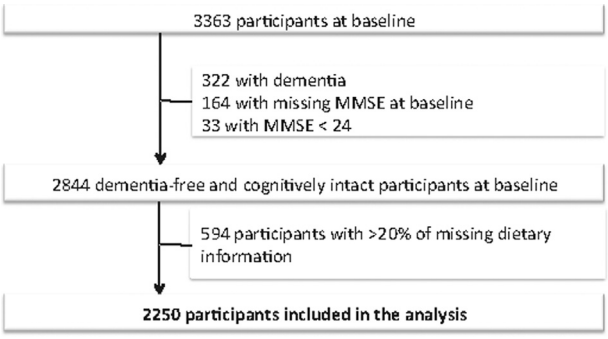

Figure 1. Flow chart and timeline of SNAC-K Study sample
Abbreviation: MMSE, Mini-Mental State Examination.

Table 1
Baseline characteristics of free-dementia and cognitive intact participants according to availability of dietary information on the SFFQ (n=2844)

Characteristics	Included (n=2250)*	Excluded (n=594)	*P-value*
Age			
60-66	1128 (50.1)	135 (22.7)	
72-78	721 (32.0)	142 (23.9)	
81-87	323 (14.4)	163 (27.4)	
90+	78 (3.5)	154 (25.9)	<0.001
Sex			
Males	873 (38.8)	192 (32.3)	
Females	1377 (61.2)	402 (67.7)	<0.001
Educational level			
Elementary	284(12.6)	134 (22.8)	
High school	1130(50.2)	311 (52.9)	
University	896 (39.8)	143 (24.3)	<0.432
Civil status			
Married	1,139(50.7)	186 (31.6)	
Widow (er)/divorced	749 (33.3)	291 (49.4)	
Unmarried	359(16.0)	112(19.0)	<0.001
BMI (mean ± SD)	25.91 ± 4.0	24.90 ± 4.4	<0.001
Physical activity			
Inadequate	479 (21.3)	292 (49.2)	
Health-enhancing	1,195 (53.1)	245 (41.3)	
Fitness-enhancing	576 (25.6)	57 (9.60)	<0.001
Smoking status			
Never	982 (43.9)	302 (51.5)	
Former	912(40.8)	199 (34.0)	
Current smoker	341 (15.3)	85 (14.5)	0.003
Alcohol consumption			
No/occasional	612(27.3)	302 (51.7)	
Light-to-moderate	1,377 (61.3)	250 (42.8)	
Heavy drinking	256(11.4)	32 (5.5)	<0.001
Vascular disorders	1,695 (75.3)	480 (80.8)	0.005
Metabolic disorders	1,278 (56.8)	290 (48.8)	0.001
Cancer	179 (8.0)	73 (12.3)	0.001
Depression symptoms	169(11.5)	68 (7.5)	0.002
APOE ε4	631 (29.5)	134 (27.8)	0.466
MMSE score (median, iqr)	29(1)	28 (2)	<0.001

Data are presented as means ± standard deviations or numbers (proportions). Abbreviations: BMI, body mass index; APOE ε4, apolipoprotein ε4 allele; MMSE, Mini-Mental State Examination. * Number of people with missing values: 1 for education, 3 for civil status, 26 BMI, 15 for smoking, 5 for alcohol consumption, 109 for *APOE* ε4.

Table 2
Factor loading matrix for the four nutrient dietary patterns identified by PCFA carried out on 30 nutrients (n=2250).

	Nutrients-based patterns			
Nutrients*	Plant-derived nutrients	Animal- derived nutrients	Milk- derived nutrients	Animal and Plant Fats
Total protein	-	0.77	0.54	-
Fibre	0.82	-	-	-
Disaccharides	-	-0.43	0.51	-
Monosaccharides	0.72	-0.37	-	-
Starch	0.31	-	-	-0.30
Cholesterol	-	0.50	-	0.61
SFAs	-0.63	-	-	0.48
MUFAs	-0.56	-	-	0.65
PUFAs	-	-	-0.37	0.48
Iodine	-	0.69	-	-
Phosphate	-	0.42	0.82	-
Iron	0.28	0.40	-	-
Calcium	-	-	0.89	-
Potassium	0.81	-	0.36	-
Sodium	-	0.79	-	-
Magnesium	0.73	-	0.32	-0.34
Selenium	-	0.70	0.43	0.35
Vitamin C	0.85	-	-	-
Vitamin B1	0.57	0.34	0.33	-
Vitamin B2	-	-	0.90	-
Vitamin B3	0.34	0.79	-	—
Vitamin B6	0.72	0.29	-	-
Folic acid	0.86	-	0.30	-
Vitamin B12	-	0.49	0.54	0.26
Vitamin K	-	0.26	-	0.45
Vitamin D	-	0.51	-	0.33
Vitamin E	0.44	-	-	0.67
Zinc	-	0.72	0.45	-
Retinol	-0.39	-	0.28	0.30
β-carotene equivalents	0.70	-	-	-
Total var. expl. %	***28.16***	***21.67***	***17.93***	***10.96***
Cumulative var. expl. %	***28.16***	***49.83***	***67.75***	***78.72***

* Energy-adjusted nutrients.

Abbreviation: SFAs, saturated fatty acids; MUFAs, monounsaturated fatly acids; PUFAs, polyunsaturated fatty acids. The importance of the corresponding nutrient to the factor was measured by the magnitude of each loading measures. Loadings $\geq$0.40 in absolute value define the main nutrients for each factor and were shown in bold typeface; loadings <0.25 in absolute value were suppressed.

pattern was observed. In secondary analyses, the slower decline in global cognitive function associated with high intake of plants- and animal-derived nutrient pattern, was stronger in *APOE* ε4 carriers than in ε4 non-carriers. **Conclusions:** Plant- and animal -derived nutrients are associated with preserved cognitive function especially among the *APOE* ε4 carriers, whereas nutrients derived from milk products may accelerate cognitive decline in the Swedish older adults.

Table 3
Association between nutrients-based patterns and three years mean change in MMSE scores

Variables	Model[1]		Model[2]	
	β-coefficient (95% CIs)	*P-value*	β-coefficient (95% CIs)	*P-value*
Nutrients-derived patterns (continuous) x time[a]				
Plant- derived nutrients	0.085 (0.033, 0.137)	0.001	0.081 (0.029, 0.133)	0.002
Animal- derived nutrients	0.098 (0.047, 0.150)	<0.001	0.098 (0.046, 0.149)	<0.001
Milk- derived nutrients	-0.064 (-0.116, -0.013)	0.014	-0.064 (-0.116, -0.013)	0.014
Animal/plant fats	-0.021 (-0.073, 0.032)	0.437	-0.019 (-0.071, 0.033)	0.467

Abbrevation: MMSE, Mini Mental State Examination

β-coefficients (95% confidence intervals) and *P*-values represent three years change in mean MMSE score per 1-unit increase in nutrients pattern score. Positive β-coefficients indicate that an increase in the nutrients pattern intake was associated with the decreased rate of decline in cognitive function during follow-up.[a] Change in MMSE score over time attributable to nutrients patterns. Model[1] included terms for age, sex, education, and included all the nutrients patterns simultaneously. Model[2] included also terms for civil status, physical activity, smoking, and total energy intake (Kcal).