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Research Article

Declining Incident Dementia Rates Across Four Population-Based Birth Cohorts

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Abstract

Background: Incidence rates of dementia appear to be declining in high-income countries according to several large epidemiological studies. We aimed to describe declining incident dementia rates across successive birth cohorts in a U.S. population-based sample and to explore the influences of sex and education on these trends.

Methods: We pooled data from two community-sampled prospective cohort studies with similar study aims and contiguous sampling regions: the Monongahela Valley Independent Elders Survey (1987–2001) and the Monongahela-Youghiogheny Healthy Aging Team (2006–Ongoing). We identified four decade-long birth cohorts spanning birth years 1902–1941. In an analysis sample of 3,010 participants (61% women, mean baseline age = 75.7 years, mean follow-up = 7.1 years), we identified 257 cases of incident dementia indicated by a Clinical Dementia Rating of 1.0 or higher. We used Poisson regression to model incident dementia rates by birth cohort, age, sex, education, and interactions of Sex × Cohort and Sex × Education. We further examined whether cohort effects varied by education, testing a Cohort × Education interaction and stratifying the models by education.

Results: Compared to the earliest birth cohort (1902–1911), each subsequent cohort had a significantly lower incident dementia rate (1912–1921: incidence rate ratio [IRR] = 0.655, 95% confidence interval [95% CI] = 0.477–0.899; 1922–1931: IRR = 0.387, 95% CI = 0.265–0.564; 1932–1941: IRR = 0.233, 95% CI = 0.121–0.449). We observed no significant interactions of either sex or education with birth cohort.

Conclusions: A decline in incident dementia rates was observed across successive birth cohorts independent of sex, education, and age.

Keywords: Epidemiology, Cognition, Cognitive aging, Community-based.

Worldwide, 10 million people develop incident dementia annually. Women make up the majority of these cases. The World Health Organization predicts that prevalence will continue to increase as the population age distribution shifts to increasingly older ages (1).

Cohorts are groups of individuals that share a common experience or characteristic, such as decade of birth. Birth cohort analyses have explored trends in incident dementia as more recent birth cohorts (born in the 1930s or 1940s) have started to age into dementia risk age ranges, allowing for rate comparison with earlier birth cohorts. Several large population-based studies in high-income countries have demonstrated declining incident dementia rates among more recent birth cohorts (2–6), although others have not (7,8). This observation is consistent with cohort studies demonstrating improving cognitive function and resistance to cognitive decline across successive cohorts (9–11). Factors potentially explaining this trend include better cardiovascular risk factor control and declining smoking habits, both of which have occurred in the 20th century (12). Additionally, education has been demonstrated to be a protective

factor for dementia diagnosis in late life (13). Although general educational attainment has improved overall in the United States, the greatest improvements have been observed in women (14). There is, therefore, a need to investigate more nuanced trends in cohort effects for dementia incidence, including potential moderating effects of sex and education.

We have previously reported improving age-associated cognitive trajectories in several cognitive domains across four successive birth cohorts born between 1902 and 1941 within a U.S. population of relatively low education and socioeconomic status (15,16). Notably, these cohort effects remained significant even after adjustment for educational attainment. In the same study, we now investigated birth cohort effects in incident dementia rates. Specifically, we examined four aims: (1) whether the birth cohort effect would be attenuated after adjusting for education, (2) whether the birth cohort effects could differ by sex, (3) whether education effects could differ by sex with adjustment for cohort, and (4) whether the trends observed are applicable only to a specific education group.

Methods

Study Participants

We pooled data from two large prospective population-based studies conducted between 1987 and present. The Monongahela Valley Independent Elders Survey (MoVIES) ran from 1987 until 2001, with biennial assessments of an initial sample of 1681. The Monongahela-Youghiogheny Healthy Aging Team (MYHAT) study is ongoing since 2006, with annual assessments of an initial sample of 1982. Both studies were conducted in the rural Monongahela Valley region of southwestern Pennsylvania. Both recruited individuals aged 65 and older by age-stratified random sampling from the voter registration lists for the targeted communities. The MoVIES study focused on the epidemiology of dementia, whereas the ongoing MYHAT study focuses on the epidemiology of mild cognitive impairment and its transition to dementia. MoVIES required participants to be fluent in English, have at least sixth grade education, not have severe hearing or vision impairment, and not be decisionally incapacitated at enrollment. MYHAT also required sufficient hearing, vision, and decisional capacity, and although not required, all participants had at least sixth grade education and were fluent in English. However, given its mild cognitive impairment focus, MYHAT excluded individuals who scored less than 21 on the age- and education-corrected Mini-Mental State Examination (MMSE) at enrollment (17,18). Further details regarding recruitment and assessment procedures have been reported previously for MoVIES (19,20) and MYHAT (21,22). Both studies were approved by the University of Pittsburgh Institutional Review Board and all participants provided written informed consent.

Between MoVIES and MYHAT, we identified four decade-long birth cohorts of substantial sample size: 1902–1911, 1912–1921, 1922–1931, and 1932–1941. The present analyses excluded 14 participants born before 1902 and 48 participants born after 1941. Education was categorized into three levels: less than high school, completed high school but no college education, and some college education or higher.

Assessment Protocols

In MoVIES, all participants underwent a detailed interview every 18–24 months, including neuropsychological and functional assessments. Those meeting operational criteria for cognitive impairment at baseline and follow-up visits, and for cognitive decline at follow-up visits, and a random sample of unimpaired participants were selected for a second-stage examination consisting of a dementia assessment including a Clinical Dementia Rating (CDR) (19). In MYHAT, all participants underwent a single-stage annual comprehensive assessment including a CDR (23).

Incident Dementia Diagnosis

In this study, dementia was operationally defined as a CDR of 1.0 or higher. This standard definition (24), based on cognitively driven everyday functional abilities, allows independent evaluation of neuropsychological data as predictors of dementia. Incident cases were those who were free of dementia at study entry and developed dementia subsequently. Estimated date of onset was calculated as the midpoint date between a study visit where a participant was scored a CDR of at least 1 and previous study visit with CDR of less than 1. In MYHAT, this was straightforward because all participants were rated on the CDR annually. However, because of the two-stage design in MoVIES, there were 98 incident dementia cases whose first CDR was at least 1 and, therefore, did not have a previous CDR date. In these cases, onset date was estimated using the midpoint between the study visit when the participant was scored a CDR of at least 1 and the most recent previous study visit that included cognitive and functional assessments, but not a CDR rating. We excluded these 98 cases from the primary analyses to ensure consistency of method across all incident cases (23), but later included them in a sensitivity analysis. We identified 257 incident dementia cases across both studies.

Statistical Analyses

We first described demographics, follow-up, and distribution of MoVIES and MYHAT for the total analysis sample and by birth cohort. We then calculated person-year incidence rates by birth cohort and age at dementia diagnosis. We used Poisson regression to model incident dementia as a function of birth cohort, age at study entry, and sex, with a natural log person-years follow-up offset term. To examine Aims 1–3, we built a series of nested models adding education and interaction terms of Sex × Cohort and Sex × Education. To test Aim 4, we tested a separate model including a Cohort × Education interaction term while adjusting for sex and age. We further explored this aim by modeling cohort, sex, and age stratified by education level. We performed sensitivity analyses including the remaining 98 MoVIES participants with incident dementia without a prior CDR and separately included covariate adjustment for baseline unadjusted MMSE to account for study exclusion criteria differences. We used SAS, version 9.4 (SAS Institute), and SPSS, version 24, for all statistical analyses.

Results

The final analysis sample included 3,010 participants with at least one follow-up visit, among whom we identified 257 incident dementia cases over 21,266.2 person-years of follow-up. Table 1 displays demographic statistics for the analysis sample by birth cohort. As shown in Table 2, the overall incidence rate per 100 person-years was 3.227 in the 1902–1911 birth cohort, 1.305 in the 1912–1921 cohort, 1.000 in the 1922–1931 cohort, and 0.300 in the 1932–1941 cohort.

As shown in Table 3, Poisson regression analyses of incident dementia as a function of birth cohort, baseline age, sex, education,

Table 1. Description of the Overall Analysis Sample (N = 3,010) and by Birth Cohort

	1902–1911	1912–1921	1922–1931	1932–1941	Total
Analysis sample, *n*	308	1,170	931	601	3,010
Incident dementia cases, *n*	63	123	59	12	257
Baseline age, mean (*SD*)	80.37 (3.70)	74.93 (7.84)	78.61 (5.81)	70.27 (2.90)	75.69 (6.93)
Sex (female), %	61.00	59.70	61.70	60.70	61.00
Education, %					
Less than HS	59.10	35.50	14.10	6.20	25.40
HS	16.90	40.20	47.00	44.40	40.80
Higher than HS	24.00	24.40	38.90	49.40	33.80
Person-years, mean (*SD*)	6.34 (4.06)	8.06 (4.52)	6.32 (3.62)	6.66 (2.98)	7.07 (4.01)
Study (MYHAT), %	2.30	22.60	85.80	100	55.50

Note: HS = high school; MYHAT = Monongahela-Youghiogheny Healthy Aging Team.

Table 2. Incident Dementia Rates by Birth Cohort and Age at Diagnosis

	Age at Diagnosis, y						
Birth Cohort	<70	70–75	75–80	80–85	85–90	≥90	Total
1902–1911 (*n* = 308)							
Cases	0	0	0	15	36	12	63
PY	0	0	328.04	850.69	591.17	182.55	1,952.46
Rate per 100 PY	0	0	0	1.763	6.090	6.574	3.227
1912–1921 (*n* = 1,170)							
Cases	0	7	35	34	21	26	123
PY	663.53	2,684.76	3,233.16	1,561.3	663.6	618.74	9,425.1
Rate per 100 PY	0	0.261	1.083	2.178	3.165	4.202	1.305
1922–1931 (*n* = 931)							
Cases	0	1	5	15	36	2	59
PY	500.9	548.36	1,031.21	2,015.22	1,563.51	227.44	5,886.65
Rate per 100 PY	0	0.182	0.485	0.744	2.303	0.879	1.000
1932–1941 (*n* = 601)							
Cases	1	3	6	2	0	0	12
PY	588.19	1,740.84	1,391.4	281.5	0.15	0	4,002.08
Rate per 100 PY	0.170	0.172	0.431	0.710	0	0	0.300
Total (*N* = 3,010)							
Cases	1	11	46	66	93	40	257
PY	1,752.62	4,973.96	5,983.82	4,708.72	2,818.43	1,028.73	21,266.3
Rate per 100 PY	0.057	0.221	0.769	1.402	3.300	3.889	1.208

Note: PY = person-years

and interaction terms of Sex × Education and Sex × Birth Cohort revealed the following results. Compared to the 1902–1911 referent cohort, each subsequent cohort had a lower incident dementia rate in every model tested. In an age- and sex-adjusted model (Model 1), the cohort effect was significant, with lower incident dementia rates in the more recent cohorts. Results were consistent in Model 2, which tested whether this cohort effect would be attenuated by adjustment for education (Aim 1). Older baseline age, but not sex or educational levels, was related to higher incident dementia rates. In Bonferroni-adjusted pairwise comparisons, the incident dementia rates for the 1932–1941 and 1922–1931 birth cohorts were also significantly lower than the rate for the 1912–1921 cohort ($p < .001$). However, there was no significant difference between the 1932–1941 and 1922–1931 birth cohorts ($p = .121$).

We further tested the hypothesis that education or cohort effects could differ by sex (Aims 2 and 3) by including interaction terms of Sex × Education and Sex × Cohort (Model 3), but observed no significant interaction terms. Although college-educated women had a marginally lower incidence rate than women who did not graduate high school, this rate ratio was not statistically significant with adjustment for multiple comparisons ($\alpha = .017$; incidence rate ratio [IRR] = 0.648, 95% confidence interval [CI] = 0.391–1.076, $p = .041$).

To test Aim 4, whether the observed declining incident dementia rate was applicable only to a certain level of education, we tested a separate model (not shown) with an interaction term of Cohort × Education, which was not significant. Additionally, we stratified our sample into two education groups: those with at least some college education and those with no college education (Table 4). We observed the same trend in both education groups, with a significantly lower incident dementia rate observed in the more recently born cohorts. Effect sizes were slightly higher in the college-educated sample compared to the no college education sample when comparing the 1912–1921 and 1932–1941 cohorts to the 1902–1911 referent cohort.

To test the Poisson regression assumption of constant rate, we also modeled an interaction term of person-years follow-up (median split at 7.78 years) by birth cohort. More years of follow-up were associated with higher incident dementia rates, but there was no significant interaction between years of follow-up and birth cohort, suggesting this effect was consistent across each cohort.

Table 3. Poisson Regression Models Predicting Incident Dementia From Birth Cohort (N = 3,010)

Basic Models	IRR	95% CI for IRR	p
Model 1			
Born 1902–1911 (referent)			
Born 1912–1921	0.639	0.469–0.870	.005
Born 1922–1931	0.358	0.251–0.511	<.001
Born 1932–1941	0.212	0.111–0.402	<.001
Baseline age, y	1.089	1.070–1.109	<.001
Female sex	0.982	0.761–1.268	.891
Model 2			
Born 1902–1911 (referent)			
Born 1912–1921	0.655	0.477–0.899	.009
Born 1922–1931	0.387	0.265–0.564	<.001
Born 1932–1941	0.233	0.121–0.449	<.001
Baseline age, y	1.088	1.069–1.108	<.001
Education: less than HS (referent)			
HS graduate	0.883	0.653–1.194	.419
College or higher	0.773	0.558–1.072	.122
Female sex	0.990	0.766–1.280	.940
Interaction model	**β**	**95% CI for β**	**p**
Model 3			
Born 1902–1911 (referent)			
Born 1912–1921	−0.740	−1.286 to −0.194	.008
Born 1922–1931	−1.372	−2.037 to −0.708	<.001
Born 1932–1941	−1.723	−2.763 to −0.683	.001
Baseline age, y	0.085	0.067–0.102	<.001
Education: less than HS (referent)			
HS graduate	0.168	−0.379 to 0.714	.548
college or higher	0.126	−0.429 to 0.680	.657
Female sex	−0.076	−0.619 to 0.468	.785
Female and HS graduate	−0.409	−1.066 to 0.248	.223
Female and college or higher	−0.559	−1.251 to 0.133	.114
Female and born 1912–1921	0.429	−0.238 to 1.096	.208
Female and born 1922–1931	0.586	−0.225 to 1.396	.157
Female and born 1932–1941	0.313	−1.000 to 1.625	.641

Note: IRR = incident rate ratio; HS = high school; referents: born 1902–1911, less than HS education, male sex. Estimated coefficients (β) are reported for the interaction model as the exponentiated coefficients (IRR) are non-informative for the interaction terms.

Sensitivity Analyses

We refit Model 2 after adding to the analytic sample the 98 MoVIES incident dementia cases without a previous CDR. Among them, 89 were born between 1902 and 1941, bringing the final analysis sample for the sensitivity analyses to 3,099 participants with 346 incident dementia cases. The observed birth cohort effect did not differ from the primary analysis, including the results of pairwise comparisons. However, higher educational attainment was associated with lower incident dementia rates when comparing the college-educated participants to those who did not graduate high school (IRR = 0.731, 95% CI = 0.5480.974, p = .033). Additionally, those who graduated high school had a marginally lower risk than those who did not, although this was not statistically significant (IRR = 0.775, 95% CI = 0.590–1.017, p = .066).

To account for differences in study inclusion criteria between MYHAT and MoVIES, we tested the influence of baseline function in the model by adjusting Model 2 for baseline-unadjusted MMSE score. Higher baseline MMSE score was associated with a lower incident dementia rate (IRR = 0.854, 95% CI = 0.825–0.884, p < .001). The birth cohort effect was unchanged from the primary analysis, with successive birth cohorts still evidencing significantly lower incident dementia rates compared to the 1902–1911 referent cohort (1912–1921: IRR = 0.698, 95% CI = 0.5060.963, p = .028; 1922–1931: IRR = 0.406, 95% CI = 0.2770.594, p < .001; 1932–1941: IRR = 0.226, 95% CI = 0.1170.436, p < .001).

Discussion

By pooling data from two large methodologically similar population-based studies in southwestern Pennsylvania, we compared incident dementia rates across four birth cohorts born in the early 1900s. We observed a decline in the incident dementia rate for each subsequent cohort when compared to those born in the earliest 1902–1911 cohort, which persisted after adjustment for age, sex, and educational attainment. Specifically, we report a 77% reduction in the incident dementia rate when comparing the latest born 1932–1941 cohort to the earliest born 1902–1911 cohort. This trend is consistent with several other epidemiological studies demonstrating a decline in incident dementia in Western countries (2–6), although not all studies have observed this trend (7,8).

In the Einstein Aging Study in New York, a decrease in dementia incidence among individuals born after 1929, most notably an 85% lower incidence rate in older white women, was unexplained by education, race, sex, or cardiovascular comorbidities (2). The Rotterdam study observed a 25% dementia rate reduction between

Table 4. Education-Stratified Poisson Regressions Predicting Incident Dementia From Birth Cohort

	No College Education (N = 1992)			Some College Education (N = 1018)		
	IRR	95% CI for IRR	p	IRR	95% CI for IRR	p
Born 1902–1911 (referent)						
Born 1912–1921	0.704	0.495–1.000	.050	0.404	0.205–0.799	.009
Born 1922–1931	0.325	0.207–0.509	<.001	0.376	0.199–0.709	.003
Born 1932–1941	0.316	0.151–0.663	.002	0.098	0.027–0.363	.001
Female sex	1.060	0.784–1.432	.707	0.754	0.456–1.247	.272
Baseline age, y	1.087	1.065–1.108	<.001	1.094	1.049–1.141	<.001

Note: IRR = incident rate ratio; referents: born 1902–1911, male sex.

the subcohort starting in 2000 compared to the subcohort starting in 1990 (3). The Framingham heart study observed a rate reduction as high as 44% comparing epochs in the late 2000s to early 2010s and the late 1970s to early 1980s (4). A report from Stockholm inferred a decrease in dementia incidence based on prevalence remaining stable despite increasing survival times (25). It is difficult to compare the educational levels of all these studies because they are reported differently and may also be qualitatively different between European and American populations. However, the Framingham population had a higher average educational level (64%–95% high school graduates) compared to our Monongahela Valley population sample (41%–94% high school graduates).

Importantly, these declining trends have not been universally reported. No recent change in risk for Alzheimer's disease was found in an urban community–based representative cohort in Chicago (7). In a national sample from Wales, the incidence rate of Alzheimer's disease was estimated to be 1.3 times higher in 2010 than in 1999 (8). Studies using U.S. Medicare claims data suggested a significant increase in clinically diagnosed Alzheimer's disease from the 1980s to 2000 (26,27). In the national diagnostic databases, the increasing trend may reflect enhanced awareness of Alzheimer's disease.

Regarding sex differences, a 20% overall reduction in dementia incidence in the Cognitive Function and Ageing Study (England and Wales) appeared to be largely driven by a reduction in rates among men (28). Conversely, evidence from the Personnes Agées QUID study in France reported a decline in algorithmically diagnosed dementia that was entirely driven by a decreasing rate in women (6). We observed trends of declining incidence in both men and women.

Racial/ethnic differences could not be investigated in our largely white study populations; however, the Washington Heights-Inwood Columbia Aging Project reported a 41% reduction in dementia hazard rate comparing a 1999 cohort to a 1992 cohort, with reduction being greatest among non-Hispanic whites and African Americans and lowest among Hispanics (29).

Regarding educational levels, we observed no significant education effect predicting incident dementia, nor did the inclusion of education in our models significantly attenuate the observed cohort effect. In stratified analyses, we observed declining incident dementia rates in both college-educated and noncollege-educated participants, but the cohort effect appeared to be stronger in those who were college educated. Higher educational attainment may reflect higher intellectual capacity, where educational opportunities are uniform, but education may also promote synaptic density and dendritic branching, resulting in higher cognitive reserve and resilience against late-life decline (30). Education patterns have changed considerably across the four birth cohorts we examined. For example, 41% of participants born in the 1902–1911 cohort graduated high school compared to 94% among those born in the 1932–1941 cohort. Additionally, higher education may promote continued intellectual attainment and building of cognitive reserve, but also positions individuals for cognitively stimulating careers requiring lifelong education, and potentially better financial and health care security.

Changes in education patterns for women deserve specific attention. The proportion of both men and women attaining college degrees has dramatically increased in the 20th century, but women have made particularly great strides transitioning from traditional homemakers to highly educated skilled laborers (14). This trend is critically relevant to the burden of dementia in the population, as longer-living women make up the majority of cases. In our study, across all birth cohorts, college-educated women had a 35% lower incident dementia rate than women who did not graduate high school, but we lacked sufficient power to observe a statistically significant interaction between sex and education.

The Flynn Effect is a phenomenon in which earlier birth cohorts are consistently outperformed on intelligence quotient tests by subsequent birth cohorts (31). Consequently, later birth cohorts may simply perform better on cognitive assessments and avoid dementia diagnosis later into old age than earlier cohorts. It is also possible that the intelligence quotient trend reflects brain mechanisms consistent with cognitive reserve theory, especially considering the substantial changes in education patterns across 20th-century birth cohorts. However, we diagnosed dementia using the CDR, which classifies dementia severity based on cognitively driven everyday functioning rather than neuropsychological test performance, avoiding confounding by education and intelligence quotient. Additionally, adjustment for baseline MMSE did not explain the observed cohort effect.

Strengths and Limitations

The Monongahela Valley comprises several Rust Belt communities in southwestern Pennsylvania that have been economically depressed since the collapse of the steel industry in the late 1970s. As an underserved small-town area, it represents a type of population that is rarely targeted for health research. As a consequence of its lower socioeconomic status, the region has a stable population with minimal in- and out-migration, which facilitates longitudinal research. As both MoVIES and MYHAT samples comprised predominantly white population, reflecting the stable demographics of the region, our findings will need to be replicated in ethnic minority populations.

Both studies were performed sequentially in the same region, providing a rare opportunity to pool their samples and identify four substantially sized birth cohorts with a collective follow-up of more than 21,000 person-years. Both aimed to study cognitive impairment in the general older population and were large community-based samples, randomly selected from the public electoral rolls. Thus, the influence of selection bias, which threatens the external validity of clinic-based studies (32), was minimized.

Both studies had almost identical inclusion criteria and methods, with the exceptions described and accounted for earlier; thus, we were able to detect incident cases and estimate their dates of onset consistently. The biennial MoVIES assessments, compared to the annual MYHAT assessments, potentially introduced a bias toward earlier dementia detection in MYHAT. However, this bias is toward the null as we predicted higher incident dementia rates in the earlier born cohorts, which primarily come from MoVIES.

Notably, during a five-year gap from 2001 to 2006 between the end of MoVIES and start of MYHAT, we collected no data and identified no incident cases. Thus, we are likely missing some incident cases for the 1912–1921 and 1922–1931 cohorts, the two cohorts with the most overlap between studies. We could not statistically adjust for a study effect because of collinearity with birth cohort (see Table 1).

The relatively few (n = 12) cases of incident dementia identified in the most recent cohort (1932–1941) likely contribute to some imprecision in our estimated effects for this group. Although this low count is likely related to younger age, total years of follow-up were comparable in this cohort and the preceding three cohorts; thus, the fewer cases may also reflect a true declining rate. Furthermore, when comparing the two cohorts with the highest overlap in age during follow-up (1922–1931 vs 1912–1921), we found a significantly lower incidence rate in the more recent cohort, even though a higher

proportion of this cohort's total years of follow-up were after age 80 (1922–1931: 64.7%, 3,806.2 person-years) than that of the earlier cohort (1912–1921: 30.2%, 2,843.6 person-years; Table 2).

For the present analyses, we focused on all-cause dementia rather than etiological subtypes, given the high likelihood of mixed etiology and the equal public health importance of all dementias (33). We have thus far examined only demographic factors in relation to the observed incidence trends; in the future, we will examine other potential risk factors such as depression, cardiovascular factors, smoking, and lifestyle variables.

Conclusions

Decreasing incident dementia trends are promising for the future of preventive care for a presently irreversible condition. Yet, as overall life expectancy increases and survival with dementia increases, dementia prevalence will remain high even though age of onset may have been delayed. Thus, the welcome reduction in the incidence rate is only one aspect of many developments that will be necessary to avoid a public health crisis. Further research should examine incidence trends in ethnic minorities and low- to middle-income countries, and investigate whether current cardiovascular risk trends, including the obesity and diabetes epidemics, may alter this declining rate in high-income countries.

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Conflict of Interest

None declared.

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