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ARTICLE

Private payer coverage policies for *ApoE-e4* genetic testingJalayne J. Arias, JD, MA¹✉, Ana M. Tyler, JD, MA¹, Michael P. Douglas, MS² and Kathryn A. Phillips, PhD²

PURPOSE: *ApoE-e4* has a well-established connection to late-onset Alzheimer disease (AD) and is available clinically. Yet, there have been no analyses of payer coverage policies for *ApoE*. Our objective was to analyze private payer coverage policies for *ApoE* genetic testing, examine the rationales, and describe supporting evidence referenced by policies.

METHODS: We searched for policies from the eight largest private payers (by member numbers) covering *ApoE* testing for late-onset AD. We implemented content analysis methods to evaluate policies for coverage decisions and rationales.

RESULTS: Seven payers had policies with positions on *ApoE* testing. Five explicitly state they do not cover *ApoE* and two apply generic preauthorization criteria. Rationales supporting coverage decisions include: reference to guidelines or national standards, inadequate data supporting testing, characterizing testing as investigational, or that testing would not alter patients' clinical management.

CONCLUSION: Seven of the eight largest private payers' coverage policies reflect standards that discourage *ApoE* testing due to a lack of clinical utility. As the field advances, *ApoE* testing may have an important clinical role, particularly considering that disease-modifying therapies are under evaluation by the US Food and Drug Administration. These types of field advancements may not be consistent with private payers' policies and may cause payers to reevaluate existing coverage policies.

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INTRODUCTION

ApoE-e4 status is the most significant genetic risk factor for late-onset Alzheimer disease.¹ Alzheimer disease research has seen critical advances that could present a shift in the clinical usefulness of *ApoE* genetic testing. Clinical access to *ApoE* genetic testing for predictive or diagnostic testing related to Alzheimer disease is determined, in part, by payers' coverage policies. Yet, there is a gap in understanding private payers' policies and their rationale for their coverage policies regarding *ApoE* genetic testing. This study examines private payers' coverage policies that apply to *ApoE* genetic testing for late-onset Alzheimer disease. Our objective is to report on our evaluation of private payer coverage policies for *ApoE* genetic testing, examine the rationales, and describe supporting evidence referenced by policies.

Individuals have one of six permutations of the *ApoE* alleles (e2/e2, e2/e3, e2/e4, e3/e3, e3/e4, e4/e4). Among these, one copy of e4 increases the risk for Alzheimer disease by two- to threefold above the general risk for Alzheimer disease.² Two copies of *ApoE-e4* (or e4 homozygote) may increase lifetime risk by up to 15-fold; however, recent studies have shown that the risk might be lower than anticipated and complicated by other risk factors.³ Over the past quarter-century, researchers have continued to provide a clearer understanding between *ApoE* genotypes and other risk factors (age, gender, lifestyle).⁴ *ApoE-e4* status is associated with earlier onset of symptoms and an increased rate of disease progression.² Despite the relationship between *ApoE* and Alzheimer disease, clinical guidelines and standard of care do not support genotyping for *ApoE* for diagnostic or predictive purposes.⁵ Two factors diminish the clinical utility of *ApoE*. First, the lack of disease-modifying therapy in Alzheimer disease reduces the value of identifying individuals who are at an increased risk. This point has been emphasized by professional societies recommending against ordering *ApoE* testing for predicting Alzheimer disease, including the American College of

Medical Genetics and Genomics.⁶ Similarly, in those who are symptomatic, knowing the individual's *ApoE* status bears no relevance for treatment options that currently prioritize symptom management. Finally, *ApoE* is not a causative gene, nor is it necessary or sufficient to cause Alzheimer disease (not everyone who is *ApoE-e4* positive develops Alzheimer disease and not everyone with Alzheimer disease is e4 positive).^{2,4}

Yet, the field is evolving and with it so may the perceived clinical usefulness of *ApoE* genetic testing. *ApoE* genotyping may serve important roles in determining who is at increased risk for adverse effects of future therapies, determine pretest probability for preclinical biomarker screening, and is available through direct-to-consumer (DTC) testing.⁷ As the field advances, payer coverage for testing will be critical for determining who can access *ApoE* genetic testing, which may be qualification criteria for future disease-modifying therapy.²

To date, no other study has evaluated private payer coverage policies for *ApoE* genotyping. This paper fills a significant gap in the literature regarding access to *ApoE* genetic testing. Filling this gap is essential to understanding how *ApoE* testing, and its payer coverage, will evolve alongside the field.

MATERIALS AND METHODS

This study analyzed private payers' coverage policies for *ApoE* genetic testing as a risk factor for late-onset Alzheimer disease. We evaluated each policy to determine (1) the coverage policy for *ApoE* genetic testing clinically for either symptomatic or asymptomatic policyholders, (2) the rationale provided for the coverage policy, and (3) the evidence, including professional guidelines, cited to support the coverage policy.

Sample and data collection

We identified the largest private payers, by membership, from a list of the top eight US private payers on Statista.com and valuepengium.com

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(Supplementary Material).⁸ These eight payers represent approximately 50% (162.8 million members) of the total US population and 57% of the US population eligible for private insurance. We then identified publicly available coverage policies that applied to *ApoE* testing for risk of Alzheimer disease by searching each payer’s website with relevant terms (i.e., “Alzheimer’s” and/or “*ApoE*”) as of 1 January 2020. If a search function was not available, one investigator (M.P.D.) reviewed the list of the payers’ policies for those that may apply. We downloaded applicable policies and two investigators (M.P.D., J.J.A.) abstracted necessary data. Investigators met to reach a consensus regarding the relevance of the abstracted data.

Data analysis

We adopted a content analysis approach to identify themes and phenomena in the text of each policy.⁹ This process included an inductive coding approach to identify themes among the policies, creation of a codebook, and application of the codebook. We uploaded the policies and the codebook into NVivo 12.¹⁰ The codebook included two layers of code. First, we coded whether the payer’s policy covered, did not cover, or used preauthorization criteria for *ApoE* genetic testing. Second, the codebook identified five distinct rationale codes: (1) the association with risk, (2) explicit references to a professional guideline or standard (within the text of the policy), (3) classifying *ApoE* as having “inadequate data” to support testing, (4) labeling *ApoE* as “investigational,” or (5) impact on patient management. Two investigators (J.J.A., A.M.T.) independently coded each policy before meeting to reach consensus on the coding decisions. Any disagreements regarding coding were resolved as a team of investigators. For all coding decisions, agreement was met without requiring a third investigator’s input. The coding results were then transferred to an Excel spreadsheet to chart data for comparison.

RESULTS

Seven of the eight identified payers have policies specific to coverage for *ApoE* testing, accounting for 113.3 million members (ranging from 4.4 million members to 40.2 million members). Of these seven, one payer utilizes a laboratory benefit management (LBM) company to draft its coverage policy.¹¹ Policies were published between 1999 and 2020, with the last year of review (i.e., not all payers review/update their policies on an annual basis) ranging between 2014 and 2020 (Table 1).

Scope and characteristics of policies

Policies vary in whether their scope is specific to Alzheimer disease, Alzheimer disease and related dementias (ADRD), or genetic testing more broadly. To understand the scope and context of the policies, we documented whether policies reference genetic markers and biomarkers besides *ApoE*, including markers specific to early-onset Alzheimer disease (Table 1). Three policies reference risk or causal genetic markers for Alzheimer disease. Among these three policies, two policies reference risk genetic markers for late-onset Alzheimer disease (*ApoE*) and causal genetic markers for early-onset familial Alzheimer disease (*APP*, *PSEN1*, *PSEN2*), as well as nongenetic biomarkers (i.e., amyloid or tau), in the coverage decision. A third policy references Alzheimer disease and genetic testing within the coverage decision and considers *ApoE*, *APP*, *PSEN1*, and *PSEN2* in its rationale. Next, one policy applies to ADRD (e.g., including frontotemporal lobar degeneration or vascular dementia) in combination with genetic markers (*ApoE*, *APP*, *PSEN1*, *PSEN2*) and biomarkers. Last, three of the policies are general genetic testing coverage policies, one of which specifically references late (*ApoE*) and early-onset Alzheimer disease (*APP*, *PSEN1*, *PSEN2*) genetic markers. All seven policies are relevant to our analysis—whether *ApoE* genetic testing is covered for purposes of late-onset Alzheimer disease. Additionally, all seven policies apply similarly to asymptomatic (risk assessment) and symptomatic (diagnostic) enrollees. Three policies do not differentiate between asymptomatic versus symptomatic policy enrollees. Four policies include language that differentiates between asymptomatic and symptomatic testing. However, within these policies, coverage decisions are not impacted by the distinction between asymptomatic and symptomatic testing.

Coverage decisions and rationales

Among the seven payers with policies relevant to *ApoE* genetic testing, five explicitly do not cover *ApoE* genetic testing (Table 2). For example:

Table 1. Policy characteristics.

Payers		1	2	3	4	5	6	7	8	Total enrolled members
Year of last review		2014	2020	2019	2019	2019	2019	2019	No policy	
Number of members (in millions)		4.4	4.4	12.2	14	15.9	22.2	40.2	49.5	162.8
Policy application										
Policy focus (disease or syndrome)	General genetic testing		•	•		•			No policy	
	ADRD						•			
	AD	•			•			•		
Policy focus (specific genetic marker or biomarker)	<i>ApoE</i>	•	Not specified	Not specified	•	•	•			
	<i>APP</i>	•			•	•	•			
	<i>PSEN1/PSEN2</i>	•			•	•	•			
	Other biomarkers or genetic markers for related dementias	•			•		•	•		

AD Alzheimer disease, ADRD Alzheimer disease and related dementias.

Table 2. Private payer coverage decisions and rationales.

Testing coverage	No coverage (policies 1, 2, 4, 5, 7)	Preauthorization requirement (policies 3, 6)
Rationales		
Association with risk	• • •	
Guideline reference	• • • •	
Inadequate data	• • •	
Investigational		•
Patient management	• • • •	
Authorization criteria		• •
Guidelines		
AHRQ (formerly AHCPR)	•	
AAN		•
APA		•
Alzheimer's Association		•
ACMG/NCGC		•
US Task Force		•
Nonspecific		•

AAN American Academy of Neurology, *ACMG/NCGC* American College of Medical Genetics and Genomics/National Society of Genetic Counselors, *AHRQ* Agency for Healthcare Research and Quality, *AHCPR* Agency for Health Care Policy and Research, *APA* American Psychological Association.

“members may NOT be eligible under the Plan for genetic testing for AD including, but may not be limited to, any genes associated with AD (e.g., *ApoE*, *APP*, *PSEN1*, *PSEN2*).”

The seven payer policies include background and supporting sections that include rationales to support the coverage decision (Table 2). We provide definitions of the six coded rationales and example quotes for policy rationales in Table 3. While the wording in the rationales differs, the prominent rationale, “patient management,” reflects a perception that *ApoE* testing would not alter clinical management. This rationale is further expanded by framing *ApoE* genetic testing as investigational or a lack of data to justify coverage for clinical purposes.

Two policies, both with a broader genetic test scope, do not provide rationales but utilize preauthorization criteria (Table 2). These policies provide criteria to determine whether genetic testing is *medically necessary* to warrant authorization. For example:

“Genetic testing is considered medically necessary and may be authorized when all of the following criteria are met [...]”

Guidelines or standards explicitly referenced in payer policies
 Three payer policies explicitly reference professional guidelines or standards as supporting their coverage policy (Table 4). None of these three policies reference the same professional guidelines. One policy does not specify or cite any guideline, describing a lack

of support for use of testing by “nationally recognized peer-reviewed medical literature.” The other two payer policies each cite a total of six standards or professional guidelines, as summarized in Table 4. In addition to the three policies that explicitly cite standards or guidelines within their rationales, all policies included a list of cited references. While policies have been updated as recently as 2020, policies reference data and guidelines that are no longer used within the field. For example, one policy references the 1984 McKhann Diagnostic Criteria—which were updated in 2011 by an Alzheimer’s Association and National Institutes on Aging Workgroup.¹² Additionally, only two policies cite prominent standards for genetic testing by Goldman et al.⁵ Only one policy referenced the 2011 Diagnostic Guidelines (NIA-AA) that reflect the current, evolving view of Alzheimer disease. While the cited standards and professional guidelines are different, they consistently recommend against *ApoE* genotyping.

Preauthorization criteria

Two policies provide preauthorization criteria, all of which must be met, to support coverage for any clinical genotyping covered under their policy. Preauthorizations are done on an individual patient level using standard information and clinical indication for testing (e.g., family history of known genetic marker). The payer will then issue an approval or denial of coverage for the test. A review of the preauthorization criteria within the two policies when applied to *ApoE* genotyping, based on currently available evidence, leads to the presumed conclusion that these two payers would not provide coverage (Table 5). However, these criteria provide nuanced insight into these policies’ rationales in determining that a specific test is medically necessary.

DISCUSSION

This study evaluated coverage policies from seven of the eight largest private payers and found that none cover or would be likely to cover genetic testing for *ApoE*, a risk factor for Alzheimer disease, in either asymptomatic (risk assessment) or symptomatic (diagnostic testing) policyholders. The results demonstrate that five of these private payers explicitly do not cover genetic testing for *ApoE*. The remaining two payers utilize a set of preauthorization criteria, which would unlikely be met in the context of *ApoE* genotyping, to determine coverage for clinical genotyping. Policy rationales supporting coverage decisions focus on clinical utility. The primary rationales include an effect on patient management and whether data are sufficient to support genetic testing. Four of the policies cite that the test would not alter patient management. Three of the policies explicitly reference policies, guidelines, or national standards as part of the rationale. However, these three policies do not reference the same sources. As the field advances, consistency among payers regarding sources to support coverage decisions may advance more equitable access to testing. Policies that used preauthorization include clinical utility, defined as a change in patient management, as one element necessary to justify covering genetic testing. Although this is consistent with the current state of the science, ongoing developments in the field may alter the current interpretation that *ApoE* genotyping does not offer clinical utility.

Efforts to identify disease-modifying therapy for Alzheimer disease are tightly linked to understanding the etiology of the disease, the underlying pathology, and risk factors—including genetic risk factors. The focus on the underlying causes of Alzheimer disease introduces two future clinical uses for *ApoE* genotyping that may increase the likelihood that testing could be medically necessary or offer clinical utility. First, research focusing on biological biomarkers redefined Alzheimer disease according to biological criteria (amyloid and tau).¹² These biomarkers may be identified preclinically in asymptomatic individuals more than a

Table 3. Coding definition and example quotes for policy rationale.

Coded rationale	Definition	Example quote
Association with risk	The insurer's policy discuss the associated risk of APOE for AD or ADRD associated with the specified gene.	<i>APOE is a susceptibility polymorphism; the presence of one or two e4 alleles increases the risk but does not guarantee that someone will develop AD.</i>
Guideline reference	The insurer's rationale for their decision refers to professional guidelines and standards of care for treatment of the specified condition.	<i>The Quality Standards Subcommittee of the American Academy of Neurology (AAN) concluded that there are no laboratory tests (e.g., APOE genotyping, genetic markers or biomarkers) suitable for evaluating and diagnosing patients with AD; genotyping, biomarkers, and imaging are areas to conduct further research for diagnosis.</i>
Inadequate data	The insurer's rationale refers to inadequate data linking genetic testing for the condition(s) with disease management or therapy.	<i>There is insufficient evidence in the peer-reviewed, scientific literature to support the use of APOE testing for the screening, diagnosis or management of cardiovascular disease.</i>
Investigational	The insurer's rationale refers to genetic testing as investigational and medically unnecessary.	<i>These are considered experimental/investigational...</i>
Patient management	The insurer's rationale indicates that pursuit of genetic testing does not affect clinical management of the patient.	<i>APOE genotyping does not reduce the risk of developing Alzheimer disease, change the clinical treatment, or substantially modify disease progression in individuals with Alzheimer disease.</i>
Authorization criteria	Rationale are embedded in criteria for preauthorization	See Table 5.

AD Alzheimer disease, ADRD Alzheimer disease and related dementia.

Table 4. Guidelines or standards explicitly referenced in payer policy.

Source	Cited language provided in payer policies
Ref. ³¹	"The guideline stated that 'it is not yet possible to depend on apoE genotyping for definitive guidance about diagnosis or treatment of Alzheimer disease.'"
Ref. ³¹	The American Academy of Neurology "concluded that there are no laboratory tests (e.g., APOE genotyping, genetic markers or biomarkers) suitable for evaluating and diagnosing patients with AD; genotyping, biomarkers, and imaging are areas to conduct further research for diagnosis."
Ref. ³²	"According to the American Psychiatric Association, providing an Alzheimer disease requires clinical symptomology and microscopic examination of the brain postmortem; 70%–90% of clinical diagnoses match pathological diagnosis postmortem."
Ref. ³³	"The Alzheimer's Association position on genetic testing applies to current tests for early-onset genes and to reliable tests that may eventually be developed to predict late-onset Alzheimer's. Having the APOE-e4 gene goes not mean a person has or will develop AD."
Ref. ³⁴	"The United States Preventive Services Task Force (USPSTF) concludes that the evidence is insufficient to recommend for or against routine screening for dementia in older adults."

AD Alzheimer disease.

decade before the onset of symptoms.¹³ The 2018 NIA-AA Research Framework proposes that evidence of an increased amyloid burden, measured through positron emission tomography (PET) or cerebrospinal fluid (CSF), would indicate that an individual is within the "Alzheimer continuum"—indicating an Alzheimer pathological change.¹³ If this Framework is clinically adopted and biomarker testing becomes standard of care, clinicians will need factors to guide determinations regarding who should be screened. While multiple factors could increase an individual's pretest probability for having an increased amyloid load, ApoE genotype has been shown to be directly related.⁴ Alongside age, ApoE genotype has shown to have a dose-

dependent effect on amyloid brain deposition, making individuals heterozygous or homozygous for an ApoE-e4 allele more likely to be "positive" for amyloid than counterparts.¹⁴ Given this relationship and relevance of ApoE status for age on onset—it is plausible to see ApoE genetic testing as clinically useful to establish pretest probability before pursuing amyloid imaging. Similarly, recent advancements increase the potential for blood-based biomarker tests for predicting and diagnosing Alzheimer disease.¹⁵ As blood-based biomarkers tests become clinically available, ApoE genotyping may serve as a mechanism to narrow the pool of individuals tested. However, it is unclear yet whether amyloid imaging or blood-based biomarker tests would have clinical purposes on their

Table 5. Preauthorization criteria and estimated potential of *ApoE* testing to meet criteria.

Policy	Preauthorization criteria	Supports <i>ApoE</i> testing (authors' estimates)
Policy 1	Test is ordered by board-certified physician within the scope of their practice or a board-certified MD medical geneticist	○
	Pre- and post-test genetic counseling is performed by a board-certified MD medical geneticist, certified genetic counselor, or appropriate MD specialist	○
	The clinical testing laboratory must be accredited by CLIA, the state, and/or other applicable accrediting agencies	○
	Documented key risk factors that suggest a genetic disorder is present (ONE of the following): (1) clinical features indicative of a condition or disease; or (2) high risk of inheriting the disease based upon personal history, family history, documentation of a genetic pathogenic variant, and/or ethnic background; or (3) following history, physical examination, pedigree analysis, and completion of conventional diagnostic testing, a definitive diagnosis remains uncertain and a hereditary diagnosis is suspected	○
	Carrier or predictive testing requires documentation confirming that a causative genetic change has been identified in an affected family member	X
	Documentation is provided that supports the clinical utility of test results that will be used to significantly alter the management or treatment of the disease (e.g., surgery, the extent of surgery, a change in surveillance, hormonal manipulation, or a change from standard therapeutic or adjuvant chemotherapy)	X
Policy 3	Member displays clinical features or is at risk of inheriting pathogenic variant	○
	Results will be used to develop a clinically useful approach or course of treatment OR to cease unnecessary monitoring or treatments for the individual being tested.	X
	Clinically useful test results allow providers to do at least one of the following: (1) inform interventions that could prevent or delay disease onset, (2) detect disease at an earlier stage when treatment is more effective, (3) manage the treatable progression of an established disease, (4) treat current symptoms significantly	X
	Disorder could not be diagnosed through completion of conventional diagnostic studies, pedigree analysis and genetic counseling consistent with the community standards	X
	No previous genetic testing for the disorder (unless significant changes in testing technology or treatments indicate that test results or outcomes may change due to repeat testing	○

Key: For both policies, all criteria must be met prior to pre-authorization for *ApoE* genetic testing. ○ indicates that it is possible for *ApoE* to meet this criteria given current evidence of *ApoE* as a susceptibility gene associated with Alzheimer's disease. X indicates that current evidence would not meet this criteria.

own that would warrant screening. In fact, amyloid imaging, or CSF analysis, has seen similar limitations on payer coverage as *ApoE*.¹⁶ A complete analysis of how *ApoE* and amyloid preclinical biomarker testing compare in the context of private payer coverage should be reserved for future analysis.

However, prospective disease-modifying therapy heightens the future clinical relevance of *ApoE*. Efforts to identify disease-modifying therapy have focused on secondary prevention treatments for individuals who are in the earliest disease stages.¹⁷ If successful, clinicians will need a measure to identify individuals who are most likely to benefit from treatments without the ability to rely on symptom presentation to justify testing. This approach is exemplified in the Biogen aducanumab application submitted to the US Food and Drug Administration (FDA). aducanumab is a monoclonal antibody treatment that targets amyloid.⁷ If approved, aducanumab will be the first disease-modifying therapy on the market.⁷ Clinical trials of aducanumab, like other agents targeting amyloid deposition, recruited individuals in a prodromal stage of Alzheimer disease using a positive amyloid result as an inclusion criterion. Additionally, the study distinguished between those that were *ApoE-e4* positive versus those who were not. First, individuals who were *ApoE-e4* experience different outcomes. Second, and perhaps more important, in the clinical trials, amyloid related imaging abnormalities (ARIA), an adverse event, was found to be dose-dependent and a greater risk for individuals who are *ApoE-e4* positive.¹⁸ This would make *ApoE* genotyping important for clinical adoption of aducanumab. Our study shows that current private payers' coverage policies would inhibit implementation of

ApoE genotyping. Policies that rely on preauthorization criteria may be the exception. Under a circumstance where a disease-modifying therapy is available for individuals, the preauthorization criteria may be met because *ApoE* status would inform clinical decision-making (e.g., dose and monitoring for adverse consequences).

In addition to the relevance of *ApoE* genotyping for implementation of screening measures and treatment, the growth of the DTC genetic testing market may raise immediate challenges for clinicians. Access to genetic testing through DTC products, including 23andMe, increases the likelihood that individuals may learn their *ApoE* status. In 2017, 23andMe received FDA approval for DTC genetic tests, including *ApoE* status.¹⁹ While we lack specific data on 23andMe users who sought out their *ApoE* status, 23andMe reports over 12 million users.²⁰ The DTC market is expected to continue to grow as individuals are seeking more control over their health and health-related information.²¹ The growth and uptake of DTC testing for *ApoE* status indicate that people are interested in learning more about their genetic risk for Alzheimer disease. This will create unique challenges for clinicians who may be unable to order a confirmatory *ApoE* test for their patients due to a lack of payer coverage of testing.²² This may inhibit appropriate pretest and post-test counseling to help individuals interpret the results of DTC results and confirm accuracy. This is particularly important given prior evidence that DTC has been inaccurate, including in cases of *BRCA1/2* testing in which women have had mastectomies based on inaccurate results.^{23,24}

Lastly, beyond the assessment of clinical utility, there is additional evidence that genetic testing, and *ApoE* specifically, may offer some *personal utility*. The REVEAL study demonstrated that individuals would be interested in learning the *ApoE* status.²⁵ One study shows that approximately 80% of respondents would pursue genetic testing for Alzheimer disease if it were paid for by insurers and nearly 59% would undergo testing if there was “at least a \$100 out-of-pocket cost for testing.”²⁶ Similarly, evidence supports that individuals would use *ApoE* status to inform personal decisions.²⁷ This study assessed participants’ behaviors and responses to learning their *ApoE* status, including efforts to obtain long-term care insurance.²⁸ There is mixed evidence about whether these changes are maintained and realistic. For example, while participants may report that they would obtain long-term care insurance, it is also likely that an individual with a known risk for Alzheimer disease may be ineligible for long-term care insurance.²⁹ Despite this, it does not eliminate the potential that *ApoE* may offer personal utility—even if the test results would not inform clinical decisions. Previous work has shown that personal utility alone typically does not drive coverage decisions.³⁰

There are several limitations to this study. We reviewed seven policies from the largest payers of more than 200 private payers. While these payers represented 113 million covered lives, the results could differ across private payers or Medicare/Medicaid policies not included in this review. Additionally, we did not evaluate outcomes of requests for *ApoE* genetic testing by plan members or their clinicians, and thus we do not know if payers applied their written policy to actual claims for testing. Therefore, these data are not broadly generalizable. Our future studies will broaden the number of payers and consider genetic risk factors for early-onset dementia and other dementias. Despite these limitations, the themes identified in our analysis provide insight into whether private payer policies would support *ApoE* testing as the field further evolves.

This study serves as an initial step in considering private payer coverage decisions for *ApoE*. The results here provide important themes regarding the rationales that support coverage decisions. These rationales and the preauthorization criteria will be important in determining whether coverage policies should be revised in the context of the evolving field.

DATA AVAILABILITY

The data reported in this paper were collected through publicly available resources. Therefore, the authors did not deposit these data into another repository. Requests for tools or frameworks used in our analysis will be considered on a case-by-case basis.

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COMPETING INTERESTS

Competing interests have been reviewed by the University of California–San Francisco. K.A.P. receives consulting income from Illumina, Inc. M.P.D. receives consulting income from Illumina, Inc. The other authors declare no competing interests.

ADDITIONAL INFORMATION

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