Genetic Testing for Lipoprotein(a) Variant(s) as a Decision Aid for Aspirin Treatment

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Services Are Considered Investigational
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Based on review of available data, the Company considers the use of genetic testing for the *LPA* rs3798220 allele (*LPA*-Aspirin Genotype)[i] in patients who are being considered for treatment with aspirin (ASA) to reduce risk of cardiovascular events to be **investigational.***

Background/Overview

LIPOPROTEIN(A)
Extensive epidemiologic evidence has determined that lipoprotein(a) (LPA) blood level is an independent risk factor for cardiovascular disease. The overall risk associated with LPA appears to be modest, and the degree of risk may be mediated by other factors such as low-density lipoprotein (LDL) levels and/or hormonal status.

Over time, a person's LPA levels remain relatively stable; however, levels have been known to vary up to 1000-fold between different people, and this is most likely due to genetics. A single-nucleotide variant in the *LPA* gene, *LPA* rs3798220, has been associated with both elevated LPA levels and an increased risk of cardiovascular disease. This variant substitutes methionine for isoleucine at amino acid position 4399 and is also called I4399M. Mendelian randomization studies have supported the hypothesis that this genetic variant, and the subsequent increase in LPA levels, are causative of cardiovascular disease.

Aspirin is a well-established treatment for patients with known coronary artery disease. It also is prescribed as primary prevention for some patients who are at increased risk of coronary artery disease. Current recommendations for primary prevention consider the future risk of cardiovascular events weighed against the bleeding risk of aspirin. The U.S. Preventive Services Task Force 2009 Guidelines recommended aspirin for men between the ages of 45 and 79 years when the benefit in reducing myocardial infarction exceeds the risk of bleeding, particularly gastrointestinal hemorrhage; and for women between the ages of 55 and 79 years when the benefit in reducing stroke exceeds the risk of gastrointestinal bleeding. Given such guidelines that recommend individualizing the risk-benefit ratio of aspirin therapy, additional tools that would aid in better defining the benefits of aspirin, and/or the risk of bleeding, have potential utility for clinicians who are making decisions about aspirin therapy.

LPA Aspirin Genotype is a commercially available genetic test (Berkeley HeartLab, a Quest Diagnostics service) that detects the presence of the rs3798220 allele. Patients with a positive test for rs3798220 have

a higher risk for thrombosis and therefore may derive more benefit from the antithrombotic properties of aspirin. It has been proposed that the additional information obtained from the LPA Aspirin Check®‡ test may aid physicians in better estimating the benefit/risk of aspirin therapy and therefore may aid in deciding whether to prescribe aspirin for individual patients.

FDA or Other Governmental Regulatory Approval

U.S. Food and Drug Administration (FDA)
Clinical laboratories may develop and validate tests in-house and market them as a laboratory service; laboratory-developed tests must meet the general regulatory standards of the Clinical Laboratory Improvement Amendments. Berkeley HeartLab/Quest Diagnostics is certified under the auspices of the Clinical Laboratory Improvement Amendments. Laboratories that offer laboratory-developed tests must be licensed by the Clinical Laboratory Improvement Amendments for high-complexity testing. To date, the U.S. FDA has chosen not to require any regulatory review of this test.

Medicare National Coverage
There is no national coverage determination (NCD). In the absence of an NCD, coverage decisions are left to the discretion of local Medicare carriers.

Rationale/Source

Assessment of diagnostic technology typically focuses on 3 categories of evidence: (1) analytic validity (test-retest reliability or interrater reliability); (2) clinical validity (sensitivity, specificity, positive and negative predictive values) in relevant populations of patients; and (3) clinical utility (ie, demonstration that the diagnostic information can be used to improve patient outcomes. The following is a summary of the key findings to date.

Genetic testing for lipoprotein(a) (*LPA*) allele rs3798220 can be evaluated in a framework similar to other novel cardiac risk factors. There are several conditions that must be met for a cardiovascular risk factor to demonstrate clinical utility. A 2002 TEC Assessment summarized 3 steps necessary for clinical utility:

- Standardization of measurement of the risk factor.
- Determination of its contribution to risk assessment. As a risk factor, it is important to determine whether the novel risk factor contributes independently to risk assessment compared with established risk factors.
- Determination of how the novel risk factor will be used in the management of the patient, compared with standard methods of assessing risk, and whether any subsequent changes in patient management lead to improved patient outcomes.

GENETIC TESTING FOR *LPA* RS3798220 VARIANT
Analytic Validity
Testing for the *LPA* rs3798220 allele is commercially available through Berkeley HeartLab under the name, LPA Aspirin Check. DNA is extracted from a buccal swab sample taken from the inner cheek. Genetic

testing is performed by real-time polymerase chain reaction (PCR) in conjunction with several control samples. Real-time PCR is expected to be more accurate than traditional PCR, because it preserves the exquisite sensitivity of PCR while reducing the probability of cross-contamination that can result in false-positive results. The main limitations to real-time PCR accuracy are human factors such as improper assay development, incorrect data analysis, or unwarranted interpretation; to ensure reliability of results, "real-time PCR primer sets must be designed and validated by stringent criteria."

No published studies were identified that evaluated the accuracy of real-time PCR testing for the specific rs3798220 allele. Test performance data are unavailable on the manufacturer website.

Section Summary: Analytic Validity
This limited information suggests that real-time PCR is likely to be an accurate method for identifying genetic variants such as the rs3798220 allele but is not sufficient to conclude that the measurement of *LPA* rs3798220 is standardized.

Clinical Validity
Several observational studies have evaluated whether *LPA* rs3798220 is an independent risk factor for coronary artery disease (CAD). Shiffman et al (2008) used data from the Cardiovascular Health Study, a prospective cohort study of risk factors for myocardial infarction in 4522 subjects who were 65 years or older, to examine the association of rs3798220 with myocardial infarction. These authors tested 74 single-nucleotide variants (SNVs) that had been genotyped as part of the Cardiovascular Health Study. After 13 years of follow-up, 539 (12%) patients had developed myocardial infarction. There were 8 SNVs that were independent predictors of myocardial infarction, with hazard ratios (HRs) ranging from 1.13 to 1.62. The rs3798220 variant was one of the independent predictors and had the highest HR (1.62; 95% confidence interval [CI], 1.09 to 2.42). The authors calculated the false-positive reporting rate for each SNV and estimated the rate to be 1% for rs3798220.

Clarke et al (2009) used a case-control design to examine the association of rs3798220 with CAD in 3145 case patients and 3352 controls from 4 European countries. They initially examined 48,742 SNVs in 2100 genes that had some association with heart disease, including 40 SNVs from the *LPA* gene. The rs3798220 SNV was found in 2% of patients and had the strongest association with CAD, with an HR of 1.92 (95% CI, 1.48 to 2.49). This association was then replicated in 3 independent patient samples from cohort studies, with a total of 4846 case patients and 4594 controls. In these patients, the rs3798220 variant remained an independent risk factor for CAD, with an odds ratio (OR) that was somewhat lower than in the derivation population (OR=1.68; 95% CI, 1.43 to 1.98).

Luke et al (2007) examined the association between SNVs and severe CAD as determined by coronary angiography. These authors used patient samples from 3 case-control studies in sequence to determine the SNVs that were most strongly associated with severe CAD. Starting with more than 12,000 SNVs, the authors identified 302 SNVs associated with severe disease; after verification in the second study, 5 SNVs remained independent predictors; and after verification in the third study, only rs3798220 remained as the

SNV most strongly associated with severe CAD. The adjusted odds for rs3798220 was 3.14 (95% CI, 1.51 to 6.56).

In a similar case-control design, Shiffman et al (2008) examined the association between the rs3798220 allele and myocardial infarction in 3 case-control studies totaling 762 cases and 857 controls. Starting from a total of 1949 SNVs associated with myocardial infarction, the authors identified 5 SNVs that were mostly strongly associated with myocardial infarction. One of these was rs3798220, which had odds in the 3 separate studies of 1.59 (95% CI, 1.03 to 2.48), 1.72 (95% CI, 1.19 to 2.49), and 3.52 (95% CI, 1.85 to 6.69).

The risk associated with genetic variants of *LPA* in diabetic patients may differ from that in the general population. A large prospective study performed in 2012 evaluated *LPA* variants in 2308 patients who had diabetes. There was no significant association between genetic variants and cardiovascular risk or mortality. ORs for coronary heart disease (CHD), cardiovascular disease, and cardiovascular death were 0.94 (95% CI, 0.69 to 1.28), 0.97 (95% CI, 0.72 to 1.29), and 1.23 (95% CI, 0.79 to 1.92), respectively. The authors also examined the degree of variability in risk between diabetic and nondiabetic patients and reported that there was significant heterogeneity between groups (p=0.006).

A 2013 case-control study of 2136 cases and 1211 controls evaluated whether SNVs rs3798220 and rs10455872 were associated with an increased risk of coronary disease. Genotyping of these 2 SNVs and 7 other *LPA* variants believed to be associated with coronary disease was done using the TaqMan assay. After adjusting for conventional risk factors, the authors found increased odds of myocardial infarction of 2.14 (95% CI, 1.37 to 3.33, p<0.001) for rs3798220 and 1.45 (95% CI, 1.36 to 2.24; p<0.001) for rs10455872. Two additional SNVs, rs3127599 and rs9346818, also were found to be associated with risk of myocardial infarction, with an odds of 1.18 (95% CI, 1.06 to 1.32) and 0.88 (95% CI, 0.79 to 0.97), respectively.

Kamstrup et al (2013) followed a Danish cohort of 8720 participants for 10 years to determine whether *LPA* variants or LPA levels increased the risk of a first-time myocardial infarction or CHD event. Genotyping of rs3798220, rs10455872, and *LPA* kringle IV type 2 (KIV-2) copy number variants was performed by PCR. The authors found that 27% of the total variation in LPA levels was explained by the rs10455872 genotype, 21% of the variation was explained by the *LPA* KIV-2 copy number variant, and 5% of the variation was explained by the rs3798220 genotype. Hazard ratios for rs3798220 carriers were 1.3 (95% CI, 0.8 to 2.1) for myocardial infarction and 1.4 (95% CI, 1.1 to 1.9) for CHD compared with noncarriers. *LPA* rs10455872 carriers had HRs of 1.3 (95% CI, 1.1 to 1.6) for myocardial infarction and 1.1 (95% CI, 0.9 to 1.3) for CHD compared with noncarriers, whereas homozygous rs10455872 patients had HRs of 1.2 (95% CI, 0.5 to 3.3) for myocardial infarction and 1.1 (95% CI, 0.5 to 2.1) for CHD compared with noncarriers.

Wang et al (2014) conducted a case-control study and did not find an association between rs3798220 genotype and myocardial infarction risk in a Chinese population. Cases (n=2365) were patients who had experienced a first myocardial infarction, drawn from hospitals in 15 cities in China. (This was the Chinese

cohort of the global INTERHEART study.) Age- and sex-matched controls (n=2678) were healthy adult visitors to the hospitals; these individuals had no history of cardiovascular disease. In logistic regression analysis adjusted for age, sex, and body mass index, the odds for myocardial infarction in rs3798220 carriers compared with noncarriers was 1.12 (95% CI, 0.57 to 2.22; p=0.73).

Similarly, Anderson et al (2013) did not find an association between rs3798220 genotype and prevalence of CAD in 1235 patients in the Intermountain Heart Collaborative Study Registry who underwent coronary angiography. CAD was defined as stenosis of 70% or more in coronary artery diameter, and non-CAD as stenosis less than 10% plus no history of CAD, myocardial infarction, or coronary artery revascularization. By these definitions, 801 (65%) patients had CAD, and 434 (35%) did not. In logistic regression analysis adjusted for age, sex, body mass index (natural log), hyperlipidemia, hypertension, diabetes, smoking history, and family history of premature CAD, the odds for CAD in rs3798220 carriers compared with noncarriers was 1.74 (95% CI, 0.84 to 3.59; p=0.36). In contrast, the rs10455872 genotype was significantly associated with CAD (OR in rs10455872 carriers vs noncarriers, 1.77; 95% CI, 1.22 to 2.57; p=0.003).

Section Summary: Clinical Validity
This data on the clinical validity of testing for the *LPA* rs3798220 allele is sufficient to conclude that it is an independent risk factor for cardiovascular disease. It has not been determined whether measurement of their genetic variant is superior to measurement of LPA levels as an independent risk factor for cardiovascular disease.

Clinical Utility
The Women's Health Study examined the comparative efficacy of aspirin and placebo for primary prevention of cardiovascular events in healthy women. In 2009, Chasman et al published a post hoc analysis of 28,345 participants in the Women's Health Study who were genotyped for the presence of the *LPA* rs3798220 minor allele. The allele was present in 3.7% of the population, 3.6% who were heterozygotes and 0.06% who were homozygotes. As expected, LPA levels in carriers of the allele were markedly elevated compared with noncarriers, and carriers had a 2-fold increased risk for subsequent cardiovascular events compared with noncarriers.

The authors reported on an interaction between the presence of the *LPA* rs378220 allele and response to aspirin therapy. In carriers, a significant risk reduction was associated with aspirin treatment, with cardiovascular events occurring in 4.8% of patients in the placebo group compared with 2.1% in the aspirin group (HR=0.44; 95% CI, 0.20 to 0.94; p=0.03). For noncarriers of the allele, there was no significant reduction in cardiovascular events associated with aspirin treatment, with cardiovascular events occurring in 2.3% of the placebo group compared with 2.1% of the aspirin group (HR=0.91; 95% CI, 0.77 to 1.08; p=0.30).

Shiffman et al (2009) reported on the interaction of the *LPA* rs3798220 variant and aspirin use from the Atherosclerosis Risk in Communities (ARIC) study. The ARIC study assessed a prospective cohort of risk factors for CAD in 15,792 subjects. The *LPA* genetic substudy of ARIC included 6752 subjects who had

data available for *LPA* genotype and aspirin use, including 221 subjects with the *LPA* rs3798220 genotype. Among carriers of rs3798220, the risk of cardiovascular events was compared in aspirin users and nonusers. The HR for nonaspirin users (n=168) was elevated at 1.57 but was not statistically significant (95% CI, 0.92 to 2.69); HR for aspirin users was not elevated at 0.86 (95% CI, 0.38 to 1.95).

Section Summary: Clinical Utility
These data are supportive, but not conclusive, of the hypothesis that carriers of the rs3798220 allele may derive greater benefit from aspirin therapy than noncarriers. It is unclear how this information would be used in clinical care. For patients who are currently recommended to receive aspirin, a negative genetic test is probably insufficient to warrant withholding aspirin. Similarly, for patients who are not currently recommended to receive aspirin, a positive genetic test is probably insufficient to warrant starting aspirin. Therefore, it remains to be determined whether results of rs3798220 testing leads to changes in management and whether these changes in management improve outcomes.

SUMMARY OF EVIDENCE
The *LPA* minor allele, rs3798220, is associated with higher levels of LPA and a higher risk for cardiovascular events. This allele is infrequent in the population and is associated with a modest increase in cardiovascular risk in the general population. Testing for this allele is commercially available, but performance characteristics are uncertain, and standardization of testing has not been demonstrated. Several observational studies have reported that this genetic variant is an independent risk factor for cardiovascular disease, but some studies have not reported a significant association. Evidence from a post hoc analysis of the Women's Health Study reported that carriers of the allele may derive greater benefit from aspirin treatment compared with noncarriers. It is unclear whether this information derived from genetic testing leads to changes in management. In particular, it cannot be determined from available evidence whether deviating from current guidelines on aspirin treatment based on *LPA* genetic testing improves outcomes. Therefore, measurement of the *LPA* rs3798220 variant as a decision aid for aspirin treatment is considered investigational.

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06/02/2011	Medical Policy Committee review
06/15/2011	Medical Policy Implementation Committee approval. New policy.
06/14/2012	Medical Policy Committee review
06/20/2012	Medical Policy Implementation Committee approval. Coverage eligibility unchanged.
02/19/2013	Coding updated
06/06/2013	Medical Policy Committee review
06/25/2013	Medical Policy Implementation Committee approval. Coverage eligibility unchanged.
06/05/2014	Medical Policy Committee review
06/18/2014	Medical Policy Implementation Committee approval. Coverage eligibility unchanged.
06/04/2015	Medical Policy Committee review
06/17/2015	Medical Policy Implementation Committee approval. Updated test name to LPA-Aspirin Genotype. Coverage eligibility unchanged.
08/03/2015	Coding update: ICD10 Diagnosis code section added; ICD9 Procedure code section removed.
06/02/2016	Medical Policy Committee review
06/20/2016	Medical Policy Implementation Committee approval. Coverage eligibility unchanged.
01/01/2017	Coding update: Removing ICD-9 Diagnosis Codes

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Next Scheduled Review Date: 06/2019

Coding

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Codes used to identify services associated with this policy may include (but may not be limited to) the following:

Code Type	Code
CPT	81479
HCPCS	No codes
ICD-10 Diagnosis	All related diagnoses

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A. Whether the medical treatment, procedure, drug, device, or biological product can be lawfully marketed without approval of the U.S. FDA and whether such approval has been granted at the time the medical treatment, procedure, drug, device, or biological product is sought to be furnished; or
B. Whether the medical treatment, procedure, drug, device, or biological product requires further studies or clinical trials to determine its maximum tolerated dose, toxicity, safety, effectiveness, or effectiveness as compared with the standard means of treatment or diagnosis, must improve health outcomes, according to the consensus of opinion among experts as shown by reliable evidence, including:
    1. Consultation with the Blue Cross and Blue Shield Association TEC or other nonaffiliated technology evaluation center(s);
    2. Credible scientific evidence published in peer-reviewed medical literature generally recognized by the relevant medical community; or
    3. Reference to federal regulations.

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