510(k) SUBSTANTIAL EQUIVALENCE DETERMINATION
DECISION SUMMARY
ASSAY ONLY

I Background Information:

A 510(k) Number

K253069

B Applicant

Diazyme Laboratories Inc

C Proprietary and Established Names

Lipoprotein (a) Molarity Assay

D Regulatory Information

Product Code(s)	Classification	Regulation Section	Panel
DFC	II	21 CFR 866.5600 - Low-Density Lipoprotein Immunological Test System	IM - Immunology

II Submission/Device Overview:

A Purpose for Submission:

New device

B Measurand:

Lipoprotein (a) [Lp(a)]

C Type of Test:

Quantitative immunoturbidimetric assay

III Intended Use/Indications for Use:

A Intended Use(s):
See Indications for Use below.

B Indication(s) for Use:
The Lipoprotein (a) Molarity Assay is intended as an in vitro test for quantitative determination of Lipoprotein(a) [Lp(a)] concentrations in human serum and plasma on validated automated analyzers. The measurement of Lp(a) is useful in evaluating lipid metabolism disorders and assessing atherosclerotic cardiovascular disease risk, when used in conjunction with clinical evaluation and other lipoprotein tests.

C Special Conditions for Use Statement(s):
Rx - For Prescription Use Only

D Special Instrument Requirements:

Beckman Coulter AU680 Chemical Analyzer

IV Device/System Characteristics:

A Device Description:

The Lipoprotein (a) Molarity assay consists of the following reagents:
Reagent 1: Tris buffer solution: pH 7.5; BSA; preservative
Reagent 2: Latex particles coated with anti-Lipoprotein (a) antibodies; Tris buffer solution: pH 8.2; BSA; preservative

B Principle of Operation:

The Lipoprotein (a) Molarity Assay is based on a latex enhanced immunoturbidimetric assay. Lipoprotein (a) in the sample binds to specific anti-lipoprotein (a) polyclonal antibody, which is coated on latex particles, and causes agglutination. The degree of the turbidity caused by agglutination can be measured optically and is proportional to the amount of lipoprotein (a) in the sample.

V Substantial Equivalence Information:

A Predicate Device Name(s):
Diazyme Lipoprotein (a) Assay

B Predicate 510(k) Number(s):
K180074

C Comparison with Predicate(s):

Device & Predicate Device(s):	K253069	K180074
Device Trade Name	Lipoprotein (a) Molarity Assay	Diazyme Lipoprotein (a) Assay
General Device Characteristic Similarities		
Intended Use/Indications For Use	Intended for quantitative determination of Lipoprotein(a) [Lp(a)] in human serum or plasma	Same
Sample type	Serum EDTA plasma Lithium heparin plasma	Same
Test Principle	Immunoturbidimetric assay	Same
General Device Characteristic Differences		
Lp(a) units of measurement	nmol/L	mg/dL
Analytical measuring interval	4.85 to 240 nmol/L	5.4 – 100 mg/dL
Traceability	Standardized to CDC CSP materials with Lp(a) measured by IFCC WG-APO MS reference measurement procedure (RMP)	Calibrators are traceable to the predicate device

VI Standards/Guidance Documents Referenced:

- Clinical and Laboratory Standards Institute (CLSI) EP05-A3 (Reaffirmed: September 2019). Evaluation of Precision of Quantitative Measurement Procedures; Approved Guideline – Third Edition.
- CLSI EP06 2nd Edition. Evaluation of the Linearity of Quantitative Measurement Procedures.
- CLSI EP07 3rd Edition. Interference Testing in Clinical Chemistry.
- CLSI EP17-A2. Evaluation of Detection Capability for Clinical Laboratory Measurement Procedures; Approved Guideline – Second Edition.

VII Performance Characteristics (if/when applicable):

A Analytical Performance:

1. Precision/Reproducibility:

A precision study to assess repeatability and within-laboratory precision was conducted according to CLSI EP05-A3. Five human serum samples and two controls were run in replicates of two per run, with two runs per day, over 20 days on the Beckman AU680 analyzer using one operator at one site and three different reagent lots. Results are shown below.

Sample	Mean (nmol/L)	Repeatability		Between-Run		Between-Day		Between-Lot		Total	
		SD	CV(%)	SD	CV(%)	SD	CV(%)	SD	CV(%)	SD	CV(%)
Control 1	34.24	0.58	1.70%	0.405	1.18%	0.512	1.50%	0.110	0.32%	0.714	2.09%
Control 2	94.21	0.62	0.66%	0.455	0.48%	0.532	0.57%	0.180	0.19%	0.769	0.82%
Sample 1	34.57	0.61	1.76%	0.444	1.28%	0.392	1.13%	0.218	0.63%	0.680	1.97%
Sample 2	74.59	0.62	0.83%	0.422	0.57%	0.557	0.75%	0.102	0.14%	0.766	1.03%
Sample 3	115.55	0.69	0.60%	0.552	0.48%	0.484	0.42%	0.149	0.13%	0.794	0.69%
Sample 4	148.85	1.43	0.96%	0.949	0.64%	0.827	0.56%	0.176	0.12%	1.467	0.99%
Sample 5	198.84	1.80	0.91%	1.271	0.64%	1.056	0.53%	0.432	0.22%	1.906	0.96%

An additional precision study was conducted per CLSI EP05-A3 using samples with known apolipoprotein a (apo(a)) isoform sizes (as determined by SDS agarose gel electrophoresis and Western blot analysis). Five human serum sample pools were tested in two replicates, two runs per day for over 21 days on the Beckman Coulter AU680 Chemistry Analyzer using one lot of reagents. Results support that precision is not dependent on Apo(a) isoform size.

2. Linearity:

A linearity study was conducted to demonstrate that measurements across the claimed assay reportable range are linear. The study was conducted according to CLSI EP06 2nd Edition. Dilution series were prepared from a serum and a plasma sample containing 257.3 nmol/L of Lp(a) by diluting with 0.9% NaCl to create 16 levels (ranging from ~4.0 to 257.3 nmol/L). The sets of dilutions were run on a Beckman AU680 analyzer using three reagent lots with three replicates per level. Linear regression was conducted for each dilution series and the deviation from linearity was calculated. The linear regression results are shown below.

Sample type, Lot	Linear regression equation	R^2
Serum, Lot 1	y = 0.9913x	0.9996
Serum, Lot 2	y = 0.9930x	0.9997
Serum, Lot 3	y = 1.0028x	0.9998
Plasma, Lot 1	y = 0.9955x	0.9994
Plasma, Lot 2	y = 0.9971x	0.9998
Plasma, Lot 3	y = 0.9963x	0.9994

The results support that the Lipoprotein (a) Molarity Assay is linear throughout its analytical measuring interval of 4.85 to 240 nmol/L.

An additional linearity study using serum samples with known isoform size was also provided to support that the assay is linear irrespective of Lp(a) isoform size.

Dilution study

A dilution study was conducted to validate the automatic rerun function on Beckman AU680 analyzer. Five native human serum samples with concentrations exceeding the measuring interval (based on a validated Lp(a) ELISA assay that measures Lp(a) in nmol/L) were diluted manually and automatically (via the automatic rerun function) using a 1:3 dilution. Each sample was measured in singlicate with three lots of Lipoprotein (a) Molarity assay on the Beckman AU680 analyzer.

3. Analytical Specificity/Interference:

Endogenous interference

An endogenous interference study was conducted according to CLSI EP07 3rd Edition to assess whether endogenous substances interfere with assay results. Two human serum pools with two different Lp(a) concentrations (low level ranged from 45.2 to 53.5 nmol/L; high level ranged from 104.1 to 119.46 nmol/L) were spiked with each of the interferents by proportionally mixing with interferent pool and solvent to generate four different concentrations of interferent (test samples), and one with solvent only (control sample). Each sample was measured in triplicate on Beckman AU680 analyzer and the mean value of test samples with interferent was used to calculate % Recovery from control sample. No interference was defined by the sponsor as less than ± 10% recovery differences between test sample and control sample.

The following substances produced less than 10% differences from the control sample:

Substance	Highest level of substance at which no interference was observed
Albumin	7400 mg/dL
Conjugated bilirubin	70 mg/dL
Unconjugated bilirubin	77 mg/dL
Hemoglobin	1220 mg/dL
Immunoglobulin G	6700 mg/dL
Intralipid	2500 mg/dL
Rheumatoid Factor	1330 IU/mL
Triglyceride	1000 mg/dL

Exogenous interference

An exogenous interference study was conducted to assess whether the presence of common drugs substances interferes with assay results. Two human serum samples with either low level ranged from 45.2 to 53.5 nmol/L or high level ranged from 104.1 to 119.46 nmol/L were split into two parts with one part spiked with potential interferent and the other part spiked with solvent. Each sample was measured in triplicate on Beckman AU680 analyzer and the mean value of test samples with interferent was used to calculate % Recovery from

control sample. No interference was defined by the sponsor as less than ± 10% recovery differences between test sample and control sample.

The following substances produced less than 10% differences from the control sample:

Substance	Highest level of substance at which no interference was observed
N-Acetylcysteine	15 mg/dL
Acetylsalicylic acid	3 mg/dL
Ampicillin-Na	7.5 mg/dL
Cefoxitin	660 mg/dL
Doxycycline	1.8 mg/dL
Heparin	3300 IU/L
Levodopa	0.8 mg/dL
Methyldopa	2.3 mg/dL
Metronidazole	12.3 mg/dL
Rifampicin	4.8 mg/dL
Acetaminophen	15.6 mg/dL
Cyclosporine	0.2 mg/dL
Ibuprofen	21.9 mg/dL
Theophylline	6 mg/dL
Phenylbutazone	32.1 mg/dL
Ascorbic acid	176.1 mg/dL

Cross-reactivity study

A cross-reactivity study was conducted to assess whether substances structurally related to Lp(a) cross-react with the assay. Two human serum pools with two different Lp(a) concentrations (low level ranged from 40.28 to 41.85 nmol/L Lp(a); high level ranged from 107.05 to 112.49 nmol/L Lp(a)) were split into two parts with one part spiked with potential cross-reactant and the other part not spiked with cross reactant. Each part was measured in 5 replicates, and the mean values were used to determine the absolute difference between matched samples with or without potential cross-reactant. Percent cross reactivity was then calculated using the following formula: (100 * absolute difference) / cross-reactant concentration. No cross-reactivity was detected for apolipoprotein B at 223 mg/dL or plasminogen at 160 mg/dL at either level of Lp(a).

High dose hook effect

A high dose hook effect (prozone effect) study was conducted using samples with Lp(a) concentrations up to 1000 nmol/L and the study demonstrated no prozone effect up to 800 nmol/L. The results support the labeling claim of no false results up to an Lp(a) concentration of 432 nmol/L. The sponsor included the following limitation in the labeling: Samples with >800 nmol/L may exhibit prozone behavior.

4. Detection Limit and Assay Reportable Range:

Limit of blank (LoB), limit of detection (LoD), and limit of quantitation (LoQ) studies were conducted according to the recommendation in CLSI EP17-A2.

For determination of LoB, one analyte-free saline (0.9% NaCl) sample was measured with three reagent lots in six runs, with ten replicates per run, over three days on the Beckman AU680 analyzer for a total of 60 determinations per lot. The LoB was determined as the 95th percentile of the 60 determinations. LoB was determined to be 0.485 nmol/L which is the highest value of the three lots.

For determination of LoD, five human serum samples with low Lp(a) concentrations were measured with three reagent lots with four replicates per day for each sample over three days on the Beckman AU680 analyzer for a total of 60 determinations for each lot. The LoD was determined as the lowest amount of analyte in a sample that can be detected with 95% probability using equation: LoD = (LoB) + 1.65 * (SD_L) where SD_L is the estimated standard deviation of the sample distribution at a low level. LoD was determined to be 1.5018 nmol/L, which is the highest value of the three lots.

For determination of LoQ, five human serum samples with low Lp(a) concentrations were measured with three reagent lots, five replicates per day for each sample, over three days on the Beckman AU680 analyzer for a total of 15 determinations per sample per lot. The LoQ was determined as the lowest concentration of analyte that can be quantified with an intermediate precision of no more than 20% CV. The LoQ for the Lipoprotein (a) Molarity Assay on Beckman AU680 analyzer was determined to be 4.85 nmol/L (highest value from the three lots).

The results of the studies are shown below.

nmol/L	Lot 1	Lot 2	Lot 3	Claimed
LoB	0.465	0.485	0.475	0.485
LoD	1.1689	1.5018	0.9943	1.50
LoQ	4.85	4.32	4.00	4.85

The reportable range of the assay is 4.85 – 240 nmol/L.

5. Traceability, Stability, Expected Values (Controls, Calibrators, or Methods):

Traceability
This method is traceable to CDC CSP materials with Lp(a) measured by IFCC WG-APO MS reference measurement procedure (RMP).

Sample stability
The sponsor provided information to support the following labeling claims:

Samples are stable for 6 days at 15-25°C, 21 days at 2-8°C, and 14 weeks at -20 °C (± 5 °C). One freeze-thaw is recommended. Re-peated freeze/thaw cycles should be avoided to minimize potential protein degradation.

On-board stability and calibration frequency
Diazyme provided information to support that reagent on-board stability is 54 days at 4-12°C; calibration frequency is 15 days.

6. Assay Cut-Off:

Not applicable.

B Comparison Studies:

1. Method Comparison:

Thirty-nine serum samples with assigned value determined by the IFCC Working Group for Apolipoproteins by Mass Spectrometry's (IFCC WG APO-MS) LC-MS/MS method, were tested in singlicate on one Beckman AU680 analyzer by one operator using 3 reagent lots of the Lipoprotein (a) Molarity Assay. These samples evenly span the measuring range (8.36 – 233.11) with known isoform(s) in each sample. Deming regression analysis was conducted for the comparison between the IFCC LC-MS/MS method and the Diazyme Lp(a) Molarity assay. The results of the analysis, including slope with 95% confidence interval (CI), intercept with 95% CI, and correlation coefficient, are shown below.

Lot	Slope (95% CI)	Intercept (95% CI) (nmol/L)	Correlation Coefficient (r)
1	0.978 (0.949 to 1.008)	1.752 (-0.971 to 4.475)	0.9959
2	0.962 (0.941 to 0.984)	2.146 (0.163 to 4.128)	0.9978
3	0.977 (0.953 to 1.002)	1.662 (-0.580 to 3.904)	0.9972

An additional method comparison study using 209 samples was also conducted and the results support that the Diazyme Lipoprotein (a) Molarity Assay is equivalent to a validated Lp(a) apo(a) size insensitive ELISA assay measuring Lp(a) in nmol/L.

2. Matrix Comparison:

A matrix comparison study was conducted to demonstrate equivalence between sample types (serum, K2EDTA plasma, K3EDTA plasma, lithium heparin plasma) when used with the Diazyme Lipoprotein (a) Molarity Assay. Matched samples from up to 71 donors were tested on the Beckman AU680 analyzer in singlicate using one reagent lot. Deming regression analysis was conducted for each sample type relative to the reference serum values. The results of the analyses, including slope with 95% confidence interval (CI), intercept with 95% CI, and correlation coefficient, are shown below for each sample type comparison.

Comparison	N	Sample range (nmol/L)	Slope (95% CI)	Intercept (95% CI)	R
serum vs. K2EDTA	71	6.29-224.40	1.002 (0.983 to 1.020)	-0.957 (-2.574 to 0.659)	0.9970
serum vs. K3EDTA	71	6.07-213.59	0.961 (0.94 to 0.976)	0.288 (-1.072 to 1.648)	0.9977
serum vs. Li-Hep	71	6.53-216.53	0.974 (0.955 to 0.993)	-1.283 (-2.970 to 0.404)	0.9966

C Clinical Studies:

1. Clinical Sensitivity:

   Not applicable.

2. Clinical Specificity:

   Not applicable.

3. Clinical Cut-Off:

   Not applicable.

4. Other Clinical Supportive Data (When 1. and 2. Are Not Applicable):

   The sponsor cited several clinical studies showing associations between Lp(a) and ASCVD risk, including the UK Biobank study[1].

   In the UK Biobank study, which included 460,506 participants between the ages of 40 and 69 years, the relationship of Lp(a) concentrations to incident ASCVD was studied. ASCVD was defined as a composite of coronary artery disease (myocardial infarction and its acute complications, coronary artery bypass graft surgery, or percutaneous angioplasty/stent placement) and ischemic stroke (cerebral infarction due to thrombosis or cerebral atherosclerosis or cerebrovascular syndromes).

   The event rate per 1000 person-years near 0 nmol/L Lp(a) was ~0.4% and the event rate increased to ~0.45% at 75 nmol/L Lp(a) and ~0.5% at 125 nmol/L Lp(a).[1] In addition, Cox proportional hazards regression model (using covariates of enrollment age, sex, race, and Lp(a) concentration) was performed. This analysis showed an adjusted hazard ratio (HR) of ~1.2 at 75 nmol/L Lp(a) and ~1.4 at 125 nmol/L Lp(a) [1].
   Incidence rates per 1000 person-years were also stratified by racial subgroup (White, Black, South Asian)[1].

   - The event rate per 1000 person-years was ~0.4% for the White group near 0 nmol/L Lp(a) and increased to ~0.45% at 75 nmol/L Lp(a) and ~0.5% at 125 nmol/L Lp(a).

- The event rate per 1000 person-years was ~0.35% for the Black group near 0 nmol/L Lp(a), 75 nmol/L Lp(a) and 125 nmol/L Lp(a) and increased for Lp(a) > 250 nmol/L.
- The event rate per 1000 person-years was ~0.65% for the South Asian group near 0 nmol/L Lp(a) and increased to ~0.75% at 75 nmol/L Lp(a) and 125 nmol/L Lp(a).

The 95% confidence intervals were larger for the Black and South Asian groups. The authors of the study concluded that there is a linear risk gradient of Lp(a) across different races with standardized ASCVD risk found to be 11% higher for each increment of 50 nmol/L (HR = 1.11 per 50 nmol/L [95% CI, 1.10-1.12]), independent of adjustment for traditional risk factors, and with similar effect estimates in all race and ethnicity groups.

Similar association between Lp(a) and ASCVD risk was also demonstrated in the Dallas Heart Study[2]. Information was provided to support that the associations between Lp(a) and ASCVD risk apply to the candidate assay.

The labeling states the following:

Scientific Statements on Use of Lp(a)

The National Lipid Association (2024)[3] provided updated risk classification guidelines from the National Lipid Association (NLA): individuals with Lp(a) levels <75 nmol/L are classified as low risk, those with Lp(a) levels ≥125 nmol/L as high risk, and those with Lp(a) levels between 75 and 125 nmol/L as intermediate risk. These thresholds align with the 2018 AHA/ACC/AACVPR multi-society guideline on cholesterol management[4] and the 2019 ACC/AHA guideline on primary prevention of cardiovascular disease[5], both of which identify Lp(a) ≥125 nmol/L as a risk-enhancing factor for atherosclerotic cardiovascular disease (ASCVD), particularly at higher values[6].

For an individual's ASCVD risk assessment, American societies (American Heart Association (AHA) and American College of Cardiology (ACC)) recommend starting with a 10-year risk estimation which includes an individual's age, race and other established risk factors[4,5]. To determine a patient's overall ASCVD risk, the patient's baseline cardiovascular risk and Lp(a)-attributable risk should be considered together. The Lp(a)-attributable ASCVD risk is independent of a patient's baseline ASCVD risk, as Lp(a) can be regarded as a risk enhancer and a patient's baseline ASCVD risk is dependent on several risk factors including family history of premature ASCVD, primary hypercholesterolemia, metabolic syndrome, sex-specific risk enhancing factors, chronic inflammatory conditions, chronic kidney disease, diabetes, hypertension, age, smoking and high-risk ethnicities[7,8].

The table below shows NLA recommended Lp(a) thresholds, alongside Lp(a) attributable ASCVD risk[3].

	NLA Scientific Statement	Lp(a)-attributable ASCVD risk
< 75 nmol/L	Individuals with Lp(a) levels <75 nmol/L are classified as low risk.	Low Lp(a)-attributable ASCVD risk
75 - < 125 nmol/L	Individuals with Lp(a) levels between 75 and 125 nmol/L are classified as intermediate risk.	Intermediate Lp(a)-attributable ASCVD risk
≥ 125 nmol/L	Individuals with Lp(a) levels ≥125 nmol/L are classified as high risk.	High Lp(a)-attributable ASCVD risk, with increasing risk with higher Lp(a) values.

Similar associations between Lp(a) and ASCVD risk have also been demonstrated in the Dallas Heart Study (DHS) [2,9]. The increasing ASCVD risk with higher Lp(a) values is supported by prospective cohort data from the UK Biobank study [1]. The risk of incident ASCVD across the spectrum of Lp(a) concentrations were quantified, and a linear relationship is demonstrated between Lp(a) concentrations and incident ASCVD risk. In this large UK Biobank study, which included 460,506 participants, each 50 nmol/L increment in Lp(a) was associated with a hazard ratio of 1.11 (95% CI, 1.10 - 1.12) for ASCVD. This linear risk relationship was consistent across racial groups, with hazard ratios of 1.11, 1.10, and 1.07 per 50 nmol/L for white, South Asian, and black individuals, respectively, despite substantial differences in median Lp(a) concentrations (19, 31, 75, and 16 nmol/L for white, South Asian, black, and Chinese individuals, respectively).

Lipoprotein (a) values should be interpreted in conjunction with clinical evaluation and other lipoprotein tests when assessing atherosclerotic cardiovascular disease in specific populations.

The measured Lp(a) value of a patient's sample can vary depending on the Lp(a) assay used and the units reported. The laboratory result report should therefore contain a statement on the Lp(a) assay used. Values determined in samples by different Lp(a) assays cannot be used interchangeably.

References:

1. Patel AP, Wang (汪敏先) M, Pirruccello JP, et al. Lp(a) (Lipoprotein[a]) Concentrations and Incident Atherosclerotic Cardiovascular Disease: New Insights From a Large National Biobank. Arterioscler Thromb Vasc Biol. 2021;41(1):465-474.
2. Lee SR, Prasad A, Choi YS, Xing C, Clopton P, Witztum JL, Tsimikas S. LPA Gene, Ethnicity, and Cardiovascular Events. Circulation. 2017 Jan 17;135(3):251-263.
3. Koschinsky ML, Bajaj A, Boffa MB, et al. A focused update to the 2019 NLA scientific statement on use of lipoprotein(a) in clinical practice. J Clin Lipidol. 2024;18(3):e308-e319.
4. Grundy SM, Stone NJ, Bailey AL, et al. 2018 AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA Guideline on the Management of Blood Cholesterol: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Circulation. 2019;139(25):e1082-e1143.

5. Arnett DK, Blumenthal RS, Albert MA, et al. 2019 ACC/AHA Guideline on the Primary Prevention of Cardiovascular Disease: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Circulation. 2019;140(11):e596-e646.
6. Tsimikas S. A Test in Context: Lipoprotein(a): Diagnosis, Prognosis, Controversies, and Emerging Therapies. J Am Coll Cardiol. 2017;69(6):692-711.
7. Kronenberg F, Mora S, Stroes ESG, et al. Frequent questions and responses on the 2022 lipoprotein(a) consensus statement of the European Atherosclerosis Society. Atherosclerosis. 2023;374:107-120.
8. Wong ND, Budoff MJ, Ferdinand K, et al. Atherosclerotic cardiovascular disease risk assessment: An American Society for Preventive Cardiology clinical practice statement. Am J Prev Cardiol. 2022;10:100335.
9. Tsimikas S, Clopton P, Brilakis ES, et al. Relationship of oxidized phospholipids on apolipoprotein B-100 particles to race/ethnicity, apolipoprotein(a) isoform size, and cardiovascular risk factors: results from the Dallas Heart Study. Circulation. 2009;119(13):1711-1719.

D Expected Values/Reference Range:

A reference range study was conducted and Lp(a) levels among various sub-populations were measured in singlicate using one lot of reagents by one operator. The expected ranges were determined using samples from apparently healthy adults in the United States.

The reference ranges are not intended to be used as medical decision thresholds for cardiovascular risk. Each laboratory should investigate the transferability of the expected values to its own patient population and, if necessary, determine its own reference ranges.

Lp(a) by race

	n	Median	2.5th percentile	97.5th percentile	Unit
All	534	52.7	< 4.85	357.4	nmol/L
White	123	27.4	< 4.85	296.5	nmol/L
African-American/ Black	211	89.2	10.0	382.5	nmol/L
Asian	120	38.9	5.3	169.4	nmol/L
Other	80	48.3	< 4.85	210.7	nmol/L

VIII Proposed Labeling:

The labeling supports the finding of substantial equivalence for this device.

IX Conclusion:

The submitted information in this premarket notification is complete and supports a substantial equivalence decision.