

Prior Authorization Review Panel

MCO Policy Submission

A separate copy of this form must accompany each policy submitted for review.

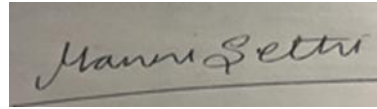
Policies submitted without this form will not be considered for review.

Plan: AmeriHealth Caritas Pennsylvania & Keystone First	Submission Date: 8/1/25
Policy Number: CCP.1238	Effective Date: 10/1/25 Revision Date: 7/1/25
Policy Name: Lipoprotein apheresis	
Type of Submission:	Type of Policy:
☐ New Policy	☒ Prior Authorization Policy
☒ Revised Policy*	☐ Base Policy
☐ Annual Review- no revisions	☐ Experimental/Investigational Policy
	☐ Statewide PDL
	☐ Other:
*All revisions to the policy must be highlighted using track changes throughout the document. Please provide any clarifying information for the policy below:	

Name of Authorized Individual (Please type or print):

Manni Sethi, MD, MBA, CHCQM

Signature of Authorized Individual:

A photograph of a handwritten signature in black ink on a light-colored surface. The signature is written in a cursive style and reads "Manni Sethi".

Lipoprotein apheresis

Clinical Policy ID: CCP.1238

Recent review date: 7/2025

Next review date: 11/2026

Policy contains: Familial hypercholesterolemia; HELP; Liposorber; low-density lipoprotein apheresis; primary focal segmental glomerulosclerosis.

AmeriHealth Caritas Pennsylvania has developed clinical policies to assist with making coverage determinations. AmeriHealth Caritas Pennsylvania clinical policies are based on guidelines from established industry sources, such as the Centers for Medicare & Medicaid Services (CMS), state regulatory agencies, the American Medical Association (AMA), medical specialty professional societies, and peer-reviewed professional literature. These clinical policies along with other sources, such as plan benefits and state and federal laws and regulatory requirements, including any state- or plan-specific definition of "medically necessary," and the specific facts of the particular situation are considered by AmeriHealth Caritas Pennsylvania on a case by case basis when making coverage determinations. In the event of conflict between this clinical policy and plan benefits and/or state or federal laws and/or regulatory requirements, the plan benefits and/or state and federal laws and/or regulatory requirements shall control. AmeriHealth Caritas Pennsylvania clinical policies are for informational purposes only and not intended as medical advice or to direct treatment. Physicians and other health care providers are solely responsible for the treatment decisions for their patients. AmeriHealth Caritas Pennsylvania clinical policies are reflective of evidence-based medicine at the time of review. As medical science evolves, AmeriHealth Caritas Pennsylvania will update its clinical policies as necessary. AmeriHealth Caritas Pennsylvania clinical policies are not guarantees of payment.

Coverage policy

Low-density lipoprotein apheresis, using heparin-induced extracorporeal precipitation or dextran sulfate adsorption, is clinically proven and, therefore, may be medically necessary for severe familial hypercholesterolemia when there is an inadequate response to, or intolerance of, maximum drug therapy (a six-month trial of $\geq$ two hypolipidemic agent classes) and one of the following criteria (American Heart Association [Gianos, 2024]; American College of Cardiology [Grundy, 2019]; National Lipid Association Expert Panel on Familial Hypercholesterolemia [Goldberg, 2011, Jacobson, 2015]):

- Functional homozygous form with low-density lipoprotein cholesterol $\geq$ 500 mg/dL.
- Functional heterozygous form with low-density lipoprotein cholesterol $\geq$ 300 mg/dL and no known cardiovascular disease.
- Functional heterozygous form with low-density lipoprotein cholesterol $\geq$ 200 mg/dL and cardiovascular disease documented as either:
 - History of myocardial infarction, coronary artery bypass surgery, percutaneous transluminal coronary angioplasty, or alternative revascularization procedure.
 - Angina with coronary heart disease documented by stress test.
- Primary focal segmental glomerulosclerosis recurring after kidney transplantation (Gianos, 2024; U.S. Food and Drug Administration, 2013, 2018).

High-density lipoprotein apheresis is investigational/not clinically proven and, therefore, not medically necessary.

Limitations

All other uses of low-density lipoprotein apheresis are investigational/not clinically proven and, therefore, not medically necessary.

The frequency of low-density lipoprotein apheresis considered medically necessary varies, but typically averages about once every two weeks to obtain an intrapheresis low-density lipoprotein cholesterol (low-density lipoprotein-C) level ≤ 120 mg/dL. It may be medically necessary to treat members with homozygous familial hypercholesterolemia more frequently.

Alternative covered services

- Lifestyle management.
- Intensive lipid-lowering drug treatment.
- Surgery for members with severe familial hypercholesterolemia — ileal bypass and liver transplantation.
- For treatment of focal segmental glomerulosclerosis — corticosteroids, cyclophosphamide, or cyclosporine in members refractory to prednisone therapy, plasmapheresis, and renal transplantation.

Background

Familial hypercholesterolemia is a congenital metabolic disorder resulting in severe elevations of blood cholesterol levels (Youngblom, 2022). Left untreated, it can lead to early development of atherosclerosis and coronary heart disease. Total cholesterol concentrations in patients with heterozygous familial hypercholesterolemia typically range from 350 to 550 mg/dL, and in homozygous familial hypercholesterolemia range from 650 to 1,000 mg/dL. Long-term intensive cholesterol-lowering drug therapy significantly reduces or removes the excess lifetime risk of coronary heart disease, lowering the level of risk to that of the general population. Some remain intolerant of or refractory to cholesterol-lowering therapy and require adjunct therapy (Goldberg, 2011; Youngblom, 2022).

Familial hypercholesterolemia impacts women through life differently than men. Although cholesterol-lowering therapy is equally effective at diminishing cardiovascular risk in both sexes, women have higher untreated and treated low-density lipoprotein cholesterol levels, resulting in part from sex-specific hormonal and genetic determinants of lipoprotein metabolism. Women receive less intensive treatment, experience off-treatment periods due to family planning, and may experience premature atherosclerotic cardiovascular disease (Klevmoen, 2023; Zimodro, 2024).

Apheresis is the extracorporeal process of removing one or more blood constituents from whole blood and returning the remainder to the circulation. Therapeutic apheresis (also called blood component therapy) removes the abnormal pathogenic component, which, theoretically, should improve the disease course. Depending on clinical use, apheresis may be performed as a one-time-only treatment or several times per week for several weeks. For some, it may be a lifelong commitment.

Lipoprotein apheresis involves the selective extracorporeal removal of low-density lipoproteins, lipoprotein(a) particles, very low-density lipoproteins, or high-density lipoproteins from either whole blood or plasma using a series of membrane filtering devices (Feingold, 2023). It is used for disorders with marked hyperlipidemia.

Selective removal of the low-density lipoproteins can occur through several processes. The U.S. Food and Drug Administration (2025) has approved two systems for lipoprotein apheresis. Both are regulated as Class III devices indicated for removal of low-density lipoproteins from the plasma of high-risk patients for whom a lipid-lowering diet and maximum drug therapy have been ineffective or not tolerated:

- Dextran-sulfate adsorption, which selectively binds apolipoprotein B-containing lipoproteins (low-density lipoprotein, lipoprotein(a) particles, and very low-density lipoproteins). Marketed as the Liposorber® LA-15 system (Kaneka Pharma America Corp., New York, New York) (U.S. Food and Drug Administration, 2013).
- Heparin-induced extracorporeal low-density lipoprotein precipitation, which selectively precipitates out apolipoprotein B-containing lipoproteins from plasma at a given pH level in the presence of heparin. Marketed as HELP® (B. Braun Avitum AG, Melsungen, Germany).

Approval for Liposorber was extended as a Humanitarian Use Device for treatment of pediatric patients with primary focal segmental glomerulosclerosis either before renal transplantation or after renal transplantation when there is recurrence of the disease (U.S. Food and Drug Administration, 2013). Approval was extended again in 2018 for treatment of adult patients with nephrotic syndrome caused by primary focal segmental glomerulosclerosis.

Selective high-density lipoprotein apheresis involves selective removal of cholesterol from high-density lipoprotein, converting the major alpha high-density lipoprotein to pre-beta-like high-density lipoprotein, which is then re-infused to the patient. The pre-beta-like high-density lipoprotein is a form of high-density lipoprotein that enhances cholesterol transport to the liver and is thought to reduce atherosclerosis development and burden. No extracorporeal apheresis device for high-density lipoprotein apheresis has been approved for clinical use.

Findings

Guidelines

Several evidence-based guidelines recommend low-density lipoprotein apheresis for patients with severe familial hypercholesterolemia inadequately controlled with drug therapy. Early detection and treatment are essential to lowering the risk of cardiac morbidity and mortality.

The American Society for Apheresis recommends low-density lipoprotein apheresis for severe familial hypercholesterolemia (Connelly-Smith, 2023).

The National Lipid Association Expert Panel on Familial Hypercholesterolemia issued patient selection criteria for low-density lipoprotein apheresis based on low-density lipoprotein cholesterol levels, risk factors, and comorbidities. The Panel recommend low-density lipoprotein apheresis: for patients with homozygous familial hypercholesterolemia and low-density lipoprotein cholesterol ≥ 300 mg/dL (or non-high-density lipoprotein cholesterol ≥ 330 mg/dL); for patients with heterozygous familial hypercholesterolemia and low-density lipoprotein cholesterol ≥ 300 mg/dL (or non-high-density lipoprotein cholesterol ≥ 330 mg/dL) with zero to one risk factors; low-density lipoprotein cholesterol ≥ 200 mg/dL (or non-high-density lipoprotein cholesterol ≥ 230 mg/dL) with $\geq$ two risk factors; or high lipoprotein(a) ≥ 50 mg/dL, or low-density lipoprotein cholesterol ≥ 160 mg/dL (or non-high-density lipoprotein cholesterol ≥ 190 mg/dL) with very high-risk characteristics such as established coronary heart disease, other cardiovascular disease, or diabetes (Goldberg, 2011).

The National Lipid Association also issued a strong recommendation for treating women with familial hypercholesterolemia with low-density lipoprotein apheresis during pregnancy and breast feeding (Jacobson, 2015).

The Writing Committee for the American College of Cardiology (2016) suggested that low-density lipoprotein apheresis be reserved for patients with homozygous familial hypercholesterolemia, severe heterozygous familial hypercholesterolemia that is inadequately responsive to pharmacotherapy, or either homozygous familial hypercholesterolemia or severe heterozygous familial hypercholesterolemia and concomitant atherosclerotic cardiovascular disease during pregnancy. A subsequent American College of Cardiology guideline confirmed

the role of low density lipoprotein apheresis in selected patients with severe hypercholesterolemia whose low density lipoprotein cholesterol is inadequately controlled with drug therapy (Grundy, 2019).

A scientific statement by the American Heart Association supports lipoprotein apheresis as safe and effective for lowering low density lipoprotein levels in patients with familial hypercholesterolemia, including patients as young as 2 years with homozygous familial hypercholesterolemia, pregnant or breastfeeding women with severe atherosclerotic cardiovascular disease and/or familial hypercholesterolemia, and patients as young as 2 years with refractory focal segmental glomerulosclerosis (Gianos, 2024).

An expert consensus issued jointly by the European Rare Kidney Disease Network and the European Society of Pediatric Nephrology recommends low-density lipoprotein apheresis in children with homozygous familial hypercholesterolemia and provides guidelines for initiating treatment and managing low-density lipoprotein cholesterol levels. The consensus statement suggests starting low-density lipoprotein apheresis in children with low-density lipoprotein cholesterol levels exceeding 7.8 millimoles per liter (300 mg/dL) despite optimal lipid-lowering therapy, or in those with subclinical or clinical atherosclerotic cardiovascular disease and low-density lipoprotein cholesterol levels over 3.4 millimoles per liter (130 mg/dL). The guidelines recommend beginning treatment as early as possible in life. The quality of evidence for these recommendations is generally low due to the lack of randomized controlled trials (Reijman, 2024a).

Evidence review

The evidence for the impact of lipoprotein apheresis on atherosclerotic cardiovascular disease outcomes is derived from prospective and retrospective observational studies showing a consistent benefit across various cohorts when using major adverse cardiovascular events as primary end points. With a single session, lipoprotein apheresis can reduce lipoprotein (a) and low density lipoprotein cholesterol levels by approximately 65% to 85%. Reduction in inflammatory markers, prothrombotic factors, atherogenic high-density lipoprotein cholesterol components, blood viscosity, and endothelial dysfunction may be achieved, along with improvement in microvascular myocardial perfusion. Preeclampsia and clinical atherosclerotic cardiovascular disease with low density lipoprotein cholesterol levels above goal in the absence of familial hypercholesterolemia remain off-label uses with limited evidentiary support (Gianos, 2024).

Several systematic reviews and meta-analyses have examined the safety and efficacy of low-density lipoprotein apheresis for familial hypercholesterolemia.

Alothman (2022) conducted a systematic review and meta-analysis of seven studies of 194 children with homozygous familial hypercholesterolemia. Low-density lipoprotein apheresis dramatically reduced cholesterol levels, but also imposed a high treatment burden on patients, with most reporting it to be tiring, painful, uncomfortable, and time-consuming, compromising educational attainment. The review noted inadequate psychological support for these patients.

Wang (2016) systematically reviewed 38 studies of patients with familial hypercholesterolemia and found that low-density lipoprotein apheresis reduced low-density lipoprotein cholesterol levels by an average of 57% to 75% for homozygous cases and 58% to 63% for heterozygous cases. Luirink (2019) conducted a systematic review of 76 case series and case reports (n = 209 participants) and found that lipoprotein apheresis was safe and substantially reduced low-density lipoprotein cholesterol and xanthomata in children with homozygous familial hypercholesterolemia.

Click (2015) conducted a systematic review and identified five small randomized controlled trials and observational studies supporting the safety and efficacy of low-density lipoprotein apheresis for reducing serum cholesterol levels in patients with familial hypercholesterolemia who do not respond to diet and intensive drug treatment. A few studies found improved coronary blood flow and halted or reversed stenosis progression, but

long-term follow-up was lacking. Belanger (2022) systematically reviewed the literature and documented improvement in survival among persons with hypercholesterolemia treated with lipoprotein apheresis.

Gu (2024) conducted a systematic review of 25 seminal studies on treatments for homozygous familial hypercholesterolemia. They found that low-density lipoprotein apheresis reduced low-density lipoprotein cholesterol levels by around 40% compared to baseline based on the best available evidence from five observational studies. However, these studies were considered at high to very high risk of bias and limited by confounding, with the largest study including only 30 patients. In contrast, high-quality evidence from randomized controlled trials demonstrated that monoclonal antibody therapy using evinacumab, evolocumab, and alirocumab reduced low-density lipoprotein cholesterol by 49%, 31%, and 36%, respectively, compared to placebo in trials totaling 184 patients. While low-density lipoprotein apheresis is effective, the authors concluded that it places a significant burden on patients and healthcare systems. They recommend that combinations of newer pharmacologic agents be considered and individualized for patients with homozygous familial hypercholesterolemia to optimize treatment outcomes.

Several observational studies support the safety and efficacy of low-density lipoprotein apheresis for familial hypercholesterolemia. Two registry studies (Luirink, 2020; Pottle, 2019) confirmed the safety and efficacy of lipoprotein apheresis for treating participants with familial hypercholesterolemia, and one intervention study found lipoprotein apheresis safe and effective for treating patients with primary focal segmental glomerulosclerosis (Raina, 2019a). Two observational studies conducted in Germany found that lipoprotein apheresis had a lasting effect on preventing cardiovascular events and improving peripheral circulation, pain level, walking distance, and the need for repeat peripheral revascularizations in patients with lipoprotein(a)-hyperlipoproteinemia (Poller, 2017; Roeseler, 2016).

An international registry study of children with homozygous familial hypercholesterolemia compared the long-term outcomes of participants who initiated high-frequency lipoprotein apheresis in childhood (lipoprotein apheresis group) and those who only received lipid-lowering drugs (pharmacotherapy-only group), matched by sex and untreated plasma low density lipoprotein cholesterol concentrations ($n = 125$ per group). Lipoprotein apheresis was associated with greater mean reductions in plasma cholesterol concentrations between baseline and final follow-up ($P < .0001$), longer atherosclerotic cardiovascular disease-free survival, and longer cardiovascular death-free survival. However, outcomes in the pharmacotherapy-only group trended toward a lower rate of cardiovascular death and a lower median age at coronary artery bypass grafting (Reijman, 2024b).

In 2024, we reordered and condensed the findings section by evidence type and added a position paper (Reijman, 2023) and a new systematic review (Gu, 2023).

In 2025, we updated the references with no policy changes warranted.

References

On May 7, 2025, we searched PubMed and the databases of the Cochrane Library, the U.K. National Health Services Centre for Reviews and Dissemination, the Agency for Healthcare Research and Quality, and the Centers for Medicare & Medicaid Services. Search terms were “blood component removal” (MeSH); “hyperlipoproteinemia type II” (MeSH), “therapeutic apheresis,” and “lipoprotein apheresis.” We included the best available evidence according to established evidence hierarchies (typically systematic reviews, meta-analyses, and full economic analyses, where available) and professional guidelines based on such evidence and clinical expertise.

Alothman L, Belanger AM, Ruel I, et al. Health-related quality of life in homozygous familial hypercholesterolemia: A systematic review and meta-analysis. *J Clin Lipidol*. 2022;16(1):52-65. Doi: 10.1016/j.jacl.2021.11.014.

- Belanger AM, Akioyamen LE, Ruel I, Hales L, Genest J. Aortic stenosis in homozygous familial hypercholesterolaemia: A paradigm shift over a century. *Eur Heart J*. 2022;42(34):3227-3239. Doi: 10.1093/eurheartj/ehac.339.
- Connelly-Smith L, Alquist CR, Aqui NA, et al. Guidelines on the use of therapeutic apheresis in clinical practice – Evidence-based approach from the writing committee of the American Society for Apheresis: The ninth special issue. *J Clin Apher*. 2023;38(2):77-278. Doi: 10.1002/jca.22043.
- Feingold K, Grunfeld C. Lipoprotein apheresis. In: De Groot LJ, Chrousos G, Dungan K, et al., editors. Endotext [Internet]. South Dartmouth (MA): MDText.com, Inc. <https://www.ncbi.nlm.nih.gov/books/NBK425700/>. Published 2000. Last updated February 19, 2023.
- Gianos E, Duell PB, Toth PP, et al. Lipoprotein apheresis: Utility, outcomes, and implementation in clinical practice: A scientific statement from the American Heart Association. *Arterioscler Thromb Vasc Biol*. 2024;44(12):e304-e321. Doi: 10.1161/atv.0000000000000177.
- Gidding SS, Champagne MA, de Ferranti SD, et al. The agenda for familial hypercholesterolemia: A scientific statement from the American Heart Association. *Circulation*. 2015;132(22):2167-2192. Doi: 10.1161/CIR.0000000000000297.
- Goldberg AC, Hopkins PN, Toth PP, et al. Familial hypercholesterolemia: screening, diagnosis and management of pediatric and adult patients: Clinical guidance from the National Lipid Association Expert Panel on Familial Hypercholesterolemia. *J Clin Lipidol*. 2011;5(3 Suppl):S1-S8. Doi: 10.1016/j.jacl.2011.04.003.
- 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. *J Am Coll Cardiol*. 2019;139(25):e1082-e1143. Doi: 10.1016/j.jacc.2018.11.003.
- Gu J, Gupta RN, Cheng H, Xu Y, Raal FJ. Current treatments for the management of homozygous familial hypercholesterolemia: A systematic review and commentary. *Eur J Prev Cardiol*. 2024;31(15):1833-1849. Doi: 10.1093/eurjpc/zwae144.
- Klevmoen M, Mulder J, Roeters van Lennep JE, Holven KB. Sex differences in familial hypercholesterolemia. *Current atherosclerosis reports*. 2023;25(11):861-868. Doi: 10.1007/s11883-023-01155-6.
- Luirink IK, Determeijer J, Hutten BA, et al. Efficacy and safety of lipoprotein apheresis in children with homozygous familial hypercholesterolemia: A systematic review. *J Clin Lipidol*. 2019;13(1):31-39. Doi: 10.1016/j.jacl.2018.10.011.
- Luirink IK, Hutten BA, Greber-Platzer S, et al. Practice of lipoprotein apheresis and short-term efficacy in children with homozygous familial hypercholesterolemia: Data from an international registry. *Atherosclerosis*. 2020;299:24-31. Doi: 10.1016/j.atherosclerosis.2020.01.031.
- Poller WC, Berger A, Dreger H, Morgera S, Enke-Melzer K. Lipoprotein apheresis in patients with peripheral artery disease and lipoprotein(a)-hyperlipoproteinemia: 2-year follow-up of a prospective single center study. *Atheroscler Suppl*. 2017;30:174-179. Doi: 10.1016/j.atherosclerosis.2017.05.007.
- Pottle A, Thompson G, Barbir M, et al. Lipoprotein apheresis efficacy, challenges and outcomes: A descriptive analysis from the UK Lipoprotein Apheresis Registry, 1989-2017. *Atherosclerosis*. 2019;290:44-51. Doi: 10.1016/j.atherosclerosis.2019.09.006.
- Raina R, Krishnappa V, Sanchez-Kazi C, et al. Dextran-sulfate plasma adsorption lipoprotein apheresis in drug resistant primary focal segmental glomerulosclerosis patients: Results from a prospective, multicenter, single-arm intervention study. *Front Pediatr*. 2019;7:454. Doi: 10.3389/fped.2019.00454. (a)

Raina R, Young C, Krishnappa V, Chanchlani R. Role of lipoprotein apheresis in cardiovascular disease risk reduction. *Blood Purif.* 2019;47(4):301-316. Doi: 10.1159/000497447. (b)

Reijman MD, Kusters DM, Groothoff JW, et al. Clinical practice recommendations on lipoprotein apheresis for children with homozygous familial hypercholesterolaemia: An expert consensus statement from ERKNet and ESPN. *Atherosclerosis.* 2024;392:117525. Doi: 10.1016/j.atherosclerosis.2024.117525.(a)

Reijman MD, Tromp TR, Hutten BA, et al. Cardiovascular outcomes in patients with homozygous familial hypercholesterolaemia on lipoprotein apheresis initiated during childhood: Long-term follow-up of an international cohort from two registries. *Lancet Child Adolesc Health.* 2024;8(7):491-499. Doi: 10.1016/s2352-4642(24)00073-7. (b)

Roeseler E, Julius U, Heigl F, et al. Lipoprotein apheresis for lipoprotein(a)-associated cardiovascular disease: Prospective 5 years of follow-up and apolipoprotein(a) characterization. *Arterioscler Thromb Vasc Biol.* 2016;36(9):2019-2027. Doi: 10.1161/atvbaha.116.307983.

U.S. Food and Drug Administration. FDA Summary of Safety and Probable Benefit (SSPB). Liposorber® LA-15 System. http://www.accessdata.fda.gov/cdrh_docs/pdf12/h120005b.pdf. Published 2013.

U.S. Food and Drug Administration. Humanitarian device exemption (H170002) approval letter. Liposorber® LA-15 System. https://www.accessdata.fda.gov/cdrh_docs/pdf17/H170002A.pdf. Published March 20, 2018.

U.S. Food and Drug Administration. Premarket Approval (PMA) database searched using product code MMY. <http://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfPMA/pma.cfm>. Page last updated April 28, 2025.

Wang A, Richhariya A, Gandra SR, et al. Systematic review of low-density lipoprotein cholesterol apheresis for the treatment of familial hypercholesterolemia. *J Am Heart Assoc.* 2016;5(7):e003294. Doi: 10.1161/JAHA.116.003294.

Writing Committee, Lloyd-Jones DM, Morris PB, Ballantyne, CM, et al. 2016 ACC Expert Consensus Decision Pathway on the role of non-statin therapies for LDL-cholesterol lowering in the management of atherosclerotic cardiovascular disease risk: A Report of the American College of Cardiology Task Force on Clinical Expert Consensus Documents. *J Am Coll Cardiol.* 2016;68(1):92-125. Doi: 10.1016/j.jacc.2016.03.519.

Youngblom E, Knowles JW. *Familial Hypercholesterolemia*. In: Pagon RA, Adam MP, Ardinger HH, et al., editors. GeneReviews® [Internet]. Seattle (WA): University of Washington, Seattle; 1993-2016. <http://www.ncbi.nlm.nih.gov/books/NBK174884/>. Published January 2, 2014. Last Revision January 30, 2025.

Zimodro JM, Mucha M, Berthold HK, Gouni-Berthold I. Lipoprotein metabolism, dyslipidemia, and lipid-lowering therapy in women: A comprehensive review. *Pharmaceuticals (Basel).* 2024;17(7)Doi: 10.3390/ph17070913.

Policy updates

6/2016: initial review date and clinical policy effective date: 10/2016

6/2017: Policy references updated.

7/2017: Policy references updated.

7/2018: Policy references updated.

7/2019: Policy references updated. Policy ID changed.

7/2020: Policy references updated.

7/2021: Policy references updated.

7/2022: Policy references updated.

CCP.1238

7/2023: Policy references updated.

7/2024: Policy references updated.

7/2025: Policy references updated.