targets NPC1L1, causes a further reduction in cholesterol absorption. Thus, LIMA1 could be a novel target for cholesterol-lowering strategies.

To be sure, this discovery is big news in the lipid world. Any time a new mediator of an important biological process is revealed, it is meritorious; the discovery of LIMA1 adds meaningful and valuable mechanistic insights to the biology of cholesterol absorption. Moreover, it adds another puzzle piece toward elucidating the mechanisms that control cholesterol homeostasis. Many patients are intolerant to ≥1 of the several classes of lipid-lowering medications or they fail to achieve desired cholesterol lowering, making novel cholesterol-controlling proteins appealing potential therapeutic targets.

This seems to be a prolific time for CVD risk mitigation. The last major discovery of a cholesterol homeostasis modulator (namely, PCSK9) uncovered an entirely new mode of lipid control by directing the degradation of LDL receptors. This finding was subsequently translated into a successful immunotherapy. Another targeted biologic, canakinumab, confirmed the long-debated theory that inflammation by itself is causal to CVD. With half of daily cholesterol input coming from the diet, might targeting the gastrointestinal brush border be equally efficacious? At present, ezetimibe treatment offers only relatively modest lipid-lowering capabilities, but there may still be potential for substantial therapeutic advancement. Perhaps inhibiting dietary absorption has not been fully optimized with current pharmacotherapy targets, and LIMA1 will serve as a more effective (or at least complementary) therapeutic target. Time will tell. For now, we have another piece of a complex puzzle to aid us going forward.

Author Contributions: All authors confirmed they have contributed to the intellectual content of this paper and have met the following 4 requirements: (a) significant contributions to the conception and design, acquisition of data, or analysis and interpretation of data; (b) drafting or revising the article for intellectual content; (c) final approval of the published article; and (d) agreement to be accountable for all aspects of the article thus ensuring that questions related to the accuracy or integrity of any part of the article are appropriately investigated and resolved.

Authors’ Disclosures or Potential Conflicts of Interest: No authors declared any potential conflicts of interest.

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