The first paper emphasizes that smooth-muscle proliferation is the key event in atherogenesis, which begins as a result of endothelial injury that alters endothelial structure and function. The second paper demonstrates early observations in hypercholesterolemia and the ways that lesions of atherogenesis caused by hypercholesterolemia result in specific forms of endothelial injury and cellular interactions. These precede the development of the intimal proliferative lesions of atherogenesis. [The SCI indicates that these papers have been cited in over 700 and 425 publications, respectively.]

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These two papers represent the initiation and early steps in the process of our conceptualization of the "response to injury hypothesis of atherogenesis." When John A. Glomset and I wrote the 1973 article, we were aware of endothelial changes that might precede platelet interactions and of increased cellular interactions, but we chose to emphasize the importance of intimal smooth-muscle proliferation as the key event in advanced lesion formation in atherogenesis. The research that led to these data included studies demonstrating that smooth-muscle cells are collagen-forming cells, studies showing that it is possible to grow differentiated smooth muscle in culture, and, in the second paper with Laurence Harker, studies incorporating the concept that growth factors released from platelets and ultimately from numerous cells in addition to platelets, nor was it clear how often platelets in-