The skeletal muscle tissue can adapt to exercise and environmental stressors with a remarkable plasticity. Prolonged cold stress exposure has been associated to increased skeletal muscle capillarization. Angioadaptation refers to the coordinated molecular and cellular processes that influence the remodeling of skeletal muscle microvasculature. Two cell types are central to angioadaptation: the myocytes, representing an important source of angiokines; and the skeletal muscle endothelial cell (SMECs), targets of these angiokines and main constituents of muscle capillaries. The influence of cold stress on skeletal muscle angioadaptation remains largely unknown, particularly with respect to myocyte-specific angiokines secretion or endothelial cell angioadaptive responses. Here, we use an *in vitro* model to investigate the impact of cold stress (28°C *versus* 37°C) on C2C12 myotubes and SMECs. Our main objectives were to evaluate: 1) the direct impact of cold stress on C2C12 cellular expression of angiokines and their release in the extracellular environment; 2) the indirect impact of cold stress on SMECs migration *via* these C2C12-derived angiokines; and 3) the direct effect of cold stress on SMECs angioadaptive responses, including migration, proliferation, and the activation of the vascular endothelial growth factor receptor-2 (VEGFR2). Cold stress reduced the secretion of angiokines in C2C12 myotubes culture media irrespective their pro-angiogenic or angiostatic nature. In SMECs, cold stress abrogated cell proliferation and reduced the activation of VEGFR2 despite a greater expression of this receptor. Finally, SMECs pre-conditioned to cold stress displayed an enhanced migratory response when migration was stimulated in rewarming conditions. Altogether our results suggest that cold stress may be overall angiostatic. However, cold stress accompanied by rewarming may be seen as a pro-angiogenic stressor for SMECs. This observation questions the potential for using pre-cooling in sport-performance or therapeutic exercise prescription to enhance skeletal muscle angioadaptive responses to exercise.