This international collaborative review set out to explore the mechanisms that underlie prolonged low-frequency force depression (PLFFD). In short, PLFFD is characterized by the slow recovery of submaximal muscle strength following various types of exercise (covered in this review). This prolonged impairment in submaximal strength can have drastic effects on increasing perceived exertion during subsequent tasks (i.e., everyday locomotion or sport specific demands). Our review discusses the differences in PLFFD following metabolically and mechanically demanding exercise. Metabolically demanding exercise encompasses exercise at high intensities (HIT) which generates reactive oxygen/nitrogen species whereas on the other spectrum, prolonged low intensity exercise such as endurance exercise causes muscle glycogen depletion. In contrast, mechanically demanding exercise involves eccentric muscle contractions that can be accompanied by severe muscle damage. Herein we show that PLFFD is explained by intramuscular mechanisms and is due to either reduced sarcoplasmic reticulum calcium (Ca^{2+}) release or reduced myofibrillar Ca^{2+} sensitivity, and the contribution of these mechanisms to PLFFD differs depending on whether metabolically or mechanically demanding exercise is performed. With this review, we hope to highlight some of the existing mechanisms, areas of future research, and potential interventions to combat PLFFD to better help accelerate post-exercise recovery.