Platelet rich plasma (PRP) is defined by the Red Cross as plasma with a 2 or more fold increase in platelet concentration above baseline levels or $>1.1\times10^6$ platelets/μl. PRP is generated primarily by centrifugation or gravity filtration. Between the numerous commercial preparations, there are differences in the volume of blood required, time and speed of centrifugation, addition of an activating agent, leukocyte concentration, method of delivery, and qualitative/quantitative differences with respect to final PRP volume and final platelet and growth factor concentrations. Overall, the final PRP platelet concentration is 2-8 times over baseline while leucocytes can be reduced or concentrated in the final PRP preparation. A key concept to understand is that PRP is a milieu of growth factors, cytokines, perhaps some RBC, and all the proteins, electrolytes, hormones, etc that are present in plasma.

The concept that PRP would improve repair of musculoskeletal tissues is based on the physiologic role of platelets in wound healing. Through modulation of the inflammatory response, promotion of local angiogenesis, attraction of fibroblasts and local stem cells to the site of injury and an induction of autocrine growth factor production by uninjured adjacent cells, platelets and their products are instrumental in normal tissue repair and regeneration. However, the positive effects of the growth factors will be tempered if the PRP contains high concentrations of leucocytes which synthesize and store catabolic cytokines such as interleukin-1. The exact ratio of platelets:leukocytes required for optimal healing is not known, nor is the absolute number of platelets or leucocytes.

An emerging paradigm shift is that PRP acts to decrease inflammation and pain in part through down-regulation of TNF-alpha and increase in synthesis of hyaluronic acid. The magnitude of this anti-nociceptive appears to be related to the platelet:leukocyte ratio with PRP preparations of higher ratios being more anti-nociceptive than those containing higher concentrations of leucocytes. This suggests that PRP could potentially serve as an endogenous source of chondroprotection and joint lubrication following intra-articular application.

Once isolated, the PRP can be injected into a joint or tendon with or without an activating (clotting) agent. When exposed to collagen or cells, platelets will be naturally activated and the clotting cascade will begin. A PRP clot serves as a fibrin matrix which serves as a scaffold for tissue repair and a reservoir for retention and slow release of growth factors. Our preliminary data indicates
that PRP can also act to recruit local stem cells.

The application of PRP in joints and tendons is relatively new and therefore there are limited clinical publications investigating its use. It is known that due to a variety of reasons, PRP is not always generated following centrifugation of blood. This means that the clinician does not know if the patient is being treated with PRP at all, and even if PRP is generated, the “dose” delivered is not known. When treating patients, clinicians should generate simple smear of both the blood and of the PRP for retrospective manual counting of the PRP in order to more accurately assess PRP technology in the treatment of musculoskeletal disorders.