Osteoarthritis is one of the most common, debilitating, musculoskeletal diseases in the world. Currently, there is no cure. It is well-known that a traumatic, joint injury increases the risk of developing post-traumatic osteoarthritis (PTOA). Therefore, in order to improve clinical treatment and prevention strategies for post-traumatic osteoarthritis (PTOA), a series of translational studies were conducted to develop research models to evaluate the effects of impact injury.

The first section of this dissertation (Ch. 1-2) provides a comprehensive introduction and literature review related to both clinical PTOA as well as previous research investigations of PTOA. The second section of this dissertation (Ch. 3-6) describes the methodology of optimizing a servo-hydraulic test machine to deliver a controlled impact injury (Ch. 3) as well as subsequent studies using this device to injure articular cartilage (Ch. 4) and cartilage-bone explants (Ch. 5-6). Further, the effects of dynamic, compressive loading to mimic walking after impact injury of cartilage-bone explants was investigated (Ch. 6). The third section of this dissertation (Ch. 7-8) details the development of an impactor device that may be used for pre-clinical, animal models.

Many significant findings were discovered through this dissertation work. Specifically, by using the proportional-integral-derivative (40, 0, 0) values, a large (25kN) servo-hydraulic test machine may be used to deliver a controlled impact injury to explants (Ch. 3). Biomarkers glycosaminoglycan (GAG) and prostaglandin E2 (PGE2) were elevated after cartilage impact injury with PGE2 having the highest mechanosensitivity than any other biomarker (Ch. 4). Energy absorbed during cartilage-bone injury is dependent upon trauma severity; PGE2 and monocyte attractant protein (MCP-1) were elevated following cartilage-bone injury (Ch. 5). Dynamic, compressive loading retained cell viability in non-impacted cartilage-bone explants and mitigated GAG release in impacted explants; GAG and PGE2 were elevated due to cartilage-bone injury whereas matrix metalloproteinase-2 (MMP-2) and interleukin-8 (IL-8) were elevated due to injury plus dynamic, compressive loading (Ch. 6). The development of a 8mm diameter impactor does create articular cartilage damage (Ch. 7), albeit a smaller, 2mm diameter impactor creates higher impact stresses and may be used arthroscopically for pre-clinical animal models (Ch. 8).