INTRODUCTION: Quantification of stability reserve (or margin) of intact/injured human musculoskeletal systems is crucial in performance evaluation, injury prevention and treatment managements. Dynamic stability of the knee joint in daily activities is maintained by a delicate interplay between the passive tissues and active musculature (voluntary and reflex). In clinical context, the mechanical stability of a biological system is usually rated by its hypermobility when excessive laxities are detected under external loads and disturbances. Joint hypermobility has been associated with pain and OA [1]. Generally greater muscle antagonist contractions are recorded via surface EMG in OA patients. Here, we aim, for the first time, (1) to quantify the mechanical stability of human knee joint in gait using a hybrid musculoskeletal model of the lower extremity and (2) to investigate the role of muscle antagonistic coactivation at different levels on both the joint stability margin as well as muscle/contact/ligament forces. We hypothesize that higher antagonistic coactiveness increases the knee joint stability margin but at the cost of augmenting also muscle forces, joint internal loads and tissue stresses and hence the risk of (further) injury.

METHODS: A validated iterative kinematics-driven musculoskeletal finite element (FE) model of the lower extremity is employed [2]. This model simulates the hip (3D) and ankle (1D) as spherical joints with no passive moment resistance but the knee joint in details as a complex nonlinear FE model consisting of bony structures (tibia, patella, and femur), their articular cartilage layers, menisci, major ligaments and patellar tendon. Cartilage and menisci are represented by nonlinear depth-dependent fibril-reinforced tissues while ligaments are modeled with nonlinear properties in tension and initial strains. The mean hip/knee/ankle joint rotations/moments and ground reaction forces (GRF) at the heel strike of gait collected on normal subjects drive the model [3]. Subject to gait kinematics-kinetics at HS with body weight of 606.6 N, the model is iteratively analyzed and the muscle forces are estimated using optimization. Muscle-tendon units are then each replaced by springs with a force-proportional tangent stiffness $K = q F/L$ [4,5] with $q$ as a dimensionless

RESULTS: At the heel strike and due mainly to the knee abduction rotation, lateral plateau carries a larger portion (~70%) of the joint load. The knee joint is stable at muscle stiffness coefficients $q \geq 14$ (i.e., $q_0 = 14$). With antagonistic coactivity at 3% and 5% levels, the joint stabilizes with critical $q$ dropping to ~13.5 and ~13, respectively while muscle forces (Fig. 1) as well as contact forces (Fig. 2) substantially increase. The force in ACL increases also from 238 N to 257 N and 283 N, respectively.

DISCUSSION: Coactivity in antagonist muscles has been recognized as a neural strategy to increase knee joint stability in both intact [6] and ACL deficient/OA [7,8] joints. Any injury or malfunction would further disturb the delicate mechanical balance between the external loads and internal passive/active resistances, demand additional compensatory cocontraction and increase the risk of secondary injuries and OA. Antagonistic cocontraction in uni- and bi-articular muscles crossing the knee joint increases the joint stability (as evident when critical $q$ decreases by ~7% at 5% coactivity level) albeit at the cost of additional activity in agonist muscles and increases in joint contact forces. The total joint compression force of 1.17 BW evaluated at HS with no coactivation (0%) substantially increased by 22% and 39% to 1.42 BW and 1.62 BW when 3% and 5% minimum activation levels were introduced, respectively. The increases in muscle coactivation have opposing effects on the system stability; on the one hand the stability margin improves due to increases in passive (due to larger joint compression) and active muscle stiffnesses while on the other hand it deteriorates due to larger compression on the joint. In this study, the net effect was positive in reducing critical $q$ from 14 to 13 which are within lower range of stiffness coefficient values found for different muscles [5]. The latter repercussion, observed also in human trunk with coactivity in abdominal muscles during lifting [9], points to a threshold of antagonistic coactivation beyond which the net effect on the knee joint stability would turn negative.

SIGNIFICANCE: The antagonistic coactivation is a neural strategy that augments margin of safety and control by increasing stiffness and stability in both intact and injured joints and that acts as an adaptation to reduce hypermobility in an injured joint. These increases in stability and control, however, are made at the price of substantial increases in agonist muscle forces, joint loads and tissue stresses and hence a higher risk of (further) injury.


ACKNOWLEDGEMENTS: Supported by the NSERC-Canada.