

Master student:

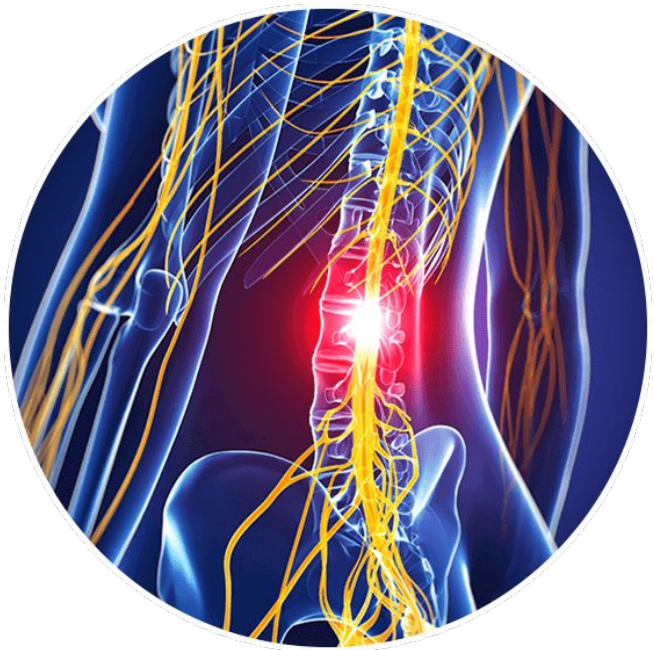
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**Muscle compensation and  
synergy reorganization in  
spinal cord injury using  
musculoskeletal models**

**Semester Project -  
Translational Neural  
Engineering Lab**

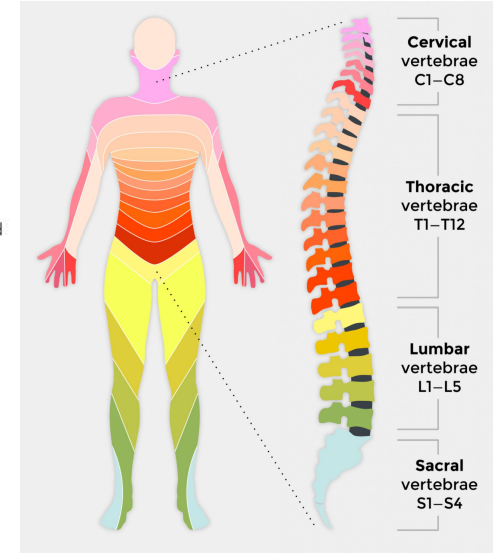
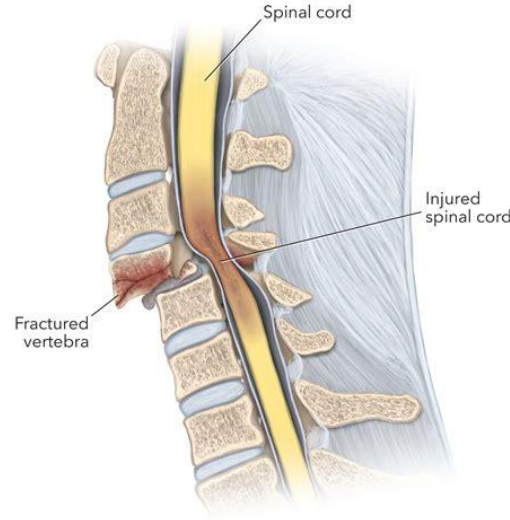


# Introduction

- Spinal Cord Injury (SCI)
- EES for Motor Recovery
- Use of MSK models for tailored neuromodulation

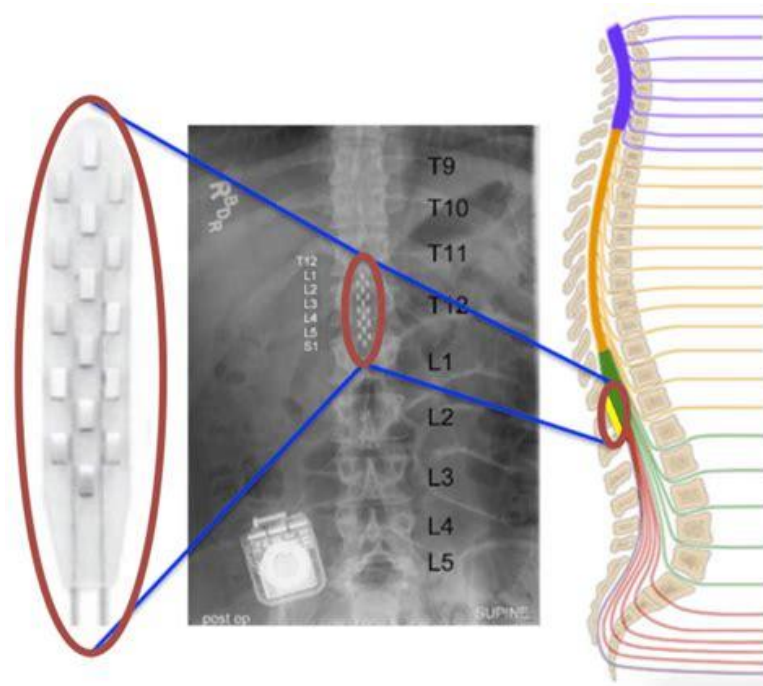
# Spinal Cord Injury (SCI)

- SCI is the damage to the spinal cord disrupting motor control
- **Consequences:** paralysis, weakness, spasticity, loss of coordination
- **Major challenge:** CNS has limited regeneration capacity
- Motor recovery requires restoring or bypassing disrupted pathways



# EES for motor recovery

- Epidural Electrical Stimulation (EES): patterned electrical stimulation to lumbar spinal cord
- Reactivates dormant circuits below the injury
- Enhances signal propagation and reflex modulation
- Enables voluntary movement in some individuals with SCI
- Strong evidence for walking recovery

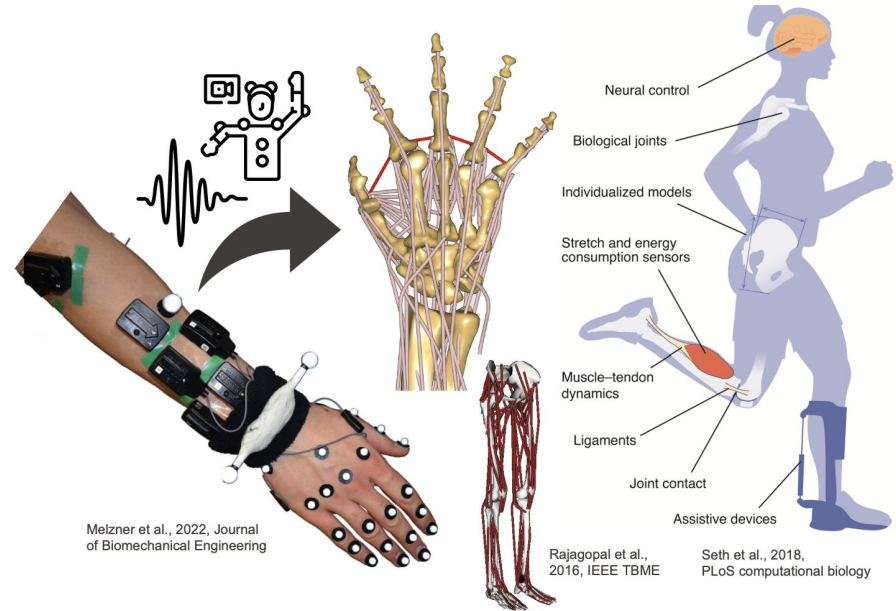


Wagner et al., 2018, *Targeted neurotechnology restores walking in humans with spinal cord injury*, **Nature**.

Rowald et al., 2022, *Activity-dependent spinal cord neuromodulation rapidly restores trunk and leg motor functions after complete paralysis*, **Nature Medicine**.

# Use of MSK models for tailored neuromodulation

- Simulate muscle activation and joint movement from neural or electrical input
- Integrate spinal stimulation (e.g., EES) to study neuromuscular response
- Predict biomechanical outcomes under healthy and pathological conditions
- Explore compensation strategies and synergy reorganization
- Support the design of personalized neuromodulation therapies

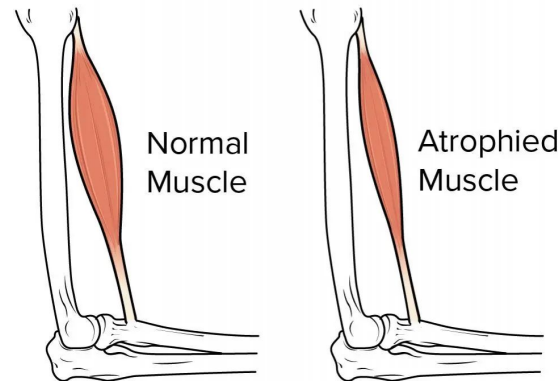
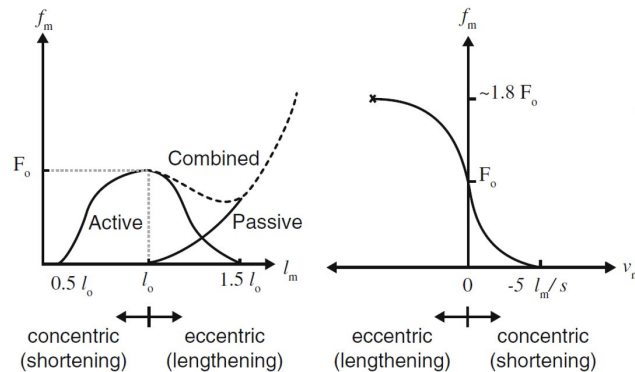




# Problem

- MSK models have default physiological parameters
- Understand muscle compensations with weak muscles

- Default MSK models assume **healthy muscles**
- SCI alters:
  - **Strength** ( $\downarrow$  force capacity)
  - **Spasticity** (abnormal tone/reflexes)
  - **Atrophy** ( $\downarrow$  muscle mass)
- Leads to inaccurate simulations if not adapted
- Must tailor models to reflect pathological muscle behavior



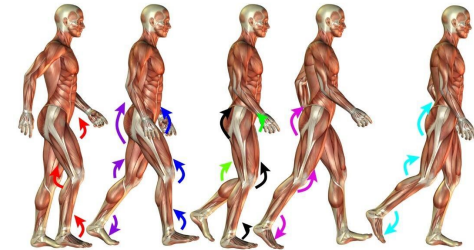
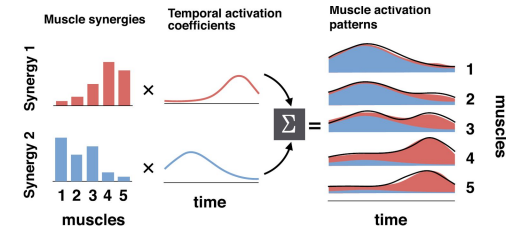


The aim of this project is to **adapt MSK models** to simulate **muscle weakness** and understand **compensation strategies** by:

- 1) Observing **muscle compensation** in single joint movements
- 2) Analyzing **muscle synergies changes** in the gait cycle

## Why muscle synergies?

- Muscles work in coordinated groups (synergies) to simplify motor control and enable efficient movement
- Understanding synergies reveals:
  - Neural control strategies
  - Adaptations to parameter changes







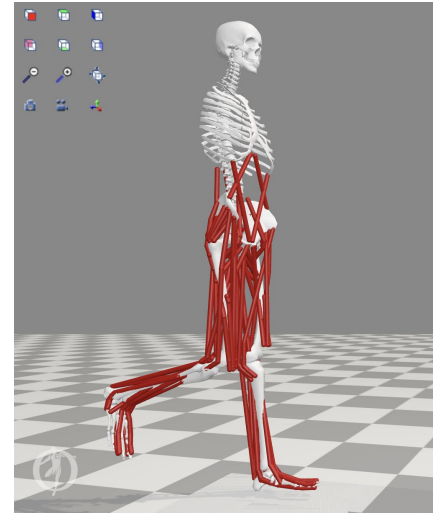
## Research Methodology

- **MSK models (OpenSim vs Myosuite)**
- **Forward and Inverse dynamics**
- **Implementing muscle weakness in simulation**

# MSK model used

## OpenSim – Gait2392

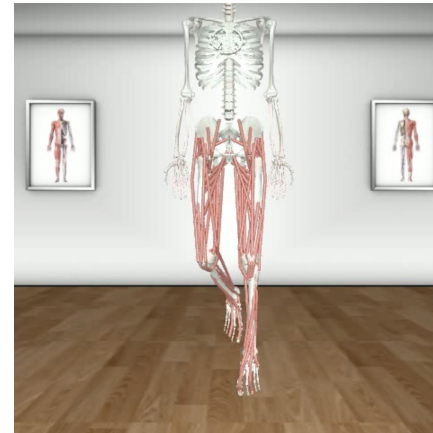
- 92 muscle-tendon units, 23 DoFs
- Validated model for lower-limb gait
- Used as biomechanical reference



OpenSim

## MyoSuite – MyoLeg Suspended

- 80 muscles-tendon units, 20 DoFs
- Single leg suspended in MuJoCo
- Flexible control of muscle parameters
- Real-time FD/ID & synergy analysis



MyoSuite

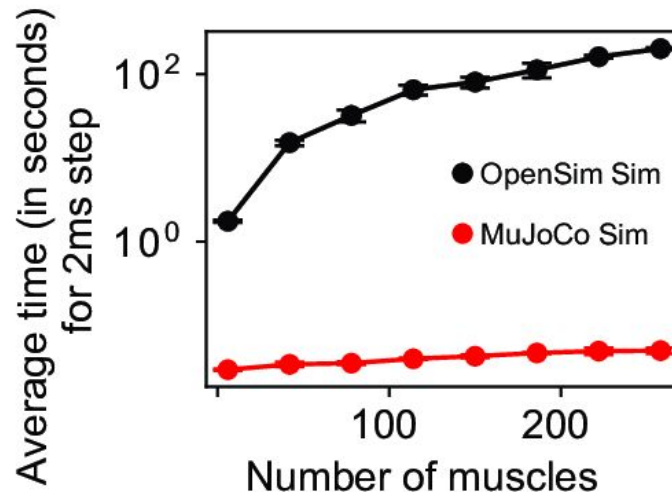
# OpenSim vs. Myosuite

## OpenSim

- Detailed anatomical models
- Computationally heavy
- Better for validation and analysis

## MyoSuite

- Real-time or faster-than-real-time dynamics
- Flexible control of muscle parameters (e.g., muscle weakening)
- Ideal for rapid prototyping and synergy analysis

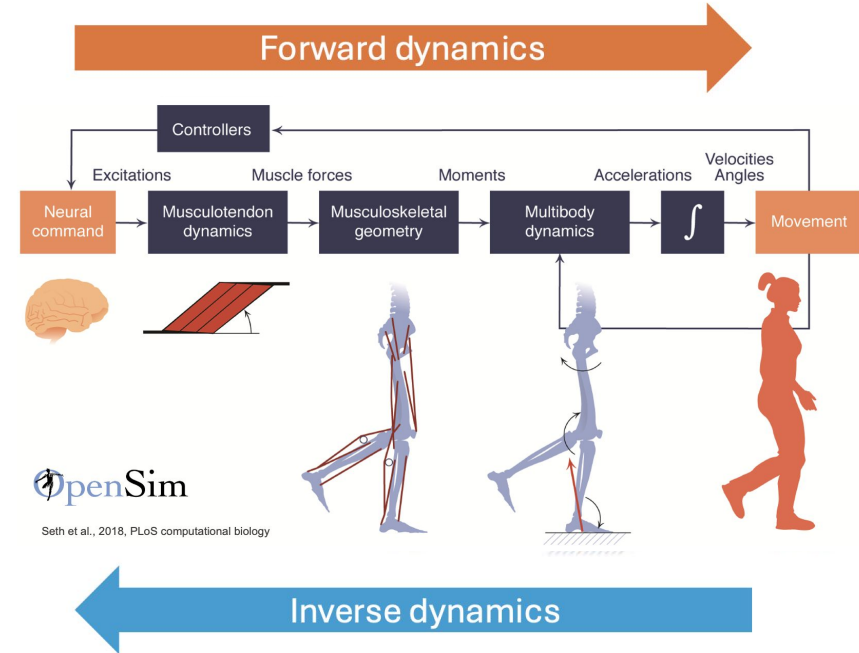


## Forward Dynamics (FD)

- Predicts motion from muscle activations
- Simulates joint angles, velocities, and forces

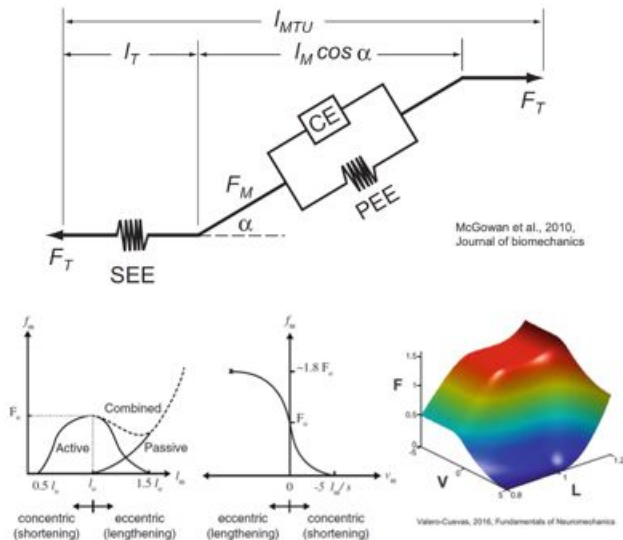
## Inverse Dynamics (ID)

- Computes muscle activations from motion
- Uses joint trajectories to estimate required forces



# Implementing muscle weakness in simulation

- Weakness implemented by scaling down the **maximum isometric force**  $F_{\max}$



$$F_{MTU}(l, v, act) = F_L(l) * F_V(v) * act + F_P(l)$$

- Can also be modulated with **optimal fiber length, tendon slack length, optimal pennation angle, ...**

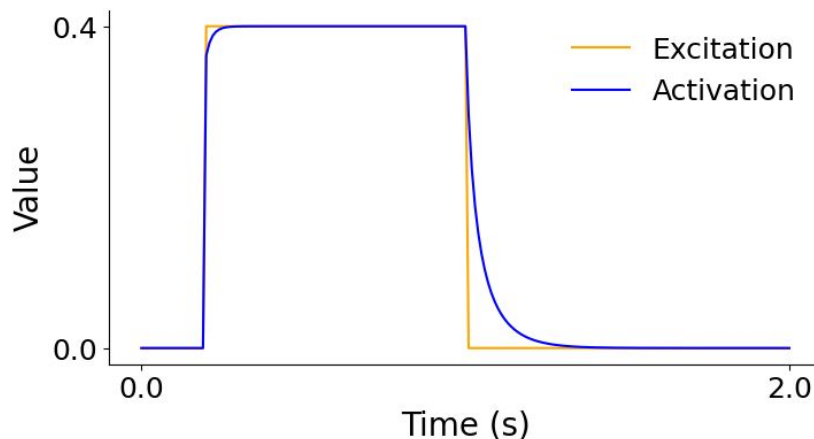
	Joint	Muscle	Maximum isometric force (N)	Optimal fiber length (m)	Optimal pennation angle (rad)
Type	Movement				
Hip	flex, inrot	iliacus	1073	0.100	0.122173
	flex, inrot	psoas	1113	0.100	0.139626339
	flex, add	add.long	627	0.138	0.10471976
	ext, add	add.mag1	381	0.0869	0.087266460
Hip/Knee	h.flex, k.ext	rect.fem	1169	0.114	0.08726646
	h.ext, h.add, k.flex	bifemilh	896	0.109	0
	h.ext, h.add, k.flex	semimem	1288	0.080	0.26179939
	h.ext, h.add, k.flex	semiten	410	0.201	0.08726646
Knee	flex	vas.med	1294	0.0889	0.08726646
	flex	vas.int	1365	0.0869	0.052359879
	flex	vas.lat	1871	0.084	0.08726646
	flex	bifemsh	804	0.1729	0.401425729
Knee/Ankle	k.flex, a.pf	med.gas	1558	0.059	0.296705969
	k.flex, a.pf	lat.gas	683	0.064	0.139626339
Ankle	df, inv	tib.ant	905	0.098	0.08726646
	df, inv	ext.hal	162	0.111	0.10471976

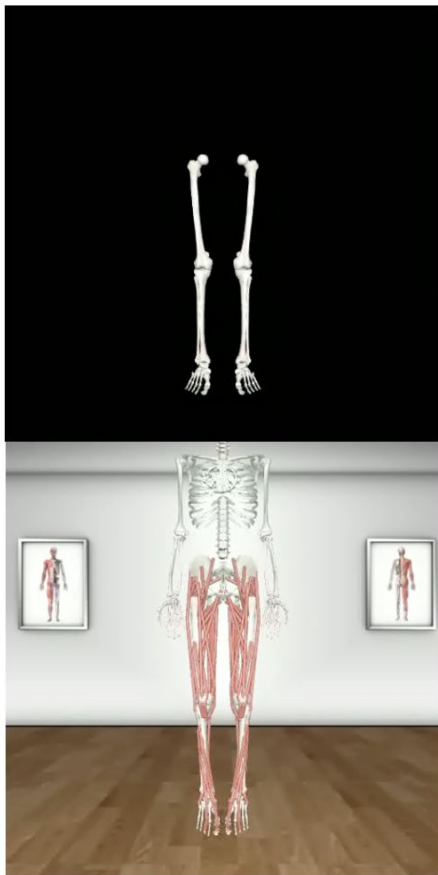
**Table:** Gait2392 Hill-type muscular model parameters

# Muscle excitation and activation dynamics

- Predefined excitation curves control timing & intensity
- Activation dynamics computed with time-dependent model
- $\tau_{act} = 10\text{ ms}, \tau_{deact} = 40\text{ ms}$
- Example: Excitation at 0.4 from 0.2s to 1.0s

Muscle Excitation and Activation Dynamics





## Results & Discussion

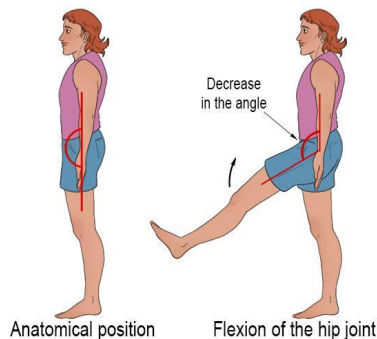
- **Simulating muscle activations in single joint movements**
- **Stronger activations of agonist muscles**
- **Synergies changes in gait cycle**



# Simulating muscle activations and joint movements

- Simulates muscle activation and joint movement using the MyoSuite model.
- Movements of interest:
  - Hip flexion
  - Hip adduction
  - Knee flexion
  - Ankle dorsiflexion
- Tracks:
  - joint angles
  - muscle activations
  - coordinates over time

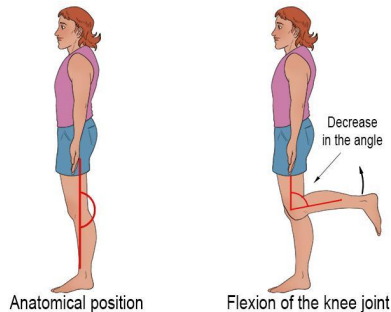
## Hip flexion



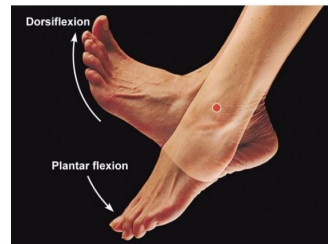
## Hip adduction



## Knee flexion



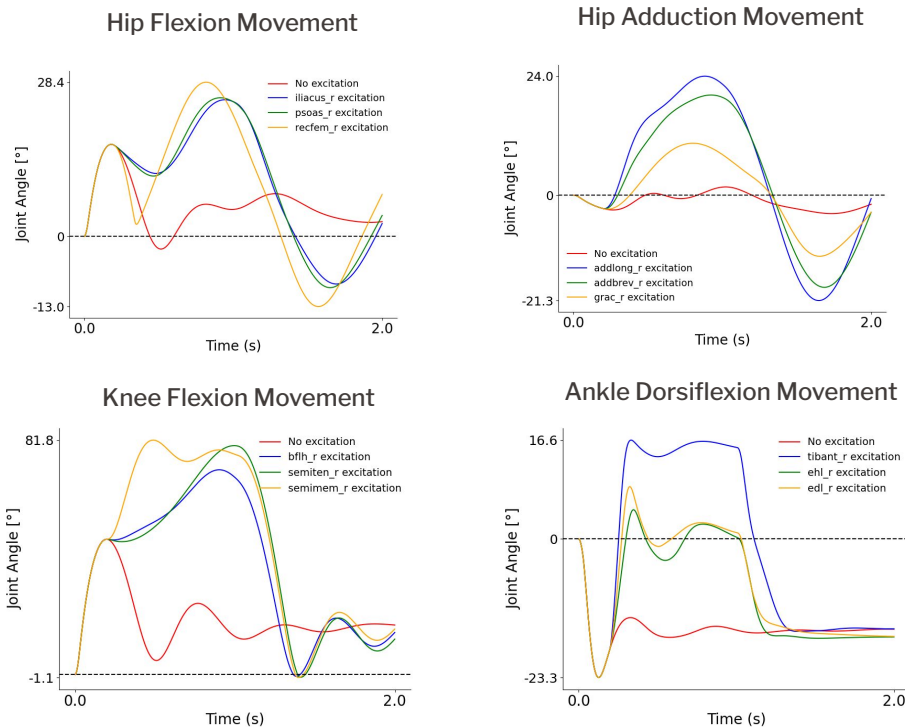
## Ankle dorsiflexion



# Simulating muscle activations and joint movements

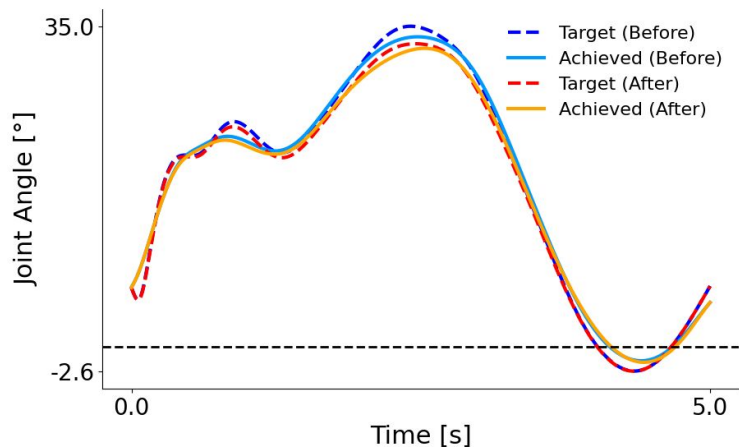
- Apply the **same excitation curves** to individual muscles
- Compute joint angles under:
  - Muscle excitation
  - No excitation
- Visualize joint trajectories for each movement
- Observe how **individual muscle stimulation affects kinematics**

## Joint Movements and Muscle Excitations

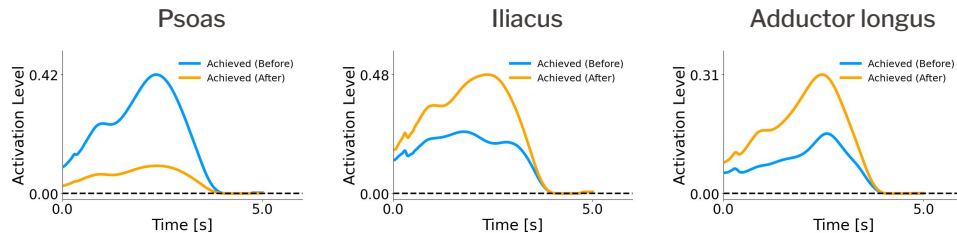


- Weak primary muscles lead to **increased activation of synergistic muscles**
- For instance, a **weak psoas** led to an increased activation in **iliacus** and **adductor longus**
- Observed across:
  - Hip flexion
  - Knee flexion
  - Ankle dorsiflexion

Kinematic comparison – Hip flexion  
(Right psoas's max isometric force set to 30% of original value)



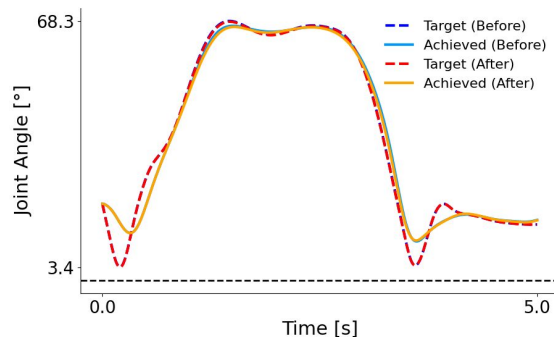
Muscle activations – Hip flexion  
(Right psoas's max isometric force set to 30% of original value)



# Stronger activations of compensatory muscles

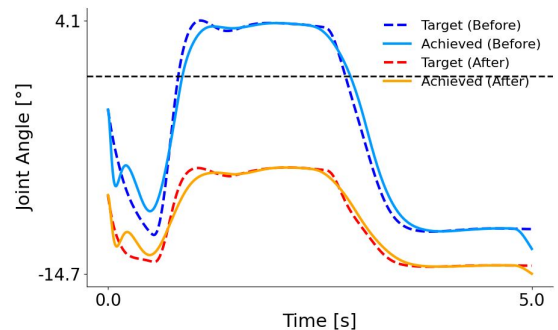
## Kinematic comparison – Knee flexion

(Right semimembranosus' max isometric force set to 30% of original value)



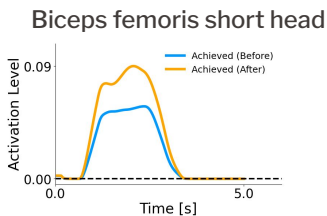
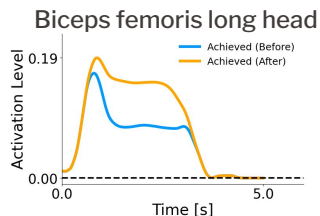
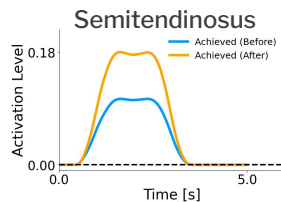
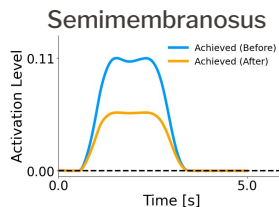
## Kinematic comparison – Ankle dorsiflexion

(Right tibialis anterior's max isometric force set to 30% of original value)



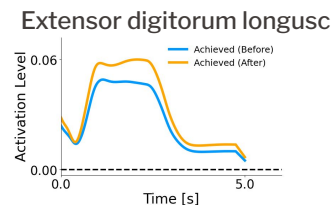
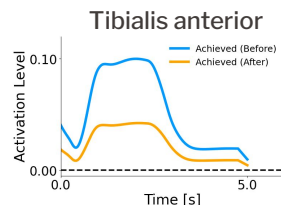
## Muscle activations – Knee flexion

(Right semimembranosus' max isometric force set to 30% of original value)



## Muscle activations – Ankle dorsiflexion

(Right tibialis anterior's max isometric force set to 30% of original value)

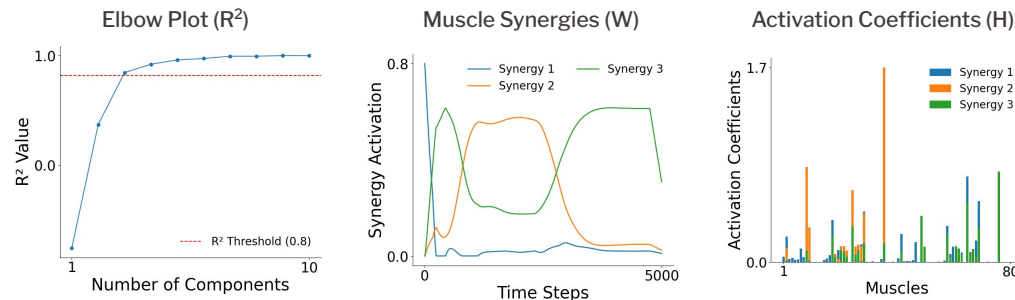


- Some movements can be compensated by other muscles
- Some don't !

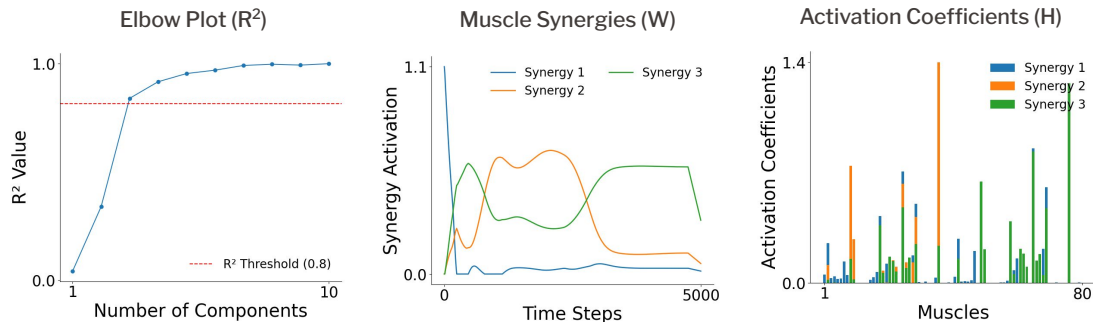
# Muscle synergies extraction

- Use of **Non-negative Matrix Factorization (NMF)**
- Determine the optimal number of synergies using  $R^2$  threshold
- Decomposes activation matrix into:
  - W: synergy patterns (muscle groups)
  - H: activation timing
- Compare synergies before vs. after changes (e.g., muscle weakness)

## Ankle dorsiflexion synergies before muscle parameter change



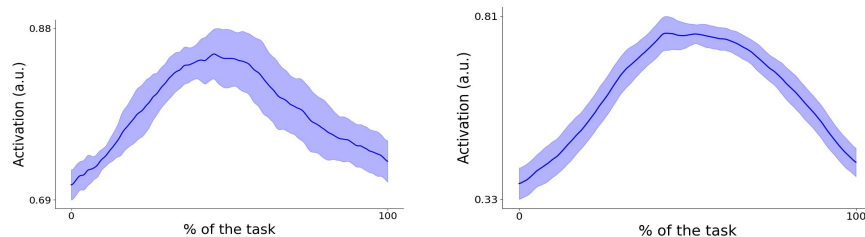
## Ankle dorsiflexion synergies after muscle parameter change (Right tibialis anterior's max isometric force set to 30% of original value)



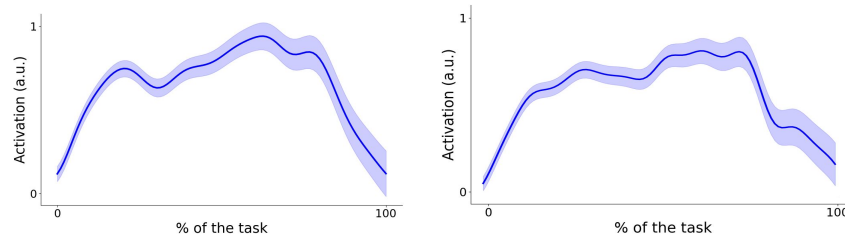
# Muscle activations & synergies changes

- Real human walking gait
- ID to segment gait cycles and compute mean + SD
- Iliacus and psoas peak during the swing phase (hip flexion)
- Hip flexors synergy shape preserved before/after psoas weakening
- Activation coefficients show reduced psoas contribution → **muscle compensation**
- Highlights **adaptive muscle recruitment**
- Synergies offer a **compact, interpretable view of motor control** and compensation

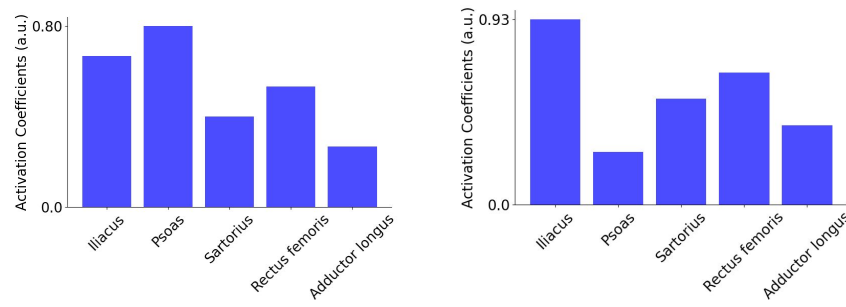
Muscle activations over task — Iliacus (left) and Psoas (right)



Synergy activation over time (before vs after parameter change) - Hip flexion synergy



Muscle contributions to synergy (before vs after) - Hip flexion synergy





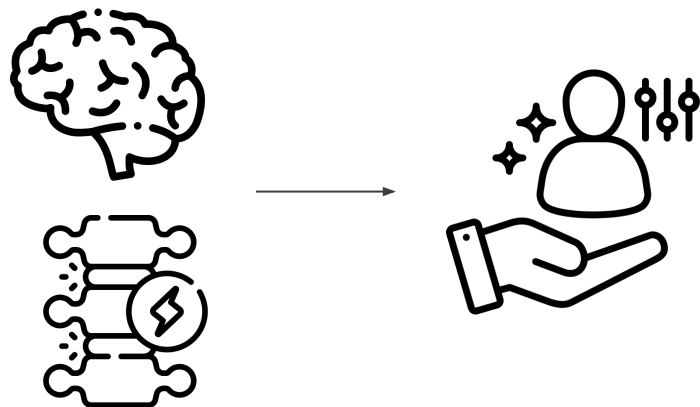
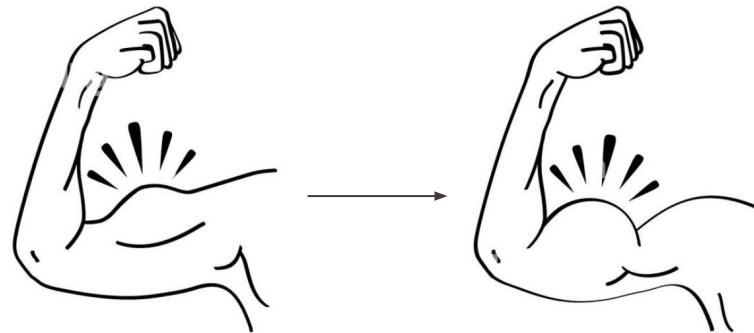
# Conclusion

- **Summary of achievements**
- **Limitation and further perspectives**



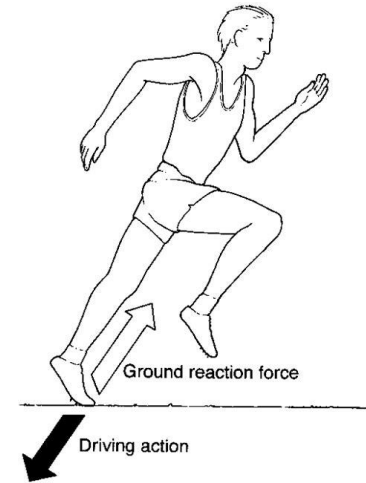
# Summary of achievements

- Weak muscles trigger increased activation in synergistic muscles, indicating **neural compensation**
- Synergy patterns are largely preserved, with subtle changes in timing and intensity
- Reorganization reflects **adaptive motor control** under impaired conditions
- Simulations provide insights into how coordination changes after injury
- Supports development of **personalized rehabilitation strategies** and **optimization of assistive devices**



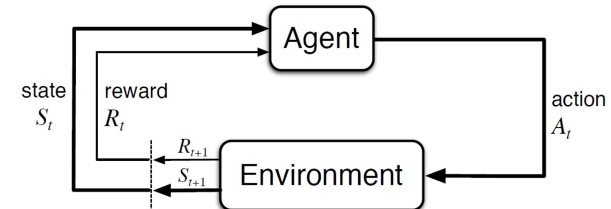
## Limitations:

- No Ground Reaction Forces → Limits stance, balance, and propulsion modeling
- Simplified neural control → No reflexes, spasticity, or patient-specific tone



## Future Perspectives:

- Add GRF simulation (foot contact models)
- Add neuromuscular pathologies (spasticity, hypertonia)
- Implement closed-loop EES with RL-trained controllers
- Modify multiple muscles/parameters based on patient data



# Thank you for listening!

## Questions?