

# Skeletal muscle extracellular matrix (ECM) and cytoskeleton in chronic kidney disease (CKD) – a role in insulin resistance?

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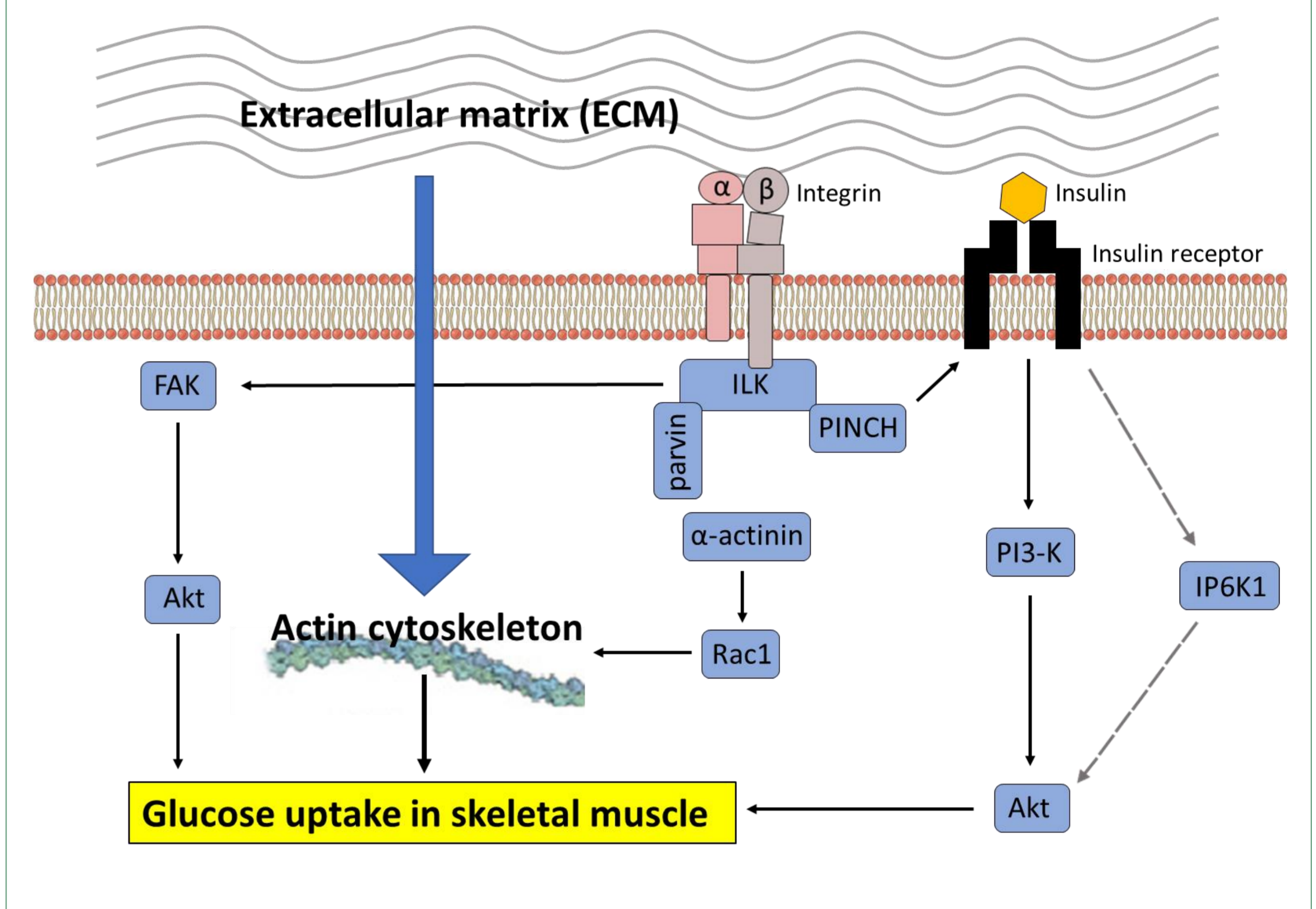
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## Background and Aim

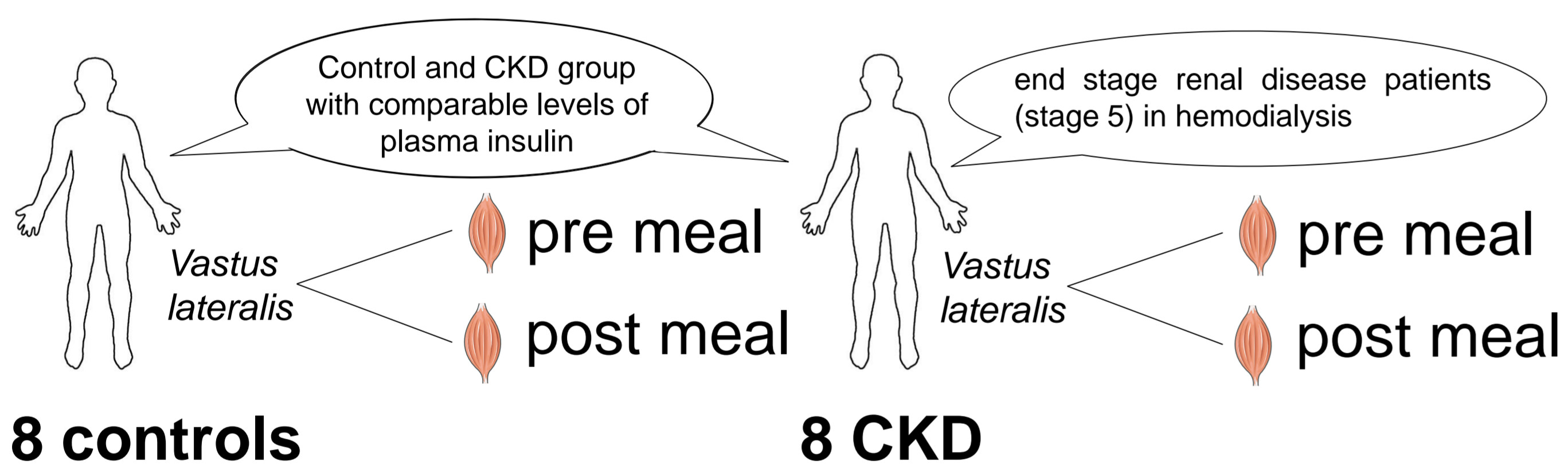
- **Chronic kidney disease (CKD)** is a worldwide public health problem associated with insulin resistance and muscle wasting.
- Novel research suggests that alterations or disruption to the linkage binding the cytoskeleton and the extracellular matrix (ECM) may contribute to muscle atrophy and insulin resistance in skeletal muscle.
- This research aims to investigate which proteins of the ECM could contribute to muscle wasting and insulin resistance development in CKD patients, among which:
  - The Rho GTPase **Rac1**,
  - The **ILK-PINCH-parvin** (IPP) complex,
  - The focal adhesion kinase (**FAK**),
  - The serine/threonine-specific protein kinase **Akt**
  - and the inositol phosphatase **IP6K1**.

## Hypothesis

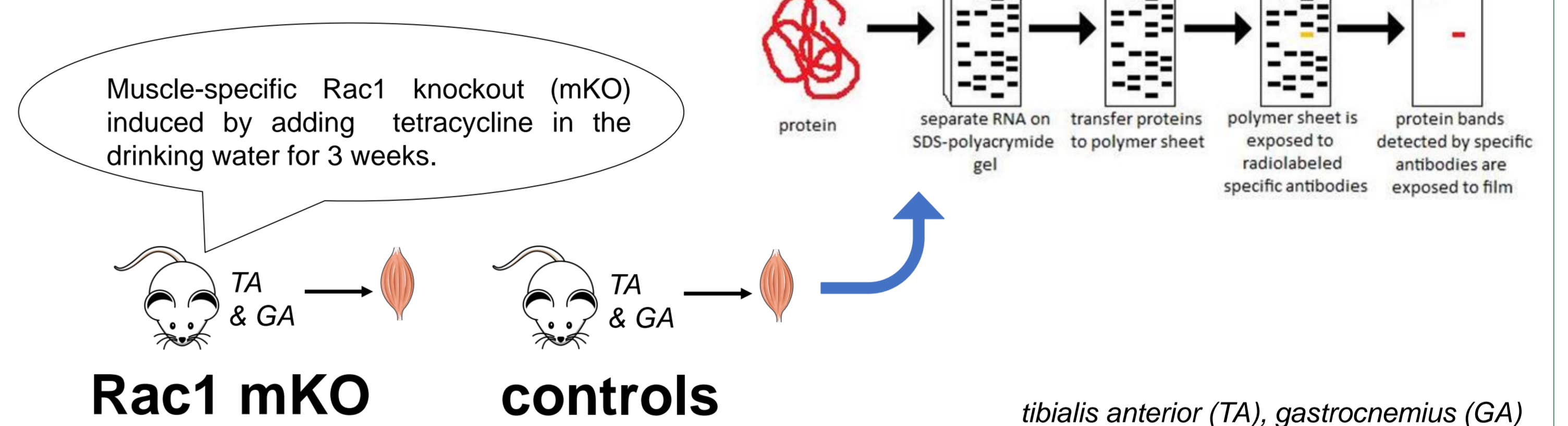


## Methods

### Human muscle samples

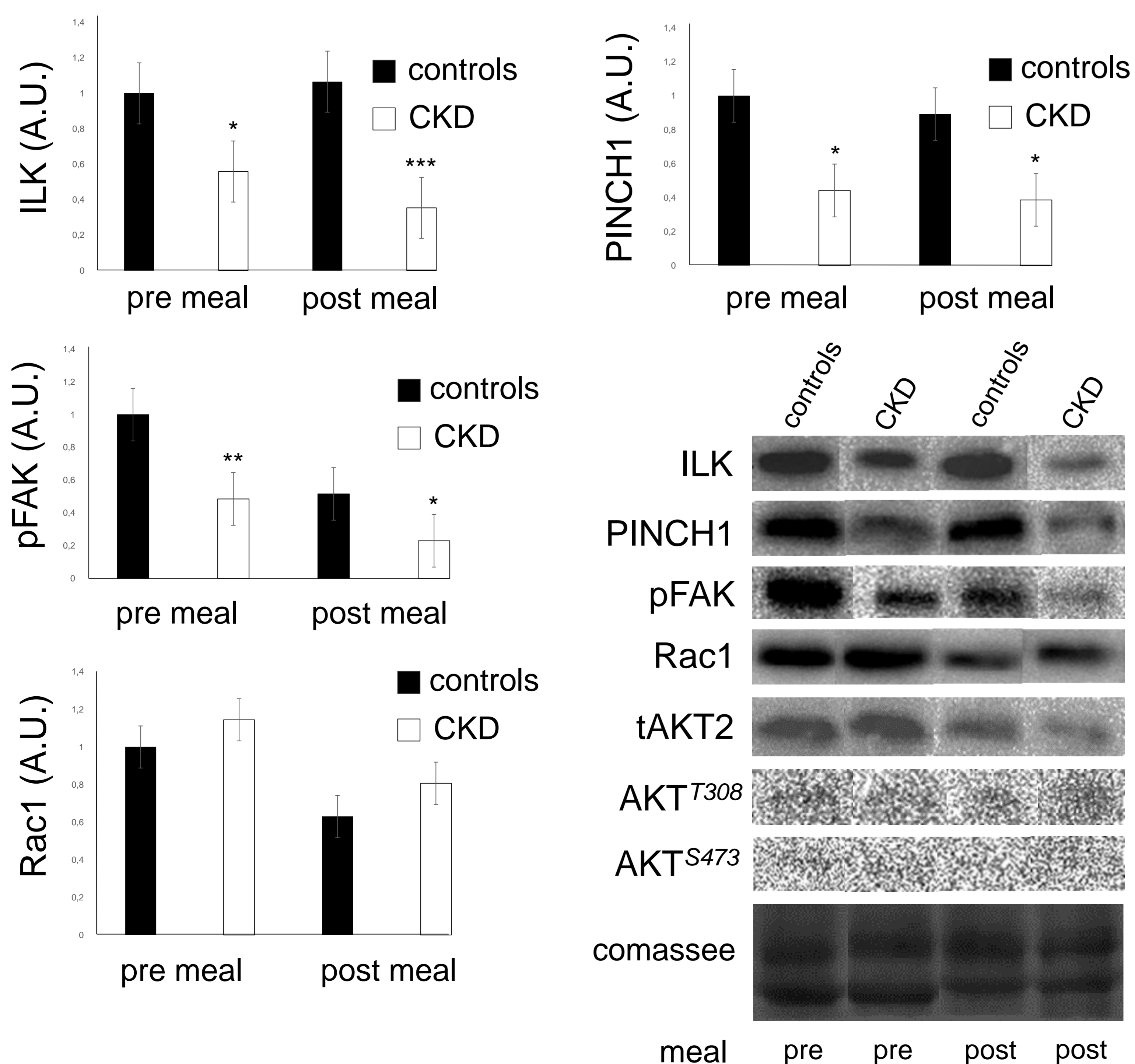


### Mouse muscle samples



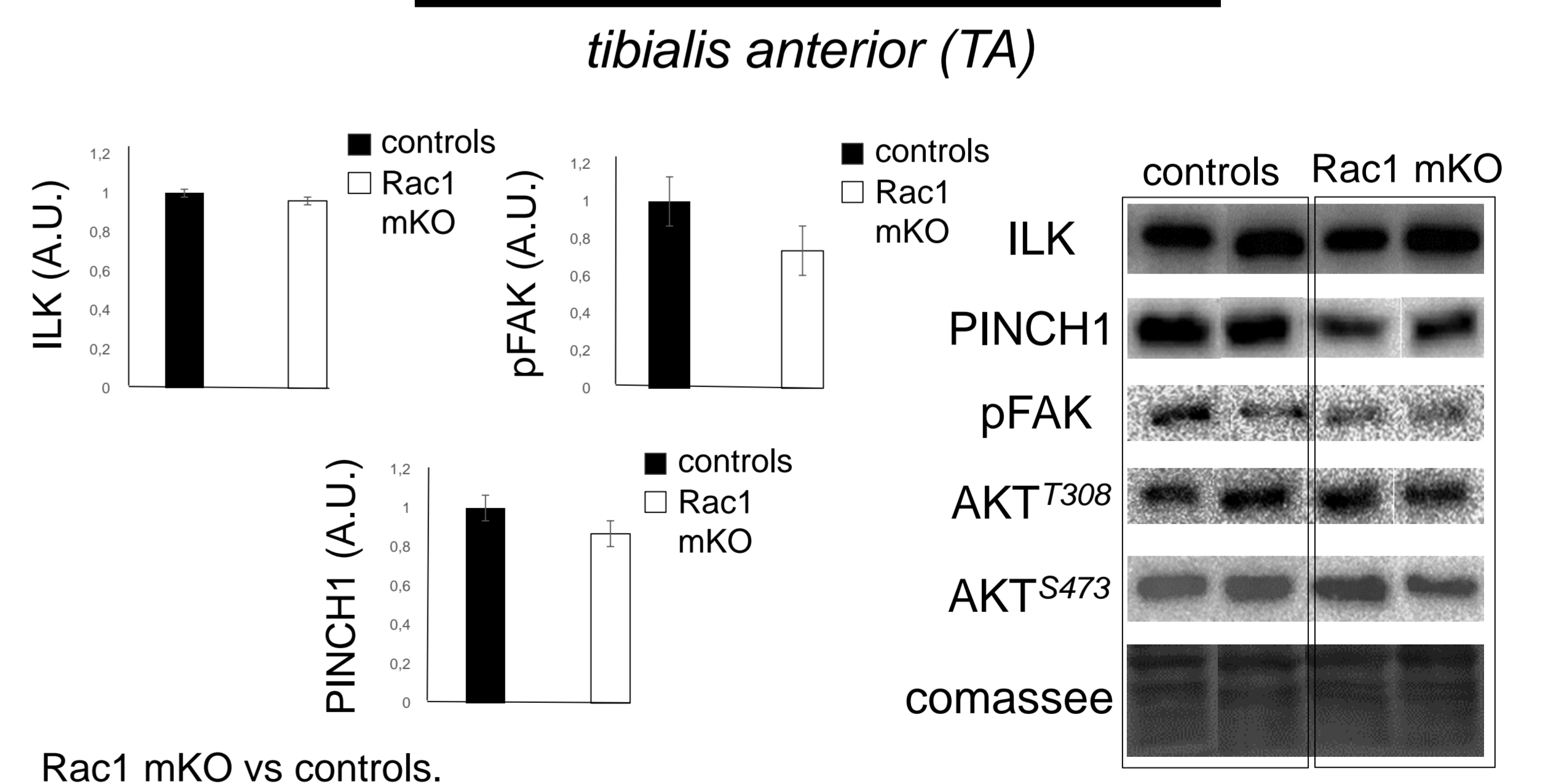
## Results

### Human muscle samples



\* P < 0.05. \*\* P < 0.01. \*\*\* P < 0.001. CKD vs controls.

### Mouse muscle samples



Rac1 mKO vs controls.

## Conclusions

- ILK, PINCH1 and pFAK levels are decreased in CKD muscles, which may contribute to the muscle degeneration and development of insulin resistance in people on hemodialysis.
- Rac1 is not altered in CKD subjects.
- Rac1 protein does not regulate ILK, PINCH1 or pFAK protein expression.

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