



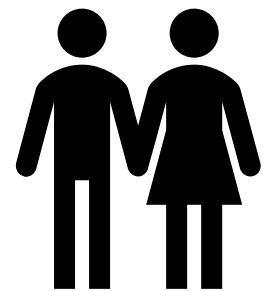
INTRODUCTION

The haemostatic imbalance remains an enigma in chronic kidney disease (CKD), and is associated with both bleeding and thrombotic complications, yet no reliable biomarker exists to predict individual risk. Understanding how CKD progression affects haemostasis is essential for improving risk prediction.

AIM

The study aimed to evaluate the impact of CKD progression on haemostatic biomarkers and global coagulation assays (GCAs).

METHOD



N=311 (> 18 years of age)
(Known Cardiovascular Risk Factors)
Northern Health, Victoria, Australia

Excluded: Use of anticoagulation, Active Malignancy, Infection

Sample Collection

Sodium Citrate Specimen (Double spun 2500g x 10 mins) to obtain platelet poor plasma

Global Coagulation Assays (GCA)

Prothrombin Time (PT), Activated Partial Thromboplastin Time (APTT), D-Dimer, Fibrinogen, Von Willebrand factor antigen (VWF: Ag), Factor VIII, Protein C, Protein S, Antithrombin, C-reactive protein (CRP)

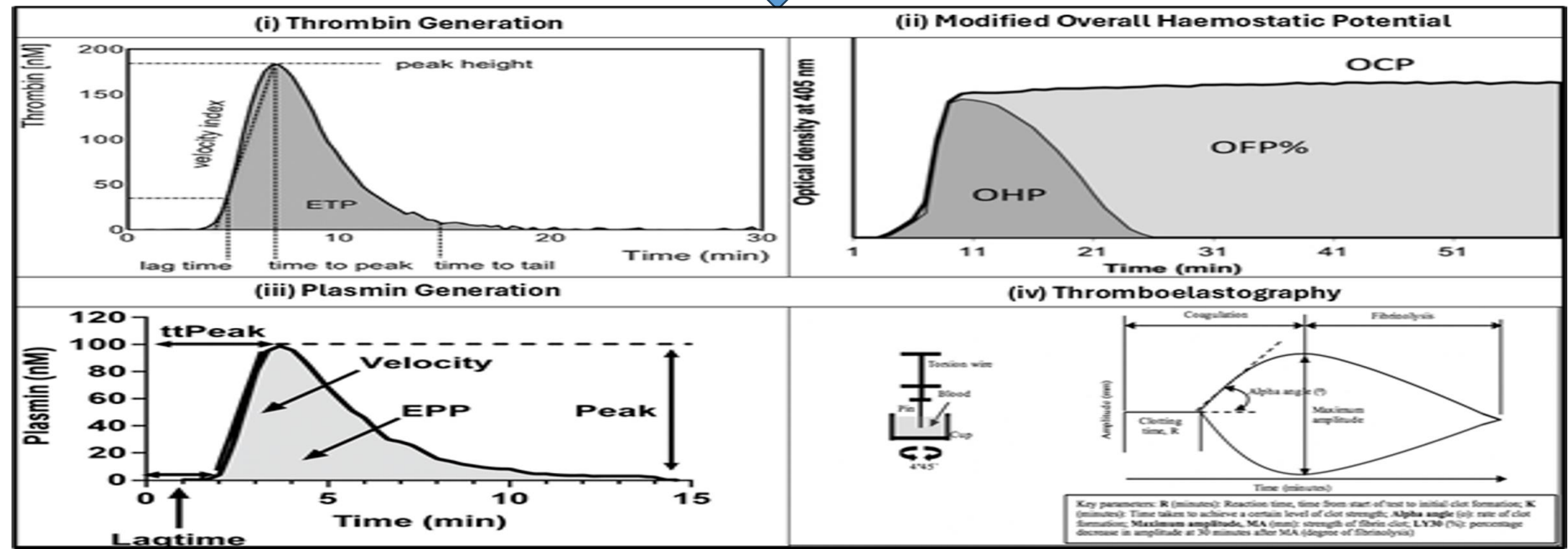


Fig 1 Global coagulation assays Curves

Haemostatic Biomarkers

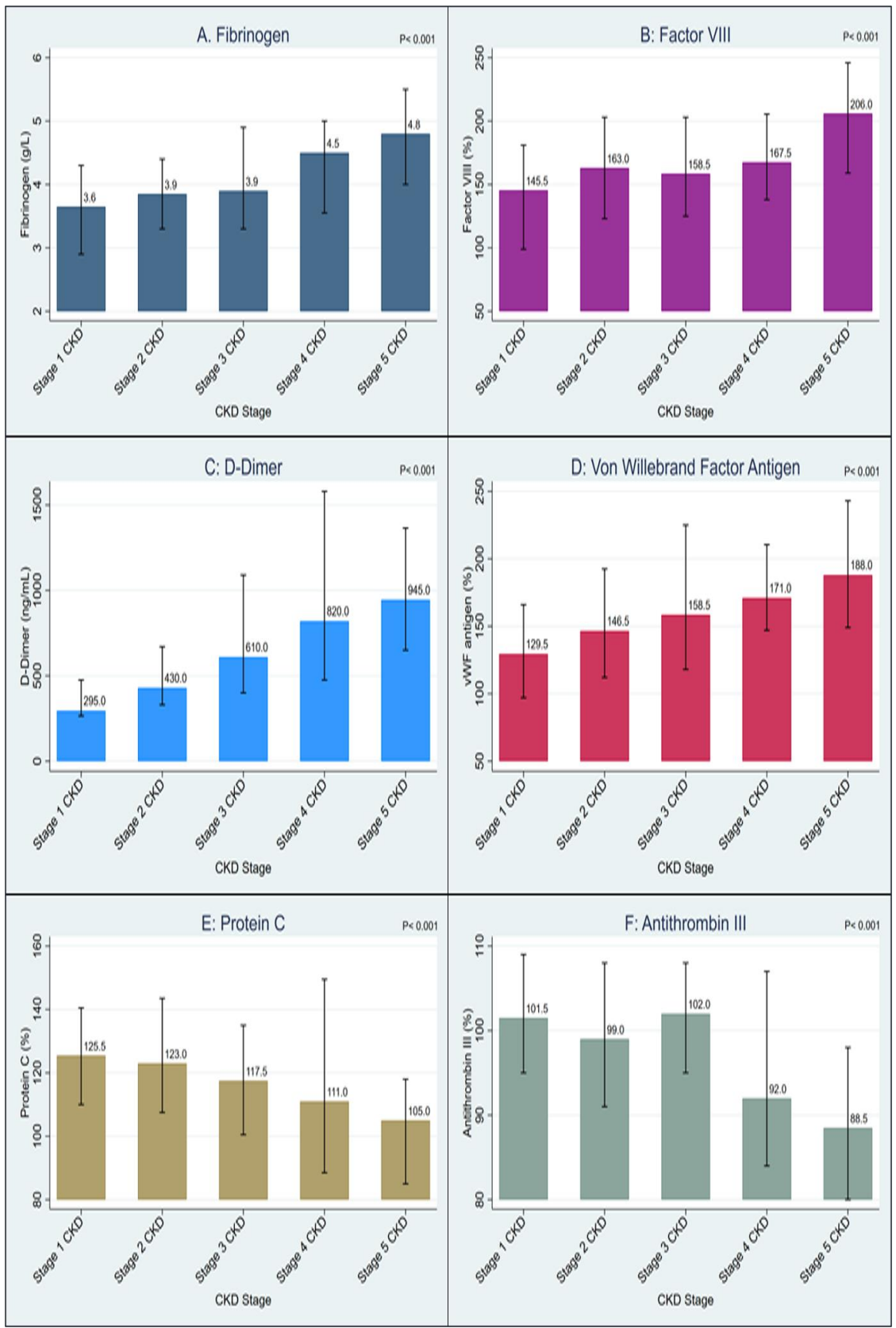


Figure 2: Alteration of haemostatic biomarkers across CKD stages
All bar graphs display median values with interquartile ranges.

A-B: Fibrinogen and Factor VIII remain stable from stages 1-3 and increase from stage 4.

C-D: D-Dimer and von Willebrand factor antigen progressively increase from stage 2.

E-F: Protein C and antithrombin remain stable from stages 1-3, begin to decrease in stage 4 through to stage 5.

Not included in the figure: PT, APTT, Protein S and CRP remained unchanged and within the healthy reference range across stages.

Global Coagulation Assays

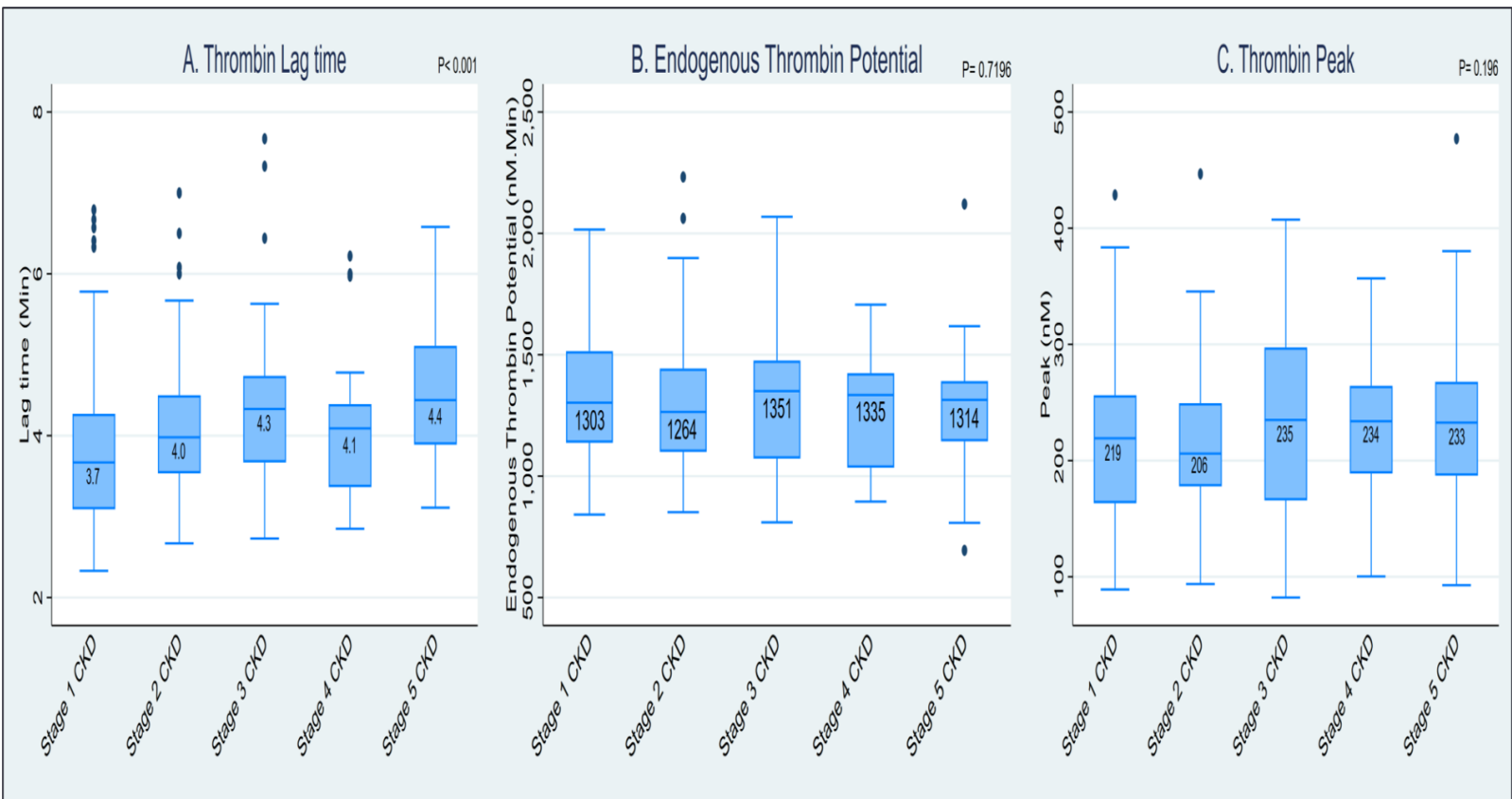


Figure 3: Thrombin Generation Across CKD Stages

All box plots display median values, interquartile ranges, outliers, and P value from the Kruskal-Wallis test.

A: Increase thrombin lag time in stage 3 and stage 5 as compared to stage 1.

B-C: No statistically significant difference in ETP and thrombin peak across stages.

Not included in figure - Time to peak and Velocity index remained unchanged across stages.

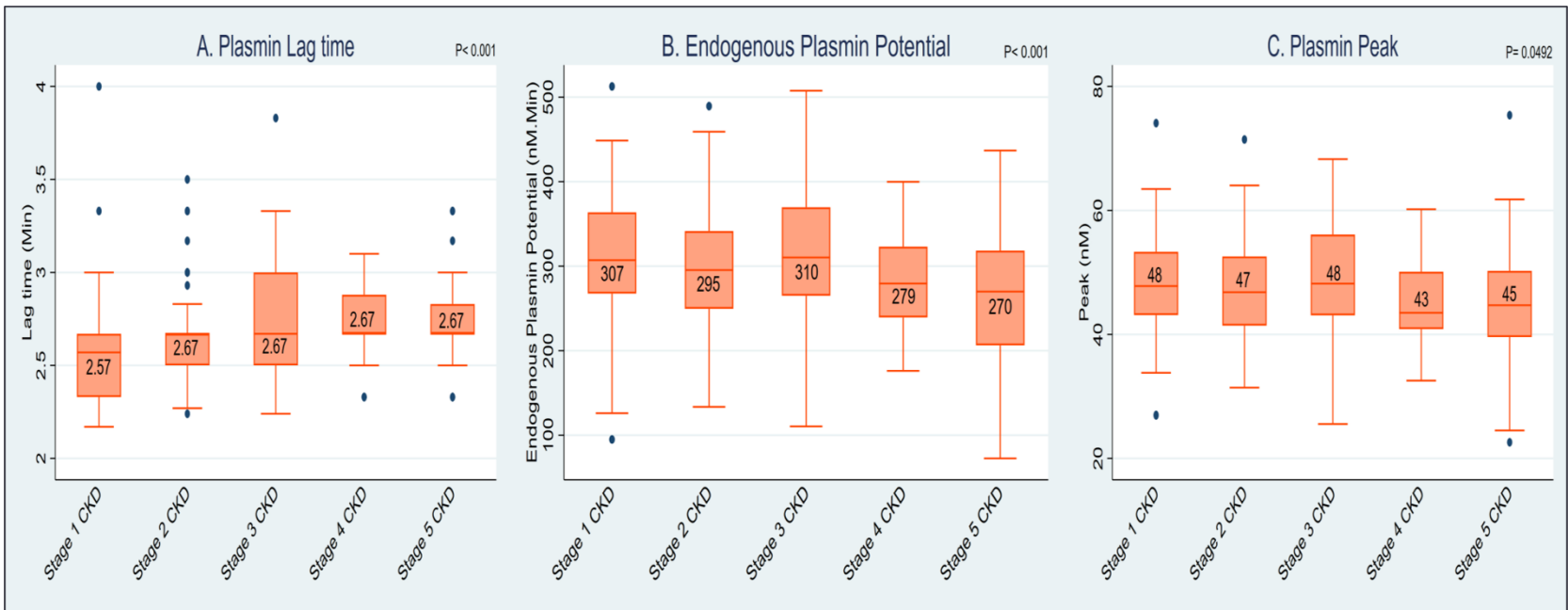


Figure 5: Plasmin Generation Across CKD Stages

All box plots display median values, interquartile ranges, outliers, and P value from the Kruskal-Wallis test.

A: Plasmin lag time remains unchanged from stages 1-2 and increases from stage 3.

B: Endogenous plasmin potential remains unchanged from stages 1-3 and begins to decrease from stage 4, with a statistically significant difference in stage 5.

C: Small decrease in plasmin peak from stage 4.

Not included in the figure - No stage-dependent changes in plasmin time to peak and velocity index across stages

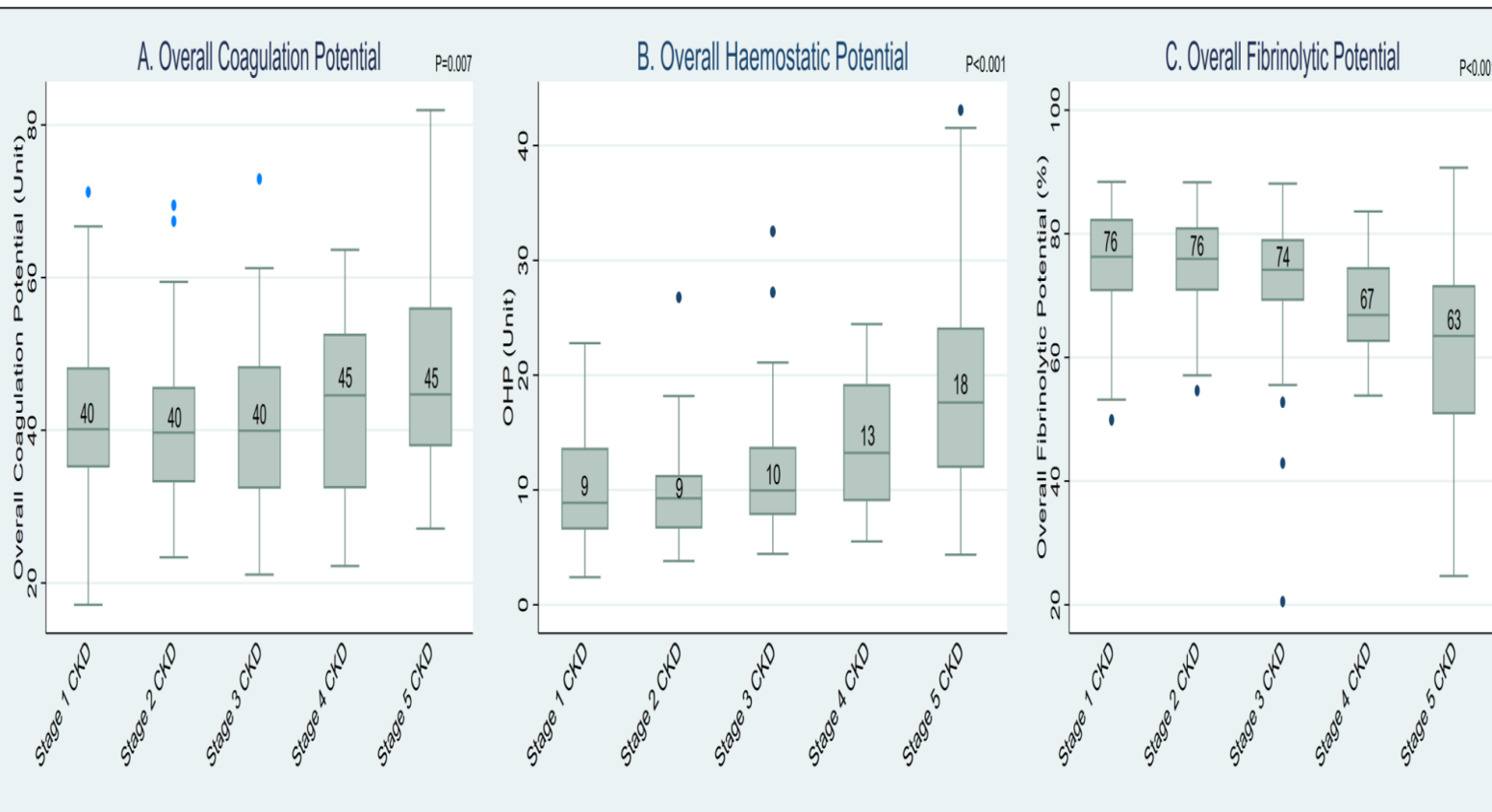


Figure 4: Modified Overall Haemostatic Potential Across CKD Stages.

All box plots display median values, interquartile ranges, outliers, and P value from the Kruskal-Wallis test.

A: OCP remains unchanged from stages 1-3 and begins to increase from stage 4, with statistical significance in stage 5 only.

B-C: OHP and OFP remain unchanged from stages 1-3. From stage 4 onward, OHP increases while OFP decreases.

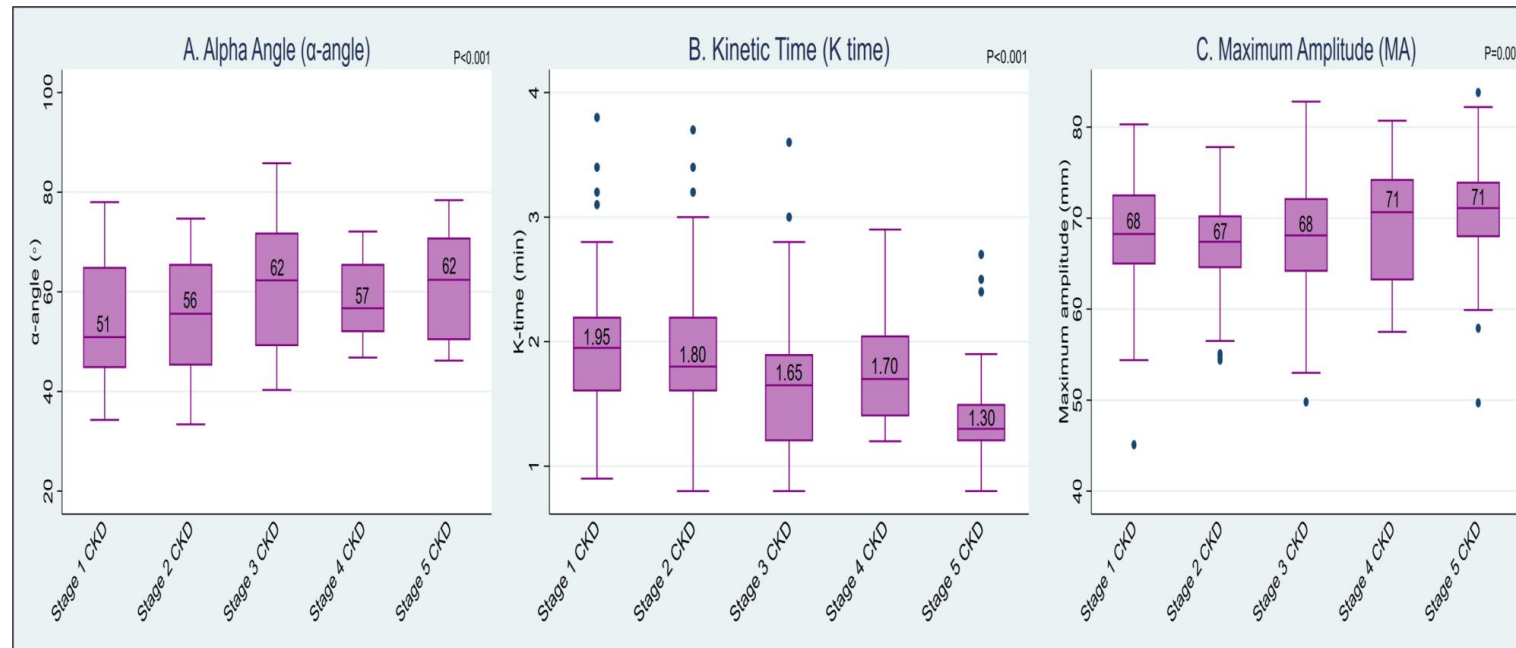


Figure 6: Thromboelastography Across CKD Stages

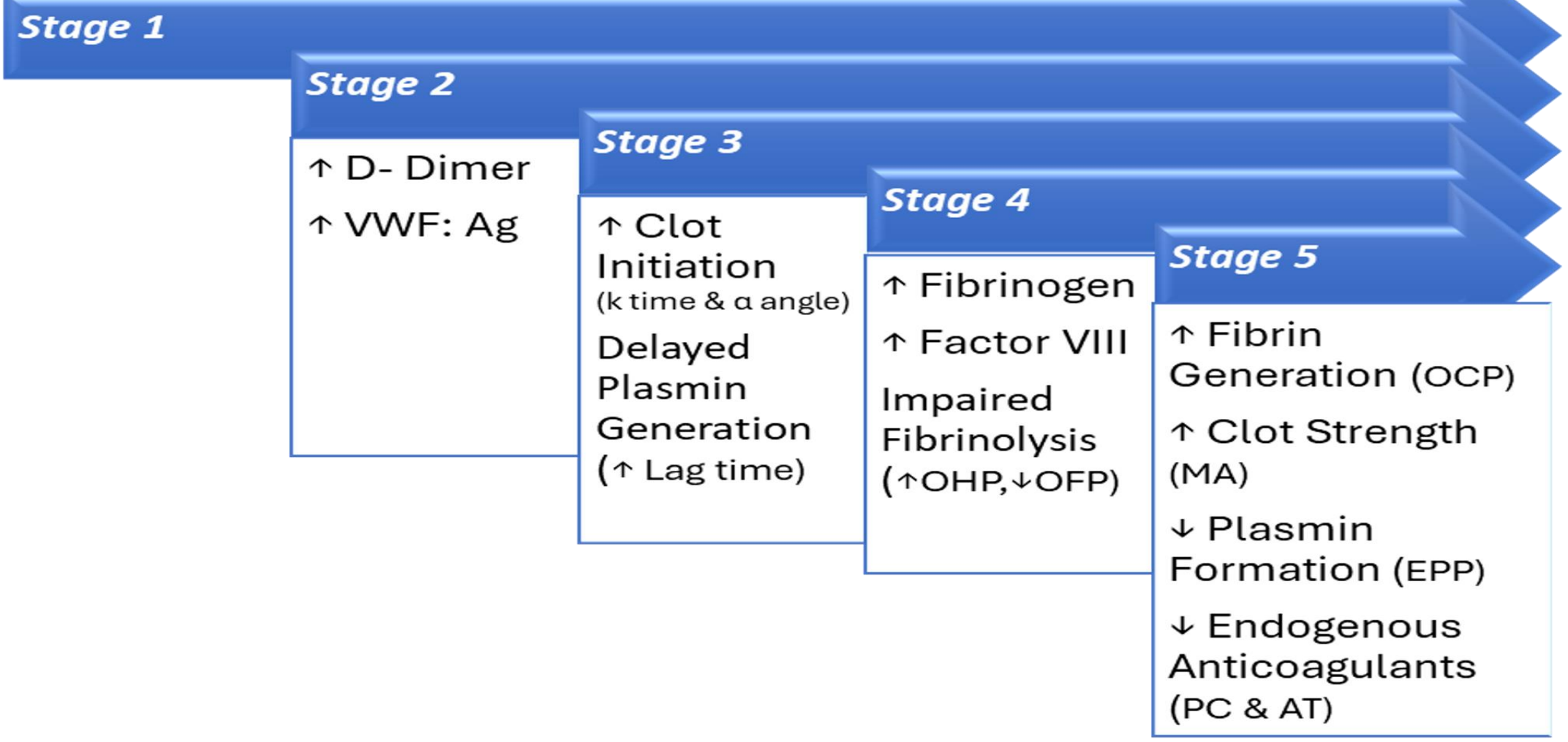
All box plots display median values, interquartile ranges, outliers, and P value from the Kruskal-Wallis test.

A-B: Increase in alpha angle and decrease in kinetic time from stage 3 onward.

C: Maximum amplitude begins to increase in stage 4.

Not included in the figure - No statistically significant changes in reaction time and lysis at 30 minutes across stages

SUMMARY



CONCLUSIONS

CKD progression is characterised by increasing hypercoagulability and fibrinolytic impairment at advanced stages. GCA, especially assays assessing both fibrin formation and lysis, demonstrated stage-dependent changes and may provide prognostic value for thrombotic and bleeding risk across CKD stages.

CONTACT INFORMATION

Vincent Lu, Email: vincent.lu2@nh.org.au