# POLIMORPHONUCLEARS AS A MARKER OF INFLAMMATION IN PATIENTS WITH CHRONIC KIDNEY DISEASE



BARRETO-SILVA, MARIA INES<sup>1</sup>; CARLA LEMOS<sup>3</sup>; SIMONE VARGAS<sup>2</sup>; THEREZA CHRISTINA BARJA-FIDALGO<sup>2</sup> AND BREGMAN, R<sup>3</sup>

NUTRITION INSTITUTE 1; PHARMACOLOGY 2; NEPHROLOGY 3 STATE UNIVERSITY OF RIO DE JANEIRO, BRAZIL



## INTRODUCTION

- Chronic kidney disease (CKD) is associated immune activation systemic and inflammation that seems to occur early in the course of the disease.
- Polymorphonuclears (PMNs) play a critical role in chronic inflammation and migration is a marker of their function. The best marker for inflammation in CKD is not defined.

## **OBJECTIVE**

To evaluate PMNs as inflammatory markers in CKD patients

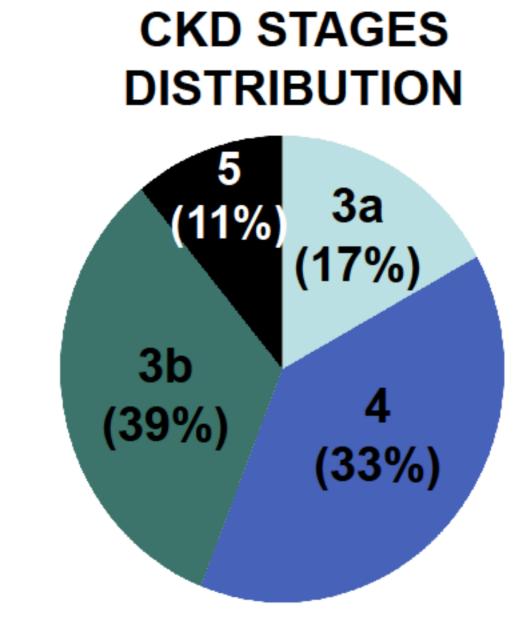
### **METHODS**

- Patients: non dialysed CKD patients under regular treatment with interdisciplinary team, for at least 1 year, at the Hospital of the Rio de Janeiro State University, Brazil
- \* estimated glomerular filtration rate (eGFR): CKD-EPI equation
- Polimorphonuclears-PMN evaluation:
  - ability to migrate:
    - formyl-methionyl-leucylphenylalanine (fMLP, 100 nM)
    - II. toward medium (random migration)
  - PMN basal ROS production
  - experimental conditions: 37 C; 5%CO<sub>2</sub>; for 1 hour
- Statistical analysis:
- PMNs migration and ROS production according to CKD stages-by ANOVA and T- test.

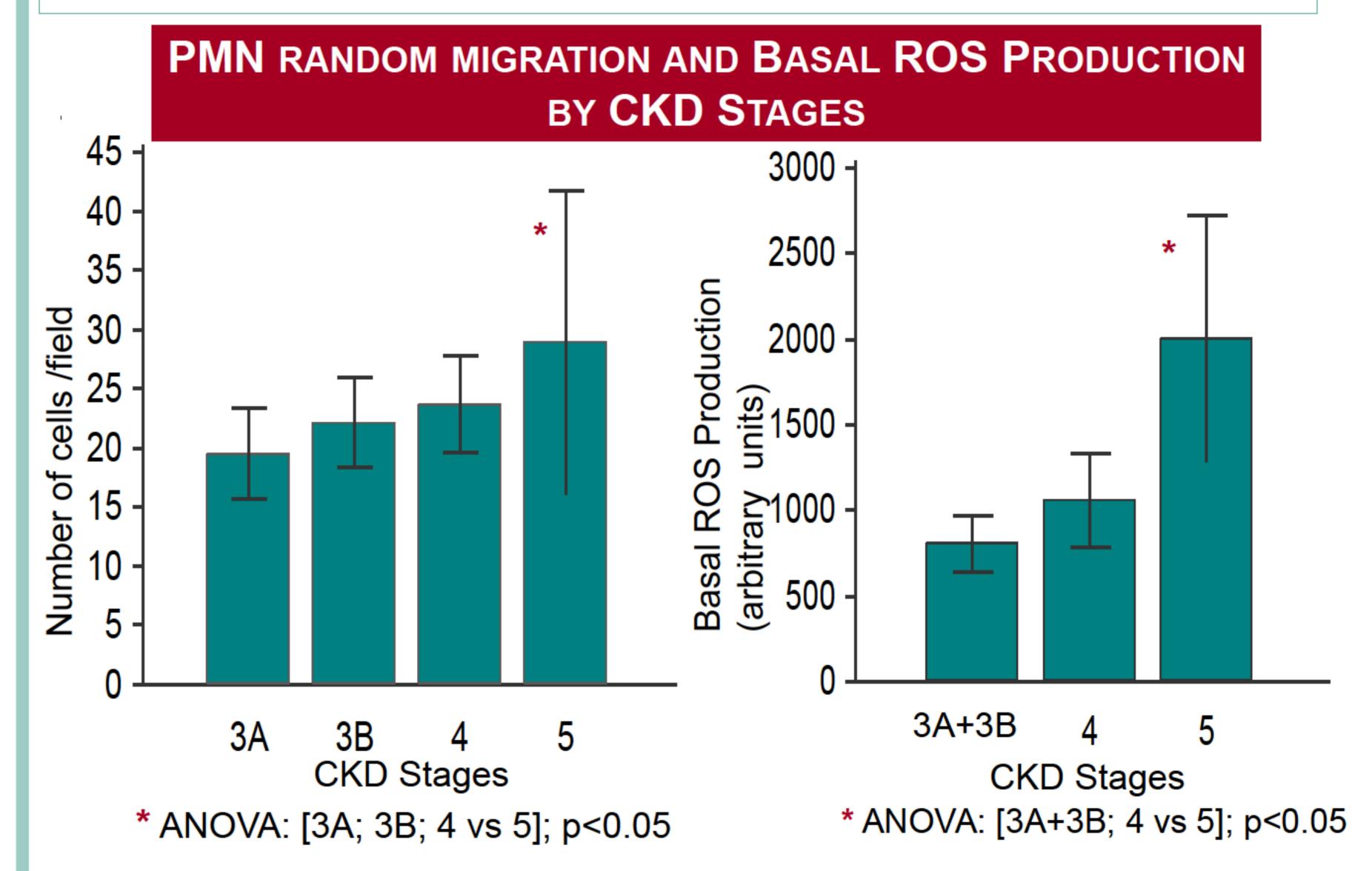
## RESULTS

PATIENTS: • n= 54 {53% men} • age: 66.3±12.2 years (32 - 87)

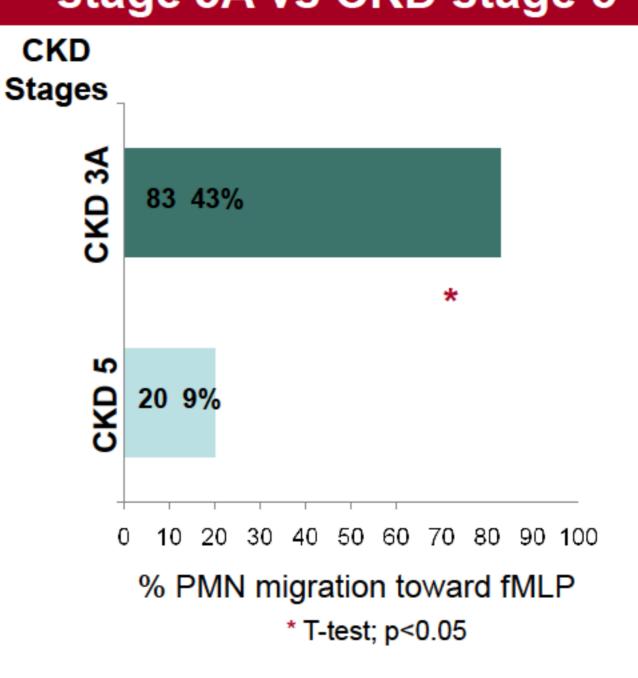
• treatment period: 4.9± 3.2 years



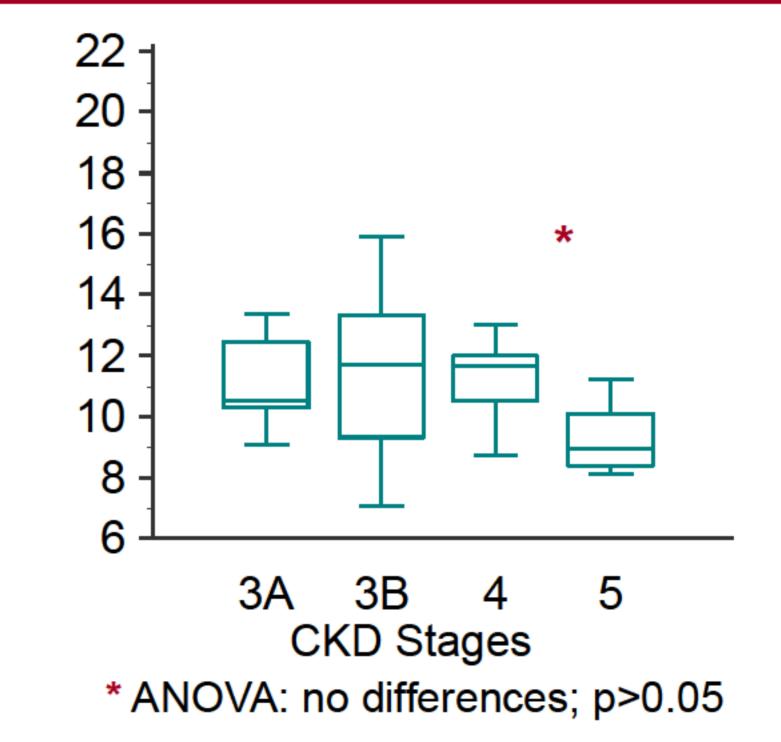
Laboratorial Parameters	Mean±standard deviation
eGFR (mL/min.)	<b>32</b> ±13 (min. 8.5-max.: 50)
Urea (mg/dL)	<b>87</b> ±32
Uric acid (mg/dL)	<b>7.9</b> ±1.9
Glucose (mg/dL)	<b>121</b> ±82
Total cholesterol (mg/dL)	<b>189</b> ±48
Hemoglobin (g/dL)	<b>12.3</b> ±1.4
Ferritin (mg/L)	<b>142</b> ±114
Albumin (g/dL)	<b>4.5</b> ±0.1
Ca (mg/dL)	<b>9.5</b> ±0.4
PO4 (mg/dL)	<b>3.8</b> ±0.7







#### IL8 LEVELS ACCORDING TO CKD STAGES



## CONCLUSIONS

Data suggest that eGFR is associated with the desensitization of circulating PMNs to fMLP, which may contribute to CKD- associated inflammation. Interleukin was not able to identify these alterations.



