

# The role of PVAT in vascular ageing in chronic kidney disease and type 2 diabetes.

No registrations found.

<b>Ethical review</b>	Positive opinion
<b>Status</b>	Recruiting
<b>Health condition type</b>	-
<b>Study type</b>	Observational non invasive

## Summary

### ID

NL-OMON23667

### Source

NTR

### Brief title

Vascular Ageing Study

### Health condition

Accelerated vascular ageing  
Chronic kidney disease  
Type 2 diabetes

## Sponsors and support

**Primary sponsor:** University Medical Center Groningen

**Source(s) of monetary or material Support:** Astellas

## Intervention

## Outcome measures

### Primary outcome

- Characterization of the pro-inflammatory and pro-calcifying environment of PVAT as

compared to subcutaneous fat (SAT)

- Identification of potential differences in inflammatory profile between PVAT obtained from 'healthy' and calcified arterial wall
- Assessment of the effects on SMC calcification, dedifferentiation and contractile function in vitro of PVAT (compared to SAT)

### **Secondary outcome**

- Role of type 2 diabetes mellitus in PVAT dysfunction
- Differences between end-stage renal disease and pre-emptive renal transplant recipients in its role of PVAT dysfunction

## **Study description**

### **Background summary**

Chronic kidney disease (CKD) is associated with a strong increase in cardiovascular risk, which is a consequence of accelerated vascular ageing. This process is hallmarked by vascular remodeling, chronic low-grade inflammation, calcification, and increased vascular stiffness. Vascular ageing is more pronounced in CKD patients who are also suffering from diabetes. The majority of type 2 diabetes (T2D) patients are obese with visceral adipose tissue (VAT) playing a central role in causing insulin resistance and metabolic syndrome. VAT is distributed through the abdominal cavity and is present surrounding the abdominal organs and the vasculature, the latter also called perivascular adipose tissue (PVAT). PVAT may be protective at some sites but it may also promote vascular ageing at other vascular sites because of its pro-atherogenic effects. This deranged function of PVAT may serve as a link between accelerated vascular ageing in CKD and T2D. I hypothesize that CKD and/or T2D derange PVAT function results in aggravated vascular ageing including development of atherosclerosis and calcification. In the current proposal, I will assess the pro-atherogenic environment of PVAT in patients with CKD with or without T2D.

In this study we will assess the role of T2D and CKD (end stage renal disease and pre-emptive) in PVAT dysfunction.

### **Study objective**

PVAT plays an important role in driving vascular ageing manifested by medial smooth muscle cell (SMC) dedifferentiation into an osteogenic phenotype that induces intimal and/or medial calcification. I furthermore hypothesize that this process of vascular inflammation and calcification is most severe in patients with both chronic kidney disease and Type 2 diabetes (diabetic nephropathy).

## Study design

T0: informed consent

T1: venapuncture 1 day before transplantation

## Intervention

Not applicable.

## Contacts

**Public**

**Scientific**

## Eligibility criteria

### Inclusion criteria

Kidney donors:

- Men and women
- Age above 17 years

Kidney recipients:

- Men and women
- Age above 17 years
- Kidney failure leading to transplantation

### Exclusion criteria

- Inadequate speaking of Dutch language
- Age below 18 years
- Incompetent

## Study design

### Design

Study type:	Observational non invasive
Intervention model:	Parallel
Allocation:	Non controlled trial
Masking:	Single blinded (masking used)
Control:	N/A , unknown

### Recruitment

NL	
Recruitment status:	Recruiting
Start date (anticipated):	01-01-2018
Enrollment:	0
Type:	Anticipated

## Ethics review

Positive opinion	
Date:	01-10-2018
Application type:	First submission

## Study registrations

### Followed up by the following (possibly more current) registration

No registrations found.

## Other (possibly less up-to-date) registrations in this register

No registrations found.

## In other registers

Register	ID
NTR-new	NL7442
NTR-old	NTR7684
Other	Research register UMCG : 201500869

## Study results

### Summary results

None yet.