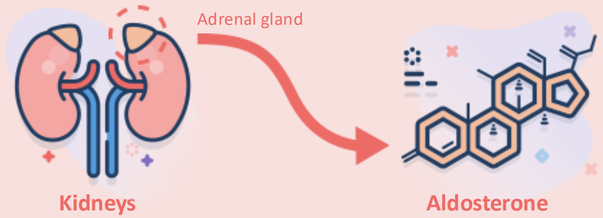
Aldosterone exhibits effects on the blood vessels, heart and kidneys through multiple pathways

Where it begins: aldosterone production[1]



Aldosterone is a **mineralocorticoid** primarily produced by **aldosterone synthase** (CYP11B2) in the **zona glomerulosa cells** in the **adrenal cortex**[1]

Aldosterone is a key regulator of BP[1]

- Aldo
- Na^+ reabsorption
- Plasma volume
- BP

Aldosterone regulation in normal physiology[2,3]

Elevations in BP and sodium lead to:

- **Inhibition of renin release**
- **Decreased RAAS activation**
- **Reduced aldosterone production**

Aldosterone remains high in abnormal physiology[3]

Despite suppression of the RAAS, aldosterone secretion continues, resulting in plasma volume expansion and increased BP[3]

Aldosterone dysregulation is abnormal aldosterone production despite excess sodium and fluid in the body[4–6]

MRAs

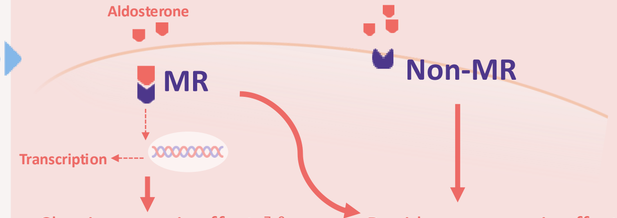
(spironolactone, eplerenone)[18]

ACEi/ARBs

indirectly reduce aldosterone by inhibiting the effects of angiotensin II, but aldosterone levels may remain high in some clinical scenarios[19]

The **non-MR** effects of aldosterone persist, continuing to drive **CV** and **kidney damage** via MR-independent effects[18,19]

Preclinical model of dual aldosterone signalling pathways



Classic genomic effects[7–9]

- Aldosterone binds to intracellular MRs
- The aldosterone–MR complex enters the nucleus to regulate gene transcription
- Lead to protein synthesis involved in electrolyte and fluid balance
- Delayed onset: effects occur after hours

Rapid non-genomic effects[8,10]

- Act independently of gene transcription
- Involve membrane-bound MRs and other receptors (e.g. GPER)
- Regulate electrolyte channels
- Trigger rapid cellular responses (seconds to minutes)

Did you know?

Pathological aldosterone activity or levels can drive HTN and organ damage[3,4,10–17]

Vasculature

Excess aldosterone drives **vascular remodelling** and leads to **CV damage**[8,9,11,15]

Heart

Excess aldosterone can impact **LV cardiac remodelling** and **cardiac function**, leading to CV morbidity[11–13,15,16]

Kidneys

Excess aldosterone may induce **fibrotic** and **inflammatory** changes in the kidneys, leading to damage[9,14,17]

View footnotes | View references | View abbreviations

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Think Aldo

Aldosterone exhibits effects on the blood vessels, heart and kidneys through multiple pathways

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Did you know?

Mineralocorticoid receptors are only involved in ~50% of aldosterone activities[20,a]

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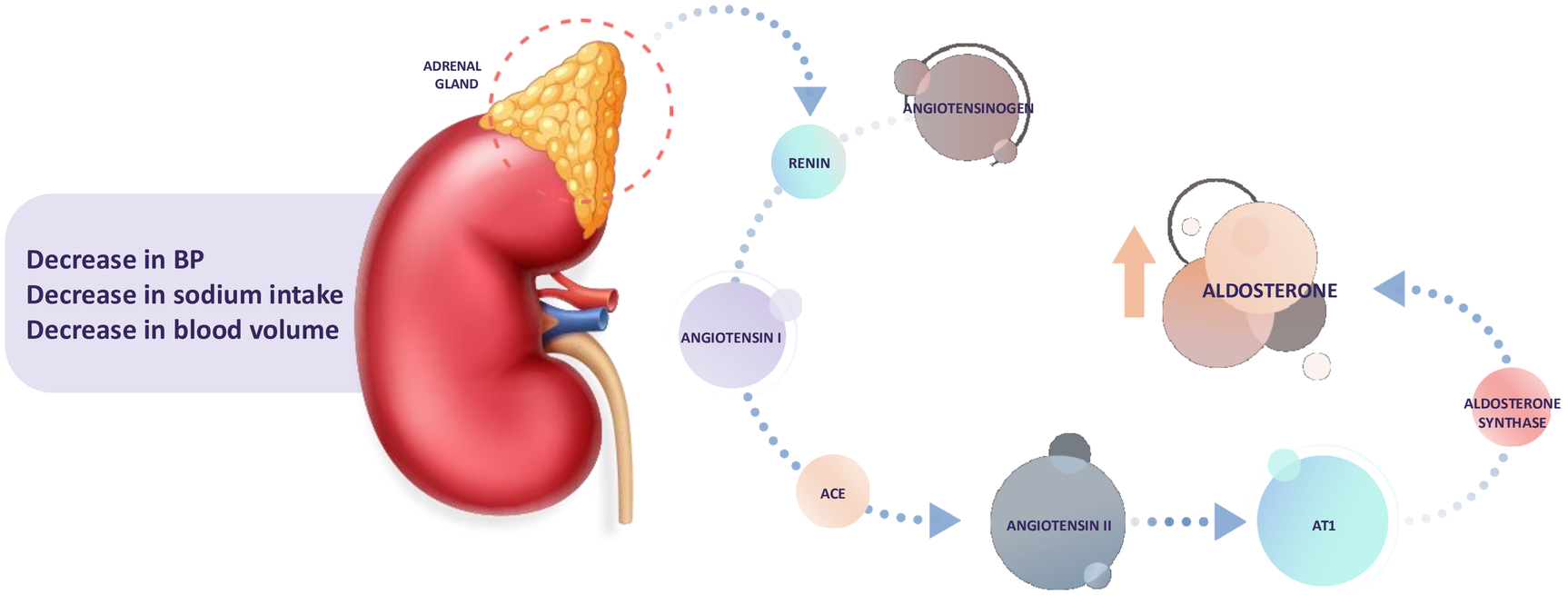
Aldosterone synthesis[1]



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Where it begins: aldosterone production[1]

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Aldosterone is a **mineral**
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Aldosterone regulation in normal physiology[2,3]

Reduced aldosterone production

nal physiology[3]

etion continues,
creased BP[3]

production despite
4–6

y or levels
ge[3,4,10–17]

Kidneys

Excess aldosterone may induce **fibrotic** and **inflammatory** changes in the kidneys, leading to damage[9,14,17]

indirectly reduce aldos
but aldosterone leve

The **non-MR** effec
CV and **kidney**

after hours

Did you know?

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Aldosterone is a key regulator of BP[1]

after hours

Did you know?

Aldosterone regulation in normal physiology[2,3]

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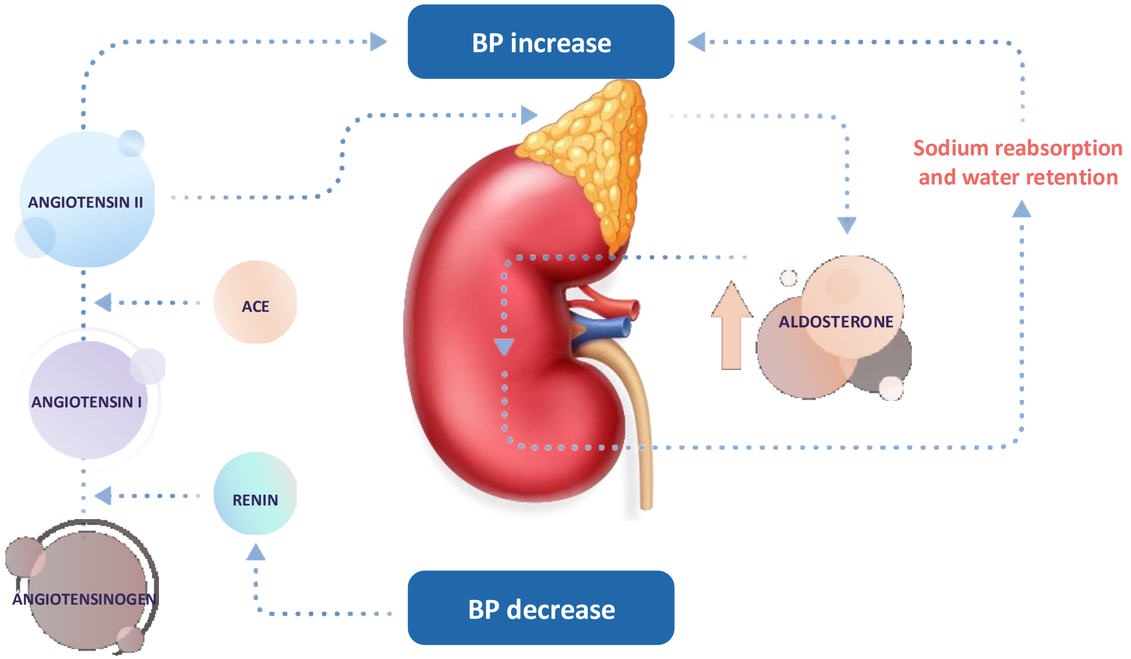
View footnotes | View references | View abbreviations

Aldosterone regulation in normal physiology

X

1. Activation of RAAS in response to decreased BP[2]
2. Increased aldosterone production causes water and sodium reabsorption, leading to plasma volume expansion and increased BP[2,3,21]
3. Elevations in BP and sodium lead to:[2,3]
   - Inhibition of renin release
   - Decreased RAAS activation
   - Reduced aldosterone production



In normal physiology, aldosterone production is increased by low sodium concentrations, low BP and low renal perfusion[2]

View abnormal physiology

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Where it begins: aldosterone production[1]

Kidneys

Aldosterone is a **mineral**
(CYP11B2) in the

Aldosterone is a key regulator of BP[1]

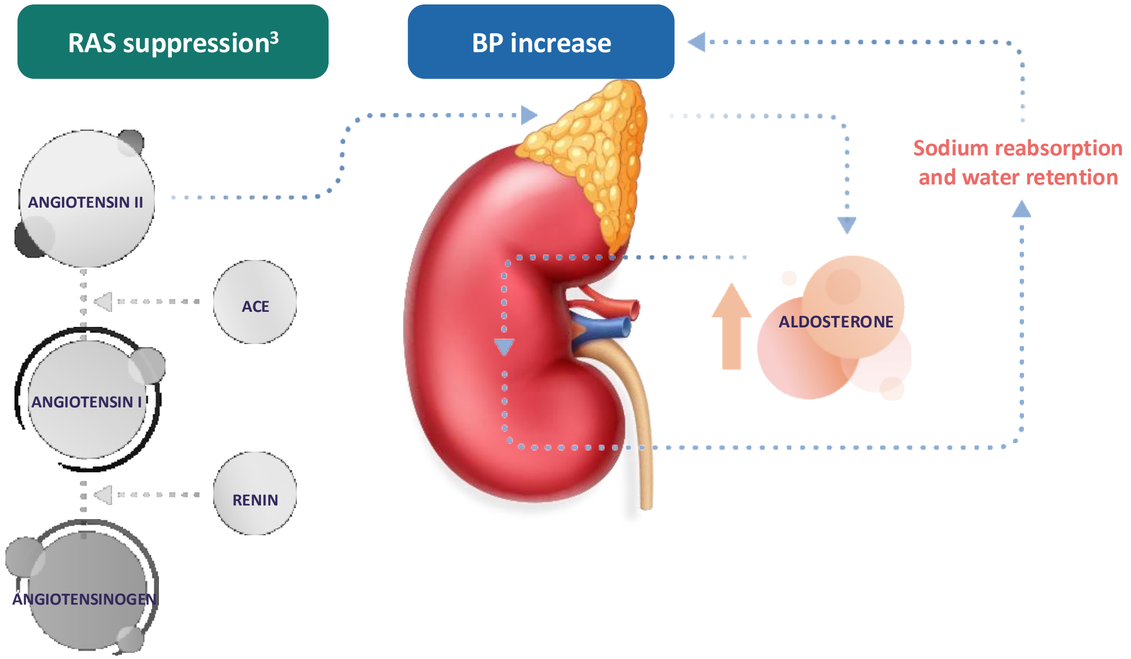
Aldosterone regulation in normal physiology[2,3]

Reduced aldosterone production

Aldosterone dysregulation in abnormal physiology

X

1 **Increased aldosterone production causes water and sodium reabsorption, leading to plasma volume expansion and increased BP[2,3,21]**



In abnormal physiology, aldosterone secretion often continues, despite suppression of the RAAS. This results in plasma volume expansion and increased BP.[3] Aldosterone dysregulation is abnormal aldosterone production despite excess sodium and fluid in the body.[4–6]

View normal physiology

indirectly reduce aldos
but aldosterone leve

The **non-MR** effec
CV and **kidney**

after hours

Did you know?

Kidneys

Excess aldosterone may induce **fibrotic** and **inflammatory** changes in the kidneys, leading to damage[9,14,17]

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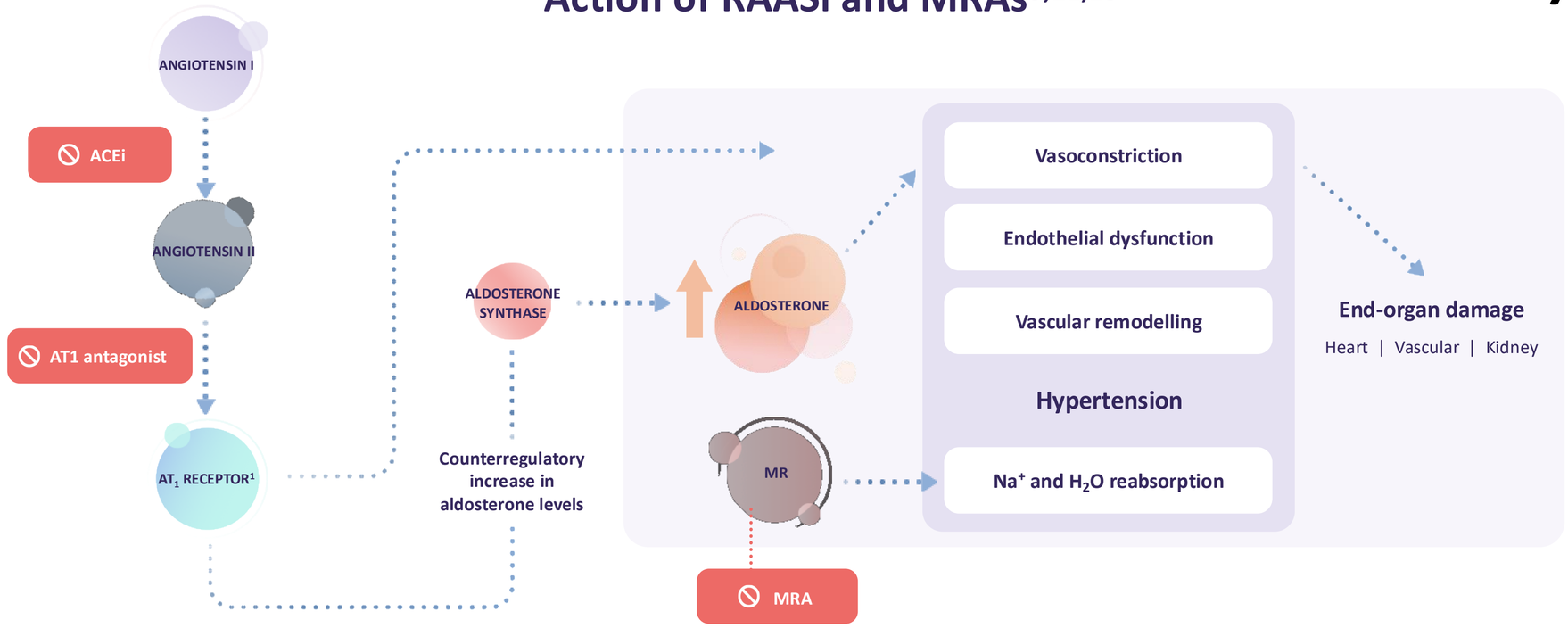
Action of RAASi and MRAs[9,22,23]



MRAs block the MR but do not reduce aldosterone production, meaning some downstream pathways are left undisrupted.[23,24]
ACEi and ARBs may lower aldosterone initially, but aldosterone levels remain elevated in some patients, allowing CV and kidney damage to persist.[19]

Where it begins: aldosterone production[1]

Kidneys

Aldosterone is a **mineral**
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The **non-MR** effec
CV and **kidney**

Aldosterone is a key regulator of BP[1]

after hours

Did you know?

Aldosterone regulation in normal physiology[2,3]

Reduced aldosterone production

al physiology[3]

etion continues,
creased BP[3]

production despite
4–6

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Kidneys

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Think Aldo

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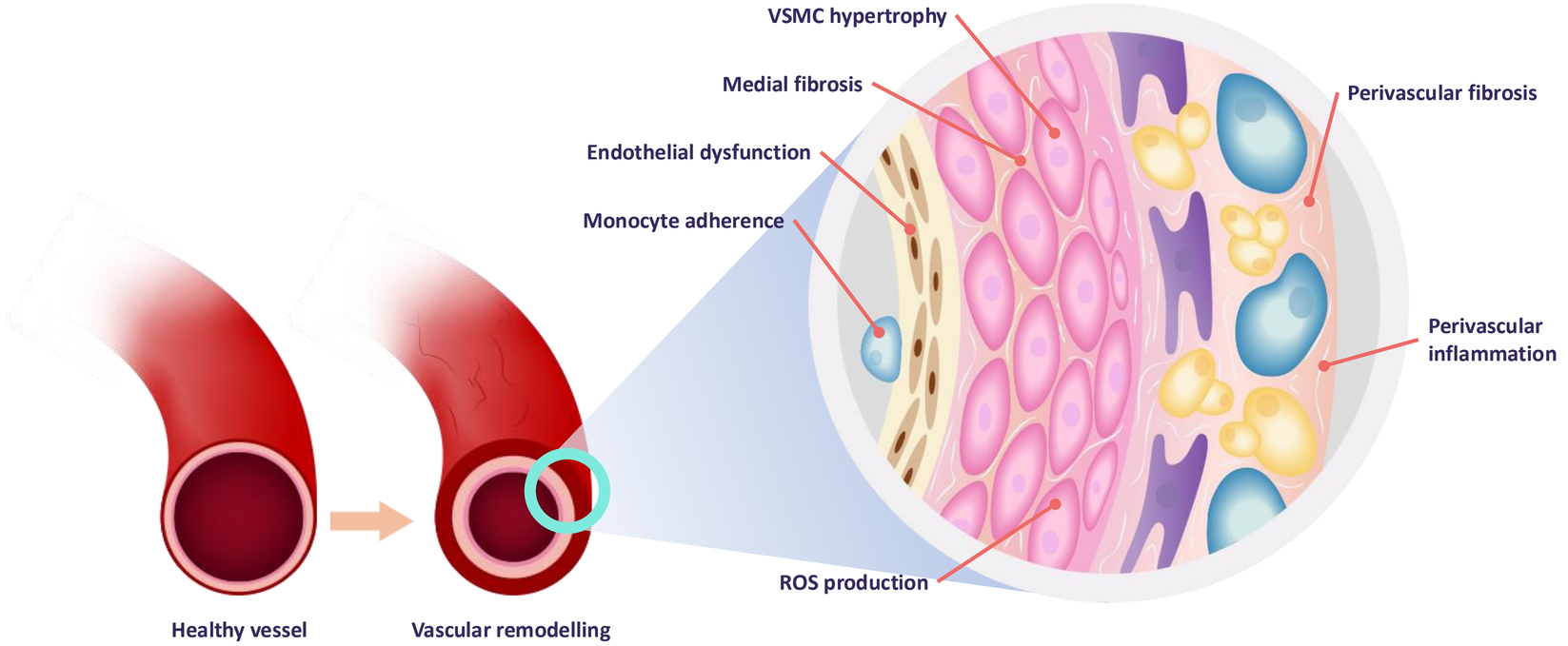
Key effects of aldosterone on the blood vessels[8,9,11,22,25–27]



Excess aldosterone drives vascular remodelling and leads to CV damage[8,9,11]

Where it begins: aldosterone production[1]

Kidneys

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Aldosterone is a key regulator of BP[1]

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after hours

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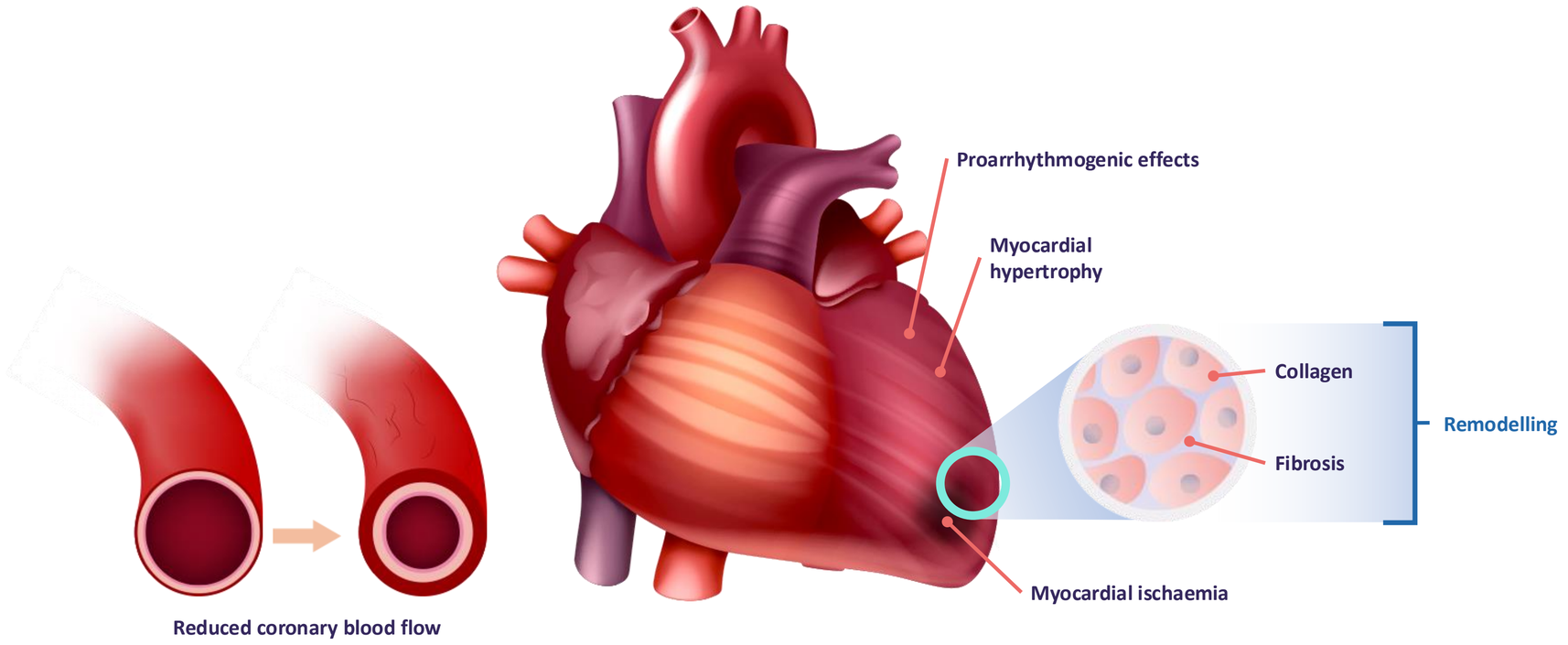
Key effects of aldosterone on the heart[8,9,11–13]



Excess aldosterone can impact LV cardiac remodelling and cardiac function, leading to CV morbidity[11–13]

Where it begins: aldosterone production[1]

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Aldosterone regulation in normal physiology[2,3]

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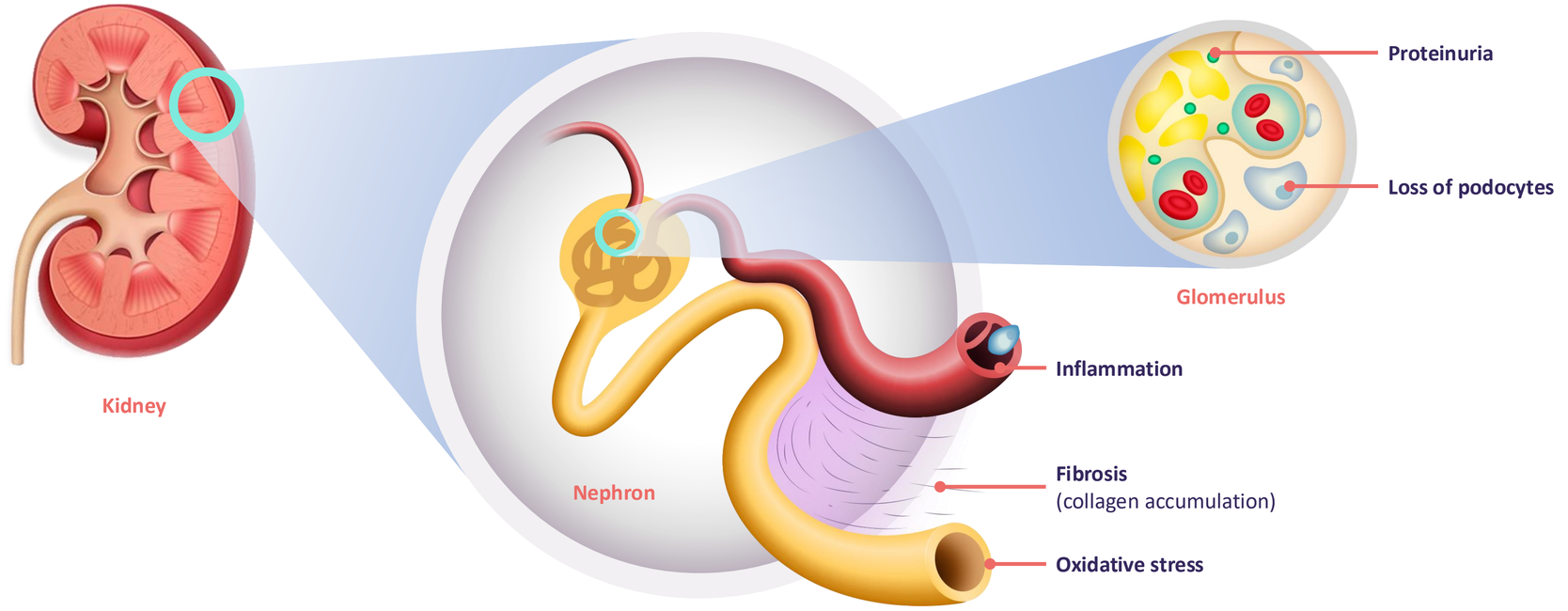

Excess aldosterone may induce fibrotic and inflammatory changes in the kidneys, leading to damage[9,14]

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indirectly reduce aldos
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The **non-MR** effec
CV and **kidney**

after hours

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Footnotes

[a]Percentage based only on studies involving spironolactone[20]

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Abbreviations

ACE, angiotensin-converting enzyme
ACEi, angiotensin-converting enzyme inhibitor
ARB, angiotensin II receptor blocker
AT1, angiotensin II type 1 receptor
BP, blood pressure
CV, cardiovascular
GPER, G protein–coupled oestrogen receptor
H_2O, water
HTN, hypertension
LV, left ventricle / left ventricular
MR, mineralocorticoid receptor
MRA, mineralocorticoid receptor antagonist
Na^+, sodium
RAAS, renin–angiotensin–aldosterone system
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ROS, reactive oxygen species
VSMC, vascular smooth muscle cell

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