

# Cardioprotective effect of thyroid hormone is mediated by AT<sub>2</sub> receptor and involves nitric oxide production via Akt activation in mice

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## Abstract

Studies have demonstrated that thyroid hormone (T<sub>3</sub>) can precondition the heart against ischaemic injury and improve post-ischaemic recovery. This study investigated whether the AT<sub>2</sub> receptor (AT<sub>2</sub>R) is involved in cardioprotection and the potential molecular mechanism responsible for this effect. Hyperthyroidism was induced in male wild-type (WT) and AT<sub>2</sub>R knockout (KO) mice by administering daily intraperitoneal injections of T<sub>3</sub> (7 µg/100 g body weight) for 14 days. The mouse hearts were harvested and perfused with a Krebs–Henseleit solution at a constant flow in a Langendorff set-up. After 30 min of stabilization, the hearts were subjected to global ischaemia for 20 min and reperfused for 45 min. Baseline cardiac function was assessed by measuring four parameters: LVDP (mmHg), heart rate (bpm), +dP/dt and –dP/dt (mmHg/s). After reperfusion, the total protein from cardiac ventricles was obtained, and the Akt signalling pathway and NO production were evaluated. Post-ischaemic functional recovery was significantly greater ( $p < 0.05$ ) in the T<sub>3</sub>-treated WT mice compared to the control, demonstrating the cardioprotective effect of T<sub>3</sub>. This effect was abolished in T<sub>3</sub>-treated KO mice, demonstrating the physiological relevance of AT<sub>2</sub>R to the cardioprotective phenotype induced by T<sub>3</sub>. Akt activation, iNOS expression and NO production increased in cardiac tissue after T<sub>3</sub> treatment in the WT animals, but no difference was observed after treatment in the KO mice. This study indicates that AT<sub>2</sub>R acts as a cardioprotector in the case of hyperthyroidism. Strategies targeting AT<sub>2</sub>R agonists might improve cardiac function through NO production and suggest potential therapeutic targets for heart diseases.

## Keywords

Angiotensin II   Cardiac hypertrophy   Cardioprotection   Isolated heart perfusion  
Thyroid hormone

## Abbreviations

AMI

Acute myocardial infarction

Ang II

Angiotensin II

AT1

Angiotensin II type 1 receptor

AT2R

Angiotensin II type 2 receptor

AT2R-KO

AT2R knockout mice

C57BL/6

C57black6

+  $dP/dt$

First derivative of the positive ventricular pressure

-  $dP/dt$

First derivative of the negative ventricular pressure

HR

Heart rate

I/R

Ischaemia/reperfusion

IU

International unit

KO

Knockout

KO-C

Control knockout mice

KO-T<sub>3</sub>

T<sub>3</sub>-treated knockout mice

LV

Left ventricle

LVDP

Left ventricular developed pressure

MI

Myocardial infarction

NO

Nitric oxide

RAS

Renin–angiotensin system

T<sub>3</sub>

Triiodothyronine

T<sub>4</sub>

Thyroxine

TH

Thyroid hormones

TTBS

Tween 20 tris-buffered saline

WT

Wild-type

WT-C

Wild-type control mice

WT-T<sub>3</sub>

WT T<sub>3</sub>-treated mice

WT-T<sub>3</sub>

WT T<sub>3</sub>-treated mice

## Notes

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## Compliance with ethical standards

## Conflict of interest

The authors declare that there are no competing or financial interests associated with the manuscript.

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