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# Mitochondrial-targeted antioxidant MitoQ ameliorated metabolic syndrome features much better than Apocynin and Allopurinol in obesogenic diet-fed rats

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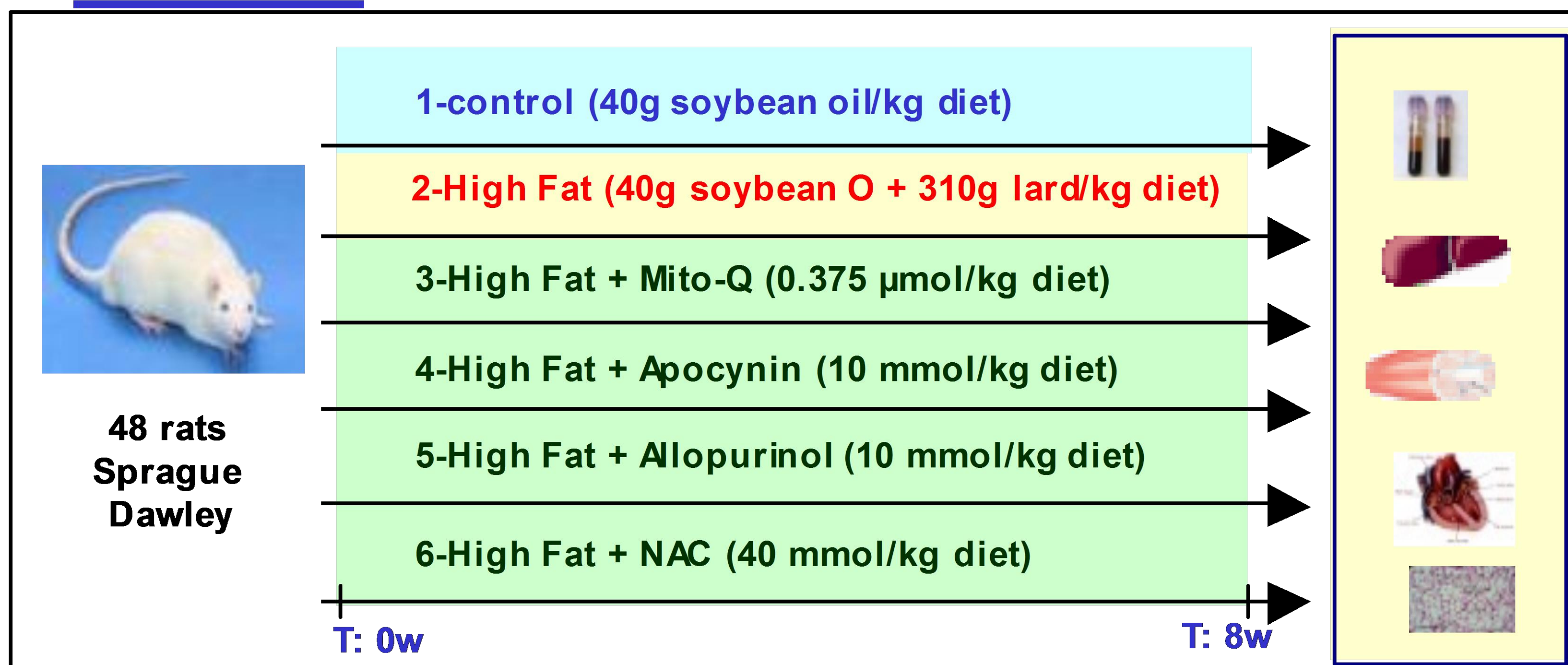
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## Introduction

The prevalence of metabolic syndrome is increasing rapidly in both developing countries and wealthy societies. This is mainly due to over-nutrition and sedentary lifestyle. It is admitted that oxidative stress is involved in the initiation or/and the evolution of this syndrome.

The present work aimed to explore the individual contributions of major cellular ROS production systems, mitochondria, NADPH oxidase and xanthine oxidase, to this oxidative stress.

## Protocols

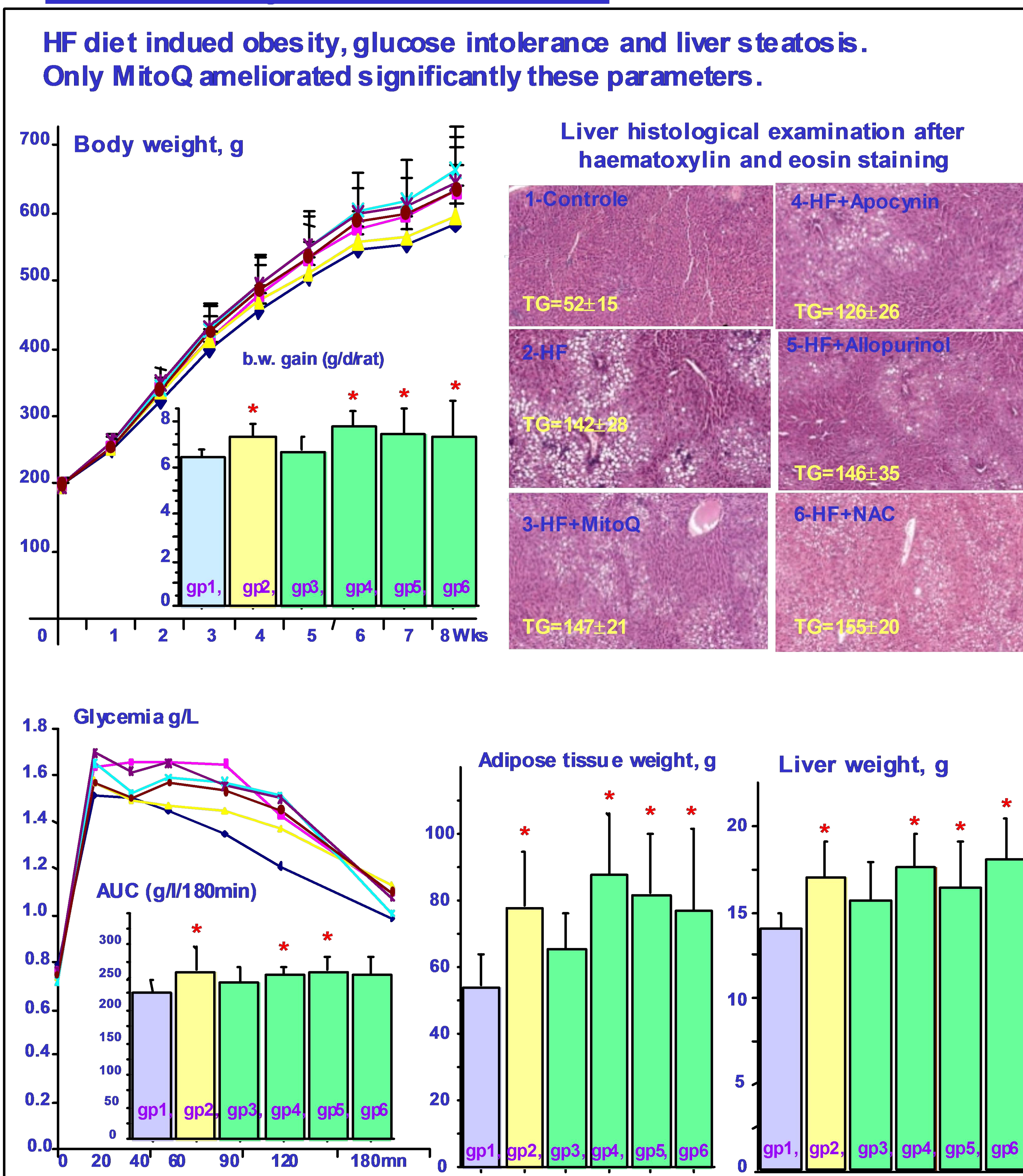


## Oxidative stress

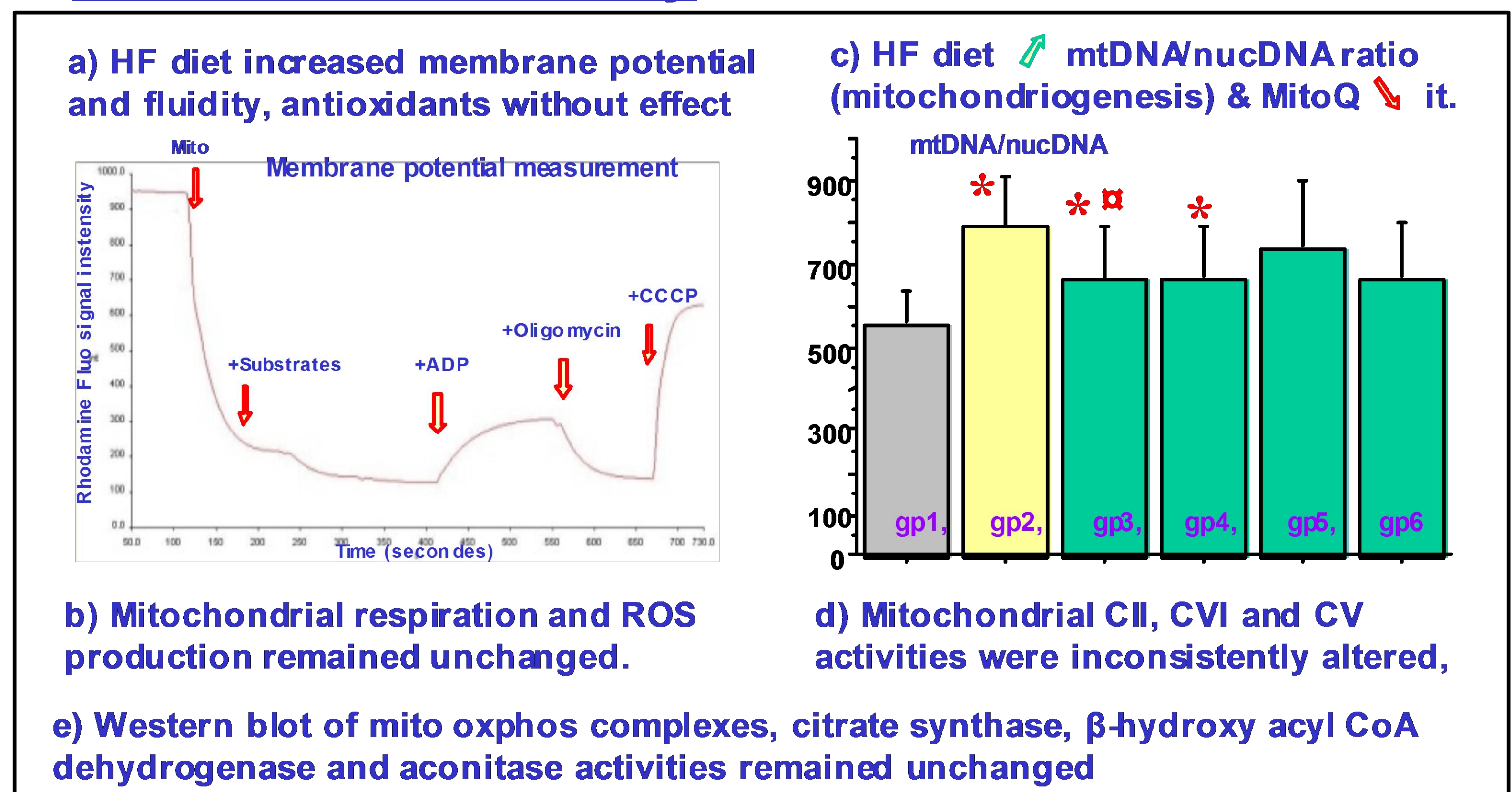
	HF vs C	Antiox vs HF		HF vs C	Antiox vs HF
<b>Plasma:</b>					
TB ARs	↓	=	<b>Liver</b>	TB ARs	↑ (MitoQ)
IsoP	↓	=		IsoP	not measured yet
AGEs	=	↓ (MitoQ)		HNE	↓ (NAC)
AOPP	↓	=		AOPP	↓ (NAC)
XO	=	=		Oxyblot	=
-SH	=	↑ (MitoQ)		XO	↓ (Allop)
SOD	=	↑ (MitoQ)		NADPH ox	(al) ↓ (AntiOx)
GPx	↓	=		-SH	↑ (NAC)
				GSH	=
				Catalase	=
<b>RBC:</b>					
Catalase	=	=	SOD	=	
SOD	=	↑ (MitoQ)	MnSOD	=	
GPx	=	=	GPx	↓	
GSH/GSSG	=	=	GRx	=	

HF diet ↓ plasma OS and ↑ liver OS, while MitoQ ↓ pl OS and NAC ↓ liver OS

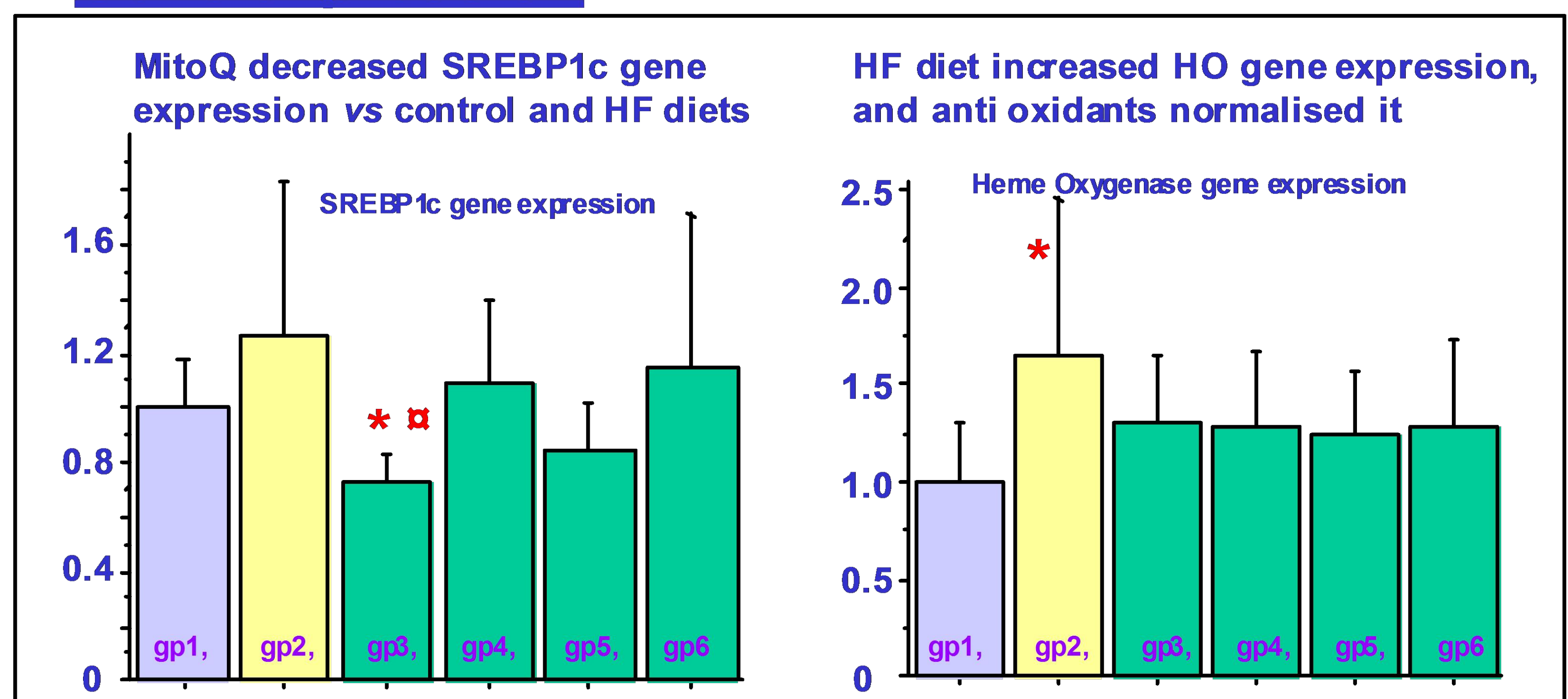
## Metabolic syndrome features



## Mitochondrial activity



## Gene expression



## Conclusions

The HF diet induced moderate obesity, hepatic steatosis, glucose intolerance, oxidative stress and mitochondrial function alterations. MitoQ showed the most important effects by decreasing body weight gain and improving glucose tolerance and some oxidative stress markers.

The MitoQ results point out the possible implication of mitochondrial ROS production in the oxidative stress-dependent obesity and insulin resistance installation and evolution, although other ROS-independent mechanisms of MitoQ action may be involved.

This work was supported by



AlimH

GLN