Spontaneous obesity in a captive-bred nonhuman primate model

SPONTANEOUS OBESITY IN A CAPTIVE-BRED NONHUMAN PRIMATE MODEL

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INTRODUCTION

- Obesity is a medical and social problem worldwide, and a significant risk factor for non-communicable diseases (NCDs) such as hypertension, cardiovascular disease (CVD) and type 2 diabetes mellitus (T2DM) (Thaker, 2017).
- Captive-bred colony of rhesus macaques (Macaca mulatta) maintained at the Primate Unit and Delft Animal Centre of the South African Medical Research Council (SAMRC/PUDAC) has shown a prevalence of spontaneous obesity.
- This study aimed to identify biomarkers that might be responsible for the observed obesity phenotype in rhesus monkeys through environmental, biochemical, and molecular approaches.

STUDY DESIGN

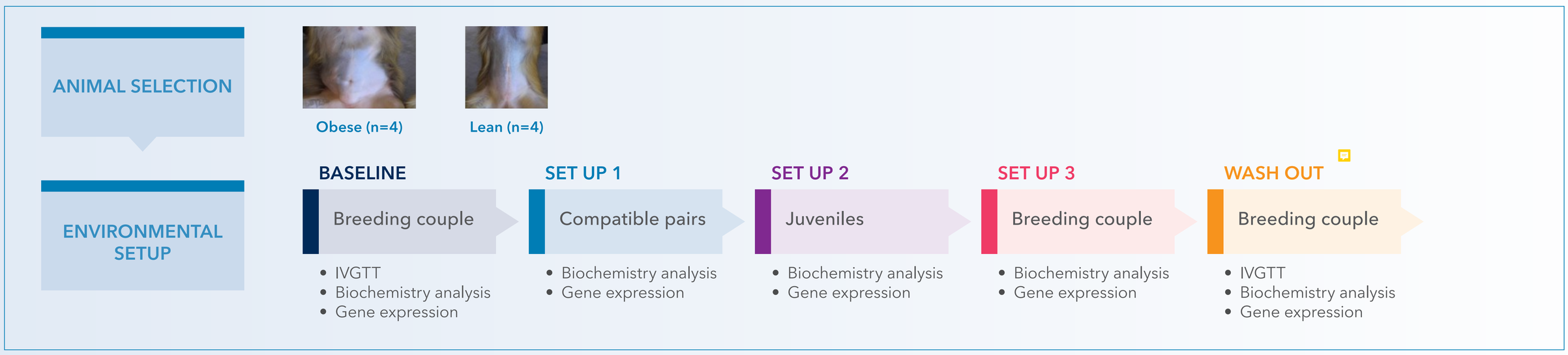

Figure 1 Study overview

RESULTS

Table 1: Biochemistry characteristics between the lean and obese rhesus females

Parameters	Groups	Baseline	Setup 1	Setup 2	Setup 3	Washout
Insulin (µIU/mL)	Lean	11.40±3.82	12.10±10.81	0.00±0.00	5.90±2.15	9.53±1.03
	Obese	14.73±6.35	14.13±7.83	0.00±0.00	15.80±16.46	9.83±1.85
	P-value*	0.40	0.77	-	0.42	0.81
Glucose (mmol/L)	Lean	3.53±0.32	3.98±0.71	3.78±0.88	3.17±0.42	4.23±0.35
	Obese	3.53±0.66	3.38±0.46	2.78±0.66	3.30±0.20	3.38±0.13
	P-value*	1.00	0.21	0.12	0.60	0.01
Cortisol (nmol/L)	Lean	698.00±224.29	672.00±245.91	0.00±0.00	851.33±160.73	1072.33±172.05
	Obese	590.00±235.82	656.75±105.87	0.00±0.00	626.50±61.87	802.75±219.12
	P-value*	0.53	0.91	-	0.05	0.14
Triglycerides (mmol/L)	Lean	0.98±0.35	0.80±0.18	0.75±0.26	0.55±0.07	0.75±0.22
	Obese	0.68±0.56	0.94±0.50	1.78±0.80	0.80±0.30	0.80±0.24
	P-value*	0.40	0.62	0.05	0.23	0.79
HbA1c (%)	Lean	4.35±0.25	4.58±0.49	0.00±0.00	4.27±0.06	4.43±0.12
	Obese	4.88±0.21	4.90±0.12	0.00±0.00	4.45±0.19	4.25±0.19
	P-value*	0.02	0.25		0.18	0.21

Descriptive mean ± standard deviation; n = 8 (4 control and 4 experimental); (-): not analyzed.
**P-value* for differences at individual time points
- P< 0.05 is considered significant.
- Estimates of the average difference between experimental-control group

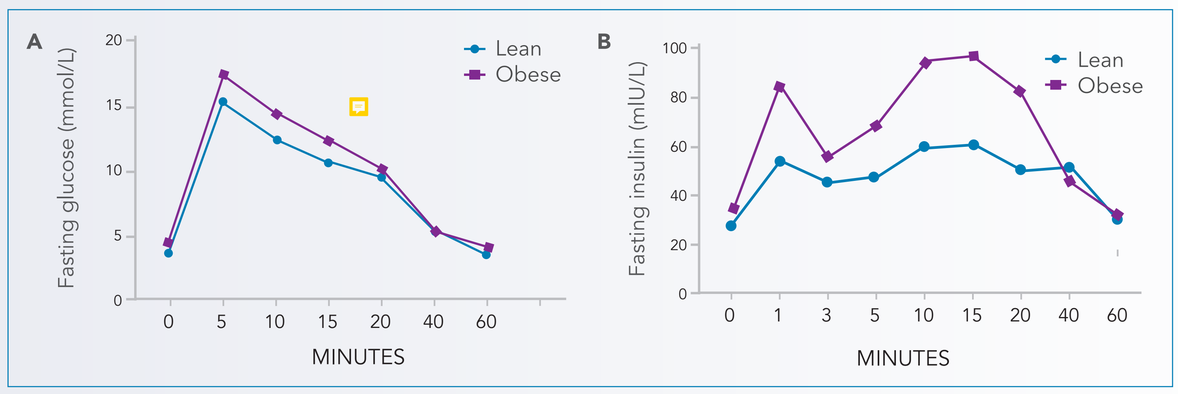

Figure 2: Intravenous glucose tolerance tests (IVGTTs) between lean and obese rhesus monkeys for glucose (A) and insulin (B) at baseline. *The values are presented as mean ± standard deviation. Statistical significance is depicted as * p≤ 0.05.*

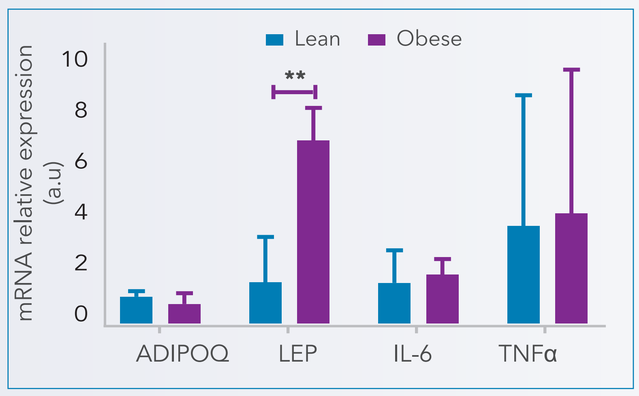

Figure 3: mRNA gene expression between lean and obese rhesus monkeys. *The relative gene expression data (arbitrary units: a.u) was normalized to the expression of two housekeeping genes (GAPDH and ACTB) and presented as mean ± standard deviation. Statistical significance is depicted as * p≤ 0.05, ** p ≤ 0.01.*

DISCUSSION AND CONCLUSION

- The different environmental housing conditions had an impact on the biochemical parameters and regulation of obesity-related genes.
- The HOMA-IR confirmed that the obese group was insulin resistant throughout the study.
- Therefore, it can be concluded that the selected obese females might be at risk of developing T2DM.

CITED REFERENCE

- Thaker VV 2017. Genetic and epigenetic causes of obesity. Adolescent medicine: state of the art reviews, 28, 379.