

The difference in glutathione peroxidase activity on arteries of a bi-ethnic population: The SABPA study

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BACKGROUND

From the literature it is evident that increased oxidative stress is integral in the development of cardiovascular disease. The immense contribution of behavioural risk factors to the high prevalence of CVD in South Africa further support the notion that increased oxidative stress may be central to this problem (1,2).

Objectives

The objectives of this sub-study were to compare glutathione peroxidase (GPx) and glutathione reductase (GR) activity between black and white Africans, and to investigate whether the activity of these enzymes are linked with cardiovascular function.

MATERIALS AND METHODS

This sub-study was embedded in the Sympathetic Activity and Ambulatory Blood Pressure in Africans (SABPA) study, included 188 black and 203 white teachers from the Dr Kenneth Kuanda Education District in the North West Province of South Africa. Ambulatory blood pressure (BP) and carotid dorsalis pedis pulse wave velocity (cdPWV) were measured. Biochemical analyses included GPx and GR enzyme activity (Cayman Chemical Company), serum peroxide (3) and total glutathione levels (OxisResearch™).

RESULTS

Table 1 Characteristics of the study population

	Africans (n= 188)	White (n=203)	P-values
Age, years	44.5 ± 8.27	44.8 ± 10.9	0.75
Gender, male, n, (%)	93 (49.5)	98 (48.3)	0.81
Body mass index, kg/m ²	30.4 ± 7.09	27.7 ± 5.94	< 0.001
CV measurements			
Ambulatory SBP, mmHg	133 ± 16.4	124 ± 12.0	< 0.001
Ambulatory DBP, mmHg	83.4 ± 10.8	76.7 ± 8.1	< 0.001
Ambulatory PP, mmHg	49.9 ± 9.18	47.3 ± 7.00	< 0.001
cdPWV, m/s	8.70 ± 1.99	8.01 ± 1.43	< 0.001
Oxidative stress markers			
GR activity, nmol/min/l	7.06 (2.55; 16.8)	2.54 (0.25; 7.64)	< 0.001
GPx activity, nmol/min/l	33.3 ± 14.1	36.2 ± 8.03	0.01
ROS, Units	92.5 (57.1; 145)	86.5 (57.4; 137)	0.049
Total glutathione, μM	889 ± 188	821 ± 174	< 0.001

Table 2 Multiple regression analysis

	Pulse wave velocity (m/s)			
	African (n=188)		White (n=203)	
Adjusted R ²	0.30		0.39	
	β (±95%CI)	P-value	β (±95%CI)	P-value
Age, years	0.22 (0.09; 0.35)	<0.01	0.37 (0.25; 0.48)	<0.01
Gender	-0.14 (-0.27; 0.00)	0.054	-0.28 (-0.41; -0.15)	<0.01
BMI, kg/m ²	-	-	-0.20 (-0.35; -0.06)	<0.01
Ambulatory MAP, mmHg	0.26 (0.13; 0.40)	<0.01	0.24 (0.09; 0.38)	<0.01
Glucose, mmol/l	0.26 (0.13; 0.40)	<0.01	0.15 (0.02; 0.28)	<0.05
CRP, mg/l	-	-	0.14 (0.02; 0.27)	<0.05
Cotinine, ng/ml	-0.09 (-0.21; 0.03)	0.16	-	-
GPx, nmol/min/l	0.08 (-0.04; 0.21)	0.20	-0.18 (-0.29; -0.07)	<0.01

BMI, body mass index; MAP, mean arterial pressure, CRP, c-reactive protein; GPx, glutathione peroxidase.

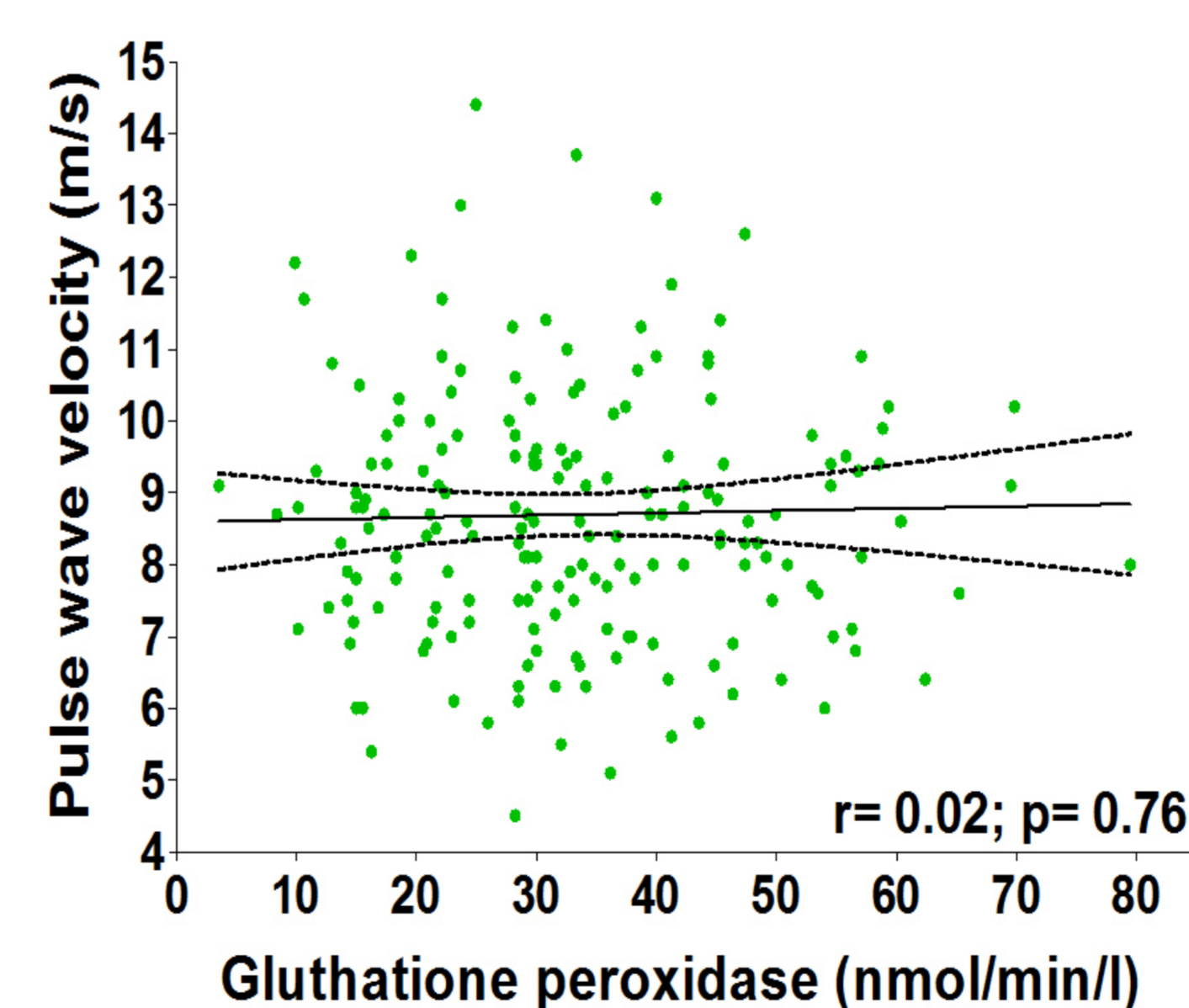
SBP, DBP and PP, systolic-, diastolic- and pulse pressure respectively; cdPWV, carotid distalis pedes pulse wave velocity; GR, glutathione reductase; GPx, glutathione peroxidase and ROS, reactive oxygen species.

The blacks presented with an unfavourable cardiovascular profile (see Table 1), with systolic- and diastolic BP, pulse pressure and cdPWV being significantly higher ($p \leq 0.006$) when compared to their white counterparts. The black group also displayed significantly higher levels of serum peroxides ($p=0.049$) with concomitant lower GPx activity ($p=0.01$), while their total glutathione levels ($p<0.001$) and GR activity ($p<0.001$) were significantly higher. In single regression analysis, an inverse relationship between GPx activity and cdPWV ($r=-0.16$; $p=0.023$) were indicated, only in the white group. The link between higher GPx activity and lower cdPWV was confirmed to be independent in multiple regression analyses ($R^2=0.39$; $\beta=-0.18$; $p=0.005$), (see Table 2) while it was absent in the black group.

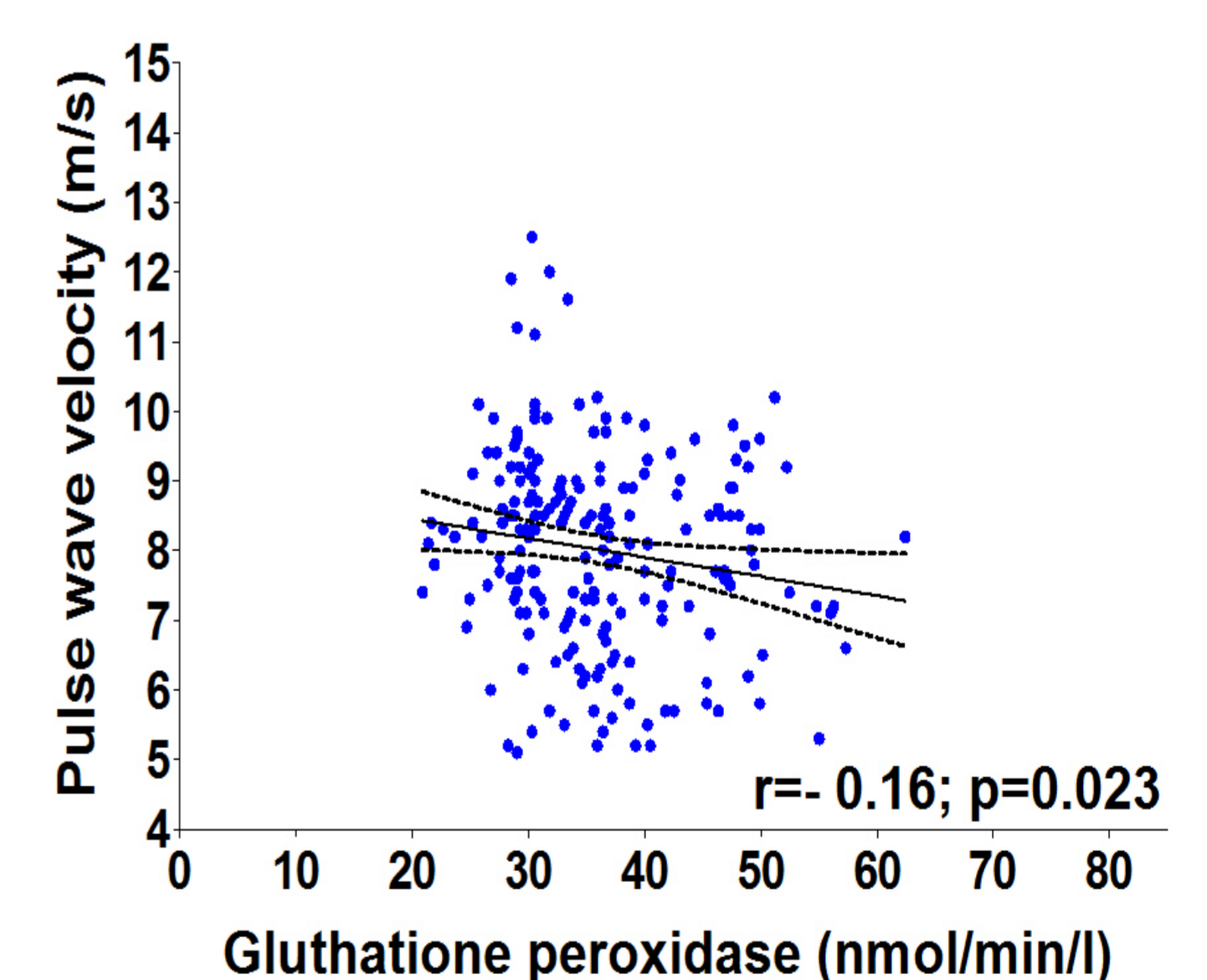
CONCLUSIONS

- An inverse relationship between GPx activity and cdPWV was encountered, suggesting:
- A protective role of higher GPx activity against arterial stiffening in the white group.

Black Africans



White Africans



Pulse wave velocity versus glutathione peroxidase activity in black and white participants.

REFERENCES

1. Kruger et al. *J Human Hypertens* 2012.
2. Schutte et al. *Am J Hypertens* 2009.
3. Hayashi et al. *Mutat Res.* 2007.

