

Effect of Post-Prandial Lipidaemia on Cerebrovascular Function

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Background: The debilitating effect that post-prandial lipidaemia (PPL) has on endothelial function within the systemic arteries has been well documented (1). However, its impact on cerebrovascular function is yet to be investigated.

Methods: Twenty young (age = 25 ± 6 years) and 20 aged (age = 67 ± 5 years) healthy males participated in the study. Cerebrovascular function and circulating triglycerides were assessed prior to, and 4 hours after the consumption of a standardised high-fat meal (2). Middle cerebral artery velocity (MCA_v; transcranial Doppler ultrasound), mean arterial pressure (MAP; photoplethysmography) and end-tidal CO₂ (PET_{CO2}; capnography) were continuously recorded throughout each testing session, while venous blood samples were obtained from an indwelling catheter. Cerebrovascular resistance (CVR) and cerebrovascular conductance (CVC) were calculated as MAP/MCA_v and MCA_v/MAP respectively, following 5 minutes seated rest. Cerebrovascular reactivity to carbon dioxide (CVR_{CO2}) was assessed in response to 3 minutes of breathing 5% CO₂ (balanced air), as described previously (3). Data were analysed using a 2 way repeated measures ANOVA and Bonferonni corrected paired sample *t*-tests and independent samples *t*-tests. Significance was established at $P < 0.05$ and data are expressed as mean \pm SD.

Results: Circulating triglycerides increased from 0.86 ± 0.49 to 2.19 ± 1.48 mmol/L in the young and from 1.44 ± 0.7 to 2.96 ± 1.6 mmol/L in the aged, following the consumption of the high-fat meal ($P < 0.05$). PPL impaired CVR_{CO2} in the aged, but not in the young (Table 1; $P < 0.05$). There were no changes in resting MCA_v, MAP, CVR or CVC in either age group.

Conclusion: These findings are the first to identify that the debilitating effects of PPL on the systemic vasculature extend to the brain. However, this effect was only present in the aged. While the underlying mechanisms remain undetermined, these observations have clear implications to the clinician, given the established relationship between impaired CVR_{CO2} and stroke risk.

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References

- (1) Wallace *et al.* (2010) *Int J Clin Pract*; **64**, 389-403.
- (2) Patsch *et al.* (1983) *PNAS*; **80**, 1449-1453.
- (3) Bailey *et al.* (2013) *Clin Sci (London)*; **124**, 177-189.

Table 1: Changes in cerebrovascular function following a high-fat meal.

Age Group	Young		Aged		P Values	
	Pre High-Fat Meal	Post High-Fat Meal	Pre High-Fat Meal	Post High-Fat Meal	Main Effect	Interaction
MCAv (cm.s⁻¹)	62 ± 13	62 ± 10	48 ± 12	47 ± 11	0.59	0.84
MAP (mmHg)	82± 10	79 ± 11	80± 18	79 ± 17	0.43	0.56
CVR (mmHg/cm.s⁻¹)	1.38 ± 0.3	1.31 ± 0.25	1.74 ± 0.51	1.77 ± 0.54	0.75	0.37
CVC (cm.s⁻¹/mmHg)	0.76 ± 0.15	0.79 ± 0.14	0.63 ± 0.22	0.63 ± 0.26	0.54	0.54
CVR_{CO2} (%.mmHg⁻¹)	2.68 ± 0.83	2.47 ± 0.89	5.34 ± 1.75†	3.99 ± 1.1*†	0.00*	0.00*

Mean ± SD; */† = difference within/between age groups ($P < 0.05$).