Effect of Post-Prandial Hyperlipidaemia on Cerebrovascular Function: Gender Difference?

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***Background*:** The consumption of a high-fat meal is characterised by a state of post-prandial hyperlipidaemia (PPH) with an exaggerated increase in triglycerides (Tg) that peaks at 4 hours[1]. We recently demonstrated that PPH was associated with impaired cerebrovascular reactivity in aged, but not young males[2]. However, to what extent PPH impacts the cerebral circulation in females, who are more prone to cognitive decline and dementia in later life[3], remains to be established.

Methods: Eighteen males (age: 24 ± 6 years; body mass index (BMI): 23.2 ± 3.6 kg.m^2) and 8 females (age: 21 ± 2 years; BMI: 23.7 ± 3.1 kg.m^2) participated in the study. Cerebrovascular function and Tg were assessed prior to, and 4 hours after the consumption of a standardised high-fat meal[1]. Middle cerebral artery velocity (MCAv; transcranial Doppler ultrasound), mean arterial pressure (MAP; finger photoplethsmography) and end-tidal CO_2 (capnography) were continuously recorded throughout each testing session. Serum Tg were determined via established methods from venous samples obtained from an indwelling cannula. MCAv and MAP were assessed following 5 minutes of seated rest. Cerebrovascular reactivity to carbon dioxide was assessed in response to 3 minutes of breathing 5% CO_2 (balanced air; $CVR_{CO2HYPER}$) and following 3 minutes of controlled hyperventilation (15 breaths per minute; $CVR_{CO2HYPO}$). Cerebrovascular range ($CVR_{CO2RANGE}$) was calculated as $CVR_{CO2HYPER}$ + $CVR_{CO2HYPO}$. Data were analysed using a 2-way repeated measures ANOVA and Bonferonni corrected paired sample *t*-tests and independent sample *t*-tests. Significance was established at $P < 0.05$ and data are expressed as mean ± SD.

Results: At baseline, females were characterised by elevated MCAv, $CVR_{CO2HYPER}$, $CVR_{CO2HYPO}$ and $CVR_{CO2RANGE}$ compared to the males (Table; all $P < 0.05$). During PPH, Tg increased relative to baseline in both groups and was associated with impaired $CVR_{CO2HYPER}$ and $CVR_{CO2RANGE}$ (Table; $P < 0.05$). Though this was independent of gender (Table; $P > 0.05$). Furthermore, PPH did not influence changes in resting MCAv or MAP (Table; $P > 0.05$).

Conclusion: Contrary to our previous findings[2], PPH has the capacity to impair cerebrovascular function in young adults. Though, it appears to be independent of gender. These observations are important given that a reduction in CVR_{CO2} may enhance the risk of stroke and neurodegenerative disease[4].

References

[1]Patsch *et al.* (1983) *PNAS*; **80**, 1449-1453.
[2]Marley *et al.* (2017) *Clin Sci*; **131**, 2807-2812.
[3]Andersen *et al.* (1999) *Neurology*; **53**, 1992-1997.
[4]Gupta *et al.* (2012) *Stroke*; **43**, 2884-2891.

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

Gender	**Males (*n* = 18)**		**Females (*n* = 8)**		***P* Values**		
	Pre-meal	**Post-meal**	**Pre-meal**	**Post-meal**	**Gender**	**Meal**	**Interaction**
Triglycerides (mmol.L)	0.88 ± 0.50	2.32 ± 1.49*	0.97 ± 0.43	1.48 ± 0.34*	0.30	**0.00**	**0.03**
MCAv ($cm.s^{-1}$)	62 ± 13	61 ± 10	73 ± 11	71 ± 10	**0.04**	0.45	0.71
MAP (mmHg)	88 ± 9	86 ± 7	80 ± 11	81 ± 11	0.09	0.73	0.39
$CVR_{CO2HYPER}$ ($\%.mmHg^{-1}$)	2.77 ± 0.77	2.40 ± 0.85	3.80 ± 0.96	3.19 ± 0.66	**0.01**	**0.01**	0.52
$CVR_{CO2HYPO}$ ($\%.mmHg^{-1}$)	2.47 ± 0.46	2.05 ± 0.50	3.45 ± 0.94	3.39 ± 0.76	**0.00**	0.24	0.36
$CVR_{CO2RANGE}$ ($\%.mmHg^{-1}$)	5.24 ± 0.87	4.45 ± 1.13	7.24 ± 1.77	6.59 ± 0.98	**0.00**	**0.01**	0.81

Values are mean ± SD; * = $P < 0.05$ *vs.* pre-meal.