Effect of acute hypoxia on dynamic response characteristics of ventilation in males and females

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The biphasic response (growth and decay) of ventilation during acute hypoxia is linked to effects of blood gases on peripheral and central chemoreceptor activity, yet the effect of sex on the size and timing of these phases is not known.

We used a two-factorial design (sex, CO_2) to study the effect of hypoxia ($F_IO_2 \approx 0.10$) on ventilation ($\dot{V}_i$) in eight female and seven male subjects. $\dot{V}_i$ was measured during two 15-minute periods of hypoxia under poikilocapnic (P) and isocapnic (I) conditions. These two periods of hypoxia were preceded by at least 15 minutes of normoxia and presented in a counterbalanced manner. Individual $\dot{V}_i$ responses were fitted to the biexponential function,

$$Y(t) = a + A_1(1 - e^{-(t-TD_1)/\tau_1}) - A_2(1 - e^{-(t-TD_2)/\tau_2}),$$

and parameters defining the size and timing of growth (1) and decay (2) phases were estimated. End-tidal PO_2 and PCO_2, as well as arterial O_2 saturation, were monitored throughout the protocol.

With respect to the growth phase, none of the parameters were affected by sex and the amplitude normalized to baseline $\dot{V}_I$ was similar between females and males during P (48.3 ± 22.2 *vs* 46.2 ± 19.2 %; mean ± SD) and I (72.2 ± 35.6 *vs* 67.8 ± 26.1 %). There was a main effect of CO_2 on the growth phase amplitude, A_1 ($F = 6.75$, $p = 0.02$), which was smaller during P compared with I in females (3.8 ± 1.9 *vs* 5.8 ± 3.4 $l \cdot min^{-1}$) and males (3.9 ± 1.5 *vs* 6.0 ± 2.5 $l \cdot min^{-1}$). With respect to the decay phase, there was a tendency for a main effect of sex on its amplitude normalized to the growth phase amplitude ($F = 4.08$, $p = 0.07$), which was larger in females than males during P (57.3 ± 26.5 *vs* 42.0 ± 29.0 %) and I (42.0 ± 29.0 *vs* 12.8 ± 31.9 %). There was also a tendency for the delay of this phase (TD_2) to increase more during I *vs* P in females (sex×CO_2: $F = 3.07$, $p = 0.1$). There was a main effect of CO_2 ($F = 6.3$, $p < 0.05$) on the amplitude of the decay phase (A_2), which was larger during P than I in females (2.4 ± 2.0 *vs* 1.3 ± 1.1 $l \cdot min^{-1}$) and males (1.7 ± 1.2 *vs* 0.6 ± 1.8 $l \cdot min^{-1}$).

These preliminary findings support other evidence of a lack of sex influence on growth of the ventilatory response in humans during shorter, incremental protocols of acute hypoxia (*e.g.* MacNutt *et al.*, 2012), but raise the possibility of sex-dependent effects on the decay of this response that is evident only during longer exposure to a fixed level of hypoxia. Some of these effects might depend on the control of end-tidal CO_2, suggesting that the use of poikilocapnic *and* isocapnic conditions is important to identification of underlying mechanisms.

MacNutt MJ, De Souza MJ, Tomczak SE, Homer JL & Sheel WA. (2012). Resting and exercise ventilatory chemosensitivity across the menstrual cycle. *J Appl Physiol* **112:** 737-747.