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Ventilation differences in the menstrual cycle: A systematic review and meta-analysis

Catherine A. Rattley ^{a,1,*}[©], Paul Ansdell ^b, Louise Burgess ^a, Malika Felton ^a, Susan Dewhurst ^a, Matthew Armstrong ^c, Rebecca Neal ^a

^a Department of Rehabilitation and Sport Sciences, Faculty of Health and Social Science, Bournemouth University, Bournemouth, UK

^b Department of Sport, Exercise and Rehabilitation, Faculty of Health and Life Sciences, Northumbria University, Newcastle-upon-Tyne, UK

^c Department of Sport and Exercise Science, Faculty of Social Sciences, Durham University, Durham, UK

ARTICLE INFO	A B S T R A C T
<i>Keywords:</i> Female sex hormones	Background: Minute ventilation (VE) may vary across the menstrual cycle due to cyclical changes in sex hor- mones, potentially exerting an exercise intensity dependent effect.
Exercise physiology	<i>Objective</i> : This systematic review and meta-analysis aimed to quantify differences in VE, respiratory frequency (RF), and tidal volume (VT) between the follicular and luteal phases at rest, during submaximal exercise (≤90 % VO _{2max}), and at incremental maximum (100 %VO _{2max}). <i>Methods</i> : The systematic review adhered to PRISMA guidelines for conducting and reporting systematic reviews. Studies included healthy eumenorrheic females (≥18 years) not using hormonal contraceptives. Studies that reported VE in the follicular and luteal phases were included. RF and VT were extracted as secondary outcome measures. Searches were conducted in Cochrane, PubMed, and EBSCO databases in January 2025. Study quality was assessed using the modified Downs and Black checklist for menstrual cycle research. Data were extracted and analysed using maximum likelihood random-effect meta-analyses followed with meta-regressions with intensity and duration as a moderator in submaximal exercise. Publication bias was assessed using Egger's test. Meta-
	regressions were also conducted for resting and submaximal datasets using change in progesterone as a moderator. <i>Results:</i> A total of 35 studies inclusive of 743 participants and 64 paired datasets were included. VE was lower in the follicular phase than the luteal phase at rest (ES = -0.64 , $p < 0.001$, $I^2 = 75.74$ %) and during submaximal exercise (ES = -0.89 , $p < 0.001$, $I^2 = 76.58$ %), with no effect of study quality. Meta-regression revealed that duration and intensity did not influence menstrual cycle effect on VE in submaximal exercise. At incremental maximum, a small but significant effect was observed towards higher VE in the luteal phase than the follicular phase (ES = -0.33 , $p = 0.03$, $I^2 = 61.5$ %) however this effect was no longer observed after removal of low-quality studies. All analyses were coupled with significant heterogenetic. $(p = 0.02)$ but not at particular to the properties of the properties of the properties of the properties of the properties.
	($p = 0.02$), RF and VT analyses suggested minimal menstrual cycle effect, with only VT reductions at rest contributing to VE differences, this was likely related to the low number of studies included. <i>Conclusions:</i> The menstrual cycle significantly influences VE, with lower values in the follicular phase at rest and during submaximal exercise. Changes in progesterone partially explain the differences in VE between menstrual phases during exercise but not at rest. At maximal intensities, hormonal influence on VE is minimal which corroborates the hypothesis that physiological mechanisms override hormonal mediation of ventilatory re- sponses at high intensities. These findings suggest potential implications for exercise performance, particularly endurance activities in the luteal phase due to the increased likelihood of hyperventilation.

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^{*} Correspondence to: Department of Rehabilitation and Sport Sciences, Human Performance Laboratory, Talbot Campus, Fern Barrow, Poole BH12 5BB, UK. *E-mail address:* krattley@burnemouth.ac.uk (C.A. Rattley).

¹ ORCID: 0000-0003-1445-3249

1. Introduction

The effect of the menstrual cycle on ventilation has been studied widely; some conclude no effect of menstrual cycle (Assadpour et al., 2020; Barba-Moreno et al., 2022; Bemben et al., 1995; Brutsaert et al., 2002; Casazza et al., 2002; Citherlet et al., 2024; De Souza et al., 1990; Dombovy et al., 1987; Eston and Burke, 1984; Hackney et al., 1991, 1994; Itoh et al., 2007; Janse De Jonge et al., 2012; Lebrun et al., 2003, 1995; Mohsenzadeh et al., 2013; Oosthuiyse and Bosch, 2006; Oxfeldt et al., 2024; Preston et al., 2001; Redman et al., 2003; Smekal et al., 2007; Smith et al., 2015; Takase et al., 2002; Williams and Parsons, 2011; Williams et al., 2023), some that there is an elevated minute ventilation (VE) in the luteal phase (Brutsaert et al., 2002; Bandyopadhyay and Dalui, 2012; Beidleman et al., 1999; Das, 1998; Hayashi et al., 2012; Jurkowski et al., 1981; Slatkovska et al., 2006; Williams and Krahenbuhl, 1997) and few that there is elevated VE in the follicular phase (Kaygisiz et al., 2003; Tagliapietra et al., 2024; Vaiksaar et al., 2011). Where there is effect, it is primarily considered to result from the stimulatory effects of progesterone on ventilation. Evidence of this progesterone-mediated effect has also been demonstrated in studies investigating natural elevations of progesterone in pregnancy (Jensen et al., 2005) and in studies administering progesterone to rats (Tatsumi et al., 1991) and males (Mikami et al., 1989). Oestrogen is also suggested to moderate ventilation and can act to upregulate the actions of progesterone on ventilation (Behan et al., 2003), highlighting the importance of considering hormone ratio alongside concentration (Hackney et al., 2022). As the effects of progesterone on ventilation are likely mediated by oestrogen (Behan et al., 2003; Bayliss and Millhorn, 1992; Tatsumi et al., 1997; Scott et al., 2000) the effect of the menstrual cycle on ventilation may require study of female participants in the early follicular, late follicular, ovulatory and midluteal phase to study four hormone profiles due to the complex interplay of sex hormones (Elliott-Sale et al., 2021).

In the mid-luteal phase, when oestrogen is moderate and progesterone is high, ventilation is reported to increase (Schoene et al., 1981). Hyperventilation in the luteal phase at rest was demonstrated by Das (Das, 1998); as pulmonary mechanics did not change, the author concluded that the elevation of ventilation resulted from the stimulatory effects of progesterone on respiratory drive. This is confirmed in work by Slatkovska et al (Slatkovska et al., 2006). whereby females in the luteal phase evidenced higher VE and lower arterial partial pressure of carbon dioxide (PaCO₂) compared to the follicular phase and this was not the result of differences in central and peripheral chemoreflex drives to breathe. Sex hormones instead may modulate the function of central and peripheral chemoreflexes via neural control mechanisms (Behan et al., 2003) in the medulla and hypothalamus (Loiseau et al., 2018), structures involved in central respiratory drive. Progesterone may act in these brain regions by enhancing CO2 chemosensitivity of central chemoreceptors (Loiseau et al., 2018; Georgescu, 2023). For instance, in rats and males, progestin administration improved CO2 chemosensitivity (Janes et al., 2024; Zwillich et al., 1978), and in pregnant females, CO2 chemosensitivity also increased (Jensen et al., 2005). This suggests that in the luteal phase when oestrogen and progesterone are elevated, increased responsiveness to elevating CO2 during exercise leads to increased VE. Additionally, the ventilatory recruitment threshold, which is the PaCO₂ at which ventilatory drive increases to respond to elevated PaCO₂, is lowered in pregnancy (Jensen et al., 2005). This taken with the finding that the ventilatory recruitment threshold is lower in females compared to males (MacNutt et al., 2012) suggests this lowering is hormonally driven, however this has not been corroborated by menstrual cycle changes in ventilatory recruitment threshold (Dutton et al., 1989). A lower ventilatory recruitment threshold results in higher ventilation at lower PaCO2 which, under exercise conditions, could lead to hyperventilation. Moreover, this menstrual cycle effect on ventilation is intensity dependent, with physiological mechanisms overriding the effect of sex hormones at high and maximal intensities (MacNutt et al.,

2012; Prado et al., 2021).

Sex hormone induced hyperventilation in exercise may increase respiratory muscle fatigue and result in decreased exercise tolerance (Harms et al., 2000; Romer and Polkey, 2008), which is supported by findings that exercise-induced asthmatic symptoms worsen in the midluteal phase (Stanford et al., 2006) and resulted in increased use of bronchodilators. While hyperventilation can lead to enhanced performance in some contexts such as near maximal or maximal exercise, as shown in males (Sakamoto et al., 2014) it can increase metabolic demand and the oxygen cost of breathing (MacNutt et al., 2012). This could limit oxygen delivery to working muscles and contribute to greater fatigue (Harms et al., 2000) in endurance contexts. Conversely, if pulmonary ventilation is reduced in the follicular phase compared to the luteal phase, it may reduce capacity to meet the metabolic demands of working muscles (MacNutt et al., 2012) alongside increases in metabolic byproducts such as lactate, as shown utilising voluntary hypoventilation in males (Woorons et al., 2010). Hypoventilation may also reduce metabolic demand and improve performance in assessments such as repeated sprints and running 200 m time trials (Woorons et al., 2020 Feb 27). Previously, a lower ventilatory response to stressors in athletes, such as hypoxia, hypocapnia or exercise, has been highlighted as beneficial, subsequently progesterone induced hyperventilation may hinder performance in some contexts (Schoene et al., 1981). Thus, the implications of the effect of the menstrual cycle on ventilation and subsequently performance are sport or activity dependent.

This is the first systematic review and meta-analysis to study the effect of the menstrual cycle on ventilation at rest, during submaximal exercise and at incremental maximum. This analysis seeks to clarify whether menstrual cycle phase significantly affects VE at rest and during exercise and the contribution of changes in RF or VT to this effect. This review also seeks to establish whether the effect is duration- and intensity-dependent due to the hypothesis that, with increasing intensity, the corresponding cardiopulmonary response overrides sex hormone effects. It is expected that the menstrual cycle will have greater effect on VE at low exercise intensities.

2. Methods

This review conforms to the PRISMA statement guidelines for conducting and reporting systematic reviews.

2.1. Eligibility criteria

Eligible studies included a sample of healthy eumenorrheic female participants who were over 18 years old and not utilising hormonal contraceptives. Studies were required to report absolute VE from the follicular phase and the luteal phase at rest or in a dose of exercise at a reported percentage of VO_{2max} . Where studies had reported respiratory frequency (RF), and tidal volume (VT) these variables were also extracted. Studies were excluded which utilised a nutrition or breathing intervention. Where studies had included more than two phases, the earlier follicular and midluteal phase values were utilised as a paired dataset. Datasets were included both at sea level and at altitude, datasets at altitude or in hypoxic conditions were removed for sensitivity analysis. Studies reporting exercise conducted at or below 90 %VO_{2max} were categorised as submaximal exercise. Where a maximal test had been conducted, maximum values were categorised as incremental maximum.

2.2. Information sources and search strategy

Searches were conducted of Cochrane, PubMed and EBSCO databases in January 2025, searching for papers from inception. The following search terms were utilised: ("menstrual cycle" OR "menstrual phase" OR "follicular phase" OR "luteal phase" OR "ovulatory phase" OR "mid-cycle") AND ("minute ventilation" OR "VE" OR "ventilation" OR "ventilat*" OR "ventilatory" OR "respiratory rate" OR "breathing frequency" OR "respirat*" OR "fb" OR "RR" OR "tidal volume" OR "VT" OR "TV" OR "pulmonary ventilation" OR "breathing rate" OR "airflow") AND ("rest" OR "exercise"). Following database searching, citations were hand-searched for further relevant papers.

2.3. Selection process

Papers were screened (CR) for duplicates, after which titles and abstracts of studies were screened and studies that did not meet inclusion criteria were removed. Following full-text retrieval and requests for supplementary datasets, papers were assessed for inclusion of relevant outcome variables with required details such as menstrual cycle phase and exercise intensity.

2.4. Data collection process

Data were extracted from papers (CR), where numerical data and supplementary data had been sought and not been provided, data were extracted from graphs (Image J, Maryland, USA, Imagej.net). Data were extracted in pairs, where two groups had been utilised, for example high and low fitness levels, both datasets were extracted and assigned a or b. The primary outcome variable was VE, with RF and VT as secondary variables. Intensity as $%VO_{2max}$, and duration in minutes were also extracted alongside outcome variables. Sample size, environmental conditions, participant characteristics, modality of exercise, and progesterone concentrations for each phase were also extracted.

2.5. Risk of bias

Studies were assessed for quality by two independent researchers (CR, LB) and discrepancies resolved in meetings. The studies were assessed using the modified Downs and Black checklist for menstrual cycle research (McNulty et al., 2020).

2.6. Data synthesis and analysis

Maximum likelihood random-effect meta-analyses were conducted on Jamovi (*jamovi* Version 2.3.28.0, retrieved from https://www. jamovi.org) providing mean difference and confidence intervals. Figures were prepared in Jamovi. Heterogeneity was assessed by I^2 statistic for an estimate of variability. 30–60 % was interpreted as moderate heterogeneity, 50–90 % as substantial and 90–100 % as considerable, in line with Cochrane Handbook recommendations (9.5.2, accessed at handbook-5–1.cochrane.org). Publication bias was assessed using Egger's test. Sensitivity analysis removed low- and very lowquality studies to assess effect of quality on effect size. Additionally, studies under hypoxic conditions were removed to assess influence on effect size. These sensitivity analyses assess risk of bias from study quality. Effect sizes were interpreted in line with Cohen (2013) whereby 0.8 was considered large, between 0.5 and 0.8 as moderate, between 0.2 and 0.5 as small and between 0 and 0.2 as minimal.

For submaximal exercise, mixed-effects meta-regressions using continuous covariates were conducted to examine the effects of duration (minutes) and intensity (%VO_{2max}) on VE. The analysis was performed using the restricted maximum likelihood (REML) estimator. Duration and intensity were continuous variables, three datasets were excluded for examining the effect of duration and the interaction of duration and intensity due to undisclosed duration. A mixed effects meta-regression was also conducted to assess the effect of change (Δ) in progesterone between phases, 25 datasets were included across resting and submaximal exercise that had provided blood hormone levels of progesterone in both phases. These meta-regression analyses were conducted in R (The R foundation 2021, version 4.4.2, retrieved from cran.r-project.org) utilising the standardised mean difference from Jamovi output.

3. Results

3.1. Study selection

Thirty-five studies were eligible for meta-analysis, inclusive of 743 participants, giving 64 datasets in total. 18 at rest, 22 during submaximal exercise, and 23 at incremental maximum. Duration varied from 3 to 100 minutes and intensity from 35 % to 90 % VO_{2max}. A flow diagram of the literature search and included studies is provided in Fig. 1.

3.2. Study characteristics

The included studies are described in supplementary materials (S1). 11 studies did not provide exercise as a percentage of VO_{2max} so only resting datasets were used (Citherlet et al., 2024; Itoh et al., 2007; Preston et al., 2001; Tagliapietra et al., 2024), and seven were excluded (MacNutt et al., 2012; Rael et al., 2021; Benito et al., 2023; Birch and Reilly, 1997; Ferguson et al., 2019; Lee et al., 2024; Matsuo et al., 2003) (Fig. 1).

3.3. Risk of Bias

The study quality was assessed by modified Downs and Black checklist for menstrual cycle research which considers the quality of the methods used for the determination of menstrual cycle phase (McNulty et al., 2020). A priori, 17 studies (49 %) were considered high quality, 15 moderate (43 %), 3 low (9 %) and 0 very low. Following assessment of menstrual cycle control by blood hormones or urinary ovulation kit usage, 14 were high quality (40 %), 8 were moderate (9 %), 3 were low (9 %) and 10 were very low (29 %). Only 8 studies provided an a priori power calculation and 11 completed familiarization trials prior to experimental conditions. Complete quality analysis is available in the supplementary materials (S2).

3.4. Results of syntheses

The full table of data extraction is available in supplementary materials (S3).

3.4.1. Minute ventilation

A mean effect size towards VE being lower in the follicular phase than the luteal phase was found at rest, during submaximal exercise, and at maximum (Table 1). This effect size was moderate at rest (ES -0.64), large under submaximal exercise (ES -0.89) and small at maximum (ES -0.33). Substantial heterogeneity was present in this analysis for all conditions (Table 1).

3.4.1.1. *Resting.* At rest, there was a significant moderate effect size (ES -0.64 [-1.03 to -0.25], p < 0.001) towards lower VE in the follicular phase, accompanied with substantial heterogeneity (I²=75.74 %; Fig. 3).

When very low- and low-quality studies were removed, of which there were five (Assadpour et al., 2020; Brutsaert et al., 2002; Itoh et al., 2007; Das, 1998; Hayashi et al., 2012), the effect size remained the same (ES -0.76 [-1.32 to -0.22]) and heterogeneity was not significantly altered (I²=80.58 %). Additionally, when datasets involving measurement under hypoxia were removed, of which there were two (Citherlet et al., 2024; Takase et al., 2002), there was no alteration in effect size (ES -0.64 [-1.05 to -0.23]) nor heterogeneity (I²=77.95 %).

Thirteen datasets provided blood hormone values for progesterone in the follicular and luteal phase with resting VE. From this, Δ progesterone between phases was calculated (follicular progesterone - luteal progesterone). A mixed-effects meta-regression model was used to examine the effect of hormonal changes (follicular - luteal progesterone) on resting VE. The analysis showed significant residual heterogeneity in the data



Fig. 1. PRISMA flow diagram of study inclusion in the systematic review.

Table 1 Effect size, heterogeneity and publication bias for each intensity analysis of minute ventilation.

Intensity	Effect size [95 % CI]	р	I^2	Eggers p value
Rest	-0.64 [-1.03 to -0.25]	< 0.001	75.7 %	0.029
Submaximal	-0.89 [-1.26 to -0.51]	< 0.001	76.6 %	0.034
Incremental Maximum	-0.33 [-0.62 to -0.03]	0.03	61.5 %	0.926

(74.96 %, p < 0.001), the model accounted for 19.37 % of the observed variability. The relationship between Δ progesterone and menstrual cycle differences in resting VE was not significant ($\beta = 0.11$ [-0.01–0.23], p = 0.07). This suggests that larger changes in progesterone between the follicular and luteal phases are not significantly associated with changed resting VE. Mean change in resting VE between menstrual cycle phases in eligible datasets was 1.70 L·min^{-1} whilst mean Δ progesterone was 9.0 ng/ml.

3.4.1.2. Submaximal exercise. In submaximal exercise, there was a large effect of menstrual cycle on VE (ES -0.89 [-1.26 to -0.51]; Fig. 4). A meta-regression revealed that intensity (residual heterogeneity 77.83 %, p < 0.001) and duration (residual heterogeneity 65.99 %, p = 0.002,

Table 2

Effect size, heterogeneity, and publication bias for respiratory frequency and tidal volume at rest (n = 13), during submaximal exercise (n = 5) and at maximum (n = 6 for RF and n = 7 for VT).

Intensity	Effect size [95 % CI]	I^2	р	Eggers p value
Respiratory frequency				
Rest	-0.35 [$-0.70 - 0.01$]	63.5 %	0.06	0.68
Submaximal	-0.32 [-0.65 -0.02]]	0.0 %	0.06	0.95
Incremental	-0.26 [-0.74 -0.22]	56.1 %	0.28	0.76
Maximum				
Tidal volume				
Rest	-0.52 [-0.90 to -0.13]	67.3 %	0.01	0.02
Submaximal	-0.49 [-1.34-0.36]]	83.0 %	0.26	0.04
Incremental	-0.29 [-0.89 -0.33]	56.5 %	0.05	0.01
Maximum				

three studies did not provide duration) did not have a significant effect on VE (p > 0.05).

When very low- and low-quality studies were removed, of which there were 3 (Brutsaert et al., 2002; Eston and Burke, 1984; Hayashi et al., 2012) comprising six datasets, the effect size remained the same (ES -0.73 [-1.12 to -0.34]) and heterogeneity was not significantly altered (I²=64.4 %).

Twelve datasets provided blood hormone values for progesterone in the follicular and luteal phase with submaximal exercise VE. The mixedeffects meta-regression analysis of these 12 datasets revealed a





Williams & Krahenbuhl, 1997a	├──-	-2.64 [-3.98, -1.30]
Slatkovska et al., 2006	├── ■──┤	-2.12 [-3.17, -1.08]
Preston et al., 2001	⊢_ ∎{	-1.92 [-2.82, -1.03]
Kaygisiz et al., 2003a	⊢	-1.75 [-2.84, -0.66]
Redman et al., 2003a	■	-0.99 [-1.78, -0.21]
Takase et al., 2002a	⊢	-0.84 [-1.80, 0.12]
Hayashi et al., 2012a*	⊢_ ∎	-0.77 [-1.68, 0.13]
Takase et al., 2002b	⊢	-0.72 [-1.67, 0.23]
Oosthuyse & Bosch, 2006a	—	-0.59 [-1.48, 0.31]
Smekal et al., 2007b	— —	-0.43 [-1.08, 0.21]
Brutsaert et al., 2002a*	⊢ ∎-	-0.42 [-1.01, 0.16]
Itoh et al., 2007a*	⊢	-0.35 [-1.49, 0.79]
Das et al., 1998*		-0.33 [-0.77, 0.11]
Williams et al., 2023a	⊢ −	-0.07 [-0.72, 0.58]
Clitherlet et al., 2024	⊢ −∎	-0.06 [-0.83, 0.70]
Assadpour et al., 2020*	⊢ −	0.00 [-0.80, 0.80]
Hackney et al., 1991a	F-	0.65 [-0.51, 1.81]
Tagliapietra et al., 2024		0.88 [0.15, 1.60]
RE Model	•	-0.64 [-1.03, -0.25]
_	-4 -3 -2 -1 0	1 2
Towards lower in follicu	Ilar phase Effect size	e Towards lower in luteal phase

Fig. 3. Standardized mean difference in minute ventilation at rest between the follicular and luteal phases of the menstrual cycle (n = 18), * indicates low or very low-quality studies removed for sensitivity analysis.

Williams & Krahenbuhl, 1997b		4	-3.2	21 [-4.70, -1.73]
Janse De Jonge et al., 2012	⊢		-2.	56 [-3.81, -1.32]
Brutsaert et al., 2002c*	├─■		-2.5	25 [-2.99, -1.51]
Brutsaert et al., 2002b*	├──∎	н	-2.0	02 [-2.73, -1.31]
Williams & Krahenbuhl, 1997c	•	⊷	-1.9	93 [-3.11, -0.74]
Brutsaert et al., 2002d*	⊢-1	B	-1.8	87 [-2.56, -1.18]
Hackney et al., 1994b	F		-1.1	18 [-2.18, -0.18]
Hackney et al., 1994a	F		-1.1	10 [-2.09, -0.11]
Hayashi et al., 2013b*	I	├──■──┤	-0.9	99 [-1.92, -0.06]
Barba-Moreno et al., 2022		├──■──┤	-0.9	96 [-1.72, -0.20]
Hackney et al., 1994c		├── ■ ──┤	-0.	76 [-1.72, 0.20]
De Souza et al., 1990a		├──■──┤	-0.	58 [-1.58, 0.42]
Beidleman et al., 1999a		┝──■──┤	-0.	25 [-1.24, 0.73]
Oxfeldt et al., 2024a		├──■──┤	-0.5	22 [-1.06, 0.61]
Beidleman et al., 1999b		├──■ ──┤	-0.	19 [-1.17, 0.79]
Hackney et al., 1991b		├──■──┤	-0.	17 [-1.31, 0.96]
Oosthuyse & Bosch, 2006b		├──■ ──┤	-0.	17 [-1.05, 0.71]
Eston & Burke, 1984b*		⊢ ∎1	-0.	16 [-0.77, 0.44]
Williams et al., 2023c		⊢∎⊢	-0.	14 [-0.79, 0.52]
Williams et al., 2023b		⊢∎	-0.	12 [-0.77, 0.54]
Eston & Burke, 1984a*		-∎-1	0.	01 [-0.59, 0.62]
Oxfeldt et al., 2024b		⊢	0.	05 [-0.78, 0.89]
RE Model		•	-0.8	89 [-1.26, -0.51]
-6	-4 -2	2 0	2	
Towards lower in follicul	ar phase Effect	ct size Toward	s lower in luteal ph	ase

Fig. 4. Standardized mean difference in minute ventilation during submaximal exercise between 35 % and 90 % VO_{2max} between the follicular and luteal phases of the menstrual cycle (n = 22). * indicates low or very low-quality studies removed for sensitivity analysis.

significant positive relationship between Δ progesterone and submaximal VE ($\beta = 0.18$ [0.0211–0.2138], p = 0.02). Residual heterogeneity was moderate (I² = 58.33 %, p = 0.01), with hormonal changes accounting for 41.95 % of the variability in VE. This suggests that larger changes in progesterone between the follicular and luteal phases are associated with increased VE. Meta-regression output revealed that with each 1 ng/ml increase in progesterone, VE increases by 0.18 L·min⁻¹. Mean change in VE between menstrual cycle phases in eligible datasets was 2.26 L·min⁻¹ and mean Δ progesterone was 11.45 ng/ml. However, the intercept was non-significant (p = 0.48), suggesting that VE was not significantly different from zero without hormonal changes.

towards lower VE in the follicular compared to the luteal phase (ES-0.33 [-0.62 to -0.03]), with considerable heterogeneity (I²=61.5 %; Fig. 5). This heterogeneity was removed (I²=0 %), and the effect size reduced to minimal (ES-0.23 [-0.46–0.00]) when very low- and low-quality studies were removed, of which there were 6 (Brutsaert et al., 2002; Dombovy et al., 1987; Itoh et al., 2007; Mohsenzadeh et al., 2013; Williams and Parsons, 2011; Bandyopadhyay and Dalui, 2012). Study quality was greatly influential on the effect of menstrual cycle on VE at maximum.

3.5. Respiratory frequency and tidal volume

3.4.1.3. Incremental maximum. There was a small significant effect

Thirteen studies provided RF and VT at rest, five during submaximal exercise, and six and seven, for RF and VT respectively, at maximum. All

Brutsaert et al., 2002e*	⊢	-2.44 [-3.46, -1.42]
Banduopadhyay & Dalui, 2012*	┝╼╾┥	-1.52 [-1.99, -1.05]
Jurkowski et al., 1981	⊢	-1.50 [-2.55, -0.45]
Takase et al., 2002c	⊢ ∎1	-0.63 [-1.57, 0.32]
Takase et al., 2002d	⊢ ∎1	-0.60 [-1.54, 0.34]
Dombovy et al., 1987*	⊢ ∎́-1	-0.55 [-1.54, 0.45]
Casazza et al., 2002	⊢ -	-0.51 [-1.66, 0.64]
LeBrun et al., 1995	⊢_ ∎1	-0.37 [-1.07, 0.33]
Smith et al., 2015	⊢	-0.25 [-1.23, 0.74]
Redman et al., 2003b	⊢_ ∎ <u></u> 1	-0.22 [-0.96, 0.53]
Beidleman et al., 1999c	⊢	-0.20 [-1.19, 0.78]
LeBrun et al., 2003	⊢	-0.11 [-1.16, 0.94]
Smekal et al., 2007a	⊢ ∎1	-0.11 [-0.74, 0.53]
Bemben et al., 1995	⊢	-0.10 [-1.34, 1.14]
Vaiksaar et al., 2011a	↓ <u> </u>	-0.07 [-1.05, 0.91]
Williams & Parsons et al., 2011*	⊢	-0.05 [-0.97, 0.87]
Itoh et al., 2008b*	⊢ I	-0.05 [-1.18, 1.08]
De Souza et al., 1990b	⊢	-0.04 [-1.02, 0.94]
Kaygisiz et al., 2003b	⊢	0.11 [-0.82, 1.03]
Mohsenzadeh et al., 2013a*	↓ _	0.28 [-0.34, 0.91]
Vaiksaar et al., 2011b	⊢ ∎1	0.32 [-0.73, 1.37]
Mohsenzadeh et al., 2013b*	⊢ ∎−−1	0.53 [-0.10, 1.16]
Beidleman et al., 1999d	⊢_ ∎	0.53 [-0.46, 1.53]
RE Model	•	-0.33 [-0.62, -0.03]
I		
-	4 -3 -2 -1 0 1 2	
Towards lower in follic	ular phase Effect size Towards lower ir	າ luteal phase

Fig. 5. Standardized mean difference in minute ventilation at maximum between the follicular and luteal phases of the menstrual cycle (n = 23). * indicates low or very low quality studies removed for sensitivity analysis.

analyses of RF and VT evidenced a low to moderate effect size towards lower values in the follicular than the luteal phase. There was a significant effect of menstrual cycle on VT at rest, which was coupled with very high heterogeneity and significant Egger's p values suggesting publication bias. Similarly, high heterogeneity was evidenced in RF analysis at rest and at maximum. Taken together these results evidence a limited effect of menstrual cycle VT and RF during exercise, limiting inferences about their role in VE changes. However, at rest, VE changes may be related to a menstrual cycle effect on VT.

4. Discussion

Minute ventilation (VE) is reduced in the follicular phase of the

menstrual cycle compared to the luteal phase at rest and during submaximal exercise. These changes are the result of reductions of tidal volume (VT) at rest, whereas during submaximal exercise, it appears to be a combination of both respiratory frequency (RF) and VT reductions. The high heterogeneity observed in these analyses was not linked to study quality, as sensitivity analyses removing very low- and low-quality studies did not alter the findings. At incremental maximum, the menstrual cycle had limited effect on VE, and the effect present resulted from very low- and low-quality studies. This corroborates the hypothesis of Prado et al. (2021) that at maximum intensities, increasing cardiopulmonary response overrides the actions of sex hormones on ventilation. Therefore, performance of maximal exercise is not limited by menstrual cycle related constraints on ventilation, this may further elucidate why there are limited menstrual cycle effects on maximal aerobic capacity (Rael et al., 2021).

Exercise intensity and duration did not account for any variability in the model, hence the menstrual cycle effect on VE during submaximal exercise appears to occur regardless of exercise intensity or duration. This contrasts the conclusion of MacNutt et al., 2012). who suggested that the effects of hormones on ventilation are intensity dependent, specifically, only occurring at low intensities. Some variability in submaximal analyses was accounted for by change in progesterone, reinforcing that there is an effect of hormones on exercise VE. During submaximal exercise across all intensities, there was a significant positive relationship between change in progesterone and VE with a 1 ng/ml increase in progesterone corresponding to a $0.18 \text{ L} \cdot \text{min}^{-1}$ increase in VE. This relationship between change in progesterone and VE was not significant at rest, although did account for 19 % of the variability in the model, implying some hormonal influence on effect size. It is possible that ventilation is affected not only by absolute hormone concentrations but also by relative fluctuations between high and low hormone levels (Real et al., 2007; Skobeloff et al., 1996). This hypothesis is supported by a lack of difference in ventilatory drive between active and inactive pill phases of oral contraceptive users when fluctuations are suppressed (Nettlefold et al., 2007) and further reinforced by reports of exacerbation of asthma symptoms in the early follicular phase and late luteal phase when sex hormones are low or declining (Graziottin and Serafini, 2016; Murphy and Gibson, 2008; Brenner et al., 2005). Premenstrual asthma is more common in those with shorter menstrual cycles (Sánchez-Ramos et al., 2017), where the usual sex hormone fluctuations occur in a shorter time window and overall exposure to sex hormones per cycle is reduced (Mumford et al., 2012). These findings reinforce the hypothesis that it is not just absolute hormone concentrations, but also their fluctuations that influence respiratory control, including ventilation. This would explain why progesterone supplementation elicits changes in VE (Tatsumi et al., 1991; Mikami et al., 1989) but research is inconsistent across female menstrual cycle studies (Assadpour et al., 2020; Barba-Moreno et al., 2022; Brutsaert et al., 2002; Citherlet et al., 2024; De Souza et al., 1990; Eston and Burke, 1984; Hackney et al., 1991, 1994; Janse De Jonge et al., 2012; Oosthuiyse and Bosch, 2006; Oxfeldt et al., 2024; Preston et al., 2001; Williams et al., 2023; Beidleman et al., 1999; Das, 1998; Hayashi et al., 2012; Slatkovska et al., 2006; Williams and Krahenbuhl, 1997; Tagliapietra et al., 2024), where individual variability in hormone concentrations likely plays a role.

The exact mechanism for this increase in ventilation as a result of sex hormones is unclear, it is suggested that the effect is not primarily exerted by influence on central or peripheral chemoreflex (Slatkovska et al., 2006), but rather through direct influence on neural control via cortical, cerebellar or hypothalamic nuclei (Behan et al., 2003), increasing chemosensitivity to CO2 (Loiseau et al., 2018; Georgescu, 2023), however this has not been evidenced by a reduced ventilatory recruitment threshold in the luteal phase (Dutton et al., 1989). The stimulatory effects of progesterone primarily act to correct elevated PaCO₂ by increases in VT (Marcouiller et al., 2014; Skatrud et al., 1980) with supplementary increases in RF (Mockridge and Maclennan, 2022; Bairam et al., 2013), as shown in pregnancy (Mockridge and Maclennan, 2022), rats (Marcouiller et al., 2014; Bairam et al., 2013), and patients with chronic ventilatory failure (Skatrud et al., 1980). Therefore, in submaximal exercise (Fig. 4), VT may increase at earlier exercise intensities in the luteal phase compared to the follicular phase, however maximal VT remains the same, hence the limited differences in VT and VE at high intensities and at maximum (Fig. 5). This increased ventilation during submaximal exercise in the luteal phase may increase the risk of respiratory muscle fatigue, metabolic demand, cost of breathing, and therefore exercise tolerance (MacNutt et al., 2012; Harms et al., 2000; Romer and Polkey, 2008) which could place limitations on aerobic performance (Schoene et al., 1981). Despite this, menstrual cycle differences in performance have not been observed (McNulty et al.,

2020), which might be explained by the mixing of tasks requiring anaerobic and aerobic capacities. Indeed, a further systematic review in 2021, highlighted current evidence for improved endurance performance in the luteal phase (Meignié et al., 2021) and the importance of considering sports or activities separately.

4.1. Strengths and limitations

All analyses involved high variability, and the meta-regression revealed this variability did not result from intensity or duration. Hence, it likely results from an external variable, this could include the measurement method, for example use of ventilatory hood or use of breath-by-breath system or modality, such as treadmill or cycle ergometer. Heterogeneity could also result from different training statuses of the samples included in addition to the small sample sizes of the included studies, the average number of participants in each study was 13. Janse De Jonge et al (Janse Jonge et al., 2019). noted that studies with under 15 participants were less likely to find differences compared to studies with 22-100 participants. Larger samples in menstrual cycle investigation account for individual variability in hormone levels and smaller samples lead to inconclusive results (Schmalenberger et al., 2021). This may also explain why this meta-analysis identified a large effect of menstrual cycle on resting and exercise VE where individual studies had not, due to the inclusion of 247 and 276 participants respectively. Similarly, as fewer studies reported VT and RF with VE, confirmation of the causative factor for increased VE is restricted, particularly in submaximal exercise where only five studies provided these outcome measures.

As frequently reported, different verification methods for menstrual cycle have differing effects on quality (Elliott-Sale et al., 2021) and this meta-analysis included all methods of verification. This work also involved all studies with a follicular to luteal comparison with preference towards early follicular and mid luteal however in some cases mid-follicular was reported instead. This could lead to differing hormonal profiles being presented under the follicular phase.

Finally, this review involved searching of supported databases by the institution, meaning some relevant databases may have been excluded. Future studies could expand the scope by incorporating a broader range of sources.

5. Conclusions

The menstrual cycle has a moderate effect on resting VE and a large effect on submaximal exercise VE regardless of exercise intensity and duration. In submaximal exercise, this effect was moderated by change in progesterone concentrations between phases consolidating reports of progesterone as a ventilatory stimulant. Maximal VE was unaffected by menstrual cycle phase, corroborating the hypothesis that cardiopulmonary response overrides hormonal regulation of ventilatory drive at high intensities. Subsequently, there is potential for endurance performance to be compromised in the luteal phase of the menstrual cycle, due to increased blood flow competition between respiratory and locomotor muscles and risk of respiratory muscle fatigue, which could combine to reduce exercise tolerance.

Other information

This review was not registered, nor a protocol published.

Study inclusion and characteristics are available in S1, the quality assessment in S2, and data extraction in S3.

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CRediT authorship contribution statement

Rattley Catherine A: Writing – review & editing, Writing – original draft, Methodology, Formal analysis, Data curation. Paul Ansdell: Writing – review & editing, Visualization, Supervision. Susan Dewhurst: Writing – review & editing, Supervision. Matthew Armstrong: Writing – review & editing, Visualization. Louise Burgess: Writing – review & editing, Formal analysis. Malika Felton: Writing – review & editing, Supervision, Funding acquisition. Neal Rebecca A: Writing – review & editing, Supervision, Funding acquisition.

Declaration of Competing Interest

The authors have no relevant financial or non-financial interests to disclose.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.resp.2025.104468.

Data availability

Data extraction is available in supplementary materials.

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