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The effect of treadmill incline on maximal oxygen uptake, gas exchange and the metabolic response to exercise in the horse

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In healthy man, conditions that change muscle O$_2$ delivery affect the achievable maximum rate of O$_2$ uptake (V$\text{O}_{2\text{,max}}$) as well as the metabolic (e.g. lactate threshold, LT) and gas exchange (e.g. gas exchange threshold, Tge) responses to incremental exercise. Inclined (I) compared to level (L) running increases locomotory muscle EMG at a given speed in the horse, indicative of elevated metabolic demand. To our knowledge, the effect of treadmill incline on V$\text{O}_{2\text{,max}}$, LT and Tge has not been addressed in the exercising quadruped. We used blood sampling and breath-by-breath expired gas analysis to test the hypothesis that I (10 % gradient) would increase V$\text{O}_{2\text{,max}}$ and the rate of O$_2$ uptake (V$\text{O}_2$) at LT and Tge in six Thoroughbred horses during incremental running to volitional fatigue. V$\text{O}_{2\text{,max}}$ was significantly higher for I (I, 77.8 ± 4.1; L, 65.5 ± 5.3 l min$^{-1}$; P < 0.05), but peak plasma lactate concentration was not (I, 28.0 ± 3.7; L, 25.9 ± 3.0 mM). Arterial P$_{O_2}$ increased to 62.1 ± 3.3 and 57.9 ± 2.7 Torr (I vs. L; P < 0.05), yet despite this relative hypoventilation, a distinct Tge was present. This Tge occurred at a significantly different absolute (I, 49.6 ± 3.2; L, 42.4 ± 3.2 l min$^{-1}$; P < 0.05), but nearly identical relative V$\text{O}_2$ (I, 63.6 ± 1.2; L, 63.9 ± 1.6 % V$\text{O}_{2\text{,max}}$) in I and L. Similarly, LT occurred at a significantly greater absolute (I, 47.9 ± 2.1; L, 43.9 ± 4.5 % V$\text{O}_{2\text{,max}}$), but a relative V$\text{O}_2$ (I, 37.3 ± 2.8; L, 26.9 ± 2.1 l min$^{-1}$), that was not different (I, 47.9 ± 2.1; L, 43.9 ± 4.5 % V$\text{O}_{2\text{,max}}$). In addition, Tge occurred at a significantly higher (P < 0.05) absolute and relative V$\text{O}_2$ than LT for both I and L tests. In conclusion, V$\text{O}_{2\text{,max}}$ is higher during inclined than level running and both LT and Tge in the horse occur at a similar percentage of V$\text{O}_{2\text{,max}}$. In contrast to humans, LT is a poor analogue of Tge in the horse. Experimental Physiology (2002) 87, 4, 499–506.

In healthy man, both V$\text{O}_{2\text{,max}}$ and the metabolic (i.e. blood lactate) response to exercise are influenced by O$_2$ delivery to the muscle and, to a certain extent, by the conditions of that delivery (i.e. arterial O$_2$ content, pressure and flow; Hogan & Welch, 1986; Bebout et al. 1989; Hogan et al. 1989, 1992; for review see Wagner, 2000). Specifically, a reduction in oxygen delivery (Q$_{O_2}$) to the exercising muscle amplified the reduction in phosphocreatine (PCr) and intracellular pH at submaximal workloads (Hogan et al. 1998) while also causing a downregulation of maximal force production. Acute adjustments of Q$_{O_2}$ also cause V$\text{O}_{2\text{,max}}$ to change in a coordinated fashion in accordance with the strong linear relationship between Q$_{O_2}$ and both workload (Hogan et al. 1998) and V$\text{O}_2$, as well as between the maximum oxygen delivery (Q$_{O_2\text{,max}}$) and V$\text{O}_{2\text{,max}}$ (Hogan et al. 1992). In addition, chronic adjustments to training (Musch et al. 1987), detraining (Ferreti et al. 1997) or disease (Sullivan et al. 1988) also cause changes in V$\text{O}_{2\text{,max}}$ that are linearly related to Q$_{O_{2\text{,max}}}$ and workload.

The lactate threshold (LT; i.e. the point at which an accelerated increase in blood lactate concentration is noted; Beaver et al. 1985) occurs at ~45–55 % V$\text{O}_{2\text{,max}}$ in humans, even when V$\text{O}_{2\text{,max}}$ is acutely altered due to variations in the fraction of inspired O$_2$ (F$\text{i}O_2$) or Q$_{O_2}$ (Hughson et al. 1995; Engelen et al. 1996; Koga et al. 1999). However, chronic adaptations of V$\text{O}_{2\text{,max}}$ due to training increases both the absolute and relative LT (Poole & Gaesser, 1985; Gaesser & Poole, 1986; Poole et al. 1990).

The gas exchange threshold (Tge; i.e. the inflection point of the carbon dioxide production (V$\text{CO}_{2\text{,max}}$–V$\text{O}_2$) relationship; Beaver et al. 1986b), occurs at a slightly higher percentage of V$\text{O}_{2\text{,max}}$ (~55 %) than LT in human subjects under a variety of experimental conditions (Beaver et al. 1986a,b;
Koike et al. 1990; Hughson et al. 1995). Similar to LT, only endurance training causes both the absolute and relative Tge (Casaburi et al. 1987) to increase.

In the Thoroughbred horse, LT occurs at 45–50% $V_{\text{O2, max}}$ (Langsetmo et al. 1997), which is similar to humans, despite the 2- to 4-fold higher $V_{\text{O2, max}}$ (120–180 vs. 40–70 ml kg$^{-1}$ min$^{-1}$). Tge in the Thoroughbred occurs at ~60–65% $V_{\text{O2, max}}$ (McDonough et al. 2002), which is somewhat higher than the value reported most commonly for man (Wasserman et al. 1990). This latter finding is most likely to be due to the dependence of Tge on elevated arterial $P_{\text{CO2}}$ in the horse (McDonough et al. 2002).

The above findings in both humans and Thoroughbred horses suggest that alterations in $O_2$ flow to the contracting musculature can have profound effects upon the $V_{\text{O2}}$ response and force production capability of the recruited muscle. According to this rationale, an exercise mode (i.e. inclined vs. level running) which may increase muscle $Q_{\text{O2}}$ will reduce the degree of intracellular perturbation at any given absolute $V_{\text{O2}}$. Thus, exercise on the inclined treadmill could increase $Q_{\text{O2}}$ and $V_{\text{O2}}$ compared to the level treadmill due to augmented venous return (Magder, 1995) and/or reduced afterload (all due in part to increased muscle activity or larger intrapulmonary pressure swings; Butler et al. 1993) and altered muscle activity patterns (Robert et al. 2000, 2001). However, LT and Tge should occur at a similar percentage of $V_{\text{O2, max}}$ as has been noted in humans in a study comparing arm and leg ergometry (Schneider et al. 1990).

Therefore, the purpose of this experiment was to determine $V_{\text{O2, max}}$ during level and inclined running and to analyse the relationship between $V_{\text{O2, max}}$, LT and Tge during incremental exercise on both the level and inclined treadmill. It was hypothesized that $V_{\text{O2, max}}$ would be increased on the inclined treadmill, and that the proximity of LT and Tge to $V_{\text{O2, max}}$ would be unchanged.

**METHODS**

**Animals**

Six healthy geldings (all Thoroughbreds; age, 4–10 years; body mass, 470–600 kg) were used in this study. The animals were housed in a dry lot with free access to water and salt and were fed alfalfa, grass hay and concentrate twice daily. They were dewormed and vaccinated at regular intervals and exercised on a high-speed treadmill (SATO, Uppsala, Sweden) at least twice weekly, year-round. All training sessions for this study were designed to be similar to the incremental exercise protocol described in the section ‘Experimental protocol’ below. All procedures were approved by the Kansas State University Institutional Animal Care and Use Committee.

**Animal preparation**

At least 2 months prior to beginning the experimental protocol, four of the horses had their left carotid artery exteriorized in the Kansas State University large animal clinic. This surgery is commonly performed in our clinic and is a relatively minor procedure. All animals were pretreated with ampicillin (10 mg, i.v.) and phenylbutazone (2 g, i.v.). Anaesthesia was then induced with ketamine HCl (1000–1500 mg), xylazine HCl (250–300 mg, i.v.) and guaifenesin (50 g, i.v.), after which the anaesthetic plane was maintained with halothane or isoflurane. Liberal amounts of fluid (Lactated Ringer Solution, ~2% body mass) were given throughout to prevent dehydration. Immediately post-surgery, butorphanol tartrate (10–15 mg, i.v.) and phenylbutazone (2 g, i.v.) were given to manage pain and reduce inflammation. Oral phenylbutazone (1 g per day) and sulfamethoxazole/trimethoprim (960 mg per day) were given over the next 5–10 days to prevent infection and reduce the inflammatory response. There were no post-operative sequelae in any of the experimental animals.

Immediately prior to the experimental protocol, each horse had one 7-F introducer catheter placed in the right jugular vein and one 18 gauge, 2.0 inch catheter placed either in a previously elevated portion of the left carotid artery or the transverse facial artery (20 gauge, 1.5 inch, 2 horses), using aseptic techniques. Lidocaine (2%, i.e. ~2 ml) was administered subcutaneously at the site of catheter placement for the carotid and jugular vein only. No anaesthesia was administered for the transverse facial artery cannulation because of the risk of damage to the facial nerve. A thermistor catheter was advanced through the 7-F introducer catheter into the right pulmonary artery, 8 cm past the pulmonary valve, for measurement of core body temperature (Physitemp thermocouple BAT-10, Clifton, NJ, USA). An extension tube (i.d., 1.6 mm; o.d., 3.2 mm) was connected to the arterial catheter to facilitate withdrawal of arterial blood.

**Experimental protocol**

Each horse completed two maximal runs in a randomized, cross-over design; one on the level and one on the inclined (10%) treadmill. Following collection of resting cardiorespiratory measurements and blood samples, the horses were warmed up for both protocols at a trot (3 m s$^{-1}$ for 800 m). Following this warm-up, the speed was rapidly increased to either 4 (inclined) or 7 m s$^{-1}$ (level) for 1 min, after which the speed was increased (both protocols) by 1 m s$^{-1}$ min$^{-1}$ to maximal effort as judged by an inability to keep up with the treadmill despite vigorous but humane encouragement. Horses were then cooled down (3 m s$^{-1}$) for at least 4 min. Cardiorespiratory measurements were collected continuously throughout exercise and cool-down. Blood samples were collected during the last 10 s of each stage and during minutes two and four of recovery.

**Ventilation**

Expired ventilation was measured using ultrasonic phase-shift flowmeters (Model FR-41eq; Flowmetrics-BRDL, Birmingham, UK), that have been described in detail elsewhere (Woakes et al. 1986). Briefly, a light fibreglass mask (< 1 kg) was placed on the muzzle of the horse. This mask was fitted internally with silicone rubber and foam gaskets to provide an airtight seal. The flow-tubes were then placed in the front of the mask, approximately opposite each nostril, so that airflow for each nostril could be measured. The flow-tubes were fitted with two ultrasonic transducers, which quantify the velocity of airflow at a resonant frequency of 40 kHz. In addition, the effects of temperature and gas composition upon zero stability were negated by the dual transducer design (Woakes et al. 1986). Each transducer pair was calibrated prior to each experiment according to the manufacturer’s standards (Woakes et al. 1986). Expiratory minute ventilation ($V_{\text{E}}$) was converted to standard temperature and pressure when dry (STPD) using standard equations for determination of $V_{\text{E}}$ and $V_{\text{CO2}}$ (Wasserman et al. 1987; McDonough et al. 2002). Alveolar ventilation ($V_{\text{A}}$) was calculated using the equation $V_{\text{A}} = (V_{\text{CO2}}/P_{\text{ACO2}})k$, where $k$ is 0.863 when converting from body temperature and pressure when saturated with water vapour. (BTPS; $V_{\text{A}}$) to STPD ($V_{\text{CO2}}$). $P_{\text{ACO2}}$ is arterial $P_{\text{CO2}}$. 

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Oxygen uptake and carbon dioxide production

Oxygen uptake ($\dot{V}_O_2$) and carbon dioxide production ($\dot{V}_{CO_2}$) were calculated as the product of $\dot{V}_E$ (STPD) and change in fractional $O_2$ concentration ($\Delta F_{O_2}$) ($F_{I,O_2} - F_{E,O_2}$, and correcting for $R$ value) or change in fractional $CO_2$ concentration ($\Delta F_{CO_2}$) ($F_{I,CO_2} - F_{E,CO_2}$), respectively (where subscript $E$ represents 'expired', and $R$ is the respiratory exchange ratio) after integration of the ultrasonic flow meter and mass spectrometer signals (McDonough et al. 2002). $\Delta F_{O_2}$ and $\Delta F_{CO_2}$ were determined from the difference between inspired and expired gas measured via mass spectrometry (Perkin-Elmer, model 1100, Pomona, CA, USA) by sampling from a port located on the fibreglass mask midway between the nares. $\dot{V}_{O_2,max}$ was defined in each experimental condition as a clear plateau in the $\dot{V}_O_2$–speed relationship.

Blood analysis

Following anaerobic withdrawal (~5 ml into plastic, heparinized syringes), blood samples were placed immediately on ice. After completion of the experimental protocol (within 1–2 h), arterial blood gases, pH and plasma lactate concentration were measured with a blood gas analyser (Nova Stat Profile, Waltham, MA, USA). Blood gases and pH were corrected for pulmonary arterial blood temperature (Fedde, 1991). The above measurements were performed on each occasion by a single technician in order to maintain internal consistency. Equipment was calibrated before and after each exercise test according to the manufacturer’s standards.

Threshold analyses

$T_{ge}$ was determined using a simplified version (Schneider et al. 1993) of the $V$-slope method of Beaver et al. (1986b). The method allows visual identification of the point (i.e. $\dot{V}_O_2$) where the slope of the $CO_2$ identity line departs from linearity with the slope of the $O_2$ identity line. This method is consistent with that of Sue et al. (1988), but has the additional benefit of preventing ‘pseudo-threshold’ responses due to anticipatory hyperventilation as described by Ozcelik et al. (1999).

The lactate threshold (LT) was determined by visually selecting the point where a slope change in the relationship between plasma lactate concentration and work rate occurred (Ivy et al. 1980; Poole & Gaesser, 1985; Gaesser & Poole, 1986; McDonough et al. 2002). This point was then verified by plotting the linear segments of plasma lactate concentration against $\dot{V}_O_2$ and using least squares regression analysis to choose the point of intersection, which was then recorded as LT.

Statistical analysis

A one-way analysis of variance was used to determine whether differences existed between level and inclined running for LT, $T_{ge}$ and $\dot{V}_{O_2,max}$. When significance was revealed, the point of significance was identified using the Student–Newman–Keuls post hoc test. Differences between arterial blood gases, plasma lactate concentration, arterial pH and haematocrit (Hct) at maximal exercise for level and inclined running were determined via paired $t$ tests, as were the differences in time to fatigue and the velocity at fatigue. Multiple linear regression was used for LT analysis. Statistical significance was set at $\leq 0.05$.

RESULTS

$\dot{V}_O_2$ exhibited a pronounced plateau for both incline and level conditions indicative of $\dot{V}_{O_2,max}$ (Fig. 1). $\dot{V}_{O_2,max}$ was significantly greater for I than L ($I, 77.8 \pm 4.1$; $L, 65.5 \pm 5.3$ l min$^{-1}$; $P < 0.05$). In addition, absolute LT ($I, 37.3 \pm 2.8$; $L, 26.9 \pm 2.1$ l min$^{-1}$; Fig. 2) and $T_{ge}$ ($I, 49.6 \pm 3.2$; $L, 42.4 \pm 3.2$ l min$^{-1}$; Fig. 3) both occurred at a
level of $\dot{V}_{O_2}$ that was greater for I than L ($P < 0.05$). However, when compared relative to $\dot{V}_{O_2,max}$, both LT (I, 47.9 ± 2.1; L, 43.9 ± 4.5 %; Fig. 4) and Tge (I, 63.6 ± 1.2; L, 63.9 ± 1.6 %; Fig. 5) occurred at a similar percentage $\dot{V}_{O_2,max}$. For both I and L, Tge occurred at an absolute and relative $\dot{V}_{O_2}$ that was greater than that at which LT occurred ($P < 0.05$). $P_{a,CO_2}$ at Tge was significantly greater than that at LT for both I (Tge, 49.5 ± 2.3; LT, 46.9 ± 1.7 Torr; $P < 0.05$) and L (Tge, 46.8 ± 2.1; LT, 41.5 ± 1.5 Torr; $P < 0.05$). Peak values for plasma lactate (I, 28.0 ± 3.7; L, 25.9 ± 3.0 mM), $P_{a,CO_2}$ (I, 66.1 ± 2.9; L, 68.1 ± 3.6 Torr), arterial pH (pH$_a$) (I, 7.226 ± 0.014; L, 7.257 ± 0.033) and Hct (I, 60.3 ± 1.8; L, 59.7 ± 1.6) were not different between tests, while the degree of hypercapnia ($P_{a,CO_2}$) (I, 62.1 ± 3.3; L, 57.9 ± 2.7 Torr; $P < 0.05$) was significantly greater for I than L at $\dot{V}_{O_2,max}$. Run time to fatigue was slightly but significantly longer for I (I, 697.0 ± 13.3; L, 735.0 ± 12.9 s; $P < 0.05$) and, as expected, the velocity at fatigue was much greater for L (I, 10.7 ± 0.2; L, 14.7 ± 0.2 m s$^{-1}$; $P < 0.05$).

DISCUSSION

The main findings of the present investigation are as follows. $\dot{V}_{O_2,max}$ was increased by ~20% for I compared with L. In addition, while $\dot{V}_{O_2}$ at LT, Tge and maximal exercise was significantly greater for I, LT and Tge occurred at a similar percentage of $\dot{V}_{O_2,max}$ for I and L. Also, Tge occurred at a significantly greater absolute and relative $\dot{V}_{O_2}$ than LT in the Thoroughbred horse. The finding that Tge occurs at a significantly greater absolute and relative $\dot{V}_{O_2}$ than LT in the Thoroughbred horse, confirms prior research from our laboratory (McDonough et al. 2002). The following three sections summarize the experimental conditions (acute and chronic) under which $\dot{Q}_{O_2}$ to the muscle has been altered as a necessary prelude to exploring the mechanism(s) underlying the results presented here.

Effects of endurance training on $\dot{V}_{O_2,max}$, LT and Tge

Aerobic training increases $\dot{V}_{O_2,max}$, LT and Tge (Poole & Gaesser, 1985; Casaburi et al. 1987; Tyler et al. 1996; Carter
et al. 2000; for review see Poole, 1997), while detraining reduces $\dot{V}O_{2,max}$ (Saltin & Gollnick, 1983; Tyler et al. 1996) and LT (Wasserman et al. 1973). In addition, training causes the relative LT and Tge to increase (Poole & Gaesser, 1985; Casaburi et al. 1987). These findings suggest that both alterations in oxygen delivery ($QO_2$) and the capacity to utilize oxygen are either responsible for, or co-vary with the changes in $\dot{V}O_{2,max}$, LT and Tge following training. Endurance training increases whole body $QO_2$ by increasing cardiac output at maximal exercise, this effect being manifested primarily through an augmented stroke volume (Saltin et al. 1968; Musch et al. 1987). In addition, mitochondrial volume is increased with training (Baldwin et al. 1995), a reduction in $\dot{V}O_{2,max}$ of approximately 11–12%, such that relative Tge was increased. Hammond et al. (1992) performed a pericardiectomy on dogs and noted that maximal cardiac output (~15%; due mainly to augmented stroke volume) and $\dot{V}O_{2,max}$ (~20%) were significantly increased. Hammond et al. (1992) performed a similar experiment on pigs (which more closely resemble humans in terms of mass-specific $\dot{V}O_2$ and cardiac structure–function relationships). The two groups reported similar findings in that cardiac output (29%) and $\dot{V}O_{2,max}$ (31%) were both increased substantially following pericardiectomy.

3. Body position. Koga and co-workers reduced leg blood flow by having subjects exercise in the supine position (Koga et al. 1999). They noted that $\dot{V}O_{2,max}$ and absolute Tge were reduced by 11–12%, such that relative Tge was unchanged.

4. O$_2$-carrying capacity of the blood. Wagner and colleagues (1995) found that O$_2$ delivery was reduced in splenectomized horses during exercise. This procedure reduced $\dot{V}O_{2,max}$ (by ~24%), O$_2$ delivery (by ~28%), muscle diffusing capacity for O$_2$ (by ~10%) and LT (by ~15–20%). Thus, in a variety of species, and under different experimental conditions, there is direct evidence that $\dot{V}O_{2,max}$ and $QO_2$ are closely related.

Effect of altered muscle recruitment

Another method of altering O$_2$ delivery is by performing modes of exercise that recruit varying amounts of the available muscle mass. In the experiments of Koga and colleagues (2001), subjects performed both one- and two-legged exercise on a specially designed cycle ergometer that was designed to alter the recruited muscle mass and the $\dot{V}O_{2,max}$ response to exercise. They noted that $\dot{V}O_{2,max}$ and the $\dot{V}O_2$ at Tge were increased by ~50% for two-legged cycling, but the relative Tge was unchanged. Thus, altering the recruited muscle mass during a particular exercise mode changes $\dot{V}O_{2,max}$ (Hermansen & Saltin, 1969) and the absolute Tge, such that the relative Tge remains unchanged.

Another method of altering O$_2$ delivery through altered muscle recruitment is to compare arm and leg ergometry. $\dot{V}O_{2,max}$ and absolute LT are significantly reduced during arm ergometry, but relative LT is unchanged (Schneider et al. 2000). Therefore, in agreement with the results of Koga et al. (2001), altering the recruited muscle mass during exercise with a similar duty cycle (60 r.p.m.), $\dot{V}O_{2,max}$ and LT...
Mechanistic basis for elevation of $\dot{V}O_{\text{max}}$ and constancy of relative LT and Tge

While it is possible that the level running protocol may have limited the maximal attainable $\dot{V}O_2$, due to the high frequency of muscle contraction (Hermansen & Saltin, 1969), the following scenario could be used to explain the increased $\dot{V}O_2$ at LT, Tge and at maximal speed on the inclined treadmill in the Thoroughbred horse. First, cardiac output is increased on the incline. Indeed, we have preliminary information confirming that cardiac output is increased by ~22% on the inclined treadmill ($n = 3$; I, 333.8 ± 23.7; L, 273.2 ± 26.6 l min$^{-1}$; $P < 0.05$; authors’ unpublished data), in line with the 19% increase in $\dot{V}O_{2,\text{max}}$ noted in the present study. In addition, $V_t$ and tidal volume ($V_t$) were augmented by a similar degree (~25 and 15%, respectively, authors’ unpublished data). Furthermore, Robert et al. (2000) noted that stride duration (i.e. the percentage of time spent on the treadmill as measured for the left hindlimb) was increased as a function of treadmill slope and the integrated EMG was increased at nearly equivalent workloads on the inclined treadmill (Eaton et al. 1995). Thus, it is plausible that altered muscle recruitment patterns may result in an increased venous return (due to the augmented action of the muscle pump (Magder, 1995) and the respiratory pump (Harms et al. 1998)), thereby increasing cardiac output via the Starling mechanism. These adjustments on the incline would serve to increase $O_2$ delivery to the working musculature allowing an augmented absolute $\dot{V}O_2$ at LT and Tge and also $\dot{V}O_{2,\text{max}}$. However, as mitochondrial and capillary volume densities are unaltered through changes in treadmill slope, LT and Tge occur at the same relative $\dot{V}O_2$ for both I and L. Indeed, prolonged training is needed to increase the relative LT and Tge, as adaptations in $\dot{V}O_{2,\text{max}}$ plateau after 4–8 weeks of training, while mitochondrial enzyme activity levels continue to increase (Saltin & Gollnick, 1983).

Conclusions

The current study demonstrates that running the Thoroughbred horse on a 10% incline increases $\dot{V}O_{2,\text{max}}$ substantially (~20%). This acute elevation of $\dot{V}O_{2,\text{max}}$ for I in the Thoroughbred horse increased the absolute $\dot{V}O_2$ at LT and Tge, but not the relative LT or Tge, such that both occurred at a similar percentage $\dot{V}O_{2,\text{max}}$ for both I and L. This has important implications for exercise testing in the Thoroughbred horse in that comparisons of $\dot{V}O_{2,\text{max}}$, $QO_{2,\text{max}}$ and $\dot{V}O_2$ at LT and Tge between different exercise trials should take treadmill inclination into account.


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