Exercise performance in acute and chronic cold exposure

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Abstract

This review focuses on the suppression of physical performance in a cold environment and the underlying physiological mechanisms. There are many situations where humans have to perform physical activities in a cold environment. Cold environments often limit exercise and working performance by impairing functions such as force production, velocity, power and manual dexterity. A muscle temperature of around 27°C is assumed to be a critical temperature below which maximal voluntary isometric force starts to decrease. The endurance time of submaximal isometric contractions peak at muscle temperatures of 27 to 28°C and decrease rapidly above and below these temperatures. Dynamic exercise performance, especially fast velocity movement, is generally more disturbed by cooling than isometric contractions. Additionally, the effect of cold adaptation on exercise performance, and the potential related mechanisms are summarized here based on a limited number of studies. Since the involuntary muscle contraction of shivering disturbs fine motor control, habituation of shivering, which is an example of cold adaptation, potentially improves exercise performance. Higher hand skin temperatures, induced by greater cold induced vasodilatation after local cold adaptation, could improve manual dexterity. Since there have been few studies testing the effect of cold adaptation on exercise performance in a cold environment, further studies seem warranted.

Introduction

Humans are exposed daily to various climatic conditions, from mild to extreme hot and cold, and sometimes experience meteorological disasters like cold snaps, heavy snow fall, flooding and tsunamis, which results in cold-associated, sometimes fatal, health conditions. In the case of accidental cold water immersion, the initial drop of skin temperature induces “cold shock” responses including hyperventilation, gasping and tachycardia[1]). These responses peak in the first 30 seconds, and are probably responsible for the majority of drowning deaths[1]). Following skin cooling, incapacitation caused by a reduction in temperature of superficial nerves and muscle, can impair performance and even result in death before hypothermia. The impairment of muscle performance of the hypothermic skeletal muscle induces swimming failure[2]) and drowning. Even in recreational or competitive sports events in a cold environment like skiing, snowboarding, diving and long distance swimming, exercise performance can be restricted by the cold. In addition, decreased performance due to a reduction of the superficial tissue temperature is commonly experienced by workers in cold workplaces like the food industry, cold storage, fisheries, forest industry, Coast Guard and military, especially in cold countries in the winter season[3,4]).

There are many situations where humans have to perform physical activities in a cold environment. Cold environments often limit exercise and work performance by affecting force production, velocity, power and manual dexterity[3,5]). Chronic cold exposure and repetitive work might lead to cold-related health problems like musculoskeletal pain, Raynaud's phenomenon (recurrent vasospasm in the fingers and toes), and, on some occasions, non-freezing cold injuries and frostbite[4,6]). On the other hand, the benefits of cooling on exercise performance have been studied especially for prolonging endurance exercise in the heat, for example, pre-cooling or cooling between exercise sessions[7-9]). The major focus of this review is the cold-induced impairment of exercise performance, excluding these possible positive effects. Additionally, the greater cold-induced vasodilatation (CIVD) response observed in people working daily in cold environments[10-12]), has been suggested to be beneficial for preventing cold injuries and improving manual dexterity in the cold[13]).

This review article focuses on the impairment of physical performance in cold environments and underlying physiological mechanisms. Additionally, the effect of cold adaptation on exercise performance and related potential mechanisms are summarized based on a limited number of studies.

Exercise performance in a cold environment

The impairment of exercise performance in hypothermic skeletal muscle has been studied and well summarized in several review articles[5,14-16]). In this review, the exercise performance of static isometric contraction, dynamic exercise, and the more practical swimming exercise are summarized below.

Isometric contraction

The classic studies of Clarke et al.[17]) reported a small or no effect of muscular temperature on maximal voluntary contraction (MVC) during isometric handgrip within the muscle temperature (Tm) range of 27 to 40°C, but a decrease of 60% when Tm reached around 20°C. Similarly, in the first dorsal interosseous muscle, MVC was relatively constant, or even greater, within the range of 25 to 35°C, but decreased by about 30% when T_m was 12 to 15°C. Others have reported a small decrease of MVC during isometric knee extension (2.1% MVC per °C muscle temperature reduction) within the range of 30 to 39°C[18]). Several studies reported a reduction in MVC when T_m dropped below 27°C[17,19]). While there is some variation between studies, a muscle temperature of around 27°C is assumed to be a critical temperature for performing maximal isometric voluntary contraction[5,16]).

Meanwhile, endurance performance follows a bellshaped pattern with muscle temperature. The endurance time of submaximal isometric contraction peaks at a T_m of 27 to 28°C and decreases rapidly above and below these temperatures[17,20]). When T_m, measured at a 2 cm depth of the brachioradialis, was decreased to 23°C or elevated to 38°C, the duration of sustained hand grip contraction (33% MVC) was shorten by around 60% of the peak observed at 27°C17). It was suggested that at a T_m below 27°C superficial muscle fibers do not contract well, and fewer fibers located more centrally have to generate the same force. When Tm increased above 27°C, the increase in metabolic rate caused earlier fatigue17).

Dynamic exercise

Bergh and Ekblom18) measured the peak torque of knee extension at the different angular velocities of 0° (isometric), 90° and 180°·sec^{-1}, within a Tm range of 30 to 39°C. The decrease in peak torque per °C reduction of T_m was greater in dynamic conditions (4.7%·°C-1 at 90°·sec^{-1} and 4.9%·°C^{-1} at 180°·sec^{-1}) than in an isometric condition (2.1%·°C-1 at 0°· sec-1). It was suggested that dynamic exercise performance, especially fast velocity movement, is generally more disturbed by cooling than isometric contractions5,14,18).

Maximal 20-sec sprint efforts at 95 revolutions per min on an isokinetic cycle ergometer were tested under different muscle temperature conditions following leg immersions21). When T_m was reduced to 31.9 and 29.0°C, the maximal peak force was reduced by 12 and 21%, respectively, compared to a non-cooling condition at a Tm of 36.6°C; this corresponded to a 2-3% reduction per °C fall in T_m 21). The height of a maximal vertical jump was decreased with a Tm reduction within the range of 30 to 39°C at the rate of 4.2%·°C^{-1} 18). Similarly, the flight time, force production during shortening phase and take-off velocity of maximal drop jump were decreased in a dose-dependent manner with degree of cooling22). It was suggested that very fast movements like the drop jump are especially susceptible to cooling14). Sargeant21) tested exercise performance at different pedaling velocities, and found a T_m dependence on the optimum pedaling rate for maximal power production. This velocity dependence of performance in cold was also reported in a ball throwing exercise23); cold-induced impairment was greater with lighter balls (faster movement) than with heavier balls (slower movement).

Time to exhaustion during short-term intense leg cycling exercise was reduced by 38% when leg muscles were cooled to 29°C, compared to a warm condition of a T_m of 34°C24). Similarly, the endurance performance of intense dynamic exercise was impaired by cooling25,26).

Swimming

Swimming in cold water can significantly impair exercise performance; the greater thermal conductance of water quickly decreases body temperature. Swimming needs coordination of the dynamic movement of different body parts, proprioceptive feedback and tactile sensation for perceiving motion in the water. These functions are also impaired by cooling. In the well-known studies on the metabolic response to swimming in varying water temperatures (18, 26 and 33°C), greater oxygen uptake for a given submaximal swimming speed (less efficient swimming) was observed in colder water27,28). It was suggested that the additional energy expenditure was attributable to shivering and lower mechanical efficiency due to impaired neuromuscular function in cold limbs. Additionally, lower oxygen uptake during maximal swimming was observed in lean participants[27,28]). The details of swimming failure (stroke parameters and efficiency) were analyzed during breaststroke swimming in cold water at 10, 18 and 25°C, for a maximum of 90 min[2]). A more frequent stroke rate (number of strokes in a minute) and shorter stroke length (distance swum per each stroke) were observed in 10°C water than in warmer water. Impaired swimming performance can partly explain the greater oxygen consumption and diminished swimming efficiency (meters swum per liter of oxygen consumption) in 10°C water.

Mechanism of performance impairment in a cold environment

The reduction in hypothermic skeletal muscle performance described is, in part, attributable to slowing of the nerve conduction velocity[29]). Reduction of the muscle contraction velocity is partly explained by slowed adenosine triphosphate (ATP) utilization[30,31]), slowed Ca^{2+} release and uptake from the sarcoplasmic reticulum[32,33]). In this section, the potential mechanisms of impairment of exercise performance in a cold environment are summarized based on studies focusing on neuromuscular functions and muscle metabolism.

Neuromuscular function

The neuromuscular function of hypothermic skeletal muscle has been studied using electromyography (EMG). Several studies have reported decreased amplitude of EMG due to cooling[34-36]), while others have reported increased amplitude[37,38]). The discrepancy could be explained by different exercise types or cooling procedures. During a brief biceps contraction at 30% maximum voluntary contraction (MVC), greater amplitude was observed when the upper arm was moderately cooled by a 20°C water circulating cuff, whereas, the amplitude was reduced when water temperature was reduced to 0°C[39]). When muscle was cooled from the skin surface, relatively cooler superficial muscle fibers did not contract well, and warmer fibers located more centrally with the muscle compensated for the required force. Thus, the reduction of the amplitude of surface EMG might reflect the lower activity of superficial muscle fibers40). On the other hand, the increase of amplitude may indicate that more muscle fibers are recruited to maintain the given work load level[41]). It has been reported that faster muscle fibers in swimming carp are recruited at relatively lower velocity in low temperature conditions to maintain swimming speed[42,43]). The maximal shortening velocity of rat fast (extensor digitorum longus) and slow (soleus) twitch fibers in vitro has been examined at temperatures between 35 and 10°C[44]). It was found that slow twitch fibers showed a greater decrease in the shortening velocity per °C reduction of T_m than fast twitch fiber. Because of the greater cold sensitivity and lower power output of slow twitch fibers in a cold environment, less cold sensitive and more powerful fast twitch fibers are recruited earlier. Therefore, to generate the muscle power to maintain work load, a greater number of fast twitch fibers need to be recruited in cold compared to a normothermic condition. This results in a greater amplitude of EMG.

A shift in the EMG frequency to lower frequencies with lower muscle temperatures has been reported more uniformly[22,34,35,37,39,45]). The shift has been connected with the decrease in muscle conduction velocity in the cold[39,46]). Since the reduction of conduction velocity with muscle cooling paralleled the reduction in the median of EMG frequency, the shift to lower frequencies would reflect a change in muscle action potential conduction velocity[35]). Since a similar reduction of EMG frequency was observed in fatigued muscle, the mechanism of the lower EMG frequency in cold was explained with the fatigue-induced 'muscular wisdom' hypothesis[15]). Marsden et al.[47]) developed the 'muscular wisdom hypothesis' that was a phenomenon of the decrease in the motor unit discharge rate during continuous muscle contraction for minimizing fatigue. Since fast twitch fibers, which are less involved in endurance activity, are recruited earlier in the cold[42,43]), it is suggested that optimizing the firing frequency is beneficial for fatigue resistance[15]).

There are only a few studies examining the co-contraction of the agonist and antagonist muscle pair after cooling[22,23]). During the concentric phase of muscle contraction, the EMG activity of the antagonist muscle is increased by cooling, whereas activity of the agonist is suppressed. This phenomenon called the 'braking effect' has been suggested as one reason for the impairment of exercise performance[14,16]). Similarly, involuntary shivering of the antagonist muscles can disturb control of motor activity in a cold environment[48-50]). Hong and Nadel[51]) tested the effect of exercise intensity on the shivering of neck muscles which were not involved in exercise. The slope relationship of neck muscle EMG activity to esophagus temperature was suppressed by increasing exercise intensity.

Muscle metabolism

Abramson et al.[52]) examined resting local oxygen uptake and blood flow in human forearm at a T_m within the range of 25 to 40°C. The oxygen uptake was calculated using the Fick principle on blood samples from the forearm vein, and forearm blood flow was measured by plethysmography. Lower local oxygen uptake and forearm blood flow was seen in cold muscle. The suppression of muscle metabolism in a cold environment could partly be explained by a reduction of oxygen supply by blood flow restriction in the cold[53-55]). Thorsson et al.[53]) reported a reduction of intramuscular blood flow (^{133}Xe clearance technique) after local cooling of the quadriceps by applying cold packs. Rennie et al.[55]) showed a similar reduction of muscle blood flow during exercise in cold water.

There have been large numbers of studies measuring oxygen uptake in the lungs to evaluate the effect of cold muscle temperature on the metabolic response. Temperature dependence on oxygen uptake kinetics at the start of exercise has been studied. Shiojiri et al.[56]) found a significantly greater time constant (the duration to the end of phase 1, defined as inflection points in respiratory exchange ratio, end-tidal PO_2 and end-tidal PCO_2 [57])) after onset of moderate (50 W) cycle exercise under a cold muscle condition (vastus lateralis T_m = 30.2°C) than in a neutral condition (T_m = 36.8°C). On the other hand, Ishii et al.[58]) reported no difference between cold (vastus lateralis T_m = 28.0°C) and neutral (T_m = 35.5°C) conditions in oxygen uptake kinetics (half-times of oxygen uptake kinetics) at the onset of cycle exercise (75 W and 125 W). In the cold conditions of both studies, rectal temperature was also decreased before starting exercise, which would induce shivering. Significantly greater oxygen consumption before starting exercise was observed in the cold conditions of the study by Ishii et al.58), whereas Shiojiri et al.[56]) reported similar resting oxygen consumption in both temperature conditions. Thus, in the work of Ishii et al.[58]) a deviation in the initial level of oxygen consumption due to shivering may have masked the difference in oxygen kinetics between muscle temperature conditions. Recently, the kinetics of intracellular oxygen pressure (PO_2) following the onset of contraction was evaluated in isolated single Xenopus skeletal muscle fibers maintained at different temperatures[59]); a greater time constant and delayed onset for the decline of intracellular PO_2 was observed in cold muscle (T_m = 15.4°C) than in muscle kept in a neutral (T_m = 20.5°C) or hot condition (T_m = 25.9°C).

It was suggested that developing techniques of measuring local oxygen uptake was essential in order to study human muscle metabolism over a large range of T_m [60]). Recently, several studies directly measured the temperature dependence of muscle metabolism using near infrared spectroscopy (NIRS)[61-63]). It was reported that oxygen consumption of human forearm at rest and during 4% MVC isometric handgrip (by measuring the slope of deoxyhemoglobin change during 20-sec arterial occlusion) decreased as a function of reduction in T_m from 36 to 26°C[61]). Using the NIRS technique, a significant reduction of total hemoglobin level and a tendency for lower muscle oxygenation were observed in resting human ankle dorsiflexor muscles cooled from the skin surface63). Hom et al.[62]) also reported a decrease in resting muscle oxygen saturation and total hemoglobin levels after 1-hour cooling with an ice bag.

More recently, diffusion-weighted magnetic resonance images were used to evaluate the effect of cooling on intramuscular water movement, including water diffusion and microvascular circulation in the capillary network[9,64,65]). It was reported that resting intramuscular water diffusion and perfusion were decreased by local cooling[64]), and the increased intramuscular water movement after exercise showed greater recovery after local cooling treatments than in a non-cooling condition[65]). This magnetic resonance technique could be applied to directly evaluate microscopic water movement in hypothermic skeletal muscle, which is associated with muscle metabolism.

Cold adaptation and exercise performance

Cold adaptation of human thermoregulatory response has been studied extensively, and there have been several categorizations based on the differences in thermoregulatory response following a period of adaptation[66-70]). In this section, the effect of repeated cold exposure on exercise performance in a cold environment and potential related mechanisms are summarized based on a limited number of studies.

Habituation of shivering

It has been reported that shivering can disturb fine motor control because of the co-contraction of the agonist and antagonist muscles with the superimposition of shivering on exercise in a cold environment[48-50]). Thus, the habituation of shivering (less shivering response after cold adaptation) could improve fine motor control in the cold. There are a lot of studies reporting the habituation of shivering response after repeated exposure to a cold environment[71-73]). However, only a limited number of studies tested the influence of adaptation on exercise performance.

Makinen et al.[74]) found a significant reduction of postural sway with a significant increase in mean skin temperature (0.4°C) over 10 days repeated exposure to 10°C air for 2 hours per day. However, since the postural sway at 10 and 25°C ambient temperature conditions improved in parallel, it was concluded that this improvement was attributed to motor skill learning and the repeated cooling had no effect on postural control[74]). During cold exposure to 10°C air, muscle tone measured by EMG was increased by 140-260% (visible shivering in some participants); but no habituation was observed over the 10-day exposure period. The temperature condition of 10°C air might not have been sufficient to habituate shivering. If the environmental condition was much colder, inducing strong shivering, an improvement of neuromuscular performance might be observed with the habituation of shivering. A recent study also reported that athletes who practiced in cold weather had no improvement in their manual dexterity, despite the smaller reduction in their finger skin temperature during cold exposure to 5°C air[75]).

Research has examined neuromuscular adaptation after repeated 2-min whole-body exposure to extremely cold air (-110°C) for 3 months[76]). At the first trial before the adaptation, the flight time of a drop jump was significantly shortened by a single 2-min cold exposure, but this impairment of performance disappeared after the repeated exposure to cold air. Based on the simultaneously measured EMG data, the improvement in performance was explained by the pronounced increase in activity of the agonist muscle (gastrocnemius medialis) during the shortening phase of the drop jump after repeated cold exposure; no change was observed in the antagonist (tibialis anterior). The diminished co-contraction of the agonist and antagonist muscles[48]), or braking effect, by the increased antagonist muscle activity[22,23]) in a cold environment, might improve the drop jump performance. Additionally, maximal voluntary contraction (MVC) of isometric wrist flexion was evaluated with the agonist and antagonist EMG analysis. The averaged

EMG activity of both the agonist and antagonist muscles was significantly increased following the 2-min single cold exposure after the 3-month repeated exposure to cold. Westerlund et al.[76]) commented that an increase in EMG activity tended to be greater in the agonist and less in the antagonist, which is similar to the reduced co-contraction during drop jump after repeated cold exposure. However, wrist flexion exercise performance did not change significantly either following a single 2-min cold exposure or after repeated exposures. The discrepancy between the results was suggested to be due to the difference of muscle contraction type (isometric or dynamic), since the maximal isometric force was relatively stable when the muscle temperature was over 27°C[17]) and the impairment of muscle performance in the cold was greater during dynamic than static exercise[5,14,26]).

Enhancement of cold induced vasodilatation

During cold-water immersion of extremities, higher finger skin temperature, more rapid cold induced vasodilatation (CIVD) and enhanced blood flow to the extremities are generally, but not always[77-80]), observed in populations living and working in a cold environment[10-12]) and also after repeated cold exposure in laboratory controlled studies[81,82]). The greater CIVD response was suggested to be beneficial for improving manual dexterity and tactile sensitivity in the cold[13]). Manual dexterity is affected by finger skin temperature[83]) or finger blood flow[84]) depending on the temperature and duration of the task. Krog et al.[10]) reported that the impairment of grip strength and finger tapping speed following 30-min hand immersion in 0°C water tended to be smaller in Norwegian Lapps and fishermen than in control groups. In their study, cold-adapted groups showed a significantly rapid onset of CIVD, however, no difference was observed in maximum and minimal finger skin temperature compared to a control group. Thus, it was not clear whether the manual dexterity of cold-adapted populations were affected by habituation of local vasoconstrictor response.

In a series of laboratory controlled studies[79,81,85]), local cold acclimation on neuromuscular functions was tested after repeated hand immersion in 8°C water for 30 min, 5 days a week for 2 to 3 weeks. No improvement in manual dexterity, grip strength, voluntary and evoked twitch force of the first dorsal interosseus muscle was observed after cold immersion[79,81,85]), even with a greater CIVD response after the repeated cold water exposure[81]). Concerning these observations, there seems to be no significant effect of repeated local cooling on exercise performance in cold, at least in the conditions that were tested in these studies[79,81,85]).

Muscle metabolism

Savourey et al.86) examined the effect of repeated local cold immersion of hand and forearm on skeletal muscle metabolism. After local cold adaptation by repeated 5-min water immersion (5°C) of the arm twice a day, 5 days a week for 2 months, finger skin temperature was kept higher during the immersion, but no adaptive change of muscle metabolism, measured by ^{31}Pnuclear magnetic response, was observed during water immersion and during 10% MVC handgrip exercise after the immersion. Since the duration of each immersion (5 min) was too short to induce a significant reduction of Tm and muscle metabolism, the experimental protocol and small number of subjects (n=5) may have been insufficient to induce adaptive change in muscle metabolism or identify any such change.

Muller et al.[87]) reported that cold weather athletes, who practiced American football in cold air (0°C on average), showed a greater exercise economy (smaller oxygen uptake for a given submaximal workload) during bicycle exercise in 5°C air compared to a physically active control group. It was speculated, without supporting data, that improved buffering of lactate or utilization of different muscle fiber types could be a mechanism underpinning the greater exercise economy. The following are potential mechanisms which might induce cold adaptive changes in muscle metabolism.

The distribution of muscle fiber type was assessed in Korean diving women who routinely exposed themselves to cold water[88]). Divers had a greater percentage of type IIx (fast glycolytic) and lower proportion of type IIa (fast oxidative glycolytic) fibers in the vastus lateralis than physically active controls, whereas, no group difference was observed in the percentage of type I (slow oxidative) fibers. This result suggested that repeated cold-water immersion might induce the shift of type II muscle fibers to the faster subgroup. It was probably because the faster types of muscle fibers were recruited at a relatively lower velocity in low temperature conditions to maintain the required activity[42,43]). A similar shift in fiber type from type I to type IIa fibers was observed in rat soleus muscle (predominantly Type I) after intermittent cold exposure[89]). Since animal studies reported that the shift of muscle fiber type composition after repeated cold exposure was specific to the predominant fiber type[89,90]), variation in the cold adaptive change in the different muscle groups should be considered.

Another potential mechanism for improving muscle metabolism after cold adaptation is an increment of oxygen delivery caused by an increase in capillary density. Bae et al.[88]) reported that Korean diving women had a significantly greater capillary density and number of capillaries per fiber of the vastus lateralis compared to that of an active control group. In animal studies, chronic cold exposure has been reported to induce a growth of capillary density and/or the capillary to fiber ratio in rats[91,92]) and in guinea pigs[93]). Deveci and Egginton[94]) considered muscle group specificity for angiogenesis (growth of capillary) after repeated cold exposure. Since a significant increase in the capillary to fiber ratio was observed only in the soleus of rats, and not in the tibialis anterior, the greater oxidative capacity and muscle activity was suggested to be a key factor controlling cold-induced angiogenesis[94]). An increased oxygen supply to muscle tissues by microvascular remodeling might be a potential mechanism for improving muscle metabolism.

Recently, several research groups have focused on the adaptation of non-shivering thermogenesis (NST) in human brown adipose tissue (BAT) after repeated cold exposure[95-98]). Skeletal muscle potentially contributes towards NST, since it constitutes up to 40-50% of total body mass and contains large numbers of mitochondria. There was a significant positive relationship between an increase in total daily energy expenditure during mild cold exposure (16°C air for 48 hours) and an increase in mitochondrial uncoupling (state 4 respiration) of isolated human skeletal muscle biopsies taken after cold exposure99,100). However, a more recent study reported no significant contribution of human skeletal muscle mitochondrial uncoupling to an increase of NST after repeated mild cold exposure[97]). Concerning these observations, acute mild cold exposure would activate mitochondrial uncoupling in skeletal muscle, though, it seems to be insufficient to induce an adaptive change that enhances NST in the skeletal muscle after chronic cold exposure.

Further investigation is required to clarify the effect of chronic cold exposure on adaptation in exercise performance and physiological mechanisms.

Conclusion

This article reviewed the impairment of physical performance in a cold environment and underlying physiological mechanisms. A muscle temperature of around 27°C is thought to be a critical temperature below which maximal isometric voluntary force starts to decrease; the endurance time of submaximal isometric contraction peaks at a muscle temperature of 27 to 28°C and is decreased below and above the temperature. Dynamic exercise performance is generally more disturbed by cooling than isometric contractions.

Decreased nerve conduction velocity and slowed ATP utilization could be potential mechanisms for impaired exercise performance. When skeletal muscle is cooled, a shift in the EMG frequency to lower frequencies has been uniformly reported, whereas the amplitude of EMG decreased or increased depending on the study. More muscle fibers, especially the faster type, are recruited in low temperature conditions to maintain a given workload. Several studies reported a reduction in resting muscle metabolism in the cold, but studies of exercising muscle in the cold are scarce.

Habituation of shivering and enhanced CIVD after repeated cold exposure could be hypothesized to improve exercise performance in a cold environment. Further investigation has to be carried out to clarify the effect of chronic cold exposure on adaptation in exercise performance and physiological mechanisms.

Conflict of Interests

The authors declare that there is no conflict of interests regarding the publication of this article.

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