Review

Protective Facemask Impact on Human Thermoregulation: An Overview

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The use of protective facemasks (PFMs) negatively impacts respiratory and dermal mechanisms of human thermoregulation through impairment of convection, evaporation, and radiation processes. The relatively minor reported increases in core temperature directly attributable to the wearing of PFMs suggest that associated perceptions of increased body temperature may have a significant psychological component or that regional or global brain temperature changes are involved. Modifications in PFM structure, components, and materials might allow for improved heat dissipation and enhanced compliance with use.

Keywords: comfort; core temperature; PFMs; thermoregulation; tolerance

INTRODUCTION

The spate of serious viral respiratory infectious agent outbreaks (e.g. severe acute respiratory syndrome, avian influenza, and pandemic influenza) has placed significant impetus upon the use of protective facemasks (PFMs), including filtering facepiece respirators (FFRs), surgical/medical facemasks (FM), and elastomeric air-purifying respirators (EAPRs) by healthcare workers (HCWs) and the public. The most commonly employed PFMs in these situations are FFRs and FMs. FFR are tight-fitting particulate respirators with a filter as an integral part of the facepiece or with the entire facepiece composed of the filtering medium that covers at least the mouth and nose and filters out harmful particles (NIOSH, 2004). FMs are loose-fitting disposable masks that cover the nose and mouth and are referred to by various nomenclatures, such as surgical mask, medical mask, procedure mask, dental mask, and laser mask.

FFRs were initially introduced into surgery to not only prevent surgical personnel from contaminating the surgical field with respiratory droplets expelled during speaking, coughing, and sneezing but also protect the wearer from splashes or sprays (TFAH and AAP, 2009). Because of their loose fit, FMs are unable to provide a high degree of protection from airborne particulates of small dimensions (i.e. droplet nuclei) that can harbor pathogens (Oberg and Brosseau, 2008). EAPRs are reusable, air-purifying respirators (APR) with facepieces made of pliable materials (e.g. silicone, rubber, and plastic) that employ one or two particulate cartridge filters and come in full facepiece or half-mask models (Roberge et al., 2010d). Although there is currently some ongoing debate and investigation into the relative merits of FFR versus FM in protecting the wearer from pathogens (Loeb et al., 2009; Srinivasan and Perl, 2009; Gralton and McLaws, 2010), there is less controversy regarding their being of some efficacy in preventing the transmission of respiratory pathogens (Cowling et al., 2009; MacIntyre et al., 2009; Aiello et al., 2010). However, the use of PFMs will not be effective if not used appropriately.

One of the more frequently cited reasons for intolerance and associated lack of compliance with appropriate PFM use is the discomfort related to buildup of facial heat (Jones, 1991; Laird et al., 2002; Radonovich et al., 2009). In a recent study (Baig et al., 2010),
increased facial heat was experienced ‘frequently-to-always’ by 56% of HCWs wearing N95 FFR. PFM-associated facial heat complaints may represent any of a variety of effects, including local dermal effects, increased temperature of breathing air, elevated core temperature, or psychophysiological responses. This review will examine the etiology of PFM-associated increases in the body’s heat perception and burden and suggest potential mitigation strategies.

METHODS

A computerized literature search was undertaken for the period 1950–2010 with the search engines Medline®, OvidSP®, EMBase™, PsycINFO®, Compendex®, and Google®. A web-based search of relevant electronic references was also performed and the bibliographies of selected articles and textbooks were perused for pertinent articles (Fig. 1). References selected for inclusion in the review were those that included information relating to heat, comfort, and tolerance associated with the use of PFMs.

RESULTS

A total of 195 articles from the literature was retrieved along with 42 web-based relevant articles and one textbook chapter. Of these, 84 literature references serve as the database for this study, including 80 journal articles, 3 electronic references from medical, governmental, and news agency sources, and 1 book chapter. There is a paucity of data available on the influence of PFMs upon body thermoregulation.

DISCUSSION

The genesis of PFM-associated changes in body temperature is a composite of several inputs of variable prominence that includes respiratory heat exchange mechanisms, the impact of nasal versus oral respiration, metabolic cost and thermal load of PFMs, facial skin heat load of PFMs, ambient climate and PFM microclimate (i.e. PFM dead space) heat and humidity, and psychophysiological heat response components.
Respiratory heat exchange mechanisms

Excess heat generated by the body’s metabolism and transferred from environmental heat sources (e.g. radiation) must be released to the surrounding environment in order to maintain thermal homeostasis. While human heat balance can be conceptually explained in various forms, the following heat balance equation, re-written from Parsons (2003) provides a practical approach for its estimation:

\[
S = M - W - \left( C + R + E_{sk} \right) + \left( E_{res} + E_{res} \right),
\]

where \( S \) = rate of heat storage (W·m\(^{-2}\)), \( M \) = rate of metabolic energy production, \( W \) = rate of the body’s mechanical work, \( C \) = rate of convective heat loss from the skin, \( R \) = rate of radiative heat loss from the skin, \( E_{sk} \) = rate of evaporative heat loss from the skin, \( E_{res} \) = rate of convective heat loss from respiration, and \( E_{res} \) = rate of evaporative heat loss from respiration. Thus, the body achieves heat balance when \( S \) equals zero. As a point of interest, heat exchange (loss) through respiration consists of two components: convective heat loss as a function of cool air inhalation in which heat from the lungs is transferred in exhalation (\( C_{res} \)) and evaporative heat loss as a function of moisture saturation in exhaled air (\( E_{res} \)). In practice, the amount of respiratory heat loss can be quantified using the following equation (Parsons, 2003):

\[
C_{res} + E_{res} = (0.0014 M [34 - T_a] + 0.0173 M [5.87 - p_a]),
\]

where \( T_a \) = ambient temperature (°C) and \( P_a \) = ambient pressure (kPa). Under thermoneutral environmental conditions, inspired air is warmed and saturated in the lungs which are generally at core temperature, but exhaled air temperature is lower (e.g. 34–35°C, Tozer, 1924; Winslow et al., 1943) because some warmth and moisture are reclaimed in transit through the nasal passages. The proportion of heat loss through each component of the respiratory heat loss mechanism has not yet been determined, but it is generally agreed that a greater amount of heat is lost through \( E_{res} \) than \( C_{res} \) due to the fact that the latent heat of water evaporation is much greater than the specific heat of air. While a number of previous investigations have shown that respiratory heat loss is dependent on several variables such as temperature and vapor gradients of inspired air (McCutchan and Taylor, 1951; Cole, 1953), respiratory minute volume (Cole, 1953), changes in body temperature (Hanson, 1974), health status (e.g. asthma) (Burch, 1945; Deal et al., 1979), and working/exercise status (Cain et al., 1990; Livingstone et al., 1994), the total amount of respiratory heat loss as a function of \( C_{res} \) and \( E_{res} \) under normal condition is 10–15 Watts (W), which accounts for \( \sim10\% \) of total heat loss from the body (Burch, 1945; Ingelstedt, 1956; Hanson, 1974).

Nasal and oral respiratory pathways of thermostasis

The majority of healthy adults are nasal breathers at resting tidal breathing or light exertion (Niinimaa et al., 1980; Hallani et al., 2008), but changes in the partitioning of the breathing cycle among nasal, oronasal, and oral components can impact the respiratory portion of heat exchange, as well as microclimate (i.e. respirator dead space) heat and moisture content. The use of PFMs results in a switch from nasal to oral breathing in most adults (Harber et al., 1997) and respiratory heat exchange is impacted variably by the route of respiration. Nasal breathing is associated with less heat loss to the environment than oronasal and mouth breathing because some expired heat and humidity are reclaimed by the rich vasculature and mucosal surfaces of the nasal passages and paranasal sinuses (Harber et al., 1997; Holden et al., 1999). The nasal mucosa normally recovers one-third of the water delivered to the inspiratory airflow from the expiratory airflow (Martins De Araujo et al., 2000). When the metabolic rate is significant (e.g. during strenuous physical activity), a shift to oronasal breathing occurs that is associated with a greater respiratory minute volume (Niinimaa et al., 1980) and the percentage of mouth breathing increases as the metabolic rate increases (Harber et al., 1997). Increases in core temperature of \( \sim1^\circ\text{C} \) are associated with induction of hyperventilation (increase in pulmonary ventilation of \( \sim35\% \)) relative to metabolic needs (White, 2006). Results from a previous investigation (Varene et al., 1986) showed that the temperature and amount of water delivered on expired air are significantly greater with mouth breathing than nasal breathing. Therefore, there would likely be an increase in the net respiratory heat loss to the environment with oronasal breathing over that noted with nasal breathing only, especially during strenuous physical work and hyperventilation. The net respiratory heat loss through oronasal breathing at a high workload (150 W), under temperate ambient temperature (25°C), has been reported as 103 ± 12 W, which accounts for \( \sim46\% \) of total cephalic heat loss (Rasch et al., 1991).

The contribution of the breathing pathway to central nervous system temperature regulation has been an area of interest for some time. Hirata et al. (1978)
observed that tympanic temperatures (considered an indirect measure of core temperature) were consistently higher with mouth breathing, implying that the vascular supply to the head had been cooled more by normal nasal breathing. There have been studies proposing a mechanism of selective brain cooling (SBC) in which venous blood is cooled in the facial area and delivered through a direct venous pathway to the cranium to directly cool the brain and serve as a protective mechanism, especially in hyperthermic states (Cabanac and Caputa, 1979; Cabanac, 1993). This mechanism can be enhanced by nasal breathing and sweat evaporation on the head (Nagasaki et al., 1998). Supporting this concept of SBC is the finding of a cooling effect (0.4–0.8°C) on the frontobasal aspects of the human brain (a site in proximity to the hypothalamus, the major thermoregulatory area of the brain) in post-operative, fully conscious neurosurgical patients with mild hyperthermia spontaneously nasal breathing for 3 min (18–20 breaths min⁻¹) in ambient temperature of 22°C (Mariak et al., 1999). This can perhaps be partially explained by the fact that the distance between the roof of the nose and the floor of the anterior cranial fossa is less than a millimeter (Mariak et al., 1999). Thus, evaporative cooling of the nasal mucosa through intensive nasal breathing directly impacts temperature on the frontobasal aspects of the brain. However, it is worth noting that the issue of whether an effect of SBC is only limited to a local brain region or to the entire brain (which constitutes a significant reduction in thermal gradients of body core temperature) still remains unresolved. Of note, some studies showed that mouth breathing results in a lowering of oral temperature readings due to the cooling effects of ventilatory air on the oral mucosa in a lowering of oral temperature readings due to the cooling effects of ventilatory air on the oral mucosa in a lowering of oral temperature readings due to the cooling effects of ventilatory air on the oral mucosa (Cooper and Abrams, 1984). Others have also reported on elevated tympanic temperatures associated with mouth breathing (Neff et al., 1989; Dezell, 1994). Thus, although it is apparent that different anatomic pathways for respiration (i.e. nose, mouth) influence thermoregulation to different degree, the use of multiple types of temperature monitoring methodologies (e.g. oral, tympanic, brain, and skin temperature measurements) reported in the research literature makes it difficult to determine accurately the full impact of nasal and oral respiration on core temperature. In general, based on available data, at low-to-moderate work rates, PFM-related increases in core temperature will likely be minor, irrespective of the route of respiration.

Metabolic cost and thermal load of protective facemasks

The direct contribution of PFMs to the metabolic cost is considered to be minor: PFMs with low/moderate filter performance (typically resulting in lower levels of airflow resistance) [i.e. European classification P1 and P2 filters (80% and 94% filtration, respectively, at test conditions of 95 1 min⁻¹ constant air flow rate)] add a metabolic cost of 20 W m⁻², and for PFMs with high performance filters [i.e. European classification P3 filters (99.95% filtration at test conditions of 95 1 min⁻¹ constant air flow rate)], the metabolic cost is 40 W m⁻² (based on 1.8 m² body surface area) (Hanson, 1999). This mild effect of PFMs on energy expenditure at low-to-moderate work rates is supported by a recent study of HCWs wearing low-resistance PFMs [i.e. surgical masks and P2 equivalent FFR (i.e. N95 FFR)] during usual work activities for 30 min that showed increases in tympanic temperature of only 0.07 and 0.03°C, respectively (Yip et al., 2005). Similarly, no added metabolic/thermal load was demonstrated for tight-fitting powered air-purifying respirators (PAPR) or a negative pressure full facepiece APR used in warm environments (33.9–35°C dry bulb temperatures) at low–moderate treadmill work rates for 20 min (Caretti, 2002; Caretti and Gardner, 2003). Under high heat/high work conditions (43.3°C/116 W h⁻¹) over 1 h, oral temperature increased only 0.33°C when wearing a full facepiece APR and no significant effect was noted under high heat/low work (58 W h⁻¹), low work/high heat, and low work/low heat (25°C) scenarios, whereas conditions remained basically unchanged with a half facepiece APR (James et al., 1984). At a work rate of 200–300 Kcal h⁻¹, no significant differences were noted in core (rectal) temperature over 2 h for subjects wearing a full facepiece APR compared with not wearing a respirator (Martin and Callaway, 1974). Similarly, Guo et al. (2008) also reported that tympanic temperature rose only 0.2°C for FFR with an exhalation valve (N95FFR-EV) and 0.6°C for FFR during staggered treadmill exercise at 3.2 km h⁻¹ × 20 min, 4.6 km h⁻¹ × 10 min, and 6.4 km h⁻¹ × 10 min, with interspersed 10-min rest periods. In another study (Hayashi and Tokura, 2004), tympanic temperatures in four female subjects, at the end of performing 3 series of 15-min stepping exercises interspersed with 5-min rest periods at environmental conditions of 28°C temperature and 60% relative humidity (RH), showed increases ranging from ~0.25 to 0.5°C for N95 FFR-EV and 0.25–1.4°C for N95 FFR; increases in rectal temperature for the same exercise period were ~0.7 and 0.9°C, respectively. However, the timing of the menstrual cycle was not identified which could have
impacted temperature measurements, and the subjects were wearing protective garments (Gore-Tex) which have somewhat limited vapor permeability that could also result in heat retention, so that it is difficult to partition out the FFR component of the rise in temperatures. Thus, it would appear from the limited available data that PFM use for periods ≤1 h, under varying workloads (low, moderate, and high), has, in and of itself, limited metabolic impact and is generally associated with only minimal-to-mild increases in body temperature as measured by oral or tympanic routes. While it is a common practice to reference a level of tympanic temperature as core body temperature in determining the thermal impact of PFMs, consideration must be given to the fact that studies have demonstrated that significant variability exists between concurrent measurements at both ears and between tympanic temperatures and pulmonary artery temperature (the ‘gold standard’ for core temperature) (Fulbrook, 1997; Sanderson et al., 2010). Logically, the impact on body temperature is likely to be augmented with longer, uninterrupted periods of PFM use in high ambient temperatures and humidity and at higher work rates.

Facial skin temperature changes with protective facemasks

The head is an area of very high metabolic activity and is a critical structure for cooling, especially when the remainder of the body is impeded in normal heat dispersal (James et al., 1984). The heat flux per unit area of bare facial skin is 104 W m⁻², approximately double the 50 W m⁻² flux of the rest of the body (DuBois et al., 1990). In moderate environmental conditions, the average temperatures of peripheral tissues are 2–4°C lower than core temperature (Lenhardt and Sessler, 2006). Facial skin temperature in an adult can vary considerably by anatomic region, with the nasolabial and perioral areas (those areas most frequently covered by PFMs) having been reported as having the highest baseline facial temperatures in young adults (34.6 ± 1.7, 34.1°C ± 1.7) and older adults (35.3 ± 1.4, 35.2°C ± 1.3) (Marrakchi and Maibach, 2007). Body temperatures are regulated, in large measure, by the exchange of heat through the body’s skin where radiation, convection, and evaporative processes occur, as described earlier. Obviously, in the facial region, these processes can only occur to their optimal extent with adequate facial skin exposure to the ambient environment, a situation that is impeded by the barrier effect of PFMs (Hanson, 1999). PFM facepiece materials and design significantly impact overall comfort (Caretti and Coyne, 2008) and EAPRs, with their larger non-breathable sealing areas, are likely to have a greater impact on facial skin temperature than the more permeable FFRs and FMs. In addition to the barrier effect, the venous flow from the head and face to the cranial cavity that plays a role in brain cooling (Cabanac and Caputa, 1979) could theoretically be compromised by pressure from the straps and head harness of tight-fitting PFs (i.e. EAPRs). It has been posited that the discomfort of respirator wear is related to elevations in facial skin temperature (DuBois et al., 1990). Multiple studies have reported on the impact of PFMs upon facial skin temperature, but most do not report concurrent core temperatures that would assist in clarifying the central versus peripheral impact of PFM use upon body temperature. The contribution of facial skin temperature upon EAPR comfort parameters at 25°C ambient temperature is found in a formula derived by linear regression analysis of data from multiple studies (Caretti and Coyne, 2008):

\[
\text{Comfort} \leq 25 = 0.59 + \left( 0.06 \times \text{TS}_{\text{face}} \right) \\
+ \left( 0.20 \times \text{facepiece} \right) + \left( 0.29 \times \text{nose cup} \right) \\
+ \left( 0.25 \times \text{harness} \right) + \left( 0.22 \times \text{breathing} \right),
\]

where \( \text{TS}_{\text{face}} \) = thermal sensation of the face, facepiece is a subjective rating of facepiece comfort (unit less), nose cup is subjective rating of nose cup comfort (unit less), harness is head harness comfort rating (unit less), and breathing is breathing comfort score (unit less).

At >25°C, the formula is: Comfort >25 = 0.40 + (0.12 × TS\(_{\text{face}}\)) + (0.17 × facepiece) + (0.32 × nose cup) + (0.17 × harness) + (0.36 × breathing).

At ambient temperatures of 18.9–25.5°C and 49–63% RH, skin temperatures at the tip of the nose and at the chin increased 3.7–7.3 and 2.6–3.6°C, respectively, during sedentary activity while wearing surgical masks over a 15-min period (Enerson et al., 1967). Laird et al. (2002) reported that wearing a filter-type respirator during the last 15 min of a 30-min laboratory study at a low work rate (50 W) resulted in a 1.9°C increase in upper lip temperature, but had no effect on cheek temperatures not covered by the respirator. The second portion of that study, a simulated work environment at ambient temperatures of 17–24°C and 60–80% RH, led to increases in upper lip temperature of 0.5–2.4°C, but again had no effect on cheek temperatures measured outside the respirator. Johnson et al. (1997) noted that, at ambient conditions of 35°C and 90% RH, and sedentary activity for 90 min, skin temperature under a full facepiece APR rose by 2°C. Over a 30-min period at ambient temperatures of 21–26°C, skin temperatures taken
under rubber EAPRs and dust-mist fiber masks (a type of FFR in common use before 1995) rose 1.5 and 1.1°C, respectively, above baseline values (DuBois et al., 1990). From a subjective perception, PFM-related upper lip temperatures >34°C elicited sensations of warmth (and associated discomfort), whereas those below this level were sensed as cool-to-neutral (Gwosdow et al., 1989; DuBois et al., 1990), despite the fact that these temperatures are within the realm of normal facial temperatures. However, consideration must be given to the 90–100% RH levels attained in PFM that result in a PFM microenvironment heat index (combination of temperature and humidity effects) that may be quite high. For example, at air temperature of 34°C and 95% RH (e.g. equivalent to expired air under normal conditions), the PFM microenvironment heat index could be 62°C on exhalation, though it would subsequently be diminished variably by the admixture of inhaled ambient air.

Studies documenting the effect of PFMs upon facial skin and core temperatures concurrently are rare. Following 3 series of 15-min stepping exercises interspersed with 5-min rest periods at ambient temperature of 28°C and 60% RH, cheek temperatures under N95 FFR and N95 FFR-EV rose ~2.0 and 1.5°C, respectively, with concomitant increases in rectal temperature ranging from ~0.6 to 1.1°C and tympanic temperature increases from 0.3 to 1.3°C, but the subjects were wearing protective clothing ensembles (Gore-Tex) that could have added to the heat load (Hayashi and Tokura, 2004). It is perhaps not surprising that increases in temperature of skin under PFMs would not have a dramatic impact on core temperature given that PFMs cover a portion of the face that accounts for only 1–2% of body surface area, so that the amount of heat transfer to the core from this heated facial skin should only approximate similar percentages (McCaffrey et al., 1975). Importantly, tympanic temperature measurements cannot be relied upon as accurate indicators of central blood temperature because they are susceptible to modification by the local environment such as when localized regions of heating are present on the face (e.g. when wearing PFMs) (McCaffrey et al., 1975) or when the face is cooled (Shiraki et al., 1988). Thus, all forms of negative pressure PFMs elevate the underlying skin temperature to variable degree based upon the PFM type, fit (gaps in the seal might allow for more cooling), composite materials (e.g. silicone, polypropylene fibers, etc.), work rate, ambient conditions, and duration of use. However, this effect is noted only for skin covered by the PFM and does not seemingly impact the facial skin that is not covered; uncovered facial skin mean temperature is a linear function of ambient temperature (Nielsen et al., 1987a). Conversely, PAPR have been shown to actually decrease core temperature due to the cooling effects of their fan-supplied air (Caretti and Gardner, 2003). The limited currently available data do not allow for determination of any distinct correlation between elevated facial skin temperature underneath PFMs and concurrent core temperature, but the small area of the face covered by PFMs suggests that its contribution to core temperature would not be excessive, but may have a significant impact on the perception of thermal comfort.

**PFM dead space heat and humidity**

Facial skin temperatures are impacted by the temperature and humidity of the surrounding air under normal conditions (Nielsen et al., 1987b). When ambient temperatures are lower than facial skin temperature, radiation is the main source of heat loss. In hot conditions, especially combined with significant physical activity, when temperatures approach or exceed body temperature, evaporative cooling (sweat evaporation) becomes a dominant heat exchange mechanism (Hanson, 1999). Wearing PFMs creates a microenvironment (i.e. PFM dead space) that then becomes the wearer’s breathing environment. This microenvironment has a significant impact on heat exchange processes of the facial skin. PFM microenvironment temperature has been considered a key parameter indicating thermal stress (Li et al., 2005). In ambient conditions of high temperatures, the dissipation of heat from the PFM dead space can be negatively impacted due to a decreased temperature gradient between the ambient environment and the PFM microenvironment (Li et al., 2005). The PFM dead space ‘effective temperature’ (a single quantitative index of environmental discomfort that incorporates air temperature and humidity) can be quite high. The relatively high heat and humidity of the expired air can cause moisture to condense on the outer surface of the FFR due to the temperature difference between the FFR and the environment (Li et al., 2006). This phenomenon can negatively impact the vapor and air permeability of the FFR, which consequently impairs respiratory heat loss and imposes an increased heat burden. Consideration must also be given to the amount of sweat formed within the dead space of PFMs. Sweat rates for the head, face, and neck averaged 0.203 gm⁻¹ min⁻¹ sedentary while wearing a full facepiece APR in a warm humid environment (35°C, 90% RH) over 90 min, but most of the sweat came from the neck. It was estimated that 7.5 gm h⁻¹ of sweat could accumulate in the respirator (Johnson et al., 1997). At wet bulb globe
temperature of 19.3°C and moderate treadmill exercise at 75% maximum heart rate while wearing a full facemask EAPR, facial sweat was 1.05 g min⁻¹ (Caretti and Gardner, 1999). Increased retention of water vapor and sweat within PFMs has other important ramifications in addition to effects on comfort because it can affect the facial seal of the PFMs (Caretti and Gardner, 1999), potentially increase the breathing resistance (Roberge et al., 2010a), and theoretically increase the risk of transmission of infectious agents to the wearer via a wicking mechanism (Yi et al., 2005). However, recent studies have demonstrated that, over the course of 1 h of low work rate exercise, FFR with and without an exhalation valve and EAPR with an exhalation valve retained very little moisture, which was attributed to the use of hydrophobic fibers (i.e. polypropylene) and exhalation valves, as well as the use of low work rates in some studies (Roberge et al., 2010b,c,d). Thus, at low-to-moderate work rates, the microenvironment of PFMs develops mild-to-moderate increases in temperature with concurrent high humidity levels that increase the effective temperature to uncomfortable levels, impact comfort and tolerance, and potentially reduce respiratory heat exchange.

**Psychophysiological heat responses**

The face is relatively uniform in its sensitivity to warming when compared to the mouth (Green and Gelhard, 1987), but the area of the face that is covered by PFMs is very thermosensitive (Laird et al., 1999). This may be possibly due to a higher facial thermolreceptor density, as has been demonstrated in animals (Cheung, 2010). The microenvironment air temperature increases the temperature of facial skin covered by PFMs that, in turn, significantly influences thermal sensations of the whole body, a phenomenon that may have a neurological component that has been explained as being due to the possibility that afferent impulses from the face to the central nervous system may be weighted more than those from other areas (Nielsen et al., 1987b). Also, the impairment of heat exchange in the facial and head regions may have a more profound impact given the fact that these areas are so critical for thermal regulation (James et al., 1984). The highly thermosensitive nature of the face is exemplified by the fact that cooling of the face is two to five times more effective at suppressing sweating and thermal discomfort than cooling an equivalent dermal area elsewhere on the body (Cotter and Taylor, 2005).

Purely psychological phenomena can indirectly impact the thermal load associated with PFM use. Individuals with underlying anxiety disorders (e.g. panic attacks) are at risk of provoking same when wearing PFMs. The respiratory subtype of panic disorder displays prominent respiratory symptomatology during panic attacks that is probably linked to a false suffocation alarm in the central nervous system (Freire et al., 2010). Individuals with panic disorder are deemed to be very sensitive to increases in CO₂ levels in the body and PFM use is associated with retention of CO₂ in some individuals (Roberge et al., 2010c,d) that could potentially serve as a trigger to a panic attack (Morgan, 1983). Indeed, single breath 35% CO₂ inhalation is a standard provocation test for panic disorder (Valenca et al., 2002). Wearing PFM (e.g. gas masks) can cause claustrophobic sensations and has been used as a provocative maneuver in mild-to-moderate cases of claustrophobia (Rachman, 1993; Radomsky et al., 2001). The usual response to the onset of a panic attack or claustrophobic reaction, irrespective of the triggering event, is a sympathomimetic one brought about by the release of neurotransmitters (e.g. catecholamines such as adrenalin and noradrenalin). Release of these neurotransmitters results in increased metabolic activity manifested physically as elevated heart rate and respiratory rate, palpitations, elevated blood pressure, etc., the so-called ‘fight or flight’ phenomenon. An associated sensation of warmth in these events may be due to actual increases in body temperature brought about by the increase in metabolic activity, by neurosensory phenomena (flushing of the skin), increased respiratory effort associated with overcoming perceived increases in PFM-related breathing resistance, or by increased sweating in the PFM microenvironment brought about by psychological stress that could increase the effective temperature of that area of the face. It may be that, in temperate environments, some (possibly significant) portion of the sensation of excess heat and warmth associated with the use of PFMs has a psychological basis given that the PFM metabolic and facial heat contributions themselves are not excessive. Much of the available research supports the notion that the primary thermal effect of wearing a respirator is subjective discomfort (Caretti and Coyne, 2008). Conversely, increased body temperature associated with thermal stress can itself lead to decrements in psychomotor performance in those with no recognized psychopathology (Morgan, 1983). The psychology of PFM use has received some limited investigation in the past and would benefit from significantly more study.

**Potential mitigation strategies for protective facemask-associated heat retention**

Mitigation of PFM-associated heat is desirable for comfort that results in greater PFM tolerance and ultimately translates to greater protection for the wearer.
Some strategies aimed at decreasing the heat burden attributed to PFMs could be explored, including (but not limited to):

(i) ‘Promotion of nasal breathing when wearing PFMs’—Because nasal breathing likely results in less heat and humidity retained in the microclimate of PFMs and may have favorable effects on cooling of some brain structures, it may be desirable from a thermal perspective to promote nasal breathing through the education of wearers of PFMs. This would be feasible only for low-to-moderate work rates, as higher energy expenditures cause a switch to oronasal breathing (Harber et al., 1997).

(ii) ‘Investigation of the effect(s) of pre-use refrigeration of PFMs on facial and body temperatures’—It has been anecdotally mentioned that cooling of EAPR might be a simple method of decreasing the impact of heat on wear (Laird et al., 2002). Although silicon and rubber used to construct the body of EAPR could be cooled in such a manner, research exists neither on the length of duration of the cooling effects nor on the impact of cooling on the fit of the PFMs. Future research could be directed toward identifying PFM-compatible materials with cooling-retention features, especially in light of the fact that facial cooling is two to five times as effective at reducing thermal discomfort than equivalent areas of the skin in other body regions (Cotter and Taylor, 2005).

(iii) ‘Use of exhalation valves’—PFMs with exhalation valves are touted as increasing wearer comfort through facilitated dispersal of PFMs dead space heat and humidity to the environment. However, at the low and moderate work rates that most current workers experience (Meyer et al., 1997; Harber et al., 2009), the benefits of exhalation valves in FFR may not be realized because the development of the requisite streamlined air currents to activate the valve may not occur (Roberge et al., 2010c,d) as it does with EAPR. Improvements in design and function could potentially lead to exhalation valves that function with lesser airflow gradients that might afford greater heat and humidity losses at lower energy expenditures.

(iv) ‘Investigate the breathability of PFM filtration materials’—While there is likely a tradeoff between breathability (vapor and air permeability) and PFM filtration efficiency (that is critical to reducing the risk of exposure to harmful particles and infectious agents), it would be of importance to fully investigate the material properties of PFMs to ensure optimal breathability that could lead to subsequent lowering of PFM dead space humidity levels that impact comfort and tolerance. For example, nanofibers offer filtration efficiency with a concomitant decrease in breathing resistance over that noted with other meltblown and spunbonded filter materials (Qion and Wang, 2006; Lee and Obendorf, 2007).

(v) ‘Development of PFMs with miniaturized battery-powered fans’—Fan-derived air currents, as exemplified by PAPRs and surgical hoods, cool the head and facial regions and the inhaled air resulting in minimal increase or a decrease in body temperature (Caretti and Gardner, 2003). Miniaturized (8 × 8 × 3 mm) battery powered fans currently exist for cooling various small electronic appliances (e.g. smart phones, GPs modules, etc.) and could potentially be adapted to PFMs (http://www.sunonamerica.com/pdf/mm_fan_catalog.pdf). One such model currently on the market, the BL-50 from Koken, Ltd. (Tokyo, Japan), is a half-mask that contains a battery-powered integral blower triggered by inhalation and used to maintain constant pressure within the facepiece (Richardson and Hofacre, 2008). In addition to cooling the face, the development of positive pressure by integral fans could serve to enhance respiratory protection by preventing ingress of harmful particles or organisms into the PFM.

(vi) ‘PFM dead space parameters’—Re-breathing of retained warm expired air within the dead space of PFMs increases facial heat discomfort. Some styles of PFM (e.g. cup shaped and duckbill FFR and FM) have larger dead spaces and can thus result in greater volumes of retained warmed air than other styles (e.g. flat fold and pleated FFR and FM). A recent study of a cohort of HCWs using PFM reported that 81% of HCWs interviewed used either a cup-shaped or duckbill N95 FFR and that 56% of all interviewees indicated that they experienced increased facial heat ‘most or all of the time’ (Baig et al., 2010). Therefore, it would be important to study the impact of various styles of PFMs on facial heat in order to determine those styles with lower associated increases in facial heat.

(vii) Anxiety-related perceptions of PFM-associated heat—The retention of CO$_2$ with the use of PFMs is a possibility (Roberge et al., 2010d), and panic disorder can be triggered by elevated CO$_2$ levels. Some of the symptoms of panic disorder include hot flashes and sweating.
Response to the 35% single breath CO₂ inhalation provocation test is quite specific for panic disorder and is routinely utilized for this diagnosis. Individuals who demonstrate intolerance to PFMs could undergo non-invasive transcutaneous CO₂ monitoring and a CO₂ provocation test to assist in determining if CO₂ retention is the source of their symptoms.

The large number of PFM users (private industry, HCWs, the public) and the increased use of PFMs in certain scenarios (e.g. infectious agent outbreaks, environmental disaster remediation efforts, etc.) should make PFM-related effects on thermoregulation a major focus for researchers and should serve as a significant impetus for additional investigation. Intolerance to the thermal effects of PFMs leads to decreased use and concomitant decreased protection for the user.

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