AIS

AUSTRALIAN INSTITUTE OF SPORT (AIS) POSITION STATEMENT:

EXERCISE IN BUSHFIRE SMOKE FOR HIGH PERFORMANCE ATHLETES

Endorsed by Australasian College of Sport and Exercise Physicians (ACSEP) and Sport Medicine Australia (SMA)

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INTRODUCTION

Impacts of the 2019-20 bushfires

Australia experienced the most severe bushfire season on record during the 2019-20 summer season. Thirty three people lost their lives and 17 million hectares were burned across New South Wales (NSW), Victoria (VIC), Queensland (QLD), Australian Capital Territory (ACT) Western Australia (WA) and South Australia (SA).[1] The health burden associated with the 2019–20 bushfires in eastern Australia included an estimated 417 excess deaths, 2,027 hospital admissions for all respiratory problems and 1,305 emergency department (ED) presentations related to asthma.[2] The number of ED presentations in the ACT and Victoria increased when the particulate matter with a diameter ≤2.5 micrometres ($PM_{2.5}$) concentration increased above 25ug/m^3.[3]

During the 2019-2020 bushfires, several sporting events were cancelled or postponed due to concerns for high performance athletes' health and wellbeing.

- On 21 December 2019: a Domestic T20 cricket match in Sydney (NSW) between Sydney Thunder and Adelaide Strikers was abandoned mid-match with poor visibility a major concern.[4]
- On 3 January 2020: a Domestic youth soccer match in Newcastle between Newcastle and Canberra due to be played on 5 January was postponed due to concerns for poor air quality.
- On 5 January 2020: The Women's soccer league round 8 match between Canberra United and Sydney FC, scheduled in Canberra, was postponed.[5]
- On 6 January 2020: The 2020 Canberra (ACT) international tennis tournament was relocated to Bendigo (VIC) due to uncertainty as to if or when the ACT air quality would improve.[6]
- On 15 January 2020: Australian Open qualifying matches at Melbourne Park were postponed until improvements in air quality were observed.[7]

While exercising outdoors athletes will increase the magnitude (both time and intensity) of their exposure to any pollutants present in outdoor environments. Therefore, they are likely to be at an elevated risk for acute adverse health events relating to air pollutants, including bushfire smoke. At a population level, it is generally accepted there is a linear relationship with increasing prevalence of health consequences with increasing exposure to air pollutants, with no known safe lower level of air pollution in general, nor specifically with regard to bushfire smoke. [8] The World Health Organisation's 2005 Air Quality Guidelines recommend developing tailored health advice for athletes. [8] During the eastern Australian 2019–20 bushfires, the Australian Institute of Sport (AIS) provided athlete specific advice to the National Institute Network (NIN) and National Sporting Organisations (NSOs). To do so, available public health guidelines for physical activity in smoke affected environments were interpreted to accommodate the type of physical activity levels occurring in the high-performance sport context. The frequency of bushfires in Australia is increasing [9] and it is expected bushfire smoke will be a more prevalent phenomenon impacting air quality. The current position statement has evolved from the initial AIS 2020 guidelines and summarises empirical evidence relating to health and performance effects of bushfire smoke with a focus of acute to sub-acute health events during periods of poor air quality secondary to bushfire smoke. The current position statement aims to provide practical advice to high performance athletes and coaches needing to train or compete in environments impacted by short term bushfire smoke conditions. Long-term consequences of bushfire smoke exposure, and other sources of air pollution, are beyond the scope of this position statement.

Constituents of bushfire smoke

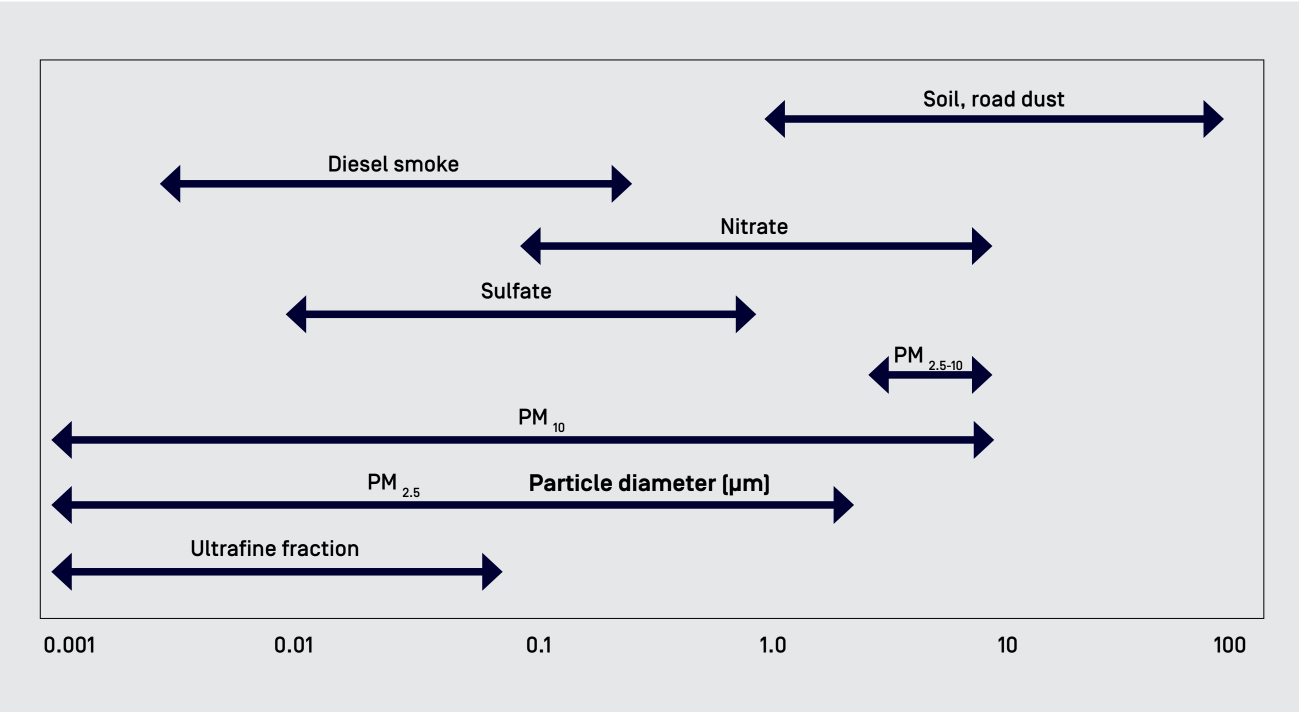
The chemical components and the size and particulate concentration of the emissions within air pollution can potentially affect health and physical performance. [10] Bushfire smoke is generated from the combustion of biomass, and its constituents differ from other sources of urban pollution such as traffic and industrial emissions. Smoke from Australian vegetation is composed primarily of carbon dioxide (CO_2), water vapor, carbon monoxide (CO), particulate matter (PM), hydrocarbons, nitrogen oxides and trace minerals and volatile organic compounds. [10-14] Water soluble metals sodium (Na) and potassium (K) have been reported as the most common metals, accounting for 97% of metals in particles that have a diameter of ≤2.5 micrometres (i.e., $PM_{2.5}$ particles). Heavy metals such as Aluminium (Al), Chromium (Cr), Manganese (Mn), Iron (Fe), Nickel (Ni) and Cadmium (Cd) can be present in bushfire smoke but are often below the limit of detection. [15] In contrast, heavy metals are more commonly associated with industrial pollution, [16] petrol and diesel emissions. [17]

Particulate matter refers to the small particles characterised according to their diameter as either less than 2.5um ($PM_{2.5}$) or less the 10um (PM_{10}). The smaller particles carry other pollutants such as heavy metals and NO_2 deeper into the lungs increasing their systemic absorption. The creation of particulate matter in the combustion of biomass generally results in a relatively stable $PM_{2.5}$ to PM_{10} ratio, with the small variance largely being explained by modified combustion efficiency which reflects the amount of oxygen (O_2) available at the time of combustion. $PM_{2.5}$ generated by Australian vegetation combustion accounted for 98.7% of the overall PM_{10}. [15] It should be noted that PM_{10} was predominantly reported in early research however more recently both $PM_{2.5}$ and PM_{10} were reported.

Air quality measurements

It is not practical or useful to continuously measure all the pollutant constituents in all locations. Particulate matter measurements have been widely adopted as an overall indicator of air pollution and are a commonly used metric for quantifying bushfire smoke severity. $PM_{2.5}$ overlap with PM_{10} because PM_{10} measures include $PM_{2.5}$ (Figure 1). $PM_{2.5}$ is likely a better indicator of health risk, although either can be considered a reasonable proxy for air pollution exposure. [8]

FIGURE 1:
Size range of airborne particles, showing the health-related ultrafine, PM2.5 and PM10 fractions and the typical size range of some major components [8] Reproduced with permission from the WHO Regional Office for Europe on behalf of the WHO subject to the terms and conditions of the non-exclusive licence.



Australian State and Territory Public Health Authorities uniformly publish hourly online air quality data from base stations spread across their jurisdictions. Each jurisdiction includes $PM_{2.5}$ and PM_{10} concentrations (micrograms (mcg) per meter cubed) as a factor in determining the overall air quality index (AQI) rating. The AQI is a unitless metric to benchmark the upper limit of acceptable air quality against a score of 100, this usually references the single worst current pollutant. The intention is to give an intuitive appreciation compared to normal air quality in the region of interest. However, reporting and inclusion of other metrics such as total suspended particles, NO_2, SO_2, ozone, visibility index and CO changes the reported air quality index (AQI) for the same relative air quality. Given variability in the AQI across Australian States and Territories and potential for misinterpretation, the current guidelines will be framed in the context of $PM_{2.5}$ and PM_{10} concentrations.

Measurement of particulate matter

Smoke concentrations in the atmosphere can markedly vary within a short distance (e.g., 2 km) and rapidly change over time with changing weather conditions. Based on the air quality measurement principles outlined above, the real time hourly $PM_{2.5}$ average is the most useful measure to evaluate the acute potential for exposure. The rolling 24-hour average can be considered an indicator of residual elevated risk for an acute medical event in susceptible individuals. Elevated 24-hour average levels, despite low real time levels, should trigger monitoring of athletes for any early evolving symptoms or previous bushfire smoke sensitivity. This is important as epidemiological studies have demonstrated Emergency Department (ED) presentations can lag following elevated bushfire smoke periods, [18] indicating smoke exposure health related events do not only occur at the immediate time of bushfire smoke exposure.

Handheld devices for measurement $PM_{2.5}$

A range of handheld measurement devices are available to measure atmospheric $PM_{2.5}$ concentration. It is important that the device used is designed for measurement of outdoor rather than indoor concentrations of $PM_{2.5}$. Teams and sporting clubs can utilise these handheld measurement devices to ascertain real-time measures of $PM_{2.5}$ concentration at a specific location when they wish to exercise. The real-time $PM_{2.5}$ value can then be used to advise athletes and officials regarding appropriate exercise activity. However, this may not be an affordable option for many sporting teams and clubs. If pitch-side measurements are not an option, it is reasonable to default to accessing the nearest State or Territory government monitoring station where hourly online readings are available. It should be noted that many of the public monitoring sites are using continuous measurements and then publish hourly average online.

The State and Territory website that provide real time information on air quality.

- **ACT:**
  https://www.health.act.gov.au/about-our-health-system/population-health/environmental-monitoring/monitoring-and-regulating-air
- NSW:
  https://www.dpie.nsw.gov.au/air-quality/air-quality-concentration-data-updated-hourly
- Victoria:
  https://www.epa.vic.gov.au/for-community/airwatch/airwatch-table-data-page#tab-standard-monitoring-sites-2
- Queensland:
  https://apps.des.qld.gov.au/air-quality/
- Tasmania:
  https://epa.tas.gov.au/environment/air/monitoring-air-pollution/monitoring-data/real-time-air-quality-data-for-tasmania/all-sites-table
- South Australia:
  https://www.epa.sa.gov.au/environmental_info/air_quality/new-air-quality-monitoring?view=table
- Northern Territory:
  http://ntepa.webhop.net/NTEPA/Default.ltr.aspx
- Western Australia:
  https://www.der.wa.gov.au/your-environment/air/air-quality-index

URLs to each State and Territory website that provide real time information on air quality were checked on 20 October 2022 and confirmed as live.

Apps

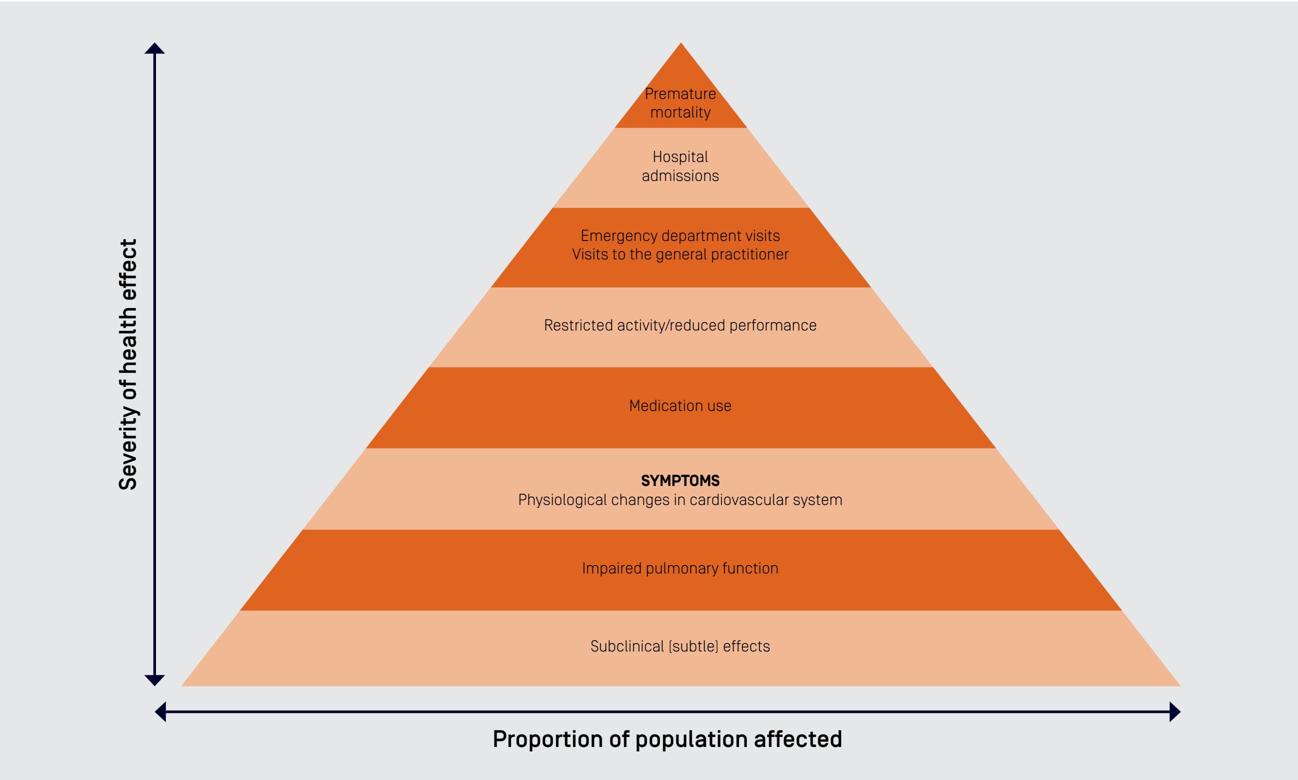
The AirRater App uses real-time air quality measurements being gathered at monitoring sites by each of the State and Territory governments and makes it available in a convenient format, on a smart phone app. (https://airrater.org/). Data is presented as $PM_{2.5}$ concentration in mcg per meter cubed.

Atmospheric "visibility" as a proxy for $PM_{2.5}$ concentration

It may not be feasible nor affordable for sporting teams, clubs and/or athletes to implement specific hardware devices that monitor ambient $PM_{2.5}$ on site. On this point, it is known that atmospheric visibility is correlated with ambient $PM_{2.5}$. [19-21] it may therefore be tempting to use atmospheric visibility as a surrogate index of $PM_{2.5}$ concentrations in lieu of any dedicated air-quality measurement devices. However, it is emphasized that although the presence of fine particulate matter does indeed alter light scattering and absorption of the ambient air, the degree to which "visibility" correlates with $PM_{2.5}$ concentration is dependent on the prevailing meteorological conditions (i.e., relative humidity) and the chemical species present within the fine particulate matter itself [20, 22, 23] thus, rather than using atmospheric visibility as a proxy for on-site $PM_{2.5}$ concentrations, one might instead use an acute reduction in visibility as a marker of worsening air quality. This warning would then demand that athletes, coaches and/or support staff should immediately seek out $PM_{2.5}$ concentrations from their relevant state and territory air-quality monitoring service. Health effects of bushfire smoke

Severity of health effects from pollution can be observed on a continuum as described by the American Thoracic Society. [24] In periods of elevated pollution due to bushfires, it is important to monitor for signs and symptoms at the bottom of the pyramid and actively limit exposure to prevent progression of severity of health effects escalating.

FIGURE 2:
Pyramid of health effects associated with air pollution. Adopted from the American Thoracic Society [24]



Effects of chemicals within bushfire smoke

Carbon monoxide (CO) is toxic due to its asphyxiant effects. It binds to haemoglobin with a 200% greater affinity than O_2, producing a stable compound called "carboxyhaemoglobin". CO also inhibits cytochrome oxidase in the electron transport chain inhibiting ATP production. [25] The SafeWork NSW workplace exposure standard of CO is 30 parts per million (ppm) averaged over 8 hours. Increased concentrations can be tolerated for shorter durations if the 8 hour average does not exceed 30 ppm. [26] During the 2019-2020 bushfire season, Canberra's worst period of air quality (PM 2.5ug/m_3 of 826.4 at 22:00) was on January 5 with an 8 hour CO average of 11.58 ppm. Even though elevated CO levels associated with bushfires may not exceed occupational safety standards, elevated levels may be expected to have an acute net performance impairment in athletes. [27]

Nitrogen dioxide (NO_2) is measured as a proxy for nitrogen oxides. Nitrogen dioxide can react with water and produce nitric acid (HNO_3) which is a respiratory irritant. Breathing air with a high concentration of NO_2 can irritate airways in the human respiratory system. Such exposures over short periods can aggravate respiratory diseases, particularly asthma, leading to respiratory symptoms (such as coughing, wheezing or difficulty breathing), hospital admissions and visits to emergency rooms. Longer exposures to elevated concentrations of NO_2 may contribute to the development of asthma and potentially increase susceptibility to respiratory infections. [28] People with asthma, as well as children and the elderly are generally at greater risk for the health effects of NO_2. If systemically absorbed, it can transform haemoglobin into methemoglobin, thereby reducing O_2 carrying capacity. When Canberra's air quality was at its worst, the NO_2 level was 0.48 ppm averaged over 1 hour. The upper limit for occupational exposure over a working day is 3 ppm. [29]

Smoke inhalation: a spectrum of illness

Smoke inhalation injury is defined as the injury resulting from a high intensity exposure at or very close to the source of a fire. It can result in a supraglottic thermal injury, chemical irritation of the upper and lower respiratory tract, asphyxiation and systemic toxicity from lung mediated absorption of smoke constituents. [30] Athletes performing heavy exercise in bushfire smoke affected environments are most likely to experience a moderate and dose proportional form of respiratory tract chemical irritation, systemic toxicity and possibly performance limiting effects of exposure to asphyxiants.

Bushfire smoke has local toxic effects throughout the respiratory tract which can reduce surfactant production, [31] impair muco-ciliary clearance [32] and result in preferential damage to secretory and ciliated cells. [33] These factors collectively may impair lung function and render an individual more susceptible to infection and secondary pneumonia. Moreover, chemical irritation of the respiratory tract can result in bronchoconstriction and asthma-like symptoms resulting in increased perceptions of breathing effort and/or difficulty (i.e., acute breathlessness).

Acute smoke exposure also results in dose dependent changes to the local immune cell presence in the lower respiratory tract. In patients admitted to an intensive care burns unit, bronchoalveolar lavage (BAL) fluid demonstrated a shift towards a predominant neutrophil presence in those with worse smoke inhalation injury. [34] The normal macrophage percentage from BAL fluid is 95% and 0.7% for neutrophils. [35] Patients with a grade 0 (no injury) according to the Abbreviated Injury Score demonstrated 41% of neutrophils and 54% macrophages, compared to 94% neutrophils with 6% macrophages in the highest severity grade 4 category [34] BAL neutrophilia is an indication of the acute inflammatory response caused by the bushfire smoke.

Similar, but lower magnitude changes have also been observed in young healthy firefighters who had a controlled exposure to woodfire smoke. After woodfire smoke exposure standardised at a $PM_{2.5}$ of 485ug/m^3 for two hours including a light cycling activity. Bronchoalveolar lavage was conducted 20 hours post intervention. A significant increase of absolute neutrophil percentage to above 8% [36] was observed. Moreover, when healthy participants were exposed to woodfire smoke at 224 ug/m^3 for 3 hours with mild aerobic exercises, no changes to BAL cell counts 24 hours after exposure was observed. [37] It is possible that exposure at a level between these two exposure conditions is where healthy individuals begin to experience subclinical effects.

An absolute neutrophil percentage equal to or greater than 50% is generally considered diagnostically supportive of acute lung injury, aspiration pneumonia or suppurative infection. [38] The exposure studies noted above indicate the minimum exposure that can initiate a localised neutrophilia is likely between 224 ug/m3 and 485 ug/m3, for 2-3 hours. [36, 37] Both studies [36, 38] reported there were no changes in pulmonary function, and a paucity of symptoms. Importantly, these studies were only a one-off exposure without an elevated background exposure. Currently there is no evidence regarding the time required to recover from a one-off exposure or compounding effects of repeating the exposure prior to recovery.

Systemic toxicity effects

Beyond acute lung irritation, bushfire smoke has the potential to exert systemic effects. The mechanisms proposed include increased oxidative stress, inducing a pro-coagulative state, elevating pro inflammatory cytokines and modulation of the autonomic nervous system. [39] There is a paucity of research into systemic toxicity associated with bushfire smoke in athletes. It is expected these health effects would be variable, usually mild, and consequently difficult to identify outside of the research setting. The training requirements and as a result, minute ventilation throughout the duration of activity varies greatly between athletes across different sports, and sometimes within the same sport. It is possible therefore that high performance athletes may experience one or more of an increased risk of respiratory infections, impaired recovery metrics, or a mismatch between expected internal and external load.

Systemic effects of short term woodfire smoke exposure

Modest increases of blood oxidative stress have been observed in young fit healthy firefighters (aged between 18-40 years) following acute woodfire smoke (up to 500ug/m^3 $PM_{2.5}$ emissions for 1.5 hours), [40] although no spirometry changes were observed. [41] The acute effects of exposure to asphyxiants in bushfire smoke can start to be observed at $PM_{2.5}$ concentrations of 1000 ug/m^3 after 1 hour of exposure with a statistically significant increase in carboxyheamoglobin from 0.9% to 1.3%. [42] In spite of the $PM_{2.5}$ being a relatively high concentration (for a short duration) the carboxyhaemoglobin did not exceed the normal range of 0-2%. [43] In isolation, the asphyxiant effect of short intense exposures to high concentrations of $PM_{2.5}$ may exert only a trivial effect on maximal aerobic capacity (i.e., VO_{2max}) and thus competitive exercise performance. The systemic effects of short-term bushfire smoke are summarised in **Table 2** and are largely confined to subtle subclinical effects. Also, what is known regarding the chronic effects are also summarised in **Table 2**.

Acute health impact of bushfire smoke

Averaged PM recordings over a day from State and Territory governments, with hospital or ambulance dispatch records, were used in 13 Australian bushfire smoke studies. Six studies included $PM_{2.5}$ and the remaining seven included PM_{10}. Five out of the 13 studies analysed periods with average PM exceeding the maximal daily standard for PM set by the NEPM (**Table 1**). A small positive association was observed with an increased risk of presenting to hospital for an exacerbation of asthma across the broader population, at air quality levels usually deemed as good quality. was observed. Younger (< 20 years) and working age (20-49 years) people are more likely to use medical services associated with their bushfire smoke exposure, relative to older age groups (> 49 years). During the Latrobe Valley coal mine fire over 6 weeks in 2014, the peak $PM_{2.5}$ concentration was 31.2 ug/m^3 with an average of 5.2 ug/m^3. [44] During this period for individuals aged 20-34 years, every 10ug/m^3 increase in $PM_{2.5}$ resulted in an increased frequency of long general practitioner (GP) consults (range from 20 to 40 minutes) and specialist respiratory services presentations by 14% and 120% respectively. However, there were no increases for standard GP consults or cardiology services, [44] consistent with international observations. [45] A study during Canadian bushfires (2003 to 2010 forest fire seasons i.e., 1 April to 30 September), reported the average $PM_{2.5}$ concentration was 5.9ug/m^3 with extreme fire days measuring 10.2 ± 11.1 ug/m^3. Every 10ug/m^3 increase in $PM_{2.5}$ from bushfire smoke increased salbutamol dispensation by 4%, asthma consultations by 6%, physician reported lower respiratory tract infections by 3% and otitis media by 5%. [46]

TABLE 1:
Bushfire smoke and associated health events in Australia 1994-2007.

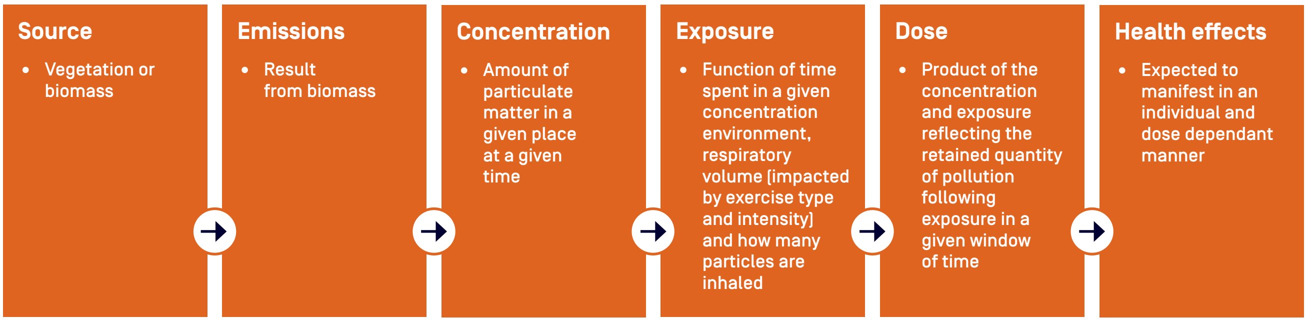
PM <diameter um	Location	Study	Duration	Age	Daily Mean um/m³	Max um/ m³	Spread	Outcome
2.5	**Melbourne**	Dennekamp et al., (2015) [47]	July 2006-June 2007	>35	32.4	247.2	IQR 6.1	OHCA in men increased 4.7% lag 1 day and 8.1% lag 2 day for every 6.1ug/m³
	Melbourne	Haikerwal et al., (2016) [48]	2 months Jan 2007		15.81	294	IQR 8.6	Asthma ED presentations increased by 1.96% same day for every 8.6 ug/m³ increase
	Sydney	Martin et al., (2013) [18]	1994 - 2007		43	100	27.4-100.2 (min-max)	Respiratory admissions asthma day 0, to lag 2 days OR 1.1-1.12, nil cardiovascular on smoke days (PM_{10}> 47.3) vs no smoke days
	Sydney	Johnston et al., (2014) [49]	1996 - 2007	15-64	39.1	100.2	14.6-100.2 (min-max)	ED presentations subgroup analysis Asthma 1.38- 1.18 OR increased lag 0- lag 3 for smoke days ($PM_{2.5}$ >27ug/m³ or PM_{10} >47 ug/m³ vs non smoke days
	Sydney	Salimi et al., (2017) [50]	2004 - 2015		16.4	56.1	IQR 5.4, SD 6.8	Ambulance call outs for breathing difficulty increased; 10ug/m³ increase RR 1.04, RR 1.05 cardiac call outs lag 2 days
	Newcastle	Martin et al., (2013) [18]	1994 - 2007		34.5	61.9	25.2-61.9 (min-max)	Nil association with asthma or CVSR
	Wollongong	Martin et al., (2013) [18]	1994 - 2007		38.3	112	24.7-112 (min-max)	Nil association with asthma or CVSR
10	**Brisbane**	Chen et al., (2006) [51]	1997-2000		18.28	60.6	7.5-60 (min-max)	Respiratory hospital admissions increased for PM 10 >15, 15-20 and above 20 at lag 0 1 and 5 RR 1.09 between 15-20ug/m³, RR 1.19 for >20
	Darwin	Johnston et al., (2002) [52]	April to 31 Oct 2000		20.84	70	2-70 (min-max)	Asthma ED presentations PM_{10} above 40 RR 1.92- 2.56 for current day to lag time 5 days
	Darwin	Johnston et al., (2007) [53]	Fire season 2000, 2004, 2005		19.1	70	6.4-70 (min-max)	ED presentations subgroup analysis only showing effect COPD OR 1.21, indigenous with COPD 1.98, indigenous cardiovascular lag 3 days OR 1.71
	Darwin	Hanigan et al., (2008) [54]	1996 - 2005		15.31	31.12	6.93-31.12 (min-max)	Same day total respiratory admissions 4.81% increase for 10 ug/m³
	Darwin	Crabbe et al., (2012) [55]	1993 - 1998		16.9	42.5	SD 7.7	ED admissions for respiratory conditions, increase 10ug/m³ RR 1.025 lag 1 days including flu admissions
	Melbourne	Tham et al., (2009) [56]	Jan-March 2003		22.21	181.7	SD 16.4	ED respiratory presentations increased accounting for day of week, temperature and humidity RR 1.028 and 1.018. no effect on admissions
	Melbourne	Dennekamp et al., (2015) [47]	July 2006-June 2007		55.2		IQR 13.7	OHCA in men increased 8.3% lag 1 day, and 11.1% lag 2 days for every 13.7ug/m3
	Moe	Tham et al., (2009) [56]	Jan-March 2003		24.78	288	SD 27.82	no significant associations with respiratory hospital admissions or ED presentations
	Traralgon	Tham et al., (2009) [56]	Jan-March 2003		21.97	237.8	SD 22.53	no significant associations with respiratory hospital admissions or ED presentations
	Newcastle	Martin et al., (2013) [18]	1994 - 2007		67.1	160.9	49.9-160.9 (min-max)	Nil association with asthma or CVSR
	Sydney	Morgan et al., (2010) [54]	1994 - 2002		62	117	117-43 (min-max)	Asthma admissions increase 5.02% on bushfire smoke days at lag 0
	Sydney	Johnston et al., (2011) [57]	1997 - 2004		67.3	114.8	IQR 11.7	Mortality increase 1 day lag OR 1.05, no specific effects on cardiovascular or respiratory mortality. (*temp model did show effect)
	Sydney	Martin et al., (2013) [18]	1994 - 2007		67.3	114.8	47.3- 114.8 (min-max)	Respiratory admissions asthma day 0, to lag 2 days OR 1.1-1.12, nil cardiovascular on smoke days (PM_{10}> 47.3) vs no smoke days
	Sydney	Johnston et al., (2014) [49]	1996 - 2007		60.5	114.8	32.0-114.8 (min-max)	All non-trauma ED presentations OR 1.02- 1.04 Lag 0- lag3 days for smoke days ($PM_{2.5}$ >27ug/m³ or PM_{10} >47 ug/m³ vs non smoke days
	Wollongong	Martin et al., (2013) [18]	1994 - 2007		68.8	280.5	50.9-280.5 (min-max)	Nil association with asthma or COPD

The results from one off woodfire exposure studies [**Table 2**] conflict with the findings reported from epidemiological studies [**Table 1**]. The exposure studies indicate that for a young fit healthy person without co-morbidity, a one-off exposure of 2-3 hours with moderate intensity exercise at a high $PM_{2.5}$ (i.e., >224 ug/m^3) is not likely to result in adverse outcomes. However, at a population level only a modest increase in $PM_{2.5}$ has an observable increase in respiratory specialist presentations and medication use in the young adult population. This may indicate duration of exposure averaged over days is more relevant than shorter intense exposure or rather that the participants of the exposure studies are a self-selecting population who have knowingly volunteered for a smoke exposure study, have no known co-morbidities and are fit enough to complete an exercise protocol. As it is unclear what exposure is required to observe adverse health and performance outcomes a broad range should be considered as it is likely that some individuals will be more susceptible to medical sequalae from exercising in bushfire smoke. Therefore, guidelines will take into account the need for consideration of individual risk, and the need to consider the worst possible outcomes when providing guidance for large group.

TABLE 2:
Systemic effects of short term woodfire exposure based on the American Thoracic Society Pyramid of Health Outcomes.

Study	Duration	Exercise	Highest $PM_{2.5}$ exposure arm	Subclinical subtle effects	Impaired pulmonary function	Physiological changes in cardiovascular system	Symptoms
Sehlstedt et al., (2010) [37]	3 hours	yes	224 ug/m^3	BAL GSH increased, mild nose/ throat irritation	x	n/a	x
Sällsten et al., (2006) [58]	4 hours	yes	279ug/m^3	mild eye irritation 10/13 subjects, slight increase nose symptoms in 5/13 participants	n/a	n/a	n/a
Barregard et al., (2006) [59]				acute phase reactant SAA, factor VIII and VIII/ vWf ratio at 20hrs, reduced IL-6			
Danielsen et al., (2008) [60]				increase DNA damage and repair			
Stockfelt et al., (2012) [61]	3 hours	no	295ug/m^3	minor nose irritation, increase biomarkers CC16 which is thought to indicate acute epithelial damage and inflammation	n/a	n/a	eye irritation
Stockfelt et al., (2013) [62]				no systemic inflammation or coagulation or oxidative stress			
Unosson et al., (2013) [63]	3 hours	yes	314ug/m^3	x	n/a	increase arterial stiffness, decrease HRV, increase HR, no change in BP	x
Riddervold et al., (2011) [64]	3 hours	no	385ug/m^3	minor mucosal irritation time does not dose dependant, changes in subjective symptoms			x
Forchhammer et al., (2012) [64]				no effect on markers of oxidative stress, DNA damage, cell adhesion, cytokines or micro-vascular function in atopic subjects.			x
Bønløkke et al., (2014) [65]				Decrease IL-6	n/a	x	
Riddervold et al., (2012) [66]				limited local inflammation exhaled breath condensate	x		
Walker et al., (2020) [67]	2 hours	no	462 ug/m^3	x	x	n/a	x
Ghio et al., (2012) [36]	2 hours	yes	485ug/m^3	IL-1B + LDH increase post, BAL increase Neut @ 20 hours	x	16.8% decrease in max HR normal by 20 hours	x
Peters et al., (2018) [40]	1 hour	yes	500ug/m^3	x	x	x	x
Hunter et al., (2014) [42]	1 hour	Yes	1000ug/m^3	increase CO at 1 hour	n/a	x	x

FIGURE 3:
Combustion to health effects: Understanding the factors (including exercise) that augment risk. Adapted from the WHO Air Quality Guidelines [8].



The effect of exercise on bushfire smoke exposure

Figure 3 illustrates the combustion to health effects pathway. Airway responsiveness to bronchial challenge testing (e.g., inhaled methacholine or histamine) varies greatly among healthy adults. Indeed, the dose required to evoke bronchoconstriction varies between individuals by up to four or more orders of magnitude. [68] This observation defines individuals with asthma, whose tolerance occurs within a much narrower range; i.e., within 1-2 doubling doses. [69] Due to this large span of airway sensitivity, it is difficult to estimate the threshold dose of smoke inhalation that can be expected to trigger an acute health episode.

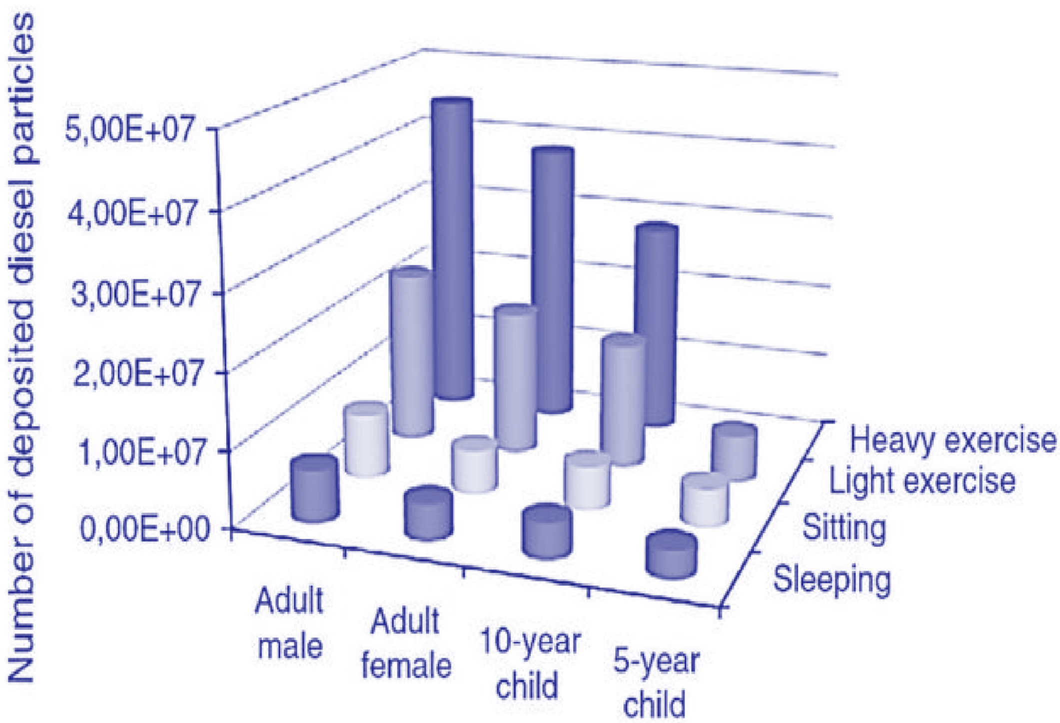
Elite athletes have similar [70] or higher [71] rates of asthma relative to the general population. Endurance and aquatic sports specifically have higher rates of asthma. [7, 70] Further, elite athletes have high rates of bronchial hyperresponsiveness, with 14-75% of elite athletes (cross country skiers, ice hockey, swimmers and floorball players) reporting bronchial hyperresponsiveness to a methacholine challenge (PD20 at 1.6-2mg of methacholine). [68] Another form of airway dysfunction that may occur in athletes is exercise-induced laryngeal obstruction (EILO). Indeed, the prevalence of EILO among adolescent athletes has been estimated at 5-10%. [72] Although the precise stimuli for exacerbation of EILO remain unclear, irritation of the upper airways (e.g., via smoke inhalation) is a probable candidate. [73] Unfortunately, it is not always possible to identify who is and is not likely to be sensitive to bushfire smoke. Asthma exacerbations can occur due to bushfire smoke even at levels below official warning levels, and this effect can lag for up to five days after the peak exposure. [52] Therefore, multiple individuals in a group can potentially be sensitive to bushfire smoke at different times from exposure. When managing athletes with recent symptom flairs or who are known to be airway sensitive, noting the PM concentration over the previous week may also be helpful.

Exercise increases the exposure of bushfire smoke through three main mechanisms. [74]

1. Tidal volume increases with increasing exercise intensity.
2. Increasing tidal volumes reduce the amount of air that is filtered through the nasal cavity as breathing switches from predominantly nasal to oral, consequently meaning more particulate matter may be deposited in the lower airways.
3. Increasing minute ventilation increases the particle deposition fraction in the lungs. [74]

The net effect of a 6 fold increase of ventilation rate during high intensity exercise can increase particle deposition by 10 fold [75] (**Figure 4**). Moreover, exercise intensity alters the location of deposition of various sized particles, with larger quantities of smaller particles potentially being deposited deeper into the lung [76]. Taken together, these findings suggest that one hour of high intensity exercise would yield an equivalent amount of particle deposition within the lungs that would otherwise be observed after 10 hours of exposure at rest.

FIGURE 4:
Particle deposition and ventilation adopted from Oravisjärvi et al.[75]



The choice to exercise or not in ambient conditions of elevated particulate matter is highly relevant for athletes with asthma or low tolerance levels (with a history of high airway responsiveness). In these athletes, a one order of magnitude increase in exposure may be sufficient to trigger acute bronchoconstriction. However, in other athletes who have low airway responsiveness, a single order of magnitude exposure could result in no observable changes, as the athlete may be able to tolerate 4 to 5 orders of magnitude, making the exposure increase from exercise inconsequential. Knowledge of individual history of airway responsiveness is important to real world decision making.

It is important that athletes, coaches and support staff are aware of the risks associated with exercise in environments affected by bushfire smoke. Where bushfire smoke is present, an individual's history of elevated airway responsiveness combined with the type of exercise under consideration should be weighed against the prevailing $PM_{2.5}$ levels (**Table 3**). Athletes, coaches and support staff should also be familiar with accessing information on $PM_{2.5}$ concentration via State and Territory websites (as above) or via reliable hand-held measuring devices. Athletes who have any history suggestive of elevated airways responsiveness should seek medical review. A decision can then be made as to whether the athletes clinical picture warrants bronchoprovocation testing.

There are several different grading systems used by different jurisdictions, health organisations and sport organisations around the world. They are all based on the agreed proposition that AHR increases with increasing concentrations. The studies conducted are not powered sufficiently to give definitive, evidence-based cut offs. The cut offs used in **Table 3** are consistent with those recommended by most health bodies in Australia.

The amber threshold is the first level at which significant restriction of activity is recommended. The broad range for the amber zone in **Table 3** is reflective of the large variation in bronchial hyperresponsiveness described. The wide For instance, some individuals may experience symptoms at < 50 $PM_{2.5}$ mcg/m^3 where as others may not experience any symptoms until the concentration is > 150 $PM_{2.5}$ mcg/m^3.

TABLE 3:

Exercise risk categories and exercise-specific recommendations according to atmospheric $PM_{2.5}$ concentration

Exercise risk category	Rationale	Exercise-specific Recommendations	$PM_{2.5}$ mcg/m³
Normal exercise conditions	• Highest "safe" level over a 24-hour period as per the NEPH	• All forms of exercise are encouraged. • Consider the previous 5 days air quality exposure for susceptible individuals • At risk individuals may still get increased symptoms and need to seek medical advice	<25
Moderate **Caution for those who are sensitive to air pollution**	• Epidemiological studies indicate increased risk of emergency presentations for asthma and breathing difficulty independent of exercise this increased risk may last 3 to 5 days • Short-term one-off exposure studies of non-susceptible people at concentrations including this level with moderate intensity exercise have not demonstrated clinically relevant changes	• Individuals who are sensitive to air pollution may need to alter their training to reduce their potential exposure. Prolonged high intensity endurance exercise (e.g., rowing, cycling, long-distance running) may need to be completed indoors. If exposure is unavoidable (ie scheduled match) defer the next exposure 3-5 days to allow recovery • Most individuals will tolerate exercise as normal, without symptoms.	25-50
Poor conditions for exercise	• For asthmatics and individuals sensitive to air pollution, expert opinion is it highly likely that any exercise in these conditions will contribute to worsening symptoms, increase use of reliever medications and need for medical review • Short-term on- off exposure studies of non-susceptible people at concentrations including this level with moderate intensity exercise have not demonstrated clinically relevant changes • It is unclear what effects repeated exposures prior to recovery have at these concentrations and how long it takes to recover. Estimated recovery time may be 3-5 days following high exposure	• It is recommended that individuals who are sensitive to air pollution limit their exposure as much as practical • It is highly recommended to complete prolonged high intensity endurance activities (e.g., rowing, cycling, long-distance running) in a facility with better air quality • Intermittent exercise (e.g., tennis, netball, beach volleyball, cricket) and power activities (e.g., sprint training, javelin training, jump training, rugby skills training) represent less risk than prolonged high intensity endurance activities. However, risk remains elevated above baseline and susceptible athletes should have a current asthma management plan and relevant medications accessible during the session • Non susceptible individuals may unexpectedly develop symptoms at these concentrations and should seek medical review early	51-150
Likely to be hazardous to exercise outdoors	• No population data describes prolonged exposure at these consequences • Expert opinion indicates the likely harm associated with exercising at or above this concentration will outweigh the potential benefits associated with exercise. • Expert opinion is that individuals who are not known to be sensitive (and therefore not prepared) to air pollution may become symptomatic.	• All efforts should be made to reduce smoke exposure as much as is practical • Reschedule events, relocate them indoors, shorten overall time outdoors etc. • Where there is an intention to play organised high level sport and there are medical staff on site to advise, these levels of pollution should trigger a discussion between medical staff and officials about the advisability or otherwise of proceeding with the event.	>150

Guidelines for exercise in smoke affected environments

The activity levels in smoke affected environments should be based on the air quality health category and the individual's history of smoke sensitivity. Of note is that there is significant variability in $PM_{2.5}$ across relatively short distances and rapid changes can occur. Those wishing to exercise should be aware of their own individual experience of sensitivity to smoke pollution and consider any safety aspects of visibility prior to participating in exercise. The Victorian Environment Protection Authority uses a visibility rating scale to advise on activity levels (**Table 4**).

Atmospheric visibility can be used as a proxy for air quality. Visibility can decrease with an increasing $PM_{2.5}$ levels. [19-21] The AIS strongly recommends however that where there is any decrement in visibility due to bushfire smoke, athletes, coaches and support staff should seek out and use atmospheric $PM_{2.5}$ levels to guide exercise levels, as per **Table 3**. Where live $PM_{2.5}$ levels are not available, an acute deterioration in visibility may be reasonable to use as rough guide as per **Table 4**.

All athletes who suffer from asthma should have an updated asthma management plan and consult their doctor prior to exercising in smoke-affected environments. Recent respiratory infection increases the risk for development of smoke-related symptoms, even in non-asthmatics. Moreover, smoke exposure increases the risk of respiratory infection. Increases in exercise intensity and duration result in increased airway exposure to polluted air. The AIS therefore, recommends modifying training or choosing training locations based on the exercise risk categories and exercise-specific recommendations according to atmospheric $PM_{2.5}$ concentration (**Table 3**) and activity levels recommendations from Victorian Environment Protection Authority based on visibility (**Table 4**).

Exposure to poor air-quality exerts both a delayed and cumulative effect on cardiorespiratory health (**Table 1**) which may, in turn, lower an athlete's threshold for symptoms. There is no research into the effects of repeated smoke exposure. This should be considered if a geographical region has been exposed to increased smoke for several days in succession. If athletes are not performing as expected in elevated $PM_{2.5}$ from bushfire smoke, this should be a trigger to evaluate their planned outdoor exercise and consider discussion with the team doctor.

TABLE 4:
Activity levels recommendations from the Victorian Environment Protection Authority based on visibility

Visible landmark	Air health category	Activity levels – people sensitive to smoke	Activity levels – everyone else
About 20 km	Good	It's a good day to be outside.	It's a good day to be outside.
About 10 km	Moderate	It's okay to be outside but watch for changes in air quality around you.	It's okay to be outside but watch for changes in air quality around you.
About 5 km	Poor	Reduce prolonged or heavy physical activity.	Normal activity, but be alert to changes in air quality
About 1.5 km	Very poor	Avoid physical activity outdoors.	Reduce prolonged or heavy physical activity.
Less than 1.5 km	Hazardous	If you can, stay indoors and keep physical activity levels as low as possible.	Avoid all physical activity outdoors.

SUMMARY STATEMENTS AND KEY CONCEPTS

Constituents of bushfire smoke air pollution

- Health and performance effects are influenced by the chemical components of pollution, the size and concentration of particles. $PM_{2.5}$ and/or PM_{10} concentrations are useful indicators of the severity of the bushfire smoke and the inherent level of risk.
- Bushfire smoke contains particulate matter, carbon monoxide, carbon dioxide, water vapor, hydrocarbons and other organic chemicals, nitrogen oxides, trace minerals.
- It is unclear from the current evidence how the toxicity of bushfire smoke compares to other sources of air pollution

Health effects and outcomes

- Some athletes are at an elevated risk for adverse health events of air pollution, including bushfire smoke, due to increased exposure from prolonged higher intensity physical activity and impaired visibility.
- Severity of health effects from bushfire pollution can be observed on a continuum.
- In periods of elevated bushfires smoke with $PM_{2.5}$ less then 25 ug/m^3 as depicted by the "green" zone, athletes should monitor for signs and symptoms for short term health outcomes such as irritated eyes, breathing difficulties and actively limit exposure to prevent escalating of severity of health effects.

Smoke inhalation can cause a spectrum of illness

- Smoke inhalation injury experienced by athletes may include a combination of any of chemical irritation of the upper and lower respiratory tract, asphyxiation and systemic toxicity from lung mediated absorption of smoke constituents.

Mechanism

- Airway inflammatory changes have been observed from short term exposure. This can exacerbate several health conditions such as asthma – a respiratory condition that is common in athletes.
- Exercise may increase the dose of bushfire smoke the lungs are exposed to through increases in tidal volume and airflows. Furthermore, breathing predominantly through the mouth during exercise increases the probability that particulate matter will deposit in deeper regions of the lungs.
- High intensity physical exertion can increase the dose of bushfire smoke that athletes are exposed to by 4 to 5 orders of magnitude compared to the same duration of rest.
- Asthmatic and airway sensitive individuals will likely experience symptoms with a pollution dose increase of only 1 order of magnitude of increased dose exposure. Given this increase is within the dose increase that exercise can contribute to, this population should monitor their symptoms and the current particulate matter ($PM_{2.5}$) concentrations.

Bushfire smoke and its impact on health and performance for athletes

- The athletic population like the general population will have a broad spectrum of tolerance to $PM_{2.5}$. One size does not fit all.
- While there is no "safe" level of $PM_{2.5}$, it is expected most people will be able to exercise without adverse health outcomes at $PM_{2.5}$ concentrations below 25 ug/m^3. Close monitoring of athletes is recommended as the PM concentration increases above this level.
- Considering the pyramid of health effects described by the American Thoracic Society, small athletic performance decrements (a subtle subclinical effect) are expected to be observed prior to overt symptoms.
- While there are plausible links to impaired physical performance in elevated bushfire smoke, the dose-response relationship between $PM_{2.5}$ and any performance decrement is yet to be established.

Factors to consider when assessing the safety of exercise in smoke

- Air quality information on State and Territory government websites is generally updated hourly. Therefore, there can be a lag between official measurements and what is occurring in real time. This can cause limitations when it comes to determining the air quality in your local environment. If smoke is affecting usual visibility within your area, it is likely that the air quality will fall into a higher risk category. Sporting clubs/organizations should consider investing in portable air quality monitors to enable measurement of $PM_{2.5}$ concentrations at their precise geographic location. Real time $PM_{2.5}$ levels can be used to inform rapid decision making.
- All athletes who suffer from asthma should have an updated asthma management plan and consult their doctor prior to exercising in smoke-affected environments.
- Increases in exercise intensity and duration result in increased airway exposure to polluted air. The AIS recommends modifying training or training locations based on **Table 3** above.
- Where there is no access to live $PM_{2.5}$ readings, **Table 4** above can be used as a approximate guide.
- Recent respiratory infection increases the risk for development of smoke-related symptoms, even in non-asthmatics. Moreover, smoke exposure increases the risk of respiratory infection.
- Consecutive days of exposure to polluted air can have a cumulative effect, lowering an athlete's threshold for symptoms. There is no research into the effects of repeated smoke exposure. This should be considered if your region has been exposed to increased smoke for several days in succession.

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