Effects of wildfire smoke exposure in cattle: a review of the current state of knowledge and future directions

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Abstract

Wildfires burn millions of acres of land annually in the United States and experts predict the number and intensity of wildfires to continue to increase. Beyond destruction of land, vegetation, and structures in the immediate path of the fire, wildfires emit a variety of particulates and other pollutants that are hazardous to breathe. Associations between exposure to wildfire particulates and many negative health outcomes, such as hospital admissions, respiratory disease, cardiovascular morbidity, and premature mortality, have been reported in humans. The western U.S. is particularly prone to wildfire breakouts and is home to more than 15 million beef and dairy cattle that are also exposed to wildfire smoke each year. Health and production issues related to wildfire smoke inhalation in cattle have not been thoroughly researched despite the susceptibility of cattle to respiratory disease and greater potential health risk from inhaled pollutants relative to other mammals. An emerging body of literature is, however, unequivocally demonstrating that wildfire smoke exposure is a threat to cattle health and performance. This review summarizes the current state of knowledge regarding the effects of exposure to wildfire particulate matter on cow and calf health and production. Implications of wildfire smoke exposure on the industry are also discussed.

Keywords: air quality, air pollution, pulmonary disease, reproduction, dairy cattle

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Introduction

Wildfires have become more frequent and severe over the past several decades, as exemplified by a number of recent high-profile conflagrations, such as the 2022 Calf Canyon blaze in New Mexico that burned an area larger than New York City and the 2021 Dixie fire that burned almost 1 million acres, becoming the second largest wildfire in California's history. Since the beginning of 2022, there have been more than 61,390 wildfire events in the U.S. resulting in 7.25 million acres burned (NICC, 2022). In the U.S., further increases in the number and intensity of large wildfires and a 10-30% extension in the duration of the wildfire season is expected (IPCC, 2014). Annual area burned by wildfires is also expected to increase by 76-152% by the end of the 21st century (Flannigan et al., 2006). Warmer temperatures combined with lower precipitation and earlier spring snow melts contribute to dry vegetation, providing ample fuel for wildfires to ignite and spread (IPCC, 2014).

In the U.S., wildfires occur often in the western states, home to more than 12 million beef cattle and 3.5 million dairy cattle that produce approximately 40% of the nation's milk (NASS, 2019). It is predicted that wildfires will disrupt the U.S. agricultural industry because of declines in crop and forage harvest and quality, and livestock health and production (USGCRP, 2016), although the economic consequences of wildfires on agriculture are currently unknown because of a lack

of research. It is estimated that economic losses just in the Utah cattle industry attributable to wildfires are over $1.4 million for an average fire year, related to premature cattle sale and necessity of feed purchasing because of scorched rangeland (Jakus et al., 2017). However, the actual costs are likely much higher as this estimate was for a single state and does not account for adverse effects of wildfire smoke on cattle productivity and health.

The detrimental effects of wildfires extend beyond the direct area burned and include the release of toxic gases and particulates into the atmosphere that are hazardous to breathe. Fine particulate matter ($PM_{2.5}$) is thought to be one of the greatest contributors to adverse health outcomes after wildfire smoke exposure (Black et al., 2017a). According to the U.S. Environmental Protection Agency, the total annual amount of $PM_{2.5}$ produced by wildfires was more than 3.3 million tons (U.S. EPA, 2020). Liu et al., (2015) estimated that exposure to PM from wildfire smoke will increase by 160% by the year 2051 in the U.S. As the number of wildfire occurrences and intensity increases, the likelihood of adverse health impacts is also expected to increase (USGCRP, 2016).

Inhalation of wildfire PM is associated with pulmonary disease and mortality in humans (Liu et al., 2015; Reid et al., 2016; Stowell et al., 2019), largely attributed to inflammation (Nakayama Wong et al., 2011). Cattle may be especially vulnerable to air pollutants owing to several unique characteristics of their pulmonary system (Veit and Farrell, 1978) and the inability of cattle to seek refugia from smoke. Indeed, ambient (i.e., non-wildfire) air pollution is associated with an increased risk of mortality in mature dairy cows and calves (Cox et al., 2016; Egberts et al., 2019). Further, research by our group found an increase in cow disease and calf mortality, and reduced milk yield when exposed to wildfire smoke PM (Anderson et al., 2020; Anderson et al., 2022).

This review will summarize the literature to date of the known consequences of wildfire smoke exposure on health, production, and mortality of cattle. This topic is particularly timely as large, catastrophic wildfires continue to increase in frequency in the U.S. and as animal producers are becoming more aware of the threats of wildfires to their agricultural operations (O'Hara et al., 2021). Because of the limited data available in cattle, we will also present research in other domesticated and free-ranging animals as well as studies rooted in human epidemiology to highlight remaining questions and areas of focus for future research endeavors involving cattle. While the focus of the review is specifically on wildfire-derived PM, relevant studies of non-wildfire PM sources and other air toxics are also discussed.

Wildfire smoke composition, dispersion, and toxicity

Wildfire smoke is a mixture of gaseous chemical compounds and particulate matter (PM) that reduce air quality (Michel et al., 2005; Wentworth et al., 2018). Chemical components of wildfire smoke include water vapor, carbon monoxide and dioxide, nitrogen oxides, sulfur dioxide, polycyclic aromatic hydrocarbons (PAH; e.g., formaldehyde, acenaphthene, napthelene, pyrene) and volatile organic compounds (VOC; e.g., benzene, ethylbenzene, toluene, xylenes), which are known neuromuscular toxins, teratogens, and carcinogens in humans (Urbanski et al., 2009; Wentworth et al., 2018; Sokolik et al., 2019; Miller et al., 2022). Particulate matter consists of liquid and solid particles that are categorized based on size. The

coarse particulates (PM_{10}) are smaller than 10 μm in diameter and fine particulates ($PM_{2.5}$) are smaller than 2.5 μm in diameter (Wilson and Suh, 1997). Wildfire emissions contain both PM_{10} and $PM_{2.5}$, but typically emit more $PM_{2.5}$ (Groß et al., 2013; Vicente et al., 2013).

Particulate matter is one of the most harmful pollutants in wildfire smoke. PM is inhaled into the respiratory tract where it can cause tissue inflammation and damage (Löndahl et al., 2007; Huttunen et al., 2012). $PM_{2.5}$ is particularly hazardous because it can penetrate deep into the respiratory tract and deposit into the lower airways and lungs (Carvalho et al., 2011). Additionally, $PM_{2.5}$ is capable of crossing the lungs to enter the blood (Fu et al., 2011; Schulze et al., 2017). PM in wildfire emissions can reach daily average concentrations that far exceed the established standards for ambient air quality (24-hour average of 35 $\mu g/m^3$ for $PM_{2.5}$ and 150 $\mu g/m^3$ for PM_{10} for human outdoor exposure; (U.S. EPA., 2013; Landis et al., 2018).

Wildfire smoke can travel great distances via air plumes, thus a single wildfire can reduce air quality in geographical locations far removed from the area burned (Stowell et al., 2019). The area affected by wildfire smoke can be 50 times larger than the areas that were directly burned by the fires (Knowlton, 2013). $PM_{2.5}$, because it is lighter than other particles, spends the longest amount of time in the atmosphere (~100 days), can travel farther distances, and therefore has a larger geographic distribution relative to other pollutants in wildfire smoke (U.S. EPA, 2010). Because of the capacity of wildfire smoke to disperse across vast distances, populations of humans and animals far from the wildfire may suffer from adverse health consequences (USGCRP, 2016).

Effects of wildfire smoke on mortality

Associations between wildfire smoke exposure and increased mortality risk in humans, including cardiovascular, pulmonary, and all-cause mortality, are well established. In a systematic review of 63 human epidemiological studies, 13 studies quantified mortality risk from wildfire smoke exposure, and of those, 9 studies reported elevated mortality rates upon exposure to wildfire smoke (Liu et al., 2015). Similarly, in a critical assessment of more than 300 studies on human health effects from wildfire smoke, positive associations between wildfire smoke exposure and mortality were found in most studies (Reid et al., 2016). Analyzing over 13 years of air quality data in Australia, including a total of 48 days affected by wildfire smoke, Johnston et al., (2011) found a 5% increase in human mortality on smoke-affected days.

Related data on mortality risk in cattle exposed to wildfire smoke are limited. In a preliminary study by our group, we collated data from two dairy farms in the Pacific Northwest across a five-year period, and found that mortality of dairy calves, but not cows, was greater on days when $PM_{2.5}$ concentration from wildfire smoke was elevated (Anderson et al., 2020). Similarly, specific human subpopulations, such as children, are more vulnerable to negative health outcomes from wildfire smoke than are adults (Shaposhnikov et al., 2014; Liu et al., 2015). Air pollution from non-wildfire sources is also associated with an increased risk of mortality in dairy cows and calves (Cox et al., 2016; Egberts et al., 2019). In mature dairy cows, ambient, non-wildfire-PM_{10}, ozone, and nitrogen dioxide concentrations were positively correlated with risk of mortality on the day of exposure and up to two weeks later, especially in the warm summer months (Cox et al., 2016). In a more recent analysis, Egberts et al., (2019) found acute and

cumulative effects of ambient, non-wildfire sources of ozone on risk of mortality in beef and dairy calves, young stock, and lactating cows in the warm season. As the chemical composition of wildfire smoke can be radically different from other sources of air pollution, and wildfire emissions can vary spacially and temporally within and across wildfire seasons, further research is warranted in cattle to assess links between mortality risk and wildfire smoke exposure.

Health effects from wildfire smoke inhalation

Exposure to wildfire smoke has been linked to impaired lung function, pulmonary disease, and pulmonary-specific hospitalizations in humans (Reid et al., 2016; DeFlorio-Barker et al., 2019; Stowell et al., 2019). In Washington state, increases in wildfire smoke $PM_{2.5}$ concentrations were positively correlated with increased risk of adverse respiratory outcomes, specifically in asthma hospitalization, chronic obstructive pulmonary disease hospitalization, and pneumonia cases (Gan et al., 2017). Assessing three years of health records in Colorado, Stowell et al., (2019) found that for every 1 $\mu g/m^3$ increase in wildfire-$PM_{2.5}$ there was a 10% increase in the risk of asthma and combined respiratory disease. Further, there was a rise in the number of cases of acute bronchitis and pneumonia after exposure to smoke from brushfires in Sydney, Australia between 1994 and 2014 (Morgan et al., 2010). These and other studies clearly point to an elevated risk of respiratory morbidity from wildfire smoke inhalation.

Respiratory illness is a leading cause of cow and calf deaths in the U.S. (USDA, 2017; Dubrovsky et al., 2019). In 2014, 12% of pre-weaned and 5% of weaned dairy calves were diagnosed with respiratory disease in the U.S. Among adult dairy cattle, on average 2.8% of cows (257,600 cows) were diagnosed with respiratory disease in 2014, 10.5% of which subsequently died and respiratory disease was cited for the removal of an average 2.1% of dairy cows across herds (USDA, 2017). Cattle are prone to respiratory disorders, such as Bovine Respiratory Disease, in part because of unique anatomical and physiological characteristics of the respiratory tract relative to other mammals (Veit and Farrell, 1978). The same characteristics that predispose cattle to respiratory infection may also contribute to greater susceptibility to inhaled PM. For example, cattle also longer trachea and bronchi, increasing pathogen (and presumably PM) retention time in the respiratory tract, which increases the likelihood of deposition (Kirschvink, 2008). Furthermore, the bovine respiratory system contains lower levels of lysozyme and fewer alveolar macrophages, which play a role clearing and breaking down inhaled pathogens and PM (Mariassy et al., 1975; Veit and Farrell, 1978; Lohmann-Matthes et al., 1994).

In a recent review of articles related to wildfires and health in cattle, no relevant peer-reviewed studies were found, leading the authors to conclude that wildfire smoke inhalation was not likely to have much impact on cattle (Eid et al., 2021). However, across two commercial farms in the U.S. Pacific Northwest, there was an increase in the number of mastitis and general illness cases among mature dairy cows on days when $PM_{2.5}$ from wildfires was elevated (Table 1; Anderson et al., 2020). In a further empirical study by our group, we found that immune cell populations, such as basophils and eosinophils in systemic circulation were higher, whereas neutrophils were lower, when cattle were exposed to wildfire $PM_{2.5}$ along with elevated air temperature and humidity (Anderson et al., 2022). Similarly, captive bottlenose dolphins housed in the San Diego Bay exposed to wildfire smoke in 2003 and 2007 had lower circulating neutrophils and elevated eosinophil counts during and 1 month following the fires (Venn-

Watson et al., 2013). However, these studies did not focus specifically on respiratory illness and thus, research is critically needed to characterize the pulmonary response to PM exposure in cattle.

Table 1. Summary of health and productive outcomes in cattle associated with exposure to gaseous and particulate matter pollutants

Exposure	Observed effect	Reference
Naturally occurring wildfire-$PM_{2.5}$	Increased incidence of mastitis and general illness in cows Increased calf mortality	Anderson et al., 2020
Naturally occurring wildfire-$PM_{2.5}$	Decreased milk yield and milk protein content	Anderson et al., 2022
Naturally occurring wildfire-$PM_{2.5}$ and elevated THI	Fewer circulating neutrophils, increased circulating eosinophils and basophils Initial decrease in blood urea nitrogen and increase in plasma non-esterified fatty acids. Opposite effects with subsequent exposure days	Anderson et al., 2022
Ambient (non-wildfire) PM_{10}	Increased relative risk of mortality in mature dairy cows	Cox et al., 2016
Ambient (non-wildfire) and wildfire $PM_{2.5}$	Decreased milk yield and increased SCC	Beaupied et al., 2022
Ambient (non-wildfire) ozone	Increased relative risk of mortality in beef and dairy calves, heifers, and mature cows	Egberts et al., 2019
PM_{10} in dust	Increased neutrophil count in lung lavage, increased risk of pneumonia	van Leenen et al., 2021
Naturally occurring wildfire-$PM_{2.5}$ and elevated THI	Decreased circulating concentration of total leukocytes, neutrophils, and eosinophils Increased eye discharge and coughing	Pace et al., 2022

$PM_{2.5}$ = fine particulate matter less than 2.5μm in diameter, PM_{10} = course particulate matter between 2.5 and 10 μm in diameter, THI = temperature-humidity index, SCC = somatic cell count

Effects of wildfire smoke exposure on inflammation

Accumulating evidence suggests that the pulmonary disease associated with exposure to wildfire-PM derive from systemic and local inflammatory responses. Production and release of leukocytes from bone marrow was elevated in healthy participants during the 1997 Southeast Asia wildfires compared to a month later after the smoke had dissipated (Tan et al., 2000). Healthy wildland firefighters had an increase in white blood cell count, peripheral blood mononuclear cell (PBMC) count, and serum concentrations of the pro-inflammatory cytokines interleukin (IL)-6 (IL-6) and IL-8, one day after fighting wildland fires compared to the day before exposure (Swiston et al., 2008). Similarly, cytokines, such as IL-1β and IL-6, which induce white blood cell production in bone marrow and production of acute phase proteins in liver, were higher in the blood of healthy individuals following acute exposure to wildfire-PM (van Eeden et al., 2001). Cytokine release plays an important role in initiating and regulating the inflammatory response and inducing neutrophil migration to the site of injury or infection (Ferreira et al., 2018).

A plethora of research also implicates local inflammation in respiratory disease following wildfire smoke exposure. In wildland firefighters, there was an increase in granulocytes, specifically neutrophils, in sputum samples collected the day after compared to the day before

the firefighters were combating an active wildfire (Swiston et al., 2008). Intratracheal instillation of wildfire-PM in mice resulted in inflammatory cell influx, including monocytes and neutrophils, to the lungs and evidence of lung damage within 24 h (Wegesser et al., 2009). A follow-up study by the same group reported an increase in the concentration of pro-inflammatory cytokines, such as TNF-α and IL-8, in lung lavage samples collected 6 h after instillation, indicating induction of a local inflammatory response (Wegesser et al., 2010). These studies indicate that exposure to wildfire emissions is associated with both local and systemic inflammation.

In cattle, there is currently little evidence to indicate that wildfire smoke induces an inflammatory response, but further research is needed in this area. Before and after exposure to wildfire smoke in dairy cattle, there were no detectable changes in circulating acute phase proteins, such as serum amyloid A and haptoglobin, which play a role in the inflammatory response (Anderson et al., 2022). Although a drop in neutrophil concentration in the blood of dairy cows and calves have been documented after wildfire smoke exposure, the mechanisms contributing to the decline are unknown (Anderson et al., 2022; Pace et al., 2022). Given that neutrophils migrate to the site of tissue injury and infection, it is possible that neutrophil declines in the blood after wildfire smoke inhalation are associated with migration to the lungs and local inflammation, as observed in humans and rodents (Swiston et al., 2008; Wegesser et al. 2009), but this hypothesis has yet to be tested in cattle.

Effects of wildfire smoke exposure on pathogen susceptibility

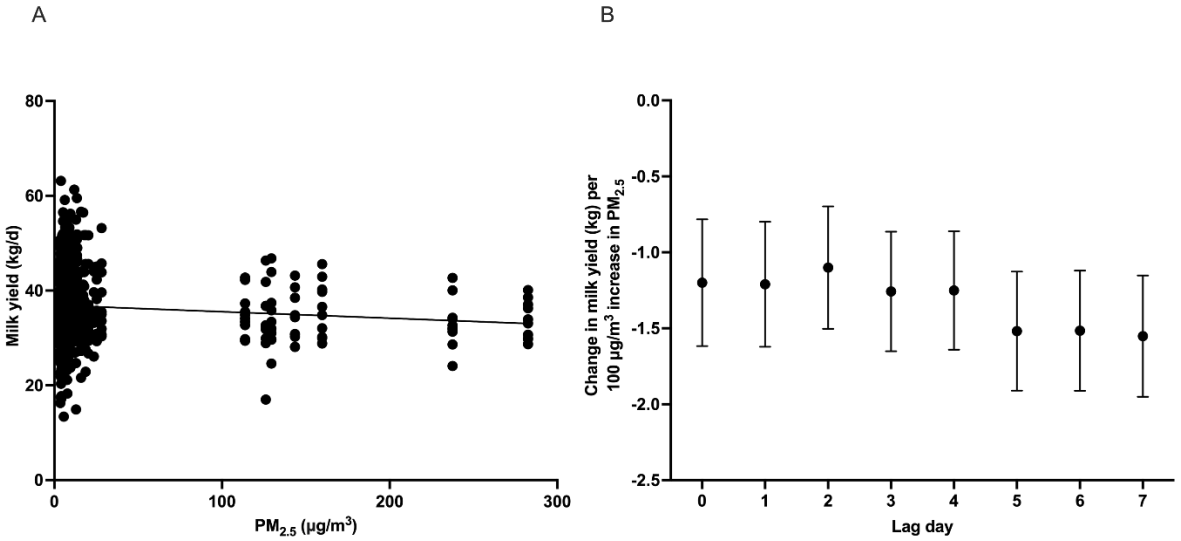
Inhalation of wildfire smoke increases the susceptibility of the respiratory system to microorganisms that contribute to disease. A retrospective study found that the number of influenza cases diagnosed during the winter was positively correlated with $PM_{2.5}$ concentrations during the previous summer wildfire season in western Montana (Landguth et al., 2020). Furthermore, Migliaccio et al., (2013) showed that the lungs of mice pre-exposed to wood smoke had a greater pathogen burden when inoculated with *Streptococcus pneumoniae* compared to mice that were not pre-exposed to smoke. Although specific effects of wildfire smoke exposure on pathogen invasion in cattle have not yet been explored, inadequate ventilation and poor air quality on farm, including elevated aerosolized bacterial counts and ammonia, are known risk factors for BRD development in cattle (Lago et al., 2006; Peek et al., 2018; Zhao et al., 2021). It is plausible that inhalation of toxic gases or PM in wildfire smoke increases cattle vulnerability to contracting viruses that contribute to BRD (Fulton, 2020). It is also worth noting that researchers recently discovered the transport of viable microbes through wildfire-smoke plumes; however, the impacts of inhaling smoke-microbes on human and animal health are largely unknown (Kobziar et al., 2018; Kobziar et al., 2019).

Effects of particulate matter on lactation performance

A few studies have investigated the impacts of PM and other air pollutants on milk production. Beaupied et al (2022) reported reduced milk yield and higher somatic cell count (SCC) in dairy cattle experiencing elevated ambient- (i.e., non-wildfire) and wildfire-PM concurrent with elevated air temperature and humidity index (THI). Our group found that for every 100 ug/m^3 increase in wildfire-PM, milk yield decreased by 1.2-1.5 kg/cow/day and milk protein concentration decreased by 0.14% independent of THI, and these effects persisted for at least 7

days after the last day of exposure to wildfire smoke (Figure 1; Anderson et al., 2022). Similarly, lactating ewes housed in a moderately ventilated showed a reduced exposure to PM and increased milk yield compared to that of a poorly ventilated barn (Sevi et al., 2003).

Figure 1. Relationship between $PM_{2.5}$ from wildfire smoke and milk production in early-lactation Holstein cows. Cows experienced 7 consecutive days of elevated $PM_{2.5}$ from wildfires. A) Milk yield was lower with increasing daily $PM_{2.5}$ concentration (P = 0.004). B) Change in average milk yield per cow per day for every 100 μg/m3 increase in $PM_{2.5}$ across lag d-0 (day of exposure) through lag d-7 (7 days after last exposure). From Anderson et. al., 2022.



The causal linkages between poor air quality and lactation performance are unclear but may be associated with interacting changes in metabolism, immune status, and feed intake. The immediate metabolic response to wildfire-PM exposure in lactating dairy cattle includes a decrease in blood urea nitrogen and an increase in plasma non-esterified fatty acids, which are reversed with continued exposure to elevated PM (Anderson et al., 2022). As the induction of an immune response is energetically demanding, it is also possible that immune activation in response to inhaled PM or secondary pathogen infection following PM exposure occurs at the expense of other energetically-demanding physiological processes such as milk production (Bird, 2019). Previous work showing positive correlations between wildfire-PM concentrations in the atmosphere and incidences of mastitis in dairy cows (Anderson et al., 2020) and increases in milk SCC with elevated PM (Beaupied et al., 2022) suggest that air pollutants contribute to or worsen mammary infection. Finally, changes in feed intake may precipitate or exacerbate metabolic shifts during exposure to wildfire-PM with a consequent reduction in milk yield, such as occurs in response to other environmental stressors (e.g., heat stress; Rhoads et al., 2009; Wheelock et al., 2010). Although loss of body condition and body weight occur during and following wildfire-PM exposure in dairy cows, feed intake has yet to be directly measured. Further research is warranted to investigate the physiological mechanisms underlying changes in milk synthesis during and following episodes of poor air quality in cattle.

Effects of wildfire smoke exposure during pregnancy and early postnatal life

Adverse environmental conditions experienced during critical windows of early development,

such as during the prenatal and postnatal period, can alter the developmental trajectory of the offspring (i.e., developmental programming), leading to lifelong or permanent impacts on physiology and health (Lucas, 1991; Barker et al., 2002). Developmental programming of offspring performance associated with prenatal exposure to PM has not been studied in cattle, but there are multiple reports in humans of premature birth and low birth weight in babies gestated under conditions of poor air quality (O'Donnell and Behie, 2013; Abdo et al., 2019; Zhu et al., 2019). For example, Abdo et al., (2019) reported a 13.2% increase in the likelihood of preterm birth with every 1 $\mu g/m^3$ increase in average wildfire smoke $PM_{2.5}$ experienced during the second trimester. Further, although there is evidence that low birthweight is a risk factor for the development of metabolic dysfunction and disease in adulthood (Barker et al., 2005), investigations of the long-term consequences of prenatal exposure to wildfire-PM are lacking both in cattle and humans.

Postnatal exposure to wildfire-PM impacts offspring physiology and health. In rhesus macaques, exposure to wildfire-$PM_{2.5}$ for 10 consecutive days during infancy decreased immune function and lung capacity at adolescence and adulthood relative to an unexposed group (Miller et al., 2013; Black et al., 2017b; Bassein et al., 2019). Furthermore, white blood cells collected from adolescent monkeys exposed to wildfire smoke early in life produced fewer cytokines, such as IL-6 and IL-8, in response to a lipopolysaccharide challenge relative to control monkeys, indicating long-term immune dysfunction (Miller et al., 2013; Black et al., 2014; Black et al., 2017b). In dairy calves, elevated wildfire-$PM_{2.5}$ concentrations in combination with elevated THI during the pre-weaning period were associated with changes in immune cell populations in systemic circulation including reduced total white blood cell, neutrophil, and eosinophil counts, indicative of infection or immune suppression (Pace et al., 2022). The calves also exhibited signs of respiratory and ocular irritation including increased ocular discharge and coughing. Furthermore, across a 5-year period and two dairy farms in the Pacific Northwest, dairy calf mortality was higher on days when $PM_{2.5}$ was elevated from wildfires. Other air pollutants, such as ozone from non-wildfire sources, also contribute to mortality risk in pre-weaned and weaned calves on the day of exposure and up to several weeks later (Egberts et al., 2019). Van Leenen et al., (2021) also documented increased neutrophil influx to the lungs and greater incidence of pneumonia in beef and dairy calves associated with elevated PM concentrations in dust in the calf barns. Future research is needed to assess long-term or permanent impacts of early life exposure to wildfire smoke on productivity and health in calves.

Conclusions

As wildfires continue to burn more intensely and across larger areas, the numbers of cattle exposed to, and affected by, wildfire smoke will continue to expand. This review summarized the current state of knowledge regarding the health and production impacts of wildfire-PM exposure in cattle and the many gaps in knowledge. Specific areas worthy of further exploration in cattle are pulmonary immune responses to inhaled wildfire-PM, opportunistic infections following wildfire-PM inhalation, and short and long-term phenotypic consequences from wildfire-PM exposure in utero and in early postnatal life. Armed with this information, preventative and responsive measures can be developed to aid producers and protect livestock herds.

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