

# education

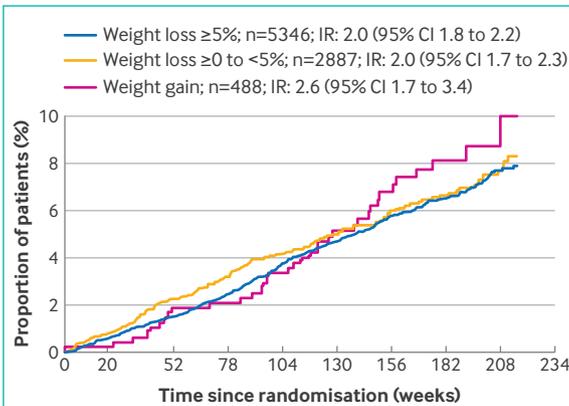
**RESEARCH REVIEWS** Fortnightly round up from the leading medical journals

## No benefit from carbocysteine or hypertonic saline in bronchiectasis



It's common for people with bronchiectasis to be prescribed carbocysteine or hypertonic saline to reduce symptoms and exacerbations. A new trial, funded by the National Institute for Health and Care Excellence, found that neither treatment reduced the incidence of exacerbations over a 52 week period in non-smokers with bronchiectasis, frequent exacerbations, and sputum production. Quality of life and adverse events were also similar between the intervention groups (carbocysteine, hypertonic saline, or both) and standard care.

• *N Engl J Med* doi:10.1056/NEJMoa2510095



Cumulative incidence plots showing time from randomisation to first major adverse cardiovascular event by body weight loss at week 20 in the semaglutide group of the SELECT study

## Unpicking the cardiovascular benefits of semaglutide

You might expect that the biggest reductions in cardiovascular risk in people taking semaglutide would occur in those who lose more weight. However, a secondary analysis of the SELECT trial suggests otherwise. It found that those with higher levels of weight loss (over 5%) after 20 weeks treatment with semaglutide didn't have a lower subsequent risk of major adverse cardiovascular events than those with less weight loss (0-5%). Those with greater reductions in waist circumference ( $>8$  cm) did have lower subsequent cardiovascular risk, but this only accounted for about a third of the observed benefit.

• *Lancet* doi:10.1016/S0140-6736(25)01375-3

## Cancer rates in younger people

News headlines often raise concern about increased rates of cancer in younger people. A surveillance study using the International Agency for Research on Cancer's GLOBOCAN database calculated the average annual percentage change (AAPC) in cancer incidence rates for 13 types of cancer across 42 countries and 14 years (2003-2017). Although cancer incidence rates have increased for several cancer types in younger adults (defined as aged 20-49), these were typically matched by increased incidence rates in older people. The one exception was colorectal cancer, where the increased rates of diagnosis in younger adults was greater than that in older adults in 69% of countries studied (median AAPC 1.66% v 0.37%).

• *Ann Intern Med* doi:10.7326/ANNALS-24-02718

DEANFIELD J, LINCOTT AM, KAHN SE, ET AL. *LANCET* DOI:10.1016/S0140-6736(25)01375-3

## CLINICAL PICTURE



## Erythematous erosive nasolabial lesion

A man in his early 60s presented with a four year history of an erythematous and erosive lesion to the left nose and upper lip. The lesion was not tender or pruritic. It was associated with intermittent purulent discharge and had gradually progressed despite treatment with topical corticosteroids. The patient reported progressive nasal congestion. Systemic examination was unremarkable, with no evidence of hepatosplenomegaly, lymphadenopathy, or cutaneous hyperpigmentation. Laboratory test results showed normal complete blood count, normal

hepatic and renal function, and negative HIV serology. Nasal endoscopy demonstrated mucosal erosion. Nasopharyngeal biopsy showed lymphoid tissue hyperplasia and granulomatous inflammation. Target enriched next generation sequencing detected *Leishmania infantum* (read counts: 374 and 495), and results from subsequent rK39 serological testing were positive, confirming the diagnosis of mucocutaneous leishmaniasis. The patient was treated with sodium stibogluconate, resulting in complete healing without recurrence on follow-up 10 months after treatment.



## Full time for GPs

An average session of work for general practitioners in England in 2021 lasted 6.2 hours, according to survey data published in 2024 (*Br J Gen Pract* 2024, doi:10.3399/BJGP.2023.0432). On that basis, any GP who attempts to work nine sessions a week—the traditional definition of full time—would be working for more than 55 hours. A new cross-sectional study estimates that a full time primary care physician in the Boston area of the United States works 61.8 weekly hours, around 1.7 hours per patient per year. It seems that the workload in the US isn't any better, although the remuneration—on average well above \$200 000 [£150 000, €170 000]—probably is.

• *Ann Intern Med* doi:10.7326/ANNALS-25-01412

## The sound of silence

Offer me either two hours listening to slow-tempo instrumental music a day or listening to silence and I'd definitely choose the latter. These were the intervention and control arms of a multicentre trial examining whether slow tempo music might reduce delirium in people undergoing mechanical ventilation. No difference in delirium duration or severity, pain, or anxiety symptoms were found between the two groups after seven days.



• *JAMA Intern Med* doi:10.1001/jamainternmed.2025.5263

Tom Nolan, clinical editor, *The BMJ*, London; sessional GP, Surrey

Cite this as: *BMJ* 2025;391:r2253

Leishmaniasis is a vector borne parasitic disease transmitted by the bite of infected sand flies. Different species of the parasite are associated with varied clinical manifestations including mucosal, cutaneous, and visceral leishmaniasis. Climate change is thought to be affecting the geographic distribution of the disease, with changes in temperature, rainfall, and humidity enabling the establishment of sand flies in previously non-endemic territories.

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Patient consent obtained.

Cite this as: *BMJ* 2025;391:e085967

## MINERVA From the wider world of research

### Employment after bariatric surgery

Although the benefits of bariatric surgery for health are well established, outcomes concerning work and employment remain in doubt. A systematic review (42 studies from 15 countries) finds that, in the short term, employment rates rise and that absenteeism and sick leave fall (*JAMA Surg* doi:10.1001/jamasurg.2025.3611). Disappointingly, these improvements don't seem to be sustained. Employment rates had dropped to pre-surgery levels within five years.

### Undiagnosed G6PD deficiency

Glucose-6-phosphate dehydrogenase deficiency is a sex linked genetic condition, common in black and Asian populations. Often asymptomatic and undiagnosed, it's an occasional cause of haemolysis after exposure to anti-malarial drugs or sulphonamides. Less well known is that, even in the absence of an overt haemolytic trigger, red cell turnover may be increased, which in turn leads to lower levels of HbA<sub>1c</sub>. Data from two longitudinal studies show that people with G6PD deficiency are more likely to experience delay in diagnosis of diabetes and a higher risk of microvascular complications (*Diabetes Care* doi:10.2337/dc25-0556).

### Magnets

Small magnets are easily swallowed by children either unintentionally or deliberately. A survey of foreign body ingestion from the UK documented more than 300 cases over a year of observation (*Arch Dis Child* doi:10.1136/archdischild-2024-328195). The most common source of the magnets was toys. No child who ingested a single magnet came to harm, but in children who swallowed more than one, surgery was needed in around 10%.



### Stable cortical maps after limb amputation

It's generally thought that when the brain loses a large sensory input after, say, the amputation of a limb, extensive

neural reorganisation occurs so that the relevant area of somatosensory cortex becomes responsive to inputs from neighbouring body parts. A longitudinal study of three adults before and after planned amputation of an arm suggests otherwise (*Nat Neurosci* doi:10.1038/s41593-025-02037-7). Functional magnetic resonance imaging showed persistence of cortical hand maps, which could be activated by voluntary phantom limb movements. Despite its physical absence, the neural representation of the amputated limb seemed to remain intact.



### Prematurity and hypertension

Infants born prematurely are more likely to develop hypertension in childhood and adolescence than infants born at term, according to longitudinal data from the Boston Birth Cohort (*JAMA Netw Open* doi:10.1001/jamanetworkopen.2025.27431). The risk was higher if they had required intensive care, and higher still if they had also experienced prematurity-related complications such as sepsis, respiratory difficulty, necrotising enterocolitis, or intraventricular haemorrhage.

### Aortic stenosis

Optimum timing of valve replacement in asymptomatic patients with aortic stenosis must balance the long term benefits of early intervention against the short term harms of the operation. A meta-analysis (four trials, 1500 patients) provides helpful data (*J Am Heart Assoc* doi:10.1161/JAHA.125.041283). Early surgery halved the incidence of a composite endpoint of mortality, stroke, and myocardial infarction when compared with conservative management. Composite outcomes sometimes hide important caveats, but here the individual components were all improved by early intervention.

Cite this as: *BMJ* 2025;391:r2256

# Effects of extreme heat on physiology, morbidity, and mortality under climate change: mechanisms and clinical implications

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This is a summary of Clinical Review. The full version can be read here: <https://www.bmj.com/content/391/bmj-2025-084675>



Extreme heat events have increased in frequency, duration, and severity because of climate change.<sup>1</sup> Heat exposure poses serious threats to human health, driving elevated risks of morbidity and mortality by inducing multisystem physiological dysfunction.<sup>3 4</sup> Understanding the multifaceted effects of heat exposure under climate change is crucial for minimising its detrimental effects on population health.

## Mechanisms of multisystem damage from heat exposure

When exposed to heat, human physiology initiates adaptive responses via two primary mechanisms: cutaneous vasodilation to enhance core to surface heat dissipation and increased sweating to promote evaporative cooling.<sup>1</sup> Specifically, the hypothalamus regulates body temperature by integrating peripheral temperature receptors distributed in the skin, core organs, and spinal cord, enabling real time thermoregulatory adjustments.<sup>3 9</sup> The endocrine system contributes to heat adaptation through the release of hormones such as antidiuretic hormone, aldosterone, and cortisol, and activation of the autonomic nervous system exerts systemic effects.<sup>12 40</sup> However, extreme heat exposure can overwhelm these compensatory thermoregulatory mechanisms, leading to systemic damage.

### WHAT YOU NEED TO KNOW

- Climate change is escalating the frequency and severity of extreme heat events, augmenting disease burden through heat exposure
- This review summarises the multifaceted mechanisms by which heat exposure induces systemic and organ-specific damage. These mechanisms drive shifts in disease profiles towards acute heat related illnesses, cardiovascular diseases, renal disorders, and other conditions, particularly affecting vulnerable groups
- Susceptibility to heat exposure spans the entire life course, from prenatal stages to old age, and is amplified by socioeconomic disparities
- The review proposes initiatives to reduce negative health outcomes and advocates for the integration of heat exposure into clinical practice guidelines, to safeguard public health in an era of unprecedented thermal challenges.

Heat induced fluid loss leads to electrolyte imbalances.<sup>41</sup> Thermal stress triggers systemic inflammation, characterised by elevated concentrations of pro-inflammatory cytokines.<sup>11 42</sup> Concurrently, oxidative stress activates protective pathways, notably the up regulation of heat shock proteins, which preserve protein integrity, inhibit apoptosis, and enhance thermotolerance.<sup>43 44</sup> The underlying mechanisms of heat induced damage vary across organs, leading to diverse patterns of functional impairment and disease risks.

### Cardiovascular system

Heat exposure elevates heart rate and blood flow, leading to higher cardiovascular workload. Cutaneous vasodilation increases cardiac output, requiring the heart to pump harder and faster, thereby increasing oxygen demand in coronary tissues. Elevated core temperature and resultant volume depletion trigger sympathetic nervous system activation to maintain blood pressure by increasing heart rate and stroke volume.<sup>46</sup> In people with pre-existing cardiovascular disease, this response may precipitate demand ischaemia or plaque rupture.<sup>47</sup> Concurrently, heat induced electrolyte imbalances can disrupt myocardial ion channel function, impairing electrical stability and increasing the risk of arrhythmia.<sup>48</sup>

Extreme heat causes myocardial injury through multiple mechanisms, including cardiomyocyte apoptosis, oxidative damage, and myocardial ischaemia, thereby impairing endothelial function.<sup>49 50</sup> Hyperthermia induced dehydration leads to haemoconcentration and a hypercoagulable state, synergistically increasing the risks of thrombosis and myocardial infarction.<sup>46 48</sup> Chronic heat exposure also disrupts lipid metabolism by increasing low density lipoprotein and decreasing high density lipoprotein concentrations, thereby promoting dyslipidaemia.<sup>53</sup>

### Respiratory system

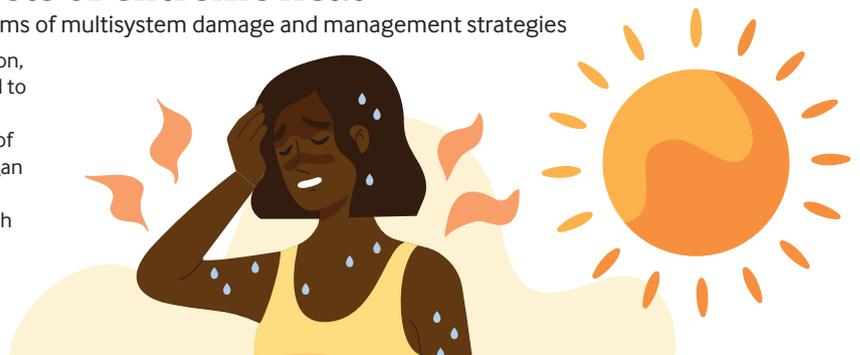
Heat derived lung damage, manifesting as pulmonary oedema and acute respiratory distress syndrome, coupled with pulmonary stress due to heat related hyperventilation, significantly contributes to respiratory dysfunction.<sup>57</sup>

Heat exposure has been associated with decreased pulmonary function, particularly among people with chronic respiratory diseases. Beyond exacerbating chronic respiratory diseases, heat exposure also heightens the risk of respiratory infections through dysregulation of pulmonary immunity. Chronic heat exposure increases susceptibility to respiratory pathogens through depletion of alveolar macrophages and consequent local immune dysfunction.<sup>67</sup>

# Impacts of extreme heat

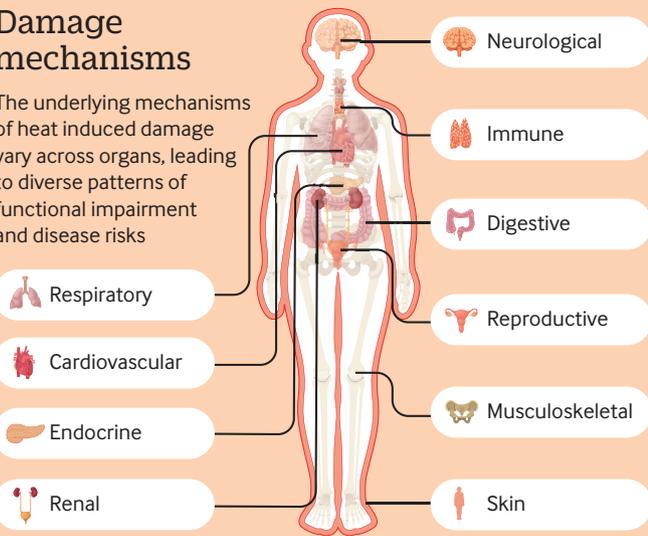
Mechanisms of multisystem damage and management strategies

Extreme heat events have increased in frequency, duration, and severity because of the climate crisis. Clinicians need to be prepared for the increasing impacts on heat sensitive conditions. This graphic summarises the health impacts of heat exposure through multifaceted mechanisms for organ specific damage. It also introduces individual and community level initiatives for a coordinated public health response, and outlines essential clinical management strategies for both acute heat related illnesses and chronic conditions exacerbated by extreme heat



## Damage mechanisms

The underlying mechanisms of heat induced damage vary across organs, leading to diverse patterns of functional impairment and disease risks



## Shifts in disease spectrum and burden



Historical data reveal a marked increase in heatwave related mortality over time, and future projections under various climate scenarios suggest a continued rise in health risks

Future climate change is projected to shift the overall disease spectrum toward higher morbidity and mortality from heat sensitive diseases



Heat exposure affects individuals across all stages of life, with varying vulnerabilities due to factors including:

Physiological characteristics

Behavioral patterns

Social determinants

## Clinical recommendations

### Acute heat exposure

Rapid recognition and management of acute heat related illnesses are critical. Clinicians should proficiently distinguish between:



### Cooling strategies, such as:

- Ice water immersion (1-5°C)
- Cold water immersion (9-12°C)

Assess and manage complications

Continuous monitoring of multi-organ function

### Managing chronic conditions

During patient consultations, heat exposure history can be routinely incorporated into clinical assessments

#### Pharmacological modifications

Medications used for chronic diseases may impair thermoregulatory capacity. Therefore, adjustments in dosage and administration timing may be necessary

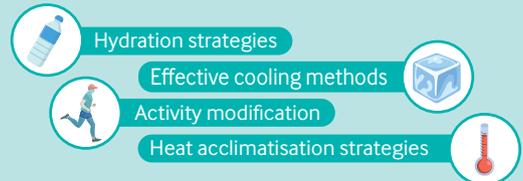
#### Personalised heat health management

Tailored to account for individual pathophysiology and comorbidities

## Adaptation strategies

### Individual strategies

Practical adaptation behaviours can help people to mediate heat related health risks. These measures vary in their affordability and effectiveness, and may be more or less appropriate for different people in different circumstances



### Community based interventions

These range from population-wide policies and guidelines, through to targeted, localised initiatives.



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### Renal system

Extreme heat exposure leads to dehydration, not only reducing circulatory renal blood volume but also impairing glomerular filtration rate, potentially progressing to acute kidney injury or even renal failure.<sup>68</sup> Acute kidney injury, often resulting from renal hypoperfusion and hypoxia, is a common complication of heatstroke. Reduced renal blood flow impairs delivery of nutrients to tubular cells and triggers inflammation and necrosis.<sup>71</sup>

Evidence suggests that recurrent acute kidney injury, triggered by heatstroke, may contribute to development of chronic kidney disease.<sup>72,73</sup> Clinical findings indicate that heat and dehydration promote the production of concentrated and acidic urine, which predisposes to urate crystallisation.<sup>75</sup> Elevated urate concentrations also increase synthesis of reactive oxygen species and promote endothelial damage, further exacerbating progression of chronic kidney disease.<sup>78</sup> Importantly, people with pre-existing chronic kidney disease, particularly those undergoing dialysis, are at increased risk during heat exposure owing to impaired thermoregulation.<sup>79</sup>

Reduced renal blood flow associated with heat exposure activates the renin-angiotensin-aldosterone system, which not only exacerbates renal vasoconstriction but also triggers cardiovascular events among patients with chronic kidney disease.<sup>68,80</sup> Urine concentration and acidification increase calcium supersaturation, promoting crystallisation and stone formation, thereby raising the risk of nephrolithiasis.<sup>81</sup> Reduced urine output caused by dehydration further impairs bacterial clearance and enhances susceptibility to urinary tract infections.

### Endocrine system

Heat stress stimulates the hypothalamic-pituitary-adrenal axis, increasing the release of cortisol, which closely correlates with acute rises in core temperature.<sup>83</sup> Thyroid hormones, which regulate metabolic rate, are also influenced by ambient temperature.<sup>12</sup> Epidemiological and animal studies both indicate that heat stress may suppress plasma concentrations of thyroid stimulating hormone, tetraiodothyronine, and triiodothyronine.<sup>84,85</sup> Moreover, heat exposure can impair the peripheral conversion of tetraiodothyronine to triiodothyronine, with the resultant low triiodothyronine concentrations aiding thermoregulation by lowering metabolic heat production.<sup>12</sup>

Heat stress activates the renin-angiotensin-aldosterone system, increasing aldosterone, and stimulates temperature sensitive arginine vasopressin release.<sup>68</sup> Elevated concentrations of aldosterone and arginine vasopressin contribute to the regulation of water balance by reducing sodium and water excretion, respectively.<sup>86,87</sup> Epidemiological evidence also shows a dose dependent association between ambient heat exposure and disrupted glucose homeostasis.<sup>88</sup> Heat induced insulin resistance mainly arises through two pathways: dehydration directly impairs insulin signalling,<sup>89</sup> and reduced brown adipose tissue activity increases fatty acid flux to metabolically active tissues such as skeletal muscle, thereby decreasing insulin mediated glucose uptake.<sup>20</sup>

### Neurological system

Heatstroke can cause cerebral injury through metabolic encephalopathy, vasogenic oedema, ischaemic hypoxia, and hypernatraemic encephalopathy, leading to acute neuropsychiatric disturbances such as delirium, ataxia, and coma.<sup>22</sup> Heat stress also elicits neuroinflammation, thereby exacerbating cerebral injury.<sup>90</sup> Hyperthermia further damages cerebrovascular endothelial cells, compromising the integrity of the blood-brain barrier. Subsequent leakage of serum proteins and inflammatory mediators exacerbates cerebral oedema, culminating in elevated intracranial pressure.<sup>93</sup>

Peripheral vasodilation induced by thermal stress diverts cerebral perfusion, impairing neurovascular coupling and precipitating hypoperfusion related cognitive deficits. Simultaneously, heat stress amplifies neuronal energy demands during cognitive processing and accelerates depletion of metabolic reserves.<sup>94,95</sup> In addition, hyperthermia suppresses synthesis and release of 5-hydroxytryptamine, attenuating its neuromodulatory effects on emotional and cognitive processing. Heat induced dopaminergic dysfunction may further exacerbate neuropsychiatric comorbidities.<sup>96,97</sup> Extreme heat also disturbs sleep architecture, subsequently impairing cognition and emotional regulation.<sup>99</sup>

### Digestive system

Hyperthermia directly damages intestinal mucosal integrity through structural disruption of epithelial tight junction proteins.<sup>100</sup> The resulting increase in gut permeability enables the translocation of endotoxins and inflammatory mediators into the systemic circulation, amplifying systemic inflammation.<sup>101,102</sup> The increased pro-inflammatory cascades can damage other organs, including the pancreas and liver.<sup>103</sup>

Redistribution of blood flow diverts perfusion from the gut to the skin, leading to intestinal hypoperfusion.<sup>105</sup> Additionally, heat stress disrupts the hypothalamic-pituitary-adrenal axis and parasympathetic signalling, suppressing intestinal peristalsis and prolonging retention of digestive enzymes.<sup>106,107</sup>

Additionally, hyperthermia disrupts the gut microbiome and increases susceptibility to gastrointestinal infections and inflammation.<sup>111,112</sup> Notably, extreme heat exacerbates water contamination by accelerating the proliferation of pathogens, thereby increasing the incidence of infectious diarrhoeal diseases.<sup>4,113</sup>

### Immune system

Emerging evidence links sustained environmental heat exposure to immune cell activation and the systemic release of pro-inflammatory cytokines.<sup>114</sup> Additionally, heat induced increases in gut permeability facilitate the translocation of endotoxins into the systemic circulation, further amplifying pro-inflammation.<sup>115,116</sup>

Heat exposure also disrupts immune function through the endocrine-immune axis. Activation of the hypothalamic-pituitary-adrenal axis increases cortisol, which induces apoptosis of T and B lymphocytes, reduces neutrophil and natural killer cell activity, and promotes polarisation of M2 macrophages.<sup>119,120</sup> Consequently, prolonged heat stress leads to immunosuppression, characterised by impaired pathogen clearance and reduced immune cell recruitment to infection sites, thereby increasing susceptibility to pathogens.<sup>118,119</sup>

## Reproductive system

Heat exposure can decrease plasma testosterone and diminish fertility.<sup>12</sup> Human spermatogenesis is temperature sensitive, with scrotal hyperthermia decreasing sperm output and quality.<sup>122</sup> In women, heat exposure can suppress the hypothalamic-pituitary-ovarian axis, lower serum oestradiol and progesterone concentrations, and disrupt menstrual cyclicality.<sup>12</sup> High temperatures induce excessive reactive oxygen species production, causing DNA damage that compromises embryo viability, blastocyst formation, and implantation potential.<sup>125</sup>

Thermal stress reduces uterine perfusion, leading to placental hypoxia and lysosomal instability, which stimulates prostaglandin synthesis.<sup>35</sup> Prostaglandins contribute to myometrial contractility and cervical ripening, whereas elevated oxytocin concentrations induce rhythmic uterine contractions.<sup>126</sup> Dehydration induced hypovolaemia activates the Henry-Gauer reflex, stimulating posterior pituitary release of antidiuretic hormone, which can synergistically amplify uterotonic effects.<sup>127</sup> Extreme heat also impairs placental perfusion and induces localised hypoxia, leading to decidual ischaemia and necrosis.<sup>128 129</sup>

Extreme heat exposure during pregnancy can disrupt maternal thermoregulation, activating the sympathetic nervous system and increasing vasoconstrictive reactivity,<sup>133</sup> which may lead to gestational hypertension.<sup>127 134</sup> This can impair uteroplacental perfusion, resulting in intrauterine growth restriction.<sup>135</sup>

## Musculoskeletal system

Dehydration leads to depletion of critical electrolytes, disrupting cellular membrane potentials and resulting in neuromuscular dysfunction that manifests clinically as muscle cramps, weakness, and compromised physical capacity.<sup>140</sup> Additionally, heatstroke causes skeletal muscle membrane damage, subsequently leading to the release of myoglobin and creatine kinase and ultimately triggering rhabdomyolysis.<sup>141</sup>

## Skin

The skin plays a critical role in thermoregulation through vasodilation and eccrine sweating.<sup>143</sup> Miliaria, a common and typically mild heat related disorder, exemplifies direct cutaneous responses to excessive heat.<sup>144</sup> Excessive sweating during heat exposure can lead to skin erythema, maceration, and exacerbation of pre-existing dermatological conditions.<sup>145 146</sup> Elevated temperatures may also promote bacterial and fungal growth, increasing the risk of cutaneous infections and autoinflammatory skin disease.<sup>147 148</sup>

## Interconnections

Critically, these multisystem damage mechanisms do not occur independently but through interconnected crosstalk. For instance, heat induced activation of the hypothalamic-pituitary-adrenal axis results in elevated cortisol concentrations, which in turn leads to immunosuppression.<sup>149</sup> The enhanced gut mucosal permeability triggers systemic inflammation, further exacerbating other organ injuries.<sup>101</sup> People with chronic kidney disease are particularly vulnerable to increased cardiovascular strain during heat exposure.<sup>150</sup> Recent evidence suggests that extreme heat exposure may induce epigenetic modifications and DNA damage.<sup>151 152</sup>

Specifically, heat exposure in one generation can result in accumulation of risk, increasing the susceptibility of subsequent generations to heat exposure.

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## Susceptibility of vulnerable populations

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### Life course vulnerability to heat exposure

Heat exposure affects individuals across all stages of life, with varying vulnerabilities owing to factors including physiological characteristics, behavioural patterns, and social determinants.

*Prenatal period and infancy*—The embryonic and fetal stages represent critical developmental windows, characterised by rapid cellular proliferation and organogenesis, during which increased susceptibility to heat stress occurs.<sup>155</sup> A US multicentre case-control study in 5848 cases of congenital heart defect and 5742 controls associates maternal heat exposure during post-conception weeks 3-8 with increased risk of congenital heart defects.<sup>156</sup> Infants, as a result of immature thermoregulatory systems and higher surface-area-to-mass ratios, absorb more heat and experience a faster rise in body temperature under hot conditions.<sup>157</sup> Although sweating enables some evaporative cooling, it also accelerates dehydration, which is often more severe in infants.<sup>157</sup>

*Children and adolescents*—Children are more frequently exposed to heat owing to increased outdoor activities, and their underdeveloped thermoregulatory systems make them less capable of coping with thermal stress.<sup>158</sup> Adolescents, likewise, often participate in outdoor activities, and the physiological stress of heat exposure can trigger robust autonomic responses to heat, a key manifestation of which is hyperhidrosis.<sup>12</sup> Beyond physiological effects, heat exposure may also adversely affect adolescents' behavioural and mental health.<sup>159</sup>

*Adults*—In adults, occupational heat exposure is an important contributor to heat related health risks. In male adults, who predominantly comprise occupational heat exposed groups, androgen associated traits such as sweat rates potentiate the risk of dehydration under humid conditions.<sup>157</sup> Female adults face elevated vulnerability during specific physiological states. During pregnancy, rising progesterone elevates core temperature and, together with increased maternal metabolism and fetal heat production, heightens maternal susceptibility to heat stress.<sup>35</sup> In menopausal women, reduced oestrogen impairs heat dissipation by inhibiting cutaneous vasodilation, increasing vulnerability to heat related cardiovascular strain.<sup>163</sup>

*Older adults*—Among older adults, age related declines in sweat gland function reduce sweating capacity.<sup>157</sup> Chronic conditions and medications further exacerbate vulnerability;<sup>157</sup> for instance, hypertension impairs vasodilation, and diabetes compromises sweating via microvascular dysfunction. Additionally, declining physical and cognitive functions hinder the ability to effectively respond to heat stress.

### Sociodemographic factors related to heat effects

Sociodemographic factors fundamentally shape heat exposure, response capacity, and underlying vulnerability. People with lower education and income levels often face higher risks

of heat related morbidity and mortality.<sup>166</sup> Lower educational attainment is associated with reduced awareness of heat risks and preventive behaviours, and limited income restricts access to essential cooling resources.<sup>167</sup> Ethnic minorities often bear disproportionate burdens owing to intersecting social, economic, and cultural disadvantages.

Although populations in tropical regions may show some degree of physiological adaptation to heat, they remain vulnerable, particularly where infrastructure is insufficient for heat adaptation.<sup>170 171</sup> Rural areas often lack access to cooling resources and healthcare services, whereas urban populations, although benefiting from better infrastructure, are increasingly affected by urban heat island effects.<sup>172</sup> Within cities, socioeconomic inequalities are evident: low income communities can experience temperatures up to 5°C higher than wealthier neighbourhoods, largely owing to dense housing and limited green space.<sup>3</sup> In slums and refugee shelters, inadequate ventilation, overcrowding, and heat absorbing materials such as corrugated metal roofing further amplify indoor heat exposure.<sup>173</sup>

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## Synergistic threats

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The health impacts of extreme heat are rarely isolated; rather, they are often exacerbated by co-occurring environmental and socioeconomic stressors that are being reshaped by climate change. Understanding synergistic threats is essential for assessing the full spectrum of heat related health burdens, and insights into the biological mechanisms enable a deeper interpretation of environmental co-exposure effects.

**Interactions between ambient heat, air pollution, and allergens**  
Climate change and air pollution are closely interconnected, as fossil fuel combustion, the primary driver of climate change through greenhouse gas emissions, is also a major source of air pollutants.<sup>174</sup> Extreme heat exacerbates ambient air pollution, particularly ground level ozone and particulate matter, through enhanced photochemical reactions and stagnant atmospheric conditions.<sup>175 176</sup> Strong evidence shows that extreme heat and air pollution act synergistically to elevate health risks.

Climate change also alters the distribution and concentration of airborne allergens such as pollen and mould.<sup>181</sup> Rising temperatures and elevated CO<sub>2</sub> concentrations not only prolong pollen seasons but also enhance the allergenic potency of pollen.<sup>182</sup>

### Amplification effect of extreme weather events

Other extreme weather events, including heavy precipitation events, floods, storms, droughts, and wildfires, often compound the physiological stress of heat exposure.<sup>184</sup> Floods create stagnant water, elevating risks of infectious diseases when followed by extreme heat.<sup>185 186</sup> Additionally, extreme heat increasingly overlaps with wildfire smoke, which releases large quantities of particulate matter and toxic gases.<sup>184 187</sup> Prolonged drought intensifies thermoregulatory strain by elevating cardiac workload and electrolyte depletion.<sup>188 189</sup> Beyond direct health effects, compound drought-heatwaves also trigger secondary disasters, including wildfires and agricultural losses that worsen malnutrition, especially in economically disadvantaged regions.<sup>190 191</sup>



MIGUEL RIOPA/AFP/GETTY IMAGES

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## Initiatives to reduce negative health outcomes due to heat exposure

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### Individual adaptation measures

Individual adaptation measures primarily focus on enhancing knowledge about heat health and facilitating practical adaptation behaviours.<sup>197</sup> These measures vary in their affordability and effectiveness. For instance, although the use of electric fans can reduce heat strain and improve thermal comfort, these devices accelerate body heating and increase physiological heat strain when used at temperatures above 45°C with low humidity.<sup>167</sup> The use of air conditioning is effective at promoting thermal comfort, but the cost of operating these units is an important barrier.<sup>198 199</sup> The use of ice towels and similar devices reduces heat strain, but they can become ineffective within 30 minutes as the ice melts. For the treatment of heatstroke, clinical guidelines recommend active cooling methods, especially cold water or ice water immersion to achieve the fastest cooling rate.<sup>200</sup> Self-pacing and taking regular breaks from work can be an effective strategy to reduce metabolic heat production and lower body temperature, particularly in situations where other cooling strategies cannot be implemented.<sup>167</sup>

### Community based interventions

**Heatwave early warning systems**—Heatwave early warning systems are widely implemented globally to provide communities with advanced notice of predicted heatwaves on the basis of location-specific temperature thresholds, along with the communication of graded alerts, enabling timely implementation of adaptive measures.<sup>197 214 215</sup> Elements of such systems that contribute to their effectiveness include components of heat health action plans, such as the use of media announcements to warn, targeted messaging for at-risk groups, and daily outreach with pre-identified vulnerable individuals throughout the heatwaves.<sup>202</sup>

**Community heat health action plans**—Heat health action plans involve the delivery of targeted education, prevention, and adaptation measures at different levels, ranging from community groups and organisations to local and national

governments.<sup>204 210 214 216</sup> Often integrated with or triggered by heatwave early warning systems, these plans emphasise raising awareness of heat related health risks through targeted health education, encouraging protective behaviours. A 2021 scoping review indicates that heat health action plans incorporating multiple components such as heat monitoring systems, information campaigns, and surveillance and management of individuals with identified vulnerabilities are highly effective in reducing heat related mortality and morbidity.<sup>217</sup>

*Occupational health and safety policies*—To reduce the risk of heat related injuries and illnesses among outdoor workers, occupational health and safety policies may include targeted guidance on working safely in high temperatures. A 2017 review of Australian policies emphasises the need for such guidance to be informed by location-specific research on the effects on physical and mental health of occupational heat exposure, characteristics of vulnerable worker groups, and meteorological thresholds associated with adverse outcome.<sup>208</sup> Furthermore, heat health guidelines and policies should account for occupation-specific factors, such as the use of uniforms or personal protective equipment that may impede heat dissipation.<sup>199</sup>

*Healthcare system preparedness*—Healthcare system preparedness must be guided by local heat vulnerability assessments, with a focus on at-risk populations such as those with chronic conditions, rural and remote residents, and low socioeconomic groups, to ensure allocation of appropriate staffing and resources that meet community-specific needs, equity, and cultural acceptability.<sup>209 210</sup> Systems for coordinated information and resource sharing before, during, and after extreme heat events should be strengthened to enhance readiness, response, and collaboration among healthcare providers, first responders, and government agencies.<sup>209</sup>

*Infrastructure and resources*—Examples of infrastructure and resource allocation initiatives to reduce the negative impacts of extreme heat include cooling centres, which are particularly critical for at-risk populations such as those without air conditioning at home.<sup>212</sup> Urban planning initiatives focusing on green infrastructure such as cool roofs, green walls, and urban forests mitigate urban heat island effects and enhance climate resilience.<sup>213</sup>

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## Recommendations for health professionals and clinical management

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### Managing acute heat related illnesses

Global warming exacerbates health risks, necessitating enhanced preparedness of clinicians for heat sensitive conditions, especially acute heat related illnesses.<sup>218</sup> Clinicians, particularly emergency physicians, should proficiently distinguish between heat cramps, heat exhaustion, and heatstroke. Early identification of heatstroke, characterised by a core body temperature exceeding 40°C, central nervous system dysfunction (such as confusion, seizures, or coma), and multi-organ involvement, is crucial.<sup>222 223</sup> Prompt evidence based cooling within the “golden 30 minutes” is essential to control core temperature, stabilise circulation, and maintain

organ function.<sup>200 219</sup> Concurrently, rapid assessment and management of complications, including rhabdomyolysis, disseminated intravascular coagulation, acute kidney injury, and cerebral oedema, are vital, along with continuous monitoring of multi-organ function.

Effective management requires a coordinated multidisciplinary response involving emergency medicine, intensive care, nephrology, neurology, and other relevant specialties. Given the complexity and variability in managing heat sensitive conditions, treatment strategies must be meticulously tailored to account for individual pathophysiology and comorbidities.

### Integrating heat exposure into disease risk assessment

Beyond acute symptoms, clinicians can consider integrating heat exposure as a potential environmental risk factor into chronic disease management frameworks. Systematically assessing the possible contribution of heat exposure to disease progression may be helpful. Heat exposure history can be routinely incorporated into clinical assessments, encompassing environmental conditions such as access to cooling and housing insulation,<sup>167</sup> behavioural patterns such as engagement in heat intensive activities, and quantitative exposure characteristics including temperature thresholds and duration of exposure.<sup>223</sup>

### Adjusting drug therapy during hot weather

Clinicians should consider the effect of heat exposure on both drug therapy and the management of heat sensitive illnesses. Many drugs used for chronic diseases, including antidepressants and cardiovascular drugs, may impair thermoregulatory capacity.<sup>221</sup> Therefore, adjustments in the timing of administration may be necessary. Particular attention should be given to diuretics, which can worsen dehydration and may be warranted during heatwaves to prevent metabolic complications.<sup>226 227</sup> Nephrotoxic agents, including NSAIDs and aminoglycosides, may also pose increased risks under heat induced dehydration.<sup>228</sup>

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## Guidelines

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The Society of Critical Care Medicine provides acute clinical guidance for heatstroke, outlining cooling modalities and drug treatments.<sup>200</sup> Preventive recommendations from the European Society of Cardiology and the American College of Obstetricians and Gynecologists highlight the effects of climate change on cardiovascular and maternal-fetal health, emphasising population level interventions.<sup>230 231</sup> The World Health Organization provides broader guidance on climate change and health, focusing on systemic, large scale strategies.<sup>232</sup> Additionally, government issued occupational heat exposure guidelines provide practical preventive measures.<sup>233</sup>

Although existing guidelines show strengths in heatstroke and targeted prevention, few clinical guidelines provide evidence based recommendations on other heat related health risks, with a notable lack of standardised protocols for heat adaptive care.<sup>234</sup>

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# The impact of noise pollution on health

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0.5 HOURS

**A 52 year old man presents with persistent sleep disturbance, tiredness, and difficulty concentrating. His partner reports increasing irritability. The couple have recently moved to a house near a busy motorway. After managing his immediate concerns, the GP considers cardiometabolic risk reduction and the health effects of long term exposure to transport noise pollution from road, rail, and aircraft.**

Environmental noise contributes to a wide range of adverse health outcomes, including sleep disturbance, cardiovascular disease, and premature death.<sup>1-3</sup> In the UK in 2018, about 40% of the population were exposed to road traffic noise, 4.5% to rail noise, and 4.8% to aircraft noise exceeding 50 decibels (dB)  $L_{den}$  (day-evening-night average over 24 hours). These exposures were associated with a loss of approximately 130 000 healthy life years in that year.<sup>6</sup>

This article reviews the evidence of its impact on health, outlines how to identify patients whose symptoms may be linked to noise exposure, and suggests strategies to mitigate its adverse effects.

## What is noise pollution?

Noise pollution is unwanted or excessive sound that negatively affects health and wellbeing (box 1).<sup>2</sup> Sources of environmental noise include transportation (road traffic, railway, and aircraft noise), industrial (ports, sites of industrial activity, wind turbines), construction, and leisure activities. Additionally, everyday domestic sounds, such as those from household appliances and noise from neighbours, may contribute to noise pollution.<sup>1</sup>

### WHAT YOU NEED TO KNOW

- Noise pollution affects over 100 million people in Europe and is estimated to contribute to around 12 000 premature deaths and the loss of over one million healthy life years annually
- Long term exposure to environmental noise is associated with a wide range of adverse health outcomes, including sleep disturbance and cardiovascular diseases
- Effective management requires a multidisciplinary approach at the patient, provider, and policy levels to restore health, restrict exposure, reduce noise at its source, or, as a last resort, remodel the receiver's environment

## How does noise pollution affect health?

Noise pollution can disturb sleep, disrupt daily activities such as communication and rest, and cause negative cognitive and emotional reactions such as feelings of displeasure, nuisance, disturbance, or irritation (termed noise annoyance).<sup>9 10</sup> Noise pollution is a psychosocial stressor that acts through direct and indirect pathways, triggering physiological responses that activate the body's stress systems: the hypothalamic-pituitary-adrenal and sympathetic-adrenal axes (see figure).<sup>9 11 12</sup> These processes can influence cardiometabolic disease precursors and risk factors.<sup>13</sup> For example, noise events, especially aircraft noise at night, can raise blood pressure even without waking the individual,<sup>14</sup> and adversely affect small blood vessels and arterial function, increase stress hormones, and worsen cardiac function.<sup>15</sup>

This article addresses the non-auditory effects of environmental noise, which can also cause hearing loss and tinnitus.<sup>39</sup> Depending on the source, intensity, and duration, auditory effects can be temporary or permanent and may not be restricted to occupational settings.<sup>39</sup> However, adverse physical health effects associated with noise occur at lower levels than those that cause hearing damage (typically  $\geq 80$  dB).<sup>10</sup>

## Who is affected?

Exposure to noise pollution is widespread. People living in urban areas or near busy transportation routes have higher exposures, as do those working in environments with substantial ambient noise or those in industries with machinery.<sup>4</sup> Disadvantaged and marginalised communities may be more likely to be exposed to harmful levels of environmental noise.<sup>4</sup> Health inequalities related to noise pollution may result not only from differences in exposure, but also from differential vulnerability to the effects of noise exposure. Disadvantaged groups are more likely to live in low quality housing and to have fewer resources to implement mitigations such as noise insulation, while those with lower quality sleep—such as pregnant women, elderly people, and shift workers—are more vulnerable to noise-associated sleep disturbance.<sup>4</sup>

## How do patients present?

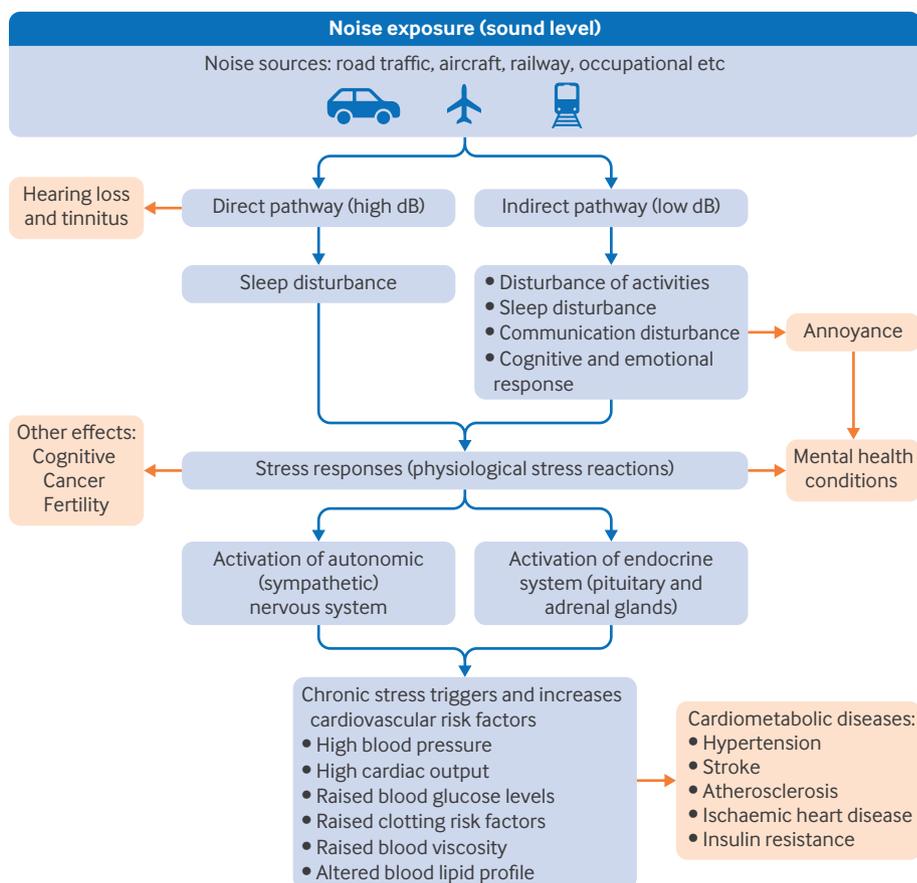
Exposure to noise pollution can lead to symptoms such as sleep disturbance and mental ill health, and may cause or exacerbate conditions such as cardiovascular diseases, psychological stress, and cognitive impairment.

Noise exposure may also be considered in patients with:

- Persistent or unexplained complaints such as fatigue, stress and annoyance, particularly if there is a temporal relationship between symptoms and noise exposure.
- Sleep disturbance without other apparent causes.
- Reports of heightened irritability or difficulties in concentrating, especially in high noise

### Box 1 | Sound properties, noise levels, and harm

- Sound intensity (amplitude, or volume) is typically measured in decibels (dB) on a logarithmic scale. For instance, rustling leaves measure about 10 dB, average office noise is around 70 dB, a hairdryer can reach 90 dB, and a jet engine can produce sound at 110 dB.<sup>7</sup>
- The human ear does not respond equally to sounds at different frequencies. Humans tend to perceive sounds at a lower frequency as having a lower intensity. To account for this, studies on environmental noise use a weighting system (A-weighting) to try and replicate the response of a human ear.<sup>8</sup> This upweights frequencies that humans can perceive.
- The nature of sound—whether continuous or intermittent, and its timing and duration—also influences its potential annoyance.
- WHO sets the threshold at which significant harmful effects from long term noise exposure occur at 55 dB(A)  $L_{den}$  (this is the day-evening-night indicator, averaged over 24 hours) or 50 dB(A)  $L_{night}$  averaged overnight.<sup>1</sup>  $L_{den}$  is a metric used to evaluate noise exposure, with higher weights for evening and night periods to account for increased human sensitivity at those times.<sup>2</sup>



Noise exposure and adverse health outcomes, adapted from Münzel 2021<sup>11</sup>

environments (such as near busy roads, railways, airports, or industrial areas).

- Worsening of pre-existing conditions such as anxiety, depression, cognitive decline, or cardiometabolic diseases.

There is no definitive way to identify noise pollution as a causative factor in a patient's presentation. Patients may themselves identify noise as a factor contributing to their symptoms, particularly if they are noise sensitive. About one in three people is noise sensitive. Noise sensitivity is associated with anxiety disorder, major depressive disorder, and autistic spectrum disorder.<sup>40</sup> Noise-sensitive people attend more to noise, discriminate more between noises, perceive noises as more threatening and out of their control, and adapt to noise more slowly than those less sensitive.<sup>41</sup> Noise sensitivity is an independent predictor of annoyance.<sup>42</sup>

To identify noise exposure and sensitivity, consider asking:

#### 1. General exposure:

- “Do you live near a busy road, railway, or airport?”
- “How much does noise affect your daily life?”
- “Does noise interfere with your sleep or ability to concentrate?”
- “Do you wear earplugs to sleep?”

#### HOW PATIENTS WERE INVOLVED IN THE CREATION OF THIS ARTICLE

The case vignette was written with input from a patient affected by noise pollution. Their comments regarding the impact of everyday domestic sounds (including those from neighbours), and the role of practical support from primary care in advocating for patients, have been incorporated into the final manuscript.

#### 2. Noise sensitivity:

- “Are you more sensitive to noise than others?”<sup>10</sup>
- Noise sensitivity can be formally quantified with scales such as Weinstein's Noise Sensitivity Scale.<sup>43</sup>

#### How can the impact of noise on health be mitigated?

Tackling the impacts of noise pollution on human health requires a multidimensional approach, encapsulated by the “Three Ps” framework: patient, provider, and policy level measures using the adapted “Four Rs” strategy (restoring, restricting, reducing, and remodelling noise impact).<sup>1,2</sup> The mitigation suggestions in the table and box 2 are mostly based on expert opinion.

#### Patient level

Understanding noise pollution as a factor in illness enables a targeted and personalised management plan, addressing the underlying issue as well as managing symptoms and associated diseases. At the patient level, mitigating environmental noise effects requires a combination of restorative and restrictive measures to improve health and provide psychosocial support. However, evidence for the benefits of noise restriction, such as earplugs, on health outcomes remains uncertain. For example, very low to moderate quality evidence from a systematic review of 229 studies in adult inpatient settings showed that non-pharmacological interventions such as earplugs, white noise generators, and relaxation

## Four Rs (restore, restrict, reduce, remodel) strategy of noise pollution management at the patient, provider, and policy levels

Patient	Provider	Policy
<b>Restore</b>		
<ul style="list-style-type: none"> <li>• Increase your awareness and understanding of the risks associated with prolonged noise exposure</li> <li>• Immerse yourself in natural sounds such as birdsong and water features to reduce stress and alleviate annoyance<sup>45,46</sup></li> <li>• Consider mindfulness practices to help manage stress and response to noise</li> </ul>	<ul style="list-style-type: none"> <li>• Provide restorative psychological therapies, counselling, and support systems</li> <li>• Use “green prescribing” (recommending outdoor activities in nature for health benefits)<sup>2,47</sup> such as forest bathing to improve wellbeing and reduce symptoms of anxiety and depression<sup>48</sup></li> </ul>	<ul style="list-style-type: none"> <li>• Develop clinical guidelines, care pathways, and resources to support the assessment and management of patients affected by noise pollution</li> <li>• Implement sound urban planning and green mitigation measures—such as green barriers, tree planting, and water features—to create favourable soundscapes that promote relaxation and support health, even in densely populated areas<sup>13</sup></li> </ul>
<b>Restrict</b>		
<ul style="list-style-type: none"> <li>• Use measures such as noise-restricting devices (like earplugs, earmuffs) and quieter spaces</li> <li>• Use mobile apps or personal soundscapes (such as white noise, nature sounds) to mask intrusive noise and improve concentration, sleep, or relaxation in noisy settings</li> </ul>	<ul style="list-style-type: none"> <li>• Encourage urban green spaces and calming acoustic environments to buffer harmful noise,<sup>13</sup> although access remains limited in many low income countries and deprived communities</li> </ul>	<ul style="list-style-type: none"> <li>• Install physical noise barriers along major roads and railways to reduce environmental noise exposure in residential areas</li> <li>• Restrict noise exposure in schools through outdoor shelters and noise-reducing structures, particularly in areas affected by transport noise such as flight paths</li> </ul>
<b>Reduce</b>		
<ul style="list-style-type: none"> <li>• Use social prescribers (professionals who connect patients to non-medical support services to improve their overall wellbeing) to facilitate communication and negotiation with local authorities through both statutory and voluntary support to reduce noise at source</li> </ul>	<ul style="list-style-type: none"> <li>• Seek proactive support from housing officers and local councillors to tackle persistent residential noise issues and advocate for mitigation measures</li> </ul>	<ul style="list-style-type: none"> <li>• Minimise noise at its source through measures such as implementing quieter machinery, improving vehicle and aircraft design, using low-noise road surfaces, and strategic urban planning and zoning laws to separate noisy industries from residential areas</li> <li>• Enforcing curfews for noisy activities such as night-time flights and entertainment venues, tackling neighbourhood noise through regulations and community engagement</li> </ul>
<b>Remodel</b>		
<ul style="list-style-type: none"> <li>• Adapt personal living spaces with soundproofing and insulation to mitigate the impact of environmental noise on daily life</li> </ul>	<ul style="list-style-type: none"> <li>• Advocate for home or environmental assessments, including noise mapping, to identify patients in high-noise settings who may benefit from targeted environmental adaptations</li> </ul>	<ul style="list-style-type: none"> <li>• Mitigate the impact of noise exposure on the receiver when reduction and restriction are insufficient: acoustic retrofitting of windows, using noise-cancelling building materials, and increasing insulation in homes and workplaces</li> <li>• Intensify efforts to meet the Zero Pollution Action Plan goal of reducing transport noise exposure by 30% by 2030<sup>49</sup></li> </ul>

### Box 2 | Policy measures to mitigate the effects of noise pollution<sup>1,2,13</sup>

#### Traffic noise control

- Implementing lower speed limits, such as 20 mph in residential areas
- Using low-noise road surfaces
- Restricting heavy commercial vehicles movements in residential areas and promoting the use of electric vehicles
- Improving regulatory frameworks for tyres, vehicles, railways, and aircraft
- Integrating noise action plans into sustainable urban mobility plans
- Expanding clean public transport and encouraging active mobility

#### Night-time restrictions

- Enforcing curfews for flights and heavy traffic during night time to reduce sleep disturbance for residents living near airports and busy roads

#### Infrastructure enhancements

- Building noise barriers along motorways and railways and investing in quieter rail and train technology to shield residential areas from traffic noise

#### Enforcement and regulation

- Strengthening noise regulation and increasing resources for local authorities to manage complaints effectively. Setting a specific noise reduction target based on reducing exposure and disease burden from noise pollution within a defined timeframe

#### Community engagement and education

- Promoting public awareness about the health impacts of noise pollution and encouraging community participation in noise management strategies

#### Integration of noise mitigation in urban planning

- Incorporating noise assessment in the planning stages of infrastructure projects. Using mapping and modelling tools to identify areas for retrofitting and effective noise reduction interventions such as green screens, parks, and nature reserves

techniques improved sleep quality.<sup>50</sup> Although evidence certainty is limited, such low cost, low risk interventions may be acceptable to patients and could be suggested to help mitigate noise exposure, particularly at night when it is most likely to disrupt sleep.

#### Provider level

Healthcare providers are essential in supporting and advocating for patients affected by noise pollution. They should offer holistic advice to restore health and restrict noise exposure, including recommending social prescribers for community based support.

Referral to another specialist may sometimes be necessary for patients with conditions adversely affected by noise pollution, particularly for noise-sensitive individuals. Although this will not be different from routine primary care practices, it may be helpful to emphasise noise exposure as a potential underlying cause of the symptoms during the referral process.

#### Policy level

Effective policy interventions for noise pollution should follow the reduce, restrict, and remodel hierarchy.<sup>2</sup> Reducing noise at the source through legislation, planning, and engineering is generally more effective than restricting transmission with sound barriers or, as a last resort, remodelling the receiver's environment with sound insulation.<sup>2</sup> For example, a Swiss repeated measures study involving 1300 residents found that reducing speed limits from 50 kph to 30 kph on 15 streets lowered noise levels, significantly reducing noise annoyance and sleep disturbances and moderately improving perceived road safety.<sup>51</sup>

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### EDUCATION INTO PRACTICE

- How can you incorporate noise exposure assessments into clinical patient evaluations?
- What resources do you need to implement practical multidisciplinary approaches for tackling the health effects of noise pollution?
- How can public awareness and community engagement be improved to support noise management strategies, and what role could healthcare professionals play?

# Brazil and the climate emergency: why the medical curriculum must include climate education

**T**he health impacts of climate change are becoming increasingly evident in Brazil.

The country's vast territorial and social heterogeneity makes it highly vulnerable to climate extremes, with severe droughts in the north, excessive rainfall in the south, and intensifying heatwaves and other extreme weather events further straining public health infrastructure and exacerbating existing vulnerabilities.

The health consequences have been stark: recent years have seen surges in deaths related to dengue, illnesses related to heat, and cardiorespiratory diseases caused by air pollution from wildfires. In May 2024, the state of Rio Grande do Sul experienced flooding that submerged over 15 000 km<sup>2</sup> and led to more than 15 000 cases of leptospirosis.

It is imperative that Brazil equips future professionals with the knowledge and skills to confront and deal with these realities. The health ministry has recently published a climate change guide, a practical resource aimed at equipping healthcare workers with knowledge and tools to respond to health issues that are climate sensitive. Medical society initiatives, such as *Médicos pelo Clima* (Doctors for the Climate), have played a pivotal role in mobilising clinicians and students to act on climate advocacy and sustainable healthcare practices.

Despite the urgency, medical education worldwide has lagged behind the pressing realities of the climate crisis. Until recently, few medical schools offered systematic instruction on how climate change affects health, especially across Latin

America. However, this situation is changing. Schools around the world are beginning to add climate health content and curricular models are multiplying.

Understanding the impacts of climate change on health is essential to being a well prepared physician. In 2022, we launched the elective discipline *Saúde e Mudanças Climáticas* to provide students with foundational concepts in climate science, emphasising the study of pathologies that are climate sensitive while integrating principles of planetary health, health system resilience, and environmental factors such as air pollution and microplastics.

Students also engaged in practical mitigation activities, including preparing meals aligned with the Planetary Health diet and exploring nature based solutions in the city, such as rain gardens and urban parks. These activities help students understand the complex strategies needed to tackle climate related health impacts.

Given Brazil's territorial and socioeconomic disparities, where the impacts of climate

**Future clinicians and leaders must be trained to assume responsibility for decarbonising services**

change are unevenly distributed and deeply intertwined with issues of environmental and climate justice, our approach tries to incorporate these dimensions explicitly. As an example, in a class on poor mental health related to the climate, we presented a case study on young people living in precarious conditions on the outskirts of São Paulo who reported higher levels of eco-anxiety than wealthier medical students during episodes of severe summer storms.

Medical students must also be prepared to understand not only how climate change affects illness and disabilities, but also how the health sector itself contributes to the crisis. Future clinicians and system leaders must be trained to recognise this climate footprint and assume responsibility for decarbonising services. This includes adopting sustainable clinical practices, advocating for institutional change, and making decisions that reduce medical waste and the sector's carbon impact.

The integration of climate change in medical curricula faces substantial challenges. One barrier is the lack of space within an already dense programme. Additionally, students often prioritise core clinical subjects over emerging topics like climate and health. These challenges are compounded by the limited

availability of faculty with specific training in climate and health.

## Overcoming barriers

To overcome some of these barriers we implemented workshops with course directors and faculty members aimed at enhancing climate literacy and codesigning concise learning components that are specific to each discipline. These were designed to be integrated into the existing curricular to avoid a new mandatory module. For example, air pollution, global warming, and wildfire smoke are presented as risk factors for cardiovascular disease in the cardiology module. These practical decisions offer a transferable model for other medical schools.

Brazil's newly approved national curricular guidelines for the medical course define the expected profile and competencies of graduates. They explicitly incorporate climate change as a challenge to be addressed in medical training. This regulatory shift will require medical schools to include and expand climate health content within their curricula.

These developments reinforce the feasibility and urgency of elective courses, faculty development programmes, and hospital based initiatives that embed climate health competencies across disciplines. Transversal curricular integration—supported by competency frameworks and targeted faculty training—offers a scalable and context sensitive strategy to prepare future physicians for the clinical and civic demands of a rapidly changing world.

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KATH XAPI PURI

# Help me cope with new motherhood in the heat



**Nur Yanayirah** describes how extreme heat affected her pregnancy, birth, and early motherhood

In 2013 I was heavily pregnant with my daughter during a period of extreme weather in Indonesia. A long dry season had caused drought, and the water where I lived had become full of sediment and was undrinkable.

The day I gave birth to my daughter was hot, and the air conditioning in the delivery room wasn't working properly. The room was poorly ventilated, and there was no privacy, which made me feel uncomfortable and short of breath. I had previously had a traumatic experience with the stillbirth of my first baby, and the heat compounded my stress and discomfort, so the doctors and I decided to opt for a caesarean section.

Breastfeeding in the hot weather was challenging because I needed so much water to stay hydrated and to regulate my body temperature. I was thirsty, irritable, and I grew annoyed and angry at my baby's cry. She was often fussy, irritable, and less inclined to feed in the heat, making it harder for me to get her to latch or to nurse comfortably. As a result, she wasn't gaining much weight, so in desperation I gave her formula. She also had a heat rash caused by blocked sweat ducts, which caused red bumps and blisters on her skin, and her discomfort was made worse by the lack of clean water in which to bathe.

## Emotional burden

Following the heat, heavy rain and flooding hit our region for three days. The water became even dirtier and insects and rats entered the house. The flooding cut off food supplies, and we had to wait for help from the village chief. The water I used to wash bottles was unsanitary, and my baby developed severe diarrhoea and had to be admitted to hospital. I felt like a failure, and my days were filled



PRIVA SUNDARAM

with sadness, anger, loss of appetite, insomnia, and overwhelming fear. This culminated in a suicide attempt, and after consulting a psychiatrist I received a diagnosis of postpartum depression.

Climate change significantly impacted my mood and wellbeing postpartum. When the weather was

very hot, I rarely socialised with friends, and I became lonely and isolated. The extreme weather events left me feeling hopeless and lacking confidence as a mother. I was losing myself and was plagued by constant anxiety about my child's health.

## Importance of community

After two years of treatment with a psychiatrist, I recovered and dedicated myself to providing support for women with similar experiences. In 2015, I founded MotherHope Indonesia, a Facebook page which now has over 58 500 members from across 100 cities in Indonesia. Mothers share their concerns about the climate crisis in our peer support group sessions. Many have found caring for their babies in the hot weather extremely challenging, especially because they lack air conditioning and adequate support from their partners or family. They find it difficult to sleep at night in the heat, which has resulted in mental health problems, and some have had thoughts of self-harm. We provide counselling and online classes from clinical psychologists to support the mental health of mothers in dealing with the climate crisis. We have also created a men's cooking class where we improve our husbands' cooking skills and confidence with various eco-friendly cooking methods. We educate families on sustainable food practices, promote nutritious diets for postpartum women and their babies, and encourage conscious food choices. We hope the programme will increase climate change literacy among new parents and promote gender equality, providing additional help for new mothers.

By empowering women through education, training, and enabling meaningful participation in policy making (such as input to Indonesia's climate change action plan), we can create more effective, inclusive, and equitable climate action for all. The needs of women, babies, and children should be integrated into climate crisis mitigation, adaptation, and disaster risk reduction strategies to fulfil their rights.

Patient author

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## WHAT YOU NEED TO KNOW

- Effects of climate change can impact the physical and mental health of pregnant and postpartum women and their babies
- Managing the effects of climate change, such as extreme heat, may help protect the mental wellbeing of new mothers
- Engaging women in community climate action harnesses local knowledge and innovative capacities for sustainable solutions

## EDUCATION IN PRACTICE

- What practical support could you offer to pregnant women and new mothers affected by extreme heat or other effects of climate change?
- How could you ensure you are learning from and working with local community groups to support new mothers?

CASE REVIEW

Red nodule on the umbilicus

A 3 year old boy presented with a red nodule on his umbilicus. The lesion gradually enlarged over a period of two months, occasionally rupturing and releasing small amounts of bloody discharge. Initially clinically diagnosed as a pyogenic granuloma by a dermatologist, the lesion had no associated history of trauma or inflammation. The child was in good general health with no associated abdominal pain, distension, vomiting, or abnormal defecation or urination.

On examination, a red nodule measuring 0.5 cm × 0.6 cm × 0.3 cm was observed at the umbilicus. The lesion had a soft consistency, with a slight accumulation

of white scales on the surface and surrounding skin; the base remained intact (figure). Ultrasound imaging showed no subcutaneous sinus or cyst and revealed a hypovascular lesion. The results of the histopathology examination indicated epidermal hyperplasia, with two distinct epithelial layers (cup-shaped cells in the inner layer) surrounded by lymphoid tissue infiltrated by plasma cells.

- 1 What are the differential diagnoses?
- 2 What is the most likely diagnosis?
- 3 How is this condition treated?

Submitted by Jingyi Yang, Qianyue Xu, and Dan Deng  
Parental consent obtained.

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A red nodule at the umbilicus, with a slight buildup of white scales on the surface and surrounding skin, the base remains intact

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answers

**LEARNING POINTS**

- Umbilical omphalomesenteric duct polyps are rare embryological remnants that can mimic common umbilical lesions in children.
- Accurate diagnosis requires careful imaging and histopathological confirmation to exclude intra-abdominal involvement.
- Surgical excision is the preferred treatment; misdiagnosis might lead to inadequate management or recurrence.

**PATIENT OUTCOME**

See [bmj.com](https://bmj.com).

to serous/serosanguinous umbilical discharge, local infection, lower abdominal pain, and/or urinary tract infection.

**2 What is the most likely diagnosis?**

Omphalomesenteric duct polyp, which is a rare remnant of the omphalomesenteric duct. Umbilical omphalomesenteric duct polyp typically presents at birth as a bright red papule or nodule with a smooth surface, which may bleed or have serous discharge, thereby irritating the surrounding skin. It is important to note that umbilical omphalomesenteric duct polyp might also coexist with other complications, such as an underlying sinus, cyst, fistula, or a fibrous band connecting to the intestine.

**3 How is this condition treated?**

Perform a preoperative evaluation before any treatment to rule out intra-abdominal connections. Surgical excision is essential for omphalomesenteric duct polyps, because it establishes the diagnosis and ensures complete removal, preventing recurrence and eliminating ileal remnants that could cause life threatening complications. Topical silver nitrate treats umbilical granulomas but is ineffective for omphalomesenteric duct polyps.

**1 What are the differential diagnoses?**

The main differentials are omphalomesenteric duct polyps, umbilical granulomas, omphalocele (also known as exomphalos), and urachal anomalies. The omphalomesenteric duct connects the developing gut to the yolk sac and typically disappears by the ninth week of gestation. If it fails to regress completely (in about 2% of cases), it can result in anomalies such as Meckel's diverticulum, which occurs at the ileal end, and umbilical omphalomesenteric duct polyps, found at the umbilical cord's end.

Umbilical omphalomesenteric duct polyp can be difficult to differentiate from an umbilical granuloma. The umbilical granuloma appears as a soft, fragile, vascular lesion, whereas the umbilical omphalomesenteric duct polyp manifests as a hard, reddish nodule, possibly with mucinous secretions. Omphalocele, a condition characterized by congenital cystic contents within the abdominal cavity, should be considered in the differential diagnosis.

Urachal anomalies include cysts, fistulas, and sinuses. Presentation can be asymptomatic or lead



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