Réchauffement climatique

Réchauffement climatique

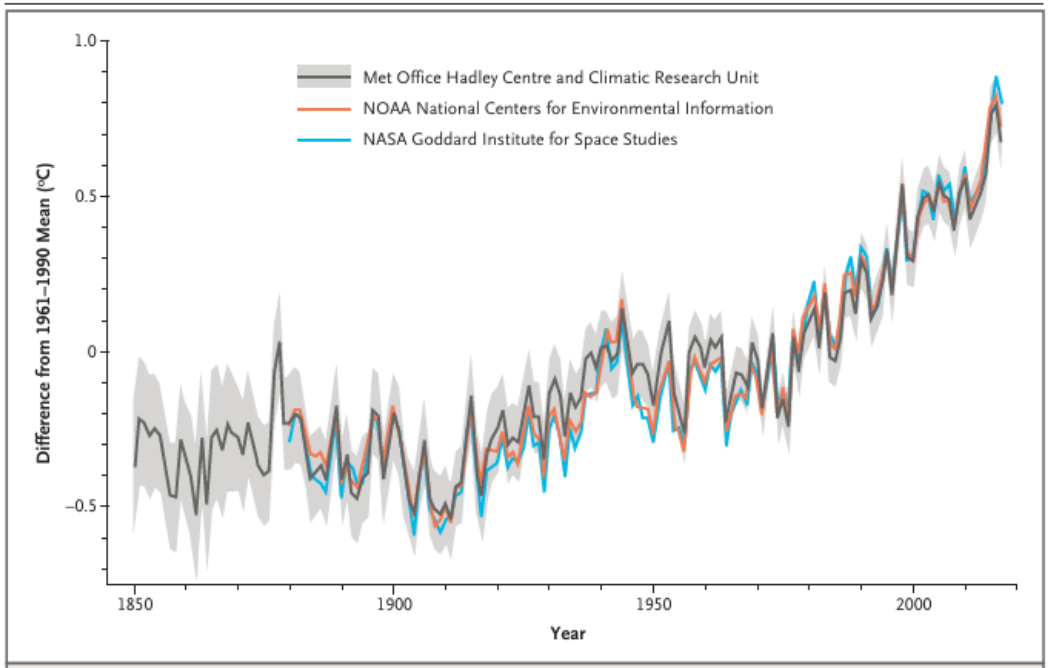
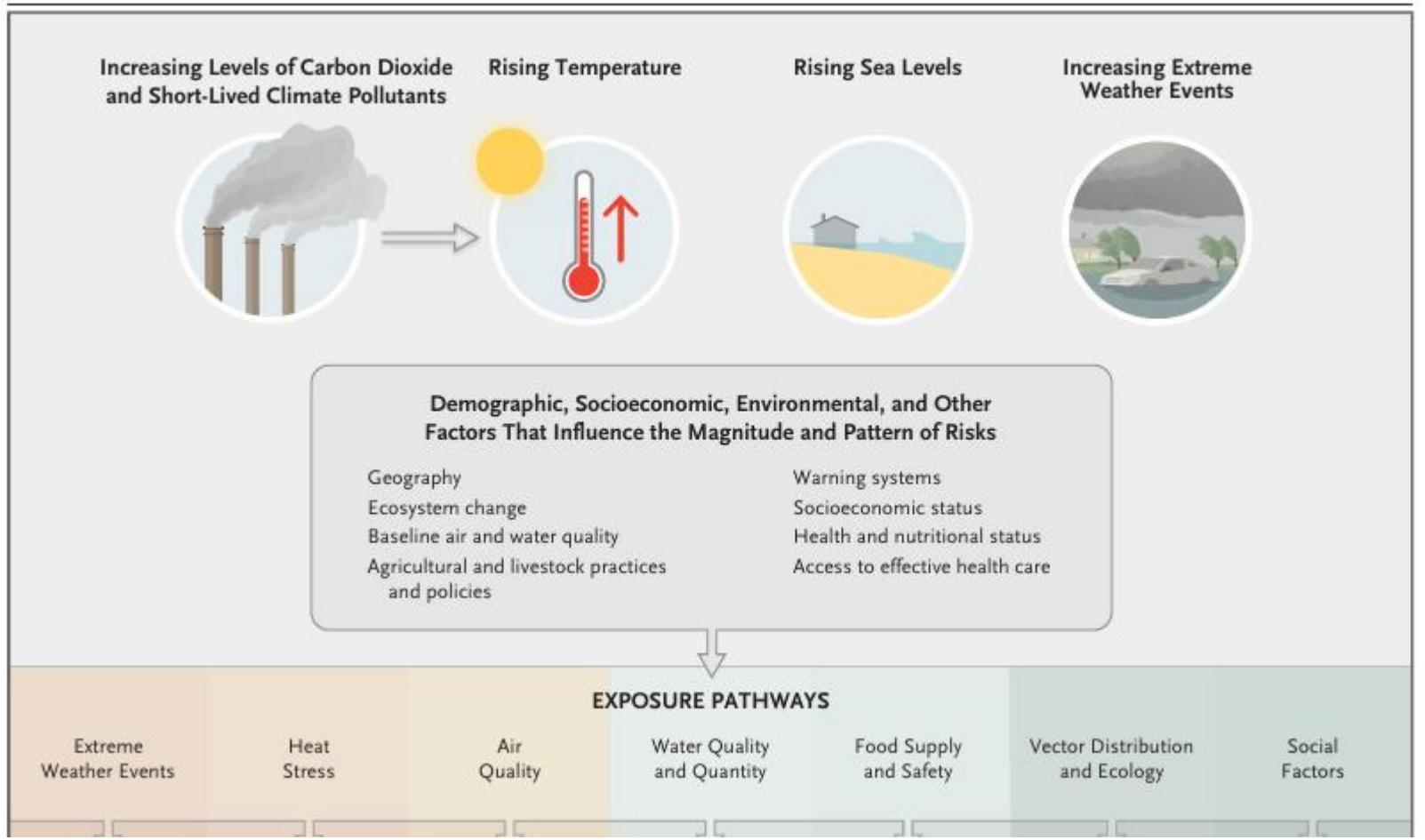


Figure 1. Changes in Surface Temperatures, 1850–2017.

The plot shows annual global mean temperature anomalies relative to the means during 1961–1990 from the HadCRUT4 data set, a global surface temperature data set produced by the Met Office Hadley Centre in collaboration with the University of East Anglia Climatic Research Unit (the gray shaded area indicates 95% confidence intervals). Plots of the mean anomalies from two other data sets — from the National Aeronautics and Space Administration (NASA) and the National Oceanic and Atmospheric Administration (NOAA) — show similar patterns. Adapted from the Met Office Hadley Centre for Climate Science and Services (www.metoffice.gov.uk/research/monitoring/climate/surface-temperature).

N Engl J Med 2019;380:263-73.
DOI: 10.1056/NEJMra1807873



N Engl J Med 2019;380:263-73.
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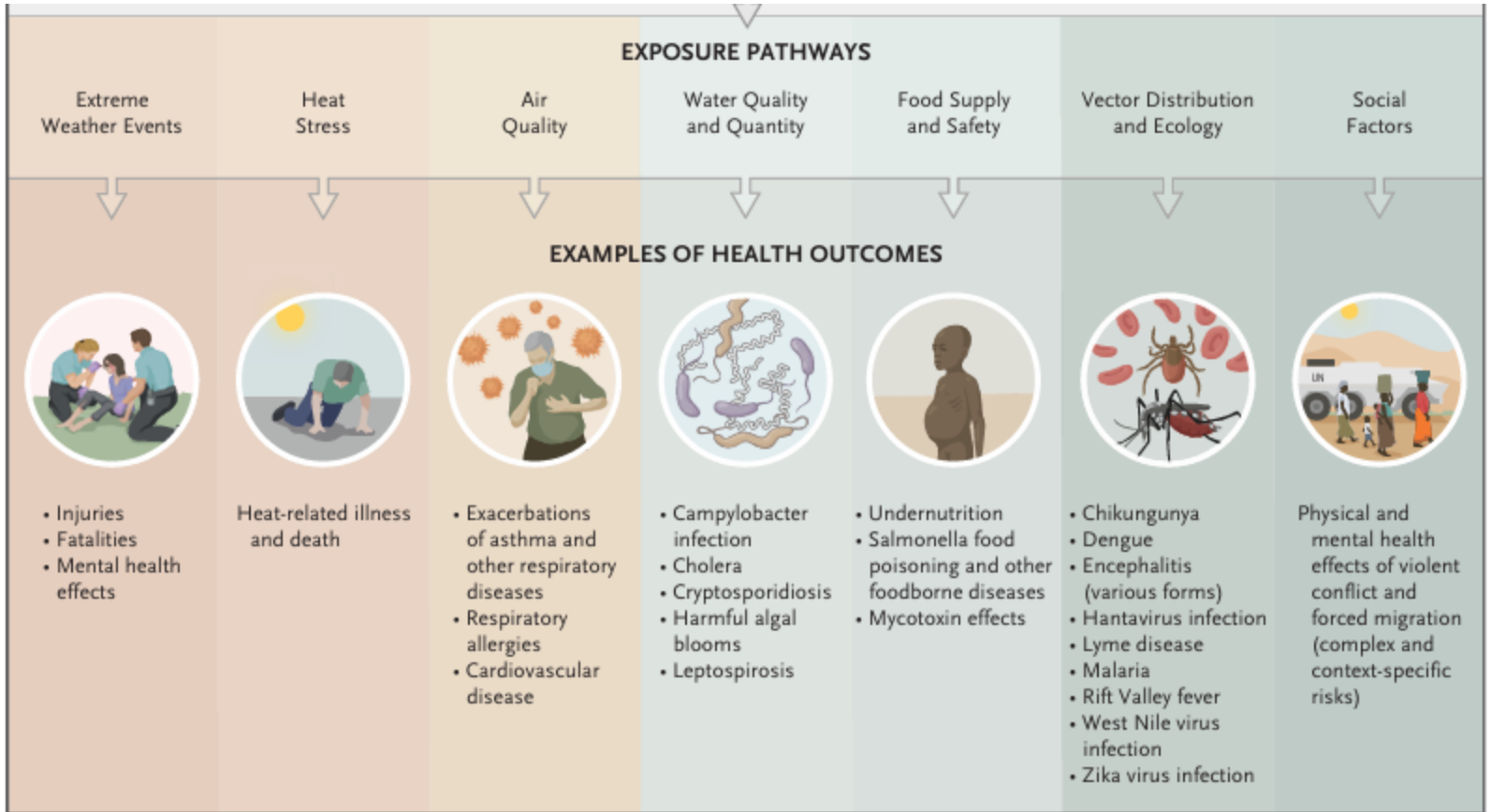

Figure 3. Major Health Risks Associated with Climate Change.

Examples of potential health outcomes and exposure pathways linking climate change with human health are shown, together with factors that can influence the magnitude and pattern of risks. The figure is based on several sources (e.g., Ebi et al.,[3] Smith et al.,[4] the World Health Organization,[16] Hallegatte et al.,[17] and Missirian and Schlenker[18]) and is not intended to be comprehensive.

N Engl J Med 2019;380:263-73.
DOI: 10.1056/NEJMra1807873

Table 1. Health Conditions Associated with Extreme Heat.*

Category of Health Condition	Representative Conditions Affected by Heat	Examples of Findings
CVD	Acute coronary syndrome with or without myocardial infarction, stroke, congestive heart failure, arrhythmia	A meta-analysis of 60 studies showed that the relative risk of death from CVD was 1.12 (95% CI, 1.09 to 1.14) with heat wave days vs. non–heat wave days; a 1°C increase in temperature was associated with a 21% increase (95% CI, 20 to 23) in the risk of death from CVD; heat-related CVD risks were higher for low-to-middle-income countries.[16] A meta-analysis of 4 studies showed that the relative risk of death from myocardial infarction was 1.64 (95% CI, 1.09 to 2.47) with heat wave days vs. non–heat wave days.[17] A meta-analysis of 13 studies showed that a 1°C increase in temperature was associated with a 16% increase (95% CI, 4 to 28) in the risk of hospital admission for myocardial infarction.[17]
Kidney disease	Acute renal failure, nephrolithiasis, electrolyte abnormalities, rhabdomyolysis with renal insufficiency or failure, urinary tract infections	A meta-analysis of 4 studies showed a relative risk of death from kidney disease of 1.03 (95% CI, 1.02 to 1.04) for a 1°C increase in temperature.[18] A meta-analysis of 32 studies showed that a 1°C increase in temperature was associated with an 11% increase (95% CI, 9 to 13) in kidney failure.[18]

N Engl J Med 2024;390:1793-801.
DOI: 10.1056/NEJMra2210769

Respiratory disease	Acute respiratory distress, asthma and COPD exacerbations, pulmonary hypertension, increased respiratory infections, pulmonary edema	An epidemiologic analysis of 452 locations in 24 countries showed that respiratory mortality was increased by a factor of 1.34 (95% CI, 1.22 to 1.47) for the 99th percentile warm-season temperature vs. MMT.[19,20]
Mental disorder	Anxiety, symptoms in people with bipolar disorder or depression, suicide attempts, suicide completion, aggressive behavior, mental fatigue	A meta-analysis of 12 studies showed that a 1°C increase in temperature was associated with a 22% increase (95% CI, 15 to 20) in the risk of mental health–related death.[21] An epidemiologic analysis of 341 locations in 12 countries showed that the risk of suicide increased by 33% (95% CI, 30 to 36) with the 93rd percentile of temperature vs. the 1st percentile (the temperatures with the maximum and minimum suicide risks, respectively).[22]
Adverse birth outcomes	Preterm birth, low birth weight, stillbirth, congenital heart defects	A meta-analysis of 6 studies showed that a 1°C increase in temperature increased the risk of preterm birth by 5% (95% CI, 3 to 7); the risk of preterm birth was 16% (95% CI, 20 to 23) higher on heat wave days vs. non–heat wave days.[23] A meta-analysis of 8 studies showed that a 1°C increase in temperature was associated with a 5% (95% CI, 1 to 8) increase in the risk of stillbirth.[23]

* The list of health effects from heat is not comprehensive. CI denotes confidence interval, COPD chronic obstructive pulmonary disease, CVD cardiovascular disease, and MMT minimum mortality temperature (i.e., the temperature associated with the lowest mortality).

N Engl J Med 2024;390:1793-801.
DOI: 10.1056/NEJMra2210769

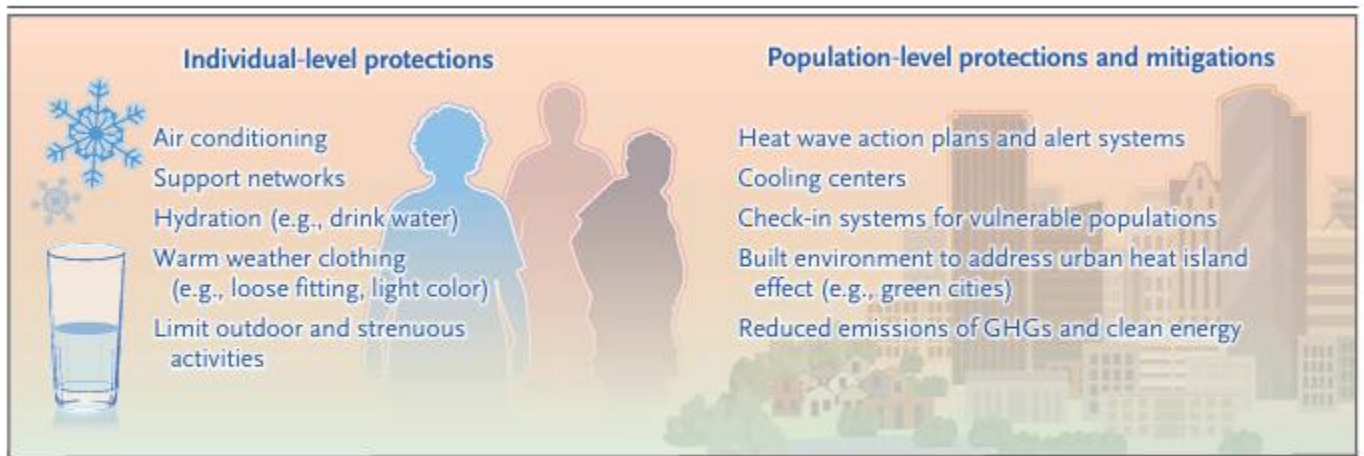

Figure 2. Protective Measures for Heat.

The listed measures are examples; the lists are not exhaustive. GHGs denotes greenhouse gases.

N Engl J Med 2024;390:1793-801.
DOI: 10.1056/NEJMra2210769

Coup de chaleur

Table 1. Epidemiologic and Clinical Features of Classic and Exertional Heatstroke.

Feature*	Classic Heatstroke	Exertional Heatstroke
Age group	Prepubertal, elderly	Postpubertal and active
Occurrence	Epidemic (heat waves)	Sporadic (any time of year)
Concurrent activity	Sedentary	Strenuous
Health status	Chronically ill	Generally healthy
Medications	Often being used (prescribed medications)	Usually none being used (sometimes ergogenic aids, illicit drugs)
Mechanism	Absorption of environmental heat and poor heat dissipation	Excessive heat production, which overwhelms heat-loss mechanisms
Sweating	May be absent (dry skin)	Usually present (wet skin)
CNS dysfunction	Common	Common
Acid–base disturbance	Respiratory alkalosis	Metabolic acidosis
Rhabdomyolysis	Unusual	Frequent
Liver dysfunction	Mild	Marked to severe
Renal failure	Uncommon (<5%)	Common (25–30%)
DIC	Mild	Marked to severe
ARDS	Common	Common
Creatine kinase	Mildly elevated	Markedly elevated
Calcium	Normal	Low (hypocalcemia)
Potassium	Normal	Usually high (hyperkalemia)

N Engl J Med 2019;380:2449-59.
DOI: 10.1056/NEJMra1810762

Table 2. Risk Factors Underlying Heatstroke.*

Heatstroke Type and Risk Factor	Explanation
Classic	
Weather	Heat waves, with successive hot days and nights
Physiological factors	Cardiovascular insufficiency impeding normal cardiovascular adjustments to heat stress: inability to maintain acceptable stroke volume in the heat, inadequate peripheral vasodilatation due to structural changes and compromised nitric oxide–mediated vasodilatory mechanism, reduced capillary density and quality of cutaneous microcirculation, decreased sweat rate and sweat-gland output in response to heat stress
Social factors	Social isolation, unventilated and non–air-conditioned living space, inability to care for oneself, confinement to bed
Underlying illness	Exacerbation of mental, cardiovascular, cerebrovascular, and pulmonary illnesses and multiple sclerosis by exposure to heat stress
Medications	Beta-blockers, diuretics, calcium-channel blockers, laxatives, anticholinergic drugs, salicylates, thyroid agonists, benztropine, trifluoperazine, butyrophenones, α-agonists, monoamine oxidase inhibitors, sympathomimetic medications, tricyclic antidepressants, SSRIs
Exertional	
Social factors	Overmotivation, peer and coach pressure
Functional factors	Low physical fitness (physical effort unsuited to physical fitness; "killer workouts"), lack of acclimatization (habituation) to heat, low work efficiency, overweight (reduced ratio of skin area to mass and greater heat-storage capacity in fat layers), protective clothing (reduced sweating efficiency)
Acquired factors	Viral or bacterial infection (even if subclinical), dehydration, sleep deprivation, sweat-gland dysfunction (e.g., deep burns, scarred skin on >40% of total body-surface area)
Congenital factors	Chronic idiopathic or familial anhydrosis, ectodermal dysplasia
Drug abuse	Amphetamines and amphetamine-like agents (e.g., ephedra), MDMA, cocaine, PCP and LSD, synthetic stimulants of the cathinone class (e.g., α-PHP), alcohol

* LSD denotes lysergic acid diethylamide, MDMA 3,4-methylenedioxymethamphetamine (ecstasy), PCP phencyclidine, α-PHP α-pyrrolidinohexanophenone, and SSRI selective serotonin-reuptake inhibitor.

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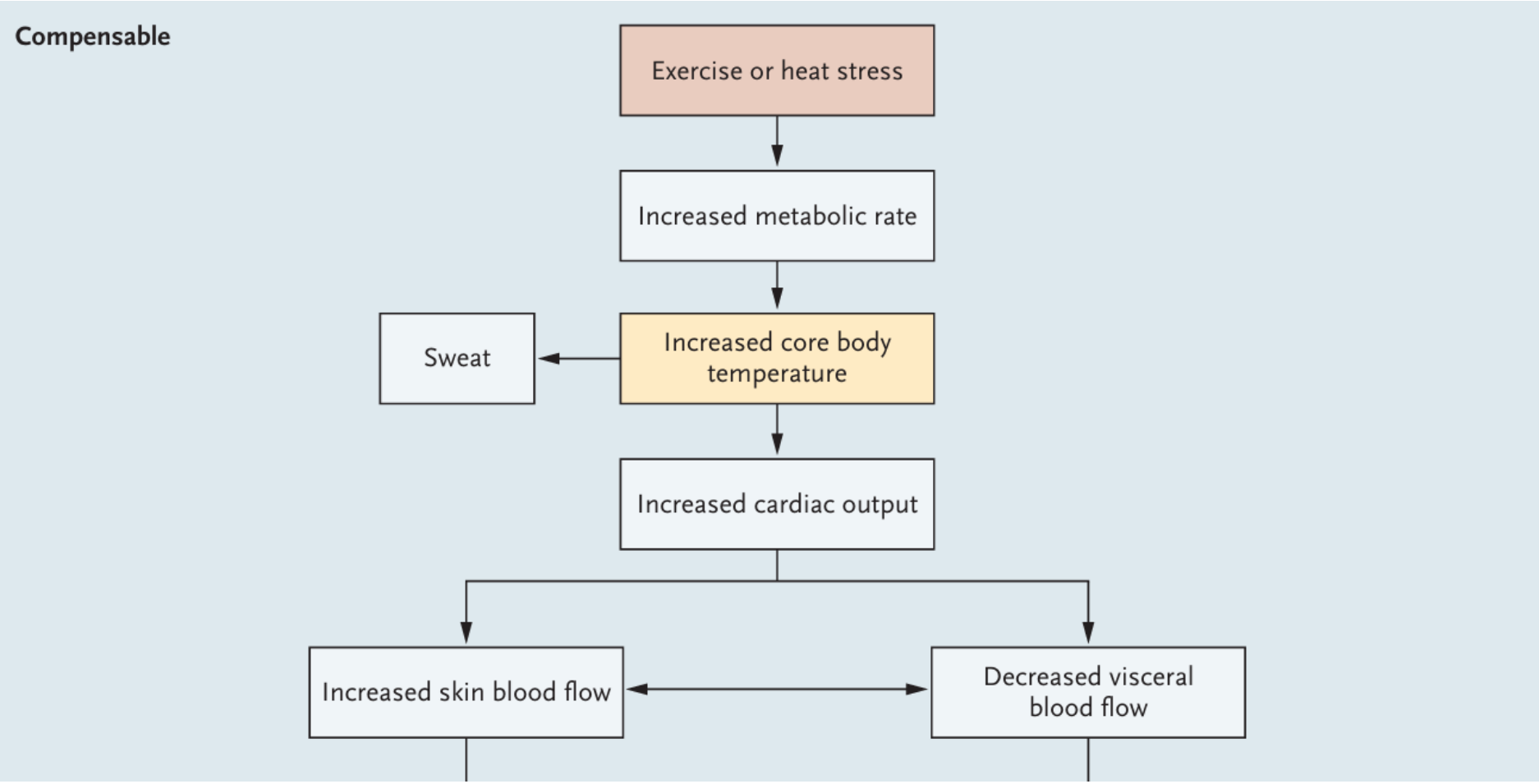
DOI: 10.1056/NEJMra1810762

Voie physiopathologique menant au coup de chaleur



N Engl J Med 2019;380:2449-59.
DOI: 10.1056/NEJMra1810762

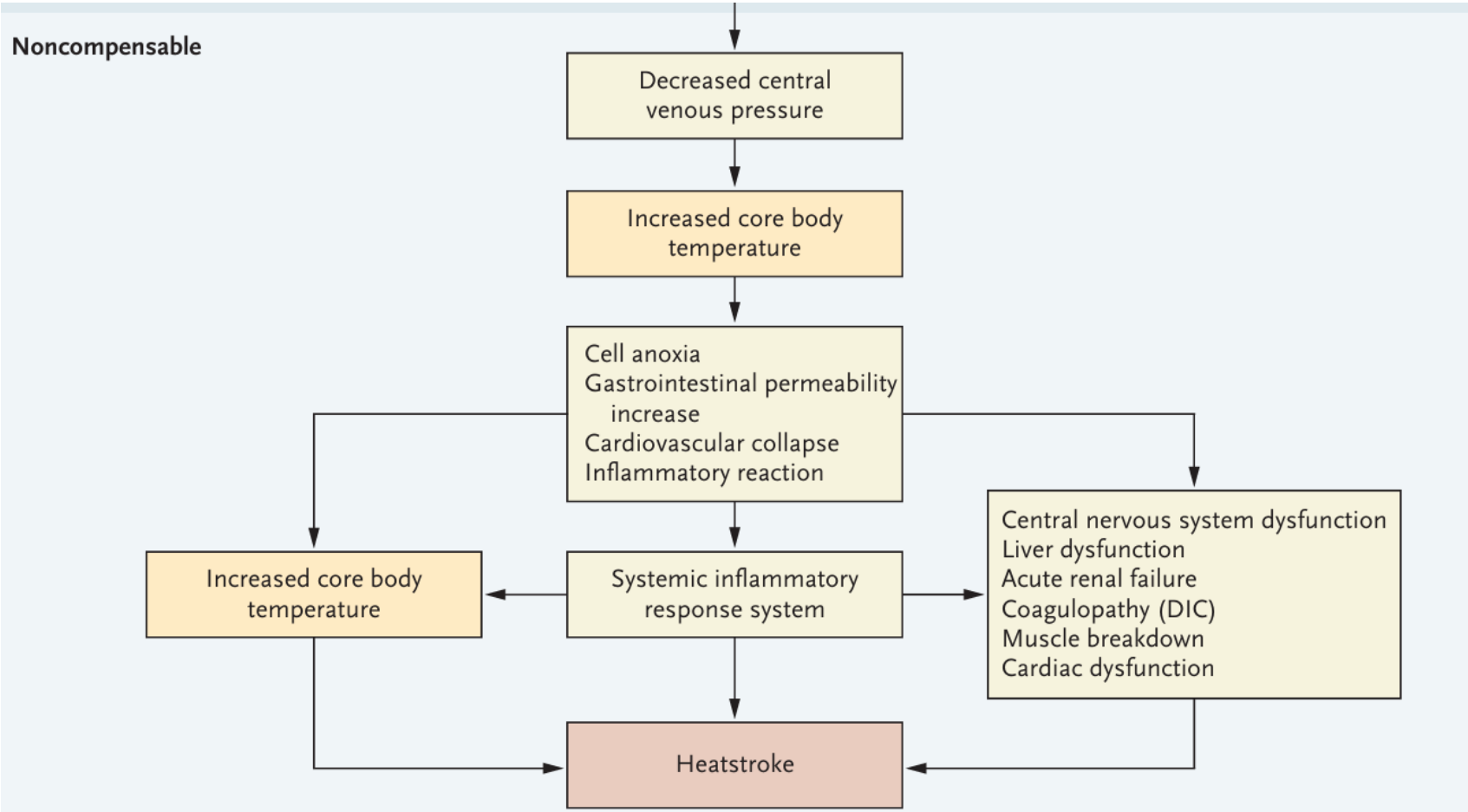


Figure 1. Pathophysiological Pathway Leading to Heat Stroke.

The sequence of events that leads to heat stroke involves a transition from a compensable thermoregulatory state to the noncompensable condition. Heat stress initiates a thermoregulatory response to increased cardiac output and redistribution of blood flow. When central venous pressure begins to decrease substantially, core temperature begins to increase rapidly and becomes noncompensable. The thermoregulatory failure aggravates pathophysiological processes at the cellular level, including an inflammatory reaction, and multiorgan failure occurs as a result of the combination of high body temperature and circulatory collapse, and ultimately is expressed as heatstroke. DIC denotes disseminated intravascular coagulation.

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Table 3. Guidelines for the Treatment of Heatstroke.*

Treatment	Comments
Treatment on site	
CPR	Perform according to ACLS protocol; administer oxygen at 4 liters/min to increase oxygen saturation to >90%
Core body temperature	Monitor rectal temperature and perform cooling in cases of hyperthermia; for exertional heatstroke, cold-water immersion; for classic heatstroke, conductive or evaporative cooling
Fluids	Administer isotonic saline IV (1–2 liters/hr); dehydration is not a major issue
Seizure medication	Administer benzodiazepines IV (5 mg) until seizures cease (not more than 20 mg)
Evacuation	For classic heatstroke, transport immediately to ED; for exertional heatstroke, transport to ED after cooling to body temperature <39.0°C
Treatment in the ED	
Core body temperature	Monitor rectal or intravesical temperature and perform cooling until core temperature <38.0°C; use either a cooling suit or cold fluids (4°C, 1000 ml/30 min) infused through central catheter; antipyretics are toxic and should be avoided; dantrolene has not been proved to be effective
Seizure medication	Administer benzodiazepines IV (5 mg, repeated) or phenytoin IV (loading dose, 15–20 mg/kg in 15 min) until seizures cease
Laboratory testing	Perform CBC, urinalysis, blood cultures, kidney-function and liver-function tests (ALT, AST, ammonia, INR); test for glucose, electrolytes, arterial blood gases and acid–base balance, clotting function, CK, LDH, myoglobin, CRP

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Table 3. (Continued.)

Treatment	Comments
Monitoring of circulation	For circulatory failure, administer fluids (30 ml/kg), monitor CVP or perform invasive hemodynamic monitoring, maintain mean arterial pressure at >65 mm Hg (or >75 mm Hg if patient is elderly or has hypertension), all with a goal of normal lactate level and urine output >50 ml/kg/hr; vasopressors should be considered if fluid therapy fails
Treatment in the ICU	
General	Perform CPR according to ACLS protocol; ECMO may be used as needed
	Monitor rectal, intravesical, or blood temperature; continue cooling to maintain core temperature at <38.0°C by infusing cold fluids (4°C, 1000 ml/30 min) through central catheter or use extracorporeal blood cooling for resistant hyperthermia; antipyretics are toxic and should be avoided; dantrolene has not been proved to be effective
	Perform laboratory tests: CBC, glucose, arterial blood gases and acid–base balance, clotting function, CK, LDH, liver function (ALT, AST, ammonia, INR), myoglobin, kidney function, urinalysis, CRP, blood cultures; repeat every 12 hr during the first 48 hr, then every 24 hr
Heart failure	Perform CPR according to ACLS protocol; perform invasive hemodynamic monitoring and echocardiography; for mild multiorgan failure, administer dobutamine IV (1 μg/kg/min, then 2–20 μg/kg/min as needed) or milrinone IV (loading dose, 50 μg/kg in 10 min, then 0.2–0.75 μg/kg/min) or adrenaline IV (1 μg/min); for severe multiorgan failure, ECMO may be used as needed
Acute kidney injury	Administer crystalloid solution to maintain urine output >50 ml/kg/hr; administer furosemide IV (10–20 mg in patients without previous exposure to diuretics; follow-up dose depends on urine output); provide hemodialysis or CVVH in cases of volume overload, severe acidosis, hyperkalemia, or uremia; adjust fluid infusion rate according to blood pressure and urine output; monitor electrolytes and correct as needed

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Encephalopathy and brain edema	For a score of <8 on the GCS,† intubate and ventilate; for mild hyperventilation (Pco_2, 34–36 mm Hg) administer hypertonic saline 3% IV (starting dose, 100 ml/30 min, then according to patient's total body water to reach sodium level increase of 12 mmol/day) or mannitol 20% IV (0.25–2 g/kg in 30 min); keep head at 45-degree angle, administer tranquilizers; patients with hyperammonemia require hemofiltration or MARS therapy; condition improves with cooling; consider monitoring ICP
Rhabdomyolysis	Administer IV fluid infusion, 1–2 liters/hr (aggressive fluid treatment in the first hour), then 300 ml/hr; furosemide IV (10–20 mg in patients without previous diuretic treatment; follow-up dose depends on urine output) in case of fluid overload; sodium bicarbonate, 30 mmol/hr (to achieve urine pH >6.5); myoglobinuria is expected; hypercalcemia and metabolic alkalosis (pH >7.5) should be avoided
DIC and other coagulation abnormalities	For bleeding and thrombosis, administer fresh-frozen plasma (bolus dose, 10–15 ml/kg, then 200–400 ml according to coagulation indexes); administer cryoprecipitate (5–10 U each time) for fibrinogen level of <180 mg/dl; administer platelet concentrates (infusion of one therapeutic dose) if platelet count <20 per mm^3 or if there is bleeding and platelet count <50 per mm^3; in patients with hepatic failure, consider PCC to achieve a target INR ≤1.5; inject PCC dose according to INR and patient's weight; avoid heparin; beware of hypothermia and metabolic acidosis
ARDS	Perform intubation and mechanical ventilation; avoid fluid overload
Liver failure	Monitor liver function and mental status for at least 4 days; provide supportive treatment: hemodynamic stability, *N*-acetylcysteine IV (bolus dose, 150 mg/kg in 200 ml of 5% glucose solution for 20 min, then 50 mg/kg in 500 ml of 5% glucose solution for 4 hr, then 100 mg/kg in 1000 ml of 5% glucose solution for 16 hr); administer hypertonic saline 3% IV or mannitol IV (0.25–2 g/kg in 30 min in 20% solution), hemofiltration, laxatives (e.g., oral lactulose, 30 ml every 2 hr until diarrhea occurs), oral rifaximin (400 mg 3 times a day) in case of fulminant liver failure; liver transplantation rarely needed, and there is no evidence that it is effective
ECG changes	Monitor continuously for possible arrhythmias; ECG changes are nonspecific
SIRS	Treat the same as sepsis; consider antibiotics

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En Europe en 2022

nature medicine

Article

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Heat-related mortality in Europe during the summer of 2022

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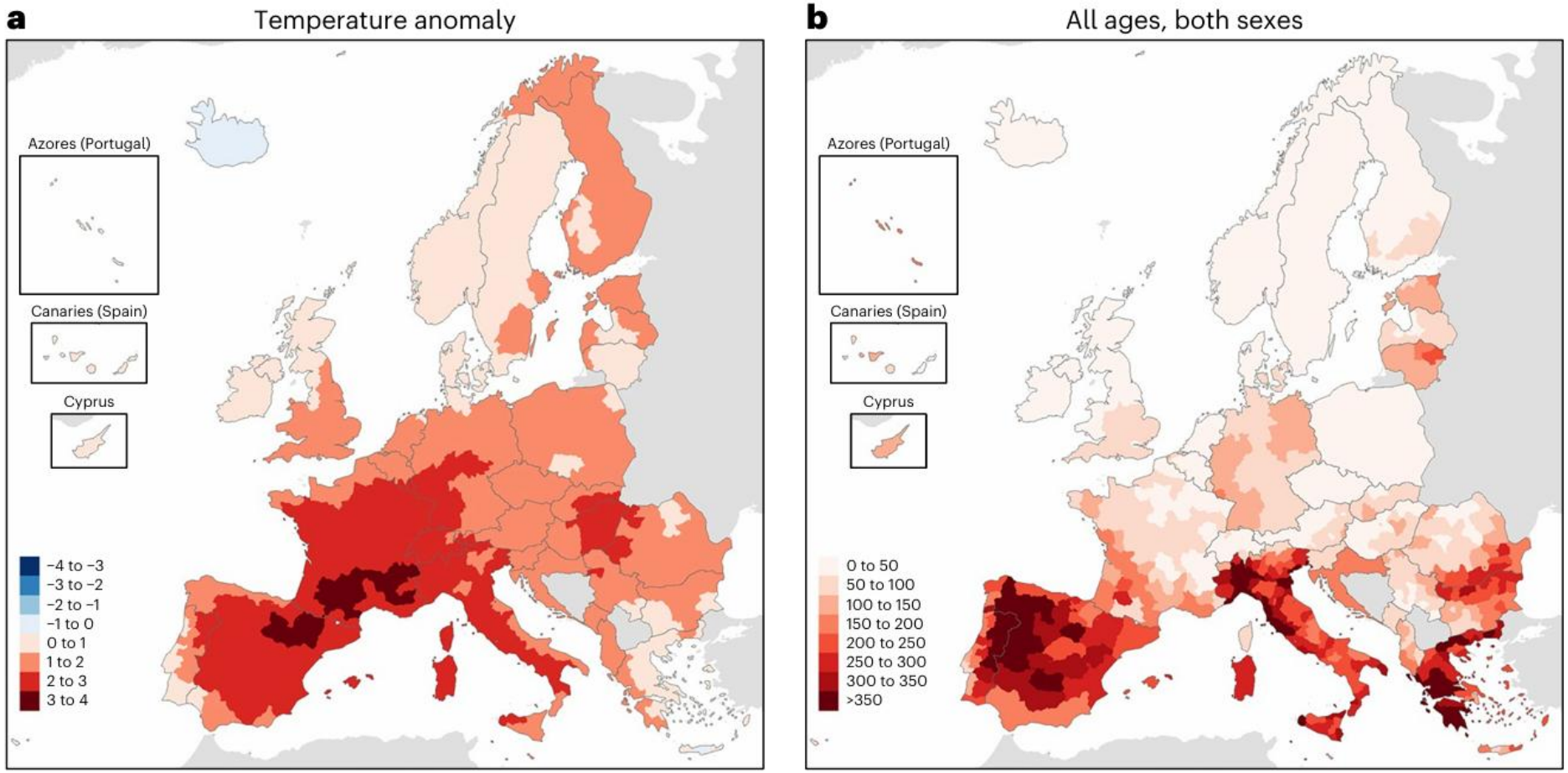
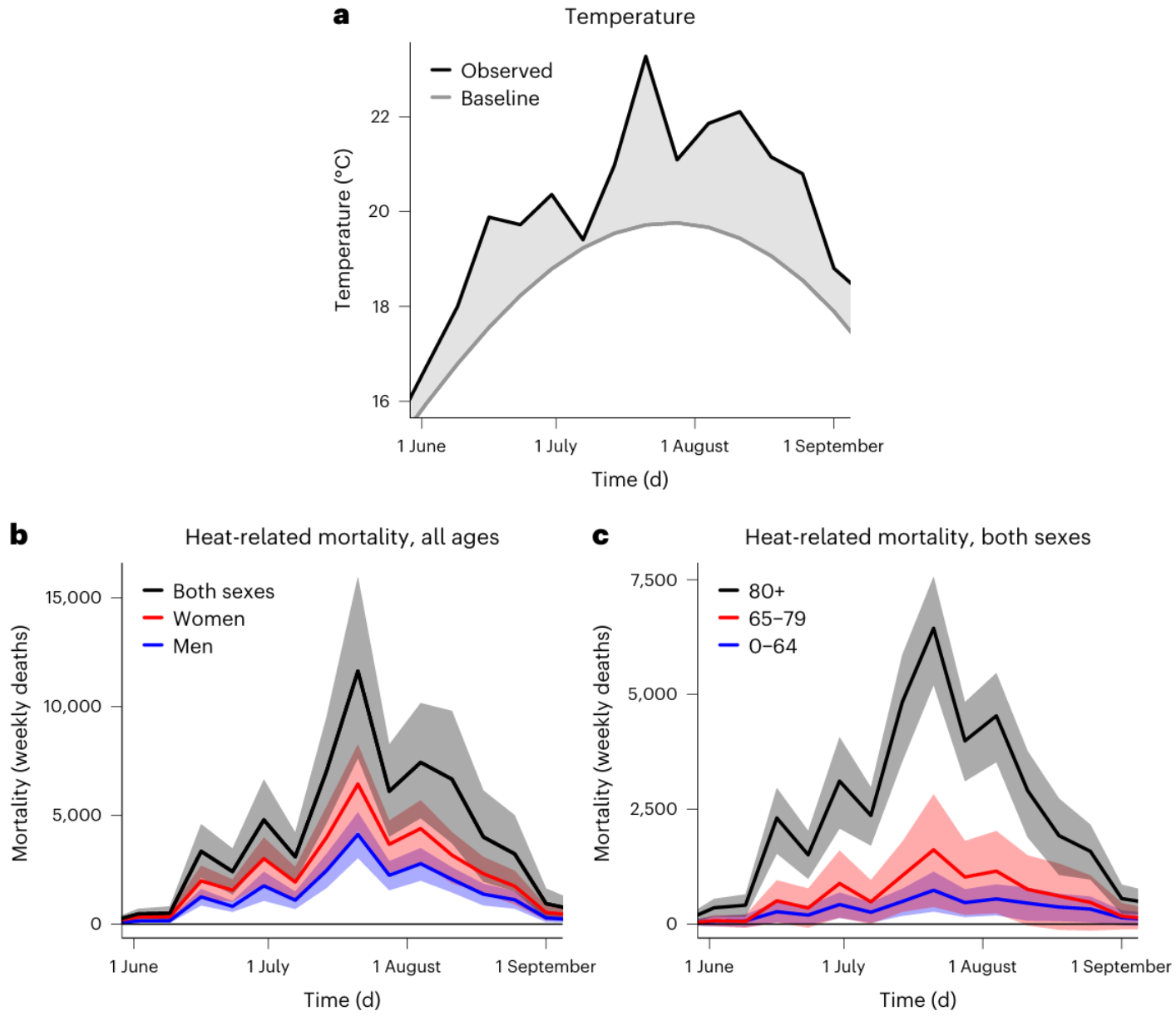
Entre le 30 mai et le 4 septembre 2022

- Analyse de la base de données de mortalité d'Eurostat : 45.184.044 cas de décès provenant de 823 régions contiguës dans 35 pays européens, représentant l'ensemble de la population de plus de 543 millions de personnes.
- Nombre de décès liés à la chaleur en Europe entre le 30 mai et le 4 septembre 2022 : estimé à 61.672 (intervalle de confiance [IC] à 95 % : 37.643–86.807)

Anomalies de température régionales et taux de mortalité liés à la chaleur durant l'été 2022




a
Temperature
Observed
Baseline
Temperature (°C)
22
20
18
16
1 June
1 July
1 August
1 September
Time (d)
b
Heat-related mortality, all ages
Both sexes
Women
Men
Mortality (weekly deaths)
15,000
10,000
5,000
0
1 June
1 July
1 August
1 September
Time (d)
c
Heat-related mortality, both sexes
80+
65–79
0–64
Mortality (weekly deaths)
7,500
5,000
2,500
0
1 June
1 July
1 August
1 September
Time (d)

- L’Italie (18.010 décès ; IC à 95 % : 13.793–22.225), l’Espagne (11.324 ; IC à 95 % : 7.908–14.880) et l’Allemagne (8.173 ; IC à 95 % : 5.374–11.018) ont enregistré les nombres de morts les plus élevés liés à la chaleur
- L’Italie (295 décès par million d’habitants, IC à 95 % : 226–364), la Grèce (280 ; IC à 95 % : 201–355) et l’Espagne (237 ; IC à 95 % : 201–355) ont enregistré des taux de mortalité liés à la chaleur les plus élevés.
- 56 % de décès liés à la chaleur de plus chez les femmes que chez les hommes, avec des taux plus élevés chez les hommes âgés de 0 à 64 ans (+41 %) et de 65 à 79 ans (+14 %), et chez les femmes âgées de 80 ans et plus (+27 %)

En France

Rev Prescrire 2026 ; 46 (509) : 213-217

- Les vagues de chaleur entraînent un excès de mortalité : de 2014 à 2022, 33.000 morts, dont 70 % de personnes âgées de plus de 75 ans
- Principaux facteurs d'aggravation de la vulnérabilité aux vagues de chaleur : âges extrêmes (très jeunes enfants et personnes âgées), l'isolement, les comorbidités (cancers, maladies cardio vasculaires, respiratoires chroniques, neurodégénératives ou métaboliques, dont le diabète et la maladie d'Alzheimer)
- Cette vulnérabilité s'explique par un plus grand risque de déshydratation et ses conséquences : hémoconcentration, déséquilibre électrolytique pouvant retentir sur la fonction rénale, le tout aggravé par la prise de certains médicaments (diurétiques, hypotenseurs, etc.) pouvant conduire à un état de choc

Recommandations de la Cour des Comptes (France, 2024)

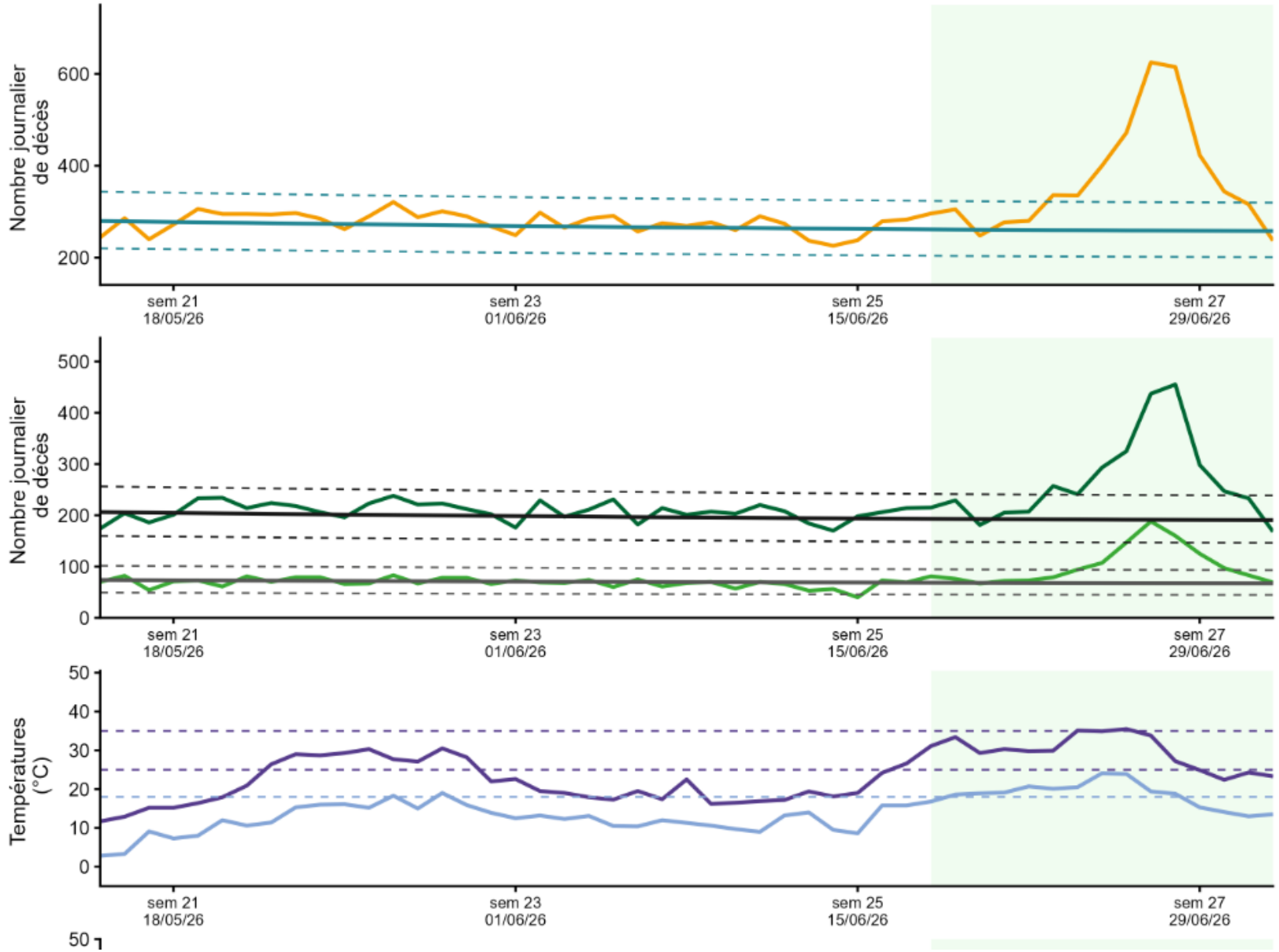
1. conduire les travaux nécessaires à une meilleure connaissance des conséquences des vagues de chaleur sur la santé des personnes vulnérables, adapter ces travaux aux spécificités de l'outre-mer et améliorer les indicateurs de veille et d'alerte
2. se doter des moyens de mieux connaître la situation sanitaire des personnes sans domicile fixe, en développant une base de données de santé adossée au système d'information du service intégré d'accueil et d'orientation, en coopération avec les organismes gestionnaires de structures d'hébergement et de soins

3. associer les sociétés savantes et les associations d'usagers à l'élaboration, par l'agence nationale de sécurité du médicament, de la liste des médicaments d'intérêt en cas de vague de chaleur et la diffuser systématiquement aux professionnels de santé (médecins, pharmaciens, infirmiers)
4. élargir les critères d'inscription des personnes les plus vulnérables sur le registre municipal et substituer à un accord préalable annuel un droit d'opposition permanent
5. réaliser un inventaire du parc immobilier des établissements sanitaires, sociaux et médicosociaux pour évaluer son adaptation aux vagues de chaleur

En Belgique

Sciensano (8 juillet 2026)

Sur l'ensemble de la vague de chaleur, qui s'est étendue du 18 juin au 1er juillet, 1.747 décès supplémentaires ont été enregistrés par rapport au nombre attendu, soit une surmortalité de 47,8 %. La Wallonie a été la région la plus durement touchée, avec une surmortalité de 76 %. Cette vague de chaleur n'a pas uniquement affecté les personnes âgées : une surmortalité de 61,3 % a également été observée chez les personnes âgées de 15 à 64 ans. Ces résultats proviennent de Be-MOMO, le système de surveillance de la mortalité toutes causes confondues de Sciensano.


Nombre journalier de décès
600
400
200
sem 21
18/05/26
sem 23
01/06/26
sem 25
15/06/26
sem 27
29/06/26
Nombre journalier de décès
500
400
300
200
100
0
sem 21
18/05/26
sem 23
01/06/26
sem 25
15/06/26
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29/06/26
Températures (°C)
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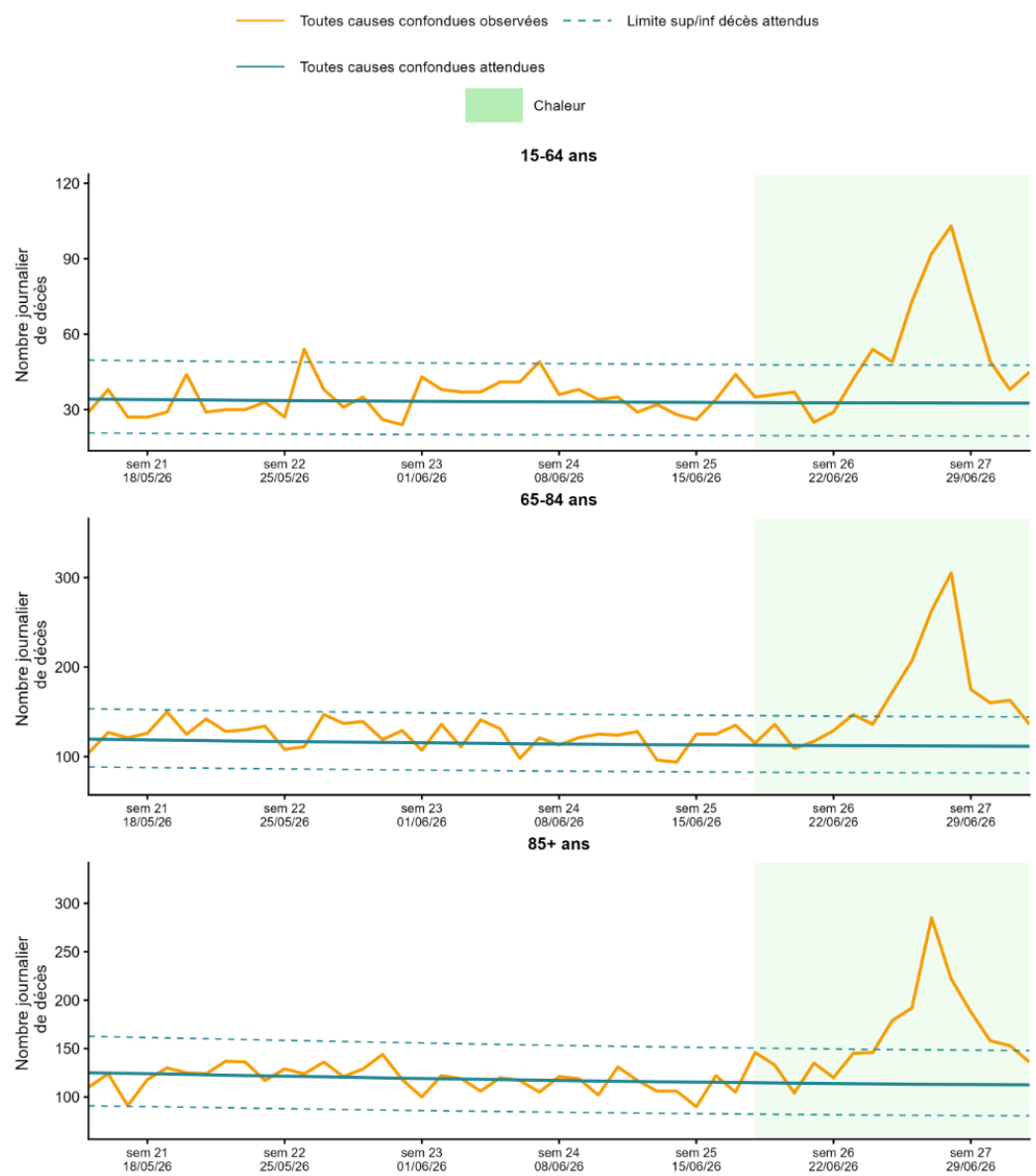


igure 2. La mortalité par groupes d'âge, du 15 mai 2026 au 1 juillet 2026, Belgique

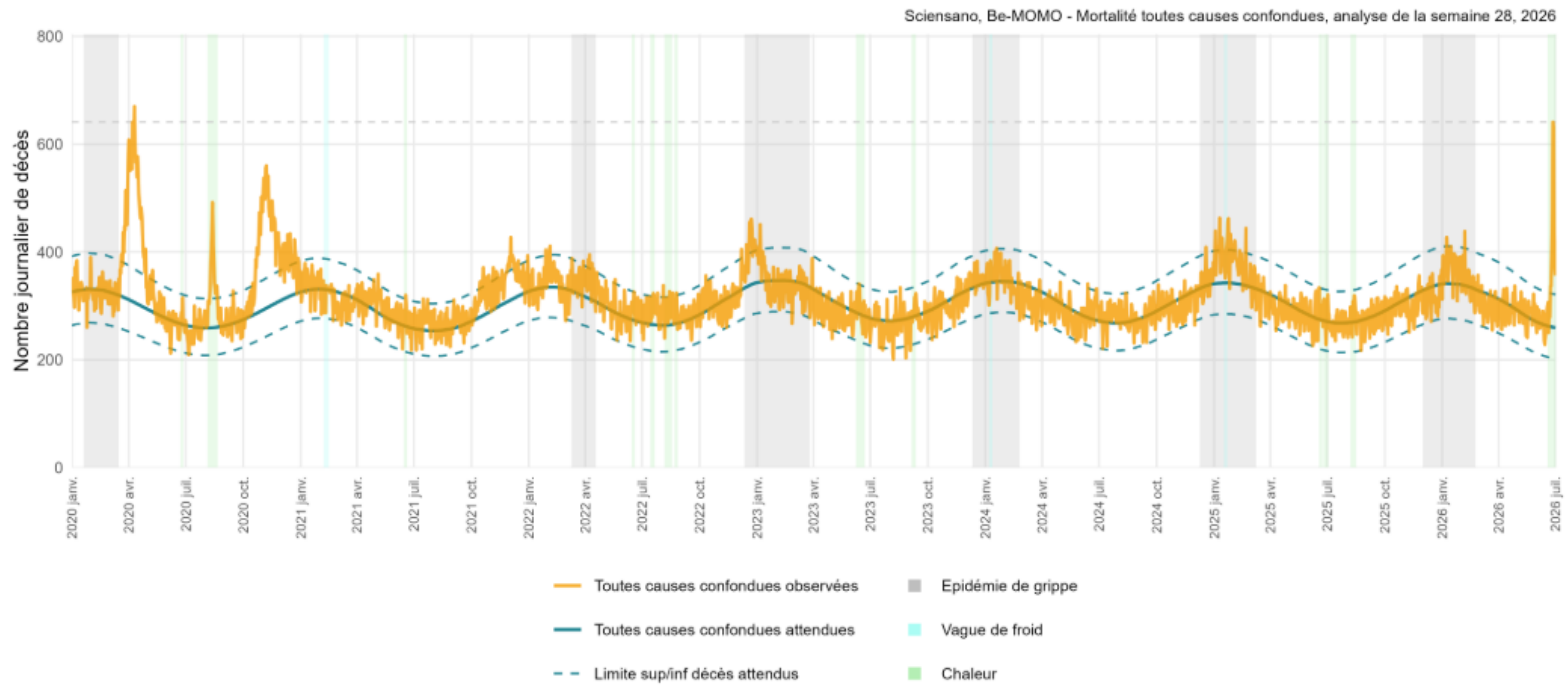


Figure 3. Mortalité toutes causes confondues, de janvier 2020 au 1 juillet 2026, Belgique

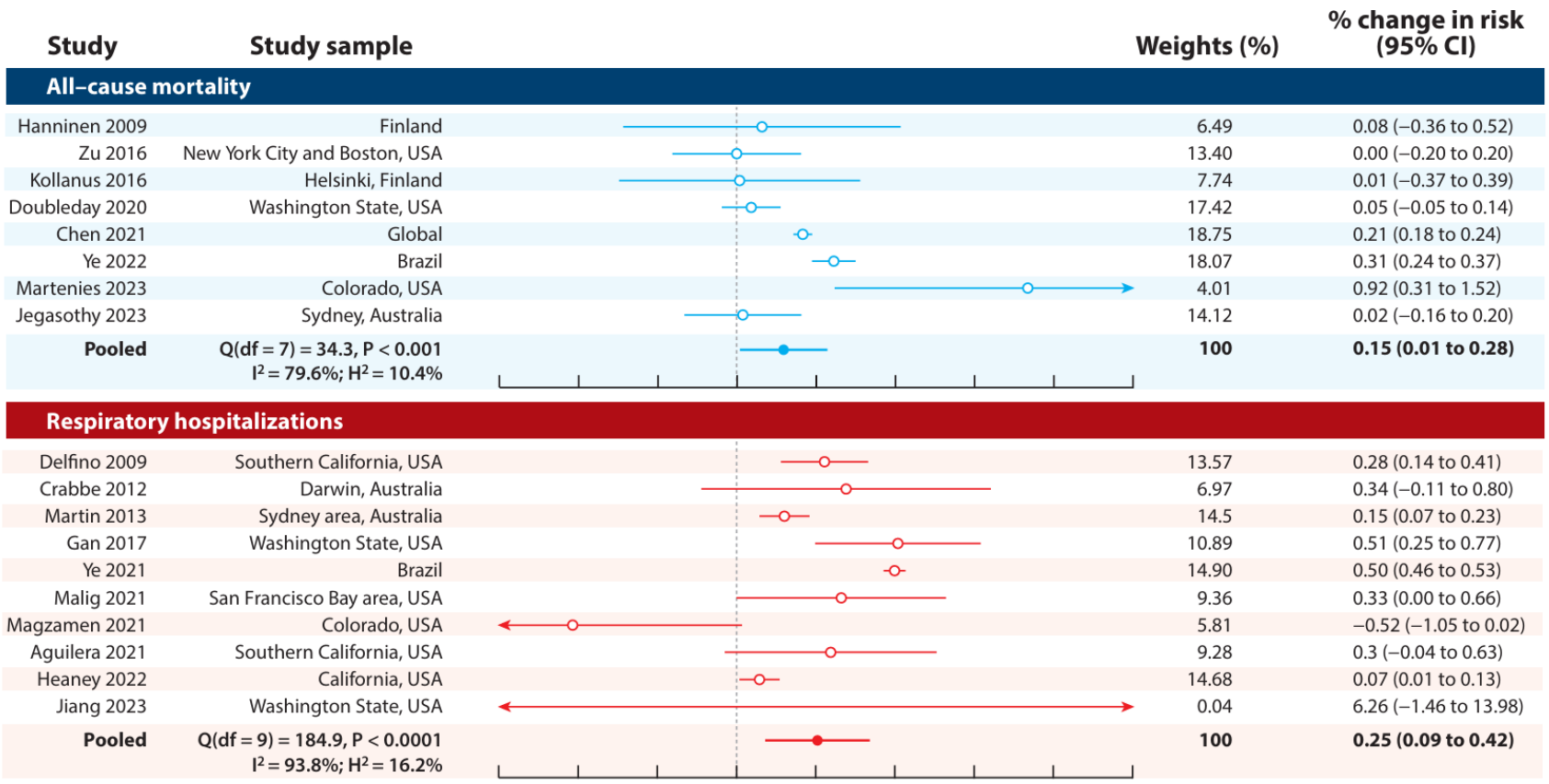
Médicaments qui causent ou aggravent un coup de chaleur, ou ses conséquences (Prescrire, 2026)

- neuroleptiques tels que la chlorpromazine
- atropiniques dont la scopolamine, autorisée notamment comme antinauséeux dans le mal des transports ; des médicaments de l'incontinence urinaire par impériosité tels que l'oxybutynine ; des antihistaminiques H1 tels que la prométhazine
- hormones thyroïdiennes
- inhibiteurs de l'anhydrase carbonique antiépileptiques, le topiramate et le zonisamide ; un diurétique, l'acétazolamide
- médicaments sérotoninergiques, notamment des antidépresseurs tels que la fluoxétine, la venlafaxine, la vortioxétine ou l'amitriptyline, avec des opioïdes tels que le tramadol
- cholinergiques : la pilocarpine ; les anticholinestérasiques utilisés dans la maladie d'Alzheimer : le donépézil, la galantamine, la rivastigmine ; les anticholinestérasiques utilisés dans la myasthénie : la néostigmine, la pyridostigmine, le chlorure d'ambénonium
- alcool, les amphétamines, la cocaïne, l'ecstasy
- par insuffisance rénale fonctionnelle en cas de déshydratation : les diurétiques ; les anti-inflammatoires non stéroïdiens (AINS) ; les inhibiteurs de l'enzyme de conversion (IEC) et les sartans ; les gliflozines
- par aggravation d'une hypotension artérielle : les bêtabloquants et les autres hypotenseurs

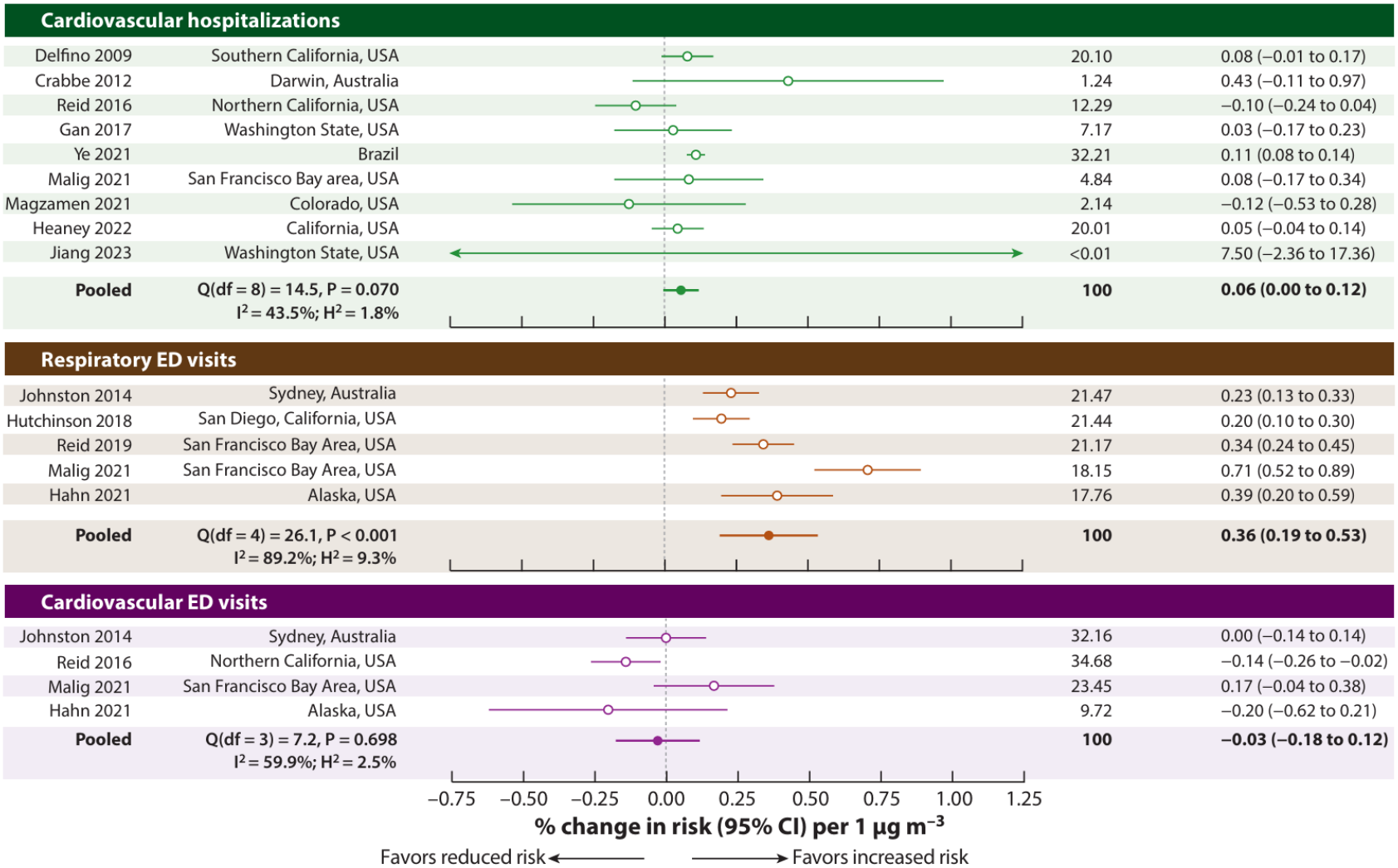
Les incendies de forêt

- Grands incendies de forêt récents (JAMA September, 2024, 332, pp 1011-2) :
  - États-Unis (2017-2023)
  - Australie(2019-2020)
  - Canada (2023)
  - Grèce (2023)
  - Russie (2021)
  - Amazonie (2019)
  - Bolivie (2010)

Méta-analyse des associations entre les particules fines ambiantes spécifiques aux feux de forêt et les résultats sanitaires le jour même par 1 µg /m³.



Study	Study sample	Weights (%)	% change in risk (95% CI)
All–cause mortality			
Hanninen 2009	Finland	6.49	0.08 (–0.36 to 0.52)
Zu 2016	New York City and Boston, USA	13.40	0.00 (–0.20 to 0.20)
Kollanus 2016	Helsinki, Finland	7.74	0.01 (–0.37 to 0.39)
Doubleday 2020	Washington State, USA	17.42	0.05 (–0.05 to 0.14)
Chen 2021	Global	18.75	0.21 (0.18 to 0.24)
Ye 2022	Brazil	18.07	0.31 (0.24 to 0.37)
Martenies 2023	Colorado, USA	4.01	0.92 (0.31 to 1.52)
Jegasothy 2023	Sydney, Australia	14.12	0.02 (–0.16 to 0.20)
Pooled	**Q(df = 7) = 34.3, P < 0.001** **I^2 = 79.6%; H^2 = 10.4%**	**100**	**0.15 (0.01 to 0.28)**
Respiratory hospitalizations			
Delfino 2009	Southern California, USA	13.57	0.28 (0.14 to 0.41)
Crabbe 2012	Darwin, Australia	6.97	0.34 (–0.11 to 0.80)
Martin 2013	Sydney area, Australia	14.5	0.15 (0.07 to 0.23)
Gan 2017	Washington State, USA	10.89	0.51 (0.25 to 0.77)
Ye 2021	Brazil	14.90	0.50 (0.46 to 0.53)
Malig 2021	San Francisco Bay area, USA	9.36	0.33 (0.00 to 0.66)
Magzamen 2021	Colorado, USA	5.81	–0.52 (–1.05 to 0.02)
Aguilera 2021	Southern California, USA	9.28	0.3 (–0.04 to 0.63)
Heaney 2022	California, USA	14.68	0.07 (0.01 to 0.13)
Jiang 2023	Washington State, USA	0.04	6.26 (–1.46 to 13.98)
Pooled	**Q(df = 9) = 184.9, P < 0.0001** **I^2 = 93.8%; H^2 = 16.2%**	**100**	**0.25 (0.09 to 0.42)**


Cardiovascular hospitalizations
Delfino 2009 Southern California, USA 20.10 0.08 (−0.01 to 0.17)
Crabbe 2012 Darwin, Australia 1.24 0.43 (−0.11 to 0.97)
Reid 2016 Northern California, USA 12.29 −0.10 (−0.24 to 0.04)
Gan 2017 Washington State, USA 7.17 0.03 (−0.17 to 0.23)
Ye 2021 Brazil 32.21 0.11 (0.08 to 0.14)
Malig 2021 San Francisco Bay area, USA 4.84 0.08 (−0.17 to 0.34)
Magzamen 2021 Colorado, USA 2.14 −0.12 (−0.53 to 0.28)
Heaney 2022 California, USA 20.01 0.05 (−0.04 to 0.14)
Jiang 2023 Washington State, USA <0.01 7.50 (−2.36 to 17.36)
Pooled Q(df = 8) = 14.5, P = 0.070 I² = 43.5%; H² = 1.8% 100 0.06 (0.00 to 0.12)
Respiratory ED visits
Johnston 2014 Sydney, Australia 21.47 0.23 (0.13 to 0.33)
Hutchinson 2018 San Diego, California, USA 21.44 0.20 (0.10 to 0.30)
Reid 2019 San Francisco Bay Area, USA 21.17 0.34 (0.24 to 0.45)
Malig 2021 San Francisco Bay Area, USA 18.15 0.71 (0.52 to 0.89)
Hahn 2021 Alaska, USA 17.76 0.39 (0.20 to 0.59)
Pooled Q(df = 4) = 26.1, P < 0.001 I² = 89.2%; H² = 9.3% 100 0.36 (0.19 to 0.53)
Cardiovascular ED visits
Johnston 2014 Sydney, Australia 32.16 0.00 (−0.14 to 0.14)
Reid 2016 Northern California, USA 34.68 −0.14 (−0.26 to −0.02)
Malig 2021 San Francisco Bay Area, USA 23.45 0.17 (−0.04 to 0.38)
Hahn 2021 Alaska, USA 9.72 −0.20 (−0.62 to 0.21)
Pooled Q(df = 3) = 7.2, P = 0.698 I² = 59.9%; H² = 2.5% 100 −0.03 (−0.18 to 0.12)
−0.75 −0.50 −0.25 0.00 0.25 0.50 0.75 1.00 1.25
% change in risk (95% CI) per 1 µg m⁻³
Favors reduced risk
Favors increased risk

Excédent de décès attribuables aux incendies de forêt de Los Angeles du 5 janvier au 1er février 2025

Table 1. Estimation of Weekly and Total Excess Deaths Attributable to the California Wildfires in Los Angeles County From January 5 to February 1, 2025[a]

Calendar week	Observed deaths, No.	Expected deaths, No. (95% CI)	Excess deaths, No. (95% CI)	Pr(Excess >0)	PAF, % (95% CI)
2	1570	1484 (1357 to 1618)	86 (−48 to 213)	0.91	6.0 (−3.0 to 15.7)
3	1592	1492 (1360 to 1616)	100 (−24 to 232)	0.94	6.9 (−1.5 to 17.1)
4	1570	1484 (1355 to 1621)	86 (−51 to 215)	0.90	6.0 (−3.1 to 15.9)
5	1639	1472 (1342 to 1597)	167 (42 to 297)	>0.99	11.6 (2.6 to 22.1)
Total	6371	5931 (5627 to 6240)	440 (131 to 744)	>0.99	6.9 (2.1 to 11.7)

Abbreviation: PAF, population attributable fraction.

[a] Expected deaths, excess deaths, and PAFs are stochastic quantities. To illustrate the uncertainty in the current estimation, the mean followed by 95% confidence intervals in parentheses are reported. Also reported is the probability of positive excess in the Pr(Excess >0) column. Excess deaths were calculated as the difference between observed deaths and deaths expected based on prior trends. PAFs were calculated as excess deaths divided by observed deaths, multiplied by 100 to get percentages. Uncertainty intervals are percentile based, obtained by computing each metric separately for a sample from the asymptotic distribution of modeled death counts and then computing the 2.5th and 97.5th percentiles. The probability of positive excess mortality was similarly calculated as the proportion of samples from the asymptotic distribution in which observed deaths exceeded expected deaths and there was a positive number of excess deaths.

Table 2. Results From Sensitivity Analyses[a]

Specification	Observed deaths, No.	Expected deaths, No. (95% CI)	Excess deaths, No. (95% CI)	Pr(Excess >0)	PAF, % (95% CI)
Primary analysis					
All causes (2020-2023 excluded)	6371	5931 (5627 to 6240)	440 (131 to 744)	>0.99	6.9 (2.1 to 11.7)
Sensitivity analyses					
Removing influenza and pneumonia (2020-2023 excluded)	5999	5612 (5344 to 5861)	387 (138 to 655)	>0.99	6.5 (2.3 to 10.9)
Removing all respiratory diseases (2020-2023 excluded)	5693	5302 (5063 to 5557)	391 (136 to 630)	>0.99	6.9 (2.4 to 11.1)
Removing all external deaths (2020-2023 excluded)	5965	5546 (5231 to 5875)	419 (90 to 734)	>0.99	7.0 (1.5 to 12.3)
All causes CITS design (2020-2023 excluded)	6371		422 (9 to 807)	0.98	6.6 (0.1 to 12.7)
All causes (2020-2022 excluded)	6371	5815 (5547 to 6078)	556 (293 to 824)	>0.99	8.7 (4.6 to 12.9)
All causes, weeks 2-9 (2020-2023 excluded)	12 009	11 677 (11 197 to 12 143)	332 (−134 to 812)	0.92	2.8 (−1.1 to 6.8)
All causes placebo (2020-2023 excluded)	6966	7134 (6809 to 7481)	−168 (−515 to 157)	0.16	−2.4 (−7.4 to 2.3)

Abbreviations: CITS, controlled interrupted time series; PAF, population attributable fraction.

[a] Expected deaths, excess deaths, and PAFs are stochastic quantities. To illustrate the uncertainty in the current estimation, the mean followed by 95% confidence intervals in parentheses are reported. Also reported is the probability of positive excess in the Pr(Excess >0) column. Excess deaths were calculated as the difference between observed deaths and deaths expected based on prior trends. PAFs were calculated as excess deaths divided by observed deaths, multiplied by 100 to get percentages. Uncertainty intervals are percentile based, obtained by computing each metric separately for a sample from the asymptotic distribution of modeled death counts and then computing the 2.5th and 97.5th percentiles. The probability of positive excess mortality was similarly calculated as the proportion of samples from the asymptotic distribution in which observed deaths exceeded expected deaths and there was a positive number of excess deaths. See the eMethods in Supplement 1 for additional information on each sensitivity check.