Infectious Medicine 4 (2025) 100184

Contents lists available at ScienceDirect

Infectious Medicine

journal homepage: www.elsevier.com/locate/imj

Perspective

# Occupational zoonoses, neurological diseases, and public health: A one health approach

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ARTICLE INFO

*Keywords:*
Occupational zoonoses
Ecohealth
One Health
Workplace safety
Neurological diseases
Zoonotic pathogens

ABSTRACT

Zoonotic diseases, which constitute 60% of all human infectious diseases, present substantial risks to public health, economies, and livelihoods. These diseases emerge at the human-animal-environment interface, with occupational exposure representing a critical yet underexamined dimension of zoonotic risk. Workers in high-risk sectors such as agriculture, wildlife management, and laboratory research face elevated exposure to zoonotic pathogens, often under conditions of inadequate preventive measures and resource constraints. Neurological disorders resulting from zoonotic infections, including Guillain-Barré syndrome, encephalitis, and meningitis, illustrate the severe health consequences for occupational groups. Cases linked to swine hepatitis E virus, West Nile virus, *Streptococcus suis*, and *Baylisascaris procyonis* underscore the urgent need for robust surveillance and targeted interventions.

The Ecohealth approach, integrated with the One Health framework, provides a transformative model for managing zoonotic risks by addressing the upstream drivers of disease emergence. By emphasizing environmental stewardship, ecological balance, and socio-economic equity, Ecohealth fosters sustainable preventive strategies. Occupational medicine is crucial in linking workplace safety with public health through tailored risk management, enhanced surveillance, and targeted education.

Despite these frameworks, significant barriers persist, including data gaps, underreporting of occupational diseases, and insufficient coordination among health sectors. Addressing these challenges requires implementing standardized occupational health surveillance systems, enhancing reporting mechanisms through digital tools, and promoting cross-sectoral data-sharing initiatives. Successful models, such as sentinel surveillance programs in agricultural sectors and integrated biosurveillance networks, demonstrate the feasibility of these strategies. Leveraging these approaches can facilitate early detection, improve reporting accuracy, and support evidence-based interventions.

## 1. Understanding occupational zoonoses: Risks, challenges, and global health strategies

Zoonoses are infectious diseases transmitted naturally between vertebrate animals and humans and constitute 58% of all human infectious diseases, pose significant risks to both human and animal health, and impact economies and livelihoods.[1,2] Transmission occurs at the human-animal-environment interface, where interactions in shared environments facilitate pathogen spread.[2] The emergence of zoonoses in humans traces back to the advent of agriculture and domestication, as humans began living close to animals. Historically, zoonoses were classified as anthropozoonoses, transmissible from animals to humans, and zooanthroponoses, transmissible from humans to animals. However, due to frequent confusion, the

*Abbreviations:* WHO, World Health Organization; HEV, hepatitis E virus; WNV, West Nile virus; CNS, central nervous system; PPE, personal protective equipment; NV, Nipah virus; PEP, post-exposure prophylaxis.

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https://doi.org/10.1016/j.imj.2025.100184
Received 30 December 2024; Received in revised form 15 April 2025; Accepted 20 April 2025
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term zoonosis has been universally adopted to refer to diseases shared between humans and animals.[3]

Currently, the European Union's Directive 2003/99/EC offers a broad definition of viruses, bacteria, fungi, parasites, or other biological entities causing zoonoses, even those involving non-vertebrate hosts.[4] Transmission occurs through direct contact, environmental contamination, vectors, as well as foodborne and waterborne routes. Various occupational contexts, including veterinary clinics, farms, meat processing plants, and wildlife rehabilitation facilities, present higher risk due to frequent exposure to zoonotic agents. Occupational exposure to zoonotic agents in particular refers to the risk of encountering zoonotic pathogens in a work environment, either through direct or indirect contact. Direct exposure involves physical interaction with infected animals, biological materials, or contaminated surfaces (e.g., handling livestock, processing meat, conducting laboratory research). Indirect exposure, on the other hand, occurs through environmental contamination, vector-borne transmission, or exposure to airborne pathogens in occupational settings where direct animal contact is absent (e.g., farm workers inhaling dust particles carrying infectious agents, wildlife officers working in contaminated areas). Even workers without direct contact with animals, such as those in outdoor environments, are at risk because of soil, water, and air contamination or vectors.[1] Additional occupational risk factors are animal health status, frequency and type of contact, environmental conditions, and occupational training level.[5]

Despite the recognition of zoonotic threats, gaps in epidemiological knowledge still hinder effective prevention. Zoonotic diseases are sill often misdiagnosed. Moreover, quantifying occupational risk requires reliable estimates of both infection probabilitiy and consequences. In many contexts, lack of data on the likelihood of infection hampers comprehensive risk assessments, limiting the ability to provide workers with evidence-based guidance.[6] Furthermore, demonstrating the association between zoonoses and occupational exposure is often complex and requires detailed epidemiological investigations. As a result, many occupational zoonoses are underreported.[7]

Nevertheless, control and surveillance of occupational zoonoses have been longstanding concerns. As early as 1975, the World Health Organization (WHO) recognized zoonoses as occupational hazards, emphasizing the need for targeted prevention.[8] Subsequent meetings, such as the 1977 expert consultation in Rome, identified high-risk occupational groups and classified zoonoses based on their socio-economic and public health impacts.[9] These classifications highlighted zoonoses with severe consequences for humans and economically important animals. Moreover, recent global events, such as the Ebola, influenza, and COVID-19 outbreaks have taxed global health systems, food supply chains, and economies underscoring the escalating threat of zoonotic pathogens. Environmental changes, including agricultural intensification, climate change, and encroachment into wildlife habitats are key drivers of zoonotic emergence.[10]

The One Health approach emerged as a pivotal strategy for addressing zoonotic diseases. This collaborative multidisciplinary framework emphasizes the interconnectedness of human, animal, and environmental health and seeks to optimize resource use, healthcare delivery, and prevention at local, national, and global levels.[11] International organizations, including the WHO, the Food and Agriculture Organization (FAO), and the World Organisation for Animal Health (WOAH), have endorsed One Health principles through initiatives like the Tripartite Zoonoses Guide.[12] These efforts promote sustained collaboration among sectors to enhance zoonoses surveillance and prevention.

Despite the progress facilitated by the One Health approach, significant barriers remain. In many cases, coordination among human health, animal health, and environmental sectors are insufficient, hindering comprehensive strategies. Additionally, information specific to the occupational risks of certain zoonoses remains incomplete or unavailable, limiting the capacity to inform public health policies and protect workers effectively. Addressing these gaps requires identifying high-risk occupations, integrating prevention into workplace health frameworks, and fostering collaboration between stakeholders to ensure evidence-based decision-making.[13]

By leveraging the One Health approach, public health authorities can prioritize zoonosis prevention in occupational settings, mitigating risks to workers and reducing the broader societal and economic impact. Enhanced surveillance and improved occupational training are critical for safeguarding the health and livelihoods of workers while contributing to global health security.

## 2. Bridging knowledge gaps in occupational zoonoses: Public health and economic imperatives

Zoonotic diseases impose profound economic and public health burdens, with direct costs exceeding $20 billion globally and indirect losses surpassing $200 billion over the past decade.[14] These impacts are felt across diverse sectors, from agriculture to healthcare, and disproportionately affect workers in high-risk occupations. The interplay between occupational exposure and zoonotic diseases remains poorly understood, particularly regarding long-term health outcomes such as neurological complications and the broader implications for public health systems. Addressing these gaps is critical to mitigating the cascading effects of zoonoses on both individuals and economies.[15]

Occupational exposure to zoonotic pathogens occurs in agriculture, livestock management, forestry, and laboratory settings, where contact with infected animals, contaminated environments, or biological materials are frequent. Workers in these sectors often face heightened risks due to inadequate prevention and limited resources. For instance, agricultural workers frequently handle bioaerosols, infected tissues, and arthropod vectors, exposing them to respiratory diseases, allergic reactions, and zoonoses. Yet, the absence of robust occupational health programs and the challenges of tracing exposure hinder effective management.[16]

The economic consequences of zoonotic outbreaks extend into agricultural productivity, food security, and trade. At the household level, diminished livestock productivity due to zoonoses reduces income and impacts national economies through decreased revenue and higher consumer prices.[17] Recurring outbreaks, such as avian influenza, further strain global trade and highlight the pandemic potential of certain livestock pathogens, some of which require only minor genetic mutations to threaten human populations.[18] However, these calculations often exclude the hidden costs associated with zoonotic epidemics, such as psychological stress, healthcare system disruptions, and postponed treatments, which amplify the burden on affected communities.

The occupational dimension of zoonoses also intersects with ecological disruption, where human activities such as farming, hunting, and wildlife management increase pathogen exposure risks. For example, studies on tick-borne typhus demonstrate that 42% of affected individuals are from exposure-prone professions.[19] These activities alter ecological balances, facilitating the spread of zoonotic agents and exacerbating public health risks. Furthermore, the industrialization of farming practices has intensified worker exposure to antibiotic-resistant bacteria, contributing to complex health challenges that remain underexplored.

The global response to zoonotic risks reflects a disparity between industrialized and developing nations. While industrialized countries invest in vaccination, health education, and food safety measures, resource constraints in developing nations hinder the integration of surveillance systems bridging veterinary and human medicine. This gap complicates the identification and management of zoonotic outbreaks, particularly in high-risk occupational settings, where poor hygiene, inadequate ventilation, and insufficient access to personal protective equipment exacerbate vulnerabilities.[20]

Addressing the gaps in knowledge and prevention of occupational zoonoses requires a coordinated, multidisciplinary approach rooted in the One Health framework. Enhanced surveillance, education, and access to preventive resources are essential to protect workers and reduce the broader impacts of zoonotic diseases. By prioritizing research into the health effects of occupational exposures and strengthening public health systems, it is possible to mitigate zoonoses' dual economic and health burdens, fostering resilience in vulnerable populations and industries alike.

## 3. Neurological risks of occupational zoonoses and the relevance of workplace exposure for public health

Neurological diseases arising from occupational zoonoses are a critical yet underexplored dimension of workplace health, reflecting the intricate interplay between exposure to zoonotic pathogens and neurological outcomes in high-risk occupational environments. These conditions frequently emerge in settings where individuals are in direct or indirect contact with infectious agents, posing multifaceted challenges due to their diverse etiologies and the potential for severe, long-term neurological sequelae. Understanding this intersection is imperative for advancing public health and occupational safety, emphasizing the urgency of interdisciplinary approaches that integrate neurology, infectious disease, and occupational health frameworks. The main zoonotic pathogens associated with neurological manifestations are listed in Table 1.

The hepatitis E virus (HEV), a single-stranded RNA virus, is primarily associated with hepatitis E, an acute liver disease. Approximately 12.47% of the global population has been exposed to HEV, equating to about 939 million individuals. Recent or ongoing infections are estimated in 15–110 million people worldwide.[21] While some genotypes exclusively infect humans, others, such as genotypes 3 and 4, have been identified in both animals and humans. Pigs, in particular, act as a significant reservoir, raising concerns about occupational exposure among people who work with pigs. HEV is primarily transmitted via the fecal-oral route, often through the consumption of contaminated water or undercooked pork products. Occupational exposure occurs through direct contact with infected swine or their excretions, particularly in farm and slaughterhouse environments, where workers handle raw meat and animal waste. This zoonotic potential of HEV extends its impact beyond hepatic involvement, as recent evidence suggests that it can also cause neurological complications, which are still poorly recognized in clinical practice.[22]

HEV has been linked to a series of neurological disorders, including Guillain-Barré syndrome, neuromuscular junction disease, encephalitis, myelitis and other neuropathies. Guillain-Barré syndrome, an autoimmune disease characterized by progressive weakness of the limbs and, in severe cases, respiratory failure, has been observed with a disproportionately higher frequency in subjects with recent HEV infections.[23] Neuralgic amyotro-

**Table 1**
Summary of current evidence on occupational zoonotic diseases presenting neurological manifestations.

| Pathogen | Epidemiological data | Transmission | Occupational context | Neurological manifestations | Preventive measures |
|---|---|---|---|---|---|
| Hepatitis E virus | Global prevalence: 939 million<br>15–110 million active infections | Fecal-oral<br>Direct contact pigs<br>Undercooked pork | Swine farming<br>Slaughterhouses | Guillain-Barré syndrome<br>Polyradiculitis<br>Encephalitis | Enhanced protective measures<br>Improved diagnostics |
| West Nile virus | 51,607 cases in the US 1999–2019<br>25,161 neuroinvasive cases | Mosquito bites<br>Direct contact infected animals | Veterinary<br>Laboratory<br>Agriculture | Meningoencephalitis<br>Cognitive deficits | Stringent safety protocols<br>Occupational surveillance |
| Nipah virus | Fatality rate: 40%–75% | Direct contact infected animals<br>Bat-contaminated food<br>Human-to-human | Abattoir workers | Encephalitis | Protective measures in abattoirs |
| Rabies | Annual global deaths: 59,000 | Animal bites, scratches<br>Exposure to saliva, neural tissue | Veterinarians<br>Animal handlers<br>laboratory workers | Encephalitis<br>paralysis | Public health strategies |
| *Streptococcus suis* | Global cases: Over 1,600 documented | Direct contact infected pigs<br>Contaminated pork products | Swine farming<br>Slaughterhouses | Meningitis<br>Hearing loss<br>Paralysis | Early and accurate diagnostics<br>Safety training |
| *Campylobacter jejuni* | Annual incidence:<br>96 million symptomatic infections | Fecal-oral | Poultry farming | Guillain-Barré syndrome<br>Paralysis | Improved hygiene measures<br>Occupational surveillance |
| Group C *Streptococcus* | n.a. | Direct contact infected horses<br>Unpasteurized dairy products | Equine care<br>Dairy farming | Meningitis<br>Cognitive deficits | Awareness campaigns |
| *Baylisascaris procyonis* | Seroprevalence wildlife rehabilitators: ≈ 7% | Ingestion of eggs (fecal-oral)<br>Direct contact raccoons | Wildlife rehabilitation | Neuroinvasion<br>Encephalitis | Protective equipment<br>Stricter biosecurity practices |

phy, which manifests itself with sudden and severe pain followed by weakness and muscular atrophy, has been associated in particular with HEV genotype 3, with cases often being bilateral, an unusual characteristic for this disorder. Cases of encephalitis and myelitis have been reported in immunocompromised individuals, with HEV RNA detected in the cerebrospinal fluid, suggesting the possibility of a direct viral invasion of the central nervous system. Other neurological manifestations include multiple mononeuritis, myositis, vestibular neuritis and Bell's palsy, further reinforcing the need to consider HEV in patients presenting with unexplained neurological symptoms.[24]

The precise mechanisms underlying the neurological damage associated with HEV remain incomplete, although several plausible explanations have emerged. An immune-mediated response, where HEV-induced antibodies cross-react with gangliosides, has been implicated in cases such as Guillain-Barré syndrome, leading to demyelination and nerve dysfunction.[25] Additionally, evidence of HEV RNA in cerebrospinal fluid suggests direct neurotropism, with potential for encephalitis and myelitis, particularly in immunocompromised individuals.[26] Viral quasi-species evolution and systemic inflammation may further exacerbate neural damage by promoting localized replication, cytokine dysregulation, and blood-brain barrier disruption, facilitating viral entry into the central nervous system.[27]

The occupational risk associated with HEV exposure is particularly concerning for individuals employed in the swine industry. Research indicates that workers in this sector exhibit a significantly higher prevalence of HEV infection compared to the general population, with meta-analysis findings revealing that 32.85% of swine industry workers tested positive for HEV IgG antibodies, in contrast to 21.70% in the general population. This disparity suggests that individuals in these occupations are 52% more likely to contract the virus.[28] The increased risk is particularly pronounced in Asia and Europe, where HEV is highly endemic and infection rates among swine industry workers are especially elevated. Notably, no significant difference in prevalence has been observed between industrialized and resource-limited countries within these regions, underscoring that occupational exposure remains a primary determinant of infection risk, irrespective of broader socioeconomic conditions.[28]

The strong evidence for zoonotic transmission of HEV underscores the necessity of enhanced protective measures in high-risk occupational settings. Workers in the pork industry, including butchers, veterinarians, and meat processors, should adopt stringent hygiene protocols to reduce infection risk. Although an HEV vaccine has been available in China since 2012, its use remains limited, and the WHO has not recommended routine vaccination. Targeted immunization programs for high-risk workers in endemic regions could be beneficial.[29] A comprehensive public health strategy is essential to address HEV's dual hepatic and neurological impact and its occupational implications.

Similarly, West Nile virus (WNV) has emerged as a significant cause of neurological disease, particularly since its introduction to North America in 1999. While most infections are asymptomatic or present as a mild febrile illness known as West Nile fever, a subset of cases progress to neuroinvasive disease, manifesting as meningitis, encephalitis, or poliomyelitis-like paralysis.[30] Between 1999 and October 2019, a total of 51,607 cases were documented in the United States, with nearly half (25,161) involving central nervous system (CNS) manifestations.[31] More recently, research has underscored the occupational risks associated with WNV exposure, particularly in veterinary, laboratory, and agricultural settings, highlighting the need for targeted preventive measures.[32]

Among the neuroinvasive forms of WNV, meningitis presents classical symptoms of viral meningitis, including fever, headache, and neck stiffness. At the same time, cerebrospinal fluid analysis typically reveals pleocytosis with early neutrophil predominance. Though the majority of cases resolve with supportive care, many patients report lingering symptoms such as fatigue and persistent headaches. More severe is West Nile encephalitis, characterized by altered mental status, confusion, and movement disorders, including tremor, myoclonus, and parkinsonian features. In some cases, cerebellar ataxia and seizures occur, while neuroimaging frequently reveals lesions in the basal ganglia and thalami.[33] An equally debilitating manifestation is West Nile poliomyelitis, a syndrome resulting from viral damage to the anterior horn cells of the spinal cord, leading to acute flaccid paralysis. This condition, which can progress rapidly to quadriplegia, also affects respiratory muscles in severe cases, necessitating mechanical ventilation.[34]

Neuropathological studies indicate that WNV preferentially targets neurons in the brainstem, basal ganglia, and spinal cord, with histological findings including microglial nodules, neuronophagia, and perivascular inflammation. Persistent viral presence in the central nervous system further contributes to ongoing neuronal damage, with a substantial proportion of survivors reporting lasting cognitive impairment, including memory deficits and executive dysfunction. Mood disturbances are also common, with depression affecting between 21% and 56% of WNV survivors, suggesting a potential link between neuroinflammation and neuropsychiatric outcomes.[35]

Beyond its impact on public health, WNV also poses a notable occupational risk, particularly for individuals with direct or indirect exposure to infected vectors, animals, or biological materials. Military personnel, veterinarians, agricultural workers, and laboratory researchers are among the highest-risk groups. Those engaged in outdoor work, particularly in endemic areas, face heightened exposure to mosquito bites, while laboratory and veterinary professionals are at risk through direct contact with infected animal tissues, fluids, or aerosols. Seroprevalence studies indicate that occupationally exposed individuals have higher rates of WNV antibodies compared to the general population, reinforcing the need for stringent protective measures.[32] Although no human vaccine is currently available, workplace safety strategies—including enhanced biosafety protocols, personal protective equipment (PPE), and mosquito control initiatives—are critical in mitigating infection risk in high-exposure settings.[32]

Nipah virus (NV) exemplifies the occupational hazards associated with zoonotic diseases and their neurological impact. Transmission primarily occurs through direct contact with infected animals, particularly pigs, or exposure to their bodily fluids.[36] Additionally, consumption of contaminated food, such as raw date palm sap exposed to bat secretions, has been implicated in outbreaks.[37] While human-to-human transmission was not observed in the 1999 outbreak, subsequent outbreaks in South and Southeast Asia have demonstrated person-to-person spread, highlighting the need for stringent infection control measures. NV enters the body via the respiratory tract, disseminating through the bloodstream and targeting brain endothelial cells via Ephrin-B2 and Ephrin-B3 receptors. Once in the CNS, the virus induces severe inflammation, increasing blood-brain barrier permeability and promoting neuronal infection. The resultant inflammatory response, driven by cytokines and chemokines, exacerbates neural damage, contributing to vasculitis, thrombosis, and encephalitis.[38] Neurological manifestations include seizures, cognitive impairment, gaze palsy, and coma, with case fatality rates ranging from 40% to 75%. Survivors frequently experience long-term neurological sequelae, such as motor dysfunction and cognitive decline.[36] These findings underscore the necessity of enhanced protective measures in high-risk occupational settings, particularly abattoirs, given the continued risk of zoonotic spillover.[39]

Rabies, a fatal viral disease endemic in many regions, is primarily transmitted through dog bites. Once neurological symptoms emerge, the infection is almost invariably lethal. It is estimated that rabies causes 59,000 human deaths per year globally.[40] Despite its severity, rabies induces minimal inflammation and only mild structural damage to the CNS. Pathological changes include ganglion cell degeneration, neuronophagia, and perivascular infiltration of mononuclear cells, predominantly affecting peripheral nerves, the spinal cord, and the brain. Inflammation is most pronounced in the midbrain and medulla, while vascular damage, including thrombosis and hemorrhages, is observed in the brainstem, hypothalamus, and limbic system.[41]

A defining pathological feature is the presence of Negri bodies in infected neurons, which facilitate viral evasion of apoptosis. As the disease progresses, neuronal damage manifests as axonal swelling, mitochondrial abnormalities, and vacuolation in cortical neurons. Rabies impairs neurotransmission by disrupting ion channels, reducing neuronal excitability. Early in the infection, the virus inhibits apoptosis, allowing undetected CNS spread, but apoptosis is induced in the later, invariably fatal stages. Routine histopathology often fails to detect significant structural damage, complicating diagnosis and delaying treatment.[42]

Occupational exposure remains a critical concern, particularly for veterinarians, wildlife researchers, and healthcare workers treating rabies patients. These individuals are at risk through bites, scratches, or contact with infectious bodily fluids. Pre-exposure prophylaxis is advised for those frequently exposed to potentially infected animals, reducing the need for rabies immunoglobulin and ensuring a rapid immune response. Post-exposure prophylaxis (PEP) remains essential following any suspected exposure.[41] While human-to-human transmission has never been conclusively documented, theoretical risks exist, particularly in healthcare settings. A hospital study identified 222 healthcare workers with potential exposure, with 4% requiring PEP due to high-risk interactions. Despite established guidelines, challenges persist, including limited awareness, delays in seeking PEP, and inadequate access to rabies biologics. Adherence to preventive measures, including vaccination and rigorous wound care, is essential for mitigating occupational risks.[43]

*Streptococcus suis* infections further reveal the devastating consequences of occupational exposures in agriculture and animal processing industries. Over 1,600 human cases of *S. suis* infection have been documented worldwide. However, this number is likely an underestimation due to underdiagnosis and underreporting, especially in regions with limited healthcare resources.[44] Transmission occurs primarily through direct contact with infected pigs or contaminated pork products. The bacterium can enter the body via skin wounds, mucosal surfaces, or inhalation of contaminated aerosols, posing a particular risk to pig farmers, slaughterhouse workers, and butchers. Pig farmers and slaughterhouse workers are especially vulnerable to *S. suis* meningitis, which is associated with neurological sequelae such as hearing loss and paralysis.[45] The infection triggers severe neuroinflammation, leading to neuronal apoptosis and microglial activation, particularly in the hippocampus and neocortex. Despite immune system activation, neural regeneration remains inadequate, resulting in irreversible neuronal damage. Experimental models reveal a significant increase in apoptotic neurons and activated microglia, yet without substantial microglial proliferation, suggesting a dysregulated immune response. Neural progenitor cell proliferation and young neuron formation show slight increases but are insufficient to counteract neuronal loss. The infection progresses rapidly, leaving minimal time for neural repair.[46]

Clinical cases highlight high rates of neurological sequelae, with up to 85% of survivors experiencing hearing loss, paralysis, and visual impairment. Hearing loss is the most common outcome, likely due to auditory nerve damage. Motor deficits and paralysis result from widespread neuroinflammatory damage.[46]

The aggressive progression of *S. suis* infections underscores the need for early diagnosis and intervention. Current diagnostic methods relying on bacterial culture may underestimate the true disease burden, necessitating improved PCR-based detection. Enhanced surveillance in pig-farming regions is crucial for early case identification. Although diagnostic advances like metagenomic next-generation sequencing have proven effective in identifying pathogens in complex cases, their accessibility remains limited. For instance, metagenomic next-generation sequencing identified *S. suis* in a slaughterhouse worker presenting with fever and progressive neurological symptoms after conventional diagnostic methods failed. This case exemplifies the need for early, accurate diagnostics to mitigate long-term sequelae.[47]

*Campylobacter jejuni* is considered to be one of the 4 key global causes of diarrheal disease worldwide and it is estimated it caused ≈ 96 million symptomatic cases in 2010.[48,49] Occupational exposure to *C. jejuni* presents a significant health risk to poultry workers and animal farmers due to repeated contact with contaminated carcasses, feces, and processing environments. The primary mode of transmission for *C. jejuni* is fecal-oral, often through the consumption of undercooked poultry or unpasteurized milk. Occupational exposure occurs in poultry processing plants and farms, where workers come into direct contact with contaminated carcasses, feces, and processing environments. Cross-contamination of food and inadequate hand hygiene further contribute to transmission. Research indicates that workers in these industries develop heightened immune responses, reflected in elevated IgG and IgA antibody levels, yet they rarely experience symptomatic infection.[50] This suggests that sustained low-level exposure may induce a degree of immune tolerance. However, the long-term consequences of chronic exposure remain uncertain, particularly concerning the risk of autoimmune disorders.[50]

Beyond its role in gastroenteritis, *C. jejuni* is a major trigger of Guillain-Barré syndrome, a severe autoimmune condition that leads to neuromuscular paralysis. The bacterium's ability to mimic human nerve gangliosides can provoke an immune response that mistakenly attacks the peripheral nervous system, particularly in genetically susceptible individuals. Evidence from outbreaks, including the 2019 epidemic in Peru, demonstrates how rapidly neurological symptoms can emerge, often progressing to widespread paralysis within days. The axonal variant of GBS known as acute motor axonal neuropathy (AMAN) is more frequently associated with *C. jejuni* infection and tends to result in more severe and lasting nerve damage.[51]

For workers in high-exposure settings, the combination of frequent bacterial contact and immune activation raises concerns about the potential for occupationally induced-autoimmune complications. While no direct link has been established between occupational exposure and an increased risk of GBS, the known mechanisms of *C. jejuni*-induced neuropathy suggest a need for further investigation. Given the severity of GBS and its potentially fatal outcomes, implementing protective measures in processing plants and farming environments is essential. Strengthening hygiene protocols and monitoring immune responses in high-risk workers may help mitigate long-term neurological risks associated with *C. jejuni* exposure.[52]

Group C *Streptococcus* (GCS), particularly *Streptococcus equi*, adds to the spectrum of occupational zoonoses with neurological outcomes. Transmission occurs through direct contact with infected horses or exposure to contaminated materials such as horse saliva, nasal secretions, or unpasteurized dairy products. Workers in equestrian environments, veterinarians, and dairy farmers face increased occupational risks due to frequent animal interactions and exposure to infected biological fluids. Cases of GCS meningitis have been linked to horse trainers, equestrians, and those consuming unpasteurized dairy products.[53] The pathogen's neuroinvasive potential and association with high mortality and residual deficits, such as hearing loss and cognitive impairments, highlight the occupational risks in equine and dairy farming contexts. Despite effective treatments with beta-lactam antibiotics, diagnostic delays often compromise outcomes. This underscores the need for greater awareness among healthcare providers and the implementation of stringent infection control measures.[54]

*Baylisascaris procyonis*, the raccoon roundworm, represents a significant occupational hazard for wildlife rehabilitators due to their frequent contact with raccoons and their feces. Although human infections are considered rare, recent serological studies in the United States and Canada indicate that approximately 7% of rehabilitators have antibodies to *B. procyonis*, suggesting prior exposure without overt clinical manifestations. Occupational risk factors include inadequate hygiene practices, inconsistent use of PPE, and environmental contamination within rehabilitation facilities. The parasite's eggs are highly resilient, persisting in the environment for years and remaining a continuous source of potential infection. Once ingested, *B. procyonis* larvae penetrate the intestinal wall, enter the bloodstream, and exhibit a strong predilection for neural tissue, resulting in neural larva migrans (NLM).[55] This condition leads to eosinophilic meningoencephalitis, characterized by severe neuroinflammation and often irreversible neurological damage. Clinical sequelae include seizures, paralysis, cognitive decline, and, in severe cases, coma. Survivors frequently experience long-term neurological deficits, including developmental disabilities, visual impairment, and motor dysfunction. Unlike other nematodes, *B. procyonis* larvae continue to grow during migration, exacerbating tissue damage and inflammatory pathology. Given these risks, improved biosecurity measures, including enhanced PPE adherence, rigorous decontamination protocols, and targeted educational initiatives, are essential to mitigate occupational exposure and protect the health of wildlife rehabilitators.[56]

The here reviewed zoonotic pathogens, the animals most commonly associated with each zoonotic pathogen, the most common transmission routes, the most affected worker populations and the most common neurologic symptoms are illustrated in Table 2 and Fig. 1. These studies collectively illuminate the profound burden of neurological diseases stemming from occupational zoonoses, with significant implications for workers' productivity and well-being. The occupational contexts of these exposures, whether in swine farming, poultry processing, wildlife rehabilitation, or equine care, highlight the necessity of tailored protective measures and enhanced diagnostic capabilities. Persistent knowledge gaps, particularly regarding pathogen transmission, immune evasion mechanisms, and effective interventions, call for sustained interdisciplinary research. An integrated One Health approach, encompassing occupational safety, infectious disease management, and neurology is essential to mitigate the long-term impacts of these diseases on public health.

## 4. Ecohealth: A proactive paradigm for sustainable zoonotic disease prevention

The Ecohealth approach, complementing the One Health framework, provides a critical perspective on zoonotic disease prevention by addressing the complex interplay between human, animal, and environmental health. Recognizing the interconnections among ecological, biological, social, and economic factors, Ecohealth prioritizes upstream interventions to mitigate zoonotic spillovers. Environmental degradation, habitat encroachment, and high-risk human-animal interactions are key drivers of zoonotic disease emergence. By targeting these factors, Ecohealth fosters proactive strategies that emphasize prevention rather than reactive containment measures.[57]

This preventive paradigm necessitates integrating environmental stewardship with public and animal health systems. For instance, reducing deforestation and preserving biodiversity help maintain ecological barriers that limit pathogen spillovers. Simultaneously, addressing socio-economic disparities that drive high-risk behaviors—such as bushmeat hunting and unregulated livestock farming—can reduce zoonotic risks. These measures highlight the importance of holistic interventions that balance environmental conservation with socio-economic

**Table 2**
Comparison table of occupational zoonotic diseases presenting neurological manifestations.

| Pathogen | Transmission | | | | | Workers at risk | | | | Neurological involvement | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | Fecal-oral | Direct contact | Animal bite | Contaminated food | Human-to-human | Meat industry | Veterinary medicine and Wildlife care | Laboratory workers | Agriculture workers | CNS | PNS |
| Hepatitis E Virus | √ | √ | | √ | | √ | | | | √ | √ |
| West Nile Virus | | √ | √ | | | | √ | √ | √ | √ | |
| Nipah Virus | | √ | | √ | √ | √ | | | | √ | |
| Rabies | | | √ | | | | √ | √ | | √ | √ |
| *Streptococcus suis* | | √ | | √ | | √ | | | | √ | |
| *Campylobacter jejuni* | √ | | | | | √ | | | | | √ |
| Group C *Streptococcus* | | √ | | √ | | | √ | | | √ | |
| *Baylisascaris procyonis* | √ | √ | | | | | √ | | | √ | |

*Abbreviations*: CNS, central nervous system; PNS, peripheral nervous system.

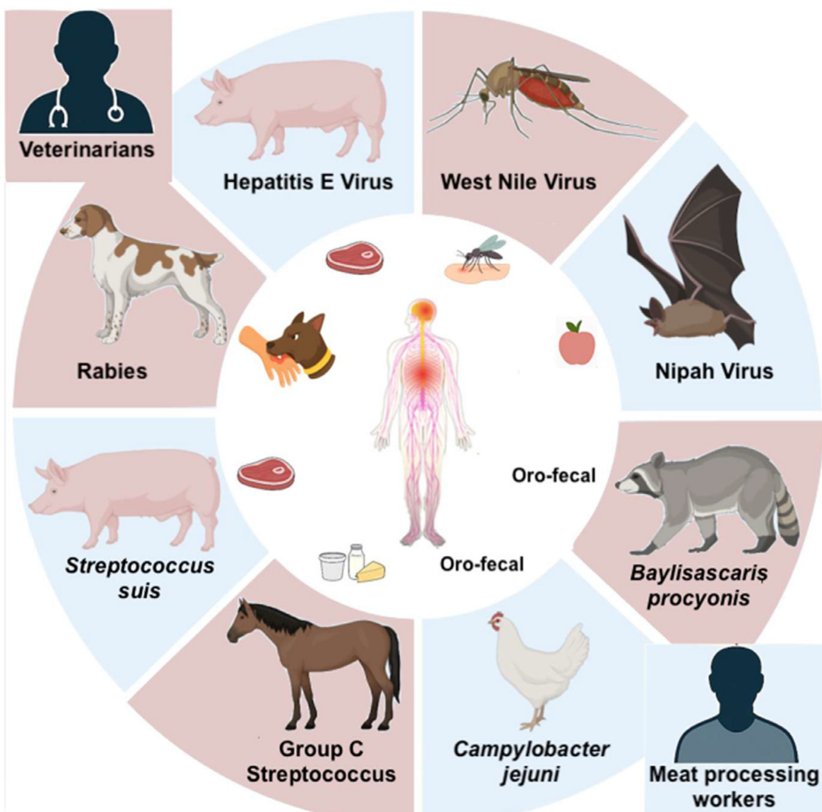


**Fig. 1.** The illustration summarizes key zoonotic pathogens, the animals most commonly associated with each zoonotic pathogen, the most common transmission routes, the most affected worker populations and the most common neurologic symptoms in zoonoses. These zoonotic pathogens pose serious occupational health risks, particularly in agriculture, veterinary medicine, and food processing industries. Preventive measures, improved hygiene, and targeted vaccination programs are essential to mitigate risks. (Created with Preview, Inkscape, and ChatGPT 4.0).

development, ensuring both health and livelihood sustainability.[58]

A key strength of Ecohealth lies in its capacity to incorporate uncertainty into decision-making processes. While the precise timing and location of zoonotic spillovers remain unpredictable, the approach advocates for investments in structures and practices aimed at outbreak prevention. Strengthening ecosystem resilience and promoting sustainable agricultural practices not only mitigate zoonotic risks but also yield additional benefits, including enhanced food security, climate adaptation, and biodiversity conservation. These multidimensional advantages enhance the cost-effectiveness of Ecohealth interventions, particularly in resource-limited settings.[59]

Occupational medicine plays a pivotal role within this integrated framework, serving as the critical link between workplace safety and public health in zoonotic disease management. Many occupations place individuals at the interface of human, animal, and environmental interactions, making occupational medicine uniquely positioned to address risks associated with zoonotic pathogens. By focusing on the health and safety of workers in high-risk environments, this field ensures that preventive measures are tailored to specific occupational exposures in agriculture, wildlife management, laboratory research, and other critical sectors.[60]

The integration of Ecohealth principles within the broader One Health framework underscores the significance of occupational medicine as a core component of sustainable health strategies. This synergy facilitates interventions that not only protect workers from immediate hazards but also address the ecological and socio-economic drivers of zoonotic disease emergence. Surveillance, risk assessment, and education initiatives within occupational medicine serve as essential mechanisms for bridging environmental stewardship with health security, fostering resilience against current and future zoonotic threats.[61]

Both the Ecohealth and One Health frameworks provide comprehensive, evidence-based strategies for mitigating occupational zoonotic risks by addressing the intersecting environmental, biological, and socio-economic determinants of disease transmission. Preserving ecological buffers, such as intact forests and wetland ecosystems, is a fundamental component of these strategies, as these natural barriers limit pathogen spillover at the human-animal-environment interface. Such measures are particularly crucial for agricultural and wildlife-sector workers, who face disproportionate exposure to zoonotic pathogens due to habitat encroachment and intensified human-animal interactions.[62] Additionally, strengthening biosafety protocols in research laboratories handling neurotropic zoonoses—such as rabies, NV, and arboviral encephalitides—ensures rigorous containment and minimizes occupational exposure to high-risk pathogens. Real-time epidemiological surveillance in high-risk occupational settings, including continuous exposure monitoring for farmworkers and routine serological screening for veterinarians and animal handlers, further enables the early detection of zoonotic infections. This facilitates timely interventions, preventing neuroinvasive disease progression and reducing long-term neurological sequelae.

The integration of occupational medicine into Ecohealth-driven interventions extends beyond individual worker protection, functioning as a sentinel system for identifying and mitigating emerging zoonotic threats at the population level. By fostering collaboration among public health authorities, environmental scientists, and occupational health specialists, these frameworks enable targeted preventive measures, such as pre-exposure vaccination programs (e.g., rabies immunization for high-risk professionals) and enhanced psychosocial support services for individuals experiencing neurological or cognitive impairments following zoonotic infections. These interventions exemplify a proactive, systems-based approach that safeguards human health while reinforcing ecological resilience and public health preparedness. Ultimately, this integrated strategy contributes to a more sustainable and adaptive response to the complex challenges posed by zoonotic diseases.[63]

Occupational medicine further strengthens public health infrastructure by functioning as an early warning system for zoonotic spillover. This proactive approach aligns with the Ecohealth and One Health vision by prioritizing upstream solutions that prevent outbreaks, enhance health equity, and promote ecological balance. The inclusion of occupational medicine within these frameworks is thus essential for achieving the integrated and sustainable health outcomes envisioned by global health initiatives.

## 5. Conclusions

Addressing gaps in occupational zoonotic disease surveillance requires a holistic approach. Implementing real-time digital reporting platforms, expanding sentinel surveillance in high-risk occupational settings, and incentivizing reporting through regulatory frameworks can significantly enhance data collection. Additionally, fostering cross-sectoral collaboration—such as partnerships among occupational health agencies, veterinary services, and public health institutions—can facilitate integrated data-sharing mechanisms. Investing in these solutions not only mitigates underreporting but also strengthens early warning systems, enabling proactive responses to emerging zoonotic threats.

## CRediT authorship contribution statement

**Angela Stufano:** Writing – review & editing, Writing – original draft, Conceptualization. **Valentina Schino:** Writing – review & editing, Conceptualization. **Domenico Plantone:** Writing – review & editing, Conceptualization. **Guglielmo Lucchese:** Writing – review & editing, Writing – original draft, Conceptualization.

## Informed consent

Not applicable.

## Organ donation

Not applicable.

## Ethics statement

Not applicable.

## Data availability statement

Not applicable.

## Animal treatment

Not applicable.

## Generative AI

ChatGPT 4.0 was used for optimizing graphical aspects of Fig. 1.

## Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

## Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

## Acknowledgments

Not applicable.

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