Review

Comprehensive analysis of the global impact and distribution of tick paralysis

Yuan-Ping Deng[1†], Yi-Tian Fu[1,2†], Hany M. Elsheikha[3*], Mei-Ling Cao[1], Xing-Quan Zhu[4], Jin-Lei Wang[5], Xue-Ling Zhang[1], Shi-Chen Xie[1], Chaoqun Yao[6*], Guo-Hua Liu[1*]

[1]Research Center for Parasites & Vectors, College of Veterinary Medicine, Hunan Agricultural University, Changsha, Hunan Province 410128, China

[2]Department of Parasitology, Xiangya School of Basic Medicine, Central South University, Changsha, Hunan, China.

[3]Faculty of Medicine and Health Sciences, School of Veterinary Medicine and Science, University of Nottingham, Sutton Bonington Campus, Loughborough LE12 5RD, UK

[4]College of Veterinary Medicine, Shanxi Agricultural University, Taigu 030801, Shanxi, China

[5]State Key Laboratory of Veterinary Etiological Biology, Key Laboratory of Veterinary Parasitology of Gansu Province, Lanzhou Veterinary

Research Institute, Chinese Academy of Agricultural Sciences, Lanzhou, Gansu, China.

[6]Ross University School of Veterinary Medicine and One Health Center for Zoonoses and Tropical Veterinary Medicine, Ross University School of Veterinary Medicine, P.O. Box 334, Basseterre, St. Kitts& Nevis

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[†] These authors contributed equally

* E-mail: liuguohua5202008@163.com; chyao@rossvet.edu.kn; Hany.Elsheikha@nottingham.ac.uk

CONTENT

SUMMARY Tick paralysis is a potentially fatal condition caused by neurotoxins secreted by the salivary glands of certain ticks. Documented cases have been reported worldwide, predominantly in the United States, Canada, and Australia, with additional reports from Europe and Africa. This condition also affects animals, leading to significant economic losses and adverse impacts on animal health and welfare. To date, 75 tick species, mostly hard ticks, have been identified as capable of causing this life-threatening condition. Due to symptom overlap with other conditions, accurate diagnosis of tick paralysis is crucial to avoid misdiagnosis, which could result in adverse patient outcomes. This review provides a comprehensive analysis of the current literature on tick paralysis, including implicated tick species, global distribution, tick toxins, molecular pathogenesis, clinical manifestations, diagnosis, treatment, control, and prevention. Enhancing awareness among medical and veterinary professionals is critical for improving the management of this condition's health impacts on both humans and animals.

KEYWORDS Tick paralysis, ticks, human cases, toxin, neurotoxin, clinical manifestations

INTRODUCTION

Tick paralysis is a condition caused by neurotoxins secreted by the salivary glands of certain female or male ticks. Of the 900 known tick species, 61 hard ticks and 14 soft ticks are implicated in tick paralysis (see Table 1) (1). The condition affects both humans and animals, typically presenting with initial gait instability (2), followed by flaccid ascending motor weakness and paralysis (3). The term “tick paralysis” was first introduced in 1824 during the expedition of Hume and Hovell from Sydney to Port Phillip Bay (Melbourne) (4). Whether the condition identified by these explorers was indeed tick paralysis remains debatable.

The first documented human cases of tick paralysis appeared in the scientific literature from Canada in 1912, reported by parasitologist John Todd. He documented 14 cases observed by nine medical doctors in British Columbia (BC), all in children, with five fatalities (5). Hadwen meticulously investigated tick paralysis in both humans and animals, proposing three key findings: first, paralysis occurred only in animals on which female ticks had been feeding for approximately a week; second, the paralysis symptoms were likely caused by a toxin; and third, without tick removal, the progression from general weakness to death from respiratory paralysis was rapid (6). Subsequent studies corroborated Hadwen's observations. Rose (7), Mail and Gregson (8), and Schmitt et al. (9) reviewed prior cases of tick paralysis in humans, noting a fatality rate of over 10%.

In this study, we reviewed a substantial number of human cases documented worldwide, with a focus on those primarily reported in

the United States, Canada, and Australia (Fig. 1 and Table S1), along with sporadic cases from Africa, Asia, and Europe (10). The disease typically occurs in spring and summer but can be observed year-round (11, 12). It most commonly affects children younger than eight years, though it has been diagnosed in individuals of all ages (10). Tick paralysis also induces general weakness or neurological symptoms in birds, mammals, reptiles, and rodents (Table 1), with significant economic implications for the livestock industry (13).

Given the substantial impact of tick paralysis on both human and animal health, analyzing current literature is crucial for understanding the condition's magnitude. Building on the insights gained from historical cases, global distribution, and the impact on animals, this review synthesizes the latest research on tick species, toxins, and strategies for prevention and control. We hope this review will enhance the knowledge of medical and veterinary professionals, as well as the public, regarding tick paralysis.

LITERATURE SEARCH METHODOLOGY AND DATA EXTRACTION

A comprehensive literature search was conducted on tick paralysis cases published up to December 31, 2023, across eight electronic bibliographic databases: PubMed, Google Scholar, ScienceDirect, Wiley, CABI (https://www.cabi.org/), CNKI (https://www.cnki.net/), VIP Chinese Journal Database (http://qikan.cqvip.com/), and Wanfang Data (https://g.wanfangdata.com.cn/index.html). For the first five databases (PubMed, Google Scholar, ScienceDirect, Wiley, and CABI), the search utilized keywords such as "tick paralysis," "paralysis case,"

"tick bite," and "tick infection". In the three Chinese databases (CNKI, VIP, and Wnfang), the search was conducted using the Chinese translations of these keywords. There were no restrictions on the publication year. Duplicate cases and publications with inaccessible full texts were excluded after a thorough review. Information was extracted regarding the first author, year and region of the report, patient age, clinical manifestations, tick attachment site, prognosis, and the likely source of tick infestation, as detailed in Table S1.

TICK PARALYSIS AND ASSOCIATED TICK SPECIES

Although tick bites are common, tick paralysis is extraordinarily rare, with most tick bites not leading to this condition. Since the first case of tick paralysis was described in 1824, 75 out of over 900 known tick species have been implicated in causing tick paralysis (2). These include 61 hard-tick species (Family: Ixodidae) and 14 soft-tick species (Family: Argasidae). Among the hard ticks, the implicated species include 19 *Ixodes* spp., 10 *Dermacentor* spp., 10 *Rhipicephalus* spp., 8 *Amblyomma* spp., 7 *Haemaphysalis* spp., 6 *Hyalomma* spp., and 1 *Rhipicentor* sp. have been identified. The soft ticks implicated are 10 *Argas* spp., 3 *Ornithodoros* spp., and 1 *Otobius* sp. (Table 1). Hard ticks are primarily responsible for causing paralysis in mammals, including humans, cats, cattle, coyotes, deer, dogs, goats, porcupines, sheep, and skunks. They are also occasionally involved in tick paralysis cases in reptiles and birds (Table 1).

Of the species causing tick paralysis, at least 26 are associated with human cases (Table S2). Most of these are ixodid ticks, with their

location, detection year, and related clinic manifestations detailed in Table S1. Most reported cases (212 of 288) occurred in North America (*n* = 177) and Australia (*n* = 35). Most patients are affected by a single tick species, with the most common being *Dermacentor andersoni*, *De. variabilis*, and *Ixodes holocyclus* (Table S1-S2, Fig. 2). In North America, *De. andersoni* (primarily found in the Rocky Mountain states and Pacific Northwest of the USA) and *De. variabilis* (found in the eastern half of the USA and along the West Coast) are the most common ticks causing human paralysis (13). Cases involving *De. andersoni* and *De. variabilis* typically present with ascending flaccid paralysis, diminished reflexes, and ataxic symptoms, although rare symptoms like typhus-like rash and dilated pupils have also been recorded (14, 15). Paralytic symptoms usually improve following tick removal. Additionally, *Amblyomma americanum*, *Am. maculatum*, and *Ixodes scapularis* are prevalent in the southeastern United States and are associated with human cases (16, 17).

In Australia, *Ix. holocyclus*, which has a wide host range (> 30 species) (18), is the most common tick causing the death of thousands of domestic and wild animals annually, including dogs and cats, with up to 10% mortality (19). It is also a significant cause of tick paralysis in both animals and humans (Table 1). Most cases of paralysis due to this tick progress for at least 24 hours and require a long recovery period after tick removal, sometimes necessitating supportive care such as mechanical ventilation. Although *Ix. holocyclus* is widely distributed along the eastern coasts of Australia, *Ixodes cornuatus* poses a significant risk of tick paralysis in Tasmania and Victoria (18). Recently, this species was found on birds for the first time, causing their death and expanding its host ranges from mammals to birds (20).

Other hard tick species cause sporadic cases in different regions or countries (Table S2).

There have also been reports of human patients infested with more than two tick species simultaneously (Table S1), though single-species infestations are more common. In Sri Lanka, *Hyalomma marginatum isaaci* and *Dermacentor auratus* are prevalent among animal populations, but they were also detected in the ear canals of 29 patients simultaneously (21). Co-infestations of *Rhipicephalus sanguineus*, *Hyalomma dromedarii*, *Hyalomma anatolicum excavatum*, and *Haemaphysalis* sp. were recorded in two children in Egypt (22). These co-infestations are more frequent in families who farm livestock or live near tick-endemic regions.

Tick paralysis is caused by neurotoxins secreted by adult female ticks or immature stages during blood feeding (2). Generally, adult male ticks (hard ticks) are not considered significant contributors to paralysis due to their relatively small toxin output. During the same feeding period, females secrete toxic contents in quantities several times higher than males (23). However, some clinical cases suggest that adult male ticks might play a role in tick paralysis. As early as 1914, Todd (24) documented a 2-year-old girl who was unable to walk and lacked deep tendon reflexes, with an adult female tick and an adult male tick found on her neck. The Centers for Disease Control (CDC) also recorded a case involving a little girl who presented with unsteady gait, inability to walk, generalized paralysis, and weak grip; a male and a female *De. variabilis* were detected on her scalp (25). Hadwen proposed that only animals fed by female ticks for around a week experienced paralysis (6), leading to most research focusing on females while the role of males was neglected.

However, isolated reports have linked male ticks to paralysis. In the literature by Brown (26), a male *De. andersoni* was collected from the left axilla of a young man exhibiting tick paralysis signs, including spotted fever, difficulty lifting arms, weakness, and pain in arms. Erasmus (27) and Swanepoel (28) observed a male *Hyalomma truncatum* tick in the right axilla of a 21-year-old man and a 16-year-old man, respectively, both suffering from paralysis symptoms. In 1962, Ben-Bassat (29) recorded a 19-year-old boy with complete right arm paralysis who had a male tick found in his right axilla. A male *De. variabilis* was implicated in the paralysis of an 82-year-old patient (30). More recently, in 2003, Edussuriya and Weilgama (21) reported a male *Hy. marginatum isaaci* in a patient's ear. These instances suggest that male ticks may also produce neurotoxins that cause tick paralysis, though such cases are rare. Interestingly, male tick-induced paralysis has mostly occurred in adult men, with most ticks being engorged. This implies that male ticks can induce tick paralysis only when they feed continuously until neurotoxins reach a threshold. Given that a single male tick produces one-seventh as many neurotoxins as a female over the feeding period (23) and that paralysis typically does not occur until females have fed for 4 to 5 days, it may take approximately 20 days for a male tick to accumulate enough neurotoxins to cause paralysis.

Soft ticks can also cause tick paralysis, though less frequently compared to hard ticks. Argasid ticks mainly induce paralysis of fowls. Unlike adult female hard ticks, larvae of soft ticks can cause paralysis due to their long feeding period, which may extend to several days. Peacock (31) described a case of nymphal *Otobius megnini* found in the right ear canal of a 16-month-old boy with a paralysis illness that

lasted about 2 weeks. No additional human tick-paralysis cases caused by soft ticks have been reported, although paralysis-like symptoms and tick-borne diseases involving argasids are documented (32–34), likely because argasids rarely use humans as hosts.

TICK PARALYSIS AND ASSOCIATED TOXICOSES

Tick paralysis is a serious condition caused by neurotoxins from various tick species. It leads to ascending muscle paralysis and, in severe cases, respiratory failure (35). This condition is triggered by neurotoxins produced in the salivary glands of ticks and secreted into the host during blood-feeding. The toxins are primarily produced by female or immature stages of hard ticks and immature stages of soft ticks (35–43). These toxins adversely affect the host's neurological system, with toxin levels peaking after several days of tick engorgement (44). The first identified toxin causing tick paralysis, holocyclotoxin, was discovered in *Ix. holocyclus* by Stone et al. in 1983. It is produced in the large salivary glands of feeding female ticks (45). Other paralytic toxins have since been identified in *Argas walkerae*, *De. andersoni*, and *Rh. evertsi evertsi* (Table 2). In ixodid ticks, the salivary glands are composed of acini, divided into four groups (I, II, III, IV), with Group-III acini (E type cells) being the most likely source of paralysis toxins (46). Toxins first appear in hosts on the third day of tick attachment and increase as the feeding duration extends (47).

Ticks can cause various forms of toxicoses, including paralysis and general toxicoses, during blood meals (34, 44, 48–50). These

toxicoses are associated with tick feeding (51, 52), such as sand tampan toxicoses from *Ornithodoros savignyi* (53), *Hy. truncatum* toxicoses, which includes sweating sickness, Mhlosinga and Magudu, and necrotic stomatitis nephrosis syndrome (54, 55), and other toxicoses from *Rh. microplus* (56), *Rh. appendiculatus* (57), *De. marginatus* (51, 58), *Ix. redikorzevi* (51, 59), and *Or. gurneyi* (60). Understanding the diverse range of toxins and their effects on hosts highlights the importance of researching tick-induced toxicoses. This understanding will be further explored through the specific mechanisms of paralysis caused by different tick families. The molecular characterization of these toxins and their pathogenesis is crucial and will be discussed in the following section.

Paralysis Caused by Ticks of Family Ixodidae

The Ixodidae family comprises several tick species that cause paralysis through the release of neurotoxins, with each species exhibiting unique characteristics and timing of paralysis onset. For example, paralysis in *Rh. evertsi evertsi* occurs between days 4 and 5 of feeding, when the tick reaches a body mass of 15-21 mg (61, 62). Similarly, paralysis caused by *Ix. holocyclus* is detected after 4-5 days of feeding, while in *De. variabilis*, paralysis occurs approximately 6-8 days after attachment (9, 63). Paralysis generally occurs during rapid engorgement, between 3-7 days of tick attachment, when the feeding site is firmly established (64). The toxins released by three genera of hard ticks are discussed in further detail below. Detailed examination of ixodid (hard) ticks reveals the complexity of their toxic effects and the need for targeted research into their

specific mechanisms. Next, we will explore how paralysis differs among various Ixodidae ticks and compare their effects.

Paralysis associated with *Amblyomma* ticks. Eight *Amblyomma* species have been documented to cause paralysis (Table 1). These ticks affect a wide range of hosts, including cattle, dogs, goats, humans, sheep, and snakes, with recovery occurring a few days after tick removal. However, there is a lack of experimental research to confirm their paralysis-inducing capabilities, toxicosis, or the underlying mechanisms involved.

Paralysis associated with *Dermacentor* ticks. Ten *Dermacentor* species are implicated in tick paralysis (64). *De. andersoni* toxins affect efferent motor neurons and can parasitize dogs, sheep, cattle, guinea pigs, hamsters, and humans (64, 65). These toxins act as presynaptic targets by suppressing acetylcholine (ACh) release at neuromuscular junctions (48). Early removal of *De. andersoni* ticks can reverse paralysis (66). Studies on hamsters have shown that the virulence of *De. andersoni* colonies increases with selection over generations, posing a significant threat to the cattle industry due to their paralysis-inducing potential (67). The salivary gland proteins of these ticks increase with feeding, with antigen levels positively correlated to tick weight gain between 25 and 250 mg (68). Wikel observed that *De. andersoni* infestation can reduce T-lymphocyte proliferation in vitro, suggesting an immunosuppressive effect likely related to salivary

gland proteins (69, 70). Further experiments confirmed this effect, with salivary gland extracts from unmated female ticks reducing T-lymphocyte responsiveness by up to 68.4% (71). Bergman et al. detected soluble proteins in the salivary gland supernatants of wood ticks that suppressed murine splenocyte proliferation *in vitro* (72). A 36 kDa immunosuppressive protein was later isolated from the wood tick salivary gland, and its amino acid sequence was characterized (73). This protein, present in both male and female *De. andersoni*, showed temporally regulated expression during feeding (74). However, it is unclear whether this protein is associated with paralysis immunity (75). Additionally, a 39 kDa triplet and a 74 kDa doublet were identified in the salivary gland extracts of *De. andersoni*, although these proteins appeared non-paralyzing and less prevalent (68). No reports have yet documented the development of immunity against *De. andersoni*.

Paralysis associated with *Haemaphysalis* ticks. Seven *Haemaphysalis* species have been implicated in paralysis. *Ha. cinnabarina* was reported to cause paralysis in a girl (76). Both *Ha. kutchensis* and *Ha. punctata* have been confirmed to lead to animal paralysis. *Ha. chordeilis* and *Ha. sulcata* are suspected of causing paralysis in humans, possibly due to mixed infestations with other tick species (22, 77). Other *Haemaphysalis* species may also play a role in paralysis, but research on their toxins and pathobiology remains limited.

Paralysis associated with *Hyalomma* ticks. *Hy. truncatum* is the only species in its genus linked to tick paralysis (64). However, emerging

cases suggest other *Hyalomma* species, such as *Hy. excavatum*, *Hy. dromedarii*, and *Hy. marginatum*, might also be involved in causing paralysis in humans (Table 1). Additionally, *Hy. detritum* and *Hy. aegyptium* have been associated with paralysis in sheep and tortoises (27, 34, 78). While *Hy. truncatum* is known for causing sweating sickness — a non-paralytic toxicosis affecting cattle, particularly young calves (79, 80) and occasionally other coven-hoofed animals such as sheep, goats, and pigs (81) — it is important to note that this condition is distinct from paralysis. In southern Africa, *Hy. truncatum* is responsible for sweating sickness, and two related but milder toxicoses, Mhlosinga and Magudu (77). The toxin responsible for sweating sickness is neither derived from the salivary glands nor associated with paralysis, and thus will not be discussed further here.

Paralysis associated with *Ixodes* ticks. Among tick species implicated in paralysis, *Ixodes* species, particularly *Ix. holocyclus*, are the most significant. Female *Ix. holocyclus* are the primary culprits responsible for tick paralysis in humans, dogs, and cats, although other animals such as cattle, horses, sheep, goats, and swine can also be affected (2, 82). The severity of tick paralysis increases with the duration of tick feeding (83, 84). Ross observed that the salivary gland extracts from *Ix. Holocyclus* induced symptoms similar to tick paralysis in mice, suggesting that these extracts are responsible for the condition (85, 86). Serum from dogs heavily infested with these ticks can mitigate the toxic effects (85), supporting the development of canine anti-tick serum (87), though it is only effective in the early stages of paralysis.

Kaire purified a toxin from homogenates of engorged *Ix. holocyclus* using ammonium acetate buffer on DEAE cellulose columns and confirmed its toxicity and ability to cause paralysis (88). This isolated toxin was stable at pH 3–9 but was inactivated at 100°C within 15 minutes. Further purification efforts identified a high molecular mass protein (40–80 kDa) associated with neurotoxic activity (63). The holocyclotoxin complex comprises three polypeptides—HT1, HT2, and HT3—each with an approximate molecular weight of 5 kDa (89), comparable to toxins found in other arachnids such as spiders (3–11 kDa) and scorpions (5–8 kDa). Additionally, a 20 kDa toxin is linked to cardiovascular failure in affected individuals (89). HT-1 and its genes were isolated, defined, and characterized through N-terminal sequencing, showing significant homology with scorpion neurotoxins (63, 90).

HT-1 contains an 18-residue signal peptide, complete initial and terminal codons, a polyadenylation signal, and a poly-A tail. Nicholson et al. proposed that HT-1, HT-2, and HT-3 may be parts of a gene complex (91). It is generally concluded that paralysis caused by *Ix. holocyclus* results from the inhibition of ACh release at neuromuscular junctions (63). Experiments revealed that muscle contraction induced by the toxin was normal at room temperature (23°C) but progressively declined and ceased under nerve stimulation when temperatures exceeded 35°C (92). This indicates that holocyclotoxin binds to neural synapses in a temperature-dependent manner, with paralysis being temperature-sensitive. Although hyperimmune serum against *Ix. holocyclus* holocyclotoxin has been developed, it does not address paralysis caused by *De. andersoni* (49), suggesting that the toxins from these tick species are either unrelated or very distantly

related evolutionarily.

Paralysis associated with *Rhipicephalus* ticks. In the genus *Rhipicephalus*, several species are known to cause tick paralysis in ungulates, including *Rh. warburtoni*, *Rh. tricuspis*, *Rh. punctatus*, *Rh. exophthalmos*, and *Rh. bursa* (34, 93–96). For humans, *Rh. annulatus*, *Rh. simus*, *Rh. sanguineus*, and *Rh. praetextatus* have been implicated in tick paralysis (22, 34, 97). *Rh. appendiculatus* causes a leucocytotropic disease in cattle, known as brown tick toxicosis (52), while *Rh. evertsi evertsi* induces motor polyneuropathy in sheep, referred to as spring lamb paralysis (98). The condition of sheep paralysis caused by *Rh. evertsi evertsi* was first reported by Hellier (1892) and Ralph (1890) (99, 100). In 1917, Du Toit documented a neuropathic syndrome in South Africa that was potentially linked to *Rh. evertsi evertsi*, although this association was not confirmed (101). Clark's research later confirmed that *Rh. evertsi evertsi* affects young lambs, who typically recover within days following tick removal (102). A protein fraction with an isoelectric point (pI) of 6 was found to inhibit nerve pulse propagation, with a molecular mass of approximately 68 kDa determined through chromatofocusing, SDS-PAGE, and gel permeation chromatography (23). Experiments showed that salivary gland extracts from female *Rh. evertsi evertsi* ticks weighing 15–21 mg could induce nerve block (23, 62, 103). Dissected nerves exposed to these extracts or purified neurotoxin exhibited muscle contraction (23, 64). The paralysis mechanisms of *Rh. evertsi evertsi* differ from those in *Ix. holocyclus* (which inhibits ACh release at the synaptic junction); instead, the neurotoxin from *Rh. evertsi evertsi* affects nerve cells and impairs

nerve impulse conduction along peripheral nerve fibers (23, 64, 104). Two proteins identified during mating and feeding were implicated as toxic components (62). A monoclonal antibody identified a paralysis toxin with an approximate molecular weight of 11 kDa, and amino acid analysis indicated similar protein mass and composition between *Rh. evertsi evertsi* and *Ix. rubicundus* (64). This suggests that the previously identified 68 kDa paralysis toxin is a trimer composed of ~11 kDa proteins.

Paralysis Caused by Ticks of Family Argasidae

Argasidae ticks exhibit a different pattern of paralysis compared to Ixodidae ticks, primarily affecting early developmental stages and demonstrating varied toxic profiles. While Ixodid ticks typically induce paralysis in adults, argasid ticks are more likely to cause paralysis during early developmental stages, such as third-stage nymphs (49). Additionally, adult argasid ticks have smaller Type II alveoli with a wrinkled surface compared to their nymphal counterparts (105). In the following sections, we will explore specific examples of paralysis caused by *Argas*, *Ornithodoros*, and *Otobius* ticks, and discuss their implications for affected hosts (Table 1).

Paralysis associated with *Argasi* ticks. Several *Argas* species are known to cause paralysis in fowl and other birds, including *Ar. africolumbae*, *Ar. walkerae*, *Ar. sanchezi*, *Ar. robertsi*, *Ar. reflexus*, *Ar. radiatus*, *Ar. persicus*, *Ar. monolakensis*, *Ar. miniatus*, and *Ar. arboreus* (Table

1). In laboratory settings, larvae of these species can induce paralysis, with symptoms emerging rapidly during the engorgement phase (106). Among these, *Ar. walkerae* has been particularly well-studied. Its toxic components have molecular masses ranging from 43 to 115 kDa, with a notable shift to 11 kDa under lower pH conditions (107). Similar to *Ix. holocyclus* and *De. andersoni*, paralysis caused by *Ar. walkerae* is attributed to reduced ACh production or release at neuromuscular junctions (108). Research indicates that only replete larvae of *Ar. walkerae* are capable of inducing paralysis in chickens (109). Gel permeation chromatography has identified macromolecular complexes and two bands of approximately 32 kDa and 60 kDa in purified fractions (109). A monoclonal antibody (mAb, 4B12), previously used to detect *Rh. evertsi evertsi* paralysis toxins, was employed to purify the neurotoxin from *Ar. walkerae*. Western blot analysis identified a 68 kDa protein, while an 11 kDa protein, not detected by Western blot, was isolated via ELISA and demonstrated cross-reactivity with 4B12 (107). Lower pH conditions facilitated the formation of a more uniform toxin complex (108). Additionally, extracts from *Ar. walkerae* larvae inhibited the release of [^{3}H] glycine from rat brain synaptosomes, stimulated by both veratridine- and potassium (107).

Paralysis associated with *Ornithodoros* ticks. In the genus *Ornithodoros*, *Or. lahorensis* has been implicated in causing paralysis in sheep and cattle, while the paralysis of marine birds due to *Or. capensis* remains uncertain (110). Paralysis usually develops in the third stage of nymphal ticks. Other species in this genus do not cause paralysis but can induce pain, blisters, local steroid, or edema, including *Or. amblus*,

Or. capensis, *Or. coniceps*, *Or. coriaceus*, *Or. gurneyi*, *Or. muesbecki*, *Or. savignyi*, and *Or. rostratus* (110). One of the earliest reports of toxicosis from sand tampans (*Or. savignyi*) described the death of ten cows within six hours of exposure (64). Kone (1948) first described tampan toxicosis from *Or. savignyi*, noting 10 bovine deaths from a herd of 98 cattle (111). Neitz et al. (1969) improved purification methods (gel and DEAE-cellulose chromatography) for the toxin components in *Or. savignyi* salivary secretions, detecting toxic fractions with undetermined LD50 (112). The toxin mixture was proteinaceous in nature (112).

Fresh oral secretion from *Or. savignyi* revealed high protein nitrogen content and was heat-stable up to approximately 80°C (113). An acidic toxin with a molecular mass of approximately 15 kDa was purified and characterized, and four putative-abundant proteins (TSGP1–4) involved in granule biogenesis in the salivary gland were confirmed (114). MALDI-MS and N-terminal sequencing identified three TSGPs as an acidic toxic homolog (TSGP2, ~15 kDa), a nontoxic homolog (TSGP3, ~16 kDa), and a basic toxin (TSGP4, ~17 kDa) (53, 114). Toxic components isolated by paper and thin-layer chromatography induced lethal effects in animals under experimental conditions (113). Prior to discovering a protein toxin in the salivary gland and larval extracts, the mechanisms and symptoms of paralysis were unclear. Howell suggested that heart failure might be a cause of death, and Mans et al. (2002) observed arrhythmic heartbeats leading to cardiac arrest following salivary gland extract injections (53, 113). Subcutaneous injection of purified toxins showed that TSGP2 primarily caused ventricular tachycardia and TSGP4 induced Mobitz-type ventricular block (53), suggesting a distinct pathogenesis for sand tampan toxicosis

by *Ornithodoros savignyi* compared to tick paralysis.

Paralysis associated with *Otobius* ticks. Most argasid ticks infest and cause paralysis primarily in poultry and livestock, including chickens, camels, cattle, and sheep. However, *Ot. megnini* is an exception, as it parasitizes equines and humans and has been associated with paralysis (31). In one case, a single *Ot. megnini* nymph found in the right ear of a 16-month-old baby resulted in weakness in the right leg, respiratory difficulty, and other paralysis-like symptoms (31). In equines, *Ot. megnini* infestations have been linked to more severe symptoms, including elevated creatine kinase levels and, in some cases, death (115). Despite these severe cases, not all *Ot. megnini* infestations cause paralysis; some only result in irritation and non-paralytic symptoms (116, 117). A recent case involving an 11-month-old cat demonstrated paralysis induced by an infestation of *Ot. megnini* nymphs. The cat exhibited depression, flaccid paralysis, and tachycardia (118). While no specific toxins have been identified in *Ot. megnini*, the consistent presence of nymphs in the ear canal of affected hosts suggests that the location of the infestation may be crucial in causing paralysis. Inflammatory reactions in the ear could potentially disrupt the host's neurological system, leading to paralysis.

MECHANISMS AND NEUROLOGICAL EFFECTS OF TICK-INDUCED PARALYSIS

Tick-induced paralysis typically manifests as ascending flaccid paralysis (Fig. 3) (2). This condition arises from a conduction block in motor nerve fibers, particularly the smaller ones, disrupting the secretion and release of ACh at the neuromuscular junction and at the nodes of Ranvier, without affecting ACh biosynthesis (119–121). Normally, motor neurons generate action potentials that travel down the axon (47). ACh, synthesized and stored in intracellular vesicles, is released into the extracellular space at the presynaptic end of the neuromuscular junction (47). It binds to receptors on muscle cells, causing muscle contraction (13, 47, 122). Flaccid paralysis occurs when tick toxins block ACh secretion and/or release. Despite similar clinical manifestations across different tick species, the mechanisms of toxin action can vary. These variations in mechanisms highlight the complexity of tick-induced paralysis, necessitating a closer look at how different species produce their specific effects on the nervous system.

In *De. andersoni*, paralysis manifests as motor polyneuropathy that primarily affects efferent pathways, with minimal impact on afferent pathways. This condition impairs impulse transmission from muscular nerves or spinal cord synapses (34, 123), potentially due to inhibited ACh release or destruction by anticholinesterase. This process affects the terminal motor fibers at the neuromuscular junction while leaving ACh receptors largely unaffected (49). The specific targeting of efferent pathways by *De. andersoni* underscores the diverse ways ticks can impair neuromuscular function, indicating other tick species may differ in their approach.

In *Rh. evertsi evertsi*, paralysis also presents as motor polyneuropathy of the peripheral nervous system, similar to *De. andersoni*. However, it predominantly affects slow nerve fibers and respiratory muscles, potentially leading to respiratory failure (124). Neurotoxins from this tick act on the nerves rather than directly at the neuromuscular junctions, with no reported inhibition of ACh release (23, 64, 125, 126). This shows the life-threatening potential of tick paralysis and the importance of understanding species-specific toxin actions.

Ix. holocyclus induces paralysis by affecting anterior horn neurons and cranial nerve cells, with minimal impact on peripheral nerves and the cerebral cortex (127). The holocyclotoxin appears to antagonize ACh secretion in a temperature-dependent manner, inhibiting ACh release above 30°C, likely by targeting processes between terminal membrane depolarization and release (119). It has minimal effect on the neuromuscular microendplate potential of the mouse extensor digitorum longus muscle, suggesting it does not target calcium-independent release vesicles or postsynaptic ACh receptors. However, it reduces the endplate potential in a calcium-dependent manner, indicating a presynaptic mechanism involving voltage-gated calcium channels (127). Similar presynaptic inhibitors of potassium and sodium channels are found in spiders and scorpions (128–131). Differences in neurotoxin action between *Ix. holocyclus* and *De. andersoni* likely exist, with *Ix. holocyclus* affecting the spread of nerve impulses along axons and *De. andersoni* affecting motor neurons in the efferent pathway. Despite these differences, both toxins lead to decreased presynaptic ACh and ascending flaccid paralysis, although the exact cellular mechanisms remain unclear. The role of presynaptic inhibition in *Ix. holocyclus* paralysis highlights a unique mechanism among

ticks, with implications for treatment and intervention strategies.

Ar. walkerae larvae cause paralysis in poultry by affecting the peripheral nervous system, particularly fast-conducting nerve fibers, which reduces the motor velocity of the median-ulnar and sciatic nerves (48, 123, 132, 133). This effect ranges from slight to moderate motor polyneuropathy, with minimal impact on the conduction velocity of afferent fibers (134). Efferent nerve fibers in respiratory muscles are also affected, leading to respiratory arrest, while cardiac muscles are generally unaffected (2). Additional studies suggest that these toxins impact not only sensory nerves but also motor nerves, affecting ACh release at neuromuscular junctions and receptor sensitivity at muscular synapses (48, 108). Larval extracts inhibit ACh release by affecting potassium-stimulated and veratrole-stimulated release of [^{3}H] glycine from rat brain synaptosomes (108). Understanding the effects of *Ar. walkerae* on both motor and sensory nerves enhances our knowledge of tick paralysis in animals and should inform future research on its broader implications for both human and animal health.

GLOBAL ANALYSIS OF TICK PARALYSIS CASES

Human Cases of Tick Paralysis

Although human cases of tick paralysis are rare, they have been well-documented across various regions (135–137). A total of 288 tick paralysis cases were identified in reports spanning from 1898 to 2023. Most cases originated from North America, Australia, and Europe,

with fewer reports from Asia and Africa. Most patients had a history of travel or prolonged stays in forested or grassland areas, emphasizing these as potential risk factors for tick exposure. These findings underscore the geographical and demographic factors associated with tick paralysis in humans, highlighting the need for a deeper exploration of specific risk factors involved.

Demographic Characteristics

Of the 288 cases, gender information was recorded for 249 individuals: 96 males (38%) and 153 females (61%), with the remaining 39 cases of unknown gender (Table S1, Fig. 4a). The data indicate a significant gender association with the illness (Fig. 4a and 4b, $P < 0.001$), with females more frequently affected across all age groups. This trend is consistent with observations in tick-borne Lyme disease (138), where females are more often affected, in contrast to diseases like Rocky Mountain spotted fever, Colorado tick fever, and tick-borne encephalitis, which are more prevalent in males (139, 140).

Patient age was recorded for 268 cases, excluding 20 with unrecorded ages, and was categorized into three age groups: under 12 years ($n = 199$), 12 to 50 years ($n = 38$), and over 50 years ($n = 30$). The age distribution was statistically significant, with children under 12 years representing the most affected group (Fig. 4b, $P < 0.001$). This contrasts with tick-borne encephalitis, which predominantly affects adults. The high incidence in children may be attributed to their reduced ability to detect and remove ticks, leading to prolonged tick attachment

and greater toxin accumulation. Additionally, children's lower body weight increases their relative exposure to the paralytic toxin. Among the 199 child patients (under 12 years, including 37 with unrecorded sex), girls (n = 119, 60%) were more frequently affected (Fig. 4c, P < 0.001). This may be attributed to long hair providing cover for ticks, which can hinder their detection. In patients older than 12 years, a male predominance was observed (Fig. 4d, P < 0.001), with 39 males (68%) and 18 females (32%) affected, indicating that males over 12 are more susceptible to tick infestation (Table S1). The demographic analysis highlights the increased vulnerability of certain groups, particularly females and children, highlighting the need for a closer examination of environmental and seasonal influences.

Seasonality, *Environmental* and Geographical Risk *Factors*

Tick paralysis exhibits a strong seasonal pattern. Among the 111 cases with documented occurrence timing, most (n = 71, 64%) occurred in summer (Fig. 4e), with May being the most common month (P < 0.001). Fall (n = 20) and spring (n = 7) accounted for fewer cases, indicating season as a significant epidemiological risk factor. This seasonality may vary based on regional weather patterns and environmental factors.

Most patients with a clear history (66/77, P < 0.001) had direct exposure to natural environments, including rural living, mountain hiking, coastal trips, bushwalking, or outdoor work (Table S1). Close contact with tick-infested animals or humans was another risk factor (n = 11,

Table S1). Residence in areas with abundant vegetation was identified as a significant risk factor. Geographical factors also play a role, with many cases reported from the eastern coast of Australia and the Pacific Northwest of North America. Several patients had traveled to these regions prior to the onset of paralysis. Understanding the seasonality and environmental risks is crucial for developing prevention strategies, which is closely linked to the clinical manifestations of tick paralysis.

Clinical Manifestation

Initial symptoms and progression. Tick paralysis can be severe and potentially fatal; however, it is often reversible with timely tick removal and prompt treatment (141). Initial symptoms are typically mild and may include fatigue, pain, paresthesia, and ataxia (142, 143). Neurological symptoms, such as symmetric ascending flaccid weakness, usually begin 5-7 days after tick attachment (120). If the tick remains attached, paralysis may progress from the legs to the torso and arms, eventually compromising respiratory muscles within hours (142). In severe cases, muscles controlled by cranial nerves may weaken, leading to respiratory depression. Some children have died from respiratory failure without intervention (Table S1). Often, when respiratory symptoms appear, the tick has engorged and dropped off, explaining why some patients recover without the need for artificial ventilation or intubation. Recognizing the early signs and progression of tick paralysis is vital for effective intervention, and these symptoms can vary significantly depending on the tick species and attachment

site.

Variations in clinical presentation. Tick paralysis is marked by acute, rapidly progressive muscle weakness (144), with lateral staring-induced nystagmus reported in children (143, 145). Different tick species may cause distinct clinical manifestations due to variations in toxin pathophysiology (47). For instance, blood pressure is typically normal in cases of paralysis linked to *De. andersoni*, whereas hypertension is often associated with paralysis caused by *Ix. holocyclus* (34).

The clinical presentation may also vary based on the tick attachment site. The scalp and ears (including ear canals) are common attachment sites (Fig. 5a). Ticks on the scalp primarily affect extremity nerves, leading to weakness or even paralysis of all extremities (Table S1, Fig. 5b). Deep tendon reflexes in the limbs are often diminished or absent (Fig. 5b). As the illness progresses, extremity weakness may be accompanied by bulbar symptoms, such as ophthalmoplegia, dysarthria, and dysphagia. Ticks in the ear canal mainly affect facial nerves, causing severe partial facial weakness, paresthesia, and potentially unilateral facial palsy. Common symptoms include ear pain and facial asymmetry (Fig. 5c). Fever and hearing loss are rare initially but may develop with worsening inflammation in the ear canal. A Weber test shows lateralization, and a Rinne test is normal under tuning fork examination (146). These symptoms may result from neurotoxins spreading into the middle or inner ear through a perforated tympanic membrane. Ticks can also attach to other parts of the body, such as the axilla (15, 147), back (136, 148, 149), chin (150), eyelids (151, 152), limbs (153–156), necks (5, 142, 149), and shoulder (157).

Similar to scalp ticks, ticks attached to other body parts or cases with unrecorded tick locations typically cause ascending flaccid extremity weakness or paralysis (Fig. 5d and 5e). The diversity in clinical presentations emphasizes the need for tailored treatment approaches, which is particularly important when considering the severity and potential fatality of tick paralysis.

Severity and Fatality

Fatal cases. Clinical manifestations in children tend to be more severe than in adults due to their weaker immune systems. A total of 29 fatal cases were documented in Australia and North America (5, 15, 76, 158–160) (Table S1), all involving young patients. The tick species responsible for these deaths included *De. andersoni* (*n* = 3), *De. variabilis* (*n* = 5), *Ix. holocyclus* (*n* = 6), and *Ha. cinnabarina* (*n* = 1). The oldest fatality was a nearly 14-year-old boy who succumbed to respiratory failure, while the youngest was a 10-month-old infant (158). Most fatalities resulted from respiratory failure or paralytic complications, with no adult fatalities reported. The severity of tick paralysis, especially in children, highlights the importance of early detection and treatment, and the necessity to examine how these factors vary across different regions.

Regional Variation in Tick Paralysis

Prompt tick removal usually reverses tick paralysis, often reducing clinical symptoms within hours. However, in some countries, symptoms can significantly worsen after tick removal (143). Patients from North America and Australia experience different clinical progressions and prognoses after tick removal. Australian cases tend to worsen before gradually improving, in contrast to North American patients (47). However, clinical progression and prognosis appear more closely related to the specific neurotoxins of the tick species involved. For example, 13 cases caused by *Ix. holocyclus* deteriorated after tick removal for several days before symptoms began to regress, eventually leading to complete recovery. Of these, 11 were from Australia (147, 149, 152, 161–163), with the remaining two from Japan (164) and Singapore (165). Both the Japanese and Singaporean patients had traveled to Australia, where they were infested by Australian ticks. The holocyclotoxin secreted by *Ix. holocyclus* accumulates and increases in concentration, reducing presynaptic ACh release. Holocyclotoxin onset may require a time-dependent intracellular step, which could explain the delayed development of symptoms despite tick removal. The pathophysiological process persists until the neurotoxin is reduced, at which point patients begin to recover.

Besides *Ix. holocyclus*, species from *Dermacentor* (14, 15, 49, 166) and *Haemaphysalis* (76) have also been linked to cases where symptoms worsened after tick removal, although such deterioration is rare. Gregson (49) documented irregular respiration and an imperceptible pulse in a young boy after tick removal. Taylor (14) reported a 3-year-old Canadian child bitten by *De. andersoni* who remained in critical condition for at least 24 hours and developed a typhus-like rash after tick removal. Both Costa (166) and Mccornak (15)

recorded young children whose conditions worsened post-tick removal, with one case improving and another resulting in death from bulbar paralysis. These differences may be due to varying toxin pathologies, though the mechanism remains unclear. The regional differences in clinical outcomes suggest a strong influence of tick species and their neurotoxins.

Long-Term Effects and Co-Infections

Persistent symptoms. In most cases, patients fully recover within days after tick removal, but some experience lasting effects. A 78-year-old woman experienced persistent fatigue following a tick bite (167), although the tick species was not identified. Similar complaints of drowsiness and weakness were observed in an adult man infested by *Rh. simus* (168) and a nearly 4-year-old boy bitten by *De. variabilis* (15) who complained they were drowsy and weak always. Swanepoel (28) reported a 16-year-old boy bitten by *Hy. truncatum* who suffered permanent weakness in his wrist, fingers, and right-hand muscles. Gregson (49) summarized five cases where paralysis persisted for days after tick removal, only resolving when the skin at the attachment site was excised, suggesting that tick toxins may accumulate in the skin and continue to affect the body.

Co-infections. Additionally, ticks may transmit pathogens while secreting neurotoxins during feeding, leading to co-infections with tick-borne viral or bacterial diseases (164). Therefore, when patients develop fever, lymph node enlargement, or other atypical symptoms, concomitant infection with tick-borne pathogens should be considered. While most cases resolve after tick removal, some patients

experience persistent symptoms or co-infections, underscoring the need for ongoing monitoring and research into tick-borne diseases in both humans and animals.

Tick Paralysis in Animals

This condition has been documented in various domestic and wild animals, including canids (169–172), felids (135, 173), cattle (174), caprines (175–178), horses (179), gray fox (180), snake (181), llama (182), mouse (183), and birds (184, 185) (Table 1). A case of tick paralysis was also reported in a wapiti (*Cervus elaphus*) in China, although the tick species responsible was not identified. Tick paralysis in animals shares similarities with human cases, particularly in its progression and clinical outcomes, which is evident in species-specific patterns that further our understanding of the condition.

Onset and progression of tick paralysis. In animals, tick paralysis usually manifests 4 to 7 days after tick attachment, though symptoms can take up to 13 days to appear in some cases (172). Clinical signs include weakness, gait incoordination, voice changes, and increased respiratory effort. Most animals recover with prompt treatment and supportive care (135, 169, 173, 179). However, in acute cases, symptoms can progress rapidly within 12 hours to 5 days, beginning with limb paresis and advancing to hind-quarter or generalized

paralysis, respiratory depression, and, in severe cases, death—even after tick removal and antiserum treatment (171, 172).

Feline tick paralysis cases. Ellie Leister and colleagues analyzed 2,077 feline cases of tick paralysis caused by *Ix. holocyclus* between 2008 and 2016. Of these cases, all from the coastal city of Queensland, 46 experienced recurrent paralysis, and 1,742 resulted in death despite emergency treatment (186). These findings underscore the high mortality rate associated with tick paralysis in felines. The high mortality rate in feline tick paralysis cases highlights the severity of this condition in animals, leading to a broader comparison with other species and geographical patterns.

Comparative mortality and geographic patterns. A comparison between feline and canine tick paralysis cases reveals a higher mortality rate in felines. Both species' cases were reported predominantly from coastal areas, with canines also showing a significant occurrence at the city's edge near jungles. This similar geographic distribution highlights the environmental factors contributing to tick exposure and the subsequent risk of paralysis. Comparing mortality and geographic patterns across species provides valuable insights into the environmental factors driving tick paralysis, which can inform prevention and treatment strategies.

Seasonal discrepancies: Canine vs. human tick paralysis. In contrast to the feline cases, canine tick paralysis cases in Australia peaked at 1,124 during spring (out of a total of 1,650 cases), with a 42% recovery rate (187). This contrasts with the higher mortality rate observed in felines (186). Interestingly, the seasonality of canine tick paralysis cases contrasts with that of human cases. Over half of human paralysis cases (67 out of 105) occurred in the summer, while most canine cases were observed in spring. This discrepancy may be due to ticks seeking dogs as blood meal sources in spring when *Ixodes* spp. adult females are most active, whereas humans are more frequently exposed to tick bites during summer outdoor activities.

DIAGNOSIS AND RECOGNITION OF PATIENTS AT RISK

Early recognition of clinical manifestations and recent tick exposure is crucial for diagnosing tick paralysis. Diagnosis is often confirmed by identifying a tick along with generalized weakness and/or respiratory distress. A history of the patient being in tick-endemic regions or having recently visited forests is also important for accurate diagnosis. Physicians should carefully inspect areas where ticks commonly attach to humans, such as the scalp (especially under long, thick hair), ears, ear canals, groin, axilla, and perineum (Table S1). Early identification of the tick can prevent unnecessary clinical and laboratory investigations, reducing the risk of deterioration, fatal outcomes, and costly tests. Using a fine-toothed comb can be particularly useful in diagnosing tick paralysis, especially in individuals with thick, long

hair, who may be more susceptible to infestation. In some cases, paralysis may develop even after the tick has detached, which is more common in Australia. Cerebrospinal fluid tests typically yield normal results. The level of tick engorgement can affect imaging results, with highly engorged ticks showing a striking T1-hyperintense signal and T2-hypointensity on MRI , 214).

Clinical Manifestations

Tick paralysis typically presents as ascending flaccid paralysis that progresses over hours to days. Initial symptoms include fatigue, irritability, paresthesias, and muscle weakness, while fever and pain are uncommon (13). Some patients may exhibit apathy and loss of appetite, along with tingling and numbness in both the lower and upper extremities. Weakness usually begins in the lower limbs and ascends to the upper body as the toxin accumulates. Reflexes may become diminished or absent, and the condition can progress to generalized paralysis. Partial facial palsy, particularly involving the lower motor neurons, may occur, especially in cases of tick infestation in the ear canal (149, 190). Without prompt intervention, the paralysis may involve respiratory muscles, leading to respiratory depression and, ultimately, death. Mental status typically remains intact until hypoxia and hypercarbia set in, potentially causing convulsions (5, 97). Although rare, dilated pupils can be observed (15, 158). Gastrointestinal or other general intoxication symptoms are infrequent.

Differential Diagnosis

Due to the rarity of tick paralysis and limited clinical experience, the condition is often misdiagnosed, causing physicians to miss the narrow therapeutic window for effective treatment (191). In cases of rapidly progressive paralysis, it is crucial to rule out tick attachment first through a thorough skin inspection. Imaging can also help detect small nodules that may indicate tick presence, preventing oversight (191). A study of 143 children with non-traumatic acute flaccid paralysis in Australia over 4.5 years found that 3 had tick paralysis (192). Guillain-Barré syndrome (GBS) accounted for 67 cases (47%), with transverse myelitis contributing to 27 cases (19%). Other conditions presenting with acute flaccid paralysis included acute disseminated encephalomyelitis, spinal cord lesions, botulism, myasthenia gravis, and viral encephalitis. The overlapping symptoms of these conditions, such as irritability, paresthesia, fatigue, and muscle weakness, often lead to misdiagnosis of tick paralysis (193–198). Some diseases are particularly challenging to differentiate from tick paralysis, and rare disorders may not be present in their traditional form. As summarized in Table 3, several conditions present with symptoms that overlap with tick paralysis, making differential diagnosis challenging. However, the table is not exhaustive and should be used as a guide alongside clinical judgment (193–198).

GBS is a significant differential diagnosis. Although treatments such as plasmapheresis and intravenous immunoglobulin are available, there is no definitive cure. The progression of GBS is slower than that of tick paralysis, with weakness developing over weeks rather than

hours or days (199). GBS may present with oropharyngeal weakness, respiratory failure, or autonomic nervous dysfunction (200). Symptoms often include prior gastrointestinal disturbances, such as emesis or diarrhea. GBS typically involves areflexia or hyporeflexia and elevated protein levels with normal cell counts in cerebrospinal fluid (200). Additionally, GBS presents with reduced nerve conduction velocity, decreased compound muscle action potential, and a prolonged "F" wave, indicating proximal nerve root demyelination, which does not occur in tick paralysis (11).

Poliomyelitis, also known as infantile paralysis, is another differential diagnosis often confused with tick paralysis, particularly in cases involving travel to polio-endemic areas. While rare due to vaccination efforts, polio is still endemic in regions of Afghanistan and Pakistan, according to WHO reports. Poliomyelitis typically presents with fever, meningitis, and asymmetric weakness, a major cause of lameness (201, 202). Cerebrospinal fluid analysis may show mildly elevated protein and lymphocytosis (203). MRI findings may also be abnormal.

Acute spinal cord lesions, which present with flaccid paraplegia or quadriplegia, differ from tick paralysis by including symptoms such as urinary retention and bowel incontinence at the sensory level (204). These lesions typically result in decreased sensation and organ function below the affected area, and protein levels can be significantly elevated in cerebrospinal fluid (11).

Botulism, caused by *Clostridium botulinum* toxin, also presents with flaccid paralysis. Like tick paralysis, botulism reduces ACh release. Symptoms often begin with cranial nerve involvement, leading to descending paralysis that primarily affects extraocular muscles and

pupils (199). Patients may develop dysphonia and dysphagia due to lower cranial nerve involvement (205), and gastrointestinal symptoms may also occur. Hematological exams in botulism typically do not show abnormal protein or white blood cell elevations.

Transverse myelitis, which can be triggered by viral, bacterial, or parasitic infections, presents with pain, paresthesia, motor deficits, and bladder dysfunction 210, 211). Elevated protein levels and T-lymphocytes are often seen in cerebrospinal fluid (207). MRI of the spinal cord typically shows lesions with high signal intensity on T2-weighted sequences, particularly in the cervical region, conus medullaris, or thoracic spinal cord (206, 207).

TREATMENT

The primary treatment for tick paralysis is the prompt removal of the tick(s) (208). A thorough examination of the patient's skin is essential, including areas such as the scalp, ear canal, nose, and armpits. The recommended method involves using fine-toothed tweezers to grasp the tick parallel to the skin and applying steady, even force to remove it intact, including the mouthparts, which can be challenging to extract (17, 216). Care must be taken to avoid squeezing the tick, which could inject additional toxins and worsen the condition. When removing ticks from the ear canal, combining tick removal with antibiotic treatment may be advisable to prevent otitis media. If patients exhibit atypical symptoms during tick paralysis, a combination treatment may be necessary to address potential co-infections.

Patients should be observed for at least 24 to 48 hours, or longer if necessary, after tick removal to ensure steady and clear improvement. For example, in cases of paralysis caused by *Ix. holocyclus*—particularly common in Australia—there is a risk of significant deterioration after tick removal, including life-threatening angioedema (210). Patients with respiratory failure may require mechanical ventilation in an intensive care unit to manage dyspnea.

Antitoxins derived from dogs are another treatment option, though they carry risks of acute anaphylaxis and neuropathy. These antitoxins are generally administered in small amounts and reserved for severely ill patients in Australia (143). The injection of anti-paralysis tick serum has demonstrated efficacy in neutralizing paralysis toxins. Recently, a synthetic anti-venom vaccine derived from holocyclotoxins has shown effectiveness in immunizing dogs (211). This suggests that synthetic or recombinant vaccines could be valuable in preventing tick paralysis and may contribute to future vaccine development, although further testing in animals is needed to assess their immunological efficacy.

CONTROL AND PREVENTION

To reduce the risk of tick-borne diseases and tick paralysis, effective repellents should be applied to the skin, such as trans-p-methane-3,8-diol and N,N-Diethyl-3-methylbenzamide (212, 213). Minimizing tick attachment is the most effective way to prevent tick paralysis. In

addition to using repellents, individuals should wear appropriate outfits — such as long-sleeves, long socks — when participating in outdoor activities. It is advisable to avoid walking through dense vegetation and to take precautions when working in bushy areas. Following outdoor activities, particularly in forested environments, individuals should thoroughly check their skin for ticks. For controlling tick paralysis in dogs and cats, the development of anti-tick vaccines represents an effective strategy.

CONCLUDING REMARKS

Tick paralysis is a potentially fatal condition with significant implications for human and animal health. Its epidemiology is influenced by factors such as seasonality, geography, age, and gender. Clinical outcomes are determined by several variables, including the species and number of ticks, the rate and amount of toxin secretion, host immune response, and the sensitivity of the tick attachment site. The condition arises from neurotoxins secreted by the salivary glands of various tick species, which accumulate in the host when the tick remains attached for several days. These toxins primarily inhibit acetylcholine release at neuromuscular junctions, leading to symptoms such as lethargy, weakness, unsteady gait, dilated pupils, ascending symmetrical paralysis, slurred speech, and reduced deep tendon and gag reflexes. Severe cases may involve respiratory distress, bradycardia, decreased oxygen saturation, and asystole. Fortunately, tick paralysis is generally reversible with prompt intervention, primarily through tick removal. However, removal can sometimes exacerbate

the condition, requiring respiratory support and intensive care. Immunoassays to detect and quantify antibodies against tick toxins can also be valuable in assessing immune status and monitoring post-immunization responses. Future efforts should focus on advancing our understanding of the molecular mechanisms underlying tick paralysis, improving diagnostic tools, and developing targeted therapies to enhance patient outcomes and reduce the health impacts of this condition.

SUPPLEMENTARY MATERIAL

Supplemental material is available online only.

Supplemental file 1

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AUTHORS CONTRIBUTORS

GHL, CY, HME, YPD, and YTF conceived the review. YPD, YTF, and GHL wrote the initial draft. CY and HME reviewed and abstracted data from the selected articles, and revised the manuscript. YPD, YTF, MLC, and GHL produced the figures and verified the data. XQZ, JLW, XLZ, and SCX assisted with editing. All authors read and approved the final version of the manuscript.

DECLARATION OF INTERESTS

The authors declare no competing interests.

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FIGURE LEGENDS

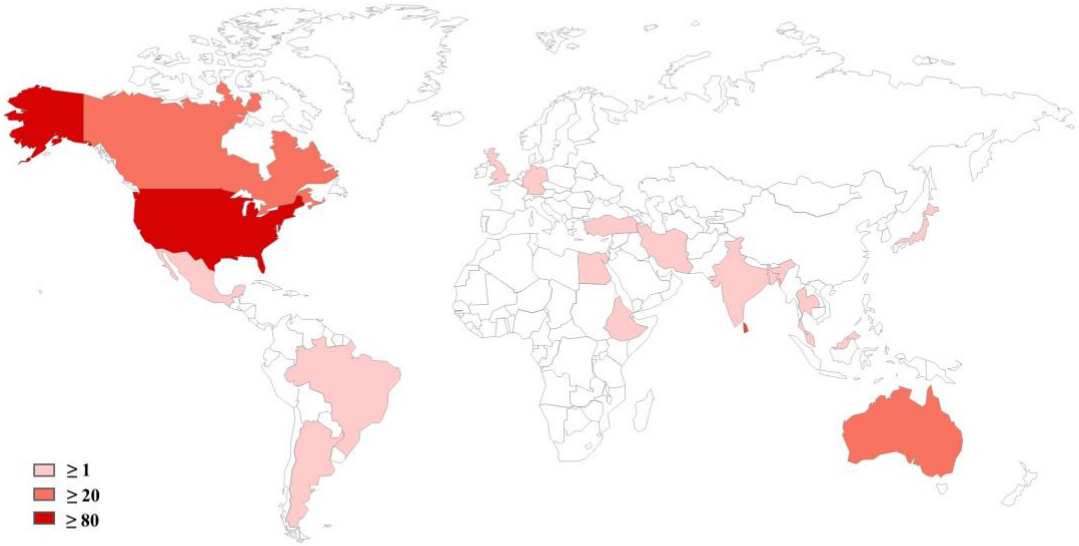

FIG.1. Global distribution of reported tick paralysis cases in humans. Areas shaded in darker red indicate a higher number of reported cases, while lighter red areas have fewer cases. Regions with no color represent areas where no cases have been reported.

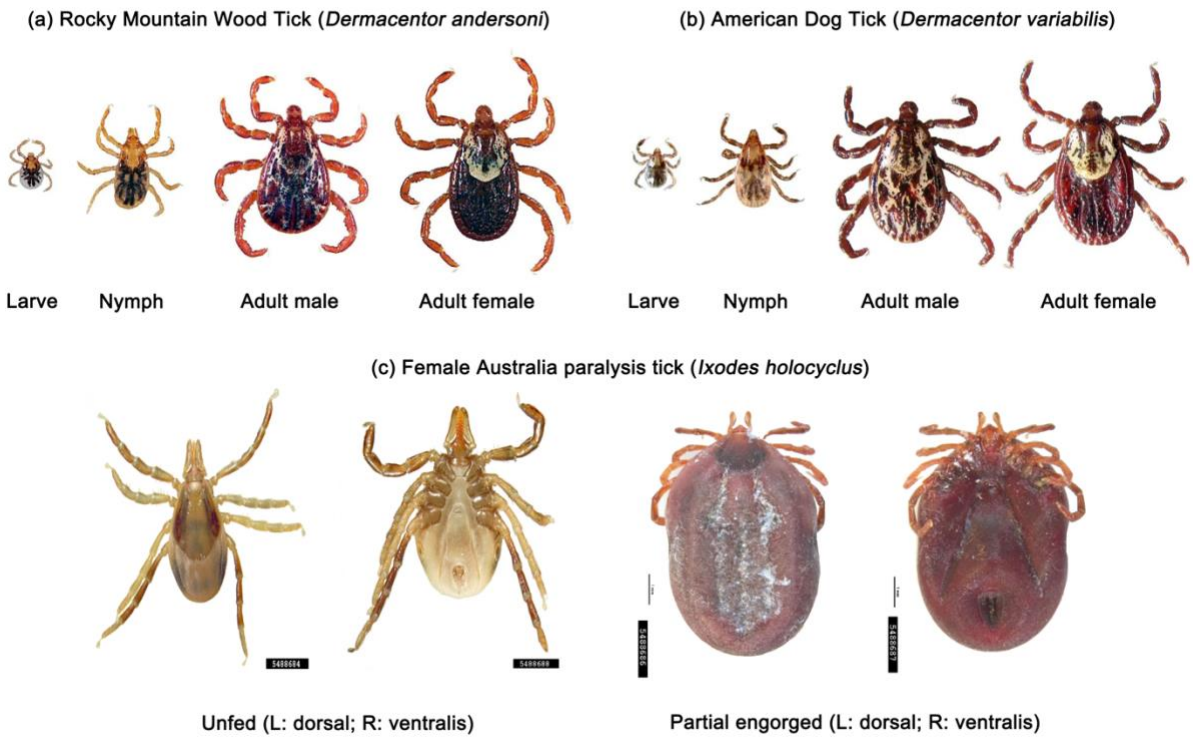

Fig. 2. Images of the three most common ticks causing human paralysis globally. (**a**) Life stages of the Rocky Mountain Wood Tick (*Dermacentor andersoni*) (Adapted from: Tick Encounter). (**b**) Life stages of the American Dog Tick (*Dermacentor variabilis*) (Adapted from: Tick Encounter). (**c**) Unfed and partially engorged female Australia paralysis tick (*Ixodes holocyclus*) (Adapted from the Pest and Diseases Image Library, Bugwood.org)

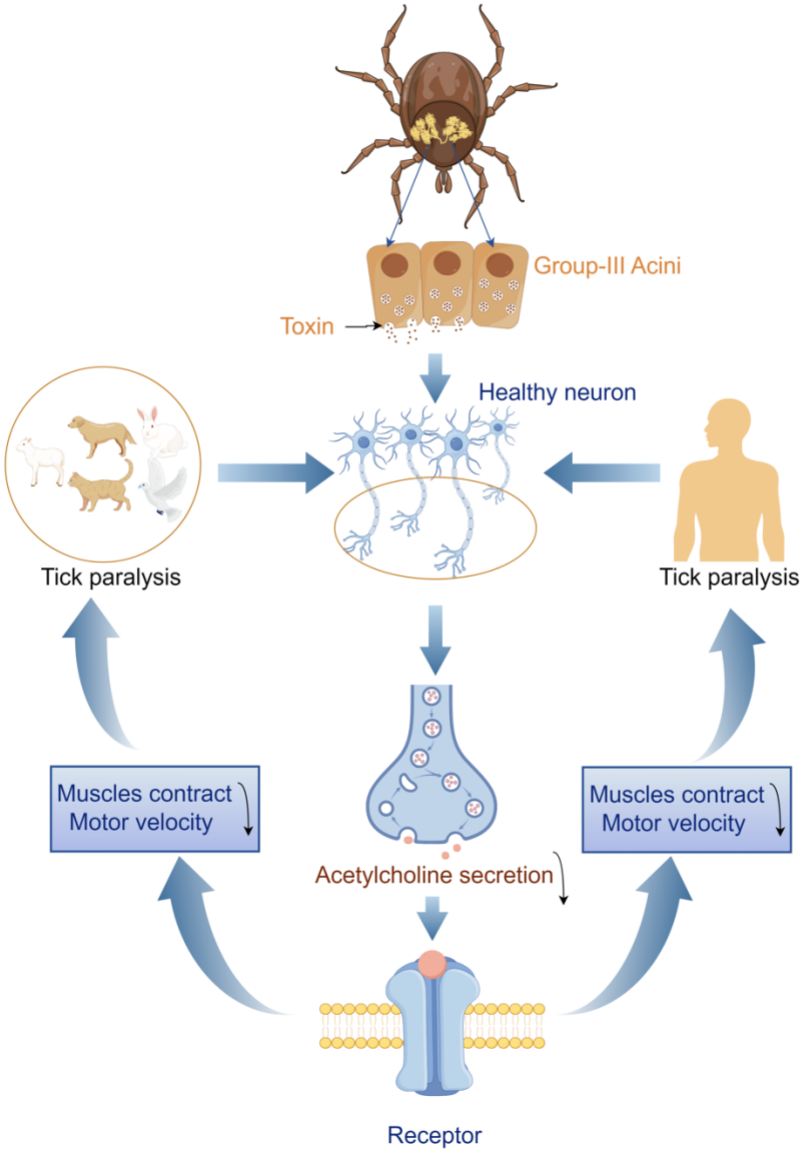
Group-III Acini
Toxin
Healthy neuron
Tick paralysis
Tick paralysis
Muscles contract
Motor velocity
Muscles contract
Motor velocity
Acetylcholine secretion
Receptor

FIG. 3. Schematic illustration of the molecular pathogenesis of tick paralysis in humans and animals (Illustration designed using FigDraw).

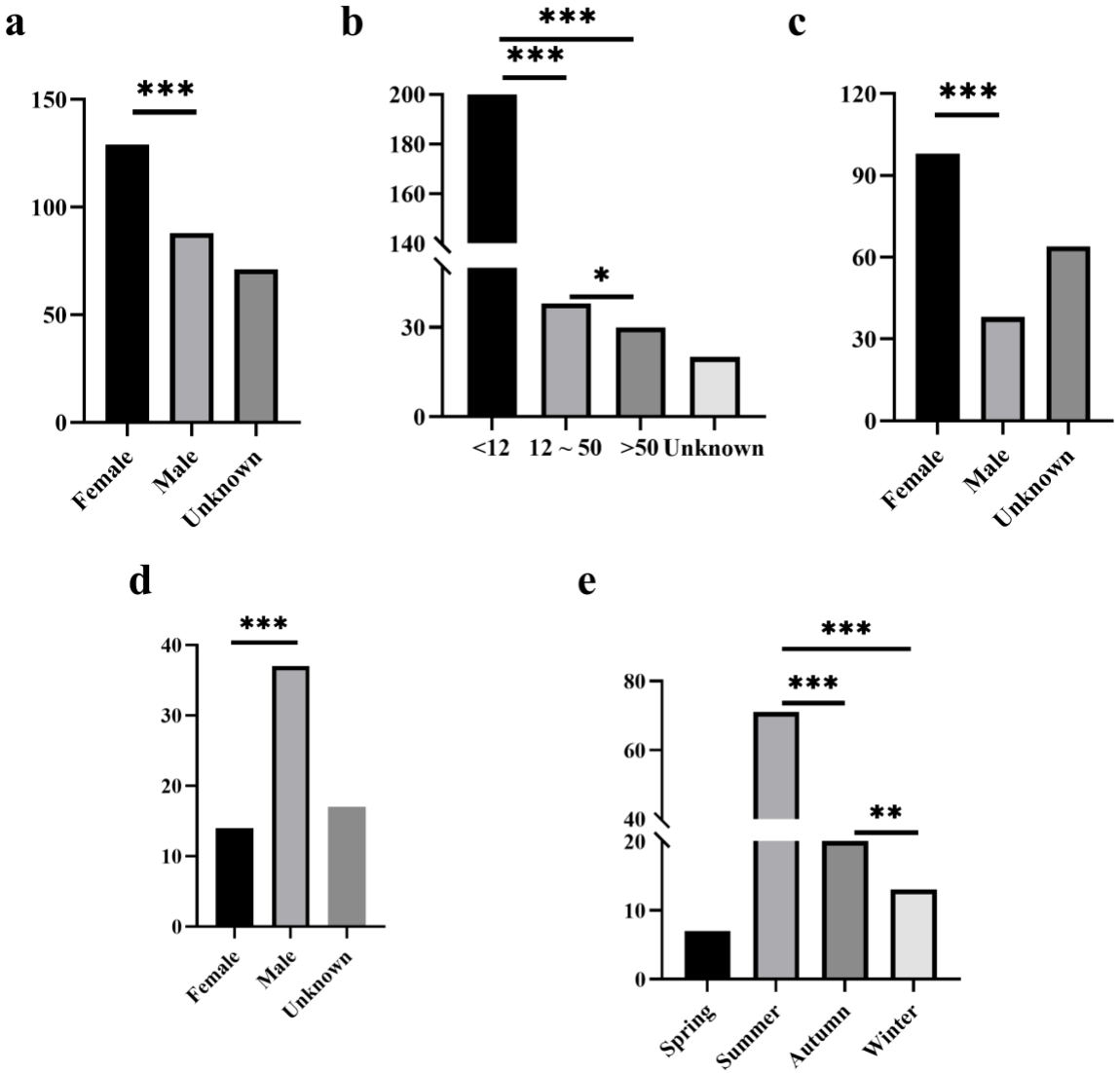
a
***
150
100
50
0
Female
Male
Unknown
b
***
***
*
200
180
160
140
30
0
<12
12 ~ 50
>50
Unknown
c
***
120
90
60
30
0
Female
Male
Unknown
d
***
40
30
20
10
0
Female
Male
Unknown
e
***
***
**
80
60
40
20
10
0
Spring
Summer
Autumn
Winter

FIG. 4. Global distribution of reported human cases of tick paralysis by gender, age, and season, analyzed by Pearson's chi-squared test. (**a**) Gender distribution. (**b**) Age distribution (<12, 12 to 50, and >50). (**c**) Gender distribution of cases in individuals under 12 years old. (**d**) Gender distribution of cases in individuals aged 12 years and older. (**e**) Seasonal distribution. *: $P < 0.05$; **: $P < 0.01$; ***: $P < 0.001$

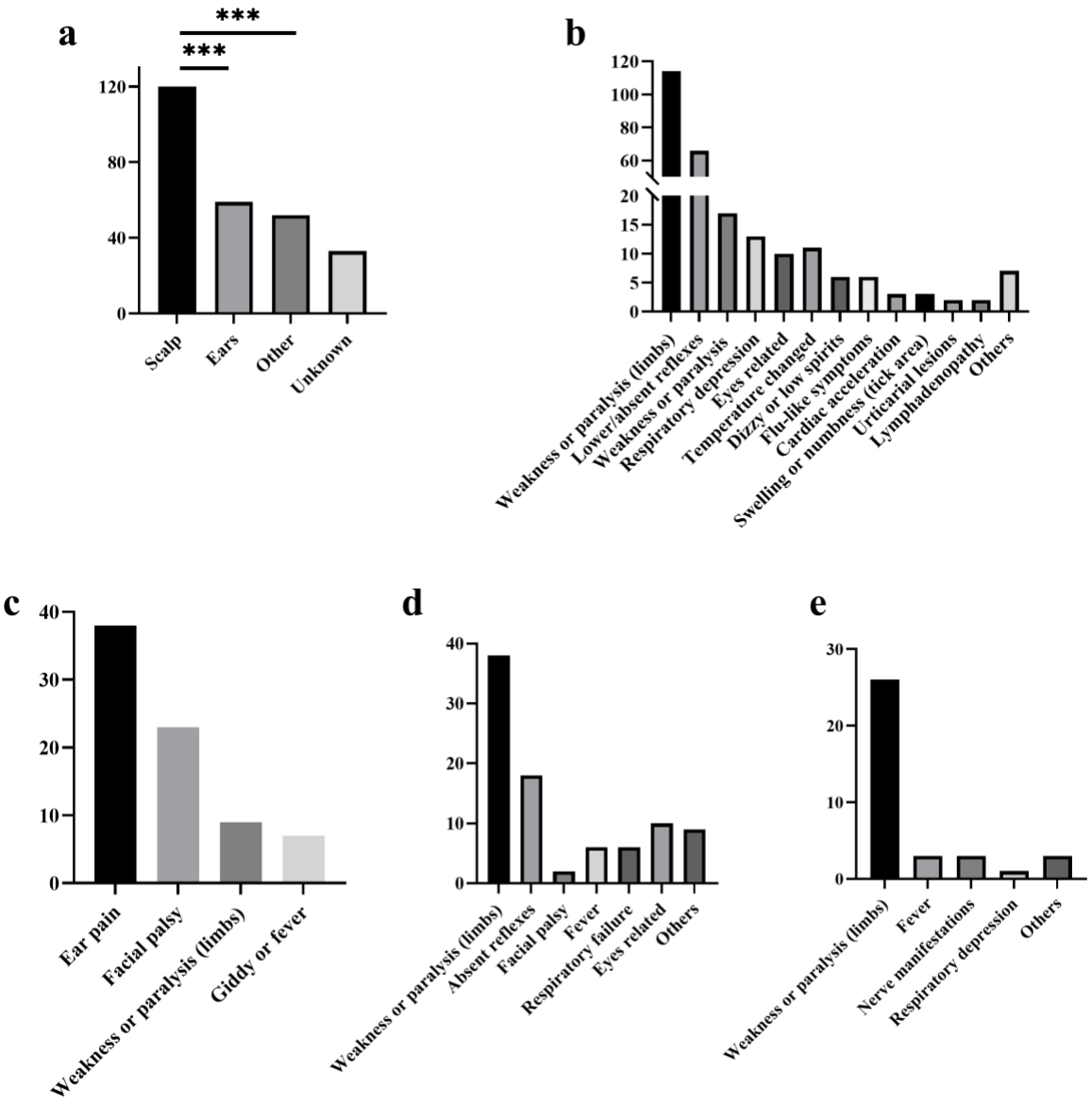
a
***
***
120
80
40
0
Scalp
Ears
Other
Unknown
b
120
100
80
60
20
15
10
5
0
Weakness or paralysis (limbs)
Lower/absent reflexes
Weakness or paralysis
Respiratory depression
Eyes related
Temperature changed
Dizzy or low spirits
Flu-like symptoms
Cardiac acceleration
Swelling or numbness (tick area)
Urticarial lesions
Lymphadenopathy
Others
c
40
30
20
10
0
Ear pain
Facial palsy
Weakness or paralysis (limbs)
Giddy or fever
d
40
30
20
10
0
Weakness or paralysis (limbs)
Absent reflexes
Facial palsy
Fever
Respiratory failure
Eyes related
Others
e
30
20
10
0
Weakness or paralysis (limbs)
Fever
Nerve manifestations
Respiratory depression
Others

FIG. 5. Clinical manifestations of human tick paralysis based on tick attachment locations. (**a**) Distribution of tick attachment sites (statistical analysis performed using Pearson's chi-squared test, ***: $P < 0.001$). (**b**) Clinical symptoms associated with ticks infesting the scalp. (**c**) Clinical symptoms associated with ticks infesting the ears. (**d**) Clinical symptoms associated with ticks infesting the back, neck, axilla, and other parts (excluding scalps and ears). (**e**) Clinical symptoms of tick paralysis with unrecorded tick attachment sites.

TABLE 1 Tick species causing tick paralysis worldwide

Family	Genus	Species (Common name)	Geographical location	Hosts reported on	Reference
Ixodidae	*Amblyomma*	*Am. americanum* Linnaeus, 1758 (Long Star tick)	USA (mainly coast)	Canids, humans	(214, 215)
		Am. argentinae Neumann, 1905	Argentina	Reptiles	(51)
		Am. cajennense Fabricius, 1787 (Cayenne tick)	America	Cattle, goats, sheep	(216)
		Am. hebraeum Koch, 1844 (Bont tick)	South Africa	Humans, goats, sheep	(217)
		Am. maculatum Koch, 1844 (Gulf Coast tick)	Central America	Canids, humans	(153, 218)
		Am. ovale Koch, 1844	America	Humans	(219)
		Am. rotundatum Koch, 1844	America	Poikilotherm	(181)
		Am. variegatum Fabricius, 1794 (Tropical bont tick)	Africa	Sheep	(175)
	Dermacentor	*De. albipictus* Packard, 1869	North America	Ungulates	(34)

	De. andersoni Stiles, 1908 (Rocky Mountain wood tick)	North America	Humans, mammals	(6)
	De. auratus Supino, 1897	Oriental realm	Humans	(21)
	De. variabilis Say, 1821 (American dog tick)	North America	Canids, humans	(5, 15, 220)
	De. silvarum Olenev, 1931	Eurasia	Sheep	(77)
	De. rhinocerinus Denny, 1843	Africa	Rabbits	(221)
	De. reticulatus Fabricius, 1794 (Ornate cow tick)	Palearctic realm	Sheep	(222)
	De. occidentalis Marx, 1892 (Net tick)	North America	Canids, humans, ungulates	(49)
	De. nuttalli Olenev, 1928	Palearctic realm	Ungulates	(1)
	De. marginatus Sulzer, 1776 (Ornate sheep tick)	Palearctic realm	Humans, ungulates	(178, 223)
Haemaphysalis	*Ha. chordeilis* Packard, 1869 (Bird tick)	North America	Humans	(77)
	Ha. sulcata Canestrini & Fanzago, 1878	Eurasia	Humans, ungulates	(22)
	Ha. punctata Canestrini & Fanzago, 1878 (Red sheep tick)	Palearctic realm	Ungulates, birds	(178, 224)
	H. parva Neumann, 1897	Middle East	Sheep	(120)
	Ha. kutchensis Hoogstraal & Trapido, 1963 (Kutch haemaphysalid)	Oriental realm	Rabbits	(225)
	Ha. inermis Birula, 1895 (Winter tick)	Europe	Ungulates	(34)
	Ha. cinnabarina Koch, 1844	Brazil	Humans, ungulate	(76)
Hyalomma	*Hy. truncatum* Koch, 1844 (Shiny Hyalomma)	South Africa	Humans, sheep	(28, 95)
	Hy. detritum Schulze, 1919	Oriental realm	Sheep	(78)

	Hy. aegyptium Linnaeus, 1758 (Bont leg tick)	Palearctic realm	Sheep, tortoise	(27, 34)
	Hy. excavatum Koch, 1844	Turkey, Egypt	Humans, sheep	(22, 94)
	H. dromedarii	Egypt	Humans	(22)
	H. marginatum	Turkey, Sri Lanka	Humans	(21, 226)
Ixodes	*Ix. arboricola* Schulze & Schlottke, 1930 (Tree-hole tick)	Europe	Birds	(34)
	Ix. brunneus Koch, 1844	North America	Birds	(227)
	Ix. gibbosus Nuttall, 1916	Palearctic realm	Ungulates	(228)
	Ix. frontalis Panzer, 1798 (Passerine tick)	Europe	Birds	(34)
	Ix. eudyptidis Maskell, 1885 (New Zealand seabird tick)	Tasmania, New Zealand	Albatrosses, petrels	(229)
	Ix. crenulatus Koch, 1844	Europe	Sheep	(66)
	Ix. cornuatus Roberts, 1960 (Tasmanian paralysis tick) (synonym of *I. robertsi*)	Australia (mainly Tasmania)	Canids, felids, humans	(171, 230, 231)
	Ix. cookei Packard, 1869 (American Castor Bean tick)	North America	Humans	(34)
	Ix. hexagonus Leach, 1815 (European dog tick)	Europe and USA	Humans	(120)
	Ix. tasmani Neumann, 1899 (Common marsupial tick)	Australia	Marsupials	(232)
	Ix.tancitarius Cooley and Kohls, 1942	Mexico	Humans	(155)
	Ix. scapularis Say, 1821 (Black-legged tick)	North America, Europe, and China	Canids, humans	(233)
	Ix. rubicundus Neumann, 1904 (Karoo paralysis tick)	South Africa	Humans, mammals, rabbits	(234–236)

		Ix. ricinus Linnaeus, 1758 (Castor bean tick)	Europe, North America, Africa	Humans, sheep	(178)
		Ix. redikorzevi Olenev, 1927	Palearctic realm	Humans	(237)
		Ix. pacificus Cooley & Kohls, 1943 (California black-legged tick)	North America	Canids, hare, humans	(220, 238)
		Ix. muris Bishopp & Smith, 1937 (Mouse tick)	North America	Canids, felids	(239)
		Ix. holocyclus Neumann, 1899 (Australian paralysis tick)	Australia and Malyasia	Canids, humans, sheep, mouse	(84, 230, 240)
		Ix. hirsti Hassall, 1931 (Hirst's marsupial tick)	Australia (mainly Tasmania)	Felids	(66)
	Rhipicentor	*R. nuttalli* Cooper & Robinson, 1908	Africa and Australia	Canids	(241)
	Rhipicephalus	*Rh. annulatus* Say, 1821 (Cattle tick)	Eurasia, Africa, and North America	Humans	(34)
		Rh. warburtoni Walker & Horak, 2000	South Africa	Goats, sheep	(96)
		Rh. tricuspis Dönitz, 1906	Africa	Ungulates	(34)
		Rh. simus Koch, 1844	Africa	Humans, sheep	(95, 97)
		Rh. sanguineus Latreille, 1806 (Brown dog tick)	Worldwide	Canids, humans	(22)
		Rh. punctatus Warburton, 1912	Africa	Ungulates	(95)
		Rh. praetextatus Gerstäcker, 1873	Africa	Humans	(34)
		Rh. evertsi Neumann, 1897 (Red-legged tick)	Africa	Sheep	(242)
		Rh. exophthalmos Keirans & Walker, 1993	Botswana	Goats, sheep, rabbits	(93)
		Rh. bursa Canestrini & Fanzago, 1878	Palearctic realm, South Africa, North America	Sheep	(94)
Argasidae	*Otobius*	*Ot. megnini* Dugès, 1883	Nearctic and Palearctic	Cats, horse, humans	(31, 118)

	(Ear tick)	realms, South Africa		
Ornithodoros	*Or. savignyi* Audouin, 1827	Africa, Asia	Camel, cattle, sheep	(114)
	(African eyed tampan)			
	Or. lahorensis Clifford, Kohls & Sonenshine, 1964	Africa and Central Asia	Cattle, sheep	(243)
	Or. capensis Neumann, 1901	Oceans and coastal	Seabirds	(110)
	(Seabird soft tick)	realm		
Argas	*Ar. africolumbae* Hoogstraal, et al., 1975	South Africa	Birds	(244)
	Ar. walkerae Kaiser & Hoogstraal, 1969	Africa	Birds	(125)
	(Southern Fowl tampan)			
	Ar. sanchezi Dugès, 1887	North America	Birds	(245)
	(Adobe tick)			
	Ar. robertsi Hoogstraal, Kaiser & Kohls, 1968	Australasia and Asia	Birds	(246)
	(Robert's Australian bird argasid)			
	Ar. reflexus Fabricius, 1794	Palearctic realm	Birds	(246)
	(African bird Argasid)			
	Ar. radiatus Railliet, 1893	North America	Birds	(247)
	(North American bird Argas)			
	Ar. persicus Oken, 1818	Palearctic	Birds	(248)
	(Bluebug)			
	Ar. monolakensis Schwan, Corwin & Brown, 1992	USA	Gulls	(249)
	Ar. miniatus Koch, 1844	America	Birds	(250)
	(Chicken tick)			
	Ar. arboreus Kaiser, Hoogstraal & Kohls, 1964	Egypt	Egrets	(251)
	(Egyptian heron rookery argasid)			

TABLE 2 Characteristics and comparison of the most common tick toxins, including tick paralysis toxin, sweating sickness toxin, tampan toxin, and ivotoxin. #: Mr: Molecular mass; pI: isoelectric point; PD: protease digestion *: the structure might be suspected.

Disease	Tick	Structure*	Mr#	pI#	Immunity	PD#	Mechanism of toxin
Sweating sickness, Mhlosinga, Magudu	*Hyalomma truncatum*	Three immunogenic and three non-immunogenic proteins (possible)	27 – 33 kDa 24 – 42 kDa (non)	-	Partial	-	-
Paralysis	*Rhipicephalus evertsi evertsi*	A trimeric complex resulting in toxin	~ 11 kDa; toxin ~ 68 kDa	6	Limited	Inactivate	Impair the conduction along peripheral nerve fibers
Paralysis	*Ixodes holocyclus*	Holocyclotoxin, three polypeptides of HT1 (with structure fold), HT2, and HT3	HT-1 ~ 6 kDa HT2/HT-3 ~ 5 kDa	8.86 4.5-5	Full	Resistant	Inhibit the release of acetylcholine from the neuromuscular junctions
Paralysis	*Argas walkerae*	An oligomer	Complexes within 43 - 115 kDa; 11 kDa at acid pH (cross-reactivity with the 4B12)	4.5	Partial	-	Inhibit the release of [^{3}H] glycine from brain synaptosomes
Tampan toxicosis	*Ornithodoros savignyi*	TSGPs	TSGP1, ~ 18 kDa; TSGP2, ~ 15 kDa; TSGP3, ~ 16 kDa; TSGP4, ~ 17 kDa	-	Partial	-	Ventricular tachycardia by TSGP2; Mobitz-type ventricular block by TSGP4
Paralysis	*Dermacentor andersoni*	Soluble polypeptides	36 – 43 kDa	-	Dose-dependent	-	Affect efferent pathway and suppression of acetylcholine release from the synapse
Protease inhibitors	*Amblyomma hebraeum*; *Rh. evertsi evertsi* *Rh. microplus* *Rh. decoloratus* *Hy. truncatum*	Four peptide fractions	~ 10 kDa ~ 5 – 6 kDa ~ 30 – 35 kDa ~ 40 kDa ~ 27 kDa	-	Limited	Resistant	1. Specific non-competitive fast-binding inhibition of trypsin (*Am. hebraeum*) 2. Competitive fast-binding inhibitor of trypsin (*Rh. evertsi evertsi*) 3. Competitive slow-binding inhibition of trypsin and fast tight-binding inhibition of chymotrypsin (*Rh. decoloratus* & *Hy. truncatum*) 4. Competitive slow-binding inhibitor of

TABLE 3 Differential diagnosis and clinical characteristics of tick paralysis and closely related disorders that may present with similar clinical features.

*: TP: tick paralysis; GBS: Guillain-Barré syndrome; Polio: poliomyelitis; ASCI: acute spinal cord injury; TM: transverse myelitis.

a: cerebrospinal fluid; b: white blood cells; c: magnetic resonance imaging; d: intravenous immunoglobulin; e: plasma exchange; f: intravenous.

Characteristics	**TP***	**GBS***	**Botulism***	**Polio***	**ASCI***	**TM***
Fever	Rare	Rare	Rare	Present	Absent	Variable
Pain	Rare	Rare	Absent	Present	Frequent	Frequent
Paresthesia	Rare	Present dominantly	Absent	Present	frequent	Frequent
Dilated pupils	Rare	Rare	Present	Absent	Absent	Absent
Gastrointestinal signs	Absent	Present	Present	Rare	Absent	Absent
CSF[a] protein	Normal	Elevated	Normal	Mildly elevated	Variable	Elevated
CSF WBC[b]s	Normal	Normal	Normal	Elevated	Variable	Elevated
MRI[c]	Normal	Generally normal	Normal	Abnormal	Abnormal	Abnormal
Spread	Tick bite	-	Eating food with toxins or bacterial spores	Fecal-oral route	-	-
Cause	Tick toxins	Problem with the immune system	*Clostridium botulinum* toxin	Poliovirus (types 1, 2, and 3)	Trauma to the spinal cord	Viral, bacterial, and fungal infections all included
Treatment	Tick removal, antibiotics	IVIg[d], PE[e], and breathing aids can ease symptoms	Antitoxin, antibiotics, severe with mechanical ventilation	No cure, only prevented by the Polio vaccine	Surgery, antibiotics, long-term hospitalization and rehabilitation	IV[f] steroids, PE, medication of virus
Prognosis	Generally complete recovery within hours to days	Recovery within months to years, some have lasting effects	Most patients recover, and a few die from breathing failure or secondary infections	Most people fully recover, some can be permanent paralysis or develop post-polio syndrome	Decrease or loss of sensation and organ function below the normal level	Varying lengths of time, pain or TM recurrent mainly depending on the cause of TM

TABLE S1. Reported human cases of tick paralysis worldwide and the associated tick species.

Tick species	Geographical Location	Patients	Clinical symptoms	Site of tick attachment	Reported exposure
Dermacentor andersoni Stiles	USA Princeton	11yrs* F*	Quadriplegia, muscles twitched, dyspnea, cardiac acceleration, rapid heart	Near the crown of the head	-
	USA Washington	8yrs F	Dizzy, gait staggering, eyelid swollen and inflammation, leukocytosis	The right axilla	-
	USA Carolina	8yrs -	Paralysis of arms, hands, back, neck, and legs, sensation changed	-	-
	USA	63yrs M	Bilateral paralysis of legs, numbness, absent reflexes	The right axilla	-
	Canada British Columbia	18months F	Ataxia, limbs paralysis, stopped breathing	The occipital region	Visiting tick popular region
	Canada	4yrs M	Unable to walk, tingled legs, losing eyesight in left eye, paralysis	The head	-
	Canada Alberta	20yrs M	Spotted fever, difficulty to lift arms, pain in arms	The left axilla (male tick)	Staying a tick popular region
	USA Idaho	6yrs F	Difficulty in standing and walking, pain in legs, absent patellar reflexes, ataxia in extremities	The scalp	Playing outside
	USA Colorado	4yrs F	Paralysis of all extremities	The scalp	-
	USA Colorado	49yrs M	Diplopia, numbness and prickling on fingers, unsteady gait, urinary retention, dysphagia, dyspnea	The groin	Visiting Rocky Mountain National Park
	USA	4yrs F	Weakness of legs, drowsy, unable to walk and swallow, respiratory distress, dysphagia	The right ear	-
	Canada Alberta	4yrs M	Irritable, clumsy, unsteady gait, incoordination of extremities, partial paralysis of lower extremities and developed to arms, diminished muscle strength and muscle tone, absent superficial reflexes	The scalp	

Canada Calgary	3yrs F	Dizzy, typhus-like rash, tingling in hands, inability to walk, headache, absent reflexes	The right occipital region	-
Canada British Columbia	3yrs M	Unable to stand, complete paralysis, acute bulbar poliomyelitis, absent abdominal reflexes, flaccid paralysis of neck and pharynx, absent cough and swallowing reflexes	The scalp	-
Canada British Columbia	66yrs F	Numbness of tongue and hands, tingled feet, inability to stand, diplopia, difficulty in swallowing, diminished DTRs	The pubic area	-
Canada British Columbia	5yrs F	Weakness of extremities, absent DTRs, unable to walk	The scalp	-
USA Oregon	3.5yrs F	Weakness, ataxia, difficulty in standing, sitting or walking	The hairline of the occipital region	Having a trip to Oregon
Canada British Columbia	8yrs F	Peculiar gait, absent reflexes, incoordinate respiration, abnormal abdominal muscles, elevated plasma creatine phosphokinase	The right temporal area of the scalp	-
USA Colorado	4yrs M*	Fever, inability to walk and feed himself, flaccid paralysis of all extremities, striking weakness, leukocytosis	The scalp	Camping in the mountains
USA Washington	6yrs F	Weak grip, bilateral weakness of extremities, decreased bilateral tendon reflexes, and mild facial weakness	The scalp	Live close to rivers or creeks, or experience mountains or parks
USA Washington	3yrs (n=5); 6yrs (n=1); 2yrs (n=1); 4yrs	Weak grip, bilateral weakness of extremities, decreased bilateral tendon reflexes, and mild facial weakness	The scalp or behind the ear	Live close to rivers or creeks, or experience mountains or parks

		(n=3); 5yrs (n=1); 82yrs (n=1); 54yrs (n=1)			
	USA Los Angeles	1.5yrs F	Deteriorated neurologic condition, distal lower extremity areflexia, weakness, drooling, absent DTRs* of upper and lower extremities	Below the occiput	vacationing on a dude ranch
	USA Colorado	6yrs F	bilateral lower extremity weakness, tingling sensation, ophthalmoplegia, dysarthria, lower nerve conduction, and areflexia	Hairline	Hiking in mountains
	Canada British Columbia	83yrs M	Diplopia, dysarthria, limb weakness, dysmetria, areflexia	Mid-back	Working in a cabin in the Thompson-Nicola region
	Canada British Columbia	2yrs F	Lower/absent extremity reflexes, mild generalized hypotonia, and weakness	Left occipital scalp	-
	Canada British Columbia	6yrs M	Ataxia, extremity paresthesia	Scalp	Living on a farm
Dermacentor variabilis Say	Canada British Columbia	2 infants	Convulsions	Necks	-
	Canada British Columbia	5yrs -	Convulsions	The back of the head	-
	USA Washington	22yrs F	Complete motor paralysis of extremities	The skin over the coccyx	-
	Canada British Columbia	4yrs -	Complete paralysis of extremities	The nape of the neck	-
	Canada British Columbia	4yrs -	Almost complete paralysis	Neck	-
	USA Washington	4yrs F	Loss of appetite, abdominal pain, great restlessness, severe pains in the knees, inability of the lower extremities	The scalp above the left ear	Living in a small "jack" pine grove
	USA Idaho	7yrs M	The inability of lower extremities, unable to stand, subnormal temperature, fast and thready pulse, vomiting	The occipital region	Exposure in the woods

USA Idaho	3.5yrs M	Unable to walk, generalized weakness of muscles and increased over time, (atypical infantile paralysis was suspected)	The head	Previously infested by wood ticks during spring
USA Idaho	5yrs F	Unable to stand or walk, pain in legs, can't control muscles	The occiput	Walking through the forest
USA Idaho	9yrs F	Unable to stand, dizzy, vomit	The scalp	-
USA Washington	4yrs F	No appetite, drowsy	The scalp	-
USA Montana	-	-	The hair	-
USA Montana	5yrs F	All extremities paralysis, absent reflexes, unable to talk and swallow	The scalp	-
USA Georgia	9yrs F	Legs paralysis, arms weakness, thick speech, nystagmus	The scalp	-
USA Georgia	7yrs F	Ataxia, no sensation of ear, eyes, nose, and throat, unsteady walk	The scalp	-
USA Carolina	4yrs F	Ataxia, weakness, general malaise, unable to stand	The scalp	-
USA New York	3yrs F	Weakness in legs, unable to walk or stand, ataxic gait, low spirits, no DTRs in extremities	Behind the right ear	Been a suburb
USA Texas	2.5yrs F	Fever, unable to move	The posterior cervical glands on the left side	-
USA Kentucky	4yrs F	Weakness progressed to complete paralysis of all extremities, diminished reflexes, ankle,	The scalp in the occipital region	Having a dog infested with many ticks

		and knee jerks, miliaria rubra		
USA	9yrs F	Onset or irritable and progressed ataxia, weakness in legs rapidly progressed to other parts, ascending flaccid paralysis	The scalp	Visiting a rural area
USA Tennessee	4yrs F	Uncoordinated extremities, absent superficial reflexes	The left occipital region	-
USA New York	3.5yrs F	Headache, pain in the right leg, unable to walk, absent DTRs, diminished reflexes	The left frontotemporal region	Petting a dog
USA Florida	5.5yrs F	Unable to stand or walk, large, tender posterior cervical lymph nodes bilaterally, left parietal region of the scalp	The left parietal region of the scalp	Having a dog infesting ticks severely
USA Georgia	7yrs F	Unsteady gait and inability to walk, generalized paralysis, weak grip	The scalp (a male and a female)	-
USA Wisconsin	3yrs M	Afebrile, mild bilateral otitis media, slight truncal titubation, moderate flaccid weakness of lower extremities, absent DTRs, mild reflexes in arms	The scalp	Exposed on woods near rural
USA Georgia	6yrs F	Truncal instability, ataxic gait, diminished reflexes, hypoventilation	The left parietal area of the scalp	-
USA New Jersey	5yrs F	Diplopia and progressive weakness	Left parietal scalp	Possibly petting dogs'
USA New Jersey	7yrs F	Ascending paralysis and ataxia	The posterior scalp	Possibly petting dogs'
USA North Carolina	5yrs F	Unsteady gait progressed to inability to walk, absent DTRs	The occipital region of the scalp	-
USA Pennsylvania	4yrs F	Progressive lower extremity weakness, compressive thoracic arachnoid cyst, respiratory depression	Behind the left ear	-
USA New York	2yrs F	Rapidly progressing ascending paralysis, hyporeflexia	The right retroauricular	Living in the forest and petting dogs

				area	
	USA Mississippi	84yrs M	Subacute ataxia, bulbar palsy, unilateral weakness, and absent DTRs	Gluteal fold	Traveling coastal Mississippi
	USA Tennessee	82yrs M	Dizziness, nausea, and vomiting upon standing up, slight weakness in multiple muscles, ataxic gait	The right anterior thigh (male tick)	Gardening in parks
	USA Tennessee	5yrs F	Fever, progressive gait difficulty, lower reflexes, diplopia	Scalp	-
	USA Los Angeles	88yrs M	Gait instability, progressive truncal ataxia	Left lateral chest	On a road trip, arriving in Coachella Valley
	USA Florida	4yrs F	Diffuse weakness of all extremities, unable to stand, absent sense	Posterior aspect of the scalp	-
Dermacentor marginatus	Iran Tehran	48yrs F	Facial edema, headache, head itching, swelling behind the ear, disequilibrium, fever	The vertex region of the skull	Working to the mountainous countryside
	Turkey Niğde	71yrs M	Itching and localized redness	The medial aspect of the right lower eyelid	Living in countryside
Dermacentor sp.	Malaysia	65yrs F	Right facial palsy, haemorrhagia in the right tympanic membrane	The right ear (nymph)	-
	USA Pennsylvania	4yrs F	Areflexic in lower extremities, unsteady gait, Increasing respiratory distress, unable to move upper extremities	The superior retro auricular scalp	-
Ixodes scapularis	USA Mississippi	11yrs M	Numbness and tingling over the left parietal area, movement difficulty, ataxia	Left occiput	Camping in the forest
Ixodes tancitarius	Mexico Valle de Bravo	- M	Paralysis symptoms	The hand	Working in Valle
Ixodes rubicundus	Republic of	4yrs -	Lossing sensory of arms and hands, weakness	The axilla	-

	Sierra Leone		of arm muscle		
Ixodes holocyclus	Australia	13months F	Motor paralysis, fever	Behind left ear	Hugged by a man who walked through the jungle
	Australia Sydney	35yrs F	Pain and weakness of legs, blurring vision	The inner side of the right labium majus	-
	Australia Sydney	2months F	Severely paralyzed in all extremities, difficulty in swallowing, absent jerk-knees and reflexes, fever, respiratory distress	The right breast	-
	Australia Sydney	7yrs M	Unsteady gait, double vision, difficulty in swallowing, breathing distress, absent reflexes and knee jerks, fever	Behind the ear and on the occipital region	-
	Australia Sydney	Nearly 6yrs F	Lump on head, unsteady gait, flaccid paralysis, absent knee jerks, breathing failure	The scalp	Having a picnic
	Australia New South Wales	45yrs M	Left upper limb discomfort	The lateral aspect of the left axilla	Bushwalk
	Australia Queensland	67yrs M	Left-sided facial weakness	The posterior superior aspect of the lateral EAC	Working outdoor
	Australia New South Wales	57yrs M	Right facial nerve palsy	Right parietal scalp	Hiking on the Central Coast
	Australia Sydney Auburn	16 months M	Dilated pupils, muscle paresis, swallow hard, stertorous breath	The scalp	-
	Australia Erina Shire Gosford	2yrs M	-	-	-
	Australia Matcham	10m M	-	The left carotid region	-
	Australia Queensland	19mon F	Serous nasal discharge and a slight cough, weakness, respiratory depression	The left ear	Camping in the rainforest area
	Australia Goomeri	3yrs F	Unsteady gait	The left ear	-

Australia Yandina	2yrs F	Irritable, weakness, and paralysis	The scalp	-
Australia Brisbane	3yrs F	Cough, weak, and stridor	The scalp	-
Australia Brisbane	6yrs F	Left-sided facial palsy	The left ear	-
Australia Bunya	3yrs M	Unsteady gaits, weakness of lower extremities	The scalp	Staying in the mountains
Australia Brisbane	3yrs F	Weakness, glassy eyes	The scalp	-
Australia	22yrs F	A firm lump	The scalp	-
Australia Brisbane	63yrs M	-	Eyelids (larva)	Living in a rural area
Japan Yamaguchi	59yrs M	Sore throat and nasal discharge, fever, lethargy, edema, and sensory loss around eschar on the scalp	Scalp	Staying at a farm
Australia	48yrs M	Lethargy, myalgia, unsteadiness, urticarial lesions, generalized lymphadenopathy	The scalp, face, neck, back, and limbs (with 44	Hiking through bushes and creeks

				ticks)	
	England London	- F	Small black lump' in the groin area	The lower leg	Traveling to the coast
	Singapore	63yrs F	Swelling and pain over the right temporal region, febrile, nerve weakness	Temporal scalp	Visiting a house near scrubland
	Turkey Aydin	45yrs M	Facial palsy	External ear canal	-
Ixodes ricinus or *Ixodes holocyclus*	Australia	4.5yrs F	Absent appetite, vomiting, nearly unconscious, absent reflexes, weakness of muscles, the knee jerks were absent	The scapula of the right shoulder	-
Amblyomma americanum	America	young M	Ataxia, diminished reflexes, vomiting	- (2 nymphs)	-
	USA Florida	38yrs M	Complete paralysis of legs, decreased reflexes of arms, missing sensory from legs to inguinal areas	The scalp, the right thigh, and the left axilla	Working and living in the forest
Amblyomma maculatum	USA Louisiana	7yrs F	Generalized, symmetrical flaccid paresis, weakness, lethargy, ataxia, diminished reflexes, adiadokokinesia	The scalp	-
	Mexico Jalisco	22yrs M	Flaccid paraplegia, areflexia, dyspnea, difficulty in breathing	Feet	Living in a rural area and contacting cattle with ticks
	Canada	3yrs M	Ataxia, extremities weakness, hyporeflexia	Back (over 50 ticks)	Visiting rural for 1 week
Amblyomma spp.	Brazil Minas Gerais State	28yrs M	Febrile, respiratory depression, erythematous papular lesions, mild bilateral ptosis, intense gastralgia, nausea, profuse sweating, malaise,	Right inguino-femoral region	Contacting animals with ticks

			muscle weakness, blurred vision, severe bilateral ptosis, dry cough, headache, and dysphagia		
	Thailand	5yrs F	acute unilateral right complete facial paralysis and right otalgia	Right tympanic membrane	Traveling to the park
Haemaphysalis cinnabarina	Canada British Columbia	~10yrs F	knee jerks absent, consciousness unimpaired, quickened pulse, paralysis of legs and abdominal muscles developed, respiratory depression	The scalp	-
Haemaphysalis sp.	India	Newborn M	lethargy and reduced activity, no appetite, yellowish discoloration of extremities, poor respiratory, lower DTRs, and generalized hypotonia	Under the chin	Living ticks popular region
Hyalomma marginatum marginatum	Turkey	3yrs F	Facial palsy (HB* IV), ear pain, unable to close eyelids, lisping, paresthesia on the left face	The external ear canal	-
	Turkey	47yrs M	Localized facial palsy (HB III)	The ear	-
Hyalomma truncatum	South Africa	16yrs M	Inability to move hands and fingers, diminished elbow flexion, paralyzed long flexors of fingers, unfunctional muscles	The right axilla (male tick)	-
	South Africa Pretoria	21yrs M	Burning pain from the right axilla o elbow, and developing to the entire arm, weakness in lifting arm, weakness of arm muscles, fever	The right axilla (male tick)	Working in farms
	South Africa Pretoria	22yrs M	Anaesthesia of a large area of arms, motor weakness	The axilla	Working in farms
	South Africa	23yrs M	Pain in ear	The ear	Staying in forest
Hyalomma sp.	Turkey Central Anatolia	33yrs M	Facial palsy (HB II), onset pain of the right ear, unable to close eyelid, afebrile	The right external	-

				auditory canal	
Dermacemor auratus + *Hyalomma marginatum isaaci*	Sri Lanka	9mons-67yrs (29 patients, 12 M, 17 F) (2 infants, 15 kids aged < 12yrs, 12 adults)	Sudden onset of acute ear pain, edematous ear canal, enlarged and tender lymph nodes	The ears (most tympanic membrane) (29 nymphs of *D. auratus*, 1 unfed male adult of *H. m. Isaac*i)	-
Rhipicephalus simus	Somalia	4yrs M	Weakness in legs, diminished reflexes, absent knee jerks	The leg	-
	Somalia	6yrs F	Tonic and clonic convulsions of the neck and face muscles, tachycardia, and tachypnea	The neck	-
	Spain	-	-	-	-
	Greece	-	-	-	-
	South Africa Transvaal	Adult M	Headache for 3 weeks, got weaker, inability to walk and swallow, drowsy, generalized weakness, absent abdominal reflexes	The lower abdomen	-
Rhipicephalus sp.	Ethiopia	7yrs M	Left ear pain, left-sided facial swelling, facial pain and weakness, facial palsy (HB IV)	The ear	Closely contact with cattle
Otobius megnini	South Africa	16months M	Pain in knees, refusing to walk, weakness of the right leg, respiration difficulty, unable to sit or lift head	The right ear canal (nymph tick)	-
Possible *Rhipicephalus sanguineus*, *Hyalomma dromedarii*, *H. anatolicum excavatum*, and *Haemaphysalis*	Egypt Giza Governorate	- (4 kids unrecorded ages)	Diarrhea, vomiting, irritability, and mild intermittent fever, and the smaller two experienced nervous manifestations	-	

sulcata					
Unclassified	Canada British Columbia	2 kids	Suddenly paralysis	-	-
	Canada British Columbia	-	Symptoms of acute ascending paralysis	The nape of the neck	-
	Canada British Columbia	-	Symptoms of acute ascending paralysis	The right temple	-
	Canada British Columbia	4yrs F	Unable to stand and control leg muscles	The nape of the neck	-
	Canada British Columbia	3.5yrs -	Almost complete paralysis of legs, missing reflexes	The neck	-
	Canada British Columbia	-	Weaker	The nape of the neck	-
	Canada British Columbia	3yrs F	Complete paralyzed legs and missing reflexes, paresis	The nape of the neck	-
	Canada British Columbia	3~4yrs	Legs paralysis and absence of reflexes	The neck	-
	Canada British Columbia	2 kids	Symptoms of paralysis	-	-
	USA Ashcroft	-	Paralysis	-	-
	Canada Bella Coola	-	Paralysis	-	-
	Canada British Columbia	5yrs F	Unable to stand	The head	-
	Canada British Columbia	6yrs F	Flaccid paralysis of all extremities, aphonia, dysphagia, labored breathing	Skin (2 ticks)	-
	Canada British Columbia	10yrs F	Complete flaccid paralysis	-	-
	Canada British Columbia	9 cases	-	-	-
	USA Florida	24yrs M	Sudden onset of pain in the lower back, severe weakness and developed to paralysis of all extremities, difficulty in opening mouth and swallowing, muscle deglutition, labored respirations	-	Fishing and across the forest
	Canada British Columbia	2yrs M	Unable to walk, restless, agitated, generalized ataxia, lethargy, poor sucking reflexes, difficulty to swallow	The scalp	-

USA	2yrs M	Paralysis	-	-
USA	2yrs M	Moribund suddenly	The occiput	-
USA	2yrs F	Tired legs, difficulty to stand	The head	-
USA	11yrs F	Bulbar paralysis	-	-
Israel	19yrs M	Complete paralysis of the right arm	The right axilla (male tick)	-
Canada British Columbia	4yrs M	Flaccid paralysis from the hips down, weak arms, hyperesthesia, conditions deteriorated	The left axilla	-
Canada British Columbia	3yrs F	Slight paralysis of the right leg onset, conditions stuporous 24 hours later, and legs and arms were entirely paralysis, unable to swallow, rapid pulse, fever	The scalp	-
Canada British Columbia	28yrs M	Rapid pulse, rash, fever, missing sensation from the hip joint in right leg, paralysis in the left leg	The left leg	-
USA Montana	2yrs F	Unable to walk and stand, rapid pulse, absent DTRs	The nape of the neck (a male and a female tick)	-
USA Montana	5yrs F	Unable to walk for at least 48 hours, missing reflexes	The scalp	-
Canada British Columbia	8yrs F	General paralysis, rapid pulse, febrile	The arm	-
Canada British Columbia	6yrs F	General paralysis of the whole body	The head	-
Canada British Columbia	5yrs F	Unable to walk and stand	-	-
Canada British	8yrs M	Legs numbness, unable to stand and move,	The skull	-

Columbia		sleepy and dull		
Canada British Columbia	40yrs M	Difficulty to speak, incoordination, weak muscle strength, absent knee jerks	The back	-
USA Wyoming	10yrs M	Double vision, unable to lift legs and head, ptosis of left upper eyelid, absent DTRs	The back	-
Australia Melbourne	45yrs M	Facial paralysis	The auditory canal	Fishing nearby lake
Australia New South Wales	2yrs -	Facial paralysis lasting 2 weeks	The auditory canal	-
Australia Sydney	Nearly 2yrs F	Unsteady gait, weakness of the right leg, absent reflexes and muscle strength, conditions worsen	The left occipital region	-
Australia Sydney	10yrs M	Weakness in the right arm, paralysis of the dorsiflexor, paresis of arms and legs	The right axilla	-
Australia Sydney	7yrs F	Fever, pain in the right head, paralysis of the right frontalis and orbicularis oculi muscles, absent DTRs	The right temple	-
Australia Sydney	10yrs M	Pain in legs, weakness of legs, paresis of all muscles of the legs, sluggish knee jerks	The scalp	Living in suburb
Australia	11yrs M	Giddy, generalized weakness, facial palsy	The ear	Walking through bushes
Canada British Columbia	3yrs	Their body was limp and muscular weakness	-	-
USA Washington	Nearly 2yrs M	Difficulty in walking and progressed to unable to walk and control arms	The scalp in the right temporal region	Spending several days at a nearby mountain lake
USA Idaho	2yrs F	Sudden inability, unable to stand, complete paralysis of legs	The scalp	-
USA Idaho	-	Vomiting, fever, prostration, loss of appetite, and other general symptoms of an intoxication	-	-
USA Washington	6yrs F	No appetite, unsteady walk, dilated pupils	The scalp	-

Australia Bellingen	3yrs M	A great deal of edematous swelling over both supraorbital margins	The center of the forehead	-
Australia Eden	13months F	Paralysis, respiration depression	Behind the left ear	-
Australia Mullumbimby	13~14yrs M	Semi-comatose, legs and muscles paralysis	Membrana tympani	
Australia Wagonga	3yrs F	High fever, convulsive, muscle paresis	The chest on the left side is nearly under the arm	-
Australia Eastwood	3yrs M	High fever, extreme cardiac weakness, pulse softness, and threading	-	-
USA Carolina	Adult F	Tingling and numbness in the lower and upper extremities, unable to stand, difficulty in talking and swallowing, decreased muscle strength, uncoordinated movement	The scalp	Visiting the farm
France	48yrs M	Weakness and pain in right arm	-	-
USA New York	4yrs -	Flaccid paralysis of all extremities, advanced to cranial nerves severely, respiratory depression	-	-
USA Georgia	4yrs F	Paralyzed legs	The scalp	-
USA Kentucky	6months M	Generalized flaccid weakness	Scalp	Visiting areas where ticks are prevalent
USA	10yrs F	Inability to walk, fever, nausea, leg weakness developed to flaccid paralysis, increased ataxia, absence of reflexes	The scalp	Living in rural region
Australia Sydney	3yrs F	Unsteady gait	The scalp	-
USA Philadelphia	5yrs F	Ascending paralysis, symmetric flaccid paralysis, mild bilateral facial weakness, absent reflexes in the lower extremities, and diminished in the upper extremities	The right ear	-
USA	1.5yrs	Difficulty in walking, swallowing, and	Behind the left	-

	M	drinking, flaccid weakness of all extremities, leg paralysis, incoordination of arms	ear	
Canada British Columbia	1.5yrs F	General weakness, drowsiness, irritability, and right-leg paresis	-	-
Canada British Columbia	5yrs F	Progressive weakness in legs, unable to stand, transitory weakness, able to raise legs but unable to control their directions	The scalp near the vertex	-
Canada British Columbia	5yrs F	Unable to stand	The scalp	-
-	2yrs F	Complete paralysis suddenly	The head	-
--	4yrs F	Weakness of extremities, flaccid paralysis, breathing distress, absent reflexes, vomiting	-	-
USA California	3yrs F	Paralysis	-	Having a trip to Oregon
USA	5.5yrs F	Unable to stand muscle weakness, severe ataxia with incoordination, dysmetria, intention tremor, hypotonia, and absent muscle stretch reflexes	Behind the right ear lobe	-
USA Mississippi	5yrs M	Continuous ataxia, uncoordinated extremities, pronounced weakness with absent DTRs in lower extremities	The left temporal area behind the ear	-
USA Mississippi	3yrs F	Walking difficulty, profound muscular weakness, absent DTRs in lower extremities	The scalp of the occipital region	-
USA Florida	6yrs F	Ascending paralysis, afebrile, unable to walk or control legs, extremely weak muscles, absent reflexes	The scalp	-
USA	2yrs F	Fever, unable to walk, DTRs decreased, slight unsteadiness	Behind the right ear	-
USA Washington	2yrs (n=4) 4yrs (n=5) 8yrs (n=1) 54yrs	Weak grip, bilateral weakness of extremities, decreased bilateral tendon reflexes, and mild facial weakness	The scalp, behind ear, and groin	Live close to rivers or creeks, or experience mountains or parks

	(n=1) 1yrs (n=1) 3yrs (n=3) 51yrs (n=1) 76yrs (n=1) 5yrs (n=1) 7yrs (n=1)			
USA	5yrs F	Dizzy, absent and muscle strength DTR in the lower extremities, unable to perform Romberg test	Behind left ear	-
USA Carolina	7yrs F	Ataxic, unsteady, and wide-based gait, weakness bilaterally in legs, absent reflexes, dysmetria	On the left lateral scalp	Living in rural and petting outdoor dogs
USA Carolina	5yrs F	Minimal non-fatigue ptosis, lateral rectus muscle paresis bilaterally, neck flexor and extensor weakness, absent stretch reflexes	The scalp behind her ears (2 ticks)	Living in a rural area
USA Los Angels	5yrs F	Generalized weakness	The scalp	-
USA Colorado	86yrs M	Difficulty in standing and moving, deteriorated weakness, absence of DTRs	The back	Petting a dog often outside
USA Colorado	78yrs F	General weakness, difficulty in walking, facial weakness, slurred speech, confusion, decreased DTRs	The neck	Hiking or walking outside every day
USA Colorado	58yrs M	Tingling sensation in hands, perioral numbness, unable to stand	The back	Working in the yard and outdoor recreational activities
Germany	47yrs M	Diplopia, blurred vision, dizziness, difficulty	The right leg	-

		walking, nerve palsy on the right-hand side, positive Romberg test		
Turkey	66yrs M	Weakness of the left arm, decreased DTRs, reduced muscle strength	The left supraclavicular area	-
Malaysia	5yrs F	Left otalgia, left-sided facial asymmetry (HB IV)	Posterior canal wall near the tympanic membrane	-
Malaysia	1yrs F	High-grade fever, rigor, irritable behaviors, right-sided asymmetry (HB IV), and the weakened right angle of the mouth	The right ear canal	-
Malaysia	78yrs F	Generalized lethargy and weakness, slight right-sided facial weakness (HB II)	The tympanic membrane of the right ear canal	-
USA Florida	42yrs M	Flu-like symptoms, fatigue, and developed to generalized weakness	The right occipital area	Hiking in Colorado
India	3yrs M	The angle of the mouth towards the right side, saliva drooling, pain in the left ear, left-sided isolated lower motor neuron facial palsy (HB IV), inflammation of the left ear canal	The tympanic membrane of the left ear	Living in rural
Malaysia	64yrs F	Sudden onset of facial asymmetry, facial nerve palsy (HB III)	The left ear	-
Ethiopia Shewa	60yrs M	The right arm flaccid monoplegia	The right arm	Living in rural
USA Georgia	7yrs F	Acute onset proximal muscle weakness	The scalp	-
Malaysia	73yrs F	Spinning sensation, nausea, vomiting, and mild headache, developed left-sided facial asymmetry and facial weakness, reduced	The ear canals	-

			hearing		
	Malaysia	61yrs F	Left ear pain associated with foreign body sensation and left facial weakness, left facial asymmetry (HB II), unable to control eye muscles	The tympanic membrane of the left ear	Contacting with a cat
	-	4yrs F	Facial nerve paralysis left otalgia, left-sided facial asymmetry	The ear	-
	USA Mississippi	2yrs F	Lower extremities weakness, unsteady gait, truncal ataxia	The ear	Living in rural
	USA	4yrs M	Progressive weakness of all extremities, afebrile, absent DTRs, diffuse hypotonia	The scalp	-
	Malaysia	7yrs F	Left otalgia left facial asymmetry	The ear	-
	Malaysia	71yrs F	Sudden-onset giddiness and left facial nerve palsy with progressively reduced hearing bilateral for a week duration, profound sensorineural hearing loss with right mild-to-moderate sensorineural hearing loss	External ear	-
	Malaysia	3yrs M	Ear pain progressed, right facial asymmetry, reduced hearing, unable to close eyes, absence of the right nasolabial fold	The ear tympanic membrane	-
	Turkey	10yrs F	Pain and itching in the right ear, unable to close the right eye, facial palsy (HB VI)	The ear	-

14 *yrs: years old; F: female; M: male; CPK: plasma creatine phosphokinase; DTR: deep tendon reflexes; HB:

15 House-Brackmann grade
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TABLE S2. Summary of human tick paralysis cases by country, including the number, frequency, and associated tick species.

Tick species	Number	Realms	Frequency
Amblyomma americanum	2	USA	2
Amblyomma maculatum	3	Mexico	1
		Canada	1
		USA	1
Amblyomma spp.	2	Brazil	1
		Thailand	1
Dermacentor andersoni Stiles	38	USA	26
		Canada	12
Dermacentor variabilis Say	37	USA	32
		Canada	5
Dermacentor marginatus	2	Iran	1
		Turkey	1
Dermacentor sp.	2	USA	1
		Malaysia	1
Haemaphysalis cinnabarina	1	Canada	1
Haemaphysalis sp.	1	India	1
Hyalomma marginatum marginatum	2	Turkey	2
Hyalomma sp.	1	Turkey	1
Hyalomma truncatum	3	South Africa	3
Hyalomma marginatum isaaci + *Dermacemor auratus*	29	Sri Lanka	29
Ixodes rubicundus	1	Republic of Sierra Leone	1
Ixodes scapularis	1	USA	1
Ixodes tancitarius	1	Mexico	1
Ixodes holocyclus	25	Australia	21
		Japan	1
		Singapore	1
		England	1
		Turkey	1
Ixodes ricinus or *I. holocyclus*	1	Australia	1
Otobius megnini	1	Europe	1
Rhipicephalus simus	5	Somalia	2
		Greece	1
		Spain	1
		South Africa	1
Rhipicephalus sp.	1	Ethiopia	1

Possible *Rhipicephalus sanguineus*, *Hyalomma dromedarii*, *H. anatolicum excavatum*, and *Haemaphysalis sulcata*	4	Egypt	4
Unknown	125	USA	56
		Canada	37
		Australia	13
		Malaysia	9
		France	1
		Germany	1
		India	1
		Ethiopia	1
		Israel	1
		Turkey	2
		unknown	3

Number: The total number of cases caused by the corresponding tick; Frequency: The clinical case reported in the country by the corresponding tick.

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