Estimated Illness Linked to Animal Contact for 7 Pathogens, United States, 2019

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Diseases transmitted through direct or indirect animal contact are a public health concern in the United States. Improvements in laboratory detection methods and illness estimates warrant an update of illness estimates linked to animal contact. We used surveillance data and other sources to estimate the annual number of US illnesses, hospitalizations, and deaths for 7 pathogens. We adjusted for underreporting and underdiagnosis by using established methods. *Campylobacter*, *Cryptosporidium*, Shiga toxin–producing *Escherichia coli*, *Listeria monocytogenes*, nontyphoidal *Salmonella*, and non-pestis *Yersinia* caused an estimated 995,022 (90% credible interval [CrI] 476,251–1,833,238) animal contact illnesses around 2019. Together with *Toxoplasma gondii*, those illnesses caused an estimated 9,592 (90% CrI 5,603–15,557) hospitalizations and 236 (90% CrI 119–411) deaths in the United States. Increased awareness among health professionals, government officials, industry partners, and the public about animal contact as a mode of disease transmission can aid prevention efforts and enhance surveillance.

Zoonotic diseases are spread between animals and humans, causing an estimated 60% of infectious diseases and 75% of emerging infectious diseases globally (*1*). Zoonotic diseases spread through direct or indirect animal contact, or foodborne, waterborne, and vectorborne transmission (*2*). In this analysis, we focus on direct (e.g., touching animals) and indirect (e.g., contact with contaminated objects or environments) animal contact. An estimate published in 2012 attributed 14% of US enteric illnesses to animal contact, corresponding to an estimated 450,000 illnesses, 5,000 hospitalizations, and 70 deaths annually based on circa 2006 data (*3*).

Campylobacter outbreaks have been attributed to pet store puppies and raccoons from a wildlife rehabilitation center (*4*,*5*). *Cryptosporidium* (*6*,*7*) and Shiga toxin–producing *Escherichia coli* (STEC) (*8*–*12*) outbreaks have been linked to farm animals, and nontyphoidal *Salmonella* outbreaks have been linked to pet hedgehogs (*13*,*14*), guinea pigs (*15*,*16*), live poultry (*17*,*18*), wild songbirds (*19*), dairy calves (*20*), and various reptiles and amphibians (*21*–*29*).

Toxoplasma gondii is an important parasitic pathogen; felids are definitive hosts (*30*). Transmission can occur through indirect animal contact through ingestion of environmentally matured oocysts from contaminated cat feces or soil (*31*). The prevalence of *T. gondii* infection in the United States is relatively low (*32*); however, serious health consequences linked to pregnancy and immune compromise underscore a need to better understand the proportions of hospitalizations and deaths that may be linked to animal contact (*31*).

The Centers for Disease Control and Prevention recently updated the estimates for 7 major foodborne pathogens by using improved methods (*33*). By using this methodology, we updated the estimated annual prevalence of US illnesses linked to animal contact for the pathogens from the previous animal contact estimates (*Campylobacter*, *Cryptosporidium*, *Listeria monocytogenes*, nontyphoidal *Salmonella*, STEC, and *Yersinia*). We expanded the *Yersinia* estimates to include non-pestis *Yersinia* spp. beyond *Y. enterocolitica* and added estimates of hospitalizations and deaths attributable to *T. gondii*.

Methods

We estimated the annual prevalence of illnesses, hospitalizations, and deaths linked to animal contact

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DOI: https://doi.org/10.3201/eid3212.260787

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for *Campylobacter*, *Cryptosporidium*, *L. monocytogenes*, nontyphoidal *Salmonella*, STEC, and non-pestis *Yersinia* and of hospitalizations and deaths for *T. gondii* by adapting the modeling approach for the recent estimate of foodborne pathogens to account for the proportion attributable to animal contact (*33*). We did not estimate *T. gondii* infections because most were asymptomatic and the mild illnesses were unquantified. We based estimates on the average US population during 2017–2019. We described model inputs by pathogen (Appendix Table 1, https://wwwnc.cdc.gov/EID/article/32/12/26-0787-App1.pdf).

Estimating Illnesses

Laboratory-Confirmed Illnesses Reported to Surveillance Systems

The Foodborne Diseases Active Surveillance Network (FoodNet) conducts active surveillance of laboratory-confirmed infections in 10 sites that are the historic catchment area and represent 15% of the US population (*34*). We used 2017–2019 data from FoodNet for *Campylobacter*, nontyphoidal *Salmonella*, STEC, and non-pestis *Yersinia*. We used 2016–2019 data from the National Notifiable Diseases Surveillance System (NNDSS) for *Cryptosporidium* and 2017–2019 data from the *Listeria* Initiative for invasive *L. monocytogenes*; both are national passive surveillance systems (*35*,*36*). We defined invasive *L. monocytogenes* as isolation of *Listeria* from a specimen collected from a normally sterile site (cerebrospinal fluid or blood) or product of conception (for pregnancy-associated illnesses, products were amniotic fluid, placenta, chorion, or umbilical cord). We counted illness in mother and infant as separate if *L. monocytogenes* were isolated from that person's specimen or if the mother or infant of a person with listeriosis had symptoms (e.g., vomiting, fever, or diarrhea) (*33*). We proportionally distributed infants with unknown outcomes according to infants with known outcomes.

Estimating Total Domestically Acquired Animal Contact Illnesses

The methods were reported in the recent estimate of foodborne pathogens and applied to this analysis (*33*). In brief, we estimated illnesses as follows.

Projected US Laboratory-Confirmed Illnesses

We used laboratory-confirmed illnesses caused by a pathogen by year (by site and year for FoodNet pathogens) to project US illnesses. We multiplied the ratio of the average US population during 2017–2019 to the population in the surveillance area in that year.

Accounting for Underreporting

We assumed no underreporting in FoodNet active surveillance (i.e., all diagnosed illnesses in the FoodNet surveillance area were reported to public health authorities). For NNDSS (*Cryptosporidium*), we adjusted for underreporting by using a multiplier estimated by comparing FoodNet-projected annual laboratory-confirmed illnesses with annual illnesses reported to NNDSS for parasites in both surveillance systems (i.e., *Cryptosporidium* and *Cyclospora*). Because FoodNet stopped collecting *Cryptosporidium* data after 2017, we used 2016–2017 *Cryptosporidium* data and 2016–2019 *Cyclospora* data from FoodNet and NNDSS. For *L. monocytogenes*, we did not adjust for underreporting because we assumed all diagnosed cases were reported given the severity of listeriosis.

Adjusting for Underdiagnosis

We created underdiagnosis multipliers to account for underascertainment from care-seeking, specimen submission, and laboratory reporting. The 2018–2019 FoodNet Population Survey randomly selected residents from the 10 FoodNet sites, collecting information on symptoms and medical care sought for an acute diarrheal illness (*37*). By using age, sex, race/ethnicity, bloody diarrhea, and fever, Bayesian regression models predicted the probabilities of seeking medical care among persons reporting an acute diarrheal illness and of submitting a specimen for laboratory testing among persons who sought medical care. We applied those models to each person with a laboratory-confirmed infection in the 2017–2019 FoodNet surveillance data to inflate the count to the reciprocals of the probabilities. Because NNDSS did not collect symptom information, we used models with age, sex, and race/ethnicity to predict those probabilities for persons with a *Cryptosporidium* infection.

The proportion of laboratories routinely testing for each pathogen, except for *Listeria*, was available from the FoodNet Laboratory Survey. We estimated test sensitivity for *Campylobacter*, non-typhoidal *Salmonella*, STEC, and non-pestis *Yersinia*. Most culture-independent diagnostic tests (CIDTs) are PCR-based and highly sensitive (*38*); therefore, we assumed 100% CIDT sensitivity. We estimated culture sensitivity using CIDT and reflex culture (cultures performed after a positive CIDT) data from FoodNet. We assumed the proportion of positive reflex culture as the lower parameter of the program evaluation and review technique (PERT) distribution. For *Cryptosporidium*, estimated test sensitivity determined by using a combination of conventional (microscopic analysis and antigen tests) was 74% and test sensitivity

determined by using molecular tests with proportional weighted random sampling was 26% (*38*–*40*). For listeriosis, we assumed high probabilities of medical care seeking, specimen submission, and testing. We based test sensitivity on a published study of blood culture sensitivity (*41*).

Estimating Domestically Acquired Illnesses

We used FoodNet, the *Listeria* Initiative, and NNDSS data to estimate the percentage of illnesses acquired while traveling outside the United States. We considered the remainder to be domestically acquired.

Estimating Illnesses Acquired through Animal Contact

We applied pathogen-specific animal contact attribution proportions derived from a structured expert judgment (SEJ) of 5 transmission pathways (foodborne, waterborne, person-to-person, animal contact, and environmental) (*42*). Because *Listeria* was not included in the SEJ, we used the proportion from the previous animal contact estimates (*3*).

Estimating Hospitalizations and Deaths

We estimated the percentage of illnesses causing hospitalizations and deaths and applied them to projected US laboratory-confirmed illnesses after accounting for underreporting. We used the 2017–2019 data from FoodNet to estimate hospitalizations and deaths caused by *Campylobacter*, STEC, nontyphoidal *Salmonella*, and non-pestis *Yersinia*. We used data from the 2017–2019 *Listeria* Initiative to estimate hospitalizations and deaths caused by invasive *L. monocytogenes.* We used the 2016–2019 data from the National Inpatient Sample (NIS) to estimate hospitalizations and deaths caused by *Cryptosporidium* and *T. gondii* (*43*,*44*). NIS is nationally representative of all short-term, nonfederal hospitals, representing 97% of all discharges in the United States, and includes all patients, regardless of payer. Because NNDSS did not collect hospitalization or death data during the study period, we estimated the percentage of cryptosporidiosis causing hospitalization or death as the ratio of inpatients diagnosed with cryptosporidiosis in the 2016–2019 NIS (using codes from the International Classification of Diseases, 10th Revision, Clinical Modification) to the illnesses in NNDSS during the same period. For *T. gondii*, we estimated the hospitalization and death rates in the US population on the basis of the NIS during 2016–2019 and applied those data to the average US population during 2017–2019. We adjusted for underdiagnosis for hospitalizations or deaths, domestic acquisition, and animal contact as the mode of transmission.

Uncertainty Analysis

Models described in each step are multiplicative. For each factor, we drew a random sample (n = 10,000) from an empirical, degenerate, PERT, or posterior distribution. We applied successive factors by multiplication to obtain proportional increases or decreases in the count. That calculation usually produces wider ranges in the final distribution than additive models. We summarized the final distribution by using a point estimate (posterior mean) and 90% CrI. We report values at the precision of the direct model output; those values should not be interpreted as reflecting equivalent scientific precision, given the assumptions and uncertainty in model inputs.

We applied empirical distribution to laboratory-confirmed illnesses, percentages of hospitalization or death, and proportion of laboratories that routinely tested a pathogen from surveillance data. We applied simple nonparametric bootstrapping to illnesses in FoodNet by site and year and to illnesses in national surveillance systems by year. We used degenerate distribution for the ratio of US population to population in the surveillance area, the underreporting multiplier for active surveillance, and sensitivity of CIDTs. We used a modified PERT distribution with 4 parameters when data from surveillance were not available or insufficient to account for uncertainty. The first 3 parameters were low, modal, and high; the fourth parameter controls the flatness of the distribution, and the default value was 4. We used mean and confidence limits from literature as mode and the lower and upper bounds (Appendix Table 1). We used the default value for the fourth parameter. When only 1 of the low, modal, and high parameters was available, we calculated the other 2 parameters assuming that the upper bound was 1.5 and the lower bound was the reciprocal (1/1.5 times of the mode) on the odds scale. When combining distributions from 2 sources, we used random sampling with weights equal to the number of events from each source.

We used R version 4.1.1 for analyses (The R Project for Statistical Computing, https://www.rproject.org). We generated posterior distributions from Bayesian regression models by using the brms package in R.

Results

Campylobacter, *Cryptosporidium*, *L. monocytogenes*, nontyphoidal *Salmonella*, STEC (O157 and non-O157 serogroups), and non-pestis *Yersinia* caused an estimated 995,022 (90% CrI 476,251–1,833,238)

Table 1. Estimated annual number of domestically acquired animal contact illnesses caused by *Campylobacter* spp., *Cryptosporidium* spp., *Listeria monocytogenes*, nontyphoidal *Salmonella*, STEC, and *Yersinia* spp., United States, circa 2019*

Pathogen	No. laboratory-confirmed†	Underreporting multiplier	Underdiagnosis	International travel, %	Animal contact, %	Mean (90% CrI)
Campylobacter spp.	72,218‡	1	52.8	19	16	520,832 (86,949–1,333,098)
Cryptosporidium spp.	8,984§	1.15	42.3	11	21	82,757 (20,491–172,973)
Listeria monocytogenes, invasive¶						14 (8–20)
NPA	656#	1	1.7	3	1	11 (7–17)
PA, mothers	66#	1	1.7	3	1	1 (1–2)
PA, infants	57#	1	1.7	3	1	1 (1–2)
Salmonella, nontyphoidal¶						249,139 (110,141–468,266)
Enteritidis	11,240‡	1	38.8	22	5	19,333 (2,661–59,187)
I 4,[5],12:i-	3,721‡	1	38.5	10	12	17,153 (4,504–39,443)
Javiana	4,324‡	1	39.2	5	14	22,659 (3,272–63,051)
Newport	5,906‡	1	38.4	7	8	20,159 (3,859–53,952)
Typhimurium	6,287‡	1	39.0	7	13	30,926 (8,385–67,175)
Other	24,504‡	1	39.6	11	14	138,909 (23,667–345,624)
STEC¶						134,738 (31,457–315,575)
O157	5,069‡	1	38.9	9	12	17,261 (3,573–45,939)
Non-O157	15,648‡	1	42.7	24	22	117,477 (18,902–296,123)
Yersinia spp.	4,183‡	1	44.1	7	4	7,543 (2–29,706)
All pathogens¶						995,022 (476,251–1,833,238)

*Values are reported at the precision of the direct model output and should not be interpreted as reflecting equivalent scientific precision. CrI, credible interval; NPA, non–pregnancy-associated; PA, pregnancy-associated; STEC, Shiga toxin–producing *Escherichia coli*.
†Estimates based on the average US population during 2017–2019.
‡Active surveillance data from Foodborne Diseases Active Surveillance Network (2017–2019), adjusted for geographic coverage.
§Passive surveillance data from the National Notifiable Diseases Surveillance System (2016–2019).
¶Total number of illnesses and the number of domestically acquired illness through animal contact were the sum of the estimates for corresponding subgroups.
#Passive surveillance data from the Listeria Initiative (2017–2019).

domestically acquired illnesses linked to animal contact (Table 1). Together with *T. gondii*, those pathogens caused an estimated 9,592 (90% CrI 5,603–15,557) hospitalizations and 236 (90% CrI 119–411) deaths (Table 2).

Illnesses

Campylobacter caused the most domestically acquired estimated illnesses linked to animal contact (520,832 [90% CrI 86,949–1,333,098]), followed by nontyphoidal *Salmonella* (249,139 [90% CrI 110,141–468,266]), STEC (134,738 [90% CrI 31,457–315,575]), *Cryptosporidium* (82,757 [90% CrI 20,491–172,973]), non-pestis *Yersinia* (7,543 [90% CrI 2–29,706]), and *L. monocytogenes* (14 [90% CrI 8–20]). For nontyphoidal *Salmonella*, 138,909 (90% CrI 23,667–345,624) estimated illnesses were caused by serotypes other than the 5 we estimated: Typhimurium (30,926 [90% CrI 8,385–67,175]), Javiana (22,659 [90% CrI 3,272–63,051]), Newport (20,159 [90% CrI 3,859–53,952]), Enteritidis (19,333 [90% CrI 2,661–59,187]), and I 4,[5],12:i- (17,153 [90% CrI 4,504–39,443]). STEC non-O157 serogroups caused an estimated 117,477 (90% CrI 18,902–296,123) illnesses, and STEC O157 caused an estimated 17,261 (90% CrI 3,573–45,939) illnesses. *L. monocytogenes* caused an estimated 11 (90% CrI 7–17) non–pregnancy associated illnesses and 2 (1 mother [90% CrI 1–2] and 1 infant [90% CrI 1–2]) pregnancy-associated illnesses.

Hospitalizations

Campylobacter caused the highest estimated number of hospitalizations from domestically acquired illnesses linked to animal contact (3,601 [90% CrI 619–9,199]), followed by nontyphoidal *Salmonella* (2,415 [90% CrI 1,067–4,530]), *T. gondii* (1,773 [90% CrI 939–2,607]), STEC (960 [90% CrI 291–2,069]), *Cryptosporidium* (753 [90% CrI 185–1,565]), non-pestis *Yersinia* (80 [90% CrI 0–327]), and *L. monocytogenes* (12 [90% CrI 7–17]). STEC non-O157 caused 613 (90% CrI 91–1,549) hospitalizations, and STEC O157 caused 346 (90% CrI 69–951) hospitalizations. An estimated 10 non–pregnancy-associated hospitalizations (90% CrI 6–15) were attributed to *L. monocytogenes*, compared with 2 pregnancy-associated hospitalizations (1 mother [90% CrI 0–1] and 1 infant [90% CrI 0–1]).

Deaths

T. gondii caused the highest estimated deaths from domestically acquired illnesses linked to animal contact (91 [90% CrI 46–144]), followed by *Campylobacter* (55 [90% CrI 0–184]), nontyphoidal *Salmonella* (46 [90% CrI 1–138]), STEC (19 [90% CrI 0–66]), *Cryptosporidium* (20 [90% CrI 4–49]), non-pestis *Yersinia* (3 [90% CrI 0–11]), and *L. monocytogenes* (2 [90% CrI 1–3]). For nontyphoidal *Salmonella*, 25 (90% CrI 0–99) estimated deaths were caused by serotypes other than the 5 we estimated: Typhimurium (9 [90% CrI 0–43]), I 4,[5],12:i− (5 [90% CrI 0–26]), Enteritidis (4 [90% CrI 0–21]), Newport (2 [90% CrI 0–12]), and Javi-

ana (1 [90% CrI 0–6]). STEC non-O157 caused 11 (90% CrI 0–45) deaths, and STEC O157 caused 8 (90% CrI 0–39) deaths annually. Two non–pregnancy-associated deaths (90% CrI 1–3) and no pregnancy-associated deaths were attributed to *L. monocytogenes*.

Discussion

We estimated that *Campylobacter*, *Cryptosporidium*, STEC, *L. monocytogenes*, nontyphoidal *Salmonella*, and non-pestis *Yersinia* caused nearly 1 million illnesses linked to animal contact annually in the United States. Together with *T. gondii*, those pathogens caused ≈9,500 hospitalizations and ≈230 deaths. *Campylobacter* caused most illnesses and hospitalizations, followed by nontyphoidal *Salmonella*. *T. gondii* led in deaths, followed by *Campylobacter* and nontyphoidal *Salmonella*. Despite methodologic differences between studies, *Campylobacter* was the leading cause of animal contact illnesses in both analyses (*3*). However, in this report, estimated hospitalizations and deaths attributable to *Campylobacter* surpassed those caused by nontyphoidal *Salmonella*, which previously caused most hospitalizations and deaths. That change is attributable to a markedly higher estimated number of illnesses for *Campylobacter* versus nontyphoidal *Salmonella* in this study than in the 2012 estimates (*3*). Although *Campylobacter* accounts for a large proportion of the illnesses, various animal types (e.g., ruminants, poultry, and swine) contribute to exposure pathways. However, *Campylobacter* outbreaks linked to those animal types are poorly characterized, highlighting the need for further investigations to inform prevention strategies (*45*,*46*).

The first syndromic gastrointestinal panel tests, which are types of CIDTs, were approved by the US Food and Drug Administration in 2013. As described in a recent estimate of 7 major foodborne pathogens (*33*), the number of laboratories using CIDTs for enteric illness diagnosis has increased (*40*). CIDTs generally are considered more sensitive than traditional culture-based diagnostics. Consequently, the widespread adoption of CIDTs has increased detection of illnesses that would previously have gone undiagnosed and unreported, probably improving the accuracy of the estimates and contributing substantially to the higher illness estimates linked to animal contact (*34*,*47*).

We saw higher estimated illnesses for certain pathogens in this study than in the 2012 estimates (*3*). For *Campylobacter*, a total of 187,481 (90% CrI 66,259–372,359) illnesses were previously estimated, but

Table 2. Estimated annual number of domestically acquired animal contact hospitalizations and deaths caused by *Campylobacter* spp., *Cryptosporidium* spp., *Listeria monocytogenes*, nontyphoidal *Salmonella*, STEC, *Toxoplasma gondii,* and *Yersinia* spp., United States, circa 2019*

	Hospitalizations			Deaths		
Pathogen	% Cases	Underdiagnosis multiplier	Mean no. cases (90% CrI)	% Cases	Underdiagnosis multiplier	Mean no. cases (90% CrI)
Campylobacter spp.	22.1†	1.7	3,601 (619–9,199)	0.3†	1.7	55 (0–184)
Cryptosporidium spp.#	25.2‡	1.5	753 (185–1,565)	0.7‡	1.5	20 (4–49)
Listeria monocytogenes, invasive¶			12 (7–17)		1.7	2 (1–3)
NPA	87.4§	1.7	10 (6–15)	15.8¶	1.7	2 (1–3)
PA, mothers	70.4§	1.7	1 (0–1)	0¶	1.7	0 (0)
PA, infants	80.6§	1.7	1 (0–1)	5.8¶	1.7	0 (0)
Salmonella, nontyphoidal¶			2,415 (1,067–4,530)			46 (1–138)
Enteritidis	27.7†	1.5	192 (24–606)	0.6†	1.5	4 (0–21)
I 4,[5],12:i-	28.7†	1.5	176 (35–434)	0.7†	1.5	5 (0–26)
Javiana	25.2†	1.5	218 (13–719)	0.1†	1.5	1 (0–6)
Newport	28.0†	1.5	193 (31–525)	0.3†	1.5	2 (0–12)
Typhimurium	29.3†	1.5	348 (77–794)	0.8†	1.5	9 (0–43)
Other	27.1†	1.5	1,288 (213–3,255)	0.5†	1.5	25 (0–99)
STEC¶			960 (291–2,069)			19 (0–66)
O157	43.1†	1.5	346 (69–951)	1.0†	1.5	8 (0–39)
Non-O157	16.2†	1.5	613 (91–1,549)	0.3†	1.5	11 (0–45)
Toxoplasma gondii#	<0.001	1.5	1,773 (939–2,607)	<0.001	1.5	91 (46–144)
Yersinia spp.	28.5†	1.7	80 (0–327)	1.4†	1.7	3 (0–11)
All pathogens¶			9,592 (5,603–15,557)			236 (119–411)

*Values are reported at the precision of the direct model output and should not be interpreted as reflecting equivalent scientific precision. CrI, credible interval; NPA, non–pregnancy associated; PA, pregnancy associated; STEC, Shiga toxin–producing *Escherichia coli*.
†Percentage hospitalized and died from the Foodborne Diseases Active Surveillance Network (2017–2019)
‡Percentage hospitalized and died were estimated using hospitalizations and deaths from the National Inpatient Sample (2016–2019) using International Classification of Diseases, 10th Revision, Clinical Modification, code A072 (cryptosporidiosis) compared with illnesses reported to the National Notifiable Diseases Surveillance System (2016–2019).
§Percentage hospitalized and died from the *Listeria* Initiative (2017–2019).
¶Total number of illnesses and the number of domestically acquired hospitalizations and deaths through animal contact were the sum of the estimates for corresponding subgroups.
#Based on the rates of hospitalizations and deaths from the National Inpatient Sample (2016–2019) applied to the United States.

520,832 (90% CrI 86,949–1,333,098) illnesses were estimated in this report. *Campylobacter* is fastidious and requires specific culture conditions, and increased use of CIDTs probably has improved detection. For STEC O157, previous estimates were 5,960 (90% CrI 1,518–14,606) illnesses, but 17,261 (90% CrI 3,573–45,939) illnesses were estimated in this report. The 117,477 (90% CrI 18,902–296,123) STEC non-O157 estimated illnesses in this report was >10 times higher than the 10,097 (90% CrI 1,005–27,073) illnesses in the previous estimates. Identifying specific pathotypes causing *E. coli* infections other than O157 is difficult using culture-based methods; however, CIDTs have helped overcome many challenges, contributing to the higher estimated STEC illnesses in the current report. Nontyphoidal *Salmonella* also showed higher estimated illnesses: 127,155 (90% CrI 66,502–219,886) in the previous estimates and 249,139 (90% CrI 110,141–468,266) in this report. Diagnostic specificity of clinical and CIDT-based tests across these pathogens detected in ill persons who sought care are assumed to be accurate; thus, differences in estimates are probably not driven by false-positive results. Instead, observed increases are probably attributable to improved case detection.

Cryptosporidium illnesses were lower than in previous estimates; 113,344 (90% CrI 22,570–331,635) illnesses were estimated previously and 82,757 (90% CrI 20,491–172,973) illnesses in this report. However, the estimated 753 (90% CrI 185–1,565) hospitalizations and 20 (90% CrI 4–49) deaths in this report were higher than the 412 (90% CrI 108–1,039) hospitalizations and 7 (90% CrI 0–37) deaths previously estimated. The lower estimate of *Cryptosporidium* illnesses is primarily attributable to a lower underdiagnosis multiplier, reflecting a new approach for accounting for medical care seeking and specimen submission (*33*). A higher underdiagnosis multiplier was used in the 2012 estimates under the assumption that most illnesses caused by *Cryptosporidium* were mild. *L. monocytogenes* estimates were similar between the previous estimates and this report; most illnesses were attributable to foodborne transmission (*33*). *Yersinia* illnesses, hospitalizations, and deaths estimated in this report represent all non-pestis *Yersinia* species, whereas the 2012 estimates included only those caused by *Y. enterocolitica*. Increased use of CIDTs has improved detection of yersiniosis cases that would have previously gone undiagnosed, serving as the probable explanation for the higher estimated illnesses, hospitalizations, and deaths caused by *Yersinia* in this report (*45*).

T. gondii was not included in the previous estimates but showed the highest number of estimated deaths linked to animal contact in this report (91 deaths annually). An SEJ identified animal contact as the primary transmission pathway for *T. gondii* (58%), compared with foodborne (28%), environmental (9%), and waterborne (5%) sources (*42*). Of note, the SEJ defined animal contact transmission as direct or indirect, including contact with excreta in the local environment. Toxoplasmosis is thought to be primarily transmitted through ingesting the parasite after this type of indirect contact. Direct animal contact transmission is less likely because oocysts shed in felid feces require 1–5 days to sporulate and become infective, and felids typically shed oocysts for only 1–3 weeks after exposure. In contrast, environmental transmission refers to exposure or contact with contaminated soil that is not attributable to animal contact. The recent estimate of 7 major foodborne pathogens reported both hospitalizations and deaths caused by *T. gondii* were lower than in previous estimates (*33*). Immunocompromising conditions, such as HIV, are risk factors for severe illness from *T. gondii*, and increased access to antiretroviral drugs might contribute to lower estimates of hospitalizations and deaths in recent reports (*48*). The high percentage of animal contact transmission might explain the higher number of deaths associated with *T. gondii* compared with other pathogens.

The zoonotic pathogens in this report are linked to different animal types and settings where persons might interact with those animals. Although we did not distinguish illnesses by animal type or setting, a report on enteric pathogen outbreaks in the United States linked to animal contact reported to the Animal Contact Outbreak Surveillance System during 2009–2021 identified 557 outbreaks, highlighting the most common animal types and settings (*49*). The etiologies in order of highest to lowest number of outbreaks included *Salmonella*, *Cryptosporidium*, STEC infections, and *Campylobacter* and caused 14,377 illnesses, 2,656 hospitalizations, and 22 deaths. Outbreaks of *T. gondii*, non-pestis *Yersinia*, and *L. monocytogenes* linked to animal contact were not reported to the Animal Contact Outbreak Surveillance System during that timeframe. Backyard poultry–associated salmonellosis outbreaks and illnesses were the most common and have increased in recent years (*18*,*49*). Reptiles were the second most common source of salmonellosis outbreaks, including those linked to small turtles despite a federal ban on their sale (*50*). Overall, most animal contact during outbreaks occurred in private homes (*49*). Although pet ownership rates

remained relatively unchanged during 2012–2022 (https://faunalytics.org/u-s-pet-ownership-demographics-sourcebook-2012, https://ebusiness.avma.org/files/ProductDownloads/eco-pet-demographic-report-22-low-res.pdf), the higher estimated illnesses for most pathogens in this study underscore the need to improve prevention knowledge among pet owners. National data on pet ownership does not include wildlife or animals housed in public settings, but enteric disease outbreaks associated with animal contact have been linked to additional settings outside the private home, including farms and dairies, festivals or fairs, and petting zoos (*49*). Therefore, stable pet ownership rates may not fully reflect opportunities for animal contact and associated transmission because national estimates do not capture exposures in those settings (https://faunalytics.org/u-s-pet-ownership-demographics-sourcebook-2012, https://ebusiness.avma.org/files/ProductDownloads/eco-pet-demographic-report-22-low-res.pdf). In addition, advancements in diagnostic testing and outbreak detection may have contributed to the higher prevalence estimated in our analysis. Future analyses describing estimated illnesses by animal type and setting might identify higher-risk exposures to these pathogens and guide targeted prevention measures.

One limitation of our study is that we used similar methods to those in the estimate for 7 major foodborne pathogens, preventing direct comparison with previous animal contact estimates (*3*). The source data have many unquantified uncertainties, several of which are described in methodologic limitations elsewhere (*33*). For example, the FoodNet catchment area is a critical contributor of unquantified uncertainty because of geographic variation in incidence rates for all pathogens. Limited health department personnel, funding, and laboratory capacity across jurisdictions might reduce the detection and reporting of animal contact–associated outbreaks, potentially affecting disease prevalence estimates. Although national reporting definitions exist, variations in how health departments define animal contact might affect how illnesses and outbreaks are categorized and reported (*47*). In addition, differences in surveillance, outbreak detection, and pathogen transmission might influence model estimates, limit direct comparisons, and contribute to underreporting. Estimates derived from SEJ are subject to expert opinion and might not reflect evidence that emerged later. Furthermore, our analysis focused on the primary pathogens causing animal contact illnesses in the United States. As national surveillance data become available, future analyses should include additional pathogens that cause animal contact illnesses to improve disease prevalence estimates and associated economic costs.

Our study found that ≈1 million illnesses, ≈9,500 hospitalizations, and ≈230 deaths linked to animal contact occur each year. Those findings underscore the need for enhanced education, awareness, and prevention measures to reduce the prevalence of disease in the United States. Continued surveillance, outbreak investigations, and epidemiologic studies are needed to improve understanding of transmission pathways and guide future disease prevalence estimates and prevention priorities. Recognizing animal contact as an important mode of transmission is essential for disease prevention priorities in addition to other routes, including foodborne, waterborne, environmental, and person-to-person transmission.

Acknowledgments

We thank Erin Imada and Susan Montgomery for providing data and expert advice for estimates described in this article. We also thank Sean Stapleton for assistance in verifying the accuracy of tabular data.

This activity was reviewed by CDC, deemed not research, and was conducted consistent with applicable federal law and CDC policy (e.g., 45 C.F.R. part 46.102(l)(2); 21 C.F.R. part 56; 42 U.S.C. §241(d); 5 U.S.C. §552a; 44 U.S.C. §3501 et seq.).

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