Cat-dependent diseases cost Australia AU$6 billion per year through impacts on human health and livestock production

Sarah Legge, Pat L. Taggart, Chris R. Dickman, John L. Read and John C. Z. Woinarski.

Abstract

Context: Cats are the definitive or primary host for pathogens that cause diseases in people and livestock. These cat-dependent diseases would not occur in Australia if cats had not been introduced, and their ongoing persistence depends on contacts with cats. *Toxoplasma gondii* is a protozoan parasite that cycles between cats and any other warm-blooded animals. People infected by *T. gondii* may appear asymptomatic, or have a mild illness, or experience severe, potentially lethal symptoms; the parasite may also affect behaviour and mental health. *T. gondii* is also a major contributor to spontaneous abortion in sheep and goats. Two species of *Sarcocystis*, another genus of protozoan parasite, cycle through cats and sheep, causing macroscopic cysts to form in sheep tissues that reduce meat saleability. *Toxocara cati*, the cat roundworm, causes minor illnesses in humans and livestock, and the bacterium *Bartonella henselae* causes cat scratch disease, an infection that can be contracted by people when scratched or bitten by cats carrying the pathogen.

Aims: We estimated the economic costs of cat-dependent pathogens in Australia.

Methods: We collated national and global data on infection rates, health and production consequences.

Key results: We estimated the costs of two cat-dependent diseases (toxoplasmosis, cat scratch disease) in people at AU$6.06 billion (plausible range AU$2.11–10.7 billion) annually, and the costs to livestock production from toxoplasmosis and sarcocystosis at AU$11.7 million (plausible range AU$7.67–18.3 million). Most of the human health costs are due to the associations between *T. gondii* and higher rates of traffic accidents and mental illness in people. The causality behind these associations remains uncertain, so those costs may be overestimated. Conversely, our estimates are incomplete, infections and illness are under-reported or misdiagnosed, and our understanding of disease outcomes is still imperfect, all of which make our costs underestimated.

Conclusions: Our analysis suggests that substantial benefits to public health and livestock production could be realised by reducing exposure to cats and breaking parasite transmission cycles.

Implications: Reducing feral cat populations in farming and urban areas, reducing the pet cat population and increasing rates of pet cat containment could help reduce the burden of cat-dependent diseases to people and livestock.

Additional keywords: disease, human dimensions, invasive species, pest management.

Introduction

Domestic cats *Felis catus* carry a diverse assemblage of bacterial, viral, fungal and parasitic pathogens (Day *et al.* 2012). Some of these pathogens rely on cats for part of their life cycle and would not exist in Australia if cats had not been introduced when Europeans colonised the continent from 1788 (Woinarski *et al.* 2019). These novel pathogens may have contributed to declines in some Australian endemic mammal species (Pan *et al.* 2012; Hillman *et al.* 2016). They also have substantial impacts on human health and livestock production (Chomel and Sun 2011; Lepczyk *et al.* 2015; Dubey 2016); however, these impacts have not previously been collectively estimated.

There are five pathogens in Australia with known impacts on human health and livestock production, for which cats are the definitive or primary host. The most significant of these is *Toxoplasma gondii*, a protozoan parasite that reproduces sexually in felids (the definitive host) and cycles asexually through any other warm-blooded animal (the intermediate host; Aguirre *et al.* 2019). *Sarcocystis gigantea* and *S. medusiformis* are also protozoan parasites for which domestic cats are the definitive host; they cycle through sheep as the intermediate host (Collins *et al.* 1979). The roundworm *Toxocara cati* is a nematode that infects felids, and can affect other mammals (Fisher 2003). The bacterium *Bartonella henselae* lives in the blood cells of domestic cats, and can temporarily infect people (Chomel *et al.* 2006). *Toxoplasma gondii*, *T. cati* and *B. henselae* have impacts on human health; *T. gondii*, *T. cati*, *S. gigantea* and *S. medusiformis* affect livestock production.

Estimates of the incidence, and health and economic impacts of *T. gondii* in people have been made in some countries (Roberts *et al.* 1994; Scharff 2012; Schurer *et al.* 2016), and an attempt to estimate the disease burden of *T. gondii* globally concluded that it was substantial (Torgerson and Macpherson 2011). In Australia, estimates are available for the prevalence of the parasite in the human population; however, a national collation of the incidence of health impacts and an estimate of the economic costs of this parasite to human health have not previously been attempted. Similarly, although some data are available on the prevalence of *T. gondii* in livestock from some regions of Australia, there has been no attempt to estimate the production costs nationally. Data on the incidence, impacts and associated economic costs of *S. gigantea*, *S. medusiformis*, *T. cati* and *B. henselae* are even more fragmentary.

In the present paper, we collate disparate information on the incidence of these cat-dependent pathogens in humans and livestock, and estimate their economic costs on human health and livestock production in Australia. Below, we summarise the impacts of these cat-dependent pathogens, before describing how we estimated the associated costs.

Toxoplasma gondii

Felids, including domestic cats, mostly become infected with *T. gondii* by eating infected prey or carrion. The parasite reproduces sexually in the cat gut, releasing oocysts, an egg-type life stage, in the cat faeces. Oocysts can remain viable in the environment for at least 18 months under favourable conditions (Dubey 2016; Aguirre *et al.* 2019). Intermediate hosts, including people, become infected with *T. gondii* by accidentally ingesting oocysts (for example, when gardening, handling kitty litter, drinking contaminated water; Bowie *et al.* 1997; Torrey and Yolken 2013), or by eating raw or undercooked meat containing the parasite. After a phase of rapid replication and migration, a form of the parasite encysts in the body, often in the brain, and persists there indefinitely (Tenter *et al.* 2000).

Toxoplasma gondii prevalence in people is highly variable, ranging between 3% and 84% among countries, but with an average global prevalence of ~30% (Tenter *et al.* 2000; Flegr *et al.* 2014), making it one of the most common infections in the world. The prevalence rates of *T. gondii* in Australians are close to the global average, with studies showing infection rates of 23–66% (Johnson *et al.* 1980; Sfameni *et al.* 1986; Karunajeewa *et al.* 2001; Molan *et al.* 2020).

Most *T. gondii* infections in people are asymptomatic; however, because infection rates are so high, *T. gondii* still imposes a substantial health burden (Torgerson and Macpherson 2011; Aguirre *et al.* 2019). For example, toxoplasmosis, the disease caused by a *T. gondii* infection, is a leading cause of food-borne illness and food-borne death in some countries (Scallan *et al.* 2011; Bouwknegt *et*

al. 2018). Toxoplasmosis usually manifests with non-specific symptoms that are easily misdiagnosed (Aguirre *et al.* 2019). However, in some cases, neural and eye tissue can be affected (Montoya and Liesenfeld 2004), and in immunocompromised people (cancer, transplant, AIDS patients; some elderly people), new infections or reactivation of latent infections can cause severe effects, especially to the heart and nervous system, potentially leading to death (Frenkel 1990).

Toxoplasma gondii infections in pregnant women who have not previously been infected with the parasite can be transmitted across the placenta to the fetus, in some cases causing miscarriage or congenital defects, especially to the nervous system, ears and eyes, that can have lifelong repercussions (Wong and Remington 1994; Montoya and Liesenfeld 2004). Babies exposed to infection *in utero* may appear asymptomatic at birth, but experience vision, hearing and development difficulties later in life (Wong and Remington 1994).

Latent *T. gondii* infections in humans are associated with behavioural shifts, including decreased conscientiousness, extrovert and entrepreneurial behaviour, and slower reaction times (Flegr *et al.* 2013; Johnson *et al.* 2018). The latter might explain the greater representation of people seropositive to *T. gondii* in accidents, including car accidents (Flegr *et al.* 2002; Gohardehi *et al.* 2018; Sutterland *et al.* 2019). There is increasing evidence of links between latent *T. gondii* infection and psychiatric diseases including depression, bipolar disorder, schizophrenia, behavioural disorders and poor cognitive function (Henriquez *et al.* 2009; Torrey *et al.* 2012; Pearce *et al.* 2014; Fuglewicz *et al.* 2017; Houdek 2017; Akaltun *et al.* 2019). *Toxoplasma gondii* infection is also correlated with higher rates of suicide (Ling *et al.* 2011; Pedersen *et al.* 2012; Sutterland *et al.* 2019). These associations may be underpinned by *T. gondii*-induced changes to the concentrations of brain chemicals such as dopamine, serotonin and tryptophan (Vyas 2015), and possibly to neuro-inflammation (Boillat *et al.* 2020). There are also correlations between *T. gondii* infections and many other diseases including epilepsy, cardiovascular disease, endocrine disease and brain cancer (Thomas *et al.* 2012; Flegr *et al.* 2014); however, plausible causal links are less well determined than are those between *T. gondii* and behaviour or mental illness.

Livestock become infected by *T. gondii* by ingesting feed or water contaminated with oocysts (Dubey 2016). In horses, cattle, pigs, ducks, turkeys, geese and chickens, *T. gondii* rarely causes disease (Stelzer *et al.* 2019). In contrast, small ruminants including sheep and goats can experience fever or diarrhoea for up to a fortnight (Charleston 1994; Dubey 2009*b*). If female sheep and goats contract *T. gondii* for the first time while pregnant, there is a risk of spontaneous abortion, still-birth and neonatal mortality (Plant *et al.* 1982; Dubey 2009*b*). *Toxoplasma gondii* infections are one of the main causes of infectious abortion in sheep in Australia and New Zealand, parts of Europe and the USA (Dubey 2009*b*). The contribution of the parasite to abortion rates locally can be high (e.g. 66% of abortions as a result of *T. gondii* infection in a study in Iran, Habibi *et al.* 2012), so the impacts of *T. gondii* infections can potentially be devastating for producers.

Toxocara cati

Adult female *T. cati* worms produce eggs that are expelled in cat faeces, then develop over about 2 months into larvae encased in a resistant shell, remaining viable and infective for years (Despommier 2003; Jarosz *et al.* 2010; Fan *et al.* 2015). People become infected with *T. cati* by accidentally ingesting the encased larvae. Most infections are asymptomatic; however, symptoms that do develop arise from the migration of *T. cati* larvae through the body, damaging tissue and causing inflammation. Larval migration through the eye, central nervous system and heart can be serious (Torgerson and Macpherson 2011). If livestock contract *T. cati*, larval migration can cause lesions in meat; however, the extent of this problem is unclear (Torgerson and Budke 2006).

Bartonella henselae

People can develop cat scratch disease, or cat scratch fever, after being bitten or scratched by a cat infected with *B. henselae*. Symptoms range from having a small sore near the scratch site, to mild fevers, aches and swollen glands (especially near the bite or scratch site). Symptoms generally begin within a few days of the scratch or bite and last for approximately 1 month. The infection can be more serious in immunocompromised people, with symptoms including encephalitis, severe muscle pain and skin lesions (Chomel 2000; Klotz *et al.* 2011).

Sarcocystis gigantea and S. medusiformis

Sarcocystis gigantea and *S. medusiformis* reproduce sexually in the gut of domestic cats, releasing sporocysts via the cat faeces. Sheep are infected by ingesting sporocysts. The parasites migrate through the sheep tissue, forming macroscopic sarcocysts, mostly in the muscles of the oesophagus, tongue, diaphragm and abdomen. Macroscopic sarcocysts can take up to 4 years to reach their full size (Moré 2019). Infection appears non-pathogenic to sheep, but, after slaughter, macroscopic sarcocysts are trimmed from infected carcasses for aesthetic reasons, causing direct loss or downgrading of meat products. Infected carcasses may also be boned-out to ensure the removal of macroscopic sarcocysts not immediately visible to meat inspectors, and, hence, sold as a lower value bone-out product. Rarely, heavy infections can result in the entire carcass being condemned. Macroscopic sarcocystosis in sheep may contribute to entire meat shipments being rejected when sarcocysts are missed at the meat-processing facility and discovered instead at the shipment destination (McMahon 1978). Carcass condemnation and trimming is rare in lambs relative to older sheep, as the risk of infection is cumulative and cysts have less time to form.

Methods

Cat-dependent pathogens of people

For each cat-dependent pathogen, we searched the literature for reports of infection prevalence and disease incidence in humans. We used Australian studies when available, but judiciously supplemented this with work from other countries if Australian studies were not available. For each disease, we developed basic cost-of-illness estimates that included medical costs, ongoing medical or other care, and the cost of lost productivity (i.e. lost income) to the affected person (and carers, where relevant). We based costs for each of these consequences from various sources, as detailed below. Where a range of values or measures of variation around mean estimates were available, we propagated this uncertainty through the calculations to generate a plausible range of cost values. Estimates of error around values were not always available, such as, for example, for the cost of types of hospital care.

Where necessary, we used online currency conversion to transform cost estimates from non-Australian studies into Australian dollars (https://www.xe.com/currencyconverter/, accessed 12 August 2020). We converted all costs to 2019 values by using an inflation calculator (https://www.rba.gov.au/calculator/, accessed 12 August 2020). To estimate lost productivity costs, we multiplied the number of days a sick person would require off work, the probability that a person is employed (using the participation rate, ABS 2020), and the average weekly wage (ABS 2019*b*). For some disease outcomes, we also estimated the lost productivity of caregivers (details below). Costs are in Australian dollars unless otherwise indicated.

Toxoplasma gondii

Toxoplasmosis is not a notifiable disease in many countries, including Australia, so estimates for the costs of *T. gondii* infection are constrained by poor and inconsistent disease reporting (FSANZ 2017), as well as an incomplete understanding of the impacts of long-term infection. Nevertheless, we estimate some components of the human health costs in Australia by considering the following three types of consequence: acute illness, congenital infection, and impacts of long-term latent infection on behaviour and mental health. For acute and congenital infections with complex and sometimes delayed physical-health impacts, we developed outcome trees to help clarify and organise the frequencies of each health impact and their costs.

Acute illness. *Toxoplasma gondii* prevalence in middle-aged to older Australians (40+ years old) has been reported as 40% (Johnson *et al.* 1980). We used population census data from 2019 (25 500 000, ABS 2019*a*) to estimate the number of people infected with the parasite, then divided this by an average lifespan (80 years) to estimate the number of new infections occurring annually.

We developed an outcome tree to establish the proportion of new infections that cause illness (Montoya and Liesenfeld 2004), the proportion of ill people that are hospitalised (Scallan *et al.* 2011), and the proportion of those that die as a consequence of infection (Scallan *et al.* 2011). We used values from a study in the USA, but note that these estimates accord with studies in Canada (Thomas *et al.* 2015; Schurer *et al.* 2016). We propagated the mean proportion (and the 90% credible intervals around this statistic) of symptomatic cases that are hospitalised, and those that later die, through the cost analysis, to generate a plausible range of cost values.

For medical costs, we assumed that 60% of symptomatic mild cases visit a general medical practitioner (GP), and that 20% visit an ophthalmologist, on the basis of the frequency with which symptomatic people sought medical care, and developed eye disease, during a monitored toxoplasmosis outbreak in Canada (Bowie *et al.* 1997). We used the average consultation costs for GPs and ophthalmologists set out in the fee schedule of the Australian Medical Association (https://ama.com.au/). To estimate the medical costs of people hospitalised as a result of toxoplasmosis, we used two data sources. First, the Australian Institute of Health and Welfare (AIHW) reports that the average cost of treating admitted patients with parasitic disease is AU$9650 (AIHW 2019*a*). Second, average hospital admission costs reported by the Independent Hospital Pricing Authority (NHCDC 2019) range from AU$4885 for patients requiring short-term treatment, to AU$13 397 for patients needing longer-term care. We assumed that cases of toxoplasmosis are evenly distributed across these two categories of admission (i.e. an average of AU$9141), because ~50% of toxoplasmosis patients are immunocompromised and require more complex care (e.g. transplant recipients, cancer patients, HIV carriers; Schurer *et al.* 2016). Because estimates from both data sources were similar, we used the AIHW data for simplicity. Terminal illnesses entail greater hospital costs; therefore, for these cases, we added the cost (Hicks *et al.* 2019) of 4 days in intensive care, which is the average duration of stay in intensive care (AIHW 2019*a*), to the general cost of treating parasitic disease.

To estimate productivity loss, we assumed that mild cases of toxoplasmosis resulted in 5 days off work; that serious cases resulted in 30 days off work; given that a study in Canada reported that toxoplasmosis patients spent 17–37 days in hospital (Schurer *et al.* 2016); and that if death was the outcome of the serious illness, this resulted in 25 years of lost work (given an average age of infection of 40 years (Schurer *et al.* 2016), and a retirement age of 65). To account for caregiver time off work when a relative has toxoplasmosis, we assumed the caregiver's lost productivity was 20% that of the sick patient; we note this is much more conservative that the approach taken by Scharff (2012), who assumed that the caregiver's lost productivity was similar to that of the patient's.

We assumed that infected people express illness only once, during initial infection, but note that some individuals may become ill more than once, such as, for example, if immunosuppression results in reactivation of the disease.

Congenital infections. Australia lacks prenatal screening for *T. gondii*, which leads to under-reporting of infections and removes the opportunity to calculate statistics over very large samples. We, therefore, estimated the annual number of congenital *T. gondii* infections in Australia in two ways. First, we reviewed the literature for Australian studies that have reported the proportion of infected livebirths from serological surveys, and applied this proportion to the most recent data on the annual number of livebirths (ABS 2018). These studies were not conducted over a large sample of births. So, we also calculated the number of congenital infections expected, given that (1) the average age of mothers giving birth in 2017 (AIHW 2019*b*), (2) the proportion of mothers of that average age who are not immune to *T. gondii* (Johnson *et al.* 1980), (3) the infection rates of non-immune mothers, based on an annual increase in infection rates for women of child-bearing age (Johnson *et al.* 1980; Sfameni *et al.* 1986), and (4) the overall frequency with which maternal infections are transferred to the fetus (Dunn *et al.* 1999).

We summarised the health consequences of congenital *T. gondii* infections and their frequencies in an outcome tree, on the basis of a literature review (Roberts *et al.* 1994; Dunn *et al.* 1999; Kortbeek *et al.* 2004; Havelaar *et al.* 2007; Dubey 2016; McLeod *et al.* 2020). Following Roberts *et al.* (1994), we assumed that babies that die first spend 3 weeks in intensive care, babies with severe symptoms require 2 weeks in intensive care, babies with moderate symptoms need 4 days in intensive care, and babies with mild symptoms incur only the average hospital admission cost for neonates (without intensive care). Costs of intensive care were taken from Hicks *et al.* (2019), and costs of general hospital care and medical interventions to treat eye, ear, and nervous-system disease were taken from the most recent report prepared by the AIHW (2019*a*). The lifetime costs for ongoing medical care (visits to eye and ear specialists) of babies that were symptomatic at birth, and also for additional individuals who were asymptomatic at birth but developed conditions later in life, were taken from the AIHW report and from the fee schedule of the Australian Medical Association. Severely affected babies may have intellectual disabilities with associated lifetime costs for residential, educational and therapeutic care; these costs to the family of the affected person as well as the government have been summarised previously (Doran *et al.* 2012).

We estimated lost-productivity costs of affected individuals by using estimated proportional income losses for intellectual and physical disabilities provided in Roberts *et al.* (1994), applying these to the mean weekly wage for employed Australians, and considering the participation rate, over a working life of 45 years. To account for family caregiver time off work, arising from cases of congenital *T. gondii* infections that cause life-long impairment, we used the estimates for the lost opportunity times of caregivers for different levels of disability in Doran *et al.* (2012), and applied them to our estimates of the number of cases of each type of disability.

Congenital infections, particularly those early in pregnancy, can result in fetal or neonatal death; we do not attempt to estimate the frequency and cost of such miscarriages and deaths to mothers or other family members, but acknowledge that they may be considerable.

Latent infection, behaviour and mental health. Recent meta-analyses across large numbers of studies have estimated the population-attributable fractions (PAFs) of *T. gondii* infections to car accidents, suicides and suicide attempts, and schizophrenia. The PAF is defined as the proportion of cases that would not occur in a population if a particular risk factor was eliminated. We used these estimates

(Smith 2014; Sutterland *et al.* 2019) to estimate how the incidence and costs of traffic accidents, suicides and schizophrenia may be reduced in the absence of *T. gondii*.

Traffic accidents cause 1200–1400 deaths and 40 000 hospitalisations annually in Australia (Commonwealth of Australia 2019). For the present analysis, we used the road-trauma values for 2015, the latest year for which accurate data on medical costs, costs to government, and a range of others costs (disrupted workplace, delayed travel times) were available for the same year (AAA ECON 2017). To estimate lost productivity, we used the number of life years lost as a result of road trauma presented by AAA ECON (2017), halved this (because the average age of hospitalised people was 40, and of people that died was 45, so the average road-accident victim was halfway through their working life), and multiplied that by the average annual income (using the weekly average wage and participation rate from ABS).

For the annual number of suicides, we averaged suicide deaths from 2016 to 2018 (Mindframe 2019). We followed Kinchin and Doran (2017) and assumed that for every suicide death, there were 15 suicide attempts. The costs of suicide and suicide attempts in Australia have recently been summarised (Kinchin and Doran 2017). The analysis by Kinchin and Doran (2017) included direct costs from coronial inquiries, funeral costs, medical costs, counselling costs, disability care costs, as well as the indirect costs of lost productivity and the costs to government from lost taxes. Their analysis of lost productivity accounts for lower employment rates of people who attempt or commit suicide.

We based the annual number of new schizophrenia cases with psychotic events from data available in McGrath *et al.* (2008), and the costs per patient for treatment, care (such as accommodation and other crisis support) and lost productivity for these patients from data provided by Neil *et al.* (2014).

In this part of our cost analysis, lost productivity of carers is incompletely estimated. The costs of employed carers are given in Kinchin and Doran (2017), but there is no information on the lost productivity of family members who act as unpaid carers. We also lacked information on the extent of unpaid caregiver time following car accidents and mental illness, on which to base cost estimations.

Bartonella henselae

The incidence of *B. henselae* infections and cat scratch disease in Australia has not been quantified. To find a plausible range of values for disease incidence, we reviewed studies in the USA and Europe (where ownership rates for pet cats are broadly comparable to those in Australia) and used the mean and range of values for the annual incidence of cat scratch disease diagnoses (9–12 people per 100 000), and the proportion of diagnoses that need hospitalisation (Jackson *et al.* 1993). We assumed that mild cases incurred costs for one visit to a GP plus one diagnostic test, and sourced those costs from the fee register of the Australian Medical Association. For the costs of hospitalisations, we used the average admission costs for patients with circulatory disease available from AIHW (2019*a*). To estimate productivity loss, we assumed that mild cases of cat scratch disease involved 4 days off work, and hospitalisations involved 2 weeks off work (Jackson *et al.* 1993). We used the mean weekly wage, discounted by the participation rate (as above), to find the cost of time off work. To account for caregiver time off work when a relative has cat scratch disease, we again assumed that the caregiver's lost productivity was 20% that of the sick patient.

Toxocara cati

Estimates for the prevalence of *T. cati* in Australia are few and immunological tests usually do not differentiate between *T. cati* and *T. canis*, another common helminthic parasite (but mostly of dogs; Fisher 2003). For example, a survey for exposure to *Toxocara* spp. conducted in Canberra reported that 7% of adults and 23% of children had antibodies to *Toxocara* spp., but did not distinguish between species (Nicholas *et al.* 1986). Also, there is no information available on the proportion of infected people that develop symptoms. Thus, estimating the health costs of *T. cati* infections in Australia is currently not possible.

Cat-dependent pathogens of livestock

We compiled reports of infection prevalence and disease incidence for each pathogen in livestock, and the impacts on production. We used Australian studies when available, supplemented by studies from other countries. The costs of production losses were obtained from sources described below.

Toxoplasma gondii

We confined the analysis to the costs of lost production in sheep. Although *T. gondii* also causes abortion in goats, its occurrence is less frequently reported, and goat flock size and, therefore, the cost of disease is smaller. There are no Australia-wide data available for the prevalence of *T. gondii* infection or *T. gondii*-induced abortions in sheep. Accordingly, we used the mean, and lower and upper ranges of estimates for the proportion of sheep abortions that are caused by *T. gondii* from a comprehensive global review by Dubey (2009*b*). We considered the proportion of *T. gondii*-induced abortions in the context of an overall abortion rate in Australian sheep (DPIRD 2019), the number of lambs that are slaughtered each year (ABS 2019*d*), and the price for a 20-kg lamb sold at market (MLA 2019).

Sarcocystis gigantea and S. medusiformis

Macroscopic sarcocystosis is not spread evenly across sheep-growing regions in Australia and infection rates are high in some regions with high cat densities, such as Kangaroo Island, South Australia, and low in other regions (Hernandez-Jover *et al.* 2013; DPIPWE 2016; Phythian *et al.* 2018; Taggart *et al.* 2019*b*). We, therefore, estimated the costs of sarcocystosis to the sheep industry by combining figures for the proportion of carcasses affected by macroscopic sarcocystosis in each state, as reported by the National Sheep Health Monitoring Program (NSHMP 2018), the numbers of adult sheep and lambs slaughtered in those states (adults and lambs were considered separately because the incidence of sarcocystosis varies with age, ABS 2019*c*), the average trim weight (including carcass condemnations, Hernandez-Jover *et al.* 2013), and the cost of that meat (MLA 2019).

Toxocara cati

In Australia, helminth infections cost the meat industry AU$532 million per year, including the costs of drenching to kill helminths at AU$144 million per year (Lane *et al.* 2015). Drenching is mainly directed towards other more economically potent species of helminth, but would nevertheless also act on *T. cati* infections. However, it is not possible to apportion a fraction of these costs to *T. cati* infection; so, *T. cati* costs were not estimated here.

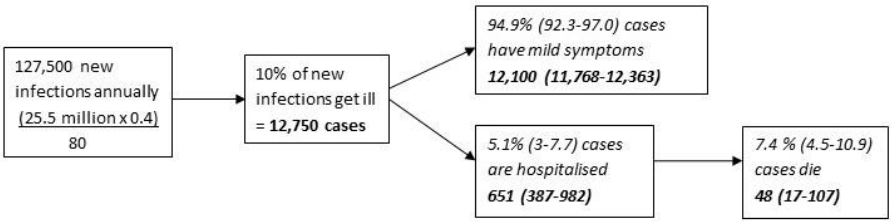
Results

Cat-dependent pathogens of people

Toxoplasma gondii

Acute illness. Based on the logic and sources described above, we estimated that each year ~127 500 Australians are infected with *T. gondii*. Of these, 12 750 are likely to be symptomatic, and, of these, 12 100 have mild illnesses, 651 cases require hospitalisation, and 48 people die as a direct consequence of acute infection (Fig. 1).

Figure 1. Outcome tree, with health consequences and their mean frequencies, for non-congenital *Toxoplasma gondii* infections. The numbers in parentheses represent 90% credible intervals for each box in the tree



The annual medical cost for acute toxoplasmosis is estimated at AU$8.68 million (AU$1 million for mild cases and the balance for serious cases; Tables 1, S1, available as Supplementary material to this paper). The estimated annual cost of lost productivity as a result of toxoplasmosis is AU$67.5 million, with most of those costs arising from serious cases, particularly terminal cases (Tables 1, S1).

Table 1. Summary of the annual medical and lost-productivity costs from acute toxoplasmosis (in AU$ million)

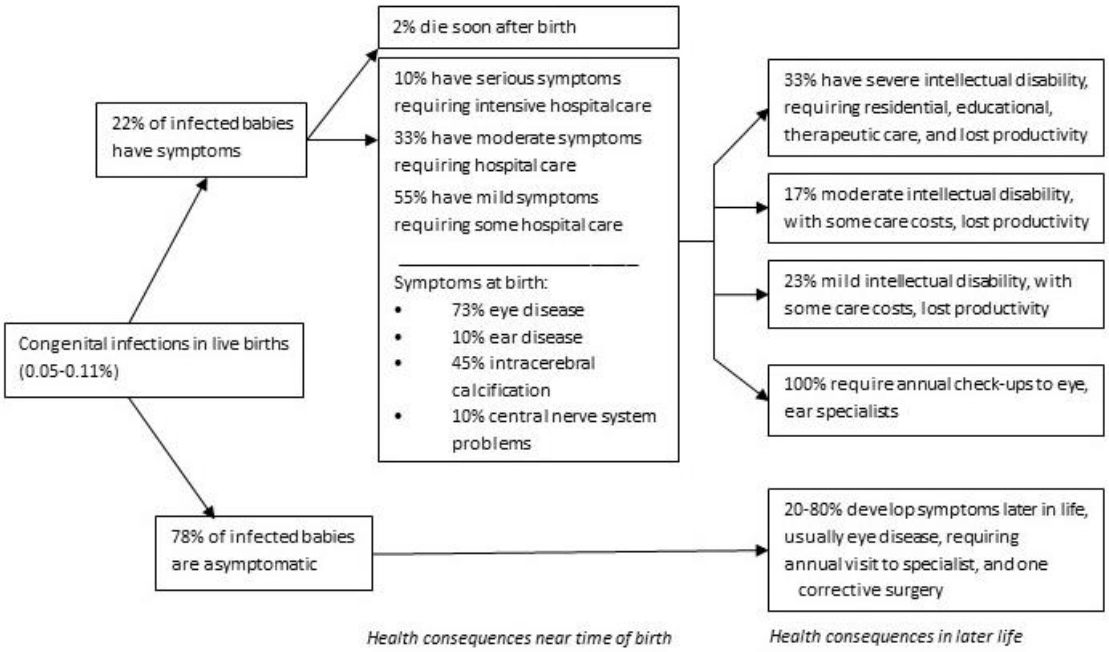
Lower and upper values around the estimate are based on the 90% credible intervals around the mean given by Scallan *et al.* (2011). Full details, including the sources for figures, are given in Table S1

Parameter	Estimate	Lower bound	Upper bound
Medical costs			
Mild cases	AU$1.00	AU$0.97	AU$1.02
Serious cases	AU$7.68	AU$4.54	AU$11.5
Lost-productivity costs			
Mild cases	AU$12.0	AU$11.7	AU$12.3
Serious cases	AU$55.5	AU$33.7	AU$81.9
Total	AU$76.2	AU$50.9	AU$107

Congenital infections. Our first estimate of the prevalence of congenital infection is based on three Australian studies that report infection rates in newborns, with results ranging from 0% (9037 babies, Munday 1978); to 0.016% (3 out of 18 908 babies, Walpole *et al.* 1991) to 0.12% (2 out of 1684 babies, Jayamaha *et al.* 2012). The mean of these three studies is 0.05%. Using the 2017 records for the number of livebirths in Australia, this equates to 140 infected newborns annually.

Our second estimate of the prevalence of congenital infection is based on demographic information, parasite prevalence in women, and transplacental transmission rates. The mean age of mothers giving birth in 2017 was 30 years (AIHW 2019*b*), and at this age 72% of mothers have not previously been infected with *T. gondii*, and are thus vulnerable to an initial infection during pregnancy (Johnson *et al.* 1980). The risk of contracting a *T. gondii* infection during 9 months of pregnancy is 0.53%, given that the annual increase in *T. gondii* infection rate in women of child-bearing age is 0.7% (derived from averaging two Australian studies that document age profiles for the prevalence of *T. gondii* antibodies in women between 15 and 45 (annual increase of 0.67%, Johnson *et al.* 1980); and (0.73%, Sfameni *et al.* 1986). The likelihood of transferring a new infection to the fetus depends on the stage of pregnancy, but overall, 29% of such maternal infections are transferred to the fetus (noting this may be an underestimate, because the mothers in the study were being treated for toxoplasmosis, Dunn *et al.* 1999). Thus, the frequency of fetal infection in vulnerable pregnancies is 0.15% (0.53% × 0.29%). Of the 309 142 births in 2017, we estimate the number of congenital *T. gondii* infections in 2017 was 339 (0.15% × 72% × 309 142), giving a prevalence of infection across all births of 0.11%, or 339 infected babies. We used these two estimates, of 140 and 339 congenitally infected babies per year, as the lower and upper bounds of a plausible range around their mean. The health consequences of congenital *T. gondii* infections and their mean frequencies are summarised in the outcome tree of Fig. 2.

Figure 2. Outcome tree, with health consequences and estimated frequencies, for congenital *Toxoplasma gondii* infections.



The annual cost incurred as a result of congenital *T. gondii* infection is estimated at AU$222 million (Tables 2, S2). The largest contributor to this total is from the costs of lost productivity in more severely affected individuals and their family carers. Although the number of severely affected individuals is small relative to the Australian population, the costs per case are high because costs

are incurred over entire lifetimes. Thus, small changes in the number of congenital *T. gondii* cases will have a large impact on the overall cost.

Table 2. Summary of the medical, ongoing care and lost-productivity costs (in AU$ million) from congenital *Toxoplasma gondii* infections

Lower and upper bound values are based on the range of plausible values for congenital infection rates, and the estimate is their mean. Full details, including the sources for figures, are given in Table S2

Parameter	Estimate	Lower bound	Upper bound
Number infections each year	240	140	339
Number symptomatic newborns	53	31	75
Number asymptomatic newborns that later develop health problems	93	37	150
Costs			
Medical care at birth and over lifetime	AU$2.57	AU$1.50	AU$3.63
Ongoing residential, educational and therapeutic costs (to family and government)	AU$1.90	AU$1.11	AU$2.69
Medical costs for late-onset health problems	AU$1.39	AU$0.55	AU$2.22
Lost productivity	AU$216	AU$127	AU$306
Total	AU$222	AU$130	AU$314

Latent infection, behaviour and mental health. The PAF of *T. gondii* to traffic accidents is 17% (95% confidence interval: 6, 29%) (Sutterland *et al.* 2019). In 2015 there were 1205 fatalities, 37 964 hospitalisations, and 227 575 non-hospitalised injuries due to traffic accidents (AAA ECON 2017). The costs of immediate and ongoing medical care, other costs (e.g. vehicle damage, insurance administration, disruption to workplaces, travel delays) and lost productivity totalled AU$18.9 billion in 2019 currency. In the absence of *T. gondii*, these traffic accident tolls would be reduced by 17% (Sutterland *et al.* 2019), hence we estimate the contribution of *T. gondii* to road trauma costs in Australia is AU$3.21 billion annually (Tables 3, S3).

Table 3. Summary of the costs (in AU$ million) from the impacts of *Toxoplasma gondii* infection on human behaviour and mental illness

Lower and upper bounds are the 95% confidence intervals around the mean estimate for the population-attributable fraction (PAF). Full details, including the sources for figures, are provided in Tables S3, S4 and S5.

Parameter	Total (AUS$ millions)	Estimate (PAF)	Lower bound	Upper bound
Traffic accidents		*(17%)*	*(6%)*	*(29%)*
Medical costs, direct and ongoing disability care	AU$3905	AU$664	AU$234	AU$1130
Other costs (e.g. travel delay, insurance admin, vehicle unavailability)	AU$11 690	AU$1690	AU$598	AU$2890
Costs to government	AU$3960	AU$673	AU$238	AU$11 150
Lost productivity	AU$1081	AU$184	AU$64.9	AU$314
Total	AU$18 910	AU$3210	AU$1130	AU$5480
Suicides and suicide attempts		*(10%)*	*(3%)*	*(19%)*
Direct costs of suicides	AU$901	AU$90.1	AU$27.0	AU$171
Lost productivity and cost to government (suicide)	AU$4670	AU$467	AU$140	AU$886
Direct costs of suicide attempts	AU$2090	AU$209	AU$62.8	AU$397

Lost productivity and cost to government (attempts)	AU$16 900	AU$1690	AU$508	AU$3220
Total	AU$24 600	AU$2460	AU$738	AU$4670
Schizophrenia		*(21.4%)*	*(13.7%)*	*(30.6%)*
Medical and crisis care costs of 3880 new cases of psychosis per year, each costing AU$47 604	AU$185	AU$39.5	AU$25.3	AU$56.5
Lost productivity costs for the year, each costing AU$47 840	AU$185	AU$39.7	AU$25.4	AU$56.7
Total	AU$370	AU$79.2	AU$50.7	AU$113

The PAF of *T. gondii* to suicide and suicide attempts is 10% (Sutterland *et al.* 2019). Over 2016–2018, there was an average of 3028 suicides, and 45 400 suicide attempts in Australia annually (Kinchin and Doran 2017; Mindframe 2019). The annual economic cost of suicide deaths and attempts in Australia is estimated at AU$24.6 billion. In the absence of *T. gondii*, these values would be reduced by 10%, or 303 deaths and 4540 attempts. Hence, the contribution of *T. gondii* to suicide and suicide attempts is estimated at AU$2.46 billion per year (Tables 3, S4).

The PAF of *T. gondii* to schizophrenia is 21.4% (confidence limits: 13.7–30.6%, Smith 2014). In Australia, schizophrenia affects seven people of every 1000 in their lifetimes, with a median incidence rate of new cases of 15.2 per 100 000 people per year (McGrath *et al.* 2008). For Australia's population (at 2019), this equates to 3880 new schizophrenia diagnoses annually. The annual cost of treating psychosis in Australia has been estimated at AU$95 444 per patient, including AU$47 604 per patient for medical care and other support, with the balance for lost productivity (converted to 2019 values; Neil *et al.* 2014). In the absence of *T. gondii*, the number of new schizophrenia cases each year would be reduced by 21.4%, or 813, with an economic saving of AU$79.2 million per year (Tables 3, S5).

Bartonella henselae

With an incidence rate of 10.5 (range 9–12) diagnosed illnesses per 100 000 people per year, and 10% of these being serious, 2680 (range 2290–3060) people would present with cat scratch illness annually, and 268 (range 230–306) of these would require hospitalisation. The costs of medical treatment are estimated at AU$2.79 million annually (range AU$2.39 million to AU$3.19 million), and the costs from lost productivity estimated at an additional AU$2.67 million annually (range AU$2.47 million to AU$3.29 million), giving a total cost of AU$5.45 million (range AU$4.86 million to AU$6.48 million; Table S6).

Cat-dependent pathogens of livestock

Toxoplasma gondii

With an overall spontaneous abortion rate in sheep of 1.75% (range 1.5–2%) per year, and with 16% of these abortions caused by *T. gondii* infection, and given annual lamb production of 22 million, then 62 300 lambs are lost annually because of *T. gondii* induced abortion. Given a cost of AU$160 per lamb, this represents a cost of AU$9.97 million (Tables 4, S7). This estimate masks disproportionately higher impacts in some regions with higher than average infection rates (Munday 1975; Taggart 2019). We note that despite the potentially serious consequences, toxoplasmosis is under-reported in livestock, and we lack estimates of rates for *T. gondii*-induced neonatal deaths, so our estimate for production losses due to *T. gondii* infections is conservative.

Table 4. Summary of the annual costs (in AU$ million) of *Toxoplasma gondii*-induced abortions to the sheep industry in Australia

The lower and upper bounds are based on the range of plausible abortion rates in sheep, and the proportion of these that are caused by *T. gondii*; the estimate is their mean. Full details, including the sources for figures, are given in Table S7.

Parameter	Estimate	Lower bound	Upper bound
Number of lambs slaughtered each year	22 million		
Abortion rate in sheep (%)	1.75	1.50	2
Proportion abortions caused by *T. gondii* (%)	17	11	23
Price for a 20-kg lamb	AU$160		
Number of lambs aborted per year	391 858	335 025	448 980
Number of *T. gondii*-induced abortions per year	62 305	36 853	103 265
Total	AU$9.97	AU$5.90	AU$16.5

Sarcocystis gigantea and S. medusiformis

We estimate a loss of AU$1.78 million annually from carcass condemnation and meat trimming as a result of macroscopic sarcocystosis (Table 5, S8). These lost production costs do not include a contribution to the costs of maintaining the meat-inspection system in abattoirs, or the extra labour to bone out infected meat.

Table 5. Estimates for the annual costs (in AU$) of macroscopic sarcocystosis to the sheep industry in Australia, by state

Mutton includes all animals >2 years of age and flocks of sheep with mixed age animals. The average trim weight for every affected carcass is 5.6 kg. Sources for figures are given in Table S8.

State	Age class	Number produced	Estimated proportion affected	Value per kilogram	Estimated cost in 2018
South Australia	Lamb	2 526 100	0.02	AU$7.96	AU$22 500
South Australia	Mutton	1 090 600	3.47	AU$5.58	AU$1 183 000
New South Wales	Lamb	5 032 100	0.02	AU$7.96	AU$44 900
New South Wales	Mutton	2 756 200	0.02	AU$5.58	AU$17 200
Victoria	Lamb	11 866 400	0.01	AU$7.96	AU$52 900
Victoria	Mutton	4 184 100	0.21	AU$5.58	AU$275 000
Queensland	Lamb	70 400	0	AU$7.96	AU$0
Queensland	Mutton	112 000	0.01	AU$5.58	AU$345
Western Australia	Lamb	2 822 900	0.01	AU$7.96	AU$12 600
Western Australia	Mutton	1 277 200	0.04	AU$5.58	AU$16 000
Tasmania	Lamb	406 900	0.26	AU$7.96	AU$47 200
Tasmania	Mutton	101 100	3.28	AU$5.58	AU$104 000
Total					AU$1.78 million

Summary costs

The costs of each cat-dependent disease to people and livestock are summarised in Tables 6 and S9.

Table 6. Summary of annual costs (estimate with plausible ranges in brackets, in AU$ millions), where estimation is possible, to human health and livestock production across four cat-dependent pathogens (*Sarcocystis gigantea* and *S. medusiformis* are considered together)
Where the cost of disease could not be estimated, the cell is marked as 'data deficient'. Where the disease is not applicable, the cell is left blank

Parameter	*Toxoplasma gondii*	*Bartonella henselae*	*Sarcocystis* spp.
Human health			
Acute Illness	76.2 (50.9–107)	5.46 (4.86–6.48)	
Congenital infection	222 (130–314)		
Behavioural impacts			
Car accidents	3210 (1130–5480)		
Other accidents	Data deficient		
Suicide, attempted suicide	2460 (738–4670)		
Schizophrenia	79.2 (50.7–113)		
Other mental illness	Data deficient		
Livestock production			
Sheep	9.97 (5.9–16.5)		1.78
Goats, other livestock	Data deficient		
Overall total		6070 (2120–10 700)	

Discussion

We estimated the economic cost to human health and livestock production of four cat-dependent pathogens (*T. gondii*, *S. gigantea*, *S. medusiformis*, *B. henselae*) that were introduced to Australia with the domestic cat, at AU$6.07 billion per year (range AU$2.12 billion – AU$10.7 billion; Table 6). We were unable to estimate the costs of a fifth pathogen, *Toxocara cati*. Estimating the economic cost of pathogens is often challenging. For the cat-dependent pathogens of interest to our study, infection and disease rates are often uncertain because of under-diagnosis and under-reporting (Buzby and Roberts 2009; Dubey 2009*b*). Changes to key estimates such as prevalence rates, congenital transmission rates, and frequencies of disease outcomes would affect our cost estimates, although we attempted to capture this uncertainty using value ranges where possible. We estimated costs for only a subset of the human-health and livestock-production impacts; for example, we considered only a subset of the mental health consequences that may be amplified by *T. gondii* infection, and we considered the costs of *T. gondii*-induced abortion only in sheep, although other livestock are affected. We were unable to collate the costs to government across all diseases, nor did we consider the costs of preventative care and regulatory practices to limit pathogen prevalence, nor the costs of research to improve our response to disease. Given these complexities, the true cost of cat-dependent pathogens may exceed the figures we compile here. Conversely, causality between *T. gondii* and human behaviours and mental health has yet to be conclusively proven, and so these costs for cat-dependent pathogens may be inflated. Below, we discuss the costs associated with each pathogen in turn, and identify the limitations of our estimates and areas for future research. We then consider the costs of the cat-dependent pathogens in the context of

the other economic costs (and benefits) of cats. Finally, we discuss some options for reducing the incidence of cat-dependent pathogens in people and livestock.

Toxoplasma gondii

Toxoplasma gondii causes the most significant human health outcomes of all the cat-dependent pathogens. We estimated the costs of medical care, social support, insurance and lost productivity to be AU$6.06 billion annually (AU$2.11–10.7 billion; Table 6). A large proportion of these costs is based on research demonstrating causal links between *T. gondii* infection and behavioural and mental health impacts, resulting in higher rates of car accidents, suicide and schizophrenia. These associations, and the estimates for the PAFs, rest on robust meta-analyses across many studies (Torrey *et al.* 2012; Smith 2014; Sutterland *et al.* 2019). However, causality is still uncertain and associations between *T. gondii* and mental health and behaviour could be underpinned by other common factors. For example, in Brazil, rates of traffic accidents and suicide are both higher in lower-income households, as is the risk of acquiring a *T. gondii* infection; so, perhaps, at least some of the relationship between *T. gondii* and accidents or suicide is partly explained by the common factor of low income (Mareze *et al.* 2019). However, the consistency and strength of the correlations, the evidence from experimental animal models, and the plausibility based on the understanding of how *T. gondii* affects concentrations of brain chemicals and the immune response, provide support for causality (Smith 2014; Vyas 2015; Sutterland *et al.* 2019; Boillat *et al.* 2020).

Comparing the costs of human *T. gondii* infections in Australia to estimates from other countries is difficult, given the age of some of these studies, the variation in health costs among countries, and the different methods used to estimate cost. For example, the costs of congenital *T. gondii* infections were estimated in the UK in 1980 (GBP£4.8 million; Henderson *et al.* 1984), and in the USA in the 1980s (US$0.4–8.8 billion per year, with the wide range owing to uncertainties about congenital transmission rates, Roberts and Frenkel 1990). An updated analysis of the US data produced an overall cost estimate for congenital toxoplasmosis of US$5.3 billion per year (Roberts *et al.* 1994), equivalent to ~AU$47 per capita in 2019 currency. These values suggest that our cost estimate for congenital infections in Australia (AU$222 million annually overall, or AU$9 per capita) is plausible or even conservative. Scharff (2012) derived an overall annual cost of acute *T. gondii* illness in the USA of US$3.5 billion (with a large confidence interval of US$1–6.6 billion). This is equivalent to AU$19 per capita in 2019; our estimate for the cost of acute toxoplasmosis is AU$76.2 million, equivalent to AU$3 per capita, again suggesting that our estimate is plausible or conservative. To sidestep the issues of trying to harmonise medical costs that vary considerably among countries, disease burden is sometimes estimated using disability adjusted life years (DALY), a unit that sums the years lost as a result of premature death or disability. Globally, the disease burden of *T. gondii* is estimated at 2–8 million DALYs; the comparable figure for malaria is 39 million DALYs (Torgerson and Macpherson 2011), reinforcing that the economic cost of *T. gondii* to human health is substantial.

In livestock, *T. gondii* is again the cat-dependent pathogen with the largest economic cost in Australia, primarily through reductions in lamb production, estimated at AU$9.97 million annually. The costs of *T. gondii* infection to sheep production are of comparable magnitude in other countries. In New Zealand, with lamb production rates similar to those in Australia, Charleston (1994) estimated that *T. gondii* costs AU$25.1 million (equivalent to NZ$14 million in 1992) annually, on the basis of a 2% median increase in lambing success rates after vaccination trials were conducted across many flocks. Bennett *et al.* (1999) estimated a cost of AU$56 million per year (accounting for inflation since 1999) to sheep production in the UK, and Freyre *et al.* (1997) estimated an annual cost of AU$11.8 million in Uruguay (again, converted into today's currency value).

Sarcocystis gigantea and S. medusiformis

We estimated that macroscopic ovine sarcocystosis costs the Australian meat industry AU$1.78 million annually. This figure lies between two other evaluations of national impact. Lane *et al.* (2015) estimated that across Australia, 0.01% of adult sheep carcasses were condemned and 0.3% of sheep carcasses were subject to trimming, amounting to annual lost production costs of AU$0.9 million. Animal Health Australia suggest a cost of AU$4.9 million per year, but without presenting the underlying data (NSHMP 2018).

Because the incidence of *Sarcocystis* spp. varies geographically, the costs of sarcocystosis are also concentrated regionally, and local-scale impacts may be proportionally much greater than the national values imply. If a high incidence of macroscopic sarcocystosis in a flock is suspected, some abattoirs refuse to receive and handle entire consignments, which could have serious impacts for local producers (Taggart *et al.* 2019*b*). Kangaroo Island is a region with a high incidence; the density of sarcocystosis-positive farms is 15 times greater on the island than on the adjacent mainland (Taggart *et al.* 2019*b*), and visible cysts on the oesophagus have been reported on 66% of slaughtered adult sheep (Taggart *et al.* 2020). Unfortunately for farmers, the prevalences of *Sarcocystis* spp. and *T. gondii* in sheep flocks are correlated, possibly because the survival of the parasites in the environment is favoured by similar conditions and these species share a common definitive host (Taggart *et al.* 2019*a*, 2020). Tasmania appears to be another hotspot for both *Sarcocystis* spp. and *T. gondii* (Hernandez-Jover *et al.* 2013; DPIPWE 2016; Phythian *et al.* 2018).

Bartonella henselae

Our estimate of an annual cost of AU$5.46 million for *B. henselae* infections, or about AU$2040 per case, is comparable to the only other national estimate we are aware of, from the USA in the 1980s, which reported an annual cost of US$12 million (1992 currency), or AU$1400 per case in today's currency (Jackson *et al.* 1993). We note that cat scratch disease is heavily under-reported, and the range of recognised symptoms is expanding as diagnostic techniques for the pathogen improve (Breitschwerdt 2008; Chomel and Kasten 2010). In Australia, antibodies to *B. henselae* were found in 5% of a small sample of blood donors (Gilbert *et al.* 1997), suggesting much higher exposure rates than what medical records indicate (1.28 million exposed, or ~16 000 exposures a year, assuming a lifespan of 80 years, in contrast to the 2680 annual cases we used as the basis of our estimation). Although the sample in the study by Gilbert *et al.* (1997) was small, it accords with reported seroprevalence rates of up to 6% in the USA, Japan, Thailand and Europe (Chomel *et al.* 2006).

Taxocara cati

We were unable to find estimates for the incidence of *T. cati* infections, as distinct from *T. canis* infections, in either people or livestock, and, thus, we were unable to separate the cost of their impacts. We suspect the health and cost impacts are modest at the national scale compared with other cat-dependent pathogens, but prevalence in both cats and people is known to be demographically and geographically clustered. For example, children are more likely to pick up infections because they play in gardens and sandpits and readily transfer soil to their mouths (Uga *et al.* 1996). One survey across multiple locations estimated prevalence in cats of less than 4% (Palmer *et al.* 2008). However, a survey of cats in Indigenous communities in the Kimberley region of Western Australia found a prevalence of 18.2%, suggesting that people in these remote communities may be at a higher risk (Meloni *et al.* 1993).

Other parts of the balance sheet

We have focussed the present study on cat-dependent pathogens that were absent in Australia before the introduction of the cat. Cats carry many other viral, bacterial, fungal and parasitic zoonoses that also occur in other reservoir species; however, the high contact rates between cats and people can facilitate high transmission of these pathogens, such as methicillin-resistant *Staphylococcus aureus*, *Capnocytophaga* spp., *Cryptosporidium* spp., *Giardia* spp., ringworm-causing fungi, and *Sarcoptes scabei* (scabies; Chomel and Sun 2011; Day *et al.* 2012; Chomel 2014). We have not attempted to estimate the fraction of these zoonoses that are transmitted by cats, but are not dependent on them. Cats are also a potential reservoir for serious diseases not yet present in Australia, such as plague, bird flu and rabies (Chomel 2014).

We also spend a substantial amount of money on cat care annually. In Australia, cat owners collectively spend about AU$3.6 billion per year on food, veterinary care and other care products for their pets (AMA 2019). We devote a substantial proportion of our resource-growing efforts to supporting cats; a recent analysis suggested that a pet cat requires the equivalent of 0.4–0.6 ha of agricultural land to produce its dry cat food for a year (Su *et al.* 2018). Australian conservation agencies spend considerable sums on feral cat management, with actions ranging from building cat-proof fencing, to aerial deployment of poison baits over large areas, to intensive trapping and shooting programs at key sites. The costs of some of these actions have been summarised in Woinarski *et al.* (2019), but the cumulative cost of these actions implemented at the national scale has not been synthesised. Cats also have economic impacts by preying on native species that provide benefits to agriculture (e.g. insectivorous birds), and by degrading natural values that are attractive to ecotourists and birdwatchers (Pimentel *et al.* 2005).

Of course, pet cats also have positive impacts on human health and the economy. Comparative surveys suggest that pet owners can have reduced cardiovascular risks, better post-surgery recovery, and better general physical and mental health than do people without pets (Wood *et al.* 2005; Barker and Wolen 2008; Serpell 2015). Long-term studies show that people with pets make fewer visits to the doctor (Headey *et al.* 2002). Pets may help reduce loneliness and improve social connection (Wood *et al.* 2005; Krause-Parello 2012). Experimental studies have suggested that pets improve the mood and social behaviour of patients with a range of psychiatric conditions, including helping children with social behaviour and developmental disorders (Barker and Wolen 2008; Friedmann *et al.* 2015). The saving to the Australian health system from pet ownership has been estimated at AU$3.9 billion annually (Headey *et al.* 2002), with an unknown fraction attributable to pet cats. However, the health benefits described above come from owning and interacting with any pet.

Cats provide little or no economic benefit to livestock production. Farmers sometimes encourage cats to occupy farm dwellings in the hope they will control rodent populations, and feral and stray cats consume large numbers of small ‘pest’ animals such as rabbits and introduced rodents (Murphy *et al.* 2019). However, there is little evidence that predation by pet or feral cats has a significant controlling impact on the population size of (or the economic impact caused by) such introduced mammal species (Elton 1953; Newsome *et al.* 1989; Pech *et al.* 1992; Cruz *et al.* 2013; Parsons *et al.* 2018).

Limitations and future research

Our collation highlights substantial knowledge gaps in our understanding of the impacts and costs of cat-dependent diseases. Untangling causality in the relationships between *T. gondii* and human

behaviour and mental health would better resolve the true costs of this pathogen, and, therefore, the benefits of using more concerted approaches to reduce infection rates in people. Similarly, better documentation of the acute illnesses caused by *T. gondii* infection, and the rates and consequences of congenital infection, are needed to evaluate the benefits of investments to reduce *T. gondii* prevalence. Our estimates for the incidence, and, thus, costs, of *B. henselae* may be too low; improvements in the diagnosis and reporting of cat scratch disease, as well as for *T. cati* infections, are required.

There are indications that cat-dependent diseases may be more common in Australia's remote Indigenous communities, on the basis of studies reporting higher rates of *T. gondii*-related eye disease, and high rates of *T. cati* infections in cats, at some communities (Meloni *et al.* 1993; Thompson *et al.* 1993; Palmer *et al.* 2008; Henderson 2009; Chang *et al.* 2012). Further research to confirm how widespread these patterns are, and their key contributing risk factors, could help reduce disease in these vulnerable communities, particularly given that the number of pet cats living in many Indigenous communities is increasing (Kennedy *et al.* 2020).

Reducing impacts and costs

A key risk factor for contracting cat-dependent pathogens, for both people and livestock, is exposure to cats. Although owners of pet cats may be more exposed to cat-dependent diseases, people without pet cats can also be exposed to feral cats or roaming pet cats (Legge *et al.* 2020). Pet cat owners are more likely to contract cat roundworm infections (Jarosz *et al.* 2010), and contact with cats is associated with a higher incidence of cat scratch disease (Flexman *et al.* 1997). *Bartonella henselae* is more common in the high-density cat populations of urban areas, increasing its risk of transmission to people (Bevins *et al.* 2012). *Toxoplasma gondii* prevalence in people depends on many factors, including cooking practices and climate, but is positively associated with cat exposure (Tenter *et al.* 2000; Jones *et al.* 2009; Dickson 2018; de Wit *et al.* 2019). *Toxoplasma gondii* and *Sarcocystis* spp. infections are higher in sheep in regions with a high cat density (Taggart *et al.* 2019*b*), and possibly in sheep-growing areas near towns that also support higher cat densities (Animal Health Australia 2019).

Common advice for reducing the risk of being infected by *T. gondii* and *T. cati* includes improving food-preparation and cooking habits, and hygiene practices during and after gardening and handling kitty litter (Dubey 2016). However, these approaches appear much less effective at decreasing the incidence of cat-dependent diseases than does eliminating contact with cats. For example, *T. gondii* is absent from islands that lack cats (Wallace *et al.* 1972), and *T. gondii* infection rates in pigs in Europe decreased during the 1990s from rates of 20–60% to under 1% as intensive farming practices were adopted that prevented pigs from accessing areas with cats (van Knapen *et al.* 1995; Tenter *et al.* 2000; Dubey 2009*a*).

As the human population grows, the population of cats (both pet and feral) living in heavily modified urban and rural areas also grows (Su *et al.* 2018; AMA 2019), increasing the opportunities for cat-dependent pathogen transmission, and thus increasing the health impacts and costs associated with their diseases (Lafferty 2005). Within cats, the prevalence of cat-dependent pathogens may generally be higher in feral than pet cats. This seems to be the case for *T. cati* (Fisher 2003), *B. henselae* (Branley *et al.* 1996; Boulouis *et al.* 2005; Chomel *et al.* 2006) and *T. gondii* (Wu *et al.* 2011).

Minimising the density of cats living around farms and in our towns and cities could reduce disease incidence. The size of the pet cat population could be reduced if potential pet owners opted not to

have a pet cat. Management options for reducing the population of feral cats include the following: preventing them from accessing superabundant food (e.g. fencing off rubbish dumps); avoiding supplementary feeding of feral ('stray') cats; strategic physical barriers or trapping; shooting or poisoning programs in and around areas of human and livestock habitation; and enhanced management of pet cats to stop leakage of pets into the feral population (Woinarski *et al.* 2019). Actions to interrupt disease-transmission pathways (for pets and feral cats) are important, such as preventing cats from accessing areas used for livestock production, vegetable gardens and children's sandpits. Finally, keeping pet cats indoors, or in secure outdoor areas, will lower the risk that a pet cat will contract pathogens that they can pass on to people (Must *et al.* 2015; Read 2019).

Conflicts of interest

Sarah Legge is an associate editor for *Wildlife Research* and was the guest Editor-in-Chief for this special issue. John Woinarski and Chris Dickman were also guest editors for this special issue. Despite this relationship, they did not at any stage have editor-level access to this manuscript while in peer review, as is the standard practice when handling manuscripts submitted by an editor to this journal. *Wildlife Research* encourages its editors to publish in the journal and they are kept totally separate from the decision-making process for their manuscripts. The authors have no further conflicts of interest to declare.

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