A QUANTITATIVE RISK ASSESSMENT FOR HUMAN *TAENIA SOLIUM* EXPOSURE FROM HOME SLAUGHTERED PIGS IN EUROPEAN COUNTRIES

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ABSTRACT

Taenia solium (*T. solium*) is a zoonotic tapeworm that is responsible for about a third of all preventable epilepsy in humans, mainly in developing countries. In Europe, adequate biosecurity of pig housing and proper meat inspection management have decreased the incidence of *T. solium* taeniosis and cysticercosis. Pigs that are slaughtered at home may be raised poorly and not undergo meat inspection. As a result, home slaughtered pork could be a risk factor for exposure to *T. solium*. The aim of this study was to quantify the risk of human *T. solium* exposure from home slaughtered pork, in comparison to the risk from controlled slaughtered pork, in European countries. A quantitative production-to-consumption risk assessment model (QMRA) was developed. Porcine prevalence data, the percentage of pigs slaughtered at home, sensitivity of meat inspection, the cyst distribution in pork and pork consumption in five different European countries were included. This was combined with literature about cooking of pork, to calculate the number of infected pork portions eaten per year in a country. Recognizing the uncertainties in the data, the model still clearly shows a ten times higher prevalence of infected portions from home slaughtered pork compared to controlled slaughtered pork. This difference is brought about by the higher prevalence of cysticercosis in the pigs that are home raised and slaughtered. Meat inspection does not affect the higher exposure from home slaughtered pork, because the sensitivity of meat inspection is low in general when pigs have a mild infection. The model demonstrates that cooking meat effectively decreases the number of infected pork portions and thus lowers the risk of exposure. Besides the findings, this QMRA has shown the knowledge gaps and what kind of future research is needed to improve the QMRA. This includes systematically reporting porcine cysticercosis cases in slaughterhouses and studies on raw meat consumption in different countries and cultures. Moreover, developing a dose response model for *T. solium* to estimate the incidence of human taeniosis is recommended. When more data becomes available, this QMRA model could be implemented in intervention strategies concerning *T. solium* in Europe and beyond.

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INTRODUCTION

Taenia solium is a zoonotic tapeworm, with pigs as intermediate hosts and humans as definitive hosts. The adult tapeworm manifests in the human intestines, causing taeniosis. Thousands to ten thousands of eggs are excreted during defecation. Exposure of pigs to the eggs is enhanced by unhygienic conditions, for example when the human tapeworm carrier defecates outdoors, and thereby contaminates vegetables, running waters or soils [1]. When pigs ingest the eggs, oncospheres hatch from them, penetrate the intestinal walls and migrate towards the muscles. The oncospheres develop into cysticerci within 60 to 70 days [2]. The cysts can survive in striated muscles for weeks to years. It is assumed that pigs do not experience any clinical symptoms from the infection. Humans may become infected when pork with cysticerci cellulosae is eaten raw or undercooked [3]. Human taeniosis is often undiagnosed, with only abdominal pain and bloating as reported symptoms [4].
Besides as end hosts, humans can occasionally serve as intermediate hosts. Humans can obtain cysticercosis from direct contact with tapeworm carriers, contaminated food, or autoinfection [5]. The oncospheres might migrate towards the striated muscle as it occurs in pigs, but other known humane predilection sites are the eyes, subcutaneous tissue and brain. In contrast to human taeniosis, human cysticercosis causes major health problems. Neurocysticercosis (NCC) is a severe form of human cysticercosis, localized in the central nervous system. NCC is responsible for almost a third of all preventable epilepsy cases in the developing world [6]. The symptoms most frequently found in NCC patients are seizure, epilepsy, perilesional brain edema, intracranial hypertension, vascular damage, stroke, cognitive deficit and depression [4]. The Food and Agriculture Organization (FAO) has put *T. solium* highest in a multicriteria based ranking of 24 food-borne parasites of global importance, where public health, animal health, microbial ecology, agribusiness, trade and socio-economic impact have been considered [7]. The reason of the ranking is the large impact of the tapeworm on public health and the endemicity of *T. solium* in many regions of the world, such as Latin America, Sub-Saharan Africa and Asia [6].

The risk factors for human cysticercosis include poor personal hygiene, poor pig-raising practices [8], a lack of potable water and sanitary latrines [9], consumption of infected, undercooked pork and poor knowledge about cysts in meat products [8, 10]. In Europe, 4% of all pig holders raise 91% of all pigs [11]. These farms all hold at least 200 pigs and have a biosecurity that is designed to minimize the transmission of pathogens like *T. solium*. Besides the structure and hygiene of European farms, meat inspection is obligatory at slaughterhouses in the European Union (EU), according to European Regulation 854/2004, chapter IV [12]. As a result, every pig carcass in the slaughterhouse is being checked for cysticerci. Since almost no cases are reported in Europe[13], *T. solium* seems to be only a minor foodborne agent in Europe. Nevertheless, various recently published papers conclude differently [14-18]. Systematic review on the epidemiology of *T. solium* and *Taenia saginata* (*T. saginata*) shows that *T. solium* taeniosis is diagnosed in 7 out of 18 countries in Western Europe. Human cysticercosis was even reported in all countries except Iceland. Most of these patients have visited endemic countries, which might explain the acquired infection. But there are also patients that have never left their country [15, 17]. As explained before, humans can transmit eggs to others, so autochthonous cysticercosis cases, could come from travelers with a taeniosis infection. But, this does not explain the porcine cysticercosis, that is notified in Austria, Bulgaria, Germany, Poland, Romania, Serbia and Spain, all between 1999 and 2015 [14, 15, 18].

Apparently, the conditions necessary for the transmission of *T. solium* between pigs and humans are still present in some European countries. Risk factors that are considered to influence this are human migration and travel towards the EU and the increasing trend in pig farming with

outdoor access [19]. Another risk factor is improper or lack of meat inspection. This is for instance possible when pigs are held and slaughtered at home.

Cystinet, the scientific European network on taeniosis and cysticercosis, aims to advance the knowledge on the zoonotic disease complex, by collaborations within Europe. The COST action (European cooperation in science and technology) consists of three working groups. The one that focuses on control and prevention addressed the question if home slaughter could be a risk factor for the spread of *Taenia spp.* In response, an online questionnaire was developed to identify home slaughter practices and meat inspection management in Europe [20]. The questionnaire was forwarded to experts on the topic in every country by COST members and response was received from 21 European countries. It resulted in a better insight into the slaughter practices in the European Union. In particular, home slaughtering of pigs is allowed in all countries, mainly for own consumption, but the order of magnitude varies considerably and was often denoted unknown. Moreover, thirteen countries answered that meat inspection of home slaughtered pigs is not applied. So, home slaughter of pigs, without adequate meat inspection, could be a relevant risk factor, which should be further assessed. The aim of this study is to give a quantitative estimation of the risk of *Taenia solium* exposure from home slaughtered pork in European countries, in comparison to controlled slaughtered pork. This was done by means of a quantitative production-to-consumption risk assessment model (QMRA).

METHOD

The QMRA model followed the steps from porcine cysticercosis prevalence up until exposure of humans to infected pork portions. Firstly, a general description of the steps taken in the model is given (Figure 1). Secondly, the data sources and the calculations necessary to assess the risk of exposure per country are described thoroughly. The calculation steps are visualized in Figure 2.

MODEL DESCRIPTION

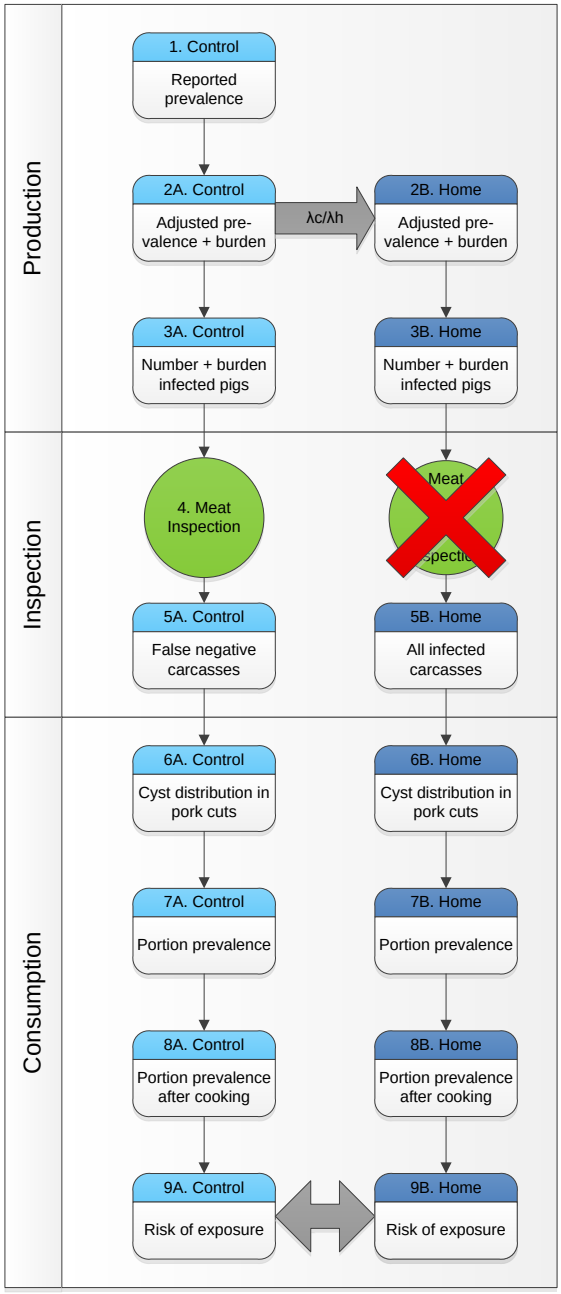

FIGURE 1 CONCEPTUAL RISK CHAIN *T. SOLIUM* EXPOSURE

The model was divided in three subsections: production, inspection and consumption. All steps were made at country level. The following steps were included (Figure 1): 1. The model sets off with the reported prevalence in pigs. 2A. With calculations to determine the exposure rate and sensitivity of meat inspection the adjusted prevalence and burden of infection of the porcine cysticercosis cases was defined. 2B. The adjusted prevalence and burden of home slaughtered pigs was obtained using prevalence data of a country where home slaughtered pork meat is inspected. 3. National data of the number of pigs slaughtered in slaughterhouses and outside slaughterhouses was multiplied by the prevalences of porcine cysticercosis to calculate the number of infected pigs for both controlled conditions (control) and home slaughtered conditions (home) apart. 4. Meat inspection, with a certain detection sensitivity was included in the 'controlled' branch of the model. For the 'home' branch, a comparison was made between leaving meat inspection out and including meat inspection practices. 5. All carcasses which tested false negative were not withdrawn from the food chain and passed on to the section consumption. 6. With the aid of the burden of the infected carcasses and the cyst distribution in pork cuts, the probability of a cyst to enter a cut[1] was predicted. 7. The weight of the cuts and a standard portion size were obtained to calculate the cyst distribution of the portions. By taking into account the total number of portions eaten in a country in a year, the portion prevalence and total number of infected portions could be obtained. 8. A subdivision between portions cooked and portions eaten raw was estimated. The portions eaten well-cooked were assigned zero risk, to calculate the final portion prevalence after cooking and thus to calculate 9. The risk of exposure.

[1] By "cut" we denote an anatomical part of the pig, such as "brain", "loin", etc.

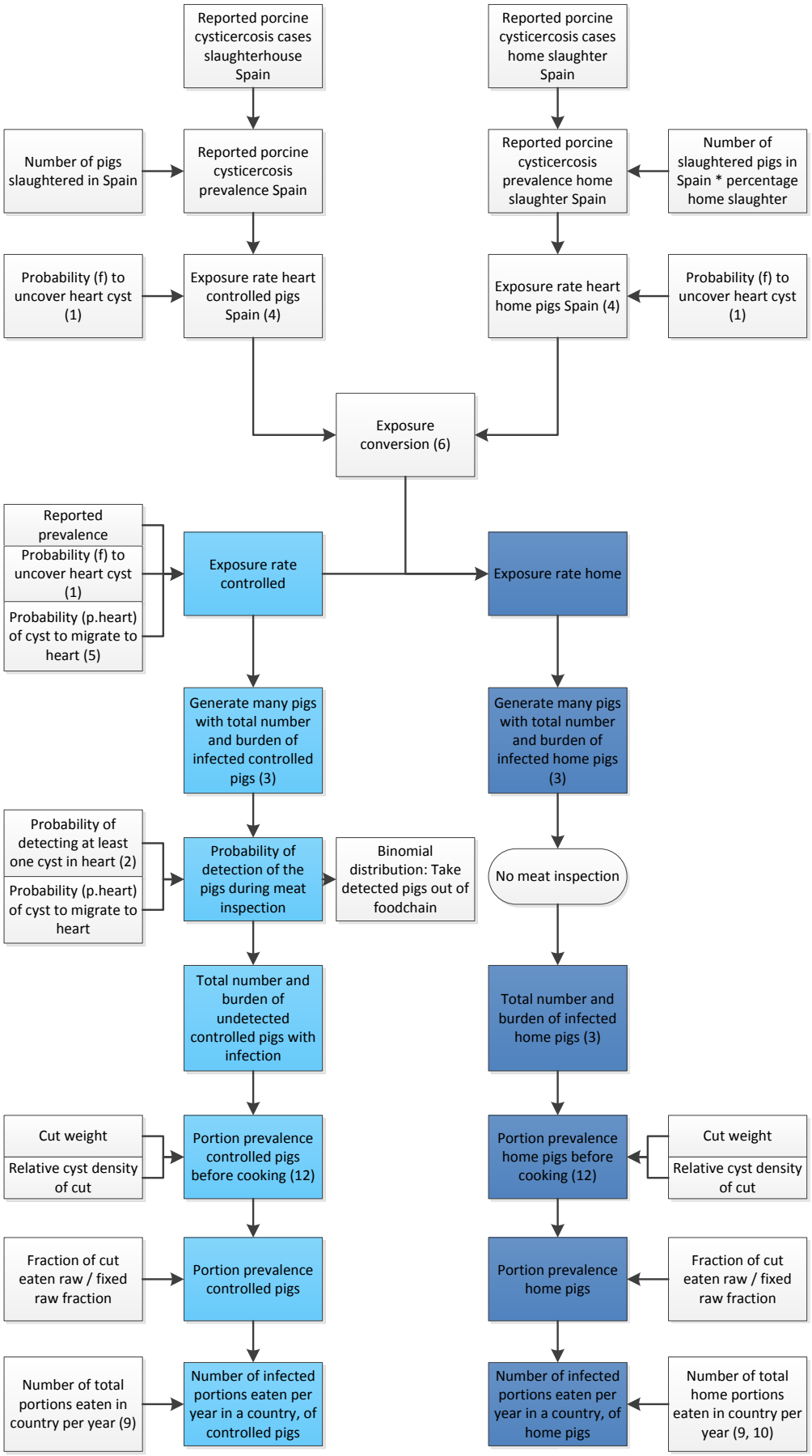

FIGURE 2 MODEL LAYOUT (FORMULA NUMBERS BETWEEN BRACKETS)

DATA SOURCES AND CALCULATIONS

TEST SENSITIVITY MEAT INSPECTION

Official European examination of swine carcasses is described in chapter IV of the European Regulation 854/2004 [12]. This regulation lists all organs and muscles that need to be visually inspected. Regarding *T. solium*, the organs that matter are the tongue, diaphragm, pericardium and heart. The heart has to be incised lengthwise once, in order to view the ventricles and septum of the heart. As only the heart is cut to detect cysts, we assumed that the cut in the heart is the basis of European meat inspection for *T. solium* and that the other organs named are not involved in meat inspection.
Research about meat inspection concerning *T. solium* shows that the inspection sensitivity depends on the pig's burden of infection [21]. In other words, the probability of a cyst to be found in the heart becomes higher when the heart contains more cysts. We used this fact to calculate the detection probability of a heart cyst. The probability (f) to uncover a single cyst in the heart is estimated, using formula 1.

$$f = \frac{\text{Mean heart surface revealed by meat inspection } (cm^2)}{\text{Mean heart surface revealed by total slicing } (cm^2)} \quad (1)$$

The surface revealed by meat inspection is the area that can be inspected after the lengthwise incision mentioned above. Total slicing is the golden (standard) method to find cysticerci cellulosae. Organs and muscles are sliced in 0,5 cm thick slices, so that all cysts are uncovered. So, total slicing gives the largest possible area that can be checked for *T. solium* cysts. The surfaces of formula 1 are adopted from another paper [22].

The probability to find at least one cyst in the heart during detection was obtained with formula 2. When the total number of cysts in the heart (n_{heart}) increases, the detection probability follows.

$$P(\text{detect} > 0 \text{ cysts} \mid \text{cysts} = n_{\text{heart}}) = 1 - (1 - f)^{n_{\text{heart}}} \quad (2)$$

EXPOSURE RATE AND INFECTION BURDEN

The exposure of pigs to *T. solium* eggs depends on certain risk factors that differ between countries, or even regions. We supposed that pigs are exposed to the eggs, resulting in an exposure rate (λ_{heart}), of eggs in the heart per lifetime. The probability of having an infection with n_{heart} cysts was described by a Poisson distribution (formula 3), which is the appropriate distribution for events that happen at random with a constant rate to an individual, i.e. an animal [23]. A higher exposure to eggs gives a higher probability of infection and a higher burden of infection. When the exposure rate of a country is known, the formula can be used to determine the adjusted number and burden of infected pigs with cysticercosis in that country.

$$P(\text{cysts} = n_{\text{heart}}) = \text{Poisson}(\lambda_{\text{heart}}) = \frac{\lambda_{\text{heart}}}{n_{\text{heart}}!} e^{-\lambda_{\text{heart}}} \quad (3)$$

Note that the exposure rate is the rate of exposure of the heart, since this is the muscle that the prevalence is derived from. Later we will introduce scaling factors to derive burdens in other muscles. When combining formula (2) and (3), the following formula results:

$$P(\text{detect}) = 1 - e^{-f\lambda_{\text{heart}}} \quad (4)$$

P(detect) is the probability to find a positive pig, given a certain *f* and λ_{heart}.

This probability of finding a positive pig is analogous to the reported prevalence in European countries, as the sensitivity of meat inspection and the exposure rate lead to found cases in the slaughterhouse.

We entered *f* and the reported prevalences as *P*(detect) in formula 4, yielding λ_{heart} for each country. To derive λ_{pig}, the exposure rate of the whole pig instead of the heart, the exposure rate was divided by the probability of a cyst to enter the heart (p_{heart}) (formula 5). Derivation of p_{heart} is further described under 'Cyst distribution and weight of pork cuts'.

$$\lambda_{pig} = \frac{\lambda_{heart}}{p_{heart}} \quad (5)$$

A binomial distribution was used to find all infected and non-infected pigs in the model, with *n* the number of pigs and *P*(detect) (Formula 4) the probability of detection.

PREVALENCE

The reported prevalences mentioned above were acquired in three steps. In the first place, the number of porcine cysticercosis cases per country was adopted from two reviews about the epidemiology of *T. solium* and *T. saginata* [15, 18]. An additional literature search was done for European countries that were lacking from the reviews [24, 25]. In the second place, for all countries that reported an annual number of cases but no total number of tested pigs, the total number of pigs slaughtered in slaughterhouses was taken from Eurostat [26]. In the third place, the annual number of cases was divided by the annual number of slaughtered pigs to generate a prevalence of reported cases. This is the controlled reported prevalence, because all reported cases were found in slaughterhouses [15, 18, 24, 25].

As was already stated, the adjusted number of infected pigs was calculated with the Poisson distribution (formula 3). The adjusted number of controlled infected pigs was divided by the total number of pigs assessed to obtain the adjusted prevalence in a controlled setting.

Home slaughtered pigs have a high chance to have been reared in uncontrolled housing. This could imply that home slaughtered pigs have also had a higher exposure to *T. solium* in their lives. This assumption is supported by data from Spain, where home slaughtered animals are inspected according to the same method as regularly slaughtered animals. Although the same method is applied, the reported prevalence in Spanish pigs under controlled conditions ranges from 0.02% to 0.03%, while amongst home slaughtered pigs, a prevalence of 0.16% to 0.43% is recorded (2011-2013) [15]. The ratio between controlled and home reported prevalence in Spain was used to calculate the home prevalence in other countries in our model.

Initially, the controlled and home reported prevalence of Spain were entered in formula 4, attaining two exposure rates, the controlled exposure rate called λ^{c}_{heart} and the home exposure rate λ^{h}_{heart}. These were divided by p_{heart} to obtain λ^{c}_{pig} and λ^{h}_{pig} as explained before (formula 5).

Formula 6 demonstrates the step to the exposure conversion. The exposure conversion was applied in the model for all countries to convert the adjusted controlled prevalence in the adjusted home prevalence.

$$\text{Exposure conversion} = \frac{\lambda^{c}}{\lambda^{h}} \quad (6)$$

NUMBER OF SLAUGHTERED PIGS

The database Eurostat records the annual number of slaughtered pigs per European country, as well as the number of pigs slaughtered at other places than the slaughterhouse [26, 27]. Slaughtering 'outside the slaughterhouse' was adopted as home slaughtering in our calculations. The yearly slaughter records taken into account are the same years for which the national number of porcine cysticercosis cases is known. The average of these years was used in the model to calculate an average prevalence.

CYST DISTRIBUTION AND WEIGHT OF PORK CUTS

The distribution of *T. solium* cysticerci in pig carcasses is not homogeneous. The predilection places described are for instance the pork shoulder, pork leg and psoas muscle [28]. To take into account the cyst distribution in the model, literature data was used [22]. In a paper of Boa et al. naturally infected pigs were slaughtered and in every half carcass the cysts per muscle group or organ were counted by the total slicing method illustrated before. The average amount of cysts per cut was divided by the average total cysts of the 24 pigs. The mean percentage of total cysts in the cut was divided by the mean percentage of the weight of that cut to calculate the relative cyst density [22]. The relative cyst density is the probability of a cyst to enter a cut. The relative cyst density of the heart was used in formula 5 as p_{heart}. Also the relative cyst density was used in a binomial function that is defined in the paragraph 'Cysts per consumed portion'.

The weight of the pork cuts was not available from literature. Only the weights relative to the average carcass weight were given (Mean Weight %) [22]. To obtain the actual cut weights in kilograms, literature about porcine brain weights of pigs in the same age class was used [29]. This $\text{Weight}_{\text{brain}}$ was taken to convert the Mean Weight% of cuts to $\text{Weight}_{\text{cut}}$. This is shown in formula 7.

$$\text{Weight}_{\text{cut}} = \frac{\text{Weight}_{\text{brain}}}{\text{Mean Weight \%}_{\text{brain}}} * \text{Mean Weight \%}_{\text{cut}} \quad (7)$$

The trunk muscles, *musculus psoas*, *musculus triceps brachii*, forelimb, abdominal muscles and hindlimb were not conducted from the brain weight, because those are only parts of the pork cuts loin, tenderloin, shoulder, foreleg, belly and ham respectively. For these cuts we assumed a homogeneous distribution within the complete cut, so that the relative cyst distribution of the muscles described in Boa et al. 2002 could be used for the entire pork cuts that we assessed. The weight of these cuts was collected from literature [30-33].

CYSTS PER CONSUMED PORTION

A couple of steps were followed to determine how many cysts end up in the consumed portions of all pork cuts. First of all, the number of portions per cut was calculated. Therefore, the cut fraction and the total number of portions consumed in a country were determined with the following formulas:

$$\text{Cut fraction} = \frac{\text{Weight}_{\text{cut}}}{\text{Weight}_{\text{carcass}}} \quad (8)$$

$$\text{Total portions} = \text{Populationsize} * \frac{\text{Pork consumption } (\frac{kg}{inhab}/yr)}{\text{Portion size } (g)} * 1000\ (g) \quad (9)$$

$$\text{Total portions home slaughtered pigs} = \text{Total portions} * \text{Fraction home slaughter} \quad (10)$$

Using samples from a multinomial distribution with probabilities given by formula 8, and the number of trials by formula 9 ('controlled') or 10 ('home'), a distribution of cuts compliant to formula 8 was generated.
Second, a binomial function was used to calculate the probability that a cyst is in a cut. The number of trials of the binomial function is the number of cysts in the pigs, calculated in step 2 of the risk chain model. The probability of a cyst entering a cut is equal to the relative cyst density that was described before.
Third, the probability of a cyst in a cut being present in a portion from this cut is equal to the fraction portion (formula 11).

$$\text{Fraction portion} = \frac{\text{Weight}_{\text{portion}}}{\text{Weight}_{\text{cut}}} \quad (11)$$

With this proportion as probability, and the cysts per cut as number of trials, a second binomial function provided the number of cysts in a portion. The abovementioned binomial distributions were applied to every portion that was annually eaten in a country thus giving the total of infected portions. The total number of portions eaten from controlled pigs was derived from formula 9. This number was multiplied by the fraction home slaughtered pigs to obtain the total number of home slaughtered portions in a country (formula 10). The final outcome is the portion prevalence (formula 12).

$$\text{Portion prevalence} = \frac{\text{Infected portions}}{\text{Total portions}} \quad (12)$$

COOKING

As only raw or undercooked meat confers an actual risk to public health, cooking practices were appraised in the model, as also shown in Figure 1. From literature it did not become clear what cuts of pork are eaten raw, so two different approaches were taken to differentiate between raw and cooked consumed portions. The first approach (cooking scenario 1) was an indicative estimation of raw consumption, with the aid of personal communication with elderly family members and a couple of websites addressing pork cuts and cooking methods [34-38]. In this approach, a specific estimation is given of what fraction of a cut is eaten raw. The second approach (cooking scenario 2) was based on three scenarios (2A, 2B and 2C): cooking 10, 50 and 90% of the cuts. In this approach a standard fraction of every cut is assumed eaten raw. We assumed perfect inactivation of cysts during cooking. So, only the fractions of the cuts estimated eaten raw have viable cysts according to the model.

After the step of cooking the final portion prevalence and total number of infected portions, for controlled and home slaughtered pigs, could be determined for every country included in the model. Also the separate attribution of the cuts to the total portion prevalence was assessed.

SOFTWARE

The quantitative risk assessment model was run in R 3.4.3 [39], with data that was stored in Microsoft Excel 2010 spreadsheets.

RESULTS

For five countries data was available on the prevalence of porcine cysticercosis and the number of home slaughtered pigs. These are Bulgaria, Germany, Poland, Romania and Spain. The results of these countries are presented henceforth.

TEST SENSITIVITY MEAT INSPECTION

As mentioned in the methods section, European meat inspection does not reveal all present cysticerci in pig carcasses. According to Boa et al. 2002, the lengthwise incision of the heart that is performed during meat inspection, gives access to 136 cm^2 of the heart. Total slicing reveals 425 cm^2. The inspection proportion of the area is 32%. In other words, when cutting the heart, each heart cyst has a probability of $f = 0.32$ to be exposed [22]. By filling in formula 1, with $f = 0.32$, the relation between the burden of infection of the heart and the sensitivity of the current method of European examination of swine carcasses was obtained. Figure 3 demonstrates this relationship.

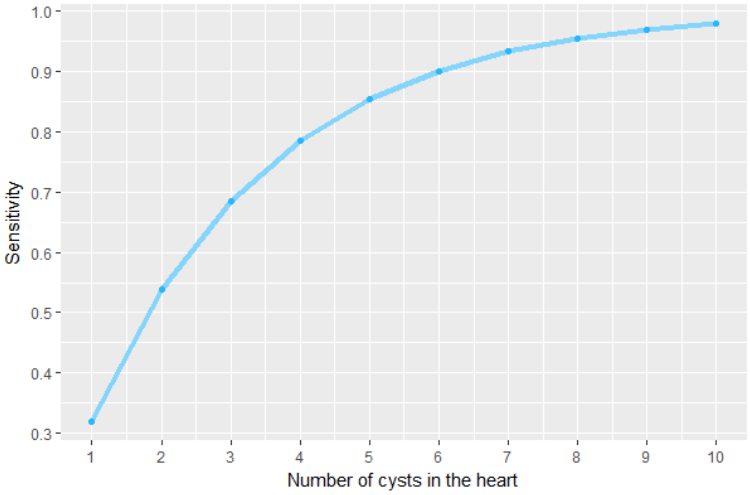

FIGURE 3 SENSITIVITY OF MEAT INSPECTION

EXPOSURE RATE AND INFECTION BURDEN

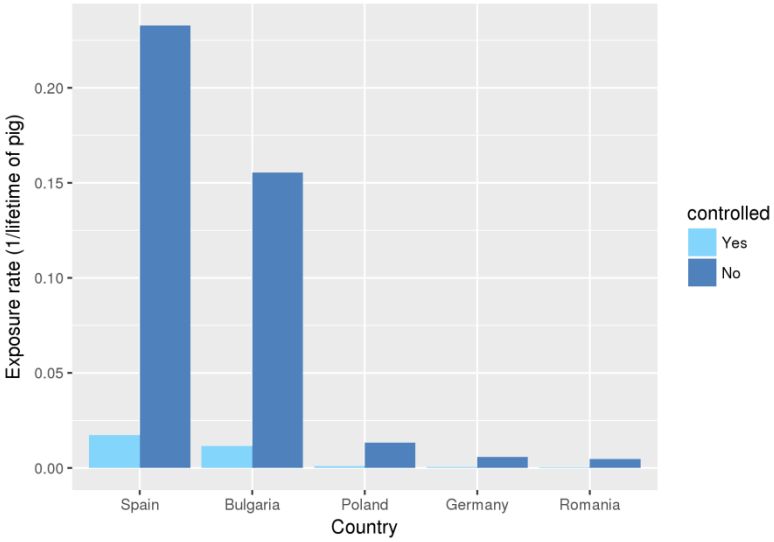

FIGURE 4 EXPOSURE RATE OF PIGS IN A LIFETIME, BY COUNTRY AND HOUSING

With the aid of reported prevalences and the probability to find a cyst in the heart, formula 3 led to the exposure rate of pig hearts to *T. solium* eggs (Figure 4). The reported prevalences are given in Table 1. The calculated heart exposure rates for every country were corrected for the probability of any cyst to be located in the heart, $p_{heart} = 3{,}6\times10^{-2}$ [22] to obtain the λ^c_{pig}. The calculated values of λ^c_{pig} are given in Table 1.

TABLE 1 DATA INPUTS AND PARAMETERS

Model part	Symbol	Unit	Bulgaria	Germany	Poland	Romania	Spain	Reference
Test sensitivity meat inspection	*f*: probability of revealing a heart cyst	Proportion	$3{,}2 \times 10^{-1}$	$3{,}2 \times 10^{-1}$	$3{,}2 \times 10^{-1}$	$3{,}2 \times 10^{-1}$	$3{,}2 \times 10^{-1}$	Boa et al. 2002
Exposure rate	*Pheart*: probability of a cyst to enter the heart	Proportion	$3{,}6 \times 10^{-2}$	$3{,}6 \times 10^{-2}$	$3{,}6 \times 10^{-2}$	$3{,}6 \times 10^{-2}$	$3{,}6 \times 10^{-2}$	Boa et al. 2002
	$\lambda^{c}pig$: exposure rate pigs in controlled housing	Rate	$2{,}7 \times 10^{-2}$	$9{,}8 \times 10^{-4}$	$2{,}3 \times 10^{-3}$	$8{,}2 \times 10^{-4}$	$4{,}0 \times 10^{-2}$	This study: Table 2
	$\lambda^{h}pig$: exposure rate pigs in uncontrolled housing	Rate					$5{,}4 \times 10^{-1}$	This study: Table 2
Prevalence	*P*(detect): reported prevalence	Proportion	$1{,}3 \times 10^{-4}$	$4{,}8 \times 10^{-6}$	$1{,}1 \times 10^{-5}$	$4{,}0 \times 10^{-6}$	$2{,}0 \times 10^{-4}$	Laranjo-Gonzales et al. 2017; Trevisan et al. 2017; Oleleu et al. 2016; WAHIS interface OIE; Table 3
Slaughter data	Proportion home slaughter of pigs	Proportion	$2{,}3 \times 10^{-1}$	$4{,}0 \times 10^{-3}$	$6{,}4 \times 10^{-2}$	$5{,}5 \times 10^{-1}$	$6{,}0 \times 10^{-4}$	Eurostat; This study: Table 4
Cyst distribution and weight pork cuts	Relative cyst density	Proportion	0,006-0,02	0,006-0,02	0,006-0,02	0,006-0,02	0,006-0,02	Boa et al. 2002
	Weight of cuts	Kilograms	0,09-14,0	0,09-14,0	0,09-14,0	0,09-14,0	0,09-14,0	This study: Table 5
	Cut fraction	Fraction	0,002-0,34	0,002-0,34	0,002-0,34	0,002-0,34	0,002-0,34	This study: Table 5
	Fraction portion	Fraction	0,007-1	0,007-1	0,007-1	0,007-1	0,007-1	This study: Table 5
Cysts per portion	Average population	Inhabitants	$7{,}5 \times 10^{6}$	$8{,}1 \times 10^{7}$	$3{,}8 \times 10^{7}$	$2{,}0 \times 10^{7}$	$4{,}7 \times 10^{7}$	Eurostat
	Pork consumption	Kg/inhabitant/yr	20,1	53,6	48,7	28,8	48,7	Faostat
	Portionweight	Grams	100	100	100	100	100	This study
	Total portions	Portions/yr	$1{,}5 \times 10^{9}$	$4{,}4 \times 10^{10}$	$1{,}9 \times 10^{10}$	$5{,}8 \times 10^{9}$	$2{,}3 \times 10^{10}$	This study
Cooking	Scenario 1	Raw fraction	0-0,47	0-0,47	0-0,47	0-0,47	0-0,47	This study: Table 6
	Scenario 2a	Fraction	0,1	0,1	0,1	0,1	0,1	This study
	Scenario 2b	Fraction	0,5	0,5	0,5	0,5	0,5	This study
	Scenario 2c	Fraction	0,9	0,9	0,9	0,9	0,9	This study

PREVALENCE

The reported prevalence of the countries included in the model is shown in Table 1. The adjusted prevalence of pigs that were raised uncontrolled and slaughtered at home was calculated via the exposure conversion (Figure 1: Step 2A to 2B). The steps to obtain the exposure conversion are shown in Table 2.

TABLE 2 STEPS TO EXPOSURE CONVERSION

	Input data		Calculated data		
Year	**Prevalence control Spain (%)**	**Prevalence home Spain (%)**	λ_c	λ_h	λ_c / λ_h
2011	$1{,}6 \times 10^{-2}$	$2{,}0 \times 10^{-1}$	$4{,}9 \times 10^{-4}$	$6{,}4 \times 10^{-3}$	$7{,}6 \times 10^{-2}$
2012	$1{,}2 \times 10^{-2}$	$1{,}6 \times 10^{-1}$	$3{,}8 \times 10^{-4}$	$5{,}1 \times 10^{-3}$	$7{,}4 \times 10^{-2}$
2013	$3{,}1 \times 10^{-2}$	$4{,}3 \times 10^{-1}$	$9{,}8 \times 10^{-4}$	$1{,}3 \times 10^{-2}$	$7{,}3 \times 10^{-2}$
Average	$2{,}0 \times 10^{-2}$	$2{,}7 \times 10^{-1}$	$6{,}1 \times 10^{-4}$	$8{,}3 \times 10^{-3}$	$7{,}4 \times 10^{-2}$

In Table 3, column 4 is shown that the calculated adjusted prevalence of pigs in controlled housing is approximately 86 times higher than the reported prevalence due to the low sensitivity of meat inspection, especially with a low burden of infection. The calculated adjusted prevalence of home slaughtered animals is another 11 to 14 times higher (Table 3, column 6). The highest prevalences are found in Spain and Bulgaria (Figure 5).

TABLE 3 PREVALENCE OF CONTROLLED AND HOME SLAUGHTER

	Input data	Calculated data			
Country	**Reported prevalence controlled (%)**	**Adjusted prevalence controlled (%)**	**Adjusted prevalence controlled / reported prevalence controlled**	**Adjusted prevalence home (%)**	**Prevalence home / prevalence controlled**
Bulgaria	$1{,}3 \times 10^{-2}$	1,1	86	13	12
Germany	$4{,}8 \times 10^{-4}$	$4{,}2 \times 10^{-2}$	87	$5{,}7 \times 10^{-1}$	13
Poland	$1{,}1 \times 10^{-3}$	$9{,}8 \times 10^{-2}$	87	1,3	13
Romania	$4{,}0 \times 10^{-4}$	$3{,}4 \times 10^{-2}$	85	$4{,}7 \times 10^{-1}$	14
Spain	$2{,}0 \times 10^{-2}$	1,7	86	18	11

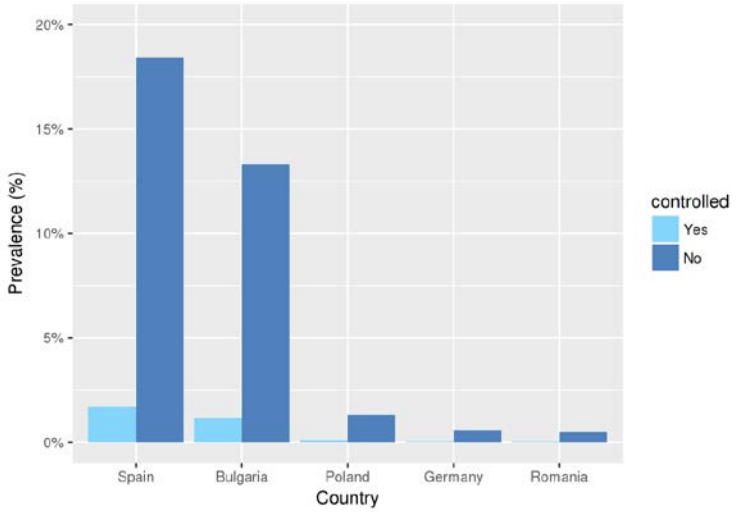

FIGURE 5 ADJUSTED PREVALENCE OF CONTROLLED AND HOME SLAUGHTERED PIGS BY COUNTRY

SLAUGHTER DATA

The number of slaughtered pigs in- and outside slaughterhouses is presented in Table 4, per country and year [26, 27]. The last column shows the average home slaughter fraction in the different countries.

TABLE 4 SLAUGHTER DATA

		Input data		Calculated data	
Country	**Year**	**Home slaughtered pigs (*10³)**	**Commercially slaughtered pigs (*10³)**	**Fraction home slaughter**	**Average fraction home slaughter**
Bulgaria	2006	40	1010	$3,9 \times 10^{-2}$	$2,3 \times 10^{-1}$
	2007	35	599	$5,8 \times 10^{-2}$	
	2008	33	993	$3,3 \times 10^{-2}$	
	2009	432	549	$7,9 \times 10^{-1}$	
Germany	2007	317	53311	$6,0 \times 10^{-3}$	$4,0 \times 10^{-3}$
	2009	247	56068	$4,4 \times 10^{-3}$	
	2010	211	58154	$3,6 \times 10^{-3}$	
	2011	184	59590	$3,1 \times 10^{-3}$	
	2012	153	58198	$2,6 \times 10^{-3}$	
Poland	2009	1365	18678	$7,3 \times 10^{-2}$	$6,4 \times 10^{-2}$
	2010	1275	19966	$6,4 \times 10^{-2}$	
	2011	1062	20979	$5,1 \times 10^{-2}$	
	2012	1377	19216	$7,2 \times 10^{-2}$	
	2013	1143	19120	$6,0 \times 10^{-2}$	
Romania	2009	2809	2888	$9,7 \times 10^{-1}$	$5,5 \times 10^{-1}$
	2010	1887	2901	$6,5 \times 10^{-1}$	
	2011	1620	3257	$5,0 \times 10^{-1}$	
	2012	1298	3474	$3,7 \times 10^{-1}$	
	2013	940	3753	$2,5 \times 10^{-1}$	
Spain	2011	27	41436	$6,6 \times 10^{-4}$	$6,0 \times 10^{-4}$
	2012	26	40609	$6,5 \times 10^{-4}$	
	2013	14	39323	$3,4 \times 10^{-4}$	

CYST DISTRIBUTION AND WEIGHT OF PORK CUTS

The relative cyst density and weight of cuts can be reviewed in Table 5. Pork organs or cuts that did not contain any cysts, are not named as they are not relevant for the model. These are for instance liver and kidneys [22]. The output of formula 6 is shown in column 4 of Table 5. The $Weight_{brain}$ was set at 0,135 kg [29].

CYSTS PER CONSUMED PORTION

The cut fraction determined with formula 8 and the fraction portion with formula 11 are shown in the last two columns of Table 5. The $Weight_{portion}$ is 100 grams. The number of portions that is annually eaten in the five included countries is demonstrated in Table 1. The results from the binomial distributions used in this step, present the number of infected portions that consumers are actually exposed to, if all portions would be eaten raw.

TABLE 5 CYST DISTRIBUTION AND WEIGHT OF PORK CUTS

	Input data		Calculated data		
Organ / cut	**Relative cyst density**	**Mean Weight (%)**	**Weight (kg)**	**Cut fraction**	**Fraction portion**
Brain	$1{,}7 \times 10^{-2}$	$3{,}4 \times 10^{-1}$	$1{,}3 \times 10^{-1}$	$3{,}2 \times 10^{-3}$	$7{,}4 \times 10^{-1}$
Head muscles	$6{,}8 \times 10^{-2}$	3,0	1,2	$2{,}8 \times 10^{-2}$	$8{,}4 \times 10^{-2}$
Internal masseter	$1{,}6 \times 10^{-1}$	$2{,}3 \times 10^{-1}$	$9{,}1 \times 10^{-2}$	$2{,}2 \times 10^{-3}$	1,0
External masseter	$1{,}4 \times 10^{-1}$	$4{,}2 \times 10^{-1}$	$1{,}7 \times 10^{-1}$	$3{,}9 \times 10^{-3}$	$6{,}0 \times 10^{-1}$
Tongue	$5{,}9 \times 10^{-2}$	1,1	$4{,}4 \times 10^{-1}$	$1{,}0 \times 10^{-2}$	$2{,}3 \times 10^{-1}$
Esophagus	$5{,}5 \times 10^{-3}$	$2{,}4 \times 10^{-1}$	$9{,}5 \times 10^{-2}$	$2{,}2 \times 10^{-3}$	1,0
Heart	$3{,}6 \times 10^{-2}$	$8{,}1 \times 10^{-1}$	$3{,}2 \times 10^{-1}$	$7{,}6 \times 10^{-3}$	$3{,}1 \times 10^{-1}$
Diaphragm	$4{,}5 \times 10^{-2}$	$7{,}2 \times 10^{-1}$	$2{,}8 \times 10^{-1}$	$6{,}7 \times 10^{-3}$	$3{,}5 \times 10^{-1}$
Tenderloin	$2{,}0 \times 10^{-1}$		$5{,}0 \times 10^{-1}$	$1{,}2 \times 10^{-2}$	$2{,}0 \times 10^{-1}$
Loin	$2{,}0 \times 10^{-2}$		$1{,}4 \times 10^{1}$	$3{,}3 \times 10^{-1}$	$7{,}2 \times 10^{-3}$
Shoulder	$9{,}3 \times 10^{-2}$		3,5	$8{,}4 \times 10^{-2}$	$2{,}8 \times 10^{-2}$
Foreleg	$7{,}5 \times 10^{-2}$		4,0	$9{,}4 \times 10^{-2}$	$2{,}5 \times 10^{-2}$
Belly	$2{,}4 \times 10^{-2}$		5,4	$1{,}3 \times 10^{-1}$	$1{,}8 \times 10^{-2}$
Ham	$6{,}0 \times 10^{-2}$		$1{,}2 \times 10^{1}$	$2{,}9 \times 10^{-1}$	$8{,}2 \times 10^{-3}$
Total	**1**		**$4{,}2 \times 10^{1}$**	**1**	

The portion prevalence is highest in Spain and Bulgaria, where respectively 0.03% and 0,02% of the 100 gram portions are infected, when pigs are slaughtered at home (Figure 6). In Spain and Germany, the total of infected portions is higher under controlled conditions than when home slaughtered, while in Poland it is almost equal and in the other countries this is the other way around (Figure 7).

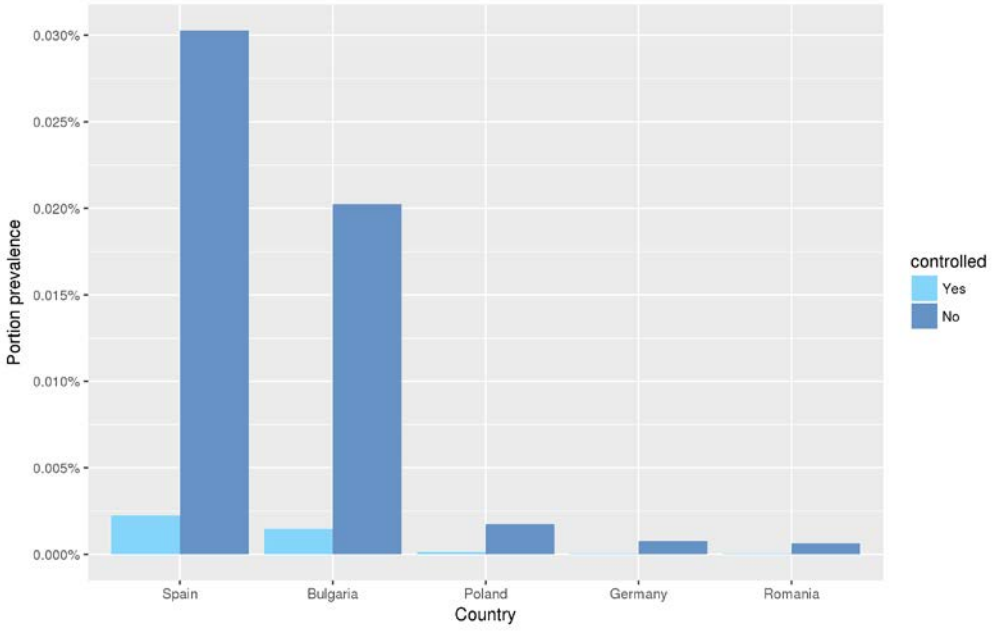

FIGURE 6 PREVALENCE OF INFECTED PORTIONS PER COUNTRY BEFORE COOKING

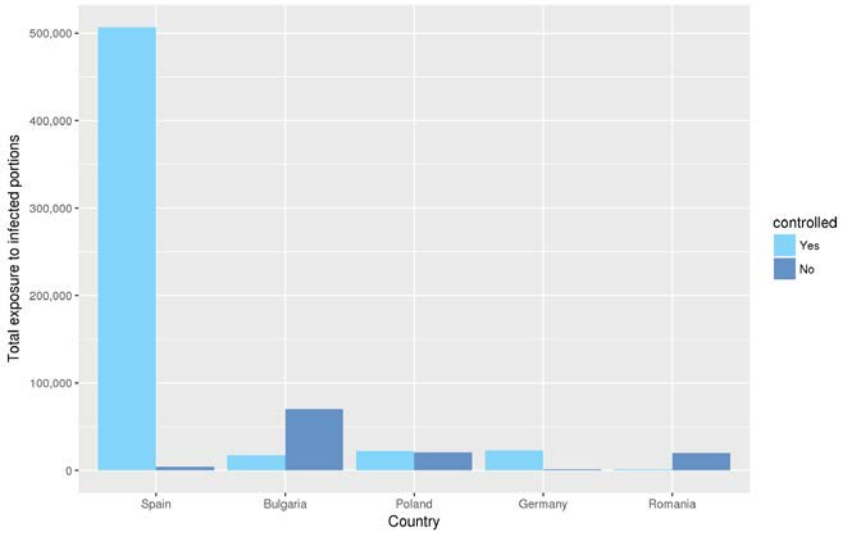

FIGURE 7 NUMBER OF INFECTED PORTIONS PER COUNTRY BEFORE COOKING

COOKING

In Table 6 cooking scenario 1 is described based on the first approach. The fraction of the cut prepared raw, is the fraction that people are expected to eat raw. For example, the esophagus that is made to ground pork. The fraction of prepared raw that is expected to be eaten raw, is what fraction of this ground pork will be eaten raw instead of cooked. For the tenderloin, the whole cut is eaten undercooked, so the $F_{raw.prep}$ is 1. Yet, the tenderloin is eaten medium/rare, so the whole cut has an $F_{prep.eaten.raw}$ of 0.4. This gives a total raw fraction of the tenderloin of 0.4. Cooking scenario 2 is based on the second approach. As was described in the methods, a fixed fraction of 0.1, 0.5 and 0.9 is considered to be eaten raw.

TABLE 6 COOKING SCENARIO 1: FRACTION OF PORK CUTS EATEN RAW

Organ / cut	Fraction of the cut prepared raw ($F_{raw.prep}$)	Fraction of prepared raw, that is eaten raw ($F_{prep.eaten.raw}$)	Total raw fraction ($F_{raw.prep}$ * $F_{prep.eaten.raw}$)	What raw products?
Brain	0	0	0	
Head muscles	0	0	0	
Internal masseter	0	0	0	
External masseter	0	0	0	
Tongue	0	0	0	
Esophagus	1	$3{,}3 \times 10^{-1}$	$3{,}3 \times 10^{-1}$	Ground pork in sausage
Heart	1	$3{,}3 \times 10^{-1}$	$3{,}3 \times 10^{-1}$	Ground pork in sausage
Diaphragm	1	$3{,}3 \times 10^{-1}$	$3{,}3 \times 10^{-1}$	Ground pork in sausage
Loin	$1{,}7 \times 10^{-1}$	$3{,}6 \times 10^{-1}$	$5{,}9 \times 10^{-2}$	Boneless top loin roast; sausage; bacon
Tenderloin	1	$4{,}0 \times 10^{-1}$	$4{,}0 \times 10^{-1}$	Baked medium/rare
Shoulder	$2{,}5 \times 10^{-1}$	$3{,}3 \times 10^{-1}$	$8{,}3 \times 10^{-2}$	Ground pork in sausage
Foreleg	0	0	0	
Belly	$5{,}0 \times 10^{-2}$	1	$5{,}0 \times 10^{-2}$	Bacon
Ham	$5{,}0 \times 10^{-1}$	$9{,}4 \times 10^{-1}$	$4{,}7 \times 10^{-1}$	Raw and cured ham; fricandeau: medium/rare

The results of cooking pork are shown in Figures 8 to 11. In Figure 8 the total exposure in a country in a year is given, when the population of that country cooks the portions as is estimated with scenario 1. These results, even as the total exposure before cooking, can also be seen in Table 7. Cooking according to scenario 1 gives a 4 times lower total exposure of infected portions. Figure 9 visualizes that decrease.

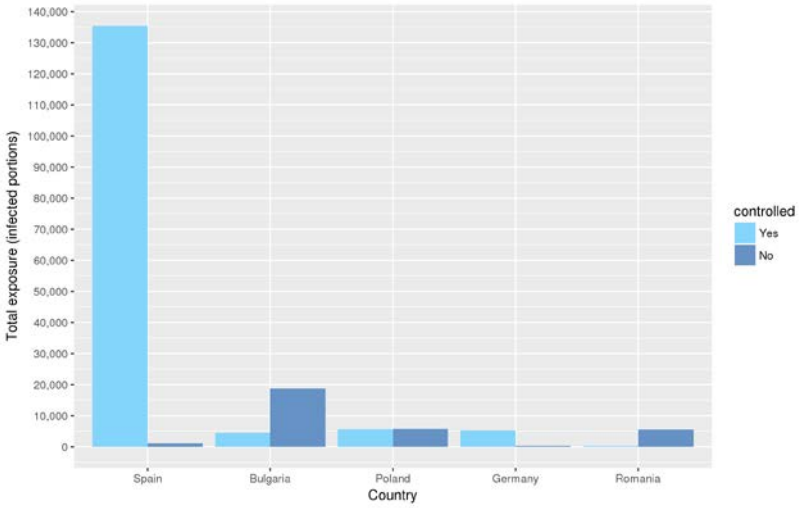

FIGURE 8 TOTAL EXPOSURE OF INFECTED PORK PORTIONS PER COUNTRY AFTER COOKING

The portion prevalence also falls after cooking scenario 1 is applied. In figure 10 the portion prevalence before and after cooking is showed. A fifty times smaller portion prevalence is left after cooking. This is only shown for controlled pork, but for home slaughtered pigs the difference between before and after cooking is the same.

Scenario 2 is compared with scenario 1 in Figure 11. This figure demonstrates that the larger the raw fraction, the more portions that are eaten contain viable cysticerci cellulosae, according to the model. Cooking according to scenario 1 leaves a higher portion prevalence than the scenario with a raw fraction of 0.1. The figure only takes into account controlled slaughter pigs in Spain, because the same scenarios were used for the other countries.

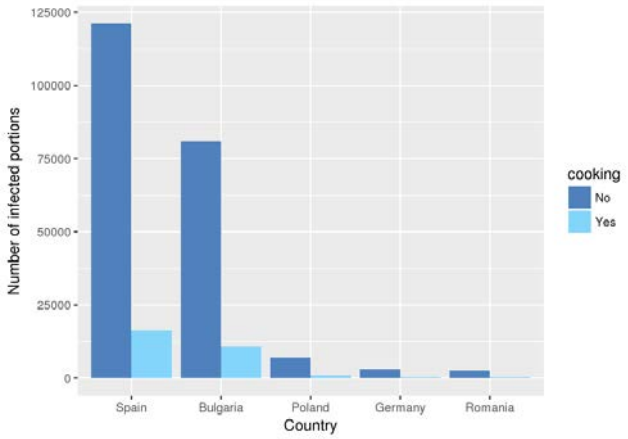

FIGURE 9 NUMBER OF INFECTED PORTIONS OF CONTROLLED PIGS, BEFORE AND AFTER COOKING COMPARED

TABLE 7 TOTAL EXPOSURE TO INFECTED PORTIONS PER COUNTRY

Country	Before cooking		After cooking	
	Controlled	Home	Controlled	Home
Bulgaria	17336	70118	4474	18668
Germany	22796	1329	5210	344
Poland	21945	20699	5638	5741
Romania	1143	20007	342	5552
Spain	506893	4133	135520	1105

The attributions of the different cuts to the total exposure of consumers are displayed in Figure 12. The muscles are responsible for 80% of the infected portions, and the organs for 20%. The organs that belong to this 20% are the esophagus, heart and diaphragm. The other organs are not eaten raw according to our sources (Table 6).

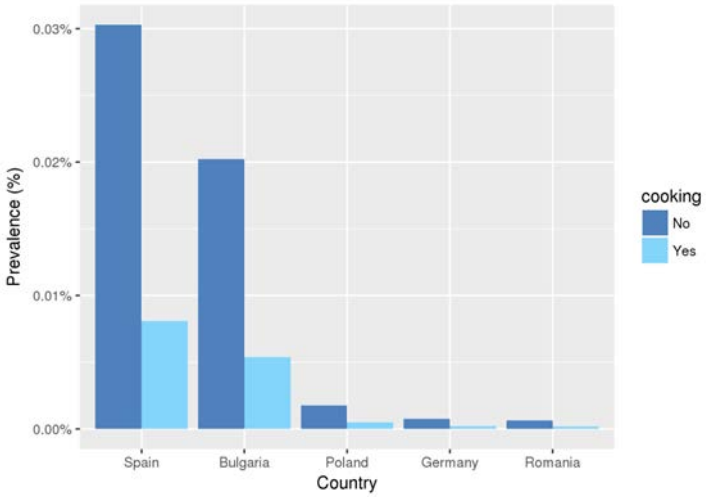

FIGURE 10 PORTION PREVALENCE OF CONTROLLED PIGS, BEFORE AND AFTER COOKING COMPARED

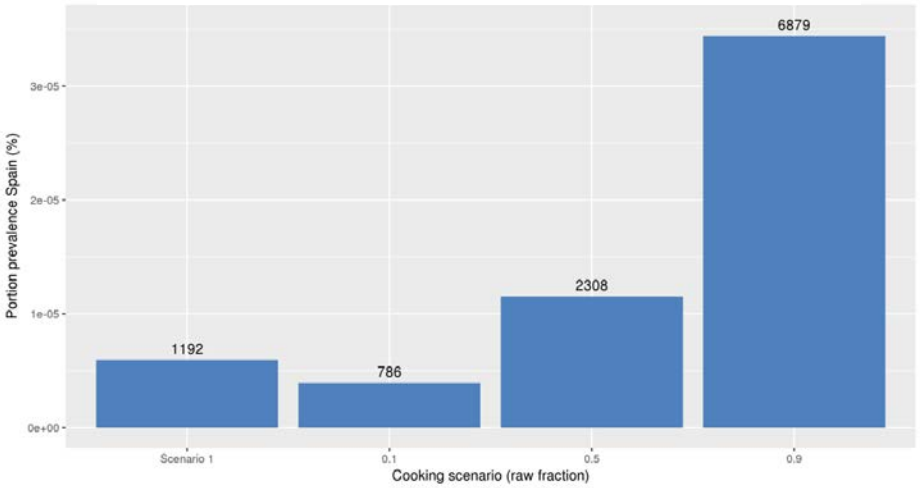

FIGURE 11 NUMBER AND PREVALENCE OF INFECTED PORTIONS AFTER DIFFERENT COOKING SCENARIOS

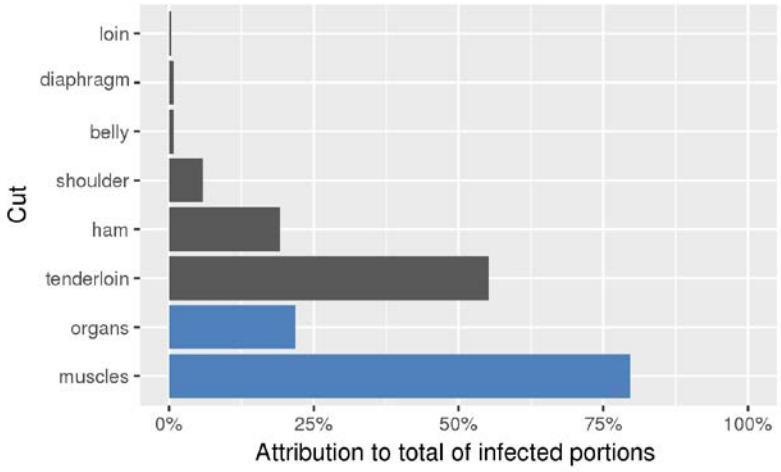

FIGURE 12 ATTRIBUTION OF CUTS TO THE TOTAL OF INFECTED PORTIONS

DISCUSSION

In this paper we have built a quantitative microbiological risk assessment (QMRA) model from pork production to consumption and implemented literature data about the porcine cysticercosis prevalence, home slaughter numbers, the distribution of cysts in pork cuts and consumption quantities of pork. We present the results of the QMRA model for the risk of human exposure to *Taenia solium* due to consumption of pork in five European countries.

Aim of the study was to estimate the risk of human exposure to *T. solium* from home slaughtered portions of pork infected with *T. solium* cysticerci in Europe and compare the portion prevalence of home and controlled pig slaughter. Although a numerical uncertainty analysis has not been performed, critical steps will be given attention.

We demonstrated that the detection of cysticerci cellulosae during meat inspection is dependent on the area of the body that is inspected and the burden of infection of the carcasses. The probability to find an infected pig during meat inspection is low, because the reported *T. solium* prevalences in European countries are very low [15, 18], and the sensitivity of meat inspection as well. This finding is in line with a paper that evaluates meat inspection and other tests for the detection of cysticercosis [21]. We obtained the original data of that study to test our model (Personal communication). The data consists of 65 pigs that were slaughtered, then inspected for *T. solium* according to the routine meat inspection protocol in that country and at last sliced to find all cysticerci cellulosae [21]. Thirty-two pigs were infected with *T. solium*. We used our p_{heart} and formula 2 to determine which infected pigs would be found with meat inspection according to our model and compared it to the pigs that were actually found with meat inspection. With an arbitrary cutoff of 0,5 in our model, distinguishing between 'detects' and ' non-detects', our model had an error rate of 4/32. The sensitivity of the model on meat inspection is 75% and the specificity is 100%. The positive predictive value is 100% too. The 4 pigs that were found infected during meat inspection, but not according to our model, could be predicted by our model per chance and due to the sharp cut off of 0,5. Besides, those four misdetects had fairly high numbers of cysts, casting doubt on the experimental outcome for those pigs. Furthermore, the meat inspection that was done in the study of Dorny et al. (2004) included the heart and other organs like the masseter muscles while we only included the heart. Altogether, the predictive value of our model, regarding meat inspection, is very high.

Nevertheless, the meat inspection sensitivity we determined constitutes an underestimation because we only included the surface of the heart incision as site of inspection, whilst according to European regulation the exterior of the heart and other predilection sites are also being checked [12, 40]. Still, we do not expect a large discrepancy, because we assume that the probability that a cyst is on the surface of an organ is small when there is a low infection burden, and with a high infection burden a heart incision alone will find the infection anyway. All articles about European meat inspection evaluation regarding *Taenia* spp. use an inspection procedure similar to that described in Regulation (EC) 854/2004 chapter IV [21, 40-42]. The regulation was amended in 2013, when only visual inspection was introduced in many European countries. As the prevalence data of the five incorporated countries do not surpass 2013, this did not affect our model. Also, European experts on slaughter practices recently answered a questionnaire where questions about official meat inspection were asked. All countries included in the model answered that their meat inspection protocol still strictly follows the procedure defined in Regulation (EC) 854/2004 [20]. So, our method to estimate meat inspection sensitivity seems relevant for the countries that were assessed.

The model is based on the assumption of a constant, but low rate of infection of pigs. An individual pig is thus exposed to a low number of cysts, at a low frequency. This is a Poisson

process, which also yields low numbers of infecting cysts. This model assumption would be violated when infection had the character of infrequent high doses of cysts. We have no data to decide between these scenarios. The second scenario would require distributions describing both abundance and prevalence (e.g. a negative binomial distribution). Such a distribution would require more parameters and is hence less parsimonious than our default choice of a Poisson process.

The exposure rates are highest in Spain and Bulgaria. This is a consequence of the reported prevalences, of which the exposure rates are derived. The exposure rates of home slaughter pigs are a factor 13,5 higher than those of controlled slaughter pigs as calculated from the exposure conversion that is derived from data of only one country, Spain. A home slaughter exposure rate based on prevalence data specific for the different countries would improve the outcomes of the model. Now the assumption was made that the home slaughtered pigs were reared the same in every country so that the exposure conversion calculated for Spain could be applied to other countries. In reality uncontrolled housing of pigs or backyard pig keeping can exist over a wide range of practices. The housing depends on cultural background, socio-economic status, climate and the housing legislation in the country. These affect the exposure of the pigs in that country. Although the exposure conversion is a substantial uncertainty in the model, the Spanish prevalence data did indicate that it is very pertinent to take into account that home slaughtered pigs can be subjected to a higher number of eggs in their lifetime than controlled slaughtered pigs. Furthermore, the Spanish data showed similar exposure conversions over the years, strengthening the idea that the estimate is robust.

By combining the sensitivity of meat inspection and the exposure rate we predicted the prevalence. That the calculated adjusted prevalences are about 86 times higher than the reported prevalences is not surprising, when we bear in mind the low meat inspection sensitivity. Nonetheless, the prediction might be an overestimation because misclassification might occur during inspection and the reported *Taenia* spp. cases that we adopted have never been confirmed by a diagnostic tool like polymerase chain reaction (PCR) [15, 18]. Other *Taenia* spp. for which pigs can serve as intermediate hosts are *T. hydatigena* and *T. asiatica*. The predilection sites of these species are different.
T. hydatigena cysticercosis establishes mainly in the omentum and the liver [43]. The cysts of *T. asiatica* prefer the liver as well [44], although the first naturally infected pig with a cyst of *T. asiatica* in muscle has recently been reported [45]. Apart from the anatomical distribution of the cysticerci throughout the body, the geographical distribution of these species does not correspond with *T. solium*. A systematic review on the global occurrence of *T. hydatigena* in pigs and cattle found only one study regarding Europe, namely Scotland. The species was detected in one of 3800 pigs in a study from 1964 to 1967 [46]. Nevertheless, *T. hydatigena* does occur in sheep and in wildlife in Europe. *T. asiatica* is not limited to South-East Asia anymore as was thought before, but still has never been detected near the European continent [47]. Therefore, we assume the contribution of *T. asiatica* to the reported European cases to be negligible and that of *T. hydatigena* to be possible although the tissue distribution differs from *T. solium*.
We do realize that a bias is possible in the prevalence differences between countries. As the prevalence data depends on what is reported in the slaughterhouses, it is reasonable that the countries with the best meat inspection and reporting system end up with the highest prevalence. The prevalence data must consequently be interpreted carefully.

The same holds for the portion prevalence. The portion prevalence is calculated highest for home slaughtered pigs in Spain and Bulgaria. In every country the home slaughter portion prevalence is a factor 10 higher than the controlled slaughter portion prevalence. Regardless, the total number of infected portions was higher under controlled than home reared conditions in Germany and Spain. The explanation is the small share of home slaughter in those countries,

giving a very small number of total portions.
The portion size of 100 grams was chosen because of the estimated consumption of pork meat in grams per capita per day that is given in the database of the FAO [48]. Here, the consumption amount reported is between 106 and 111 grams per day in the European Union (2009 to 2013). For the five countries in the model a consumption between 69 (Bulgaria) and 149 (Germany) grams per day has been reported. For the sake of clarity the variety of portion sizes over the years and countries was not included in this risk assessment.
Since the portion prevalence is very low (figure 6: the highest is 0,03%) the chance that someone gets exposed to more than one infected portion is very low. So we presume that every infected portion is eaten by someone else. For home slaughter though, this might be a false assumption. If a family keeps some pigs for their own consumption and those pigs are all reared on the same ground at the same time, all pigs might be infected and so the family has a much higher risk of exposure to *T. solium* cysticerci than the average. This illustrates that the portion prevalence for home slaughter is more complicated to translate to a quantitative risk on population level. The portion prevalence of home slaughtered pork was also assessed when meat inspection is done on home slaughtered carcasses. This is not shown in the results, because the difference between the portion prevalences was negligible and remained as high as without meat inspection. We connect this to the low sensitivity of meat inspection. In a country with a higher exposure of the pigs, more pigs would be found infected in slaughterhouses, so meat inspection then would make a difference.

Fortunately, cooking of the pork portions goes along with a conversion in the number of infective portions. If the scenario that was presented in Table 6 is a good estimation of cooking practices, the risk is decreased with a factor 3 due to cooking. We chose for scenarios where meat is either raw or perfectly cooked before being eaten, instead of a model where inactivation is a function of cooking time and temperature, as was done in a QMRA for another meat borne parasite *Trichinella* spp. [30]. Despite the fact that a publication about heat inactivation of *Taenia* cysts was available, the time to inactivation was not contemplated so we did not adopt these results for our model [49].
Estimating the fraction of a cut that is eaten raw is complex. For instance, it is known that some parts of the ham are always eaten raw, but the shoulder can be cut and prepared in multiple ways. An unknown fraction of these cuts is ground pork. Of the ground pork, again only a fraction is used in raw meat products like sausages and the rest is roasted. Also, the raw cuts are often dried, smoked or pickled with salt, and when a whole pig is slaughtered for one family a large quantity will be frozen. Freezing four days at -5 °C; three days at -15 °C or one day at -24 °C effectively kills cysticerci [50]. Salt pickling lowers the viability of *Taenia* metacestodes due to changes in the osmotic potential, causing a membrane rupture [51]. The other preparation methods have not been evaluated as far as we know. Thus, the raw fraction of pork cuts eaten is a limitation of this study.
Even so, raw meat products are eaten. The consumption preferences depend on the cultural background and personal customs. In Germany a cross-sectional survey about raw meat consumption pointed out that among 510 respondents 63,1% of the people from Eastern Germany (EG) and 34,4% of the people from Western Germany (WG) ate raw ground pork. In EG 17,8% and in WG 5,1% ate raw ground meat at least once a week [52]. In Romania raw pork consumption is very common, and although not quantitative, articles demonstrate that Romanian traditions of home slaughter causes meat borne infections like taeniosis [53, 54]. Bulgarian trichinellosis patients gave a history of eating undercooked meat products like sausages and meatballs, that were home-made [55]. So also in Bulgaria raw meat consumption is ordinary. More interestingly, a considerable number of home-made meat products come up in articles about Romanian meat borne infections [53, 54]. This could firstly mean that home slaughtered pig is risky not only regarding *T. solium* but also other pathogens, because they are

also exposed to other high prevalent parasites such as *Trichinella*. Secondly, it could mean that home slaughtered pigs are more often consumed raw. A combination of these reasons is probable and in both cases shows the relevance of our model.

We identified the heterogeneous distribution of cysticerci in the pig carcasses in our model. The cyst density was used to calculate the share of each cut in the total of infected portions. The largest attribution after cooking comes from the tenderloin and ham. This might change when cooking is performed differently. For example the masseter muscles do not add to the risk now because they are always eaten thoroughly cooked according to various sources [34-38]. But they do have a very high relative cyst density so if cooking habits are changing or someone prefers them raw the masseter would contribute to the risk. Another factor that could change the attributions of the cuts is the cyst viability in the meat. Viable cysts are distinguished from degenerated ones by the color, scolex movements and translucency [21, 56]. In this study 100% viability of cysts before cooking was assumed. However, of 24 carcasses that were naturally infected with *T. solium*, about 80% contained only viable cysticerci in the masseters and shoulder, but about 30% of the carcasses contained only viable cysticerci in the heart and hind limb and 50% in the belly [22]. Thus, the probability of viability of the cysts also seems to be dependent on the cut, which we neglected.

Aside from the viability fraction per cut we also left out viability in general. *Taenia* cysts can survive for three years after experimental infection [57]. Pigs that were slaughtered 26 weeks post infection had a mean total viability of 99% (SD ±1) [56]. Pigs are often slaughtered around twenty weeks of age, so degenerated cysts are not probable. Yet, experimentally infected pigs receive a single high dose of eggs while naturally infected pigs are likely exposed to eggs all their lives. Additional data from Dorny et al. 2004 (personal communication) demonstrates that pigs with an infection of less than 100 cysticerci often have a very low viable fraction while pigs with an infection between 100 to 24662 cysticerci have a viability fraction between 0,896 and 1 (data of 31 pigs) [21]. On the one hand, pigs with a light infection could be pigs that have just been exposed to cysts, so being young with a strong immune system and therefore with a low viable fraction of cysts. Yet, cysts need time to become mature. On the other hand, it could be pigs that were exposed to cysts a long time ago, so being old and already close to being recovered. Though both interesting options, neither can explain that the pigs with a heavy infection have such a high viable fraction. Besides the degeneration of cysticerci takes time, for resorption needs to take place. As we had no good clarification for the remarkable results, we assumed that all cysts were viable.

As discussed, many assumptions had to be made to calculate the risk of human exposure to *T. solium* in pork. To examine if the model is a good representation of the real situation human taeniosis prevalence is necessary. Three aspects make this difficult. First, some countries have reported the number of general taeniosis cases every year (Romania and Poland for example) [15, 18]. Taeniosis in humans can be caused by other *Taenia* spp. like *T. saginata*. Distinction between them (by counting the amount of primary uterine branches in gravid proglottids [58] or PCR confirmation) is often not made or recorded, for instance because treatment does not depend on the species. In that case the reported data is not useful to compare with the model. Second, the reported *T. solium* taeniosis is hard to use. A person diagnosed with taeniosis could have been infected years ago, for adult tapeworms can survive in excess of ten years [57]. Third, many people with a tapeworm are never being diagnosed, due to the mild and vague symptoms or sociological reasons (i.e. a taboo) [59]. Hence, human taeniosis prevalence can often only be estimated by means of the sales figures of specific anthelmintic drugs like niclosamide [59, 60].

Regardless of these circumstances if we look at the countries in our model for which there is human taeniosis data, we can say the following: In Poland from 2007 to 2009 a total of 278

human cases have been reported. 180 cases are due to *T. saginata* and the other 98 cases are 'other tapeworms' (i.e. 35% of the total cases) [18]. If the other tapeworms are almost all *T. solium* cases it means there are on average around 33 cases per year. This would mean that from the annual 42644 infected portions, 0,08% cause an infection. In Romania from 2007 to 2009, 1463 taeniosis cases have been reported. If also in Romania 35% is due to *T. solium*, around 170 cases per year are *T. solium* taeniosis cases [18]. This would imply that 0,8% of the total infected portions cause an infection. Although the difference between those countries is 10 times, it is not impossible when we take into consideration the earlier described food customs in Romania and the percentage home slaughter that is 9 times higher in Romania than in Poland. The number of people exposed as estimated by the QMRA model is not so odd taking into account that the number of reported cases is assumed an underestimation of the real cases.

In conclusion, we developed a model to assess the relative exposure to *T. solium* in Europe, comparing pork from home slaughter with pork from controlled slaughter. Our model takes into account different stages of the food chain, from the prevalence that starts at the farm to the portion prevalence that ends up on the consumers' plate. This makes it possible to look at the effect of every step in the chain on the final exposure. Despite the uncertainties, our model shows the importance of extensive meat inspection, good biosecurity and more importantly, proper cooking of meat, especially when pigs are slaughtered at home. The most important findings are firstly that meat inspection performed in Europe has a very low sensitivity, especially when pigs have a low infection burden. Therefore the adjusted prevalence of *T. solium* is much higher than reported, as we showed. Secondly, in home slaughtered pigs the prevalence is about 12 times higher than in controlled slaughtered pigs, because of the transmission route of *T. solium* in combination with the unhygienic living conditions of home slaughtered pigs. This means that people eating pigs that were housed in backyards or in another way uncontrolled, have a higher risk of exposure to *T. solium*. Finally, thorough cooking of pork kills cysticerci cellulosae, thereby greatly lowering the number of infected portions. So, the final exposure to *T. solium* depends on many factors and differs per country, way of meat inspection, way of housing pigs and cooking habits.

The model can be expanded if more information about the prevalence among pigs (controlled and home slaughtered) and consumer behavior regarding raw meat consumption is acquired. Therefore, it would be useful if European countries develop a better monitoring system for *T. solium*, preferably based on a more sensitive method instead of visual inspection [61] and confirmation of suspected findings. In addition, a comprehensive survey about raw meat consumption would reduce uncertainty in the estimates on the raw consumed portions and give a better perception of cultural differences (e.g.[62]). Moreover, investigating the viability of cysticerci in naturally infected pigs under the same rearing conditions but different age classes would improve knowledge about the survival of cysts in the body. These suggestions for research would increase insight in European *T. solium* occurrence. When these factors become better known, then our model could be used as basis for future QMRA studies on exposure of *T. solium* in pork. Furthermore, if knowledge becomes available about the dose-response of *T. solium* for human infections, the model could be extended to estimate human incidence of *Taenia solium* taeniosis.

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