



## Effect of climate change on runoff of *Campylobacter* and *Cryptosporidium* from land to surface water



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### ARTICLE INFO

#### Article history:

Received 4 November 2015

Received in revised form

22 February 2016

Accepted 1 March 2016

Available online 2 March 2016

#### Keywords:

Climate change

Pathogen transport

Runoff

Quantitative microbial risk analysis

Lowland catchments

### ABSTRACT

Faeces originating from wildlife, domestic animals or manure-fertilized fields, is considered an important source of zoonotic pathogens to which people may be exposed by, for instance, bathing or drinking-water consumption. An increase in runoff, and associated wash-off of animal faeces from fields, is assumed to contribute to the increase of disease outbreaks during periods of high precipitation. Climate change is expected to increase winter precipitation and extreme precipitation events during summer, but has simultaneously also other effects such as temperature rise and changes in evapotranspiration. The question is to what extent the combination of these effects influence the input of zoonotic pathogens to the surface waters.

To quantitatively analyse the impacts of climate change on pathogen runoff, pathogen concentrations reaching surface waters through runoff were calculated by combining an input model for catchment pathogen loads with the Wageningen Lowland Runoff Simulator (WALRUS). Runoff of *Cryptosporidium* and *Campylobacter* was evaluated under different climate change scenarios and by applying different scenarios for sources of faecal pollution in the catchments, namely dairy cows and geese and manure fertilization. Model evaluation of these scenarios shows that climate change has little overall impact on runoff of *Campylobacter* and *Cryptosporidium* from land to the surface waters. Even though individual processes like runoff fluxes, pathogen release and dilution are affected, either positively or negatively, the net effect on the pathogen concentration in surface waters and consequently also on infection risks through recreation seems limited.

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### 1. Introduction

Several studies have shown a positive correlation between rainfall and waterborne disease outbreaks (Cann et al., 2013; Curriero et al., 2001; Nichols et al., 2009; Signor et al., 2007). These disease outbreaks include acute gastrointestinal illness caused by exposure to water contaminated with zoonotic bacteria or parasites. Due to rainfall, *Campylobacter* and *Cryptosporidium* in animal faeces may be transported from fields to surface waters (Tyrrel and Quinton, 2003). Faeces originating from domestic animals, wildlife or manure-fertilized fields, is considered an important source of zoonotic pathogens to which people may be exposed

by water recreation or drinking-water consumption (Ferguson et al., 2003).

Climate change, causing increase in winter precipitation and more extreme precipitation events, in combination with changes in evapotranspiration, is expected to influence runoff regimes (Bergström et al., 2001). Throughout this paper, the term runoff will refer to the quick flow component of the runoff which is considered all water flowing through quick flow paths, i.e. overland flow plus fast transport through drains or biopore flow. In low relief regions like the Netherlands, the latter has a large contribution to the total amount of quick flow (Rozemeijer and Velde, 2008). Overland flow can occur either when rainfall rate exceeds the infiltration capacity of the soil or because the soil is saturated (Becker et al., 2004; Dunne and Black, 1970). The first type is often dominant in (semi-)arid regions, when soils have low initial soil moisture and/or a very low infiltration rate. Overland flow in the Netherlands largely occurs through saturated flow.

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Exceptions are found in the more hilly areas like the south of Limburg (Kwaad, 1991), during very intensive rain events or during summer when high soil moisture deficits decrease the soils' infiltration capacity (Boxall et al., 2009).

Given the correlation between rainfall and waterborne disease outbreaks, increased pathogen transport to surface waters may be plausible due to climate change. However, besides rainfall, other factors also affect pathogen transport by overland flow. For example, temperature is an important factor that affects the survival of pathogens before and during transport (Tyrrel and Quinton, 2003). Other factors include pathogen numbers on land (Dorner et al., 2004), timing of manure application to soil and subsequent rain events (Donnison and Ross, 2009; Meals and Braun, 2006; Tang et al., 2011), soil composition (Atwill et al., 2002; Donnison and Ross, 2009) and vegetation (Atwill et al., 2002; Trask et al., 2004) which may be directly or indirectly susceptible to climate change. These factors will not solely increase numbers of zoonotic pathogens transported to surface waters by runoff. For example, increase in air temperatures will be unfavourable for the persistence of pathogens deposited on the land surface (Moriarty et al., 2012; Sinton et al., 2007). To predict the sum of effects of climate change on pathogen runoff it is necessary to consider both negative and positive effects (Sterk et al., 2013).

In some studies changes in runoff were incorporated in risk modelling frameworks (Schijven et al., 2013; Smith et al., 2015), however, pathogen runoff to surface waters was not modelled mechanistically. In those studies, an increase of events with high pathogen concentrations attributable to climate change was assumed. However, since the combined effect of processes involved in pathogen transport from land to surface waters is not unequivocal, an estimate of the sum of impacts of climate change on pathogen runoff is necessary before incorporation in risk modelling frameworks.

The aim of the current study is to analyse the net effect of climate change on pathogen runoff and human infection risks in the Netherlands. Most models to determine pathogen transport are created for hill slope areas (Bhattarai et al., 2011; Guber et al., 2011; Kouznetsov et al., 2007). These models only include infiltration excess runoff and are not suitable for lowland catchments like the Netherlands. To predict pathogen transport for lowland catchments, pathogen concentrations reaching surface water through runoff were calculated by combining an input model for pathogen loads in a catchment with The Wageningen Lowland Runoff Simulator (WALRUS, see Brauer et al., 2014a). Hupsel Brook Catchment and the Cabauw polder (Brauer et al., 2014b), were used as representatives for the range of catchments in the Netherlands.

Runoff of *Cryptosporidium* and *Campylobacter* was evaluated under different climate change scenarios by applying different scenarios for sources of faecal pollution in the catchments, namely dairy cows and geese and manure fertilization. Estimated changes in pathogen concentrations in surface waters, driven by changes in runoff, were used to quantify the impact of climate change on the human infection risks due to exposure to waterborne pathogens during recreation.

## 2. Methods

WALRUS (version 1.0 Brauer et al., 2014a) was used as to determine water fluxes in the catchment. Using Mathematica (version, 9.0.1 Wolfram Research Inc., Champaign, USA), results from this model were combined with simulations of pathogen concentrations at the field and pathogen transport within the catchment.

### 2.1. Pathogen concentrations at the field

#### 2.1.1. Pathogen deposition

Depending on the scenario, pathogen deposition in the watershed was calculated as a function of either presence of animals or manure fertilisation.

Pathogen loadings from animals were determined using the method described by Dorner et al. (2004). In short, the fraction of animals infected by *Cryptosporidium* or *Campylobacter* was derived by random sampling from several (equally weighted) beta-distributions, describing the probability that an animal is infected (prev):

$$prev \sim \beta(\alpha, \beta) \quad (1)$$

Parameters,  $\alpha$  and  $\beta$ , for each of the  $\beta$ -distributions are based upon prevalence studies (See supplemental material S1). A mixture of data has been used since pathogen prevalence may differ according to geographical regions, but in most watersheds insufficient data are available regarding pathogen prevalence in local animal populations, and it is difficult to ascertain which studies of animal populations are most similar to animals in another given geographical region (Dorner et al., 2004).

For each positive animal, the number of pathogens shed was determined by multiplying hourly manure production rate  $M_f$  (kg/hr), with the pathogen concentration. Pathogen concentrations were based on random sampling of a gamma-distribution, which according to Dorner et al. (2004), can take on a variety of shapes and could be fit to published shedding intensity data. The gamma distribution represents animal shedding intensity in log number of pathogens per gram of fresh weight manure,  $C_m$ :

$$C_m \sim \Gamma(r, \gamma) \quad (2)$$

with shape and location parameters,  $r$  and  $\gamma$ , based on literature data (See supplemental material S1).

Combining Equations (1) and (2), the number of pathogens deposited per hour ( $N_{dep}$ ) could be calculated:

$$N_{dep} = N_a * prev * M_f * C_m \quad (3)$$

Where  $N_a$  is the number of animals in the catchment.

Pathogen deposition by manure fertilisation was determined multiplying an estimate of the manure spread in the area,  $M_s$  (kg), with pathogen concentrations based on random sampling of the gamma distribution (2):

$$N_{dep} = M_s * C_m \quad (4)$$

#### 2.1.2. Removal by die-off and release

Pathogens in manure are removed by two processes; die-off/inactivation and release due to precipitation. The following differential equation is used to estimate the change in pathogens over time:

$$\frac{dN_f(t)}{dt} = \underbrace{N_{dep}(t)}_{\text{deposition}} - \underbrace{\mu(t)N_f(t)}_{\text{die-off}} - \underbrace{\omega(t)N_f(t)}_{\text{release}} \quad (5)$$

Die-off is described as a first order decay reaction where  $N_f$  is number of pathogens present in manure,  $t$  is time and  $\mu$  (day<sup>-1</sup>) is the decay rate. Release is determined using release rate coefficient,  $\omega(t)$

Decay rate is modelled as function of temperature in the faeces

$T_f$  (°C) and is therefore time dependent. For *Cryptosporidium* the equation of Peng et al. (2008) was used to calculate the decay rate in faeces:

$$\mu_{crypto}(t) = 0.0018e^{0.152(T_f(t)-4)} \quad (6)$$

For *Campylobacter*, no relation was available for decay in faeces. But data from field experiments, showing the reduction of *Campylobacter* within cattle faeces during a summer, spring, fall and winter period were available from Sinton et al. (2007). Temperatures and decay rates between two subsequent measurement points were determined and nonlinear model regression was used to fit a relationship between temperature and decay rate. The result (Equation (20)) has been used to calculate the decay rate of *Campylobacter*.

Temperature in faeces was modelled as a function of solar radiation, Rad (MJ m<sup>-2</sup>), air temperature,  $T_{air}$  (°C), and soil temperature,  $T_{soil}$  (°C) (Sinton et al., 2007):

$$T_f(t) = 3.4 Rad(t) + 0.9 T_{soil}(t) + 0.13 T_{air}(t) \quad (7)$$

Release only occurs during a rainfall event, and was modelled using the release function for the release of *Cryptosporidium* and *Giardia* from Dairy Cattle Manure of Bradford and Schijven (2002). The release rate is given by:

$$\omega(t) = \alpha_f(t) \left( 1 + \alpha_f \beta_f t_{rain}(t) \right)^{-\left( 1 + \frac{1}{\beta_f} \right)} \quad (8)$$

Where  $t_{rain}$  (hr<sup>-1</sup>) is the time passed since the start of the rainfall event, and  $\alpha_f$  (hr<sup>-1</sup>) and  $\beta_f$  (-) are fitting parameters.

Equation (8) has been used to determine the release rate both for *Cryptosporidium* and *Campylobacter*. Even though the equation was not specifically designed for the latter, comparisons of Guber et al. (2006) show that this function is also suitable for other manure constituents.

To determine  $\alpha_f$ , a correlation with rainfall intensity  $P$  (mm hr<sup>-1</sup>) was used (Guber et al., 2006):

$$\alpha_f(t) = 0.0361 + 0.8603 P(t) \quad (9)$$

Because there was no clear relationship to calculate  $\beta_f$ , a value in the range of the results of Guber et al. (2006) and Bradford and Schijven (2002) was used.

With initial conditions of  $N_f = 0$  at  $t = 0$ , the concentration that was released during a rainfall event was calculated using:

$$C_{release}(t) = \begin{cases} \frac{\omega(t) N_f(t)}{P(t) \alpha_G} & P > 0 \\ 0 & P = 0 \end{cases} \quad (10)$$

Where  $\alpha_G$  is the fraction of land surface in the catchment.  $C_{release}$  [#mm<sup>-1</sup>] was used as input concentration for the transport model.

## 2.2. Transport model

The WALRUS model, developed by Brauer et al. (2014a), is a parametric conceptual rainfall-runoff model which is suitable for lowlands where shallow groundwater and surface water influence runoff generation. The model consists of three reservoirs, namely, soil, quick flow and surface water. Water is added at the land surface to the different reservoirs by precipitation ( $P$ ). The soil wetness index ( $W$ ) determines which fraction of this precipitation percolates through the soil matrix ( $P_v$ ) or via the quick flow routes to the surface water ( $P_Q$ ). Water is removed from both the vadose zone

and surface water reservoir by evapotranspiration ( $ET_v$  and  $ET_s$  resp.)

Flow to the surface waters through the quick flow reservoir is denoted as  $f_{QS}$  and depends linearly on the water level in the quick flow reservoir ( $h_Q$ , [mm]). Discharge at the catchment outlet ( $Q$  [mm h<sup>-1</sup>]) is based on a stage-discharge relation that specifies the relation between surface water level ( $h_s$  [mm]) and discharge.

The four model parameters: wetness index parameter ( $c_w$ ), vadose zone relaxation time ( $c_v$ ), groundwater reservoir constant ( $c_g$ ) and quick flow reservoir constant ( $c_q$ ) can be found by calibration using datasets of precipitation and discharge. For more detailed description on the WALRUS model, see Brauer et al. (2014a).

The WALRUS model does not include solute transport, hence pathogen fluxes were added to the model. The concentration released at the source was divided over the flow routes; proportional to the water flow and under the assumption of instant mixing for all fluxes (see Fig. 1 and Table 1 for a more detailed overview of the fluxes).

$C_{release}$  is the concentration of pathogens released from manure into the aquatic phase, resulting in a number of pathogens entering the quick flow reservoir ( $N_{fp}$ ). Because of adsorption to soil particles during transport and longer travel times resulting into more decay,

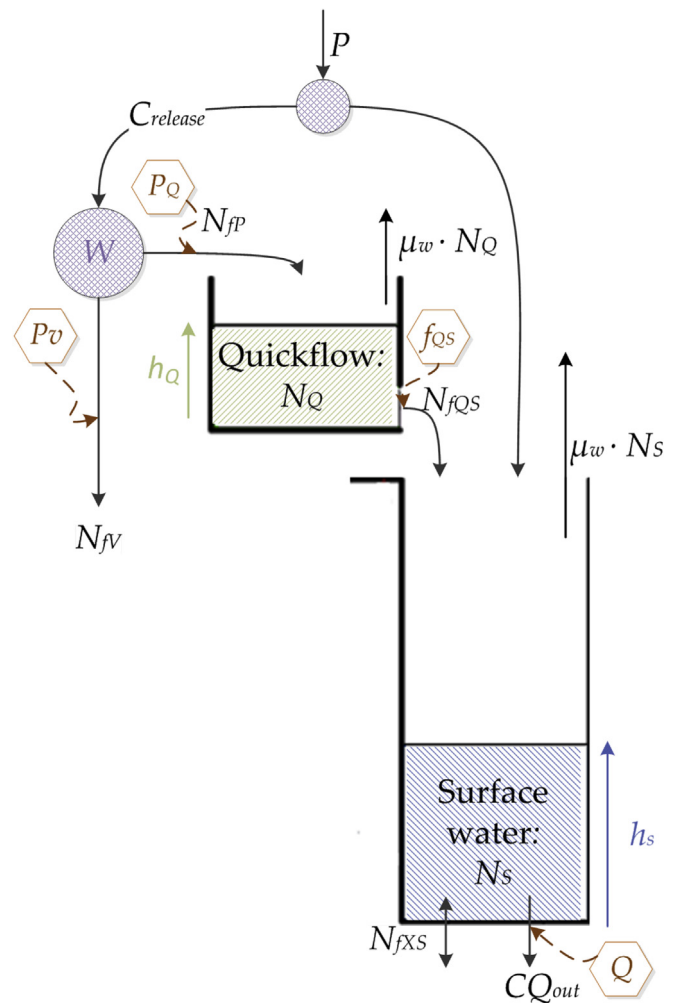


Fig. 1. Pathogen transport fluxes. See Table 1 for description of all variables. Hexagons, Wetness index ( $W$ ) and reservoir states ( $h_Q$  and  $h_S$ ) are variables following from the WALRUS model.

**Table 1**  
Description of variables and fluxes for pathogen transport.

<b>Pathogens reservoirs</b>			
$N_Q$	Pathogen number in quick flow reservoir	$\rightarrow \frac{dN_Q}{dt} = N_{fP} - N_{fQS} - \mu_w N_Q(t)$	[#]
$N_S$	Pathogen number in surface water reservoir	$\rightarrow \frac{dN_S}{dt} = N_{fQS} + N_{fXS} - N_{fS} - \mu_w N_S$	[#]
<b>External fluxes: input</b>			
$N_{fP}$	pathogen input precipitation	$= P_Q C_{release}$	[# h <sup>-1</sup> ]
$N_{fXS}$	pathogen input surface water supply	$= f_{XS} C_{fXS}$	[# h <sup>-1</sup> ]
<b>External fluxes: output</b>			
$N_{fV}$	pathogen number entering vadose zone	$= P_V C_{release}$	[# h <sup>-1</sup> ]
$C_{out}$	pathogen concentration in discharge	$= \frac{N_S}{A Q}$	[# mm <sup>-3</sup> h <sup>-1</sup> ]
<b>Internal fluxes</b>			
$N_{fQS}$	pathogen flux quick flow	$= \frac{N_Q}{h_Q} f_{QS}$	[# h <sup>-1</sup> ]
$N_{fS}$	pathogen flux discharge	$= \frac{N_S}{h_S} Q$	[# h <sup>-1</sup> ]
<b>Constants</b>			
$A$	catchment area		[mm <sup>2</sup> ]
$C_{fXS}$	pathogen concentration surface water supply		[# mm <sup>-1</sup> ]
$\mu_w$	Temperature dependent decay rate in water	$= \frac{\ln 10}{10^{a_0 + a_1 T}}$	[h <sup>-1</sup> ]
<b>Fluxes and states following from WALRUS</b>			
$P_Q$	precipitation into quick flow reservoir	$= P W \alpha_g$	[mm h <sup>-1</sup> ]
$P_V$	precipitation into vadose zone	$= P (1 - W) \alpha_g$	[mm h <sup>-1</sup> ]
$f_{QS}$	Quick flow	$= \frac{h_Q}{c_Q} \alpha_g$	[mm h <sup>-1</sup> ]
$Q$	discharge	$= \text{func}(h_S)$	[mm h <sup>-1</sup> ]
$W$	wetness index	$= \cos\left(\frac{\max(\min(\frac{dv}{c_w}, cw), 0)\pi}{c_w}\right) \frac{1}{2} + \frac{1}{2}$	[-]
$h_Q$	level quick flow reservoir	$\rightarrow \frac{dh_Q}{dt} = \frac{P_Q - f_{QS}}{\alpha_g}$	[mm]
$h_S$	surface water level	$\rightarrow \frac{dh_S}{dt} = \frac{f_{XS} - P_S - ET_S + f_{GS} + f_{QS} - Q}{\alpha_S}$	[mm]

the contribution of pathogens through groundwater fluxes was assumed to be limited compared to the surface water fluxes and hence neglected in the current model. Pathogens entering the vadose zone ( $N_{fV}$ ) were therefore considered as an external flux.

The change in the number of pathogens in the quick flow reservoir was computed as:

$$\frac{dN_Q(t)}{dt} = N_{fP}(t) - N_{fQS}(t) - \mu_w N_Q(t) \quad (11)$$

Where  $\mu_w$  is the pathogen decay rate in water and  $N_{fQS}$  is the number of pathogens transported from the quick flow reservoir into the surface water reservoir.

$$N_{fQS}(t) = \frac{N_Q(t)}{h_Q(t)} f_{QS}(t) \quad (12)$$

Pathogens could enter the surface water not only by quick flow, but also through surface water supply ( $f_{XS}$ ). The change in the number of pathogens in the surface water reservoir is:

$$\frac{dN_S(t)}{dt} = N_{fQS}(t) + N_{fXS}(t) - N_{fS}(t) - \mu_w N_S(t) \quad (13)$$

Where  $N_{fS}$  is the number of pathogens in the discharge.

$$N_{fS} = \frac{N_S}{h_S} Q \quad (14)$$

Decay rates in water differ from those in faeces, but in the model no distinction was made between the runoff and surface water. To determine the decay rate in water the following equation was used:

$$\mu_w(t) = \frac{\ln 10}{10^{a_0 + a_1 T}} \quad (15)$$

$a_0$  (log<sub>10</sub> day) and  $a_1$  (log<sub>10</sub> day °C<sup>-1</sup>) are inactivation rate parameters: 0.53 and -0.017 respectively for *Campylobacter* and 3.1 and -0.078 respectively for *Cryptosporidium* (Schijven et al., 2013).

The number of pathogens in the surface water compartment was

used to calculate the pathogen concentration in the discharged water,  $C_{Qout}$  [# / L<sup>3</sup>]:

$$C_{Qout}(t) = \frac{N_{fS}(t)}{A Q(t)} \quad (16)$$

Where  $A$  is the area of the catchment [L<sup>2</sup>].

## 2.3. Scenarios

### 2.3.1. Catchments

Brauer et al. (2014b), applied the Walrus model for two catchments in the Netherlands, namely the Hupsel Brook catchment and the Cabauw polder. They showed that after calibration, WALRUS produced good results for both catchments, with Nash-Sutcliffe efficiencies of 0.87 and 0.83 for Hupsel and Cabauw respectively.

Hupsel Brook catchment represents the part of the Netherlands that is above mean sea-level in the east and south of the country while the Cabauw polder represents the lower parts with controlled water levels in the west and north. See [supplemental material S2](#) for characteristics and calibration parameters of these two catchments.

Seepage ( $f_{GS}$ ) and surface water supply ( $f_{XS}$ ) are only present in the Cabauw catchment. Because no seepage data are available, Brauer et al. (2014b) estimated seepage as the residual of the water budget of the year Nov. 2007–Oct. 2008 assuming a constant seepage flux year-round. Surface water levels in the Cabauw polder are controlled by adjusting weir elevations and regulating surface water supply. The variable inlet is used to maintain these surface water levels. However, since data for surface water supply was only present for a limited period, and the irregularity of the decisions of water managers for changing weir levels and surface water supply made it impossible to give a reliable model for the value of  $f_{XS}$ , it was chosen to use the median of the measurements from 2007 to 2011 (Brauer et al., 2014b). The weir level ( $h_{SMIN}$ ) in the Cabauw polder was set to 500 mm (winter, Nov–June) and 600 mm (summer, June–Nov) from the channel bottom.

For Hupsel, the default stage-discharge relation given in WALRUS was used to determine discharge. For Cabauw, the specific stage-discharge relation for the weirs at the outflow point of the catchment was used (Brauer, 2014)

$$Q(h_s) = \begin{cases} 2.247h_s^2 + 0.0391h_s - 0.00006 & \text{if } h_s < 0.0307 \\ -0.4176h_s^3 + 1.409h_s^2 + 0.089h_s - 0.00008 & \text{if } h_s \geq 0.0307 \end{cases} \quad (17)$$

### 2.3.2. Input scenarios

Three different input scenarios were evaluated: 1) grazing dairy cattle; 2) wild geese; and 3) manure fertilisation. In case of the grazing animals, pathogen input is added every hour over the period that the animals are present. Numbers of animals are given in Table 2. Manure fertilisation was assumed to take place three times per year on a random day, under the conditions that this day was between February 1st – September 1st and when the soil temperature was above 0 °C. Dutch regulations prohibit manure fertilisation outside this period or when the soil is covered with snow, (partly) frozen or fully saturated with water.

The number of animals per catchment was calculated by multiplying and rounding the number of animals per ha with the total area of the catchment and the fraction of grassland.

For the fertilisation scenarios, it was assumed that the entire mais-area of the catchment is fertilised on the same day. In case of the Cabauw catchment, besides input from the animals, pathogens could also enter the system through surface water supply. Since the main focus was the effect of climate change on runoff, as a default, the inflow concentration  $C_{\text{FS}}$  was set to 0.

### 2.3.3. Climate change scenarios

For the Netherlands, the Royal Netherlands Meteorological Institute (KNMI) developed four different climate change scenarios, which predict changes in air temperature, precipitation, wind and sea level in 2050 and 2085 compared to the period 1981–2010 (KNMI, 2014b). The scenarios predict an air temperature rise of about 1–1.3 °C and 2.3–3.7 °C for 2050 and 2085 respectively. Predictions for precipitation show that on average, according to all scenarios, winters will become wetter and extreme precipitation amounts will increase. Changes in precipitation for summer are more difficult to predict, hence they are given with a large uncertainty band.

Data of the KNMI for hourly precipitation, temperature, radiation and evaporation from 1981 to 2010 were used as reference scenario (KNMI, 2015). This 30 year period is assumed to capture the natural variation. Hourly data from the meteorological stations Hupsel and Cabauw were not available for the entire period, instead data of the Bilt were used for both catchments. The data for the climate scenarios was transformed using the method developed by

the KNMI (Bakker, 2012; KNMI, 2014a). For precipitation, the hourly data were transformed using a nonlinear transformation method (Bessembinder, 2012).

Evapotranspiration was not transformed directly. Instead, transformed values for temperature and radiation were used to calculate the Makking reference evapotranspiration (Hiemstra, 2011).

The transformations resulted in a 30 year-time series, where each year represents a possible scenario for the climate in 2050 or 2085. Changes in transmission of pathogens to surface water by runoff were calculated for both years under the  $G_L$  and  $W_H$  climate scenario (see supplemental material S3). The  $G_L$  scenarios were evaluated using the lower limit of the prediction band for changes of precipitation in summer and the  $W_H$  scenarios with the upper limit to create the most conservative and extreme predictions.

### 2.4. Sensitivity of the individual processes

As explained before, different processes determine the concentration at the outlet catchment, namely: release, decay in the faeces, decay in the water and dilution. To examine the individual effect of these processes, a sensitivity analysis was conducted.

The model was run for the scenario of cattle at the field with climate data for the year 2009 and simulations were carried out subtracting 10% from and adding 10% to the process parameters (e.g.  $\omega$ ,  $\mu$ ,  $h_Q$ ,  $h_S$ ). Differences between Cabauw and Hupsel and *Campylobacter* and *Cryptosporidium* were evaluated visually.

### 2.5. Risk analysis

To get an indication of the health implications of the changes in concentrations, a risk analysis has been conducted assuming bathing takes place in the catchments.

For this calculation, it was assumed that bathing takes place during the bathing season (1st of May to 1st of October), only when water temperatures exceed 17 °C.

The ingested dose,  $D$  was calculated using:

$$D = C V \quad (18)$$

where  $C[L^{-1}]$  is the predicted pathogen concentration under the scenario examined and  $V[L]$  is the individual volume of water that was consumed. Distributions of ingested volumes of water during swimming in fresh water were based on random sampling from the gamma distribution of Schets et al. (2011).

The risk of infection ( $R_{\text{inf}}$ ) per exposure event was calculated using the following dose response relationship (Teunis et al., 2008):

$$R_{\text{inf}} = 1 - {}_1F_1(\alpha, \alpha + \beta; -D) \quad (19)$$

where  ${}_1F_1$  is the hypergeometric distribution and  $\alpha$  and  $\beta$  are the

**Table 2**  
Input data to determine pathogen production at the field per input scenario.

	Dairy cattle	Geese	Manure-fertilized
Nr of animals per ha	2.5 <sup>a</sup>	Summer: 0.7 <sup>c</sup> Winter: 5 <sup>c</sup>	–
Manure per m <sup>2</sup> [kg/m <sup>2</sup> /event]	–	–	1 <sup>a</sup>
Manure production [kg/animal/day]	58.8 <sup>b</sup>	0.25 <sup>d</sup>	–
Period	Grazing <sup>e</sup> on average 175 day/yr, 11 hr/day	Year round	Three times per year Februari–September

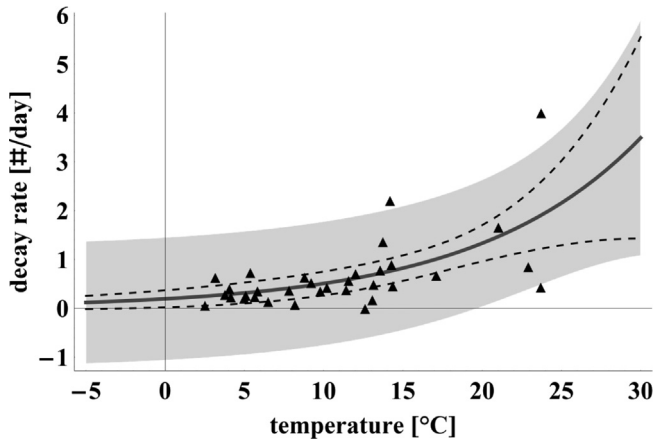
<sup>a</sup> Based on regulations of max 170 kg Nitrogen/year/ha, and assuming 0.005 kgN/kg cow manure and excretion of 58.8 kg/animal during grazing (CBS, 2014).

<sup>b</sup> (CBS, 2014).

<sup>c</sup> Based on geese counts in winter and summer (Sovon, 2014).

<sup>d</sup> Hussong et al. (1979).

<sup>e</sup> Keuper and van der Schans (2011).



**Fig. 2.** Decay rate of *Campylobacter* in faeces, depending on temperature. Triangles show decay rate between two subsequent measurement points with temperature (data: Sinton et al., 2007); Dashed line encompass the 95% confidence bands for mean predictions; the grey-shaded area encompasses the prediction interval for individual observations.

parameters of the Beta-distribution, (0.038 and 0.022 respectively for *Campylobacter* and 0.106 and 0.295 respectively for *Cryptosporidium* (Schijven et al., 2013)).

Risks of infection with *Campylobacter* and *Cryptosporidium* were calculated (Mathematica 9.0.1 Wolfram Research Inc., Champaign, USA) using Monte Carlo simulations with random sampling of 10 000 values. In order to evaluate effects of climate change for current and future summers under the different  $G_L$  and  $W_H$  scenarios, a fixed seed number was used in the Monte Carlo simulations.

### 3. Results

#### 3.1. Pathogen input

Based on the data of Sinton et al. (2007), the following equation was derived for the decay rate of *Campylobacter* in faeces (see Fig. 2):

$$\mu_{campy}(t) = 0.19e^{0.096T_f(t)} \quad R^2 = 0.65 \quad (20)$$

This result has been used, together with Equations (1)–(10), to determine the release of pathogens during a rain event.

Release of pathogens at the Cabauw and Hupsel catchment only slightly differed in absolute numbers because of differences in land use and cover, but in terms of percentage, effects of climate change were similar.

The changes in released *Campylobacter* and *Cryptosporidium* for the different input scenarios are shown in Table 3. For geese and cattle, both direction and extent of these changes differed per scenario. For the most conservative scenarios ( $G_L$ ), concentrations decrease in 2050 with approximately 9% for *Campylobacter* and 5% for *Cryptosporidium*. Predictions for 2085 showed an increase, except for the scenario of geese, which showed a slight decrease. The more extreme ( $W_H$ ) scenarios show an increase for the mean released concentration for both 2050 and 2085, with, in general, a higher increase of *Cryptosporidium* concentrations.

Calculations for release from manure fertilised fields deviated considerably (in the order of 1–2 log units) each time the model was run, depending on the timing of the fertilisation. Therefore, the result in Table 3 should be seen as one exemplary model run.

Fig. 3 shows a histogram for the released concentration of pathogen for the different input scenarios. This histogram shows that for geese, the pathogen concentration was either high or 0, while for cattle and manure fertilisation the whole range of concentrations occurred.

#### 3.2. Quick flow flux

Table 4 shows the change in quick flow for the different climate scenarios, both for the upper and lower precipitation scale. Over the year, all scenarios showed an increase in both average and extreme (.95% level) quick flow. However, as shown in Fig. 4, the amount and direction of change differed per season.

For Cabauw, mean quick flow increases more in case of the most conservative scenario ( $G_L$ ) compared to the most extreme climate scenario ( $W_H$ ), while for Hupsel the opposite is the case. The increase of the 95% levels of quick flow in the Cabauw catchment was much higher for the extreme scenario.

#### 3.3. Pathogen concentrations in river discharge

Pathogen concentrations in the river discharge decreased for all input sources and for all scenarios (see Table 5A–B), though the relative amount of decrease differed when comparing the pathogens, catchments and input sources. Overall, the highest decreases were found for the 2085 $W_H$  scenario.

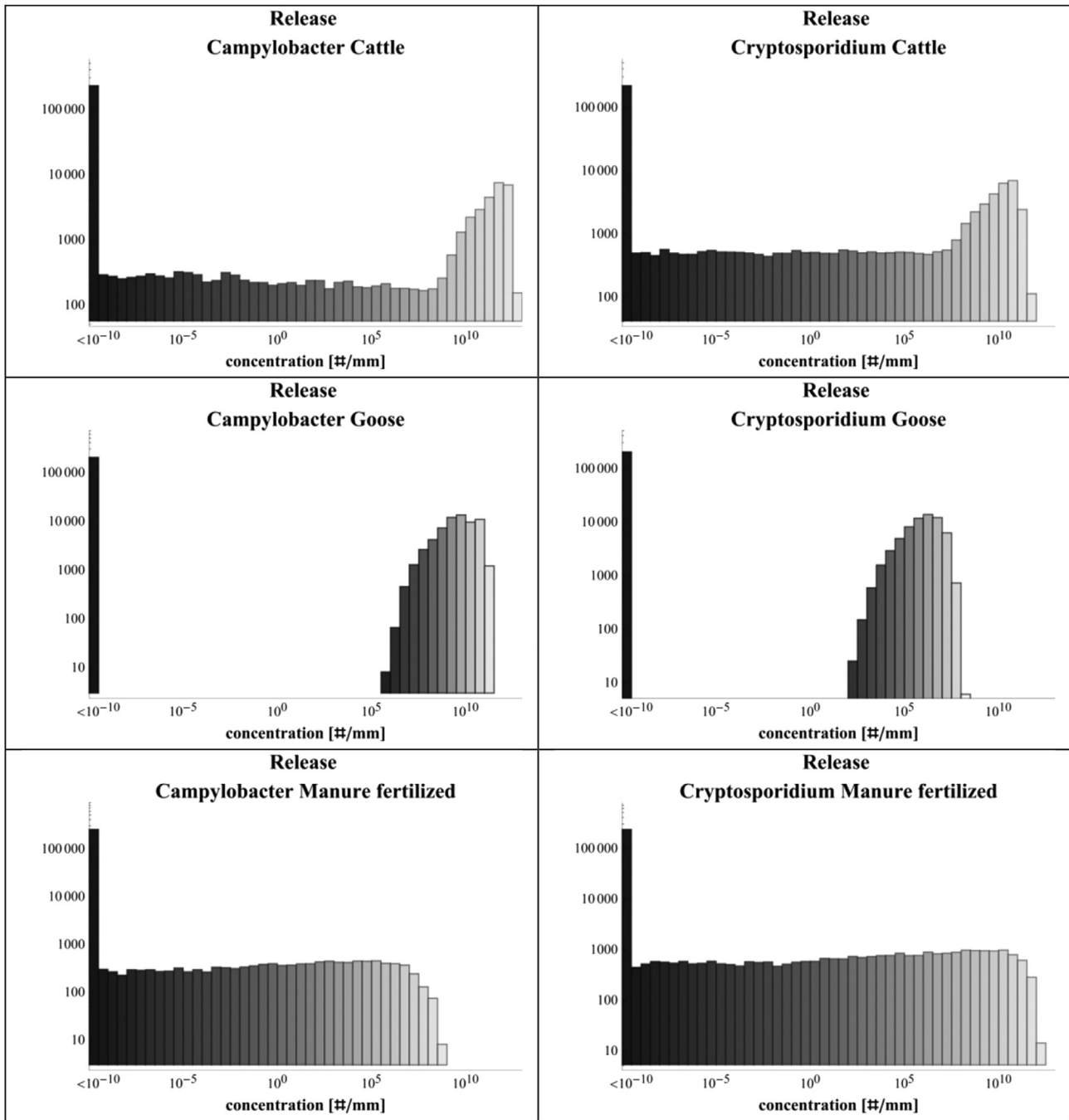
Unlike the results for release, pathogen concentration results for the input from manure fertilised fields were almost constant over each model run. Fig. 5 illustrates seasonal differences with the amount and direction of change per season in the Cabauw catchment.

#### 3.4. Sensitivity of the individual processes

The different processes (i.e. die-off, release and dilution) were

**Table 3**  
Changes in released concentrations of *Campylobacter* and *Cryptosporidium* for the different climate scenarios (change of 95% level between parentheses).

	1981–2010 Hupsel [nr/mm]	1981–2010 Cabauw [nr/mm]	2050 $G_L$	2050 $W_H$	2085 $G_L$	2085 $W_H$
<b><i>Campylobacter</i></b>						
Cattle:	6.6E+10	7.1E+09	–9.5% (–12%)	+57% (+42%)	+6.8% (+12%)	+30% (–62%)
Geese:	3.9E+09	4.3E+08	–9.9% (–8.8%)	+23% (+26%)	–2.1% (0.0%)	+50% (+50%)
Manure fertilised:	–	8.0E+04	–8.1% (–97%)	+55% (–100%)	+6.8% (–98%)	+47% (–100%)
Manure fertilised:	2.4E+06	–	–7.8% (–96%)	+36% (–100%)	–0.3% (–97%)	+59% (–100%)
<b><i>Cryptosporidium</i></b>						
Cattle:	3.7E+09	4.0E+08	–5.1% (–6.3%)	+101% (+82%)	+14% (+22%)	+68% (–46%)
Geese:	8.3E+06	9.1E+05	–4.8% (–5.0%)	+50% (+55%)	+6.8% (+7.7%)	+89% (+88%)
Manure fertilised:	–	1.4E+10	–2.8% (–27%)	+85% (+224%)	+15% (+12%)	+60% (–100%)
Manure fertilised:	3.7E+10	–	–1.5% (–29%)	+80% (+430%)	+11% (+16%)	+66% (–100%)



**Fig. 3.** Histogram of released *Cryptosporidium* (left pane) and *Campylobacter* (right pane) for the evaluated scenarios for cattle, geese and manure fertilisation. Results are shown for the Cabauw catchment only, but results for Hupsel were very similar.

**Table 4**

Increase of quick flow for the different climate scenarios (change of 95% level between parentheses).

$f_{QS}$	1981–2010	2050G <sub>L</sub>	2050W <sub>H</sub>	2085G <sub>L</sub>	2085W <sub>H</sub>
Hupsel	238 mm/yr	+8.0% (+7.8%)	+10% (+8.1%)	+11% (+11%)	+17% (+11%)
Cabauw	360 mm/yr	+6.0% (+6.1%)	+3.6% (+13%)	+9.1% (+7.7%)	+6.1% (+21%)

found to contribute differently to the concentrations of *Campylobacter* and *Cryptosporidium* in the discharge (see Fig. 6). *Cryptosporidium* seemed to be mainly affected by die-off and dilution in the surface waters, while the other processes have only minor

effects. For *Campylobacter*, again die-off and dilution in the surface water compartment have the highest influence, but contribution of the other processes is important as well. Note that for *Campylobacter*, impact of die-off and dilution in the quick flow

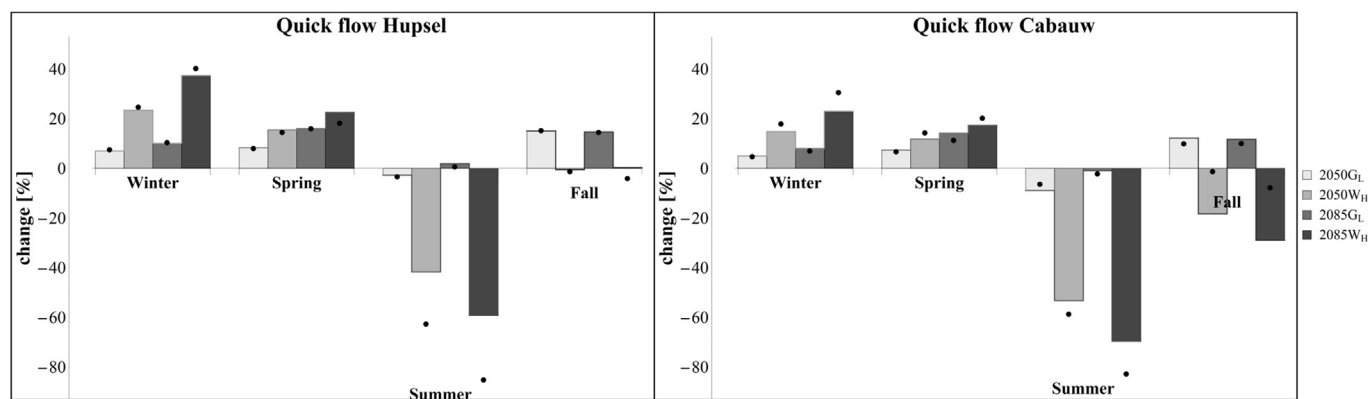


Fig. 4. Average change in quick flow as per season for the evaluated scenarios for Hupsel (left pane) and Cabauw (right pane). Black dots give the change for the 95% level.

Table 5

Changes in mean concentrations at the catchment outlet of Hupsel (A) and Cabauw (B) for *Campylobacter* and *Cryptosporidium* for the different climate scenarios (change of 95% level between parentheses).

A	Hupsel	1981–2010	2050GL	2050WH	2085GL	2085WH
	<i>Cryptosporidium</i>	[nr/L]				
	Cattle:	4.0E-01	–17% (–21%)	–32% (–31%)	–22% (–24%)	–47% (–47%)
	Geese:	1.5E-03	–15% (–18%)	–30% (–33%)	–22% (–26%)	–42% (–44%)
	Manure fertilised:	6.4E+00	–17% (–19%)	–31% (–31%)	–23% (–23%)	–46% (–44%)
	<i>Campylobacter</i>	[nr/L]				
	Cattle:	8.3E-02	–14% (–11%)	–34% (–26%)	–15% (–13%)	–46% (–34%)
	Geese:	7.2E-03	–13% (–13%)	–30% (–28%)	–18% (–17%)	–41% (–39%)
	Manure fertilised:	5.8E-06	–11% (–42%)	–26% (–79%)	–15% (–47%)	–36% (–92%)
B	Cabauw	1981–2010	2050GL	2050WH	2085GL	2085WH
	<i>Cryptosporidium</i>	[nr/L]				
	Cattle:	1.4E-02	–18% (–11%)	–58% (–38%)	–18% (–12%)	–71% (–51%)
	Geese:	2.4E-04	–7.9% (–6.3%)	–26% (–19%)	–10% (–8.5%)	–37% (–28%)
	Manure fertilised:	1.5E-01	–11% (–5.7%)	–24% (–15%)	–12% (–7.2%)	–34% (–22%)
	<i>Campylobacter</i>	[nr/L]				
	Cattle:	1.7E-03	–11% (–11%)	–47% (–48%)	–11% (–10%)	–54% (–56%)
	Geese:	6.4E-04	–14% (–15%)	–36% (–35%)	–18% (–19%)	–49% (–46%)
	Manure fertilised:	8.2E-08	–17% (–50%)	–35% (–85%)	–20% (–46%)	–44% (–94%)

compartment were much higher for Cabauw compared to Hupsel.

### 3.5. Risk analysis

Risk analysis showed a much higher risk of infection in case of the cattle input scenario compared to the geese. Predictions of the risk of infection for the climate change scenarios all showed a decrease of for most scenarios, except for the GL scenarios for *Campylobacter* in the Cabauw catchment, which showed an increase up to 59% in 2085 in case of the cattle input scenario (Table 6A–B).

Risks of infection were overall higher for *Cryptosporidium* than for *Campylobacter*. Fig. 7 shows the cumulative frequency distribution of infection risks at Cabauw, with cattle as an input source.

## 4. Discussion

According to the model calculations, quick flow fluxes will increase due to climate change for all climate scenarios. Also, the release of *Campylobacter* and *Cryptosporidium* increased for most scenarios. However, when combining pathogen release and quick flow fluxes, mean concentrations of both *Campylobacter* and *Cryptosporidium* at the catchment outlet decreased slightly, less than one log<sub>10</sub> unit, for all scenarios. Apparently, increases in water volume outweigh the increase in pathogen numbers, which leads to a lower concentration. Looking at the influence of processes

separately (section 3.4), also confirms that pathogen concentration in the discharge is largely determined by the level of dilution.

As expected, the decrease in pathogen concentration in receiving surface waters in general also leads to a decrease in infection risks upon human exposure. Surprisingly, the infection risk from exposure to surface water at Cabauw increased for the GL scenarios. For these scenarios, pathogen concentration increased for the period that people were assumed to be exposed i.e. only during summer when water temperatures exceed 17 °C. This assumption is not evidence based and indicates the need for a better estimate of probabilities of human exposure to surface waters to improve predictions of the infection risks through bathing (see also Sterk et al., 2015).

For released pathogen concentration, only slight differences were found between the two catchments, caused by variation in land use and cover. Hence, the differences in results between the two catchments were mainly caused by the differences in quick flow; predictions for the most extreme scenario showed +17% increase in mean quick flow for Hupsel compared to +6% for Cabauw. An explanation for this difference is the difference in evapotranspiration reduction in both catchments (see Brauer et al., 2014). For Cabauw, the increase in actual evapotranspiration partly compensated the increase in precipitation, especially for the extreme scenario (WH). This indicates the importance of catchment characteristics for its sensitivity to climate change.

Several studies describe predictions for runoff under future



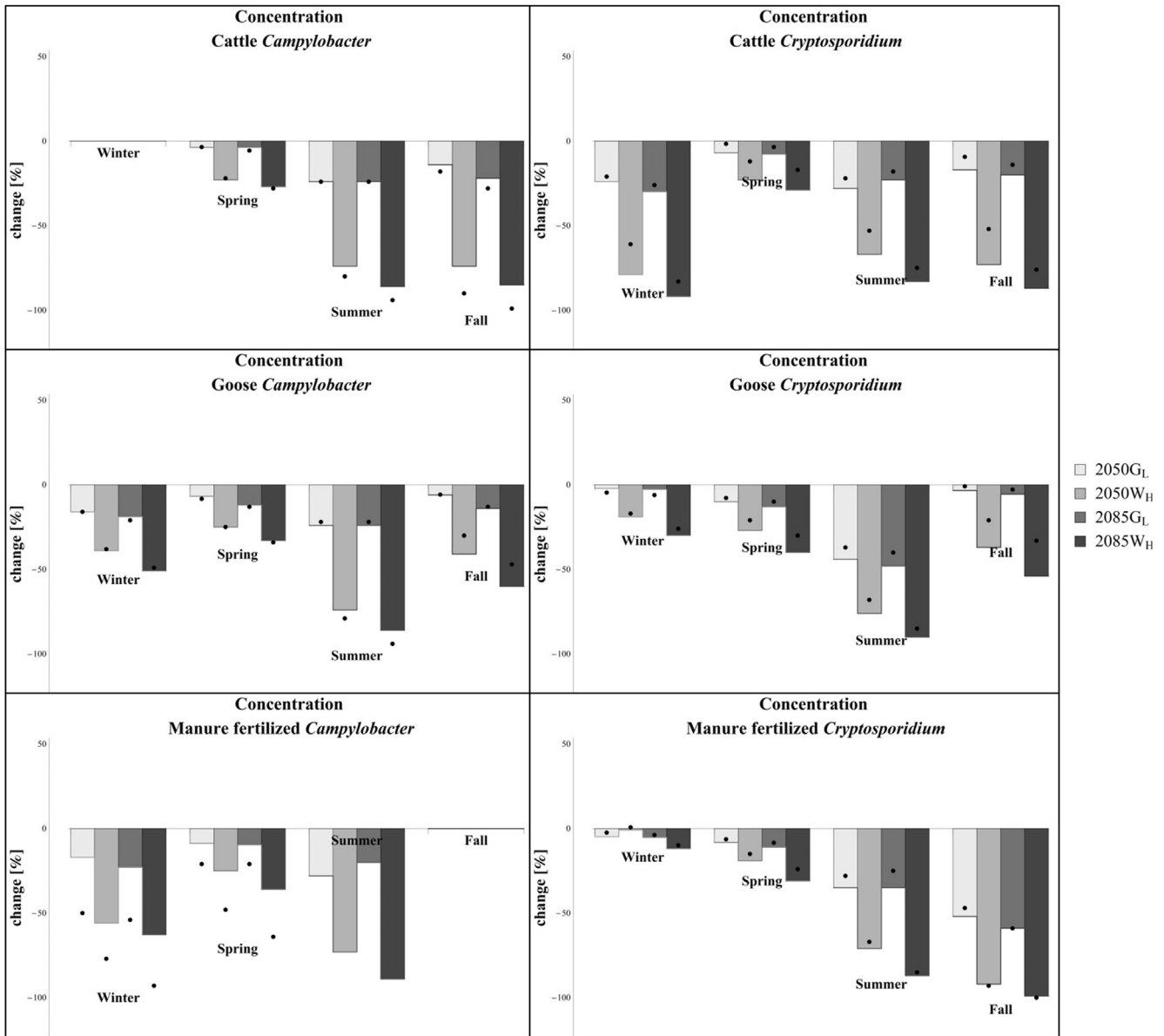
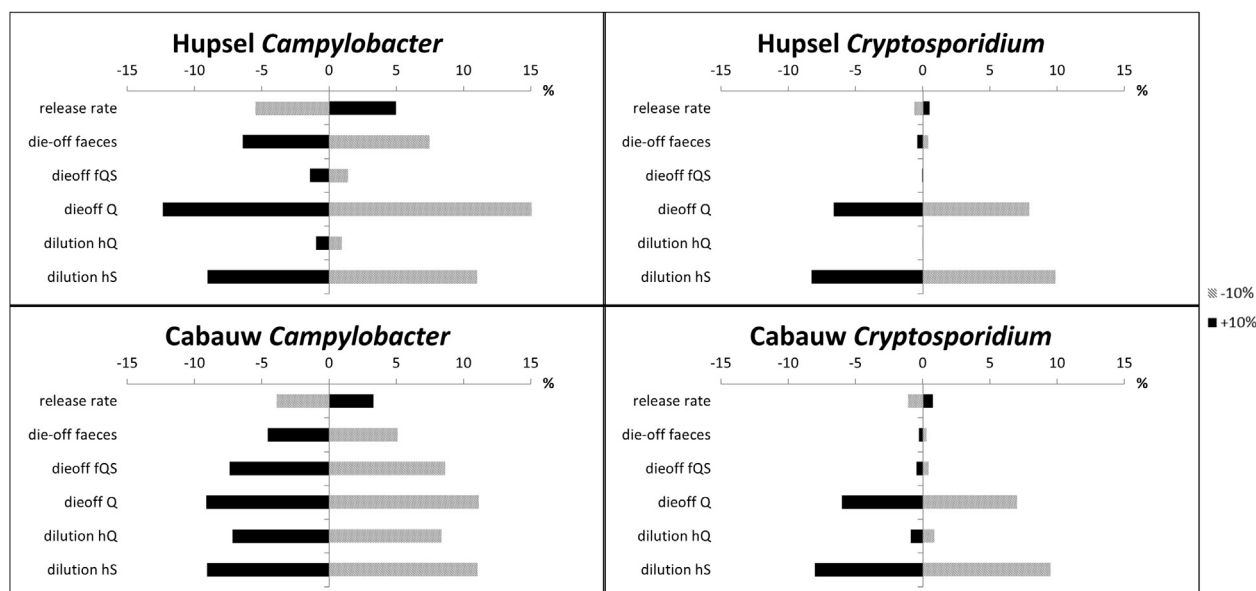


Fig. 5. Results for the Cabauw catchment: Average change in concentrations at discharge for *Campylobacter* (left) and *Cryptosporidium* (right) as per season for the evaluated scenarios for cattle, geese and manure fertilised fields. Black dots give change for the 95% level.

climate scenarios. Predictions for several streams in Denmark showed an increase in yearly average. They also showed a change in seasonal pattern, where monthly runoff generally increased in winter (December–March) and decreased in the late summer (Andersen et al., 2006; Thodsen, 2007). On the other hand, predictions for a catchment in the lower Harz Mountains in central Germany showed that total runoff and interflow will increase by 3.8% and 3.5% in the period 2021–2050 but decrease by 33% and 31% in 2071–2100 compared to the baseline scenario case (Anis and Rode, 2015). However, the catchment response cannot be compared, since this is a sloped area where discharge peaks are not only generated by rainfall but also snowmelt and soil-frost induced runoff. As also mentioned by Capell et al. (2014), lowland areas have a very different vulnerability to climate change compared to montane catchments since factors like snow accumulation do not play an important role.

Predictions for changes in pathogen concentration under

climate change scenarios thus far are not available on the catchment scale. However, there are several predictions for alterations in input for nutrients such as nitrogen and phosphate. For example, model results for the phosphate loading of Danish streams suggested a 3–17% increase within the next 100 year depending on soil type and region (Jeppesen et al., 2009). For nitrogen, Andersen et al. (2006) showed a 8% increase. Even though pathogens and nutrients have similar sources and transport mechanisms, using nutrients as an indication for pathogen concentration is difficult because of absence of biogeochemical processes such as exchange with the atmosphere and the effects of die-off on the transport of pathogens. In fact, predictions for one pathogen do not even have to correspond to another. Differences between bacterial, viral and parasitic pathogens in persistence and other characteristics complicate mutual comparison and are likewise complicating comparisons to indicator organisms (Hörman et al., 2004; Wilkes et al., 2009). For example, in this study, pathogen release increases due to climate



**Fig. 6.** Effect of a 10% increase or decrease for the processes individually on concentration of *Campylobacter* (top) and *Cryptosporidium* (bottom) at the outlet of the catchment for Cabauw (left pane) and Hupsel (right pane). Results shown for cattle.

**Table 6**

Current average risk of disease per person for *Campylobacter* and *Cryptosporidium* for Hupsel (A) and Cabauw (B) and change in percentage of average risk for future scenarios; 95th percentile between parentheses.

A	Hupsel	1981–2010	2050G <sub>L</sub>	2050W <sub>H</sub>	2085G <sub>L</sub>	2085W <sub>H</sub>
<b><i>Campylobacter</i></b>						
	Cattle	7.3E-05 (3.6E-04)	-7%	-21%	0%	-33%
	Geese	3.7E-07 (1.7E-06)	-7%	-22%	-4%	-33%
<b><i>Cryptosporidium</i></b>						
	Cattle	5.5E-03 (2.8E-02)	-24%	-44%	-31%	-58%
	Geese	1.1E-05 (4.0E-05)	-33%	-57%	-49%	-65%
B	Cabauw	1981–2010	2050G <sub>L</sub>	2050W <sub>H</sub>	2085G <sub>L</sub>	2085W <sub>H</sub>
<b><i>Campylobacter</i></b>						
	Cattle	8.2E-06 (2.7E-05)	+34%	-23%	+59%	-27%
	Geese	2.8E-08 (8.6E-08)	+25%	-29%	+39%	-25%
<b><i>Cryptosporidium</i></b>						
	Cattle	1.4E-03 (7.3E-03)	-11%	-42%	-5%	-56%
	Geese	4.6E-06 (2.3E-05)	-24%	-51%	-32%	-62%

change are, in general, larger for *Cryptosporidium*. Since *Campylobacter* is more temperature sensitive, its survival is more afflicted than *Cryptosporidium* (see Equations 6–15–20). *Cryptosporidium* suffers less due to the increase in temperature and radiation, consequently the increased precipitation is the dominant influence on its release.

The extent of the impact of climate change on pathogen concentrations in surface waters is also affected by the type of input source. This mainly has to do with the period in which the different types of input source is present. Climate change is not equal in each period of the year, therefore when the source is mainly present in winter, like the geese, this will have a different effect than when the source is mainly present in summer, e.g. the cows. With respect to the third scenario, the percentage of decay for release from manure fertilised field depends mainly on the moment of fertilisation. In the model, fertilisation occurs at three random days under conditions when fertilisation is allowed. Coincidence of fertilisation and rain events influences the pathogen concentration that enters the surface waters; deposition of manure before a period of drought leaves a larger period of decay before release into the water course

and vice versa.

The model has several limitations that may influence the study outcomes. WALRUS has been shown to predict the water fluxes in the catchments of Hupsel and Cabauw quite well (Brauer et al., 2014b). However, the model predictions have been made using measurements for fXS. In the current model situations, a constant value has been used for fXS, and pathogen concentration in fXS was ignored. Also, WALRUS only includes saturation excess runoff. As mentioned in the introduction, runoff in the Netherlands is largely determined by saturation excess, except during dry periods in summer when high soil moisture deficits decrease the soils' infiltration capacity. Neglecting of infiltration excess runoff may have underestimated the consequences of climate change.

For inclusion of pathogen transport in the model, several assumptions have been made which affected the results. First of all, transport of pathogens was simplified to fluxes proportional to the water flow, i.e. instant mixing was assumed. In reality, dilution is not instant (Fischer et al., 1979) which could lead to an underestimation of pathogen concentration in areas where runoff has just entered the surface water.

Furthermore, attachment and immobilisation to vegetation and the soil, that is found to take place during overland transport (Guber et al., 2007; Trask et al., 2004), was ignored. This could have been included in the model using an attachment rate, as implemented in e.g. the STWIR module by Guber et al. (2011). However, since an estimate for this rate at the catchment scale is not available, nor how it would change under climate change, this would not necessarily improve the predictions. Another limitation regards the absence of a spatial component in the model. Input of pathogens is assumed to be evenly spread at surface of the catchment, and prevalence and pathogen concentration in the animals are randomly taken from the distribution every hour, while in reality this may be clustered in space and time (White et al., 2001). Injection of manure (which is common practice in many countries), instead of broadcasting at the soil surface is ignored, while experiments show that injection decreases the amount of faecal microorganisms in runoff to surface waters (Heinonen-Tanski and Uusi-Kämpä, 2008). Also, input data for the pathogen model were collected from a lot of different studies i.e. including different

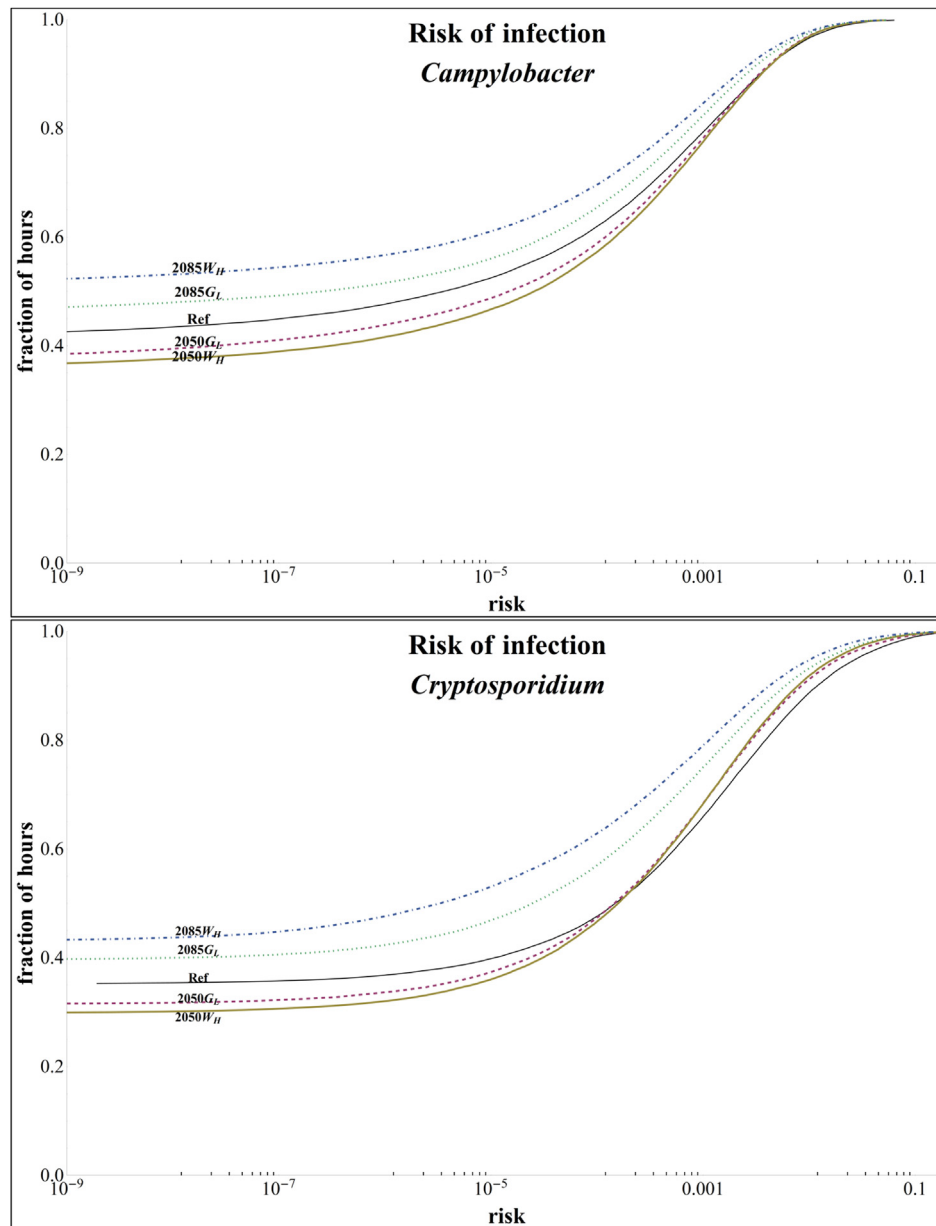


Fig. 7. Fraction of time that infection risk is below a certain value. Results shown at Cabauw for *Campylobacter* (top) and *Cryptosporidium* (bottom) for the scenario of presence of cattle in the catchment and under the different climate scenarios.

countries, methods, conditions and pathogen strains (see supplemental material S1). Unfortunately, no study was available that collected all necessary data for the Netherlands while, as also mentioned by Dorner et al. (2004), geographical differences in concentration of pathogens in faeces are likely.

Accuracy of the absolute results of the model is unknown since pathogen transport in the model was not calibrated nor verified. Concentrations in the order of 0–87 #/L for *Cryptosporidium* have been found in Dutch waters (Hoogenboezem et al., 2000; Schets et al., 2008), but for verification of the model a sequence of measurements at the catchment outlets of Cabauw and Hupsel over a certain period would be needed. Furthermore, estimates of the actual number of animals present in these catchments would be needed. In absence of these data, current results are based on scenarios only.

Without absolute numbers, the model can still be used to

estimate the relative effect of climate change. However, to determine the extent of the health effects, absolute numbers are needed to determine the risk of infection. When absolute risks are small, risks still remain small even given a large relative increase, though when risks are already large, even a small increase could have a significant impact on human health.

Furthermore, the presented predictions are based only on direct effects of climate change (e.g. changes in temperature, precipitation, evapotranspiration and radiation). It is expected that climate change has indirect effects on transport in runoff as well, for example through changes in soil conditions, vegetation or perhaps prevalence in animals (Sterk et al., 2013). Incorporation of such effects in model predictions is difficult since changes are not clearly defined yet and neither is their effect on pathogens. Likewise, scenarios only involved changes in climate. Changes in agricultural management in the future, like for instance the trend of decline in

grazing animals on the fields (Helming and Reijs, 2014), and changed fertilisation regimes due to altered regulations (Schröder and Neeteson, 2008) could also have a large impact on the concentration of pathogens reaching the surface waters and therefore possible public health effects both for humans and animals.

## 5. Conclusions

- This study shows that for the evaluated scenarios, climate change has little impact on concentrations of *Campylobacter* and *Cryptosporidium* transported from land to the surface waters. Even though individual processes like runoff fluxes, pathogen release and dilution are affected, either positively or negatively, the net effect on pathogen concentrations in surface waters and consequently also on infection risks seems limited.
- These model predictions are catchment specific but are assumed to be representative for the Netherlands or similar lowland catchments with little slope and a shallow groundwater level.
- In general, this study points out that the assumption of an increase in pathogen concentration in surface waters because of climate change is too simple and interplay of the different processes needs careful evaluation.

## Acknowledgements

The information Claudia Brauer provided on WALRUS is gratefully acknowledged. Thanks to Lester Sinton for providing the data on die-off experiments with *Campylobacter* in faeces. Valuable help was provided by Jannette Bessembinder and Rudmer Jilderda (KNMI) for the transformation of climate data according to the KNMI scenarios. Thanks to Eri van Heijnsbergen and René Wijn-gaarden for critical reading of the manuscript. The work was funded through the RIVM strategic Research Project Climate Cascades (S/607021).

## Appendix A. Supplementary data

Supplementary data related to this article can be found at <http://dx.doi.org/10.1016/j.watres.2016.03.005>.

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