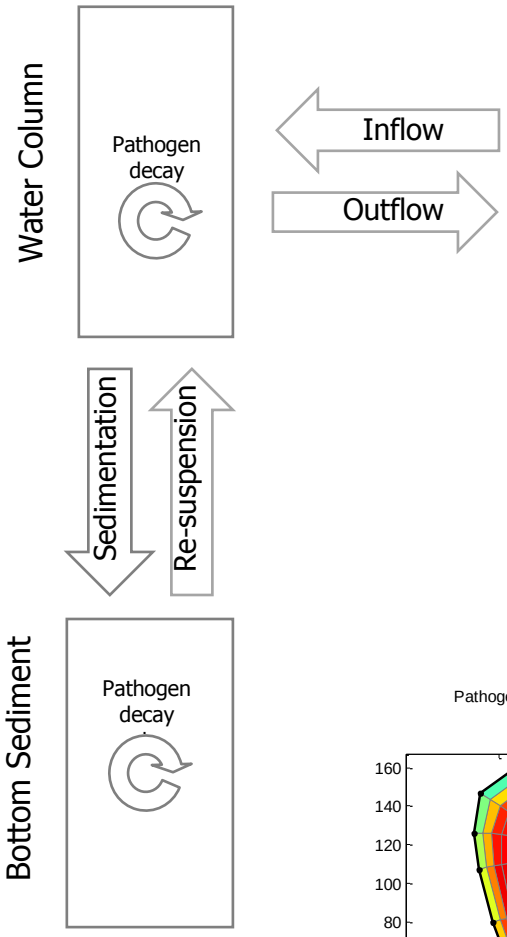
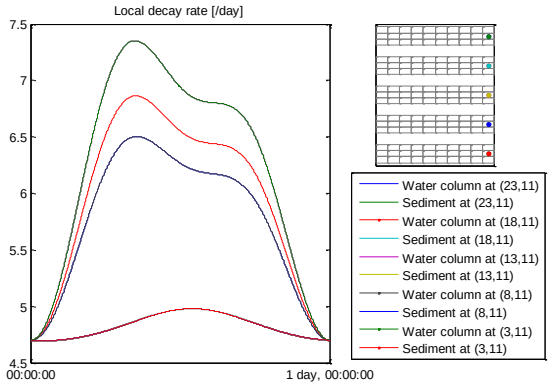
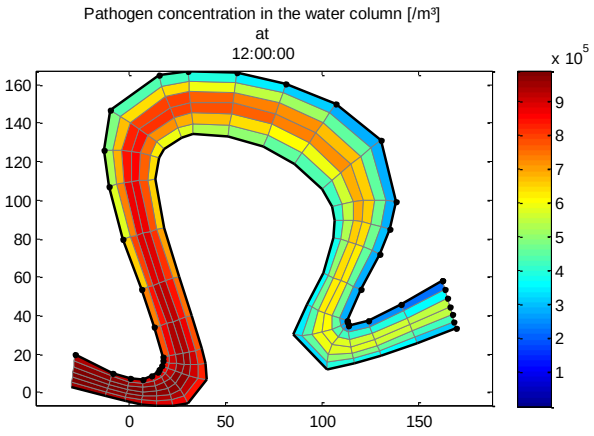
Master Thesis:

Reducing the survival of pathogens in urban water by making optimal use of natural processes







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Contents

Abstract

Pathogens are present in our urban water system, and may pose a health risk to swimmers and playing children. Although the knowledge of pathogens has increased over recent years, little is known about their behaviour in the urban water system. The goal of this thesis is to investigate this.

First, the behaviour of pathogens under the influence of environmental variables such as sunlight intensity and temperature will be investigated. Then, this will be incorporated into a hydrological model to simulate the movement of pathogens through a water system.

The pathogen population is assumed to decay according to the linear model shown in equation (1).

$$c(t) = c_0\, e^{-kt} \tag{1}$$

The decay rate, k, is influenced by the species of pathogen and the conditions in the environment. To estimate the value of the decay rate under influence of these factors, equation (7) is proposed.

$$k_{overall} = (c_1 + c_2 \cdot S) \cdot {c_3}^T + \left(c_4 + c_5 \cdot {c_6}^{|A-6.5|}\right) \cdot I_{avg} \cdot O + c_7 \qquad (7)$$
$$with \quad I_{avg} = \frac{I_0}{\tau h}(1 - e^{-\tau h})$$

The factors that have been shown to be relevant are temperature, seawater ratio, acidity, light intensity and oxygen content.

The shape of the formula according to which these factors influence the overall decay rate has been formulated by gathering data and conclusions from existing literature. Values of the different pathogen constants (c_1 through c_7) have been estimated for faecal coliforms by least square curve fitting to data from literature. The values shown in Table 6 were found.

c_1	c_2	c_3	c_4	c_5	c_6	c_7
0.21	0.16	1.07	-7.93E-04	1.59E-03	1.8	4.3707

Table 6: Pathogen constants for faecal coliforms

A sensitivity analysis was done on both the pathogen constants and the environmental variables. The conclusion was that c_3, the pathogen constant controlling the influence of the temperature was most sensitive. This is also the constant that has the best support in literature. For the environmental variables, the oxygen content and light intensity were shown to influence the decay rate the most.

A 2D finite element model was set up by using hydrological calculated using Delft3D and post processing it with a Matlab script. The communication file, produced by the Delft3D suite, is used to transfer data to Matlab. The script adds three types of the same pathogen to the hydrological model, pathogens that are free floating in the water column, pathogens that are adsorbed to particles in the water column, and pathogens that are adsorbed to particles that have settled to the sediment. These types of pathogen are moved through the grid depending on the flow calculated by Delft3D, and adsorbed pathogens can switch between the water column and sediment type according to formulas for sedimentation and resuspension.

Three water systems have been examined to test both the model itself and the behaviour of pathogens in such a system. In the first, the influence of channel depth was examined, in the second, the shape of an inflow of pathogens, and last, the influence of natural banks.

The removal of pathogens from the water column was found to be caused mostly by the sedimentation and not so much by pathogen decay. The effect of this is that in an equilibrium state, the concentration of pathogens in the sediment is much higher than that in the water column. This is a claim that is supported by several studies.

As sedimentation is the largest factor when considering the removal of pathogens from the water column, any measures that decrease the flow velocity decrease pathogen concentrations. However, pathogens are still present in the system, and care should be taken that the water column is not reinfected through resuspension.

Validation of the model has not been possible, though is definitely desirable.

The main weakness of the model is the presence of three different timescales for flow, sedimentation and pathogen decay. Calculation could perhaps be improved by introducing artificial factors, boosting all three to have a similar timescale.

Set up and approach

Goal of the thesis

In recent years, the methods to detect bacteria and pathogens in water have increased drastically, and as a result many research has been done on the microbiologic quality of the surface water. Not very surprisingly, the conclusion was that the urban water system houses many organisms harmful to humans.

One study, performed in 2003-2004 by RIVM in the canals of Amsterdam revealed the water quality was poor according to the European Bathing Water Directive (2006/7/EC) and decreased quality after rainfall: probably due to sewer overflows and because dog faeces and other filth was washed from the streets into the canals.

At the same time there is the desire to use our surface water for recreation purposes, also in urban areas. During fine weather we enjoy swimming at local beaches, and when it's raining, we would like to temporarily store water at water squares.

The surface water could also be used to flush toilets, irrigate gardens, etc.

Knowing the quality of our urban water is not always up to standards, and that we still want our children to play in those same waters, we need to improve the surface water quality.

A lot can be gained by reducing the sources of pathogens and by disconnecting inflows, but it will be virtually impossible to prevent our water from being contaminated. Therefore it is equally important to reduce the survival chances of dangerous pathogens as much as possible.

But what are the vulnerabilities of those viruses and pathogenic bacteria? And how can we create these circumstances in our urban water?

That is the main question of this master thesis.

Main Question

- How can we arrange our urban water system to decrease the survivability for pathogens as much as possible?

To be able to answer this main question, some subquestions should first be answered. These have been divided over two phases: the first phase is mainly a literature study and will focus on the microbiological issues, the second is the incorporation into a hydrological model.

Subquestions

Phase I: Microbiological model

1) How do microbiologists estimate pathogen decay rates?
2) What are the survival rates of pathogens at living conditions that exist in the urban water system?
   a) Which of these living conditions are relevant to this thesis?
      For example: exposure to sunlight, water temperature, oxygen concentration etc.
   b) How does each pathogen species react to each living condition?
   c) Which environmental factors have the biggest influence?
   d) What happens to the survival rates when multiple conditions are examined at the same time?
      Is the die off in sunlight and a certain temperature equal to the sum of both factors separately, or does the presence of both increase the decay rate?

Phase II: Hydrological model

3) How can the microbiological model be integrated into a hydrological model?
4) How can the model be used to calculate pathogen concentrations in water systems?

Approach

Phase I: Microbiological model

This phase addresses the pathogens and their survival rates. Much is known on this topic, mostly due to research in related fields of expertise, such as drinking water and sanitation. Therefore, most of these subquestions can be answered by doing a literature study.

It is to be expected that the survival rates can't be found for each of the pathogens and for each of the living conditions. If this is the case, and these knowledge gaps are thought to influence the validity of the water quality model used in Phase II, additional research may be required.

Phase II: Hydrological model

Here, the knowledge obtained at the previous subquestions should be applied to a water system. Which characteristics will stimulate a high die off rate?

Decreasing the influx of pathogens is not considered to be in the scope of this thesis.

First, a model must be created that combines the results of Phase I with a currently existing hydrological model, for instance Delft3D. Then, this model can be used to calculate the pathogen concentrations under many conditions, and the effects of various interventions can be studied.

Phase I: Microbiological model

The decay formula

It is commonly presumed that population growth and decay occur exponentially, so that the size of a decreasing population is described by equation (1).

$$c(t) = c_0\, e^{-kt} \quad (1)$$

with c the population size, c_0 the population at t=0, k the decay rate [/ [time]], and t the time

In this model, the variable k, refered to as the decay rate, governs the rate at which the population is decreasing.

The model described in equation (1) is not entirely correct as often a so-called shoulder can be observed before the pathogen population starts decaying with a constant decay rate. However, it is commonly simplified to a single constant decay rate, and thus equation (1) will be used.

Thomann & Mueller, 1987 were the first to propose a formula splitting the decay rate based on environmental factors: the decay in the dark (influenced by temperature), the additional decay in sunlight, and the decrease of pathogens attributed to the sedimentation and filtration of particles.

$$k_{overall} = k_{dark} + k_{light} + k_{sedimentation} \quad (2)$$

The decay rate partitions are then filled in with equations based on environmental factors.

For the dark decay rate, the Arrhenius-van 't Hoff equation is usually applied, assuming the influence of temperature.

$$k_{dark} = k_{T20}\theta^{T-20} \quad (3)$$

with $k_{t,20}$ the decay rate in the dark at 20°C, θ a constant, and T the temperature in °C

(equivalent to $k_{dark} = (k_{T20} \cdot \theta^{-20}) \cdot \theta^{T} = c\theta^{T}$)

The effect of light is usually assumed to be linear with the sunlight intensity the pathogens receive. This can be expressed as equation (4).

$$k_{light} = \varphi I_{avg} \quad (4)$$

with φ a constant and I_{avg} the sunlight intensity, averaged over the water column

The depth averaged sunlight intensity can be derived from the solar intensity at the water surface according to equation (5).

$$I_{avg} = \frac{I_0}{\tau h}(1 - e^{-\tau h}) \quad (5)$$

With I_0 the sunlight intensity at the water surface, τ the light attenuation factor or turbidity and h the water depth

I will not go into detail about the equation for the decay partition due to sedimentation and filtration, because although it makes sense to a microbiologist to include these processes in the decay rate, to a hydrologist it does not. Sedimentation and filtration does not neutralise the pathogen, it merely changes the location of it. Furthermore, the sediments have been reported to function as a reservoir for pathogens, as within the sediments they are protected from predation and photoinactivation. (Crabill et al., 1999) It makes more sense to take into account the effects of sedimentation and filtration in the hydrological model.

As there are very few good measurements in sediment, I will use the same equation for pathogen decay, but assume that the influence of light is negligible. I will construct the hydrological model in such a way that this can be changed easily should better insights arise.

Substituting equations (3) through (5) into equation (2) and leaving out the effects of sedimentation gives us equation (6).

$$k_{overall} = k_{T20}\theta^{T-20} + \varphi\frac{I_0}{\tau h}(1 - e^{-\tau h}) \quad (6)$$

However, the natural world is a complex one, and not only are there more factors influencing the overall decay rate of pathogens than just temperature and sunlight intensity, their effects are not always independent of one another.

Approach

In order to study the relation of the pathogen decay rate and the pathogen species and environmental variables, the aim is to formulate an equation, based on equation (6) that is as complete as possible within the scope of a natural water system.

In theory, the shape of this formula should be applicable to any type of pathogen, with constants (θ, φ and τ in equation (6)) controlling the amount of influence a variable has, depending on the pathogen species. I will attempt to find a set of suitable constants for faecal coliforms, as most data is available for this particular range of bacteria.

To determine the shape of this formula, conclusions and data from the literature will be used.
To determine the constants within this formula, a least squares curve fit will be performed, first on formulas depending on single variables, where data was available in which only this variable was varied, while the other environmental variables were kept constant.
Then, to find the constants that could not be calculated with this approach, the data for which all environmental variables are known will be used to fit the complete formula.

The data used is selected from the literature I could find. In some cases, data could not be used because the overall decay rate could not be calculated from the values provided in the paper, while the shape of the values and the conclusions drawn in the paper could still be used to support the shape of the formula.

Many papers use a different indicator for the decay rate, for example the T90, the time it takes for the population to reduce to 10% of the starting value. The sunlight intensity is often used cumulatively as the sum of energy over a time period. In this case the sunlight intensity was calculated by dividing with the approximate duration of the experiment.

The formula I propose is shown in equation (7). It is based on equation (6), with several variables added, and some constants rearranged to have as few as possible. The shape of the influences of the separate variables and the values of the constants for faecal coliforms will be discussed per variable.

$$k_{overall} = (c_1 + c_2 \cdot S) \cdot {c_3}^T + \left(c_4 + c_5 \cdot {c_6}^{|A-6.5|}\right) \cdot I_{avg} \cdot O + c_7 \qquad (7)$$
$$with \quad I_{avg} = \frac{I_0}{\tau h}(1 - e^{-\tau h})$$

with S the ratio of seawater (0 = freshwater, 1 = seawater), T the temperature in °C, A the acidity on the pH scale, I_0 the sunlight intensity at the water surface in W/m^2, τ the light attenuation factor in /m, h the water depth in m and O the dissolved oxygen concentration in mg/l
c_1 through c_5 are constants depending on the pathogen species

If several measurements are provided for variables which are not included in equation (7), the data with variables which most closely resembles a natural water system will be used.

All data is listed in Appendix V, including its source and notes on the validity.

Environmental variables

Temperature

As temperature is so easy to measure and control, many studies have investigated the influence of temperature or at least noted down the average value during their research.

Their results however, vary. Some report that temperature is a significant factor, others that there does not seem to be an effect.

However, as many studies have not paid any attention to the water's oxygen content, (less oxygen will dissolve in warm water), while allowing light, which influences the decay rate (see next chapter), their results can be explained.

Opinions on the processes involved with temperature's effect on pathogen inactivation differ. It is likely to be a combination of several factors influenced by temperature, such as the activity of predatory microorganisms (bacteriophages) or the bloom of algae, which in turn may affect turbidity, dissolved oxygen and acidity.

Mancini, 1978 has done a great job at gathering data from various literature sources and fitting the data to equation (3). He has also found that k_{T20}, the dark decay rate at 20°C, differs between fresh- and seawater. His findings are shown in Figure 1.

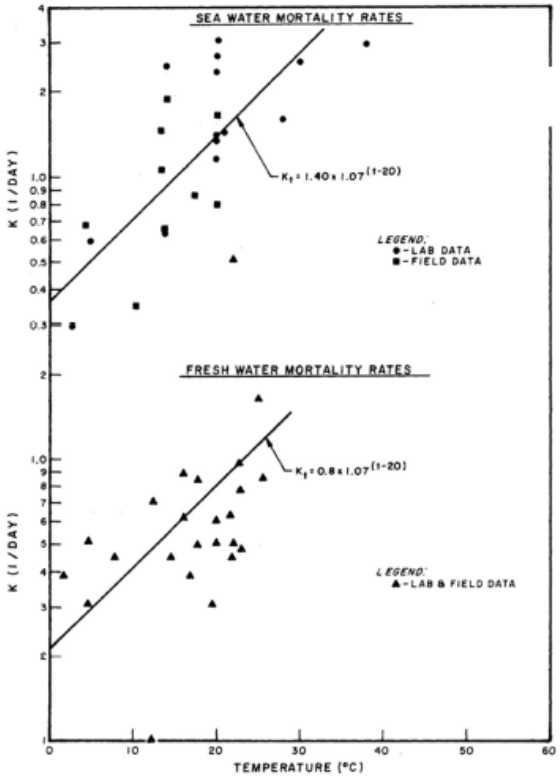

Figure 1: Coliform mortality as a function of temperature, Mancini, 1978

In some literature the authors have measured decay rates while varying temperature and keeping other variables constant. By applying this to equation (7), equation (8) is found.

$$k_{overall} = c_1 \cdot {c_2}^T + c_3 \tag{8}$$

The only relevant constant in this case is c_2, as the others are influenced by other variables than temperature, and thus vary between datasets. However, c_1 and c_3 will correlate greatly for values of c_2 that are close to 1, which we expect from Manicini's results.

Therefore, to obtain a more stable solution, the results will be fitted to equation (9) instead. As a result of the simplification, assuming c_3 is positive, we expect the value for c_2 to be slightly lower.

$$k_{overall} = c_1 \cdot {c_2}^T \tag{9}$$

I have fitted each data set to using the least square approach and calculating the values for the constants, using the function 'lsqcurvefit' in Matlab.

The curve fits and their results are shown in Figure 2 and Table 1.

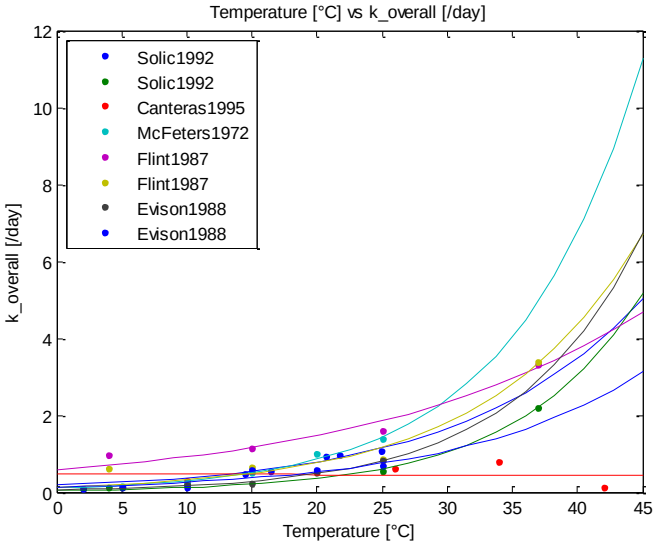

Figure 2: Lsqcurvefit on data with varying temperature

	c_1	c_2	n	$\Sigma(f(c,x_i)-y_i)^2)/n$
Average		**1.078**		
Šolić et al., 1992	0.180	1.077	5	4.69E-03
Šolić et al., 1992	0.042	1.113	4	3.59E-03
Canteras, 1995	0.459	1.000	4	6.91E-02
McFeters et al., 1972	0.110	1.108	5	5.39E-03
Flint, 1987	0.580	1.048	4	3.28E-02
Flint, 1987	0.132	1.091	4	7.98E-02
Evison, 1988	0.058	1.111	6	6.06E-04
Evison, 1988	0.126	1.074	6	1.13E-02

Table 1: Calculated values of constants for equation (8). n indicates the number of points in the dataset and $\Sigma(f(c,x_i)-y_i)^2)/n$ is the squared error averaged over the data points

As can be seen the values for c_2 are all reasonably close to 1.07, the value which was calculated by Mancini, 1978.

It should be noted that because of the power, the data points with high temperatures have a relatively high influence on the fit of the curve, while temperatures over 30°C are not likely to occur in a natural water system. To investigate if this is a problem, the fitting was repeated with only data points below a temperature of 30°C. The curve fits and their results are shown in Table 2 and Figure 3.

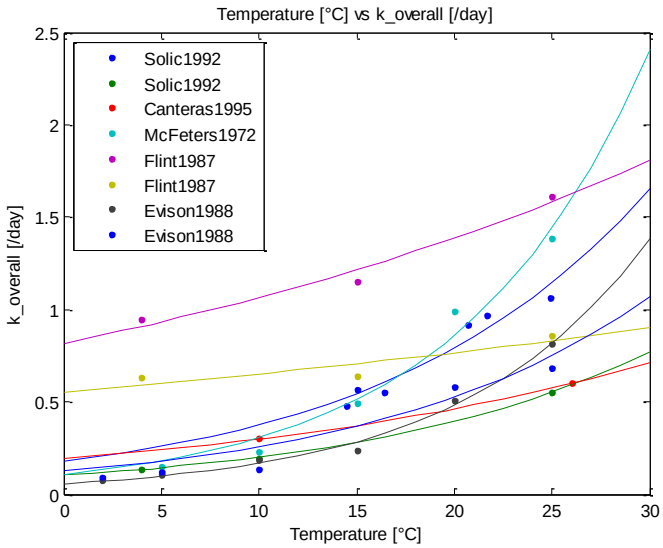

Figure 3: Lsqcurvefit on data with varying temperature (<30°C)

	c_1	c_2	n	$\Sigma(f(c,x_i)-y_i)^2)/n$
Average		**1.066**		
Šolić et al., 1992	0.180	1.077	5	4.69E-03
Šolić et al., 1992	0.103	1.070	3	9.77E-06
Canteras, 1995	0.195	1.044	2	7.24E-15
McFeters et al., 1972	0.110	1.108	5	5.39E-03
Flint, 1987	0.817	1.027	3	2.23E-03
Flint, 1987	0.555	1.016	3	2.26E-03
Evison, 1988	0.058	1.111	6	6.06E-04
Evison, 1988	0.126	1.074	6	1.13E-02

Table 2: Calculated values of constants for equation (8) for data with temperature < 30°C

Flint, 1987's data sets show a lower gradient than the other data sets do. It is possible this is caused by not having kept all other variables constant: Flint has only recorded values for temperature. For example, if the oxygen content was less at higher water temperatures (which would be plausible as less oxygen will dissolve in warmer water) this would have lowered the decay rate for higher temepratures.

The values for c_2 are slightly lower than before, but still close to Mancini's conclusion. I will assume a value of 1.07 for the corresponding constant in equation (7), c_3.

Temperature & seawater ratio

As said, Mancini, 1978 has also found that the values for seawater differed from those in freshwater. As can be seen in Figure 1 he has found that the growth rate of his function (c_2 or θ) stays the same while the multiplication constant (k_{T20} or c_1) differs.

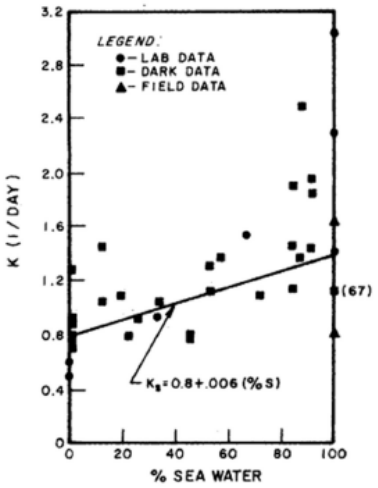

Figure 4: Coliform mortality at 20°C as a function of seawater percentage, Mancini, 1978

He has also investigated how varying the ratio of seawater to freshwater affects the decay rate, as can be seen in Figure 4. He has used the ratio of seawater to freshwater rather than the salt content, as previous research (Savage & Hanes, 1971) has shown that there appear to be more factors in seawater that accelerate pathogen decay then the salt concentration, for example the population of bacteriophages.

His conclusion is that the overall decay rate for coliforms can be estimated using equation (10).

$$k_{overall} = (0.8 + 0.6\,S) \cdot 1.07^{T-20} + k_{light} \tag{10}$$

Mancini's research has used a lot of data points from previous studies, and his attempts at a useable formula for coliform decay rate are very similar to mine. I have not been able to find enough data on seawater percentage to compare his results to my own, but as his results with temperature match mine, I see no reason to not gratefully accept his conclusions on the effects of seawater.

To fit equation (7), equation (10) can be rewritten to equation (11) using that θ=1.07.

$$k_{overall} = (0.8 \cdot 1.07^{-20} + 0.6 \cdot 1.07^{-20} \cdot\ S) \cdot 1.07^{T} + k_{light} = (0.21 + 0.16 \cdot S) \cdot 1.07^{T} + k_{light} \tag{11}$$

Conclusion

There is a reasonable consensus in the literature that the dark decay rate can be estimated according to equation (12).

$$k_{dark} = (c_1 + c_2 \cdot S) \cdot {c_3}^{T} \tag{12}$$

with, for faecal coliforms:

$$[c_1 \quad c_2 \quad c_3] = [0.21 \quad 0.16 \quad 1.07]$$

Sunlight

Sunlight is generally accepted as being the environmental factor that affects the decay of pathogens most strongly. As such, quite some research has been done on the process, and many treatment processes count on this free way of deactivating pathogens. (Wastewater stabilisation ponds, SODIS [leaving plastic bottles with water out in the sun])

Sunlight has been reported to directly damage any microorganisms when photons are absorbed by DNA (Jaegger, 1985), however, this process is insignificant compared to the process in which light puts so-called photosentisizers into an excited state. (Davies-Colley et al., 2000) These excited photosentisizers then either react directly with molecules belonging to the pathogen, or with oxygen, forming what is referred to as free radicals. These then react with components of the cell, including DNA but also proteins and building blocks of the cell wall, deactivating the micro-organism. (Bose & Chatterjee, 1995).

I've found several studies which have done experiments with varying the sunlight. Their results are shown in Figure 5. As can be seen the values vary and a clear relation can not immediately be deduced, indicating that there are more variables affecting the results.

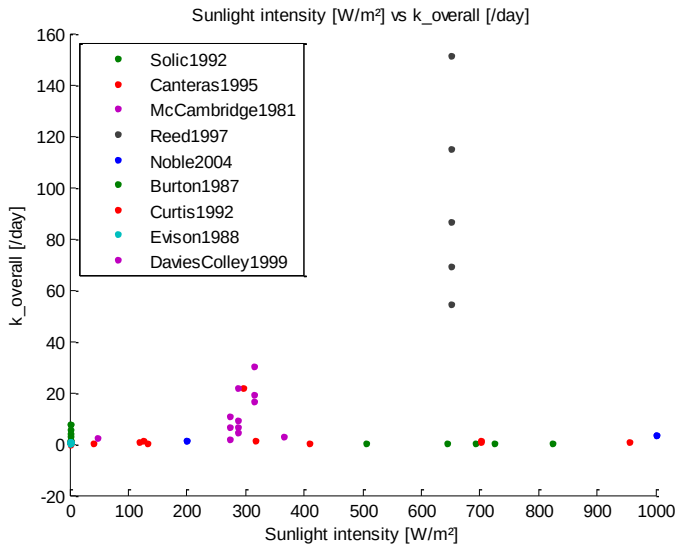

Figure 5: Results for the overall decay rate of faecal coliforms for various sunlight intensities

Many studies have worked with cumulative insolation, with the consequence that they have not really investigated if the effects of more light over a shorter period is equal to the effect of less light over a longer period. For the purpose of this thesis I shall assume this is the case.

Sunlight & oxygen

As oxygen is such an important part of the main deactivation process, it should be no surprise that researches have found that solar deactivation is not only linear with the sunlight intensity, but also with oxygen content. (Downes, 1878 (!); Reed, 1996; Curtis, 1992)

Unfortunately however, there are very few literature studies that have taken the effort of measuring the oxygen content.

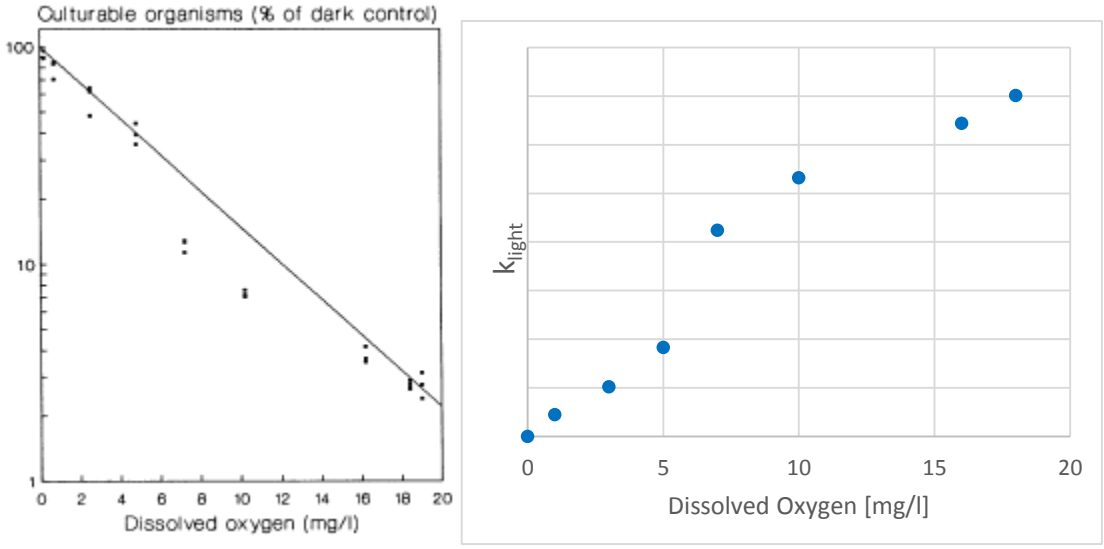

Figure 6: The effect of oxygen concentration on the impact of light measured by Curtis, 1992
Left: original - right: converted to decay rate

Curtis, 1992 has shown that the decay rate of pathogens in a reactor under the influence of sunlight is almost linear with dissolved oxygen, (Figure 6) but has shown his data in a way so that the actual value of the decay rate cannot be calculated.

When the data gathered in Figure 5 is reduced to data which has both the sunlight intensity and the oxygen content available, and it is plotted in a 3D graph, we are able to see a pattern, as can be seen in Figure 7. It seems the decay rate increases both with higher oxygen content as with higher sunlight intensity.

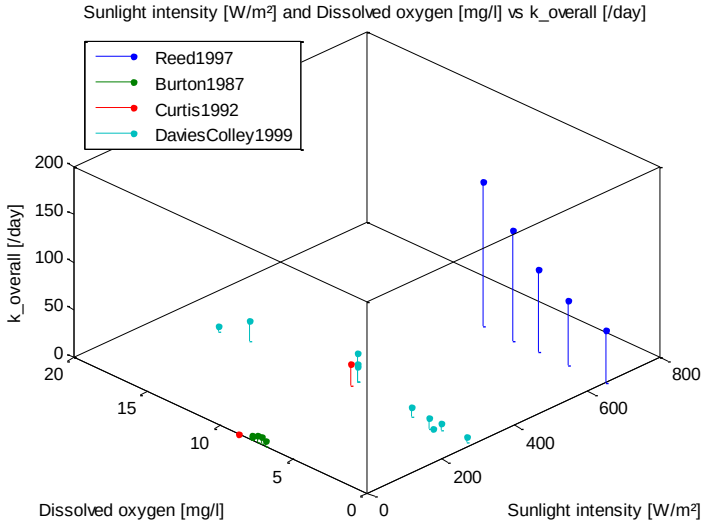

Figure 7: Results for the overall decay rate of faecal coliforms for various sunlight intensities and oxygen concentrations

This pattern is more clear when only decay rates from studies that have varied sunlight intensity or oxygen content (with both documented) while keeping other variables constant are plotted versus the product of dissolved oxygen and sunlight intensity, as in Figure 8. In this graph, the data from Curtis has a varying sunlight intensity (one measurement in the dark, one in sunlight) and constant oxygen content, while the data from Reed and Davies-Colley was obtained by varying dissolved oxygen under a constant light intensity.

The data from Davies-Colley seems to fit a simple linear equation. Reed's data seems to fit a curve, and Curtis' data (Figure 6) appears to fit a curved line with a small perturbation in the middle region.

As the data available is very limited, it seems the best solution is to assume a linear equation such as equation (13).

$$k_{overall} = c_1 \cdot I_{avg} \cdot O + c_2 \qquad (13)$$

Using Matlab I have once again used lsqcurvefit to fit equation (13) to the data. The results are shown in Table 3. The calculated values for the constants vary considerably between the studies, indicating that yet another variable could be affecting them.

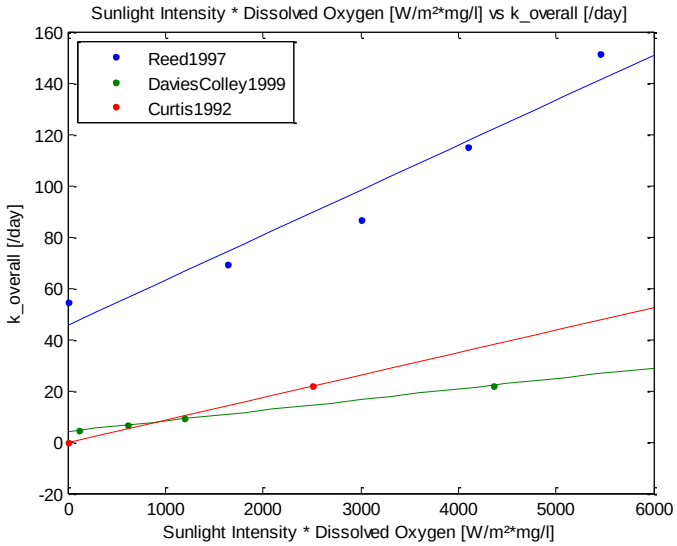

Figure 8: Lsqcurvefit on data with varying sunlight intensity * dissolved oxygen

	c_1	c_2	n	$\Sigma(f(c,x_i)-y_i)^2)/n$
Reed, 1997	0.018	45.309	5	7.06E+01
Davies-Colley, 1999	0.004	4.085	4	1.41E-02
Curtis, 1992	0.009	-0.259	2	0.00E+00

Table 3: Calculated values of constants for equation (13)

Sunlight & temperature

Some studies report a synergy between the effects of sunlight and temperature, however these are all at very high temperatures (>40-50°C). (E.g. McGuigan et al.,1998) As such this effect would seem to be irrelevant in natural waters.

Sunlight wavelength & humic substances

It has been shown that pathogen deactivation is most efficient at wavelengths in the UVA and UVB region of the spectrum (290-350nm) (Acra et al, 1984). It is also reported that humic substances may influence the process, making the pathogens venerable to longer wavelengths of light as well (Curtis, 1992), while others point out that humic substances also cause a coloration of the water, decreasing the intrusion of these wavelengths into the water column, and thus again decreasing the effect.

As I am mainly interested in natural waters, I will assume that the light affecting the pathogen decay is the full spectrum of sunlight, uninfluenced by humic compounds.

Sunlight & acidity

Davies-Colley, 1999 and Curtis, 1992 report that Escherichia coli shows an increased decay rate for high and low acidity, but only in the presence of light, while other pathogens (e.g. Enterococci) do not.

The data available is reasonably good, so I was able to include the pH in equation (7).

All available data which has varied the pH while keeping all other environmental variables constant, shows the lowest decay rate around a pH of 6.5. I propose equation (14) for the relation between acidity and the decay rate.

$$k_{overall} = c_1 \cdot {c_2}^{|A-6.5|} + c_3 \qquad (14)$$

The result for the lsqcurvefit can be seen in Figure 9 and Table 4. The value for c_2 is the interesting one, as according to equation (7) it only depends on the acidity.

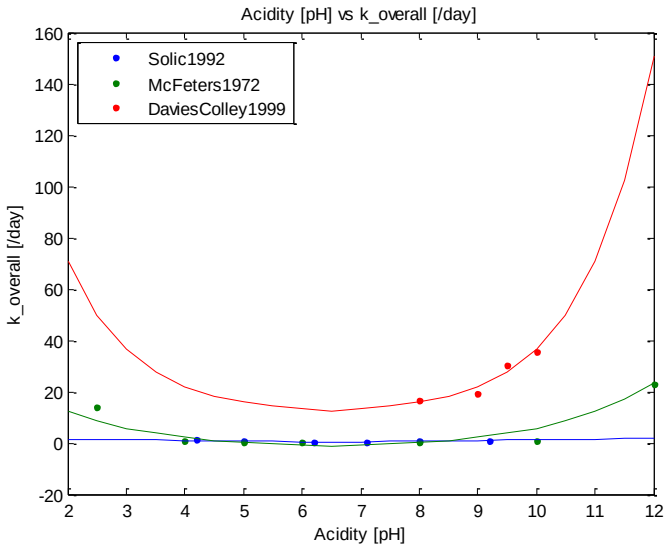

Figure 9: Lsqcurvefit on data with varying acidity

	c_1	c_2	c_3	n	$\Sigma(f(c,x_i)-y_i)^2)/n$
Average		**1.726**			
Šolić et al., 1992	9.849	1.026	-0.521	6	1.60E-02
McFeters et al., 1972	1.013	1.803	-2.253	7	8.28E+00
Davies-Colley, 1999	1.272	2.349	11.254	4	3.81E+00

Table 4: Calculated values of constants for equation (14)

As the fitted curve to data from McFeters, 1972 provides a decent fit in the most common regions (pH around 7) while still showing the expected behaviour for more extreme values, I will assume c_2 to be 1.8.

Conclusion

There is a reasonable consensus in the literature that the decay rate contributed to light can be estimated according to equation (15).

$$k_{light} = \left(c_4 + c_5 \cdot c_6^{|A-6.5|}\right) \cdot I_{avg} \cdot O \quad (15)$$

with, for faecal coliforms:

$[c_4 \quad c_5 \quad c_6] = [? \quad ? \quad 1.8]$

Predation

Presence of bacteria and algae stimulates the deactivation of viruses both in seawater (Kapuscinski et al., 1980) and in fresh water (Ward et al., 1986).

The research involving predation of pathogenic microorganisms is few and far between and populations are hardly ever recorded in studies, I am unable to include it properly in equation (7). However, as the population of predatory microorganisms is influenced by temperature and the seawater ratio, their effect should be included in the final result, even though the formula does not contain a direct reference to the variable.

The pathogen constants

In the previous chapter I have already determined 4 of the 6 constants in the decay rate formula, equation (7). The remaining variables will be estimated by performing yet another lsqcurvefit, but this time to data from literature which have a value for all the different environmental variables. It should be noted that this dataset is from freshwater sources only, so that c_2, which is the constant that increases the dark decay depending on the seawater ratio, is not being tested. The data from Burton was measured in sediments.

The data used is listed in Table 5, the results are shown in Figure 10 and Table 6.

	k_ overall [/day]	Temperature [°C]	Acidity [pH]	Seawater ratio [-]	Sunlight intensity [W/m²]	Dissolved oxygen [mg/l]
Burton, 1987	7.73	19.60	7.45	0	0.00	7.20
Burton, 1987	5.66	18.50	7.30	0	0.00	6.95
Burton, 1987	4.01	18.80	7.65	0	0.00	7.85
Burton, 1987	7.66	19.30	7.40	0	0.00	7.50
Burton, 1987	3.02	18.50	8.05	0	0.00	7.85
Davies-Colley, 1999	22.10	20.00	7.50	0	287.04	15.22
Davies-Colley, 1999	9.21	20.00	7.50	0	287.04	4.19
Davies-Colley, 1999	6.45	20.00	7.50	0	287.04	2.14
Davies-Colley, 1999	4.61	20.00	7.50	0	287.04	0.41
Davies-Colley, 1999	35.79	20.00	10.00	0	314.81	8.55
Davies-Colley, 1999	30.39	20.00	9.50	0	314.81	8.55
Davies-Colley, 1999	19.34	20.00	9.00	0	314.81	8.55
Davies-Colley, 1999	16.58	20.00	8.00	0	314.81	8.55
Davies-Colley, 1999	11.05	20.00	9.50	0	273.15	2.60
Davies-Colley, 1999	6.45	20.00	7.50	0	273.15	17.00
Davies-Colley, 1999	1.84	20.00	7.50	0	273.15	2.40

Table 5: Data used to calculate the pathogen constants for Faecal Coliforms

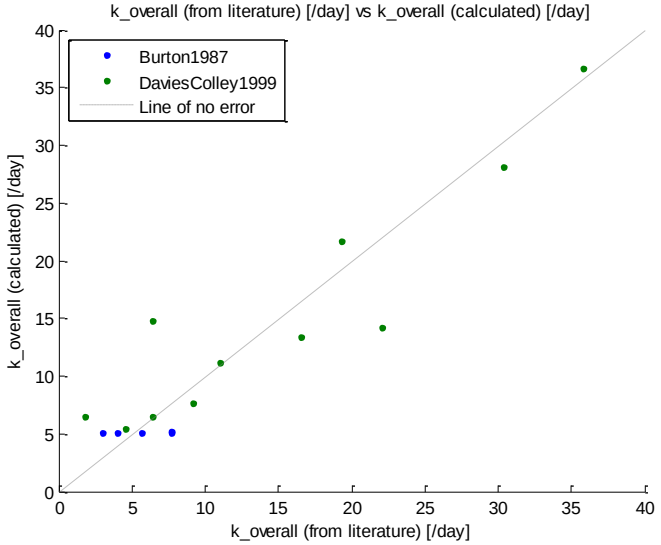

Figure 10: Comparison of decay rates calculated with the fitted constant and the values reported by literature

c_1	c_2	c_3	c_4	c_5	c_6	c_7	n	$\Sigma(f(c,x_i)-y_i)^2)/n$
0.21	**0.16**	**1.07**	-7.93E-04	1.59E-03	**1.8**	4.3707	16	12.3396

Table 6: Pathogen constants as calculated with lsqcurvefit. Values in bold were determined in the previous chapter

As can be seen, the values measured by Burton differ more than the values calculated by applying the model formula with a light intensity of 0 [W/m^2]. As such I expect the sediment reservoir effect in the final model to be stronger than it actually is. However, Burton, 1987 is just one study, and there simply isn't enough data to propose an entirely different formula.

Substituting the calculated values for the pathogen constants into equation (7) gives our end goal, the decay rate formula for faecal coliforms (equation (16)).

$$k_{overall} = (0.21 + 0.16 \cdot S) \cdot 1.07^{T} + \left(-0.0008 + 0.0016 \cdot 1.8^{|A-6.5|}\right) \cdot I_{avg} \cdot O + 4.4 \quad (16)$$

Sensitivity analysis

In order to analyse a complex multivariable function it is a good idea to do a sensitivity analysis. In such an analysis it is customary to take the formula with average values, and then, one at a time, vary each of the variables to see how much the outcome changes.

In this case there are two categories of variables in the decay rate formula: the environmental variables and the pathogen constants. It is useful to do a sensitivity analysis for both of these.

As the average starting values, for the pathogen constants the values calculated in the previous chapter were used, and for the environmental variables, I've chosen values that are sensible and near the average of the values that have been gathered in the literature studies. The values are listed in Table 7.

c_1	c_2	c_3	c_4	c_5	c_6	c_7
0.21	0.16	1.07	-8E-04	1.6E-03	1.8	4.4

Seawater ratio	**Temperature**	**Acidity**	**Sunlight Intensity**	**Dissolved Oxygen**	$k_{overall}$
[-]	[°C]	[pH]	[W/m^2]	[mg/l]	[/day]
0.5	17.5	7.5	250	7	8.99

Table 7: Average values used for the sensitivity analysis

The pathogen constants

After performing the sensitivity analysis for the pathogen constants, the values shown in Table 8 and Figure 11 have been found.

	c_1	c_2	c_3	c_4	c_5	c_6	c_7
+10%	0.8%	0.3%	45.3%	-1.6%	5.6%	5.6%	4.9%
-10%	-0.8%	-0.3%	-8.9%	1.6%	-5.6%	-5.6%	-4.9%

Table 8: Sensitivity analysis results for the pathogen constants

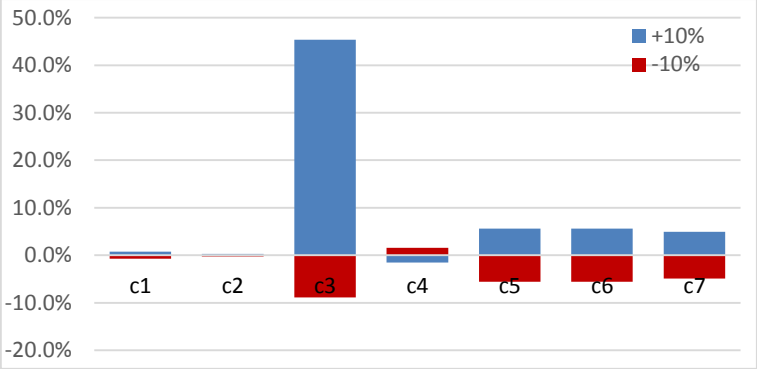

Figure 11: Sensitivity analysis results for the pathogen constants

As can be seen, the overall decay rate formula is most sensitive to inaccuracies in the value for c_3, which controls the temperature influence. Luckily, the data this constant was derived from is the best documented, and also confirmed by the work of Mancini, 1978. The constants controlling the seawater influence are the least sensitive.

The environmental variables

After performing the sensitivity analysis for the environmental variables, the values shown in Table 9 have been found.

	Seawater ratio	Temperature	Acidity	Sunlight Intensity	Dissolved Oxygen
+10%	0.3%	1.3%	31.1%	4.1%	4.1%
-10%	-0.3%	-1.2%	-20.0%	-4.1%	-4.1%

Table 9: Sensitivity analysis results for the environmental variables

The overall decay rate appears to by most sensitive to the acidity, by far, however it should be noted that although an increase of 10% of the pH is mathematically correct, the pH usually isn't expected the vary quite as much. The pH is a measure for the acidity that is actually the negative 10-based logarithm of the hydrogen ion concentration in mol/l. If instead of varying the pH, the value of $[H^+]$ is varied by 10%, the results, as shown in Table 10 and Figure 12, look very different.

	Seawater ratio	Temperature	Acidity	Sunlight Intensity	Dissolved Oxygen
+10%	0.3%	1.3%	-1.3%	4.1%	4.1%
-10%	-0.3%	-1.2%	1.5%	-4.1%	-4.1%

Table 10: Sensitivity analysis results for the environmental variables, with corrected pH variation

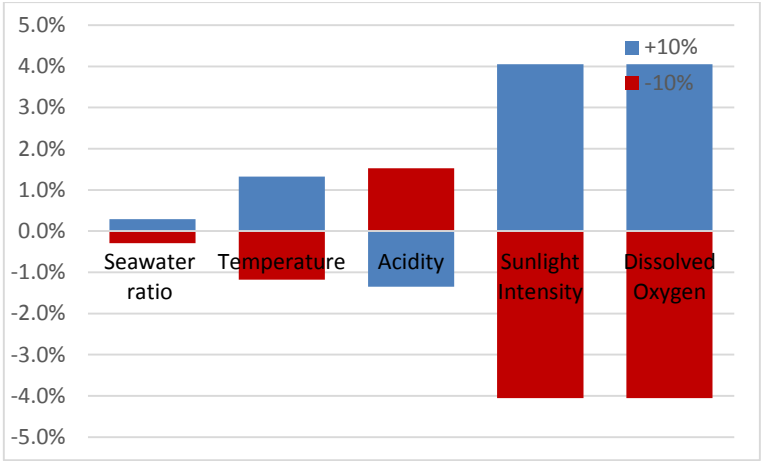

Figure 12: Sensitivity analysis results for the environmental variables

Note that the sign of the acidity influence has flipped. This is because a 10% increase in $[H^+]$ is an increase in acidity, and thus a decrease in pH. The influence of light is now the largest factor, as is the consensus in literature. As such, a water manager looking to increase fecal coliform decay rates is recommended to start by improving light, decreasing turbidity, and increasing aeration.

Phase II: Hydrological model

Model setup

To understand how a population of pathogens behaves as part of a water system, it is important to know how the pathogens move to areas which might have a different set of environmental conditions, thus influencing the decay rate. For this reason the decay formula and the formula for the decay rate need to be integrated into a hydrological model.

The obvious choice for the type of hydrological model is the 'finite element'-model, which is composed of a finite amount of compartments or cells, each with values for e.g. the water depth, pathogen concentration and temperature. These cells can interact with each other, as water can flow from one cell into the next.

Although measurements for pathogen concentrations mostly focus on the water column, both bacteria (Schillinger et al., 1985) and viruses (Rao et al, 1987) are able to adsorb to suspended solids in high quantities. Different micro-organisms prefer a different range of particle sizes and materials. (Characklis et al., 2005) The particles may settle as they normally would, taking adsorbed pathogens with them.

The pathogens seem to be concentrated at the water-sediment interface.

Resuspension of the adsorbed pathogens is possible, in particular when the sediment is perturbed. This can be caused by storm events and recreational activity (Crabill et al., 1999), dredging operations etc.

Vegetation has a stabilizing effect on sediments and therefore also to the pathogens adsorbed to it.

While in the sediment, pathogens are somewhat protected from environmental factors that would otherwise harm them if they were floating around in the water column. Sherer et al., 1992 reports the survival of indicator bacteria in the sediment to be in the order of months, when it is in the order of days for the water column.

For this reason it is important to include the existence of a pathogen reservoir in the sediment into the model.

Taking into account the requirements mentioned above, the processes affecting a single cell compartment are shown in Figure 13.

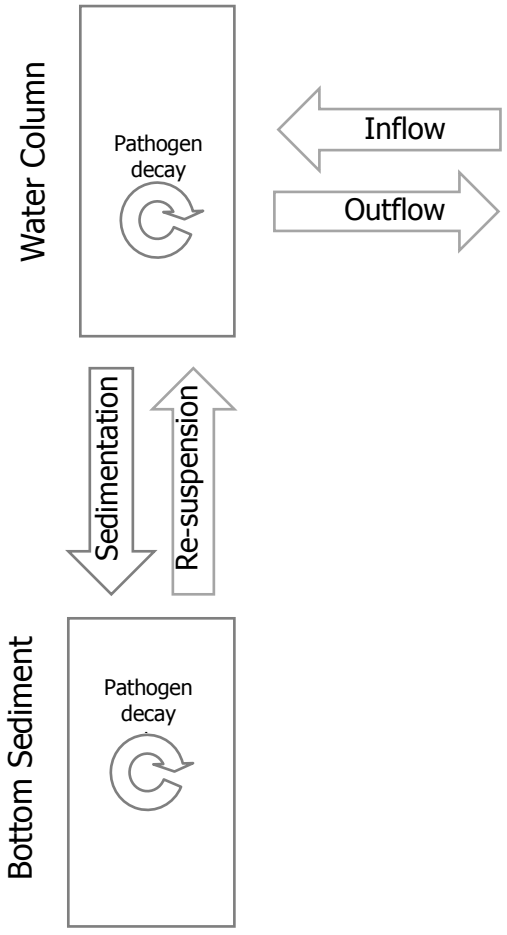

Figure 13: Schematisation of the processes affecting a single cell in a finite element model

The minimum dimension for a model of this kind to operate in would be 1D (length along a river, no variation over width or height), but not much understanding could be gained from this setup. With a 2D model (length and width of a water system, no variation over height) a range of effects can be investigated, for example the rotation of water (and the pathogens with it) in eddies. A full 3D model would also include a varied concentration over the height of the water column.
I have chosen to build a 2D model, as it allows the greatest insight relative to its complexity.

Of course it is unnecessary to reinvent the wheel: there already many programs available which can calculate a 2D hydrological model.
One of these is Delft3D, developed by Deltares, which has many features not only for the most basic models, but also to include for example morphology and ocean waves. I have worked with this model before and found it to be flexible to work with, and as the results can be relatively easily exported to Matlab, the possibilities for post-processing are endless.
Furthermore, the Delft3D suite is open source. This means it is free for anyone use and/or to modify the source code.
It would be best to include the microbiological model as an extension to the Delft3D suite, but unfortunately I do not have any knowledge of the programming languages Delft3D was written in (C++/Fortran). However, it is not necessary to perform the pathogen calculations during the calculations of the hydrological model (as pathogen concentrations do not affect hydrological variables such as flow and water depth), and therefore it is possible to add the model as post-processing, which can be done with Matlab scripts, a scripting language I am familiar with.

I have used Delft3D 4.00.01 and Matlab R2010b to compose the model.

Although I have been able to assemble a formula to calculate pathogen decay (as described in the previous section), there are still a large number of uncertainties involved. Therefore, for the model to be useable after this thesis is complete, it needs to be written in such a way that the pivotal formulas can be altered without requiring a complete rewrite of the code.

The effect of having a second reservoir in the sediment is that the pathogens need to be split up into three groups: free floating (not attached to any particles), suspended (adsorbed to particles but still suspended in the water column) and sediment (adsorbed to particles that have settled).
It is assumed that free floating particles will not adsorb to particles and that adsorbed pathogens will not detach.

I will now give a description of the methods for calculating the various processes as used in the model. A manual to set up your own model can be found in the appendices.

Setup in Delft3D

For the hydrological model, results must first be composed using Delft3D. To this end, a 2D model must be set up, including a grid for the area accompanied by its bathymetry, initial values for flow and water height, boundary conditions for flow and water levels etc.

The morphology module, which can calculate bed levels and concentrations for sediments in suspension is not used, despite its apparent usefulness for the calculation of the movement of pathogens adsorbed to suspended solids. The module would indeed be useful if the pathogen calculations could be done online (in other words: during the hydrological calculations) but as our setup with Matlab is strictly post-processing, it can't be done.
Furthermore the morphology module is currently not part of the open source.
The post-processing script does take into account variations of the bed level, but it has to calculate the movements of pathogens adsorbed to particles itself.

Delft3D needs to be set to produce both map and com files as output (please refer to the Delft3D manual), as these will be imported by the post-processing script. The time interval at which these are saved must be equal. This time interval will be the time step size which the post-processing script will use.

The post-processing script currently does not support the inflow of pathogens at point discharge operations, only at boundaries.

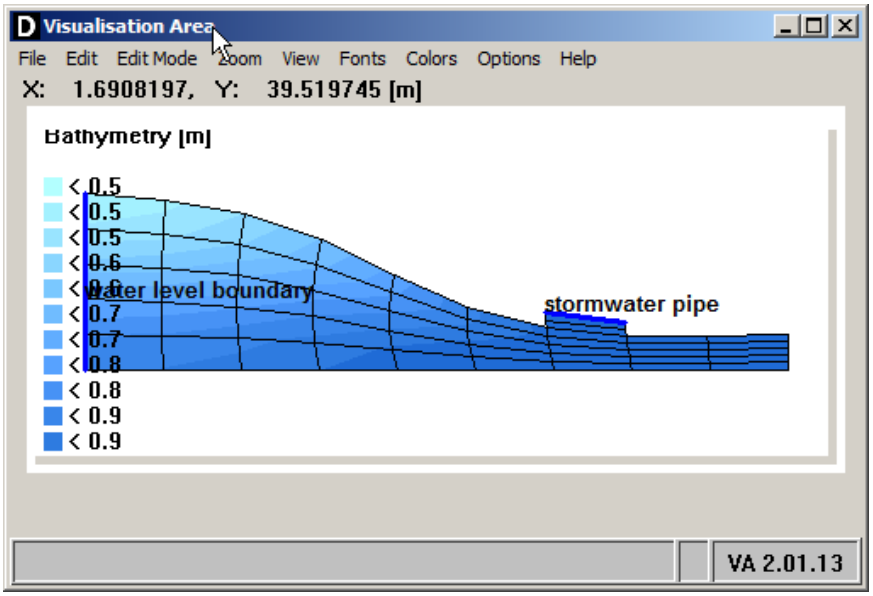

Figure 14: Example of a model setup in Delft3D

Once Delft3D has finished calculating the hydrological model, the end result is a map- and com file with matrices of values. As the grid is not necessarily uniform, specific cells are referred to with their matrix index, expressed as the index in n and m direction. Most values have been calculated at the cell centres, but flow parameters are calculated at the cell interfaces, and have a direction perpendicular to the cell wall. The direction parallel to increasing n index is referred to as the v-direction, the one parallel to increasing m is the u-direction.

Post-processing in Matlab

Setting variables

At the start of the script, many variables need to be set that are not required for the Delft3D calculation, but are for the post-processing script. Some are constant and cannot be changed, while others may vary over both space and time.

If this is the case, I've made a function which interprets the input, which may be 0D, 1D, 2D or 3D in any variation of time and 2D space, and convert it to 3D, keeping it constant along any axis that has not been specified. This enables the user to be able to quickly set these variables to a constant, while still allowing full customisability. For example, 0D: the temperature to 10°C throughout by providing 10, or 1D: as a function of time (by providing something like sin([timesteps])) or varying along the length axis of the water system.

Note that 3D in this context means time × length × width, rather than the usual length × width × height.

The variables that need to be set are:

- the path to the map and com files generated by Delft3D
- the environmental conditions
  - the temperature [°C] (3D support)
  - the seawater ratio [-] (3D support)
  - the acidity [pH] (3D support)
  - the sunlight intensity at the water surface [W/m^2] (3D support)
  - the light attenuation or turbidity [/m] (3D support)
  - the dissolved oxygen [mg/l] (3D support)
  - the gravitational constant, g [m/s^2]
  - the water density, ρ_w [kg/m^3]
  - the formula that converts the sunlight intensity at the water surface to the depth averaged sunlight intensity, equation (5) [W/m^2]
- the variables related to the particles the pathogens are adsorbed to
  - the mean particle diameter, D_{50} [m]
  - the particle diameter compared to which 90% of particles are smaller, D_{90} [m]
  - the particle density, ρ_s [kg/m^3]
  - the relative grain density, $\Delta = \frac{\rho_s - \rho_w}{\rho_w}$ [-]
  - the formula that calculates the friction coefficient, $c_f = g \cdot C^2$ [-]
  - the formula (see Figure 17) that calculates the kinematic viscosity, v [m^2/s]
  - the formula (see equation (17)) that calculates the fall velocity, w_s [m/s]
  - the formula (see equation (21)) that calculates the resuspension speed [m/s]

    - the depth to which there are pathogens in the sediment [m]
- the decay formulas
    - the formula which calculates the decay rate in the water column, equation (7) [/day]
    - the formula which calculates the decay rate in the sediment, equation (7) with $I_{avg} = 0$ [/day]
    - the formula which calculates pathogen population in the water column, equation (1) [count]
    - the formula which calculates pathogen population in the sediment, equation (1) [count]
- the initial conditions
    - the initial free floating pathogen concentration in the water column [count/m^3] (2D support, not time)
    - the initial concentration of pathogens that are absorbed to particles which are present in the water column [count/m^3] (2D support, not time)
    - the initial count of pathogens that are adsorbed to particles which are present in the sediment [count] (2D support, not time)
- the boundary conditions
    - for each boundary condition
        - n-index of this location (vector if it spans multiple cells)
        - m-index of this location (vector if it spans multiple cells)
        - the free floating pathogen concentration in the water column [count/m^3] (1D support, only time)
        - the concentration of pathogens that are absorbed to particles which are present in the water column [count/m^3] (1D support, only time)

Importing data

Only the grid data is imported from the map file, the rest is read from the com file. This is because the Matlab library provided by Deltares returned an error when attempting to retrieve x and y coordinates from the com file.

The values that are imported are:

- the coordinates of the cell corners (drawgrid())
- the area of each cell (GRID GSQS)
- the width and length of each cell (GRID GUU and GVV)
- the discharge (CURTIM QU and QV)
- the flow velocities (CURTIM U1 and V1)
- the water levels (CURTIM S1)
- the bottom depth (INITBOT, DPS)
- the Chézy roughness values (ROUGHNESS CFROU)
- the time step numbers (CURTIM TIMCUR)
- the duration of a time step (PARAMS TSCALE)
- the locations of the open boundaries (BOUNDCNST MNBND)

The script transforms the coordinates for the corners of the cell to both the coordinates in the middle of the cell walls (which is the location of the discharge and velocity values) and the coordinates in the middle of the cell (which is the location of most values such as water level), to use while plotting. It also uses the grid corner coordinates to calculate x and y components of the cell wall directions, which is later used to convert vectors with u and v components to vectors with x and y components.

The time imports are used to retrieve the passed time in seconds and the time interval between steps.

Water depths are calculated by subtracting bed levels from water levels.

The cell area and water depths are used to calculate cell volumes at each time step, which is used later on to convert pathogen count and concentration.

Velocity values (at cell walls) are interpolated to the velocity values at the cell centre, as the post-processing script uses these to calculate resuspension, which occurs there.

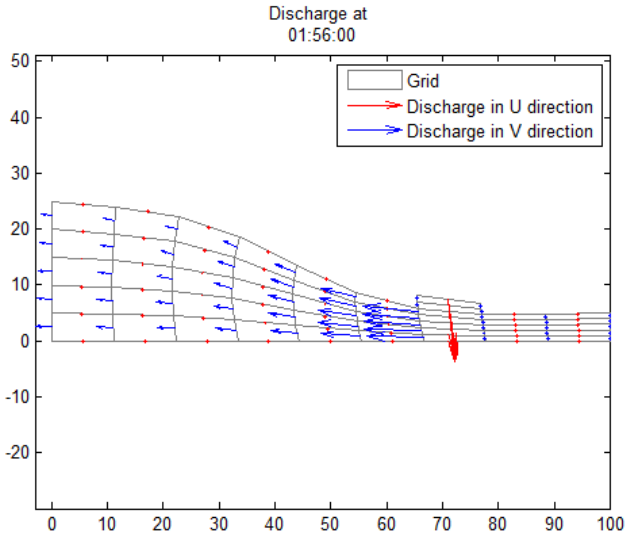

Figure 15: Example of a discharge plot made with the plotting functions after importing Delft3D values into Matlab

Pathogen movement through flow

A schematisation is shown in Figure 16.

At each time step the script loops through the array of cells, starting at 1,1. At each cell, it first sets the pathogen count to be equal to that at the previous time step.

Then, it checks the discharge in u-direction at the cell wall at index n,m-1. If it is smaller than 0, there is a flow from this cell into the previous cell at index n,m-1. In this case the script calculates the flow of pathogens (the concentration in this cell times the discharge times the time step size), distracts it from the current pathogen count in this cell and adds it to the count in the previous cell. If however the discharge in u-direction was larger than 0, it's the other way round (the concentration in the previous cell is used, added to this cell and subtracted from the previous). The script then does the same for the v-direction with the cell at index n-1,m, and moves along to the next cell.

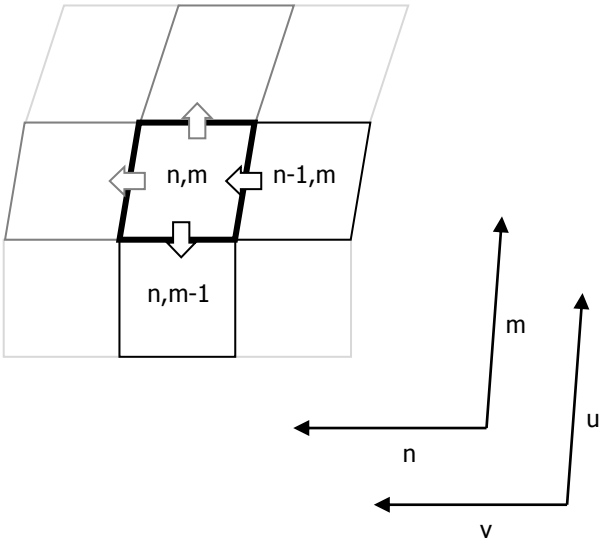

Figure 16: Schematisation of the grid with discharges at the cell walls

Pseudo code

```
for i = 2:data.time.i
  for n = 1:data.n
    for m = 1:data.m
      count(i,n,m) = count(i-1,n,m);
      if discharge.u(i,n,m-1) < 0
        count(i,n,m)   = count(i,n,m)   + dt * discharge.u(i,n,m-1) .* conc(i-1,n,m);
        count(i,n,m-1) = count(i,n,m-1) - dt * discharge.u(i,n,m-1) .* conc(i-1,n,m);
      else
        count(i,n,m)   = count(i,n,m)   + dt * discharge.u(i,n,m-1) .* conc(i-1,n,m-1);
        count(i,n,m-1) = count(i,n,m-1) - dt * discharge.u(i,n,m-1) .* conc(i-1,n,m-1);
      end
      if discharge.v(i,n-1,m) < 0
        count(i,n,m)   = count(i,n,m)   + dt * discharge.u(i,n-1,m) .* conc(i-1,n,m);
        count(i,n-1,m) = count(i,n-1,m) - dt * discharge.u(i,n-1,m) .* conc(i-1,n,m);
      else
        count(i,n,m)   = count(i,n,m)   + dt * discharge.u(i,n-1,m) .* conc(i-1,n-1,m);
        count(i,n-1,m) = count(i,n-1,m) - dt * discharge.u(i,n-1,m) .* conc(i-1,n-1,m);
      end
    end
  end
  % sedimentation and resuspension
  % pathogen decay
  % calculating the new concentrations
end
```

The downside of this approach is that the pathogens can only move to adjacent cells in one time step, never further. It is also possible that the outflow of a cell exceeds the volume it holds, which would lead to a negative pathogen count. If this is the case, the script halts and throws an error.

For this reason the time step size should not be too large, relative to the size of the discharge.

It also assumes that within the cell the pathogen concentration is constant. This assumption will hold better if grid sizes are chosen smaller.

Sedimentation and resuspension

The difficulty here is that the different flows from and to the sediment are important: we can't simply take the sum of the sedimentation and resuspension as is normal in a hydrological model.

The downward flow is relatively simple, as the fall velocity of the particle the pathogen has adsorbed to can be calculated according to the approximations for natural sediment that were proposed by Van Rijn, 1993, shown in equation (17).

$$w_s = \frac{\Delta \cdot g \cdot {D_{50}}^2}{18 \cdot \nu} \qquad for\ 1 < D_{50} \leq 100\mu m$$

$$w_s = \frac{10 \cdot \nu}{D_{50}}\left(\sqrt{1 + \frac{0.01 \cdot \Delta \cdot g \cdot {D_{50}}^3}{\nu^2}} - 1\right) \qquad for\ 100 < D_{50} \leq 1000\mu m \qquad (17)$$

$$w_s = 1.1 \cdot \sqrt{\Delta \cdot g \cdot D_{50}} \qquad for\ D_{50} > 1000\mu m$$

with w_s the fall velocity [m/s], Δ the relative particle density [-], g the gravitational constant [m/s^2], D_{50} the mean particle diameter [m] and ν the kinematic viscosity [m^2/s]

This formula also uses the kinematic viscosity, which is dependant of the temperature. As the temperature in the model can vary, we must calculate the kinematic viscosity at each location and time step. I have been unable to find a proper formula for this, however I did find a table of values, which I've fitted to a 4th power polynomial through excel, as is shown in Figure 17.

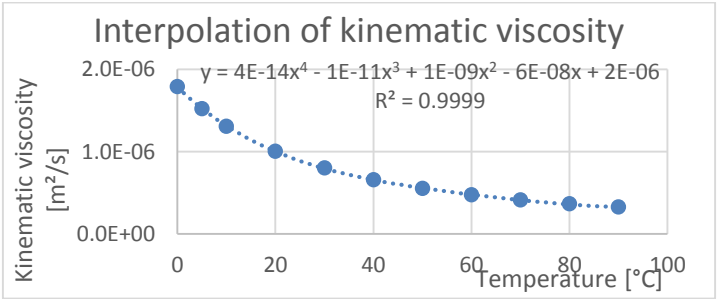

Figure 17: Fit of the kinematic viscosity as a function of temperature

Once the fall velocity is known the amount of pathogens that move from the water column to the sediment can be calculated using equation (18), assuming that the pathogen concentration is constant along the water depth.

$$S_{down} = \max\left(1 ; \frac{w_s \cdot \Delta t}{h}\right) \cdot c \quad (18)$$

with Δt the time step size [s], h the water depth [m] and c the count of pathogens adsorbed to particles in the water column [count]

The closest I've found to an approach that calculates the resuspension of sediment is the approach for bed load transport by Van Rijn, 1984. His formula for the bottom transport per unit of width is shown in equation (19).

$$s_b = \sqrt{g \cdot \Delta \cdot {D_{50}}^3} \cdot 0.053 \cdot \frac{T^{2.1}}{{D_*}^{0.3}} \quad \text{for } T < 3$$
$$s_b = \sqrt{g \cdot \Delta \cdot {D_{50}}^3} \cdot 0.1 \cdot \frac{T^{1.5}}{{D_*}^{0.3}} \quad \text{for } T \geq 3 \quad (19)$$

with D_* the dimensionless particle parameter $D_* = D_{50}\left(\frac{\Delta \cdot g}{\nu^2}\right)^{\frac{1}{3}}$ and T the dimensionless bed shear stress parameter $T = \frac{\tau' - \tau_{cr}}{\tau_{cr}}$

with τ_{cr} the bed shear stress for initiation of movement according to Shields and τ' the particle related shear stress $\tau' = \left(\frac{C}{C'}\right)^2 \cdot \tau$

with C the Chézy coefficient, τ the shear stress $\tau = \frac{g}{C^2}u^2$ with u the flow velocity,

and C' the Chézy coefficient for D_{90} $C' = 18 \cdot log\left(\frac{12 \cdot h}{D_{90}}\right)$

The bed shear stress for initiation of movement is described by Shields, 1936 as in equation (20).

$$\tau_{cr} = 0.055 \cdot g \cdot D_{50} \cdot \rho_w \quad \text{for } D_* > 150$$
$$\tau_{cr} = 0.013 \cdot {D_*}^{0.29} \cdot g \cdot D_{50} \cdot \rho_w \quad \text{for } 150 \geq D_* > 20$$
$$\tau_{cr} = 0.04 \cdot {D_*}^{-0.1} \cdot g \cdot D_{50} \cdot \rho_w \quad \text{for } 20 \geq D_* > 10$$
$$\tau_{cr} = 0.14 \cdot {D_*}^{-0.64} \cdot g \cdot D_{50} \cdot \rho_w \quad \text{for } 10 \geq D_* > 4$$
$$\tau_{cr} = 0.24 \cdot {D_*}^{-1} \cdot g \cdot D_{50} \cdot \rho_w \quad \text{for } 4 \geq D_* \quad (20)$$

with ρ_w the water density

After the script has worked through the above equations, it ends up with the bed load transport per unit of width [m^2/s]. What we need to know is the bed erosion. Normally this is calculated from the gradient of the sediment transport, but that would sum in the sedimentation, which we cannot do because the pathogen need to move both to and from the sediment.

If we assume that the bed load transport is generated in 1 meter, we can divide by 1m to find our erosion speed. The final formula is shown in equation (21).

$$S_{up} = \max\left(1 ; \frac{s_b \cdot A \cdot \Delta t}{h_s}\right) \cdot c \quad (21)$$

with Δt the time step size [s], b' the cell width perpendicular to the flow direction, A the cell area, h_s the pathogen mixing depth in the sediment [m] and c the count of pathogens adsorbed to particles in the sediment [count]

The downsides to this approach are that the bed load transport probably isn't generated purely within the cell, so that the upward sediment flux is actually lower. Furthermore, if the sedimentation rates and flow patterns are relatively high compared to the time step, an oscillation occurs, as is shown in Figure 18. This must be solved by choosing a smaller time step size.

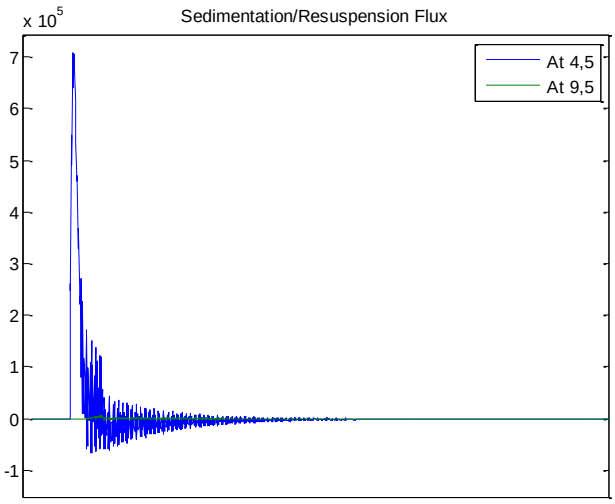

Figure 18: Oscillation in the sedimentation/resuspension flux.

Pathogen decay

The final step within the time iteration is the application of the decay formula.

$$c(t) = c_0\, e^{-kt} \tag{1}$$

with

$$k_{overall} = (c_1 + c_2 \cdot S) \cdot {c_3}^T + \left(c_4 + c_5 \cdot {c_6}^{|A-6.5|}\right) \cdot I_{avg} \cdot O + c_7 \tag{7}$$

Output

I've made a few functions that can be used to quickly plot a range of values that have been produced by the post-processing script.

There are three different kinds: plotdischarge, plotscalar and plotvaratlocation.

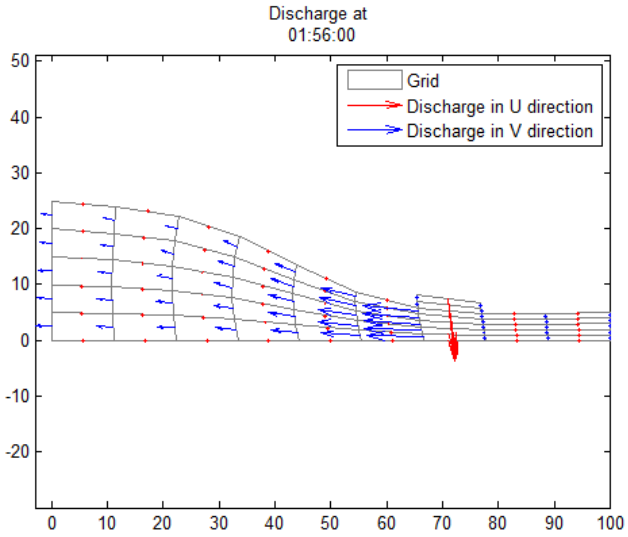

Figure 19: plotdischarge();

By calling plotdischarge(), the script plots a grid with the discharges in both u and v directions as vectors in the correct locations for the first time step. A slidebar is added at the bottom to navigate to other time steps.

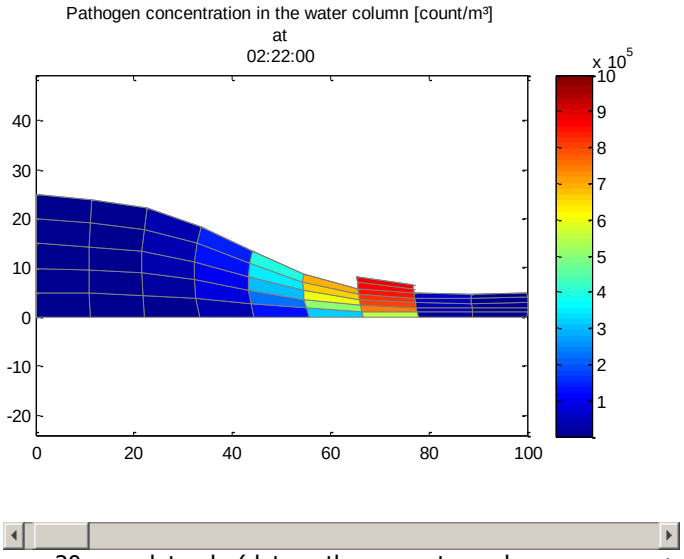

Figure 20: plotscalar(data.pathogen.water_column.conc + data.pathogen.suspended.conc, 'Pathogen concentration in the water column [count/m³]');

By calling plotscalar(name_of_the_3D_variable,'the title for the graph'), a plot is drawn for the first time step, containing the variable displayed in the grid. A slidebar is added at the bottom to navigate to other time steps.

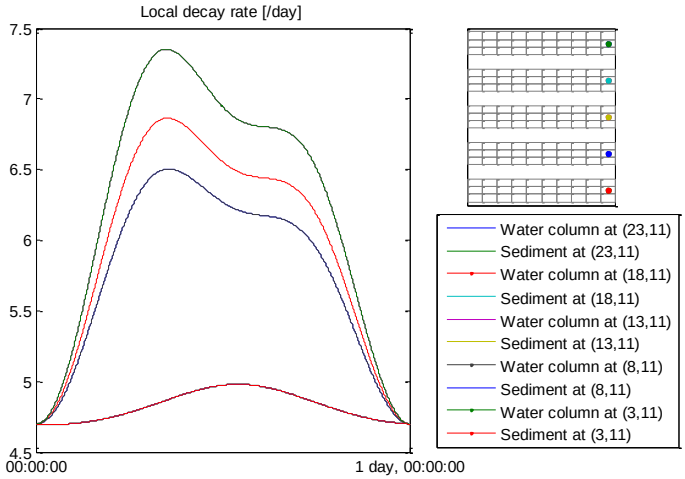

Figure 21: plotvaratlocation(vars,'Local decay rate [/day]');

By calling plotvaratlocation(vars,'the title for the graph'), a line plot is drawn showing the time variation of the vars listed in "vars", as well as a plot of the grid with a dot at the appropriate location. "Vars" should be a cell array of structs, each containing a data, location and label value. For example: vars = {

```
struct('data',data.pathogen.water_column.conc(:,4,2),'location',[4 2],'label','At 4,2'),...
struct('data',data.pathogen.water_column.conc(:,4,3),'location',[4 3],'label','At 4,3')
};
```

Model results

Several different water systems have been modelled and investigated using the model described. The goal of doing these runs has been to gain insight into the behaviour of the water system as well as testing the model itself, showing its strength and its limitations.

The influence of channel depth

In this run the goal was to investigate the influence of the water depth. On the one hand, having a small depth is a benefit because it means the decay rate in the water column is higher, while on the other hand it means that the same flow needs to pass through a smaller area, thus increasing the flow velocity, which reduces the time that the pathogens are being exposed to the higher decay rate as well as reduce the amount of pathogens that are removed from the water column through sedimentation.

Also, as this model setup is relatively simple, it is a good case scenario to explain the way the Delft3D suite and the Matlab script is used.

Model setup

Delft3D

First, the hydrological part of the model needs to be calculated with Delft3D. First, a grid needs to be made, using RGFGRID in the grid section of the Delft3D suite. In this case a rectangular grid was made to form five channels, each 3m across and 20m long, with 3 cells across and 10 along the length axis.

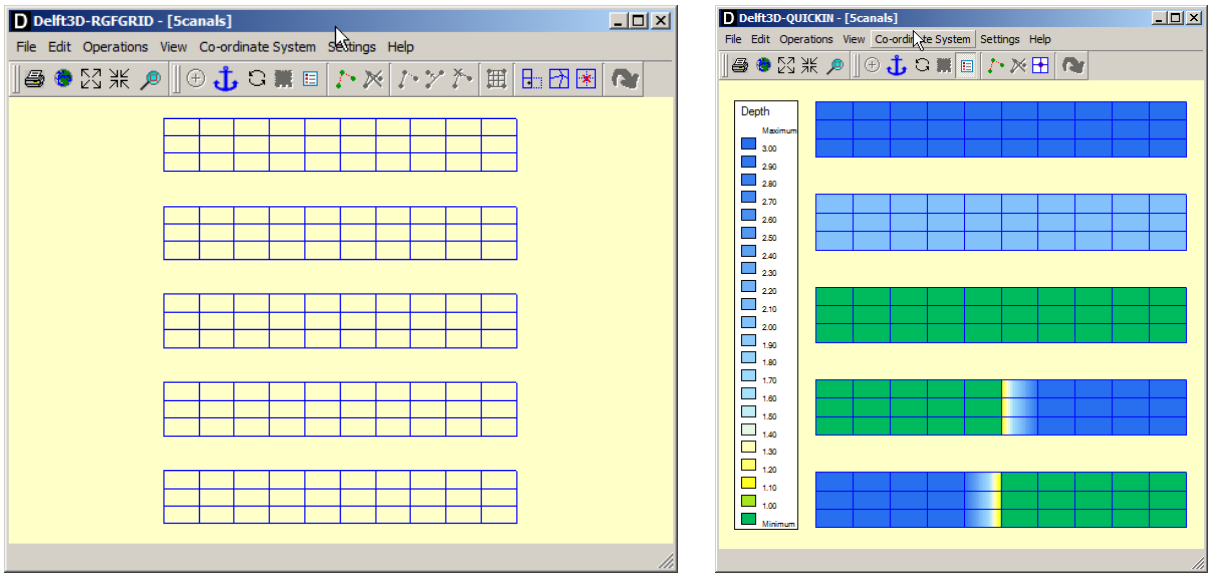

Figure 22: The grid in RGFGRID and the bathymetry in QUICKIN

Next, the bathymetry needs to be added to the grid. This is done using QUICKIN. The three topmost channels will be given a constant depth of 3m, 2m and 1m. The bottom two channels will be used to see if it matters if the channel depth changes. The fourth channel is given a depth of 1m for the first part of the channel and 3m in the last part. In the fifth channel this is reversed, with 3m in the first part and 1m in the second.

Now, we can proceed to the flow module, and begin with the flow input to create an MDF-file, which contains the configuration for the Delft3D model.

In the domain section, the grid and bathymetry files made in the previous step must be selected.

In the time frame section, the simulation start and end times and the time step size are configured. In this case I have chosen a simulation time of 1hr, as our boundary conditions will not change over time. The time step size is 0.05min, which is quite small, but is required to prevent the pathogen concentrations from oscillating in the Matlab script.

In the initial conditions section, the initial values of the water level and flow can be set, either through a file that has been generated in another Delft3D run, or as a uniform value. In this case, uniform values are used and the water level is set to 0m, relative to the reference level.

In the boundaries section, the boundary conditions must be set. It's easiest to do this through the Visualisation Area, using Edit Mode -> Add while the boundary section is opened. In this case there is a boundary at both the left and right end of each of the five channels, totalling 10 boundary sections.

All the boundaries need flow conditions. It is common to have a discharge boundary upstream of the model and a water level boundary downstream. This is also set in this case: the inflow for each of the channels is set to a total discharge of $2m^3/s$, and the downstream is set to a water level of 0m. The downstream boundaries are also

given a "reflection parameter alpha" of 100s, which is a commonly used value. This parameter prevents waves from reflecting off the downstream boundary, creating an unstable model.

The final section that needs to be edited is the output section. At the storage tab, Delft3D needs to be configured to produce the map file at at least one time, and a communication file at the time interval that the Matlab script will be using. In this case the map results interval is set to two days, larger than the total simulation time, so that only a map of the start is produced. The communication file's interval is set to 0.05min, equal to the Delft3D time step size. A history and restart file are not needed, so their interval can be set to 0.

Once the configuration has been saved the calculation can be started. Once completed, the output values can be checked by reading the map or communication file with QUICKPLOT.

When the Delft3D model is run for the first time, it is recommended to also set the communication file interval to 0, as writing to the file severely increases the calculation time. The map file interval should be increased so the output can be checked. Once the Delft3D output is satisfactory, the intervals can be set to the proper value.

If the Delft3D calculation returns an error this can be seen by opening the tri-diag.*runid* file with a text editor. Warnings about convergence in UZD should be solved by avoiding small grid cells and/or choosing a smaller time step size.

Matlab

To configure the Matlab script, create a new script file in the inputfiles folder. It's easiest to open the template file and save it under a new filename, your_runid.m.

Running this file sets a number of variables that will later be used during the calculations.

First, Matlab needs to know where to find the com and map files generated by Delft3D.

There is a command here to run the script that reads the Delft3D files, before the remaining variables are set. This allows the values calculated by Delft3D to be used, as well as the time vector and such.

Then, the system variables need to be set.

For this run we will use a value of 0 for the seawater ratio, a pH of 7, 0.5/m for the sunlight attenuation and sine functions for the water temperature, dissolved oxygen and sunlight intensity at the surface. These are plotted in Figure 23. As can be seen, all have a period of 1day. The sunlight intensity is highest at noon, while the temperature peeks an hour later, as if the water has been warmed up by the sunlight. The oxygen content is the inverse of the temperature.

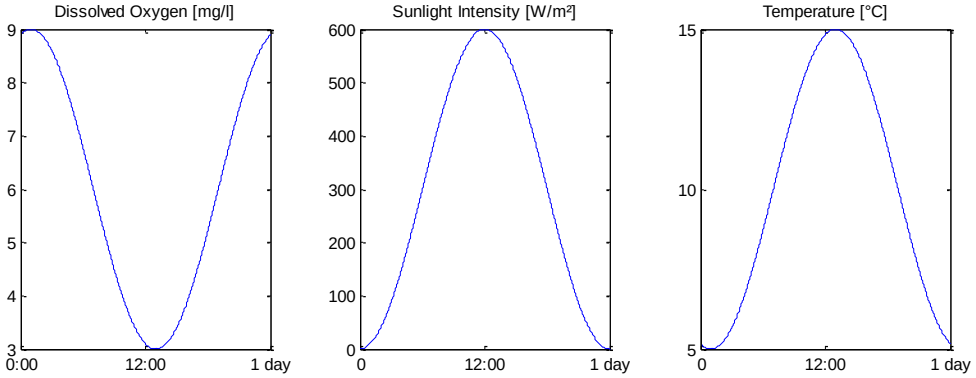

Figure 23: Time varied input for dissolved oxygen, sunlight intensity and temperature

For the water density, 1000kg/m^3 is used.

For the sediment variables we use a D_{50} of 0.2mm and a D_{90} of 0.24mm, with a density of 1100 kg/m^3. These values have been chosen because studies have shown that pathogens tend to adsorb to smaller particles. (for example: Schillinger et al., 1985)

A sediment mixing depth (the depth over which the amount of pathogens in the sediment is spread, to determine the concentration) is set to 10cm.

Several formulas are also set here, these are the formulas that have been discussed previously.

Next, the initial conditions need to be entered. The concentration is required for the free floating pathogens in the water column, the pathogens in the water column that are adsorbed to particles and the pathogens in the sediment. For this run, they are all set to 0.

Then, the boundary conditions are required. For each boundary the concentrations of the free floating and adsorbed pathogens in the water column must be supplied. The order of the boundary locations is the same as it was in Delft3D. During initialisation, the model also displays the boundaries it has read from the communication file and at which coordinates to occur. Note that Delft3D uses the format m,n for the coordinates, while Matlab uses n,m. In this run a concentration of 1 million /m^3 has been used, with 80% of those adsorbed to suspended particles and 20% free floating.

Finally several coordinates of interesting locations at which some variables will be plotted at the end of the calculation can be provided. For this run, we use the middle, most downstream cell of each channel.

Once the inputfile has been saved, the model should be initialised by typing *init_model* in the Matlab command window. The script will ask for the runid, and then read all the variables. It then shows three plots of the discharge vectors, the water depth and the velocity at the cell centres that can be used to check the script has properly imported the Delft3D data. If this is the case, *y* should be entered to continue the calculations. If this is not chosen, the calculations can be started by entering *run_model* in the command window.

After that the script will begin with the actual calculations. Every 100th timestep the progress will be printed to the command window.

Once the calculations are complete, the script shows a range of plots that might be interesting, and also saves the data to an output file. All data used and calculated by the script is stored in this file and can be opened at a later time.

```
>> init_model
Please enter the runid: varieddepth
  Clear or change the variable "runid" to reset this option.
Found 10 open boundaries
  Boundary 1  from (1,22) to (1,24)
  Boundary 2  from (1,17) to (1,19)
  Boundary 3  from (1,12) to (1,14)
  Boundary 4  from (1,7) to (1,9)
  Boundary 5  from (1,2) to (1,4)
  Boundary 6  from (12,22) to (12,24)
  Boundary 7  from (12,17) to (12,19)
  Boundary 8  from (12,12) to (12,14)
  Boundary 9  from (12,7) to (12,9)
  Boundary 10  from (12,2) to (12,4)
Model initialisation complete.
Proceed with the calculations? Enter Y to continue. Y
Calculating.. Timestep 100/1201
Calculating.. Timestep 200/1201
Calculating.. Timestep 300/1201
Calculating.. Timestep 400/1201
Calculating.. Timestep 500/1201
Calculating.. Timestep 600/1201
Calculating.. Timestep 700/1201
Calculating.. Timestep 800/1201
Calculating.. Timestep 900/1201
Calculating.. Timestep 1000/1201
Calculating.. Timestep 1100/1201
Calculating.. Timestep 1200/1201
Done. Saving..
Save complete, output stored at outputfiles/varieddepth.mat
```

Figure 24: Contents of the Matlab command window while executing the Matlab script

Model results

As all the inputs and flows in this run are constant, the pathogen concentrations are expected to reach an equilibrium state, in which the sum of the pathogen outflow and the removal is equal to that of the inflow. To compare the sedimentation rate to the decay rate, the percentage of pathogens removed from the water column in a time step through sedimentation can be converted to a decay rate. The results of this are shown in Figure 25. The spatial variation of the pathogen decay rate in the water column is caused by the water depth: a larger depth means that the depth averaged light intensity is lower. The variation of the sedimentation rate is distributed similarly, because if the water depth is larger, it takes a longer time for a particle to settle all the way to the bottom.

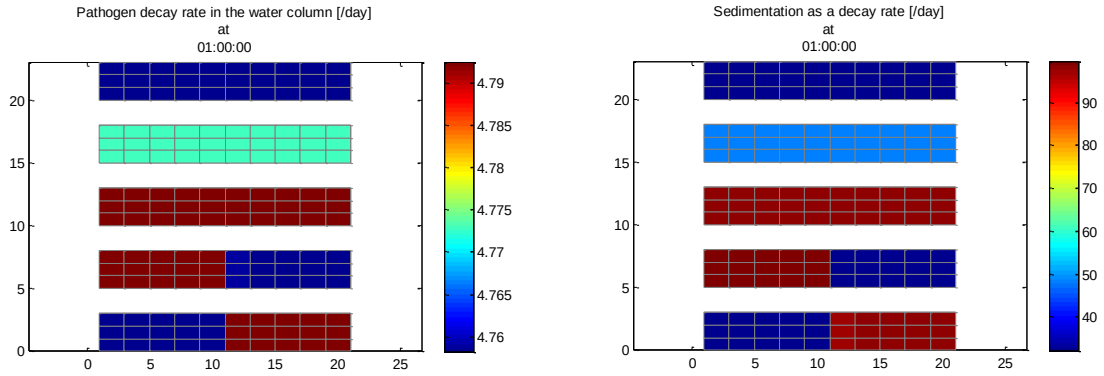

Figure 25: Comparison of pathogen decay and sedimentation as a decay rate

What should stand out is that the sedimentation decay rate (30 to 100/day) is many times higher than the pathogen decay rate (4.76 to 4.79/day). This means that in the water column, removal of pathogens is dominated by sedimentation.

In the sediment, the only way for the pathogens to be removed is through decay (and resuspension which does not occur in this run), so that there, the amount of pathogens added through sedimentation from the water column must be balanced by the pathogen decay rate. As the sedimentation is so high compared to the decay rate, the equilibrium pathogen concentration in the sediment would be many times higher than that in the water column. This result is supported by Crabill, 1999, who discovered large concentration of pathogens in the sediment (2200 times that in the water column) and that the sediments acted as a reservoir.

The decay rate also has a temporal variation caused by the temporal variation of the temperature, oxygen content and light intensity, as is shown in Figure 26. The hump is caused by the phase shift between the light intensity and the temperature and oxygen content.

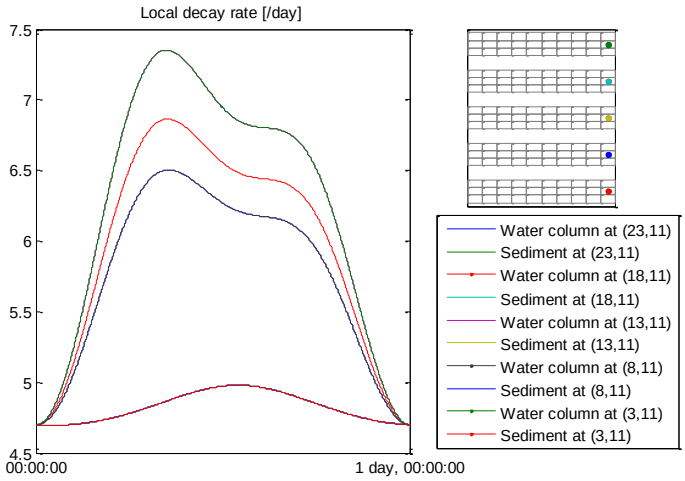

Figure 26: The decay rate over time in various locations

The equilibrium state for the pathogen concentration in the water column was reached within about 10 minutes, but the equilibrium for the pathogen concentration in the sediment was not anywhere near reached in the calculation time. Therefore, in order to be able to make a statement about the equilibrium state, the Matlab script was repeated with the pathogen concentrations at the end of the initial model run, with a much increased concentration in the sediment, until the output varied little over the time period.

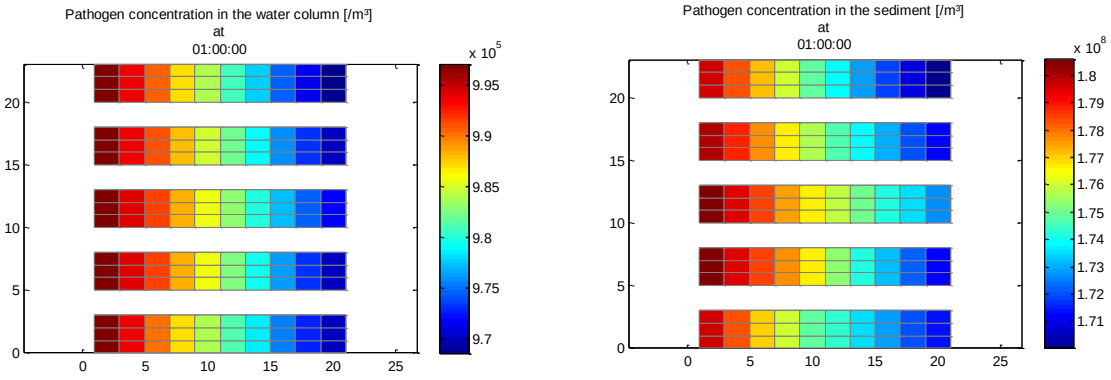

Figure 27: Pathogen concentration in the water column and in the sediment in the equilibrium state

The equilibrium pathogen concentrations are shown in Figure 27. The deeper channel has both a (slightly) lower concentration in the water column and in the sediment at the outflow, suggesting that the increase in sedimentation and decay rate with decreased depth do not outweigh the disadvantage of the increased velocity.

The channel which first is shallow and then deep performs slightly better than the channel that is deep at first and shallow later. This would be because the improved removal through decay and sedimentation occurs at a stage where more pathogens are in the water.

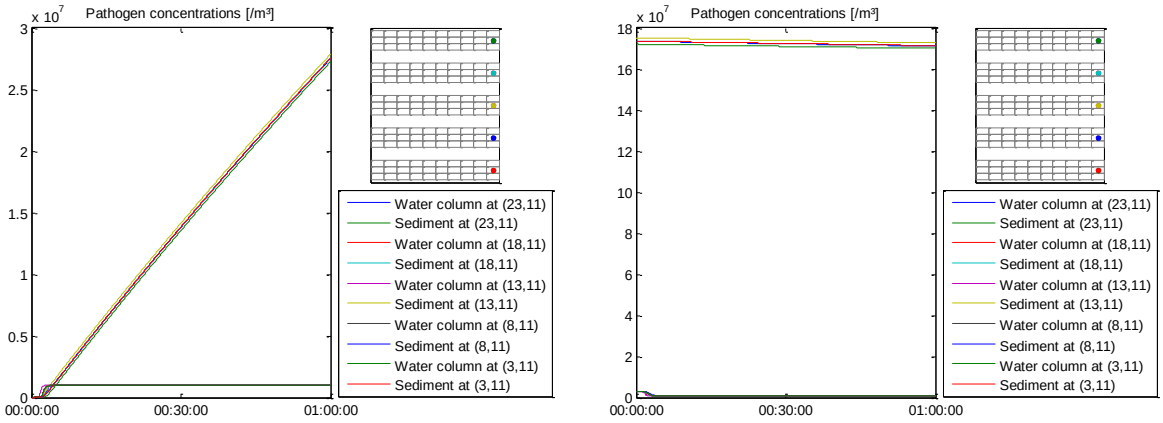

Figure 28: Temporal variation of pathogen concentrations at various locations in the initial run (left) and the equilibrium state (right)

Influence of the inflow shape

For this model I wanted to investigate if pathogens in the water column could be contained near the inflow of contaminated water, for example a stormwater outflow. I will compare a direct inflow (run A) with an inflow with a basin (run B), and an inflow with a basin and an eddy in the basin (run C).

Model setup

Three separate model setups have been prepared as described above, each with a different configuration at the pathogen inflow. The following inputs have been used:

Delft3D:

- A 15x10 cell grid, resembling a small canal section (3m wide, 30m long) with an inflow of water about one third downstream. The basin runs have an extra 4.5x2m basin next to the inflow. One has a thin dam separating the basin from the main canal to generate an eddy within the basin.
- The bathymetry is uniform with a depth of 1m throughout.
- The total run time is 2hrs and the time step size is 0.025min.
- The initial values are no flow and a water level of 0m throughout.
- The boundary conditions are:
    - Inflow: constant discharge of $3m^3/s$
    - Outflow: constant water level of 0m
    - Pathogen inflow: constant discharge of $1m^3/s$
- The horizontal eddy viscosity has been set to $0.1m^2/s$ (in all the runs) to allow the formation of an eddy

Matlab script:

- For the environmental variables the same values as for the previous runs have been used.
- For the particle variables the same values as for the previous runs have been used.
- The initial conditions are that no pathogens are present in the sediment or water column.
- The boundary conditions are:
    - Inflow: no pathogens
    - Outflow: N/A
    - Pathogen inflow: free floating pathogen concentration 200 000 $/m^3$, adsorbed pathogen concentration 800 000 $/m^3$

Model results

As shown in Figure 29, in run A the stormwater inflow enters the canal and is then quickly absorbed in the canals flow. In run B the stormwater slows in the basin area, and most of it flows into the canal at the downstream end. In run C, an eddy is formed in the basin as was intended.

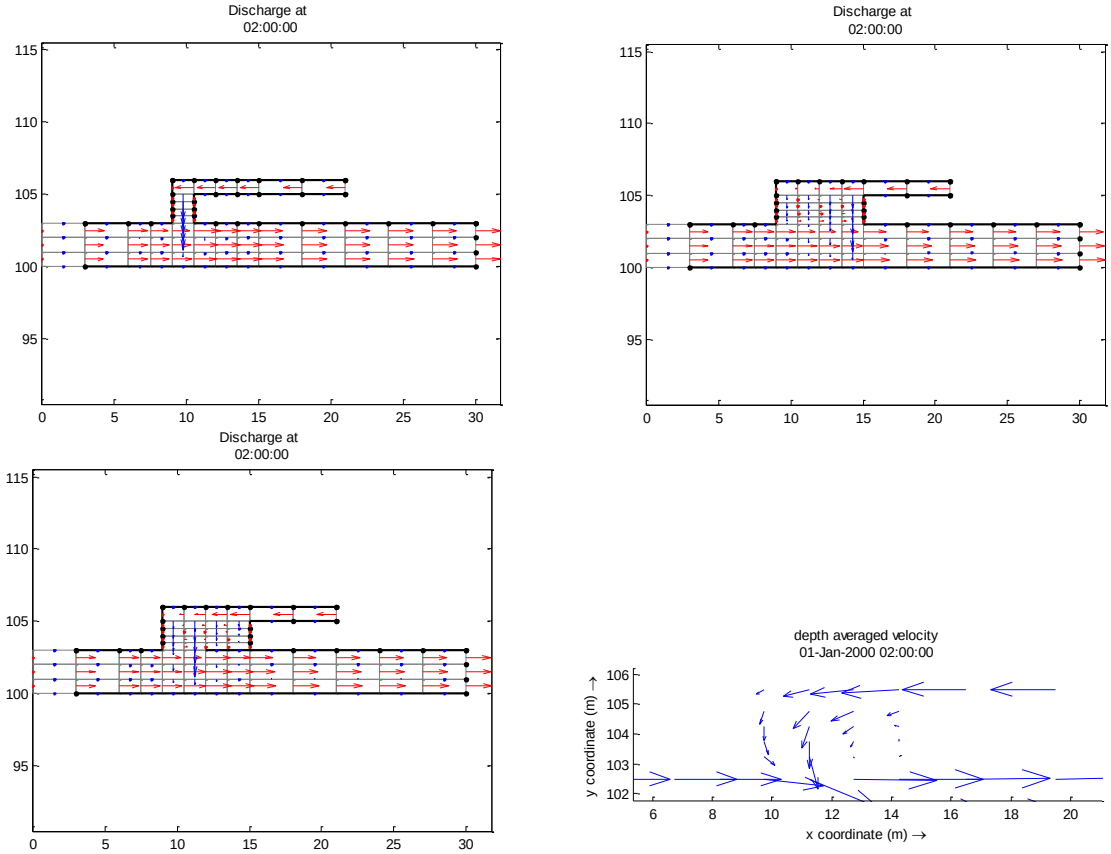

Figure 29: Plot of the discharge vectors for run A (top left), B (top right) and C (bottom left), and the interpolated velocity vectors, zoomed in at the eddy in run C (bottom right).

As can be seen in Figure 30, Figure 31 and Figure 32, run B produces the lowest pathogen concentration downstream in the canal, both in the water column and in the sediment. The pathogens are retained both in the sediment and water column in the basin.

The disadvantage of having an eddy (run C compared to B) seems to be caused by the constricted outflow from the basin. This leads to an increased velocity, removing more pathogens from the basin.

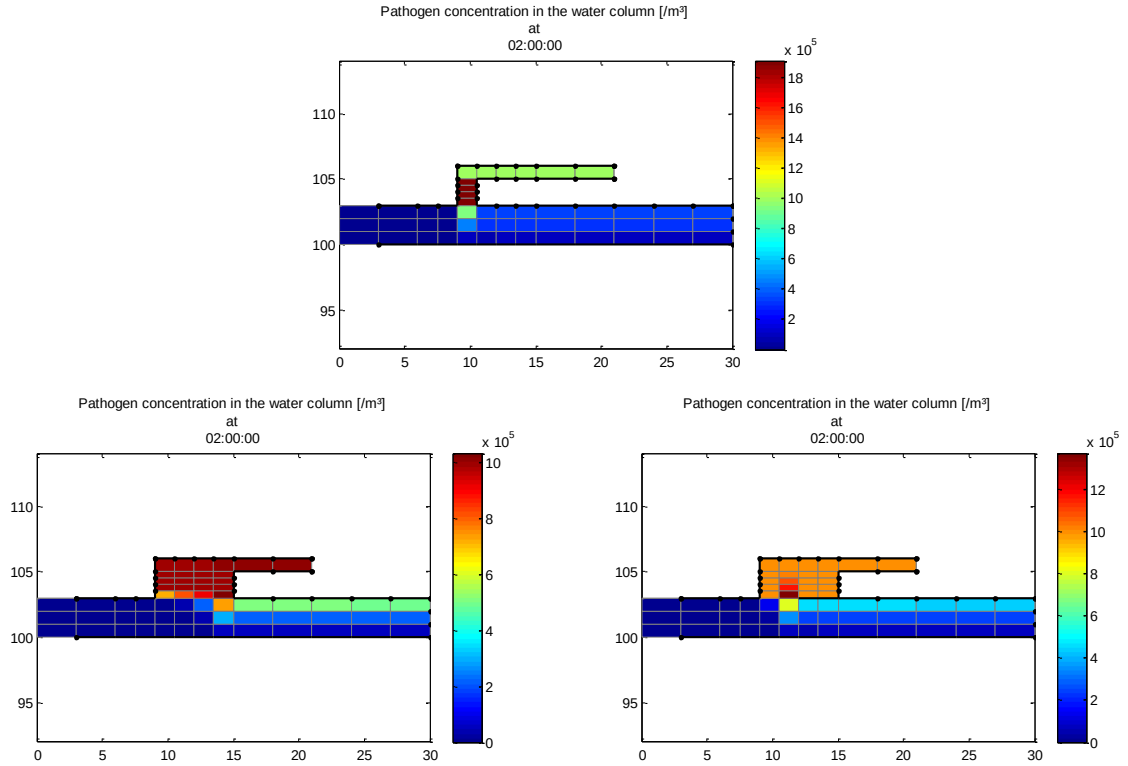

Figure 30: Plot of the pathogen concentration in the water column for run A (top), B (bottom left) and C (bottom right).

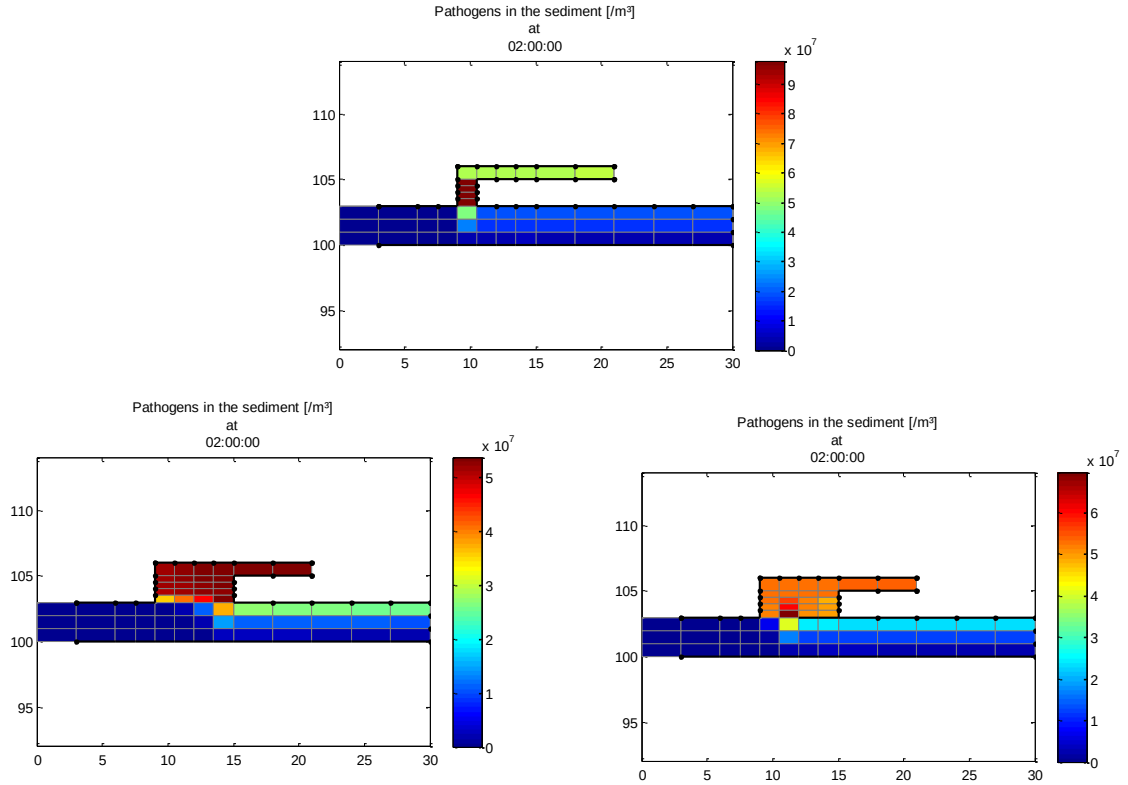

Figure 31: Plot of the pathogen concentration in the sediment for run A (top), B (bottom left) and C (bottom right).

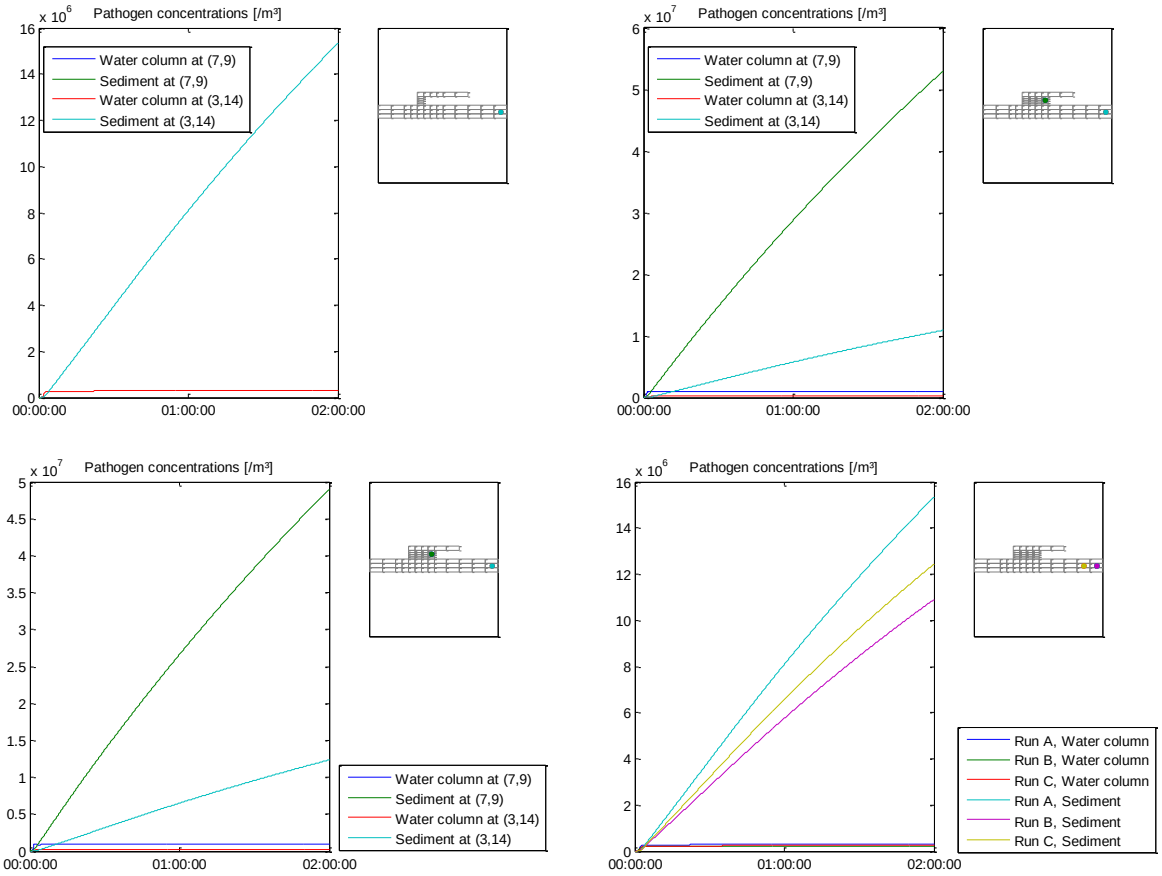

Figure 32: Plot of the pathogen concentrations for run A (top left), B (top right) and C (bottom left), and pathogen concentrations at the outflow for all the runs (bottom right).

The influence of natural banks

There is a trend among Dutch water managers to design systems with "natural banks", banks that are wide and shallow with plenty of vegetation. This provides the system with higher storage at high water levels, a haven to wildlife and is considered more pleasing to the human population as well.

Vegetation along the banks would increase the roughness, slowing down flow velocity, which would lead to an increased sedimentation rate.

This could mean that pathogens adsorbed to particles are more likely to settle along the banks (even though most water flows through the centre of the stream), creating a reservoir of pathogens in the sediments there.

The decay rate in the water column could either be increased (the water is less deep, thus the average light intensity is closer to that on the surface) or decreased (as the vegetation there would cause shading). Neither of these effects would influence the decay rate in the sediments, as it is assumed that the influence of light is negligible there.

The apparent importance of sedimentation means that this would be a good setup to test if the model can handle the pathogen fluxes related to sedimentation and resuspension properly. The desire to also test resuspension would mean that there would have to be relatively high flow velocities in the stream centre, which would differ from the normal situation in the Netherlands.

It is also possible to have a somewhat extravagant grid shape to test the models robustness when faced with cells varying in size and shape and sharp turns. Furthermore, the conversion of the vector directions from u and v to x and y for plotting could use a good check.

Model setup

Two separate model setups have been made to compare a natural bank with a steep bank. The following inputs have been used.

Delft 3D:

- A 34x8 cell grid, in the shape of a meandering stream. The stream width is roughly 20 meters and the length is roughly 480 meters.
- Two different bathymetries:
    - Width axis:
        - The "natural" bathymetry is deepest in the middle and gradually slopes to the water height at the edge of the bank.
        - The "steep" bathymetry is deeper in the middle, and at the side the bed level rises above the water level to ensure the channel is much narrower.
    - Length axis:
        - A slope of about 1:10 000 (0.05m over the 480m length) is introduced to increase the flow velocities. A slope is required increase velocity, to test the behaviour of resuspension.
- The total time is set to 12 hours, the time step is 0.025 minutes. This is quite a small time step size, which proved to be needed to reach a stable solution for sedimentation and resuspension in small cells. To keep the calculation time from being excessive (these settings the total calculation time for both runs in Delft3D and Matlab is about 3-4hrs) the total duration was chosen to be quite short: the pathogen concentrations have not yet reached an equilibrium value.
- The initial conditions are no flow and a uniform water level of 0m.
- The boundary conditions are:
    - Inflow: constant discharge of 40m^3/s
    - Outflow: constant water level at 0m
- The Chézy roughness is space varied with a value of 50 $m^{1/2}$/s (smooth) in the stream centre and 20 $m^{1/2}$/s (rough) at the edge, to simulate the presence of vegetation at the banks. The same roughness file is used for both runs, but because the bathymetry of the steep bank does not allow water to flow at the edges, this means the roughness here is 50$m^{1/2}$/s throughout.

  The roughness file was generated as if it was a depth file using QUICKIN as recommended on the forums of Delft 3D[1], because a built in method currently does not exist. The process introduces a shift as QUICKIN defines the values at the cell corners, which Delft3D then assumes to be at the cell walls. At the forums, Bert Jagers calls the effect of the shift negligible. The roughness is also used by the Matlab script, which interprets the value to be at the cell centre. If the generation of the roughness file is built in to Delft 3D at a later stage, the Matlab script should be adapted to interpolate the values at the cell walls to values at the cell centre.

[1] Link: http://oss.deltares.nl/web/delft3d/delwaq/-/message_boards/view_message/43816#_19_message_44030

Matlab script:

- For the environmental variables the same values as for the previous runs have been used, except for the sunlight intensity at the surface. This has been modified to follow a sine function along the width axis, with 100% light intensity in the stream centre and half at the banks, to simulate shading caused by the vegetation.
- D50 (1mm) and D90 (35mm) have fairly high values, while the relative grain density is quite low (0.01), to trigger the resuspension threshold.
- The initial conditions are that no pathogens are present in the water system.
- The boundary conditions are:
    - Inflow: free floating pathogen concentration 200 000 /m^3, adsorbed pathogen concentration 800 000 /m^3
    - Outflow: N/A

Model results

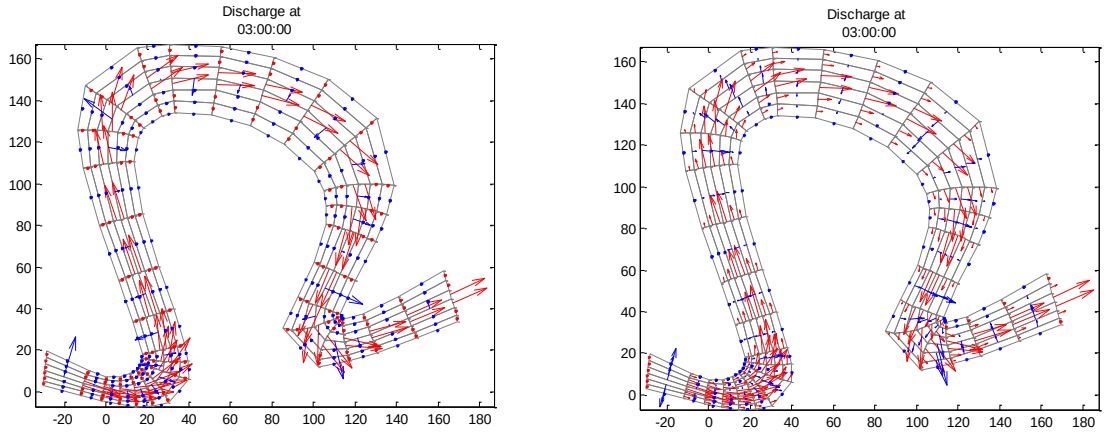

Figure 33: Plot of the discharge vectors for the steep run (left) and natural run (right)

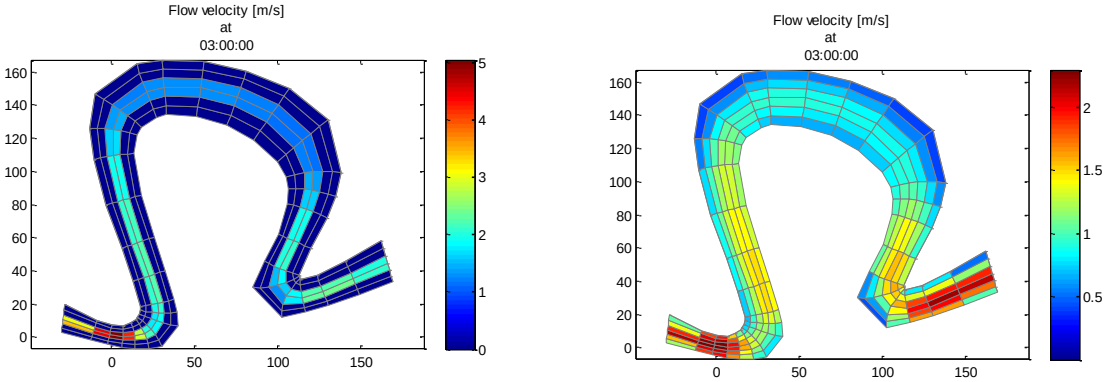

Figure 34: Plot of the flow velocities for the steep run (left) and natural run (right)

As can be seen in Figure 33 the discharge vectors calculated in both runs appear to be plotted correctly. For the natural run it can be seen that the discharge in the centre of the stream is indeed greater than at the edges, as was expected. In the straights, the flow mostly follows the length axis of the stream (u direction, red in the figure), whereas in the bends both the u and v components are significant.

The velocities, as shown in Figure 34 are also as can be expected: faster in the steep bank run because the friction is lower and a bit slower in the horizontal straight than in the vertical ones, as the stream is slightly wider there.

The pathogen concentration in the water column, shown in Figure 35, shows that at the outflow of the stream, the pathogen in the steep banks run is about 7.5e5, while that of the natural banks run is around 5.5e5.

The run with natural banks has more pathogens on the right bank at the second curve and also at the downstream end. This is caused by an increased flow from the middle of the stream to this bank. In the second curve most pathogens are on the inside of the curve, because the water of the main stream is taking a shortcut across the shallower areas, maximizing the gradient. At the last curve the opposite is seen, as this curve is much tighter and the main stream is pushed outward by the centrifugal force.

It can also be seen that the concentration decreases downstream: this is caused by the combination of sedimentation (for the largest part) and pathogen decay.

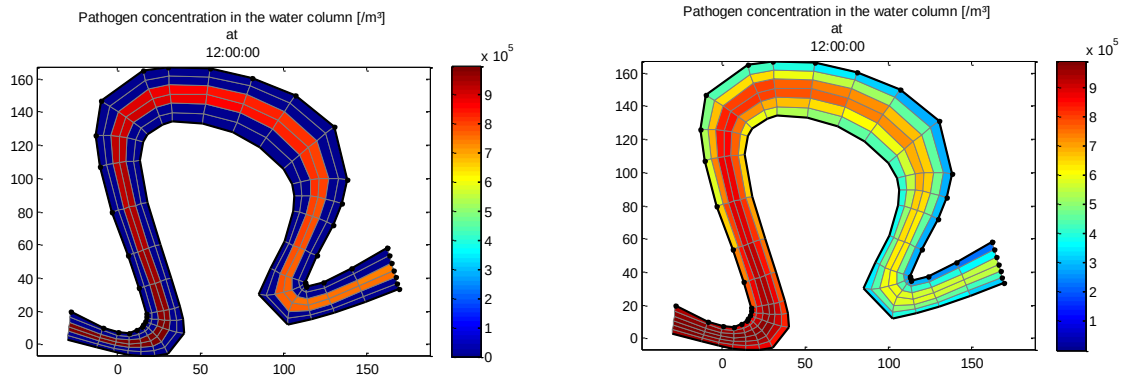

Figure 35: Plot of the pathogen concentration in the water column (both free floating and adsorbed) for the steep run (left) and natural run (right)

We can get an idea of the relative contributions to the disappearance of the pathogens by reviewing the percentage of pathogens that are free floating compared to the overall concentration in the water column, shown in Figure 36. A high percentage indicates that most of the pathogens precipitate to the sediment, as a disappearance through pathogen decay is equal for both free floating and adsorbed pathogens. The percentage at the inflow was 20%, and in the natural banks run we can see that downstream, the percentage has increased to around 40%, suggesting that about a fourth of the adsorbed pathogens have settled. The percentage along the banks is much higher, indicating more sedimentation.

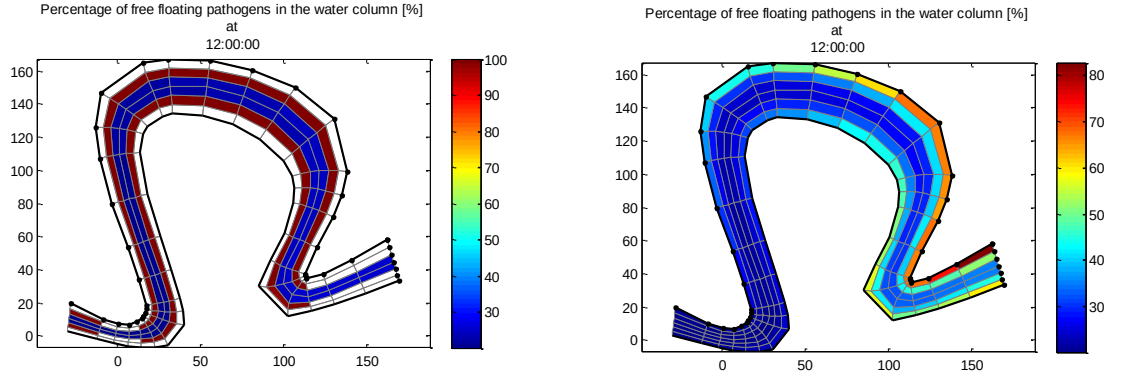

Figure 36: Plot of the percentage of adsorbed pathogens in the water column for the steep run (left) and natural run (right)

Last, we review the pathogen concentrations in several locations, shown in Figure 37. What stands out is that the pathogen concentration in the sediments is much higher than that in the water column, and also that the concentration in the stream centre is much higher than at the bank. The concentrations in the run with natural banks is lower, because they are spread out across a wider bed, and in the natural run we see that there is a larger difference in pathogen concentration between the sediments that are further downstream: (4,13) and (4,21). It seems that the location further downstream receives fewer pathogens because they have already settled out.

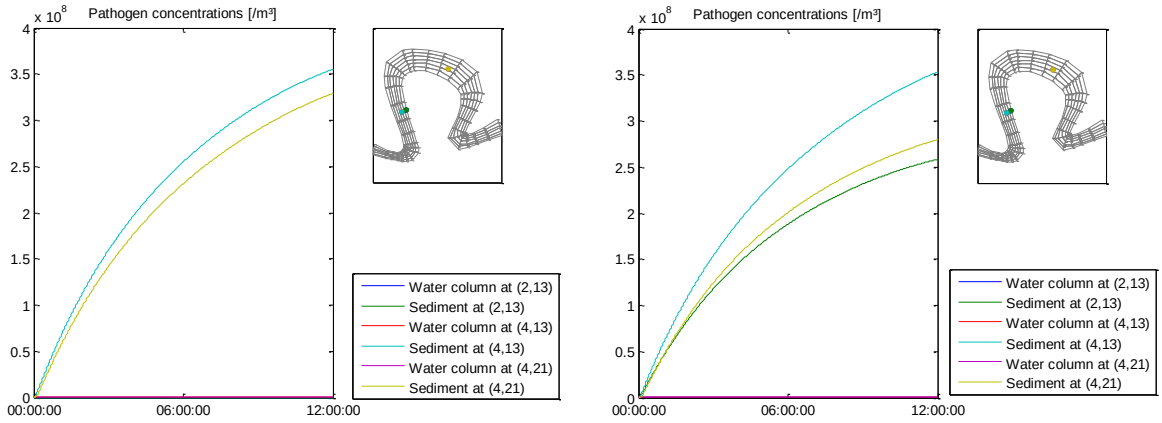

Figure 37: Plot of the pathogen concentration in several locations for the steep run (left) and natural run (right)

It seems our initial assumption that particles would settle along the banks and resuspension would take place in the main channel was partly incorrect. Although indeed the flow along the banks is reduced, the formula for resuspension also takes into account the Chézy roughness, reasoning that it is easier to initiate movement on a rough bed. When resuspension does occur in the model run, the amounts of material suspended are negligible to the material that precipitates. This can be seen in Figure 38. The sedimentation rate is constant throughout the grid and has a value of about 3.4mm/s. Resuspension occurs mostly along the banks, but even there it only reaches about 0.002mm/s. It seems it is quite difficult to get sediment to move.

The model run therefore has not produced the expected results, but nonetheless the occurrence of resuspension has helped my iron out a few bugs. The values related to the suspended particle sizes and slope could be tweaked further to increase resuspension, however I fear these values would take the model too far away from a realistic situation.

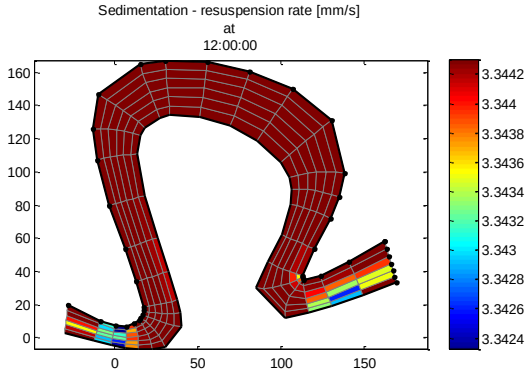

Figure 38: Plot of the sedimentation rate minus the resuspension rate for the natural run

From the model run it can be concluded that natural banks reduce the amount of pathogens in the water column by reducing the flow velocity. Most sedimentation occurs along the banks. Resuspension is not a relevant factor to consider.

The model itself withstood the test of grid curvature, although in particular the sedimentation and resuspension fluxes started oscillating in cells with a low water volume. To solve this, the time step had to be chosen smaller.

Conclusions and discussion

A formula has been proposed with which the decay of pathogens in natural water systems can be estimated. Faecal coliforms have been found to be most sensitive to the intensity of sunlight and the oxygen content in the water.

A Matlab script has been created that, using the hydrological calculations of Delft3D, allows the modelling of the pathogen concentrations in almost any water system.

With this model it has been shown that the main factor reducing the pathogen concentration in the water column is sedimentation. The main factor reducing the pathogen concentration in the sediments is the pathogen decay.

In a system where pathogens regularly enter the system, the system will strive to reach an equilibrium state in which the flux of pathogens from the water column to the sediments through sedimentation equals the pathogen decay in the sediment. As the sedimentation speed is much higher, the pathogen concentration in the sediment will be much higher than that in the water column.

If having a lot of pathogens in the sediment is not an issue, water managers can remove pathogens from the water column by encouraging sedimentation through low flow velocities or other measures.

Otherwise, the area around main inflows of pathogens can be designed in such a way that sedimentation occurs there and the pathogens are not spread through the system.

Some of assumptions the model is based on are better supported than others. This is generally caused by simplifications and gaps in the available knowledge.

For the microbiological model, the main uncertainties are:

- The relation between the decay rate and the combined effect of sunlight and oxygen.
  Shockingly few studies have been aware of the importance of the dissolved oxygen, many have not even included an approximate value in their paper.
- The decay of pathogens within the sediment has barely been studied.
  It has been assumed that the decay rate in the sediment is the same as that in the water column, except that the influence of light is assumed to be 0. However, some light may still reach the sediment, and it is also possible that the sediment protects the pathogens from other influences, such as predation
- Some known influences have not been included in the decay rate formula, including predation and the availability of nutrients.
  In particular the effect of predation is very complicated, as it is influenced by the population of bacteriophages, which may change with location and season and is likely also to be affected by the environmental variables. There simply have not been enough publications to be able to formulate a relation. These effects are combined in the constant c_7 in the formula for the overall decay rate.
- The accuracy of the pathogen constants that were calculated for faecal coliforms has not been verified.
  To obtain the pathogen constants for faecal coliforms that could not be calculated using data from the literature in which the writers have kept the environmental conditions constant except for one variable, data was used that had a known decay rate and of which also all of the environmental variables were known. These demands on known data reduced the datasets to values from only two studies. Although the fit seemed to be reasonably well for this dataset, more verification would be desirable.

For the hydrological model, the main uncertainties are:

- Again, the model could not be validated because a water system with known values for all the variables was not available.
  To properly validate the model, a case study should be done at a site, at which are known (or can be estimated):
  - The hydrological variables to set up a Delft3D model, most importantly the in- and outflow of water and dimensions and bathymetry
  - The concentration of pathogens in the water column and in the sediment, at the inflow into the system and also at several locations within the system, as well as the ratio between pathogens that are free floating and adsorbed to particles
  - The properties and range of the suspended solids the pathogens are adsorbed to
  - The values of all the environmental factors throughout the system and throughout a certain time frame
- In hydrological models it is common practice to calculate the sediment transport, however this model requires a slightly different result.
  Erosion and sedimentation is usually calculated as part of a mass balance using the gradient of the sediment transport capacity of a stream. However, this produces a sum of sedimentation and

resuspension, while the model requires both separately. After all, the resuspension flux would depend on the concentration in the sediment while the sedimentation flux depends on the concentration of adsorbed particles in the water column.

The sedimentation speed was determined with the fall velocity of a particle the pathogen is adsorbed too, which seems likely to yield correct results. However, the calculation of the resuspension is somewhat dodgy. The formulations for bottom transport by Van Rijn were used, because these are related to the Shields formula that determines the initiation of movement of sediment grains. The end result is a quantity of the bottom transport, while what is needed is an erosion speed. To deduce the erosion from the bottom transport, the fetch length to produce this bottom transport must be known, and it isn't. It was assumed to be 1meter, which would seem a short length, producing a relatively large erosion.

As the resuspension rate was found to be very low (if present at all) in the model runs that have been executed, this last assumption does not appear to be very important. However if validation shows much higher resuspension rates, the formulation should certainly be reconsidered.

The Matlab script has been made to allow relatively easy modification to the points mentioned above, as the formulations used are considered to be model inputs.

The Matlab model still has room for improvement in several elements:

- There is a difference in the timeframe of three of the processes that influence the pathogen concentration: movement with flow, sedimentation, and pathogen decay.

  The model requires a relatively small time step size to produce a stable result for the flow related movement, which reduces the simulation time length that can be calculated within a reasonable amount of time. This in turn means that effects of the sedimentation and the pathogen decay rate might not show up in the model results as they would have an effect over a larger time frame.

  This is also an issue with hydrological calculation in which the morphology is considered. This is mitigated by using the "morphological factor", which artificially increases the amount the morphology is changed so that is has a timescale that is comparable to that of the flow. Something similar could be used here, but the interpretation of the results would be much more difficult, and such implementation must be done with great care.
- As an alternative to the current calculation of the sedimentation and resuspension rates, a more traditional approach could be used, by calculating the sediment transport capacity at cell walls and using the gradient in u and v direction to determine either an erosion or sedimentation in the cell. If this is done the exchange of particles can only be in one direction, but the assumptions would be more reliable. This would require a rewrite of the sedimentation and resuspension calculation, which is a medium size job.
- This is a 2D model, and as such the concentration of pathogens has been assumed to be constant with the water depth. Usually, his is not the case. Instead it would be possible to consider there is a depth dependant gradient in the suspended particle concentration. This would require a relatively small addition to the Matlab script.
- The Delft3D model allows the modelling of flows with variable water density and temperature. The script does not consider these as values that Delft3D can produce. Implementation would require a relatively small addition to the Matlab script.
- Delft3D is capable of running 3D calculations, but the Matlab script is not. This would require a large rewrite of most of the Matlab script calculations that have to do with movement between cells and exchange with the sediment.
- The values of the Chezy roughness that are being read from the Delft3D output files are located at the cell walls, while the resuspension calculation, for which they are used, assumes they occur at the cell centres. This is currently not really an issue, because if the roughness values are not uniform they must be generated using QUICKIN, which will also put them at a slightly different location, this time at the cell corners.
- The model only considers resuspension to be caused through particles being picked up by the flow. It is also possible that sediments are resuspended in events, for example is kids play at a beach and stir up the sediments, or fish dig through the sediments looking for food, or some dredging operations are done. These sort of events could be simulated separately, which would require a relatively small addition to the Matlab script.
- The script currently does not support the inflow of pathogens in discharge points, which the Delft3D model does support. Only inflow at boundary conditions is supported. The script could be updated to read the discharge operations in the Delft3D model and request boundary conditions for them, but those must be saved differently than the boundary conditions that occur at open boundaries. This would require a medium addition to the Matlab script.
- The script could be coded as program, rather than as a part of Matlab, which should drastically increase calculation speeds. Ideally it can be written as a module into the Delft3D suite (as this is open source). If the need of the communication file could be removed this would also drastically increase

the calculation speed of the Delft3D flow calculations. This is not something I am currently capable of doing myself, as my experience is limited to scripting languages.

- Currently the Matlab script does not throw user friendly errors if there is something wrong with the input data. This is usually not a problem if the user is familiar with Matlab scripts and can understand Matlab's own default error messages. Catching potential errors and explaining the problem is not particularly difficult but would require a substantial amount of testing as many things can go wrong and everything should be caught.

Acknowledgements

Most of all I would like to thank my supervisor, Dr. ir. Van de Ven, who has been a great help throughout my research and has also been patient and kind while providing valuable feedback and inspiration. The other members of my committee, prof. dr. ir. Van de Giesen and prof. dr. Medema have been a great help as well. Lastly, Mrs Els Keizer has been kind of enough to proofread my work and motivate me when I needed it.

Thank you!

Appendices

I: Definitions

Adsorption The adhesion (joining of two different substances due to attractive forces) onto the surface of particles.

Antibiosis A biological interaction between organisms that is detrimental to at least one species. Predation is antibiosis, but not all antibiosis is predation.

Bacteria A single cell organism that reproduces by division. Some bacteria are beneficial, others are not. In this thesis, only *pathogenic* bacteria are considered, as beneficial bacteria are not a threat.

Infectivity Capability of causing disease (in humans).
Note that if a *pathogen* can be detected, it does not necessarily have to be infectious.
For example: a virus may have protein 'keys' on its shell, mimicking those of useful particles, so that a cell will mistake it for one and allow it entry. If those structures are damaged, the cell will no longer accept it and the virus is no longer infectious.

Parasite An organism that lives in another organism and uses its host for its survival, without contributing to the survival of the host.

Pathogen A micro-organism that is *infectious*. There are three groups of pathogens: *viruses*, *bacteria* and *parasites*.

Persistence The capacity of a *pathogen* to retain its *infectivity* in an environment.

Survival (..of *pathogens*)
A term often used as a synonym for *persistence* in virology, though dead and uninfectious are not technically the same.

Virus An organic structure, much smaller than a *bacteria*, that needs to hijack cells of an organism to copy its genes. Whether a virus is 'alive' or not is a subject of debate.

II: List of Equations

$$c(t) = c_0\, e^{-kt} \tag{1}$$

$$k_{overall} = k_{dark} + k_{light} + k_{sedimentation} \tag{2}$$

$$k_{dark} = k_{T20}\theta^{T-20} \quad \text{(equivalent to } k_{dark} = (k_{T20} \cdot \theta^{-20}) \cdot \theta^{T} = c\theta^{T}\text{)} \tag{3}$$

$$k_{light} = \varphi I_{avg} \tag{4}$$

$$I_{avg} = \frac{I_0}{\tau h}(1 - e^{-\tau h}) \tag{5}$$

$$k_{overall} = k_{T20}\theta^{T-20} + \varphi\frac{I_0}{\tau h}(1 - e^{-\tau h}) \tag{6}$$

$$k_{overall} = (c_1 + c_2 \cdot S) \cdot {c_3}^{T} + \left(c_4 + c_5 \cdot {c_6}^{|A-6.5|}\right) \cdot I_{avg} \cdot O + c_7 \tag{7}$$

$$k_{overall} = c_1 \cdot {c_2}^{T} + c_3 \tag{8}$$

$$k_{overall} = c_1 \cdot {c_2}^{T} \tag{9}$$

$$k_{overall} = (0.8 + 0.6\,S) \cdot 1.07^{T-20} + k_{light} \tag{10}$$

$$k_{overall} = (0.8 \cdot 1.07^{-20} + 0.6 \cdot 1.07^{-20} \cdot\ S) \cdot 1.07^{T} + k_{light} = (0.21 + 0.16 \cdot S) \cdot 1.07^{T} + k_{light} \tag{11}$$

$$k_{dark} = (c_1 + c_2 \cdot S) \cdot {c_3}^{T}$$
with, for faecal coliforms:
$[c_1 \quad c_2 \quad c_3] = [0.21 \quad 0.16 \quad 1.07]$ (12)

$$k_{overall} = c_1 \cdot I_{avg} \cdot O + c_2 \tag{13}$$

$$k_{overall} = c_1 \cdot {c_2}^{|A-6.5|} + c_3 \tag{14}$$

$$k_{light} = \left(c_4 + c_5 \cdot {c_6}^{|A-6.5|}\right) \cdot I_{avg} \cdot O$$
with, for faecal coliforms:
$[c_4 \quad c_5 \quad c_6] = [? \quad ? \quad 1.8]$ (15)

$$k_{overall} = (0.21 + 0.16 \cdot S) \cdot 1.07^{T} + \left(-0.0008 + 0.0016 \cdot 1.8^{|A-6.5|}\right) \cdot I_{avg} \cdot O + 4.4$$
$$with \quad I_{avg} = \frac{I_0}{\tau h}(1 - e^{-\tau h}) \tag{16}$$

$$w_s = \frac{\Delta \cdot g \cdot {D_{50}}^{2}}{18 \cdot \nu} \qquad for\ 1 < D_{50} \leq 100\mu m$$
$$w_s = \frac{10 \cdot \nu}{D_{50}}\left(\sqrt{1 + \frac{0.01 \cdot \Delta \cdot g \cdot {D_{50}}^{3}}{\nu^2}} - 1\right) \qquad for\ 100 < D_{50} \leq 1000\mu m \tag{17}$$
$$w_s = 1.1 \cdot \sqrt{\Delta \cdot g \cdot D_{50}} \qquad for\ D_{50} > 1000\mu m$$

$$S_{down} = \max\left(1\,; \frac{w_s \cdot \Delta t}{h}\right) \cdot c \tag{18}$$

$$s_b = \sqrt{g \cdot \Delta \cdot {D_{50}}^{3}} \cdot 0.053 \cdot \frac{T^{2.1}}{{D_*}^{0.3}} \qquad \text{for T < 3}$$
$$s_b = \sqrt{g \cdot \Delta \cdot {D_{50}}^{3}} \cdot 0.1 \cdot \frac{T^{1.5}}{{D_*}^{0.3}} \qquad \text{for T} \geq 3 \tag{19}$$
$$\text{with} \quad D_* = D_{50}\left(\frac{\Delta \cdot g}{\nu^2}\right)^{\frac{1}{3}}$$
$$T = \frac{\tau' - \tau_{cr}}{\tau_{cr}}$$

$$\tau' = \left(\frac{C}{C'}\right)^2 \cdot \tau$$
$$\tau = \frac{g}{C^2} u^2$$
$$C' = 18 \cdot log\left(\frac{12 \cdot h}{D_{90}}\right)$$

$$\begin{aligned}
\tau_{cr} &= 0.055 \cdot g \cdot D_{50} \cdot \rho_w && \text{for } D_* > 150 \\
\tau_{cr} &= 0.013 \cdot {D_*}^{0.29} \cdot g \cdot D_{50} \cdot \rho_w && \text{for } 150 \geq D_* > 20 \\
\tau_{cr} &= 0.04 \cdot {D_*}^{-0.1} \cdot g \cdot D_{50} \cdot \rho_w && \text{for } 20 \geq D_* > 10 \\
\tau_{cr} &= 0.14 \cdot {D_*}^{-0.64} \cdot g \cdot D_{50} \cdot \rho_w && \text{for } 10 \geq D_* > 4 \\
\tau_{cr} &= 0.24 \cdot {D_*}^{-1} \cdot g \cdot D_{50} \cdot \rho_w && \text{for } 4 \geq D_*
\end{aligned} \qquad (20)$$

$$S_{up} = \max\left(1 ; \frac{s_b \cdot A \cdot \Delta t}{h_s}\right) \cdot c \qquad (21)$$

III: List of symbols

Symbol	Description	Unit
Δ	The relative particle density	[-]
Δt	The time step size	[s]
A	The acidity	[pH]
A	The cell area	[m^2]
b'	The cell width perpendicular to the flow direction	[m]
c	A constant	
c	A concentration	[/l] or [$/m^3$]
c	The amount of pathogens	[-]
C	The Chézy coefficient	[$m^{0.5}/s$]
C'	The Chézy coefficient for D_{90}	[$m^{0.5}/s$]
D_*	The dimensionless particle parameter	[-]
D_{50}	The mean particle diameter	[m]
D_{90}	The particle diameter compared to which 90% of particles are smaller	[m]
g	The Earth's gravitational acceleration	[m^2/s]
h	The waterdepth	[m]
I_0	The sunlight intensity at the water surface	[W/m^2]
I_{avg}	The depth averaged sunlight intensity	[W/m^2]
k	The decay rate	[/day]
k_{dark}	The decay rate in the dark, the decay rate of a pathogen in the absence of light	[/day]
k_{light}	The partial decay rate due to the influence of light	[/day]
$k_{overall}$	The overall decay rate, which is influenced by the species of pathogen and environmental influences	[/day]
$k_{sedimentation}$	The influence of sedimentation and filtration of pathogens adsorbed to particles expressed as a decay rate	[/day]
k_{T20}	The decay rate of a pathogen in the dark at 20°C	[/day]
O	The concentration of dissolved oxygen	[mg/l]
S	The ratio of seawater (1: seawater, 0: fresh water)	[-]
s_b	The bottom sediment transport per unit of width	[m^2/s]
S_{down}	The downward flux of pathogens adsorbed to particles	[-]
S_{up}	The upward flux of pathogens adsorbed to particles	[-]
t	The time	[day] or [s]
T	The temperature	[°C]
T	The dimensionless bed shear stress parameter	[-]
u	The flow velocity	[m/s]
w_s	The falling velocity of a particle in water	[m/s]
θ	The constant that controls the influence of the temperature on k_{dark}	
ν	The kinematic viscosity	[m^2/s]
ρ_s	The density of the sediments	[kg/m^3]
ρ_w	The density of the water	[kg/m^3]
τ	The light attenuation or turbidity	[/m]
τ'	The particle related shear stress	[N/m^2]
τ_{cr}	The shear stress for initiation of movement	[N/m^2]
φ	The multiplication constant that controls the influence of light on k_{light}	

IV: How to use the model

1. Setting up

To begin using the model, download and install Delft3D. Instructions can be found online at:
http://oss.deltares.nl/web/delft3d/download

Next, make sure Matlab is installed on your system, you will be using it to run the post processing script.

Lastly, download the script files for the pathogen model at:
https://www.dropbox.com/sh/uja6my422tb3kix/MjyeHTqghH
These files can be placed anywhere on your drive. In the downloaded files you will also find example input files for the runid 'beach'.

2. Delft3D

Next, the water system you intend to model should be set up in Delft3D. I will give a very short description here, for further information, please refer to the Delft3D manual.
When running Delft3D, make sure to first set the working directory to where you want your Delft3D files to be.

First, the grid must be made with RFDGRID. This can either be a rectangular or circular grid that is generated by entering the amount of cells in n and m directions and their dimensions, or by drawing splines.
Then, the bathymetry must be entered using QUICKIN.

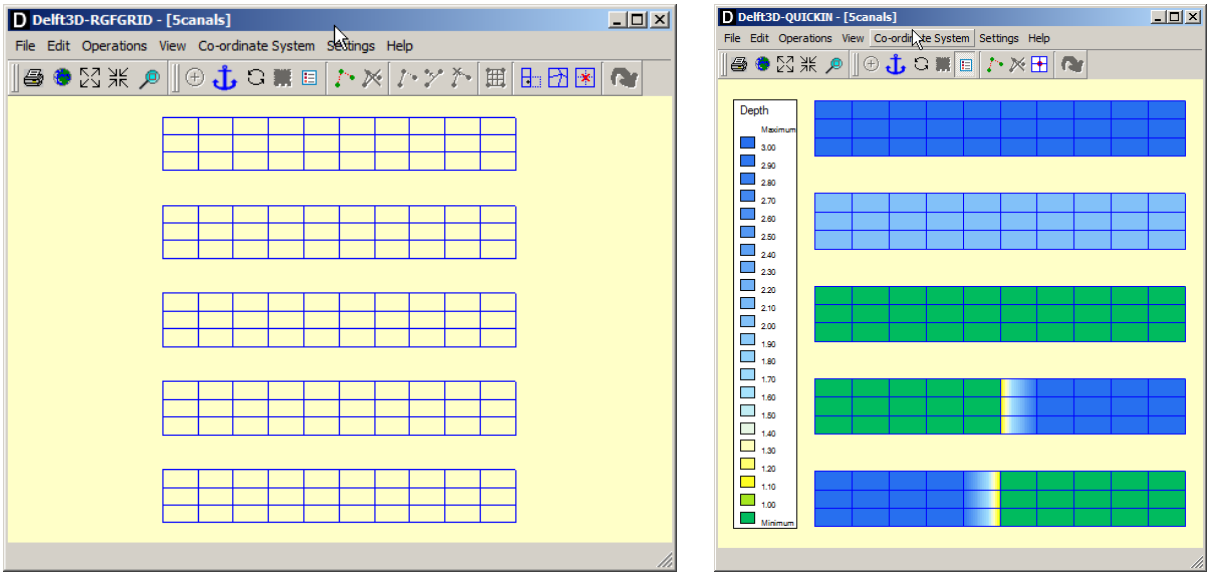

Figure 22: A grid in RGFGRID and a bathymetry in QUICKIN

Now, the MDF file, Delft3D's main input file, should be configured.
In the domain section, select the files for the grid and the bathymetry.
In the time frame section, set the desired values.
In the initial conditions section, set the desired values.
In the boundaries section, use the visualisation area to draw the open boundaries and then choose the flow conditions. It is recommended to set a discharge boundary at the upstream end of your grid, and use a water level at the downstream boundary. A reflection parameter alpha of $100s^2$ at the downstream boundary should prevent instability caused by reflection effects.
In the output section, set the model to produce the map file at at least one time, and the communication file at the time interval the Matlab script should use as a time step, usually the same as the time step for your Delft3D model. When you are running the Delft3D model calculations for the first time, you are recommended not to store a communication file, as this drastically slows down the calculation. It is better to first check the hydrological model is stable before committing to long calculation times.
When this is done, the calculations can be started.

If the Delft3D calculation returns an error, this can be seen by opening the tri-diag.*runid* file with a text editor. Warnings about convergence in UZD should be solved by avoiding small grid cells and/or choosing a smaller time step size. Generally, the Matlab script requires a slightly smaller time step size than the Delft3D model.

3. Matlab

Open Matlab and set the current folder to the folder in which the downloaded files of the pathogen model are located.

To configure the Matlab script, create a new script file in the inputfiles folder. It's easiest to open the template file and save it under a new filename, your_runid.m.
Running this file sets a number of variables that will later be used during the calculations.

First, Matlab needs to know where to find the com and map files generated by Delft3D.
There is a command here to run the script that reads the Delft3D files, before the remaining variables are set. This allows the values calculated by Delft3D to be used, as well as the time vector and such.

Then, the system variables need to be set. The values in the template file are set to their defaults, and can be changed by you if desired.
If indicated, you can use a constant value, or a vector that has the same length as one of the dimensions (time, n or m), or a matrix with 2 or 3 dimensions. The script will automatically adjust the values to the appropriate size. Any dimensions you did not specify will be kept constant.
The default values for temperature, sunlight intensity and dissolved oxygen have been set to vary with time as displayed in Figure 23.

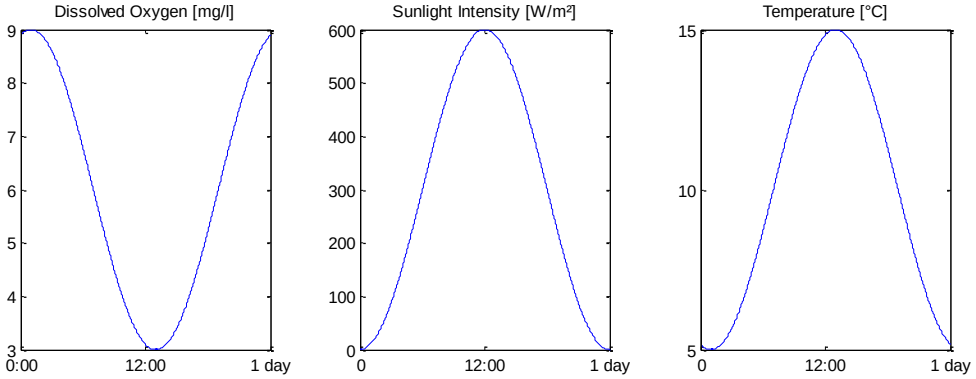

Figure 23: Time varied input for dissolved oxygen, sunlight intensity and temperature

The variables related to the particles the pathogen is adsorbed to can't change over time and space.

Next, the initial conditions need to be entered. The concentration is required for the free floating pathogens in the water column, the pathogens in the water column that are adsorbed to particles and the pathogens in the sediment.
Then, the boundary conditions are required. For each boundary the concentrations of the free floating and adsorbed pathogens in the water column must be supplied. The order of the boundary locations is the same as it was in Delft3D. During initialisation, the model also displays the boundaries it has read from the communication file and at which coordinates to occur. Note that Delft3D uses the format m,n for the coordinates, while Matlab uses n,m.
Finally, several coordinates of interesting locations at which some variables will be plotted at the end of the calculation can be provided.
Once the inputfile has been saved, the model should be initialised by typing *init_model* in the Matlab command window. The script will ask for the runid, and then read all the variables. It then shows three plots of the discharge vectors, the water depth and the velocity at the cell centres that can be used to check the script has properly imported the Delft3D data. If this is the case, *y* should be entered to continue the calculations. If this is not chosen, the calculations can be started by entering *run_model* in the command window.

After that the script will begin with the actual calculations. Every 100th time step the progress will be printed to the command window.
Once the calculations are complete, the script shows a range of plots that might be interesting, and also saves the data to an output file. All data used and calculated by the script is stored in this file and can be opened at a later time.

```
>> init_model
Please enter the runid: beach
  Clear or change the variable "runid" to reset this option.
Found 2 open boundaries
  Boundary 1  from (2,11) to (6,11)
  Boundary 2  from (9,4) to (9,4)
Model initialisation complete.
Proceed with the calculations? Enter Y to continue. y
Calculating.. Timestep 100/1201
Calculating.. Timestep 200/1201
Calculating.. Timestep 300/1201
Calculating.. Timestep 400/1201
Calculating.. Timestep 500/1201
Calculating.. Timestep 600/1201
Calculating.. Timestep 700/1201
Calculating.. Timestep 800/1201
Calculating.. Timestep 900/1201
Calculating.. Timestep 1000/1201
Calculating.. Timestep 1100/1201
Calculating.. Timestep 1200/1201
Done. Saving..
Save complete, output stored at outputfiles/beach.mat
```

Figure 39: Contents of the Matlab command window while executing the Matlab script

You can also select variables to plot yourself. The plotting library that is included in the downloaded files can be used to plot three types of graph.

The first is plotdischarge();, which can be used to plot the discharge vectors. A scroll bar is added at the bottom which can be used to navigate through the different time steps.

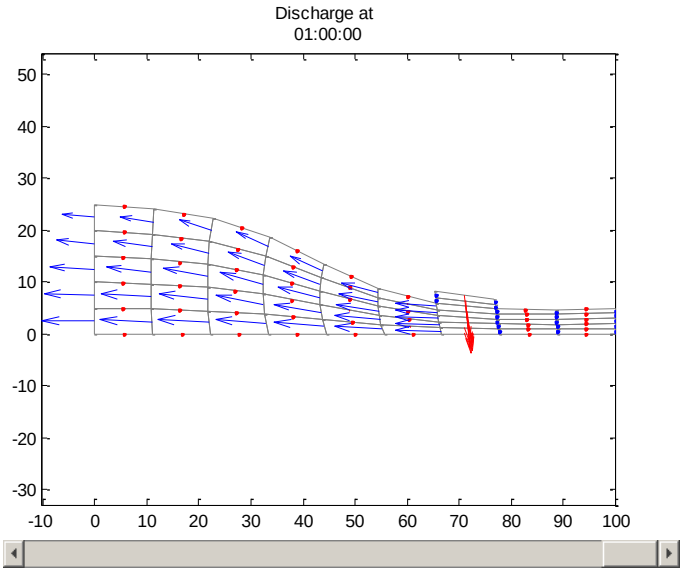

Figure 40: plotdischarge();

The second is plotscalar(*the_variable*,'The title of the graph');. This function plots any variable with dimensions of time,n,m to the grid. Again, a scrollbar is added to allow navigation through time.

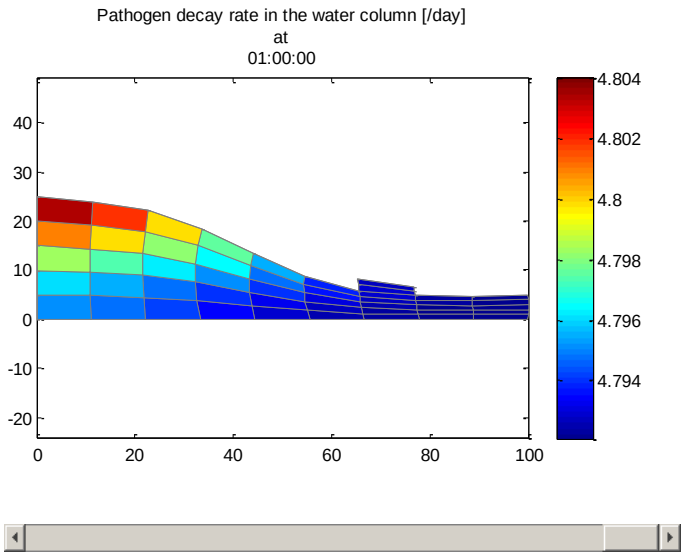

Figure 41: plotscalar(data.pathogen.water_column.decay_rate,'Pathogen decay rate in the water column [/day]');

The third is plotvaratlocation(*cell_array_of_structs_with_values_for_data_label_and_location*, The title of the graph');, meant to plot the change in time of variables, it takes input of dimension time,1,1. Each variable needs a label for the legend and a location which it represents. If the location is irrelevant, a value of [1 1] can be used, which should always return coordinates of NaN,NaN and thus will not be plotted.

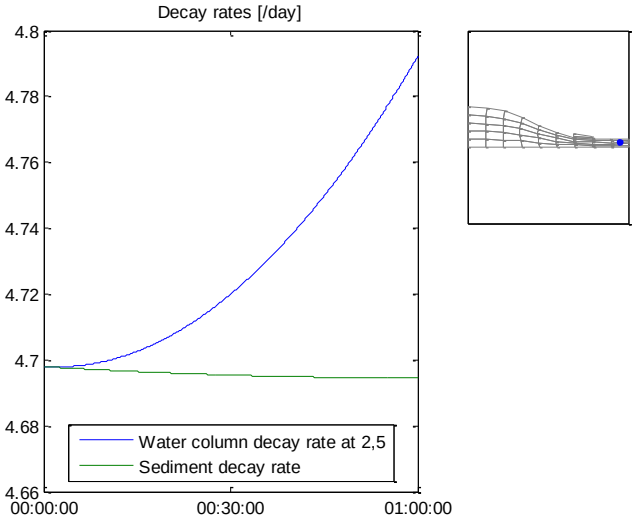

Figure 42: plotvaratlocation({ struct('data', data.pathogen.water_column.decay_rate(:,2,5), 'location', [2 5], 'label', 'Water column decay rate at 2,5'), struct('data', data.pathogen.sediment.decay_rate(:,2,5), 'location', [1 1], 'label', 'Sediment decay rate') }, 'Decay rates [/day]');

Some other plotting functions can also be used to annotate graphs. plotgrid(); will draw the grid, and plotlocation(n,m) can be used to mark a location on an already opened plot.

V: List of data used for the calculation of the pathogen constants

k_overall [/day]	Temperature [°C]	Acidity [pH]	Seawater ratio [-]	Sunlight intensity [W/m²]	Dissolved oxygen [mg/l]	Citation	notes
0.219	NaN	6.8	0	NaN	7.8	Hanes et al., 1967	pH and oxygen from table 1, k values from graphs, measurements done in the lab
0.414	NaN	7	0.33	NaN	7.2	Hanes et al., 1967	pH and oxygen from table 1, k values from graphs, measurements done in the lab
0.543	NaN	7.3	0.67	NaN	6.8	Hanes et al., 1967	pH and oxygen from table 1, k values from graphs, measurements done in the lab
1.017	NaN	7.8	1	NaN	6.5	Hanes et al., 1967	pH and oxygen from table 1, k values from graphs, measurements done in the lab
0.217	NaN	6.8	0	NaN	7.8	Hanes et al., 1967	pH and oxygen from table 1, k values from graphs, measurements done in the lab
0.274	NaN	7	0.33	NaN	7.2	Hanes et al., 1967	pH and oxygen from table 1, k values from graphs, measurements done in the lab
0.774	NaN	7.3	0.67	NaN	6.8	Hanes et al., 1967	pH and oxygen from table 1, k values from graphs, measurements done in the lab
1.332	NaN	7.8	1	NaN	6.5	Hanes et al., 1967	pH and oxygen from table 1, k values from graphs, measurements done in the lab
0.020	16.4	NaN	1	506	NaN	Solic et al., 1992	field, variables from table 1,k from table 2
0.051	21.7	NaN	1	645	NaN	Solic et al., 1992	field, variables from table 1,k from table 3
0.050	14.5	NaN	1	693	NaN	Solic et al., 1992	field, variables from table 1,k from table 4
0.055	20.7	NaN	1	725	NaN	Solic et al., 1992	field, variables from table 1,k from table 5
0.100	24.9	NaN	1	824	NaN	Solic et al., 1992	field, variables from table 1,k from table 6
0.481	14.5	NaN	1	0	NaN	Solic et al., 1992	field, fig 1 a
0.553	16.4	NaN	1	0	NaN	Solic et al., 1992	field, fig 1 a
0.921	20.7	NaN	1	0	NaN	Solic et al., 1992	field, fig 1 a
0.970	21.7	NaN	1	0	NaN	Solic et al., 1992	field, fig 1 a
1.063	24.9	NaN	1	0	NaN	Solic et al., 1992	field, fig 1 a

0.138	4	NaN	1	NaN	NaN	Solic et al., 1992	lab, fig 1 b
0.197	10	NaN	1	NaN	NaN	Solic et al., 1992	lab, fig 1 b
0.553	25	NaN	1	NaN	NaN	Solic et al., 1992	lab, fig 1 b
2.210	37	NaN	1	NaN	NaN	Solic et al., 1992	lab, fig 1 b
1.176	NaN	4.2	1	0	NaN	Solic et al., 1992	lab, fig 7
0.708	NaN	5	1	0	NaN	Solic et al., 1992	lab, fig 8
0.425	NaN	6.2	1	0	NaN	Solic et al., 1992	lab, fig 9
0.481	NaN	7.1	1	0	NaN	Solic et al., 1992	lab, fig 10
0.601	NaN	8	1	0	NaN	Solic et al., 1992	lab, fig 11
0.891	NaN	9.2	1	0	NaN	Solic et al., 1992	lab, fig 12
0.040	18	NaN	1	0	NaN	Canteras et al., 1995	lab, 8.5% salinity, table 1
0.320	18	NaN	1	40	NaN	Canteras et al., 1995	lab, 8.5% salinity, table 2
0.890	18	NaN	1	119	NaN	Canteras et al., 1995	lab, 8.5% salinity, table 3
0.910	18	NaN	1	955	NaN	Canteras et al., 1995	lab, 8.5% salinity, table 4
0.300	10	NaN	1	0	NaN	Canteras et al., 1995	lab, 8.5% salinity, table 5
0.600	26	NaN	1	0	NaN	Canteras et al., 1995	lab, 8.5% salinity, table 6
0.800	34	NaN	1	0	NaN	Canteras et al., 1995	lab, 8.5% salinity, table 7
0.120	42	NaN	1	0	NaN	Canteras et al., 1995	lab, 8.5% salinity, table 8
1.273	19	NaN	1	702	NaN	Canteras et al., 1995	field, summer, table 3
1.462	19	NaN	1	126	NaN	Canteras et al., 1995	field, summer - light intensity corrected as light intensity is not depth averaged, table 3
1.443	19	NaN	1	317	NaN	Canteras et al., 1995	field, summer - light intensity corrected as light intensity is not depth averaged, table 3
0.625	19	NaN	1	702	NaN	Canteras et al., 1995	field, summer, table 3
0.453	12	NaN	1	409	NaN	Canteras et al., 1995	field, winter, table 3
0.496	12	NaN	1	132	NaN	Canteras et al., 1995	field, winter, table 3
0.151	5	8.1	0	NaN	NaN	McFeters et al., 1972	lab, membrane chamber, figure 6
0.231	10	8.1	0	NaN	NaN	McFeters et al., 1972	lab, membrane chamber, figure 6
0.495	15	8.1	0	NaN	NaN	McFeters et al., 1972	lab, membrane chamber, figure 6
0.990	20	8.1	0	NaN	NaN	McFeters et al., 1972	lab, membrane chamber, figure 6

1.386	25	8.1	0	NaN	NaN	McFeters et al., 1972	lab, membrane chamber, figure 6
13.863	10	2.5	0	NaN	NaN	McFeters et al., 1972	lab, membrane chamber, figure 7
0.660	10	4	0	NaN	NaN	McFeters et al., 1972	lab, membrane chamber, figure 7
0.433	10	5	0	NaN	NaN	McFeters et al., 1972	lab, membrane chamber, figure 7
0.330	10	6	0	NaN	NaN	McFeters et al., 1972	lab, membrane chamber, figure 7
0.347	10	8	0	NaN	NaN	McFeters et al., 1972	lab, membrane chamber, figure 7
0.770	10	10	0	NaN	NaN	McFeters et al., 1972	lab, membrane chamber, figure 7
23.105	10	12	0	NaN	NaN	McFeters et al., 1972	lab, membrane chamber, figure 7
0.623	NaN	NaN	0	0	NaN	McCambridge et al., 1981	lab, table 2
2.280	NaN	NaN	0	48	NaN	McCambridge et al., 1981	lab, table 2
2.807	NaN	NaN	0	366	NaN	McCambridge et al., 1981	lab, table 2
0.950	4	NaN	0	NaN	NaN	Flint, 1987	lab, table 1, unfiltered, above sewage outfall
1.150	15	NaN	0	NaN	NaN	Flint, 1987	lab, table 1, unfiltered, above sewage outfall
1.610	25	NaN	0	NaN	NaN	Flint, 1987	lab, table 1, unfiltered, above sewage outfall
3.330	37	NaN	0	NaN	NaN	Flint, 1987	lab, table 1, unfiltered, above sewage outfall
0.630	4	NaN	0	NaN	NaN	Flint, 1987	lab, table 1, unfiltered, below sewage outfall
0.640	15	NaN	0	NaN	NaN	Flint, 1987	lab, table 1, unfiltered, below sewage outfall
0.860	25	NaN	0	NaN	NaN	Flint, 1987	lab, table 1, unfiltered, below sewage outfall
3.400	37	NaN	0	NaN	NaN	Flint, 1987	lab, table 1, unfiltered, below sewage outfall
54.720	NaN	NaN	NaN	650	0	Reed, 1997	lab, E coli, fig 3
69.120	NaN	NaN	NaN	650	2.52	Reed, 1997	lab, E coli, fig 3
86.400	NaN	NaN	NaN	650	4.62	Reed, 1997	lab, E coli, fig 3
115.200	NaN	NaN	NaN	650	6.3	Reed, 1997	lab, E coli, fig 3
151.200	NaN	NaN	NaN	650	8.4	Reed, 1997	lab, E coli, fig 3
0.696	20	NaN	1	NaN	NaN	Noble et al., 2004	table 3, E coli
0.504	14	NaN	1	NaN	NaN	Noble et al., 2004	table 3, E coli
0.720	20	NaN	0	NaN	NaN	Noble et al., 2004	table 4, E coli
0.504	14	NaN	0	NaN	NaN	Noble et al., 2004	table 4, E coli
3.289	NaN	NaN	1	1000	NaN	Noble et al., 2004	table 5, E coli
1.154	NaN	NaN	1	200	NaN	Noble et al., 2004	table 5, E coli

3.232	NaN	NaN	0	1000	NaN	Noble et al., 2004	table 5, E coli
1.300	NaN	NaN	0	200	NaN	Noble et al., 2004	table 5, E coli
7.728	19.6	7.45	0	0	7.2	Burton et al., 1987	sediment, plexiglass chambers, E coli, table 4
5.664	18.5	7.3	0	0	6.95	Burton et al., 1987	sediment, plexiglass chambers, E coli, table 4
4.008	18.8	7.65	0	0	7.85	Burton et al., 1987	sediment, plexiglass chambers, E coli, table 4
7.656	19.3	7.4	0	0	7.5	Burton et al., 1987	sediment, plexiglass chambers, E coli, table 4
3.024	18.5	8.05	0	0	7.85	Burton et al., 1987	sediment, plexiglass chambers, E coli, table 4
-0.259	NaN	8.9	0	0	8.8	Curtis et al., 1992	waste stabilization pond, table 2
21.816	NaN	8.9	0	295	8.5	Curtis et al., 1992	waste stabilization pond, table 2
0.075	2	NaN	0	0	NaN	Evison, 1988	lab, E coli, table 3
0.107	5	NaN	0	0	NaN	Evison, 1988	lab, E coli, table 3
0.186	10	NaN	0	0	NaN	Evison, 1988	lab, E coli, table 3
0.236	15	NaN	0	0	NaN	Evison, 1988	lab, E coli, table 3
0.512	20	NaN	0	0	NaN	Evison, 1988	lab, E coli, table 3
0.813	25	NaN	0	0	NaN	Evison, 1988	lab, E coli, table 3
0.093	2	NaN	1	0	NaN	Evison, 1988	lab, E coli, table 3
0.120	5	NaN	1	0	NaN	Evison, 1988	lab, E coli, table 3
0.134	10	NaN	1	0	NaN	Evison, 1988	lab, E coli, table 3
0.564	15	NaN	1	0	NaN	Evison, 1988	lab, E coli, table 3
0.582	20	NaN	1	0	NaN	Evison, 1988	lab, E coli, table 3
0.682	25	NaN	1	0	NaN	Evison, 1988	lab, E coli, table 3
22.105	20	7.5	0	287	15.219	Davies-Colley et al., 1999	time 5-7hrs, assumed time = 6hrs, fig 3
9.210	20	7.5	0	287	4.1895	Davies-Colley et al., 1999	time 5-7hrs, assumed time = 6hrs, fig 3
6.447	20	7.5	0	287	2.1375	Davies-Colley et al., 1999	time 5-7hrs, assumed time = 6hrs, fig 3
4.605	20	7.5	0	287	0.4104	Davies-Colley et al., 1999	time 5-7hrs, assumed time = 6hrs, fig 3
35.789	20	10	0	315	8.55	Davies-Colley et al., 1999	time 5-7hts, assumed time = 5hrs, assumed insolation = constant, fig 4
30.394	20	9.5	0	315	8.55	Davies-Colley et al., 1999	time 5-7hrs, assumed time = 6hrs, fig 4
19.342	20	9	0	315	8.55	Davies-Colley et al., 1999	time 5-7hrs, assumed time = 6hrs, fig 4
16.579	20	8	0	315	8.55	Davies-Colley et al., 1999	time 5-7hrs, assumed time = 6hrs, fig 4

11.052	20	9.5	0	273	2.6	Davies-Colley et al., 1999	time 5-7hrs, assumed time = 6hrs, fig 6
6.447	20	7.5	0	273	17	Davies-Colley et al., 1999	time 5-7hrs, assumed time = 6hrs, fig 6
1.842	20	7.5	0	273	2.4	Davies-Colley et al., 1999	time 5-7hrs, assumed time = 6hrs, fig 6

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