# Decentralised urban water reuse: The implications of system scale for cost & pathogen risk

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#### Abstract

The non-potable reuse of treated sewage in urban areas provides significant conservation of potable supplies beyond that available through water use efficiency. Effluent reuse is also an inevitable requirement in novel decentralised wastewater systems. At present, urban water reuse, where pursued, usually involves large-scale schemes based on new or existing centralised sewage treatment plants. This is despite the diseconomy of scale inherent to pipe networks that balances economies of scale in sewage treatment and negates any cost advantage for wastewater systems with more than around 1,000 connections. In light of this, the theoretical relationship between effluent reuse system scale and pathogen risks was examined at various effluent qualities. Waterborne disease was seen to be a significant factor when reusing effluent in urban areas and smaller systems were found to pose a lower risk of waterborne infection, all other things being equal. Pathogen risks were then included within an economic analysis of system scale. It was concluded that with the inclusion of pathogen risks as a costed externality, taking a decentralised approach to urban water reuse would be economically advantageous in most cases. This conclusion holds despite an exact evaluation of increased waterborne disease due to effluent reuse remaining problematic.

#### Keywords

Life cycle cost; microbial risk assessment; non-potable wastewater reuse

#### **INTRODUCTION**

Continued urban growth together with increasing requirements for the preservation and restoration of natural flows and water qualities have placed conflicting demands on urban water systems. Large-scale effluent reuse or recycling projects in urban areas with tens, or hundreds, of thousands of connections have been, and still are, promoted as the solution to these problems (Law, 1996; Thompson, 2000). Options for non-potable reuse in urban areas vary from irrigating golf courses and other open areas with conventionally treated effluent to the recycling of highly treated effluents as a secondary source of supply. All water reuse reduces wastewater disposal to aqueous environments and water recycling also conserves potable supplies. Effluent reuse will not, however, be the cheapest means of water conservation (see White& Fane, 2001). Conservation through water use efficiency does however have a limit, and this paper explores whether decentralised approaches to urban effluent reuse and recycling offer advantages over similar approaches at larger scales.

Increased consciousness of sustainable development has renewed the investigation and development of small-scale wastewater treatment. Decentralised systems are considered a less resource intensive and more ecologically benign form of sanitation (Lens *et al.*, 2001). Despite this, the historical and institutional bias against decentralised systems continues. In part this reflects a lack of understanding of the true economics of scale in relation to water systems (see Clark 1997; Booker, 1999) and an assumption of poor performance for small-scale wastewater treatment. The institutionalisation of centralised approaches is also a crucial factor. Technology has however, progressed, and small-scale treatment is now capable of reliably and consistently producing high quality effluents (Croce *et al.*, 1996).

# Urban water reuse and pathogen risk

Human pathogens, particularly viruses, can be present in high numbers in raw sewage and will continue to be present to some degree in any treated effluent, although generally below detection. Hence, the reuse of treated sewage in an urban area where exposure to the community is possible will always involve some risk of increased waterborne disease.

*Waterborne pathogens* can be broadly characterised as helminths, parasitic protozoa, bacteria or viruses. Enteric viruses are the most likely pathogens to be spread through the reuse of treated sewage because of the difficulty in physically removing them and the potential for infected individuals to excrete extreme numbers, up to  $10^{12}$  g<sup>-1</sup> faeces (Rusin *et al.* in Maier *et al.*, 2000). With exposure to poorly treated effluent, or through treatment failures, parasitic helminths, protozoa, and bacteria also become concerns.

Numerous characteristics make microbial pathogens fundamentally different from chemical toxins in terms of risks posed to human health. As living organisms, pathogens possess the potential to evolve and alter their ability to cause disease (virulence) and persistence in various environments, including wastewater treatment. This ability means our knowledge of waterborne disease and pathogen risks is never likely to be exhaustive. Pathogens multiply within affected individuals. With the exception of some pathogenic bacteria being able to grow in the environment, such as *Salmonella*, all pathogenic microbes found in sewage will have originated from an infected individual. Waterborne pathogens generally cause some form of gastroenteritis with diarrhoea and/or vomiting, but more serious diseases may also follow (such as arthritis or diabetes). Exact symptoms and severity of illness vary greatly between pathogen strains, with host factors also important. For many waterborne diseases, not all infected individuals will show disease symptoms; these asymptomatic carriers may still, however, excrete large numbers of pathogens that cause illness in others. Another characteristic of many waterborne diseases is that despite being initially contracted from water, they are contagious and spread, from person to person directly.

Infectious intestinal diseases are relatively common. Based on the Wheeler *et al.*, (1999) study in the UK, an estimated 20% of the population suffer from such illnesses each year. The proportion attributable to water was not known. In the study, 14% of all cases were caused by enteric virus with less than 1% caused by protozoa. Many of the 55% of cases where no agent could be identified are also likely to have been caused by enteric virus. Our work uses human *Rotavirus* and the pathogenic protozoan *Giardia lambia* as indicative pathogens in the modelling of increased infection due to effluent reuse. *Rotavirus* and rotaviral infection are better understood than many other enteric viruses, but may be of less importance for waterborne disease than say coxsackie viruses or hepatitis A virus (Schwartzbrod, 1995). Importantly for our work, a sound dose response relationship has been estimated (Gerba & Rose, 1993). Enteric viruses are generally much more infective than waterborne bacteria (Haas & Rose *et al.*, 1999). *Giardia* is potentially an important source of disease risk from water reuse being highly resistant to disinfection. Furthermore, immunity to both *Rotavirus* and *Giardia* does not last and reinfection can occur.

*Quantitiative microbial risk assessment* (QMRA) can be used to provide the probable number of waterborne infections in a population from estimates of pathogen exposures. Established dose response relationships are used. These dose response functions are species specific (Haas & Rose et al., 1999) having been derived from empirically based human feeding studies. Sigmoidal equations were found to best describe the relationship of the logarithm of the number of pathogens ingested to likelihood of infection (Gerba & Rose, 1993; Regli *et al.*, 1991; Haas & Rose *et al.*, 1999). Human populations are highly diverse in relation to their response to infectious microbes and a non-threshold dose response function implies that across the population as a whole there is a finite

probability of infection occurring from the ingestion of even a single pathogen, and this probability increases as more pathogens are ingested. The dose response relationship of pathogenic protozoa are described by exponential functions, and various Beta-Poisson functions have been established for species of enteric viruses and bacteria. The dose response functions for *Rotavirus* and *Giardia* used in this work are given below in equations 1 and 2 respectively (Gerba & Rose, 1993; Regli *et al.*, 1991) where D is the number of pathogens ingested.

$$P_{INFP} = 1 - e^{-\frac{D}{50.23}}$$

$$P_{INFV} = 1 - 1 + \frac{D}{5.6} \left( 2^{1/0.27} - 1 \right)^{-0.27}$$

Equation 1

#### Equation 2

*Evaluating waterborne illness*. Fatalities, 'outbreak' events and individual illnesses will all cause economic burdens to society. Evaluation via a product function approach whereby impacts are valued based on quantifiable links to actual 'markets' gives more defensible valuations than approaches such as 'willingness to pay' surveys. Illness and fatality can be linked to the employment market, with time lost to illness calculated at a figure related to the average productivity of individuals in the economy. Health care costs can also be included. This approach was used to estimate the average cost of protozoan illness in the United States (Regli *et al.*, 1999), Giardiasas being evaluated at an average case cost of US\$3,100. This figure did not include a value for lives lost to the illness. In a separate American study, Elbasha *et al.*, (2000) defined an infection outcome tree with nine possible outcomes from infection by a pathogenic strain of *E.coli*. The average present value of illness was estimated at US\$5,800, discounted at 5%. Such values for avoided waterborne intestinal illness do not, however, represent the community's willingness to pay for less disease risk or the pain and suffering, and potential for premature death from waterborne disease. Willingness to pay to avoid such risk may add considerably to economic value of illness.

#### Economies of scale in urban water systems

Economies of scale exist in both capital and operating costs of wastewater treatment. Diseconomies of scale are, however, apparent in all pipe networks (Clarke, 1997) This diseconomy is inherent and results because as the number of connections increase, the distance of pipe required per connection also increases, as does the need for larger pipes with greater volumes. The extent of this diseconomy varies with urban density and is affected by the layout of the piping network. Based on system replacement and operational costs for conventional wastewater infrastructure in Adelaide, Clark (1997) reported little difference in system life cycle cost (LCC) per household for wastewater systems with between 500 and one million connections. The decrease in average LCC of wastewater treatment being balanced by an increasing average cost of sewering. Below 500 connections treatment costs dominate and an economy of scale exists. A slight diseconomy of scale was evident above 10,000 connections. The actual optima was dependent on both household density and the discount rate applied. In an separate Australian study estimating the economics of scale for greywater recycling systems, Booker (1999) reported minimum cost per household within the range of 1,200 to 12,000 connections depending on treatment scenarios. Little difference in LCC was seen to exist in a range from 120 to 120,000 connections with capital amortised over 20 years at 5%.

# MODELLING METHODOLOGY

The theoretical relationship between waterborne disease risk and system scale was modelled for two scenarios of non-potable urban wastewater reuse. The LCC of sewering, wastewater treatment and secondary reticulation were estimated. Pathogen risk was then included a as costed externality. It was assumed that regardless of system size, a high level of reliability was possible through increased investment in maintenance and online monitoring. The two scenarios were:

*Urban irrigation*. The scenario involves conventional wastewater treatment with effluent reused on playing fields, golf courses and public parks. A relatively low level of exposure was expected.

*Urban recycling*. This scenario involves advanced levels of wastewater treatment with effluent recycled back to households via dual reticulation. As recycled effluent forms a secondary supply for all non-potable uses: garden watering; laundry; and toilet, a correspondingly high level of exposure is assumed.

# Pathogen risk and reuse system scale

Modelling of pathogen flows in reuse systems, was based on pathogens entering sewage from infected individuals. For both scenarios the factors of infection rate, pathogens shedding by infected individuals, wastewater volume generated per capita and persons per household were held constant.

*Pathogen presence in sewage.* Despite the same average pathogen inputs, pathogen concentration profiles, in sewage, will differ with system scale. This is because unlike other wastewater components, pathogens come only from infected individuals. Wastewater from systems serving small populations will therefore have higher variability in pathogen concentration and higher peak concentrations even if average numbers are the same. Pathogen numbers were modelled as point estimates based on typical shedding rates from infected individuals. *Giardia* was assumed to be shed at  $10^7 \text{ g}^{-1}$  of faeces (Rusin *et al.* in Maier *et al.*, 1999). *Rotavirus* was assumed to be shed at a rate of  $10^{10} \text{ g}^{-1}$  from infected individuals (Rusin et *al.* in Maier *et al.*, 1999; Schwartzbrod, 1995). The modelling assumed that excretion of *Rotavirus* lasted for an average of two days and for *Giardia* shedding averaged six days per infected individual. An average 200 grams of faeces per person per day was used in modelling. Wastewater generation was estimated with a simple end-use model (see White & Fane, 2001) giving an average 145 litres /capita /day (LCD).

*Microbial removal in wastewater treatment.* The urban irrigation scenario assumed a wastewater treatment chain of primary settling, biological treatment, and UV disinfection giving 4.5 log removal of enteric virus and a 2.5 log removal of pathogenic protozoan. The urban recycled scenario assumed an advanced level of wastewater treatment, including, microfiltration and UV disinfection, giving 6 log removal of enteric virus and 4 log removal of pathogenic protozoan.

*Exposure Volumes*. Exposures for each scenario are given in Table 1 below. Exposures per capita are assumptions only, based on levels that might reasonably be expected in each of the scenarios.

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Exposure Vol.	No. exposures per year		Volumes ingest	ted per year (ml)		
	Irrigation	Recycling	Irrigation	Recycling		
100 ml	0.005	0.1	0.50	10.00		
10 ml	0.025	0.5	0.25	5.00		
1 ml	0.125	2.5	0.13	2.50		
100 ul	0.625	12.5	0.06	1.25		
10 ul	30.00	625	0.03	0.63		

# Table 1: Average annual exposure to effluent per capita assumed by scenario

Total	1.0 ml	19.4 ml

# Life cycle costs, pathogen risks and system scale

For systems serving between 24 and 240,000 households, LCC of wastewater treatment was estimated based on primary sources. Household occupancy was assumed to average 2.2 persons. Small bore sewer network and secondary reticulation costs were derived from Booker (1999) based on doubling the density of dwellings used in that analysis. A septic tank effluent pump (STEP) system was assumed. All costs were discounted to present value at 5% over 50 years.

*Valuing waterborne infection.* To include the externality of waterborne disease into the LCC optimisation of urban water reuse system scale, requires both an incidence of waterborne illness and an economic value of illness. A base incidence of one person in 20,000 being infected per day without effluent reuse by both *Rotavirus* and *Giardia* was used based ,in part, on the results in the Wheeler *et al.*, (1999) study. A value of A\$2000 per infection was assumed.

# **MODELLING RESULTS**

Modelled increase in infections due to both *Rotavirus* and *Giardia* were found to vary significantly with reuse system scale. The relationship is shown in figure 1 for both pathogens and both reuse scenarios.

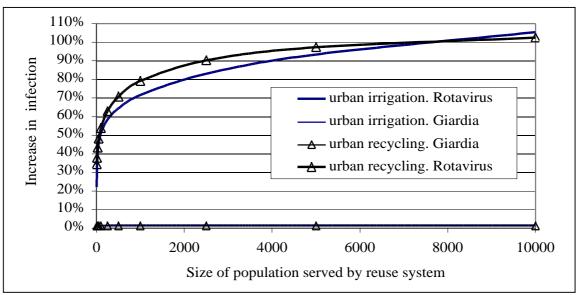


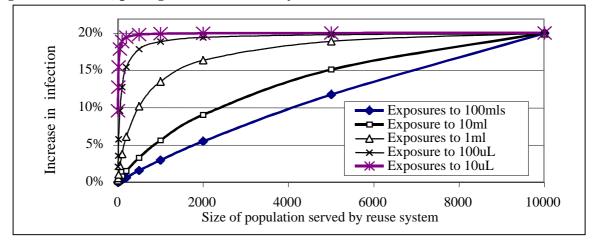
Figure 1 Urban water reuse system scale vs. increased infections

Table 2 shows the average per capita yearly volumes of effluent ingestion, which would increase rotaviral infection in the community by 20% at various exposure volumes. The results show are for the urban irrigation scenario only but were similar for the urban recycling scenario. Figure 2 illustrates these exposures in relation to reuse system scale. It can be seen that exposure volume is an important parameter in terms of the relationship between reuse system scale and pathogen risk. Risk for many small exposures, in the form of multiple aerosols ingested, is also seen to be much higher than that from a single large volume exposure, on a volume to volume basis.

# Table 2 Average per capita dose required to increase rotaviral risk by 20%

Exposure Vol.	No. exposures per year	Volume of exposure per year (ml)
100 ml	0.01	0.77
10 ml	0.02	0.22
1 ml	0.15	0.15
100 ul	1.40	0.14
10 ul	13.9	0.014

Figure 2 Dose size, pathogen risk and reuse system scale



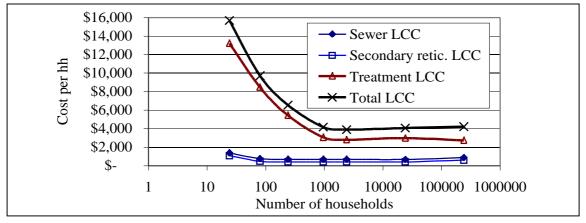


Figure 3 Life cycle costs per household for effluent reuse in the urban recycling scenario

Life cycle costs per household of sewer and wastewater reuse infrastructure for the urban recycling scenario are shown in figure 3. Based on these LCC of infrastructure but for both scenarios, figures 4 shows a total LCC of non-potable reuse against system scale that includes an estimated economic value of increased rotaviral and giardial illness.

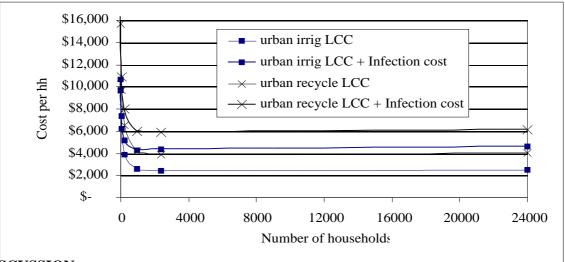


Figure 4 Life cycle cost per household for scenarios including cost of infections

# DISCUSSION

A general relationship of increasing waterborne infection risk with increasing population size served by urban wastewater reuse systems has been shown, all other factors being equal. This general relationship is evident in figure 1 for both urban reuse scenarios modelled. The impact of system scale on infection risk is particularly significant for virulent enteric pathogens such as *Rotavirus*. A similar if slighter trend is seen for the less infective parasitic protozoan *Giardia*. *Rotavirus* and similar enteric viruses, are likely to be the primary pathogens of concern when reusing sewage in urban areas, and it is significant that these are also the pathogens showing the strongest relationship between pathogen risk and system scale. The impact of scale on infection risk was somewhat dampened by increased levels of wastewater treatment ; the urban irrigation scenario showing a more pronounced scale-risk trend than the urban recycling scenario (again see figure 1).

Exposure volume was found to have a major impact on the pathogen risk - system scale relationship (see figure 2). No important scale relationship can be seen from multiply daily exposures to 10ul aerosols, but a significant relationship is observed for a low probability ingestion of 100mls of effluent. The explanation for these various relationships of exposure volume, infection risk and system scale lie in the interaction between sigmodal dose-response functions and varying pathogen concentration profiles in sewage at different scales. Populations with the same infection rates will produce different pathogen concentration profiles at different wastewater system scales. An issue does exist, in relation to the question of pathogen threshold doses required to cause infection. The current model assumes no thresholds. If thresholds do exist for significant enteric pathogens, particularly viruses, and if pathogen are evenly distributed throughout effluents then the probability of meeting a threshold level in a single dose would be higher in a small reuse system. This is because, when pathogens are present in a small system, they are like to be present at high numbers. It is however highly likely that pathogens will be found, in effluent, clumped together, and not evenly distributed. If this is the case the general pathogen risk-system scale relationship, as described, should hold true, even with a dose thresholds.

A number of further issues that could affect a general acceptance of the relationship describe were not take into account in modelling and therefore should be highlighted. Firstly, the differences in wastewater residence time between systems of differing size was not included. Some extra die-off of pathogens might be expected in a larger system with long residence times. Secondly, the potential for 'feedback' of pathogens from individuals infected due to effluent reuse back into sewage was not modelled. There is no reason to expect small systems to have a higher probability of feedback than large systems. However, if 'feedback' were to occur in a single small system, then a notable proportion of the population using that system may become infected with the same pathogen. Such an event, although not statistically significant, would be perceived as a waterborne disease 'outbreak' and would be associated with small-scale urban effluent reuse. The perception would be that decentralised effluent reuse was a high risk activity.

Life cycle costs per household for the wastewater collection, treatment and reuse systems for both scenarios were estimated. These costs are illustrated for the urban recycling scenario in figures 3. These figures are indicative only, as costs will vary greatly based on project specific factors including site, system layout, and type of sewer network (STEP or conventional). Incorporating an economic value of waterborne infection into LCC per household was seen to shift the cost optima for both urban recycling and irrigation scenarios from between 2,400 and 24,000 connections back towards around 1000 connections (see figures 4). Although slight, based only on the evaluation of increased infection from two enteric pathogens, a general conclusion can be made that with the internalisation of the pathogen risk externality, decentralised urban wastewater reuse systems will be more economic than larger and centralised wastewater reuse, all other things being equal.

# CONCLUSION

Many uncertainties exist in evaluating increased microbial illness due to urban wastewater reuse. These uncertainties include: the actual prevalence of enteric disease in the community; the likely doses that may be consumed by an exposed population; the potential of as yet unidentified pathogens to cause disease; and the proportion of infected individuals who will become ill. Another potentially significant factor not included is contagion, which would act as a multiplier for reuse system derived infections. Such uncertainties exist without addressing difficult questions around the monetarisation of human health risk ; the dollar value of human disease risk and illness will always remain contentious. Further, real moral objections can be raised about a process that seeks to place a monetary value on human fear, pain, suffering and death. Despite this, a discernible, if theoretical, economic trend in favour of small-scale decentralised systems in the order of 1000 connections has been shown if effluent is to be reused in urban areas. Alternatively, because of the economics of scale inherent to water and wastewater systems, little is gained in terms of LCC per household above 1000 connections, and a precautionary approach to pathogen risk would therefore see decentralised reuse systems favoured due the relationship of risk to scale illustrated in this paper.

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