

9-19-2005

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# **Modelling the spread of hepatitis C via commercial tattoo parlours: Implications for public health interventions**

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**Submission Date:** September 19, 2005

**Abstract:** Hepatitis C (HCV) is a serious infection caused by a blood-borne virus. It is a contagious disease spreading rapidly via a variety of transmission mechanisms including contaminated tattoo equipment. Effectively regulating commercial tattoo parlours can greatly reduce this risk. This paper models the cost-effectiveness and optimal timing of such interventions, and parameterizes the model with data for Vienna, Austria. This dynamic model of the contagious spread of HCV via tattooing and other mechanisms accounts for secondary infections and shows that regulation can be highly cost-effective.

**Keywords:** HCV epidemic, commercial tattoo parlours, cost-effectiveness, dynamic modelling, policy analysis

## Introduction

The last decade has witnessed rapid spread of hepatitis C (HCV), a highly contagious blood-borne pathogen which is characterized by substantial morbidity and mortality.<sup>1</sup> One important vector of transmission is tattooing. This paper models HCV infection via this and other means to help answer the question of whether and when commercial tattoo parlours should be regulated to reduce the spread of HCV.

Altogether, 3.1% of the world's population (170 million people) is infected with HCV.<sup>2,3</sup> Yet hepatitis C has not received attention commensurate with the magnitude of the problem, possibly because infection is usually asymptomatic for many years and less rapidly fatal than is HIV/AIDS.<sup>4</sup> Nevertheless, HCV is more virulent, contagious, and prevalent than HIV. It infects four times as many people as HIV, and is expected to kill more Americans than HIV by the year 2010.<sup>4,5</sup>

Hepatitis C can be spread in a variety of ways, including via (unscreened) blood transfusion, organ transplantation, haemodialysis, colonoscopies, use of inadequately sterilized medical, surgical or dental equipment, birth to an infected mother, injection drug use, intranasal cocaine use, piercing, tattooing (including earrings and permanent make-up), sexual contact with an infected partner, sharing razors, toothbrushes or nail-grooming equipment, and occupational exposure to blood.<sup>1,6-17</sup>

Development of HCV screening and regulation of organ transplantation and blood transfusion has been so successful that they have gone from "one of the most common" (10%) to "hardly any" (0.004% to 0.0004% per unit transfused) sources of HCV transmission in the United States.<sup>1,17</sup> A question motivating this paper is whether tattooing may be similar in this sense – a significant source of infections that are preventable via regulation.

Sexual contact is reported to be the second most common risk factor for Hepatitis C acquisition.<sup>1</sup> The risk per pairing is very low, with 0-1.8% of HCV negative individuals having sex with HCV positive partners becoming infected.<sup>16</sup> Nonetheless, because sex is a common behaviour and there are many HCV-infected individuals, the number of new infections via sexual transmission is not small.<sup>1,2</sup>

Most HCV infections in developed countries today are transmitted via injection drug use. 20-40% of intravenous drug users become infected within the first year of using needles, with this proportion reaching up to 92% for those who use longer than five years.<sup>1,2</sup> HCV-policy interventions regarding drug addicts are almost identical to the respective HIV-policy interventions, but may not be as effective because HCV is so much more virulent.<sup>18</sup> Nevertheless, since 2000, primary prevention programs, risk reduction counselling, and needle and syringe exchange programs seem to have substantially reduced infections via injection drug use.<sup>1-2,17</sup>

There are widely divergent opinions concerning the proportions of HCV infections caused by tattooing, with estimates ranging from less than 1%,<sup>19</sup> to over 3%,<sup>20</sup> and even up to more than 20%: Haley and Fischer's 1991/92 study<sup>10,11</sup> of 626 selected patients of an orthopaedic spinal clinic reports that 22% of patients with a tattoo exhibit an HCV infection (as opposed to only 3.5% of patients with no tattoos). Moreover, Haley and Fischer<sup>10</sup> report that commercially-obtained tattoos may be the source of twice as many HCV infections as is injection drug use. Other researchers argue, however, that the Haley-Fischer study misinterpreted the data and that the likelihood of acquiring an HCV infection via tattooing is rather small.<sup>6</sup> Nonetheless, there is some cause for concern that, at least in the United States, tattooing might have been grossly underestimated as a source of HCV infection.

The goal of this paper is to investigate whether and when tattoo parlours should be regulated to reduce their role in the spread of HCV. In particular, one might ask "*At what*

*point in the spread of an HCV epidemic are expensive regulatory interventions cost-effective in terms of the resulting benefits from reduced spread of HCV?” or “What is the minimum level of success for a control program to guarantee cost-effectiveness?”* Hence we present a (six-state) nonlinear dynamic model of HCV spread (and progression) that captures the effects of secondary HCV infections. The states of the model correspond to people who are uninfected and hence susceptible to infection and infected people in both the acute or chronic infection states, with all three infection states split between individuals seeking and not seeking tattoos. We parameterize our model for Vienna, Austria by building off of a pioneering study of Viennese tattoo parlours by Blaschke and Purkert<sup>21</sup> that provides unique insights into the operational practices of tattoo parlours. In 2003 the Austrian Chamber of Commerce instituted strict regulation of commercial tattoo parlours in order to control the spread of HCV,<sup>22</sup> an action that is retrospectively justified by our analysis. However, other cities and regions have not yet taken similar action. This paper may inform their deliberations concerning such actions.

Mathematical models of the spread of contagious diseases have long been used to inform public health interventions.<sup>23,24</sup> An important subset developed in response to the HIV/AIDS epidemic.<sup>25-28</sup> That work is highly relevant to HCV control because many of the transmission vectors are similar. Indeed, interesting papers have been written using essentially identical modelling methods for both diseases, but contrasting the implications that emerge because of differences in parameter values.<sup>18,29</sup> In brief, HIV has lower infectivity because that virus is less robust outside the human body, but the average cost per HIV infection is significantly higher than with HCV.

There are more differences between HCV and HIV in the course of disease progression post infection. A number of authors have developed interesting models of incidence and progression of HCV infection. Such models are important for making medical treatment decisions and for healthcare forecasting and planning purposes, but are less directly relevant here, where the focus is primarily on modelling incidence (and its prevention).

Likewise, many papers have explicitly modelled the spread of HCV via other mechanisms<sup>30-32</sup> including injection drug use,<sup>33-36</sup> sexual contact,<sup>37</sup> and blood transfusion.<sup>38</sup> This paper is, however, to the best of our knowledge, the first that models HCV transmission via tattoos.

## **A mathematical model of HCV infection including tattooing**

The discussion above suggests that governments may be contemplating regulating tattoo parlours in order to reduce the spread of HCV. Safe tattooing practices include not only simple hygiene like hand-washing and protective clothing, but also proper sterilisation techniques for reusable equipment, and using one-shot, non-reusable needles and tubes.<sup>17</sup> Thorough sterilisation is not inexpensive; as we will elaborate below, it could increase the cost per tattoo by on the order of 15%. Hence, one might expect some resistance from business interests, and quantification of the benefits vs. costs of regulation ought to enter the public debate.

Such quantification should recognize secondary infections. When people get infected by receiving a tattoo, they may subsequently infect others by having other types of HCV risky contacts. (The number of secondary infections is not simply the so-called “reproductive rate” of “the” tattoo-born epidemic because there are many HCV transmission mechanisms. An HCV-infected person who gets a tattoo participates in two interrelated epidemics, one spreads by tattooing and the other spreads by other means. Furthermore, HCV is still spreading. So,

decisions concerning tattoo parlour regulation pertain to the transient spread, not just steady state conditions.

Hence, we need a dynamic model that explicitly tracks both forms of HCV transmission in a manner that supports “what if” policy experiments to explore the effects of regulating tattoo parlours earlier or later in the spread of the HCV epidemic.

***Uncontrolled model of HCV spread***

Paradoxically, it is convenient to begin the derivation of the model equations by initially neglecting tattooing, addressing only transmission via other mechanisms (in total). Given this simplification, it is sufficient to distinguish between three states: people who are uninfected and hence susceptible to infection ( $S$ ), and infected people in both the acute ( $A$ ) and chronic ( $C$ ) infection state. Assuming, as is common in epidemiological models, that infection spreads by random mixing the state equations in the absence of tattooing would be

$$\begin{aligned} \dot{S} &= k + r_{AS}A + r_{CS}C - (\mu_S + \alpha\pi)S \\ \dot{A} &= \alpha\pi S - (r_{AC} + r_{AS} + \mu_A)A \\ \dot{C} &= r_{AC}A - (r_{CS} + \mu_C)C \end{aligned} \tag{1}$$

where

- $\pi := (A + C) / (S + A + C)$  ..... *current HCV prevalence in percent,*
- $k$  ..... *annual inflow to the population (“births”), all of whom are uninfected,*
- $r_{XY}$  ..... *annual rate of transition from state X to state Y,*
- $\mu_X$  ..... *exit rate from death or other causes from state X, and*
- $\alpha$  ..... *coefficient on random mixing’s generation of new infections.*

The coefficient on random mixing,  $\alpha$ , can be thought of as a sum over transmission mechanisms of the product of the frequency of contact between individuals and the probability that contact between an uninfected and an infected individual leads to a new infection. Those components never appear individually and cannot be estimated separately, so they are combined into a single parameter  $\alpha$ . There are many ways of intervening to reduce transmission via these routes that merit modelling and study. However, the objective here is to model the benefits of regulating tattoo parlours, so those issues are suppressed.

Considering HCV transmission via tattoo parlours requires dividing each of these three states into two groups: (a) those who visit tattoo parlours with some frequency and (b) those who do not (see Figure 1). Seeking tattoos is an activity that is usually initiated in early adolescence, at roughly the same time that the other, dominant HCV-transmitting behaviours (e.g., drug use) begin. Hence, letting  $\delta$  represent the fraction of people who will ever be interested in getting a tattoo, we split the inflow of uninfected people,  $k$ , into a stream  $\delta \cdot k$  flowing into the population of uninfected individuals who seek tattoos ( $S_t$ ) and a stream  $(1 - \delta) \cdot k$  of other uninfected people ( $S_o$ ).

\*\*\*\*\* **Figure 1 about here** \*\*\*\*\*

Transmission via means other than receiving tattoos proceeds exactly as before, but now the proportion  $\pi$  of all people who are infected involves infected people seeking tattoos ( $A_t + C_t$ ) and other infected people ( $A_o + C_o$ ), as well as the two groups of uninfected people ( $S_o$  and  $S_t$ ). Thus, prevalence is equal to

$$\pi := \frac{A_o + A_t + C_o + C_t}{S_o + S_t + A_o + A_t + C_o + C_t}. \quad (2)$$

Tattoo-induced transmission is presumed to occur at a rate that is proportional to the product of the annual rate with which susceptibles who visit tattoo parlours ( $S_t$ ) get tattoos ( $\lambda$ ), the probability that an infection results from getting a tattoo with tattoo equipment that is infectious ( $\beta$ ), and the probability that the tattoo equipment is infectious ( $\phi$ ). The last term ( $\phi$ ) is in turn the product of the probability that use of an infected tattoo recipient renders the equipment infectious ( $\theta$ ), and the probability the equipment has previously been used on someone who was HCV positive ( $f(\pi_t)$ ), where  $\pi_t$  denotes the HCV prevalence among people getting tattoos. Blaschke and Purkert's<sup>21</sup> direct observations and discussions with tattoo shop owners and equipment producers suggest that before regulation tattoo equipment was typically used five times before being replaced. Assuming the prior HCV status (before the tattoos) of successive customers is independent, this last probability can be written as

$$f(\pi_t) = \text{P}\{\text{tattooing equipment exposed to HCV}\} = \frac{1}{5} \sum_{i=0}^4 \left(1 - \frac{A_t + C_t}{S_t + A_t + C_t}\right)^i. \quad (3)$$

The final model flows reflect people “maturing out” of seeking tattoos. These are “within infection state” flows from those seeking tattoos to those no longer seeking tattoos and are presumed to be at an annual per capita rate  $g$ . Hence, the full model becomes:

$$\begin{aligned} \dot{S}_o &= (1 - \delta)k + gS_t + r_{AS}A_o + r_{CS}C_o - (\mu_S + \alpha\pi)S_o \\ \dot{A}_o &= \alpha\pi S_o + gA_t - (r_{AC} + r_{AS} + \mu_A)A_o \\ \dot{C}_o &= r_{AC}A_o + gC_t - (r_{CS} + \mu_C)C_o \\ \dot{S}_t &= \delta k + r_{AS}A_t + r_{CS}C_t - (g + \mu_S + \alpha\pi + \lambda\beta\theta f(\pi_t))S_t \\ \dot{A}_t &= (\alpha\pi + \lambda\beta\theta f(\pi_t))S_t - (g + r_{AC} + r_{AS} + \mu_A)A_t \\ \dot{C}_t &= r_{AC}A_t - (g + r_{CS} + \mu_C)C_t \end{aligned} \quad (4)$$

where

- $k$  ..... annual inflow to the population (“births”), all of whom are uninfected,
- $\delta$  ..... fraction of people who are interested in getting a tattoo,
- $g$  ..... annual rate at which people are no longer interested in getting more tattoos,
- $r_{XY}$  ..... annual rate of transition from state  $X$  to state  $Y$ ,
- $\mu_X$  ..... annual exit rate from death or other causes from state  $X$ ,
- $\alpha$  ..... annual rate of HCV infections attributed to tattooing,
- $\lambda$  ..... annual rate of getting tattoos,
- $\beta$  ..... probability an infection results from infectious equipment,
- $\theta$  ..... probability an infected tattoo recipient renders the equipment infectious,

and where  $\pi$  is defined by Eq. (2), and  $f(\pi_t)$  is determined by Eq. (3).

## Regulatory Control

One could imagine phasing in regulatory stringency. For instance, as the HCV prevalence among people receiving commercial tattoos rose, one could impose increasingly stringent rules that would in the context of this model be represented as requiring the sterilizing of

tattoo equipment for every fourth client, then every third, then every other, and only later for each client. However, practical considerations of how public health regulations are promulgated suggest adopting a simpler binary approach. Once the problem has become sufficiently severe, public health officials act to solve it, and inspecting for and enforcing compliance with a partial strategy might be more complex than with a simple policy of requiring hygienic practices for every patient. Any such (successful) sterilisation/regulation policy of tattoo parlours can be modelled as making  $\theta=0$ , but we explore implications of regulations that reduce  $\theta$  (but do not make it zero) in sensitivity analyses.

Model simulations generate two outputs: (a) the number of sterilisations  $\Sigma$  and (b) the number of HCV infections  $I$  over time. As is customary, future outcomes are discounted at a fixed annual discount rate  $r$  from the present ( $t_0$ ) to the time of regulation ( $\tau$ ) and beyond to the (possibly infinite) end of the planning horizon ( $T$ ), so:

$$\Sigma(\tau, T) = \int_{\tau}^T e^{-rt} \lambda (S_t + A_t + C_t) dt, \quad (5)$$

$$I_{\beta\theta}(t_0, \tau, T) = \int_{t_0}^{\tau} e^{-rt} (\alpha\pi S_o + (\alpha\pi + \lambda\beta\theta f(\pi_t)) S_t) dt + \int_{\tau}^T e^{-rt} (\alpha\pi S_o + \alpha\pi S_t) dt, \quad (6)$$

- $\lambda$  ..... annual rate of getting tattoos,
- $\alpha$  ..... annual rate of HCV infections attributed to tattooing,
- $\beta$  ..... probability an infection results from infected equipment,
- $\theta$  ..... probability an infected tattoo recipient renders the equipment infectious,
- $r$  ..... discount rate,
- $t_0$  ..... current time,
- $\tau$  ..... point of starting the intervention,
- $T$  ..... end of planning horizon,

where  $\pi$  and  $f(\pi_t)$  are given by Eqs. (2) and (3), respectively.

Running the model twice, with two different points ( $\tau=t_1$  and  $\tau=t_2$ ) of intervention (which includes running the model with and without control, i.e.,  $\tau=t_1=t_0$  and  $\tau=t_2=T$ ) generates an incremental cost (increase in the number of sterilisations) and an incremental effectiveness (e.g., reduction in the number of HCV infections). The ratio of these two quantities is the incremental cost-effectiveness of moving the point of intervention forward from  $t_1$  to  $t_2$ .<sup>39</sup> Given estimates of the ‘‘cost of sterilisation’’ ( $\gamma$ ) and the cost of a newly acquired HCV infection ( $\Gamma_r$ ), cost-effectiveness results can be converted into a benefit-cost ratio,

$$BC(t_1, t_2) = \frac{I_{\beta\theta}(t_0, t_2, T) - I_{\beta\theta}(t_0, t_1, T)}{\Sigma(t_1, T) - \Sigma(t_2, T)} \cdot \frac{\Gamma_r}{\gamma}, \quad (7)$$

- $\gamma$  ..... incremental cost due to regulation, including using a new needle, new ink, disposables, sterilisation of the tattooing equipment,
- $\Gamma_r$  ..... average cost of a newly acquired HCV infection,
- $t_0$  ..... beginning of planning horizon,
- $t_1, t_2$  ..... times of starting an intervention to regulate tattoo parlours, and

$T$ ..... end of planning horizon,

The optimal point in the epidemic for regulation to begin can be calculated as the time when prevalence rises to the point that delaying regulation has an adverse benefit-cost ratio.

Sensitivity analyses can show how that optimal point of intervention changes with various parameter values. We turn next to a discussion of those parameter values.

***Parameterising the model for Vienna, Austria: Population and infection-related parameters***

Most people begin receiving tattoos on average around age fifteen.<sup>20</sup> There is no clear-cut maximum age for getting a tattoo, but there is enough age-dependence that it does not make sense to lump together all adults. Although many middle-aged people get tattoos, few older people do, so we truncate the age range at 49. If there were no mortality, 15-49 year olds would exit the model (by aging) at an annual rate of  $1/35 = 0.02857$  per year. The average annual death rate for 15-49 year olds in Austria is  $0.001475$ ,<sup>40</sup> so we take  $\mu_S = 0.02857 + 0.00148$ , or  $\mu_S = 0.03$  in round numbers.

There are currently 775,000 15-49 year olds in Vienna. Official statistics project that the population of Vienna will increase by only ~6% by 2020, so for simplicity we assume the population of 15-49 year olds will be constant at 775,000. Age distribution effects stemming from the baby-boom and immigration are not critical for present purposes. If a population of 775,000 individuals is stable despite an exit rate of 3% that implies an inflow of 23,250. Tattoo shops in Vienna serve not only residents of Vienna, but also people in adjoining areas of Lower Austria and Burgenland, effectively expanding the relevant population base by 50%. Hence, we set  $k = 1.5 \cdot 23,250 = 34,875$ .

People exit the tattooing states not only by dying or turning 50 but also by no longer being interested in getting more tattoos. In this model that is parameter  $g$ . We do not have a way of estimating  $g$  directly, but we can estimate the sum of  $\mu_S$  and  $g$  indirectly from the average age of someone getting a tattoo, which is about 26 (<http://www.tattoo.dk/questionnaire/eng-cyberresults.htm> accessed 26 July 2004). To make the average age of people in a 15-49 year old age box with constant inflow and a constant per capita outflow rate equal this value, that constant per capita total annual exit rate must be  $(\mu_S + g) = 0.061$ , implying that  $g = 0.031$ . More formally, we solve

$$\sum_{i=15}^{49} i (1 - (\mu_S + g))^i = 26$$

for  $\mu_S + g$ . This combined exit rate of  $\mu_S + g = 0.061$  implies the average time someone who enters the tattooing population spends willing to get tattoos is about  $1/0.061 = 16.4$  years. Among those who get a tattoo, the average number of tattoos is 3.5 (<http://www.tattoo.dk/questionnaire/eng-cyberresults.htm> accessed 26 July 2004), suggesting the average annual rate of getting tattoos is  $\lambda = 3.5/16.4 = 0.2135$ .

Health Canada<sup>20</sup> finds that 8% of adolescents have tattoos and another 21% want to get some, so we set  $\delta = 0.29$ . Estimates from other sources, countries, and ages generally confirm that  $\delta = 0.29$  is a reasonable base value,<sup>41,42</sup> but we do sensitivity analysis for  $0.25 \leq \delta \leq 0.33$ .

Given  $\delta$ , the annual number of tattoos received by Viennese residents is  $3.5 \cdot \delta \cdot k_{Vienna} = 3.5 \cdot 0.29 \cdot 23,250 = 23,600$ . That is 65% of the 36,197 tattoos, Blaschke and Purkert<sup>21</sup>



estimate are given annually in Vienna, matching well the conventional wisdom that two-thirds of those receiving tattoos in Vienna actually living in Vienna, with the remainder predominantly from neighbouring regions. Hence, as mentioned, the population affected by regulation of Vienna's tattoo shops is 1.5 times the population of Vienna.

Transition rates from the acute to the susceptible and chronic states are set to  $r_{AS} = 0.9$  and  $r_{AC} = 1.1$  because the acute stage lasts 6 months and about 55% of those who are infected progress to the chronic state.<sup>43</sup> The transition rate from chronic to susceptible ( $r_{CS}$ ) reflects both spontaneous remission (0.002 per person per year) and treatment. According to Siebert *et al.*<sup>44</sup>, 61% of people respond to treatment over a 16-year period, suggesting an average annual rate of  $0.61/16 = 0.038125$ , for a combined rate of about  $r_{CS} = 0.04$ .

Chronic HCV can be lethal, but only over an extended period, so most HCV deaths occur after age 50. We reflect those delayed deaths in the social cost per HCV infection, but the death rate for HCV infected 15-49 year olds is not dramatically higher than it is for other 15-49 year olds. The average age of receiving a tattoo is 26 so we are interested in the cumulative HCV death rate over a period of  $50 - 26 = 24$  years, which is approximately 9.5%.<sup>40</sup> The equivalent annual death rate is  $1 - (1 - 9.5\%)^{1/24} = 0.415\%$ . Given that the background exit rate for 15-49 year-olds without HCV was  $\mu_S = 0.03$ , we set  $\mu_C = 0.03415$ . Since HCV is primarily fatal in the long run, the exit rate in the acute state is the same as in the susceptible state, i.e.,  $\mu_A = \mu_S = 0.03$ .

Parameter  $\alpha$  is difficult to estimate, but two independent, indirect calculations suggest similar values. Tattooing is not the primary source of HCV transmission, so  $\alpha$  can be estimated indirectly given a plausible steady-state prevalence. For Eq. (1) in steady state

$$\alpha = \frac{(r_{AC} + r_{AS} + \mu_A)(r_{CS} + \mu_C)}{(r_{AC} + r_{CS} + \mu_C)(1 - \hat{\pi})}.$$

The steady state prevalence  $\hat{\pi}$  is not known because the HCV epidemic has not stabilized. However, it is probably modest. Hence, given the parameter values cited above,  $\alpha$  can reasonably be modelled as being a few percent greater than the total exit rate for individuals with chronic infection ( $r_{CS} + \mu_C$ ), divided by the proportion of infected people who progress to the chronic state, or a little more than  $(r_{CS} = 0.04 + \mu_C = 0.03415)/0.55 = 0.135$ . An independent estimate can be based on the fact that in 2003 (after tattoo parlour regulation), 2,393 Austrians were diagnosed with acute HCV infection.<sup>45</sup> There are roughly twice as many acute infections as are diagnosed because only 25% are symptomatic and roughly one-third of the others are caught,<sup>46</sup> and  $0.25 + 33\% \cdot 0.75 = 0.5$ . The resulting estimate of  $2 \cdot 2,393 = 4,786$  new infections ought to match the Austrian population of 15-49 year olds (about 3.875 million), times the HCV prevalence (0.8%),<sup>45</sup> times  $\alpha$ , implying  $\alpha = 0.15$ . As a compromise we set  $\alpha = 0.14$  in the base case.

There is considerable uncertainty concerning  $\beta$ . On the one hand, literature suggests that the probability of becoming infected with HCV given one needle stick with an infectious needle is 1.8%.<sup>47</sup> On the other hand, a tattoo might be thought of as an ongoing series of pricks over 0.5-2 hours. Some Austrian HCV experts have suggested that once a tattoo is bleeding the chance of an HCV infection via an infected needle may approach 100%. There is likewise no standard figure in the literature for the probability that tattoo equipment becomes infectious when it is used on someone who is infected ( $\theta$ ). Fortunately  $\beta$  and  $\theta$  appear everywhere jointly in the model, so we treat the product of the parameters  $\beta$  and  $\theta$  as a single parameter with a base case value  $\beta \cdot \theta = 0.5$ , but explore the entire range of possible values from 0-100%.

### ***Parameterising the model for Vienna, Austria: Objective function parameters***

We use an annual discount rate of  $r = 3\%$  and consider 0% and 5% in the sensitivity analysis.<sup>39</sup> Based on Blaschke and Purkert<sup>21</sup>, Rauner *et al.*<sup>28</sup> estimate the incremental materials and other non-labour costs of sterilisation and other hygienic procedures induced by regulation would be approximately €6.11. Assuming that staff costs are about €60 per hour and sterilisation takes about 10 minutes, adds €10 in labour costs for total sterilisation costs of about €16.– per tattoo. Before regulation a typical tattoo cost about €100 so this represents an incremental cost of about 15%. We assume this is not enough of a price increase to drive significant numbers of customers to underground or unregulated tattoo sources.

For the benefit-cost analysis, we assign the present value of the expected value of all future HCV-related social and health costs to the moment of infection. We use Siebert *et al.*'s<sup>44</sup> Markov model to predict the HCV epidemic and related costs and incorporated productivity losses per life year lost.<sup>46,48</sup> The average social costs per additional infection include (a) symptomatic acute care costs of €1,814 for the 25% who are symptomatic, yielding an average of €450, (b) chronic health care costs incurred by the 55% of those infected who progress to the chronic stage, and (c) €26,310 per life year lost, reflecting productivity/consumption losses based on the average yearly Austrian gross income.<sup>48</sup> Lifetime costs are much lower for the roughly 61% of individuals exhibiting a chronic HCV infection who are successfully treated (€46,760 per case) than for those for whom treatment fails (€267,560), so the total undiscounted cost per infection is  $\Gamma_{r=0} = €450 + 0.55 \cdot (0.61 \cdot €46,760 + 0.39 \cdot €267,560) = €73,530$ . Most chronic costs occur considerably after the time of infection. Discounting back to the time of infection at a 3% (5%) annual rate reduces the total discounted costs per infection to €37,730 (€25,780). Thus, the discounted cost per infection is quite sensitive to the discount rate because those costs are distributed over 50+ years post-infection. Note that discounting calculations are done with detailed projections of the timing of future costs, but in round terms the modal chronic stage costs are about 24 years post-infection, and  $\$450 + (\$73,530 - \$450) / 1.03^{24} = \$36,400$ , which is close to the \$37,730 figure from more detailed calculations.

Total social costs are dominated by mortality. Health care costs are less one-quarter of the total, at €15,450 undiscounted, or €9,190 and €6,630 when discounting at 3% and 5%, respectively. Table 1 summarizes the parameters.

\*\*\*\*\* Table 1 about here \*\*\*\*\*

### ***Initial conditions representing Vienna before regulation***

Reflecting an overall HCV prevalence of 0.8% and the observed number of acute infections, Rauner *et al.* (2004) derive initial conditions for Vienna in 2003 of:

$$\begin{aligned} \underline{S}_o &= 989,452, & \underline{A}_o &= 539, & \underline{C}_o &= 5,435, \\ \underline{S}_t &= 162,375, & \underline{A}_t &= 409, & \underline{C}_t &= 2,907. \end{aligned} \tag{8}$$

## **Results**

Using the initial conditions for Vienna in 2003 and running the model with and without regulation suggests that regulating tattoo shops was an extremely cost-effective action. In

particular, the benefit-cost ratio of control for  $t_0 = t_1 = 2003$  and  $t_2 = T = 2063$  when discounting with the usual  $r = 3\%$  is

$$BC(2003,2063) = \frac{166,012 - 40,653}{984,245} \cdot \frac{37,730}{16.11} \cong 298.$$

Discounting at 0% and 5% the ratio becomes 844 and 157, respectively. Since health care costs are on the order of one-quarter of the total social costs, it was also cost-effective for Vienna to regulate its tattoo parlours even if no monetary value is placed on premature deaths due to HCV. In net present value terms (subtracting the incremental cost of sterilisation from the social cost savings), over the next 60 years regulation would generate a net (3%) discounted value of social benefits of approximately  $\text{€}1.518 \cdot 10^9$ .

These are, to be blunt, spectacularly high benefit-cost ratios, but one might be suspicious of projections over such a long time horizon, particularly given that the HCV epidemic has already morphed several times in terms of what are the dominant transmission vectors. For that matter, tattooing is to some extent a fad. It is entirely possible that at some point in the not too distant future, few people will even be seeking tattoos.

So we might be interested in a “semi-long term” perspective, one that focuses only on costs and infections over a shorter time horizon, but which tracks the long-term consequences of any infections created or averted within that shorter term planning horizon. With this model we can do that simply by adjusting  $T$ . Generally speaking, the shorter  $T$  is, the lower the benefit-cost ratio because some future secondary infections are ignored, but the benefit-cost ratio does not fall precipitously because shortening the horizon not only reduces benefits (credits fewer infections averted) it also reduces the costs (counts up sterilisations only through time  $T$ ). Specifically, with  $T - t_0 = 10$ ,  $BC(2003,2013) = 79$  and the present value of net social benefits is still approximately  $\text{€}0.423 \cdot 10^9$ . Reducing the planning horizon to a single Austrian legislative period ( $T - t_0 = 4$ ) still yields a cost-benefit ratio of  $BC(2003,2007) = 58$ . Even if  $T - t_0 = 1$ , the benefit-cost ratio is still highly favourable (49).

Of course the choice facing Viennese policy makers in 2003 was not so much to regulate in 2003 or never regulate, but rather to take action or not in 2003, but leaving open the possibility of enacting regulations at some subsequent time, say 2004. For regulating tattoo shops in 2003 instead of 2004, the benefit-cost ratio for  $T = 2063$  is

$$BC(2003,2004) = \frac{43,119 - 40,653}{984,245 - 949,363} \cdot \frac{37,730}{16.11} \cong 166.$$

In other words, procrastinating for a year would have saved about 35,000 sterilisations, but at the cost of about 2,500 more HCV infections. Since the social cost per HCV infection is about 200 times the cost per sterilisation, procrastinating is a bad idea. Looking at it the other way, moving regulation up from 2004 to 2003 has a benefit-cost ratio of 166.

With our base case parameters, regulating tattoo parlours is not only a cost-effective way to make a modest dent in the HCV epidemic. Now that transmission has been reduced for transfusions and other sources that were historically important, regulating tattoo parlours may make a material difference to the future course of the epidemic. In fact, regulation can not only lower the intensity of an HCV epidemic, it may also greatly reduce the endemic level of HCV prevalence.<sup>49</sup> In the long run, the HCV epidemic may, however, spread in ways quite different than what is captured in our simple model’s equations. So running the current model to steady state or over *very long* time horizons is done mostly for illustrative purposes.

As noted, there is enormous uncertainty concerning the parameter constellation  $\beta \cdot \theta$ , and there is also uncertainty concerning other parameters, notably  $\alpha$  and  $\delta$ . Does that parametric

uncertainty change our results? Holding other parameters at base case values, Table 2 shows the results for the BC ratio and for  $T_{min}$ , defined as the shortest planning horizon for which the benefit-cost ratio is larger than one when starting with initial conditions for Vienna in 2003.

The short answer is that varying these parameter values can substantially change the benefit-cost ratio of regulating tattoo parlours. However, the ratio under base case conditions was so large (298:1), that only extreme parameter variation brings that ratio down to anywhere near the critical level of 1. E.g., if  $\beta \cdot \theta > 0.0033$  regulation is always justified in terms of the cost benefit ratio (for the base case parameter values). If the overall conclusion that Viennese regulation was cost-effective is wrong, it is wrong because of structural modelling errors, not poor parameter estimates.

\*\*\*\*\* **Table 2 about here** \*\*\*\*\*

It is interesting to observe that an increase in the fraction of people, who are interested in getting a tattoo ( $\delta$ ) reduces the BC ratio and vice versa. That appears to be related to the opportunity to prevent secondary infections. More people getting tattoos means more infections averted, but it also means more expensive sterilisations. However, the larger the non-tattoo population relative to the tattoo population, the more important secondary infections are relative to primary, tattoo-induced infections. Conversely, increasing the frequency of non-tattoo transmission ( $\alpha$ ) increases the BC ratio of tattoo regulation significantly, again because more secondary infections are averted per primary, tattoo-induced infection that is averted.

So far the results have "praised" Vienna for regulating tattoo parlours in 2003 when the HCV prevalence was about 0.8%, but perhaps Viennese decision-makers should have acted even sooner when HCV prevalence was lower than 0.8%. We can investigate this by finding the smallest prevalence such that implementing regulation now is better than waiting another year. That breakeven prevalence is around 1/100 of today's value, i.e.  $\pi = 0.008\%$ . No one really knows in what year the HCV prevalence was that low, but it was probably before tattoos became as popular as they are now and almost certainly it was before the first antibody assays to identify HCV were introduced.<sup>50</sup>

Table 3 shows this breakeven prevalence for which regulation is first cost-justified depends on (1) the length of the planning horizon  $T$  and (2) whether one recognizes all HCV infections, including secondary infections, or one uses a naive model that considers only primary infections. More specifically, the naive model compares the cost of sterilisation to the product of the cost of HCV infection times the probability that someone seeking a tattoo will become infected via by receiving that tattoo.

Longer planning horizons reduce the breakeven prevalence. More dramatically, recognizing secondary injections cuts dramatically that minimum prevalence for which intervention is justified. In other words, a model such as this one that recognizes secondary infections would recommend intervening sooner than would a naive model that neglected secondary infections. How much sooner depends on the rate of change in prevalence. However, the delay in intervention from using a naive model could be considerable, as long as five years even if HCV prevalence is increasing at a compound rate of 20% per year.

\*\*\*\*\* **Table 3 about here** \*\*\*\*\*

This information is relevant for decision makers in other cities that may not yet have instituted regulations and which may be at different points in the HCV epidemics' spread.

## Conclusions

The analysis above yields three interrelated conclusions, all of which are fairly robust with respect to parameter uncertainty. (1) Regulating tattoo parlours was a cost-effective form of HCV control for Vienna in 2003 and, by extension, merits consideration in other jurisdictions. (2) It is possible to create simple, dynamic, policy simulation model that reflects both direct infections from tattoos and also secondary infections spread via additional tattoos and other, non-tattoo mechanisms. (3) The policy recommendation from such a dynamic model that incorporates secondary infections can be markedly different than that of a naïve model with regard to the stage in the epidemic at which a regulatory intervention is cost-justified. Hence, it would seem sensible for jurisdictions with both tattoo parlours and HCV to use this or some related model to inform decisions about whether and when to regulate those tattoo parlours.

The breakeven threshold prevalence for which regulation becomes cost-justified was very low when our model was parameterized for Vienna, Austria. Put another way, even with an HCV prevalence as low as 0.8%, with Viennese parameter values, regulating tattoo parlours yielded a benefit-cost ratio on the order of 300 relative to doing nothing and on the order of 150 relative to waiting another year. It is hard to imagine that benefit-cost ratios for other first-world cities with HCV prevalence on the order of 0.5 – 1.0% would not also be favourable.

The results may or may not extend to third world countries because most of the social benefit from averting HCV infections in Vienna stems from avoiding quite expensive medical interventions and from extending the lives of people with very high annual earnings. On the other hand, the majority of the cost of complying with the regulation was driven by labour rates, specifically additional time required by tattoo parlour staff to sterilize equipment. So in countries with lower wage scales, the cost per sterilisation would also be lower.

Various extensions of the model are possible, including: more detailed modelling of subpopulations to see if tattooing may serve as a “bridge” connecting what would otherwise be low- and high-risk subpopulations, crediting tattoo regulation with averting the spread of other blood-borne viruses such as HIV/AIDS, and including risk-response of tattoo customers, whereby imperfect regulation might increase the number of people who seek tattoos and, hence, who are exposed to some reduced but still positive risk of infection.

## References

1. Centers for Disease Control and Prevention (1998). Recommendations for prevention and control of hepatitis C virus (HCV) infection and HCV-related chronic disease. *MMWR Morb Mortal Wkly Rep* **47**(RR-19): 1-33.
2. Centers for Disease Control and Prevention (2004). *Hepatitis surveillance report no 59*. U.S. Department of Health and Human Services, CDC: Atlanta, GA.
3. World Health Organization (1999). Hepatitis C – global prevalence (update). *Wkly Epidemiol Rec* **74**(49): 425-427.
4. American Gastroenterological Association (2003). Stigma of hepatitis C and lack of awareness stops Americans from getting tested and treated. *AGA News Releases*, June 2003.
5. Deuffic-Burban S, Poynard T, Valleron AJ (2002). Quantification of fibrosis progression in patients with chronic hepatitis C using a markov model. *J Viral Hepat* **9**(2): 114-122.
6. Alter MJ (2002). Prevention of spread of hepatitis C. *Hepatology* **36**(5 Suppl 1): S93-S98.
7. Bronowicki JP, Venard V, Botte C, Monhoven N, Gastin I, Cone L, Hudziak H, Rhin, B, Delanoe C, LeFaou A, Bigard MA, Gaucher P (1997). Patient-to-patient transmission of hepatitis C virus during colonoscopy. *N Engl J Med* **337**(4): 237-240.
8. Conry-Cantilena C, VanRaden M, Gibble J, Melpolder J, Shakil AO, Viladomiu L, Cheung L, DiBisceglie A, Hoofnagle J, Shih JW (1996). Routes of infection, viremia and liver disease in blood donors found to have hepatitis C virus infection. *N Engl J Med* **334**(26): 1691-1696.
9. Hagan H, Thiede H, Weiss NS, Hopkins SG, Duchin JS, Alexander ER (2001). Sharing of drug preparation equipment as a risk factor for hepatitis C. *Am J Public Health* **91**(1): 42-46.
10. Haley RW, Fischer RP (2001). Commercial tattooing as a potentially important source of hepatitis C infection. Clinical epidemiology of 626 consecutive patients unaware of their hepatitis C serologic status. *Medicine* **80**(2): 134-151.
11. Haley RW, Fischer RP (2003). The tattooing paradox: are studies of acute hepatitis adequate to identify routes of transmission of subclinical hepatitis C infection? *Arch Intern Med* **163**(9): 1095-1098.
12. Ippolito G, Puro V, Petrosillo N, De Carli G, Micheloni G, Magliano E (1989). Simultaneous infection with HIV and hepatitis C virus following occupational conjunctival blood exposure. *JAMA* **280**(1): 28.
13. Pereira BJ, Milford EL, Kirkman RL, Quan S, Sayre KR, Johnson PJ, Wilber JC, Levey AS (1992). Prevalence of hepatitis C virus RNA in organ donors positive for hepatitis C antibody and in the recipients of their organs. *N Engl J Med* **327**(13): 910-915.
14. Sartori M, La Terra G, Aglietta M, Manzin A, Navino C, Verzetti G (1993). Transmission of hepatitis C via blood splash into conjunctiva. *Scand J Infect Dis* **25**(2): 270-271.
15. Terrault NA (1998). Epidemiological evidence for perinatal transmission of hepatitis C virus. *Viral Hepatitis Reviews* **4**: 245-258.
16. Terrault NA (2002). Sexual activity as a risk factor for hepatitis C. *Hepatology* **36**(5 Suppl 1): 99-105.
17. World Health Organization (1999). Global surveillance and control of hepatitis C. Report of a WHO consultation organized in collaboration with the Viral Hepatitis Prevention Board: Antwerp, Belgium. *J Viral Hepat* **6**(1): 35-47.
18. Pollack HA (2001). Cost-effectiveness of harm reduction in preventing hepatitis C among injection drug users. *Med Decis Making* **21**(5): 357-367.

19. Balasekaran R, Bulterys M, Jamal MM, Quinn PG, Johnston DE, Skipper B, Chaturvedi S, Arora S (1999). A case-control study of risk factors for sporadic hepatitis C virus infection in the southwestern United States. *Am J Gastroenterol* **94**(5): 1341-1346.
20. Health Canada (2001). *Special report on youth, piercing, tattooing and hepatitis C trends and findings*. Health Canada: Toronto.
21. Blaschke S, Purkert T (2004). *Das ökonomische Potential von Präventionsmaßnahmen im Bereich Hepatitis C: Die Rolle der Tattoo-Studios*. Master Thesis. University of Vienna: Austria.
22. Bundesministerium fuer Wirtschaft und Arbeit (2003). Ausübungsregeln für das Piercen und Tätowieren durch Kosmetik(Schönheitspflege)-Gewerbetreibende. *BGBI II (Austrian Federal Law Gazette, Part II)* **141/2003**: 647-651.
23. Layden TJ, Layden JE, Ribeiro RM, Perelson AS (2003). Mathematical modeling of viral kinetics: a tool to understand and optimize therapy. *Clin Liver Dis* **7**(1): 163-178.
24. Anderson RM, May RM (1991). *Infectious diseases of humans*. Oxford University Press: Oxford.
25. Kaplan EH, Brandeau M (eds.) (1994). *Modeling the AIDS epidemic*. Raven Press: New York.
26. Nelson PW, Perelson AS (2002). Mathematical analysis of delay differential equation models of HIV-1 infection. *Math Biosci* **179**(1): 73-94.
27. Rauner MS (2002). Using simulation for AIDS policy modeling: benefits for HIV/AIDS prevention policy makers in Vienna, Austria. *Health Care Manag Sci* **5**(2): 121-134.
28. Rauner MS, Brailsford SC, Flessa S (2004). The use of discrete-event simulation to evaluate strategies for the prevention of mother-to-child transmission of HIV in developing countries. *J Oper Res Soc* (forthcoming).
29. Deuffic-Burban S, Wong JB, Valleron AJ, Costagliola D, Delfraissy JF, Poynard T (2004). Comparing the public health burden of chronic hepatitis C and HIV infection in France. *J Hepatol* **40**(2): 319-326.
30. Pybus OG, Charleston MA, Gupta S, Rambaut A, Holmes EC, Harvey PH (2001). The epidemic behavior of the hepatitis C virus. *Science* **292**(5525): 2323-2325.
31. Salomon JA, Weinstein MC, Hammitt JK, Goldie SJ (2002). Empirically calibrated model of hepatitis C infection in the United States. *Am J Epidemiol* **156**(8): 761-773.
32. Salomon JA, Weinstein MC, Hammitt JK, Goldie SJ (2003): Cost-effectiveness of treatment for chronic hepatitis C infection in an evolving patients population. *JAMA* **290**(2): 228-237.
33. Esposito N, Rossi C (2004). A nested-epidemic model for the spread of hepatitis C among injecting drug users. *Math Biosci* **188**: 29-45.
34. Mather D, Crofts N (1999). A computer model of the spread of hepatitis C virus among injecting drug users. *Eur J Epidemiol* **15**(1): 5-10.
35. Mather D (2000). A simulation model of the spread of hepatitis C within a closed cohort. *J Oper Res Soc* **51**(6): 656-665.
36. Sheerin IG, Green FT, Sellman JD (2003). The cost of not treating hepatitis C virus infection in injecting drug users in New Zealand. *Drug Alcohol Rev* **22**(2): 159-167.
37. Struve J, Norrbohm O, Stenbeck J, Giesecke J, Weiland O (1995). Risk factors for hepatitis A, B and C virus infection among Swedish expatriates. *J Infect* **31**(3): 205-209.
38. Weusten JJ, van Drimmelen HA, Lelie PN (2003). Mathematic modeling of the risk of HBV, HCV, and HIV transmission by window-phase donations not detected by NAT. *Transfusion* **42**(5): 537-348.
39. Gold MR, Siegel JE, Russell LB, Weinstein MC (1996). *Cost-effectiveness in health and medicine*. Oxford University Press: New York.
40. Statistics Austria (2004). *Statistic Yearbook 2004*. Statistics Austria: Vienna, Austria.

41. Armstrong ML, Murphy KP (1997). Tattooing: another adolescent risk behavior warranting health education. *Appl Nurs Res* **10**(4): 181-189.
42. Armstrong ML, Roberts AE, Owen DC, Koch JR (2004). Contemporary college students and body piercing. *J Adolesc Health* **35**(1): 58-61.
43. Wiese M, Berr F, Lafrenz M, Porst H, Oesen U (2000). Low frequency of cirrhosis in a hepatitis C (genotype 1b) single-source outbreak in Germany: A 20-year multicenter study. *Hepatology* **32**(1): 91-96.
44. Siebert U, Sroczynski G, Rossol S, Wasem J, Ravens-Sieberer U, Kurth BM, Manns MP, Hutchison JG, Wong JB; German Hepatitis C Model (GEHMO) Group; International Hepatitis Interventional Therapy (IHIT) Group (2003). Cost-effectiveness of peginterferon  $\alpha$ -2b plus ribavirin versus interferon  $\alpha$ -2b plus ribavirin for initial treatment of chronic hepatitis C. *Gut* **52**(3): 425-432.
45. Jonas S, Jessner W, Rafetseder O, Wild C (2004). *Chronische Hepatitis C – Implikationen für Therapie und ökonomischen Ressourceneinsatz in Österreich*. Institute for Technology Assessment of the Austrian Academy of Sciences: Vienna.
46. Statistics Austria (2004). *Yearbook of the Health Statistic 2002*. Statistics Austria: Vienna, Austria.
47. Wasley A, Alter MJ (2000). Epidemiology of hepatitis C: geographic differences and temporal trends. *Semin Liver Dis* **20**(1): 1-16.
48. Federal Chamber of Employees (2004). *Wirtschafts- und Sozialstatistisches Taschenbuch 2004*. Federal Chamber of Employees: Vienna, Austria.
49. Rauner MS, Behrens DA, Caulkins JP (2004). An epidemic model of the spread of hepatitis C via tattoo parlors: implications for the timing of public health interventions. *ORDYS working paper 288*, Vienna University of Technology, Vienna, Austria (37 pages).
50. Kuo G, Choo QL, Alter HJ, Gitnick GL, Redeker AG, Purcell RH, Miyamura T, Dienstag JL, Alter MJ, Stevens CE, Houghton M (1989). An assay for circulating antibodies to a major etiologic virus of human non-A, non-B hepatitis. *Science* **244**: 362-364.



**Table 1: Base case parameter values**

	<b>Value</b>	<b>Description</b>	<b>Interval</b>
$k$	34,875	Annual inflow to the population (=“births”)	–
$\delta$	0.29	Fraction of people who are interested in getting a tattoo	0.25-0.33
$g$	0.031	Annual rate at which people are no longer interested in getting more tattoos	–
$r_{AS}$	0.9	Annual rate of transition from “acute” to “susceptible”	–
$r_{AC}$	1.1	Annual rate of transition from “acute” to “chronic”	–
$r_{CS}$	0.04	Annual rate of transition from “chronic” to ”acute”	–
$\mu_S$	0.03	Annual exit rate from the pool of susceptibles	–
$\mu_A$	0.03	Annual exit rate from the acute state	–
$\mu_C$	0.03415	Annual exit rate from the chronic state	–
$\lambda$	0.2135	Annual rate of getting tattoos	–
$\alpha$	0.14	Coefficient of HCV spread via methods other than tattoos	0.13-0.15
$\beta \cdot \theta$	0.5	Probability of an infection resulting from getting a tattoo with tattoo equipment that is infectious	0-1
$r$	0.03	Discount rate	0-0.05
$T$	60	Length of planning horizon (years)	1-60
$\gamma$	16.11	Cost of sterilization process (in €) including using a new needle, new ink, sterilization of the tattooing pen, etc.	–
$\Gamma_r$	37,730	Social cost of an HCV infection (in €), discounted to the time of infection for $r = 0\%$ , 3% and 5%	25,780-73,530

**Table 2: Illustration of how the specific cost-benefit ratio results BC(2003,2004) of regulate now vs. next year depend on what is assumed for the probability of an infection resulting from getting a tattoo with tattoo equipment that is infectious before regulation ( $\beta\theta$ ), on considerations reflecting HCV infection from reasons other than tattooing ( $\alpha$ ), and the length of the planning horizon  $T$ . ( $T_{min}$  is the shortest planning horizon for which intervention yields a favourable cost benefit ratio.)**

	$\alpha = 0.14$ $\delta = 0.29$		$\alpha = 0.13$ $\delta = 0.29$		$\alpha = 0.15$ $\delta = 0.29$		$\alpha = 0.14$ $\delta = 0.25$		$\alpha = 0.14$ $\delta = 0.33$	
$\beta\theta$	BC	$T_{min}$	BC	$T_{min}$	BC	$T_{min}$	BC	$T_{min}$	BC	$T_{min}$
<b>0.001</b>	0.31	–	0.27	–	0.36	–	0.31	3	0.3	–
<b>0.01</b>	3	3	3	3	4	2	3	3	3	3
<b>0.02</b>	6	1	5	1	7	1	6	1	6	1
<b>0.05</b>	15	1	13	1	18	1	15	1	15	1
<b>0.1</b>	31	1	27	1	36	1	31	1	31	1
<b>0.2</b>	63	1	55	1	73	1	63	1	63	1
<b>0.3</b>	96	1	84	1	112	1	97	1	96	1
<b>0.4</b>	130	1	113	1	152	1	131	1	130	1
<b>0.5</b>	166	1	144	1	192	1	166	1	165	1
<b>0.6</b>	202	1	175	1	235	1	203	1	201	1
<b>0.7</b>	239	1	208	1	278	1	240	1	238	1
<b>0.8</b>	278	1	241	1	323	1	279	1	277	1
<b>0.9</b>	318	1	276	1	369	1	319	1	316	1
<b>0.95</b>	338	1	294	1	393	1	339	1	337	1
<b>0.99</b>	355	1	308	1	412	1	356	1	353	1
<b>1</b>	359	1	312	1	417	1	360	1	357	1

**Table 3:** Minimum prevalence for which intervention is cost-justified as a function of planning horizon and whether secondary infections are recognized

<b>Planning horizon <math>T</math></b>	<b>Full (inclusive) Model</b>	<b>Naive Model Tracking Direct Infections Only</b>
1	0.016%	0.04%
2	0.015%	0.037%
3	0.014%	0.035%
4	0.013%	0.033%
5	0.012%	0.032%
6	0.0112%	0.03%
7	0.011%	0.028%
8	0.01%	0.027%
9	0.0099%	0.025%

Fig 1: Flow chart of the HCV model (Equation (4)) for  $\phi := \theta \cdot f(\pi_t)$  as defined by Equation (3)

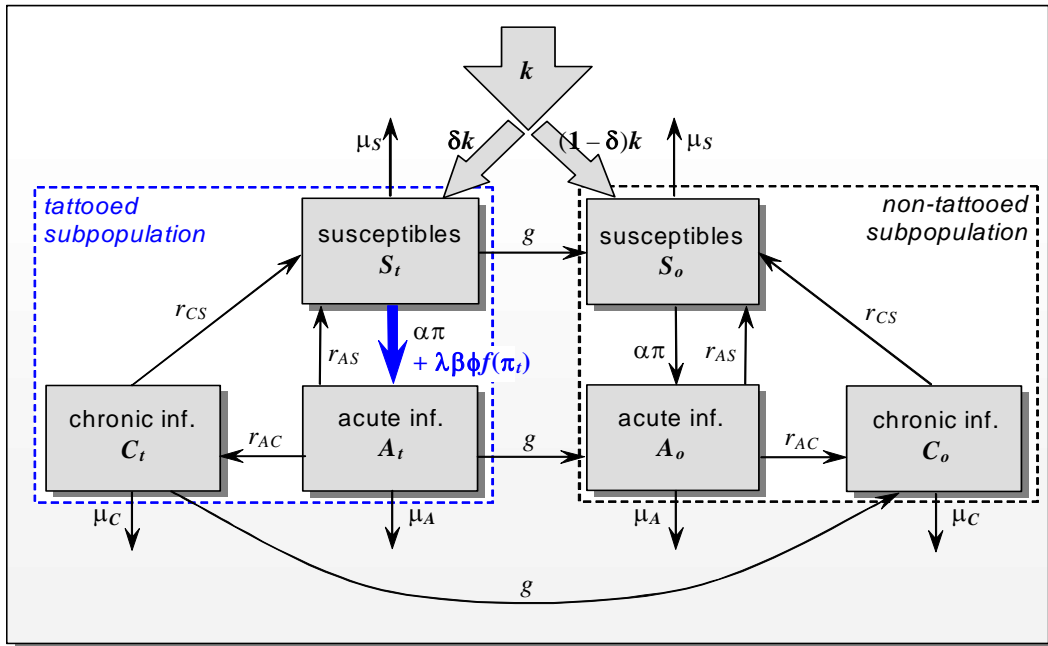


Figure 1