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The Gompertz Model for Tumour Growth
Dr. Berry

Charlene Budzinsky
6103784
April 5, 1990

Description of Problem

Most cells reproduce through mitosis whereby one parent cell produces two, virtually identical daughter cells. Since animals must at some point stop growing, "contact inhibition" [3] causes free division to stop except when necessary to repair wounds. In other words, cells usually grow to a certain degree of crowding.

However, cancer cells do not exhibit contact inhibition. DNA mutations render a cancer cell capable of uncontrolled growth and division. Cancer cells divide more often, not necessarily faster than normal cells [4]. Therefore, one cancer cell can develop into a "colony... of descendants." [3] And when this colony reaches a size of approximately 10^6 members [6], it is a detectable tumour.

Since tumour growth is an integral factor in cancer, it is important to study tumour growth in order to understand the disease and to design optimal chemotherapy and radiation schedules. Also, the study of tumour growth can lead to better understanding of normal cell division, growth and growth control.

The Gompertz model is universally used to model the growth of solid tumours and is the subject of this paper.

Overview of Gompertz Model

The Gompertz model originates as the differential equation

$$\frac{dN}{dt} = k e^{-lt} N$$

~~motivation
given later
(see p 4)~~

where

N - number of tumour cells , $N > 0$
 t - time , $t > 0$

k - 'initial' relative growth (decay) rate
 (for small t) $k \neq 0$

l - decay rate of tumour growth rate , $l > 0$

The solution yields the equation

$$N = C e^{\frac{-k}{l} t} e^{-lt}$$

where N , t , k , l are the same as above
 and as well

C - carrying capacity , $C > 0$

This is a deterministic model for population growth with a growth rate that decreases as a function of time. It is important to note that this is the model for a solid tumour whose only source of oxygen and nutrients is by diffusion. Also, the time when the tumour is first detected will be denoted as $t=0$, therefore $N(0) \geq 10^n$, this is our initial condition.

Problems with Model

1. Deterministic Model

This model has the usual disadvantages of any deterministic model.

- (i) Although time is a continuous variable, data can only be sampled at discrete time intervals. However, we do not expect to predict exactly the number of cells at any given time, but rather the population dynamics over time.
- (ii) The variable N is not continuous, it is rather an integer, so as a function of time, $N(t)$ is a step function. However, in a large population, a single birth or death represents a very small relative change in the population.
- (iii) This model treats all members in the population as average. However, since we are not trying to predict the behaviour of a single member of the population, but rather, of the population as a whole, it seems reasonable to average over the population.

2. Ethical Considerations

Since in humans, any detectable tumour is treated almost immediately, the study of tumour growth in humans is difficult. Experimentation is limited to the study of cancer cells in flasks and studies on animals. However, since medical research on animals is often considered unethical, study is again sometimes difficult. However, experiments have led to much knowledge about the kinetics of tumour growth.

Motivations for Model

In mitosis, one parent cell produces two daughter cells. If this type of division continues at a constant rate, the population will double at constant time intervals. Experimental evidence has shown that initially tumour cells appear to double in number over a constant period of time. However, as a tumour grows larger, the doubling time continuously increases. [1, 6]

This leads to, instead of modelling a constant growth rate, using one that decreases as the tumour grows larger.

Possible Reasons for Decreased Growth Rate

1. As time increases, reproducing cells mature and therefore divide more slowly. Where ageing actually depends on the number of times a cell has reproduced rather than on chronological age. ?
2. As the size of the tumour increases, there is a loss in reproducing cells. The cause may be that as a tumour grows, the supply of oxygen and nutrients to the core by diffusion becomes more difficult. Therefore, a "necrotic core" [1], or a core of non-dividing cells and debris, develops and grows rapidly as the total tumour size increases.
3. A combination of cell maturation and the necrotic core.

Assumptions

1. Large populations vary differentially with time and hence their dynamics may be approximated by differential equations.
2. No catastrophic events occur, such as treatment or death of the cancer victim.
3. All cells behave as an average cell. For example, we must assume no variation in cell maturation rates or in growth rates for a fixed cell age.
4. The only source of oxygen and nutrients for the tumour is by diffusion.
5. The concentration of oxygen and nutrients in the surrounding medium is constant.
6. We are only studying one tumour, not the population of cancer cells in the whole body.
7. A continuous model is applicable since the number of cells is so large that one birth or death has little relative effect on the population.
8. The number of cells present is positive, and is in fact, greater than 10^6 so we are dealing with a detectable tumour. In other words $N(0) \geq 10^6$ cells.
9. Measurement methods are accurate.
10. Experimental evidence applies to tumour growth in humans.

good!

Derivation of the Model

It is known that the number of cells in the tumour grows at a rate proportional to the number present

$$\therefore \frac{dN}{dt} = \gamma N$$

where N - number of tumour cells, $N > 0$
 t - time, $t > 0$
 γ - instantaneous relative growth rate, $\gamma \neq 0$

However, the growth rate slows gradually

$$\therefore \frac{d\gamma}{dt} = -l \gamma$$

where l - decay rate of $\gamma(t)$ (growth rate), $l > 0$
 leading to the system of equations

$$(1) \quad \frac{dN}{dt} = \gamma N$$

$$(2) \quad \frac{d\gamma}{dt} = -l \gamma$$

[2]

However, (2) can be solved by separation of variables

$$\frac{dY}{dt} = -\lambda Y$$

$$\frac{dY}{Y} = -\lambda dt$$

$$\ln |Y| = -\lambda t + C_1 \quad (C_1 \text{ arb. const})$$

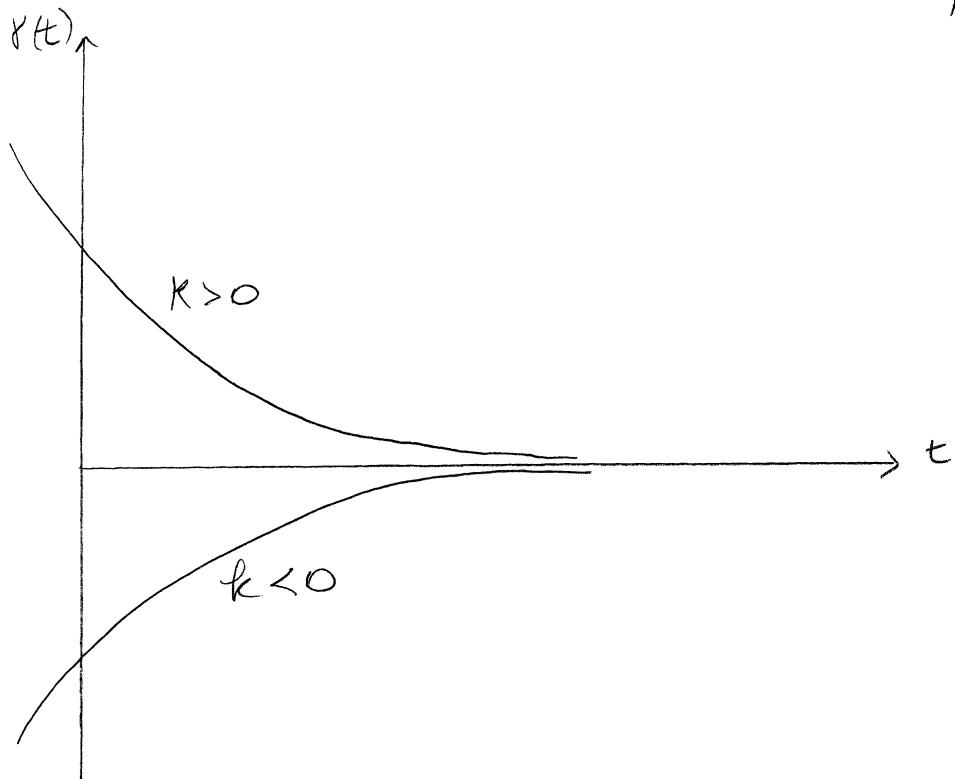
$$|Y| = e^{-\lambda t + C_1}$$

$$|Y| = K e^{-\lambda t}$$

$$Y = k e^{-\lambda t}$$

$$K = e^{C_1} > 0 \\ k \neq 0$$

$$(3) \quad \therefore Y(t) = k e^{-\lambda t} \quad k \neq 0, \lambda > 0$$



Substituting (3) into (1) yields the Gompertz model

$$\boxed{\frac{dN}{dt} = k e^{-\lambda t} N}$$

$$N > 0$$

$$t > 0$$

$$\lambda > 0$$

$$k \neq 0$$

Interpretation of k

For small t , $e^{-kt} \approx 1$, therefore the model becomes $\frac{dN}{dt} = kN$

Therefore, k can be interpreted as the 'initial' relative growth (decay) rate for small t .

It is conceivable that k is negative in the cases of lab experiments or in some real life situations.

In some lab experiments, tumours are introduced into environments not capable of maintaining them. For instance, if a large tumour is introduced into a rat, the nutrient supply may not be rich enough to support the tumour, therefore the initial relative growth rate may be negative.

In real life, if a cancer victim suddenly loses weight or reduces food intake, again the environment may be incapable of supporting a large tumour and the initial relative growth rate may be negative, using this time as the initial time $t=0$.

What does this model look like?

In this model, since the right hand side of the differential equation depends on t , we have a nonautonomous system, therefore phase plane analysis does not apply.

However, it is possible to get an idea about the solution before solving

(i) For small t , $e^{-kt} \approx 1$

$$\therefore \frac{dN}{dt} \approx kN$$

This is essentially Malthusian growth (decay) with solution

$$N(t) = \alpha e^{kt}$$

In other words, N increases exponentially as a function of time, for small t , ($k > 0$), or decreases exponentially, for small t , ($k < 0$).

(ii) For large t

As $t \rightarrow \infty$, $e^{-kt} \rightarrow 0$, so if N remains bounded, $\frac{dN}{dt} \rightarrow 0$

In other words, we expect to see dwelling off in the number of tumour cells as $t \rightarrow \infty$. Therefore, we anticipate a carrying capacity.

that the solution will exhibit a limiting value, known as

Solution of Model

$$\frac{dN}{dt} = k e^{-lt} N, \quad k \neq 0, l > 0$$

separation of variables yields

$$\frac{dN}{N} = k e^{-lt} dt$$

$$\ln |N| = -\frac{k}{l} e^{-lt} + C_2$$

(C_2 arb. const.)

$$|N| = \exp \left\{ -\frac{k}{l} e^{-lt} + C_2 \right\}$$

$$|N| = C \exp \left(-\frac{k}{l} e^{-lt} \right) \quad C = e^{C_2} > 0$$

$$N = C \exp \left(-\frac{k}{l} e^{-lt} \right) \quad \text{since } N > 0$$

∴

$$N(t) = C e^{\frac{-k}{l} e^{-lt}}$$

$C > 0, l > 0, k \neq 0$

is the final form of the Gompertz model.

Interpretation of C [C is effectively constant in graph. Can we supply physical interpretation?]

Since $\lim_{t \rightarrow \infty} C e^{\frac{-k}{l} e^{-lt}} = C$, we can

interpret C as the carrying capacity.

What does this solution look like?

There are several cases we need to address:

Case (1) $k < 0$

Case (2) $k > 0$

- (a) $\ell < k$
- (b) $\ell \geq k$

Case (1) $k < 0$

$$(i) N(t) = Ce^{-\frac{k}{\ell} e^{-\ell t}}$$

Since $k < 0$

$$-k > 0$$

$$-\frac{k}{\ell} > 0$$

$$-\frac{k}{\ell} e^{-\ell t} > 0$$

$$e^{-\frac{k}{\ell} e^{-\ell t}} > 1$$

$$N(t) = C e^{-\frac{k}{\ell} e^{-\ell t}} > C$$

$\therefore N(t) > C$ for all t

$$(ii) \frac{dN}{dt} = k N e^{-\ell t}$$

Since $k < 0, N > 0, e^{-\ell t} > 0$
it is obvious

$$\frac{dN}{dt} < 0 \text{ for all } t$$

$\therefore N(t)$ is a decreasing function of time
for all t .

$$\begin{aligned}
 \text{(iii)} \quad \frac{d^2N}{dt^2} &= \frac{d}{dt} [k N e^{-lt}] \\
 &= k e^{-lt} \frac{dN}{dt} + k N (-l) e^{-lt} \\
 &= k e^{-lt} (k N e^{-lt}) + k N (-l) e^{-lt} \\
 &= k N e^{-lt} (k e^{-lt} - l)
 \end{aligned}$$

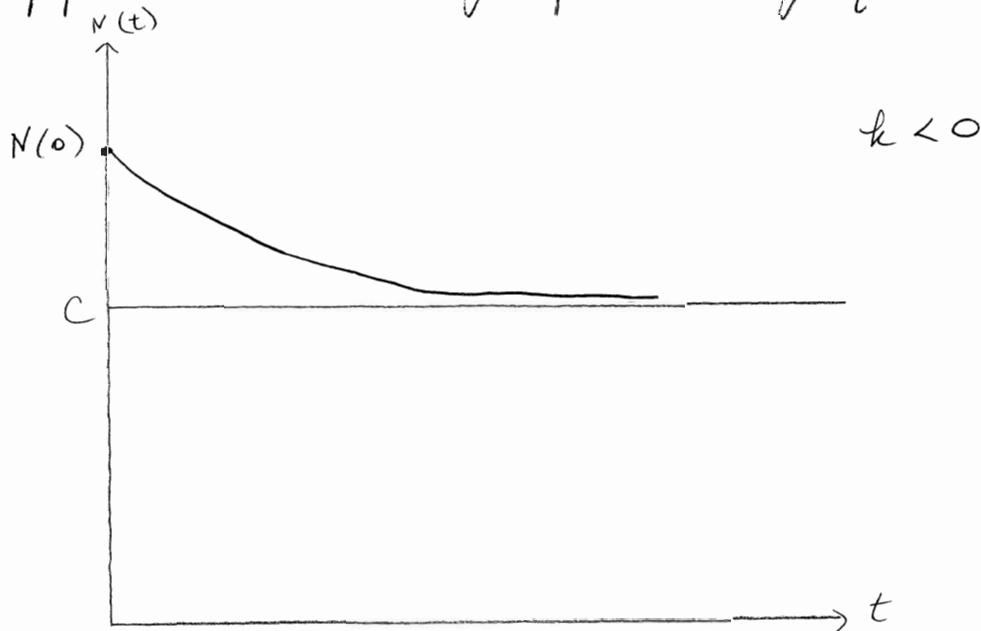
Since $k < 0$, $N > 0$, $e^{-lt} > 0$, $k e^{-lt} - l < 0$
we can see

$$\frac{d^2N}{dt^2} > 0 \text{ for all } t$$

$\therefore N(t)$ is concave up for all t

(iv) since $\lim_{t \rightarrow \infty} N(t) = C$

$N(t)$ approaches C asymptotically from above



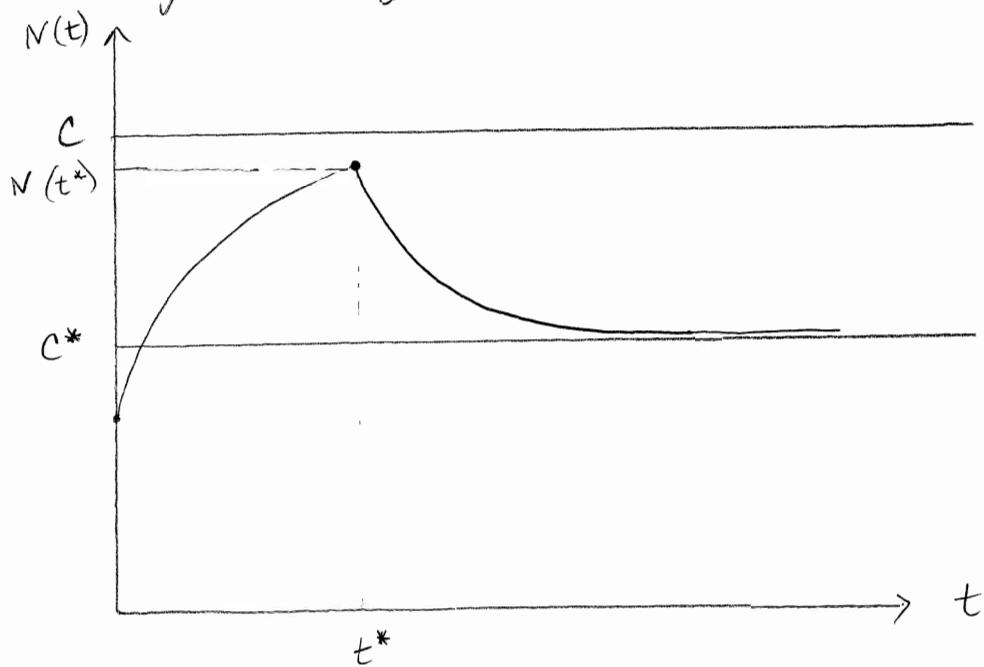
This makes sense physically since the carrying capacity may suddenly be lowered to C^* ? If the tumour is larger than C^* (where $C^* < C$), it will shrink exponentially as shown in the above diagram.

For example, say at time $t = t^*$ the cancer patient suddenly starts fasting. Therefore, nutrient supply is suddenly decreased and the carrying capacity is lowered to C^* .

\therefore The model for this case becomes

$$N(t) = \begin{cases} C e^{-\frac{k}{l} e^{-lt}} & 0 < t < t^* \\ C^* e^{-\frac{k^*}{l^*} e^{-l^*t^*}} & t > t^* \end{cases}$$

(we use k^* and l^* because these parameters will likely change also)



Case (2) $k > 0$

$$(i) N(t) = C e^{-\frac{k}{e} e^{-et}}$$

Since $k > 0$
 $-k < 0$
 $-\frac{k}{e} < 0$
 $e^{-et} < 1$

$$N(t) = C e^{-\frac{k}{e} e^{-et}} < C$$

$\therefore N(t) < C$ for all t .

$$(ii) \frac{dN}{dt} = k N e^{-et}$$

Since $k > 0, N > 0, e^{-et} > 0$, we can
 see that

$$\frac{dN}{dt} > 0 \quad \text{for all } t$$

$\therefore N(t)$ is an increasing function of time for
 all t .

(iii) Since $\lim_{t \rightarrow \infty} N(t) = C$

$\therefore N(t)$ approaches C asymptotically from below

Is there a point of inflection?

A point of inflection indicates the time of maximum tumour growth. It is important to know this time and the corresponding tumour size for the following reason.

Most tumours have the ability to develop capillaries enabling the tumour to grow almost without limit, since it is no longer limited to nutrients from diffusion, and making it easy to spread to other parts of the body through the blood. This capillary development occurs when the "tumour approaches its diffusion-limited size." [5] However, it is believed that if one could block the chemical message that induces blood vessel growth, it may be possible to maintain the tumour in its "dormant or non-malignant state." [5]

It seems reasonable that this growth of vessels begins after the growth rate starts decreasing, in other words, immediately after the inflection point occurs, since as tumour growth slows, blood vessels are sent out.

Therefore, if the tumour can be detected before the growth rate slows, it may be possible (someday) to block the chemical messages, thus preventing wild growth and the spread of cells through the body. The point of inflection also stresses the importance of early detection, since, if one can detect and treat the tumour before the growth rate slows, the prognosis is probably much better than if the tumour already has developed capillaries.

Is there a point of inflection?

$$\frac{d^2N}{dt^2} = k N e^{-lt} (ke^{-lt} - l)$$

When is $\frac{d^2N}{dt^2} = 0$? (Note: $\frac{d^2N}{dt^2} \neq \infty$)

$$\frac{d^2N}{dt^2} = k N e^{-lt} (ke^{-lt} - l) = 0$$

iff

$$k e^{-lt} - l = 0$$

since $k > 0$
 $N > 0, e^{-lt} > 0$

$$e^{-lt} = \frac{l}{k}$$

$$-lt = \ln\left(\frac{l}{k}\right)$$

$$t^* > 0 \Leftrightarrow k > l$$

$$t^* = -\frac{1}{l} \ln\left(\frac{l}{k}\right) = \frac{1}{e} \ln\left(\frac{k}{e}\right)$$

What is $N(t^*)$?

presence of pt of inflection
 depends on relative
 sizes of k & l .

$$\begin{aligned} N(t^*) &= C e^{-\frac{k}{e}} e^{-lt^*} \\ &= C e^{-\frac{k}{e}} e^{-l[-\frac{1}{e} \ln(\frac{l}{k})]} \\ &= C e^{-\frac{k}{e}} e^{\ln \frac{1}{k}} \\ &= C e^{-\frac{k}{e} (\frac{l}{k})} \\ &= C e^{-l} \end{aligned}$$

$$N(t^*) = \frac{C}{e}$$

Case 2(a) $k > 0, \ell < k$

If $\ell < k$

$$\frac{\ell}{k} < 1$$

$$\ln\left(\frac{\ell}{k}\right) < 0$$

$$t^* = -\frac{1}{\ell} \ln\left(\frac{\ell}{k}\right) > 0$$

$\therefore t^*$ is a possible inflection point.

For $\ell < k, N(0) = ce^{-k/0}$

$$\text{but } \frac{k}{\ell} > 1$$

$$-\frac{k}{\ell} < -1$$

$$e^{-\frac{k}{\ell}} < e^{-1}$$

$$ce^{-k/0} < \frac{c}{e}$$

$$\therefore 0 < N(0) < \frac{c}{e}$$

Since $\frac{dN}{dt} > 0$ for all t, N is an increasing function of t

$$\therefore 0 < t < t^* \Leftrightarrow N(0) < N < \frac{c}{e}$$

$$t^* < t < \infty \Leftrightarrow \frac{c}{e} < N < c$$

(i) For $0 < t < t^*$, $N(0) < N < \frac{c}{e}$

$$\begin{aligned}t &< t^* \\-kt &> -kt^* \\e^{-kt} &> e^{-kt^*} \\k e^{-kt} &> k e^{-kt^*} \\k e^{-kt} - l &> k e^{-kt^*} - l = 0 \\k e^{-kt} N (k e^{-kt} - l) &> 0\end{aligned}$$

$$\therefore \frac{d^2N}{dt^2} > 0 \quad \text{for } N(0) < N < \frac{c}{e}$$

(ii) For $t^* < t < \infty$, $\frac{c}{e} < N < c$

$$\begin{aligned}t &> t^* \\k e^{-kt} - l &< k e^{-kt^*} - l = 0 \\k e^{-kt} N (k e^{-kt} - l) &< 0\end{aligned}$$

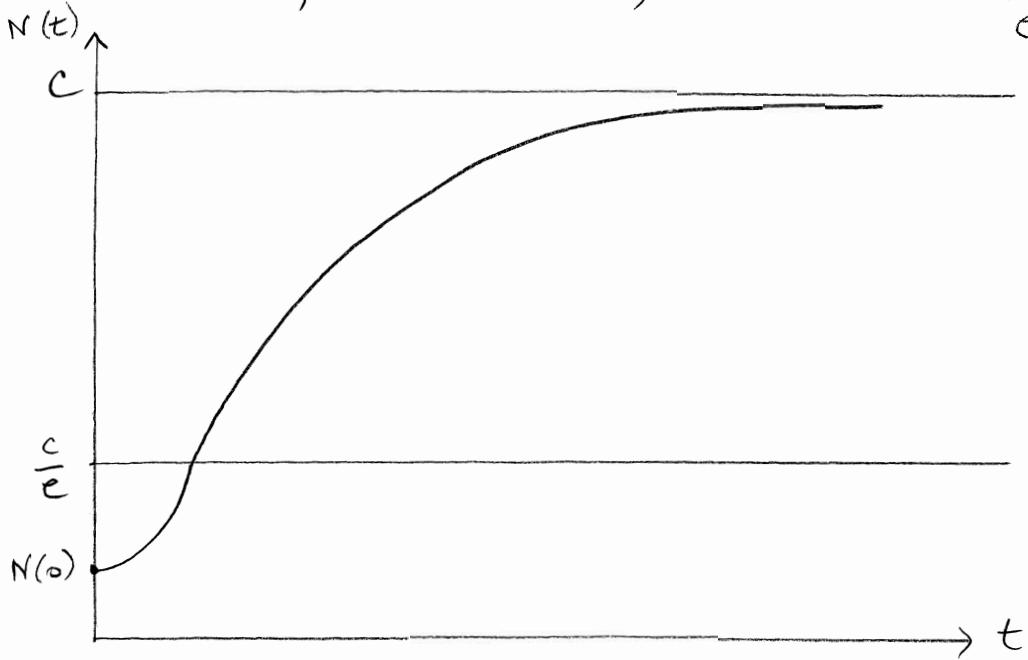
$$\therefore \frac{d^2N}{dt^2} < 0 \quad \text{for } \frac{c}{e} < N < c$$

for $k > 0$, $l < k$

	$\frac{d^2N}{dt^2}$	$\frac{dN}{dt}$	N
$\frac{d^2N}{dt^2}$	+	-	
$\frac{dN}{dt}$	+	+	
N	\nearrow	\cup	\nearrow

$\therefore N = \frac{c}{e}$ is an inflection point

For $k > c$, $d < k$, $N(0) < \frac{c}{e}$



In this case, the tumour is detected early and if treated immediately the prognosis is probably good.

Case 2(b) $k > 0, l \geq k$

If $l \geq k$

$$\frac{l}{k} \geq 1$$

$$\ln\left(\frac{l}{k}\right) \geq 0$$

$$t^* = -\frac{1}{l} \ln\left(\frac{l}{k}\right) \leq 0$$

but $t > 0$, therefore when $l \geq k$ there is no point of inflection

For $l \geq k$, $N(0) = C e^{-\frac{k}{l}}$

but $l \geq k$

$$l \geq \frac{k}{l}$$

$$l \leq -\frac{k}{l}$$

$$e^{-k/l} \geq e^{-1} = \frac{1}{e}$$

$$N(0) = C e^{-\frac{k}{l}} \geq \frac{C}{e}$$

$$\therefore \frac{C}{e} \leq N(0) < C \quad \text{for all } t$$

For $l \geq k, t > 0$

$$-lt < 0$$

$$e^{-lt} < 1$$

$$k e^{-lt} < k$$

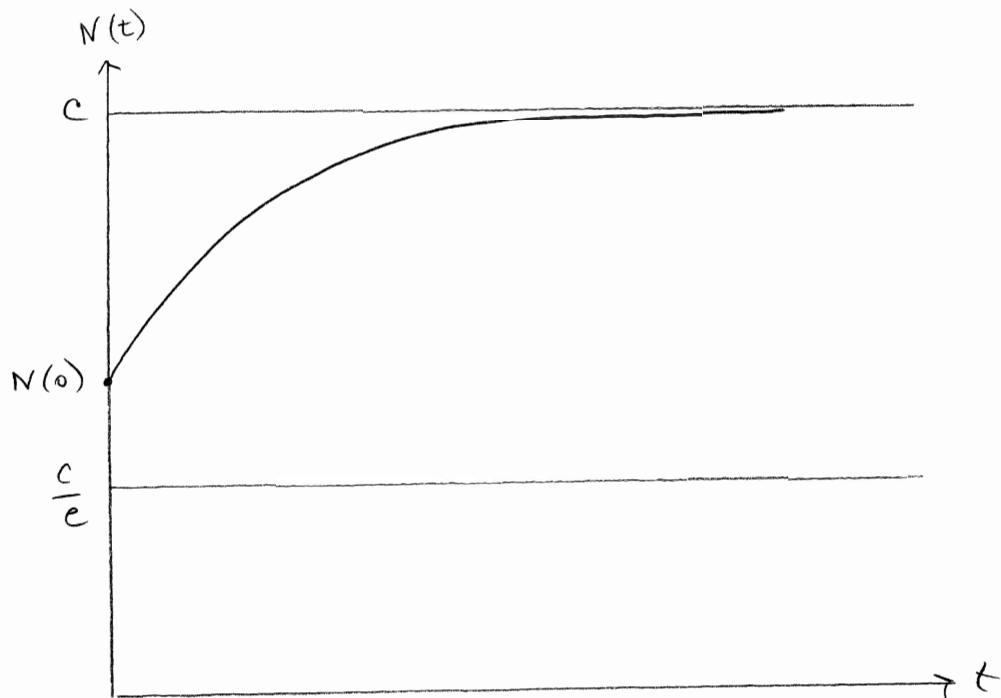
$$k e^{-lt} - l < k - l \leq 0$$

$$\therefore \frac{d^2N}{dt^2} = k e^{-lt} N [k e^{-lt} - l] \leq 0$$

for all $t > 0$

$\therefore N$ is concave down for all t .

For $k > 0$, $l \geq k$, $\frac{c}{e} \leq N(0) < c$

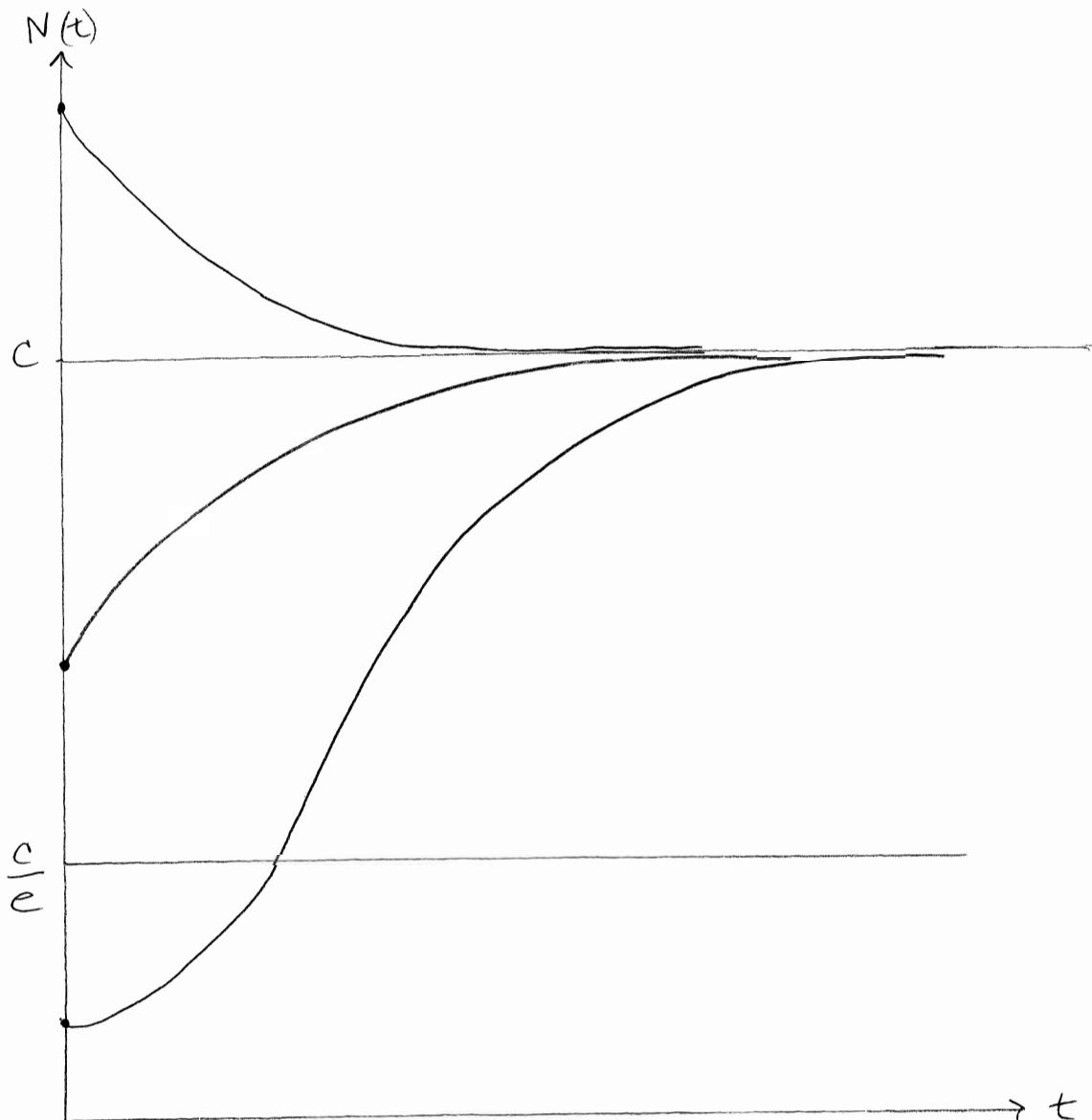


In this case, the tumour was not detected very early this may already be developing capillaries and spreading throughout the body via the blood.

The Gompertz Model

$$N(t) = C e^{-\frac{h}{k} e^{-kt}}$$

- N - number of tumour cells ; $N > 0$
- t - time ; $t > 0$
- C - carrying capacity ; $C > 0$
- h - 'initial' relative growth (decay) rate
(for small t) $h \neq 0$
- k - decay rate of tumour growth rate $k > 0$



Estimation of Model Parameters:

In estimating model parameters, it is best to use physical interpretations as much as possible. For example, if one notices a levelling off in the cell numbers or a possible inflection point (or maximum growth rate), C can be estimated. Similarly, if the initial growth rate for small t or the decay rate of the growth rate can be estimated physically, they should be, then one can solve for the remaining parameters.

Methods of Estimation

(i) Simple Mathematical Method

The simple mathematical method is not recommended since it can sometimes lead to ridiculous conclusions.

(ii) Least Squares

Either direct or indirect least squares is recommended for parameter estimation, rather than the simple mathematical method.

Direct Least Squares

Under the assumption that the given data (t_i, N_i) for $i = 1, 2, \dots, n$ may be represented by the function

$$N(t_i) = C e^{-\frac{k}{e} t_i} \quad i = 1, 2, \dots, n$$

then the function

$$S(k, l, C) = \sum_{i=1}^n [N(t_i) - N_i]^2$$

should attain a minimum value for appropriate choices of k, l, C .

Therefore, we require $\frac{\partial S}{\partial k} = \frac{\partial S}{\partial l} = \frac{\partial S}{\partial C} = 0$

or equivalently, by differentiating

$$S(k, l, C) = \sum_{i=1}^n (C e^{-\frac{k}{e} t_i} - N_i)^2$$

with respect to k, l, C we get the system

$$\frac{\partial S}{\partial k} = C \sum_{i=1}^n e^{-\frac{2k}{e} t_i} - \sum_{i=1}^n N_i e^{-\frac{k}{e} t_i} = 0$$

$$\frac{\partial S}{\partial l} = \sum_{i=1}^n \left[C e^{-\frac{k}{e} t_i} - N_i \right] \left[\frac{-k}{e} e^{-\frac{k}{e} t_i} - t_i \right] \left[\frac{1}{l} + t_i \right] = 0$$

$$\frac{\partial S}{\partial C} = C \sum_{i=1}^n e^{-\frac{2k}{e} t_i} - \sum_{i=1}^n N_i e^{-\frac{k}{e} t_i} = 0$$

now?

which can be solved numerically to get the best fit to the data with a function of the form

$$N(t_i) = C e^{-\frac{k}{e} t_i}$$

satisfying the requirement that S be a minimum.

Indirect Least Squares

It may sometimes be possible to transform the Gompertz function to a linear function. Indirect least squares can then be used to determine estimates for the parameters. This will provide the best fit possible for the linear regression of the transformed variables.

(i) C is known

$$N = C e^{-\frac{k}{l} e^{-lt}}$$

$$\frac{N}{C} = e^{-\frac{k}{l} e^{-lt}}$$

$$\ln\left(\frac{N}{C}\right) = -\frac{k}{l} e^{-lt}$$

$$\ln \ln\left(\frac{N}{C}\right) = \ln\left(-\frac{k}{l}\right) - lt$$

This is of the form

$$y = ax + b$$

where $y = \ln \ln\left(\frac{N}{C}\right)$

$$x = t$$

$$a = -\frac{k}{l}$$

$$b = \ln\left(-\frac{k}{l}\right)$$

then if we solve

$$a \sum_{i=1}^n x_i^2 + b \sum_{i=1}^n x_i = \sum_{i=1}^n x_i y_i$$

$$a \sum_{i=1}^n x_i + nb = \sum_{i=1}^n y_i$$

or equivalently

$$-\frac{k}{l} \sum_{i=1}^n t_i^2 + \ln\left(-\frac{k}{l}\right) \sum_{i=1}^n t_i = \sum_{i=1}^n t_i \ln \ln\left(\frac{N_i}{C}\right)$$

$$-\frac{k}{l} \sum_{i=1}^n t_i + n \ln\left(-\frac{k}{l}\right) = \sum_{i=1}^n \ln \ln\left(\frac{N_i}{C}\right)$$

for k and l , this will provide the best fit possible based on a double logarithmic residual sum of squares.

(ii) l is known

$$N = C e^{\frac{-k}{l} e^{-lt}}$$

$$\ln N = \ln C - \frac{k}{l} e^{-lt}$$

$$\ln N = \ln C - k \left(\frac{e^{-lt}}{l} \right)$$

This is of the form

$$y = Ax + B$$

where

$$y = \ln N$$

$x = \frac{e^{-lt}}{l}$ ← ok, since l is known

$$A = -k$$

$$B = \ln C$$

then if we solve the system

$$-k \sum_{i=1}^n \left[\frac{e^{-lt_i}}{l} \right]^2 + \ln C \sum_{i=1}^n \frac{e^{-lt_i}}{l} = \sum_{i=1}^n e^{-lt_i} \ln N_i$$

$$-k \sum_{i=1}^n \frac{e^{-lt_i}}{l} + n \ln C = \sum_{i=1}^n \ln N_i$$

for k and C , this will provide the best possible fit to the data based on a logarithmic residual sum of squares.

(iii) It may be possible, but I have not found a way to linearize the function if either k is known, or all three parameters are unknown.

Computer Simulation

Computer simulated plots were constructed in S within the framework of UNIX, for various values of the parameters k and l . A common value of 100 was used for C in order to make comparison between plots easy.

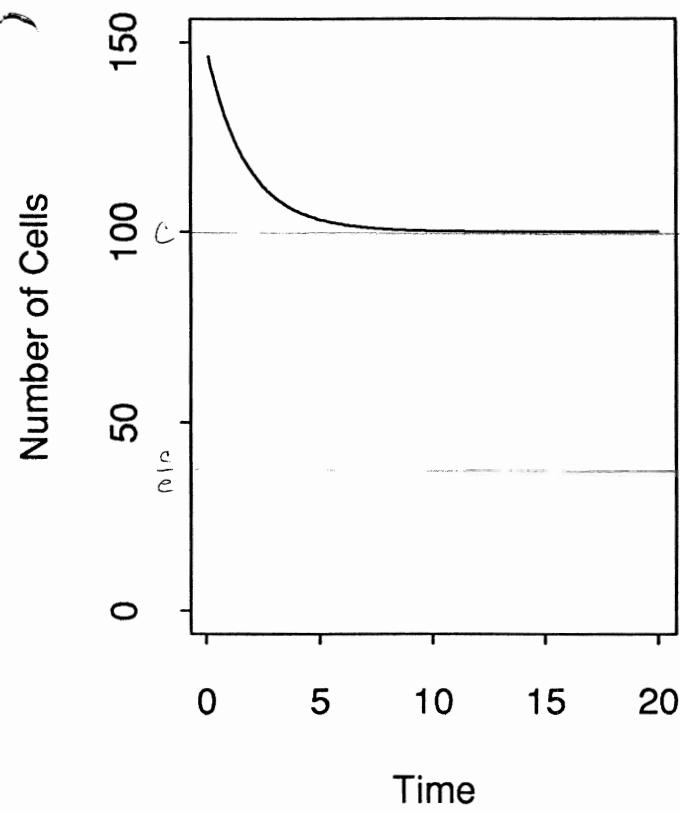
From the plots on page 28, we can see that the computer simulated plots agree with our theoretical cases (i) $k < 0$
(ii) $k > 0, l < k$
(iii) $k > 0, l \geq k$.

From the plots on page 29, we see the effect that k has on the curve for fixed values of l . Obviously, the effect of different values of k is most apparent for small values of t ($0 \leq t < 5$). Again, this agrees with our theory since k was interpreted as the 'initial' relative growth rate (decay rate) for small t . It seems, the closer k is to 0, the closer $N(0)$ is to C .

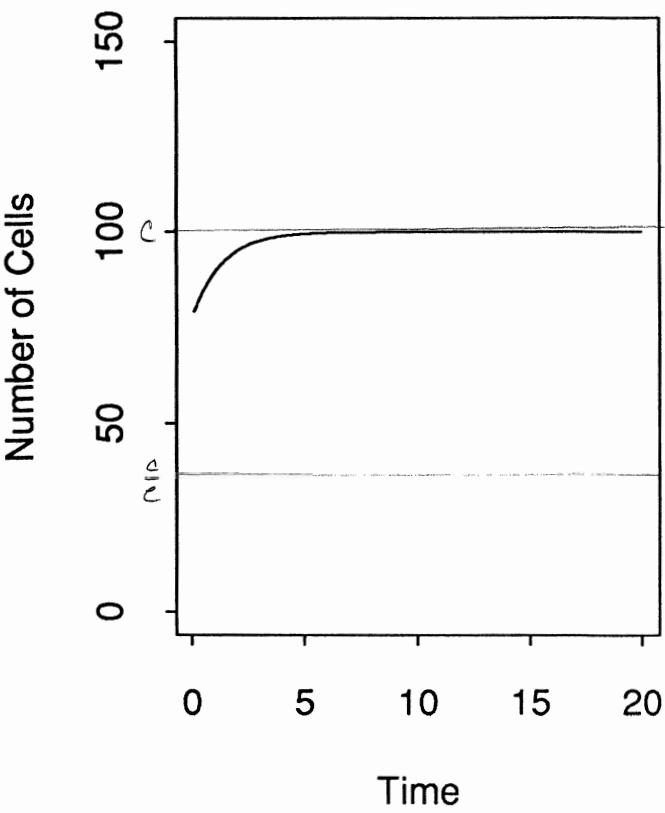
From the plots on page 30, we see the effect that l has on the curve for fixed values of k . The parameter l seems to determine how quickly the curve levels off. As l increases, the slope of the curve becomes steeper and as the slope becomes steeper, the carrying capacity is reached more quickly. This agrees with theory since l is the decay rate of the growth rate, therefore as it increases the curve levels off more quickly.

However, note the relationships are very complex between k and l .

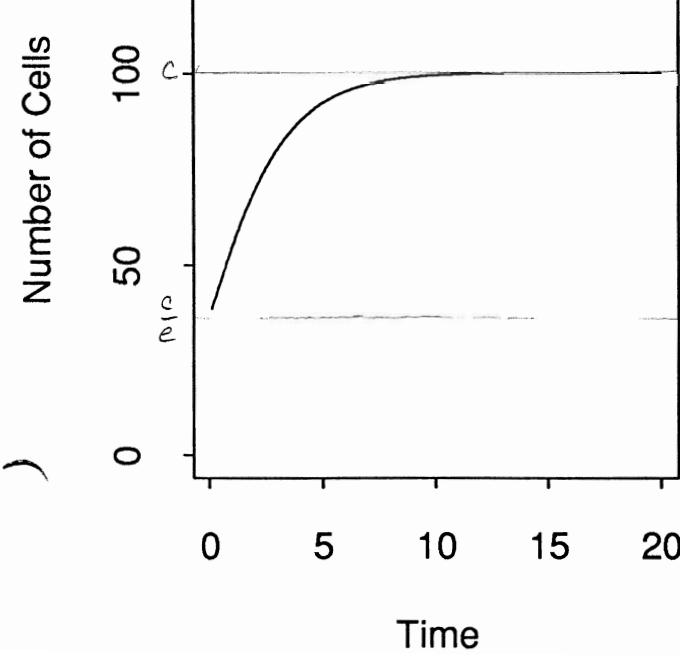
C=100, k=-.2, l=.5



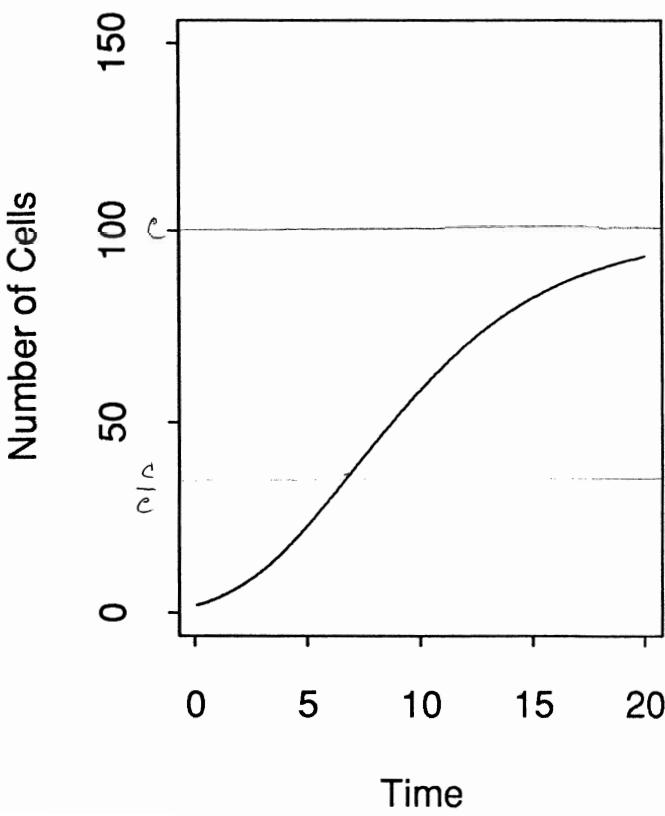
C=100, k=.2, l=.8



C=100, k=.5, l=.5



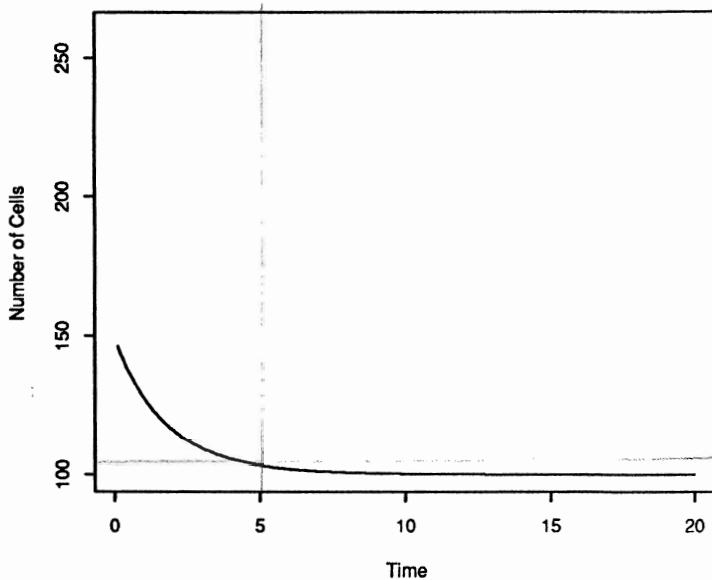
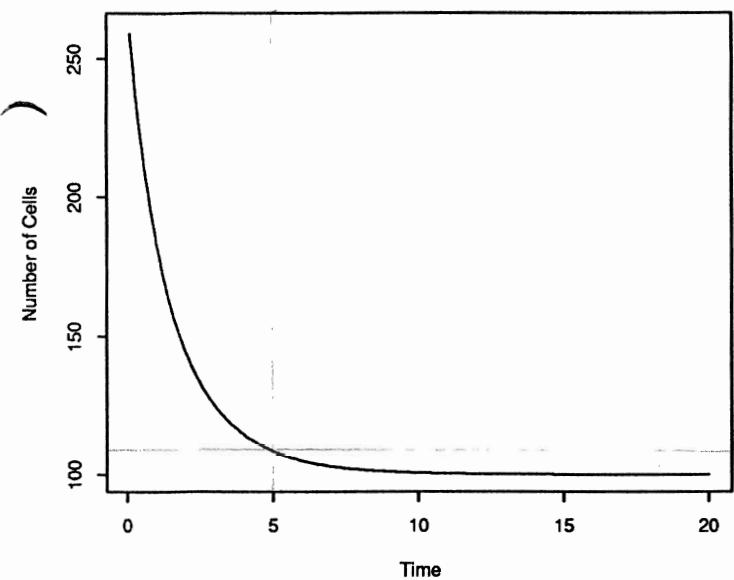
C=100, k=.8, l=.2



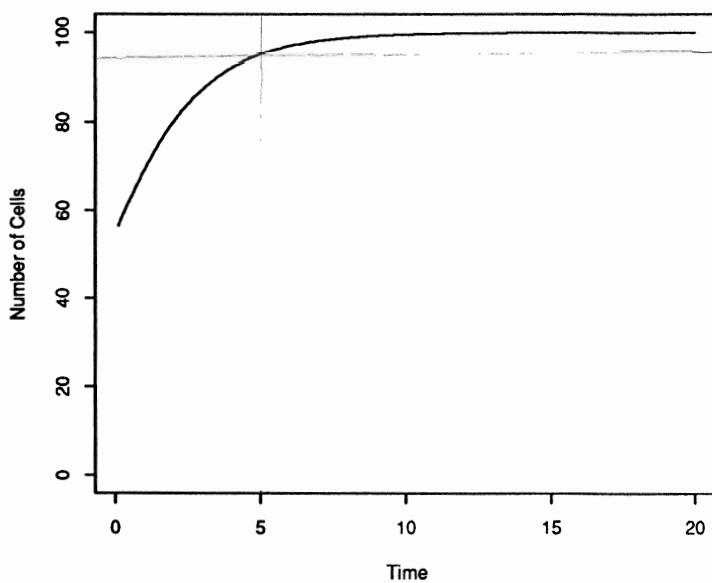
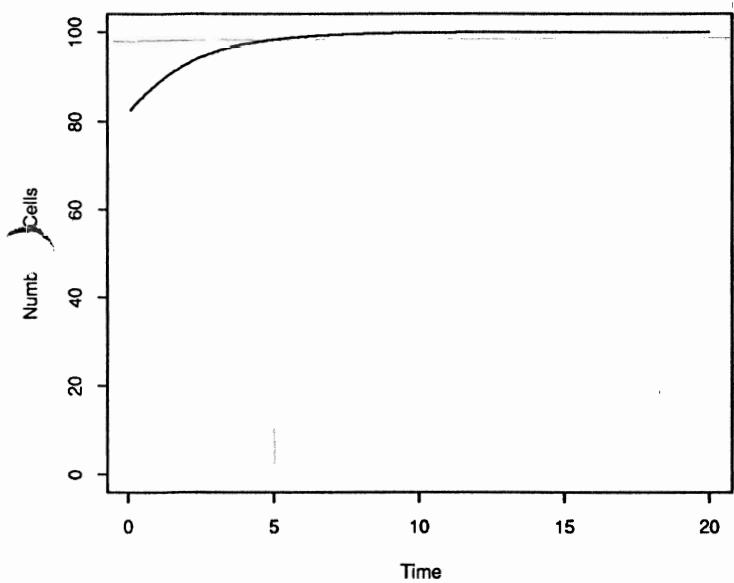
C=100, k=-.5, l=.5

C=100, k=-.2, l=.5

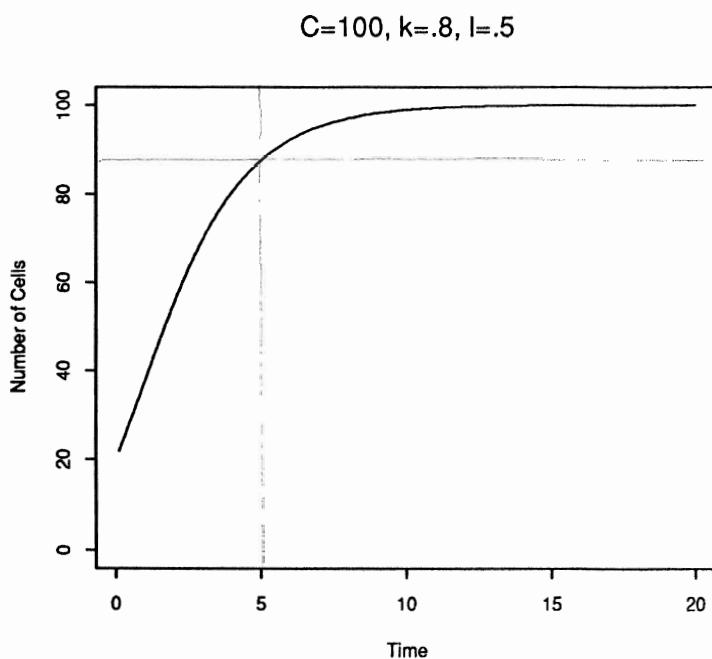
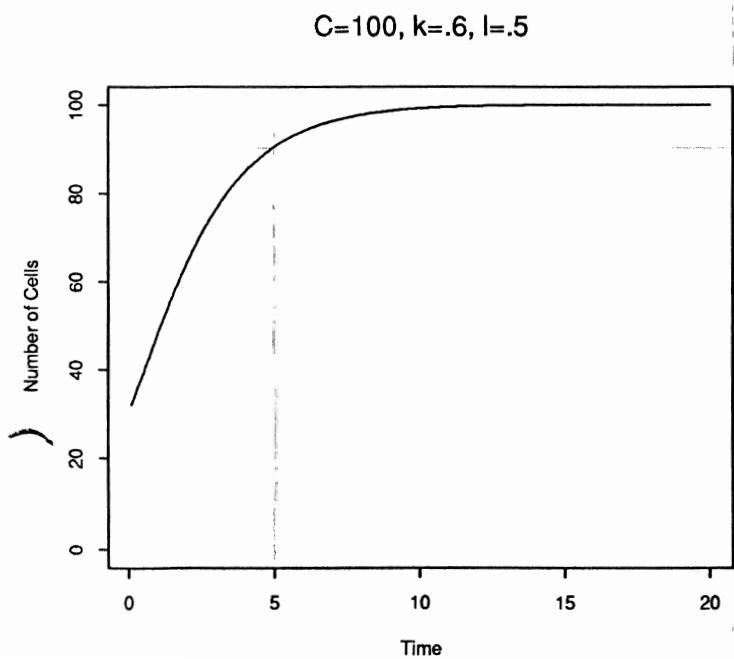
29



C=100, k=.1, l=.5



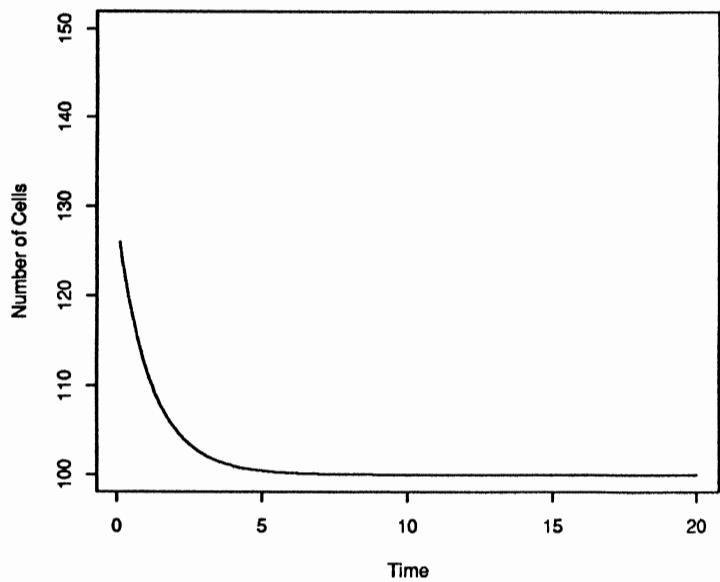
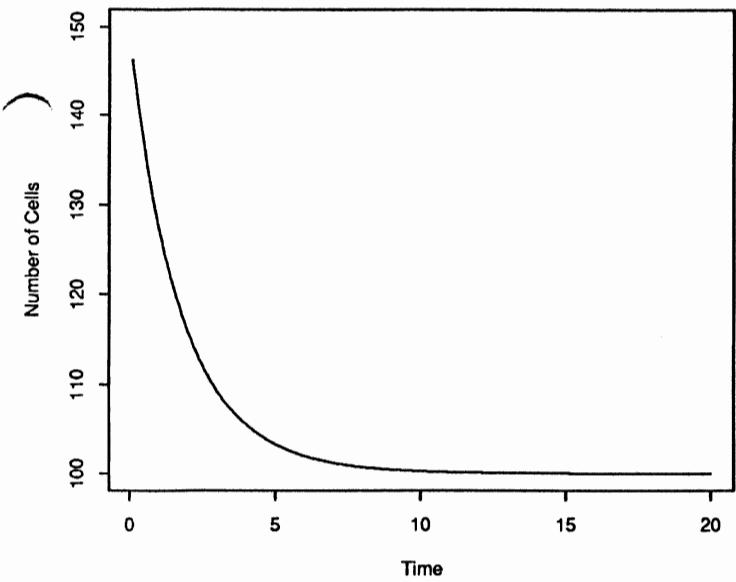
C=100, k=.6, l=.5



C=100, k=-.2, l=.5

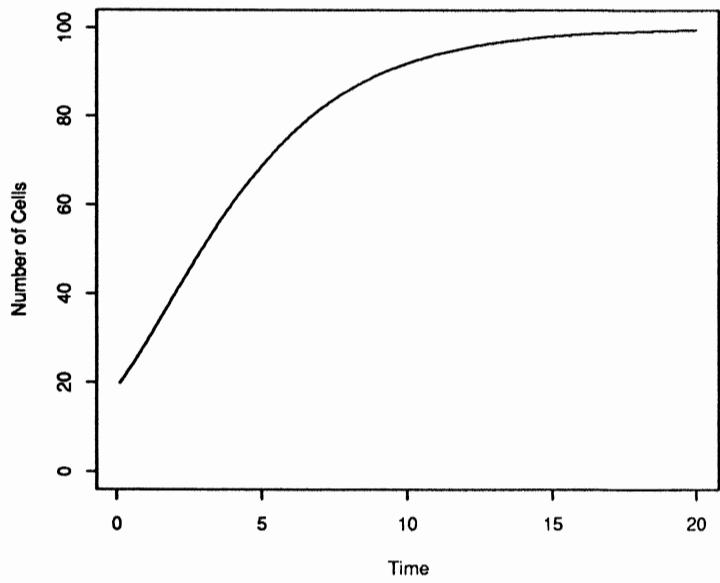
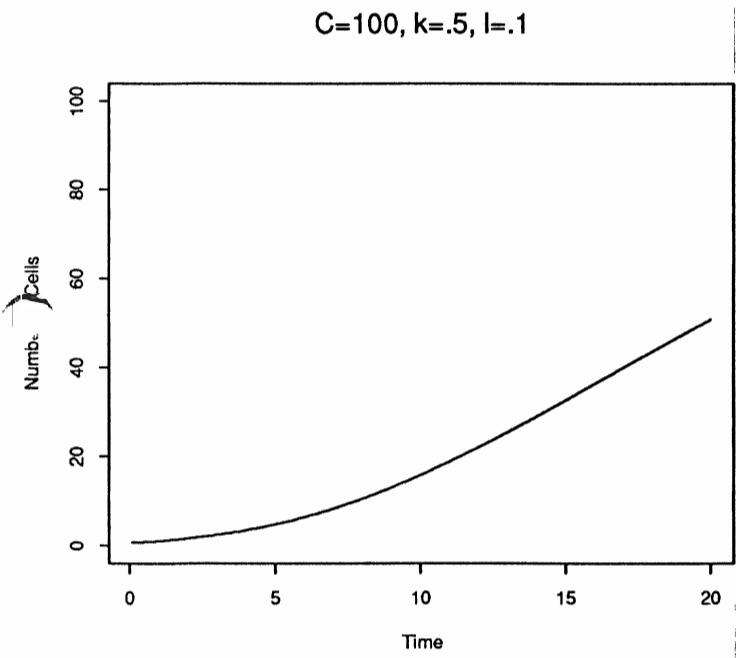
C=100, k=-.2, l=.8

30



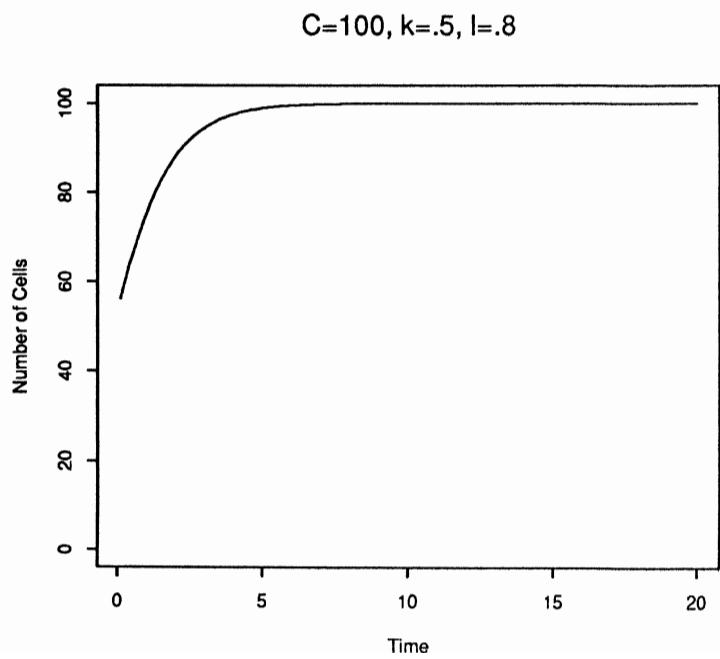
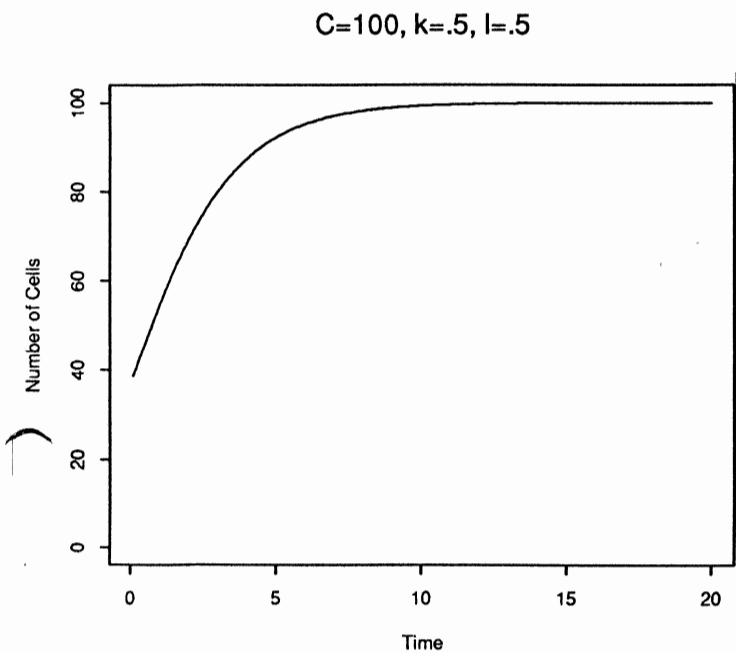
C=100, k=.5, l=.1

C=100, k=.5, l=.3



C=100, k=.5, l=.5

C=100, k=.5, l=.8



Suggestions for Improvement

1. Introduce cancer treatment at some point.
In humans, as soon as tumours are detected, treatment usually begins; therefore radiation treatment or chemotherapy could somehow be introduced into the model.
2. Incorporate increased nutrient supply from capillary growth at a certain point.
3. Extend the model to include all cancer cells in the body rather than just the one tumour. Therefore, if the tumour spreads, these colonies would also be included in the model.

Conclusions

Many sources report that the Gompertz model has proven to be very effective in predicting tumour growth in animals. [2, 5, 6] "Various researchers have shown that the data for many solid tumours is fitted remarkably well, over almost a 1000 fold increase in tumour volume" by the Gompertz model [1]. Because of its effectiveness in practice and its relative simplicity, the Gompertz model is an excellent model to use for modelling the growth of tumours.

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