

Mathematical Modelling of Church Growth

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ABSTRACT

The possibility of using mathematics to model church growth is investigated using ideas from population modelling. The use of the basic epidemic model is discussed and its results applied to a variety of church growth situations. Results show that even a simple model like this can help understand the way in which churches grow, particularly in times of revival.

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1 Introduction

1.1 Background To Church Growth

"Church Growth" is a recent subject area which seeks to analyse why Christian churches, at various levels of organisation, grow or decline. The stimulus for investigating why churches grow came from western missionary organisations in the late 1950's. They were concerned with the effectiveness of the missions they had founded, and needed to examine this effectiveness in order to determine priorities for funding. The pioneer in this field was Donald McGavran who did much to encourage these missionary organisations to see the vast potential for numerical growth in the non-western world. Although numerical growth is not a requirement of all missionary endeavours (McGavran, 1963) it was nevertheless felt that situations needed to be analysed with the factors that cause and inhibit growth identified.

Many of the church growth principles were developed at The Fuller Theological Seminary in Pasadena, California, where Donald McGavran became professor of Church Growth. The church growth movement has itself grown over the years with numerous organisations teaching church growth principles, acting as consultants to denominations and local domestic churches as well as mission organisations. In the UK the Bible Society has been teaching church growth courses for a number of years. Also the British Church Growth Association, with a membership from across the denominations, encourages this way of thinking through its books and quarterly journal.

Much of the analyses in church growth is qualitative, largely based on biblical theology, with ideas also drawn from the social and management sciences, as well as good common sense. However an important part of the analysis is quantitative, which involves measurement of the size of churches over a period of time. Local churches are encouraged to keep and examine attendance and membership figures in order to monitor their situation. Some denominations, such as the Baptist Union of Great Britain, have an annual head count, and there are periodically more ambitious attempts to assess the size of the whole church. A recent survey was conducted in England by Marc Europe¹ who produced membership and attendance figures by region, denomination, and churchmanship (Brierley, 1991a) and compared them with previous surveys. Likewise around the world a growing amount of data exists on the numerical state of churches in various countries.

The data that is gathered is analysed statistically, with projections given for the near future. This approach is essentially empirical in nature. The question can be asked if any theoretical understanding could be brought into the situation that might help explain why the figures behave as they do. To do this quantitatively, mathematics is needed to model the dynamical features in a church growth situation.

¹ Now managed by the Christian Research Association.

A simple example is found in *Christian England* pp. 56-57 (Brierley, 1991b). It has been noticed that the difference between the attendance and membership figures for a denomination is positive if the denomination is growing, and negative if it is declining. Further, the size of the gap appears to indicate the speed of the change. Is this an accident of the numbers or does it depend on the dynamics of how a church attracts attenders and turns them into members? If there is some underlying fundamental principle then more reliable forecasts can be made from that principle, and, more importantly, changes to the churches recruitment strategies suggested and their likely outcome deduced. Although beyond the scope of this paper this is the sort of problem that a mathematical model could address.

1.2 Objections to Modelling Church Growth

It could be argued at this point that this type of analysis is not appropriate for what is a highly complex social system, and, in the view of the church, a divinely controlled situation. This can be answered from at least two angles :

- 1 As a social system church growth is a form of population modelling, a well developed subject for both human and animal populations. Predator-prey, competing species, and harvesting models are well known (Murray), and epidemic modelling analyses very complex behaviour among different groups of people. Population models are successfully applied in complex social situations such as innovation diffusion (Kumar and Kumar, 1992; Mahajan, 1990).

In these types of models the complexity is handled by starting with very simple models based on numerous assumptions, and then gradually relaxing these assumptions by adding new features into the model. There is not usually one model to handle all situations; just enough features are included to extract the principles of the situation and compare with appropriate data. This type of modelling is quite unlike physics where models of all situations can be built up out of simple well-tested laws. There are no such laws in population modelling, and in that sense no equation can exactly represent a given situation. However the equations, although approximate, do shed light on the phenomena being modelled.

- 2 From a theological point of view even the aspect of data gathering has been criticised. This has been defended by a number of people, for example Gibbs (1985) p106-111. These are theological problems outside the scope of this report. A mathematical model of church growth is only modelling the numbers of people represented by this data. Given that the data is gathered, is it appropriate to use mathematics to understand the situation when there is divine control on this growth? This same argument could be used against all the sciences. In practice the models and theories developed are always subject to the proviso "given that God continues to act in the same way". If he does not the theory becomes inapplicable. The same will be true of Church Growth models, which will become apparent when the parameters in the models are identified.

In short, both the social and theological nature of Church Growth will place limits on the usefulness of the mathematical models, a situation familiar to those in social sciences.

1.3 Types of Models

The next step is to consider what sort of models can be developed. Stochastic models are closer to the truth, but more difficult to handle. For that reason deterministic models are best investigated first. Models can be developed to investigate age profiles of the church as well as its geographical spread, however these are unnecessary complications for an initial model. Instead the only feature modelled will be that of the change of numbers in the church over a period of time.

Rather than develop a new model from scratch, it is worth seeing if there is similar behaviour to church growth in other areas of population modelling. This report looks at the application of epidemic models to church growth. These models prove useful because of the similarities between the spread of a disease and the spread of beliefs which ultimately leads to growth in the church. These similarities are :

- There are at least two categories of people : those who have the disease or belief and those who do not.
- Beliefs, like many diseases, are often spread by some sort of contact between the two categories of people. In the case of diseases the contact may be physical, or via some intermediary mechanism such as air-borne droplets. For beliefs the contact is via oral communication.
- The church has frequently experienced the type of rapid growth followed by slower periods of change typical of epidemics. For example, in the last Welsh revival of 1904-5 100,000 people were added to the main Welsh denominations (Evans, 1969, p146), only to be followed by a period of slower growth and eventual decline. Over a longer period South and Latin America, Africa and some Asia countries have seen a huge growth in the Church this century, which shows no sign of slowing down. A similar rapid growth was seen in the Church during the first century AD..
- During times of revival people are noticeably different, particularly in regard to their enthusiasm to communicate their beliefs to others. This has no doubt been a factor in the rapid growth of the church during revivals. In Wales in 1904 such people were said to “have the revival” as if it were a disease that could be caught!

Thus models of the spread of infectious diseases, usually called epidemic models, should prove a useful starting point to model the change in numbers in the church over a period of time.

1.4 Aims of Church Growth Modelling

What should such models achieve? Clearly the situation in a local church has too many variable factors to allow for accurate prediction of numbers. Even at the global level of a particular country parameters can change

unpredictably. Nevertheless mathematical models will provide useful information. Four important results of modelling are :

1. Mathematical models primarily provide principles rather than numbers. An example of this from ecological modelling is the Predator-Prey model originally developed by Lokta and Volterra. There are few cases where the model can be compared with real data, but it does furnish the principle, called Volterra's principle, that moderate harvesting across both species will cause the numbers of the prey species to rise (Braun, 1975). The principle is well observed in the fishing industry and in crop-spraying programmes, even though the actual data is not fitted to the model.
2. A model can help in the understanding of the dynamical process, which can lead to a *theoretical* assessment of strategies
3. The model can help to decide what sort of data is best to measure effectiveness.
4. The model can help explain why there is such wide variation in the speed and extent of church growth and decline. For example some growth is slow and steady, whereas some, often associated with revivals, are fast. Some revivals last many years, as in the 18th century Great Awakening, some only for a year or so as in the 1859 revivals.

There is much of topical interest in church growth such as : church planting strategies; attempts to evaluate methods of evangelism; analysis of church attendance statistics; and speculation whether the recent phenomena of the "Toronto Blessing" will result in a revival among the western Christian church. The latter topic is particularly interesting because there is a great reluctance to call the "Toronto Blessing" a revival, even among its supporters, simply because there has not yet been a large number of converts (Wimber, 1994; Robinson 1994). As will be shown later in this report, epidemic type growth, so typical of a revival, can have a very slow increase in numbers in the early stages. It is hoped that some elementary mathematical analysis will shed some light on these types of areas.

2 Basic Epidemic Model - Construction

2.1 General Assumptions

In the simplest model of the spread of an epidemic, three categories of people are considered, represented by the variables :

- S - The number of susceptibles
- I - The number of infectives
- R - The number of people removed from the system after having had the infection.

A susceptible becomes infected through contact with an infective. Once infected it is assumed that they are immediately able to infect others, even if there are no symptoms. That is, the latent period of the infection is negligible. People spend a certain length of time, D , in the infected category. This is the period over which they can infect others. It may be the entire infectious period of the disease, or less if isolation takes place on or after the time symptoms show, or the disease is otherwise detected. Once a person is removed from the infectious state it is assumed they are no longer able to infect anyone again or become infected again. (Those in the "removed" category may be removed because they have been isolated, or have died, or are now immune to the infection.) Thus the epidemic model is a compartment model for the three categories whose numbers are : S , I , and R . Diagrammatically this is represented as :

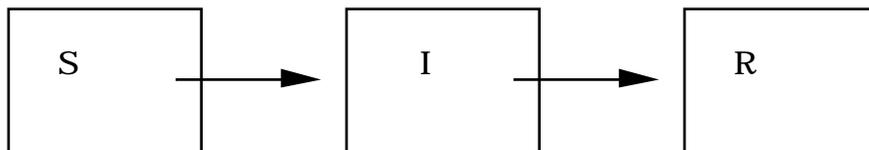


Fig 1

It will be further assumed that the total population $N = S(t) + I(t) + R(t)$ is constant.

To obtain the transmission rates between the compartments further assumptions are needed. The most fundamental, and most criticised (Anderson and May, 1987, p65), of these is homogeneous mixing. That is the

infectives are well mixed throughout the susceptibles². Thus all susceptibles are equally likely to become infected (provided the time scale is not too small) which leads to $dS/dt \propto -S$. Also all infectives will be equally likely to infect a susceptible, which gives $dI/dt \propto I$.

Thus the equation for the susceptibles is usually written (Anderson and May) :

$$\frac{ds}{dt} = -\lambda(t)S \quad (1)$$

where $\lambda(t)$ is called the *force of the infection*, and is the per capita rate at which susceptibles become infected. If δt is a small time then $\lambda(t)\delta t$ is the probability of a susceptible becoming infected in time δt .

It is possible to make assumptions to determine the form of $\lambda(t)$ from the susceptibles' viewpoint but it is easier to reason from the infectives' viewpoint. Let $n_i(t)$ be the number of susceptibles infected by one infective during the whole period they are infectious.³ The net change in the number of infectives during that period because of that single infected is $n_i(t) - 1$. Thus the per capita rate of change of infectives is

$$\frac{n_i(t) - 1}{D}$$

This gives the equation for the infectives as :

$$\frac{dI}{dt} = \frac{n_i(t) - 1}{D} I = \frac{n_i(t)}{D} I - \gamma I \quad (2)$$

where $\gamma = 1/D$ is the removal rate of infectives. As the model is a compartment one the loss of susceptibles equals the gain in infectives :

$$\lambda(t)S = \frac{n_i(t)}{D} I$$

giving an expression for the force of the infection in terms of the infectives' behaviour :

² See Anderson (1988) for models that take into account different degrees of contact amongst susceptibles and infectives.

³ This is referred to as R in Anderson and May p 17.

$$\lambda(t) = \frac{n_i(t)I}{DS} \quad (3)$$

Different assumptions for how a disease is spread will give different forms for $n_i(t)$. It is usual to make two assumptions. The first is that $n_i(t) \propto D$, the longer people are infectious for the more they infect. This is true for most diseases although not for people with a limited number of contacts such as non-promiscuous people with sexually transmitted diseases (STD's).

2.2 Crowd Model

The second assumption, which will cover most infections, is that $n_i(t) \propto S$, the more susceptibles there are the more contacts an infective has. This is typically what happens if a person with a cold enters a class of students, the larger the class the more students are likely to get the cold in a given time period. It is also true if a larger number of infectives enter a larger population, such as a university or a town, in such a way that they are homogeneously mixed, an assumption already made. This model is sometimes called the crowd model (The Open University 1988).

Thus the form of $n_i(t)$ is

$$n_i(t) = \beta DS \quad (4)$$

where β is a constant. Equation 4 with equations 1 to 3 give the classic equations for a general epidemic as given by Bailey (1975) :

$$\frac{dS}{dt} = -\beta SI \quad (5)$$

$$\frac{dI}{dt} = \beta SI - \gamma I \quad (6)$$

$$\frac{dR}{dt} = \gamma I \quad (7)$$

Basic Model of an Epidemic - Model A - The Crowd Model

Equation (7) comes from (5) and (6) and the fact that the total population $N = S + I + R$ is constant.

The rate of transfer from S to I is given by the product of the two population numbers, the “mass action” principle, originally proposed for epidemics by Hamer (1906). This type of non-linear behaviour in a compartment model is characteristic of epidemic and many ecological models.

2.3 Fixed Contacts Model

Clearly this second assumption is not true in the very early stages of an epidemic where a lone infective in a very large population will not have more contacts in a given time if the population is larger. Neither is it true for STD's where there is usually a fixed number of different contacts between an infective and other people in a given time period regardless of the size of the susceptible population. In these cases the second assumption for $n_i(t)$ is modified to $n_i(t) \propto S/N$, that is the number of contacts between an infective and a susceptible depends on the proportion of susceptibles in the total population. This has assumed that an infective does not deliberately seek out susceptibles, thus their constant number of contacts are shared between susceptibles, infectives, and the removed.⁴ Thus :

$$n_i(t) = \beta \frac{DS}{N} \tag{8}$$

where β is a different constant from the first model.⁵ Equation (8) with equations (1-3) give the basic epidemic equations familiar in the study of STD's and the spread of HIV/Aids (Anderson, 1988) :

$$\frac{dS}{dt} = -\beta \frac{SI}{N} \tag{9}$$

$$\frac{dI}{dt} = \beta \frac{SI}{N} - \gamma I \tag{10}$$

$$\frac{dR}{dt} = \gamma I \tag{11}$$

Basic Model of an Epidemic - Model B - Fixed Number of Contacts

⁴ If removal is via death or isolation, and these numbers are a significant proportion of the total population, then $n_i(t) \propto S/(S+I)$.

⁵ The number of contacts an infective has in a totally susceptible population $n_i(t) = \beta D$ is related to the basic reproductive rate of the microparasite or virus that causes the infection (Anderson and May Ch 2 where it is called R_0)

For most infectious diseases model A is usually suitable although the truth often lies somewhere between the two models, the number of contacts an infective has depending on S but not linearly. Various attempts have been made to construct a more sophisticated model of the transfer from S to I (see May and Anderson 1985 p142), however models A and B are sufficient to derive important epidemiological principles.

Of course, if the total population N remains constant, the mathematical results of the two models will be identical since all that has happened is that the transfer constant β has been redefined. However for infections that are spread over long time periods, where there may be births, natural deaths and migration, the two types of models will be mathematically different. Again this will need careful consideration for applications to church growth.

For the remainder of this report results will be quoted for model A only, however the reader should bear in mind that model B may be equally relevant.

3 Basic Epidemic Model - Results

3.1 Threshold of the Epidemic

Perhaps the most important result is that of the "threshold" of the epidemic first derived by Kermack and McKendrick (1927). Technically an epidemic is deemed to occur if the rate at which people become infected increases. This occurs when :

$$S_0 > \frac{\gamma}{\beta} \equiv \rho \tag{12}$$

where ρ is referred to as the threshold of the epidemic. This can be demonstrated by illustrating the phase paths of I against S (fig 2). Thus epidemics are more likely to occur in large concentrations of susceptibles, a fact borne out by the prevalence of epidemics in large cities and the general lack of epidemics among wild animals

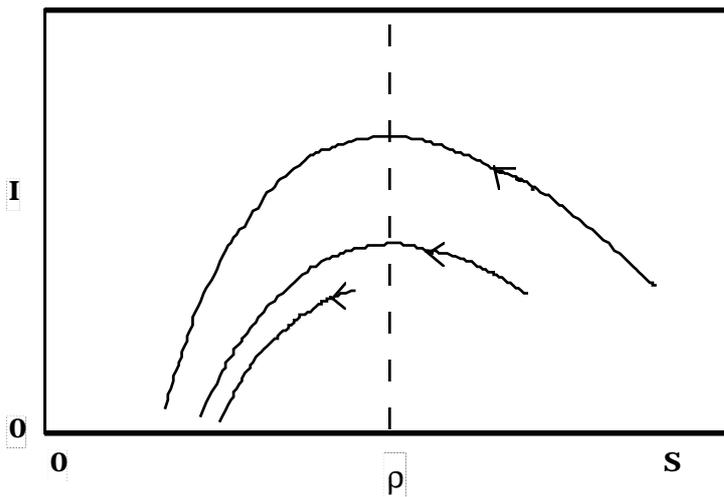


Fig 2 Typical Phase Paths of the Basic Epidemic Model

where large concentrations are unusual. Epidemics are also more likely if the contact rate between susceptibles and infectives (β) is higher, and if the duration of the infectious stage ($D = 1/\gamma$) is longer, both common sense results. This principle has been demonstrated for numerous real cases, one readable example is that of the Eyam plague of 1665-6 given in Raggett (1982).

Significantly the value of I_0 does not influence the likelihood of an epidemic (unless it is zero!). If the initial number of infectives is smaller the epidemic may take longer to start, *but it will occur*.

3.2 Early Stages of the Epidemic - Doubling Time

In the early stages of an epidemic, when the number of infectives and removed are very small compared with the total population ($S \approx N$), the growth in the number of infectives is exponential. Equation 6 becomes :

$$\frac{dI}{dt} = (\beta N - \gamma)I \tag{13}$$

giving a doubling time for the early stages of an epidemic as :

$$t_d = \frac{\ln(2)}{\beta N - \gamma} \quad (14)$$

This result is particularly useful in estimating β if data is available for those stages (e.g. Anderson, 1988, and May and Anderson, 1987, who apply it to the spread of HIV).

3.3 End of an Epidemic - Lack of Infectives

Solving equations (5)-(7) in the static case gives $(S,0,R)$ as stable equilibrium points for any S and R . Thus, on the I - S plane, the entire S axis is in stable equilibrium, all solutions ending up at some point on it. Thus some susceptibles remain at the end of the epidemic. What determines its value?

There is no analytic solution for equations (5)-(7) for S , I and R in terms of t , however I can be expressed in terms of S and their initial values :

$$I(S) = I_0 + S_0 - S + \rho \ln \left[\frac{S}{S_0} \right]$$

The spread of the infection is over when I becomes 0, the number of susceptibles remaining S_∞ can be calculated from the non-linear equation :

$$S_\infty = I_0 + S_0 + \rho \ln \left[\frac{S_\infty}{S_0} \right] \quad (15)$$

S_∞ is thus determined by the threshold and the initial values of S and I . The epidemic ends not for lack of susceptibles but for lack of infectives. The epidemic burns itself out before all susceptibles can catch the disease because the infectives have fallen to insufficient numbers to carry on the spread. This is due to there being insufficient infectives initially for the number of initial susceptibles, given the threshold of the epidemic. Increasing the number of initial infectives will always reduce the susceptibles remaining as :

$$\frac{dS_\infty}{dI_0} = \frac{1}{1 - \rho/S_\infty} \quad (16)$$

is always negative. Mathematically there is no value of I_0 that will make S_∞ zero, however there may be cases where it could be made very close.

3.4 Small Epidemics - Threshold Theorem

For a small epidemic Kermack and McKendrick derived a simpler result namely : *the number of susceptibles falls to a value as far below the threshold as it started above.* This is referred to as the threshold theorem. Thus :

$$S_0 - \rho \approx \rho - S_\infty \quad (17)$$

A small epidemic is one where the initial number of susceptibles is not far above the threshold value, and the initial number of infectives is small. However as Raggett (1982) showed the theorem is not far from the truth even for a fairly large epidemic such as the Eyam plague.

4 The Simple Church Growth Model

4.1 Use of the Basic Epidemic Model

The basic epidemic model will be used as the initial model to investigate the dynamics of how a church grows. The justification for this is as follows:

- 1 Churches grow because people undergo a process - conversion - which results in observable changes in a person, such as church attendance, enthusiasm for the new faith, adoption of a new moral code with its behavioural changes. Thus a convert can be easily distinguished from a non-believer just as a person with an infection can be distinguished from a susceptible.
- 2 Most conversions come because of a contact between an active believer and a non-believer. The active believer may lead someone to Christ⁶, or simply take the person to a church meeting or evangelistic campaign. The growth in the church is proportional to the contacts between an active believer and non-believers, just as the spread of an infectious disease is proportional to the number of contacts between infectives and susceptibles.
- 3 Not all people in the church are responsible for spreading the faith. Indeed in most churches only a small proportion of believers are involved in passing on their beliefs⁷. Thus as well as active (or "infected") believers there are also church members removed from most of the growth process. This is similar to the removed category in an infectious disease. Often it is the new converts who are most enthusiastic about spreading the faith, and who have the most non-Christian contacts. Thus as a first approximation it is assumed that all new converts go through an initial phase of enthusiasm where they are highly active in spreading the faith, but, after a period of time, lapse into a less active role in evangelism. Although the number of converts brought about by those in the "removed" category will not be zero, it is assumed that the number is very small compared to those from the infectives and thus it can be ignored.
4. Periods of revival within the church often behave in a similar fashion to an epidemic : there is a period where it builds up; it reaches a climax; and eventually it passes away. It may take place gradually or suddenly (Lloyd-Jones, 1986, pp105-106). Not all church growth is like this, neither do all diseases

⁶ *This is the conventional expression used when a believer is instrumental in another person's conversion. It is incorrect to say that the believer converts the unbeliever - theologically conversion is an act of God. See the section on terminology.*

⁷ *Even in a highly successful "Cell" Church 65% of the membership being actively involved in bringing people to Christ is deemed a very high figure (Neighbour, 1994), no doubt a key factor in their growth. For conventional churches the figure is more likely to be less than 10%.*

spread like this; there are endemic infections. However epidemics and revival church growth share these dynamical features.

- 5 For the main part of this report the timescale will be chosen so that births and deaths can be ignored to a first approximation, that is no biological growth⁸ or decay. Thus the static epidemic model will be explored and its shortcomings pointed out, where appropriate.

Further to this, growth by transfer, so significant for individual congregations, will be ignored as the model will be applied to the church as a whole rather than one small part of it.

Thus the simple church growth model is the basic epidemic model given by equations (5)-(7), or possibly (9)-(11) depending on the transmission mode and population numbers.

4.2 Identification of Variables and Parameters

Given that the simple epidemic model is a suitable starting point to analyse church growth, the variables are easily identified. S is the number of susceptibles, those not in the church, non-believers, with whom the church members have contact. Isolated susceptibles are not part of the dynamics of growth. I is the number of "enthusiasts", or "infected" believers, within the church who are active in spreading the faith, i.e. in making contacts with non-believers that lead to their conversion. R is the number in the church who have a negligible role in making converts, thus $I + R$ is the total number in the church, referred to as church members or believers. $S + I + R$ is the total population involved in dynamics of the growing church, which is a constant in the short term.

The parameter β is a measure of the effective contacts between believers and non-believers. This depends on $n_i(t)$ the number of converts (not contacts) one infected believer is responsible for during the whole of their infectious period, that is how many people they bring to Christ before they drop down to the lower level of activity characterised by the removed category. $D (= 1/\gamma)$ is the length of time they are an infected believer.

4.3 Identification of Transmission Mechanism

In section 2 two models for an epidemic were identified depending on how $n_i(t)$ depends on the number of susceptibles : the crowd model (A) and the constant contact model (B). To decide which model is more appropriate the transmission mechanism between an infected believer and an unbeliever needs to be identified.

⁸ Church growth is usually divided into three categories : biological (those born to church members, who themselves become members); conversion (those who become members having had no upbringing in the church); transfer in (those who move into the church from another). All three have their opposite in terms of decay : death, reversion and transfer out. These are explained in Gibbs (1985) Ch4 and Pointer (1987) pp19-22.

The key question is : if the population of unbelievers is increased will the church grow faster through conversion, i.e. will an infected believer be responsible for more conversions? If the church is a significant proportion of the population the answer is generally "yes" and thus model A is more appropriate. Consider the following transmission mechanisms :

- The infected believers are engaged in a systematic program of evangelism such as door to door work. The larger the population the more people will get visited - thus the more contacts will be made, i.e. model A. There are two exceptions. The first will be if the unbelieving population is huge compared to the church, in which case model B is more suitable. The second will be if believers deliberately seek out susceptibles so that they only meet a fixed number of susceptibles in a given time period. In this case $n_i(t)$ is independent of S and the equations become linear. However this is a rare form of recruitment mainly confined to cults.
- The infected believers evangelise through their network of contacts⁹. This network is unlikely to be larger if the population increases - there are only so many friends and acquaintances a person can hold down. However in a larger population this network is often more changeable over time - this increases the number of contacts, and the number of people two or more believers have in common in their network will be smaller, thus the number of global contacts for the church is bigger. This again supports model A.
- The infected believers are those caught up in a revival. In this case, in their enthusiasm, they make contact with many people outside of their normal friendship network. Indeed people whom the infected believers have never met may seek them out simply because news about them, and their behaviour, has reached those people. Again this leads to an increased number of contacts in a larger population, hence model A.

The value of D will vary according to the mechanism. In some revivals it can simply be a matter of months before the enthusiastic phase passes - short term growth. In a programme of evangelism it is more likely to be around two or more years - medium term growth. It is conceivable that the enthusiastic phase could last many years leading to long term growth, however the basic epidemic model is unsuitable as births and deaths have been excluded.

4.4 Interpretation of Epidemic Model Results

In section 3 four results were identified for the basic epidemic model. These can be applied to the simple church growth model.

⁹ *Holloway (1989) pp24-27, quoting the work of McGavran, identifies social networks as the major lines of spread of Christian faith.*

- **Epidemic Threshold.** There is a threshold above which significant church growth, or a revival, will take place, given by (12). Growth is more likely to occur in large concentrations of unbelievers, if the contact rate between believers and unbelievers is higher, or if believers are infective for longer. This agrees with common sense, an important guideline in mathematical modelling. However the number of infected believers does not determine whether growth will take place or not. A small church is as equally likely to see a revival as a larger one, it will just take longer for the revival to get going and be spread over a longer period of time. This will be investigated further in section 5.
- **Early Stages.** In the early stages a church grows exponentially, equation (13). This doesn't mean its growth is always fast. For example if it takes twenty years to double its numbers, it will double in the following twenty years. The growth is geometric rather than arithmetic. Such growth has been seen amongst South American Protestant churches throughout this century, and among the Pentecostal and New Church streams in the UK in recent years (Brierley, 1993). When the early phase is over the growth usually slows down.
- **End of Growth.** Growth eventually comes to a halt because of a lack of infected believers. The church runs out of enthusiasts, because their conversion rate is not sufficient among a falling number of unbelievers. Growth does not end because there are no more unbelievers. The history of revivals show that they stop long before all the people in a population are converted or reached. However a church with more enthusiasts at the beginning will see greater growth, all other things being equal, as equation (16) shows.
- **Threshold Theorem.** The number of converts made during a period of growth will be approximately double the difference between the number of susceptibles and the threshold.

To illustrate the last point, the threshold ρ (12) can be rewritten for model A using (4) as $\rho = S/n_i(t)$. Let the church be in a population of say 50,000, and 10 enthusiasts are responsible for making 11 converts during their enthusiastic period, i.e. $n_i(t)$ is 1.1 initially. The threshold is then about 45,000 giving a difference of 5,000. Thus around 10,000 converts are made! Sadly in a typical British town of 50,000 people many churches will contain no such enthusiasts. Thus the number of initial enthusiasts I_0 is very small, and this growth would occur over a period of time much longer than the lifetime of the individuals. The growth, therefore, has to be offset by deaths. Thus a few churches see some growth, and the rest survive or die due to biological and transfer effects alone.

5 Numerical Solutions

5.1 Scaled Equations

The simple church growth model is a non-linear system without an analytical solution in general. Thus to investigate time scales for growth, and the number converted, the differential equations need to be solved numerically. In order to avoid instabilities in the numerical method the variables I and S need to be scaled.

The simplest scaling is to let the new variables i and s be the *proportion* of non-believers and infected believers in the whole population :

$$i = \frac{I}{N} \quad s = \frac{S}{N} \quad (18)$$

Substituting these into equations (5) and (6) for model A gives :

$$\frac{ds}{dt} = -\beta N s i \quad (19)$$

$$\frac{di}{dt} = \gamma \left(\frac{\beta N s}{\gamma} - 1 \right) i \quad (20)$$

Let $n_i(0) = \beta N / \gamma$ the number of non-believers converted through one infected believer given that the whole population is susceptible. This depends on the population size following the original assumption. Using this and $D = 1/\gamma$, equations (19-20) become :

$$\frac{ds}{dt} = -\frac{n_i(0)}{D} s i \quad (21)$$

$$\frac{di}{dt} = \frac{i}{D} (n_i(0) s - 1) \quad (22)$$

Scaled Equations For Simple Church Growth

The threshold of the epidemic type growth now becomes

$$s_0 > \frac{1}{n_i(0)} \equiv \hat{\rho} \quad (23)$$

This will be the threshold for a revival to occur.

Exactly the same scaled equations result for model B except that $n_i(0)$ is β/γ and independent of population size, following its assumption. Thus as long as N is a constant the dynamics of the two models have the same behaviour. For model A the parameters $n_i(0)$ and D will be determined for a particular community, whereas in model B it is independent of the community.

The differential equations were solved numerically on a computer using a Runge-Kutta-Fehlberg method of order 3/4 (Burden and Faires, 1988, Ch 5).

5.2 Increasing Effectiveness of Evangeliser

One aim of evangelistic programs is increase a believer's *effective* witness. This can be achieved by training people to explain the gospel effectively, e.g. Teach and Reach method and Good News Down The Street. It can also be achieved by increasing the number of contacts with unbelievers, e.g. Seeker Church model¹⁰ and the Cell Church model¹¹. The idea is that, all other things being equal, an infected believer will be responsible for more conversions. Both methods can be employed together.

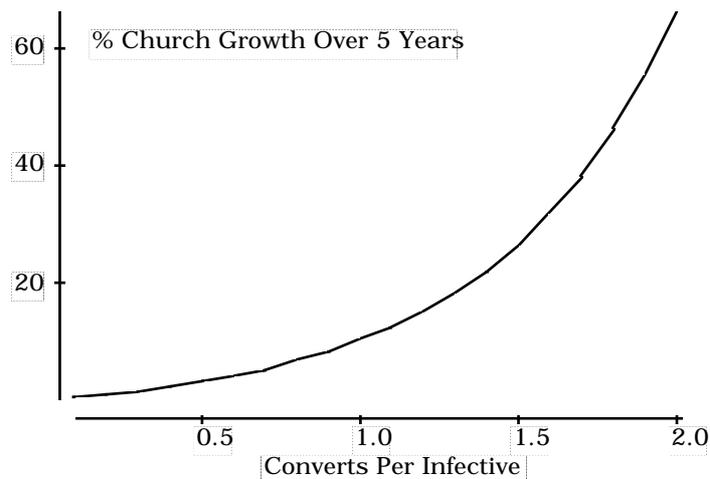


Fig 3 Effect of Changing Individual Effectiveness on Church Growth

Assume that the effectiveness of such a method is medium term say $D = 2$ years, i.e. a believer loses their evangelistic impact after two years on average. Assume also that the number of infected believers is initially 5% of the church, with 10% of the total population in the church, i.e. $s_0 = 0.9$ and $i_0 = 0.005$.

The equations can be solved with a variety of values of $n_i(0)$ from 0.1 up to 2.0 converts per infective over that two year period. The percentage church growth over a five year period is shown in

¹⁰ The Seeker Church model is pioneered by the Willow Creek Community Church near Chicago. The methods are analysed by Robinson (1993).

¹¹ There are many examples of Cell Churches, the most well know include the Yoido Full Gospel Church in Seoul, Korea, and the underground church in China. The methods are explained by Neighbour(1990) and others on the World Wide Web (Cell Church Links).

Fig 3. Note the effect is near exponential. The benefits from doubling the effectiveness of an individual believer is to more than double the growth rate of the church.

5.3 Increasing Number of Evangelisers

Another aim of evangelistic programs is to increase the number of people involved in evangelism. Keep D at 2 years and fix $n_i(0)$ at 0.5, i.e. in 2 years it takes two infected believers to produce one convert. If i_0 is now varied the percentage church growth responds in a linear fashion (figure 4). Thus increasing the number of

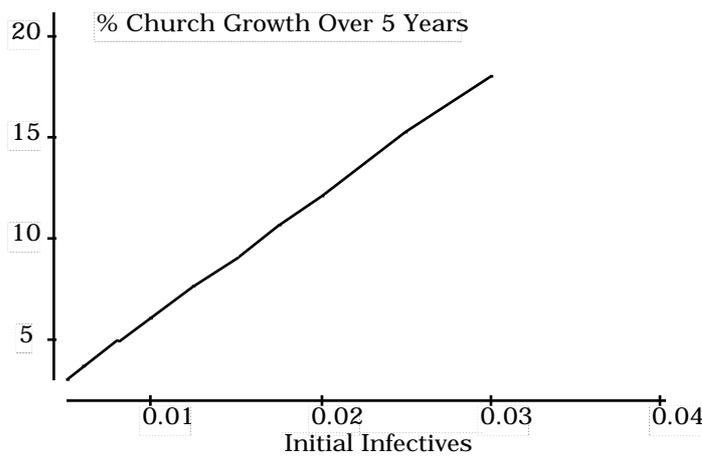


Fig 4 Effect of Changing the Number of Evangelisers on Church Growth

evangelisers does not have the same impact as increasing an evangeliser's effectiveness over periods as short as 5 years. For longer periods births and deaths need to be taken into account.

To explain this result, note that in the early stages the increase in the number of infected believers is approximately exponential in time $\propto i_0 \exp(a t)$ where a is proportional to the number of converts one infective is responsible for. This is linear in i_0 but exponential in a , thus

growth is more sensitive to changes in effectiveness than it is to the initial number of infected believers.

5.3 Medium Term Revival - Global View

Revivals in the Christian church invariably start among its members first (Lloyd-Jones, 1986, pp 99-101), with the fire being spread from believer to believer before it reaches unbelievers. This is sometimes called a renewal phase of a revival. Mathematically it requires mass action type contact between I and R to model the change of behaviour among inactive believers, a feature the simple church growth model doesn't contain. However the simple model will give some indication of the later stages of a revival when contact with unbelievers becomes the dominant behaviour.

Consider a global view, i.e. the whole of the Christian church in one country. Keeping the church as 10% of the whole population (about the UK.) figure, a revival will occur if the threshold $\hat{\rho} = 1/n_i(0)$ is exceeded by the initial percentage of non-believers as given by equation (23). Thus $n_i(0) = 1/0.9$. Of course the church will grow if $n_i(0)$ is less than this figure but it will not be revival type growth. Assume that $n_i(0) = 1.15$, just in excess of the threshold.

Typically revivals in a country start with a small number of infected believers (Lloyd-Jones pp163-166). Let $i_0 = .01\%$ i.e. only 1 in a thousand of the church are so affected.

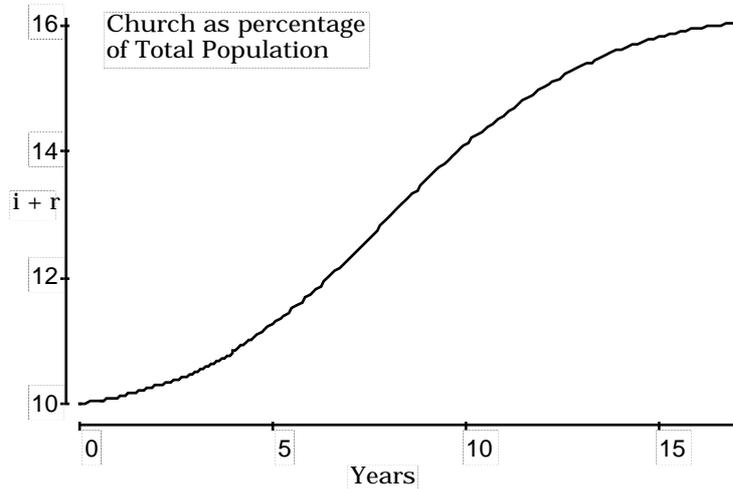


Fig 5 Growth of the Church in a Country - Medium Term Revival

The church, initially 10% of the population, increases to 16% over 15 years. However the start of the growth is slow with only 1% of the population added in the first five years. The bulk of the growth is in the following 7 years, which sees a further 4% added. Thus a revival may not be immediately noticeable in terms of a substantial increase in numbers within the church. Bearing in mind that this follows an earlier renewal phase, the time period before growth is notice could be quite lengthy.

This "slow start" behaviour typifies a medium to long term revival such as the 18th century evangelical awakening in Britain. Although it started in the 1730's the significant effects on church numbers did not occur until the middle of the century with much of the increase in the latter half. One of the reasons for the slowness of the revival is the low numbers of church members within society as a whole, together with the low number of infected believers initially. It is these conditions which prevailed in the 18th century. By contrast the revivals during the 19th century in Britain were faster, but the church was a much larger proportion of the population. This will be investigated in section 5.4.

Another significant result is that the revival is ending due to dynamical effects dependent on its initial intensity. It is not ending due to any change in spiritual conditions such as the revival work being hindered in some way. Given that infected people are only effective for a fixed period then, with a given number of susceptibles, only a certain number of conversions become possible before the number of susceptibles an infected person is likely to meet in that time period is too small to keep the revival going. Of course the believer may still be involved in conversions after his infectious period ceases, but this is at a much lower level and does not give revival type growth.

The only way to increase the number of converts in a revival is to increase the effectiveness of the infected believer $n_i(0)$ ¹². Increasing this to 1.2 converts per person sees a larger but shorter revival. The revival is over in about 10 years with the church increasing to about 22% of the population. The church sees a 10% increase in its numbers in less than 3 years, compared to 5 years with the lower figure for $n_i(0)$. Thus the revival is noticed earlier.

Increasing the parameter D, the time period over which conversions take place, slows the revival down but the numbers converted stay the same. D could be removed from the equations by scaling t.

5.4 Short Term Revival - Global View

In some periods the church has occupied a much larger proportion of the population. In Britain during the 19th century it accounted for nearly half the population. Assuming the church is 50% of the population, the threshold for a revival to occur is now higher, $\hat{\rho} = 2$, thus more converts per unbeliever are required for a revival to occur.

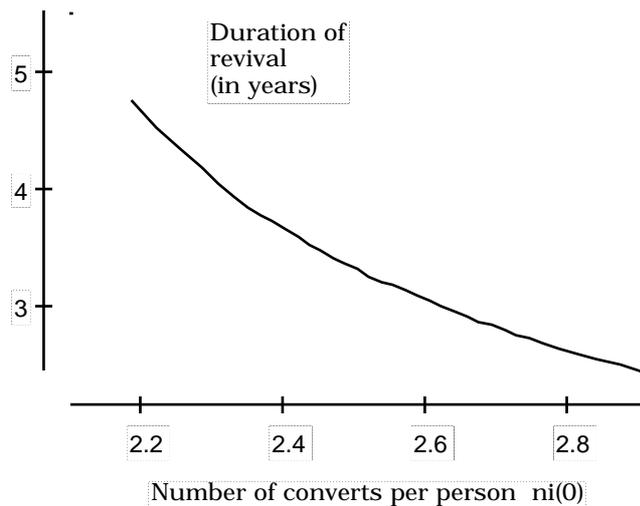


Fig 6 Duration of Short Term Revival in a Country

Whether this is "harder" to achieve cannot be answered, there are too many factors, however from a social point of view a church that is more acceptable in a culture, because of its size and therefore influence, may find it easier to make converts. Thus revivals can occur albeit with a larger value of $n_i(0)$.

Keep the number of infected believers initially at 1 in 1000 of the church thus $s_0 = 0.5$ and $i_0 = 0.0005$. Keep the effective period $D = .1$ year. For $n_i(0) = 2.1$ the revival is over in 6 years and the church increases by 5% of the population. Figures 6 and 7 show the duration

and increase in the church for values of $n_i(0)$ up to 3.

¹² That is increase the number of effective contacts between an infected believer and a non-believer. This may be done by increasing the number of contacts, or by unbelievers being more responsive to the gospel message. It is this latter method that is deemed to be a significant cause of a revival taking place. A revival is an act of God which turns believers in to effective witnesses and "opens the hearts" of unbelievers (Lloyd-Jones pp 50, 56-57, 106, 233-236,).

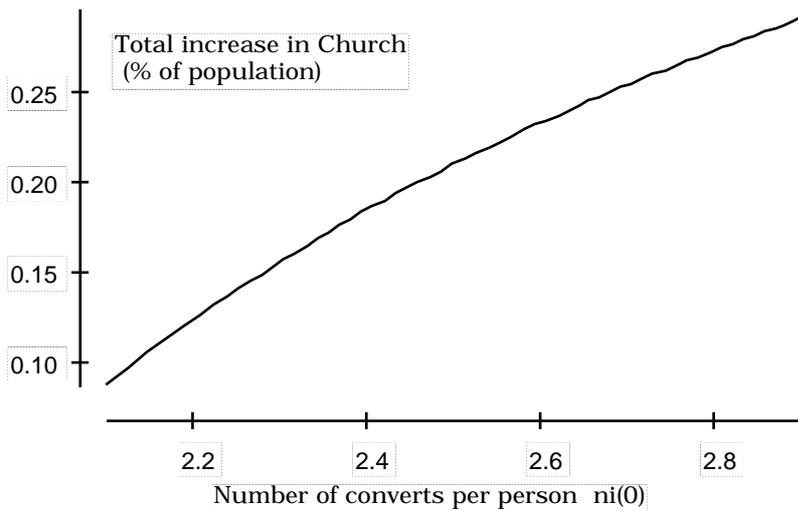


Fig 7 Growth of the Church in a Country - Short Term Revival

The larger the value of $n_i(0)$ the faster the revival and the larger the number of converts. The intensity of the revival is very sensitive to the number of converts per person. Indeed for $n_i(0) = 3$ the conversion rate is ten times that when it is 2.2. Short term revivals can become noticeable very quickly in their impact on society around them! This contrasts quite significantly with the results of

section 5.3. When the church is a larger proportion of the population then, if a revival occurs and other conditions remain the same, it is likely to be faster and more intense. This fact appears to be born out by history when the revivals of the 18th century in a numerically weak church are contrasted with the much faster ones of the following two centuries when the church was stronger.

5.5 Short Term Revival - Local View

The same equations can be applied at a local level to a single congregation. Here the total number N will be the number of people the church has access to - its parish or catchment area. One congregation is a smaller proportion of the population than the whole church because most areas contain a number of congregations. Let a congregation be 2% of the population and assume revival has come to all its members so that $i_0 = 2\%$, $s_0 =$

98% and $r_0 = 0$. The threshold for a revival is now $\hat{p} = 1.02$. Again take the infectious period to be short, $D = 0.1$.

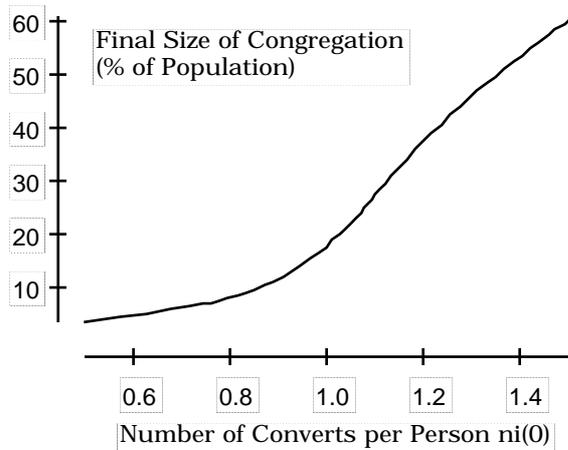


Fig 8 Growth of a Single Church - Short Term Revival

Even with $n_i(0)$ under the threshold significant growth with converts taken from the surrounding community is possible. Figure 8 shows the ultimate size of the congregation as a percentage of the population. Growth up to 20% from 2% is possible without it being strictly a revival in the sense that the rate of increase of infected people is increasing. However the church is starting with all its members infected. The period that gets to this point would

need to be modelled as well.

Once the threshold value is exceeded the growth is explosive; again it is very sensitive to the number of people converted per person. Figure 9 shows that the time period for growth starts going down once the threshold is exceeded. Thus once revival conditions prevail not only is the growth higher but the speed at which it takes place is faster. For $n_i(0) = 1.4$ the congregation goes from 2% to 60% of the community in under two years!

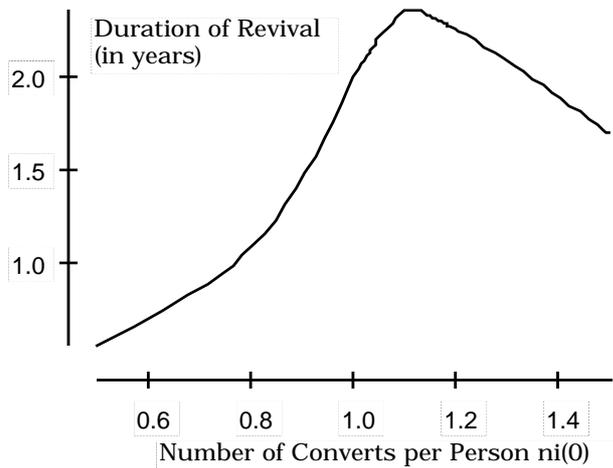


Fig 9 Duration of a Short Term Revival in a Single Church

There are examples where churches have made such a dramatic impact on their community in the past. However it must be realised that an average of 1.4 converts per person throughout a whole of the congregation is a huge figure rarely achieved. The reality is that a small number are responsible for most of the converts. The average figure then becomes difficult to achieve because individuals do not have contacts throughout the community. Individuals do not homogeneously mix in the population.

6 Conclusion

6.1 Main Conclusions

The primary aim of this report was to investigate whether population models, in particular the epidemic model, could be used to model a growing church. As shown in sections 4 and 5 the results of the model do exhibit typical church growth behaviour, particularly that seen during revival. Further the construction of the equations can be explained in terms of the dynamical process that take place between non-believers and the two categories of believers, albeit a highly simplified model. The mass action principle is well suited to modelling the dynamics of conversion. In general it can be concluded that the epidemic model is a suitable starting point for investigating the dynamics of church growth.

A number of specific conclusions can also be drawn from investigating the effects of changing parameters and initial conditions :

- 1 Improving the effectiveness of believers in evangelism has a more significant effect than increasing the number of evangelisers. Whether this has any implications for evangelism training is not clear. It may not be very easy to improve a person's evangelistic effectiveness. However it does help explain why revivals can start with such low numbers of infected believers. If the effective conversion rate increases by only modest amount, either by changes in the infected believers, or changes in the non-believers' receptivity, then growth can very quickly take off.
- 2 If the Christian church is a small proportion of the whole population, and infected believers a small proportion of the church, then revival is possible but its build up tends to be slow. The bulk of the converts in a revival come in its middle period, given that all parameters remain the same. Thus if a revival lasts 20 years the number of converts over the first 5 years may not be that noticeable. The current phenomena referred to as the "Toronto Blessing" has many of the hallmarks of a revival, but as yet no large number of conversions have been seen in the wider church. As noted earlier this has led many to refrain from calling it a revival. However, given the low numbers in the church in western countries, any revival is likely to be long term, with the characteristic slow build up. This current phenomena is 18 months old world-wide, less in the UK, thus it may need another 3 years or more before there is enough of an effect on church attendance and membership to be able to safely class it as a revival. However if it turns out to be a revival, then, dynamically, it already is one now!
- 3 When the church is a larger proportion of the population, a higher conversion rate among infected believers is needed for revivals to occur. Given other parameters remain the same such revivals are shorter than those in countries where the church is weak. The larger the revival the faster it occurs, thus for a large revival the number of conversions at its peak can be very dramatic.
- 4 Revivals can burn out for dynamical reasons, i.e. the number of susceptibles falls to a level where the conversion rate proves inadequate to sustain a revival. The longer a revival goes on the harder it is to

keep going because there are less non-believers. However it will stop before all non-believers are reached. It does not gradually get slower until everyone is converted. Of course revivals can end for other reasons, such as infected believers being "less infected". In this model this is represented by changes in the parameters of the model.

- 5 Significant growth can take place, even without it being classed as a revival. This is especially possible if a large number in the church can be placed in an infected condition, much easier for a single congregation than the church at large.

6.2 Further Work

A number of features need to be added to this model for it to be realistic in all situations :

- 1 Births and deaths - for growth in the long term.
- 2 Transfer growth - for modelling individual congregations.
- 3 Further categories of people - there may be more than one category of unbeliever some more resistant than others. Believers could be split into attenders and church members, simply because this is the sort of statistical information available.
- 4 Reversion - i.e. some believers revert back to being unbelievers, these may count as a separate class of unbeliever, easier to bring back in.
- 5 Re-infection - i.e. inactive believers become infected again. This is essential if the early phase of a revival is to be modelled.
- 6 Age groups - different age groups evangelise differently. Thus a church of young people may well stay young because it attracts other young people.
- 7 Evangelism which doesn't depend on personal contact - e.g. converts through radio broadcasts. This will no longer be a mass action principle.
8. Social Networks - much evangelism is along lines of social contacts (Holloway, 1989, pp24-27). Much research is currently being done on the effect of networks in spreading HIV.

The simple church growth model will be extended in future publications.

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Glossary

Dynamical variables :

t	Time
S	Number of susceptibles (or non-believers)
I	Number of infectives (or infected believers)
R	Number removed from epidemic (or inactive believers)
N	Total number of population under consideration
s	Proportion of susceptibles (non-believers) in total population
i	Proportion of infectives (infected believers) in total population
s_0	Initial proportion of susceptibles (non-believers) in total population
i_0	Initial proportion of infectives (infected believers) in total population
r_0	Initial proportion of removed (inactive believers) in total population

Parameters :

β	Contact rate
D	Duration of Infections
γ	$1/D$
$\lambda(t)$	Force of the infection
$n_i(t)$	The number of susceptibles (non-believers) who are infected (converted) through contact with an infective (infected believer),
$n_i(0)$	The number of susceptibles (non-believers) who are infected (converted) through contact with an infective (infected believer), given whole of population is susceptible.
ρ	Threshold of an epidemic, or revival, in the unscaled equations.
$\hat{\rho}$	Threshold of an epidemic, or revival, in the scaled equations.

Terminology

Believer	A member of a church, also referred to as a church member and a Christian in the report. How membership is defined, or whether all members really are believers, would require more sophisticated models. Also called a convert if they have recently become a believer.
Non-believer	A person who is not a member of a church, a non-Christian or unbeliever .
Infected believer	A believer with a much higher activity in passing on the faith to non-believers. Also called active believer , or enthusiast . They may be engaged in a systematic program of evangelism, or they may be caught up in a "revival-type" behaviour causing them to be effective witnesses. It is assumed in this report that they are new-converts.
Inactive believer	Those removed from the process of winning new converts, at least by comparison with infected believers
Conversion	The process by which a non-believer becomes a believer. This is represented as a transfer from category S to category I. All new converts are assumed infected straightaway. Also referred to in this report as being as a non-believer being brought or led to Christ .
Church	This may refer to an individual congregation , i.e a local group of Christians who meet together on a Sunday under a common leadership. It may also refer to all the people who belong to Christian congregations throughout a country, or the world, regardless of denomination - i.e. the whole church