Mathematical Modeling of Church Growth: An Update

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Abstract

The possibility of using mathematics to model church growth is investigated using ideas from population modeling. It is proposed that a major mechanism of growth is through contact between religious enthusiasts and unbelievers, where the enthusiasts are only enthusiastic for a limited period. After that period they remain church members but less effective in recruitment. This leads to the general epidemic model which is 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 religious revival. This is an updated version of Hayward (1999).

Key Words: Church Growth, Population Models, Diffusion, Differential Equations, Epidemics, 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. Although spiritual growth is included in the subject, numerical growth – how many attend or belong to a church – is a vital area for analysis. The stimulus for investigating the reasons 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 encourage and inhibit growth identified.

Church growth thinking is divided into two strands: the Church Growth Movement, which is based within the denominations and seminaries of the Christian church to serve their needs; and the Social Science strand whose focus is academic research. The two strands have tended to remain separate perhaps reflecting a certain amount of mutual suspicion between them. It is perhaps not surprising that those working within churches distrust sociologists as until recently the prevailing social science view was that religion had no significant place in modern society and would die out – a view often called secularisation theory (Stark and Bainbridge, 1987, pp.13–14; Warner, 1993) The comments of the anthropologist Wallace (1966, p.265) are typical: *"the evolutionary future of religion is extinction"*, as are Berger's remarks that assertions of supernaturalism would be restricted to smaller groups or backward regions (Berger, 1970). From the sociological point of view the church growth strand could also be regarded as suspect, since it is not neutral and often lacks academic rigour.

Many of the principles of the Church Growth Movement were developed at The Fuller Theological Seminary in Pasadena, California, where Donald McGavran became professor of Church Growth. This 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 USA there is the North American Church Growth Association, with a membership from across the denominations, which encourages this way of thinking through its literature. Similar work is undertaken in other countries. Much of the work is qualitative, with quantitative work restricted to data gathering, interpretation and application to churches in the way that business consultants might advise firms. Brierley (1991) in the UK is typical. There is however little attempt at general theories of church growth, only heuristic principles.

The Social Science strand grew very much as a reaction to a key, but controversial, book by Dean Kelley, originally published 1972 and revised in 1986, which put forth an explanation as to why conservative churches are strong. Kelley's thesis, stated simply, was that conservatives churches are strong and hence grow, whereas the more liberal churches decline. This has led to a flourish of research to either prove or disprove this thesis (Hoge and Roozen, 1979; Roozen and Hadaway, 1993).

This history of the two strands, and their relationship, described by Inskeep (1993). One clear distinction between them has emerged. The church growth work tends to view growth mainly influence by factors with the churches themselves – institutional factors, whereas the social science

strand views growth as primarily determined by conditions in the surrounding society – contextual factors. However the common factor of both strands is their desire to understand how churches grow.

Numerous authors have noted that in the USA the Christian churches, as well as other religions, continue to grow despite the predictions of secularisation theory. This has led to the beginnings of a paradigm shift in thinking from secularisation theory, as typified by Berger (1969) , towards one which sees religions flourishing in what is essentially an open market religious economy. This fundamental change is described by Warner (1993), and is typified by the work of Stark (Stark, 1996; Stark and Bainbridge, 1985; 1987; Fink and Stark 1992) and Iannaccone (Iannaccone, 1992; 1994; Iannaccone, Olson, and Stark, 1995), among others. Indeed Iannaccone, using a model based on rational choice theory, affirms Kelley's thesis that strictness, makes churches strong, even in modern society. This has implications for the study of church growth as it becomes increasingly accepted that religious revivals are not only facts of history, but continue to take place in modern society among all classes (Stark and Bainbridge, 1985, ch.9; Stark and Iannaccone, 1994; Warner, 1993, pp.1046–1048).

Much of church growth modelling tends to be statistical, Doyle and Kelley (1979) is typical. 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. Theories have been expressed qualitatively and tested against data (for example see Hoge (1979). Stark comes closer to a theory by computing arithmetically the implications of exponential growth (Stark and Bainbridge, 1985, ch.16; Stark, 1996, p.7). More recently Iannaccone et. al. (1995) have produced a theory of church growth based on the variables of time and money using economic production functions. However none of these approaches attempts to model the dynamics of church growth in terms of the underlying causes. The main aim of this paper is to produce such a model of church growth, using mathematics, which will describe the dynamics of the growth process. It is hoped that such models will give a deeper understanding of the way in which churches grow.

1.2 Types of Models

The next step is to consider what sort of models should 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, in this paper, the only feature modeled 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 investigating if there are similar behaviour patterns to church growth in other areas of population modeling. This paper 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 may be summarised:

- There are at least two categories of people: those who have the disease – or belief, in the church growth case – 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 airborne 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. In the churches this is usually referred to as religious "revival". For example in Wales, UK in 1904–5 100,000 people were added to the main Welsh denominations (Evans, 1969, p.146), only to be followed by a period of slower growth and eventual decline. Over a longer period South and Latin America, Africa and some Asian countries have seen a huge growth in the churches this century, which shows no sign of slowing down.

- During times of revival people are noticeably different, particularly in regard to their enthusiasm to communicate their beliefs to others. Their behaviour is affected. 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" (Lloyd-Jones 1984. pp.60–61) as if it were a disease that could be caught! Those involved in revivals have described them as "contagious", being spread from congregation to congregation (Edwards, 1990, p.89).

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

1.3 Diffusion in Populations

The diffusion process can occur in wide variety of physical, biological and social systems. As such there is a wealth of literature covering models in these areas which may be deterministic or stochastic and may include spatial spread. The model in this paper is in the style of deterministic non-spatial modelling. Banks (1994) and Murray (1989) review a range of such mathematical models and their applications. In the case of church growth the religious belief is being diffused through a population. Thus church growth is a form of social diffusion. Early mathematical models of social diffusion were studied by Coleman (1964) and applied to the spread of medical innovations. Kumar and Kumar (1992) and Mahajan et. al. (1990) review more recent work. Sociological models of innovation diffusion are described non-mathematically by Rogers (1995).

Most of the above models are variations on the logistic model of population growth. These models assume that those possessing the innovation (adopters) are responsible for its spread through contact with those without (potential adopters). However the models also assume that adopters continue to spread the innovation until it is adopted by all potential adopters, although the coefficient of influence may decline. This will be deemed to restrictive for modelling the spread of religion, as enthusiasm for spreading the faith not only wanes but effectively ceases to exist for many within the church. The fact that religious belief never spreads throughout a population lends weight to the need to limit the process of spread. Thus church growth modelling will be social diffusion where the enthusiasm to spread the "innovation" by those in possession of it is limited in duration. This leads to a third category of people who are removed from the spreading process. The need for this dropping-out effect in social diffusion was noted by Webber (1972, p.231), and by Granoveter and Soong (1983) in the context of the spread of fashion, rumours and riots. In Granoveter's model the drop-out was determined by a threshold, with the adopters giving up the adoption. In the church growth case the drop-out will be determined by a period of time, following Webber (1972), with the adopters remaining in the church but now ineffective in spreading the innovation. This is the epidemic model.

The use of the epidemic model in social diffusion was proposed by Bartholomew (1967, ch.8) to model stochastically the spread of a rumour through a population. The model was extended by Sharif and Ramanathan (1982) to incorporate other diffusion effects. They applied the epidemic model to the adoption, and then rejection, of black and white TV sets due to the rise of colour TV. However epidemic type models are not generally used in technological diffusion as the models are deemed too cumbersome, or have too many parameters for the limited available data (Mahajan et.al., 1990, p.13).

1.4 Aims of Church Growth Modeling

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. It is tempting to fit models to actual data, however the complexity of the underlying effects may make identification of the processes difficult. For example, attempts to interpret USA church growth data in terms of revival or lifecycle effects have proved controversial, (see Miller and Nakumara (1996) and references therein) and demonstrates the difficulties involved.

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

1. Mathematical models can provide principles rather than numbers. An example of this is seen in the predator-prey model originally developed by Lokta and Volterra. There are few cases where the model fits well 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, without actual data being 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 should be gathered to best measure a church's effectiveness.

4. The model can help explain why there is such a 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 1858–9 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 (Robinson, 1993; Wimber, 1994). As will be shown later in this paper, 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.

1.5 Overview of Paper

The main aims of this paper are:

1. To show that mathematics can be used to model the dynamics of growth in churches.
2. To investigate the claim that conversion growth is proportional to contact between unbelievers and active, or infected, believers (also called enthusiasts). That is the spread of religion is a form of interactive diffusion.
3. To investigate the claim that the enthusiastic, or recruitment, phase of active believers is limited in duration, after which time they become effectively removed from the conversion process. That is those who diffuse the religious innovation do so only for a limited period. The removed are called inactive believers.

The epidemic model is constructed from its foundations in section 2, with some simple conclusions presented in section 3. The justification for applying the epidemic model to church growth, together with its two claims (aims 2 and 3), is given in section 4. The model is investigated for a number of typical church growth situations, and compared with data from a past revival, in section 5.

2 General Epidemic Model – Construction

2.1 General Assumptions

Although epidemic models are well understood (Anderson and May, 1987; Bailey, 1975), the development and results of the basic three compartment model used in epidemic theory give important insight into its church growth application. Thus a simplified version of the development is given here. A fuller version can be found in Anderson and May (1987).

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 glossary of all symbols relating to the epidemic and church growth models appears in appendix A.

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. This is represented diagrammatically in figure 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, p.65; Bartholomew, 1967, pp.215f, 247f) of these is homogeneous mixing. That is, the infectives are well mixed throughout the susceptibles. (Other forms of contact can be considered, e.g. Anderson (1988), takes into account different degrees of contact amongst susceptibles and infectives.) 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, 1987):

$$\frac{dS}{dt} = -\lambda(t)S \tag{1}$$

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. (This is referred to as R Anderson and May, 1987, p.17). The net change in the number of infectives during that period because of that single infective 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:

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

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:

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

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 the period people are infectious the more people 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 (STDs).

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. Thus a larger population will lead to more personal contacts, that is a network space of greater density. 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 (Open University, 1988), also known as mass action (Hethcote,1994), or density dependent (McCallum et. al., 2001).

Thus the form of $n_i(t)$ is:

$$n_i(t) = \beta DS \tag{4}$$

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):

$$\frac{dS}{dt} = -\beta SI \tag{5}$$

$$\frac{dI}{dt} = \beta SI - \gamma I \tag{6}$$

$$\frac{dR}{dt} = \gamma I \tag{7}$$

Equation 7 comes from 5 and 6 and the fact that the total population N 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 STDs 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. Thus increasing the population size will not change the number infected by one infective if the ratio of infectives to susceptibles stays the same; the system scales with population size. In these cases the second assumption for $n_i(t)$ is modified to $n_i(t) = S/N$, 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. Thus:

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

where β is a different constant from the first model, and represents the number infected by one infective per unit time. Equation 8 with equations 1 to 3 give the general epidemic equations familiar in the study of the spread of STDs and HIV/Aids (Anderson (1988) :

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

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

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

This is referred to as the fixed contacts model, also known as standard incidence, and frequency dependent (McCallum, 2001).

For most infectious diseases the crowd model is usually suitable, although the truth often lies somewhere between the two models, the number of contacts an infective has depends on S but not linearly. Various attempts have been made to construct a more sophisticated model of the transfer from S to I (May and Anderson, 1985), however the two models 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.

It was originally argued in Hayward (1999) that the Crowd Model is the most applicable in church growth, and as such it is used for the remainder of this paper. However in later publications it has been shown that the Fixed Contacts Model is more relevant as most churches are in communities where increasing the community size does not increase the number of possible contacts a person has, because the communities are already much larger than the maximum effective friendship or contact network. The effect on this paper is minimal as the scaled equations used in section 5 are independent of the model type due to a constant total population.

3 General 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:

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

where ρ is referred to as the threshold of the epidemic. This can be demonstrated by illustrating the phase paths of I against S (figure 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 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 example is that of the Eyam plague of 1665–6 given by 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

In the early stages of an epidemic, when the number of infectives and removed are very small compared with the total population ($S \simeq N$), 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:

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

This result is particularly useful in estimating β if data is available for those stages, e.g. Anderson (1988), and May and Anderson (1987) 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 $(S, 0, N - S)$ as stable equilibrium points for any S. 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 of 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:

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

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:

$$\frac{dS_\infty}{dI_0} = \left(1 - \frac{\rho}{S_\infty}\right)^{-1} \tag{16}$$

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

For a small epidemic Kermack and McKendrick (1927) derived a simpler result from equation 16 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:

$$S_0 - \rho \simeq \rho - S_\infty \tag{17}$$

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.

3.5 Unlimited Infectious Period

When the duration of the infectious period, D, becomes very long compared to other timescales it can be treated as effectively unlimited and $\gamma \to \infty$. In this case the system reduces to two equations in S and I only with $N = S + I$ a constant. Equation 6 becomes the logistic equation:

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

Thus the epidemic eventually spreads through whole susceptible population, as in the standard models of cultural diffusion. This is sometimes referred to as the simple epidemic model.

4 The Limited Enthusiasm Church Growth Model[1]

4.1 Use of the General Epidemic Model

a. The general 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: 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. The rigid adherence to a distinct lifestyle has been recognised as an important feature of growing churches (Kelley, 1986). Thus a convert can be easily distinguished from an unbeliever just as a person with an infection can be distinguished from a susceptible. (The use of terms such as unbeliever and convert in the limited enthusiasm model are explained in appendix 2.). Further Hadaway (1993a) notes that enthusiasm among church members has a significant effect on attendance, with effects waning as enthusiasm wanes.

b. Most conversions occur because of a contact between an active believer and an unbeliever, often via an interpersonal bond (Stark and Bainbridge, 1985, pp.309, 355). This active believer will be called an *enthusiast*[2]. The enthusiast may "lead someone to Christ" – the conventional expression used when a believer is instrumental in another person's conversion. However the contact may simply be that an enthusiast takes the person to a church meeting or evangelistic campaign subsequently leading to a conversion at the hands of others. The growth in the church is proportional to the contacts between an enthusiast and unbelievers, just as the spread of an infectious disease is proportional to the number of contacts between infectives and susceptibles. Hadaway (1993b) notes that evangelism is an important predictor of church

[1]The name of the model has been updated to bring it in to line with later publications

[2]The name enthusiast was originally a derogatory term applied to people taken up with religion. In particular it was a nickname applied the the first Methodists in the 18th century who were instrumental in the conversion of others to the faith.

growth. It is this contact process that slows down the growth into a logistic behaviour. Without this process growth within a fixed size population becomes unrealistically exponential as Stark and Bainbridge (1985, p.349) note in their example of cult growth.

c. Not all people in the church are responsible for spreading the faith, i.e. not all are enthusiasts, using this paper's definition. Indeed in most churches only a small proportion of believers are involved in passing on their beliefs. For example, even in a highly successful "Cell" Church 65% of the membership being actively involved in the conversion process is deemed a very high figure (Neighbour, 1990), no doubt a key factor in their growth. For conventional churches the figure is more likely to be less then 10%.

   Thus as well as enthusiasts (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 (Stark and Bainbridge, 1985, p.363). 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. This reduction of effectiveness is part of the process of secularistion that many new churches undergo as they becoming more accommodating to the surrounding society (Stark and Bainbridge, 1985, p.100, ch.19). However it will be too simplistic to think of the removed as "secularised" believers. It is purely the recruitment potential that is limited. The reasons for this drop of enthusiasm are varied, but are usually summed up in Wesley's Law of the decline of pure religion (Kelley, 1986, p.55). Essentially the "law" says that taking up a new religion produces benefits, spiritual or material, perhaps in the form of new friends, or respect, thus making missionary zeal more costly to engage in. It becomes easier to be devoted to work *within* the church, rather than *without*. The enthusiasm can also be the result of an experience whose effects decline after a short period.

   Sometimes it is not a lack of enthusiasm that causes a drop in a believer's usefulness. After a while most new converts find that they have exhausted their network of non-Christian contacts. Some will cease to be part of that network as the new convert exchanges old friends for new Christian ones in their church (Olson, 1989).

   It is this process of a limited recruitment period that causes a church to run out of potential converts as eventually happened to the early Christian church (Stark, 1996, pp.12–13). It prevents a church from eventually taking over an entire population, leaving sections of the population untouched regardless of birth and death effects.

d. 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, pp.105–106). Not all church growth is like this, neither do all diseases spread like this; there are endemic infections. However epidemics and revival church growth share these dynamical features.

A number of different processes can be identified as causes of growth and decline in an individual 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 one church having left another). All three have their opposite in terms of decay: death, reversion and transfer out. In addition there are those who having left the church are *restored* back. These processes are explained in Pointer (1987, pp.19–22).

For the main part of this paper the timescale will be chosen so that births and deaths can be ignored to a first approximation, that is no biological growth 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 mainly applied to the church as a whole rather than one small part of it.

Thus the limited enthusiasm church growth model is the general 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 general epidemic model is a suitable starting point to analyse church growth, the variables are easily identified:

> S, the number of susceptibles, are those not in the church, unbelievers, with whom the church members have contact. Isolated susceptibles are not part of the dynamics of growth.
>
> I, the infectives, are the number of enthusiasts, or "infected" believers, within the church who are active in spreading the faith, i.e. in making contacts with unbelievers that lead to their conversion.
>
> R, the removed, are 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 enthusiasts and unbelievers. This depends on $n_i(t)$ the number of converts (not contacts) one enthusiast (infected believer) is responsible for during the whole of their infectious period before they drop down to the lower level of activity characterised by the removed category. β may be small because the church is in high tension with society permitting very few contacts (Stark and Bainbridge, 1985, p.136). However β could also be small because the church is so much like society it has nothing to offer, and although it has many contacts, few are effective (Kelley, 1986, ch.6). For example the exclusive nature of early Christianity made it far more effective than the moderate pagan religions, even though the pagans had more actual contacts (Stark, 1996, p.204f).

$D(= 1/\gamma)$ is the length of time a believer remains infected or enthusiastic. These parameters may depend on a large number of sociological factors in the surrounding society, or in church (Hoge and Roozen, 1979) as well as psychological and spiritual factors in the believer and unbeliever (Wagner, 1987). However it is assumed that for large enough numbers they remain constant over a period of time. A change in one of the underlying factors will result in a change in one these parameters and hence in the dynamics of the church growth. This is discussed in section 5.4. The dynamical model should be relevant for situations where growth depends on social context, or institutional factors, or both.

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 and the fixed contacts model. To decide which model is more appropriate the transmission mechanism between an enthusiast and an unbeliever needs to be identified. The key question is: if the population of unbelievers is increased will the church grow faster through conversion, i.e. will an enthusiast be responsible for more conversions? If the church is a significant proportion of the population the answer is generally "yes" and thus the crowd model is more appropriate. Consider the following transmission mechanisms:

(i) The enthusiast 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. the crowd model. There are two exceptions. The first will be if the unbelieving population is huge compared to the church, and the church's resources are fully stretched in its evangelism program. In this case a larger population will not mean more contacts even over a long period of time, thus the fixed contacts model is more suitable. The second exception will be if enthusiasts deliberately seek out suitable 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 in the Christian church although often found in cults.

(ii) The enthusiasts evangelise through their network of contacts. Such social networks are seen as a major means of spreading the Christian faith (Olson, 1989; Stark and Bainbridge, 1985, p.312f). This network is unlikely to be larger if the population increases – there are only so many friends and acquaintances a person can hold down; this would imply the fixed contact model. 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 some support for the crowd model.

(iii) The enthusiasts 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 enthusiasts have never met may seek them out simply because of news about them, and their behaviour, has reached those people (Edwards, 1990, pp.90–91). Again this leads to an increased number of contacts in a larger population, hence the crowd model.

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 general 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 general epidemic model. These can be applied to the limited enthusiasm church growth model.

Epidemic Threshold. There is a threshold above which significant church growth, or revival growth, will take place, given by equation 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 modeling. However the *number* of enthusiasts does not determine whether growth will take place or not. A small church is as equally likely to see revival growth as a larger one if their enthusiasts are equally effective; 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. 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 in a logistic fashion.

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 unbelievers and the threshold.

Permanent Enthusiasm. If enthusiasm is not limited in duration then religious belief spreads along the lines of classic social diffusion and eventually covers the entire population. It is the limitation of enthusiasm that prevents the whole susceptible population being ultimately converted.

An example is helpful to illustrate the last point. The expression for the threshold ρ, equation 12, can be rewritten for the crowd model as: $\rho = S/n_i(t)$, using equation 4. Let the church be in a population of say $50,000$. As an average, let 10 enthusiasts be 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! However 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.

Further, the churches may not be in effective contact with a significant proportion of the population of 50,000 for reasons of geographic location, class, race etc. Thus the actual growth is smaller, drawn from the church members' circle of influence only.

5 Numerical Solutions

5.1 Scaled Equations

The limited enthusiasm 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. This also has the advantage of transforming the equations into ones where the relevant church growth parameters appear explicitly.

The simplest scaling is to let the new variables i and s be the proportion of enthusiasts and unbelievers respectively in the whole population:

$$\begin{aligned} i &= \frac{I}{N} \\ s &= \frac{S}{N} \end{aligned} \tag{18}$$

Substituting these into equations 5 and 6 for the crowd model gives:

$$\frac{ds}{dt} = -\beta N s i \tag{19}$$

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

Let $n_i(0) = \beta N/\gamma$ the number of unbelievers converted through one enthusiast 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:

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

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

The threshold of the epidemic type growth now becomes:

$$s_0 > \frac{1}{n_i(0)} \triangleq \hat{\rho} \tag{23}$$

This will be the threshold for revival growth to occur.

Exactly the same scaled equations result for the fixed contacts model 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 the crowd model the parameters $n_i(0)$and D will be determined for a particular community, whereas in the fixed contacts model they are independent of the community.

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

5.2 Increasing the Effectiveness of an Evangeliser

One aim of evangelistic programs is to increase a believer's effective witness. One approach is to train people to explain the gospel effectively. Many methods are taught throughout the church, a number of which are reviewed by Green (Green, 1990, part 3).

The effective witness can also be improved by increasing the number of contacts with unbelievers. Two models of church organisation that attempt to achieve this are the Seeker Church model, pioneered by the Willow Creek Community Church near Chicago (Robinson, 1995), and the Cell Church model. Examples of the latter include the Yoido Full Gospel Church in Seoul, Korea, and the underground church in China, both of which have seen huge growth in recent years. Cell Church methods are explained by Neighbour (1990).

The idea behind both approaches, which can be employed together, is that, all other things being equal, a believer who has been so trained will be responsible for more conversions. Such people are candidates for being treated as enthusiasts, and will be called evangelisers in this paper. (The term evangelist has a more technical meaning within the Christian church.)

Assume that the effectiveness of such a method is medium term say $D = 2$ years, i.e. a believer loses their evangelistic impact two years after conversion on average. Assume also that the number of enthusiasts 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 figure 3. Note the effect is near exponential. (This effect was computed on arithmetic arguments by Stark and Bainbridge (1985, p.355), although it was not explicitly stated.) The benefits from doubling the effectiveness of an individual believer is to more than double the growth rate of the church.

5.3 Increasing The 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 enthusiasts 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 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 $\propto i_0 exp(at)$, 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 enthusiasts.

5.4 Medium Term Revival

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 limited

enthusiasm church growth model doesn't contain. However the model will give some indication of the later stages of a revival when contact with unbelievers becomes the dominant behaviour. Believers affected by a revival spread the gospel with considerably increased enthusiasm. Such believers are candidates for being enthusiasts, those "infected" by the revival.

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), revival growth will occur if the threshold $\hat{\rho} = 1/n_i(0)$ is exceeded by the initial percentage of unbelievers 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 with the number of enthusiasts increasing. Assume that $n_i(0) = 1.15$, just in excess of the threshold, with believers only infected for a short period of $D = .1$years. Thus converts make a significant impact on unbelievers for only a short period after their conversion.

Typically revivals in a country start with a small number of infected believers (Lloyd-Jones, 1986, pp.163–166). Let $i_0 = .01\%$ i.e. only one in a thousand of the church are so affected. The resulting growth of the church is given in figure 5.

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 noticed 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 and the USA were faster, but the church was a much larger proportion of the population. This will be investigated in section 5.5.

Another significant result is that the revival is ending due to dynamical effects dependent on its initial intensity, and the fact that a believer's enthusiastic phase is limited. 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 their 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 enthusiast $n_i(0)$, that is increase the number of effective contacts between an enthusiast and an unbeliever. 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 by the Christian Church to be a significant cause of a revival taking place. Theologically a revival is regarded as an "act of God" which turns believers into effective witnesses and makes unbelievers responsive to that witness (Lloyd-Jones, 1986, pp.50, 56–57, 106, 233–236).

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 the time t. Although having a limited duration to the enthusiastic period limits the growth to a number less than the whole population, its value does not effect the amount of growth. Over longer periods where births and deaths become significant, this result will no longer apply.

5.5 Short Term Revival

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 revival growth to occur is now higher, $\hat{\rho} = 2$, thus more converts per unbeliever are required for revival growth to occur. 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 revival growth can occur albeit with a larger value of $n_i(0)$.

For example, keep the number of enthusiasts initially at 1 in 1000 of the church, thus $s_0 = 0.5$ and $i_0 = 0.0005$. Keep the effective period $D = 0.1$ year. For $n_i(0) = 2.1$ the revival growth 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.

The larger the value of $n_i(0)$ the faster the revival growth 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 be become noticeable very quickly in their impact on society around them! This contrasts quite significantly with the results of section 5.4. 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.

5.6 Estimation of Parameters from Data

Using real data from churches poses some considerable challenges. Most churches keep a record of membership numbers, but the meaning of membership varies. At one extreme there are very strict protestant churches, such as the Methodists in the 18th century, where evidence of conversion must be shown, and membership is discontinued if commitment is lacking. At the other extreme some are very lax, such as the Roman Catholic church, where all in the religious community are members regardless of commitment. Attendance is much higher than membership in the strict churches, and much lower in the lax churches. Thus membership is rarely a measure of religious attendance, its relationship to attendance will differ between different churches and over time. Until recently few churches recorded attendance, except at untypical times such as Easter.

Revivals thrive on anecdotal evidence but reliable data is hard to come by. Even the data collecting

that does take place may prove unreliable during a revival as individual churches are otherwise distracted by events in their midst. It should also be noted that following a revival new religious groups get formed for whom no data is available, thus an accurate picture is impossible to achieve.

However an estimate can be attempted for the revival that took place in Wales in 1904–5. Annual membership figures are available for all churches apart from the Anglican church, for whom communicant figures are available (Williams, 1985). These are used as an estimate of membership. For 1904 the combined adult total for churches in Wales stood at 48.94% of the total Welsh adult population. Prior to this date the percentage had been falling very slowly, until in 1904 it had risen 1 percentage point from 1903. By the end of 1905 the percentage of people in membership of Welsh churches had risen to 53.43% of the total adult population, where it has had to be assumed that the Anglican church increased by the same percentage as the other churches. (It changing its method of measuring communicant numbers in 1905.) Anecdotal evidence would support this assumption. The total adult population had also increased over the year by 2% to stand at $1,446,447$.

The revival started in October of 1904 in at least two separate geographical locations with a very small number of people in each. A number of other churches had become involved in the revival by the end of 1904. Assume that about 1 in 500 of church people had become enthusiasts for the revival by the beginning of 1905, i.e. about $1,500$ people. The bulk of the converts came in the next 12 months, so this will be taken as the duration of the revival. The basic church growth model gives $n_i(0) = 2.02$ (i.e. $n_i(t) \approx 1$ as the church is about 50% of the population) with a duration $D = 1.04$ weeks. Thus each enthusiastic believer, on average, was responsible for bringing 1 more into the churches in the space of a week. If the number of initial infectives at the beginning of 1905 is underestimated then the value of $n_i(0)$ remains about the same but the enthusiastic period becomes shorter, that is, enthusiasts have to bring in new people faster. The duration of the enthusiastic phase is very short, much shorter than can be explained by any process of secularisation.

Note that initial number of susceptibles 51.06% starts above the threshold of $1/2.02 = 0.495$ and falls to a similar level below 46.57%. Indeed with about half the population churchgoers $n_i(0)$ would have to be at least 2 converts per person for a revival to take place – what ever the time scale. It is very likely that this value for $n_i(0)$ is only an average and that a number of enthusiasts were not responsible for anyone joining the church, and a smaller number were responsible for more than 2, but over a longer period. However this would require a more sophisticated model than the basic church growth one, and it is unlikely that any data from this period could discover these variations.

6 Conclusion

6.1 Main Conclusions

The primary aim of this paper was to investigate whether population models, in particular the epidemic model with its spread by contact and limited infectious period, 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 processes that take place between unbelievers and the two categories of believers, albeit a highly simplified model. The mass action principle (personal contacts) is well suited to modeling the dynamics of conversion, and provides the typically S-shaped behaviour found in the growth of churches. The limited duration for the enthusiastic, or recruitment, phase effectively prevents the church from growing to the whole population. 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 enthusiasts, or changes in the unbelievers' receptivity, then growth can very quickly take off.

2. If the Christian church is a small proportion of the whole population, and enthusiasts a small proportion of the church, then revival growth 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. This is a direct result of the logistic behaviour of the growth. 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 unambiguously measured 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, as such it is too early see substantial growth in a religious phenomena that is less than five years old.

3. When the church is a larger proportion of the population, a higher conversion rate among enthusiasts is needed for revival growth 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 revival growth continues the harder it is to keep going because there are less unbelievers. This is a direct result of the spread by contact, with the enthusiasts effort being increasingly "wasted" on those already converted.

   However the growth will stop before all unbelievers are reached. It does not gradually get slower until everyone is converted, this being a direct result of limiting the enthusiastic, or recruitment, phase of a believer. 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. Revivals do not end because the people involved in it become more secularised. Typical timescales for enthusiastic periods are very short, whereas secularisation is a long term process. It is quite possible the lack of enthusiasm and the inability to win converts contributes to the secularisation of the church (Stark and Bainbridge, 1985, p.364).

6. Because the model concentrates on the dynamics of the growth it is independent of whether the underlying factors which effect growth are institutional, social or theological. Of course

if any of parameters change in time then the relative merits of these factors would need to be investigated.

6.2 Further Work

The limited enthusiast church model needs to be extended to account for other mechanisms for growth, as outlined in section 4.1.

1. Growth through births and deaths are needed to model long term behaviour. As well as the supply of unbelievers being replenished, the children of believers may automatically enter the church without ever being classed as unbelievers or infected. Children of believers often have less interest in enthusiastic religion than their parents, are more socialised, and hence push the church towards ineffectiveness (Stark and Bainbridge, 1985, pp.24, 157–165).

2. Transfer growth exists between individual congregations, and between denominations. This opens up the possible need to model competition between different sorts of churches. This was characteristic of the early spread of Christianity as it competed with paganism (Stark, 1996, p.191), and is particularly true in the USA and Europe which are highly pluralistic religious markets (Fink and Stark, 1992; Iannaccone, 1991; Stark and Bainbridge, 1985, p.353; Stark and Iannaccone, 1994).

3. Churches may decline through reversion, i.e. some believers revert back to being unbelievers.

4. Inactive believers can become enthusiastic again, i.e. they are re-infected from existing enthusiasts. This process is essential if the early phase of a revival is to be modelled.

5. Further categories of people can be considered. There may be more than one category of unbeliever some less resistant than others to conversion, thus creating possible pools of potential recruits (Stark and Bainbridge, 1985, p.351f). Secondary conversions such as the spouses of initial converts may have far less enthusiasm (Stark, 1996, p.100) and be a separate category of believer.

6. The susceptible pool is not strictly homogeneous as converts often come through existing social networks (Stark and Bainbridge, 1985, p.312f).

The model will be extended in future publications.

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A Glossary

Dynamical variables:

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

Parameters:

β	Contact rate
D	Duration of Infection or enthusiastic period
γ	$1/D$
$\lambda(t)$	Force of the infection
$n_i(t)$	The number of susceptibles (unbelievers) who are infected (converted) through contact with an infective (enthusiast)
$n_i(0)$	The number of susceptibles (unbelievers) who are infected (converted) through contact with an infective (enthusiast), given whole of population is susceptible. This is the reproductive ratio of epidemiology. The reproduction potential.
ρ	Threshold of an epidemic, or revival, in the unscaled equations.
$\hat{\rho}$	Threshold of an epidemic, or revival, in the scaled equations.

B Terminology

Believer: A member of a church, also referred to as a **church member** and a **Christian** in the paper. How membership is defined, or whether all members really are believers, or whether all attenders are members, would require more sophisticated models. A believer is also called a **convert** if they have recently become a believer.

Unbeliever: A person who is not a member of a church, **a non-Christian**.

Enthusiast: A believer with a much higher activity in passing on the faith to unbelievers. Also called **active believer**, or **infected believer**. They may be engaged in a systematic program of evangelism i.e. an **evangeliser**, or they may be caught up in a "revival-type" behaviour causing them to be effective witnesses. It is assumed in this paper 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 an unbeliever becomes a believer. This is represented as a transfer from category S to category I. All new converts are assumed infected immediately. Also referred to in this report as being as an unbeliever 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**. The size of the church can be measured by attendance at the main services or by an official system of membership.

C Revised Notation

Hayward (1999), essentially this paper, was the first publication on the mathematics of church growth, based on a technical report of 1995. Later publications have undergone a change of notation, and a greater use of the fixed contacts model, partly as a result of using the System Dynamics methodology. The following expresses the limited enthusiasm model of this paper in the revised notation.

C.1 Fixed Contacts Model

The equations in the new notation equations, 24–26, are the equivalent of equations 9–11. The model is further modified by allowing some converts to become inactive rather than enthusiasts.

$$\frac{dU}{dt} = -\frac{C_p}{N\tau_a}UA \tag{24}$$

$$\frac{dA}{dt} = \frac{gC_p}{N\tau_a}UA - \frac{A}{\tau_a} \tag{25}$$

$$\frac{dB}{dt} = \frac{(1-g)\,C_p}{N\tau_a}UA + \frac{A}{\tau_a} \tag{26}$$

The notational comparison is given in table 1. The conversion potential, the number of people that

Name	Old Notation	New Notation
Unbeliever	S	U
Enthusiast	I	A
Inactive Believer	R	B
Duration of Enthusiastic Period	D	τ_a
Conversion Potential	βD or $n_i(0)$	C_p
fraction converts enthusiasts	not in model	g
Reproduction Potential	not in model	$R_p = gC_p$

Table 1: Notation Change in Fixed Contact Limited Enthusiasm Model

would be converted by one enthusiast during their enthusiastic period given the whole population are unbelievers, C_p, is constant. The reproduction potential R_p, i.e. the number converts made enthusiasts, is more useful. The condition for revival-type growth is $R_p > 1/\bar{U}_0$, where $\bar{U}_0$ is the initial fraction unbelievers.

C.2 Crowd Model

The new equations 27–29 is the equivalent of equations 5–7

$$\frac{dU}{dt} = -\frac{c_p}{\tau_a}UA \tag{27}$$

$$\frac{dA}{dt} = \frac{gc_p}{\tau_a}UA - \frac{A}{\tau_a} \tag{28}$$

$$\frac{dB}{dt} = \frac{(1-g)\,c_p}{\tau_a}UA + \frac{A}{\tau_a} \tag{29}$$

The conversion potential c_p is the equivalent of βD. The reproduction potential is $r_p = gc_p$.

The conversion potential, the *fraction* of people that would be converted by one enthusiast during their enthusiastic period given the whole population are unbelievers, c_p, is constant. Thus in crowd model the number converted is higher in a larger population. This is the density dependence. The condition for revival-type growth is $r_p > 1/U_0$. Thus revival growth depends on the actual number of unbelievers, in contrast to the fixed contacts which is a fraction.