Contingent Claims Analysis Approach for Modelling Suicide Risk in Later Life

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Abstract

Background: Affective disorder is generally regarded as the prominent risk factor for suicide in the old age population. Despite the large number of empirical studies available in the literature, there is no attempt in modelling the dynamics of an individual's level of suicide risk theoretically yet. In particular, a dynamic model which can simulate the time evolution of an individual's level of risk for suicide and provide quantitative estimates of the probability of suicide risk is still lacking. *Aims and Methods*: In the present study we apply the contingent claims analysis approach of credit risk modelling in the field of quantitative finance to derive a theoretical stochastic model for estimation of the probability of suicide risk in later life in terms of a signalling index of affective disorder. Our model is based upon the hypothesis that the current state of affective disorder of a patient can be represented by a signalling index and exhibits stochastic movement and that a threshold of affective disorder, which signifies the occurrence of suicide, exists. *Results and Conclusions*: According to the numerical results, the implications of our model are consistent with the clinical findings. Hence, we believe that such a dynamic model will be essential to the design of effective suicide prevention strategies in the target population of older adults, especially in the primary care setting.

Keywords: Contingent claims analysis, stochastic process, suicide

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1. Introduction

In the past decade suicide has become a serious health problem in our modern societies. The most recent surveys in the United States ranks suicide as the nineth leading cause of mortality, responsible for nearly 31,000 deaths (Peters, et al., 1998). According to a recent World Health Organization report, suicide was found to be the cause of 1.8% of the world's 54 million deaths in 1998 (World Health Organization, 2001). The organization also reported that self-inflicted injuries including suicide accounted for about $814,000$ deaths in 2000. This translates into a global mortality rate of about 15.1 per $100,000$ or one death every 40 seconds. Accordingly, the World Health Organization has urged its member nations to address themselves to the growing problem of suicide (U.S. Public Health Service, 1999).

In the majority of countries suicide rates are found to be higher among older adults than in any other age group. Owing to the current trend of a decreasing number of births in the industrialized world, suicide among the elderly is expected to become a major public health concern in the coming decades. Haas and Hendin estimated that there would be about $14,000$ suicide deaths in the United States in the 55 and over age group by the year 2020 (Haas and Hendin, 1983). Hence, since the release of the National Strategy for Suicide Prevention: Goals and Objectives for Action in May 2001 (U.S. Public Health Service 2001) by the Office of the Surgeon General of the United States, the prevention of suicide in later life has been a major concern of the United States.

Recently, after critically reviewing and evaluating the strength of the evidence from a number of empirical studies for whether correlates of suicide in each of three broad domains — mental health, physical health, and social factors — constitute risk factors for suicide in later life, Conwell et al. argued that affective disorder was the predominant risk factor for suicide in elders (Conwell, et al., 2002). Psychological autopsy studies from many countries also consistently show that more than 90% of suicide victims have one or more axis I major psychiatric disorder at the time of their deaths, and that the percentage for suicide victims over 65 is about 71% to 95% (MoScicki, 1997). This further suggests that affective disorder, in particular depression, is a predictive factor for self-injurious behaviour or suicide, especially among the older age groups (Conwell, et al., 2002; Isometsä, 2000; MoScicki, 1995; and Lawrence, et al., 2000). Thus, it is believed that the proportion of later life suicides would be dramatically reduced provided that affective illness was identified and treated effectively (Isacsson, 2000).

Although affective illness should be the leading target of suicide prevention efforts in the old age population, yet Conwell et al. emphasized that factors in other domains also played an important role in determining an individual's risk for suicide via their intricate interactions with the risk factor of affective illness (Conwell, et al., 2002). For instance, evidence shows that social support variables may both enhance and reduce the suicide risk in older adults, whilst physical illness is a contributing factor to the elevated risk for elderly suicide via depressive disorders. In fact, an individual's level of risk is in constant flux, reflecting the dynamic interaction of influences. Hence, a better understanding of those interactive effects will facilitate more precise preventive interventions.

Affective disorders are common in primary care practice, but often go undiagnosed and inadequately treated. According to the surveys, about 70% of older adults who committed suicide saw their primary care provider within 30 days of death, and more than one-third of older patients have visited their physician within a week (Conwell, 1994). A recent national register-based study of all suicides in Denmark (1981-1997) by Qin et al. also demonstrated that a history of hospitalization for psychiatric disorder was the prominent risk factor for suicide (Qin, et al., 2003). Furthermore, risk was extremely high for those recently discharged from the hospital; about 27% to 37% have been in-patients (King and Barraclough, 1990). Thus, the evaluation of suicide risk by the depression and suicide feelings (life-weariness, death wishes, suicidal thought and attempt) in the primary care setting is very important (Waern, et al., 1999). As the primary care setting is a significant venue for intervention, one important approach to late-life suicide prevention is, therefore, to optimize the ability of primary care providers to diagnose and treat late-life affective disorders and suicidality effectively. Unfortunately, since many primary care providers taking care of the older people lack the knowledge or have unsophisticated psychiatry training, it is very difficult for the primary care providers to determine the degree of risk for suicide accurately (Schulberg and McClelland, 1987).

In spite of the large number of empirical studies available in the literature, there is no attempt in theoretically modelling the dynamics of an individual's level of risk for suicide yet. In particular, a dynamic model which can simulate the time evolution of an individual's level of risk for suicide and provide quantitative estimates of the probability of suicide risk is still lacking. We believe that such a dynamic model will be essential to the design of effective suicide prevention strategies in the target population of older adults, especially in the primary care setting. For instance, it helps to suggest on-going case management procedures for working with suicidal patients so as to offer timely and appropriately targeted recommendation according to a treatment alogrithm. Furthermore, since the demand for risk assessment on people with suicidal tendency is currently the major task in clinical practice and research focus, the model can be useful in streamlining clinical rating results (*e.g.* at triage or priority) and making them more efficient or reliable in decision making, especially in time-sensitive situations. It is thus the purpose of our paper to propose a stochastic model for estimation of the probability of suicide risk. The formulation of our model follows the *contingent claims analysis* of credit risk modelling in the field of quantitative finance (Cossin and Pirotte, 2001). Our model is based upon the hypothesis that the current state of affective disorder of a patient can be represented by a signalling index and exhibits stochastic movement and that a *threshold* of affective disorder, which signifies the occurrence of suicide, exits. The introduction of the signalling index of affective disorder is not foreign in clinical practice (Range and Knott, 1997; and Brink, et al., 2000); in fact, a number of similar risk assessment instruments, *e.g.* Hamilton Rating Scale for Depression (Hamilton, 1960), Beck Depression Inventory (Beck, et al., 1961), Scale for suicide Ideation (Beck, et al., 1979), the SAD PERSON Score (Patterson, et al., 1983), etc., have been developed to assess an individual's suicide potential, and are found to be especially helpful for health workers with limited psychiatric training.

This paper is organized as follows. In section 2 we apply the contingent claims analysis to estimate the probability of suicide risk. Numerical results are presented and discussed in the section 3. Section 4 contains the conclusion.

2. Model

To begin with, we suppose that there exists a *signalling index* X summarizing the current state of affective disorder of a patient. This index X is a dynamical variable and changes with time t. It is justified to assume that suitable treatment will ameliorate the affective disorder of a patient. In addition, a patient's mental health will also be affected by a number of "external random forces", *e.g.* physical health, social factors, other putative risk factors, etc. (Conwell, et al., 2002). All these considerations can be summarized in the stochastic differential equation:

$$\begin{aligned} & dX = -\left(\mu - \frac{1}{2}\sigma^2\right) X dt + \sigma X dZ \\ \Longrightarrow \quad & d(\ln X) = -\mu dt + \sigma dZ \end{aligned} \tag{1}$$

where μX refers to the magnitude of preventive treatment for a patient's affective disorder, and Z represents the Wiener process with a standard deviation σ. The σX quantifies a patient's response to other influences. For simplicity, we assume both μ and σ are constant parameters. [Note: dX, dt and dZ denote infinitesimal changes in X, t and Z, respectively.] Equation (1) indicates that the stochastic signalling index X is assumed to follow a lognormal random process.[3] This is reasonable because the higher the value of a patient's signalling index X is (or equivalently, the more severe the affective disorder of a patient is), the more vulnerable to other influences the patient becomes and the stronger the necessary preventive treatment is. In essence, the stochastic differential equation tells us how the signalling index of a patient changes with time under the influence of both a deterministic effect and a resultant external random force. Furthermore, it is natural to assume that there exists a threshold X_c of the signalling index, which signifies the occurrence of suicide.

The above formulation of our model follows closely the contingent claims analysis of credit risk modelling in the field of quantitative finance. Credit risk models aim at modelling the default risk of a firm by providing reliable quantitative assessment of the firm's probability of default. The essence of the credit risk modelling is that default occurs when the leverage ratio of a firm goes beyond unity, *i.e.* when the asset value

[3]Such a lognormal random process ensures that $X \geqslant 0$.

of a firm is less than its liabilities (Hui and Lo, 2002; Hui, et al., 2003; and Hui, et al., 2003b). This is analogous to the scenario of a patient committing suicide when his/her signalling index of affective disorder goes beyond the threshold X_c.

The novelty of our model is that the estimation of the probability of suicide risk can be solved analytically. Following the standard treatment of stochastic analysis,[4] it is not difficult to write down the density distribution function of the aforesaid random process with an absorbing barrier at $X = X_c$:

$$f(x,t;x_0) = K(x,t;x_0) - K(x,t;-x_0) \cdot \exp(\gamma x_0) \quad , \tag{2}$$

where

$$\begin{aligned} K(x,t;x_0) &= \frac{1}{\sqrt{2\pi\sigma^2 t}} \exp\left\{ -\frac{(x - x_0 + \beta t)^2}{2\sigma^2 t} \right\} \quad , \\ \beta &= \mu \quad , \quad \gamma = \frac{2\mu}{\sigma^2} \quad , \\ x &= \ln\left(\frac{X}{X_c}\right) \quad , \quad x_0 = \ln\left(\frac{X_0}{X_c}\right) \quad . \end{aligned} \tag{3}$$

The density distribution function $f(x,t;x_0)$ gives the transition probability of the signalling index of a patient from the current value X_0 to the value X after a period of time $t > 0$. Then, the total transition probability from the current value X_0 to a value above the threshold X_c after a period of time $t > 0$ is given by

$$\begin{aligned} P(X_0,t) &= 1 - \int_{-\infty}^{0} dx\ f(x,t;x_0) \\ &= N\left(\frac{\ln(X_0/X_c) - \beta t}{\sigma\sqrt{t}}\right) + \left(\frac{X_0}{X_c}\right)^{\gamma} \cdot N\left(\frac{\ln(X_0/X_c) + \beta t}{\sigma\sqrt{t}}\right) \quad , \end{aligned} \tag{4}$$

where $N(\cdot)$ is the cummulative normal distribution function. This total transition probability can be interpreted as the probability of commiting suicide by a patient whose signalling index currently has the value X_0 after a period of time $t > 0$.

According to equation (4), the more severe a patient's affective disorder is, the higher his/her probability of suicide risk. For example, an individual who is characterized by the null value of the signalling index has no suicide risk, whereas a patient

[4]See, for example, the standard textbook of S.M. Ross on stochastic analysis: "Sheldon M. Ross, *Stochastic Processes*, 2nd ed. (Wiley, New York, 1996)".

with very severe affective disorder (namely $X_0 \to X_c$) has a probability of suicide risk approaching unity. Such a suicide risk system thus provides a methodology to estimate the transition probabilities of suicide risk associated with a change in the state of affective disorder of a patient. A negative change in the value of the signalling index of affective disorder of a patient diminishes the likelihood of suicide whereas a positive change increases the likelihood of suicide. Equation (4) also indicates that in the absence of any kind of preventive treatment (*i.e.* the case of underdiagnosis), namely $\mu = 0$,[5] the probability of suicide risk would approach unity asymptotically, whereas with preventive treatment (as characterized by $\mu > 0$), the probability of suicide risk can be stabilized at the level $(X_0/X_c)^\gamma$ after a sufficiently long period of time. Furthermore, we observe that for $0 < \mu < \sigma^2/2$ the stabilized level is higher than X_0/X_c, whilst for $\mu > \sigma^2/2$ it is lower than X_0/X_c. Obviously, the stronger the preventive treatment is, the lower and faster the stabilized level of suicide risk can be attained. Hence, medical and health institutions can apply the suicide risk analysis to actively manage and mitigate the levels of suicide risk of their patients by adjusting their suicide prevention strategies.

3. Numerical results

In Figures 1-4 we plot the probability of suicide risk versus the dimensionless time $\sigma^2 t$ for different values of X_0 and μ. Figure 1 indicates that in the case of underdiagnosis, *i.e.* $\mu = 0$, the probability of suicide risk of a patient grows very rapidly towards unity, and the growth rate increases with the severity of affective disorder. This is understandable because of the anticipated deterioration of the patient's affective disorder over time. In fact, Horgan has pointed out that suicide is nearly always due to untreated depression (Horgan, 2002). Moreover, according to a three year study of all coroners' records, autopsy and police reports for suicide victims aged 65+ in Ontario, over 80% of the elderly who committed suicide received no psychiatric referral. Of the sample, 87% were untreated while only 13% received antidepressants (Duckworth and McBride, 1996).

[5] In order to simulate the case of no preventive treatment, we need to eliminate any downward drift of the stochastic variable x and thus choose $\mu = 0$.

In Figures 2, 3 and 4, we examine the effect of preventive treatment in reducing the suicide risk. Figure 2 demonstrates a case of weak treatment: $\mu = \sigma^2/6$. The general trend of suicide risk profile is very similar to the one in the case of underdiagnosis, yet the growth rate of the probability of suicide risk is slower. For patients with severe affective disorder (say $X_0/X_c > 0.7$), they are still exposed to very high risk for suicide. This coincides with the fact that a large majority of the depressed patients with a history of suicide attempts, who are at higher risk for future suicide and suicide attempts, are undertreated (Oquendo, et al., 1999). Suominen also reported that few suicide attempters with major depression receive adequate treatment for depression before the suicide attempt and that, despite their well-known high risk for suicide, the treatment situation is not necessarily any better after the attempt (Suominen, et al., 1998).

On the other hand, in Figures 3 and 4 the numerical results show that the suicide risk can be efficiently improved by the adequate preventive treatment: $\mu = 3\sigma^2/2$ and $5\sigma^2/2$, and that more intensive preventive treatment leads to a lower stabilized level of suicide risk, characterized by $(X_0/X_c)^\gamma$, in a shorter duration. Thus, patients under long-term adequate preventive treatment have significantly lower suicide risk than the untreated patients. This implication is consistent with the clinical observations (Angst, et al., 2002; Keller, 2001; Carlsten, et al., 2001; and Isacsson, 2000). For instance, Keller reported that proper treatment duration is essential to maximizing outcome (Keller, 2001). In some countries the increased rate of prescribing coincides with fall in the suicide rate (Carlsten, et al., 2001; and Isacsson, 2000).

Furthermore, all of the four figures explicitly illustrate the observation that the probability of suicide risk of a patient grows with the severity of his/her affective disorder. In other words, $P(X_0, t)$ is a monotonically increasing function of X_0 at any given time t.

4. Conclusion

We have presented a theoretical model for estimation of the probability of suicide risk in terms of a stochastic signalling index of affective disorder. The model is able to provide a quantitative description of the dynamics of an individual's level of suicide risk, and numerical results show that the implications of our model are consistent with the clinical findings. Since the parameter σ essentially fixes the characteristic time scale and the measure of the strength of preventive treatment in our model, there is only one free parameter, namely μ, which characterizes the strength of preventive treatment for a patient. Hence, once we can have a good calibration of the parameter μ via the time-series analysis of existing empirical data, more quantitative theoretical predictions can be made and compared with further empirical studies so as to test the validity of our model more rigorously.

Furthermore, our model implies an interesting universality among different groups of patients in terms of age, gender, race, culture, etc. For example, while men and women can be distinguished by their different degrees of sensitivity in response to various external influences, *i.e.* men and women are characterized by different values of the parameter σ, their suicide risk follows the same profile and can be estimated from Figures 1-4, even though their characteristic time scales and their measures of the strength of preventive treatment are different. Similar ideas are also applicable to different races of people or groups of people having different cultural backgrounds.

As a final remark, in the estimation of the probability of suicide risk, our model does not consider the effect of drastic events. To simulate the possibility of the occurrence of a drastic event that triggers immediate suicide, we may extend our model with a discrete jump process, namely the Poisson process, that forms a second source of randomness in addition to the Wiener process. However, this mixed Poisson-Wiener process does not allow us to write down the probability of suicide risk in closed form.

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Figure captions:

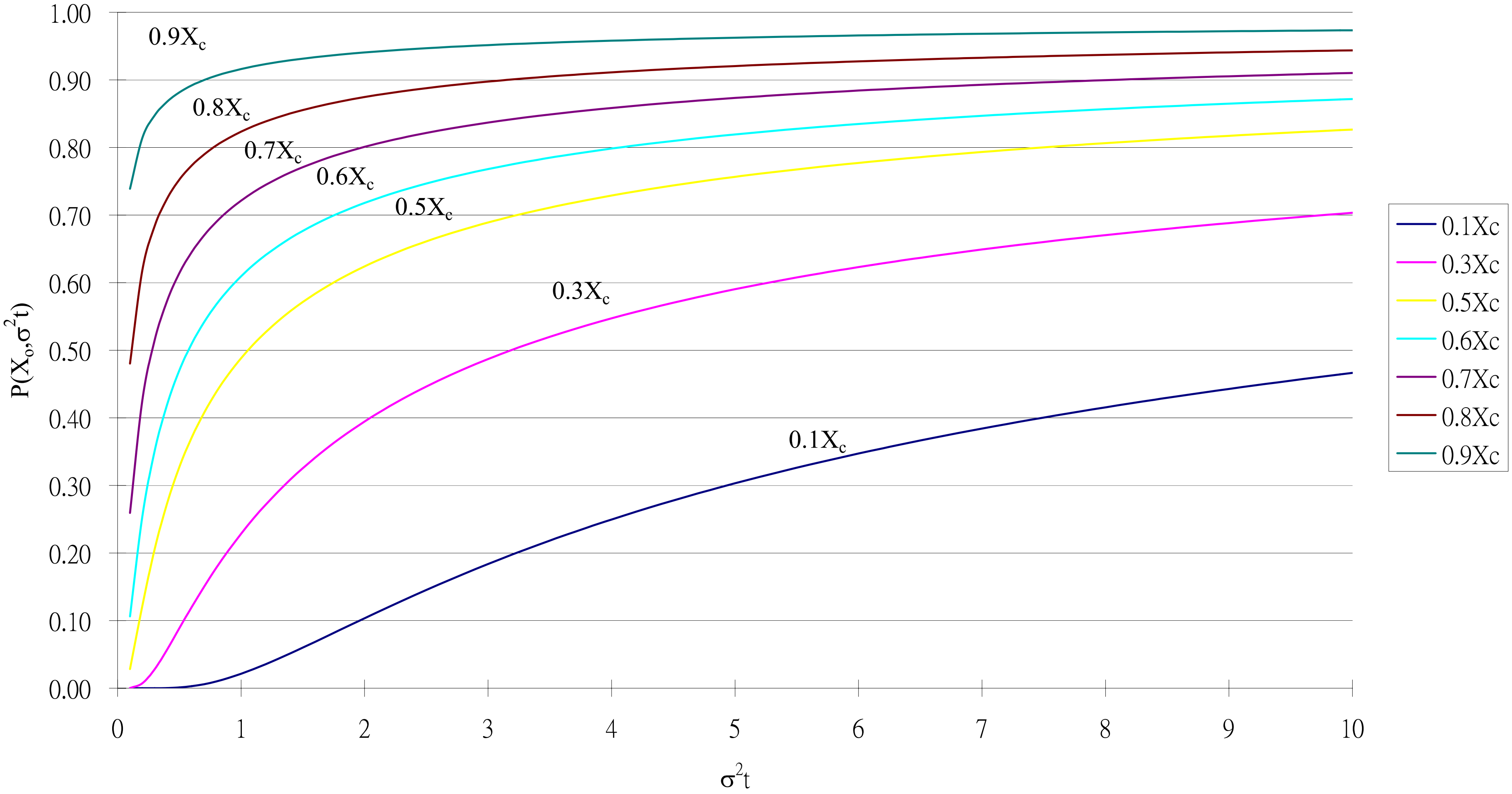
1. $P(X_0, t)$ as a function of t for $\mu = 0$.

2. $P(X_0, t)$ as a function of t for $\mu = \sigma^2/6$.

3. $P(X_0, t)$ as a function of t for $\mu = 3\sigma^2/2$.

4. $P(X_0, t)$ as a function of t for $\mu = 5\sigma^2/2$.

Figure 1 :



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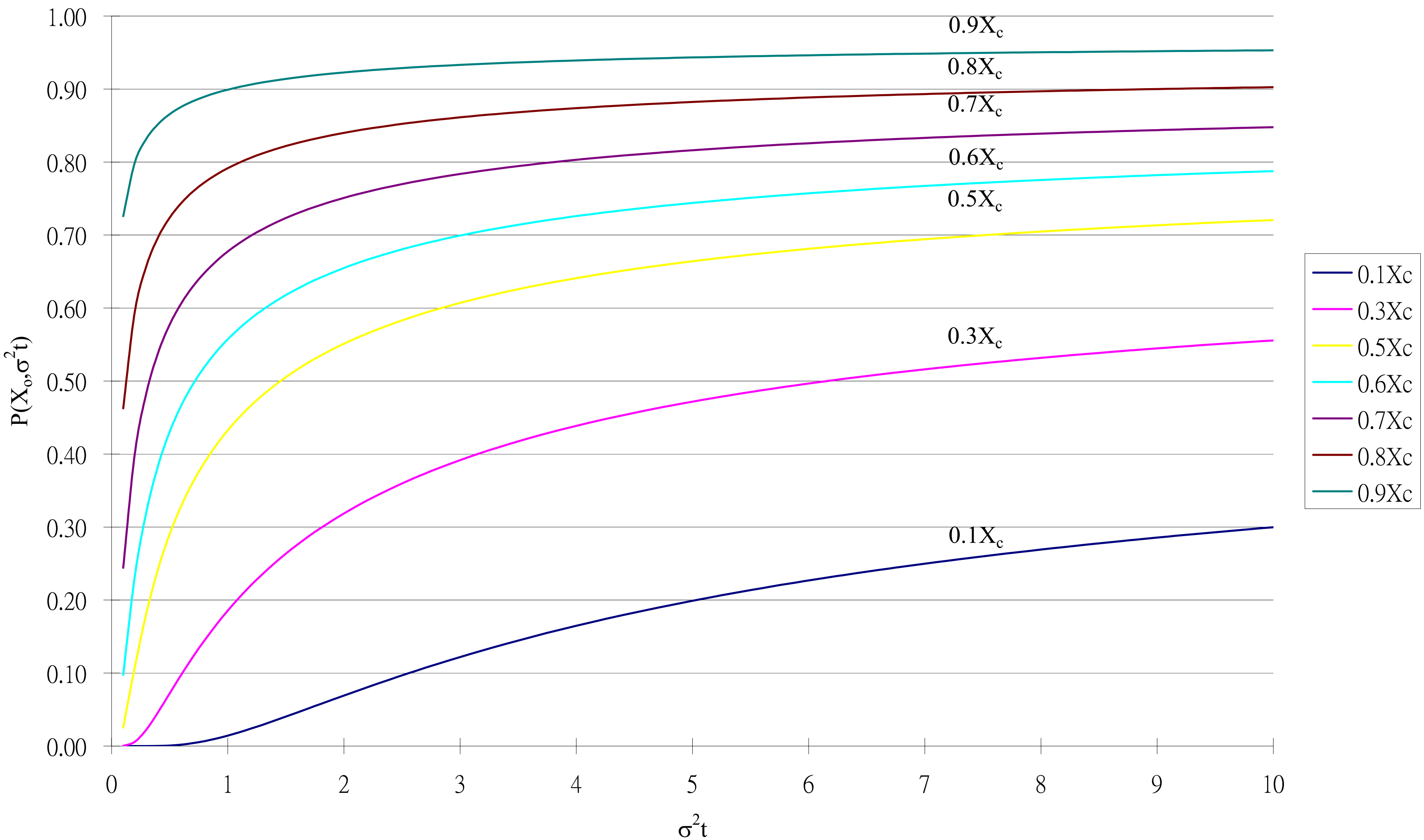

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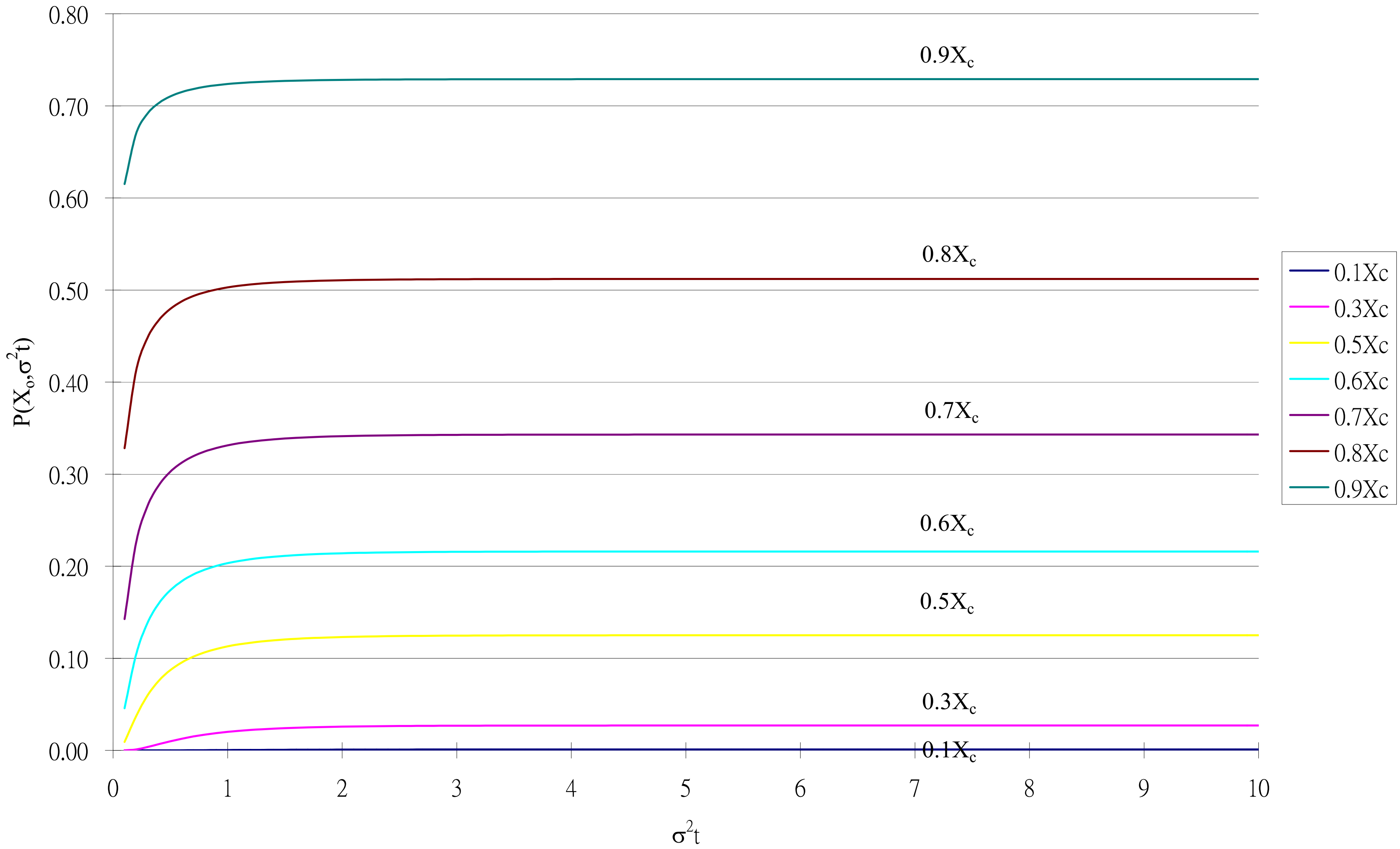
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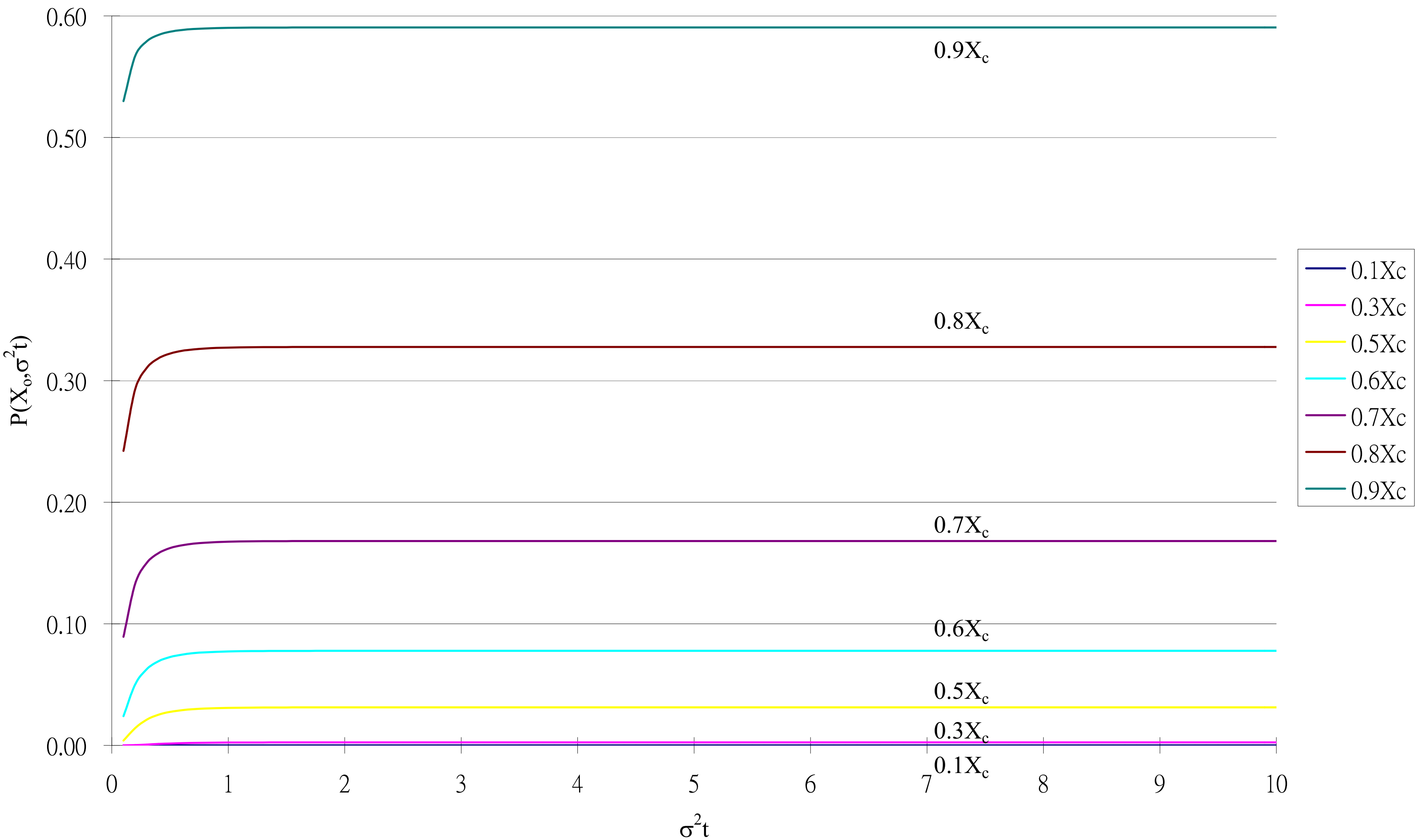

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