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PERSPECTIVE

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Will genetic test results be monetized in life insurance?

Oytun Haçarız[1] | **Torsten Kleinow**[1] | **Angus S. Macdonald**[1] | **Pradip Tapadar**[2] | **R. Guy Thomas**[2]

[1]Department of Actuarial Mathematics and Statistics, Heriot-Watt University and the Maxwell Institute for Mathematical Sciences, Edinburgh, UK

[2]School of Mathematics, Statistics and Actuarial Science, University of Kent, Canterbury, UK

Correspondence
Angus S. Macdonald, Department of Actuarial Mathematics and Statistics, Heriot-Watt University, and the Maxwell Institute for Mathematical Sciences, Edinburgh EH14 4AS, UK.
Email: A.S.Macdonald@hw.ac.uk

Abstract

If life insurers are not permitted to use genetic test results in underwriting, they may face adverse selection. It is sometimes claimed that applicants will choose abnormally high sums insured as a form of financial gamble, possibly financed by life settlement companies (LSCs). The latter possibility is given some credence by the recent experience of "stranger-originated life insurance" (STOLI) in the United States. We examine these claims, and find them unconvincing for four reasons. First, apparently high mortality implies surprisingly high probabilities of surviving for decades, so the gamble faces long odds. Second, LSCs would have to adopt a different business model, involving much longer time horizons. Third, STOLI is being effectively dealt with by the U.S. courts. Fourth, the gamble would be predicated upon a deep understanding of the genetic epidemiology, which is evolving, subject to uncertain biases, and cannot predict the emergence of effective treatments.

KEYWORDS

adverse selection, genetic tests, life settlement, one-shot gamble, stranger-originated life insurance

1 | INTRODUCTION

1.1 | Genetic testing

Since genetic tests began to enter clinical practice in the 1990s their use by life and health insurers in underwriting has been controversial (Joly et al., 2010).

In one sense this was perhaps surprising, and the reaction of life insurers at the time does suggest that some were taken by surprise. For many years, life insurers had routinely included family medical histories in underwriting. This could show an applicant to be at risk of an inherited disorder. For example:

(a) A man whose father had died of Huntington disease was at 50% risk of having inherited the gene mutation responsible for it.
(b) A woman whose mother and sister had died of breast cancer was at some risk of having inherited a gene mutation that could lead to breast cancer.

The first example is relatively straightforward, because a mutation arising in one specific gene is the sole cause of Huntington disease (Bates et al., 2002). The second example is complicated, because only a small proportion of breast cancer is caused by inherited genes, and there is more than one candidate gene (Eeles et al., 2004). This family history could have a genetic origin, but it could also have occurred spontaneously.

Genetic testing, meaning the direct examination of the DNA sequence, appeared merely to resolve the ambiguity inherent in knowing only the family history. Both individuals above could learn, definitively, whether they had or had not inherited a mutation responsible for their family history.[1]

What life insurers had perhaps failed to anticipate, in the 1990s, was the extent to which genetic test results would come to be regarded, in many quarters, as worthy of some legal protection against being used as a basis for discrimination. From the perspective of insurers, the rationale for this may not be obvious (and hence the controversy), but the strength of public sentiment is undeniable. See Prince (2019) for an excellent account of the policy debates and outcomes that have emerged in the UK, Sweden, Australia, and Canada. Broadly speaking, in all cases there has been pressure to bar insurers from using genetic test results, counter-pressure from insurers, and a variety of outcomes. Perhaps the most salient example is the UK, because government and insurers quite soon reached an accommodation, which has been stable for nearly 25 years.

1.2 | Adverse selection

Claims by insurers to have a "right to underwrite" (see Thomas, 2017 and references therein) turned out to have limited traction (see Prince, 2019) so one of the main reasons advanced by insurers to be allowed to use genetic test results was their exposure to the risk of adverse selection, if they were not.

[1]This is broadly true of the Huntington example, but to a much more limited extent of the breast cancer example, for reasons which need not detain us.

Adverse selection, as actuaries and insurers understand it, arises when an applicant for insurance is able to withhold adverse information relevant to the risk. Whether or not it matters depends on the magnitude of the difference in risk indicated by the withheld information.

(a) The difference between the mortality of males and females is comparable to the difference between the mortality of smokers (of cigarettes) and nonsmokers. Insurers for many years charged different premiums to males and females, but saw no need to distinguish between smokers and nonsmokers. And, when such a distinction was introduced (in the UK, in the 1980s) it was for marketing reasons, not because of fears about adverse selection (Rhodes & Savill, 1984).[2]
(b) The difference between the mortality of a carrier of a Huntington disease mutation and that of a noncarrier is extremely high, comparable to a difference of several decades of age (Brackenridge et al., 2004). No life insurer would offer the same premiums to two persons whose ages were several decades apart, and most would argue that to do so would present an existential risk. From this, they would proceed to argue that genetic test results should be allowed in underwriting.

One important difference between these two examples is that extreme differences in mortality tend to be associated with large differences in population size. There are many fewer people age 90 than age 20, and many fewer carriers of high-risk mutations than noncarriers.

Nevertheless, the hypothesis upon which insurers rely, in pointing to the threat from adverse selection, is that persons who know they are at higher risk and who do not have to disclose it will be incentivized to buy more life insurance (Thomas, 2017). In this respect, we introduce a distinction between two kinds of adverse selection.

(a) *Precautionary adverse selection*: The applicant's privileged knowledge gives them an incentive to ensure that their reasonable need for life insurance is met. This would lead to more life insurance being sold to higher risk persons, but not to unusually large amounts of cover for each person. There is some evidence that adverse selection of this kind may happen. Lane et al. (2015) reported that many cancer genetic counselors in Canada advise patients to put life insurance in place before taking a genetic test.
(b) *Speculative adverse selection*: The applicant's knowledge that they can take out life insurance at less than its true cost gives them such a strong incentive that they go further, and take out sums insured well in excess of any actual need.

The first of these is life insurance meeting a normal need. The second is life insurance as a financial gamble.

In the debate on genetics and insurance, speculative adverse selection is often invoked as being a likely outcome. For example, in a report to the Canadian Institute of Actuaries on the possible costs of a ban on insurer access, Howard (2014) assumed that 75% of individuals who tested positive would subsequently purchase an additional $900,000 of life insurance (compared with an assumed average before the test of $100,000).

[2]Once a single insurer had introduced the distinction, it *induced* fear of adverse selection among its competitors, where before there had been none.

It is also sometimes suggested that third parties will finance the purchase of policies. For example, a *Statement on Genetic Testing and Insurance* (Canadian Institute of Actuaries, 2014) issued in conjunction with the above report said:

> *...anti-selection should increase in provinces which allow secondary insurance markets. Currently, Québec, Saskatchewan, Nova Scotia, and New Brunswick allow secondary purchasing and selling of individual life insurance policies. In such markets, an opportunistic insurance policy purchasing program could be constructed to encourage individuals who have positively tested for genetic conditions to purchase insurance policies and sell the policies to this program in exchange for cash.*

The purpose of this paper is to examine the credibility of such suggestions that life insurance might be framed as a speculative product. In particular we ask three questions.

(a) Would individuals who have an adverse genetic test result be likely, as a result, to take out abnormally large life insurance policies?
(b) Would third parties be likely or able to finance large-scale speculative adverse selection?
(c) Does genetic epidemiology provide sufficient certainty about the risks to justify the outlay of money on a gamble, as in (a) or (b) above?

Our interest is not confined to any particular jurisdiction, but since the greatest developments in the secondary life insurance market have taken place in the United States (Gatzert, 2010), most of our examples will be drawn from there.

1.3 | The secondary life insurance market

A policyholder with a life insurance policy can sell it for cash to a third party (investor) in the secondary life insurance market. The investor takes responsibility for paying future premiums, and receives the sum insured when the insured person dies. This can be attractive to the investor if the insured person is in poor health, and attractive to the insured person if the surrender value on offer from the insurer is low. We identify four main strands of the secondary market (see Braun et al., 2018; Guttery et al., 2012).

(a) *Viatical companies* (VCs) buy policies from insureds with very short life expectancies (LEs), typically <2 years.
(b) *Life settlement companies* (LSCs) buy policies from older insureds, typically over age 65, with LEs of about 15 years or less.
(c) *Stranger-originated life insurance* (STOLI) follows the LSC business model but originates new policies intended to be sold to the investor rather than seeking out existing policyholders.[3]

[3]Unfortunately, the acronym "STOLI" has also been widely used for "stranger-*owned* life insurance," as a search of the Society of Actuaries' website with keyword "STOLI" confirms (searched on February 27, 2020). Stranger-*owned* life insurance encompasses all secondary market sales, and therefore includes the wholly legitimate business of VCs and LSCs, who purchase policies originally bought by individuals with *bona fide* insurance motivations. Stranger-*originated* life insurance, in contrast, refers to a less legitimate subset of secondary market sales, where a speculative investor

(d) *Premium financing companies* lend money to finance new life insurance policies but do not purchase them from the policyholder.

The concern expressed in the quote in Section 1.2 from the Canadian Institute of Actuaries (2014) report is about a new form of STOLI, where life insurance would be originated on individuals who have tested positive for genetic predispositions, but who may still have LEs of several decades. In this long-term aspect, it would differ from the extant business models at (a)–(d) above, which involve individuals with LEs of about 15 years or less.

1.4 | Plan of this paper

First we review the main features that define the epidemiology of a genetic disorder, with hypertrophic cardiomyopathy (HCM) as an example (the details are in the Appendix). Next, we introduce the idea of a "one-shot gamble" (Thomas, 2017) in order to discuss speculative adverse selection. We extend this discussion to LSCs and, more generally, STOLI. Then we review some features of the epidemiology of genetic disorders, and its reliability as a basis for investing in a financial gamble. We finish with our conclusions.

2 | BASIC FEATURES OF SINGLE-GENE GENETIC DISORDERS

2.1 | When is an inherited disorder of relevance to life insurance?

Here we briefly list the main features that might make a single-gene genetic disorder relevant for life insurance (see Pasternak, 2005 for genetical matters, and Macdonald, 2003 for features relevant to insurance).

(a) A small number of potentially fatal disorders are caused by abnormal variations in the DNA of a single gene, called "deleterious variants,"[4] which we abbreviate to "DV."
(b) Natural selection acts to remove DVs from the population, so they persist only if their physiological effects, called the "phenotype," are delayed until after reproductive ages. This also means that they are delayed until a carrier is economically active, and might buy life insurance. Only such "late-onset" disorders really matter for life insurance, and they are relatively rare.[5]
(c) With exceptions (which we ignore) a child inherits two copies of each gene, one from each parent. If both must have DVs to give rise to the disorder, it is recessively inherited. Such disorders tend to manifest early in life and are not significant for life insurance. Those that are significant must have late onset (see (b) above) *and* dominant inheritance, meaning that inheriting a DV from just one parent may give rise to the disorder.

provides finance to an individual with no *bona fide* insurance motivations to buy a policy, and then purchases it from the insured.

[4]We understand that the term "mutation," which we used until now, is falling out of favor among geneticists.

[5]Exceptionally, early-onset disorders might have some relevance if the insurance is on the life of a child.

(d) Genotype does not guarantee phenotype. The probability that the disorder will appear, during a DV carrier's lifetime, is called the "lifetime penetrance." It is usually <100%. More generally, the probability that the disorder will appear by any given age is called the "age-related penetrance," and this is important for life insurance. It must not be very high at early-to-middle ages, so life insurance can be purchased normally, but it must be high enough at middle-to-late ages so that, for a significant proportion of insured persons, the disorder develops and death occurs while life insurance is still in force.

To summarize, the genetic disorders of significance for life insurance are dominantly inherited single-gene disorders, of severe effect, but whose effects are delayed until middle age. As an example, we will use HCM, an inherited disorder of the heart muscle. We describe it briefly in the Appendix. It meets our criteria, because it is relatively common, mostly has high penetrance at young adult ages, and presents significantly elevated mortality risk.

2.2 | Family history and genetic testing

Before DNA could be sequenced directly, single-gene disorders could be recognized by the patterns of inheritance seen in extended families, or "pedigrees" (Cummings, 2015). Broadly speaking, it would be very unusual for two parents to carry the same rare DV, so each child of a parent who carried a DV would inherit it with probability 1/2, independently of their siblings (by Mendel's laws).

By asking about family history of disease, life insurers have long been able to assess tolerably well the probability that an applicant might carry a DV[6] (Brackenridge et al., 2004). Therefore, the underwriting of genetic disorders did not begin with the advent of DNA sequencing in the 1990s. Rather, DNA sequencing made it possible to say for sure that a person at risk of having inherited a DV either had or had not inherited it. It is important, for insurance purposes, to distinguish between "diagnostic" and "predictive" genetic testing.

(a) Once symptoms of a disorder have appeared, a genetic test may confirm the diagnosis, and identifying the precise mutation may be important for treatment (in which case, the test may also be prognostic).
(b) A genetic test taken by a completely healthy person, free of any symptoms, is purely predictive of future risk. It may also be prognostic, if prophylactic treatment for the disorder is possible. An example is inherited breast cancer. Genetic testing may reveal a deleterious mutation in the BRCA1 or BRCA2 gene decades before a single cancerous cell exists, and prophylactic treatment (mastectomy) may be considered.

3 | THE ONE-SHOT GAMBLE

In this section, we explore in more detail the possibility of individuals using life insurance as a vehicle for a financial gamble, and introduce the idea of a "one-shot gamble." The discussion here will be intuitive; for mathematical details see Chapter 11 of Thomas (2017).

[6]Life insurers usually ask only about the medical history of an applicant's parents and siblings. Clinical geneticists and epidemiologists might acquire much more extensive histories, over several generations.

3.1 | Precautionary framing *versus* speculative framing

Life insurance is typically framed principally as a precautionary product, that is it is bought to restore a quantum of wealth which the beneficiary loses consequent upon the death of the life insured. Probably the most common context is that the beneficiaries are family dependants of the life insured. But there are other possible contexts: for example, the beneficiary may be a lender, who requires life insurance (usually paid for by the borrower) to ensure the repayment of a loan if the borrower dies; or an employer, who requires "key person insurance" to compensate business lost after the death of an employee Nie et al. (2018). In each case, there exists a clearly identifiable prospective financial loss, so that any claim restores rather than increases (at least in broad-brush terms) the wealth of the beneficiary.

While the above framing as a precautionary product represents a typical view of life insurance, insurers ultimately cannot constrain how people view their products. Life insurance can alternatively be framed as a speculative product; that is a bet against the insurer at a cheap price, which is made to increase (rather than restore) the wealth of the beneficiaries. In this framing, the quantum of cover chosen will depend principally on the terms of the insurance (the price of the cover relative to its expected payoff). Where the terms are attractive, the quantum of cover chosen might be greater than any precautionary need.

3.2 | The one-shot gamble

Life insurance framed as a bet against the insurer always constitutes a one-shot gamble. That is, the payoff from the investment is determined by the occurrence or nonoccurrence of a single event, the death of the life insured. Customers cannot make the same bet many times, and obtain the average result diversified across those instances; they can make the bet only once, on a single life. Note that in this paragraph (and throughout this paper) we use the terms "gamble," "investment," and "bet" interchangeably; in the present context, none of them has any moral connotation.

In term life insurance, the single event (the death of the life during the term) is relatively unlikely; that is, the one-shot gamble is a gamble against long odds. Typically, this remains true even if the life has some private information suggesting higher risk. For example, if a 30-year-old life has private knowledge of HCM, the probability of dying during the term of a 25-year insurance is about 15% (own calculation, based on Haçarız, 2020). The life is still much more likely to survive the term than not.

This combination of features—the inability to diversify the bet, and the long odds—make a large gamble on term insurance unattractive for most realistic scenarios. For a large gamble to become attractive, at least one of the following scenarios needs to be true:

(a) The gamble is diversifiable across many lives; or
(b) The odds of the gamble are relatively short.

As an example of a diversifiable gamble, if we could buy 25-year term insurance on each of a large number of 30-year-old lives with HCM as described above, but at a price which allows for only a 5% probability of dying, then that portfolio of bets could be an attractive investment. Although it is unlikely that any particular life will die during the term, with a large number of

lives we can be fairly sure that close to 15% will die, compared with the 5% on which the premiums were based.

As an example of a gamble with short odds, if we privately know we already have a terminal illness from which we are 80% likely to die during the term of the insurance, then a one-shot gamble on term insurance at standard premium rates may be attractive. This is because there is an 80% chance that our beneficiaries will receive a payout many times the premium, which we may feel makes it worth taking the 20% risk that we shall just lose the premium.

Superficially, it may appear that the need for short odds on the one-shot gamble might be satisfied by choosing a whole life policy, under which a death payment will be received at some future time with certainty. But this apparent certainty is illusory, because for a whole life policy, receiving a death payment is not the same as "winning" the one-shot gamble. The economic gain to the customer at death is not the death benefit, but rather the *difference* between the death benefit and the accumulated value of all the premiums paid. As the customer gets older, this difference gets smaller; for deaths at high ages, the accumulated value of all premiums paid may even exceed the death benefit ultimately paid. So while choosing a whole-life policy does increase the chance of *some* economic gain, the chance of a *large* economic gain is still small; and the chance of an economic *loss* also becomes material.[7]

As an alternative to diversifying the insurance bet across many independent lives, the one-shot gamble could instead be held as part of a broader portfolio of other bets. However, provided these other bets are independent of the life insurance, then including them can only *reduce* the maximum fraction of total wealth which can sensibly be allocated to the one-shot gamble. So under this portfolio concept, the bet on the one-shot gamble remains limited by its long odds.[8]

The considerations above suggest that the potential for speculative adverse selection by a single individual is in fact quite limited. This is so whether the individual has the means to pay the premiums or must borrow from a premium financing company (see Section 1.3). Neither of the clearly favourable scenarios—a diversifiable gamble, or a gamble with short odds—corresponds closely to the situation of a single individual with some private genetic knowledge. However, in principle (and subject to any applicable legal restrictions), a diversifiable gamble might be engineered by a third party, which could assemble a group of such individuals, and then fund the purchase of policies on all their lives. The next section considers this possibility.

4 | VIATICAL AND LIFE SETTLEMENT COMPANIES

4.1 | Motivation

In this section we consider VCs and LSCs and their business models, see Section 1.3. We are motivated to do so by the Canadian Institute of Actuaries report (Canadian Institute of Actuaries, 2014) quoted in Section 1.2. We find that life insurance contracts written for long terms on relatively young lives with a reasonable prospect of a long life seem to fall well outside any of the existing business models. Nevertheless, we also consider the possibility of a different STOLI-

[7]We ignore the possibility of dividends on the whole life policy, which are not certain, and do not negate the point that the net benefit of the policy to the customer decreases at higher ages.

[8]With negative correlation between the one-shot gamble and the other bets, some increase in the bet on the one-shot gamble might be justified; but there seems no obvious rationale to anticipate negative correlation.

type business model, where a third party would finance the purchase of life insurance policies by carriers of a DV.

4.2 | Business models

For full accounts of the origin and operation of VCs and LSCs see, for example, Ray (2000), Doherty and Singer (2003), Canadian Centre for Elder Law Studies (2006), Aspinwall et al. (2009), Guttery et al. (2012), Braun et al. (2018), Sheridan (2019), or Braun and Xu (2020). Both types of company profit from creating a secondary market in life insurance policies. That is, they trade in policies that have already been issued by insurers.

VCs originated in the acquired immunodeficiency syndrome (AIDS) crisis in the 1980s, when a large number of young people were suddenly faced with dramatically reduced LEs. Any who had long-term life insurance in place could usually obtain only a relatively low surrender value from the insurer, which did not reflect their terminal illness. VCs purchased these policies for cash payments in excess of the surrender value, assumed responsibility for paying the premiums, and collected the full sum insured on death. This established the VC business model, buying policies from persons with very short LEs, usually <2 years.[9]

LSCs have a similar business model but target different sectors. They accept longer expectations of life, typically buy the policies of older persons, and seek out larger sums insured (Giaccotto et al., 2015). Based on data from the New York State Insurance Department, 1993–2009, the same authors reported the following, associated with the growth of the LSC market.

(a) Estimated LEs increasing from 13.99 to 31.12 months.
(b) The average sum insured increasing from $79,113 to $574,609.

Xu (2019) states that over 2011–2016, the average age at settlement was 76.3, and that "over the last five years" the mean sum insured settled was $1.9 million with a median of $1 million. The increase in the sums insured is particularly interesting. Giaccotto et al. (2015) say "Purchasing fewer but larger [policies] saves on transactions costs." However, this strategy is the exact opposite of the basic principle underlying insurance, because it concentrates risk rather than spreading risk across a large number of individuals.[10]

A high degree of reliance is placed on estimates of LEs which are provided by a small number of specialist underwriting companies, called "LE providers". In the 2000s, at least, these tended to provide over-optimistic (too low) estimates[11] (Bauer et al., 2018; Sheridan, 2019; Xu 2020). For LSCs in particular, the base tables in use were neither particularly reliable at high ages, nor based on the wealthiest segment of the population, also leading to underestimated LEs (Sheridan, 2019).

Note the key features of this business model.

(a) The companies buy existing policies, they do not initiate new policies.

[9]In respect of AIDS itself, this business model did not survive the introduction of antiretroviral treatments.

[10]Sheridan (2019) described how LSCs would try to avoid concentration in particular "cohorts," such as nonsmokers or specific impairments.

[11]If LE estimates were too high, the business would not be transacted. Note the analogy between LE providers competing to give acceptable LE estimates to LSCs and credit ratings agencies competing to give acceptable credit ratings to structured products issued by investment banks (McLean & Nocera, 2010).

(b) The terms involved are fairly short (very short in the case of VCs).
(c) There exists some risk of medical developments lengthening expectations of life.
(d) The companies do not depend on the life insurer mispricing the policy at issue. They target persons whose health has deteriorated since their policies were issued.

In particular, companies operating as above have no bearing on the genetics and insurance debate. This is so even if a company was able to use genetic test results in choosing which policies to buy. The time horizons are short, not long, and companies do not initiate the purchase of new contracts with very long terms and high sums insured.

At first sight, the insurers who issued the contracts originally are not affected; they need not even know that a policy has been sold. It is often argued, however, that insurers build in significant numbers of lapsed policies, receiving only the surrender value, in their profit calculations, and that investors will not usually lapse policies they have bought (Braun et al., 2018; Doherty & Singer, 2003). The first authors, in particular, regard the secondary market as a benefit, if it will "...keep incumbent insurers from the unfair, and ultimately unworkable, practice of using high lapse expectations to underprice certain policies."

4.3 | STRANGER-ORIGINATED LIFE INSURANCE

4.3.1 | STOLI in North America

Contrast the business model in the previous section with the hypothetical scenario of a third party monetizing the one-shot gamble by identifying individuals at increased long-term risk and then funding them to take out new life insurance policies. This "STOLI" overcomes the drawback of the one-shot gamble by diversifying across many lives. Glasgow et al. (2018) describes how STOLI emerged in the United States from 2000 onward, with the following features, being a twist on the basic business model of LSCs.

(a) STOLI investors typically targetted healthy elderly people, who would pass medical underwriting. They often used brokers in key geographical areas, since open advertising was impossible.
(b) LE estimates were obtained from the LE providers, as for a LSC.
(c) STOLI policies were very large, usually over $2 million and often in the range $5–$10 million.
(d) Investors often took out multiple policies on the same individual, from different insurers.
(e) While the selected persons could pass medical underwriting, the level of fraud perpetrated to pass financial underwriting, and to take out multiple policies on one life, was high.
(f) A very large volume of STOLI business was written. Glasgow et al. (2018) mention one single scheme ("the Coventry program") which generated about 7,000 policies with sums insured of about $20 billion.
(g) Life insurers only realized what these policies were in the financial crisis of 2008, when some investors were unable to pay the premiums.

Essentially STOLI is a bet on when the insured individual would die made by investors who have no insurable interest.[12] Braun et al. (2018) shows that the volume of STOLI business

[12]In most jurisdictions, the initial purchaser of a life insurance policy must have an "insurable interest" in the insured person. That is, they must stand in some relationship to them such that they will suffer financial harm were the insured

increased steeply until about 2008, then declined equally steeply. This was not a coincidence. It represented an attempt to securitize mortality risk, on an industrial scale, in an era when securitization and distribution of risk was seen as a panacea[13] (McLean & Nocera, 2010).

The type of policy was important. Conventional term and whole life insurance, under which reserves are accrued, impose a "...punitive return drag for an investor" (Sheridan, 2019). This is because conventional contracts mandate substantial premiums in the early years, which accrue at the rates of return granted under the policy; these rates are typically much lower than the rate of return anticipated by the investor from the "bet" on mortality exposure. Therefore investors purchased universal life contracts, choosing maximum death benefits with little or no savings element. It is unclear if STOLI business would be viable based on conventional contracts.

Barring outright fraud, STOLI policyholders have to pass the insurer's medical underwriting, which will be stricter for large sums insured, so at first sight it is difficult to see where the investors' profit is. Indeed, according to Sheridan (2019) STOLI investors did not realize that by targetting the wealthiest they were also targetting the healthiest, and it is not clear that the STOLI business was profitable.[14]

Below, we consider two aspects of STOLI: its legality; and whether the arithmetic is likely to work in the case of genetic disorders.

4.3.2 | STOLI and the Law

The whole point of STOLI is that an investor wishes to avoid the legal requirement that anyone taking out a life insurance policy on someone else's life has to have an insurable interest in that person. The investor suffers no financial loss or harm on the death of the insured person, so the policy is purely an investment.

Regulators in the United States have moved to curb STOLI business, and life insurers, mainly in the United States, have in many cases been successful in having STOLI policies declared void in court. Glasgow et al. (2018) describe these developments.

(a) Two regulatory bodies introduced model acts, which have since been adopted in many states. Their effect was to require certain disclosures that would alert an insurer to possible STOLI business.[15]

person to die. Examples include dependent relatives, lending institutions or businesses in which the insured person is a key employee. The purpose of insurable interest is to avoid the situation where the initial purchaser of a life insurance policy is interested only in the death of the policyholder. Most jurisdictions nevertheless permit the initial purchaser to sell the policy to a third party at a later date. For further discussion of the insurable interest doctrine as applied to secondary life insurance markets, see Richmond (2012) and Swisher (2015).

[13]Aspinwall et al. (2009) explains mortality risk to readers who are familiar with credit derivatives, and the larger part of the book is devoted to the structured and derivative products that can be created out of life settlements.

[14]One possible explanation is that insurers have built lapse assumptions into premium rates; if too few policies are lapsed, the insurer makes a loss and that is the STOLI investor's profit. Sheridan (2019) calls this the "lapse support meme" and writes "Premium rates varied so widely by carrier, vintage, product type and premium class that investors lacked a baseline from which to calibrate the extent of the lapse assumptions and determine if they were a viable cause of this magical STOLI value creation or not."

[15]One model act described the business as "viatical settlements" and the other as "life settlements," so the older terminology is perpetuated (Glasgow et al., 2018).

(b) A large amount of case law has accumulated. Glasgow et al. (2018) said "Generally speaking, insurers did well in this litigation. Indeed, during the time period from 2009 to 2012, huge numbers of policies were invalidated and new pro-insurer STOLI decisions seemed to be issued by courts across the country almost every week." Xu (2019) reports that 126 legal cases, relating to STOLI, were decided between 2010 and 2017.

Given that STOLI investors have typically underestimated LEs (Glasgow et al., 2018; Sheridan, 2019), also a feature of VCs and LSCs, as noted by Giaccotto et al. (2015), it is expected that large numbers of claims will emerge in the future and many of them will be successfully challenged by insurers. Braun et al. (2018) says: "Over the past decade, most states also passed laws to ban STOLIs. Today, STOLIs are a pariah in the life settlement industry."

Since it is the gambling aspect of STOLI that the U.S. courts found objectionable, and that would be the same if the basis of the gamble was genetic test results, we must presume that such an enterprise based on genetic test results, in the United States, would be at high risk of legal challenge.

The Canadian Institute of Actuaries (2014) report suggested that STOLI could be used to exploit genetic tests in Canada. The legal position in Canada is similar to that in the United States: an insurable interest is required when a life insurance contract is written, but the contract can then be assigned to an entity who has no insurable interest (Canadian Centre for Elder Law Studies, 2006). However, there is no evidence in the literature of STOLI activity in Canada.

4.3.3 | STOLI and genetic tests

The key point here is that STOLI based on genetic test results would be completely different from the LSC business model, adopted and expanded by STOLI investors after 2000. Essentially we would be adding a fifth, new, business model to the life settlement market. Taking HCM as an example, we note the following obstacles to STOLI based on genetic test results.

(a) Although mortality rates associated with HCM are much greater than normal, the probabilities of surviving for a long time are surprisingly high. Haçarız (2020) noted that if HCM-related mortality was 1% per annum (a widely cited figure) the probability of surviving for 40 years was about 2/3. Using the more up-to-date rate of 0.55% per annum (from Haçarız, 2020) the probability of surviving for 40 years is about 4/5.
(b) Consequently, the STOLI investor would have to buy insurance policies with very long terms, to obtain any substantial excess probability of a payout, and payouts would extend into the distant future. Meanwhile, the investor's premium outgo is continuing. This is very unlike the normal LSC business model.
(c) Because of the long policy terms, the investor is vulnerable to future advances in medicine that could destroy its profits at a stroke. The more the investor focuses on a particular disorder, the more concentrated this risk is.
(d) The investor profits *only* if the life insurer underpriced the policies, because of the missing information (but see Footnote 14). But if the insurer has priced the policies cautiously, precisely because of limited information or selection bias in the available information, the investor's advantage may be illusory. We explore selection bias in the next section.
(e) To implement such a scheme, the investor would have to locate potential purchasers. It would be unwise to assume that this could not be done; one characteristic of STOLI investors seems to be

ingenuity. But in the United States, the STOLI target market was a small proportion of individuals, those with sufficient wealth. In respect of a genetic test, the target market is also small, those with an adverse test result. If both requirements had to be met to make the business worthwhile, the target market would be the intersection of two small populations.

(f) Suppose investors do assemble a pool of carriers of HCM-related DVs who do not have a clinical diagnosis of HCM, which would be disclosable to an insurer. Some may have a family history, some may know only a genetic test result. Then the investor would have to finance the purchase of large life insurance policies, keeping within insurers' financial underwriting limits.

(g) Finally, perhaps, the investor would offload the long-term risk onto other investors, for example, by packaging the policies into a structured product, like an insurance-backed bond. Ironically, the main investors looking for yield over very long terms might be life insurers themselves.

Crucially, the entire basis of the gamble, which we emphasise is quite different from STOLI based on the LSC business model, rests on the investor's understanding of the risk associated with a genetic test result. The only source of that understanding is the genetic epidemiological literature, which we consider next.

5 | GENETIC EPIDEMIOLOGY

5.1 | How genetic epidemiology evolves

The epidemiology of a single-gene disorder typically evolves over time in the following way (see Cummings, 2015; Haçarız, 2020).

(a) The disorder may be recognized as Mendelian from studies of families with exceptional numbers of cases. Prevalence estimates are, in the absence of genotyping, based on clinically diagnosed cases. Mortality rates may be based on strongly selected populations, such as persons referred to clinics because of severe symptoms, introducing selection bias.

(b) Over time, variants in particular genes are associated with the disorder.

(c) Penetrance estimates of the identified mutations are obtained, based on the presence of clinically diagnosed individuals within families. Such studies are subject to *ascertainment bias* (see Hodge, 2002 and references therein) since only families with affected members come to the attention of researchers.[16]

(d) Over time, studies in larger and less selected populations may take place. Studies of prevalence in unselected populations are particularly important, since they may reveal significant differences between the prevalence of DVs and the prevalence of clinical diagnoses (that is, the presence of "silent" DVs). This in turn may throw light on penetrance estimates subject to ascertainment bias.

(e) If effective treatment or prevention is available, the primary purpose of genetic testing is to identify carriers of DVs so they can be treated. Then genetic testing may significantly reduce mortality.

[16]Ascertainment bias refers to the recruitment of families, rather than individuals, into a study.

5.2 | An example: HCM

We describe HCM briefly in the Appendix. Here we highlight two ways in which the epidemiology of HCM has evolved, and is still evolving (see Haçarız et al., 2020).

5.2.1 | Falling estimates of HCM-related mortality

From the first study in 1958, until the 1990s, studies of HCM typically estimated annual mortality rates of between 3% and 6% (Maron et al., 1999). These were strongly influenced by selection bias. Subsequent studies suggested an annual mortality rate of about 1%, and this was used by Howard (2014). However, there are two reasons to believe that the 1% annual mortality rate is considerably overstated.

(a) Studies of HCM-related mortality take the form of survival analyses, which measure time under observation until an endpoint is reached. The endpoint of interest is, clearly, death. But virtually all studies used an endpoint which counted certain nonfatal events as well, such as a resuscitated cardiac arrest.[17] Such "non-fatal deaths" have to be removed from the mortality rates used for actuarial purposes.
(b) Modern treatments have reduced HCM-related mortality even since the 1990s. Based on very recent and large studies, with "non-fatal deaths" removed, Haçarız (2020) estimated an annual mortality rate of 0.55% (see Footnote 20).

5.2.2 | Falling estimates of disease penetrance

We note in the Appendix, point (e), that the population prevalence of clinical HCM has been estimated to be 0.2% but that the population prevalence of known HCM-related DVs has been estimated to be about 0.6%, both based on unselected populations so not subject to selection or ascertainment bias. Allowing for as-yet-unidentified DVs the population prevalence of HCM-related DVs could be as high as 1%.

This implies that the true population penetrance of HCM DVs—that is, the probability that the disorder will appear in a DV carrier's lifetime—may be very much lower than current estimates based on studies of persons with clinical HCM. The same applies for estimates of mortality rates.

5.3 | Consequences for life insurers and STOLI investors

It is evident that the results of an actuarial study into adverse selection, based on genetic epidemiology, will depend strongly on what stage has been reached in the evolution of the epidemiology. For example, Macdonald et al. (2003) in a study of breast and ovarian cancer suggested reducing the empirical penetrances by 50–75% to allow for ascertainment bias. Lemaire et al. (2000) said, in another study of breast and ovarian cancer:

[17]For example, Elliott et al. (2006), Maron et al. (2013, 2015, 2016).

The results of our research should be applied with caution. They are based on the most recent data available from the medical literature, but new medical articles are published regularly that often provide very different estimates of BC and OC risks, depending on the demographic group studied. For instance, estimates of the lifetime probability of developing ovarian cancer for a woman with a BRCA1 mutation range from 11% to 84%. Also, there might be a systematic bias in medical studies due to the selection of the sample, usually families with a strong family history.

It would be very difficult to make reliable forecasts of the future penetrance and mortality associated with a single-gene disorder, when the epidemiology is imperfect, evolving and unknown possible treatments lie in the future. However, we can make two practical observations.

(a) A life insurer who uses up-to-date epidemiology to calculate premiums (based on family history when genetic test results may not be used) will almost certainly arrive at premiums that are on the safe side, possibly extremely so.
(b) A STOLI investor who finances the large-scale purchase of life insurance policies by carriers of DVs will have to pay these safe-side premiums. Then the investor faces all the risks associated with true penetrance and mortality being overstated in the epidemiology, and effective treatments being developed. Unless the investor can offload its risk onto other investors, perhaps through securitization, it will face these risks for decades.

Haçarız et al. (2020) modelled HCM and adverse selection,[18] and in particular considered the possibility of an insurer basing premium rates[19] on epidemiology that was subject to selection or ascertainment biases, while an investor tried to exploit the insurer by funding adverse selection. They also considered the possibility that family history may not be used. Our figures are taken from table 8 in Haçarız et al. (2020).

To represent the insurer's assumptions, we use the annual HCM mortality rate of 1% (widely cited and assumed by Howard, 2014).[20] To represent the "true" annual HCM mortality rate we use the more recent estimate of 0.55% (see Section 5.2.1), while noting that this too is based on studies from which it is unlikely that all biases have been eliminated.

The "cost" of adverse selection is defined as that uniform percentage increase in all premiums that would compensate for the insurance losses caused by adverse selection, after the insurer has charged everyone actuarially fair premiums on the basis of all information it is allowed to use.

(a) First, Table 1 considers the effect of precautionary adverse selection only, that is, insurance meeting a need and not as a financial gamble. "Mild" adverse selection means twice as

[18]The model was a multiple-state model (Macdonald et al., 2018) in which the following events in an individual life history were represented by transitions between states: taking a genetic test; purchasing life insurance; fatal and nonfatal HCM-related events; and normal mortality. The life histories of all the members of a nuclear family were modelled simultaneously, allowing family medical history to be used by insurers in underwriting.

[19]The relevant premium rates are those charged to persons with a family history of HCM. In line with UK practice, Haçarız et al. (2020) assumed that persons with a *negative* genetic test result would be offered the normal premium rates, ignoring the presence of a family history. This assumption is conservative.

[20]In our model, "rate" always means a transition intensity, analogous to the actuary's force of mortality (Macdonald et al., 2018). Howard (2014) used "rate" to mean the probability of dying over 1 year. The consequences of this distinction are not material for the numerical results and we prefer not to burden the reader with it in the text.

TABLE 1 Examples of percentage increases in premiums when the insurer calculates premiums using epidemiology subject to upward bias

Precautionary adverse selection	Premium increases with Family history disallowed %	Family history allowed %
None	−0.21	−0.24
Mild	−0.19	−0.24
Severe	−0.16	−0.25

Note: The rate at which insurance is purchased "normally" is high, representing a large insurance market.
Source: Haçarız et al. (2020).

many people as normal[21] buy insurance after testing, "severe" means five times as many. In all cases the percentage costs are small but *negative*. This means that the premiums the insurer is charging are more than adequate to meet the risk, because of the upward bias in the epidemiology upon which they relied. Since the investor faces cashflows that are the mirror image of the insurer's, they make a loss, because they are paying high premiums but death claims are fewer than expected.

(b) Next, we note that the same level of adverse selection superimposed on a much smaller insurance market should, in principle, have a proportionately greater effect. To that end, Table 2 simulates a smaller market,[22] in which the normal rate of insurance purchase is five times less than in Table 1. "Mild" adverse selection again means being twice as likely to buy insurance, but "severe" adverse selection now means 25 times more likely. The percentage premium increases are again negative, but not very different from those in Table 1. The reason is that insurers may not know genetic test results, but they are not completely ignorant. They can calculate premium rates allowing for some proportion of insurance purchasers being carriers of HCM-related DVs. If they can use family history in underwriting, they can do this with greater accuracy, which is why these results are more consistent.

(c) Finally we add speculative adverse selection, in Table 3. This takes the "small market" assumptions of Table 2 to model precautionary adverse selection and assumes that "adverse selectors" take out sums insured 10 times normal. Now, in the most extreme case when family history cannot be used, we do get a larger increase in premiums to meet adverse selection costs. If family history can be used, however, then the insurer is well-protected. In fact, the more adverse selection *behavior* the better.

Of course, excess profit for the insurer means that policyholders are paying more than their risk warrants, in statistical terms. We do not suggest the life insurer does this on purpose, although it may set out to price this insurance cautiously. The point is that a STOLI investor funding the large-scale purchase of life insurance on the terms offered would have cashflows that were the mirror image of the life insurer's, leading to a loss. This illustrates the risks that a STOLI investor might run if it tried to exploit adverse selection based on genetic tests.

[21]The "normal" rate of purchase is represented by a transition intensity of 0.05 per annum; approximately, 5% of uninsured individuals buy insurance in any year. This represents a large insurance market.

[22]Represented by a transition intensity of 0.01 per annum.

TABLE 2 Examples of percentage increases in premiums when the insurer calculates premiums using epidemiology subject to upward bias

Precautionary adverse selection	Premium increases with Family history disallowed %	Family history allowed %
None	−0.19	−0.21
Mild	−0.15	−0.22
Severe	0.13	−0.27

Note: The rate at which insurance is purchased "normally" is low, representing a small insurance market.
Source: Haçarız et al. (2020).

TABLE 3 Examples of percentage increases in premiums when the insurer calculates premiums using epidemiology subject to upward bias

Precautionary adverse selection	Premium increases with Family history disallowed %	Family history allowed %
None	0.27	−0.31
Mild	0.69	−0.40
Severe	3.39	−0.90

Note: The rate at which insurance is purchased "normally" is low, representing a small insurance market. "Adverse selectors" take out sums insured of ten times normal.
Source: Haçarız et al. (2020).

6 | CONCLUSIONS

6.1 | Genetic testing and adverse selection

Life insurers' concerns about their exposure to adverse selection if they are unable to ask about genetic test results known to applicants have been expressed for nearly 30 years. If high adverse selection costs are to arise, it would seem to be due mainly to speculative adverse selection. Therefore, in this paper we have considered three questions.

(a) Would individuals who have an adverse genetic test result be likely, as a result, to take out abnormally large life insurance policies?
(b) Would third parties such as LSCs be likely or able to finance large-scale speculative adverse selection?
(c) Does genetic epidemiology provide sufficient certainty about the risks conferred by carrying a DV to justify the outlay of money on a gamble, as in (a) or (b) above?

We summarize our answers to these questions in the following sections.

6.2 | Individuals: The one-shot gamble

For individuals with private knowledge of an adverse genetic test, life insurance might be framed as a speculative product; that is a bet against the insurer at a cheap price. This is always

a one-shot gamble: individuals cannot diversify risk by making the same bet many times; they can make the bet only once.

This particular one-shot gamble is usually a gamble against relatively long odds. For term insurance, despite increased mortality, the individual is still typically very likely to survive the entire term. For whole-life insurance, the economic gain is large only if the individual dies before too many premiums have been paid.

In most realistic scenarios involving an a"bdverse genetic test, this combination of features—inability to diversify, and long odds—make the one-shot gamble an unattractive prospect for a large bet by a single individual.

6.3 | Third parties: LSCs and STOLI

One way to overcome the long odds of a one-shot gamble on genetic information would be to turn it into a many-shot gamble. LSCs[23] possibly could do this, by financing the purchase of life insurance policies, but a completely new business model would be needed, targeting individuals with much longer LEs and smaller probabilities of premature death, and initiating new policies (STOLI). Combining the double rarity of wealthy individuals and individuals who carry a DV, it is unlikely that a sufficiently large market exists for STOLI investors.

The legal system in the United States offers some defences against STOLI, ultimately relying on insurable interest, and U.S. courts have, in recent years, invalidated large numbers of STOLI policies. Despite the concerns expressed in the Canadian Institute of Actuaries (2014) report, we have found no public record of STOLI activity in Canada comparable to that in the United States.

6.4 | Relying on genetic epidemiology for a financial gamble

In Section 5, we described the typical evolution of the epidemiology of an inherited disorder, and in particular the fact that much of it is based on studies of selected (clinically affected) populations and is subject to selection and ascertainment biases.

Consequently, the mortality risk associated with a DV may be imperfectly described by currently available epidemiology, especially if the DV is known only on the basis of DNA sequence. Ancillary information such as family history might improve the estimation of the associated risk, but might also be disclosable to a life insurer, increasing the premiums and reducing the payoff of the gamble.

Moreover, since policies with very long terms are needed to increase the likelihood of a payout, two more, implicit, assumptions come into play, namely: (a) that today's epidemiology will be reliably stable over the policy terms; and (b) that medical advances will not significantly reduce mortality. These seem risky assumptions for the long term. In the end, investing in life policies on the basis of genetic tests is a bet against the long-term progress of medical science.

[23]VCs, with their extremely short time horizons, are not relevant.

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APPENDIX A

An Example: Hypertrophic Cardiomyopathy (HCM)

Hypertrophic Cardiomyopathy (HCM) was a significant source of adverse selection costs in Howard (2014), and was studied in more detail in Haçarız (2020), and we use it to give numerical examples in this paper. Standard references on HCM are Elliott et al. (2014) and Gersh et al. (2011). Haçarız (2020) summarized the aspects of actuarial interest.

(a) *Physiology*: HCM is thickening of the muscle wall of the left ventricle of the heart.
(b) *Clinical Definition*: HCM is deemed to be clinically present if the left ventricular wall thickness (LVWT) exceeds 13–15 mm. This criterion for onset is often met in adolescence or early adulthood. Clinical HCM may be present but "silent"—symptomless and undetected.
(c) *Risk*: HCM presents significant risk of sudden cardiac arrest (SCA), heart failure, and (at older ages) stroke.
(d) *Genetics*: HCM is caused by mutations in any of several genes encoding for sarcomere proteins. All are dominantly inherited. Mutations accounting for about 1/4 to 1/3 of HCM have yet to be identified but are presumed to exist.
(e) *Prevalence*: The prevalence of clinical HCM is estimated to be about 0.2%, but the prevalence of known mutations has been estimated to be about 0.6%, implying that the prevalence of all mutations may approach 1%.
(f) *Penetrance*: The penetrance of HCM mutations has been estimated to be 69%, but that estimate is quite old and predates the discovery that mutations have much higher prevalence than does clinical HCM (see (e) above). When clinical HCM does develop, it does so before age 20–30 in most cases.
(g) *Mortality*: The annual mortality rate from HCM is widely quoted to be 1%, but this is potentially misleading as the endpoints used in survival analyses of HCM invariably include nonfatal as well as fatal events. Based on recent large studies an annual mortality rate of 0.55% was estimated by Haçarız (2020) (see Footnote 20).
(h) *Genetic Testing for HCM*: Cascade genetic testing (CGT) is used in HCM, rather than any form of screening. CGT works as follows.
    (1) An individual, who is a member of a family with no history of HCM being present, develops a heart problem (possibly fatal).
    (2) HCM is diagnosed and a known HCM-related mutation is identified. The individual is then called the "proband" or "index case."
    (3) All first-degree relatives of the proband have a 50% chance of also carrying the mutation. They are all offered genetic testing.
    (4) If any of those tested do carry the mutation, then all of their untested first-degree relatives also have a 50% chance of carrying the mutation, so they are offered genetic testing, and so on.
    (5) In this way, testing can "cascade" through an extended family.
(i) *Epidemiology*: Virtually all epidemiology of HCM is based on selected populations, that is, individuals with clinical HCM. The prevalence estimates mentioned in (e) above are exceptions. Estimates of penetrance and mortality rates are likely to be subject to selection and ascertainment biases.