CHANCE, NECESSITY, LOVE: AN EVOLUTIONARY THEOLOGY OF CANCER

by Leonard M. Hummel and Gayle E. Woloschak

Abstract. In his 1970s work *Chance and Necessity*, Jacques Monod provided an explanatory framework not only for the biological evolution of species, but, as has become recently apparent, for the evolutionary development of cancers. That is, contemporary oncological research has demonstrated that cancer is an evolutionary disease that develops according to the same dynamics of chance (that is, random occurrences) and necessity (that is, law-like regularities) at work in all evolutionary phenomena. And just as various challenges are raised for religious thought by the operations of chance and necessity within biological evolution, so this particular theological question is raised by the findings of contemporary cancer science: Where is love, divine and human, within the evolutionary chance and necessity operative in all dimensions of cancer? In this article, we contribute to the dialogue in science and religion by offering the following responses to this question: (1) the thought of Arthur Peacocke to claim that divine love may be understood to be at work in, with, and under our very efforts to make theological meaning of the chance and necessity that inform the evolution of cancers; and (2) Charles Sanders Peirce's evolutionary philosophy to make this claim: that the work of scientific communities of inquiry to understand and to find better ways to cope with the disease of cancer is itself the work of divine love amid the chance and necessity of cancer.

Keywords: cancer; chance; evolution; Jacques Monod; necessity; Arthur Peacocke; Charles Sanders Peirce; religion; science

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Jacques Monod: Chance, Necessity, Absurdity—and Cancer

When Jacques Monod published in 1970 a short treatise entitled *Chance and Necessity: An Essay on the Natural Philosophy of Modern Biology*, he had

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long been renowned among his fellow researchers in molecular biology. Furthermore, Monod's reception, along with François Jacob and André Lwoff, of the Nobel Prize five years earlier "for their discoveries concerning genetic control of enzyme and virus synthesis" also had brought him broad public recognition and even prestige, especially in France. Thus, during the pitched student-police battles in Paris in the spring of 1968, the name "Monod" was invoked by authorities as a leading citizen of the Republic in an attempt to bring calm to the capital.

But even on the basis of Monod's scientific achievements and social standing, no one could have imagined the tremors that the publication of his little work would cause throughout the world. At just under two hundred pages, much of it summarized then current findings in biochemical research and their evolutionary implications with a detail that, according to one reviewer, rendered comprehension by most general readers "a little unrealistic" (Steiner 1971, 5). Still, while many who picked up the book struggled to grasp its resume of evolutionary biology, almost all were impressed—some positively, some not—with what Monod himself claimed to be the major implication of that science: life arises, not out of any comprehensive plan or according to any comprehensible order, but out of chance which "alone is at the source of every innovation, of every creation in the realm of life. Pure chance, chance alone, a liberty absolute but blind, at the very root of the towering edifice of evolution: today this central notion of modern biology is no longer a hypothesis among others possible or at least conceivable. It is the *only* one conceivable, the only one consistent with the facts of observation and research" (Monod 1971, 112–13).

Although the title of Monod's book is *Chance and Necessity*, the text itself focuses mostly on the former evolutionary dynamism, and less on the latter. To be sure, necessary or law-like regularities of natural selection govern the outcomes of chance, but what those laws direct throughout evolution comes about from no necessary causes and for no purposes. Consequently, "the ancient covenant is in pieces; man knows at last that he is alone in the universe's unfeeling immensity, out of which he emerged only by chance. His destiny is nowhere spelled out, nor is his duty. The kingdom above or the darkness below; it is for him to choose" (Monod 1971, 180).

In his preface to *Chance and Necessity*, Monod cites a portion from Albert Camus's seminal work *The Myth of Sisyphus*, and throughout his own work Monod embraced a view of the human condition informed by Camus. As a result, some have linked Monod to, or seen him as, the prototype for the twenty-first century's spate of scientific scoffers of religion.

However, Monod was not so much a cultured despiser of religion like many current scientific atheists as he was one who, with some heaviness of

heart like Camus, forswore not only theological understandings of nature but all attempts to detect any purpose for it. Furthermore, Monod was descended from a long line of Huguenots, and it has been suggested that a trace of Stoicism sometimes at work among those Calvinists may have influenced his call to sternly face the world as it is—or, at least, as he understood it (Stanier 1977, 101). Whether or not Monod did appropriate this tradition for his own philosophy of the human being in the face of chance and necessity, he clearly did propose that the world is governed, not by inscrutable providence, but by austere fate. And he understood there to be a confluence between his biological research and Camus's thought: "Each of science's conquests is a victory of the absurd" (Monod 1967, 27).

Monod's work focused on the origins of life, with a particular emphasis on how genetic information was passed along, since genes, themselves, are inert. After much labor, Jacob and he reported in 1961 their hypothesis that a ribonucleic acid (mRNA), a substance whose base sequence is complementary to that of deoxyribonucleic acid (DNA) in the cell first transcribes and then translates (Jacob and Monod 1961). In doing so, they explained the mechanisms by which the text stacked within the genetic library actually instructs molecular activities—that is, the way in which genetic information actually informs.

While their studies did not focus on cancer, Monod and his colleague did not miss the importance of their findings for a variety of particular biological phenomena—including their origins in genetic mishaps: "Malignancy is adequately described as a breakdown of one or several growth controlling systems, and the genetic origin of this breakdown can hardly be doubted" (Jacob and Monod 1961, 354). Nor has the significance of Monod's focus on chance and necessity for evolutionary development been overlooked in current descriptions of the evolutionary features of cancer. For example, Monod's groundbreaking work on messenger RNA appears in a 2011 article, "The Biological and Therapeutic Relevance of mRNA Translation in Cancer" (Blagden and Willis 2011). In "Models of Experimental Evolution: The Role of Genetic Chance and Selective Necessity," the authors identify many selective forces in the midst of chance genetic mutations at work in the development of cancers (Krakauer and Wahl 2000). In "Chance or Necessity?: Insertional Mutagenesis in Gene Therapy and Its Consequences," Monod's landmark book is cited and then its categories employed to suggest ways to prevent leukemia that may result from therapeutic gene insertions (Baum et al. 2004). Again, Monod's legacy is evident in an assessment of the relationship between one's occupation and exposure to carcinogens in "Lung Cancer Among Silica-Exposed Workers: The Quest for Truth between Chance and Necessity" (Cocco et al. 2007). And Monod's work informed Sui Huang's essay, "Tumor Progression: Chance and Necessity in Darwinian and Lamarckian Somatic (Mutationless)

Evolution" that details much of what cannot and can be changed as cancers evolve (Huang 2012).

A clear explication of Monod's significance for contemporary cancer research has been put forward by Mel Greaves, a leading proponent of the link between the evolution of life and the development of cancers.

> The evolutionary process itself is dependent upon genetic variation arising from mutation and, for sexual species, recombination or genetic exchanges which, like mutations, generate novel variation. But this now poses a real conundrum in the context of genetically regulated restraints against cancer:
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> no changes in genes = no cancer;
>
> no changes in genes = no evolution = no us. (Greaves 2003, 47)

The underlying reason for this riddle may be located in the nature of the disease itself: cancer progresses—or more accurately, evolves—through the complex interplay of chance occurrences and the law-like regularities that govern the outcome of these occurrences. That is, just as DNA mutations and natural selection for these mutations are involved in the evolution of various species, so also mutational mechanisms and forces of selection are at work in the evolution of individual cancers.

The implications of the link between cancer and evolution may be framed with an even more ironic emphasis that suggests the absurdity of all biological being: while the operations of chance and necessity promote the evolution of life, so these very same forces drive the evolution of a disease that may end lives. From this perspective, cancer may be seen to be the quintessential disease of life since, within the flow of life itself, there is a pull like gravity toward the development of cancers. In Greaves's words, "Cancer then becomes a statistical inevitability in nature—a matter of chance and necessity, to quote Jacques Monod's memorable phrase applied to evolution" (Greaves 2003, 52).

But, more precisely, how is cancer a disease of evolution in which the principles of chance and necessity are at play?

Chance and Necessity: Cancer as an Evolutionary Phenomenon

Much like the chance–necessity description of Monod's work on evolutionary dynamisms, cancer too works via an evolutionary formula based on both chance and necessity. Cancers develop as a result of chance mutations that occur in the DNA and are necessitated by the natural selection processes that are ongoing in the person. In evolution, chance occurs at the level of a single organism that develops genetic changes; necessity works at the level of the selection of the organism that is best suited for a particular environment. With chance there is no selection, only a random

series of changes, but with natural selection only a given set of mutations will be selected for in law-like necessity. The induction of mutations by chance is not predictable (by definition), but the selection of those traits that are most fitting for a particular environment are predictable. Usually chance operates at the level of the individual, or in cancer at the level of a single cell; natural selection works on populations, or in the cancer on populations of cells.

Cancer starts with a single cell that acquires enough mutations in just the right genes to convey a growth advantage to the cancer cells compared to the normal cells—a more efficient usage of nutrients, lack of a need for growth factors, failure to inhibit, and so on. This first step involves the accumulation of chance mutations in a small number of genes (3–7, usually) in a normal cell that drives that particular cell to become a cancer cell selected with law-like predictable necessity into a population of cancer cells in that patient. In evolution, single egg or sperm cells acquire mutations randomly, and as those mutations make their way into the population there is a natural selection that operates to select those individuals in the population that are best adapted to the environment at hand. In cancers, mutations occur randomly in somatic cells (not eggs or sperm) in the body and those cells in the population that are best adapted for rapid growth in that particular person (environment) are selected for in a process very similar to natural selection. In both cases, chance allows for random generation of mutations and natural selection working on a law of necessity selects for those mutations that allow for best survival of the population. This chance–necessity relationship drives evolution of populations in a species and of cancer cell populations in a person. In both cases, chance operates at the generation of the mutations in single cells and necessity drives the selection of the optimal mutant in a population (although in cancer it is a population of cells). To be clear, evolution of species and evolution of cancers are not identical processes, but they have common mechanisms and common selection processes. Cancer is an evolutionary process in that it evolves in the patient over time through dynamic progress; as the body (and doctors) develop approaches to destroy the cancer, the cancer evolves mechanisms to evade them.

What drives the chance mutations in cancer cells? There are three main categories of factors: (1) endogenous factors (within the cell or body itself), (2) exogenous factors (environmental factors), and (3) a combination of endogenous and exogenous factors. Endogenous factors are perhaps most interesting because they are the factors that are generated in our own cells that cause mutations. This contribution of endogenous processes to mutations usually includes mutations that occur as a result of both the error rate of the DNA replication machinery of the cell and those mutations that occur as a function of the generation of reactive oxidation species (oxidative damage) created as a necessary by-product of cellular

metabolism. Why did natural selection permit the existence of error rates in essential cellular processes such as DNA repair? With the DNA being so important to the host cells, why would nature select for (and not against) error-prone processes? The answer to this complicated question is based at least in part on the idea that as evolving creatures we *need* mutation. In the absence of mutation, human beings cannot evolve, so we have built-in processes that permit evolution to occur. Cancer is a by-product of the evolutionary process.

Let us first look at the connection between mutation induction and the process of cell replication. Many investigators have explored the connection between cell division and cancer in recent years. Among the most provocative, recent work by Tomasetti and Vogelstein has examined the number of stem cells (cells capable of regenerating the entire tissue by undergoing continual cell division) in different organs and shown that organs that are capable of self-renewal with a large pool of stem cells are more likely than those with few stem cells to develop cancers (2015, 78–81). These authors suggest that the differences in cancer frequency from one organ to another may be explained solely on the basis of the number of cells capable of undergoing cell division within the organ. There have been several commentaries on this approach suggesting that Tomasetti and Vogelstein underestimated the risks of environmental factors and genetics in the process of carcinogenesis and that the subset of tumors they examined was skewed because many tumors that are especially prevalent in the U.S. population were excluded from the study (Ashford et al. 2015). Nevertheless, the idea that there is a relationship between the future number of cell divisions of a cell and its mutation rate has been known for some time and is not really disputed. The view that endogenous mutation frequencies are related to cell division status has implications not only for cancer induction; it is related to evolutionary biology.

Estimates of mutation rates vary depending on exactly what contributors are used for the calculation. Do we consider those mutations from environment, from natural aging, from the errors that are introduced by DNA replication alone? Estimates based on the error-rate of the DNA synthesis machinery (i.e., errors made during duplication of the DNA itself) comes out to be about three single base-pair changes (out of 3 billion) per round of cell division (Krebs et al. 2012). This is the mutation frequency that will occur simply as a consequence of cell division itself, with no contribution from environmental factors, viruses, or any other possible contributors. No matter what number one uses for the mutation frequency, as the cell increases the number of cell divisions, then, it increases the likelihood of that cell going on to accumulate mutations. Because mutations in specific genes can become a step toward cancer, then cell division is actually a risk for cancer.

Why does this mutation frequency associated with cell division occur at all? With evolution selecting for the best possible environment and the ways to allow for the optimal survival of the species, why would error rates for enzymes that synthesize DNA be tolerated? Evolution demands change, and one way to elicit change is to create mutations. Our cells would be relatively static over time if we did not have built-in error rates for our DNA replication machinery. There are several other ways to induce mutations from environmental influences, viruses, and other factors, but these are chance phenomena; nature clearly selected for something more sure-fire in this situation. Allowance for errors in the replication process, then, provides a mechanism to ensure that a cell can have natural mutation induction and as a consequence change that can be used to drive evolution. Mutations allow for adaptation to a changing environment while, alternatively, a static genome creates a situation that, when an environment changes, the cell/organism will not be able to adapt and thus will die. The mutations that are important in evolution are those that occur in germ cells (eggs and sperm) and are therefore passed on to the offspring. The occurrence of higher than needed mutation rates in somatic cells is merely a by-product of this occurrence in germs cells; after all, changes in somatic cells (non-germ cells) in the body will not be passed on to the offspring and thus will not drive evolution. Nevertheless it is mutations in somatic cells that give rise to cancer.

Based on this thinking, cancer appears to be a disease in humans that is inevitable, although it is not inevitable in each person and there are certainly behaviors and genetic factors that influence the risk for acquiring cancer. Tomasetti and Vogelstein have concluded that cancer induction is more about "luck" than about risky behaviors. The arguments from others in the field are not so much about whether cell division is associated with increasing risk of cancer (which is not really denied) but rather how important repeated cell division is to the process of formation of the first cancer cell in the body. (It should be noted that a cancer in a person is descended from a single cell that has gone awry, step by step). Cancer biologists all accept that there is an element of "luck" or "chance" in the process of cancer induction.

The chance induction of mutations can occur by the error rate of the proteins that lead to DNA duplication; but chance also plays a role in other endogenous processes that occur in the cell, oxidative damage that is a normal consequence not only of replication but also of oxidative processes in the cell that are a part of normal cellular metabolism. The by-products of metabolic activity are often reactive oxygen species that are generated in the cell, and when they are produced in close proximity to DNA are capable of generating DNA damage. The DNA damage from endogenous oxidative processes can also lead to mutations that contribute to carcinogenesis. Many people try to reduce the damage caused by these oxidative processes

by consuming antioxidants such as carrots, blueberries, green tea, and even red wine (which contains resveratrol); some studies have shown that they are useful in reducing oxidative stress in cells, although therapeutic benefits as cancer-reducing agents have been difficult to prove in individual patients (Hong et al. 2015).

Based on endogenous processes alone, normal cells can then acquire sufficient mutations with time to transform from normal cells into cancer cells, but a single transformed cell is not enough to make a full-blown cancer. There must be a series of selections that is driven by a law-like precision (necessity) that permits those mutant cells with the best growth advantage in that particular environment to survive. The key distinctions between the chance (mutation induction) and necessity (selection of those cells most suited to the environment) components of the response are in part based in the idea that the chance event involves no selection and necessity does, that the chance event is not predictable and that the necessity process is, and that the chance event occurs in a single cell while the necessity process works at the level of the population.

It should be noted that observing only those mutations induced by endogenous processes vastly underestimates the number of mutations that are induced in normal cells in a lifetime, but clearly the chance of having mutations accumulate in normal cells increases with age (and the risk of developing cancer increases with age). In addition, if one considers exogenous factors that may influence mutation frequency (such as virus infection, inflammatory processes, exposure to radiation and/or sunlight, and others) then the chances of mutation development are increased even further with age, since these are exposures that accumulate with age.

The "necessity"-driven process of selecting for those cancer cells from the population that are best adapted to a given environment are also much affected by the therapies that are given to the patient. As different chemotherapeutic drugs are given to the patient, they become part of the environment that influences the selection process. As a person is treated with chemotherapy, the cells that are resistant to the killing effects of that chemo combination survive, and the result is a drug-resistant cancer that can evade chemotherapy (Aktipis et al. 2011). This ability to overcome therapy is one of the evolutionary features that make cancer so difficult to treat successfully. Resistance to chemotherapy and immune response modulators is more common than the development of resistance to radiation predominantly because the former involves single gene changes and the latter appears to involve changes in several different subtypes of genes. In the case of drug resistance, again chance drives the initial mutations but it is necessity that selects for those mutant populations that are most likely to grow well in the presence of the chemotherapeutic agents.

These findings all point to the role of chance in cancer induction, but it is because this chance operates in all cells and is modulated in response

to both endogenous processes (such as the DNA replication machinery and oxidative damage mentioned above) and exogenous factors (such as smoking, radiation, viruses). In most cases, cancer is a type of chance response, resulting from the accumulation of mutations in just the right genes in just the right cells. Nevertheless, as there is a chance induction there is a selection by necessity that leads to the growth and development of the cells that are optimally suited to the particular environment in that particular person. In any individual mutation induction is a chance response, but as the cancer is selected for and grows it operates at the level of necessity. And thus, for humanity as a whole, cancer is an inevitable process that is the result of mutations that accumulate; the acquisition of mutations is a necessary process of our evolution since mutation is the driver of evolution and without it a species cannot evolve.

While cancers are not alien to, but a part of life, does that mean that they are necessarily a part of life—that is, because of life, there must be cancers? The answer might seem to be no for this reason: If all individual cancers come about through chance mutations—that is, through changes in DNA structure that may occur but do not have to do so—then it might seem that there is no necessity that any will ever come about.

While there is no absolute necessity for the existence of cancers, there appears to be a statistical inevitability to their occurring somewhere, some time. There are a variety of ways in which this truth might be explicated, but the very familiar example of coin tossing clearly illustrates why this is so. Each time a regularly minted coin is tossed, the odds are 50 percent that it will come up "tails." However, the more times it is flipped, the odds increase that one of those times it will turn up tails. This happens because, in all games of chance, there is also at work a law-like regularity—the law of large numbers. Those running gambling establishments know that while their "house of games" may lose heavily in one or several placed bets, it is predictable that their enterprise will succeed as the games go on. Hence the adage that, in the long run, "the house always wins." Within a cell, there are more nuanced and refined conditions than those directing the inanimate world of flipped coins that govern life and, thereby, strengthen the inevitability of cancerous happenstances.

To be sure, the odds that a single mutation of DNA will initiate a malignant process within a cell are very small. But trillions of mutations occurred worldwide as this sentence was written. Accordingly, it is a statistical certainty that a malignancy will develop somewhere, some time. Indeed, this inexorable quality to cancer becomes apparent when one looks at the mutational load over the course of an individual lifespan in the vast numbers of dividing cells that are undergoing these mutations. So while cancer is an extremely rare disease, it happens because of the background of the 10,000 trillion cells divisions that occur in the average lifetime. For example, in one particular site of origin—the prostate—we know that 80

percent of males over age 80 develop cancers there. This statistical loading of life for cancerous outcomes is why, in contemplating Monod's insights about evolution, Greaves employs a variety of gaming metaphors to describe the evolution of cancer under the heading "How Cancer Cells Play the Winning Game" (Greaves 2003, 53–68).

Next, we shall explore Arthur Peacocke's engagement with Monod as this biologist and religious thinker tried to understand the role of chance and necessity in various evolutionary phenomena. In doing so, we shall posit that Peacocke's life-long conversation with Monod contributed not only to the general field of religion and science but to religious thought about love amid the chance and necessity of cancer's evolutionary development.

Peacocke on Chance and Necessity

Peacocke's early scientific study of mutagenesis—the source of cancers—launched his life-long commitment to studies in science and religion. An accomplished molecular biologist, Peacocke made significant and enduring discoveries about a variety of DNA functions, and, in particular, the ways in which radiation may alter those processes. While it is a matter of chance whether or not a mutation will result from any single exposure to radiation, the effects following all mutations that happen to occur are predictable. These "deterministic" outcomes suggested first to Peacocke, and, in time, to many others, that more than "blind chance" was at work not only in the effects of mutagenesis but throughout all evolutionary events.

> Nature and life processes involve a combination of chance occurrences and processes that are mediated by necessity (or are deterministic in nature). For example, by necessity, a change in climate to a colder environment will select for a certain set of survival features that are predictable—thicker fur over thinner hair, longer sleep cycle over shorter sleep cycle, slower metabolism over faster metabolism, etc. Because these sets of . . . features can be predicted from a set of known parameters, they are deterministic or driven by necessity. (Woloschak 2008, 83)

What did Peacocke mean by chance and necessity? And why did he think that Monod's explanations of their functions in nature were inadequate?

First, chance: for which Peacocke understood there to be two senses. One refers to the force at work in events that cannot by any means be predicted with regularity to result from any cause or set of causes. To be sure, all physical occurrences have one or more proximate or distant causes—the fall of a Newtonian apple may involve the force of the wind, the ripeness of the fruit, and always includes gravity—but for some events, the attribution of clear and distinct causal factors to the production of specified results is meaningless. Peacocke employed the familiar case of coin-tossing as an example. A number of factors cause each toss to turn up heads or tails, but these factors are so many and their effects are so unpredictable that

we may say that the results of these factors are brought on by chance. The second meaning of chance applies to those events that occur, not for any reason, but by accident—that is, when two or more things meet, intersect, collide, or overlap that do not have to do so: Suppose that when you leave the building in which you are reading these pages, as you step onto the pavement you are struck on the head by a hammer dropped by a man repairing the roof. . . In ordinary parlance we would say it was due to "pure chance" (Peacocke 1979, 303).

Peacocke utilized both these meanings of chance to explain findings from his early cancer research on the processes by which radiation may cause genetic mutations. One process is the "interplay" between the genes of an organism and particular environmental pressures on them: "These two causal chains are entirely independent, and it is in the second sense of chance that Monod is correct in saying that evolution depends on chance" (1995, 126). The other sense of chance—that of unpredictability—also applies "since, in most cases we are not now in a position to specify all the factors which led to the mutated organisms being selected and, even less, the mechanism by which mutation was induced in the first place" (1995, 126).

While chance is at work in the biological world, so also is "necessity" or "law." By both of these terms, Peacocke meant "the principles and processes that govern the conditions for occurrences that are predictable and determinable." These directive dynamisms include, "the fundamental physical constants, the fundamental particles as well as the physical laws of the interrelation of matter, energy, space, and time and of other physical features of the universe" (1995, 321). From his observations that "laws arise that do structure and control events in the world," Peacocke concluded, "there is no reason why the randomness of molecular events in relation to biological consequence has to be given the significant metaphysical status that Monod attributed to it" (Pennock 2001, 476). Here, Peacocke contended with Monod, not as a metaphysician, but as a scientist in arguing that the principles of necessity (or law) are at work in the world: "As we already have seen in the behavior of matter on a larger scale, many regularities, which have been raised to the level of being describable as 'laws', arise from the combined effect of random microscopic events which constitute the macroscopic" (1979, 306).

Throughout his career, Peacocke argued for the ways in which chance and necessity together drive evolutionary developments.

> From the interaction of genetic mutations and natural selection, from the role of so-called chance events, in the emergence and development of life, many (as we saw) who have reflected on the processes of biological evolution have concluded that they are "due to chance" and therefore of no significance for man's understanding of the universe and of his place in it. These studies demonstrate that the interplay of chance and law is in fact creative, for it is

> the combination of the two which allows new forms to emerge and evolve. (Peacocke 1979, 313–14)

As a scientist, Peacocke discerned many ways in which life continued to evolve through the interplay of chance and necessity. In his earlier theological works, he concluded that God created and sustained through these same evolutionary processes: "This combination for a theist, can only be regarded as an aspect of the God-endowed features of the world. . . . God is the ultimate ground and source of both law ("necessity") and "chance" (2001, 477). In bringing life into the world, God acts like a musical composer who extemporizes "a fugue to create the world *through* what we call "chance" operating within the created order, each stage of which constitutes the launching pad for the next" (2001, 477). That is, Peacocke proposed that God's acts of creation are kenotic, "whereby *God suffers in, with and under the creative processes of the world* with their costly, open-ended unfolding in time" (2001, 477).

As his work in theology unfolded, Peacocke became a leader in a religion/science scholarship burgeoning with writings on evolution, chance, and necessity. As he drafted more theological works, each with its own melodic line, Peacocke conducted himself much like a composer of fugues by harmoniously weaving his own line of thought into the mix of others in this field. And as Peacocke was dying from cancer, the compliment was returned by a gathering of theologians who invited him to summarize his life's work on many matters including "natural evil" or the suffering brought on by creation: "When faced with this ubiquity of pain, suffering, and death in the evolution of the living world, we are impelled to infer that God, to be anything like the God who *is* Love—must be understood to be suffering in, with, and under the creative processes of the world" (2007, 25).

Like Monod, Peacocke concerned himself throughout his career with the meaning of life evolved and evolving. Indeed, Peacocke believed that, in their life-long journey of grappling with the dynamics and significance of evolution, he and Monod had a common origin: "I [have] suggested that Monod and I were at least fellow-voyagers setting out from the same home port of the scientific perspective on the world" (1973, 23). However, in his bringing theological interpretations to evolution, Peacocke understood himself to have been aiming for a very different destination than Monod. "The course I have steered approaches a very different land-fall from that of Monod. I am not pretending that the journey by the route I have indicated will be any less stormy, indeed some nights may be darker, but, if we had time to travel this route further, I would suggest that a gleam of light could be discerned on the horizon, perhaps even that 'day-spring from on high' which was promised us" (1973, 23). Monod was a careful and daring scientific thinker who highlighted the function of chance in biological being. As a scientist, Peacocke responded to Monod by emphasizing the

role of necessity alongside of chance in evolution. As a theologian, Peacocke added to his scientific observations on both random occurrences and law-like regularities that his own belief that God was at work in them but in a way that did not add anything observable to that work. Next, we shall employ his theological convictions about the presence of the divine amid such evolutionary dynamisms to develop our evolutionary theology of cancer.

Peacocke's Encounter with the Evolution of Cancers

At the close of his life, Arthur Peacocke was surrounded by a community of inquiry into the significance of his previous religious proposals that God worked in all that is. Peacocke appreciated the importance of the moment afforded him by this attention. "It was only during this time that the enormity of what I had to face up to gradually dawned on me and this catalyzed me to finishing off 'An Essay in Interpretation' concerned with a more naturalistic understanding of the Christian faith which I hoped would be congenial to more orthodox believers as well as those who are seriously challenged by the scientific world view as the norm for their thinking" (2007, 191). Peacocke's encounter with death precipitated not only his closing remarks on theology and science, but also an account of his own faith amid the turbulence of evolutionary chance and necessity. A few weeks before his death, Peacocke composed and circulated a final statement. He titled it "Nunc Dimittis," the Latin translation of the opening of Simeon's canticle in Luke 2: 29–35: "Lord, now lettest thou thy servant depart in peace, according to thy word: For mine eyes have seen thy salvation, which thou hast prepared before the face of all people . . . ." (Clayton 2007, 3). In considering Monod's thought, Peacocke had predicted that he, himself, might meet up with existential struggles and storms along his life-journey. As evident in the following summary of and commentary on this final testimony, he had such encounters—as well as experiencing his anticipated vision of the "day-spring from on high."

> Up until July 2004 I was blessed with a long, healthy and fruitful life. In July 2004, in my eightieth year I was diagnosed not only with prostate cancer, but having it in an advanced form. This was an enormous shock to myself and my wife. . . . By [2005], I was taking an enormous range of pills, bouts of nausea were becoming frequent, and it was becoming less and less possible to envisage a normal life of any kind. I was trying to be stoic and trying not to inveigh against God for what was clearly going to be my fate—a fate I had not really envisaged or imagined. (2007, 191)

After a career replete with productivity and well-being, Peacocke received his diagnosis of an "advanced" (in his case, fatal) cancer as a heavy blow. In response both to learning about his disease and to the suffering it brought him, Peacocke strove to be "stoic"—a term that could mean many

things but, in Peacocke's case, seems to signify simply that he was trying to control himself. What might Peacocke have been trying to contain within? He shared that he was attempting not to "inveigh against God"—and, perhaps despite himself, confessed thereby that he was tempted to do so. In choosing to use the word "inveigh," Peacocke informs us that, despite himself, he was drawn to protest vehemently against God.

What troubled Peacocke was an impending "fate" that he had not predicted. Twice, he uses this term that would connote to all such as himself who are familiar with Monod the lack of purpose for life given the capriciousness of the world. Peacocke's chance meeting with what he had not "envisaged or imagined" impels him to pray for something more.

> Over the years I have given much thought and spilt much ink on the nature of God and God's interaction with people. Not surprisingly the subtler nuances of my deliberations have fallen away before the absolute conviction that God is love and eternally so. This remains the foundation of my prayers and thoughts for "underneath are the everlasting arms." This is not always easily experienced and it needs much concentrated meditation—the "black dog" of depression is sometimes difficult to expel. (2007, 192)

Peacocke articulates both his foundational religious beliefs and destabilizing existential despair. To describe the state of his soul, he employs Winston Churchill's menacing canine metaphor for depression. To maintain his conviction "that God is love and eternally so," he claims the need for meditation on that conviction. It appears, therefore, that as Peacocke's inducement to inveigh increased, so did his need to focus on another reality. In his account, the tension continues to build as Peacocke, having described his understanding of cancer, his consequent physical ailments, and how his fundamental spiritual convictions were being torn at by temptations, next revealed that a major source of this existential struggle derived from a single subject with which he had wrestled throughout his career as a scientist and theologian.

> [M]y concern[s] over the years has been the recurrence of what theologians call "natural evil." I have often attempted to illustrate the ambivalence of this concept, for example showing that what we call natural evil is a consequence of a divinely created law-like structure implementing the divine purpose to bring into existence intelligent persons. *The irony is that one of the examples I took was the role of mutations in DNA which are the basic source of evolution, and so of the emergence of human beings—and also of cancer.* This [illness] is a new challenge to the integrity of my past thinking. [Emphasis Added] (2007, 192–93)

It was Peacocke's fate to have begun his career studying the chance and necessity of cancer, next to live well into a quite ripe old age with no signs of his life's ending, and then to abruptly experience both career and life undone by the very subject of his study—and in a manner that seems to

have brought him great physical discomfort along with its psychic insult. Throughout his life journey, Peacocke had argued that sufferings brought on by nature are not evil because they are part of its fabric. Yet, at his life's end, Peacocke experienced himself as being struck down by the disease that came into being through the same processes that brought him and all humanity into being—and he was struck hard by that irony which, as noted, may be summarized as:

> no changes in genes = no cancer;
>
> no changes in genes = no evolution = no us. (Greaves 2003, 47)

How might one make meaning of this—that human being cannot come to be except through the very process that also can cause it to cease to be? How might one solve this problem?

Or is this a problem that can be solved in the sense of its being a puzzle that must or even can be pieced together? Or, rather, is it more among the mysteries of human being that humans may come to accept? Peacocke offered the following answer:

> I am only enabled to meet this challenge by my root conviction that God is Love as revealed supremely in the life, death and resurrection of Jesus the Christ.
>
> However the fact remains that death for me is imminent and of this I have no fear because of that belief. (Peacocke 2007)

Peacocke continued his testimony by recollecting the famous story of spiritual transformation recounted in Bede's history of the coming of Christianity to the Kingdom of Northumbria in England (597 CE). In his own rendition of this testimony by a court advisor, Peacocke omitted Bede's concluding sentence that summarizes this Northumbrian's desire for insights into the mystery of being: "Therefore, if this new teaching has brought any more certain knowledge, it seems only right that we should follow it" (Bede 1990, 130–31). In its stead, Peacocke shared his own account of having been enlightened: "I know that God is waiting for me to be enfolded in love." This, then, is the knowledge toward which Peacocke oriented himself—or rather, the knowledge that appears to have oriented Peacocke toward his end: "Death comes to everyone and this is my time." In the end, Peacocke did not receive the fact that his life and his death were rooted within the same evolutionary process to be a problem, but to be a mystery with which he could live as he was dying. In light of this understanding of divine love for him and all creation, he claimed that he could accept their connectedness—and testified that he could depart in peace.

Next, we shall consider the significance of the evolutionary philosophy of Charles Sanders Peirce. While Peirce did not reflect on the disease of cancer

from which he did die, we shall explore his insights into the possibilities and problems of understanding love—both divine and human—in a world of chance and necessity in order to develop an evolutionary theology of cancer.

C. S. Peirce's Evolutionary Philosophy of Chance, Necessity, and Love

Among Peirce's abiding scientific/philosophical efforts were his inquiries into the first principles of evolutionary processes during an era when "agnosticism was then riding its high horse and was frowning superbly upon all metaphysics" (Peirce 1934, 12). The poet Susan Howe paraphrases Peirce's own descriptions of meetings by a "Metaphysical Club" in the late nineteenth century, reflecting how this community of inquiry strove to make meaning of evolution: "A knot of us . . . gathered to discuss metaphysical/questions force law fate Darwin" (Howe 1999, 66). The labors of this ensemble helped birth American pragmatism—a movement that proposed to a scientific age focused on facts and evidence that thoughts and ideas were not inconsequential, but had their own empirical effects and practical bearings. As pragmatism developed, its proponents continued to express this central conviction in a variety of ways, and later in his life Peirce did so this way: "It is a perfectly intelligible opinion that ideas. . . have a power of finding or creating their vehicles [in the minds of humans], and having found them, of conferring upon them the ability to transform the face of the earth" (1931, 220).

Of course, some such transformations are for the better, while others are not; for example, certain ideas may lead people to do harm, while other ideas may influence them to help others. But in these cases and in many other instances, ideas do have power to create new facts in the world. In particular, Peirce proposed as a testable hypothesis that the principle of love could affect the course of evolution. And he suggested that humanity had a role in bringing about this change for a more harmonious—and, ultimately, more loving—universe.

Peirce was a philosopher open to a mix of religious and scientific thought, and nowhere was this availability more apparent than in his analysis of evolution. He parsed out three dynamisms at work not only within life, but throughout the cosmos. "Three modes of evolution have thus been brought before us: evolution by fortuitous variation, evolution by mechanical necessity, and evolution by creative love. . . . The . . . propositions that absolute chance, mechanical necessity, and the law of love are severally operative in the cosmos may receive the names of *tychism*, *anancism*, and *agapism*" (1935, 302). Having observed and recorded regularities from his days as a coastal surveyor, Peirce thought it incontrovertible that a dynamism best described as "necessity" directs the course of events throughout the world.

Yet, while he appreciated how the concept of necessity helps in framing many mechanics of nature, Peirce found it inadequate to explain the innumerable free-flowing currents also coursing throughout creation. To account for this persistent "blooming and buzzing confusion" as William James put it, Peirce turned to chance as an enduring force: "Everywhere the main fact is growth and increasing complexity. . . there is probably in nature some agency by which the complexity and diversity of things can be increased. . . . the theory of chance merely consists in supposing this diversification does not antedate all time" (James 1981, 262; Peirce 1931, 65). Peirce believed creation itself reveled in the force of chance, because chance allowed creation to continue to create—to birth new, complicated, and often higher forms of being into the world.

While new being emerges from chance, so, too, does chaos. As a philosopher with strong moral sensibilities Peirce therefore wondered if evolutionary chance and necessity might be have some end-point or purpose. As a scientist addressing an era also struggling with the social implications of differing and frequently competing evolutionary theories, Peirce claimed he could discern in the world a third force—that of love at work through chance and necessity and directing it toward harmony. To support this claim, Peirce referenced findings by physicists that many initially chaotic conditions such as gaseous states eventually do settle down. He also pondered the statistical properties of the law of large numbers (involved in coin-tossing and gambling) for indications that the oddities brought on by chance might eventually even out. To be sure, because chance occurrences are inevitable, creation will always have instabilities; indeed, without this wild force there could be no new being. But Peirce conjectured that, over time, the power of love might gradually tame chance and render its rough places so plain that they would become negligible (Hacking 1990, 315).

Peirce used many strategies to schematize how love might bring about such a positive transformation of the natural order. He employed the mathematical figure of asymptote to exhibit his hypothesis that the harmonious practical bearings of love might, over time, lessen the effects of chance: when a line and curve are asymptotic, the distance between them gradually approaches, though does not reach, zero as they extend themselves toward infinity.

Peirce proposed that, through a similar progression, love might gradually tame chance, so that chance will come infinitesimally close to meeting up with love.

Prima facie, Peirce's notions about "evolutionary love" may seem fanciful at best—the ingredients of an exotic religion and science cocktail that do not blend well. Indeed, Peirce's science of evolution alone appears to be an incoherent mix of Darwinian and anti-Darwinian elements. Certainly, Peirce's thoughts on the power of love to make the world more peaceful contain components of a somewhat naïve progressive-era optimism—of the

sort that Monod eschewed—that the world will, sooner or later, become a better place.

In the end, however, something plausible persists in Peirce's notion that love is drawing the world toward better ends—for that idea, in true pragmatic fashion, may have the practical effect of leading persons to act lovingly toward one another and the world. Peirce himself understood it this way: that it was not only the idea of Gospel love that could transform the world, but also the conduct of those acting in sympathy with this idea that could do so. "Under this conception, the ideal of conduct is to execute our little function in the operation of the creation by giving a hand toward rendering the world more reasonable whenever, as the slang is, it is up to us to do so" (Peirce 1931, 615). Peirce's proposal about *agape* was a bold one: under its sway**,** humanity might tame chance for the purpose of greater harmony and less suffering. And his notion may appear less a flight of fancy when linked to the more general pragmatic principle that ideas may have great power— sometimes for ill and hopefully for the good.

Still, while bold and hopeful, this question remains: what might this theory about love working through chance and necessity, in fact, mean for the evolutionary developments of the disease of cancer? That is, what might be the practical bearings of Peirce's evolutionary love for a theology of cancer? At first glance, Peirce's theory of evolutionary love may not seem to offer anything helpful for our particular purpose. His philosophy of evolution may not appear to signify much for the case of cancer because, in its destructive authority over other cells, the evolution of cancer through chance and necessity does not seem to be tending toward any kind of harmonious conclusion. Thus, as cancers develop and continue along their evolutionary course, the line of love that might bring about a diminution of cancer suffering does not appear to be asymptotically curving toward the line of cancerous developments.

However, we do not conclude that Peirce's evolutionary philosophy itself suggests such a tragic finale for the evolution of cancers. Rather, we suggest that our critical review of his philosophy may be employed to propose the following: scientific understandings of the evolutionary nature of cancer and their practical bearings in better ways to intercept its evolutionary developments may testify to divine love amid the evolutionary chance and necessity of cancers.

Chance, Necessity, Love: An Evolutionary Theology of Cancer

As noted earlier, cancer is not a simple disease. To be sure, cancer may be easily described as a disease of cells—the very constitutive elements of bodies and being. Yet, cancer is not simply the disordered inner workings

of any one or many cancerous cells, but the disorder that results from the complicated network of relationships that those cells establish with normal cells. Cancer may also be simply portrayed, on the one hand, as a disease of genes within cells, and it is most certainly that. On the other hand, as cancers progress with reference to the genetic library, the genome itself is often transformed into a bewildering chaos. And, while this disease of cells and genes does unfold through space and over time according to some very identifiable evolutionary hallmarks, the occurrence and timing of these typical features appear in novel ways in every individual case of cancer—and they do so precisely because every evolutionary event is a unique phenomenon.

Indeed, as we have noted, it is quite possible that cancer is the most complex disease that exists. Since it was discovered that cancer evolves in the unique environment of each patient, the realization that no two cancers are identical and that different mutations accumulate in different cancers emerged. This means that characterization of the end-stage tumor is very different than what the characterization of the "early" tumor would have been; there are many more genes that are mutated late in the disease because the cancer is continually evolving than there are mutated early in the disease process (Forbes et al. 2015). This makes it difficult for people involved in developing anticancer therapies to develop appropriate drugs to target the disease. So, to be sure, common features do exist among cancers, but, with their intrinsic variability, cancers find ever new ways to develop (Kitano 2003, 227–35).

Nevertheless, along with their complexity, cancers have contours that may be grasped, that is, "comprehended." That is, now we have a clear and distinct idea of what cancers are, and our understanding of cancers is as sure and certain as this: cancers are cells that go their own way according to evolutionary dynamisms of chance and necessity. Running throughout the chaos of this disease of cells and genes, with its tale-tell hallmarks, are these very principles of evolutionary development that, themselves, are comprehensible and provide us a means of wrapping our understanding around the phenomenon of cancers. In addition, we can now not only outline the fundamental features of cancers, but we can also fill in details of those features more precisely. Because of our efforts, cancer is less a closed box so that we may now better determine the genetic events required to initiate it and then to move it along.

To be sure, the course of our increasingly precise knowledge of cancer has not been straight and smooth, but rather long and winding. That progress has been uneven because our knowledge of this evolutionary disease has itself been evolving through chance discoveries and subsequent integration of those findings. And as Peirce noted, all individual scientific findings may miss the mark of a final truth even as their varying conclusions may increasingly cluster around the bull's-eye of such truth. Accordingly, we

may never arrive at a complete understanding of cancers, nor may we ever arrive at a full understanding of their origins and development that would enable us to prevent or cure all of them. However, we are approaching better understandings of cancers that may enable us to better cope with them. Accordingly, Peirce's words about all scientific inquiry apply to cancer research: "Despair is insanity. True, there may be facts that will never get explained but . . . we must be guided by the rule of hope" (1931, 405). Our increasingly precise understandings of the inner workings of cancers are themselves the practical bearings of inquiry guided by hope.

Furthermore, our improved scientific understanding of both the contours and inner features of cancers has borne a particularly good fruit: the increased ability to intervene as the disease develops. To the degree that we understand cancers to be an evolutionary phenomenon, to that degree we have been thereby enabled to treat and, in some cases, to cure those with it. The first designer drug that was specifically developed to target a unique oncogenic protein was the drug Gleevec; this drug was directed against a novel protein found only in some cancers and therefore could attack the cancer cells specifically without affecting any normal cells. Understanding the molecular changes that occurred as the cancer progressed allowed for the generation of a targeted anticancer strategy that proved successful (Goldman and Melo 2003). This case and many others demonstrate that the more we understand how cancers progress according to evolutionary principles the better we are able adapt to them and, in some cases, to create ways to overcome them. Downstream studies aimed at predicting not just tumor response but also tumor evolution in a specific patient may facilitate these efforts even further; this may be difficult because of the "chance" nature of cancer evolution, but predictions about probabilities of particular evolutionary directions surviving in patients, or the "necessity" of the cancer surviving in particular patients, may be possible, particularly when we examine patients with specific diseases (diabetes, for example). The chance–necessity paradigm of cancer evolution applies not only to the development of the cancer but also to the therapeutic dimension of cancer in a patient.

To be sure, evolution is the very dynamism that predicts that cancers will always develop somewhere, some time. But with our understanding of the ways in which cancers are evolutionary events, we may now better predict their ways and, through various interventions, intercept their development. Love, divine and human, may be understood to be at work on our ever-increasing capacity to redirect the flow of cancerous chance and necessity.

While these findings of cancer science may not be able to display divine purposes in nature, they may be means by which humans may further divine purposes for nature. Accordingly, all members of the cancer science "community of inquiry"—including those who profess belief in a God and those who do not—may be, in Peirce's words, "lending a hand" for divine

purposes. From this perspective, we can understand the practical bearings of Monod's thought in each and every one of the previously catalogued articles that build on his work as instruments by which divine love may be operative in a world of chance and necessity. That is, we may review the previously cited list of research derived from Monod's atheistic speculations on chance and necessity to be, paradoxically, products of divine love by which we have been enabled to cut into the evolutionary phenomenon of cancer. To the degree that similar research may bring about better understandings of the disease that, in turn, may lead to better treatment for those suffering from it, to that degree such attempts may be received as the work of divine love in the world. In the world of the finite—and in God's time stretched out infinitely before us—we may be guided by the hope that the asymptotic distinction between the persistence of cancers and the power of divine/human love may so diminish that, to be sure, cancerous suffering would still obtain, but, being an infinitesimal distinction, might no longer be perceivable or be experienced.

From a Peircean perspective, the science of cancer may offer us tools to respond wisely to a world with cancers by helping us to learn to accept what cannot be changed about them and also to hope to change what can be. By wise responses, we mean those whose practical bearings provide us tools "of . . . reshaping as much of the environment as is within our power in order to destroy the factors in the universe that work against our well-being and even our very survival" (Smith 1983, 21). That is, through their research, scientific "communities of inquiry" do strive to understand and, increasingly succeed at understanding the evolutionary nature of cancers—and the efforts of these communities, carried out by persons both of faith and of no faith, may evidence the divine love in their ability to work on the evolution of cancers.

Conclusion: "Trying to Understand:" Divine and Human Love amid the Evolution of Cancers

Along with his many successes, Monod's life was also one of struggle. During the Second World War, he remained in Paris where he assumed leadership in the resistance. And though his name was invoked in order to bring peace during the 1968 uprising in Paris, he received a cold shoulder at a gathering of students on whose behalf he interceded. A striking, close-up photo of Monod several days afterwards portrays him carefully holding the hand of a blinded student and escorting her away from the street–fighting as he looks forward longingly while an accompanying Red Cross worker beckons for assistance. An epitome of Monod's lifelong striving for a better world may be found in the final words that, as he lay dying of leukemia, he whispered to his brother: "Je cherche à comprendre" "I am trying to understand" (Smith 1983, 616).

As he contemplated his impending death from cancer, Arthur Peacocke offered his own understanding of how one may both recognize the chance and necessity at work in cancer as well as affirm the presence of divine love for the world amid this evolutionary phenomenon. In his theological reflections on the significance of that disease at these final moments, he framed ideas on the goodness of God that extended his lifelong reflections on divine presence in a world of evolutionary changes. To be sure, in doing so he faced squarely the existential challenges brought to human being—his own, included—by the dynamisms of chance and necessity. At the same time, he was able to proffer an account of divine presence amid these evolutionary forces as they expressed themselves in the disease of cancer.

A lasting gift of Jacques Monod's "trying to understand" has been his descriptive framework for the chance and necessity operative in all evolutionary events. A major contribution of Arthur Peacocke was to offer a theological understanding of these same evolutionary processes. "Either way, his [Monod's] or mine, our duty is clear—it is that first enjoined on self-conscious thinking man by Plato through the mouth of Socrates, that our duty is to take whatever doctrine is best and hardest to disprove and embarking upon it as upon a craft, to sail upon it through life in the midst of dangers" (1973, 23). For an evolutionary theology of cancer, the implications of Peacocke's particular way can be described accordingly: divine love may be operative in, with, and under theological meaning making about the chance and necessity that inform the evolution of cancers.

To be sure some theological formulations about the meaning of cancer may not bring consolation for suffering and, in fact, not a few ill-conceived ones may be more deadly to the human spirit than anything that the disease alone might bring. Nevertheless, in the midst of existential struggles with the disease of cancer, spiritual transformation of the sort described by Peacocke is evidentially possible. And theological formulations such as Peacocke's that provide accounts of divine love through the emergence of evolutionary phenomenon like cancer are plausible, if not compelling.

> At that subtle moment when man glances backward over his life, Sisyphus returning toward his rock, in that slight pivoting he contemplates that series of unrelated actions which become his fate, created by him, combined under his memory's eye and soon sealed by his death. Thus, convinced of the wholly human origin of all that is human, a blind man eager to see who knows that the night has no end, he is still on the go. The rock is still rolling. (Camus 1955, 123).

The link that Monod himself recognized between the philosophical implications of his scientific findings with Camus' reflections on the human condition has been detailed in Sean Carrol's excellent recent work *Brave Genius* (2013). That connection also is clear in the above citation from

Camus's *Myth of Sisyphus* with which, we noted earlier, Monod began his own seminal work on chance and necessity. Near the conclusion of that essay, Camus writes: "I leave Sisyphus at the foot of the mountain! . . . He too concludes that all is well. . . One must imagine Sisyphus happy" (1955, 123). The French word for "happy" here is *heureux*, among whose meanings is that of being satisfied with one's fate or fortune—of not being crushed by chance but, instead, of being content with and consenting to what one has been handed. To first face what one has been dealt, and then to become human in the face of it, is the quintessential existential response to a world shaped by chance and necessity.

"Happiness" in the face of the fact of cancer's presence in the world—in the strictest sense of being content with and consenting to the persistence of that presence—is certainly one possibly human and humane response to this disease. Not only the statistical inevitability of cancer's appearance somewhere in the biosphere but also its sometime juggernaut quality as it may evolve within individual lives do call for acceptance of cancer's existence as something that may not always be able to be changed.

However, another response—born of the human capacity to imagine and sometimes create better conditions in the world—is also possible. This response, consonant with the pragmatic tradition of Peirce, exhorts humanity to exercise these powers in order to contend with forces that threaten "our well-being and even our very survival" (Smith 1983, 21). While the progressive thought of Peirce and other pragmatists may slip into a naïve optimism that Camus, Monod, and others of their school have rightly disdained, the effect of that philosophy also may be to impel us to look for better ways of living—of "lending a hand" in a way that may better human being. In doing so, we become co-creators as our abilities to respond to cancers themselves evolve as cancers evolve.

Accordingly, we employ Charles Sanders Peirce's evolutionary philosophy to make this novel claim: the work of scientific communities of inquiry to understand and to find better ways to cope with the disease of cancer is itself the work of divine love amid the chance and necessity of cancer. Since cancer is the inevitable consequence of human evolution, at some level it is a consequence of being evolving human beings and is of necessity required for our existence. Based on this, it is clear that cancer is a consequence that humanity must face in order to be evolving creatures that can survive in this world; as such, then, each person that suffers from cancer is paying the price for humanity's evolution and therefore deserves our love and support. Our responsibility as human persons is that we must care for those with cancer because they are suffering the consequences in place for all humanity; their disease is the disease of all humanity, and their suffering is the suffering of all humanity. We have a responsibility of love, care, and ease of pain and suffering that is a needed consequence of our responsibility to others who are suffering on behalf of all of humanity.

In this article, we have highlighted how cancer is a disease of evolutionary processes driven by the dynamics of chance and necessity throughout its development. Accordingly, we have posed this question: where is love amid these evolutionary developments? We have directed the thought of Peacocke and Peirce to claim, respectively, that meaning may be made of these evolutionary forces in cancer and that love may be discerned in the human capacity to respond to these dynamics. We further propose that it behooves the community of inquiry engaged in the religion and science dialogue to find even more ways to discern the power of love—divine and human—at work given the evolutionary development of cancers. Indeed, we suggest that the very act of religious reflection on the place of love amid the chance and necessity of cancers may, itself, be one of the ways in which love—divine and human—may be discerned in a world with cancers in it.

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