Analyses of past disasters may supply insights to mitigate the impact of recurrences. In this context, we offer a unifying causative theory of Old Testament plagues, which has present day public health implications. We propose the root cause to have been an aberrant El Niño-Southern Oscillation teleconnection that brought unseasonable and progressive climate warming along the ancient Mediterranean littoral, including the coast of biblical Egypt, which, in turn, initiated the serial catastrophes of biblical sequence — in particular arthropod-borne and arthropod-caused diseases. Located beyond the boundary of focal climate change, inland Goshen would not have been similarly affected. Implicit in this analysis is a framework to consider a possibility of present day recurrence of similar catastrophes and their impact upon essential public services.

INTRODUCTION

The vivid Old Testament saga of the 10 plagues that devastated the land of Egypt and its people (Exodus 1-12) has intrigued some to seek rational explanations for a chronicle of disasters that befall one population yet spared another. Indeed, biblical scholars in a 21st century translation of the Old Testament concede that from an historical standpoint, the first nine plagues resemble natural events well known in the Middle East, save for their patterns and rapid succession [1]. In light of present day knowledge, we offer a fresh, cohesive, and rational explanation of these events, with the implication that they could recur. The 10 plagues are: 1. the Nile River turns bloody, fouling drinking water and killing fish. 2. Frogs leave the Nile for dry land, invade Egyptian homes and die, causing a great stench. 3. Annoying small insects swarm. 4. Annoying large insects swarm. 5. An epizootic kills different types of livestock in pasture. 6. Boils afflict beasts and humans. 7. An especially severe thunderstorm with lightning and hailstones destroys crops near harvest. 8. Strong winds bear swarms of locusts to obliterate remaining crops. 9. “Palpable darkness” obscures all light. 10. Firstborn Egyptians and their

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†Abbreviations: RVF, Rift Valley fever; RVFV, Rift Valley fever virus; ENSO, El Niño-Southern Oscillation; WNV, West Nile virus.
surviving firstborn animals die, while Israelites and their livestock live.

**BRIEF LITERATURE REVIEW**

In parallel with scientific advances, succeeding authors have offered progressively rational explanations for the plagues, as well summarized by Marr and Malloy [2]. Over the past half-century, authors of four key papers have put forward competing scientific views. These are characterized by differing suggestions for origins of plagues 1 and 5, and whether there was a common source for some plagues, or alternatively serial connections between them. In the late 1950s, Hort attributed the bloody appearance of the Nile River and the fish kill of plague 1 to reddish silt and freshwater flagellates (*Euglena sanguinea, Haematococcus pluvialis*) being carried downstream by torrential waters to overflow onto the floodplain of the Nile Delta [3,4]. She proposed that the unusually strong river overflow caused anthrax spores in contaminated soil to germinate and initiate the epizootic of plague 5 and attributed all but the hail of plague 7 to Nile overflow. Several decades later, Schoental suggested that red tide due to pigment-producing marine dinoflagellates was responsible for the bloody Nile and fish kill. She, however, proposed a sequential plague connection, e.g., the flies of plague 4 transmitting an unknown infectious agent to cause plague 5 [5]. Hoyte, in 1993, postulated different fresh water dinoflagellates (Gymnodinium and Glenodinium sp.) as causing plague 1, but agreed with Schoental that the flies of plague 4 transmitted the agent of plague 5, and also caused plague 6 [6]. As origin of plague 5, Hoyte rejected previously suggested bacterial and viral agents — including *Bacillus anthracis* and Rift Valley fever virus (RVFV) — based upon differing livestock susceptibilities to infectious agents, offering instead the protozoon, *Trypanosoma evansi*. Thereafter, Marr and Malloy suggested a different species of freshwater dinoflagellates (cyanobacteria) for plague 1 and made a connection with plague 2 by proposing that dinoflagellate toxins forced frogs to flee the river. Moreover, they presented a model in which each of the first nine plagues shaped the next, thereby greatly advancing the concept of sequential plague causation. They also revived consideration of viral origin for plague 5 by suggesting that two viruses — African horse sickness and Bluetongue — were responsible, having been spread by vector insects of plague 3 [2]. In contrast to varying opinions as to plagues 1 and 5, authors of the three papers addressing plague 9 all agreed the cause was a sandstorm [2,4,6]. Therefore, while differing in detail, previous scholars have considered that living agents and abnormal climatic conditions (also manifested in the hail of plague 7 and the winds of plague 8) account for the first nine plagues.

It is the 10th plague — the seemingly inexplicably pattern of selective killing of Egyptian firstborn humans and animals yet sparing all Hebrew humans and animals — that has posed the thorniest problem for rational elucidation. Attempts to explain plague 10 vary greatly. Hort essentially avoided the issue by proposing translation and transcription errors. In her view, the original Old Testament concept of this plague was the “destruction of the first-fruits” — meaning crops, not children. Hoyte also downplayed the significance of firstborn deaths, maintaining it was symbolic for the death of the pharaoh’s eldest son from typhoid fever. (Hoyte also took note of hyperbole or poetic license in quantitative biblical descriptions, e.g., death of “all” animals or contamination of “all” waters, as do current biblical scholars [7]). Somewhat more creatively, Schoental, and later Marr and Malloy, argued that the firstborn died from lethal mycotoxins (e.g., *Stachybotrys atra*) arising in moldy granaries. The latter argued that primogeniture permitted firstborn humans to have had first and greatest exposure to the moldy food, as did dominance for firstborn animals. They attributed survival of younger human and animal siblings to their prompt perception and avoidance of dangers lurking in moldy granaries — but offered no support.

There is also contention as to the location of the biblical land of Egypt: Hort con-
sidered it to have been the whole Nile valley as far south as ancient Nubia, whereas Hoyte argued that the Hebrews of the Old Testament would have perceived it only as the Nile Delta. In either event, it would have likely included the places of forced Hebrew labor at the “treasure cities” of Rameses and Pithom in the northeastern Nile Delta and Egyptian dwelling places close to the Mediterranean coast (Figure) [7-9]. By contrast, scholars generally agree that the part of the land of Egypt known as the region of Goshen would have been further inland, north of present day Wadi Tumilat and under desert climate influences [2,3,7]. Situated at a periphery of the coastal plain, Goshen would have been a pastureland for settled and transient foreigners, including semi-nomadic Hebrew herdsmen.

Addressing the central biological or weather event in each plague, we now offer a fresh explanation of both remote and proximate causation, based on historical examples of comparable catastrophes. We also put forward a simple biological explanation for the seemingly inexplicable 10th plague.

FOCAL CLIMATE CHANGE PROPOSAL OF CAUSATION

Drawing from present day knowledge of the interplay of climatic conditions, ecological changes, and arthropod-borne and arthropod-caused diseases, we suggest that the immediate cause of all plagues — in biblical sequence — was unseasonable and progressive climate warming along the eastern Mediterranean coast where Israelites worked in forced labor. The seminal event for the warming would have been an El Niño-Southern Oscillation (ENSO) teleconnection. The inland region of Goshen — beyond the climate warming — essentially would have escaped the unseasonable effects.

The El Niño warming component of the ENSO cycle occurs when the Southern Oscillation reverses the usual low atmospheric pressure system over the western Pacific Ocean to the east, thereby raising surface temperatures of the central and eastern Pacific in the tropics and atmospheric temperatures of adjacent land masses. This climatic effect may go beyond the tropics, as when an El Niño warms the eastern Mediterranean basin during late winter and early spring [10]. Hence, biblical Egypt as perceived by Hebrews at labor in “treasure cities” near the Mediterranean coast (Figure) would have been in a credible location to experience ENSO-induced climatic warming. In Africa, India, South America, and China, ENSO atmospheric warming has caused heavy rainfall; mosquito-borne outbreaks of Rift Valley fever (RVF), malaria, and dengue; explosion of swarming locusts; and an historically severe red tide causing massive fish kills [11-16]. Indeed, a disaster of biblical proportions began in coastal Peru in March 1925 when an ENSO warmed the surface of the eastern Pacific Ocean and raised the ambient temperature of adjacent coastlands from 15° to 26° C over some three months, bringing deluging rain to a usually arid re-
Large numbers of frogs, dragonflies, crickets, and mosquitoes appeared and were followed by epidemic diseases — seemingly dengue and malaria. It is noteworthy that climate warming alone, without rain, may initiate outbreaks of mosquito-borne disease. This repeatedly occurred in Colombia during the last decades of the 20th century when ENSO-induced increases in ambient temperature together with drought led to stagnation of moving rivers, breeding of vector mosquitoes, and outbreaks of dengue and malaria.

**AUTHORITATIVE BIBLES AND TRANSLATION INCONSISTENCIES**

For consistency, we used the well-recognized and authoritative sources of two different translations of the Old Testament published in the 20th and 21st centuries by the Jewish Publication Society [18,19]. Since not all scholars agree that the word choices in these translations reflect the original Hebrew text, we also include independent scholarly opinion (personal communication, Ms. Janice Friend). The cause of plague 3, ‘kinim’ (phonetically) has been interpreted to mean either gnats or lice, and is translated as “gnats” in the 20th century edition and “vermin” in the 21st, the latter adding in annotation that ‘kinim’ is a non-specific description of small insects including mosquitoes and lice. However, in the Hebrew text ‘kinim’ is used in two ways (Exodus 8:12-15): ‘ha’aretz v’haya l’kinim’, [take the staff and strike the earth and there will be ‘kinim’] and ‘v’lo yacholu vat’hi hakinim ba’adam uvab’ha’ima’ [and the ‘kinim’ ate or bit both man and beast].

There is even less unanimity for the translation of ‘arov,’ the cause of plague 4 (Exodus 8:17-20): ‘tishacheth ha’aretz mip’nai he’arov’ [the land was devastated {destroyed, decayed, or spoiled} in the face of the ‘arov’]. The 20th century version translates ‘arov’ as “swarms of flies;” the 21st as “swarms of insects;” adding in annotation that the meaning is uncertain. Some rabbinic scholars consider the meaning to be flies; others, wild animals based in part on a literal interpretation of Psalm 78:45. “He sent among them swarms of ‘arov’, which devoured {bit or ate} them; and frogs, which destroyed them.” Most Hagaddot, however, interpret ‘arov’ to mean flies as do seven of nine articles addressing plague 4 in literature reviewed by Marr and Malloy [2].

We take ‘kinim’ to mean various small, annoying insects including mosquitoes and midges, and ‘arov’ as a contrasting general term for larger organisms, particularly flies.

There are also differences in translations in plagues 5 and 10 as to the subjects of epizootic disease. The 20th century translation employs “cattle” in both; however, the 21st century version uses “livestock” in plague 5 and “cattle” in plague 10. In the Hebrew text, plague 5 is visited upon beasts or a group of animals as ‘b’mikn’chaasher basadei, basousim, bachamorim, bag’malim babakar ovatzon’ [among your herds, among your horses, donkeys, camels, cattle and sheep] (Exodus 9:3). The victims in plague 10 are ‘b’heima’ as in ‘kol b’chor b’heima’ [every firstborn among the beasts] (Exodus 12:29) and ‘mayadam v’ad b’heima h’ [among men and among beasts] (Exodus 12:12). The word ‘b’heima’ is used throughout Exodus 11 and 12 as meaning beasts.

We take “cattle” to be a generic term for two distinct collections of livestock: animals in pasture that are killed in plague 5 and animals destroyed in plague 10 that are located elsewhere — presumably at Egyptian dwellings.

**ENSO EFFECTS AND THE FIRST SIX PLAGUES**

As a parallel to the 1925 Peruvian and more recent ENSO episodes, unseasonable and progressive focal climate warming would have lasted two to three months. This span would have permitted a biblical perspective of plagues as a concatenation of successive, and somewhat overlapping, disasters. Coinciding with the start of the lambing season and the later celebration of Passover, plague 1 would have begun near the vernal equinox as the ENSO teleconnection raised ambient temperatures above 20°
C. Plague 6 would have occurred one to two months later, when continued warming brought daily temperature highs above 23° C. At that time, tropical ocean temperatures usually would have been less than 18° C during March and cooler than now, according to a paleoclimatological model [20].

1. Water temperatures rising above 18° C at ancient debouching branches of the Nile River would have stimulated growth of dinoflagellates cysts as has historically occurred in the harbor of Alexandria, Egypt [21,22]. *Alexandrium minutum* grows well in waters of low salinity having input of nutrient-rich freshwater, conditions likely to have obtained in biblical times at the outflow of the Nile at its late winter-early spring low as human and animal waste and other sewage flowed toward the confined estuary. When temperatures exceeded 20° C, eutrophication and nutrient upwelling would have promoted massive algal blooms of red tide and their toxins. Prevailing onshore north winds would have blown the toxic blooms upriver, causing its bloody appearance, fouled drinking water, and fish kill.

2. Increasing water temperatures would have stimulated activity of frogs (e.g., *Rana ridibunda* [23]), and the toxic river environment would have forced active frogs to flee to land. Toward nightfall as temperatures fell, the frogs would have sought warmth in human dwellings; later, lethargic and dehydrated, they would have succumbed to opportunistic infection [5]. Anaerobic bacteria growing in frog carrion would have made the land stink.

3. Continued atmospheric warming would have fostered breeding in swampy areas of various annoying small insects such as biting midges (Culicoides species) and sand flies (Phlebotomus species). Culex, Aedes, and other mosquitoes would have hatched in irrigation ditches near pastures — some to become virus vectors in plague 5 [24-26].

4. Biting flies, having hatched in soil heavily polluted with animal urine and feces, also would have become abundant with warming weather. As noted by others, stable and black flies (including Stommys and Simulian species) would have been especially bothersome [2,3,6]. Eggs of some non-biting flies would have hatched on or about animals or humans — in bedding or clothes; their larvae, being capable of growth in mammalian tissue, would later cause plague 6 [27].

5. Concurrent RVFV infection in ruminants and West Nile virus (WNV) in equine species would account for the epizootic of plague 5, having been freshly introduced into the Delta at a time when neither had been present for 10 to 20 years. At temperatures generally ranging between 20° C and 22° C, vector mosquitoes systematically and slowly would have spread both viruses to livestock throughout the pastures, eventually infecting the vast majority. Mature Egyptian herdsmen exposed in the pastures would have been unaffected, having acquired active immunity in long-past exposures [28,29]. In historic times, both RVFV and WNV intermittently have been introduced into the Nile Delta without becoming established for long periods: RVFV episodically has been transported with infected livestock, and WNV intermittently spread by infected birds [30,31].

6. After hatching, specific fly larvae would burrow into skin and subcutaneous tissues of livestock and humans and develop in situ to cause furuncular myiasis, manifested as inflammatory nodules or boils. Historically in Egypt and Saudi Arabia, several species have caused myiasis in camels, goats, and sheep [27]. In humans, the tumbu fly (*Cordylobia anthropophaga*) has played a role [32,33]. Tumbu fly larvae often produce myiasis in legs and buttocks, which might explain the biblical observation that Egyptian “magicians could not stand before Moses because of the boils” [34].

**ENSO EFFECTS AND THE LAST FOUR PLAGUES**

By late April or May, progressive rises in coastal warming would have brought daily high temperatures above 25° C to initiate a supercell storm and cause plague 7.
As rising temperatures progressed and daily highs along the coast approached 29°C, lasting effects of plague 7 would set conditions for plagues 8 to 10.

7. Severe springtime storms occasionally arise in the Middle East. A dangerous hailstorm occurred in Egypt as recently as 1999 [35,36]. The storm of plague 7 was especially severe, bringing hailstones “such as had not fallen upon Egypt since it had become a nation.” It would have started as warm, moist sea air moving over the northern Delta collided with cool, dry inland air, causing thunder, lightning, heavy rains, and then large hailstones, destroying crops near harvest. Afterward, the storm would have left puddles of rainwater and promoted new vegetation from pastures to dwelling places.

8. Violent storm winds blowing first east and then west from the desert would have, respectively, conveyed swarms of desert locusts (Schistocerca gregaria) into and out of coastal Egypt. An exact parallel occurred in 1967, as swarming desert locusts were first brought to the seacoast of Egypt from the Arabian Desert by a cyclonic east wind, and then carried away by a similar west wind [37].

9. As violent storm winds subsided, prevailing warm sea winds laden with moisture would have returned to the northern Delta to overlay cool desert air left by plague 8. Condensation of moisture would yield a dense advection fog. This sea fog would have obscured all light along the coast. Absent dissipating wind or air shift for three days, darkness would have persisted. Dense fogs in Egypt have caused multiple fatal vehicular accidents as recently as 2006 [38].

10. New vegetation and water puddles resulting from the storm of plague 7 would have promoted breeding of mosquitoes and attracted birds. With ambient temperatures approaching 29°C, both Aedes and Culex mosquitoes would have become increasingly efficient for disseminating RVFV, and Culex species for disseminating WNV to Egyptian inhabitants of humble and palatial dwellings [39,40]. Immune mature Egyptians would have survived exposures to both viruses [30,31]. Non-immune younger Egyptians (including firstborn) and domiciled livestock would have died from one or the other.

**DISCUSSION**

Disturbed climatic conditions over the biblical land of Egypt clearly are evident in the Old Testament account of plagues 7 and 8, bespeaking the turbulent atmospheric events of a supercell storm bearing hailstones to destroy crops and veering winds to drive swarms of locusts to and fro. Unseasonable springtime warming would have precipitated such a storm, like the thunderstorm over the Sinai desert of Egypt in April 1998 occurring after an unusual one-week heat wave [35]. To account for an extended two- to three-month period of unseasonable climate warming that would have led to the succession of plagues, it is necessary to infer a more extraordinary climatic phenomenon. We make the case that the seminal event was an ENSO teleconnection, causing progressive warming along the Mediterranean coast of biblical Egypt for such duration. Goshen, lying beyond the boundary of climate change, would thereby escape all or most plagues.

The description of the 10 plagues depicts an increasingly severe sequence of public health catastrophes: red tide and its toxic sequel of fish kill, water pollution and expulsion of frogs from the Nile; explosions of various arthropod populations; two episodes of epizootic and epidemic diseases (the second causing deaths of firstborn); a turbulent atmosphere with thunder, lightning, rain and hail, and then violent winds; and loss of daylight. Historical records of catastrophes occurring after atmospheric disturbances caused by ENSO teleconnections mirror the Old Testament account, save for hail and loss of daylight [16,11-15,17]. We propose that the hail and loss of daylight also had an ENSO origin in a supercell storm. The Old Testament record provides an opportunity to consider the breadth of public services that might be required in event of another series of ENSO calamities.
Were a populous region to now be struck with catastrophes that parallel the biblical elements, essential public health, medical, and veterinary services likely would be overwhelmed. In fact, as preparation for adverse public health effects of climate change, Fromkin and colleagues at the U.S. Centers for Disease Control and Prevention are calling upon public health policymakers and professionals at all levels to plan coordinated responses [41]. In event of sustained increases in mean ambient temperatures, they anticipate a risk of tropical arthropod-borne infectious diseases, including RVFV and WNV outbreaks, and propose both surveillance of vector-borne diseases and the potential for vector-borne diseases, the latter to be done by monitoring temperature, rainfall and vector populations.

**VIRUS DISEASES, LIVESTOCK STRESS AND PLAGUE 5**

Bunyavirus and flavivirus outbreaks have been documented after an ENSO cycle [11,17]. Neither RVFV (a bunyavirus) nor WNV (a flavivirus) alone can account for disease in all livestock types enumerated in plague 5. Whereas WNV infection often may be fatal to equines [42], we find no reports of WNV infection causing serious disease, let alone death, in adult camels. In contrast, RVFV infection is often deadly for many ruminant species, but does not cause clinical signs in equines [43]. Some young ruminants are especially susceptible to RVFV: The mortality rate of infection for newborn lambs is greater than 90 percent, whereas for sheep it is 30 percent [44]. Healthy mature camels usually manifest no clinical findings of RVFV infection, save abortion [5,43,45]. When severely stressed, however, camels may die from RVFV infection. Early in the 1977 RVF outbreak in Egypt, there were 39 implicated RVFV deaths in 40 camels in transit from the Sudan; the animals also suffered trypanosomiasis and starvation [46,47]. Ultimately, this lot accounted for 69 percent of all 56 camel deaths in the outbreak [47]. Again in the 2000 Saudi Arabian RVF outbreak, deaths of tens of thousands of infected camels and other livestock occurred among livestock in transport at the Red Sea port of Jazan [48]. In both outbreaks, RVFV infection was the proverbial “last straw” for the camels — the ultimate in a chain of stresses that accompany livestock transportation [49]. In the Old Testament chronicle, RVFV infection would have been the ultimate stress for camels and other ruminants in pasture — already suffering exposure to inclement weather, parasitic infestations, malnutrition owing to poor quality of late winter pasturage, and dehydration due to the limited water in irrigation ditches at seasonally low levels. Livestock recently acquired from nomadic caravans additionally would have borne stresses inherent to transportation. Among severely stressed biblical equines, the virulence of WNV infection also would have been enhanced, as has been demonstrated in laboratory animals [50].

**EPIDEMIOLOGY AND PATHOGENESIS OF PLAGUE 10**

The ancient and continuing Egyptian practice of stabling livestock within dwellings would have facilitated human contact with RVFV-infected livestock [29]. Since RVFV may be secondarily spread by direct contact, susceptible biblical Egyptians would have become infected at their homes during care or butchering of RVFV-infected animals, as well as through direct contact with infected persons [51,52]. They also would have been exposed to RVFV through milk of infected animals — in pastoral communities, milk is still a food staple. Moreover, RVFV and some strains of WNV may cause fulminating hepatic and/or hemorrhagic disease in humans [46,52-55]. When hepatic disease was a feature of fatal human RVFV infection, autopsies in Egypt often showed advanced schistosomal cirrhosis [56]. As schistosomiasis was endemic among ancient Egyptians, hepatic cirrhosis due to schistosomiasis would have been expected [57]. Fulminating RVFV or WNV infection superimposed upon preexisting hepatic cirrhosis would have been rapidly
lethal to susceptible biblical Egyptians [53]. Finally, to the extent it was prevalent, a specific genetic characteristic among non-immune biblical Egyptians — the defective CCR5 allele leading to absence of chemokine receptor CCR5 — would have increased fatal outcomes of WNV infection [58].

In those parts of Africa where RVFV and WNV were enzootic during the 20th century, the prevalence of antibodies to each, although widespread, was low in persons younger than 15 to 20 years [30,31]. Lacking the active immunity of mature elders, firstborn biblical Egyptian children exposed to RVFV through contact or to either virus through mosquito transmission would have been unprotected from lethal infection. Conversely, younger siblings still or recently at breast, possessing transplacental passive immunity, would have been protected. Such a dramatic contrast would have fostered the perception of vulnerability of the firstborn in plague 10.

**EVEN TUATION OF PLAGUE 5 INTO PLAGUE 10**

Residual effects of the heavy rains of plague 7 would have extended the geographical range of transmission of both viruses from pastures to human habitations. Close to dwelling places of the biblical Egyptians, new vegetation and standing water would have increased vector mosquito numbers and attracted birds [59,60]. Around the dwellings, infected birds would have amplified WNV, and infected lambs would have amplified RVFV. In plague 10, the contrast in mortalities of RVFV-infected lambs with RVFV-infected sheep near human dwellings — absent stresses of pasture life — would have lent a perception of widespread deaths among firstborn animals.

As atmospheric warming continued, the end result of masses of newly hatched vector mosquitoes near Egyptian dwellings rapidly amplifying both viruses, in concert with secondary contact spread of RVFV, would have been the devastating plague 10. At the time of plague 5, postulated ambient temperatures greater than 20° C but less than 23° C would have permitted Aedes and Culex mosquito vectors to steadily — but not rapidly — transmit RVFV among pastured ruminants, and Culex species to slowly spread WNV from flocks of nearby birds to grazing equines. Continued climbing temperatures, along with many more mosquito vectors, would have accelerated transmission of both viruses; at ambient temperatures of 26° C, these mosquitoes would have become very efficient transmitters [39,40]. Historically, some period of warm weather is necessary for introduced RVFV to develop into a major outbreak. RVFV was brought into Egypt in 1977 and spread northward largely unnoticed through the summer and early autumn; in October, it exploded into an epidemic and epizootic in the Nile Delta [25,46,47]. Similarly, RVFV was identified in Saudi Arabia in mosquitoes in 1999, yet was recognized as an epidemic and epizootic only in August–September 2000 [48,61]. Along this line, experimental findings demonstrate a need for physical warming of vector mosquitoes for WNV transmission. No WNV could be recovered from virus-inoculated Culex mosquitoes held at 10° C; however, WNV was recovered from comparably inoculated mosquitoes additionally warmed to 26° C for three to six weeks [62].

Absent similar climatic conditions in Goshen, comparable disasters would not have occurred. Interestingly, recent studies in the Nile Delta utilizing the newer technique of thermal scanning radiometry correlate the focal distribution of filariasis, a disease also transmitted by Aedes, Culex, and other mosquitoes, with focal surface and subsurface moisture in the soil and plant canopy [63]. This finding lends support to the view that the occurrence of the 10th plague in the land of Egypt, but not in Goshen, may have been due in large part to focal differences in moisture.

That overlapping mosquito-borne outbreaks of virus disease do occur was repeatedly documented in episodes caused by Western encephalitis and St. Louis encephalitis viruses in Kern County, Califor-
nia, during the latter decades of the 20th century [64]. Recovery of WNV from mosquitoes in a setting of enzootic RVF in Mauritania in 1987 and at the end of an RVF epidemic in Egypt in 2002 provides historical support to the concept that these viruses together could have caused plagues 5 and 10 [65,26]. Additionally, observations made during an RVF outbreak in Mauritania in 1988 seem relevant: As expected, the finding of icterus was significantly greater among persons with laboratory evidence of recent RVFV infection than in those without [52]. However, this was not the case for individuals having hemorrhagic signs, which led the seasoned investigators to consider the possibility of an additional outbreak agent.

CONCLUSIONS AND OUTLOOK

The present analysis of the origin of the Old Testament plagues offers a possible unifying theory of interrelation of a single atmospheric event with a series of unseasonable climate changes causing all calamities. Continuing scientific advances may permit future development of a formal hypothesis based on our analysis. 1) Directions for new inquiry could include new scientific investigations to explore possibilities of ENSO occurrences having caused major climate changes along the southeastern Mediterranean littoral during biblical times — current measurement of flood heights of the Nile River and tree ring widths in Asia along with analyses of ice core samples offer climate estimates of only the past 500 years [66]. New measurements of isotope tracers in banded reef coral in the Red Sea and temperature at various depths in the earth’s surface might reconstruct earlier climatic conditions and permit estimations of variations across a range of inter-seasonal to inter-decadal periods in ancient times [10]. 2) If nucleic acids from mummified humans and animals permitted reconstruction of viruses, this might shed light on origins of epidemics and epizootics in biblical Egypt [67]. 3) Continuing studies of historical descriptions in ancient written sources could yield new insights into contemporary climatologically related health risks.

Outbreaks of arthropod-viruses are documented throughout tropical and subtropical regions following ENSO atmospheric reversals [11,13,14,17]. Recent dissemination of so-called “tropical” viruses such as Blue-tongue, Chikungunya fever, and WN has resulted in autochthonous infections in mid-latitude zones [68-70]. In future ENSO cycles, such viruses may be important public health concerns in temperate regions.

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