

Safety of Travel in South Africa: The Kruger National Park

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South Africa is a flourishing tourist destination and the South African Tourism Board (SATOURL) recorded a steady growth in international visitors of about 11% per annum for the period from 1994–1999.¹ The four most popular sites visited by tourists are Cape Town and environs, the Garden Route, Oudtshoorn ostrich farms, and the Kruger National Park. The Kruger National Park (KNP), which at over 1.949 million hectares, is the largest wildlife or nature reserve in a single African country, affords the visitor the opportunity to appreciate a magnificent variety of flora and fauna, in their natural environment (Fig. 1). In 1998 almost one million tourists visited the KNP.¹

In recent years, the various infamous titles bestowed on South Africa, including the dubious dual distinction of being referred to as the world's "rape and high-jacking capitals," have reputedly slowed the projected 20% per annum tourism growth in South Africa (personal communication, Dale Pretorius, CEO, SATOUR, Jan. 2000). Extensive press coverage of South African epidemics, including cholera, bovine tuberculosis, and malaria,

appear to have negatively influenced the volume of tourism to the KNP. Although it is not possible to directly measure the impact by contacting all people considering a visit but deciding against it because of perceived risk, the proportional monthly distribution of visitors is informative. When considering overnight visitor numbers, available from the Kruger National Park Commercial Development and Tourism database for the period April 1997 to March 2000 inclusive, it is noteworthy that only 17.8% of visitors stayed in the KNP during the January to March quarter. This is the time-period commonly perceived by the media to be of highest malaria risk. The impact of negative media perceptions of malaria risk is particularly apparent for January 2000. Widespread media panic followed deterioration of the malaria situation in KwaZulu-Natal. The lowest proportion (3.8%) of residential visitors of any month in the past 4 years occurred in January 2000, compared with an average of 6.6% for January during the previous 3 years (Fig. 2). Media coverage of a cholera outbreak during February 1998 in rural Mpumalanga was also accompanied by a decrease in residential visitors, decreasing to 4.6% for that month, compared with an average of 5.6% for the month of February from 1997–2000.

The image of safety in African nature reserves has been tarnished by a number of criminal activities in recent years.² Travelers have encountered life-threatening situations from armed bandits in East Africa. An increase in armed conflicts, especially in northeastern and central Africa, has made military automatic and semi-automatic weapons easily available for use in robberies and attacks. Public concern is fueled by media reports and specific features of nature reserves ensure that actual or perceived untoward events in the reserve or surrounding environment will be newsworthy. Factors found to characterize newsworthiness include novelty, seriousness, potential for epidemic spread, the bizarre nature of an event, human interest and drama, all elements that could be present.³

Although a central tenet of travel medicine is that prevention should be based on epidemiological data, limited attempts have been made to formally catalogue

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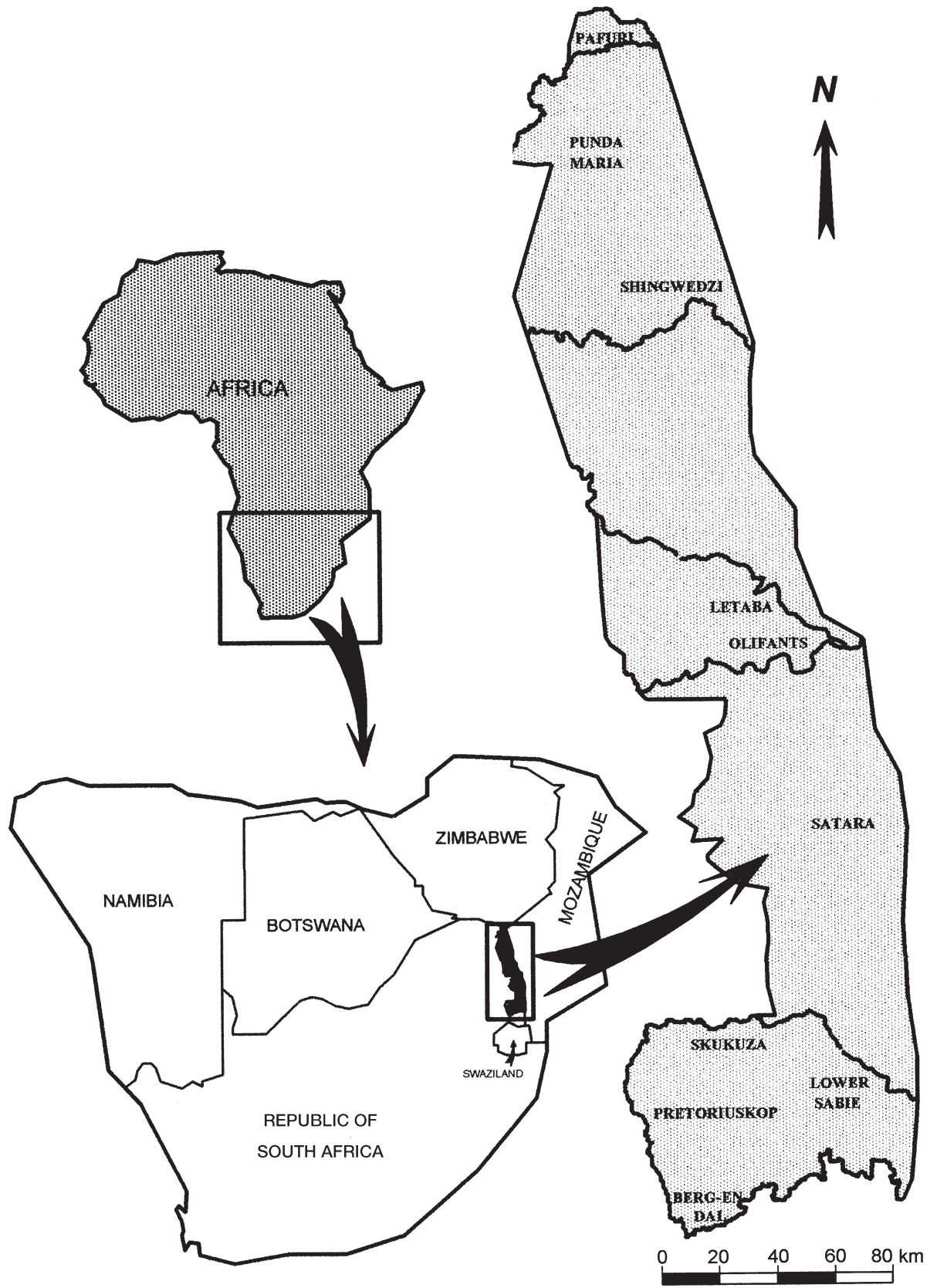


Figure 1 The Kruger National Park in relation to Africa and southern Africa.

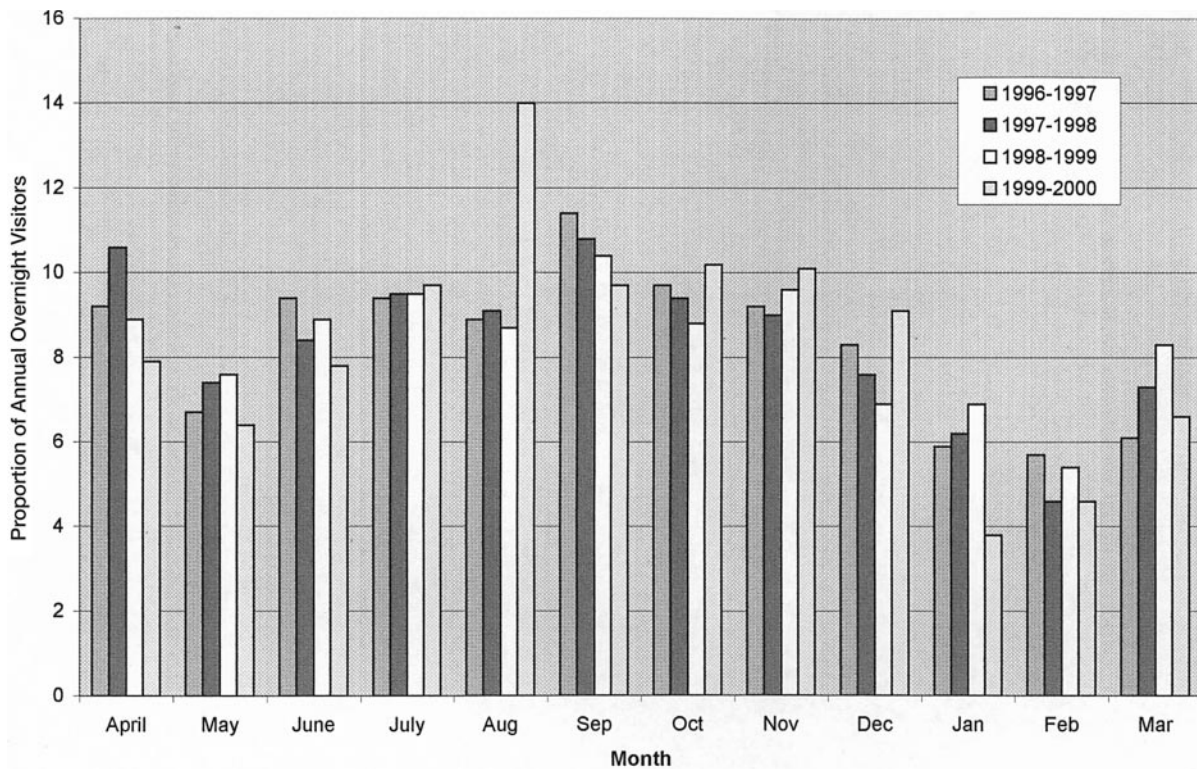


Figure 2 Proportional monthly overnight visitors, Kruger National Park, April 1996–March 2000.

and quantify the hazards encountered by tourists visiting African nature reserves, and the KNP in particular.⁴ Currently, recommendations of travel medicine advisors, and risk-modifying measures adopted by travelers, are largely based on anecdote, personal opinion, and related nonscientific approaches. An additional strategy used to estimate risk to travelers, particularly for infectious diseases, is extrapolation from local population data.⁵ This approach has major weaknesses, as routine demographic and disease notification data in African countries are notoriously inaccurate.⁶ In addition, most tourists enjoy considerably safer environments than rural populations that bear the brunt of these health-related events. Tourists typically consume food and water of higher microbiological quality, are exposed to fewer mosquito bites, have better access to medicines and medical assistance, and may have a better understanding of disease and its prevention, than most local residents in developing countries. Thus using country level malaria, schistosomiasis, rabies, food and waterborne diseases, tuberculosis, and arboviral disease prevalences, to estimate risk for travelers to the KNP may not be appropriate.

The object of the present review is to catalogue potentially important health-related conditions that exist in the KNP, attempt to provide a realistic appraisal of risk within the limits of available data, and briefly describe measures taken by the KNP administration to modify spe-

cific risks. A broad variety of information sources were exploited to obtain these data including a comprehensive review of published and unpublished scientific research conducted in the KNP, commercial press records, postal and in-flight surveys, police records of criminal and traffic offenses, local general practice records, and interviews with key informants. Various routine databases, including the food-handler screening program, abattoir records, water monitoring records, and notification records of statutory notifiable medical conditions were also used. This methodology has the limitations inherent to studies utilizing retrospective data sources not specifically designed for use in research. Although this means that exact risks could not be quantified, it was also advantageous, removing potential Hawthorne effects that might have existed if data collection had been set up prospectively.⁷

Vector-Borne Diseases

Malaria

Malaria accounts for 9% of all disease in Africa, and over one million people, mainly small children, die from malaria annually.⁸ Recently there has been an increase in malaria prevalence in Africa, and four factors are incriminated: resistance of *Plasmodium falciparum* parasites to chloroquine and sulfadoxine-pyrimethamine; resistance

of Anopheline mosquitoes to insecticides; climate changes; and large-scale migration.^{9,10} South Africa has also been affected, and the past three decades have seen a greater than 100-fold increase in the number of malaria cases notified to the National Department of Health, from 364 cases in 1971 to 51,433 cases in 1999.¹¹ This is despite a residual intradomicillary spraying program that has seen the distribution of malaria in South Africa reduced by 80% and limited to the northeastern areas bordering Mozambique, Swaziland, and Zimbabwe.¹² Novel approaches, guided by operational research, including the seminal introduction of rapid malaria diagnostic tests at program level, exploiting mosquito vector behavior for cost-effective community control measures, and employing high-resolution geographical information systems (GIS) for planning and evaluating control, have seen notable improvements in malaria control and management in Mpumalanga Province, which adjoins the southern KNP.^{13–15}

South African health authorities use seasonal malaria incidence at district level to guide malaria chemoprophylaxis recommendations for tourists.¹⁶ Although available GIS capabilities allow a finer resolution, and timely definition of risk in rural communities resident within the malaria area, it is naïve to equate the risk in rural villages with that of tourists in wildlife reserves, like the KNP. Factors contributing to this disparate risk include length of stay in the malarious area, location of dwelling in relation to vector breeding sites, disproportionate cost of most commercially available personal protection measures to local residents, and type of accommodation used. Using rates of malaria in villages adjacent to the KNP may overestimate the risk of malaria to travelers, and lead to unnecessary use of antimalarial prophylaxis, with associated drug-related adverse events.¹⁷

To more accurately quantify malaria risk to visitors to the KNP, a postal survey was conducted during 1996 in the large cohort of visitors that stayed in the KNP during the month of April.¹⁸ This was an epidemic year, and April was chosen because this is a higher risk malaria month, with a relative increase of 89.6% over the monthly average of reported malaria cases in South Africa.¹⁹ The attack rate of 4.5 cases of *P. falciparum* malaria per 10,000 visitors during April, is not only much lower than that in the highest risk rural communities in Mpumalanga Province, South Africa, 20 per 1,000 per annum, but it compares very favorably with the malaria risk defined in visitors to other popular African destinations e.g., 30 per 10,000 visitors per month for travelers to Kenya.^{15,20} In deciding whether malaria prophylaxis should be advocated, it is essential to establish whether the risk of malaria outweighs the risk of serious prophylaxis associated side-effects. Although no association was demonstrated between malaria risk and use of chemoprophylaxis,

since the risk of serious neuropsychiatric side-effects with, for example, mefloquine is 1 in 10,000, there appears to be merit in promoting use of effective anti-malarial medications at least during the summer rainfall months.²¹ This period extends from September to May in the KNP.

It would be ideal to continuously monitor travelers to generate relevant risk estimates, but unfortunately this is not feasible, as annual large-scale postal surveys are resource-intensive. However, it may be possible to approximate malaria risk trends in visitors, from the incidence of malaria in KNP employees. This proxy risk trend is probably valid because employees are also not believed to enjoy any immunity due to the seasonal nature of malaria in South Africa. Employees' risk represents an upper risk limit, as they perform duties outdoors during the peak nocturnal vector-biting period, and few, if any, employees utilize malaria chemoprophylaxis. Staff members are continually cautioned to maintain a high-index of suspicion of any febrile symptoms, and to immediately seek medical care and definitive diagnosis, that is immediately available within the KNP. These cases are notified by general practitioners located in the KNP. The rates amongst employees during the past 4 years were 12.6% (1996), 4.4% (1997), 5.8% (1998) and 8.6% (1999) per annum. The lack of an increase of cases amongst KNP employees, since the time of the traveler survey, provides reassurance that the risk of malaria to visitors is unlikely to have escalated, if their incidence is a reliable proxy measure of risk to travelers.

However, research into KNP visitors' knowledge and behavior indicates that there remain opportunities for further reducing personal malaria risk. Although the majority of visitors surveyed during the peak malaria season used either chloroquine and proguanil in combination (35.6%), or mefloquine (18.4%), a sizeable proportion used regimens not recommended in this chloroquine-resistant area, including chloroquine alone (15.7%).²² Only 30% of travelers using prophylaxis reported being fully compliant, using antimalarial drugs both regularly as prescribed and for 4 weeks after leaving the malaria area.²³ Although this finding is similar to that of a study in Kenyan travelers, it is a source of concern, as severe malaria is more often the result of premature cessation of drug, than of using the wrong drug.^{20,24}

KNP visitors' use of personal measures to avoid mosquito bites and their knowledge of malaria are also inadequate. Despite the safety record of topical N,N-diethyl-m-toluamide (DEET) containing insect-repellent, use and evidence of the value of adequate clothing cover, insecticide and/or coils, air conditioning and/or bednets, and repellents for reducing malaria risk, these measures are underutilized by KNP visitors.^{25,26} A postal survey found that 13% of visitors used no personal protection

against mosquito bites during the highest risk season, and only 17% used four or more effective measures.²⁷ A disconcerting finding of a survey amongst in-flight passengers returning from Skukuza Airport in the KNP, was that more than a third of travelers did not recognize fever or flu-like illness, as an important symptom of malaria.²⁸

A remote warm water spring with a unique breeding colony of *Anopheles arabiensis* Patton mosquitoes, the dominant malaria vector in South Africa, has proven a valuable site for seminal research on the behavior of these vectors for improved cost-effective malaria prevention.^{29,30} Research has shown that the peak biting activity of *An. arabiensis* is during the predawn period, and that 81% of bites occur on the ankles or feet.³¹ Wearing closed shoes, or raising feet off the ground, dramatically reduces vector contact, as does application of small doses of DEET containing insect repellent to the feet, without redistribution of bites to more proximal body parts.³² The value of various locally available commercial and plant-derived repellents for reducing bites is also currently being assessed.

In addition to conducting essential research, the KNP routinely embarks on a number of actions to reduce malaria. All residential units are sprayed twice per year with a residual synthetic pyrethroid insecticide (deltamethrin), and windows and doors are fitted with mosquito-proof gauze. Larvaciding with *Bacillus thuringiensis* is performed in residential camps, and to a diameter of 300 meters outside. This is extended to 800 meters during high incidence years, when a helicopter is used. The KNP advocates the use of antimalarial chemoprophylaxis during the malaria risk months and personal protection measures against mosquitoes throughout the year. Malaria information brochures are available at entrance gates, reception offices, shops, and on the Internet.^{33,34} Mosquito repellents, coils and vaporizing mats are available in all camp shops for purchase by visitors. Two doctors, equipped with malaria diagnostic tests, and effective therapy, are available at the largest camp, Skukuza.

Arboviruses

Chikungunya, an alphavirus, and dengue and yellow fever, both flaviviruses, are mosquito-borne diseases most dramatically associated with a hemorrhagic fever picture. Rift Valley fever, a phlebovirus, also relies on a mosquito vector for survival and transmission, and infrequently causes human disease in South Africa.³⁵

Chikungunya is a mosquito-borne virus of Africa and Asia that causes febrile illness with a maculopapular rash and protracted arthralgia of a variable number of joints. Wild primates serve as reservoir hosts of the virus.³⁶ Although anthropophilic populations of *Aedes aegypti*, the most efficient vector mosquito, reach high den-

sities along the KwaZulu-Natal and Eastern Cape coast during summer, such populations are rare in inland South Africa.³⁷ This absence of anthropophilic *A. aegypti* may explain why the three previously recorded human outbreaks of Chikungunya virus, in Mpumalanga and Northern Province, that border the KNP, remained focal in rural savannah areas. These outbreaks (1956, 1975/1976, and 1977) involved epizootic transmission between baboons and vervet monkeys, and the sylvatic mosquito, *Aedes furcifer*, following heavy rains and resultant high densities of these treehole breeding mosquitoes.³⁸⁻⁴³ *A. aegypti* was not involved in any of these outbreaks.

Dengue fever is not endemic in South Africa and previous occurrences resulted from importation of cases into the Durban area of KwaZulu-Natal from the Indian subcontinent.⁴⁴ Although Natal coastal *A. aegypti* have been shown experimentally to be competent vectors of Dengue virus, South African *A. furcifer*, which is found in the KNP, is a poor vector of Dengue 1 and 2 viruses.⁴⁵

In Africa, yellow fever is a tropical disease of West and Central Africa that does not occur in South Africa. Its introduction into South Africa is considered highly unlikely as this would require a person who had not received protective vaccine to be infected from a sylvatic source in rural South America or Africa and then rapidly travel to South Africa in the summer months to infect man-biting *A. aegypti*. As already emphasized, anthropophilic *A. aegypti* is restricted to the eastern coast of the country, and therefore there is no risk of yellow fever in the KNP.⁴⁶

Rift Valley fever (RVF) was first recognized as a major epizootic in South Africa, during 1950/1951 with the loss of approximately 100,000 sheep and cattle, and approximately 500,000 abortions in sheep.⁴⁷ Concurrent disease in humans, characterized by a febrile illness with transient loss of visual acuity, was recognized.^{48,49} In the second documented South African outbreak, during 1975, seven human deaths resulted and were associated with either acute hemorrhagic fever or encephalitis.⁵⁰⁻⁵²

Epizootics occur periodically following heavy rains that flood natural depressions. This allows hatching of the primary vector and reservoir (*Aedes* spp) mosquitoes that have become infected by trans-ovarial transmission of RVF virus.⁵³⁻⁵⁷ Additional RVF outbreaks have occurred in South Africa, one on the inland plateau that affected sheep farming areas following heavy rains (1974-1976) and a small outbreak in coastal northern KwaZulu-Natal in 1981.

People have rarely been affected during these RVF epizootics, and human involvement is usually restricted to veterinary workers, or those involved in the livestock industry, where exposure to viremic tissues of freshly slaughtered or aborted infectious animals, is a risk factor. This assertion is supported by experience from the

latest outbreak in South Africa, and the only one to affect the KNP. During late January 1999, abortions occurred in pregnant buffaloes, with deaths in a few other species, including giraffe. Three veterinary officials involved in conducting autopsies on these animals also developed a mild febrile disease after becoming infected with the virus. However, there is believed to be no risk to visitors to national parks (R Swanepoel, personal communication, Feb. 1999) in South Africa, as the mosquito vector is zoophilic, and seldom ventures indoors, and visitors rarely have direct contact with wild animals.

Ecologically there is a very slight likelihood of outbreaks of Chikungunya and Rift Valley fever in the KNP, but no risk exists for either dengue or yellow fever. Yellow fever vaccination is therefore not indicated if South Africa is the only African destination. If an outbreak of either Chikungunya or RVF occurs in the KNP, then measures to prevent mosquito bites would be a sensible precaution. Visitors who remain within their vehicles, as prescribed by the KNP, are believed to be at almost no risk, as they would not be exposed to preferred vector habitats, or to fresh carcasses, or aborted wildlife fetuses.

Tick-Borne Diseases

Hard ticks (Ixodidae) are vectors of tick bite fever (*Rickettsia conorii* and *Rickettsia africae*), Q-fever (*Coxiella burnetii*) and Congo-Crimean hemorrhagic fever in southern Africa. Large numbers of birds and wild mammals have been examined for infestation with ixodid ticks in the KNP. Every warthog (*Phacochoerus aethiopicus*), Burchell's zebra (*Equus burchelli*), impala (*Aepyceros melampus*), and kudu (*Tragelaphus strepsiceros*) examined was found to be infested with *Amblyomma hebraeum*, particularly the immature tick stages.⁵⁸ Larger host mammal species carried a greater burden of adult ticks, in particular eland, buffalo, giraffe, and rhinoceros.

Other ixodid tick species, including *Boophilus decoloratus*, have also been found to commonly infest large wild ruminant mammals in KNP, and *Haemaphysalis* spp and *Rhipicephalus* spp commonly infest wild carnivores, whereas *Hyalomma truncatum* predominantly infests scrub hares (*Lepus saxatilis*).⁵⁹⁻⁶³ Investigations into the seasonal abundance of ticks on wild ruminants found a distinct increase during drought years.⁶⁴⁻⁶⁶ Collection of free-living ticks, by means of drag-sampling with flannel strips, resulted in the collection of 14 tick species, with *Amblyomma hebraeum* and *Boophilus decoloratus*, predominating.⁶⁷

African tick bite fever. African tick bite fever, is a febrile disease characterized by classical eschars, with or without skin rash, and occasionally complicated by severe multisystem disease, particularly if diagnosis and effective therapy is delayed.^{68,69} The two forms seen are Mediterranean spotted fever which is caused by *Rickettsia conorii*

and transmitted by *Rhipicephalus* sp, and classical African tick bite fever, caused by *Rickettsia africae* and transmitted by *Amblyomma hebraeum*. A growing number of reports of tick bite fever being diagnosed in travelers returning from visits to South African nature reserves have appeared.⁷⁰⁻⁷⁴ However, the true incidence rate is not known because most travelers have returned home before the completion of the incubation period. Incidence amongst staff members can be used as a proxy measure of risk to travelers. Medical records indicate that this varies from 1 to 4 cases per month, with a peak in the summer months. This value is probably an extreme upper limit, because of increased opportunities for exposure of staff to ticks. However, it may indicate that tick bite fever is a more important disease entity for travelers than presently recognized. People walking in high grass should wear clothes that cover their bodies, particularly their legs, and apply effective tick repellents on exposed skin, or impregnate their clothes with permethrin.

Vervet monkeys may also be infected with *R. conorii* in the KNP.³⁶ Monkeys and other small mammals do occasionally venture into camps and carry ticks with them. However, the risk of these ticks transferring to humans within a residential camp is believed to be remote.

Crimean-Congo hemorrhagic fever. Crimean-Congo hemorrhagic fever (CCHF), caused by infection with a nairovirus, infrequently causes human disease in South Africa.³⁵ This virus occurs in eastern Europe, Asia, and Africa in a distribution that corresponds with the main vectors of the virus: ticks of the genus *Hyalomma*. These ticks are known as pied-leg ticks in South Africa with their alternating rufous-brown and white leg banding. Ticks are infected when their immature stages feed on viremic small mammals, like hares. Adult ticks, infected as larvae or nymphs, subsequently feed on large animals, like cattle, horses, zebra, and antelope, and transmit infection. There is no evidence of domestic livestock or wildlife becoming sick from CCHF infection, but they are briefly viremic.⁷⁵

During this period their tissues are infectious to people, particularly during butchering or postmortem. Fortunately, the virus does not survive well in tissues after death and so does not constitute a hazard in meat processed according to health regulations.⁷⁶

People become infected either by contact with freshly slaughtered livestock (cattle, sheep, or ostriches), contact with infected fluids from patients, from tick bites, or from squashing ticks.^{35,77,78} Despite documented wide distribution of antibody in the sera of cattle and wild vertebrates, there have been no human CCHF cases recorded as a result of contact with infectious tissues from freshly slaughtered wildlife in South Africa.^{77,79}

The majority of ticks implicated are *H. truncatum*, although specimens of *H. marginatum rufipes* and *H. m.*

turanicum have also been incriminated.^{80,81} These ticks only have one life cycle per annum, and adults are more active in summer and immature stages peak in autumn and winter.⁸² Fortunately the three species of *Hyalomma* ticks that occur in South Africa rarely feed on humans, although ticks may bite people if there is prolonged contact, e.g., when lying on a tick on the ground.^{76,83}

There is no documented occupational risk, or risk to visitors, in the KNP. Walking trail participants may choose to plan their trips for seasons when ticks are least active. Accommodation on walking trails does not include sleeping on the ground, but hikers should be cautioned to treat clothing with insecticides, e.g., synthetic pyrethroids, especially socks and pants, and apply repellent to exposed skin.

Other Zoonotic Diseases

Anthrax

Anthrax is a potentially lethal disease in humans, which may follow inhalation, or ingestion, of the spores of the causative bacterium, *Bacillus anthracis*.⁸⁴ Most often however, human disease follows direct contact with anthrax-infected animals, or anthrax-contaminated animal products, occasionally with dramatic results as corroborated by an outbreak of more than 10,000 cases of cutaneous anthrax in Zimbabwe between 1979 and 1985.⁸⁵ Vegetative bacteria have a poor survival outside a mammal host, of less than 24 hours.⁸⁶ This contrasts with the phenomenally hardy spores that can survive for decades in an inhospitable environment.⁸⁷ The spores germinate in an environment rich in glucose, amino acids, and nucleosides, such as mammalian tissues, and the vegetative bacilli form spores if there is a deficiency of these nutrients, most notably when infected body fluids are exposed to air.

Evidence from genotype grouping indicates that anthrax is an indigenous disease in the KNP, and that southern Africa may have been the geographical origin of *B. anthracis*.⁸⁸ Anthrax is an important wildlife disease and has caused serious losses in wild animal populations in the KNP. During epidemic outbreaks in 1959, 1960, 1970, 1990, 1991, 1993, and 1999, more than 5,000 wild animals succumbed.⁸⁹ The disease occurs endemically in Pafuri, in the far north of the KNP, and periodically in epidemic form in other areas.⁹⁰

A variety of animal species have been affected, with kudu antelope (*Tragelaphus strepsiceros*) being the focus of many epidemics. The density of susceptible kudu appears to be an important factor for maintaining the disease in the KNP. The contribution of blowflies as disseminators of the disease amongst kudus has been established.⁹¹ Carnivores, particularly lions (*Panthera leo*), have also been affected largely through feeding on the car-

cases of buffalo (*Syncerus caffer*) at water holes and troughs.⁹² Lions open carcasses providing the opportunity for sporulation of the anthrax bacilli, and dissemination of spores.⁹³ Vultures also play an effective disseminator role due to their scavenging habits and wide flight ranges.⁹⁴

The reassuring finding of rapidly declining numbers of anthrax spores with distance downwind from heavily infected anthrax carcass sites, makes the likelihood of infection by inhalation while in transit near a carcass site highly improbable.⁹⁵

Hides from carcasses infected with *B. anthracis* are regarded as being permanently infected and may contaminate articles with which they come into contact. Even final products made after the tanning or curing of raw hides are not necessarily free from spores and thus no products from anthrax-infected wildlife are harvested or sold in the KNP.⁹⁶ Interestingly, the infectivity of anthrax spores for humans is normally regarded as low.⁹⁷ During the anthrax epidemics in KNP wildlife, large teams of workmen were used to track down, sometimes cut up, and burn anthrax contaminated carcasses. They were definitely exposed, but only one contracted cutaneous anthrax after suffering a puncture wound from a bone fragment.⁹⁰ Further, no mishap took place during the field necropsies of more than 50 anthrax carcasses in the KNP (De Vos, in preparation). There is no risk to tourists unless they ignore KNP rules and leave their vehicle and handle an infected carcass.

Bovine Tuberculosis

Tuberculosis (TB) caused by *Mycobacterium bovis* is clinically indistinguishable from TB caused by *Mycobacterium tuberculosis*, and has been confirmed from a number of African countries.⁹⁸ Although the consumption of milk contaminated by *M. bovis* is regarded as the principal mode of transmission between animals and humans, agricultural workers may acquire the disease by inhaling coughed particles from infected cattle.^{99,100}

Bovine tuberculosis due to *M. bovis*, was diagnosed for the first time, in an emaciated African buffalo (*Syncerus caffer*), in the southern KNP in July 1990.¹⁰¹ Since that time bovine tuberculosis has been demonstrated commonly in African buffalo in the southern KNP, and in some buffalo herds the prevalence is as high as 70%.¹⁰²

The distribution and characteristics of pulmonary lesions in certain buffalo suggest that the initial route of infection is aerogenous and the cavernous state of these pulmonary lesions indicates that such animals were probably highly infective at the time of death.¹⁰² The survival time of *M. bovis* in the natural habitat of infected free ranging wildlife has been investigated, and it could only be isolated for a maximum period of 6 weeks from infected tissue specimens, and for 4 weeks from feces.¹⁰³

Spillover into other species, including cheetah (*Acinonyx jubatus*), lions (*Panthera leo*), and chacma baboons (*Papio ursinus*), has been documented, and this raised a concern about the potential of transmission of *M. bovis* to humans.¹⁰⁴

This led to an investigation amongst KNP employees considered at high risk of infection, including workers involved in slaughtering tuberculous buffalo.¹⁰⁵ Screening for active disease was performed by bacteriological investigation of sputum specimens using standard laboratory methodology, and study participants also underwent comparative intradermal skin testing to determine their level of infection. For comparative purposes, testing was also done in low risk groups, including workers involved in handling carcasses after butchering, and in a group of workers who were not considered to be at risk, including administrative workers. *M. bovis* was not isolated from any of the 206 employees tested, neither did differential skin testing show any degree of *M. bovis* infection risk, even among high risk occupations. Reasons for this low risk may include the performance of high-risk procedures, slaughtering and post mortems, in the open, with an abundance of sunlight and fresh air, and thus dilution of infectious aerosols.¹⁰⁶

As bovine tuberculosis was not found to be an occupational zoonosis in the Park, nor was aerosol transmission demonstrated as a mechanism for human infection in this study, there is not believed to be any meaningful risk to tourists.¹⁰⁵

Rabies

Rabies virus, lyssavirus 1, is generally transmitted by the bite of infected animals, and causes fatal encephalitis in humans and other warmblooded vertebrates. The disease is widely distributed in the world, and was diagnosed for the first time in Africa in 1893, in the Eastern Cape Province.¹⁰⁷ South Africa is endemic for rabies, being hyperendemic in yellow mongoose (*Cynictis penicillata*) on the central plateau of the country.¹⁰⁸ The maintenance host in the dry western areas is the bat-eared fox (*Otocyon megalotis*), and the black-backed jackal (*Canis mesomelas*) fills this niche in the northern parts of the country bordering Zimbabwe.¹⁰⁹ More than 90% of the 10–20 cases of human rabies that are recorded annually, occur in KwaZulu-Natal, where dog rabies has been intractable since re-introduction into this province in 1976.

Rabies has not been diagnosed in wildlife in the KNP despite active attempts at detection, and there is therefore no risk to visitors.¹¹⁰

Plague

In the recent past there has been a resurgence of epidemic plague globally, and this is particularly an African problem, where nearly 65% of the 3,000 annually reported

cases occur.¹¹¹ Rodents, in particular veld rats (*Aethomys chrysophilus*) in the KNP, are infested with *Xenopsylla brasiliensis*, the flea that maintains plague circulation in rodent populations in Africa, and that is capable of transmitting the infection to humans.¹¹²

Serological surveys for antibodies against the plague bacillus, *Yersinia pestis*, are useful tools for detecting plague foci, and monitoring the efficacy of control measures.^{113,114} Recently revived serological plague surveillance in dogs and rodents, with concurrent flea indices determination in Mpumalanga Province, includes a rural district, Shongwe, bordering the KNP, and no evidence of plague infection has been found.¹¹⁵ There have been no reported human plague cases in South Africa since 1982, when plague was diagnosed in the coastal regions of the Eastern Cape.¹¹⁶ The most eastern extent of plague detected in Mpumalanga is more than 200 km west of the KNP boundary.¹¹⁷

Sleeping Sickness

Tsetse fly (*Glossina morsitans morsitans*) occurred historically in the KNP until the large-scale Rinderpest outbreak during 1896 decimated its animal hosts. The Mozambican civil war caused suspension of collaborative fly control in southern Africa and currently no information exists on the southward movement of flies in Mozambique. Tsetse flies recolonize former habitats at a rate of 5–10 km per year.¹¹⁸ Therefore, an early warning surveillance system was established in the KNP in 1982 to monitor the incursion of tsetse flies into the Limpopo/Levuvhu drainage system. Twelve modified Harris traps were erected at strategic locations along the river courses to trap flies. In addition, thick blood smears are now taken in the far north of the KNP in buffaloes and other mammals, and examined for the presence of trypanosomes on an annual basis during the rainy summer period (February and March). Although numerous blood-sucking flies of the genera *Haematopota*, *Tabanus*, and *Stomoxys* have been collected, no *Glossina* spp have been collected, and no positive serology detected. There is therefore no risk of Nagana, or sleeping sickness in the KNP.

African Horse Sickness

African horse sickness (AHS) is common in zebra foals from the KNP and six serotypes have been described.¹¹⁹ However, disease, subclinical and clinical, as a result of infection with AHS has only been described once in humans. Four laboratory workers from the same vaccine-packing facility developed, at different times over an 8-year period, an illness characterized by encephalitis (in 3 workers) and uveochorioretinitis (in 4) after aerosol exposure, as a result of accidental breakage of freeze-dried vaccine bottles.^{120,121} There is therefore no risk to KNP visitors.

Encephalomyocarditis Virus

Encephalomyocarditis virus infection has been associated with cardiac failure and fatality in African elephants (*Loxodonta africana*) since 1987 in the KNP. In a single outbreak that began in December 1993, peaked in January 1994, and terminated by November 1994, 64 elephants died.¹²² Studies conducted on rodents in the KNP demonstrated a remarkable temporal correlation between the outbreak in elephants and a rodent population explosion. There was a concurrent increase in the prevalence of encephalomyocarditis virus antibodies in rodents.

All staff members who had exposure to infected rodents or elephants were carefully screened for serological activity and symptoms. Only 2 staff members who were involved in sampling over 900 mice and performing post mortems on 20 elephants, had serological evidence of infection, and only 1 of these suffered a mild headache for a few days. There thus appears to be no risk to visitors to the KNP; indeed encephalomyocarditis' status as a zoonotic disease remains to be confirmed.

Foot-and-Mouth Disease

Foot-and-mouth disease (FMD) is the most economically important viral disease of livestock. The KNP is a foot-and-mouth endemic area, and a routine surveillance program of cloven-hoofed animals is operated to monitor FMD activity.¹²³ South African Territory (SAT) types of FMD virus are maintained in buffalo (*Syncerus caffer*) in the KNP, and there are regular outbreaks of FMD in impala (*Aepyceros melampus*), probably as a result of transmission from carrier buffalo.^{124,125} In impalas FMD virus may circulate for a considerable period of time.

Measures to prevent the escape of the virus into domestic animal populations adjacent to the KNP have been implemented, in particular the erection of a double fenced off corridor. Experimental work has shown that the risk of transmission from KNP buffalo carrying SAT types 1, 2, and 3 viruses separated by a fence from FMD susceptible cattle, but sharing common drinking troughs and hay racks for a 15-month period, was low.¹²⁶

Reports of human infection with FMD are rare, and usually result from skin inoculation, or drinking contaminated milk.¹²⁷ No human cases have ever been diagnosed in the KNP.¹²⁸

Food and Waterborne Diseases

The meat from many wildlife species is pleasantly palatable and popular amongst local people and tourists.^{129–131} Fortunately, very few pathogens are sufficiently robust to survive the significant changes in temperature, pH, moisture content, and osmolality which

occur post mortem, or which are associated with preservation processes such as pickling, smoking, or drying.¹³² To ensure meat safety, a number of practices are observed in the KNP. Carcasses are eviscerated in the field immediately after slaughter, work is restricted to the later afternoons to avoid hot conditions, and carcasses are transported with their skin to avoid contamination and bacterial decomposition. In the field, workers wear overalls, industrial gloves, and gum boots. The general hygiene at the meat processing plant is maintained in terms of all conditions stipulated in the Abattoir Hygiene Act (Act 121 of 1992). This includes thorough cleaning and sterilization of equipment, regular maintenance of all cooling facilities, and because all workers involved in meat processing, including the slaughter team, are considered food-handlers, they are medically examined and tested for diseases transmissible by food handling.

The Division of Veterinary Services based in the KNP conducts formal meat inspections in terms of the Abattoir Hygiene Act. This includes inspection for parasitic cysts, tubercles, and abscesses.¹³³ Bacterial counts are routinely performed on biltong, and all canned meat is stored in brine with a pH tested below 5.5. Random sampling and culturing is done.

Since 1983, the KNP has employed a full time technician to perform testing of samples, collected from food-handlers during medical examinations, that are conducted twice annually. A person who tests positive is notified and relieved from duty immediately. They are referred to the KNP doctor for treatment and only allowed to return to their duty after they have laboratory proof of successful treatment. In addition supervisors are obliged to refer any employee with a chronic cough, gastrointestinal tract symptoms, or a skin condition, to the doctor for treatment. For the 10-year period, 1990–1999 inclusive, of 7,117 tests performed, 1 was positive for *Shigella* sp, 3 were positive for *Salmonella typhi*, and 28 were positive with *Salmonella* sp and referred for curative therapy. During the same period, 9 food-handlers were diagnosed with pulmonary tuberculosis (TB).

Only pasteurized milk is sold or used in restaurants. All fresh produce (bread, meat, fruit, and vegetables) sold inside the Park is prepacked. Camp restaurants are subjected to random quality-assurance inspections. Each region of the Park has its own Health and Safety Committee that carries out regular inspections in all camps. Monthly meetings are held to address findings. The Training Section conducts training programs throughout the year, including courses on occupational health and safety, and occupational hygiene (including personal hygiene). Each supervisor is expected to send at least 5% of his/her staff on these courses each year. The Departments of Health of Mpumalanga and Northern

Provinces also perform inspections of facilities from time to time.

Brucellosis

Brucella abortus has been isolated from free-living wild African buffalo (*Syncerus caffer*), and hippo, in the KNP.^{134,135} The serological prevalence is about 20% in hippo and 17% in buffalo.¹³⁶ No known cases have ever been detected amongst staff members, including veterinary personnel. The latter are at greatest risk when performing rectal examinations for pregnancy diagnosis.

Trichinosis

In Africa trichinosis is essentially a disease of wild carnivores.¹³⁷ The absence of trichinosis in true herbivores has been confirmed in the KNP, which is of importance given the increasing utilization of game meat as food. People would thus have to ingest undercooked infested meat from a carnivore to become infected. No human cases have ever been diagnosed in the KNP.

Echinococcosis

Echinococcosis has been diagnosed in the following wild species in the KNP: lion (*Panthera leo*), spotted hyena (*Crocuta crocuta*), Cape hunting dog (*Lycan pictus*), Burchell's zebra (*Equus burchelli antiquorum*), buffalo (*Syncerus caffer*), hippopotamus (*Hippopotamus amphibius*), and impala (*Aepyceros melampus*).¹³⁸ Infestation rates in herbivores vary from 60% in zebra to less than 1% in impala. Screening of high-risk staff has not yielded any positive serology for echinococcosis (Braack LEO, unpublished data). There is no risk to tourists.

Salmonella

Salmonella sp infection is common in many wildlife species, in particular crocodiles (*Crocodylidae* spp).¹³⁹ Testing for *Salmonella* sp infection is routinely conducted on carcasses in the abattoir. In addition, crocodiles should not be handled, and crocodile meat is not processed, or sold in the KNP.

Water Quality

Stream water is provided for domestic use in residential camps and bushcamps on walking trails are supplied by bore holes. All raw water is purified using a process including addition of flocculents, sedimentation, filtration, and disinfection with chlorine. Free residual chlorine levels are determined on a daily basis and full biochemical and microbiological monitoring is conducted on a weekly basis. Review of registers indicates excellent water quality post-treatment. In addition, investigation of river water and hippopotami (*Hippopotamus amphibius*) in the KNP revealed a lack of heavy metal pollution.¹⁴⁰ For the more wary trav-

eler, all shops, restaurants, and cafeterias, sell sealed bottled water.

Other Infectious Conditions

Review of available general practice records, an interview with the senior general practitioner at Skukuza, and perusal of statutory notifications, indicate that there have been no cases of cholera, meningococcal meningitis or other formidable contagious conditions, diagnosed in the KNP for the 10-year period, 1990–1999 inclusive.

Tuberculosis

The 1% prevalence of active disease with *Mycobacterium tuberculosis*, detected among staff members during the *M. bovis* survey, and routinely detected in food-handlers through the screening program, is typical of community trends in South Africa.^{105,141} The active detection program amongst food-handlers, and referral of all employees with chronic cough of longer than 3 weeks for medical attention by supervisors and a nursing sister located at Skukuza, limits the risk to visitors.

Bilharzia

The eastern half of South Africa is endemic for *Schistosoma haematobium*, and within this area there are hyperendemic foci of *Schistosoma mansoni* infection.^{142,143} The intermediate snail hosts exist in KNP waterways that originate in areas where infected children may contaminate them. Occasionally, Mozambican refugees could also play this role.¹⁴⁴

In southern Africa, *Schistosoma mattheei* infects cattle, horses, sheep, zebra, baboon, and antelope.¹⁴⁵ Although *S. mattheei* can infect people, it is almost always together with *S. haematobium*, or *S. mansoni*.¹⁴⁶ The female of *S. mattheei* may in fact need a adult male *S. haematobium*, or *S. mansoni* to transport it to its egg laying site.¹⁴⁷

Thus, all streams and other natural water bodies within the KNP should be treated as infested with schistosomes. Park authorities do not permit swimming, or fishing, in streams, rivers, or dams, and have provided safe chlorinated swimming pools in specific restcamps. Tourists should thus be at no risk of contracting bilharzia in the KNP, as crocodiles and hippos provide a persuasive disincentive to swimming.

Trauma

The burden of trauma as a cause of death in the age group 15–60 years is greater in subSaharan Africa than in any other region of the world.¹⁴⁸ South Africa has a particularly poor record, with the national per capita violence mortality rate being one of the highest in the

world, and six times greater than the US.¹⁴⁹ Of equal concern is the high traffic death rate per unit of distance traveled, that is only surpassed by Kenya, Morocco, and Korea.¹⁵⁰ As traumatic events are not random occurrences, they are usually preventable.¹⁵¹

Wild Mammal Attacks

Attacks by wild mammals can be exceptionally newsworthy, whether these involve workers on nature reserves, or tourists. A review of commercial press records of all reported deaths and injuries to workers on wildlife reserves in South Africa from January 1988 to December 1997 inclusive, found that 6 workers had been killed and 14 injured in encounters with wild mammals.¹⁵² Twelve of these incidents, including 4 deaths (2 soldiers, a gatekeeper, and a trail guide), occurred in the KNP. Nine incidents involved leopards or lions and elephants; buffalo and giraffe accounted for the remaining episodes. All attacks, with the exception of those involving leopards, occurred on foot, outside the safety of fenced camps.

Little was known about tourist risk of injury and death by wild mammals in South Africa's nature reserves before a recent 10-year retrospective review of commercial press records was conducted.¹⁵³ Seven tourists were killed by wild mammals in South Africa between January 1988 and December 1997 inclusive. None of these tourist fatalities occurred in the KNP, despite this being the most frequently visited nature reserve. Three of the 4 deaths ascribed to lions in this study resulted from tourists carelessly approaching prides on foot, in lion reserves, and a private reserve provided inadequate protection to a guest who was mauled. Tourist ignorance of animal behavior, and flagrant disregard of rules, contributed to the 2 fatalities involving hippopotami. A bull elephant with severe discomfort from a dental problem was responsible for the final death.

During the same period there were 14 nonfatal attacks on tourists, including 5 by hippo, 3 by buffalo, 2 by rhino, and 1 each by a lion, leopard, zebra, and musth elephant. Four of these attacks occurred in the KNP, with hippo, leopard, rhino, and elephant, each involved in a single attack. Only the incident with the elephant, occurred while the visitor was in their motor vehicle. This occurred when an amorous bull elephant, disturbed while soliciting an elephant cow's attention, charged a visitor's vehicle, and dragged it several meters after capsizing it. The injuries that resulted could have been prevented if the visitor had interpreted the warning display that precedes an elephant's charge, including the tell-tale temporal secretion characterizing musth, ground-kicking, swaying motion, extended ears, and raised trunk with trumpeting. The action of a visitor who left his motor vehicle, in direct defiance of KNP rules, to examine a supposedly injured hippopotamus, was rewarded with a trampling, and minor injuries. The clawing of a camper at the Berg-en-

Dal camping site in the southern KNP by a leopard, was unusual, as leopards seldom attack humans unless they are sick, or injured, or in defense. Leopards however, have the propensity to enter open windows, or open tent flaps, as in this case. Windows should therefore be closed and tents securely zipped.

The attack by four black rhino on a walking trail participant on the Napi walking trail is informative, as it emphasizes the aggression of these large herbivores, particularly if surprised. It is therefore essential that walking trail participants not move too quietly and insist on a well-qualified trail guide. The KNP only uses guides who are highly trained to standards set by the Field Guides Association of South Africa with SKS/DA (special skills and dangerous animal training). This is clearly worthwhile as demonstrated by the attack on a walking trail group by two angry lionesses. The experienced ranger leading the expedition shot one lioness and the other fled, without human injury.

An important reason that may explain the low number of these incidents are the measures taken by KNP authorities to prevent direct contact between wild mammals and tourists. This is largely through providing secure residential camps that are completely fenced, including electrification. Visitors are not permitted to leave their vehicles except at designated secure areas, and make themselves liable for prosecution if they contravene. The KNP employs topgraded guides to lead hiking trails, and there are rangers available at camps to enlighten visitors on important animal behavior that may herald aggression.

Motor Vehicle Accidents

Low speed limits of 50 km/h are enforced on good quality tarred roads, and the limit on dust roads is 40 km/h. A strict curfew is maintained, with no private vehicles allowed outside camps during the hours of darkness and heavy penalties are imposed to ensure the safety of travelers and wildlife.¹⁵⁴ These precautions are of particular relevance when considering hippos that exhibit nocturnal grazing behavior.

During the past ten years (1990–1999 inclusive) there have been no traffic-related human deaths in the KNP. There have been a total of 1,014 minor accidents within the KNP, adjoining private nature reserves, and on feeder public roads, but with only 45 injuries. Many of these accidents either occurred in parking areas, or resulted from sudden stops without the driver behind noticing. During this period, only 15 drivers were charged with reckless driving.

Natural Disasters

In the past quarter of a century there have been 6 fires that have affected residential camps. One in Lower

Sabie was caused by an electrical fault, and the other 5 were the result of lightning, including the razing of the Pretoriuskop Restaurant. There were no human injuries or deaths. The only wind damage in the past quarter of a century in the KNP has been to windmills.

Twice in the past 25 years there have been floods in the KNP associated with tropical cyclones, in 1976, and 2000. Fortunately, these only resulted in damage to infrastructure, particularly roads and bridges, and there were no human casualties, or injuries.

Snake-Bite

There are a number of venomous snakes in the KNP, including boomslang (*Dispholidus typus typus*), Mozambique spitting cobra (*Naya mossambica*), Egyptian cobra (*Naya haje annulifera*), and black mamba (*Dendroaspis polylepis*). In general, they avoid human contact through ferocious displays, flight, or feigning death.¹⁵⁵ A particular problem however, is the puff adder (*Bitis arietans arietans*), that relies on immobility and cryptic camouflage to avoid detection, basks in the sun on forest footpaths, and thus may pose a risk to walking trail participants. However, well-trained trail guides lead groups of people in single-file, and they carefully search for snakes. It is remarkable that only 1 trail-participant has been bitten by a snake in the past 20 years, and ironically this was by an African rock python, which is a nonvenomous snake.

Conclusions

Studies of risk perception assist us in understanding public response to health-related events in African nature reserves, and the attendant, often disproportionate, alarm that these occurrences, or even theoretical threats, invoke. Research has shown that particular categories of health events more often provoke alarm in members of the public. These include exposures that are unusual, result in deaths that are vivid, occur in environments that are foreign to the individual, and where the effects of exposure may be delayed.¹⁵⁶

This review of all available relevant data-sources indicates that tourists visiting the KNP are extremely unlikely to experience any serious health-related condition as a direct result of their visit. To minimize this risk further a number of sensible precautions should be taken:

- Careful use of personal protection measures against vector mosquitoes of malaria, from dusk to dawn, throughout the year.
- Correct use of effective malaria chemoprophylaxis, at least during the higher risk malaria months, October to May.
- Topical application of effective tick repellents, and treatment of socks and trousers with a synthetic pyrethroid to prevent tick bites, particularly if walking on a trail.
- Remaining in a secure motor vehicle, or adequately fenced precincts while in the vicinity of large mammals.¹⁵³
- Rigidly observing nature reserve instructions.
- Never approaching animals that appear ill, malnourished, displaying aggressive behavior traits, or females with young.¹⁵⁴
- Checking the credentials of game rangers before embarking on a walking trail.¹⁵²
- Ensuring adequate travel insurance, including aeromedical evacuation contingency plans.¹⁵⁷

From this review, it is clear that KNP management have taken all reasonable measures to reduce the incidence of preventable injury, or illness, in visitors. Travelers have the prime responsibility to educate themselves about risks at their destination, how to assess these risks, and to take measures to minimize their personal exposure to risky situations.

References

1. South African Tourism Board. A survey of South Africa's international tourism market. Pretoria: South African Tourism Board, 1999.
2. Leggat PA, Klein M. Personal safety advice for travelers abroad. *J Travel Med* 2001; 8:46–51.
3. de Semir V. What is newsworthy? *Lancet* 1996; 347: 1163–1166.
4. Steffen R. Travel medicine—prevention based on epidemiological data. *Trans R Soc Trop Med Hyg* 1991; 85:156–162.
5. Waner S. Health risks of travelers in South Africa. *J Travel Med* 1999; 6:199–203.
6. Durrheim DN, Harris BN, Speare R, Billingham K. Infection control nurses—sentinels for outbreak surveillance. *Bull World Health Organ* 2001; 79:22–27.
7. Koontz H, Wehrich H. Management. 9th Ed. Singapore: McGraw Hill, 1989:35–36.
8. Kondrachine AV, Trigg P. Malaria: hope for the future. *World Health* 1995; 2:26–27.
9. Nchinda TC. Malaria: a reemerging disease in Africa. *Emerg Infect Dis* 1998; 4:398–403.
10. Marsh K. Malaria disaster in Africa. *Lancet* 1998; 352:924.
11. Durrheim DN, Ogunbanjo GA, Blumberg L. Managing re-emergent malaria in South Africa. *S Afr Fam Pract* 1999; 21:19–24.
12. Sharp BL, le Sueur D. Malaria in South Africa—the past, the present and selected implications for the future. *S Afr Med J* 1996; 86:83–89.
13. Durrheim DN, la Grange JJP, Govere J, Mngomezulu NM. Accuracy of a rapid immunochromatographic card test for *Plasmodium falciparum* in a malaria control programme in South Africa. *Trans R Soc Trop Med Hyg* 1998; 92:32–33.

14. Speare R, Govere J, Durrheim DN. Malaria control in South Africa: symposium in the wilderness. *J Travel Med* 1999; 6:149–150.
15. Booman M, Durrheim DN, La Grange JJP, et al. Using a geographical information system to plan a malaria control programme in South Africa. *Bull World Health Organ* 2000; 78:1438–1444.
16. Department of Health. Guidelines for the prophylaxis of malaria. Pretoria: Department of Health, 1996.
17. Dollow S. Risk-benefit ratios must be taken into account. *BMJ* 1996; 313:1553.
18. Durrheim DN, Braack LEO, Waner S, Gammon S. Risk of malaria in visitors to the Kruger National Park, South Africa. *J Travel Med* 1998; 5:173–177.
19. Uyirwoth GC. Malaria notifications in South Africa. *Epidemiol Comments* 1995; 22:165–174.
20. Lobel HO, Phillips-Howard PA, Brandling-Bennett AD, et al. Malaria incidence and prevention among European and North American travellers to Kenya. *Bull World Health Organ* 1990; 88:209–215.
21. Lonergan G. Antimalarials for visitors to Kruger National Park? *J Travel Med* 1999; 6:210.
22. Durrheim DN, Gammon S, Waner S, Braack LEO. Antimalarial prophylaxis—use and adverse events in visitors to the Kruger National Park. *S Afr Med J* 1999; 89:170–175.
23. World Health Organization. Malaria prophylaxis for travellers. *Wkly Epidemiol Rec* 1993; 68:377–383.
24. Behrens RH. Chloroquine and proguanil prophylaxis in travellers to Kenya. *Lancet* 1992; 339:63.
25. Fradin MS. Mosquitoes and mosquito repellents: a clinician's guide. *Ann Intern Med* 1998; 128:931–940.
26. Schoepke A, Steffen R, Gratz N. Effectiveness of personal protection measures against mosquito bites for malaria prophylaxis in travellers. *J Travel Med* 1998; 5:188–192.
27. Durrheim DN, Leggat PA. Prophylaxis against malaria. Preventing mosquito bites is also effective. *BMJ* 1999; 318:1139.
28. Waner S, Durrheim D, Braack LEO, Gammon S. Malaria protection measures used by in-flight travelers to South African game parks. *J Travel Med* 1999; 6:254–257.
29. Coetzee M, Braack LEO. *Anopheles arabiensis* in the Kruger National Park. Proceedings of the 8th Entomological Congress of the Entomological Society of Southern Africa, Bloemfontein, 1–4 July 1991. Johannesburg: Entomological Society of Southern Africa, 1991:19.
30. Coetzee M, Hunt RH, Braack LEO, Davidson D. Distribution of mosquitoes belonging to the *Anopheles gambiae* complex, including malaria vectors, south of the latitude 15°S. *S Afr J Sci* 1993; 89:227–231.
31. Braack LEO, Coetzee M, Hunt RH, et al. Biting pattern and host-seeking behavior of *Anopheles arabiensis* (Diptera: Culicidae) in northeastern South Africa. *J Med Entomol* 1994; 31:333–339.
32. Govere J, Braack LEO, Durrheim DN, et al. Effects on the biting pattern of *Anopheles arabiensis* mosquitoes in the Kruger National Park, South Africa after treating human ankles and feet with a mosquito repellent. In press.
33. National Parks Board, Pretoria, South Africa. <http://www.parks-sa.co.za/malariainfopage>.
34. National Parks Board, Pretoria, South Africa. <http://www.parks-sa.co.za/knp/scientificservices/FAQS>.
35. Swanepoel R. Viral haemorrhagic fevers in South Africa: history and national strategy. *S Afr J Sci* 1987; 83:80–88.
36. Kaschula VR, Van Dellen AF, de Vos V. Some infectious diseases of wild vervet monkeys (*Cercopithecus aethiops pygerythrus*) in South Africa. *J S Afr Vet Assoc* 1978; 49:223–227.
37. Kemp A, Jupp PG. Potential for dengue in South Africa: mosquito ecology with particular reference to *A. aegypti*. *J Am Mosq Control Assoc* 1991; 7:574–583.
38. McIntosh BM, Jupp PG, Dos Santos I. Rural epidemic of chikungunya in South Africa with involvement of *Aedes* (*Diceromyia*) *fuscifer* (Edwards) and baboons. *S Afr J Sci* 1977; 73:267–269.
39. Fourie ED, Morrison JGL. Rheumatoid arthritic syndrome after chikungunya fever. *S Afr Med J* 1979; 56:130–132.
40. Morrison JGL. Chikungunya fever. *Int J Dermatol* 1979; 18:628–629.
41. Jupp PG. *Aedes* (*Diceromyia*) *fuscifer* (Edwards) and *Aedes* (*Diceromyia*) *cordellieri* Huang in southern Africa: distribution and morphological differentiation. *J Am Mosq Control Assoc* 1998; 14:273–276.
42. Jupp PG, McIntosh BM. *Aedes fuscifer* and other mosquitoes as vectors of chikungunya virus at Mica, northeastern Transvaal, South Africa. *J Am Mosq Control Assoc* 1990; 6:415–420.
43. Jupp PG, McIntosh BM, Dos Santos I, DeMoor P. Laboratory vector studies on six mosquito and one tick species with chikungunya virus. *Trans R Soc Trop Med Hyg* 1981; 75:15–19.
44. Blackburn NK, Rawat R. Dengue fever imported from India. *S Afr Med J* 1987; 71:386–387.
45. Jupp PG, Kemp A. The potential for dengue in South Africa: vector competence tests with dengue 1 and 2 viruses and 6 mosquito species. *Trans R Soc Trop Med Hyg* 1993; 87:639–643.
46. Jupp PG, Kemp A. What is the potential for future outbreaks of chikungunya, dengue and yellow fever in southern Africa. *S Afr Med J* 1996; 86:35–37.
47. Alexander RA. Rift Valley fever in the Union. *J S Afr Vet Med Assoc* 1951; 22:105–109.
48. Gear JHS. Rift Valley fever in South Africa. *S Afr Med J* 1951; 25:620.
49. Freed I. Rift Valley fever in man complicated by retinal changes and loss of vision. *S Afr Med J* 1951; 25:930–932.
50. Van Velden DJJ, Meyer JD, Olivier J, et al. Rift Valley fever affecting humans in South Africa. *S Afr Med J* 1977; 51:867–871.
51. Gear JHS. Haemorrhagic fevers of Africa. An account of two recent outbreaks. *J S Afr Vet Assoc* 1977; 48:5–8.
52. McIntosh BM, Russel D, dos Santos I, Gear JHS. Rift Valley fever in humans in South Africa. *S Afr Med J* 1980; 58:803–806.
53. World Health Organization. An outbreak of Rift Valley Fever, Eastern Africa, 1997–1998. *Wkly Epidemiol Rec* 1998; 73:105–109.
54. McIntosh BM, Jupp PG, Anderson D, Dickinson DB. Rift Valley fever. 2. Attempts to transmit virus with seven species of mosquito. *J S Afr Vet Med Assoc* 1973; 44:57–60.

55. Jupp PG, Cornel AJ. Vector competence tests with Rift Valley fever virus and five South African species of mosquito. *J Am Mosq Control Assoc* 1988; 4:4–8.
56. McIntosh BM, Jupp PG. Epidemiological aspects of Rift Valley fever in South Africa with reference to vectors. *Contrib Epidemiol Biostat* 1981; 3:92–99.
57. Linthicum KJ, Davies FG, Kairo A, Bailey CL. Rift Valley fever (family Bunyaviridae, genus Phlebovirus). Isolations from Diptera collected during the inter-epizootic period in Kenya. *J Hyg (Cambridge)* 1985; 95:197–209.
58. Horak IG, MacIvor KM, Petney TN, De Vos V. Some avian and mammalian hosts of *Amblyomma hebraeum* and *Amblyomma marmoreum* (Acari: Ixodidae). *Onderstepoort J Vet Res* 1987; 54:397–403.
59. Horak IG, De Vos V, De Klerk BD. Parasite of domestic and wild animals in South Africa. XVII. Arthropod parasites of Burchell's zebra, *Equus burchelli*, in the eastern Transvaal Lowveld. *Onderstepoort J Vet Res* 1984; 51:145–154.
60. Horak IG, Boomker J, Spickett AM, De Vos V. Parasites of domestic and wild animals in South Africa. XXX. Ectoparasites of kudus in the eastern Transvaal Lowveld and the eastern Cape Province. *Onderstepoort J Vet Res* 1992; 59:259–273.
61. Horak IG, Potgieter FT, Walker JB, et al. The ixodid tick burdens of various large ruminant species in South African nature reserves. *Onderstepoort J Vet Res* 1983; 50:221–228.
62. Horak IG, Guillardmod AJ, Moolman LC, de Vos V. Parasites of domestic and wild animals in South Africa. XXII. Ixodid ticks on domestic dogs and on wild carnivores. *Onderstepoort J Vet Res* 1987; 54:573–580.
63. Horak IG, Spickett AM, Braack LEO, et al. Parasites of domestic and wild animals in South Africa. XXXIII. Ixodid ticks on scrub hares in the north-eastern regions of northern and eastern Transvaal and of KwaZulu-Natal. *Onderstepoort J Vet Res* 1995; 62:123–131.
64. Spickett AM, Horak IG, Heyne H, Braack LEO. The effect of severe drought on the abundance of ticks on vegetation and on scrub hares in the Kruger National Park. *Koedoe* 1995; 38:59–64.
65. Horak IG, De Vos V, Braack LEO. Arthropod burdens of impalas in the Skukuza region during two droughts in the Kruger National Park. *Koedoe* 1995; 38:65–71.
66. Horak IG. The relationship between ticks, hosts and the environment in the Kruger National Park, South Africa. *Proceedings of the Second International Conference on Tick-borne Pathogens at the Host-vector Interface: A Global Perspective. Kruger National Park August 28–September 1, 1995. Pretoria: Onderstepoort Veterinary Institute* 2:413–426.
67. Spickett AM, Horak IG, Braack LEO, van Ark H. Drag-sampling of free-living ixodid ticks in the Kruger National Park. *Onderstepoort J Vet Res* 1991; 58:27–32.
68. Loubser MD, Davies VA, Meyers KE, Christianson AL. Severe illness caused by *Rickettsia conorii*. *Ann Trop Paediatr* 1993; 13:277–280.
69. Walker DH, Gear JH. Correlation of the distribution of *Rickettsia conorii*, microscopic lesions, and clinical features in South African tick bite fever. *Am J Trop Med Hyg* 1985; 34:361–371.
70. Puente S, Lago M, Subirats M, et al. Spotted fever attributable to *Rickettsia conorii*: ten cases imported from sub-Saharan Africa. *J Travel Med* 1995; 2:204–205.
71. Raeber P, Winteller S, Paget J. Fever in the returned traveller: remember rickettsial disease. *Lancet* 1994; 344:331.
72. Jensenius M, Hasle G, Henriksen AZ, et al. African tick-bite fever imported into Norway: presentation of 8 cases. *Scand J Infect Dis* 1999; 31:131–133.
73. Fournier PE, Beytout J, Raoult D. Tick-transmitted infections in Transvaal: consider *Rickettsia africae*. *Emerg Infect Dis* 1999; 5:178–181.
74. Brouqui P, Harle JR, Delmont J, et al. African tick-bite fever. An imported spotless rickettsiosis. *Arch Intern Med* 1997; 157:119–124.
75. Burt FJ, Swanepoel R, Braack LEO. Enzyme-linked immunosorbent assays for the detection of antibody to Crimean-Congo haemorrhagic fever virus in the sera of livestock and wild vertebrates. *Epidemiol Inf* 1993; 111:547–557.
76. Hoogstraal H. The epidemiology of tick-borne Crimean-Congo haemorrhagic fever in Asia, Europe, and Africa: a review article. *J Med Entomol* 1979; 15:307–417.
77. Swanepoel R, Shepherd AJ, Leman PA, et al. Epidemiologic and clinical features of Crimean-Congo hemorrhagic fever in southern Africa. *Am J Trop Med Hyg* 1987; 36:120–132.
78. Joubert JR, King JB, Rossouw DJ, Cooper R. A nosocomial outbreak of Crimean-Congo haemorrhagic fever at Tygerberg Hospital. *S Afr Med J* 1985; 68:722–728.
79. Shepherd AJ, Swanepoel R, Shepherd SP, et al. Antibody to Crimean Congo hemorrhagic fever virus in wild mammals from southern Africa. *Am J Trop Med Hyg* 1987; 36:133–142.
80. Cummings GS. Host preference in African ticks (Acari: Ixodida): a qualitative data set. *Bull Entomol Res* 1998; 88:379–406.
81. Swanepoel R, Struthers JK, Shepherd AJ, et al. Crimean-Congo hemorrhagic fever in South Africa. *Am J Trop Med Hyg* 1983; 32:1407–1415.
82. Horak IG. Parasites of domestic and wild animals in South Africa. XV. The seasonal prevalence of ectoparasites on impala and cattle in the Northern Transvaal. *Onderstepoort J Vet Res* 1982; 49:85–93.
83. Theiler G. The Ixodoidea parasites of vertebrates in Africa south of the Sahara. Report to the Director of Veterinary Sciences, Pretoria: Onderstepoort Veterinary Institute 1962, Project 9958.
84. Inglesby TV, Henderson DA, Bartlett JG, et al. Anthrax as a biological weapon. Medical and public health management. *JAMA* 1999; 281:1735–1745.
85. Myenye KS, Siziya S, Peterson D. Factors associated with human anthrax outbreak in the Chikupo and Ngandu villages of Murewa district in Mashonaland East Province, Zimbabwe. *Cent Afr J Med* 1996; 42:312–315.
86. Titball RW, Turnbull PC, Hutson RA. The monitoring and detection of *Bacillus anthracis*. *J Appl Bacteriol* 1991; 70:95–185.
87. Williams RP. *Bacillus anthracis* and other spore forming bacilli. In: Braude AI, Davis LE, Fiere J, eds. *Infectious disease and medical microbiology*. Philadelphia: WB Saunders, 1986:270–278.

88. Smith KL, De Vos V, Bryden HB, et al. Meso-scale ecology of anthrax in southern Africa: a pilot study of diversity and clustering. *J Appl Microbiol* 1999; 87:204–207.
89. De Vos V, Bryden HB. Anthrax in the Kruger National Park: temporal and spatial patterns of disease occurrence. *Salisbury Med Bull* 1996; 87:26–31.
90. Pienaar U de V. Epidemiology of anthrax in wild animals and the control of anthrax epizootics in the Kruger National Park, South Africa. *Fed Proc* 1967; 26:1496–1502.
91. Braack LEO, De Vos V. Feeding habits and flight range of blow-flies (*Chrysomyia* spp) in relation to anthrax transmission in the Kruger National Park, South Africa. *Onderstepoort J Vet Res* 1990; 57:141–142.
92. De Vos V. Anthrax. In: Coetzer JAW, Thomson GR, Tustin RC, eds. *Infectious diseases of livestock with special reference to southern Africa*. Cape Town: Oxford University Press, 1994:1262–1289.
93. McConnell EE, Tustin RC, de Vos V. Anthrax in an African buffalo (*Syncerus caffer*) in the Kruger National Park. *J S Afr Vet Assoc* 1972; 43:181–187.
94. Lindeque PM, Turnbull PC. Ecology and epidemiology of anthrax in the Etosha National Park, Namibia. *Onderstepoort J Vet Res* 1994; 61:71–83.
95. Turnbull PC, Lindeque PM, Le Roux J, et al. Airborne movement of anthrax spores from carcass sites in the Etosha National Park, Namibia. *J Appl Microbiol* 1998; 84:667–676.
96. Henning MW. Anthrax. *Animal diseases in South Africa*. Johannesburg: CNA, 1956.
97. Watson A, Keir D. Information on which to base assessments of risk from environments contaminated with anthrax spores. *Epidemiol Infect* 1994; 113:479–490.
98. Cosivi O, Grange JM, Daborn CJ, et al. Zoonotic Tuberculosis due to *Mycobacterium bovis* in developing countries. *Emerg Infect Dis* 1998; 4:59–70.
99. Collins CH, Grange JM. A review. The bovine tubercle bacillus. *J Appl Bacteriol* 1983; 55:13–29.
100. Moda G, Daborn CJ, Grange JM, Cosivi O. The zoonotic importance of *Mycobacterium bovis*. *Tuber Lung Dis* 1996; 77:103–108.
101. Bengis RG, Kriek NP, Keet DE, et al. An outbreak of bovine tuberculosis in a free-living African buffalo (*Syncerus caffer* Sparrman) population in the Kruger National Park: a preliminary report. *Onderstepoort J Vet Res* 1996; 63:15–18.
102. Keet DE, Kriek NP, Huchzermeyer H, Bengis RG. Advanced tuberculosis in an African buffalo (*Syncerus caffer* Sparrman). *J S Afr Vet Assoc* 1994; 65:79–83.
103. Tanner M, Michel AL. Investigation of the viability of *M. bovis* under different environmental conditions in the Kruger National Park. *Onderstepoort J Vet Res* 1999; 66:185–190.
104. Keet DE, Kriek NP, Penrith ML, et al. Tuberculosis in buffalo (*Syncerus caffer*) in the Kruger National Park: spread of the disease to other species. *Onderstepoort J Vet Res* 1996; 63:239–244.
105. Weyer K, Fourie PB, Durrheim D, et al. *Mycobacterium bovis* as a zoonosis in the Kruger National Park, South Africa. *Int J Tuberc Lung Dis* 1999; 3:1113–1119.
106. Weyer K, Fourie PB, Durrheim DN, et al. *Mycobacterium bovis* infection in workers in the Kruger National Park. Proceedings of the ARC-Onderstepoort OIE International Congress with WHO-Cosponsorship on Anthrax, Brucellosis, CBPP, Clostridial and Mycobacterial Diseases, Bergendal, Kruger National Park, South Africa, 9–15 August 1998. Onderstepoort: Onderstepoort Veterinary Institute, 1998:383–387.
107. Hutcheon D. Reports of the Colonial Veterinary Surgeon and Assistant Veterinary Surgeons for the year 1893. Cape of Good Hope: Department of Agriculture, 1894.
108. Swanepoel R, Barnard BJH, Meredith CD, et al. Rabies in southern Africa. *Onderstepoort J Vet Res* 1993; 60:325–346.
109. Barnard BJH. The role played by wildlife in the epizootiology of rabies in South Africa and South West Africa. *Onderstepoort J Vet Res* 1979; 46:155–163.
110. Meredith CD. Wildlife rabies: past and present in South Africa. *S Afr J Sci* 1982; 78:411–415.
111. Gubler DJ. Resurgent vector-borne diseases as a global health problem. *Emerg Infect Dis* 1998; 4:442–450.
112. Braack LEO, Horak IG, Jordaan LC, et al. The comparative host status of red veld rats (*Aethomys chrysophilus*) and bushveld gerbils (*Tatera leucogaster*) for epifaunal arthropods in the southern Kruger National Park, South Africa. *Onderstepoort J Vet Res* 1996; 63:149–158.
113. Shepherd AJ, Leman PA. Plague in South African rodents 1972–1981. *Trans R Soc Trop Med Hyg* 1983; 77:208–211.
114. Gordon HG, Isaacson M, Taylor P. Plague antibody in large African mammals. *Infect Immun* 1979; 26:767–769.
115. Govere J, Durrheim DN, Booman A. Plague surveillance in South Africa. *S Afr Med J* 1999; 89:570.
116. Shepherd AJ, Hummitzsch DE, Leman PA, Hartwig EK. Studies on plague in the Eastern Cape Province of South Africa. *Trans R Soc Trop Med Hyg* 1983; 77:800–808.
117. Hallett AF. A serological survey of the small mammals for plague in southern Africa. *S Afr Med J* 1970; 44:829–834.
118. Laid M. Tsetse: the future biological methods in integrated control. Ottawa: IDRC, 1977.
119. Lord CC, Woolhouse ME, Barnard BJ. Transmission and distribution of virus serotypes: African horse sickness in zebra. *Epidemiol Infect* 1997; 118:43–50.
120. Swanepoel R, Erasmus BJ, Williams R, Taylor MB. Encephalitis and chorioretinitis associated with neurotropic African horsesickness virus infection in laboratory workers. Part III. Virological and serological investigations. *S Afr Med J* 1992; 81:458–461.
121. van der Meyden CH, Erasmus BJ, Swanepoel R, Prozesky OW. Encephalitis and chorioretinitis associated with neurotropic African horsesickness virus infection in laboratory workers. Part I. Clinical and neurological observations. *S Afr Med J* 1992; 81:451–454.
122. Grobler DG, Raath JP, Braack LEO, et al. An outbreak of encephalomyocarditis-virus infection in free-ranging African elephants in the Kruger National Park. *Onderstepoort J Vet Res* 1995; 62:97–108.
123. Mpumalanga Directorate of Veterinary Services. Annual report. Nelspruit: Directorate of Veterinary Services, 1999.
124. Thomson GR, Vosloo W, Esterhuysen JJ, Bengis RG. Maintenance of foot and mouth disease viruses in buffalo (*Syncerus caffer* Sparrman, 1779) in southern Africa. *Rev Sci Tech* 1992; 11:1097–1107.

125. Keet DF, Hunter P, Bengis RG, et al. The 1992 foot-and-mouth disease epizootic in the Kruger National Park. *J S Afr Vet Assoc* 1996; 67:83–87.
126. Bengis RG, Thomson GR, Hedger RS, et al. Foot-and-mouth disease and the African buffalo (*Syncerus caffer*). 1. Carriers as a source of infection for cattle. Onderstepoort *J Vet Res* 1986; 53:69–73.
127. Morgan-Capner P, Bryden AS. Foot-and-mouth disease, vesicular stomatitis, Newcastle disease, and swine vesicular disease. In: Palmer SR, Soulsby EJJ, Simpson DIH, eds. *Zoonoses*. Oxford: Oxford Medical Publications, 1998:320.
128. Bengis RG, Veary CM. Public health risks associated with the utilisation of wildlife products in certain regions of Africa. *Rev Sci Tech* 1997; 16:586–593.
129. Young E, van den Heever LW. The African buffalo as a source of food and by-products. *J S Afr Vet Assoc* 1969; 40:83–88.
130. Young E, Wagener LJJ, Bronkhorst PJJ. The blue wildebeest as a source of food and by-products: the production potential, parasites and pathology of free-living wildebeest of the Kruger National Park. *J S Afr Vet Assoc* 1969; 40:315–318.
131. Young E, Wagener LJJ. The impala as a source of food and by-products. Data on production potential, parasites and pathology of free-living impalas of the Kruger National Park. *J S Afr Vet Assoc* 1968; 39:81–86.
132. Bengis RG. Animal health risks associated with the transportation and utilisation of wildlife products. *Rev Sci Tech* 1997; 16:104–110.
133. Bengis RG. Wildlife diseases of animal and public health significance in the Kruger National Park. Proceedings of the Symposium on Wildlife Utilization in Southern Africa, 28 June–6 July, 1992; Pretoria: National Parks Board.
134. Bengis RG. A review of bovine brucellosis in free-ranging African wildlife. Proceedings of the ARC-Onderstepoort OIE International Congress with WHO-Cosponsorship on Anthrax, Brucellosis, CBPP, Clostridial and Mycobacterial Diseases, Berg-en-Dal, Kruger National Park, South Africa, 9–15 August 1998. Onderstepoort: Onderstepoort Veterinary Institute, 178–183.
135. Gradwell DV, Schutte AP, van Niekerk CA, Roux DJ. The isolation of *Brucella abortus* biotype I from African buffalo in the Kruger National Park. *J S Afr Vet Assoc* 1977; 48:41–43.
136. Chaparro F, Lawrence JV, Bengis R, Myburgh JG. A serological survey for brucellosis in buffalo (*Syncerus caffer*) in the Kruger National Park. *J S Afr Vet Assoc* 1990; 61:110–111.
137. Young E, Whyte IJ. Trichinosis (*Trichinella spiralis* infestations) in wild animals of the Kruger National Park. *J S Afr Vet Assoc* 1975; 46:233–234.
138. Young E. Echinococcosis (hydatidosis) in wild animals of the Kruger National Park. *J S Afr Vet Assoc* 1975; 46:285–286.
139. Huchzermeyer FW. Public health risks of ostrich and crocodile meat. In: Ahl AS, Suttmoller P, eds. Contamination of animal products: prevention and risks for public health. *Rev Sci Tech Off Int Epiz* 1997; 16:599–604.
140. Dauth J, Dreyer JM, Raubenheimer EJ, et al. Blood lead concentrations in hippopotami (*Hippopotamus amphibius*) in the Kruger National Park. *J S Afr Vet Assoc* 1988; 59:153–154.
141. Fourie PB, Weyer K. Tuberculosis prevalence and risk of infection in Southern Africa. *S Afr J Sci* 1986; 82:387.
142. Durrheim DN, Speare R, Govere J. Southern African schistosomiasis—epidemiology and control. In: Coetzer PWW, ed. *The ecology of environmental hygiene and occupational health*. Pretoria: Easilearn CC, 1997:310–321.
143. Pitchford RJ. Temperature and schistosome distribution in South Africa. *S Afr J Sci* 1981; 77:252–261.
144. Appleton CC, Ngxongo SM, Braack LEO, le Sueur D. *Schistosoma mansoni* in migrants entering South Africa from Mozambique—a threat to public health in north-eastern KwaZulu-Natal? *S Afr Med J* 1996; 86:350–353.
145. Pitchford RJ. Some brief notes on schistosomes occurring in animals. *J S Afr Vet Med Ass* 1963; 4:613–618.
146. Pitchford RJ, Visser PS. Excretion of *Schistosoma mattheei* eggs from man, baboons and cattle living in their normal environment. *J Helminthol* 1975; 49:137–142.
147. Pitchford RJ, Visser PS, du Toit JE, et al. Observations on the ecology of *Schistosoma mattheei* Veglia & Le Roux, 1929, in portion of the Kruger National Park and surrounding area using a new quantitative technique for egg output. *J S Afr Vet Assoc* 1973; 44:405–420.
148. Murray C, Lopez A. Mortality by cause for the eight regions of the world: global burden of disease study. *Lancet* 1997; 349:1269–1276.
149. Van der Spuy JW. Trauma, alcohol and other substances. *S Afr Med J* 2000; 90:244–246.
150. Van der Spuy JW. South African road traffic statistics: 1950–1994. *Trauma Emergency Med* 1996; 13:35–38.
151. Hargarten SW. Injury prevention: a crucial aspect of travel medicine. *J Travel Med* 1994; 1:48–50.
152. Leggat PA, Durrheim DN, Apps PJ. Occupational risks posed by wild mammals in South African wildlife reserves. *J Occup Health Safety—Aust N Z* 2000; 16:47–54.
153. Durrheim DN, Leggat PA. Risk to tourists posed by wild mammals in South Africa. *J Travel Med* 1999; 6:172–179.
154. Leggat PA, Durrheim DN, Braack LEO. Traveling in wildlife reserves in South Africa. *J Travel Med* 2001; 8:41–45.
155. Broadley DG. *FitzSimons' snakes of southern Africa*. Johannesburg: Delta Books, 1983.
156. Fischhoff B, Bostrom A, Quadrel MJ. Risk perception and communication. *Annu Rev Public Health* 1993; 14:183–203.
157. Leggat PA, Carne J, Kedyarune U. Travel insurance and health. *J Travel Med* 1999; 6:252–257.