13 DDT, Pollution, and *Gomarara*: A Muted Debate

Frantz Fanon once said: "I do not know; but I say that he who looks into my eyes for anything but a perpetual question will have to lose his sight" (Fanon [1952] 1967, 29). This is the intention of this chapter: to ask questions. Could the massive aerial and ground spraying of the Zimbabwean countryside be catching up with us? Is there a connection between *gomarara* (cancer) and OCPs?

So serious is the scourge of *gomarara* in Zimbabwe (formerly Rhodesia) that on June 23, 2012, the Ministry of Health announced that it was close to finalizing a national cancer prevention and control strategy to guide the nation in fighting *gomarara* (GOZ 2013)—this against a background of rising numbers of cases in recent years, with many of the cases being readily attributed to HIV and AIDS (Moyo 2012).

I am not even sure this is a question I am qualified to answer. Is there direct evidence linking OCPs and *gomarara* in Zimbabwe? Not yet. Is there enough evidence to raise concern? Absolutely. To begin this chapter, I first will explore the state of *gomarara* in Zimbabwe briefly, drawing out the incidence of those types of *gomarara* usually associated with OCPs. The statistics are quite staggering.

In the second section, I reconstruct debates about OCPs as environmental pollutants, an issue that was muted at the height of the spraying campaigns of the 1950s–1970s and is largely forgotten now. The 1964 protests among *vatema* were confined to loss of cattle, forced resettlement, and *nhimura* (the Native Land Husbandry Act). They did not extend to DDT and other OCPs as environmental pollutants with long-term health effects. *Vachena* were more concerned with "wildlife," and politico-military struggles to save Rhodesia from "communist terrorists," according to the Rhodesians, and the struggle for self-liberation (*kuzvisunungura*) from *hudzvanyiriri* (oppression, according to *vatema*). With the end of *Chimurenga*, Zimbabwe's war of independence, the issue was not remembered.

This silence is alarming given the global banning of the chemicals, along with other synthetic products like lead-based paint and asbestos that were once deemed very safe and that now turn out to be toxic. I will examine some of the investigations made into the environmental effects of OCPs elsewhere, marshalling that evidence to ask questions and to map and follow the itineraries of these pesticides in our bodies and those of our animal cousins.

Most of the historical literature on DDT and other OCPs focuses on the United States. I am not aware of any monographs on Africa. The carcinogenicity of OCPs is still generally unexplored from a humanities and social science perspective. This is despite the fact that scholarship has focused on the toxicology factor in the banning of pesticides and environmental regulation (Dunlap 2008). The histories of the development of DDT, caught between military and civilian uses against people and pests, has received detailed attention that enables those studying the chemical's deployments in Africa to understand where it is coming from technically and culturally (Russell 2001; Nash 2007; Dunlap 1981). Beyond the United States, the histories of industrial pollution–induced diseases (Walker 2010) betray a "how DDT changed the world" tone of technological determinism that ascribes transformative powers to the artifact (Kinkela 2011), contrary to preceding chapters. We see echoes of this with respect to cell phones in Africa: "How mobile technology is changing Africa." Yet these phones did not become technology, let alone mobile, alone. They are made such by people in Africa.

As noted, *varungu* in Rhodesia were concerned with OCP pollution because it affected *mhuka*, not because it endangered the lives of *vanhu vatema* or *vanhu* in general. In this, they have much in common with their fellow *vachena* in 1960s and 1970s America. The concern over the environmental effects of OCPs on birds and other forest animals in the United States in the wake of Rachel Carson's seminal *Silent Spring* was from *vachena*, not *vatema* (Davis 2014). Some of the environmental and public health texts about the United States deal with what Susan Bohme (2015) calls "toxic justice" (see also Tarr 1996; Stine and Tarr 1998). This chapter identifies more with recent works on chemical exposure, especially on the handling of toxic chemicals, uptake in plants, and contamination from wind drift, itself a form of aerosol mobility (Harrison 2011).

"The Statistics Are Classified"

To be fair, after the initial euphoria, the residual and aerial spraying of DDT, BHC, and dieldrin came under closer scrutiny among skeptics both within

and outside the Tsetse Branch. In a detailed examination in 1951, entomologist Desmond Lovemore argued that simply because *mishonga* worked wonders "on economic farm crops growing on a few hundred acres," that did not mean automatically that they would work over "hundreds of square miles of remote and often worthless country with absolute success."[1]

Critics attacked the toxic footprint of these chemicals on the environment. The Tsetse Branch did not deny it. Nineteen years after Lovemore's criticism, Assistant Director of Veterinary Services (ADVS) Cockbill admitted: "It is a fact that we contribute to the pollution of the environment by using DDT." While defending the chemical for its effectiveness in killing *mhesvi* and its "cheaper cost than other effective insecticides," he also brought up another worrying fact: "it remains lethal … for three or four months."[2] The Tsetse Branch had carried out tests on different *mishonga* with different chemical compositions, and found them "more lethal to the tsetse fly or more persistent in their action than the organochlorine compounds, dieldrin, telodrin, thiodan, DDT and BHC." Said Cockbill:

> The toxicity of telodrin to mammals is far too high for it to be used as a general insecticide. Dieldrin and thiodan, while effective and persistent, are costlier, and more toxic to mammals than DDT. BHC is less persistent than DDT, and therefore less effective under our conditions of application. In all our tests DDT was found to be superior in use to the organophosphorus and the carbamide insecticides. Of the range of commercial insecticides available at the present time there is no satisfactory alternative to DDT for the control of *G. morsitans* by means of applications of persistent insecticide to tsetse habitat. There are undoubtedly substances used as insecticides which, in a few days, break down into products less harmful to living things than DDT, but it would be futile to undertake costly and arduous spraying operations against tsetse with insecticide that was ineffective.[3]

Cockbill was philosophical in his defense of DDT and the problem of pollution. Urging "perspective" and the need to be "realistic," he argued for a curb on "those that dispense it most liberally," who could use less toxic pesticides (or DDT doses) and still achieve their objectives—like cotton farmers, who applied it at the rate of one to one and a half pounds of DDT per acre, ten to fourteen times a season, or fruit farming, in which "much, if not most of the pesticide falls to the ground or is lost in drift."[4] By contrast, 166.5 tons of 75 percent DDT wettable powder was sprayed in the *mhesvi* habitat over 3,496 square miles of infested country at a rate of 1.8 ounces of DDT per acre.

Usually the spray was a one-off application, the second coat being applied only "in some years," especially when rainfall had diluted the effectiveness. The applications targeted tree bark, rot holes, fallen logs, and ground holes, but not foliage. Cockbill stressed that the risk of poisoning

wildlife was "not great" and was in fact "negligible compared with general farming practice."[5] However, he also stressed something telling: "The statistics are classified and are not available to me. I have been assured by the Central Statistical Office that the quantity of DDT utilized annually by this Branch is trivial compared with that used in agriculture generally."[6] DDT was also used for controlling cutworms in tobacco and maize, bollworms in cotton, and stalkworms in maize.[7] To be fair, although DDT continued to be imported as late as 1978, it had generally been replaced with "the very much more readily degradable" organochlorine compound thiodan, even though trials had shown the latter to be "very much more costly" than DDT and weaker.[8]

In 1978, Lovemore sought to assuage fears that BTTC was not concerned with the pollution issues surrounding DDT and residual sprays in general:

> No obvious effects on animal, bird, reptile, fish and other insect life have been observed during the very large scale spraying operations which have been conducted with DDT in Rhodesia. Field staff have been instructed prior to each operation to pay particular attention to this important aspect over the years, but nothing of interest has been recorded. Similarly, as regards the more insidious effects of the chemical the work done by Phelps and others at the University of Rhodesia has shown that no serious problem has developed as yet from tsetse control operations in Rhodesia. … This work continues. … It is also noteworthy that in Nigeria where DDT has been in use very extensively in tsetse control operations, in fact, probably very much more so than in Rhodesia, no serious "side-effects" have been noted.[9]

There was no mention of effects on *vanhu*—just *mhuka*.

Lovemore was also urging *vachena* to come to terms with an inconvenient fact: there simply was no other insecticide available that was "comparably as effective and sufficiently cheap to permit large scale operations." Research was ongoing on the sterilization of wild flies after attracting them in large numbers using "attractive odours" and the ultralow volume (ULV) application of thiodan by fixed-wing *ndege* operating at night. However, he cautioned: "Even in the event of one or both of these techniques being perfected tomorrow, it would be some years before one or other, or both, could be adequately applied in the field to replace … DDT."[10]

Cockbill, however, would concede later that these organochlorines had residual effect *zvipfuyo* cattle of *vatema* resettled in the reclaimed areas and that the fear of poisoning was what made the government settle *vatema*, but not *vachena*, there:

> We have no knowledge of any record that damage to wild life had occurred from a concentration of DDT, or its breakdown products, in animal fats as a result of our

insecticide spraying activities. We believe that no such records exist. However, during 1970 some donkeys belonging to this Branch died of unknown causes. They had been grazing for months within the Sebungwe spraying operations area, which had been treated with DDT. Specimens of liver and other organs taken at the postmortem examination were submitted for analysis for DDT. The Public Analyst reported that "traces of DDT were insignificant," despite the fact that modern gas chromatograph analytical methods are capable of detecting DDT in such minute quantities as one part in ten thousand million. There was thus no evidence of any accumulation of DDT in these donkeys.[11]

Even then, the reference was to *vatema*'s *zvipfuyo*, not *vatema* themselves.

Mafrayi were exposed to *chepfu*. This is clear from the "Safety Precautions" outlined in Farrell's report on the Chiredzi River spraying operation, wherein only the operator of the *mushini* wore overalls, but even he had no gloves, goggles, dust mask, headgear, or shoes. The overalls were supposed to be washed daily; each workman received daily soap allowances for the laundry and a personal bath. *Chimugondiya* (brown bar soap) was a detergent manufactured for hand-washing clothing, but it was the only soap *vatema* got, for laundry and bathing combined. By contrast, *vachena* received scented bath soap and had their clothes washed and ironed by the batmen (aides).[12] The official reports say that "no one came into contact with the neat Dieldrex 15" and that the chemical was "pumped from drum to drum with a semi-rotary petrol pump."[13]

However, the same report also says that "pouring of the 3.6% mixture was facilitated by buckets, which avoided the difficulty of pouring the insecticide from a five-gallon drum into a six inch [container]." By the end of Farrell's report, the statement that no one came into contact with the chemical is already exposed as a farrago of nonsense. "The operators frequently received spray drift in their faces. This is, of course, harmful," he begins to concede, but quickly plays this contamination down: "However, the maximum spraying time in any one month was 33 hours, and in any day 6 hours. This was divided between two operators. The WHO recommended maxima are 40 hours per week, and 8 hours per day."[14]

It was not until the late 1950s that investigations were undertaken to determine the toxicity of OCPs in Southern Rhodesia. This was to assuage the growing but nevertheless muted criticism on the possibility of poisoning the environment. The earliest tests involved the resistance of *zvipukanana* to *mishonga*. Every time that persistent (residual) pesticides were used in controlling bloodsucking *zvipukanana*, all but a few *zvipukanana* developed resistance. By 1959, no such resistance had been encountered in *mhesvi*, yet it was deemed essential to anticipate the likelihood of this

chipukanana following most others in developing resistance according to the chemical composition of the *mushonga* used. Whenever such resistance was noticed, the *mushonga* was immediately replaced with another that differed profoundly in chemical structure.[15]

Another factor pushing the research was the fear of the *mhesvi* itself developing resistance to DDT, BHC, and dieldrin in future. Therefore, BTTC undertook preliminary tests comparing the toxicity of the organophosphate Bayer S1752 with that of dieldrin in Shell Dieldrex 15 form. After sixteen weeks of repeated experiments, it became clear that dieldrin retained toxicity sixteen weeks after spraying and that the organophosphate, though toxic, was slower in its action than the organochlorine as a pesticide. There was no indication that OCPs induced resistance in *mhesvi*.[16]

Investigators looking into the toxicity of OCPs were more interested in the effects of the chemicals on *mhesvi* and in boosting the efficacy of spraying operations than the environmental effects of pollution. They were clearly not concerned with the toxicity of the chemicals on *mombe*, let alone the fact that any *zvipukanana*, *shiri*, or *mhuka* coming into skin contact with or ingesting them would die or become sick. When they thought about the effects of rain on the deposits, they were not thinking about the diminishing effectiveness against *mhesvi*, not the downstream effects of the *mushonga* being washed away and, as runoff, entering streams, large rivers, and oceans.

After *Silent Spring*: Echoes from US Debates on DDT

For purposes of the discussion in the rest of this chapter, it helps to recap the contours of the debate on OCPs in the United States that was under way as the spraying campaigns against *mhesvi* and related experiments escalated in Southern Rhodesia. Rachel Carson's book *Silent Spring* provided the lightning rod for a campaign to ban DDT use in the United States. Published in 1962, the book detailed the negative environmental impacts of chemical pesticides like DDT. Carson accused chemical corporations of lying to the public and attacked politicians for their uncritical and even complicit stance toward the "facts" the industry provided about the safety of OCPs.

The wave of criticism from environmentalists—Carson being one among many—stung the US government into action. In 1964, most OCPs were banned from use in some forests and lands. Five years later, all nonessential DDT use was ended. In 1970, the Department of Agriculture banned DDT use on fifty food crops; on wood and lumber materials; on

dogs, cats, and other domesticated animals; and in factories and on commercial and institutional premises. DDT was also outlawed in marshlands, forests, and plains of national parks and preserves of the United States. The crowning moment, that same year, came with the establishment of the Environmental Protection Agency (EPA) to oversee the enforcement of these and future environmental regulations. Two years later (in 1972), DDT and other chemical pesticides were banned from use on US soil. In 1978, the US Occupational Safety and Health Administration (OSHA) acknowledged the possibility that DDT was an occupational carcinogen (Coulston 1985, 373).

The emotions that environmental debates about OCPs in the United States raised among empowered citizens and chemical companies are instructive. To examine this, look no further than the words of biochemist and chemical industry representative Robert White-Stevens: "If man were to follow the teachings of Miss Carson, we would return to the Dark Ages, and the insects and diseases and vermin would once again inherit the earth" (McLaughlin 1998). There were those who still believed many decades later that Carson was "simply ignorant of the facts" (Bailey 2002) and must be held responsible "for almost as many deaths as some of the worst dictators of the last century," all in the name of small birds (Taverne 2005). There was big money riding on this; if DDT was banned in the United States, a global ban was next—such a huge market!

Carson's defenders pointed to an issue completely dismissed in Southern Rhodesia: that some of these besieged *zvipukanana* like *hutunga* had developed resistance to DDT (Quiggin and Lambert 2008). That is exactly what Carson had argued: that too many pesticides in the environment bred resistant types, and the less the spray the better (Carson 1962, chap. 16). In fact, DDT usage was banned neither in the United States nor internationally, whether by the US government or under the 2001 Stockholm Convention on Persistent Organic Pollutants on the basis of its connection to resistance. As we now know, within seven to ten years the pest's building genetic resistance to pesticides like DDT will be complete, and the chemicals become virtually useless (Oreskes and Conway 2010).

Defenders of DDT and other OCPs continued to argue that it was necessary and not as evil as its critics painted it to be. They argued, for example, that it degraded rapidly, its strength weakening the more it was exposed to sunlight (ultraviolet radiation) to a point at which it became a bunch of harmless chemicals. As one scientist said in 1985, that was why "levels of DDT worldwide have more or less remained constant and they have not accumulated" (Coulston 1985, 333).

Studies in the 1960s and 1970s showed that when pesticides were sprayed, besides the targeted animal the pesticide also killed land insects, aquatic insects, birds, shrews, mice, reptiles, and ants (Herman and Bulger 1979). So long as the pesticide remained, actively killing the targeted insects, other kinds of animals were also dying (Koeman et al. 1978, 55). Studies of helicopter spraying had shown it to kill predators of flies as well, such as flycatchers, spiders, ants, scorpions, grasshoppers, butterflies, hornets, wasps, and monkeys (55).

In particular, DDT and its principal metabolites, DDD and DDE, were found in nearly all *mhuka* in areas where the compound had been sprayed. Specifically, within these *mhuka* it was discovered in brain tissue, the blood stream, liver, and kidneys. It biodegraded very slowly into metabolites, at the same time also accumulating in adipose (fatty) tissue of birds and fish, making them mobile pollutants (i.e., as food for their predators). Researchers found DDT present in house sparrows, cowbirds, predatory birds, scavengers, and migratory birds. In times of extreme famine when the body needed to burn more fat to survive, DDT was found to move from its fatty deposits into the blood stream and thence into the brain, leading to death (Hill, Dale, and Miles 1971, 502).

This evidence began accumulating from the 1960s on. The concentration of DDT in the adipose tissue of egrets was discovered in Audubon Canyon Ranch (in San Francisco) as early as 1961 (Faber, Risebrough, and Pratt 1972, 111). In addition to neurological poisoning, increasing evidence was also pointing to reproductive poisoning. In Rhodesia, researchers could zone the effects of DDT poisoning according to immediate, intermediate, and secondary bioaccumulation. In the first category were *mhuka* in direct contact with spray—like small *mhuka* (including *zvipukanana*) in the air, plants, soil, and water. The second was composed of predator *shiri*, *mhuka*, and *hové* (fish) that fed directly on either contaminated water or *zvipukanana*. The third tier included predators of these predators.

Tests in the United States had shown that eggs of birds of prey like *twukodzi* (hawks), *makondo* (eagles; singular *gondo*), and *mazizi* (owls) had thinner than normal shells due to OCP poisoning (Faber, Risebrough, and Pratt 1972, 111–112; Elliott 1994). In Florida, eggs of gray herons were breaking under the birds' weight due to thin shells. In San Francisco the eggs of the peregrine falcon were observed to be disappearing mysteriously during incubation season, while among those birds whose eggs hatched, nestlings were dying in large numbers (Faber, Risebrough, and Pratt 1972, 112). The effects of DDT on populations of *hungwe* (fish eagle) and *rukodzi* (falcon) were first observed in 1983 (Tannock, Howells, and Phelps 1983).

Meanwhile, investigations in Norway from 1965 to 1983 discovered OCPs (including BHC and dieldrin) in the livers of dead *makondo* and *mazizi* (Frøslie, Holt, and Norheim 1986).

The evidence was not limited to *shiri* but extended to *mhuka* as well. Experiments with *mbeva* led to conclusions that DDT, dieldrin, and BHC also had neurotoxic, reproductive, hormonal, immunological, cardiac, renal, carcinogenic, and mutagenic effects in *mhuka* (Allen et al. 1979a, 514–518). The male and younger *mhuka* were found to be more susceptible than females and older ones, and higher dosages were more toxic. The OCPs mostly affected reproductive organs, the central nervous system, and metabolic pathways in liver and kidney. As neurotoxins, OCPs were found not serious enough to cause death. Reproductively, however, they were known to cause hormonal (estrogen and testosterone) changes that resulted in immature births in rats. In the same animal, they were also found to cause vascular congestion, tubular degeneration, and changes in kidneys. Livers meanwhile succumbed to mitochondria and fatty infiltration. In rabbits, the chemicals were discovered to limit the body's capacity to defend itself. Chronically exposed *mhuka* (especially *mbeva*) also exhibited a high risk of liver-cell tumor development. Carcinogenicity aside, the OCPs also damaged chromosomes and resulted in negative genetic (mutagenic) effects (Allen et al. 1979a, 514–521).

The Journey of OCPs in Our Bodies

OCPs are passengers and travelers in bodies. Metabolites like DDA, DDD, and DDE are formed as DDT moves through the body (or many bodies) and through food chains and ecosystems. *Vanhu* tend to excrete more DDT in their urine than do *mbeva* (mice), *imbwa* (dogs), and *tsoko* (monkeys). In order of rapidity of excretion overall, DDA was the most rapidly excreted, DDD was next, then DDT, with DDE staying longest in the body. These differences were due to each metabolite's water solubility; the higher the water solubility, the faster the ejection rate (Morgan and Roan 1971, 1972; Wallcave, Bronczyk, and Gingell 1974). The higher amount of DDE found in older women versus younger women was attributed to intake of DDT over longer periods of time (Coulston 1985, 364–365). Dietary ingestion was considered the primary source of DDT in adipose tissue, with fatty foods (poultry, milk) contributing to 95 percent of DDT intake. In the adipose tissue, DDT accumulated to as much as one hundred parts per million (ppm) and biodegraded at 4.1 mg/day; in two years, a body's DDT burden could shrink from 100 ppm to 40 ppm. It took approximately ten to twenty years

for DDT to disappear from adipose tissue, while DDE persisted many years longer, even for an entire adult life (Keifer and Mahurin 1997; Le Couteur et al. 1999).

How do DDT and other OCPs—principally dieldrin and BHC—get into the bodies of *vanhu*? Here, the first port of call is *mufrayi* himself. Workers spraying without protective clothing and entire communities are told that the pesticide is harmless and for their own good. The villagers in the countryside are exposed to spray mist, especially from airborne spraying, through wind drift, evaporation, dusty air, and rains (Gil and Sinfort 2005). This is where an appreciation of ecosystems, atmospheres, climates, rainfall, and temperature as processes and as mobilities, as first advanced in the book introduction, is helpful (Coulston 1985, 341). These OCPs concentrate in the adipose tissue of the body—in the brain, kidney, liver, and heart and, in fish, in the gill and underbelly and muscles (Zhou, Zhu, and Kong 2007; Zhou et al. 2008). When *vanhu*, *shiri*, and other living things eat other *mhuka*, like fish and smaller *shiri*, or suckle their young (as the case may be), they ingest the primary sources of OCPs. The chemicals move through the trophic web, accumulating in larger quantities all the time, so that the higher we move up the food chain, the higher the concentration (Semenza et al. 1997, 1030).

If we think of the body as moving through time and of chemicals moving and being present in the body through time, then attention to mobilities opens a new vista. The older the body gets, the more these toxic chemicals accumulate in the adipose (fatty) tissues of the body (Coulston 1985, 364–5). The journey of OCPs and their metabolites (*musvo* in *chidzimbahwe*) also disables the body's mobility (or ability to be agile), not just through death or sickness, but also by inducing neurological, behavioral, morphological, and many other abnormalities (Rowan and Rasmussen 1992; Keifer and Mahurin 1997).

Of course, the body does not retain all DDT-based chemical in its adipose tissue; some of it exits with excretions. Liver, kidney, and breast tumors have been identified among people in DDT-concentrated areas. In *vanhu*, DDT was still considered to have only minor toxic effects in the 1980s, with acute cases of poisoning resulting in extreme muscular weakness, joint pain, extreme nervous tension, anxiety, confusion, inability to concentrate, and depression. Very high doses of DDT were associated with convulsions and even death, especially when containers were not disposed of permanently and safely (Allen et al. 1979a, 513).

Studies from the 1960s and 1970s had found no signs and symptoms of DDT among workers exposed to it, even after chest X-rays (Laws et al.

1967, 766; Deichmann and McDonald 1977). By the 2000s, however, this was no longer the case. The question was how high DDT dosages could lead to convulsions and death. A study had found "a significant association … between DNA migration or percentage of damaged cells and blood concentrations of p,p´-DDT, p,p´-ðð, and p,p´-DDE" (Yáñez et al. 2004, 22). This was especially the case among spraymen in Mexico, who worked in conditions like the ones in Southern Rhodesia, with parts of their bodies exposed (18). Other scientists found DDT not to be genotoxic, even while others associated it with Type 2 diabetes.

Workers that were exposed directly to chronic contamination with OCPs experienced jerks, seizures, hearing and visual problems, anemia, leukemia, fatty infiltration of the liver, degeneration of cardiac muscle, and damage to lungs, kidneys, and brain. They then descended into convulsions, leading to death due to asphyxiation and cardiac arrest. Other symptoms of poisoning also included chronic liver damage (cirrhosis), chronic hepatitis, endocrine and reproductive disorders, allergic dermatitis, breast cancer, non-Hodgkin's lymphoma, and polyneuritis. As early as 1989, scientists made connections between benzene-containing substances like BHC and various types of leukemia (Lee, Johnson, and Garner 1989; Tompa, Major, and Jakab 1994, 159; Kumar and Kumar 2007, 2). They also noticed marked rises in frequencies of chromosomal aberrations (CAs) in peripheral blood lymphocytes of workers involved in loading, packing, and transporting OCPs (Tompa, Major, and Jakab 1994, 160)—the work *mafrayi* were doing.

During the 1990s, research made a clear connection between parental exposure to pesticides and risk of *gomarara* in children, linked mostly to the exposure of the father in the preconception phase. Specifically, it suggested links with the two commonest *gomarara* among children, acute lymphocytic leukemia and central nervous system tumors. Links with Wilms' tumor, Ewing's sarcoma, and soft-tissue sarcomas were still tenuous (Flower et al. 2004). This was not limited to *mafrayi*; farmworkers and mineworkers were similarly exposed without knowing it (Gladen et al. 1998; Fenske 1997; Gomes, Lloyd, and Revitt 1999).

The *mishonga* usually entered *mafrayi*'s bodies through their skin and noses as they sprayed. Dieldrin in particular was easily absorbed through skin contact. By the early 1970s, some scientists were warning that dieldrin, even more than DDT, was "the greatest hazard" to spraymen. Elsewhere, impotence had been noticed in four out of five farm workers constantly exposed to OCPs (Espir et al. 1973). DDT-exposed males experienced reductions in testosterone levels, sperm count, and the semen volume of ejaculate. Meanwhile, abnormal sperm and sperm motility increased (Bush,

Bennett, and Snow 1986; Ayotte et al. 2001; Martin et al. 2002). In 2012, OCPs were identified as synthetic hormone-disrupting chemicals that led to a gradual increase in two male sexual-development disorders (cryptorchidism and hypospadias), testicular dysfunction, and testicular cancer (Bergman et al. 2013. It was speculated that occupational exposure could alter the sperm genetically prior to conception, damaging fetal development (Salazar-Garcia 2004). Some scientists, however, quibbled over the absence of any experimental studies to back up the birth defect claims. High doses of DDT, up to almost 1000 mg/man/hr., were recorded in places of sustained chemical use (Wolfe and Armstrong 1971, 169). The research indicated that DDT had little chance of skin absorption unless applied to the skin in fats or oils (Rivero-Rodriguez 1997). Yet, as we have discussed, in Southern Rhodesia DDT was *mixed with oil* to achieve penetration on leaves and on bark surfaces.

The chemical could be spread in any number of ways. This often occurred during application when the mist blew into the trees and the wind direction changed, blowing right back into the sprayer's face; or the *mushonga* might spill onto the body or clothing, making skin contact inevitable. It occurred due to lack of protective clothing like boots; overalls that were not torn and that covered the legs, torso, and arms completely; and face masks and helmets. Rubber gloves certainly insulated the sprayer from contact; leather or fabric ones were like sponges, absorbing and retaining the pesticide in contact with them so that it slowly seeped into the skin. Few *vatema* operating the *mushini*, carrying *mushonga*, or walking through sprayed swaths wore any shoes, and they stepped directly on the sprayed ground.[17] However, any such information about contamination was "classified."

Understanding the routes of OCPs into the environment and then into *mhuka* is critical for a comprehension of the full impact of DDT spraying in Southern Rhodesia. When it rained, the Tsetse Branch was far more worried about limits on flying and spraying time, diminished toxicity against *mhesvi*, and the inevitable leafing of the vegetation than about pollution and public human and animal health. By 1967, researchers had discovered that drinking water and eating food were the two major ways through which *mhuka* and *shiri* ingested DDT (Nash and Woolson 1967, 924; Menzie 1972, 199). OCPs were also inhaled through aerosols and dust from dry deposits or contaminated soils in the quantity of 5 mg/year. Although some scientists urged the use of respirators (Coulston 1985, 339), these were certainly never used in Southern Rhodesia.

During spraying, about 30–50 percent of the chemical was lost to the air and resulted in atmospheric contamination. Aerial spraying in particular

deposited solid, gaseous, and liquid forms of OCP into the atmosphere through wind drift and evaporation (Gil and Sinfort 2005). Pesticides also entered the atmosphere after application when they evaporated out of crops and soil where they would have dissolved (volatilization), through degradation pathways (e.g., hydrolysis and photolysis), and through sheer wind erosion. In the air, the distribution of OCPs varied according to their chemical and physical characteristics and meteorological conditions. They exited the atmosphere as acid rain or as solid and gas injections or through intake by living things (Gil and Sinfort 2005). In Rhodesia, windy days provided optimal spraying conditions, the drift enabling the aerosol droplets to move sideways into the vegetation, touching *mhesvi* hiding on the underside of the leaf or log and in between cracks in bark.

Workmen returning with OCP-contaminated clothing brought the chemicals into the home; *tsika*/*chivanhu* (both then as now) made women responsible for washing their husbands' clothing. This exposed pregnant women—and their unborn children, through the placenta (Salazar-Garcia et al. 2004). Comet assay tests performed on pregnant women in 2004 demonstrated the correlation between DDT, DDD, and DDE levels in the blood and DNA damage in women in *hutunga* and agricultural spraying (Yáñez et al. 2004, 18).[18] Connections were found between DDT and DDE and altered menstrual cycles, fetal loss, and earlier or delayed menopause. In 2012, the hormone-disruptive activity of OCPs—especially the development of new substances mimicking the behavior of estrogen—was noted to be increasing (Bergman et al. 2013; Garcia-Rodriguez et al. 2004).

Mothers also contracted OCP poisoning by eating contaminated food or drinking contaminated water. For the latter, people and other *mhuka* ingested OCPs when they drank water from wells and rivers, where the runoff from sprayed areas accumulated. In winter, when most rivers stopped flowing and evaporation was at its peak, the ratio of animals to waterholes also increased (Castilhos et al. 2000; Zhou, Zhu, and Kong 2007). Each rainy season, the flooded rivers gathered the contaminated water and deposited it downstream and thence into the ocean. A 2010 study of dolphins on the Zanzibar coastline indicated the presence of lindane and DDT metabolites, the health effects of which were not established, but they were deeply troubling from an environmental health perspective (Mwevura et al. 2010). Studies of persistent organochlorine pesticides in Lake Tanganyika showed negative consequences to fish and fish eaters like *shiri* and *vanhu* (Manirakiza et al. 2002).

Therefore, the polluted environment becomes one source of many routes through which chemicals enter us, travel within us, lodge inside our

organs, and exit us into the food chain or the environment (Menzie 1972, 199; Nash and Woolson 1967, 924; Coulston 1985, 337). The route from the spring or well to the mouth, through the stomach(s) to the exit points (urine, fecal matter, sweat, and mucus) becomes the transportation infrastructure and transient workspace the chemical passes through, acts and is acted upon, and moves on by dint of the body's own biologically mobile workshop (Coulston 1985, 341).

(How) Do OCPs Cause *Gomarara*?

The itineraries of OCPs in the body began to raise alarm when some scientists began to associate them with *gomarara*—itself a cellular mobility of sorts, if we think of mutagenicity that way. The argument began to be made in the 1970s that, on entering bodies, certain chemical compounds trigger the growth of tumors that are simply abnormal masses of tissue that are functionless but not inflammatory, especially where preexisting health issues exist. A chemical that does this is called a *carcinogen*; the tumor does not have to be painful or harmful (if it is a benign instead of malignant tumor); just its sheer presence is enough to qualify a chemical that causes it as a carcinogen (Coulston 1985, 348).

The next question, then, is whether chemicals like OCPs cause, promote, or escalate *gomarara*. Inevitably, the first link drawn between DDT and *gomarara* was that the former promoted rather than caused the latter. DDT was not a "true" carcinogen (Coulston 1985, 349). By 2004, DDT, DDD, and DDE were confirmed to cause DNA damage in women; to alter their menstrual cycles; to cause fetal loss, early menopause, retarded childhood or pubertal growth; to induce significant structural and functional neurodevelopmental changes, testicular disorders and tumors, breast cancer, chronic liver damage, Parkinson's and Alzheimer's diseases, kidney diseases, and immunosuppression; and to cause a decrease in semen quality, increase in testicular and prostate cancer, increase in defects in male sex organs, and increased incidence of breast cancer (Yáñez et al. 2004, 18; García-Rodríguez 1996, 1093; Allen et al. 1979b, 679–680; Wolff and Toniolo 1995; Kumar and Kumar 2007; Pan et al. 2009).

As Freya Kamel and Jane Hoppin noted in 2004, the tragedy of pesticide poisoning is that it usually affects the poor—especially farm workers, who cannot afford medical consultations. For that reason, it usually goes undiagnosed; hence "workers who have never been diagnosed with pesticide poisoning may still have sustained high exposures or experienced pesticide-related illness; therefore, using diagnosed poisoning as a criterion

for inclusion in an exposed group or exclusion from a comparison group may incorrectly classify individuals. … Farm owners who employ others to apply pesticides may have limited personal exposure to pesticides" (Kamel and Hoppin 2004, 950; see also Moses et al. 1993).

OCPs are a global problem. Countries where *hutunga* and *mhesvi* are found have high temperatures and heavy rainfall that enable the rapid movement of these pesticide residues through air and water into the global environment (atmosphere, sea; Ramesh et al. 1990, 290). DDT and HCH (hexachlorocyclohexane; not to be confused with BHC—that is, benzene hexachloride), for example, are the major *mishonga* in Indian foodstuffs such as grains and vegetables. Milk and milk products are the major sources of dietary exposure to DDT and HCH in India, above FAO/WHO-approved levels. Untreated water is a potential source of DDT and HCH (Kannan et al. 1997).

It is striking—but hardly surprising—that research into chemical exposures and breast cancer effects was not a high priority in the United States. To start with, it only affects women in a predominantly male-dominated scientific field. Moreover, research into the carcinogenicity of cancer, just like research into guns and violence, tobacco and its harmful effects, or that of genetically modified crops, has met with resistance and downright suppression from the industries that stand to benefit from continued use of these drugs (pun intended). The connections between breast cancer and reproductive hormones must at the very least have justified an investigation into environmental chemicals and their effects. Yet more than four decades after the banning of DDT use in the United States in the wake of proven adverse reproductive effects in wildlife, the agenda for a rigorous debate on the carcinogenic effects of OCPs remains elusive.

There is a serious contradiction in responses to chemicals and other goods that are later found to be either toxic or defective in the United States and Africa; I specifically mean companies headquarters in the former with subsidiaries in or selling chemicals to the latter. In the United States, it is common for goods made locally or abroad to be "recalled" or banned entirely after successful class-action lawsuits are brought against them in court: goods such as lead-based paints, asbestos, pharmaceutical drugs, faulty automobiles, and so on. Adverts are floated everyday inviting claims from potential victims of mesothelioma who worked in the navy, shipyards, mills, heating, construction, or automotive industries who were exposed to asbestos to submit claims. By contrast, those same companies are never required to extend this same compensation to victims who did the actual mining, transportation, shipping, and handling in Africa, where

many of these companies extracted these ores. Gabrielle Hecht's (2012) extensive study of these commodity chains demonstrates this in the case of uranium that played such a critical role in both the military and energy projects of the United States and South Africa. There is also usually an assumption that simply banning something marks the end of the problem. Lead-based paint is a perfect example: it remains on the walls, as does the asbestos, poisoning away *vatema*, with neither consequences for those that have poisoned others nor compensation for the victims, many of whom just die at home, unable to afford a visit to the clinic or to get tested.

Worse, a ban is a door that closes a chapter of inconvenient questions investigators might ask. Put differently, it seems that investigators look for answers so long as a drug or chemical is being produced and used. The moment it is withdrawn or its production is terminated, they look for another cause célèbre. Perhaps that is exactly why science, technology, and society (STS) are essential in Africa or anywhere else. That is, to investigate and raise questions when goods are no longer here to do what is *good* for people and the environment—bar a few who get rich, consequences be damned—but have instead become the problem. Once DDT was banned, there seemed no further incentive to conduct detailed research into and evaluate its effects in the long term, especially in places remote from the United States where the *chepfu* came from.

In fact, DDT and dieldrin, and then endosulfan, continue to be used in Africa, Latin America, and Asia long after 1985, when their production in the United States for export was restricted. Translated, this means that US companies were exporting *chepfu* that the country had deemed too dangerous on its own soil, in its own atmosphere, to its own people. Are we all humans, then? Do we bleed blood too?

The problem with OCPs is that they do not kill people en masse or instantly, their gestation period being more drawn out over almost a person's lifetime. Thus, the incentive to investigate the long-term effects of OCPs did not exist, because the generation affected by them was still healthy and much alive—and in fact bought into the idea that DDT was very safe for *vanhu* (Wolff and Toniolo 1995). The Rhodesians were more worried about the chemicals' effects on *mhuka*. The way a society treats its animals is a good indicator of how it treats other people. We may assume since they called and treated *bobjaan* (baboons), they included *vatema* in their compassionate worry for *mhuka*.

The deafening silence on the possible carcinogenicity of OCPs was neither localized to Rhodesia nor limited to the mid- to late-twentieth century. Research from the 1970s and 1980s seemed to indicate that DDT, DDD, and DDE accumulations in the human body had a fifty-fifty chance of causing

any health problems, let alone *gomarara*. The same researchers, however, also admitted to the lifelong bodily burden of DDT and its metabolites once ingested, especially DDE (Coulston 1985, 366). Nearly half a century later, silence!

All of this makes an inquiry into the environmental history of DDT and other OCPs in Zimbabwe, Africa at large, and the Global South more broadly important. As Epstein (1994) noted, the definition of "environmental" causes of *gomarara* research has almost exclusively focused on viruses, vitamins, diet, tamoxifen, smoking, occupational exposures and radiation, while ignoring pollution or industrial chemicals. Almost to the end of the twentieth century, studies made no attempt to examine the obvious link between carcinogens and breast cancer, choosing instead to explore dietary factors (Wolff and Toniolo 1995). Even as recently as 2009, scientists have lamented "the lack of knowledge about human exposure and health effects in communities where DDT is currently being sprayed" (Eskenazi et al. 2009).

By 2000, breast cancer incidence rates were on the increase, and it was no longer enough to say that this was purely a result of more efficient screening and detection of *gomarara*. What accounted for the cases revealed efficiently to begin with? Could it be that environmental chemicals were responsible, just as they had already proven to be endocrine disruptors? How could it be ignored that *gomarara* figures had risen at the same time as the rise in the use of OCPs (Allen et al. 1979b, 679)? Already, epidemiological studies were showing that women who had the highest DDE levels in their blood also had a fourfold risk of breast cancer (Allen et al. 1979b).

Therefore, at the very least, Zimbabwe's *gomarara* crisis deserves notice. In the limited space of this final section, and in the absence of a systematic ethnographic study on carcinogenicity, I now seek only to raise awareness of the issue and ask questions. This is not a conclusion, but a start—not necessarily my own start, but one for all those who care for humanity. Although studies of *gomarara* in oncological wards in Africa take us to the scene of encounter between cancer and medicine (Livingston 2012), we also need to step out of the ward and explain the cases under chemotherapy. To what causes are they bearing witness?

Opening a Discussion: Environmental Pollutants and *Gomarara* in Zimbabwe

The record-keeping on *gomarara* in Zimbabwe has been erratic at best, making the availability of up-to-date statistics on the countryside difficult,

especially for cases originating from tsetse-related OCP-sprayed areas. Add to that the smallholder farmers and farmworkers that have extensively used OCPs like Gammatox (BHC) since at least the 1960s as pesticides when growing vegetables and grain, the tons of OCPs sprayed to guard against *hutunga*, and the large tracts of *mapurazi* on which fruit trees, tobacco, cotton, and maize that require pesticides grow!

Only one government agent, the Zimbabwe National Cancer Registry (ZNCR), currently collects and collates statistics on *gomarara*. The first ever cancer registry in the country was established in the second-largest city, Bulawayo, in 1963, but it closed in 1977, after which *gomarara* registration lapsed, with information confined to data from histopathology series. The registry reopened in 1985, this time in the capital, Harare (formerly Salisbury), but it only covered statistics for the city. Its numbers are drawn from routine weekly visits to the wards of the nation's two central referral hospitals, medical records with a discharge diagnosis of *gomarara*, government laboratory pathology reports, completed notifications from the Radiotherapy Department, monthly reports from the private pathology laboratory, and death certificates for Harare residents. The major limitation is that these figures are drawn only from Harare residents or patients admitted to Harare hospitals (Kadzatsa and Chokunonga 2016; Chokunonga et al. 2013).

The elderly and the poor usually retire to, or never leave, the rural countryside. They live far from Bulawayo or Harare—in Hwange, Binga, Lupane, Sanyati, Gokwe, Hurungwe, Guruve, Centenary, Muzarabani, Rushinga, Mutoko, Mudzi, Nyanga, Chipinge, or Chiredzi East. These outskirts of Zimbabwe are the same places where massive amounts of OCPs were dumped in the environment exactly when (1950s–1970s) the groups being struck down by *gomarara* now were children or young adults, including those employed in tsetse control operations as *magocha* and *mafrayi*. If any in this group go to the hospital, they go to the one nearest their village, or to a general hospital at which only painkillers such as Panado, Disprin, or paracetamol are dispensed and where no doctor, let alone testing equipment, is to be found. There are also those that go to a *n'anga* (traditional healer) or to *maprofita* (prophets) instead—not because they are ignorant of the efficacy of clinical medicine, but because they are too poor to afford a radiological test, bus fare, or hospital fees.

They die from what society commonly calls *kuroyiwa* (witchcraft), *chirwere chegomarara* (disease of the mistletoe), *ronda rinongobva pasi risingapori* (a wound that emerges from the ground and never heals), or *kutemwa nemusoro* (headache). Statements like "*Akangoti mudumbu mudumbu, ndiye*

sarai" ("He started complaining of a headache, then said goodbye"), "*Gumbo rake rakabva pasi*" ("Her leg came from the ground"), "*Rakangotanga, hapana anoziva kuti chii*" ("It just started, nobody knows what it is"), and "*Ane chitsinga*" ("He has an implant inserted by a witch") are common. These people die quietly, without even an autopsy, carrying their secrets to the grave.

Gomarara is becoming a serious policy issue in Zimbabwe, a nation of fourteen million people. This is especially true after prominent politicians Morgan Tsvangirai and Thokozani Khupe, president and deputy president of the opposition Movement for Democratic Change (MDC), respectively, admitted to colon and breast cancer in 2016 and 2011, respectively. They can count themselves lucky to have lived longer (Tsvangirai succumbed to the disease on February 14, 2018), because they are powerful people of means; up to 1,300 people die of *gomarara* in Zimbabwe every year without ever getting tested or treated. Since 2007, the number of new cases of *gomarara* per year has more than doubled from 3,349 to over 7,000 and rising; only 700–1,500 are treated. There are only two radiotherapy treatment, chemotherapy, and surgery centers for *gomarara* in a country of fourteen million citizens. The machinery at the two centers, the Mpilo and Parirenyatwa referral hospitals, is constantly broken.

The World Health Organization (WHO) has emphasized sex, tobacco use, and alcohol as contributing to 40 percent of the *gomarara* case load (Jemal et al. 2012; Shafey, Eriksen, and Mackay 2009; Glynn et al. 2010). Indeed, it has urged *vatema* to make "lifestyle changes"—to stop "risky sexual behavior," eat a healthy diet, perform regular physical activity, limit alcohol intake, avoid or reduce smoking, and have regular health checkups ("Fighting the Cancer Scourge" 2012). Completely excluded from this approach is an environmental intervention, in a country and continent whose history and landscape has been so thoroughly drenched in OCPs. The most common *gomarara* in Zimbabwe are cervical, breast, prostate, and skin cancer, but *gomarara* of the digestive system are on the rise. The focus of the Cancer Association of Zimbabwe (CAZ) is to establish the relationship between *gomarara*, HIV and AIDS, and other conditions, like diabetes and hypertension (Moyo 2012; "Call to Decentralize Cancer Treatment Services" 2012). OCPs are not on the agenda.

In a groundbreaking article on the social aspects of cervical cancer in Zimbabwe in 2006, J. F. Mangoma and colleagues said something that is as true for this specific *gomarara* as it is for breast, bladder, or prostate cancer. According to the authors, "Cancer of the cervix is a gender-sensitive condition in that only women suffer from it. Thus, its importance may easily be marginalized. Often, *gomarara* has to compete for meager resources with more dramatic diseases like HIV and AIDS, malaria and tuberculosis"

(Mangoma et al. 2006, 93). Thus, though preventable and curable, its morbidity and mortality is increasing in Zimbabwe (Chirenje et al. 1998, 1999). Most studies of *gomarara* focus on the scientific situation, not on sociological, historical, and environmental aspects. Mangoma and others thus sought "to give a sociological and anthropological insight into rural black women's understanding of cervical cancer, its symptoms and the importance of screening" (Mangoma et al. 2006, 93). These scholars used Mutoko as a case study, but limited their studies to the social environment (patriarchal societies, kinship, marital and sexual factors), perhaps oblivious to the fact that Mutoko experienced sustained anti-*mhesvi* spraying operations in the 1960s and 1970s.

The funding priorities for combating *gomarara* in Zimbabwe are decided by donor countries and organizations in the United States and Europe, as well as WHO—not by Zimbabweans, let alone the Zimbabwean government, which is broke. These priorities are heavily skewed toward communicable disease–related *gomarara*. Hence, Kaposi's sarcoma and cervical *gomarara* receive the most funding because of their association with communicable diseases like HIV/AIDS and HPV (papillomavirus), whereas those associated with noncommunicable carcinogenic agents receive less (Kachala 2010). Some of the money, just as with HIV/AIDS funding, goes toward advocacy work to convince people that *gomarara* is not "a curse unleashed by angry ancestral spirits on errant individuals" but a matter of tumors (*gomarara*; Moyo 2012).

Studies of *gomarara* among Zimbabwe's white population over thirty years found the pattern of *gomarara* roughly typical of populations with high socioeconomic status living in Europe or North America, with elevated incidence rates of breast cancer, large-bowel cancer, and, in women, lung cancer. However, there were also some rather unusual features, like higher skin cancer (including melanoma) rates and liver and bladder cancer rates than were often seen in white populations. The statistics for 1990 to 1992 found that 551 suffered from *gomarara* of the skin other than melanoma (just nineteen), with the forty-five and over age group the most vulnerable; 318 suffered *gomarara* in all other sites except the skin, again with the forty-five-plus age group most susceptible; and sixty-seven suffered prostate cancer, mostly affecting those over fifty-five. Among white women, 362 suffered *gomarara* of the skin, mostly from age thirty-five and over; 135 suffered breast cancer, again in those thirty-five and over; 339 suffered "all sites but skin" cancer, also in those thirty-five and over. The skin cancers were mostly skin tumors. Figures for 1995 showed that breast cancer in Zimbabwe was "virtually the highest in the world," and it was put down to

people's diet, along with the very high incidence of ovarian cancer (Bassett et al. 1995, 24–28).

Among black people in Harare, the 1995 statistics showed high rates of liver, prostate, and cervix cancer and low rates of large-bowel and breast cancer. Incidences of *gomarara* of the esophagus, bladder, and (in men) lungs were also high; the increase in Kaposi's sarcoma was attributed to AIDS. At that point, no evidence yet existed of non-Hodgkin's lymphoma or cervical cancer. *Gomarara* occurrence in Africa was readily attributed to "urbanization, with its accompanying changes in diet and lifestyle, as well as the recent AIDS epidemic" (Bassett et al. 1995, 29). Again, there was no discussion of environmental factors.

Another instance of this bias can be seen in research on primary carcinoma of the liver, one of the most common *gomarara* in sub-Saharan Africa. It is attributed to aflatoxin exposure, but even though scientists acknowledge the aflatoxin's contribution to geographic variations in liver cancer in the region, they make no mention of its sources or promoters—like DDT. Rural men have also tested positive for iron overload, blamed on the high iron content of home-brewed beer, but there is no attention to OCP content in such foods, especially grain or crops treated with gammatox, malathion, or DDT. Tobacco, for example, is blamed for esophageal cancer, but the high rates observed in certain regions defy this simple explanation in the absence of environmental studies (Bradshaw and Schonland 1974; van Rensburg et al. 1985; Segal, Reinach, and de Beer 1988; van Rensburg 1981; Marasas, van Rensburg, and Mirocha 1979; Sydenham et al. 1990).

There is thus far no satisfactory explanation for why *gomarara* incidence rates in Zimbabwe have been higher than anywhere else in Africa. In 1995, esophageal cancer was high, lung cancer moderately high, and liver cancer also high. Cervical cancer was the highest recorded in Africa. Breast cancer was previously not common but increasing in incidence, as was melanoma, 90 percent of it affecting the legs. There were also age-specific incidences of leukemia, non-Hodgkin's lymphoma, and myeloma (Bassett et al. 1995, 29–30). The incidence of stomach and lung cancer was moderate, with large-bowel cancer relatively rare. Kaposi's sarcoma is now the most common *gomarara* in men (23.3 percent), accounting for two-thirds of *gomarara* in men aged twenty-five to thirty-four. However, the increase in women is striking; it is ranked third in frequency after cervical and breast cancer, the most common *gomarara* among young women (29 percent at ages fifteen to thirty-four). Kaposi's sarcoma was long endemic in Zimbabwe in elderly men—62 percent of them aged forty-five or more,

a quarter of them sixty-five and over, and almost all of them skin tumors. The pattern of cases was credited to HIV and AIDS; none of them to environmental pollution factors. Non-Hodgkin's lymphoma incidence was not very high, but is increasing—again attributed to HIV (Bassett et al. 1995, 34; Gordon 1973).

The question I end with is this: How would an environmental approach—moreover, one that examines OCP use in mass campaigns against *mhesvi*, *hutunga*, *zvimokoto* (quelea birds), *hwiza* (locusts), and *mhunduru* (armyworms), as well as field uses in crop and fruit tree protection—aid understanding of the *gomarara* crisis in Zimbabwe today? I do not ask as a scholar—just as a son whose father died of it and a son-in-law whose father-in-law has it.

The Mobile Workshop

The Tsetse Fly and African Knowledge Production

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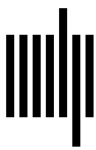

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