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The Mobile Workshop

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Published by The MIT Press

Mavhunga, Clapperton Chakanetsa.

The Mobile Workshop: The Tsetse Fly and African Knowledge Production.

The MIT Press, 2018.

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10 The Coming of the Organochlorine Pesticide

The methods of the Gaza king Mzila—from whence Rhodesian authorities derived “Umzila’s principle”—had been organic. *Hurumende* maintained his methods of clearing and keeping the land free of *mhesvi*, but the materials they used were toxic to the environment. The principles had shifted: *vatemala* managed *mhesvi* through strategic deployment within the environment, rather than exterminating *mhesvi*, its habitat, and *mhuka*, whereas the Rhodesian state sought to control and eradicate it. We have already discussed the trail of forest and animal destruction left behind in pursuit of this goal.

We now add another dimension: the extensive poisoning of the environment to exterminate *mhesvi*. The deployment of *chepfu* to eliminate or control *mhuka*, both big and microscopic, that were dangerous to *vanhu*, *zivpfuyo*, and *zvirimwa* (crops) from the beginning of the Rhodesia project in 1890 is the subject of this chapter. It argues that the use of synthetic chemical poisons, while marking a significant turning point, was not entirely a new phenomenon in the history of chemical production and usage in local societies. Such innovations in *chepfu* (poisons), as will be shown, had existed before, but—tellingly—in organic, biodegradable, hydrodegradable, and photodegradable forms.

Given the extensive destruction of trees via both mechanical and chemical means, the massive amounts of organochlorine pesticides (OCPs) dumped into the environment to kill *mhesvi*, and the poisoning of *mombe* in massive chemotherapeutic interventions, OCPs immediately present an opportunity to explore the question of pollution and its health effects. Substantial research has been done on occupational health issues related to mining in Southern Rhodesia and South Africa (Van Onselen 1982a, b; Packard 1989; Phimister 1994), but mostly concerned with the political economy of migrant labor located at the intersection of race and class. Some studies have begun to explore citizen mobilization and activism

relating to asbestosis in the Northern Cape and Swaziland, with a sustained focus on the scientific-medical aspects (Waldman 2007; McCulloch 2002, 2005). They echo Sheila Jasanoff's (1995) "science at the bar" approach (Meeran 2003), with its emphasis on locating occupational health and wellness at the intersection of law and science. In recent years, STS scholars have focused on the materiality of contamination and how it happens, with close attention paid to uranium, asbestos, and the chemicals used to extract them (Hecht 2012).

The purpose of this very brief chapter is a modest one: to introduce and account for the specific circumstances by which OCPs arrived in Southern Rhodesia. In fact, by the time organochlorines like DDT, BHC, and dieldrin and organophosphates like Thallium were deployed in combat against *mhesvi*, *hutunga*, *hwiza* (locusts), and *zvimokoto* (quelea birds) after World War II, Southern Rhodesia's farmers had been dispatching *mhuka*, *shiri*, *zvipukanana*, and *hutachiwana* with *chepfu* through ingestion, inhalation, and skin contact for over fifty years. The chapter therefore starts from this earlier history, well before DDT and its peers, in search of antecedents that profoundly shaped and offered a broader context for the use of OCPs.

Chapters 10, 11, and 12 build on and contribute to the global histories of pesticides, with the present one setting up the discussion of DDT and its organochlorine associates BHC, dieldrin, and, to a lesser extent, endosulfan. This chapter is concerned primarily with pre- and non-OCP history that places the coming of DDT and other OCPs in context. The story of DDT itself has received distinguished attention (Russell 2001; Stapleton 2005), with emphasis placed on experts and expertise, regulation, environmentalism, chemistry, and entomology. Most of the discussion centers on agriculture, armies, and indoor use, however (see also Russell 1999).

The discussion that follows does something different. First, it will introduce OCPs as material things, before discussing their coming to Southern Rhodesia, with emphasis on the circumstances of their arrival. The argument advanced is that this is not merely a case of knowledge transfer—that is, of a knowledge already developed and proven workable in Europe and North America. To start, like all other ingredients local and inbound, OCPs were coming in as raw materials to assemble stratagems that local actors were designing against *mhesvi*. OCPs were subjected to local experiments precisely because of the nature of the pest that Southern Rhodesia was fighting: a mobile *hutachiwana*-carrying *chipukanana* that inhabited climatic, geophysical, vegetational, and human environments different from those in the United States and Europe, where the chemicals had been designed.

Unlike preceding ones, this chapter has no conclusion, because it is the opening dialogue for a story continued in the next two chapters.

DDT, Dieldren, and BHC

Three organochlorine pesticides (OCPs) arrived in Southern Rhodesia just after 1945 and dominated *mhesvi* control operations for much of the late Rhodesian period. They were DDT (dichlorodiphenyltrichloroethane), lindane (gamma-hexachlorocyclohexane), and dieldrin. Later, they were largely replaced with two OCPs: thiodan (also called endosulfan) and deltamethrin. This section first explains what these chemicals were and how and for what they were originally designed in the United States and Europe, as well as the circumstances of their travel and deployment in *mhesvi*-occupied Africa, focusing on Southern Rhodesia (later Zimbabwe).

In the global OCP discussion, Africa does not exist; an impression might be created that the chemical was never applied. The most extensively used of the three OCPs in Zimbabwe, DDT is a colorless, crystalline, tasteless, and almost odorless organochloride first synthesized in 1874 through mixing chloral (CCl_3CHO) and chlorobenzene ($\text{C}_6\text{H}_5\text{Cl}$) in a sulfuric acid catalyst. When it decomposes and loses some of its constituent parts, DDT becomes DDD (dichlorodiphenyldichloroethane) or DDE (dichlorodiphenyldichloroethylene). DDT is a hydrophobic chemical that is highly soluble in fats and oils. It acts through skin absorption and ingestion; once inside the body, it attacks the nervous system by interfering with normal nerve responses. Its immediate effects in *vanhu* and *mhuka* are less toxic because it is poorly absorbed through thick skin, but laboratory animals express hyperexcitability, tremors, incoordination, and convulsions, with fatal doses producing liver lesions. In *vanhu*, exposure leads to a prickling sensation of the mouth, nausea, dizziness, confusion, headache, lethargy, incoordination, vomiting, fatigue, and tremors. Its effects in *zvipukanana* are far more rapid and lethal because the chemical is easily absorbed through its outer covering (the exoskeleton).

DDT was first used in a pesticidal role in 1942 by the US Army to kill *hutunga*, *zvikwekwe* (ticks), *inda* (lice), and *mbeva* (mice), and from 1946 onwards it became the signature pesticide for malaria and *mhesvi* control throughout Africa. After World War II ended, the chemical became a civilian pesticide in agricultural pest control and in campaigns to kill *hutunga* and stop malaria in Europe and North America (WHO 1979; de Zulueta 1990). The antimalarial role of DDT went global in 1955 under the World Health Organization's program covering North Africa, the Balkans, the Caribbean,

Northern Australia, Southeast Asia, and the South Pacific (Gladwell 2001; Chapin and Wasserstrom 1981; Sadasivaiah, Tozan, and Breman 2007). Today, DDT remains in use for malaria control in South America, Africa, and Asia; in 2016, calls intensified for its use against Zika virus-carrying *hutunga* in Brazil. In addition to its use in the control of *mhesvi*, the chemical has been extensively deployed in Africa to protect crops and fruit trees against worms and other pests and indoors to kill *masvosve* (ants), *mapete* (cockroaches), *nhunzi* (houseflies), and *makonzo* (rats). DDT has remained in use for as long as it has because it is inexpensive to make, is effective, and has a long residual effect in the environment.

First believed to kill only *zvipukanana* and liberally deployed for such, DDT has since proven to be both a pollutant and a health hazard for plants, *mhuka*, and *vanhu* alike (Allen et al. 1979a, 513). It is very “lipid soluble” (can easily dissolve in fatty substances), it has a very long “half-life” of six to ten years in *vanhu*, and it is very slowly released from fat once absorbed. That is why the Stockholm Convention on Persistent Organic Pollutants (2001) moved to limit its use to the control of public health disease vectors like *nyong’o* and *n’gana* through killing and repelling *hutunga* and *mhesvi*, respectively. DDT did not become a pesticide overnight. Even as its postwar use shifted to civilian pest control, scientists in the United States—and newspapers such as the *New York Times*—were already saying that the chemical was a health hazard (EPA 1975). They were largely ignored; however, the awareness campaign attracted the attention of the naturalist Rachel Carson. From an op-ed in the *New Yorker*, Carson produced the seminal 1962 book *Silent Spring*, which argued that OCPs were poisoning humans and the biotic environment (Lear 2007). The popularity of the book spawned the rise of the postwar environmental movement in the United States, forcing President John F. Kennedy to institute a commission of inquiry into Carson’s findings. The subsequent Scientific Advisory Committee advised the use of DDT and other OCPs to be discontinued (Greenberg 1963).

In the antipesticide and antichemical environment of the 1960s, DDT became a focal point of attack, with Carson the ammunition and rallying point. The Environmental Defense Fund (EDF), an organization composed of scientists and lawyers formed in 1967, produced increasing evidence of the lethal effects of OCPs on bird populations and successfully petitioned the government to ban the chemicals altogether. DDT use was duly suspended (*Time* 1971). Accusations of bias created such controversy that the Environmental Protection Agency (EPA) was forced to convene seven

months of hearings from 1971 to 1972. In 1972, the EPA finally banned all uses of DDT barring a few public health ones, and even those only under stringent conditions. The lawsuits from the DDT firms began; countersuits from the EDF followed, one seeking a reversal, and another a total prohibition, until the Supreme Court came down on the side of the EPA in 1973 (EPA 1975). After the decision, DDT was used only in exceptional public health cases, such as outbreaks of potentially epidemic-causing pests, like an outbreak of fleas in 1979.

One of the major paradoxes of US biosafety and industrial regulations is that the US government bans certain dangerous chemicals and pharmaceutical drugs and institutes recalls of automobiles and other products at home, but allows US companies to continue making and exporting such dangerous products and exposing countries of the Global South to them. Also striking is the fact that the United States has, for pragmatic reasons in the national interest, refused to sign the Stockholm Convention on Persistent Organic Pollutants, and, after the domestic ban, still allow its companies to make these chemicals and export them for use outside the United States despite their known health and environmental effects (“Report of the Expert Group” 2010).

In the aftermath of the DDT ban, production for the Global South market continued at an average of three hundred tons of poison a year, in response to the pestiferous mobilities of *mhesvi*, *hutunga*, and other insects (USDHHS 2002). Twenty-six countries—among them Cuba, Singapore, Chile, and Korea—had banned DDT by 1986. Over 170 countries later ratified the Stockholm Convention, which became international law in 2004 and limited DDT only to WHO-approved *hutunga* and *mhesvi* control (UNEP 2001).

The second OCP is lindane, also known as Gamatox or benzene hexachloride (BHC) in Southern Rhodesia, but more appropriately as hexachlorocyclohexane (γ -HCH). *Lindane* is a neurotoxic organochlorine that interferes with the central nervous system, the liver, and the kidneys in *mhuka*. Lindane was registered in the late 1940s. In *zvipukanana*, it kills through skin and egg contact and absorption and through ingestion. Lindane first acquired global “fame” as a pesticide used against *mhesvi* and *hutunga* in Africa in the 1950s. However, the OCP was also lethal against sucking and biting pests, grain sores, soil pests like fleas, beetles, and mushroom fleas, and *zvipukanana* that attacked crops in the soil (Kumar and Kumar 2007, 2). It was considered deadlier to *zvipukanana* than vertebrates, although

severe exposure in *vanhu* resulted in nausea, vomiting, diarrhea, muscle fibrillation, tremor, and convulsions. BHC also damaged tissue in testicles, kidneys, skin, and liver in *mhuka* (Videla, Barros, and Junqueira 1990).

Lindane was first synthesized in 1825 by the English scientist Michael Faraday (1791–1867) and is named after Teunis van der Linden (1884–1965), the Dutch chemist who first isolated and described γ -HCH in 1912. Only in 1942 were its pesticidal properties first discovered; thereafter, Imperial Chemical Industries Ltd (ICI), the largest manufacturer of chemical products in twentieth-century Britain, started manufacturing it for wider-scale use. Based out of London, from ICI's formation in 1926 the company specialized in chemicals, explosives, fertilizers, pesticides, dyestuffs, paints (specifically the brand Dulux, dominant in southern Africa), the cloth-making fibers Terylene (also called *tererini* among *vatemala*) and nylon, and nonferrous metals. In World War II, ICI participated in Tube Alloys, Britain's nuclear weapons program (Smith et al. 2008). Without doubt, the company's "gift" to Africa remained lindane, used to treat grain harvests against weevils and for mass killing of *mhesvi* and *hutunga*. In its long history, lindane was made in Britain, Europe, the United States, China, Brazil, India, and Russia (Commission for Environmental Cooperation 2006). Only in 2009 was its production and use in farming banned under the Stockholm Convention. In the United States, lindane continued to be manufactured by Morton Grove Pharmaceuticals, which as late as 2007 received a warning from the Food and Drug Agency (FDA) to correct information on its website that omitted and minimized the health risks of its products (CEC 2006; "Report of the Conference" 2009).

The last OCP, which was also used as an anti-*mhesvi* pesticide in Rhodesia from the late 1950s until independence, was *dieldrin*, a much more toxic organochlorine compound than DDT. It was first industrially produced as an alternative pesticide to DDT in Denver, Colorado, by the US company J. Hyman & Co. in 1948. In 1987, it was discontinued worldwide when the manufacturer canceled its registration. Alongside chlordane, aldrin, heptachlor, and endrin, dieldrin belongs to a family of chemical *chepfu* called *cyclodienes* that are "less acutely toxic but of greater potential for chronic toxicity than the organophosphate and carbamate insecticides" (Allen et al. 1979a, 518).

Like DDT and lindane, dieldrin killed *zvipukanana* through skin contact and ingestion of contaminated matter. This OCP was widely used in agriculture and insect pest control from the mid-1940s to mid-1960s, but, like DDT, its persistence in the environment would lead to its termination

(Boryslawsky et al. 1985). Dieldrin was named after the Diels-Alder reaction process, through which norbornadiene and hexachlorocyclopentadiene are synthesized into a chemical pesticide. It is extremely persistent in the environment and does not easily break down—again like DDT and lindane, a characteristic that made it effective as a pesticide but also dangerous. Dieldrin was used against parasitic *zvipukanana* like termites, blowflies, ticks, and lice, and widely in cattle and sheep dips, to protect fabric from moths, beetles, and carrot and cabbage root flies, and as seed dressing against wheat and bulb fly (Kumar and Kumar 2007, 2). Although dieldrin is now banned in most of the Global North, aldrin continues to be used as an anti-termite in most of Africa.

The Coming of OCPs to Southern Rhodesia

OCPs arrived in eastern and southern Africa right after the end of World War II. The East African Tsetse Research Organization conducted experiments to find out how best to use OCPs to kill *mhesvi*. The first involved finding out if the *chipukanana* could die when exposed to OCPs through skin contact. In this investigation, the pesticide was applied directly on the skin initially, and then on traps. The answer, contained in a report in 1947, was affirmative: OCPs could kill *mhesvi* through skin contact (Vanderplank 1947). These experiments have already been discussed in chapter 4 alongside other traps and need no further mention here. The second experiment involved ground and aerial spraying of the insect's hides, breeding places, and shelter. This is our concern in this and in the next two chapters.

The general concept underpinning OCP use remained the same, however; the insecticide worked through a residual killing effect built up through a lethal deposit of sprays or dust, or aerosol smokes, fogs, or fine sprays. At least until 1951, residual and aerosol spraying were not combined. Spraying was divided into two types depending on the killing effect. The *residual killing effect* was achieved through selective application to vegetation from the ground using sprays, non-selective application to vegetation from the air with sprays, impregnated screens, dusts, and bait animals sprayed with insecticide. The *immediate killing effect* was achieved using smokes from generators, aerosol fogs from the Todd Insecticidal Fog-Producing Apparatus (TIFA), and aerosols and finely atomized sprays from *ndege* (aircraft).¹ Residual killing using Four Oak machines had been tried on islands in Lake Victoria against *mhesvirupani*. DDT and BHC oil solutions were applied initially, but the residual effect was lost quickly because of absorption by

leaves. Later, DDT and BHC emulsions were used with a 98 percent kill rate during the peak *zvukukwa* period. However, this was a method ill-suited to *mhesvirutondo*, a savannah type ranging all over that could only be sprayed in its resting and breeding sites, along the game tracks, and at waterholes. Nonselective application on four Lake Victoria islands had shown that canopy and unpredictable meteorological conditions “made the difficulties of achieving anything approximating a complete kill as difficult as they could be.”²

In a bid to find a less laborious method than spraying by hand, “impregnated screens” were stationed at likely places on Lake Victoria and sprayed with a weekly dose of DDT and BHC. The 50 percent success rate, already unimpressive in *mhesvirupani*, was declared virtually useless against *mhesvirutondo*, for which stationary screens and traps were unattractive. Nonetheless, it could still be useful against the thicket-favoring *mhesvirupani*. DDT and BHC dusts had been used in the mountains and broken areas of KwaZulu unsuited to fixed-wing *ndege*; hence, they were sprayed by hand and with mechanically operated dusters. However, only DDT ended up being used because BHC “proved irritating to the operators.”³

By 1950, DDT had already begun to be sprayed indoors in both “European areas” and “African areas” against malaria in the Mazowe area. DDT was used in the former, whereas BHC was deployed for spraying huts (cottages and compounds)—66,712 huts of *vashandi vatema* (black workers) in total—and barring the “objectionable odour,” it had been very effective. Interestingly, the archives show *vatema* pleasantly surprised by the mass destruction of *hutunga*, *zvikwekwe* (ticks), and *mapete* (cockroaches; Alexander and Ranger 1998, 209). On the face of it, a Daniel Headrick-style “tools of empire” scenario, in which *ruzivo rwevatema* were enabling *vachena* to colonize the territory, seemed to be under way.

Not so with *mhesvi*, at least initially. The most intriguing aspect of residual *mushonga* involved bait oxen dipped or sprayed with *mushonga*. Both at Shinyanga in Tanzania and in KwaZulu, such *mombe* were driven everyday into a small block of bush infested with *mhesvirupani*. The Shinyanga experiment failed because *mombe* were driven in after and taken out before *mhesvi*'s feeding times (early morning and evening, respectively) to save them from marauding *shumba*. It is possible that the experiment would have succeeded against *mhesvirutondo*, which fed by day. The South Africa Division of Veterinary Services' KwaZulu experiment succeeded against *mhesvirupani*, but the residual films (DDT) were only effective for five to six days, then became harmless. A Uganda trial with BHC had shown that “the dose needed to produce toxicity in the ... tsetse is too near that point

harmful to the animal."⁴ The South Africans switched to BHC because it was more economic (du Toit 1954). Rhodesia then invited the South Africans in 1953–1954 to help stop the *mhesvirutondo* advance in Hurungwe with BHC.

Dieldrin, from dieldrex 15 percent, product of Shell Chemicals of Central Africa (Vale 1968), inaugurated a new era in *mhesvi* control operations, "in which the basis of control was dependent on the residual properties of the insecticide."⁵ The first operation of this kind in Southern Rhodesia was conducted at Kapondo in the northwestern Hurungwe District in 1958. On February 6, an experiment was completed that tested the reaction of blowflies in the laboratory to dieldrin applied as a residual film and compared it to the effects of spraying residual films in the field.⁶ The conclusion was that "a 4% concentrated dieldrin applied as a residual film is the most efficient in causing death provided the flies are exposed to it for more than 0.5 minutes."⁷

The dieldrin experiments of 1958 were followed with another hugely successful one in the Maseme River drainage area of Binga in 1960. The method used was taken from west and east Africa, and involved ground teams using pneumatic or motorized knapsack sprayers to apply *mushonga* to dry-season resting places and refuge sites of *mhesvi*. To succeed, it was critical to spray *mushonga* before the extreme dry season began—that is, before the end of September. Thus, all ground operations were conducted from the beginning of June to the end of September so that "the insecticide used ... remain[ed] strongly lethal for a minimum of six months after being placed in position."⁸ Successful ground application of dieldrin on a selective basis relied on the theory that during the hot, dry season, *mhesvi* concentrated in areas of evergreen vegetation to survive. These habitats were riverine fringes, vlei edges, and hills. *Mhesvi* retreated from open woodland to the shade and humidity of the forest's edges and riverine thickets, making them the perfect foci for selective spraying.

The success of the Maseme and Hurungwe experiments paved the way for the extension of 3.1 percent dieldrin spraying in all tsetse control areas of Southern Rhodesia until 1967, when the move to the much cheaper 5 percent DDT was made. The switch "was only made after the longevity of the residual properties" of DDT had been demonstrated in the baking-hot conditions of the Zambezi valley. The *mushonga* remained effective "well in excess of eight months and, in fact, DDT proved to be superior to dieldrin, which was being tested simultaneously." Comparatively so much cheaper was DDT that "it became possible to operate over very much more extensive

areas of country than had hitherto been possible, which went a long way to overcoming the problem of subsequent reinvasion.”⁹

In 1970, tests ongoing at Rekomichi Research Station since 1969 sought to establish whether poles treated with white DDT attracted more or fewer flies compared to those treated with gray DDT or to untreated trellis poles. The trial was inconclusive. Experiments also switched to finding the most effective spraying techniques. Further trials were conducted on whether a sticker might be used to prevent deposits of DDT WP from being washed away by rain. In October 1972, baobab and mopane trees were sprayed with several formulations of 5 percent DDT WP suspension and the sticker, but it made little to no difference. Trials were continued in 1973 using stronger DDT concentrations.¹⁰ In 1974, the experiment was discontinued altogether because the sticker proved to be unsatisfactory.¹¹

From 1971 to 1975, a total of 48,168 km² were sprayed with a total of 1.2 million kilograms of DDT. At the same time, on farms, farmers were using 1,470 kg/ha of the 75 percent wettable powder or 1,102 kg/ha of active ingredient, four times every season, year after year. The powder was also sprayed in foliage and fruit trees, for cutworm control in maize and tobacco, for stock-borers in maize, in vegetable gardens, and to control bollworm in cotton production. Most of the pesticide fell to the ground or simply drifted. The defenders of DDT use in *mhesvi*-control work pointed out that farmers were using a far higher DDT concentration compared to the “only” 5 percent DDT BTTC was using. Desmond Lovemore, assistant director of Tsetse and Trypanosomiasis Control, mounted a spirited defense of DDT in 1976–1978, citing the latest studies performed since 1970:

In tsetse control spraying, ... the minimal quantities applied are selectively placed, very often in situations where little wash can occur. It is also usual for there to be only one application ever to an area, although certain areas had to be re-treated several times over successive years before elimination was eventually achieved. ... 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. ... It is also noteworthy that in Nigeria where DDT has been used very extensively in tsetse control operations, in fact, probably very much more so than in Rhodesia, no serious “side-effects” have been noted.¹²

For these reasons, Lovemore remained adamant that DDT would continue to be the preferred choice for *mhesvi* control operations. However, concerns about its use could no longer be ignored.

In the late 1970s, therefore, the Tsetse Control Branch sought alternatives to 5 percent DDT, partly for economic reasons (it was too expensive) but also in response to public (white) outcry against DDT. The branch focused on two chemical alternatives. One was to reduce the strength of DDT from 5 percent to 2.5 percent, based on what other African countries were doing and on research at Rekomichi. In general, the research found “very little difference” in overall effect between 5 percent and 2.5 percent mixes. Flies lived marginally longer when sprayed with the latter, but still died; invaders were indeed caught, but “did not apparently survive long enough in the 2.5% block to re-colonise it.” Although investigators were optimistic, the experiment did not conclusively resolve the question of whether 2.5 percent was adequate or equivalent to the usual 5 percent, precisely because the two blocks sprayed with each dosage had different features and “required different treatment techniques.”¹³

Sounding tentative and guarded, the director of Veterinary Services concluded in his annual report:

It seems there are grounds to reduce the concentration of the suspension used to some degree, provided planners ensure all likely habitats are well treated—the objective of good planning anyway. If by reducing the concentration it is necessary to raise the application rate per unit of area, there may be no benefit in terms of active ingredient dispersed. In this trial it did seem that the lower amount of active ingredient was adequate, so routine planning principles as applied by locally trained glossinologists may not have to be modified to compensate for a decrease in concentration. It is important to ensure this point, for, if indeed a weaker suspension does require a wider distribution to be effective, then other significant cost factors such as transport and labour, currently running at approximately 34% and 15% of total cost respectively, will be elevated!¹⁴

The second move was toward replacing DDT altogether with a rapidly degradable substitute. In 1973, five years after DDT replaced dieldrin as the first-line *mushonga* against *mhesvi*, the BTTC commenced experiments to compare the effectiveness of DDT and endosulfan. The latter *mushonga* was aerially sprayed using an ultralow volume of 20 percent endosulfan, applied to a 261 km² woodland infested with *mhesvi* in Chirisa Game Reserve. Six cycles at intervals of lengths between seventeen and twenty-two days and approximating to seven liters per km² were applied between June and November. The results were impressive; 99 percent control was obtained. The tests continued into 1975, but the final lines of Assistant Director

Desmond Lovemore's 1974 report are instructive: "Effects of the insecticide on a variety of insects, fish, mice, frogs, and geckoes were also studied. All test creatures, except the fish, which were rapidly killed (but their situation was highly artificial and exposed), appeared unharmed."¹⁵

Even in the chemical-intensive agricultural sector, the importation of three hundred tons of DDT for the 1977–1978 growing season belied an already unfolding shift to the more readily degradable OCP, endosulfan. Until then, trials to determine the viability of endosulfan as an alternative had proved it to be not persistent enough and more expensive.¹⁶ The ultralow volume (ULV) application of *mushonga* using fixed-wing *ndege* operating at night was still at the experimental stage, "some years" away from being perfected enough to replace DDT (Chapman 1976). Even then, it would only complement ground spraying with DDT in areas where selective application was unavoidable.¹⁷