RIVERS STATE UNIVERSITY OF SCIENCE AND TECHNOLOGY,

NKPOLU, PORT HARCOURT, NIGERIA.

AN ENTOMOPATHOGENIC FUNGUS, *HIRSUTELLA THOMPSONII* FISHER (FUNGI IMPERFECTI) AS A POTENTIAL BIOLOGICAL CONTROL AGENT OF *MONONYCHELLUS TANAJOA*. BONDAR (ACARI: TETRANYCHIDAE).

BY

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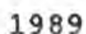
THESIS SUBMITTED IN PARTIAL FULFILLMENT OF THE THE REQUIREMENTS FOR THE AWARD OF THE DEGREE OF MASTER OF PHILOSOPHY IN APPLIED ENTOMOLOGY.



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DECLARATION.

I, Benson Odongo, hereby declare that, the work presented in this thesis is my own and has not been submitted for a degree in any other University; it is original except where indicated otherwise and in which case full references are given.

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We certify that this work has been supervised by us.

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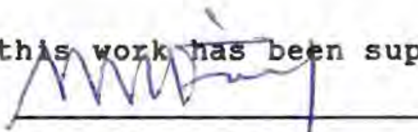

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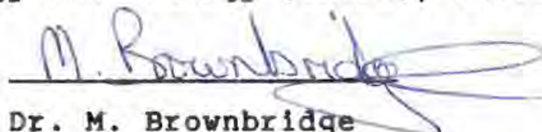

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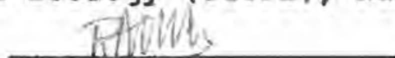

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Abstract

The infectivity of *H. thompsonii* on cassava green mite, (CGM), *M. tanajoa* was determined in laboratory, cage and field experiments.

In the laboratory, eggs, larvae and adult females were sprayed with suspensions of the fungus at concentrations ranging between 9.8×10^5 to 1.0×10^8 conidia per ml (CPM). Between 3 to 21% egg hatch reduction was recorded from *H. thompsonii*. treatment compared with the control. Phase contrast micrographs did not show that the fungus grew onto the surface of, or, penetrated into the eggs. Significant mortality (up to 34%) was recorded from *H. thompsonii* treated CGM females. The number of eggs laid per CGM female treated with the fungus was significantly lower than the number laid by the control batch. Death of infected female mites mainly occurred between 3 to 6 days after infection in the laboratory. Within this time, mite cadavers were observed to undergo some morphological changes, e.g body coloration from creamy appearence through brown, dark-brown and development of fungal growth; body became turgid, and then broke open and rapidly shrank to disappearence; they were invariably attached to the substratum; fungal mycelial penetration into the mite tissue and their conidiation could be demonstrated from 72 hours after treatment. CGM larvae were not appreciably killed by *H. thompsonii*.

In potted experiments, the number of eggs and live mites on the leaves were reduced following application of the fungus. The protection given was slightly better than those from *Amblyseius teke* (a phytoseiid mite predating on CGM) introduction.

In the field aqueous suspensions of *H. thompsonii* conidia at a concentration of $8.0x10^5$ and $3.7x10^5$ CPM were applied to *M. tanajoa* pest on field cassava at the peak of pest infestation, which occurred at the driest and hottest period of the season. The treatment reduced the number of egg and live CGM by 63 and 77% respectively. The damage symptoms caused by the pest was also reduced. This was particularly evident from the second week after treatment application.

Rainfall was a density independent mortality factor. Heavy precipitation led to a sharp reduction crash in mite numbers and subsequent levels of pest damage.

It is therefore shown here that timely application of *H. thompsonii* in coordination with other control or mortality factors, e.g under intergrated pest management practices, could greatly help reduce the level of pest damage. Pest distribution was most concentrated on the upper, younger leaves between leaves 1 to 5. The numbers fell with increasung age of the leaves. The effects of temperature, rainfall totals and number of rainy days per month on pest population and damage syptom indices are discussed.

DEDICATION.

To

Apap, Ladit Nacan Owiny

and

Aya Imat Loi Owiny

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GENERAL INTRODUCTION

The major staple and food crops in sub-saharan Africa consist of about 53% cereals, 31% roots and tubers, 6% grain legumes, and 5% groundnuts, plantain and bananas (Paulina and Yeang, 1981). Irish potatoes, sweet potatoes, cassava and sugar beets are among 15 major crops of the world that contribute over 75% of the world per capita daily calorie consumption and more than 60% of the per capita daily protein supply (Okigbo, 1986).

FAO (1985) yield records of root and tuber crops showed that, generally, these were lower in Africa as compared with the world average. Insect and mite pest attacks are major contributors to the low production of these crops.

In tropical Africa, the cassava crop, (*Manihot esculenta* Crantz) has suffered from attacks by new pests which have been introduced into the region within the last two decades. The new pests are the cassava mealy bug , *Phenacoccus manihoti* and the cassava green mite (CGM), *Mononychellus tanajoa*.Bondar (Acari: Tetranychidae). These species occur in very low numbers in their native home, South America, and

are not pests there. *P. manihoti* was first recorded on cassava in Zaire by Hahn and Williams (1973) and Sylvestre (1973). *M. tanajoa* has spread to nearly all the cassava growing regions of Africa from Uganda where it was first observed in 1971 (Nyiira, 1972; Lyon, 1973), probably through the accidental introduction of infected cassava cuttings. The pests now threaten cassava production in 31 out of 34 countries in the African cassava belt, causing yield losses of up to 80% (Herren, 1987). Furthermore, the two pests were recorded occurring together in 24 countries and it was predicted that the two pests would probably be present in all the cassava growing areas within the next 2-3 years.

Pest management studies on *P. manihoti* (Mat. Ferr.) (Homoptera: Pseudococcidae) have indicated that a parasitic wasp, *Epidinocarsis lopezi* (De Santis) (Hymenoptera: Encyrtidae) which is a natural enemy of the pest, has a high potential for the control of this pest species (Lema and Herren, 1985; Neuenschwander and Madojemu, 1986; Neuenschwander and Sullivan, 1987). However, a similar solution has not yet been developed for *M. tanajoa*.

M. tanajoa is one of more than 50 spp. of tetranychid mites identified by Belloti and

Schoonhoven (1978), attacking cassava in about 60 countries worldwide. *M. tanajoa* and two other species of the spotted mite complex, *Tetranychus cinnabarianus* (Boisduval) and *T. utricae* (L) are the most common mite pests of cassava. The complex has a wide host range and is found throughout the tropics (Byrne *et. al.*, 1983). Other species of mites found occasionally on cassava include *Eutetranychus banksi* (Andrew and Poe 1980), *E. orientallis, Oligonychus biharensis* (Lal and Pillai, 1976) and *O. peruvianus* (Salas, 1978; Byrne *et al.*, 1983).).

Yield losses attributed to *M. tanajoa* on tuberous roots, leaves and stems have been widely cited in various publications (van de Vrie *et al*, 1972; Nyiira, 1976; Shukla, 1976; Maini and Lal, 1977; Nyiira, 1978; Doreste and Apote, 1979; CIAT, 1980; Leuschner, 1980; CIAT, 1981; Byrne *et al.*, 1982; CIAT, 1983; CIAT, 1984; Odongo, 1986). Most of the observed losses were significant and have prompted investigations into ways and means of reducing the population and damage of the pest to economically acceptable levels. As a result, about 50 chemical compounds have been tested on the pest in Latin America, Africa and India (Byrne *et. al.*, 1983). Unfortunately, Helle and van de Vrie (1974) observed that mites have developed resistance to

most of the tested chemicals. Furthermore, nearly all of the chemical insecticides and the equipments required for their application are usually too expensive for small scale cassava growers to buy. Yaseen (1977) further cautioned that beneficial insects as well as other non-target organisms were adversely affected by the broad-spectrum, non-selective pesticides which normally dominate the present market.

Many other control methods have been tested in order to minimise damage caused by *M. tanajoa* on cassava. These have included cultural practices (Leefmans, 1915; Bondar, 1938), and selection and development of cassava strains for host plant resistance (IITA, 1978; CIAT, 1980; Ezumah, 1980; CIAT Ann. Rep. 1981; CIAT Ann. Rep. 1983; IITA Ann. Rep. 1983; Ndyiragije, 1986).

Research efforts into the identification and assessment of biological control agents for *M. tanajoa* have revealed 17 candidate species in 7 insect orders, and, various spiders (Yaseen *et al.*, 1982; Markham *et al.*, 1986). Unfortunately, these predators have been found to contribute little to the natural regulation of the pest. Efforts have thus been diversified to investigate the possible use of disease micro-organisms as single component control agents in

the field, or, and more preferably, as a part of an intergrated pest management programme.

The term 'disease' according to Burges and Hussey (1971) means a departure of the insect from a state of health. This phenomenom was first noticed among domesticated insects. The first recorded work on an insect disease was by Aristotle (between 384 to 322 BC- Quoted from Clerence, 1955) (Reported in Burges & Hussey, 1971) who observed that bees suffered from disease. Poinar and Thomas in 1982 regarded a 'disease' as a state in which a body, or a tissue, or organ of the body, is disturbed either functionally or structurally or both. Two types of insect diseases were distinguished (i) those that were non-infectious, being caused by abiotic factors like temperature, chemicals and injury; and, (ii) those diseases that were described as being caused by pathogens. The relevant points in both definitions are that a 'disease' is caused by a pathogen which attacks and brings about functional or structural disturbances on the infected part of the body, tissue, or body organ.

An attempt to isolate fungal pathogens attacking cassava green mite (CGM), was first made by Odindo and Bartkowski (Pers. Comm., 1987; ICIPE, Ann. Rep., 1986). The fungi were isolated

from infected CGM cadavers collected from 24 cassava fields located in various regions of Kenya. Of the 24 isolates recovered, 7 of them (coded as MP9, MP17, MP20, MP21, MP22, MP14 and MP19) were shown to cause mortalities in treated mites between 5.3 and 73.3% (ICIPE Ann. Rep., 1986). Isolates MP14, and MP19 belong to the genera of *Entomophthora* (= *Neozygites*) and *Hirsutella* respectively. The remaining 17 isolates were discarded as they were identified as saprophytic fungi, and therefore unable to actively attack living mites.

Investigations were undertaken in an attempt to establish the effect of *H. thompsonii* on *M. tanajoa* through laboratory bioassays. Further tests were carried out on populations of *M. tanajoa* infesting cage and field cassava plants to determine if the fungus could be used as a mycoinsecticide. The effect of environmental conditions on the virulence of the entomogenous fungus was also considered. In brief, the objectives of the study were

1). To study the effect of *H. thompsonii* on various developmental stages of *M. tanajoa* in the laboratory;

2). To understand the mode of action and symptoms of *H.thompsonii* infection on

M. tanajoa (Laboratory Studies);

3) To compare the effect of *H. thompsonii* to that of *Amblyseius teke* (a phytoseiid predatory mite) in controlling *M. tanajoa* on cassava potted plants;

4) To establish the pathological efficacy of *H. thompsonii* on *M. tanajoa* infecting field c assava.

LITERATURE REVIEW.

2.1.Biological Control Using Microorganisms.

There has been a resurgence of interest in work on microorganisms during the past 15 to 20 years mainly as an attempt to develop alternative agents to chemical insecticides for pest control (Hall and Papierok, 1982).

Cook (1980) observed that, in general, opportunities for biological control using insect pathogens are rapidly increasing due to (i) the recent changes in genetic engineering techniques; (ii) the discovery of special mechanisms by which one organism may influence another; (iii) the new technology on mycorrhizae; and (iv) techniques for adjusting environmental parameters.

Work on potential mycoinsecticides is discussed below. Particular emphasis has been placed on areas relevant to the understanding of the entomopathogenic fungus, *H. thompsonii*.

2.2. Mycoinsecticides.

The potential for using fungi as mycoinsecticides was first recognised by Agostino Bassi in 1835 (Reported in Burges and Hussey,

1971) who observed that *Beauveria bassiana* (Balsamo) Vuillemin caused muscardine disease in silkworms. In 1874, Le Conte (Reported in Hall and Papierok, 1982) had independently suggested the use of fungi as biological control agents. Shortly afterwards, Metschnikoff (1879), and Krassilsctchik (1888), mass-produced a fungus, *Metarrhizum anisopliae*, for the first time for the control of larvae of the grain weevil.

Entomogenous fungi attack a variety of terrestrial and aquatic insects as well other invertebrates and in some cases even vertebrates (Poinar et *al*., 1982). According to Ferron (1979), most of the fungi infecting insects and mites are found in the following classes: Fungi Imperfecti (=Deuteromycetes), Zygomycetes, Oomycetes, Trichomycetes and Chytridiomycetes.

2.2.1 Fungi Imperfecti.

This class of fungi contains more than 150 known species which attack many insects and arachnid species (Samson, 1981). They have a wide geographical distribution, and are generally considered to be easy to culture (Hall and Papierok, 1982). Most species sporulate on dead insects but *Verticillum lecanii* can even sporulate on living insects. *Beauveria bassiana* and *B.*

bringniartii attack many insects of economic importance including Lepidoptera, Coleoptera, and Diptera, particularly the mosquitoes where they have about 500 host species (Charles, 1941; Gosswald, 1938; Lipa, 1963; Muller-Kogler, 1965; Kalvish and Kikharchuk, 1974), and are also known to attack assasin bugs in the insect order Heteroptera (Parameswaram *et al.*, 1977). *M. anisopliae* and *M. flavoviridae* infect more than 200 species of insects (Veen, 1968), in the orders Coleoptera, Lepidoptera, Orthoptera, Hemiptera (Muller-Kogler, 1965; Veen, 1968), Homoptera (e.g. aphids, Foster (1975)), and Diptera (Saubenova, 1976). Another species of fungus in this class is *Nimuraea rileyi*, an important pathogen of lepidopterans e.g. *Heliothis* species and *Spodoptera littoralis* (Getzin, 1961; Ignoffo, 1981). *N. rileyi* is mainly found in the tropical and sub-tropical regions (Hall and Papierok, 1982). About 40 species of *Hirsutella* are known to infect many species of insects (Mains, 1951; Samson, 1981). *H. thompsonii* attacks eriophyid mites (McCoy, 1981). *V. lecanii* (=*Cephalosporium lecanii*) attacks aphids, scales, and white fly (Gams, 1971; Hall, 1981).

Other entomopathogenic fungi of economic importance are largely distributed in 4 more

classes of fungi: Zygomycetes (Thaxter, 1888; Weiser *et al.*, 1964; Gustefsson, 1965; Goldberg, 1969; Missoiner *et al.*, 1970; Shands *et al.*, 1972a; Dean *et al.*, 1973; Dedryver, 1978; Page, 1978; Papierok, 1978; Dedryver, 1980; Remaudiere *et al.*, 1981; Hall and Papierok, 1982); Oomycetes (Glenn and Chapman, 1978; Fetter-Lasko, 1980; Federici, 1981; Washino, 1981); Chytridiomycetes (Couch *et al.*, 1963; Roberts, 1974; Bland *et al.*, 1981) and Trichomycetes (Farr *et al.*, 1967; Williams *et al.*, 1980).

2.3. Mechanisms of Infection.

For terrestrial insects, fungal invasion usually occur directly through the cuticle, although penetration via other routes is not uncommon e.g the alimentary tract (Veen, 1968; Broome *et al.*, 1976; Schabel, 1976; Kish and Allen, 1978) or the respiratory system (Clark *et al.*, 1968; Hedlund and Pass, 1968; Pekrul and Grula, 1979). Non-preference of penetration site has also been observed (McCauley *et al.*, 1968; Fargues and Vey, 1974; Brobyn and Wilding , 1977; Mohamed *et al.*, 1978; Pekrul and Grula, 1979). It is also reported that the head capsule is less

frequently attacked than the rest of the body (Schabel, 1978).

It is at the cuticle level that host specificity may be manifested. This has been demonstrated by *Neozygites fresenii* conidia which germinate on the cuticle of susceptible and resistant aphid hosts but only infect the former (Brobyn and Wilding, 1977).

The outermost layer of the cuticle, the epicuticle, contains lipids, which appear to have antifungal properties (Sussamann, 1951; Koidsumi, 1957; Latege, 1972). The rest of the cuticle consists of the proteins such as chitin, and also lipids and phenolic complexes (Richards, 1978). Entomopathogenic fungi are known to produce exocellular proteolytic, chitinolytic and lipolytic enzymes *in vitro* (Huber, 1958; Claus, 1961; Gabriel, 1968; Kusera *et al.*, 1968; Samsinakova *et al.*, 1971; Latege, 1974; Grula *et al.*, 1978; Paris, 1980; Hall, 1981) and several histological studies strongly suggest that enzymatic activity occurs during and facilitates conidal penetration of the cuticle (Brobyn and Wilding, 1977; Lambiase and Yendol, 1977; Grula *et al.*, 1978). Chmielewski et al. (1983) also demonstrated that host invasion by *Aspergillus* spp., *Metarrhizum* spp. and *B. bassiana* was

facilitated by enzymatic and mechanical action. Competition for food resources then occured between the pathogen and the host.

Once fungi have overcome the defence barriers, they may assume a different morphology. Blastospores (Fungi Imperfecti) may be formed, possibly in response to high carbon dioxide tensions (Hall and Latege, 1980); hyphal bodies (Entomophthorales) (Thaxter, 1888; Olive, 1906; Prasertphon and Tanada, 1968) or protoplasts (Entomophthorales) (Tyrrel, 1977) have also been observed. These stages probably enable the disease to be disseminated to other tissues, resulting in the death of the host (Hall and Papierok, 1982).

2.4 Disease Symptoms of Fungal Infections

The symptoms of a fungal disease in the citrus red mite, *Phyllocoptruta olivae*, attacked by *H. thompsonii*, range from sluggish movement of the infected pest, to body discoloration, from a lemon yellow coloration, to dark yellow and and eventually to brown (McCoy *et al.*, 1974; Muma, 1958; Burditt *et al.*, 1962; Baker and Neuzig, 1968). Discoloration of the integument may not, however, be a very reliable disease diagnosis because some infected mites do not change colour. Secondly, the citrus red mite usually acquires a

darker coloration with age (McCoy, 1978). Another important consideration in the diagnosis of infection symptoms of entomogenous fungi, is the position of the host at death, e.g. the host may be fixed to a substrate as with flies or grasshoppers attacked by *Entomophthora*, or the dead host can be relaxed on the ground (Poinar and Thomas, 1982). Body consistency may also be helpful in the diagnosis of various infections e.g whether it is hollow, cheeselike, or hardened.

2.5 . Epizootiology.

Fungal diseases can manifest themselves in natural insect populations in enzootic or epizootic forms (Hall and Papierok, 1982). The presence and development of the disease depend on numerous abiotic and biotic factors some of which are outlined below.

2.5.1 Abiotic Factors.

Relative humidity is a critical factor influencing the infectivity of entomogenous fungi. Saturated or near-saturated air, or a water film, is necessary for spore germination of most fungi (Hall and Papierok, 1982). In the case of Fungi Imperfecti, which are pathogenic to terrestrial insects, *in vitro* experiments have shown that a

minimum of between 92-93% RH is required for spore germination (Walsttad *et al.*, 1970; Ferron, 1981). Despite these rather stringent conditions necessary for the establishment of an entomogenous fungal infection, it has been shown that insects may support their own microclimate and that isolated insects can become infected in conditions of low humidity (Madelin, 1963; Moore, 1973; Ferron, 1977; Doberski, 1981).

In the field, factors likely to increase microclimate humidity include irrigation techniques (Evlakhova and Voronina, 1967; Wilding, 1981) and the density of the crop canopy (Tanada, 1963; MacLeod *et al.*, 1966). Wind tends to lower the microclimate humidity towards ambient (Hall and Papierok, 1982). Heavy rains can be undesirable, lowering the density of airborne conidia and washing spores off cadavers (Kish and Allen, 1978).

Temperature is another important factor affecting infectivity with different fungal species, or strains, having different thermic preferences. The optimal temperature requirements for Fungi Imperfecti (*Beauveria, Verticillium, Paecilomycetes* and *Nomuraea*) in the laboratory have been found to revolve around 20 to 25 ^{0}C (Getzin, 1961; Hall, 1980; Ferron 1981; Ignoffo,

1981). The range is between 25 to 30^0 C for entomogenous fungi *H. thompsonii*, *Culicinomyces clavosporus* and *M. anisopliae* (Latch, 1965; Sweeney, 1978; Hall and Espinosa, 1981).

Laboratory determined optima for growth or infection may not be the same in the field (Hall and Papierok, 1982). Temperature may disproportionately influence the behaviour of the host, e.g the rate of feeding or reproduction in aphids (Wilding, 1970; Hall, 1981) are closely related to the ambient temperatute. Also, for terrestrial insects, the temperature-humidity interaction is important in germination and cuticle penetration by the fungus, and consequently important in the infection cycle and spread of the disease (Voronina, 1971).

For practical purposes, therefore, application of fungal insecticides should be made, if possible, during cool and humid evening hours as opposed to hot and dry afternoon hours.

Certain biotic factors have been found to affect epizootiology in the field and include, for example, the mobility of the host insect (Shands et al., 1972b; Hall and Burges, 1979). Some terrestrial insects feed in moist microclimates on plants and are thus highly susceptible to fungal diseases (Hall and Burges, 1979; Dedryver, 1980).

A high reproductive rate and a resulting high host density promote the epizootiology of mycosis (McLeod *et al.*, 1966; Robert *et al.*, 1973; Wilding and Lauckner, 1974; Suter and Keller, 1977; Remaudiere *et al.*, 1981). Robert *et al.* (1973) showed that a critical concentration of a host inoculum also has to be present for the development of an epizootic. Furthermore, the manner in which the inoculum is distributed, and the overall distribution of the disease within the crop affects the level of pest control obtained in the crop (Keller and Suter, 1980; Remaudiere *et al.*, 1981).

2.6 Use of Fungi in Biological Control

Ecological studies made on epizootic or enzootic infections by fungi, indicate that fungi can be manipulated in two possible ways to control pests: (i) applied to achieve a 'knock-down 'effect e.g. in the fungi producing toxins; or when a comparatively long 'lag-phase' exists between fungal application and host death can be tolerated (Hall and Papierok, 1982); and (ii) where an insect pest species has a rapid reproductive rate, e.g aphids or mites, the fungus must be able to spread more rapidly than the pest

is able to reproduce, following application (Latege and Perry, 1980; Hall, 1981).

Other important factors when employing a biological agent for pest control are (i) the virulence of the pathogen to the target pest(s); and (ii) the safety of the pathogen to man and other mammals.

2.6.1 Virulence

Of all groups of micro-organisms, entomogenous fungi pose the greatest difficulties in bioassay of virulence. Delivery of infectious propagules to the assay host in a standardised manner is often difficult and the techniques involved are often laborious and time consuming (Hall and Papierok, 1982). More research is needed in this area although several assay systems have been developed (Hall, 1976; Wilding, 1976; Puttlet et al., 1976; Papierok and Wilding, 1979; Vandenberg and Soper, 1979; Fargues, 1981; Milner and Soper, 1981). The usual bioassay parameters are LC_{50}, LD_{50}, and LT_{50}- i.e the concentration, dose or time needed to kill 50% of test insects. However, these values alone do not always give results which are meaningful with respect to control in the field (Hall and Papierok, 1982).

2.6.2. Safety of Entomogenous Fungi.

Hall and Papierok in 1982, noted that several important regulatory agencies have recognised that microbial pesticides impose inherently different potential hazards, compared to synthetic chemicals. For fungi, these are mainly infectivity, toxicity and allergenicity. Among the entomopathogenic fungi, only *Aspergillus* spp. and *Conidiobolus coronatus* have been shown to be infective to mammals (Emmons and Bridges, 1961; Ignoffo, 1973) and only *Aspergillus* spp. have proved sufficiently toxic for an LD_{50} to be obtained. *B. bassiana* has been reported as causing allergenic reactions (Hussey and Tinsley, 1981). Tests to establish if the fungi caused irritation of the eye and skin, and mutagenicity effects, showed that *H. thompsonii* (Ignoffo, 1973; McCoy and Heimpel, 1980) and *N. rileyi* (Ignoffo, 1981) were safe, giving negative results in the tests.

2.6.3 Efficiency of Pathogens in the Field.

The muscardine fungi, *M. anisopliae* and *Beauveria* spp. are the most intensely studied entomopathogenic fungi (Hall and Papierok, 1982). The application of *B. bassiana*) blastospores at

10^{14} spores ha^{-1}. was able to control a Colorado beetle, *Leptinotarsa decemlineata*, population in Normandy (Fargues *et al.*, 1976). In the USSR, *B. bassiana* is applied as 'Beauverin' for the control of Colorado beetle at a rate of approximately $4x10^{13}$ spores ha^{-1} where the fungus is recommended for use with reduced doses of insecticides (Ferron, 1981), the concurrent application of the insecticides prompting a synergistic effect. *B. bassiana* is also used in China against the European corn borer, *Ostrinia nubilalis*, and the pine caterpillar (*Nephorettix* spp.) on rice and tea (Hussey and Tinsley, 1981).

M. anisopliae has been used against the rhinoceros beetle, *Oryctes rhinoceros*, (Latch and Fallon, 1976; Anonymous, 1978). The use of this fungus as an adjunct with a baculovirus has virtually eliminated this major pest of palm in Polynesia (Bedford, 1981).

H. thompsonii has for a long time been recognised as a natural regulatory agent of eriophyid mites and was first reported by Speare and Yothers (1924), and later by Yothers and Mason (1930). Fisher et. al. (1949) reported *H. thompsonii* to be epizootic on the citrus rust mite. Further reports on the pathogenicity of the

fungus on eriophid mites was made by Muma (1955), Burditt *et. al*. (1962) and McCoy (1981) among others. *H. thompsonii* is often responsible for the natural decimation of heavy citrus rust mite populations but not before crop damage occurs. Recognition of its potential against eriophyid mites has prompted tests in several localities, mostly on citrus. In Surinam, van Brussel (1975) controlled citrus rust mite, *Phyllocoptruta oleviora*, using a blended mycelial-conidial suspension even during the dry season. The bulk of the developmental work has been done in Florida (McCoy *et al*., 1971; McCoy *et al*., 1974; McCoy, 1978, 1981; McCoy and Couch, 1982) where the growing of citrus crops is a major industry. In the first series of experiments, the fungus was grown in liquid culture. After blending, the mycelia were sprayed with different adjuvants on to the plants. Heavy mite infestations on fruit and foliage were often reduced to low levels within two weeks, and maintained at low levels for six months to a year (McCoy and Selhime, 1974). Other mite species susceptible to *H. thompsonii* include *Eutetranychus banksi* (McG), *E. sexmaculatus* (Riley), *Panonychus citri* (McG), *Typhlodromalus peregrinus* (Muma et al., 1961), *Tetranychus* utricae. Koch (Gardner et al., 1982).

H. thompsonii also attack a number of insect species including the blueberry budfly, ***Aceria vaccinii*** (Keifer) (**Baker and Neuzing, 1968**) and **wasps, *Polistes olivaceous*** (Poinar **and Thomas, 1982**).

A number of other entomogenous fungi have been in use in the field. ***V. lecanii*** is used to control aphids, such as ***Aphis gossypii*** and ***Myzus persicae*** (Hall and Burges, 1979). ***N. rileyi* has** been used against ***Plathypena scaba*** (Hall and Papierok, 1982; Ignoffo, 1981) and *C.* ***tarsalis*** (Washino, 1981). ***Coelomomycetes stegomyiae*** has been shown to be pathogenic to ***Aedes polynesiensis*** (Laird, 1967; 1981) in the Pacific, and also to ***A. gambiae*** larvae (Muspratt, 1963).

3.0 GENERAL MATERIALS AND METHODS.

3.1. Effect of *H. thompsonii* on Developmental Stages of *Mononychellus tanajoa*: Laboratory Studies.

Adult female cassava green mite (CGM) *Mononychellus tanajoa* were picked from leaves of field cassava plants using a moist fine-pointed brush. The mites were transferred onto cassava leaf discs which were free from mite infestation. A cork borer of area 2.83 cm^2 was used to obtain the leaf discs-used for feeding the experimental mite population. The leaf discs were placed lower side uppermost onto moist filter paper (Whatman Filter paper, Qualitative no. 1), placed on cotton wool. These were carried in Petri dishes, which were floated on water in plastic trays (Figure 1). The water barrier prevented ants and cockroaches attacking the experimental material.

Depending on the nature of the investigation, the adult mites which were placed on the leaf discs were either discarded after 24 hours, leaving behind freshly laid eggs or subjected to a series of treatments as described below.

To obtain the *H. thompsonii* inoculum, pure cultures of the fungus were grown on potato dextrose agar (Figure 2). Two weeks old cultures had a grey-looking mycelial matting which lay thinly over the surface of the growth medium. Numerous conidiospores were normally found dispersed among the mycellia (Figure 3). The fungus was scraped off the

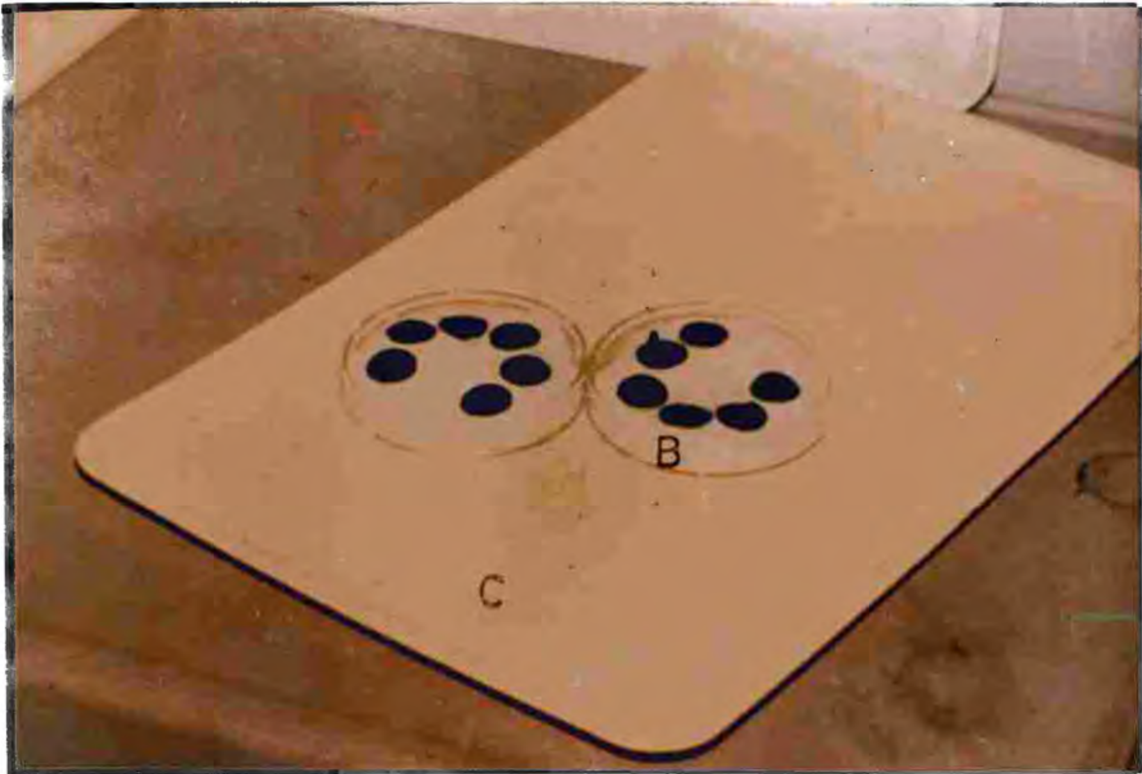

Figure 1. Experimental set-up for bioassay of *H. thompsonii* on *M. tanajoa*; A=Leaf disc for feeding mites ; B=Petri-dish; C=Plastic tray containing water.

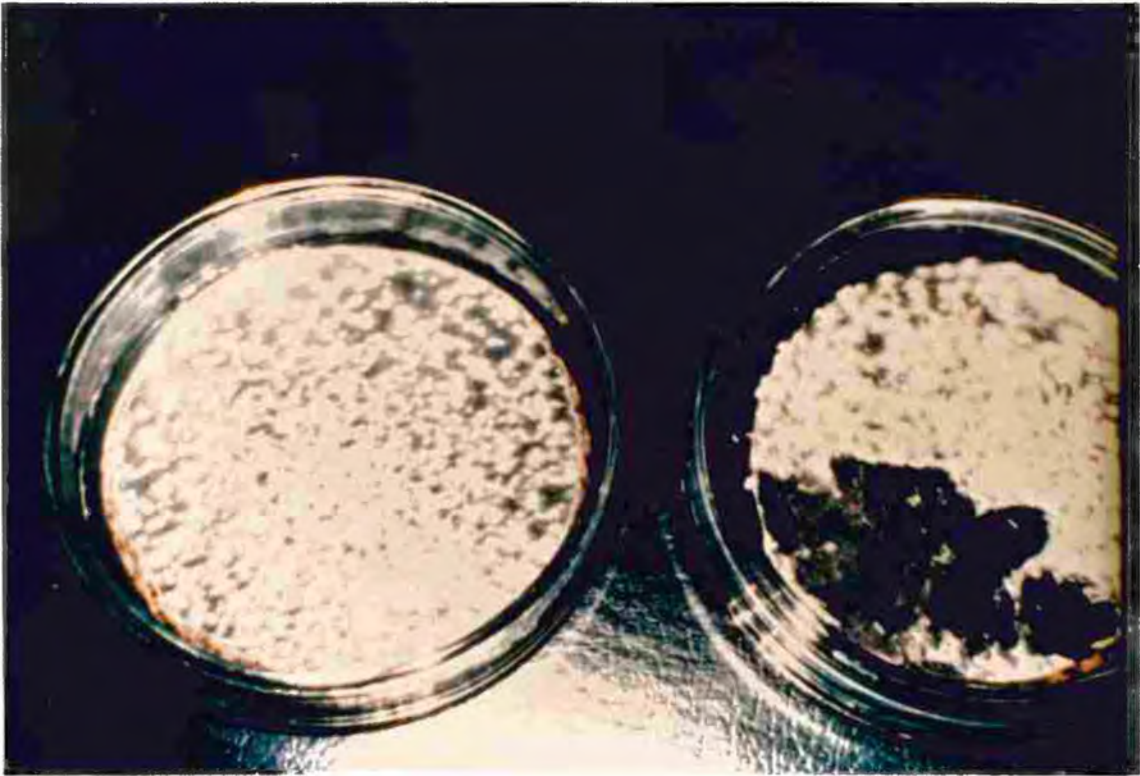

Figure 2. *H.thompsonii* culture in Petrish dishes (diameter=9.0 cm) in the laboratory.

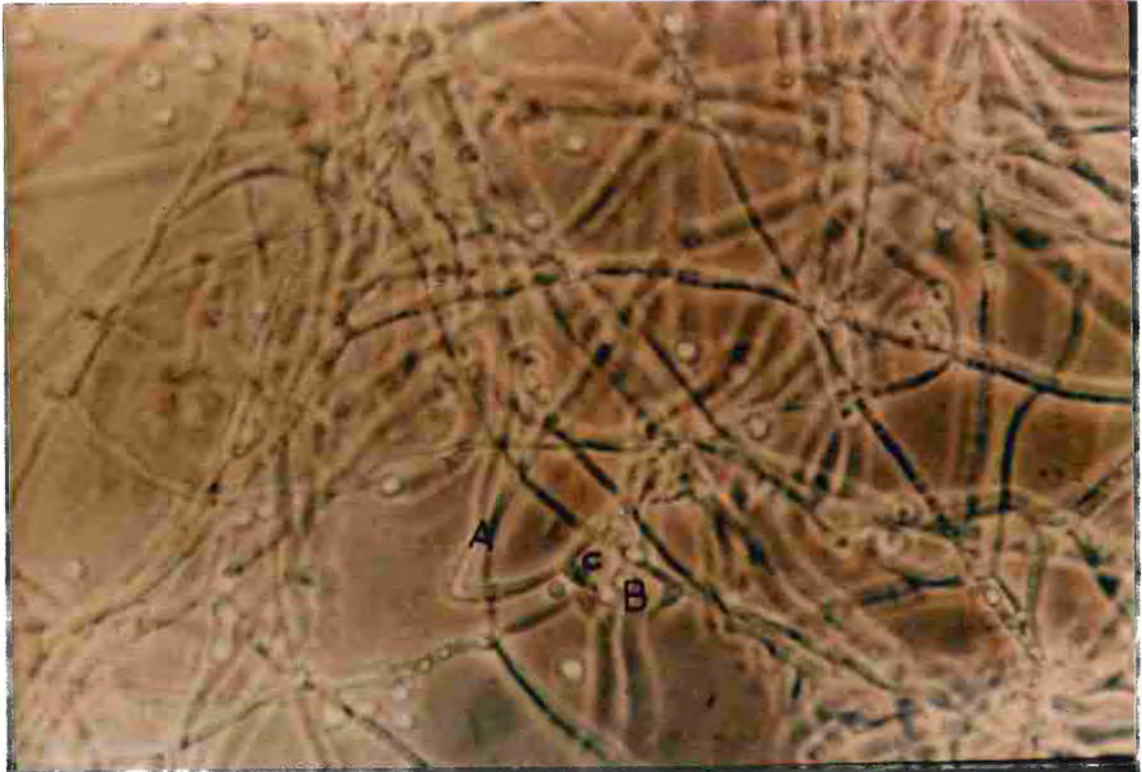

Figure 3. H .thompsonii mycelial growth (A); the infective units, the conidia (B) which are normally attached to phalides (C). (Phase contrast microscopy, (mag.=x1360).

medium using a sterilised scalpel blade, transferred to a glass mortar and ground into a homogenous aqueous suspension. The suspension was then filtered through layers of cheese cloth. The resultant suspension contained mainly fungal spores and fragments of fungal mycelia. The suspension was applied as a very fine spray onto the experimental mites, using a hand-operated laboratory sprayer (Figure 4) (Courtesy of J, Bartkowski).

H. thompsonii conidia concentrations (measured in number of conidia per per ml, (CPM)) were calculated from counts made on a hemocytometer viewed under a phase contrast microscope (Model Leitz Wetzlar ORTHOPLAN). The method of conidia spore estimation, originally developed by Cantwell (1970) for the estimation of *Nosema* sp. cells infesting bees, was adapted here for the computation of *H. thompsonii* conidiospores.

After treatment, the leaf discs, each of which represented one replicate, were allowed to dry .The Petri dishes were then covered to generate the high humidity necessary for the germination of the fungal spores. These were left for 24 hours. The petri dishes were then either put in a high relative humidity chamber, where a relative humidity of between 90 to 98% was maintained by an automatic hydrometer system, or, in a low humidity laboratory environment (R.H=75.5%; sd=5.4). Humidity chamber temperature varied between maximum 29.9^{0}C (sd=0.2) and minimum 17.6^{0}C (sd=0.9) while in the laboratory the

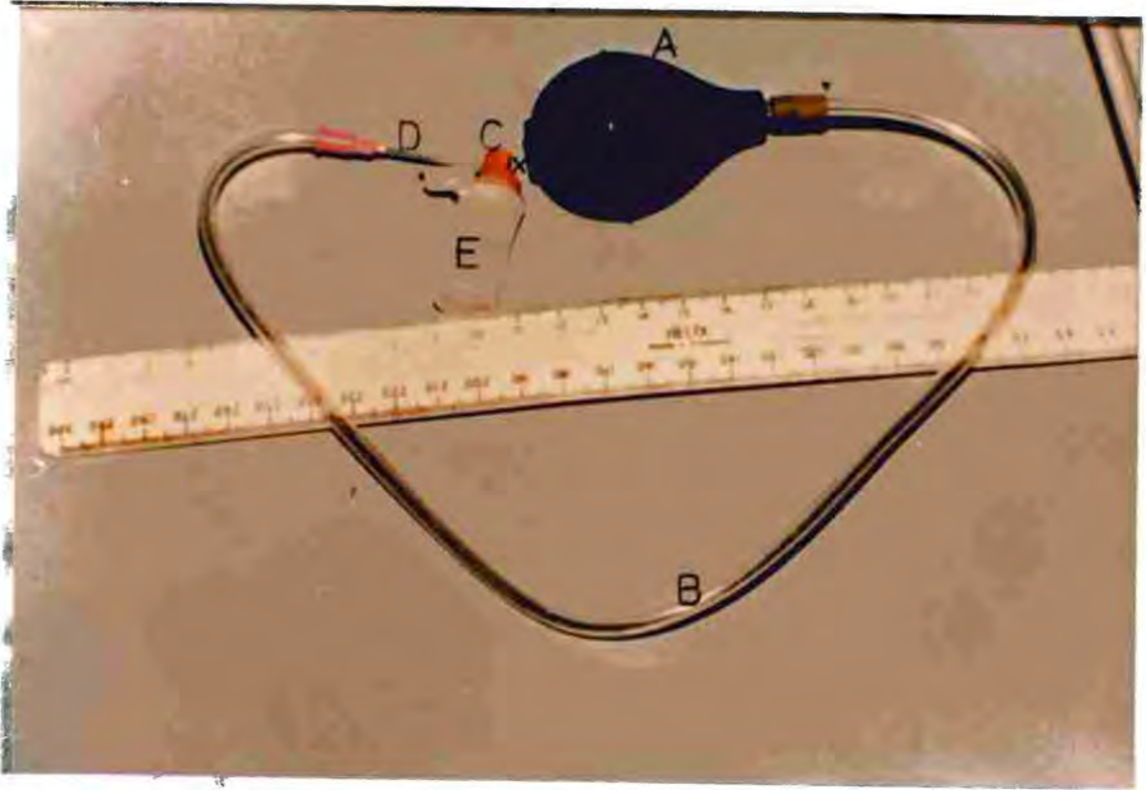

Figure 4. Laboratory sprayer used for application of H. thompsonii conidia suspensions on M. tanajoa, A=rubber bulb; B=rubber tube connection; C=rubber bung holding injection needle (D) and a second needle running vertically into plastic container (E); The two needles are in close proximity to each other at point X.

temperature fluctuated between 26.9^{0}C down to 13.5^{0}C (sd=9.3). The infectivity of the fungus on various mite stages was compared in both environments. Similarly, infectivity was compared between a growth chamber environment (RH =99%; temperature= 30^{0}C) and in the laboratory. The duration of the laboratory bioassays was between five to eight days.

The number and percent of larvae which hatched from the treated eggs; living and dead larvae and adult mites; and dead larvae and adult mite cadavers showing signs of *H. thompsonii* infection symptoms were recorded for the respective treatments.

The Statistics Analysis Systems (SAS) statistical package was used for all data analyses.

3.2 Mode of Action and Symptoms of *H. thompsonii* on Eggs and Mature Female *M. tanajoa*.

Female *M. tanajoa* were put onto the underside of cassava leaf discs according to the procedures described in section 3.1. After 24 hours, they were sprayed with suspensions of *H. thompsonii* conidiospores. The eggs which had been laid during this period receieved the same treatment. A high RH of about 100% was generated around the leaf discs by covering up the petri dishes. Micrographs and descriptions of stages of disease development were recorded.

3.3. The Effect of *H. thompsonii* on *M.tanajoa* on Cassava Planted in Pots.

Cassava cuttings were planted in well drained plastic buckets and regularly watered with tap water. The germinated plants were put in the sunshine and allowed to grow. When the plants were between one and half to two months old, they were artificially infested by placing old CGM-infected leaves, collected from the field, onto them.

Treatments were applied to the plants according to the following regimes:

1) *M. tanajoa* infested plants were sprayed with a suspension of *H. thompsonii* conidia;
2) A batch of phytoseiid mites, *A. tekae* were introduced onto cassava planys infected with *M. tanajoa*.
3) *M. tanajoa* infected plants were sprayed with laboratory tap water (Control).

Treatment 1. Pure cultures of *H. thompsonii* grown in the laboratory on potato dextrose agar were crushed down into homogenous aqueous suspension using a blender (Model: KENWOOD MAJOR). The suspension was later diluted as required, put into a hand operated knapsack sprayer (AKILI (Achelis (K) LTD.)) and applied onto the experimental plants. Late cool and humid evening hours were the preferred times of application as these provided near to idealy conditions for the germination of the spores, promoting the

chances of infection. The conidia would also not be exposed to strong ultra violet radiation at this time of day.

Treatment 2. *Amblyseius teke* (a local phytoseiid predatory mite) were collected from old cassava plants located at Siaya district, in Western Kenya. They were sorted out in the laboratory and introduced (using a fine pointed moist brush) onto the potted plants.

Treatment 3. Laboratory tap water was sprayed onto the cassava plants.

The potted plants were enclosed inside polythene covered cages in order to minimise accidental introduction of phytoseiid mites. From time to time the plants were brought out into the sunshine in order to 'sun-harden' them.

The experimental design was completely randomised with treatments 1 to 3 (described above) and were replicated by three times.

The level of CGM infestation before and after application of control treatments were taken and included the following records: (i) all *M. tanajoa* and *A. teke* eggs; (ii) the pest and predator mobile stages (a combination of larvae, nymphs and adults); and (iii) cadavers found on leaves 1 to 5 of every plant (leaf 1 being the topmost expanded or mature leaf). A minimum of at least three leaflet counts were made, but preferably counts were made on

the whole of the leaves. A leaflet in this context refers to one of the leaf's minor, partly free sub-units all joined together onto a common petiole. *M. tanajoa* damage indiceson a 1 to 5 scale were scored on each of the sampled leaves, the scores increasing with increasing leaf damage.

3.4. Pathological Efficacy of *H. thompsonii* on *M. tanajoa* Infesting Field Cassava.

Cuttings of a local cassava cultivar, 'Kibandameno', were used in the experiment, planted at a spacing of 0.6m and 1.0m between plants and rows respectively. The field was divided into two equal portions or sub-plots measuring 9mx9m. A two metre space separated the two sub-plots, the space acting as a barrier against possible spray drift from one treatment to the other. During the dry season, on 2nd Dec. 1987, the cassava plants were artificially infested with *M. tanajoa* by placing infested cassava leaves onto them. The plants were left for 2 weeks prior to treatment to allow the *M. tanajoa* infestation to be established. The plants were then treated with either

(1) *H. thompsonii* spores suspension

or

(2) Water Spray (Control).

A systematic method of sampling was used to select the plants to assess the effects of the treatments. For example, on each sampling date, a single starting plant in a given row was selected. Subsequent sample plants were picked at an equal distance from the previous one - every

third plant was picked in this case. Where there was more than one branch per plant, only the largest of them was selected for sampling. Five leaves, starting from the topmost expanded leaf and going downwards in steps of two, i.e leaves in positions no: 1, 3, 5, 7, and 9, of fifteen plants per sub-plot were sampled. Sampling continued at two weekly intervals until the conclusion of the experiment.

The data colleced included number of CGM eggs, living mites and number of cadavers and damage symptoms indices on leaves (as in potted experiment). Initially, the number of phytoseiid predatory mites and their eggs were also taken but these were found to be very few and have been excluded from the results. These parameters were recorded from at least three leaflets per sampled leaf. Procedure details are as given in the potted plant investigations (Chap. 3.3).

Daily minimum and maximum temperatures, rainfall totals and number of rainy days for five months covering the duration of the experiment were also recorded from the fields. The weather data were then (1) summarised at two weekly intervals (2) subjected to regression analyses over parameters of CGM infestation levels, i.e CGM population, egg counts and damage levels.

4. RESULTS.

4.1. Mode of Action and Symptoms of *H. thompsonii* on Eggs and Mature Female *M. tanajoa*.

Newly laid *M. tanajoa* eggs were sprayed with a suspension of *H. thompsonii* and water as control. Phase-contrast micrographs did not show fungal growth on the surface of the eggs (Figure 5).

Healthy living CGM had a typical creamy appearence (Figure 6). It was observed that from 24 hours after death (HAT) mites began moving slowly, had lot of diarrhea and also had reduced feeding rate. Most of the treated CGM started dying from 48 HAT. Cadavers of CGM which had died from *H. thompsonii* attack, began developing darker coloration earlier than those from the control, at about 48 hours after death (HAD). CGM which died from natural causes developed the dark brown coloration, later on from 72 HAD (Figure 7).

Figure 8 shows CGM cadavers which body content had bursted out and dried off, 72 HAD. The presence of dry, friable cadavers, which were stuck onto the substratum, characterised the symptoms of mites which had died from *H. thompsonii* attack. Mites that had died from natural causes, however, were not so glued onto the leaf surfaces. At about 96 HAD *H. thompsonii* mycelial growth could be demonstrated on most of the infected CGM cadavers (Figure 9). The invasion of the host mite tissue by the entomogenous fungus,

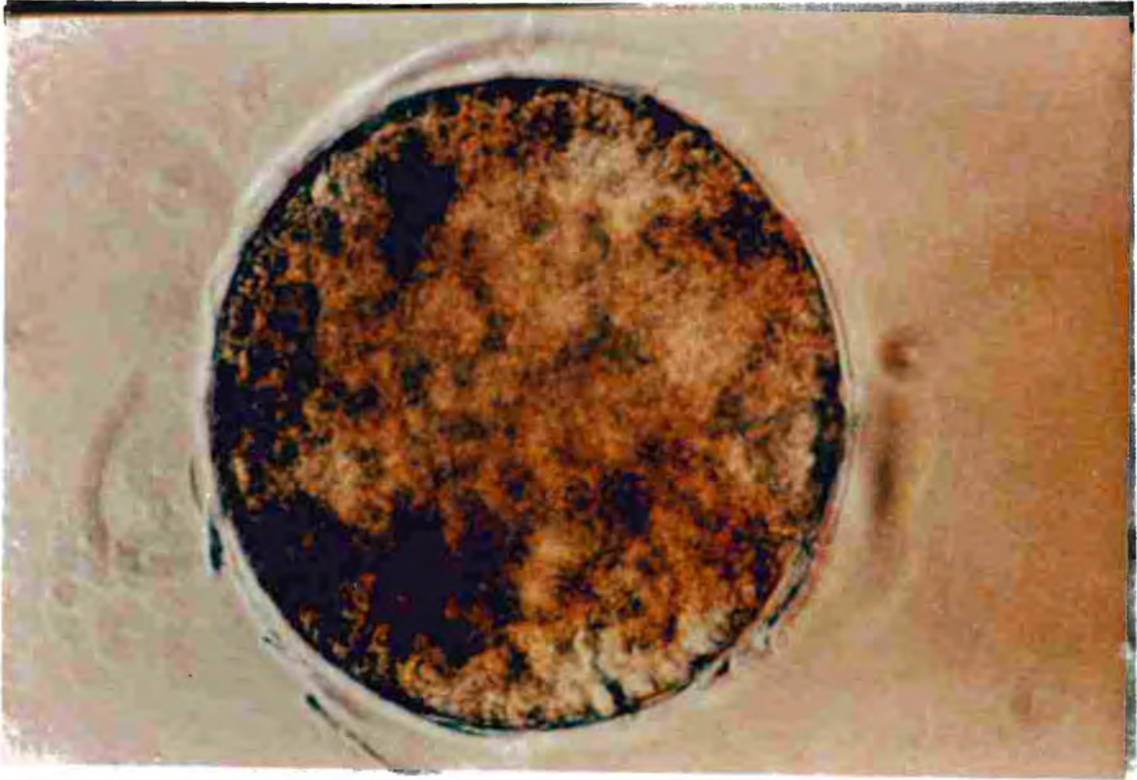

Figure 5. M. tanajoa egg, 72 hours after treatment with H. thompsonii conidia (Phase contrast microscopy; mag.=x3400).

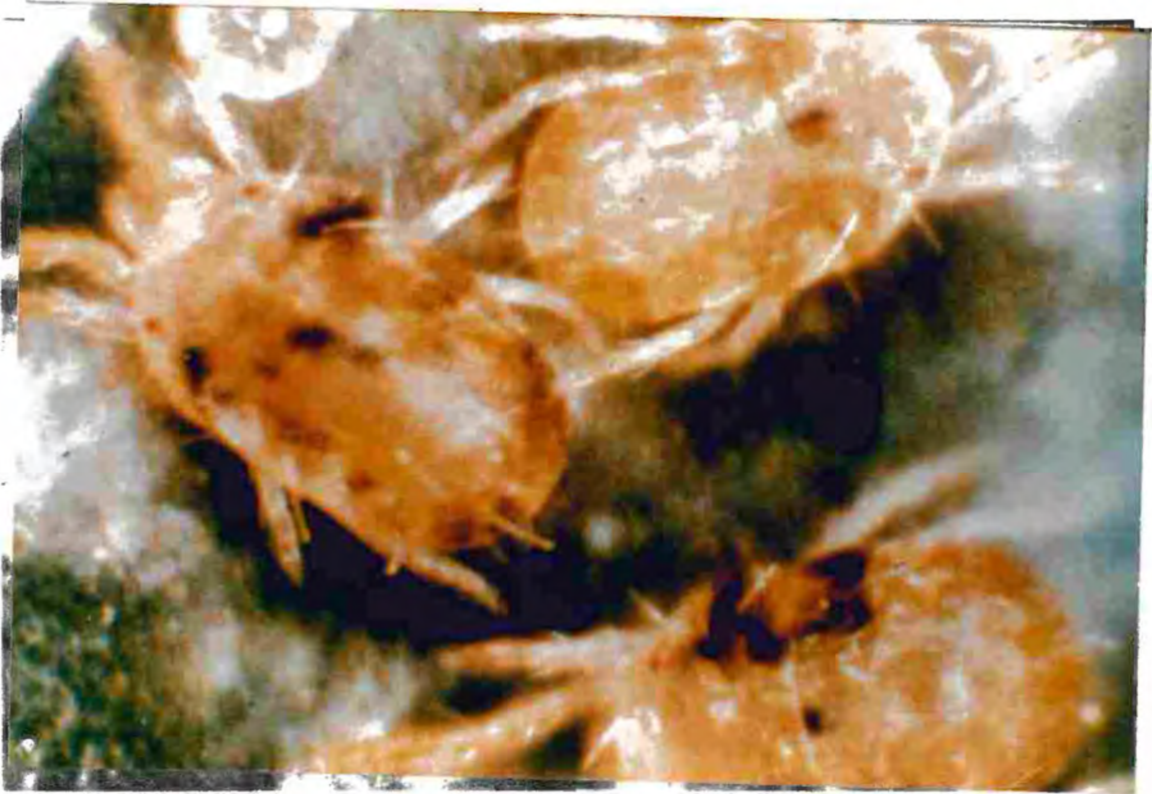

Figure 6. Healthy M. tanajoa just before H. thompsonii treatment (Phase Contrast Microscopy; mag.=x348).

Figure 7. Cadavers of M. tanajoa female, 24 hours after death following H. thompsonii infection (Phase Contrast Microscopy; mag.= x544).

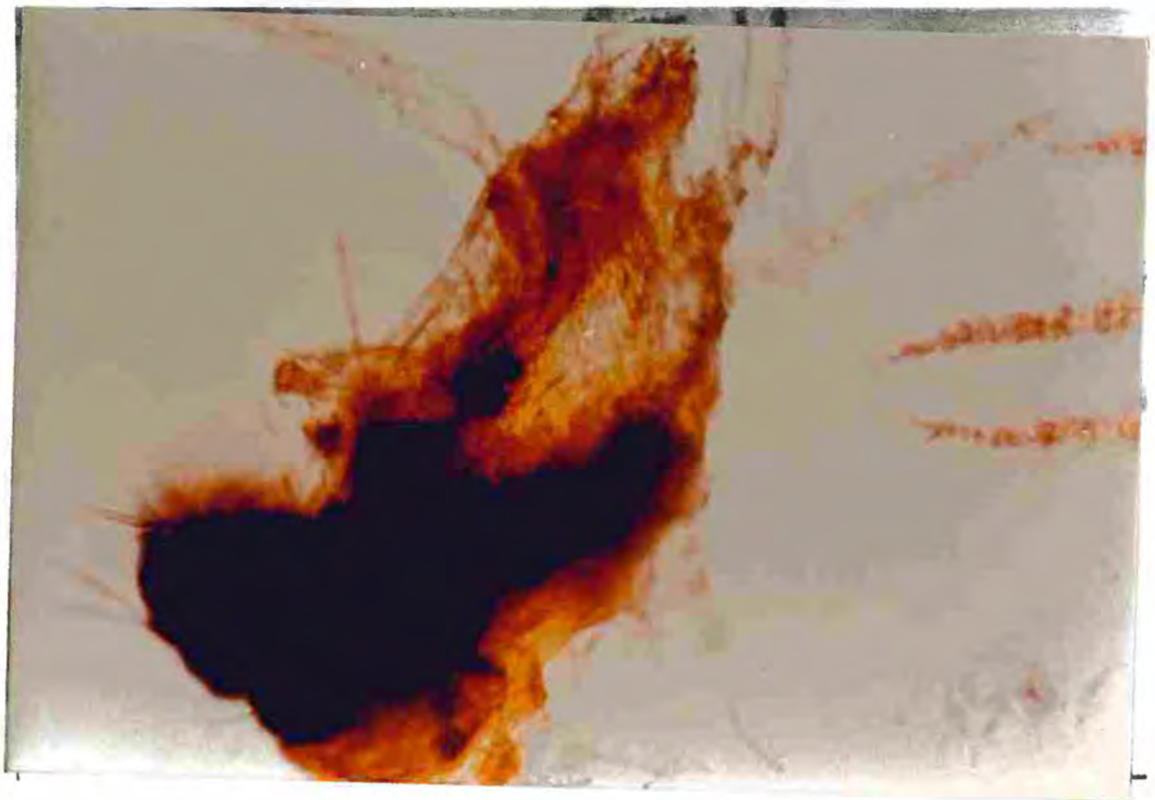

Figure 8. Typical H. thompsonii attack symptom; CGM cadavers drying up, 72 hours after death (Phase Contrast Microscopy; mag=x544).

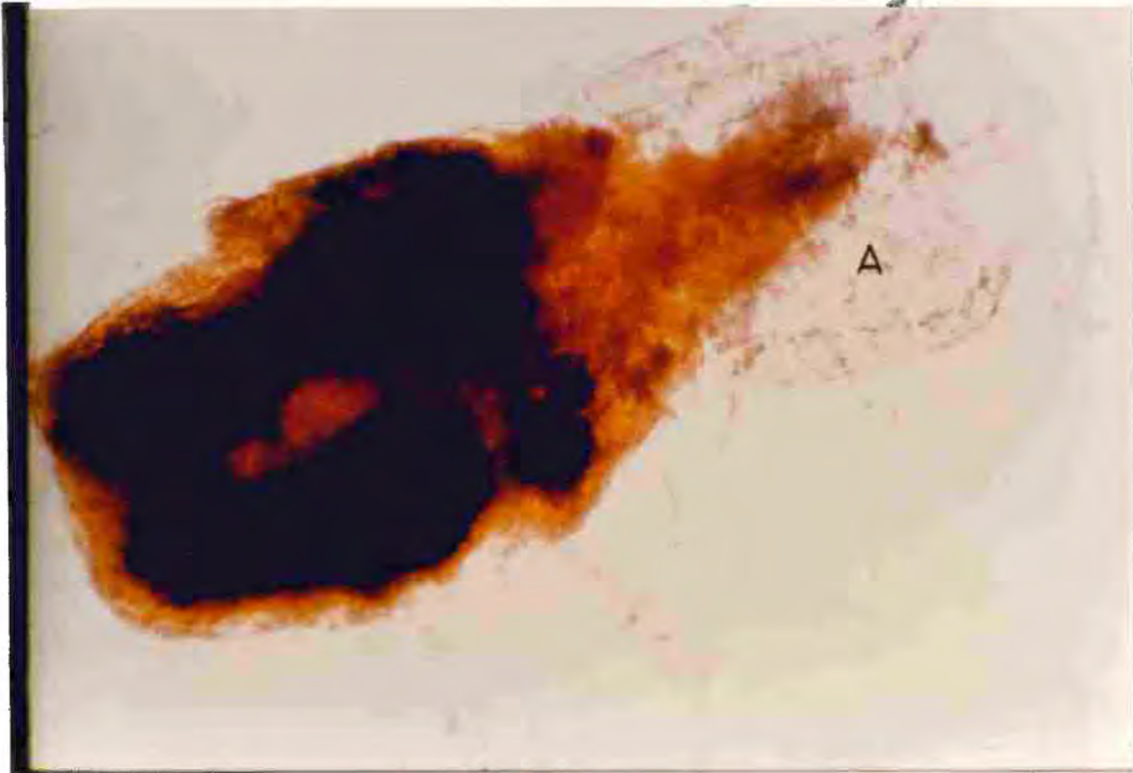

Figure 9. CGM cadavers with H. thompsonii mycelial growth (A) 96 hours after death (Phase Contrast Microscopy; mag.=x544).

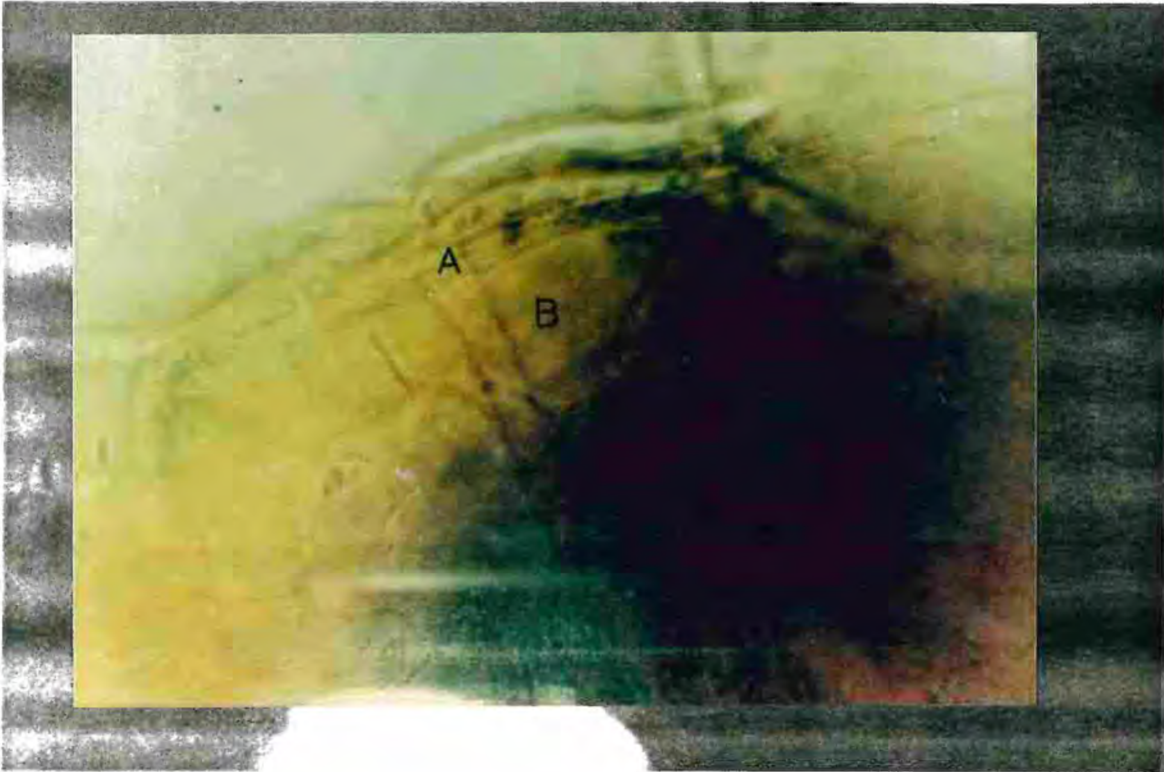

Figure 10. E.thompsonii mycelia (A) growing into the tissue of M. tanajoa (B), 96 hours after death (PCM,mag.=3400).

96 HAD is shown in Figure 10. From this time, and later on, *H. thompsonii* was found conidating freely on infected cadavers (Figure 11).

4.2. Effect of *H. thompsonii* on Developmental Stages of *M. tanajoa*: Laboratory Studies.

4.2.1. *H. thompsonii* Affecting the Hatching of *M. tanajoa* Eggs.

One day old *M. tanajoa* eggs were sprayed with *H. thompsonii* at a concentration of 7.2×10^5 (A), 5.6×10^5 conidia per ml (B), and water (C) (Figure 12). The number of larvae hatching from the treated eggs on the 5th day expressed as a percent value of the total number of eggs, were 40, 59 and 70 respectively. The difference in cumulative percent egg hatch between the three treatments could be demonstrated through days 6, 7 and 8. On day 8 for example, 73%, 84 and 89% of the eggs from the three treatments had hatched into larvae. A maximum cumulative percent egg hatch difference of 21 was recorded between *H. thompsonii* (conc.=7.2×10^5 CPM) and water on the 7th day after treatment.

A comparable investigation as the one above was carried out with a *H. thompsonii* concentration of 8.6×10^7 CPM and water (Figure 13). Eggs treated with the fungus had fewer eggs hatching into larvae than the control batch between the 5th to 8th day after treatment application. On day 6 for

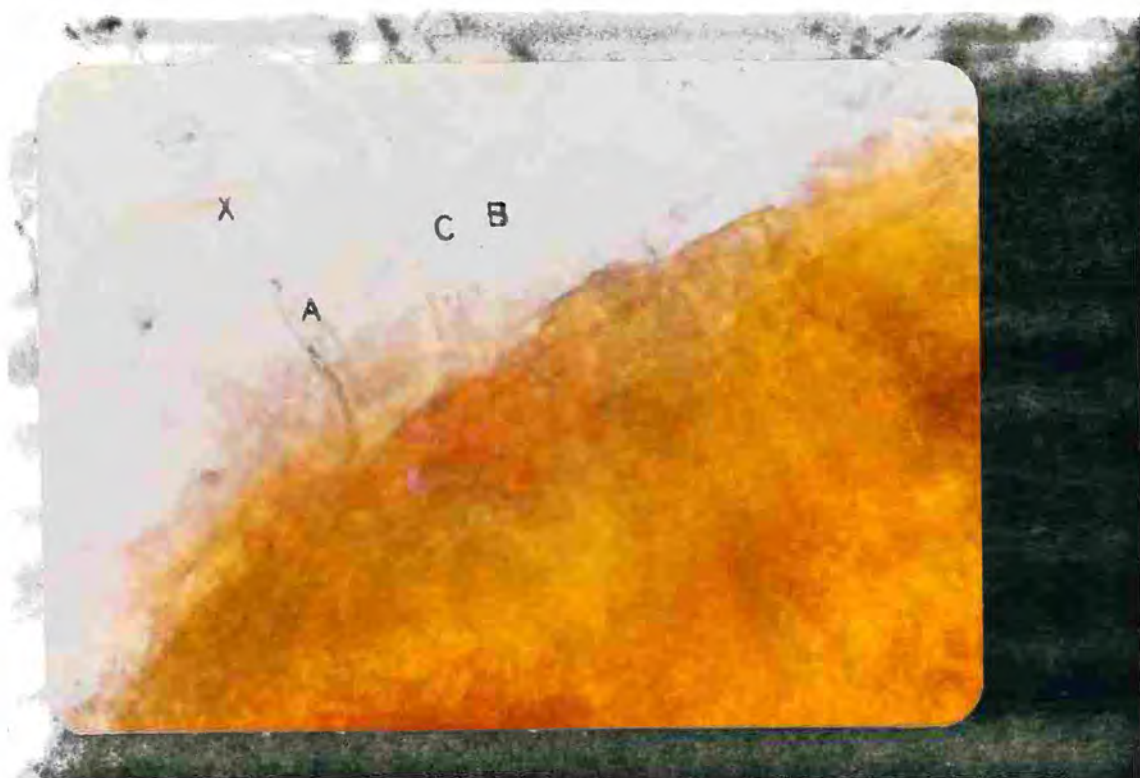

Figure 11 E. thompsonii conidiating on M. tanajoa cadavers, 96 hours after death; A=mycelium showing septation at point X; B=conidium; C=phalide.

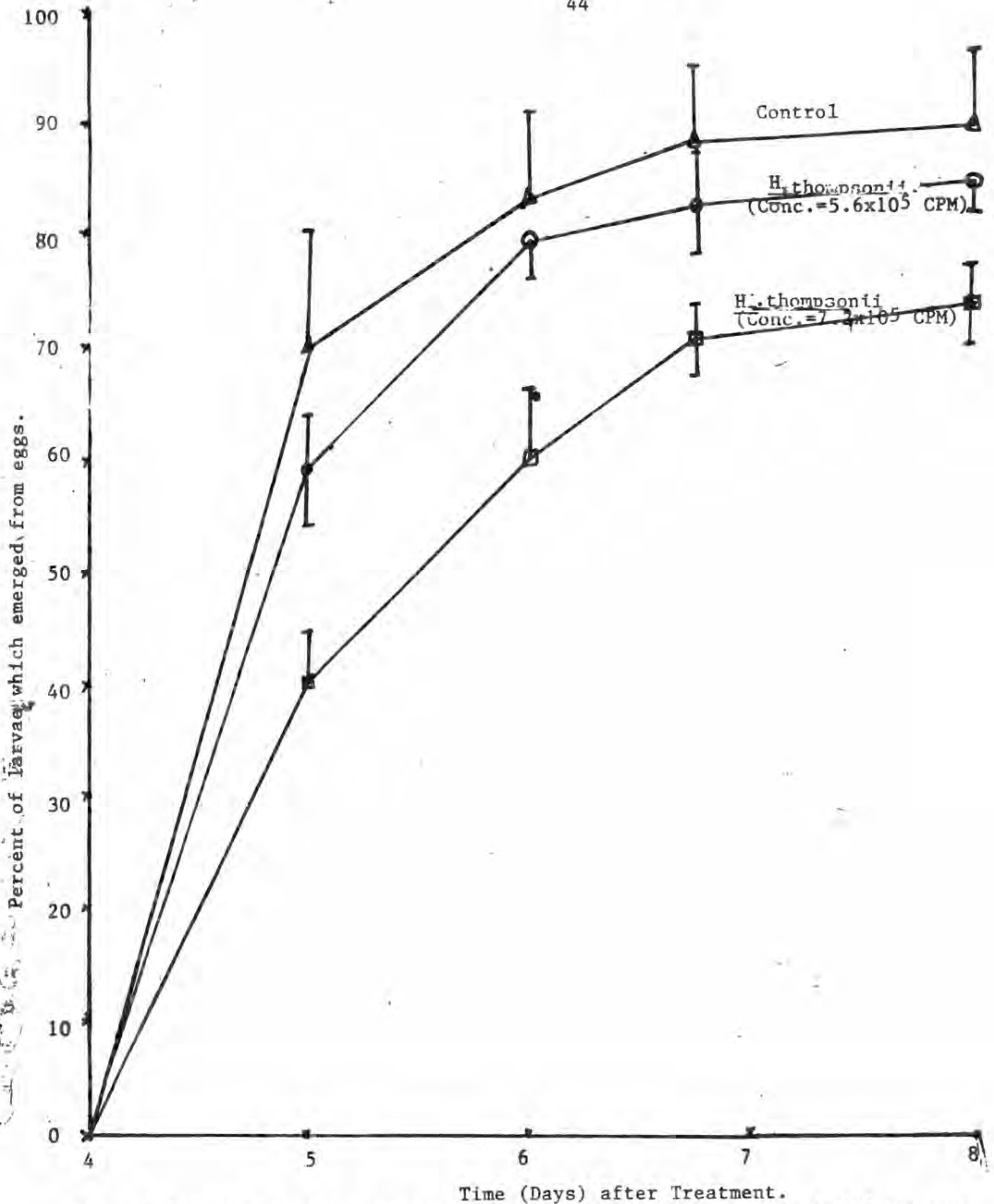

Figure 12. Cummulative percent of living CGM larvae which hatched from eggs treated at one day old stage with _H. thompsonii_ or water.

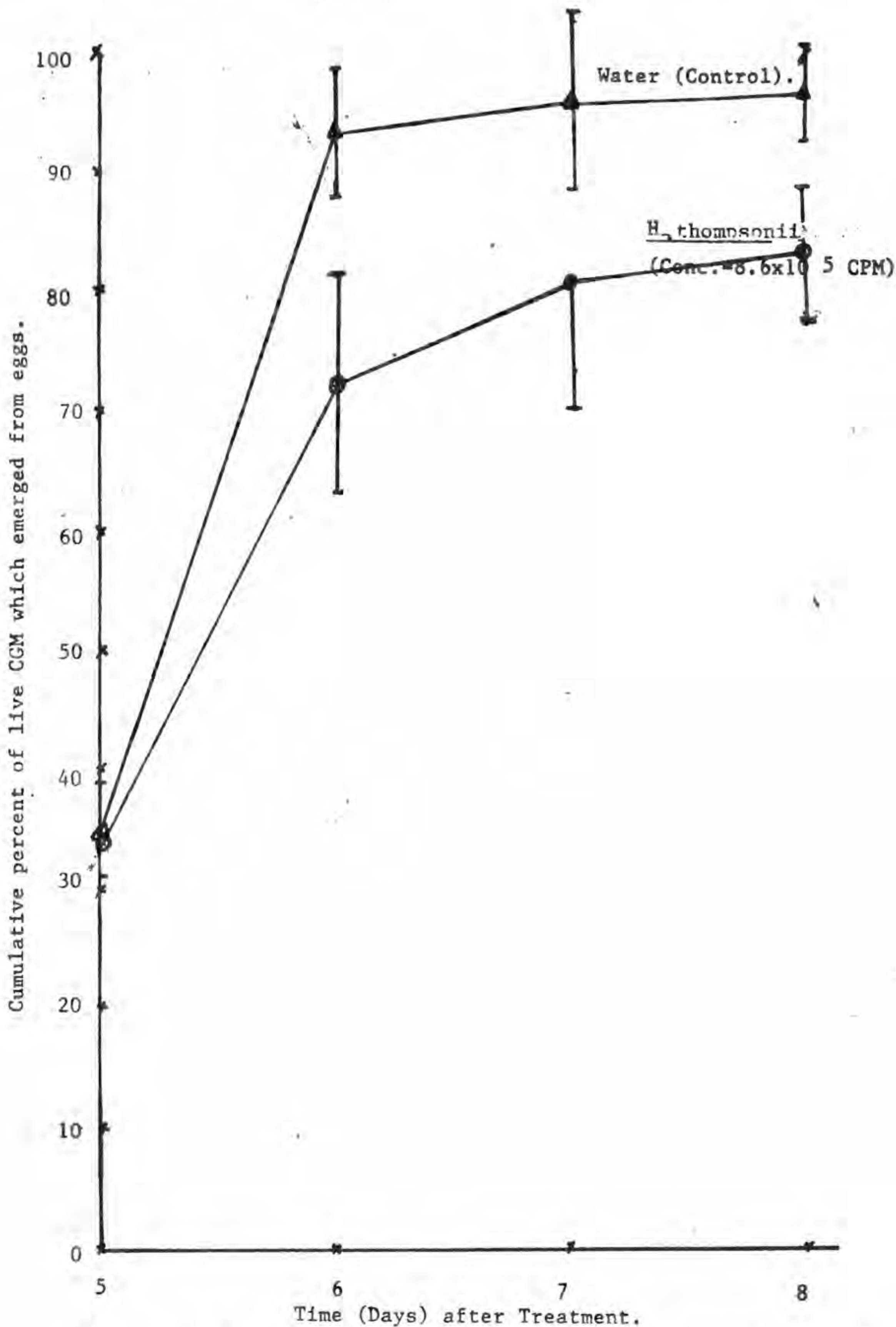

Figure 13. Cumulative percent living CGM larvae which hatched from egg sprayed at one day old stage with H.thompsonii or water.

example, 72% of the fungus sprayed eggs had produced living larvae, whereas 93% emergence was recorded from the control treatment. The difference in percent hatch between these two treatments at this time was 21. The drop in the number of living larvae from the 6th to the 8th day was a reflection of larval mortality, which was higher in the fungus treated eggs than in the control.

Further work on the effect of the entomopathogenic fungus on *M. tanajoa* is given in Figure 14 where batches of one day old eggs were sprayed with a suspension of *H. thompsonii* (Conc.=$1.0x10^8$ CPM) or water as the control. On the 5th day after treatment, 38.0 and 36.0% of the eggs from the fungus and control treatments respectively, produced living larvae. Maximum hatching was was observed between 5 and 6 DAT. On day 6, the fungus treated batch had 72% hatch and the control, 91.5%. Between 7 and 8 DAT larval mortality in the two treatments increased, while the number of eggs which were hatching obviously decreased, explaining the apparent decline in hatching.

In all the three sets of investigations, there was no significant difference in the percentage of eggs which hatched from *H. thompsonii* and water (control).

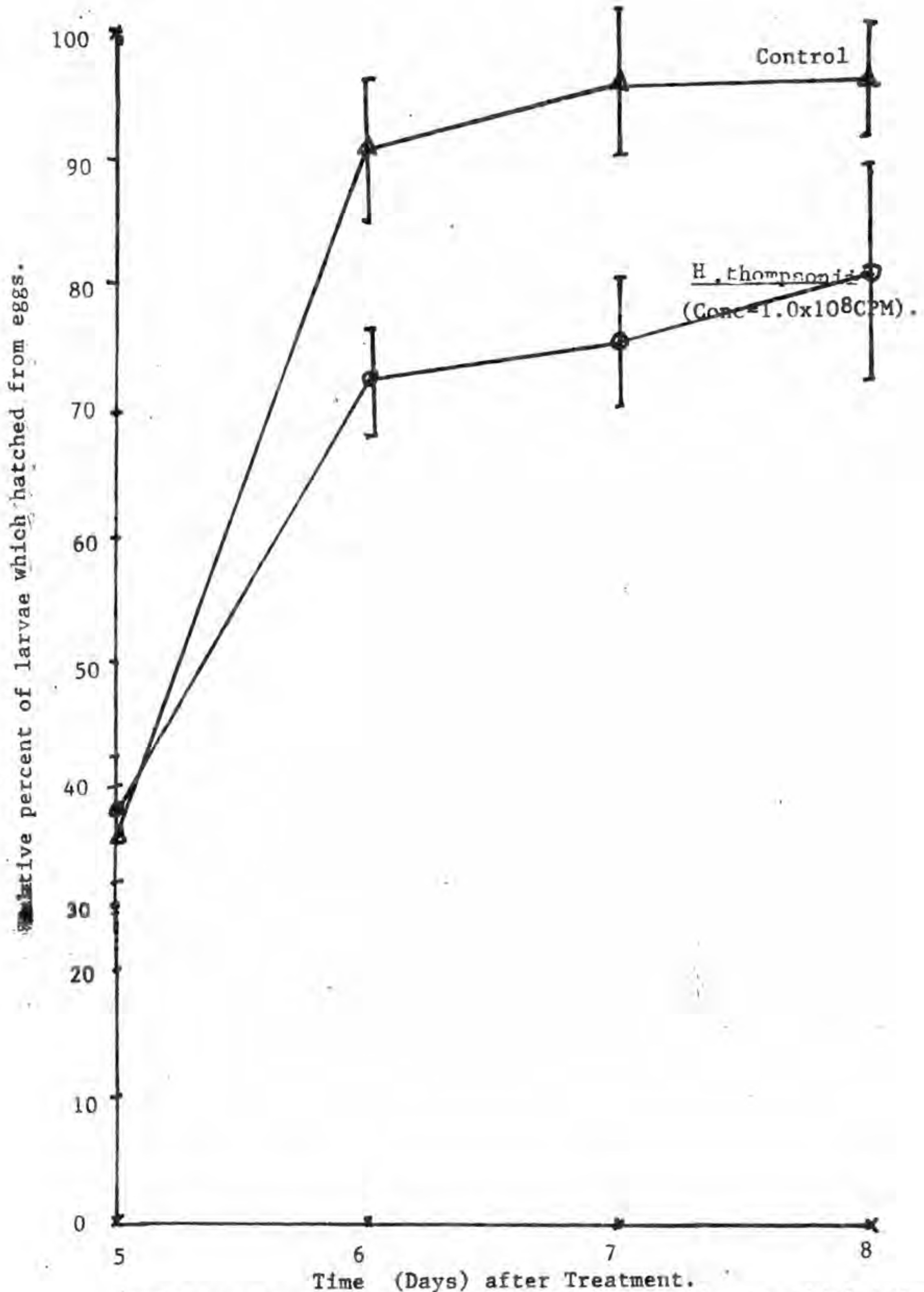

Figure 14. Cumulative percent of living CGM larvae which hatched from eggs sprayed at one day old stage with H. thompsonii or water (Control).

4.2.2. <u>CGM Larvae Cadavers Infected with *H. thompsonii*</u>

Observation of the larval cadavers recovered after treatment application showed that 27.7% (sd=9.8) of the *H. thompsonii* treated larvae were infected with the fungus compared with 12.7% (sd=8.9) in the control (Table 1).

In the humidity chamber, 30.9% and 13.8% of the cadavers from fungus and control treatments respectively were observed to be infected (Table 2).

4.2.3. <u>Number of Eggs Laid per Female CGM Attacked by *H. thompsonii*</u>

A cumulative mean of 5 daily recordings (rep=8) for the two treatments showed that, under humidity chamber conditions, each fungus treated female laid about 8.8 eggs as compared with 7.9 eggs per female in the control. These means did not vary significantly from each other (Table 3).

In the laboratory, a significantly higher egg count of 5.8 eggs per control female, as compared with less than 1.0 eggs per fungus treated female CGM, was recorded (Table 4).

Another set of investigations was conducted using *H. thompsonii* concentrations of 10.4×10^7, 5.2×10^7, 2.6×10^7, 1.3×10^7 and 0 (water control) conidia per ml on female CGM under laboratory and growth chamber conditions. In the laboratory, a mean of 3 eggs per female was counted for each of the *H. thompsonii* treatments and the control at the start of the experiment (Fig. 15). Egg counts per female CGM

Table 1. Percent infection of larval cadavers treated with *H. thompsonii* con.=$8.6x10^5$ CPM (F) and water (C): (Lababoratory Conditions) (Means from data taken daily for 6 days; rep.=6).

Treat.	Mean no. Initial CGM Larvae Treared	% Infection	Arc Sin Values	F Value	P>F	CV
F	21.8	27.7	31.0[a]	68.3	0.0001**	29.8
	sd= 8.7	9.0				
C	16.3	12.7	17.0[b]			
	sd=4.7	8.9				

n=36; Means with the same letter are not significantly different under Duncan's Multiple Range Test (DMRT) (Duncan, 1955).

Table 2. Percent infection of *M. tanajoa* larval cadavers treated with *H. thompsonii* conc.=9.8×10^5 CPM (F) and water (C): (Humidity Chamber Conditions) (Means of data taken for 6 days; rep=6).

Treat.	Initial Live	CGM % Infection	Arc Sin Value	F Value	P>F	CV
F	34.3 sd=9.8	30.9 12.3	32.0[a]	37.3	0.0001**	30.4
C	47.8 sd=12.0	13.8 8.5	20.5[b]			

n=36.* Means with the same letter are not significantly different under Duncan's Multiple Range Test statistics.

Table 3. Number of eggs laid per female CGM treated with *H. thompsonii* conc.=$1.0x10^8$ CPM (F) and water (C): in humidity chamber (Means from 5 daily recordings; rep=8).

Treatment	Mean no. of egg per female	n	F Value	P>F	CV
F	8.8 ns sd=1.7	40	1.76	0.1898	37.5
C	7.9 ns sd=2.6	40			

* ns= Means not significantly different under Duncan's Multiple Range Test statistics.

Table 4. Number of eggs laid per female *M. tanajoa* treated with *H. thompsonii* conc.=$1.0x10^8$ CPM (F) and water (C) (Laboratory conditions) (Mean from data taken for 4 days; rep=8).

Treatment	Mean of CGM Egg/Female	n	F Value	F Value	CV
F	0.6[b] sd=0.4	32	630.0	0.0001	26.1
C	5.8[a] sd=1.2	32			

* Means with the same letter are not significantly different under Duncan's Multiple Range Test statistics.

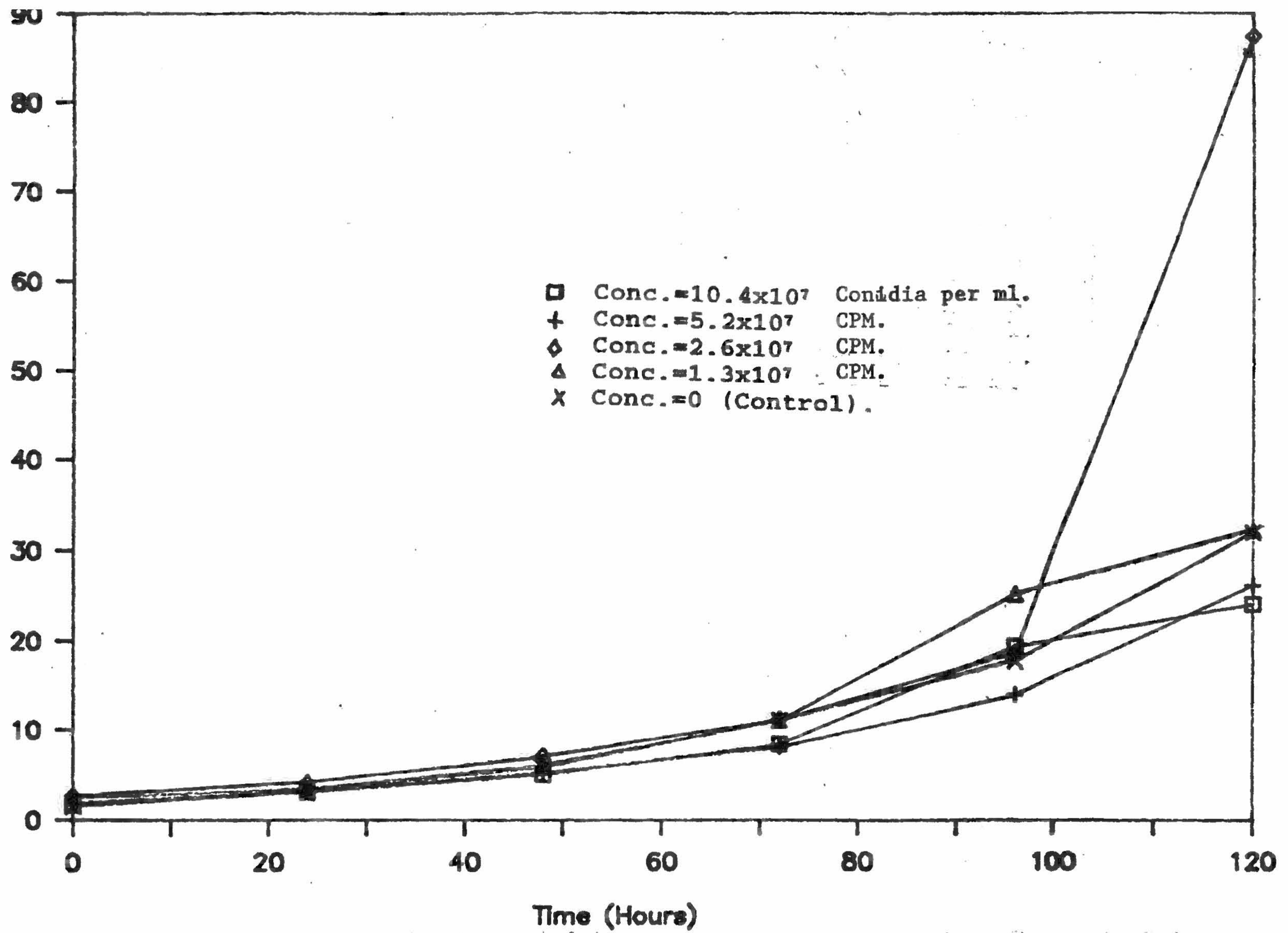

Figure 15. Eggs laid per female CGM infected with various concentrations of H. thompsonii -Laboratory condition.

increased gradually between time 40 and 120 HAT. The maximum egg count value was from fungus treatment concentration $2.6x10^7$ CPM followed by concentrations $1.3x10^7$ CPM and the control.

Under the growth chamber conditions, initial egg counts (at time 0 HAT) per female CGM treated with the fungus concentrations was 3 (Fig. 16). The counts rose relatively more sharply than was reported from the laboratory conditions, so that maximum values between 25 to 40 eggs per female CGM were obtained at 120 HAT. On the whole, a clear relationship between *H. thompsonii* concentrations and egg count values could not be established.

In both laboratory and growth chamber environments, the rise in egg counts per female CGM between 0 to 72 HAT was because of the relatively higher rate of death among female mites from all treatments. At the same time, the overall number of eggs laid was increasing. Later, between 72 to 120 HAT, hatching occured but the rate of this process was lower than the rate of mortality of female CGM. Death was particularly highest on on mites treated with concentration $2.6x10^7$ CPM (Under laboratory condition) whereas under the growth chamber conditions, a relatively higher rate of mortality was observed amongst CGM females sprayed with *H. thompsonii* at $5.2x10^7$ CPM.

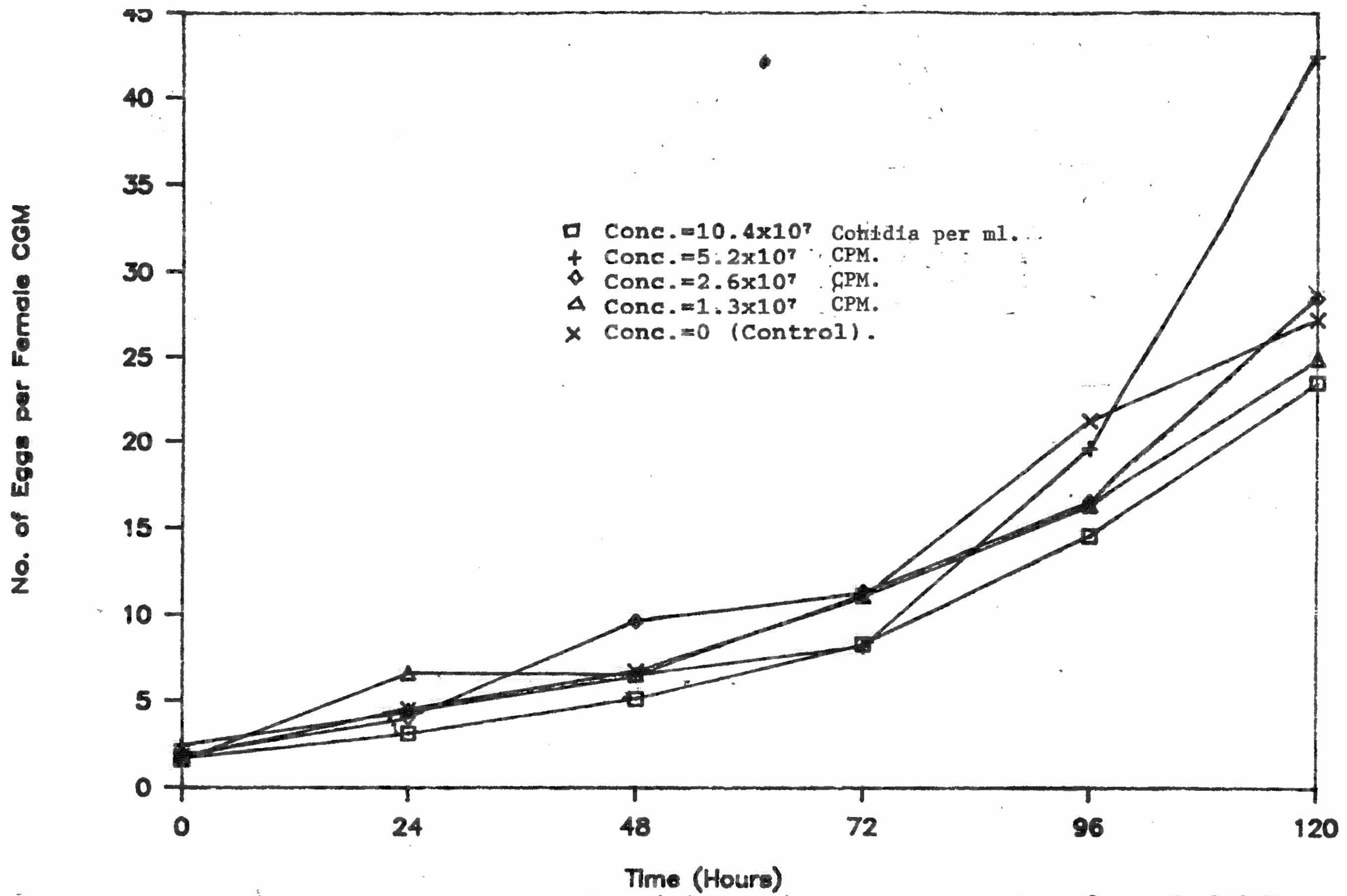

Figure 16 . Eggs laid per female CGM infected with various concentrations of H. thompsonii -Growth chamber condition.

4.3. The Effect of *H. thompsonii* on *M. tanajoa* Infesting Potted Cassava Plants.

4.3.1. Effect of *H. thompsonii* on CGM Egg Counts per Leaf of Potted Cassava Plants.

Mean *M. tanajoa* egg counts on cassava leaves taken on Jan. 6th, two weeks before the plant treatments, showed no significant difference between plants for each of the three treatments, i.e *H. thompsonii*, *A. teke* and water (control). The mean counts were between 1,600 to 4,000 egg per leaf. After treatment, there was a sharp and significant drop (P=0.0381) (Fig. 17 and Appendix 1) in mean egg numbers per leaf from all the treatments, i.e. for *H. thompsonii* mean=6.5 (sd=8.6) eggs per leaf; *A. teke* mean=271.9 (sd=480.2) eggs per leaf and from the control, 835.0 (sd=500.5) eggs per leaf. The egg count trend was then downwards until 5th of Feb., with control, *A. teke* and *H. thompsonii* treated plants having 0.2 (sd=0.5), 17.0 (sd=sd=15.8) and 41.1 (sd=23.7) eggs per leaf respectively. The differences in egg numbers were significant (P=0.0456). On dates between 2nd March until 8th May, there was no statistical variation between egg counts for each of the treatments. The counts varied from between 7 to 1,000 eggs per leaf.

A repetition of the three treatments on CGM eggs counted on a second test cassava cultivar, i.e. 'Ratenyi' is given in Appendix 2.

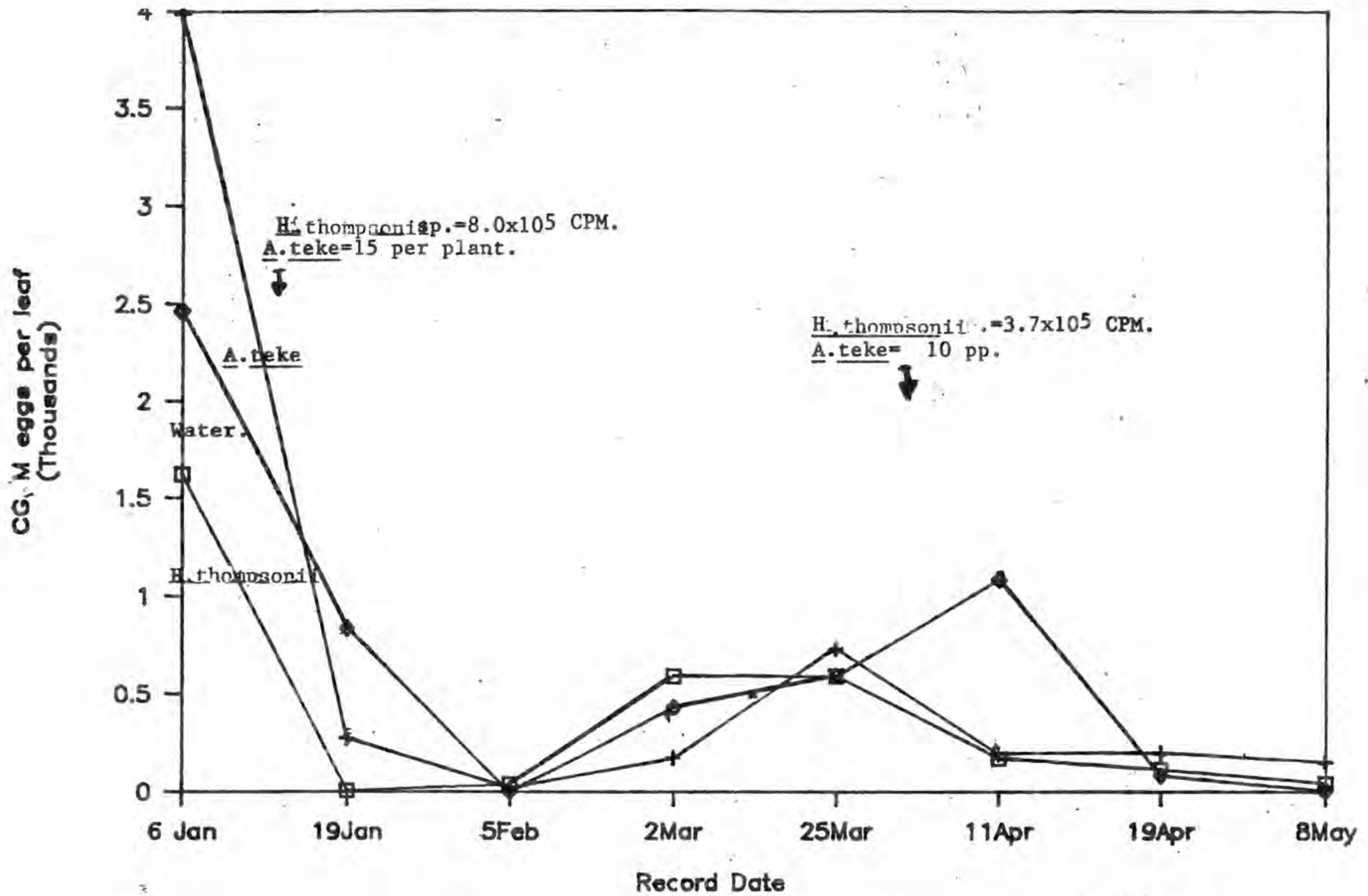

Figure .17 CGM eggs per leaf of potted cassava treated with H. thompsonii ; A. teke & water.

The distribution of CGM eggs on the first five cassava leaves on varieties 'Kibandameno' and 'Ratenyi' is given in appendices 3 and 4 respectively.

4.3.2. The Effect of *H. thompsonii*, *A. teke* and Water on *M. tanajoa* Attacking Cassava Grown in Pots.

On Jan 19th, *M. tanajoa* live counts taken from *H. thompsonii* treated plants (mean=6.8, sd=5.0 per leaf) were not significantly different from those of the control (mean=561.7, sd=916.8 per leaf) and from *A. teke* (mean=982.4, sd=827.3). The reason for the lack of statistical difference was probably due to the high standard deviations between the means (Fig. 18 and Appendix 5). On Feb. 5th, the mean number of CGM was highest on leaves treated with *H. thompsonii*, decreasing on *A. teke* and lowest in the control treatments (means=17.7, sd=12.8; 4.8, sd=5.4 and 0.6, sd=0.2) mites per leaf respectively. This trend was still evident on March 2nd. On March 25th, the mean CGM counts from *H. thompsonii*, *A. teke* and water treatments were 252.2 (sd=157.5), 311.8 (sd=255.3) and 560.4 (sd=272.4). These did not differ statistically from one another. On April 11th and 19th, relatively lower CGM counts were taken on plants treated with *H. thompsonii* and *A. teke* than those from the control treatment (i.e means=70.0, sd=69.2; 136.7, sd=218.2 and 173.3, sd=180.1 for the 11th April and 76.3,

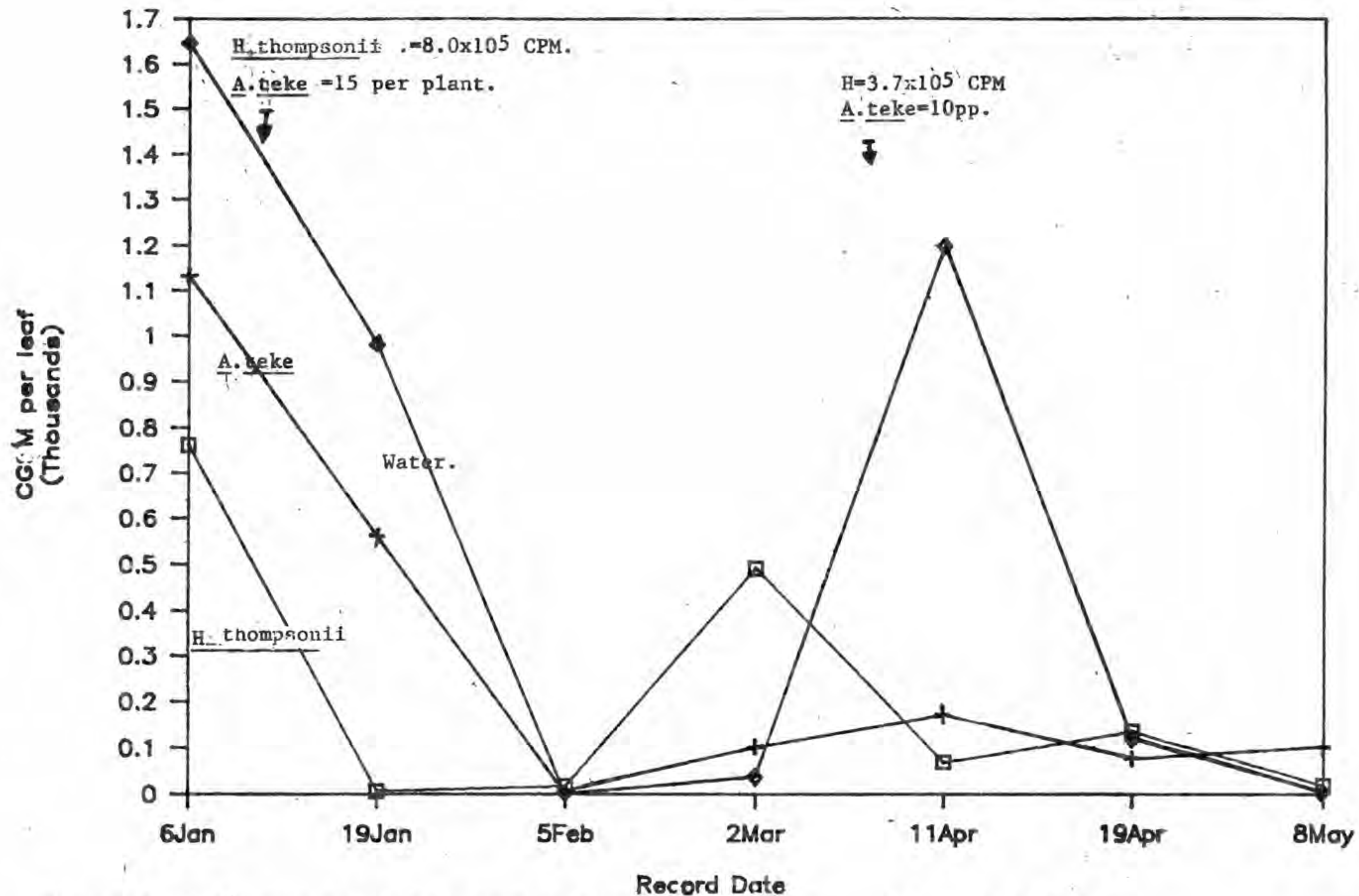

Figure 18. CGM per leaf of potted cassava treated with H. thompsonii ; A. teke & water.

sd=103.2; 1202.2, sd=640.5 and 121.6, sd=97.2 for the 19th April). On May 8th, the mean CGM counts from the fungus and control treatments were 19.9, sd=14.6 and 3.7, sd=3.7 both means being lower than that from the phytoseiid mite treatment (mean=102.9, sd=62.7) (P=0.0436).

Data on further tests made on potted 'Ratenyi' cassava cultivar is shown in Appendix 6. The distribution of mobile CGM on leaves in positions 1, 2, 3, 4 and 5 starting from the topmost expanded leaf and proceeding downwards to older lower leaves for the two cultivars are given in Appendices 7 and 8.

4.3.3. CGM Cadaver Counts on Potted Cassava Plants Treated with *H. thompsonii*, *A. teke* and Water.

Prior to treatment application onto the experimental plants, i.e on Jan. 6th, the mean number of CGM cadavers counted for *H. thompsonii* and *A. teke* phytoseiid mite treatments, were 70.0, (sd=30.5) and 98.8 (sd=36.4) per leaf respectively. These values were significantly lower than those from the control treatment plants (mean=185.7, sd=50.90). On Jan. 19th, the highest number of cadavers were recovered from *A. teke* treated plants (mean=4817.0, sd=3100.2) and were lowest on the *H. thompsonii* treated leaves (mean=144.2, sd=120.8). The three counts were highly significantly different from one another (P=0.0007). From

Table 5. Mean number of CGM cadavers per leaf of potted 'Kibandameno' cassava treated with: *H. thompsonii* (F); *A. teke* (P) and water (C).

Date	Cadavers/leaf on treat. F	P	C	F Value	P>F	CV
6Jan	$(3.1)^{b}$ 70.0 sd=30.5	$(3.3)^{b}$ 98.8 36.4	$(4.9)^{a}$ 185.6 50.9	3.50	0.0450	30.2
*12/01. *H. thompsonii* =8.0x10^{5} CPM; *A. teke*=15 PP.						
19Jan	$(4.1)^{c}$ 144.2 sd=120.8	$(8.3)^{a}$ 4817.0 3100.2	$(6.8)^{b}$ 1137.0 533.1	72.32	0.0007	9.0
5Feb	$(1.0)^{a}$ 12.0 sd=18.0	$(1.4)^{a}$ 550.0 952.2	$(3.1)^{a}$ 655.7 631.1	2.12	0.2355	100.9
2Mar	$(3.2)^{a}$ 58.3 sd=68.0	$(1.2)^{b}$ 9.4 14.4	$(0.4)^{b}$ 0.9 1.0	8.95	0.0334	75.2
25Mar	$(3.0)^{a}$ 28.3 sd=19.4	$(2.5)^{a}$ 25.3 26.2	$(2.0)^{a}$ 11.7 11.7	0.51	0.0329	41.5
*04/04. *H. thompsonii* sp=3.7x10^{5} CPM; *A. teke*=10 PP.						
11Apr	$(3.5)^{a}$ 99.6 sd=130.8	$(5.4)^{a}$ 365.4 309.3	$(4.4)^{a}$ 125.0 125.3	4.80	0.0864	20.2
19Apr	$(2.9)^{a}$ 81.3 sd=109.1	$(4.4)^{a}$ 442.1 678.9	$(5.5)^{a}$ 370.0 299.8	1.04	0.4342	31.6
8May	$(3.9)^{a}$ 109.0 sd=55.5	$(0.9)^{a}$ 7.5 7.5	$(3.9)^{a}$ 78.3 36.0	2.96	0.1641	22.7

* Dates when the levels of treatments shown were applied; means in parentheses (log(count+1))with the same letter are not significantly different under DMRT; actual means and standard deviations are indicated; n=15.

the 5th Feb., through to 11th April, cadaver counts varied from 1 to 656 per leaf both extremes being recorded from the control treatment. Between the 19th of April and the 8th of May, there was no significant variation between the three treatments.

Appendix 9 shows cadaver counts per leaf from treatments made on 'Ratenyi' cassava cultivar. Appendix 10 and 11 give a general picture of the distribution of CGM cadavers on the first five leaves of potted cassava plants of the two cultivars.

4.3.4. *M. tanajoa* Damage Index on Leaves of Potted Cassava Treated with *H. thompsonii*, *A. teke* and Water.

Scoring for damage levels of CGM on potted 'Kibandameno' cassava began on January 19th, 1988, two weeks aftet treatments had been applied onto the experimental plants. The damage index recorded from *H. thompsonii* treated plants was 1.2 (sd=0.3) and was significantly lower than those from *A. teke* and water (means=3.1, sd=0.6 and 2.6, sd=0.2) (P=0.0009). On 5th of Feb., the mean damage score on leaves treated with *H. thompsonii* (mean=1.0, sd=0.0) and *A. teke* (mean=1.4, sd=0.7) were statistically lower than that recorded from the control leaves (mean=2.8, sd=0.4) (P=0.0323). The development of damage indices through the period of until 5th May is illustrated in Table 6.

Table 6. Cassava green mite (CGM) damage index on leaves of 'Kibandameno' Cassava cultivar treated with:

H. thompsonii (F); *A. teke* (P) and water (C).

Date	CGM damage index on treat. F	P	C	F Value	P>F	CV
*12/01. *H. thompsonii* =8.0x10^5 CPM; *A. teke*=15 PP.						
19 Jan	1.2^b sd=0.3	3.1^a 0.6	2.6^a 0.2	63.70	0.0009	23.7
5 Feb	1.0^b sd=0.0	1.4^b 0.7	2.8^a 0.4	9.14	0.0323	45.5
2 Mar	1.3^a sd=0.3	1.1^a 0.1	1.0^a 0.0	2.00	0.2500	30.8
25 Mar	1.8^a sd=0.4	1.4^a 0.3	1.5^a 0.3	0.37	0.7114	20.3
*06/04. *H. thompsonii* =3.7x10^5 CPM; *A. teke*=10 PP.						
11 Apr	1.3^b sd=0.6	2.1^a 0.3	2.2^a 0.4	18.97	0.0001	22.2
19 Apr	2.1^a sd=0.3	1.2^b 0.4	1.9^a 0.1	9.80	0.0001	21.0
5 May	1.0^b sd=0.0	1.0^b 0.0	2.3^a 0.5	8.40	0.0370	29.6

*Dates when the indicated levels of treatments were applied; means with the same letter are not significantly different under DMRT; n=15.

Appendix 12 shows the scores made on a second cassava cultivar, 'Ratenyi' after treatment applications. Appendices 13 and 14 give the variation of CGM damage on leaves in positions 1, 2, 3, 4 and 5 on plants from the two test cultivars, 'Kibandameno' and 'Ratenyi' respectively.

4.3.5. Counts of *A. teke* on all Treated Cassava Plants.

The highest mean number (10.1, sd=8.4) of predatory mite, *A. teke* was recorded on the 19th Jan., 1988 from plants where phytoseiid mites were introduced. 0.8 (sd=1.1) of *A. teke* mobile mites-i.e larvae, nymphs and adults combined-per leaf was recorded from the *H. thompsonii* treated plants. The control treatment had zero counts of beneficial mites at this time. The means dwindled towards zero as shown by observations made on the 5th of Feb., 2nd and 25th of March (Table 7). The result indicates that there was an apparent migration of the phytoseiids from the plants where they were introduced towards the control plants in the later part of the investigation, e.g on the 19th April and 5th of May, 1988 the means were, *H. thompsonii* treated=3.9 (sd=3.7) and 0.1 (sd=0.5); *A. teke* treated=2.4 (sd=1.4) and 0.5 (sd=1.4); and control, 6.5 (sd=7.7) and 1.1 (sd=2.8) respectively.

The same experimental treatments were repeated on 'Ratenyi' cassava cultivar and results are given in Appendix

15. *A. teke* distribution on the first five leaves of 'Kibandameno' and 'Ratenyi' cultivars is shown in Appendices 16 and 17 respectively.

M. teke egg counts per leaf followed a similar pattern to that of the mobile phytoseiid counts described previously. These are presented in Table 8 and appendix 18 for 'Kibandameno' and 'Ratenyi' cultivars respectively. *A. teke* egg counts on the first five five leaves of the two cultivars are given in Appendices 19 and 20.

Table 7. Means of *A. teke* on leaves of potted 'Kibandameno' cultivar treated with: *H. thompsonii* (F); *A. teke* (P) and water (C).

Date	Number of *A. teke* per leaf F	P	C
*12/01. *H. thompsonii* =8.0x10^5 CPM; *A. teke*=15 PP.			
19 Jan	0.8	10.1	0.0
	sd=1.1	8.4	0.0
5 Feb	0.0	0.2	0.0
	sd=0.0	0.4	0.0
2 Mar	1.0	0.0	0.0
	sd=1.7	0.0	0.0
21 Mar	1.2	0.2	0.0
	sd=2.1	0.8	0.0
*04/04. *H. thompsonii* =3.7x10^5 CPM; *A. teke*=10 PP.			
11 Apr	2.4	5.0	0.7
	sd=4.0	5.1	1.2
19 Apr	3.9	2.4	6.5
	sd=3.7	1.4	7.7
8 May	0.1	0.5	1.1
	sd=0.5	1.4	2.8

*Dates and units of treatment application; n=15.

Table 8. Mean number of *A. teke* eggs per leaf of potted 'Kibandameno' cultivar treated with: *H. thompsonii* (F); *A. teke* (P) and water (C).

Date	*A. teke* eggs on treatments F	P	C
*12/01. *H. thompsonii* =8.0x10^5 CPM; *A. teke*=15 PP.			
19 Jan	1.3	6.6	0.0
	sd=2.4	5.9	0.0
5 Feb	0.0	0.0	0.0
	sd=0.0	0.0	0.0
2 Mar	0.7	0.0	0.0
	sd=1.2	0.0	0.0
25 Mar	0.4	1.4	0.0
	sd=1.1	3.8	0.0
*06/04. *H. thompsonii* =3.7x10^5 CPM; *A. teke*=10 PP.			
11 Apr	1.0	5.2	3.0
	sd=1.2	7.5	5.1
19 Apr	3.1	0.9	8.7
	sd=4.3	1.5	9.1
8 May	0.0	0.3	0.0
	sd=0.0	1.3	0.0

* Dates and units of treatment applications; n=15.

4.4. Pathological Efficacy of *H. thompsonii* on *M. tanajoa* Infesting Field Cassava Plants.

4.4.1 *M. tanajoa* Eggs Counts per Leaf as Affected by *H. thompsonii*: Field Experiment.

Counts made before plant treatment showed that the mean number of CGM eggs per leaf on 25th Jan, 1988, were 223.7 (sd=208.8) and 179.0 (sd=164.9) in plots to be treated with *H. thompsonii* and water respectively. These means did not significantly differ from each other (Fig. 19 and Appendix 21). The first counts made after treatment applications showed a highly significant reduction in the number of *M. tanajoa* eggs from *H. thompsonii* protected plants (mean=308.8; sd=243.6 eggs per leaf) when compared to a mean of 834.8 (sd=602.0) eggs in the control (P=0.0001) which represented approximately, a 63% reduction in the number of eggs per leaf. The difference between the egg counts became less pronounced on March the 8th and 21st. On March 21st, for example, low records of about 5.1 (sd=8.0) and 9.8 (sd=26.6) eggs per leaf were taken from plots treated with the fungus and water respectively. There was no marked difference between the counts taken in each of the treatment plots in April. *H. thompsonii*

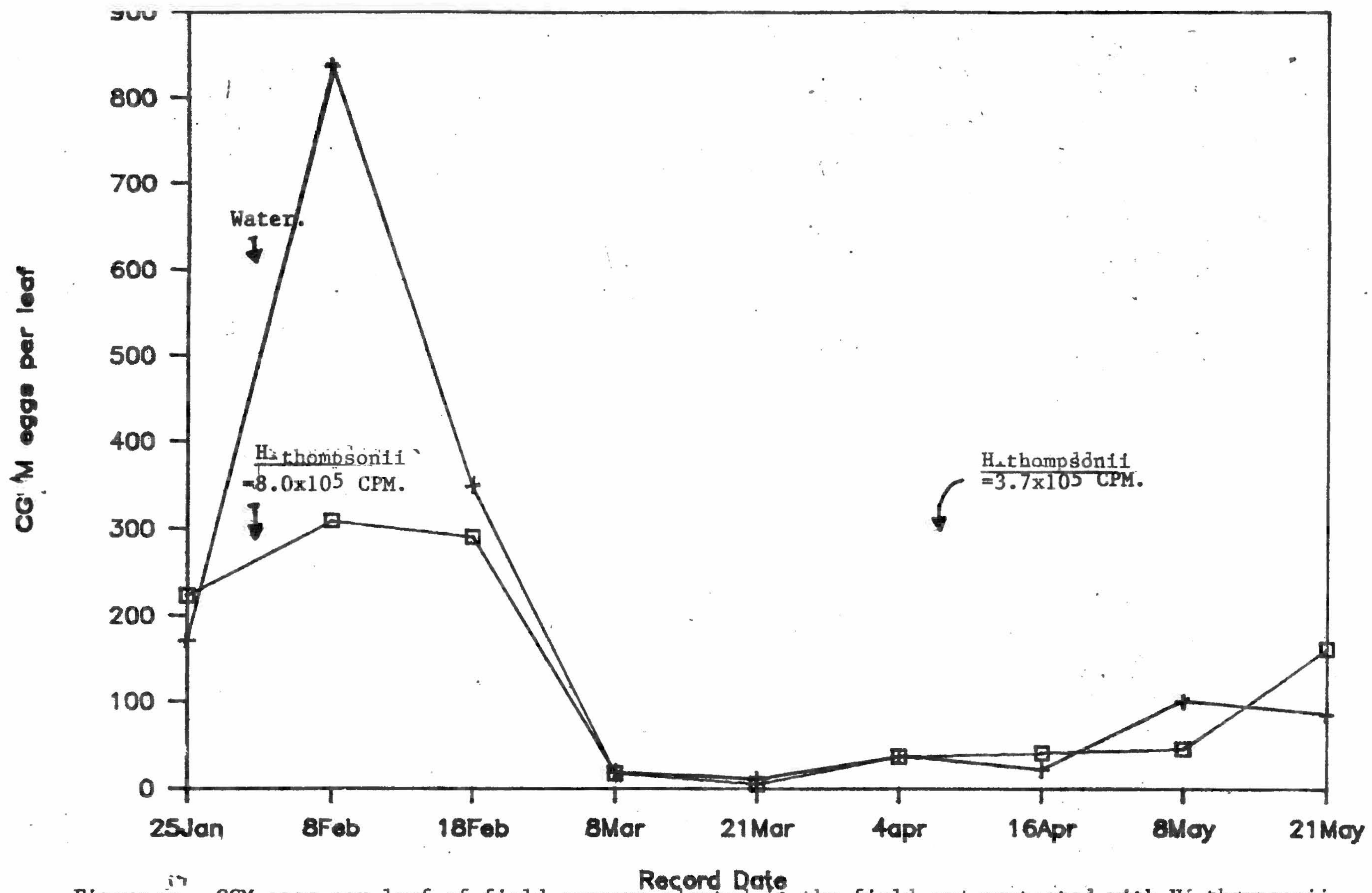

Figure 19. CGM eggs per leaf of field cassava planted in the field and protected with *H. thompsonii*

treated plots recorded 40.5 (sd=3.1) and 45.6 (sd=6.9) eggs per leaf on the 16th April and 6th of May, while the control field had 21.3 (sd=6.6) and 100.6 (sd=17.4) eggs per leaf. On 21st of May, however, there was a significantly higher number of eggs per leaf on protected plants (mean=161.7 sd=32.1) as compared with the control (mean=85.2 sd=20.6) (P=0.0481). Rainfall record showed that there was a heavy and continious downpour from about the second half of March through to May when between 20 to 130 mm of rainfall every two weeks was recorded (Appendix 25). Air temperature in the field varied between 26.8 max. and 19.4 min (Appendix 29).

4.4.2 Effect of *H. thompsonii* on *M. tanajoa* on Field Cassava

Two weeks prior to the application of *H. thompsonii* spores suspension or water (control) to field cassava, there was no difference in number of mobile *M. tanajoa* counted on plants in each of the plots i.e mean=185 (sd=149.7) per leaf in the treatment plot and 198.0 (sd=179.8) on the control plot (Figure 20 and Appendix 22). One week after treatment the mean number of mobile CGM was, 72.3 (sd=71.0) per leaf in the *H. thompsonii* treated

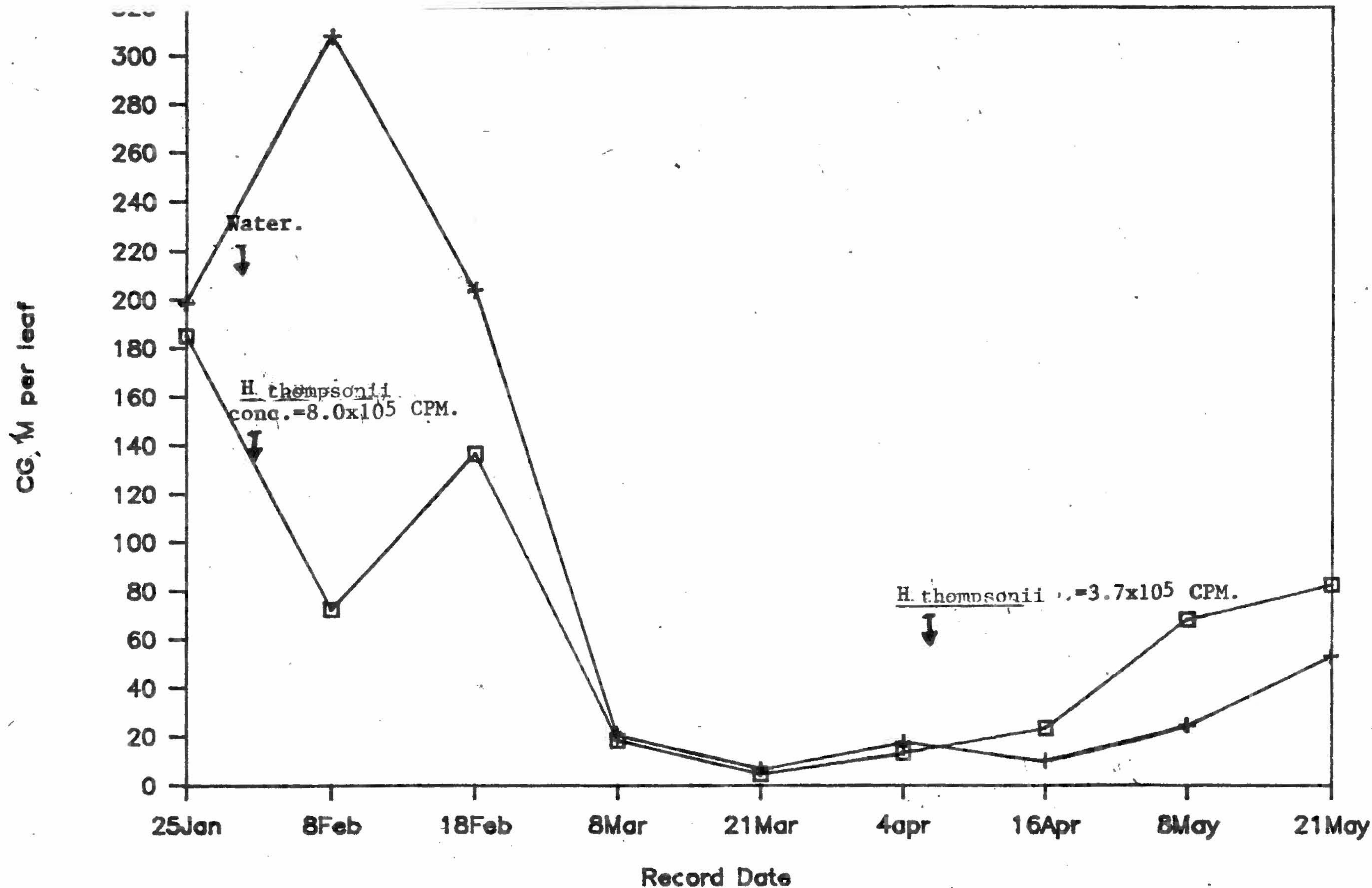

Figure 20. CGM per leaf of field cassava sprayed with *H. thompsonii* & water.

plot, and 307.9 (sd=287.1) per leaf in the control plot. This represented a 77% reduction in the number of living CGM on the *H. thompsonii* treated leaves. The means varied significantly (P=0.0001). On the 18th of Feb., the *H. thompsonii* treated plants had a mean of 136.4 (sd=140.2) mites per leaf compared with 203.8 (166.1) per leaf on the control plants. The recorded means were not significantly different.

The effects of rainfall on CGM population were manifested by a sharp drop in the number of mobile CGM recorded in each plot throughout March, continuing into the first half of April, 1988, when the means were between 5 to 24 respectively.

On May 21st, the mean numbers of mobile CGM per leaf in both treatment plots were still not significantly different, However, by the 21st of May, the mean CGM counts per leaf of 82.7 (sd=3.6) on the treated and 53.0 (sd=11.7) in the control plants, were significantly different (P=0.0001).

4.4.3 Effect of *H. thompsonii* on *M. tanajoa* Cadaver Counts.

In general, the number of cadavers observed on leaves for both fungus and control treatments was not consistent over time. Pre treatment

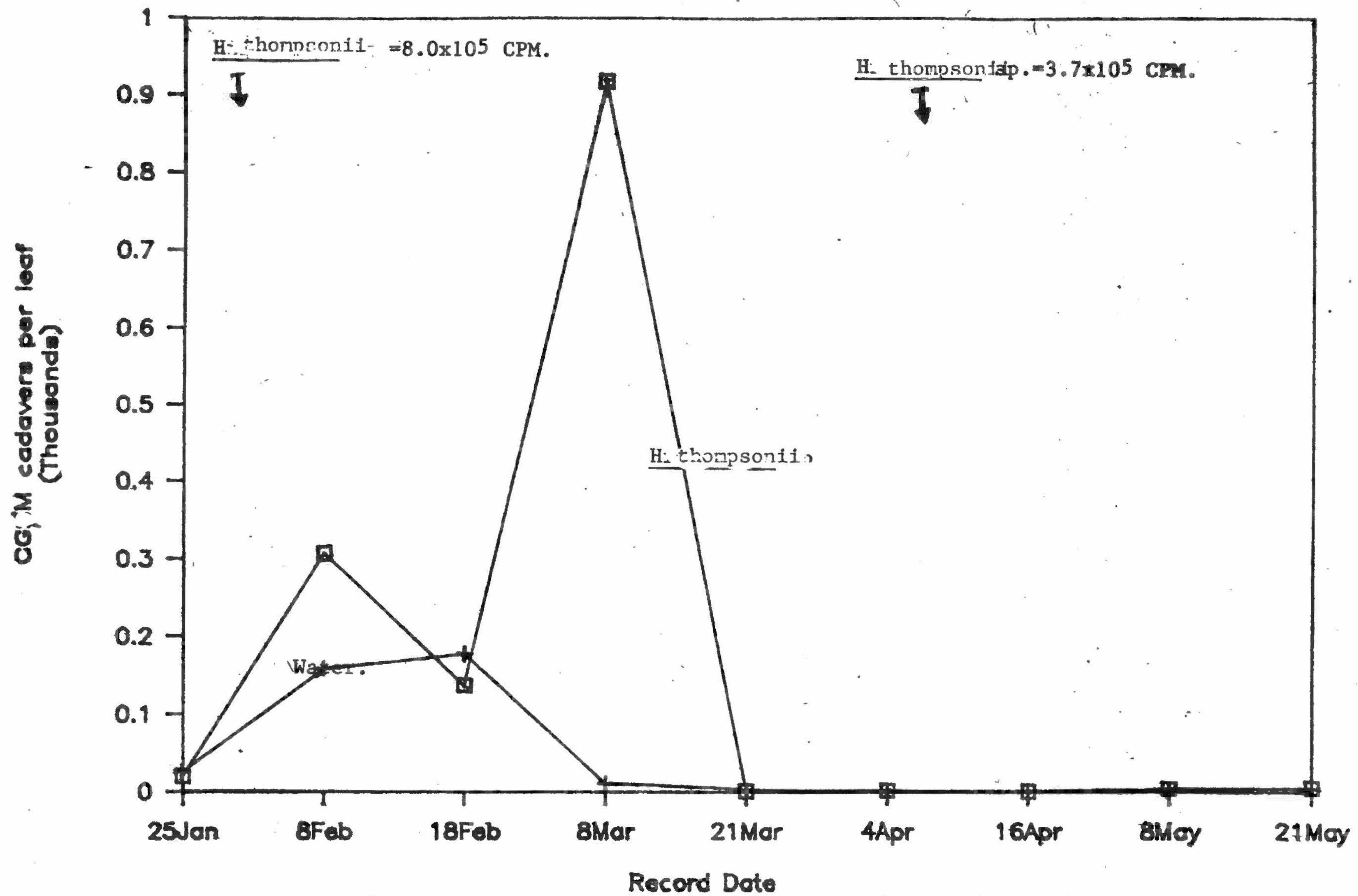

Figure 21. CGM per leaf of field cassava sprayed with H.thompsonii & water.

cadaver counts taken on the 25th of Jan., in both treatments were 20.1 (sd=27.8) and 25.3 (sd=27.5) per leaf for fungus and control plots respectively. These figures were not significantly different (Fig. 24; Appendix 24). The cadaver counts increased after application of the treatments, i.e on the 8th of Feb., 307.3 (sd=346.3) and 158.2 (sd=216.0) cadavers per leaf were recorded on fungus and control plants respectively. On the 8th of March, a mean of 9.8 (sd=11.7) cadavers per leaf was recorded in the control plot compared with 915.6 (sd=22.7) cadavers per leaf in the *H. thompsonii* treated plot.

The washing off effects of the rains caused a drastic reduction in the number of cadavers per leaf from the 21st of March. The lowest cadaver counts were made on the 16th of April, at the end of the rains. Details of the recordings are given in Appendix 24.

4.4.4 *M. tanajoa* Damage on Field Cassava Sprayed With *H. thompsonii* or Water.

H. thompsonii treated cassava leaves had damage scores (scored on a 1 to 5 index, increasing with severity of damage) of 1.7 (sd=0.5), 1.6 (sd=0.7) and 1.9 (sd=0.6) on the 25

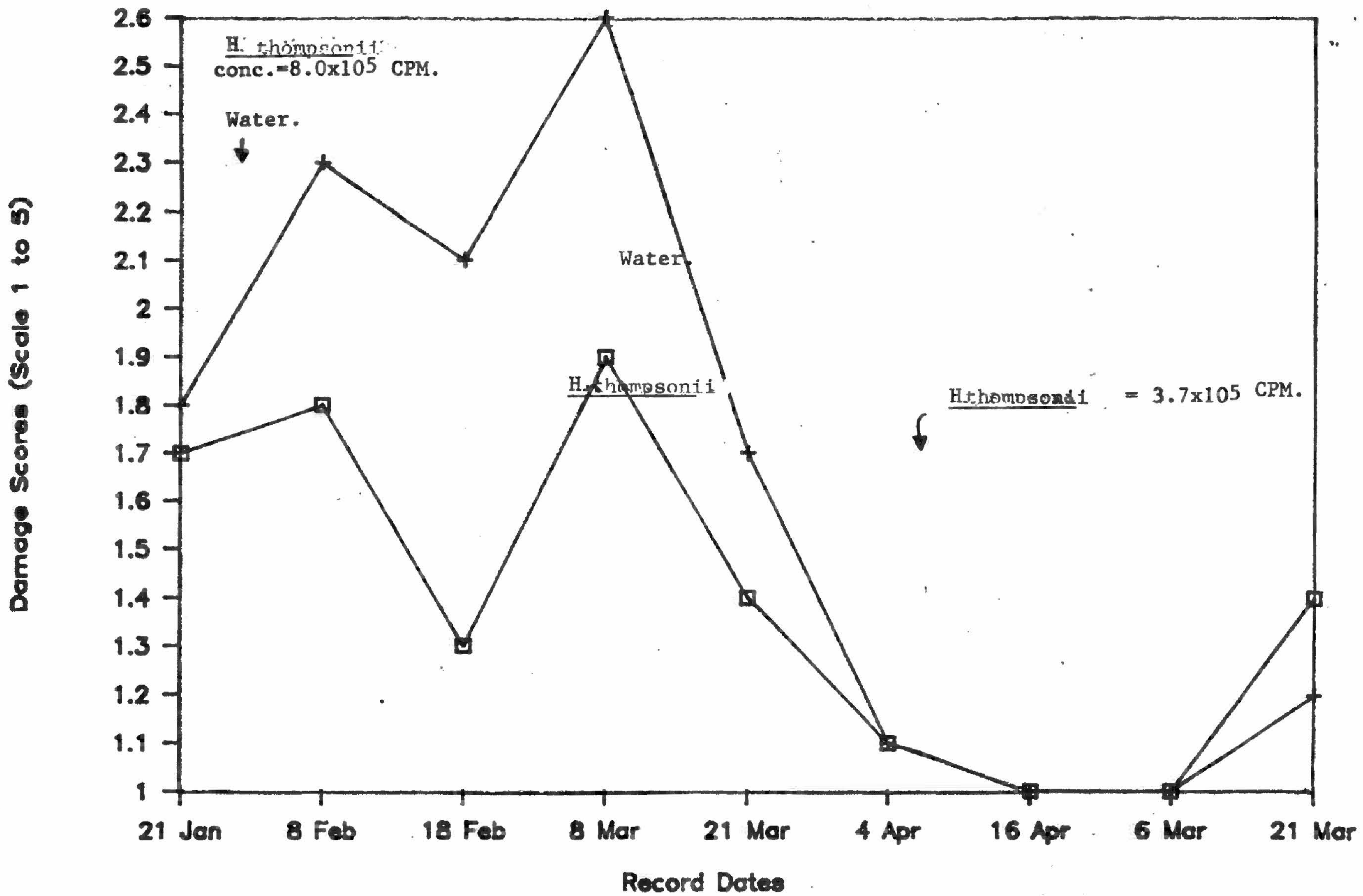

Figure 22. CGM damage levels on leaves of field cassava.

of Jan., 8th and 18th of Feb. respectively. Comparatively, leaves on the control cassava had scores of 1.8 (sd=0.7) , 1.9 (sd=0.8) and 2.0 (sd=0.5) on the same dates (Fig. 22 and Appendix 23). The damage scores in the two treatments did not significantly differ from each other over the period. Scores on the 8th and 21st of March were 1.9 (sd=0.4) and 1.4 (sd=0.3) on the *H. thompsonii* treated plants and were significantly lower than those from the control, 2.6 (sd=0.6) and 1.7 (sd=0.6) on the same dates (P=0.0001).

4.4.5 Effect of Weather on CGM Infestation Level.

Regresion analyses of weather factors affecting the number of living mites, eggs and damage indices of CGM revealed that only the frequency of the rainy days reduced the recordings. The relationships were $r=^{-}0.695^{*}$ and $r=^{-}0.700^{*}$ for living mite numbers and eggs respectively. Temperature and rainfall totals did not show a significant correlation with the levels of pest infestation (Table 9, Appendx 25).

Table 9. Regression analysis of weather factors influencing level of attack of cassava green mite (*M. tanajoa*).

	Temp.(^{0}C)	No. of rain days	Rainfall total
CGM Dam.	___	$r={}^{-}0.256^{ns}$	$r=0.111^{ns}$
CGM pop.	$r=0.232^{ns}$	$r={}^{-}0.695^{*}$	$r={}^{-}0.443^{ns}$
CGM eggs	$r=0.149^{ns}$	$r={}^{-}0.700^{*}$	$r={}^{-}0.604^{ns}$

* Level of significance=0.05%; ns=not significant.

4.4.6. Distribution of M. tanajoa on Leaves of Field Cassava Plants.

The distribution of mobile *M. tanajoa* on leaves of field cassava plants is shown in Fig. 23 and Appendix 26. Mite numbers on leaves in positions 1, 3, and 5 were generally higher than those from leaves in positions 7 and 9. On the 25th of Jan. for instance, leaf 1 had 165.5 (sd=123.9) mites; leaf 3, 253.6 (sd=140.2); leaf 5, 262.2 (sd=224.3); leaf 7, 201.5 (sd=248.3) and the lowest, and oldest, leaf number 9, had 76.3 (sd=87.1). This stratification of CGM counts was to be found even at later dates, e.g at the peak of pest infestation on the 18th of Feb., there were 282.1 (sd=223.8), 210.1 (sd=166.7), 401.1 (170.8), 109.6 (sd=105.4) and 73.9 (sd=89.3) mites (mean counts) on leaves 1, 3, 5, 7, and 9 respectively. The details of analysis of variance for CGM distribution on various leaf positions and dates are presented in Appendix 26.

4.4.7 M. tanajoa Egg Counts on Leaves 1 to 9 of Field Cassava Plants.

M. tanajoa egg distribution followed a similar trend to that noted earlier noted with the mobile active stages of the pest. The highest numbers of eggs were consistently recorded from leaves in positions 1, 3 and 5 (Fig. 24 and Appendix 27). On the 25th of Jan, the mean egg counts were 324.4

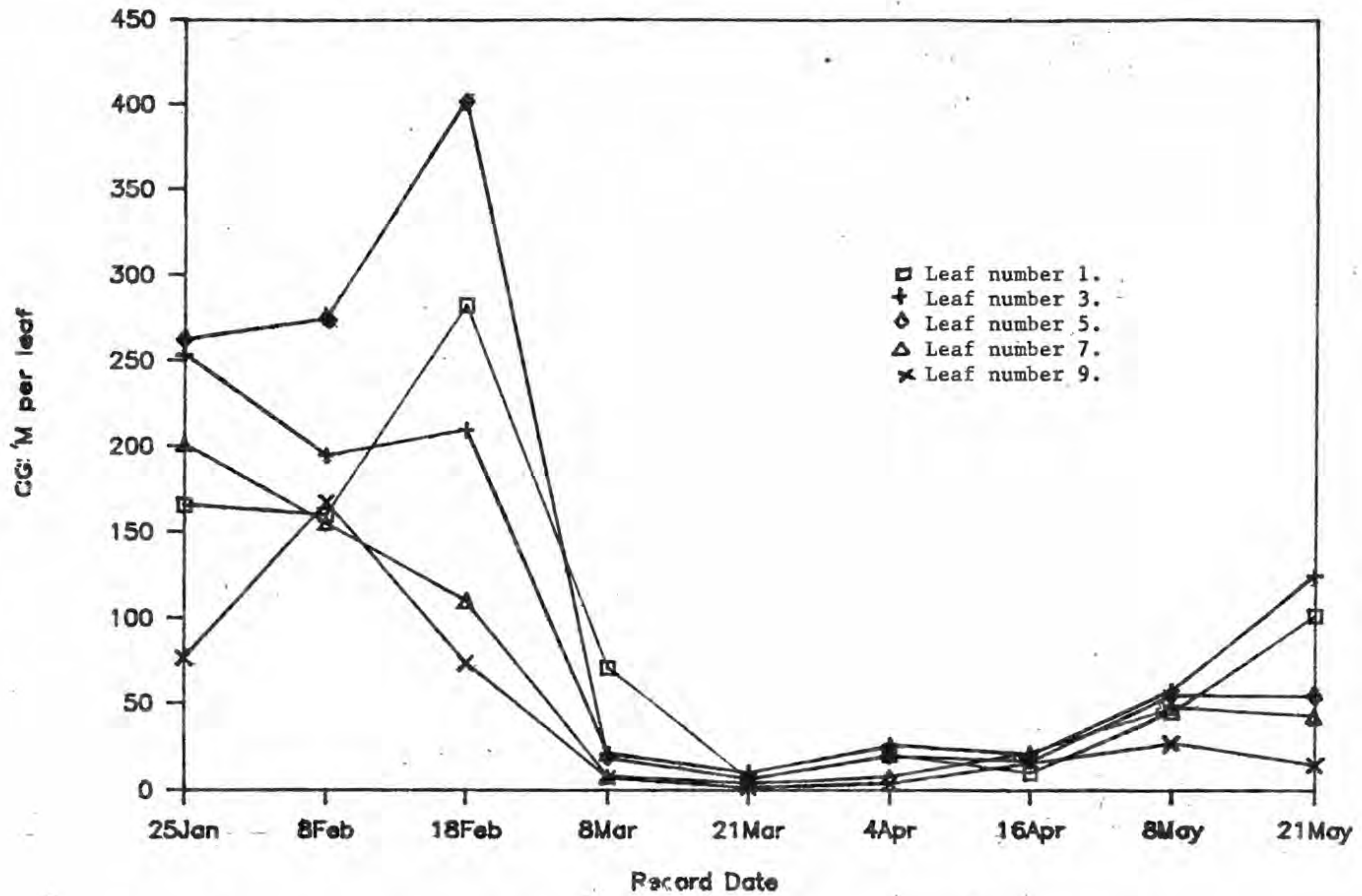

Figure 23. Distribution of CGM on leaves 1 to 9 of cassava planted in the field.

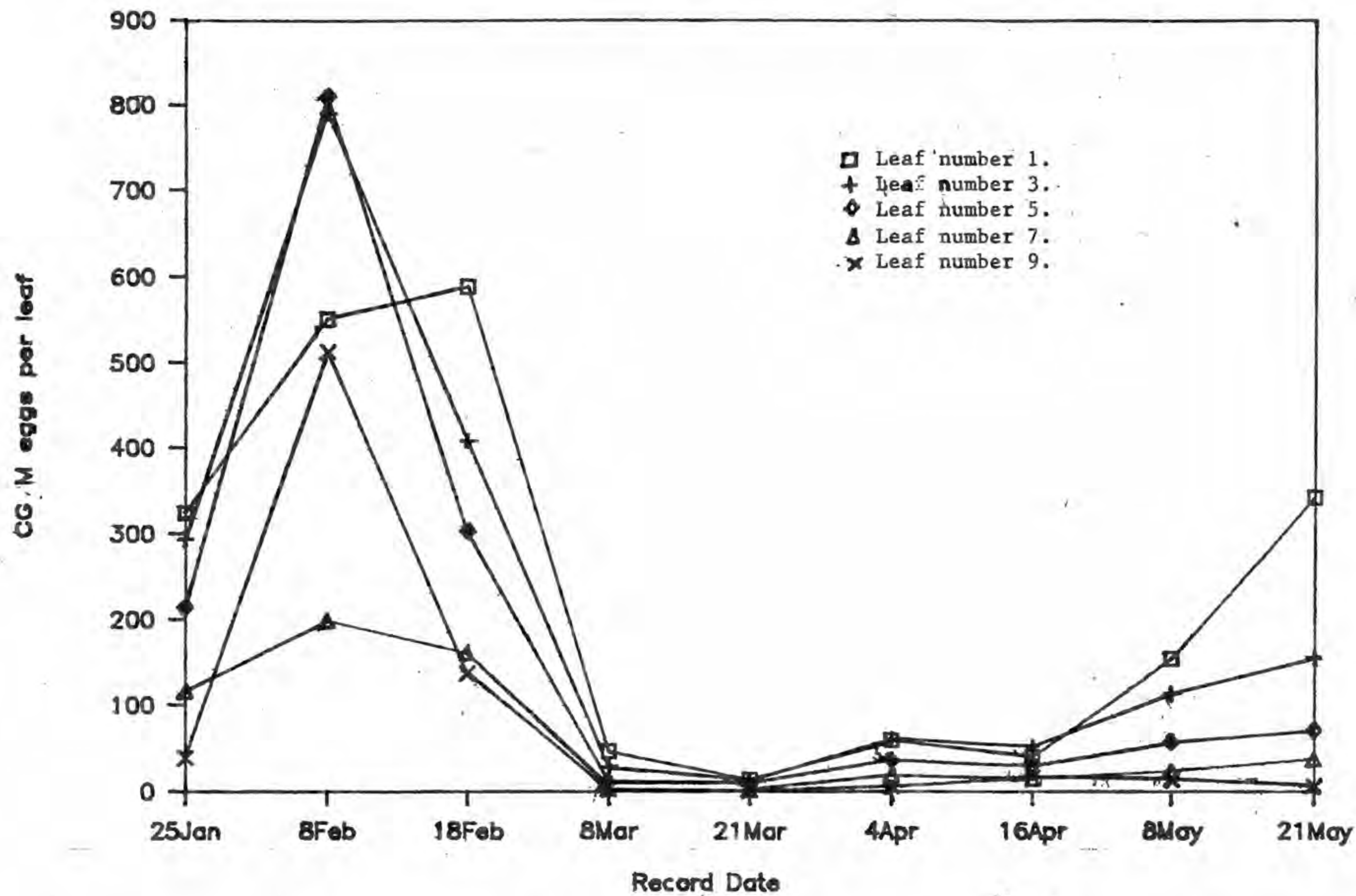

Figure 24. CGM eggs on leaf no. 1 to 9 of cassava planted in the field.

(sd=213.3), 293.6 (sd=213.1), 214.1 (246.0), 115.7 (sd=203.2) and 38.6 (sd=58.7) for leaves 1, 3, 5, 7 and 9 respectively. When most of the egg counts reached their peak on the 8th of Feb., the mean number of eggs on leaves 1, 3, 5, 7 and 7 were 550.3 (sd=325.9), 789.2 (sd=461.8), 808.4 (sd=606.9), 198.2 (sd=223.3) and 512.8 (sd=572.9) respectively. On the 21st of March, when the lowest CGM egg counts were made on cassava leaves, the trend was maintained with the following means 13.5 (sd=24.6), 12.2 (sd=21.4), 9.7 (33.2), 1.1 (sd=4.2) and 0.9 (sd=0.5) eggs per leaf in positions 1, 3, 5, 7 and 9 respectively.

4.4.8 *M. tanajoa* Cadavers Distribution on Field Cassava Leaves 1 to 9.

Leaf numbers 5, 7 and 9, i.e the lower and older leaves, generally had the highest number of cadavers for most of the record dates (Fig. 25 and Appendix 28). On the 25th of Jan., mean cadaver readings of 9.8 (sd=14.8), 21.2 (sd=29.1), 28.4 (sd=31.7), 32.5 (sd=35.5) and 21.7 (sd=27.1) per leaf on leaves in positions 1, 3, 5, 7 and 9 were obtained. On the 8th of Feb., counts made in the same leaf positions gave 170.0 (sd=263.3), 178.4 (sd=230.8), 294.7 (sd=279.4), 272.2 (sd=359.9) and 243.7 (sd=272.6) cadavers per leaf. On the 18th of

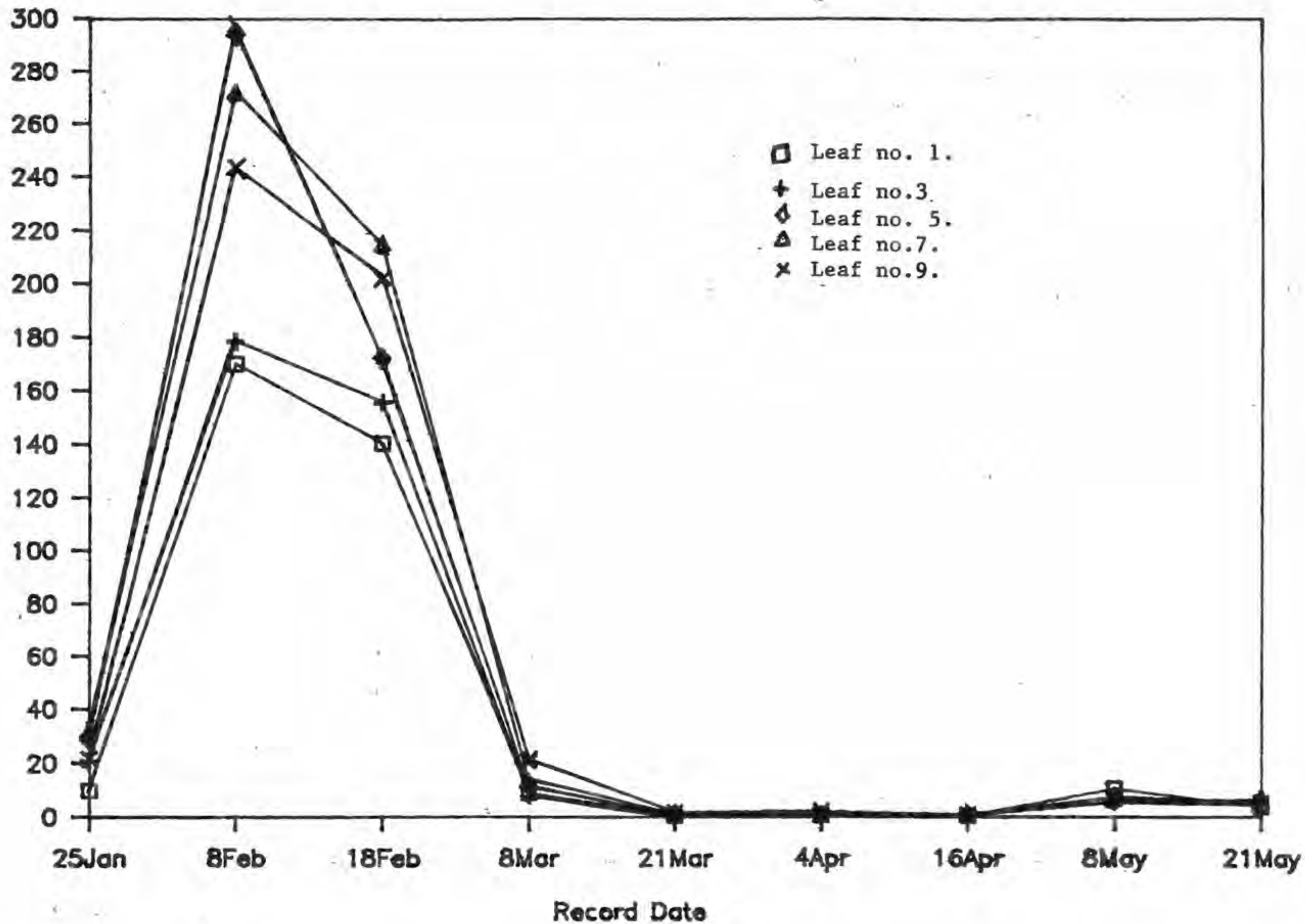

Figure 25. CGM cadaver counted on leaf no. 1 to 9 of cassava plants in the field.

Feb., leaves 1, 3, 5, 7 and 9 had 140.3 (sd=133.9); 155.9 (sd=175.3); 172.4 (sd=196.7); 214.9 (sd=228.2) and 202.3 (sd=94.9) mean cadavers per leaf respectively. Some of the lowest means were recorded on April the 4th with 0.6 (sd=1.3), 1.2 (sd=2.2), 0.9 (sd=1.4), 0.8 (sd=1.4) and 2.2 (sd=2.9) cadavers per leaf, from leaves 1, 3, 5, 7 and 9,

4.4.9. *M. tanajoa* Damage Index on Field Cassava Leaves 1 to 9.

The *M. tanajoa* damage scores increased from the younger leaves to the older leaves, down the cassava plants (Table 10). On the 25th of Jan, leaves 1, 3, 5, 7 and 9 had damage scores 1.6 (sd=0.5), 1.7 (sd=0.6), 2.1 (sd=0.3), 2.3 (sd=0.4) and 2.7 (sd=0.6) respectively, the damage levels on leaves 1 and 3 being significantly lower than leaves 5, 7 and 9 ($P=0.0001$). Following treatment application, the leaf damage scores recorded on the 8th of Feb., varied but at a lower level, between 1.7 on leaves 1 and 3 up to 1.9 on leaf 9. From late March until the 21st of May, damage indices were low and ranged between a maximum of 2.0 recorded on the lower, older leaves (7 and 9) to 1.0.

Table 10. CGM damage index on leaves 1 to 9 of field cassava plants; n=30.

Record Date (1988).	CGM damage index on leaf					F Value	P>F	CV
	1	3	5	7	9			
25 Jan	1.6^{d}	1.7^{d}	2.1^{c}	2.3^{b}	2.7^{a}	33.30	0.0001	20.3
	sd=0.5	0.6	0.3	0.4	0.6			
8 Feb	1.7^{a}	1.7^{a}	1.8^{a}	1.9^{a}	1.9^{a}	1.50	0.2078	31.6
	sd=0.8	0.7	0.7	0.9	0.8			
18 Jan	2.0^{a}	1.9^{a}	1.9^{a}	2.0^{a}	2.0^{a}	1.21	0.3097	20.7
	sd=0.5	0.5	0.5	0.6	0.6			
8 Mar	2.1^{a}	2.2^{a}	2.3^{a}	2.3^{a}	2.3^{a}	1.13	0.3438	23.3
	sd=0.3	0.3	0.4	0.3	0.4			
21 Mar	1.1^{b}	1.2^{b}	1.3^{b}	2.0^{a}	2.0^{b}	22.65	0.0001	32.2
	sd=0.4	0.4	0.4	0.7	0.4			
4 Apr	1.0^{b}	1.0^{b}	1.0^{b}	1.1^{ab}	1.3^{a}	3.84	0.0058	29.4
	sd=0.0	0.1	0.0	0.4	0.5			
16 Apr	1.0^{a}	1.0^{a}	1.0^{a}	1.0^{a}	1.0^{a}	0.90	0.1112	10.5
	sd=0.0	0.0	0.1	0.0	0.0			
6 May	1.1^{a}	1.1^{a}	1.0^{a}	1.0^{a}	1.0^{a}	1.60	1.1790	12.3
	sd=0.3	0.3	0.3	0.1	0.1			
21 May	1.3^{a}	1.3^{a}	1.3^{a}	1.3^{a}	1.2^{a}	0.78	0.5399	26.9
	sd=0.1	0.1	0.1	0.1	0.1			

* Values bearing the same letter are not significantly different, under DMRT statistics.

5. DISCUSSION.

5.1. Mode of Action and Symptoms of *H. thompsonii* Infecting Eggs and Mature Female CGM.

Phase contrast micrographs made in the laboratory did not reveal that *H. thompsonii* cnidia grew on the surface of, nor penetrated into, CGM eggs. However, eggs treated at the one day old stage with the fungus had a slightly, (but not significant) lower percent hatch than the ones from the control. The result showed that it is probable that the egg shell coating prevented fungal growth and penetration into the egg. The exocellular proteolytic, chitinolytic or lipolytic enzymes previously reported by Huber (1958) and Hall (1981) to be the mechanisms used by the fungus for gaining entry into the insect or mite body, therefore, did not seem to be effective on the eggs.

The change of behaviour of *H.thompsonii*.infected female *M. tanajoa* from active to sluggish movement, and the eventual colour change of cadavers observed here was similar to that described from *P. oleivora* (Ashmead) (Fisher 1950) infected with *H.thompsonii* (Muma 1958; Burditt *et al.*, 1963). Changes in the colour of the integument of the

blueberry bud mite infected with *H. thompsonii* had been similarly observed by Baker and Neunzing (1968).

In assessing the disease symptoms associated with the *H. thompsonii* infection, the cadavers had to be incubated in a high humidity environment to promote fungal germination and penetration into the mite tissue and the eventual conidiation.

No significant difference was realised in the number of eggs laid per female CGM treated with either *H. thompsonii* or water in the humidity chamber environment. However, investigations conducted in the laboratory showed that there was a significantly lower number of egg counts per female infected with *H. thompsonii* than from the control. The difference in records from the two environments may have occurred as a result of differences in the means of temperature and relative humidity in the two environments, i.e. laboratory temperature=20.2^{0}C (sd=6.5) and RH=75% (sd=5.4); humidity chamber temperature=23.8^{0}C (sd=5.8) and RH=95% (sd=4.0). The rather lower mean laboratory temperature (difference=3.8^{0}C) and relative humidity (difference=19.5%), compared with those from the humidity chamber, could have favoured *H. thompsonii* infectivity and the resulting high mortality among the CGM female test

population. At the same time, the rate of egg laying by female CGM could have been reduced under the cooler laboratory conditions as compared to the rate onder the warmer humidity chamber conditions.

5.2. Effect of *H. thompsonii* on Developmental Stages of *M. tanajoa*. Laboratory Studies.

Symptoms of ***H. thompsonii*** attack which was earlier given in this report was clearly demonstrated on larvae killed by the fungus. This means that the symptoms are reliable enough to enable one to distingush between cadavers that had been killed either by the entomogenous fungus or by other natural causes. It was also noted in this work that due to the short period between each larval instar, there was a relatively lower death rate caused by the fungus on these young stages, than was the case with adult female mites. This phenomenom could be understood on the grounds that the larval and nymphal moulting intervals are usually very short, between 1 to 3 days only (Yaseen and Bennett, 1977). For this reason, any fungal spores that might have landed on the larval cuticle, at the time of spray, and had started germinating would most probably be removed with

the skin cast before it could penetrate deeper into the body tissue. The opposite argument could explain why female CGM were more susceptible to the disease causing fungus than the larvae, that is, since the adults normally do not cast off their skins, the fungus can grow into, and destroy the body tissue of the mite.

H. thompsonii was very effective in reducing the number of eggs laid per female CGM. This may be due to a number of factors (1) CGM attacked by *H. thompsonii* usually become weak since the infected mites develop a low appetite (2) there is usually high mortality among the infected egg laying females.

M. tanajoa female adult mites exposed to *H. thompsonii* in both the laboratory and humidity chamber and had higher mortality levels than the corresponding control population. These results were akin to those of Gardner *et al.*, (1982), who, after topically applying conidia of *H. thompsonii* (Commercial form ABG 6065) onto *Tetranychus utricae*, obtained mortalities of up to 96.54% sd=3.54, due to the fungus mycosis. They obtained no death from the control treatment. The majority of the mites died 3 to 5 days after exposure to the conidia, which also corresponded to the findings of this report. The two results also

agree on the fact that, after death, the fungal hyphae rapidly penetrated the host tissue and sporulated. The same commercial mycoinsecticide caused mortalities of between 73 to 100% in a population of *Oligonychus ilicis*. Conflicting evidence has, however, been advanced by Impe and Pierard (1985), who conducted a laboratory bioassay on *H. thompsonii* on *T. utricae*, directly rubbing, or spraying suspensions of the conidia onto the mites. They found that the fungus did not actively attack living mites, and therefore concluded that the fungus was a saprophyte which quickly invaded bodies of mites which died from other causes.

It is important to note that eggs laid by female *M. tanajoa* treated with the fungus had a lower percentage hatch than those exposed directly to the fungus. This could imply that *H.thompsonii*, while attacking the body of a female mite, has, either directly or indirectly easier access to the unlaid soft and delicate eggs, than on the eggs which are already laid. The latter are protected with a hard egg shell coating which resists attack by the fungus. However, the exact effect of on the fungus on the lowering of CGM egg hatchability could not be fully established in this study.

5.3. *Effect of H. thompsonii on M. tanajoa Infesting Cassava Plants Grown in Pots.*

It is apparent that shifting cassava plants from from the open sunshine where they were grown, into the cages, a relatively shady environment, was responsible for the general CGM population drop in all three treatment plants. This also explains why the cadaver counts per leaf generally rose particularly on *H. thompsonii* treated plants. Open sunshine seems to be good for the growth of both plants and their pests, the CGM. The pests then readjusted to the new condition, with the population steadying at a lower level than was observed in the sunshine environment. *H.thompsonii* treatment on the infested plants caused an immediate lowering of the pest counts per leaf. Pest counts from the control leaves was higher for the corresponding time (Appendix 1). As was reported earlier, the fungus could have reduced egg counts by either (1) directly killing female CGM or (2) by reducing the number of eggs laid per female. The effect of *H. thompsonii* on CGM infesting caged cassava plants was comparable to,

and, on a number of occasions, better than the levels of control exerted by the phytoseiid mites. This was especially noticable from the time of the second fungus application (concentration $3.7x10^5$ conidia per ml.), when a drastic reduction in the pest population was realised for the whole month of April and part of May (Appendix 5).

Despite the present report on the positive effects of *H. thompsonii* on CGM infesting potted cassava plants was obtained. Gardner *et al.* (1982) found that *H. thompsonii* (Commercial form 6065) was not pathogenic on *T. utricae* infesting Irish shamrocks (*Oxalis acetocella* L.). This could mean that *M. tanajoa* is more susceptible to *H.thompsonii* than to *T. utricae*.

In this experiment,there was a problem of how to prevent *A. teke* from migrating to plants where *H. thompsonii* and water were sprayed. This movement was especially marked towards the end of the experimental period. The predators could have invaded the cassava at the time when the plants were exposed to sunshine for hardening. It must be noted that this invasion could have marred the effects of *H. thompsonii* and the actual treatment of *A. teke* with respect to the control.

Standard deviations were high for the egg, mite and cadaver counts. However, results shown on

figures 17 and 18 where there was no significant difference between the widely varying means of eggs and mobile CGM from the three treatments can not be explained on high standard deviations alone. This calls for a repetition of the experiment. Increasing the sample size could probably have helped in reducing errors attributed to aggregation nature of the pests on leaves and plants. In this case, log(count+1) transformed values greatly helped in reducing the coefficient of variation in the statistical analyses.

5.4. Pathological Efficacy of *H. thompsonii* on CGM Infesting Field Cassava.

Application of *H. thompsonii* for the control of *M. tanajoa* on field cassava plants caused a significant reduction in the number of eggs, and active live mites found on the plants within one month after treatment. Application of the fungus at times of peak pest infestation, which was also the driest and hottest part of the year, were very effective, the conditions seemingly not reducing the effectiveness *H. thompsonii* on the pest. The result conforms to an earlier argument advanced by Doberski (1980), that insect pests can be infected with fungal diseases even at relative humidities

much lower than those normally observed in the laboratory. The CGM damage index was lowered by *H. thompsonii* application nearly one and half months after the mycoinsecticide was introduced. This is because the plants take several days to develop new CGM-symptom-free leaves following the pest elimination.

The higher CGM and egg counts recorded on 'protected' cassava plants following the rains when compared with those taken on the control plants, may be explained by a number of reasons (1) mites may have migrated from the relatively more damaged control plants onto the relatively cleaner 'protected' ones at a time when (2) rainfall had washed off the mite cadavers containing *H. thompsonii* inoculum; and (3) the concentration of conidia suspension used in the second *H. thompsonii* treatment may have been too low to cause any reduction in the pest numbers.

Rainfall was a density independent mortality factor as its appearence resulted into indiscriminate reduction on the scores of both treatments (Appendix 21 and 22).

The highest cadaver counts were recorded from the *H. thompsonii* protected cassava plants as was to be expected, since the the fungus was pathogenic to mites on these plants and was absent

from the control plants. However, the difference between the two treatments was, in most cases, not statistically significant due to the high standard deviations values. Rainfall also lowered the cadaver counts, just as it did with eggs and active mite stages.

Records on the distribution of CGM eggs and active stages on various leaf positions (1 to 9), showed that the upper, younger leaves held a higher number of eggs than the older ones. Such a spatial distribution of pests has, in most cases, been attributed to the abundance of more nutritious food substances in the younger leaves (Yaseen and Bennett, 1977). In general, leaf feeding tetranychids like for example, *Oligonychus punicae* (Hirst), a pest of *Persea induca*, always tend to migrate from heavily infested leaves to younger and healthier ones (McCurthy, 1970). This argument also explains why there were more cadavers on the older leaves than on the younger ones. This indicated that while active and younger mites tend to move upwards for better feeding and breeding sites, the aged group in the population usually remain on the lower leaves of the plants where they finally die.

Results from the four and half months observation period showed that, for actively

growing plants, CGM damage indices tend to be lower on the upper younger leaves than on the lower leaves. This implied that the upper leaves are capable of of replacing the chlorophyll cells which have been damaged by the sucking mites whereas in the case of older leaves this replacement capability has already been spent.

Reports on the effects of *H. thompsonii* on beneficial arthropods and mammals showed that an application of suspensions of conidia at a concentration of 1.49×10^6, per ml did not cause infections of either larvae or and adults of *Coccidophilus oleivora* and *Lindorus lopihanthas* (=*Rhyzobius lophanthas*), both coccinelid predators on *Phyllocoptruta oleivora*, a pest of citrus in Argentina (Soza Gomez *et al.*, 1985).

The beneficial fungus, *H. thompsonii*, is not harmful to man as pertains to its toxicity, infectivity and allergicity (Ignoffo, 1981). This is one of the factors to consider, since various parts of a cassava crop, particularly the roots and leaves, are used as foodstuff for man and his domestic animals in processed and even unprocessed forms (Coursey, 1978; Lutaladio, 1983).

In conclusion, as a result of the apparent safety of the fungus, and its ability to afford protection to infested cassava plants, it appears

to represent an excellent candidate organism for use in a CGM control programme.

6. SUMMARY OF RESULTS.

1). Phase contrast microscopy did not reveal any *H. thompsonii* fungal growth on eggs of CGM.

2). *M. tanajoa* eggs exposed to *H. thompsonii* conidia under laboratory conditions, exhibited a reduction in percentage hatch from 3.2 to 21% when compared with the control.

3). Death of CGM females was recorded from 48 through 96 hours after treatment when up to 100% mortality was realised.

4). Infected, dead CGM changed colour from a creamy appearence to brown and then dark-brown from 48 to 120 hours after death (HAD).

5). From 72 HAD and afterwards, the cadavers characteristically burst open, dried and were glued onto the substratum.

6). Conidiation of *H. thompsonii* on cadavers could be demonstrated from 72 HAD and afterwards.

7). *H. thompsonii* invaded the body of the mite host by penetrating into its tissue by means of its hyphae.

8). Significantly more CGM larval cadavers from a *H. thompsonii* treated population developed the fungus infection symptoms than those from the control.

9). The rate of egg laying by CGM females infected with *H. thompsonii* was reduced. For example, in the laboratory, a mean of 5.8 and less than 1.0 eggs were laid per female CGM treated with water and *H. thompsonii* respectively. These means were significantly different from each other.

10). Application of various concentrations of *H. thompsonii* ranging from 10.4×10^7 conidia per ml down to 0 (control water spray) on female CGM reared in the laboratory and the growth chamber, did not indicate there was a significant dose/mortality response by the treated mites.

11). *H. thompsonii* concentration 8.0×10^5 CPM reduced the number of CGM eggs counted per leaf on potted cassava plants by 128 times. *A. teke* and the control plants each had reduced egg counts per

leaf by a factor of 3, all records being taken two weeks after treatment application.

12). *H. thompsonii* at a concentration of $3.7x10^5$ conidia per ml. also significantly lowered the number of living CGM counted per leaf of potted cassava. The protective effects of the fungus compared favourably with those of *A. teke*;

13). Difficulties were encountered in restricting the movement of *A. teke* between plants of all treatments. Thus they were sometimes found even on the control plants. The phytoseiids may have acted as an extra mortality factor in the treatments and hence could have contributed to the small difference between the treatments and also for the short duration of these differences.

14). The CGM damage index on potted cassava plants improved on those treated with *H. thompsonii*, beginning 1 week after treatment (WAT) and lasting for nearly 1 month. The improvement as a result of treatment with *A. teke*, on the other hand, was noticable from the 3rd WAT and was observed for only 2 weeks.

15). There was no steady and consistent count of CGM cadavers per leaf of cassava planted in pots and treated with *H. thompsonii, A. teke* or water.

16). *H. thompsonii* application caused a 63% reduction in the number of eggs counted per leaf of field cassava.

17). The number of living mites per leaf of field cassava was lowered by up to 77% following application of *H. thompsonii* at a concentration $8.0x10^5$ conidia per ml.

18). The effects of *H. thompsonii* on the number of eggs and living CGM found on leaves of field cassava were recorded within 2 WAT. During this same period, counts from the control cassava were generally higher than those from the protected plants.

19). The damage index on leaves of field cassava protected by application of suspensions of *H. thompsonii* was reduced by between 18 to 37% from the third through to the seventh WAT.

20). For about two and half months, the mean CGM cadaver counts per leaf of field cassava were

higher on *H. thompsonii* treated plants than on those plants sprayed with water.

21). The number of rainy days in a given period negatively and significantly affected CGM counts. It also reduced the level of CGM damage symptoms. Temperature and rainfall totals did not show such a correlation. These factors may be important when designing an intergrated pest management approach for CGM.

22). Most live mites and eggs were found on the upper and younger leaves number 1 to 5. Sampling for the effectiveness of a mycoinsecticide could, therefore, be concentrated on these upper leaves only.

23). Dead CGM were more abundant on the lower older leaves from from 7 to 9.

7. CONCLUSION.

The results presented here are the first of their kind to report on the field testing of *H. thompsonii* applied for the control of cassava green mite, *M. tanajoa* Bondar, a pest which is unique to the cassava crop, *M. esculenta* Cranzt in Africa. The fungus has been demonstrated to cause high pest mortality both in laboratory bioassays and in the field. Further investigations on the pathogenicity of the fungus on beneficial arthropods which attack *M. tanajoa*, or those that are helpful in various agro-ecological systems, should be made in order to determine the safety of this potential mycoinsecticide. Since *H. thompsonii* is found naturally in the environment, it does not seem to to present too great an ecological risk. So are *Bacillus anthricis, Salmonella Pseudomonas aeruginosa, Tetanus spp., Serrata marcescens* etc - virulent disease organisms. There is a chance that, with the current knowledge on genetic manipulation techniques, the virulence of this beneficial fungus could be further increased. Alternatively, following development of cheap and appropriate methods of mass production, the fungus may be

applied to synergise other control measures as
part of an intergrated pest management strategy.

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6. APPENDICES

Appendix 1. ***M. tanajoa*** **egg counts per leaf of potted 'Kibandameno' cassava treated with: *H. thompsonii* (F); *A. teke* (P) and water(C).**

Date	*M. tanajoa* eggs on treat. F	P	C	F Value	P>F	CV
6/01	$(6.7)^{a}$	$(7.7)^{a}$	$(7.4)^{a}$			
	1620.0	3987.0	2466.5			
*12/01/'88 *H. thompsonii* =8.0×10^{5}CPM;*A. teke*=10 per plant(PP)						
19Jan	$(1.1)^{b}$	$(4.0)^{ab}$	$(6.4)^{a}$	8.25	0.0381	26.4
	6.5	271.9	835.0			
	sd=8.6	480.2	500.5			
5 Feb	$(2.4)^{a}$	$(1.8)^{ab}$	$(0.1)^{b}$	7.37	0.0456	87.7
	41.1	17.0	0.2			
	sd=23.7	15.8	0.4			
2 Mar	$(5.5)^{a}$	$(4.6)^{a}$	$(5.1)^{a}$	1.02	0.4387	26.3
	593.5	174.3	434.3			
	sd=466.2	129.0	525.6			
25 Mar	$(5.7)^{a}$	$(6.0)^{a}$	$(5.6)^{a}$	2.06	0.2426	14.9
	593.7	732.4	595.2			
	sd=490.0	532.0	495.2			
*06/04/'88. *H. thompsonii* =3.7×10^{5} CPM; *A. teke*=10 PP.						
11Apr	$(3.8)^{a}$	$(3.9)^{a}$	$(6.6)^{a}$	1.88	0.2660	25.9
	174.1	197.7	1090.3			
	sd=194.4	181.8	695.2			
19Apr	$(2.3)^{a}$	$(3.0)^{a}$	$(3.7)^{a}$	0.28	0.7674	43.2
	117.6	202.7	84.0			
	sd=170.2	297.7	76.2			
8 May	$(2.9)^{a}$	$(4.6)^{a}$	$(0.7)^{a}$	5.95	0.0633	42.4
	44.7	157.7	6.7			
	sd=30.3	70.9	6.7			

*** Dates when the treatment levels shown were applied;** CPM=conidia per ml; means of (log(count+1)) in parentheses **with the same letter are not significantly different under Duncan's Multiple Range Test (DMRT) (1955); actual mean** count and standard deviations are indicated; n=15.

Appendix 2. *M. tanajoa* eggs per leaf of 'Ratenyi' cassava planted in pots.

Date	*M. tanajoa* eggs on treat. F	P	C	P>F	F Value	CV
30 Dec	(5.5)[a]	(5.7)[a]	(6.0)[a]	0.7980	0.24	13.8
	358.5	460.1	427.8			
	sd=243.8	69.3	206.9			
6 Jan	(5.7)[a]	(4.8)[a]	(5.3)[a]	0.5847	0.62	17.5
	516.1	423.0	422.9			
	sd=523.9	495.2	453.8			
12/01/'88. *H. thompsonii* .=8.0x10^5 CPM; *A. teke*=15 PP.						
19 Jan	(2.2)[a]	(4.7)[a]	(2.9)[a]	0.1807	2.71	44.7
	43.5	418.0	99.7			
	sd=63.7	743.3	108.0			
5 Feb	(4.7)[a]	(1.8)[a]	(2.7)[a]	0.0628	5.98	46.0
	182.4	29.5	178.1			
	sd=125.3	44.0	284.3			
2 Mar	(4.6)[a]	(5.0)[a]	(6.6)[a]	0.2374	2.10	24.0
	201.9	392.8	870.1			
	sd=293.7	515.5	575.5			
25 Mar	(5.5)[a]	(4.7)[a]	(5.5)[a]	0.8684	0.15	21.9
	675.8	366.6	841.4			
	sd=731.0	370.7	631.0			
* 06/04/'88. *H. thompsonii* =3.7x10^5 CPM; *A. teke*=10 PP.						
11 Apr	(4.7)[a]	(3.4)[a]	(5.5)[a]	0.4586	0.95	21.2
	639.0	73.1	535.6			
	sd= 550.0	86.5	550.4			
19 Apr	(2.6)[a]	(0.6)[a]	(4.1)[a]	0.0809	5.03	64.1
	70.1	49.2	168.3			
	sd=98.0	85.2	203.7			

* Dates when indicated levels treatments were applied; means of (log(count+1))in parentheses-with the same letter are not significantly different under DMRT; actual mean counts and standard deviations are indicated; n=15.

Appendix 3. *M. tanajoa* eggs on leaves 1 to 5 of potted 'Kibandameno' cassava plants.

Date	*M. tanajoa* eggs per leaf					F Value	P>F	CV
	1	2	3	4	5			
6Jan	$(7.3)^{a}$	$(7.8)^{a}$	$(7.8)^{a}$	$(6.7)^{a}$	$(6.8)^{a}$			
	5112.9	2966.0	2895.1	1292.6	4072.2			
	sd=2745.5	1270.1	1592.9	770.8	4797.6			
19Jan	$(3.5)^{a}$	$(4.1)^{a}$	$(4.2)^{a}$	$(3.5)^{a}$	$(3.8)^{a}$	0.92	0.4678	26.4
	191.2	338.7	523.9	581.5	440.6			
	sd=221.1	134.1	331.2	414.9	423.1			
5 Feb	$(1.4)^{ab}$	$(0.9)^{b}$	$(0.7)^{a}$	$(2.6)^{a}$	$(1.7)^{ab}$	2.96	0.0434	87.7
	9.6	3.8	2.8	46.8	27.5			
	sd=8.4	3.3	3.0	43.2	41.1			
2Mar	$(5.9)^{a}$	$(5.8)^{ab}$	$(5.2)^{ab}$	$(4.4)^{ab}$	$(4.0)^{c}$	3.67	0.0117	26.3
	927.9	576.4	258.8	125.8	114.8			
	sd=1027.9	359.5	158.5	116.1	100.1			
25Mar	$(6.5)^{ab}$	$(6.8)^{a}$	$(5.9)^{bc}$	$(5.1)^{cd}$	$(4.6)^{d}$	10.8	0.0001	14.9
	1227.0	1205. 0	456.6	220.3	129.1			
	sd=1188.7	834.2	369.2	48.3	88.2			
11Apr	$(5.4)^{a}$	$(5.5)^{a}$	(4.8)a	$(4.6)^{ab}$	$(3.6)^{b}$	3.50	0.0220	25.9
	927.2	628.6	357.5	307.4	216.2			
	sd=464.4	563.6	396.4	164.4	196.7			
19Apr	$(4.2)^{a}$	$(3.4)^{ab}$	$(3.3)^{ab}$	$(2.5)^{bc}$	$(1.7)^{c}$	4.66	0.0063	43.2
	287.6	168.5	70.0	125.7	22.2			
	sd=379.7	246.8	64.6	189.2	26.6			

* Means of (log(count+1))in parentheses-with the same letter are not significantly different under DMRT; actual mean and standard deviations are indicated; n=9.

Appendix 4. *M. tanajoa* eggs on leaf number 1 to 5 of potted 'Ratenyi' cassava plants.

Date	*M. tanajoa* eggs per leaf					F Value	P>F	CV
	1	2	3	4	5			
30 Dec	$(4.6)^{c}$	$(4.5)^{b}$	$(6.0)^{ab}$	$(6.4)^{a}$	$(5.9)^{ab}$	6.70	0.0009	13.8
	191.3	338.9	523.9	581.6	446.8			
	sd=247.9	252.2	182.6	87.9	287.7			
6Jan	$(5.5)^{a}$	$(5.4)^{a}$	$(5.3)^{a}$	$(5.3)^{a}$	$(4.8)^{a}$	0.83	0.5202	17.5
	458.9	660.0	399.8	462.9	397.8			
	sd=461.4	961.4	265.4	573.4	538.9			
19Jan	$(4.7)^{a}$	$(3.9)^{ab}$	$(2.8)^{bc}$	$(2.2)^{c}$	$(2.6)^{bc}$	4.48	0.0076	44.7
	433.0	478.1	193.6	80.2	85.1			
	sd=459.1	563.5	302.4	89.3	110.1			
5Feb.	(3.7)a	(3.2)a	(3.4)a	(3.8)a	(1.4)b	4.36	0.0086	46.0
	137.1	129.9	258.3	116.6	8.1			
	sd=141.9	132.2	384.4	90.2	7.5			
2Mar	$(6.6)^{a}$	$(5.9)^{ab}$	$(5.8)^{ab}$	$(4.6)^{bc}$	$(4.1)^{c}$	5.50	0.0027	24.0
	1050.9	611.3	578.7	414.7	337.2			
	sd=742.9	497.8	507.4	322.5	236.6			
25Mar	$(6.4)^{a}$	$(5.1)^{b}$	$(5.3)^{ab}$	$(4.8)^{b}$	$(4.6)^{b}$	3.25	0.0290	21.9
	1276.7	521.5	392.4	221.4	380.0			
	sd=763.0	1036.0	386.1	206.1	205.1			
11Apr	$(5.0)^{a}$	$(4.8)^{a}$	$(4.4)^{a}$	$(4.8)^{a}$	$(3.9)^{a}$	1.74	0.1751	21.2
	787.4	472.4	219.0	397.1	203.7			
	sd=843.8	485.4	245.4	276.8	278.5			
19Apr	$(3.4)^{a}$	$(2.4)^{a}$	$(2.1)^{a}$	$(2.4)^{a}$	$(1.9)^{a}$	1.17	0.3500	64.1
	150.7	36.0	78.9	68.4	66.0			
	sd=357.9	36.9	89.8	56.0	104.4			

Means in parentheses(log(count+1)) with same letter are not significantly different under DMRT; actual egg means and standard deviations are indicated; n=9.

Appendix 5. ***M. tanajoa*** counts on potted 'Kibandameno' cassava plants treated with H. thompsonii (F), ***A. teke*** (P), and water (C).

Date	Mean CGM/leaf on treat. F	P	C	F Value	P>F	CV
*12/01. *H.* =8.0x10^{5}CPM; *A. teke*=15 PP.						
19Jan	(1.2)^a 6.8 sd=5.0	(4.4)^a 561.7 916.8	(6.5)^a 982.4 827.3	6.12	0.0607	23.7
5Feb	(2.2)^a 17.7 sd=12.8	(1.1)^a 4.8 5.4	(0.3)^a 0.6 0.2	4.50	0.0948	65.6
2Mar	(5.7)^a 492.7 sd=281.8	(3.9)^b 101.0 113.3	(3.4)^b 37.3 25.0	28.96	0.0042	25.8
25Mar	(5.1)^a 252.1 sd=157.5	(5.3)^a 311.8 255.3	(5.9)^a 560.4 273.4	6.46	0.0559	15.4
*06/04 H. thompsonii =3.7x10^{5}CPM; *A. teke*=10 PP.						
11Apr	(3.5)^b 70.0 sd=69.2	(4.2)^b 173.3 180.1	(6.9)^a 1202.2 640.5	7.55	0.0438	18.0
19Apr	(2.8)^b 136.7 218.2	(3.0)^a 76.3 103.2	(4.2)^a 121.3 97.2	0.7269	0.35	36.8
8May	(2.1)^{ab} 19.9 sd=14.6	(4.0)^a 102.9 62.7	(0.7)^b 3.7 3.7	7.58	0.0436	51.5

* Dates when the levels of treatments shown were applied; means in parentheses (log(count+1)) with the same letter are not significantly different under DMRT; actual mean counts and standard deviations are indicated; n=15.

Appendix 6. Mean *M. tanajoa* on leaves of 'Ratenyi' potted cassava treated with: H. thompsonii (F); *A. teke* (P) and water(C).

Date	*M. tanajoa*/leaf on treat. F	P	C	F Value	P>F	CV
30Dec	$(4.8)^{a}$	$(5.7)^{a}$	$(5.2)^{a}$	2.16	0.2310	13.1
	153.9	430.7	248.8			
	sd=61.2	276.3	165.4			
6Jan	$(5.0)^{a}$	$(4.0)^{a}$	$(5.6)^{a}$	1.31	0.3645	16.3
	194.9	120.0	202.9			
	sd= 124.2	81.0	124.2			
*12/01. H. thompsonii .=8.0×10^{5} CPM; *A. teke*=15 PP.						
19Jan	$(2.3)^{a}$	$(3.8)^{a}$	$(3.6)^{a}$	0.78	0.5151	42.6
	27.8	198.5	109.0			
	sd=38.7	224.0	111.2			
5Feb	$(4.4)^{a}$	$(1.5)^{a}$	$(3.0)^{a}$	6.63	0.0537	46.0
	136.2	14.7	147.5			
	sd=66.7	19.8	238.3			
2Mar	$(4.0)^{a}$	$(4.5)^{a}$	$(6.5)^{b}$	7.37	0.0456	22.5
	168.9	275.3	761.1			
	sd=132.5	292.2	467.5			
25Mar	$(5.8)^{a}$	(4.2)a	$(6.0)^{a}$	0.87	0.4865	17.3
	555.5	249.3	776.3			

sd & mean counts and standard deviations are indicated; n=15.

Appendix 7. ***M. tanajoa*** distribution on the first five leaves potted 'Kibandameno' cassava plants.

Date	*M. tanajoa* on leaf					F Value	P>F	CV
	1	2	3	4	5			
6/01	(7.1)^{a}	(6.7)^{a}	(7.0)^{a}	(6.6)^{a}	(6.5)^{a}			
	2614.3	623.7	1341.3	1023.4	1741.3			
	sd=1676.3	80.1	359.8	319.9	1191.7			
19Jan	(4.0)^{a}	(4.5)^{a}	(4.2)^{a}	(3.5)^{a}	(3.8)^{a}	1.45	0.2473	23.7
	318.7	656.4	683.1	612.0	314.7			
	sd=334.8	541.3	862.6	902.5	280.7			
5Feb	(0.8)^{a}	(1.2)^{a}	(0.8)^{a}	(1.5)^{a}	(1.4)^{a}	3.15	0.1490	65.6
	3.5	3.7	2.5	12.3	16.4			
	sd=3.4	3.0	2.7	17.0	23.7			
2Mar	(4.7)^{a}	(4.9)^{a}	(4.6)^{a}	(3.7)^{a}	(3.7)^{b}	2.40	0.0783	25.8
	384.9	327.0	194.7	77.9	67.3			
	sd=175.9	261.5	147.1	56.0	52.4			
25Mar	(6.2)^{a}	(6.1)^{a}	(5.5)^{ab}	(4.8)^{b}	(4.6)^{b}	6.60	0.0010	15.4
	683.6	598.3	329.3	114.6	148.1			
	sd=358.9	401.9	254.4	53.0	76.0			
11Apr	(5.2)^{a}	(5.3)^{a}	(5.2)^{a}	(4.9)^{a}	(3.8)^{b}	4.47	0.0077	18.0
	668.1	527.2	591.6	345.8	276.4			
	sd=322.2	107.4	346.9	182.8	199.8			
19Apr	(4.1)^{a}	(3.5)^{ab}	(3.4)^{ab}	(3.1)^{ab}	(2.7)^{b}	1.53	0.2241	36.8
	198.9	102.3	126.1	56.0	50.2			
	sd=209.8	133.7	178.5	63.0	74.3			
8May	(2.2)^{a}	(2.4)^{a}	(2.6)^{a}	(2.2)^{a}	(1.7)^{a}	0.70	0.6027	51.7
	27.3	109.7	28.5	29.8	15.7			
	sd=7.1	86.7	9.7	22.8	8.7			

Means in parentheses (log(count+1)) with the same letter are not significantly different under DMRT; actual means and standard deviations are indicated; n=9.

Appendix 8. *M. tanajoa* on the first five leaves of 'Ratenyi' cassava plants grown in pots.

Date	*M. tanajoa* on leaf 1	2	3	4	5	F Value	P>F	CV
30Dec	(5.2)^{a}	(5.4)^{a}	(5.4)^{a}	(5.3)^{a}	(5.2)^{a}	0.43	0.7876	13.1
	248.8	182.0	319.0	281.7	299.7			
	sd=120.7	263.8	170.9	195.3	249.9			
6Jan	(5.1)^{a}	(4.7)^{ab}	(4.7)^{ab}	(4.4)^{ab}	(4.0)^{b}	2.95	0.0407	16.3
	202.9	150.0	197.2	135.6	103.7			
	sd=124.2	123.5	135.8	123.6	106.3			
19Jan	(3.9)^{a}	(3.8)^{a}	(3.1)^{a}	(2.6)^{a}	(2.8)^{a}	1.47	0.2416	42.6
	142.0	126.4	104.0	96.0	90.6			
	sd=137.1	160.0	126.5	99.1	100.6			
5Feb	(3.8)^{a}	(3.6)^{a}	(2.8)^{ab}	(2.6)^{a}	(1.9)^{b}	3.24	0.0292	46.0
	195.9	85.9	101.7	92.9	21.0			
	sd=270.7	51.5	141.9	47.5	29.6			
25Mar	(6.2)^{a}	(5.4)^{ab}	(5.2)^{b}	(5.2)^{b}	(4.9)^{b}	3.68	0.0178	17.3
	920.0	661.1	354.0	289.1	407.0			
	sd=673.0	560.2	353.4	310.3	513.0			
*6/04								
11Apr	(5.0)^{a}	(4.8)^{a}	(4.1)^{a}	(4.4)^{a}	(4.4)^{a}	1.50	0.2331	18.4
	688.3	553.1	660.0	353.4	246.7			
	sd=898.5	604.2	447.4	357.0	268.5			
19Apr	(3.9)^{a}	(3.1)^{a}	(2.5)^{a}	(3.1)^{a}	(2.3)^{a}	2.20	0.0969	41.8
	190.1	149.0	171.5	202.3	105.8			
	sd=121.6	172.7	239.3	224.4	166.8			

Means in parentheses(log(count+1)) with the same letter are not significantly different under DMRT; actual means and their standard deviations are indicated; n=9.

Appendix 9. Number of *M. tanajoa* cadavers per leaf on potted 'Ratenyi' cassava plants treated with: H. thompsonii (F); *A. teke* alone (P) and Water (C).

Date	Cadaver counts on treatments F	P	C	F Value	P>F	CV
30 Dec	$(3.4)^{a}$	$(4.4)^{a}$	$(3.2)^{a}$	4.97	0.0001	13.1
	31.6	118.1	27.3			
	sd=9.4	80.3	7.4			
6 Jan	$(4.7)^{a}$	$(5.0)^{a}$	$(4.9)^{a}$	1.73	0.2008	10.5
	126.9	304.9	158.4			
	sd=56.8	244.7	72.0			
*12/01. H. thompsonii =8.0×10^{5} CPM; *A. teke*=15 PP.						
19 Jan	$(0.9)^{b}$	$(4.0)^{a}$	$(4.6)^{a}$	9.27	0.0314	41.0
	3.2	194.1	178.7			
	sd=4.1	288.5	154.6			
5 Feb	$(2.0)^{a}$	$(4.5)^{a}$	$(2.4)^{a}$	1.97	0.0253	34.2
	19.7	271.9	62.7			
	sd=12.2	281.9	67.8			
2 Mar	$(2.3)^{a}$	$(2.7)^{a}$	$(2.1)^{a}$	0.09	0.9118	40.7
	27.8	61.1	16.5			
	sd=27.6	89.4	16.3			
25 Mar	$(2.5)^{a}$	$(3.6)^{a}$	$(3.2)^{a}$	0.67	0.5612	27.9
	36.3	96.4	32.0			
	sd=56.0	98.0	29.0			
*6/04. H. thompsonii =3.7×10^{5} CPM; *A. teke*=10 PP.						
11 Apr	$(5.0)^{a}$	$(5.5)^{a}$	$(4.7)^{a}$	0.82	0.5013	10.5
	206.7	302.7	135.1			
	sd=175.2	175.1	68.8			
19 Apr	$(4.8)^{a}$	$(4.8)^{a}$	$(5.3)^{a}$	1.02	0.4380	26.6
	202.1	228.3	253.5			
	sd=129.0	136.1	158.5			

*Dates when the levels of treatments shown were applied; means in parentheses (log(count+1)) with the same letter are not significantly different under DMRT; actual means and standard deviations are indicated; n=15.

Appendix 10. Distribution of cassava green mite cadavers on the first five leaves of potted 'Kibandameno' cassava plants grown in pots.

Date	Cadavers on leaf.					F Value	P>F	CV
	1	2	3	4	5			
6 Jan	(3.5)[a]	(3.3)[a]	(4.1)[a]	(3.8)[a]	(4.3)[a]	5.65	0.0050	30.2
	74.7	155.2	146.9	79.5	164.4			
	sd=82.0	218.9	118.9	36.4	34.0			
19Jan	(6.0)[a]	(6.3)[a]	(6.5)[a]	(6.7)[a]	(6.7)[a]	1.55	0.2197	9.0
	1815.9	3020.9	1400.3	1061.3	724.8			
	sd=633.3	2792.5	1222.5	914.4	694.1			
5 Feb	(0.2)[a]	(0.8)[a]	(0.9)[a]	(3.4)[a]	(4.4)[a]	1.87	0.1490	100.9
	0.3	5.0	5.5	751.4	642.9			
	sd=0.4	6.1	5.9	747.7	858.2			
2 Mar	(1.5)[a]	(1.()[a]	(1.5)[a]	(1.9)[a]	(1.4)[a]	0.28	0.8879	75.2
	53.9	20.6	19.2	8.3	8.8			
	sd=67.2	21.4	31.0	12.5	12.9			
25 Mar	(2.6)[a]	(2.4)[a]	(2.6)[a]	(2.1)[a]	(2.7)[a]	0.48	0.7533	41.5
	21.2	20.3	21.6	12.6	32.8			
	sd=18.3	18.8	14.0	10.7	33.8			
11 Apr	(4.8)[a]	(4.6)[a]	(4.4)[a]	(4.2)[a]	(4.1)[a]	1.03	0.4109	20.2
	161.1	180.8	186.6	200.2	249.7			
	sd=317.6	185.1	147.8	173.0	153.0			
19 Apr	(4.5)[a]	(4.0)[a]	(4.1)[a]	(4.1)[a]	(4.6)[a]	1.03	0.4109	31.6
	393.7	257.3	325.7	268.0	243.8			
	sd=455.8	399.0	475.8	285.4	256.9			
8 May	(2.7)[a]	(2.7)[a]	(2.9)[a]	(3.2)[a]	(3.1)[a]	1.06	0.3976	22.7
	41.8	43.6	53.0	64.2	102.0			
	sd=24.9	26.2	25.6	39.3	49.1			

Means in parentheses (log(count+1)) with the same letter are not significantly different under DMRT; actual means and standard deviations are indicated; n=9.

Appendix 11. Mean number of CGM cadavers on the first five leaves of potted 'Ratenyi' cassava cultivar planted in pots.

Date	Cadavers number on leaf 1	2	3	4	5	F Value	P>F	CV
30 Dec	$(3.7)^{a}$	$(3.9)^{a}$	(3.7)a	$(3.8)^{a}$	$(3.7)^{a}$	1.44	0.2518	13.1
	63.7	79.7	52.6	61.3	37.7			
	sd=51.7	65.4	39.2	38.0	19.9			
6 Jan	$(4.9)^{a}$	$(5.0)^{a}$	$(4.9)^{a}$	$(4.6)^{a}$	$(4.8)^{a}$	0.99	0.4319	10.5
	215.6	220.9	243.8	144.8	155.7			
	sd=201.3	163.6	203.3	102.7	87.4			
19 Jan	$(2.3)^{a}$	$(3.0)^{a}$	$(2.6)^{a}$	$(3.9)^{a}$	$(3.40^{a}$	2.20	0.0994	41.0
	32.8	294.7	74.0	104.6	120.8			
	sd=39.4	480.2	67.6	53.1	104.9			
5 Feb	$(2.1)^{b}$	$(3.4)^{a}$	$(3.2)^{a}$	$(3.4)^{a}$	$(4.0)^{a}$	10.02	0.0269	34.2
	97.9	118.9	126.6	120.0	127.3			
	sd=136.2	104.3	130.1	122.8	132.5			
2 Mar	$(1.6)^{a}$	$(2.5)^{a}$	$(2.20^{a}$	$(2.6)^{a}$	$(2.8)^{a}$	1.21	0.3324	54.8
	28.3	59.2	24.7	23.8	39.6			
	sd=40.7	91.7	34.0	22.7	32.7			
25 Mar	$(2.9)^{a}$	$(3.3)^{a}$	$(3.1)^{a}$	$(3.0)^{a}$	$(3.1)^{a}$	0.23	0.9171	27.9
	55.4	76.3	43.0	51.0	48.3			
	sd=75.1	71.3	49.0	47.0	61.0			
11Apr	$(5.4)^{a}$	$(5.5)^{a}$	$(4.9)^{a}$	$(4.7)^{c}$	$(4.7)^{a}$	2.52	0.0675	10.5
	312.1	307.9	166.9	127.7	159.3			
	sd=60.7	208.8	73.9	72.2	124.9			
19Apr	$(4.0)^{a}$	$(5.6)^{a}$	$(5.5)^{a}$	$(5.2)^{a}$	$(4.5)^{a}$	1.02	0.4380	26.6
	169.2	367.8	284.9	213.8	104.1			
	sd=151.7	225.0	148.5	114.4	66.6			

Means in parentheses (log(count+1)) with the same letter are not significantly different under DMRT; actual means and standard deviations are indicated; n=9.

Appendix 12. CGM damage index on leaves of potted 'Ratenyi' cassava cultivar treated with: H. thompsonii (F); *A. teke* (P) and water (C).

Date	CGM damage scale on treatments			F Value	P>F	CV
	F	P	C			
*12/04. H. thompsonii =8.0x10^5 CPM; *A. teke*=15 PP.						
19 Jan	1.5^a sd=0.7	2.4^a 0.7	2.3^a 0.6	3.16	0.1502	29.7
5 Feb	1.5^a sd=0.4	1.8^a 0.5	2.0^a 0.7	1.48	0.3303	47.6
2 Mar	1.5^a sd=0.5	1.1^a 0.1	1.8^b 0.3	31.00	0.0037	34.1
25 Mar	1.5^a sd=0.6	1.9^a 0.9	2.4^a 0.8	2.16	0.2311	22.7
*06/04. H. thompsonii =3.7x10^5 CPM; *A. teke*=10 PP.						
11 Apr	1.6^a sd=0.6	1.8^a 0.7	3.0^a 1.0	0.51	0.6350	26.1
19 Apr	1.8^a sd=0.3	1.6^a 0.6	2.0^a 0.0	1.64	0.3025	21.9

*Dates when the indicated levels of treatments were applied; means with the same letter are not significantly different under DMRT; n=15.

Appendix 13. CGM damage index on leaves in positions 1 to 5 of potted 'Kibandameno' cultivar planted in pots.

Date	CGM damage index on leaf number 1	2	3	4	5	F Value	P>F	CV
19 Jan	2.9[a]	2.6[ab]	2.0[b]	2.0[b]	2.3[b]	5.22	0.0036	23.7
	sd=0.2	0.2	0.8	0.8	0.4			
5 Feb	1.0[a]	1.0[a]	1.8[b]	2.6[b]	2.3[b]	7.64	0.0004	45.5
	sd=0.0	0.0	0.7	0.8	0.6			
2 Mar	1.1[a]	1.1[a]	1.2[a]	1.1[a]	1.1[a]	0.18	0.9456	30.8
	sd=0.2	0.2	0.2	0.2	0.2			
25 Mar	1.8[a]	1.8[a]	1.4[ab]	1.3[b]	1.4[ab]	3.89	0.0142	20.3
	sd=0.5	0.9	0.6	0.4	0.6			
11 Apr	1.9[a]	1.9[a]	2.0[a]	1.8[a]	1.8[a]	0.45	0.7702	22.2
	sd=0.7	0.4	0.6	0.2	0.2			
16 Apr	1.9[a]	1.9[a]	1.7[a]	1.7[a]	1.6[a]	1.50	0.2336	21.1
	sd=0.4	0.4	0.0	0.0	0.6			
8 May	1.2[a]	1.4[a]	1.7[a]	1.7[a]	1.3[a]	1.37	0.2722	29.6
	sd=0.7	0.7	0.0	0.7	0.6			

Means with the same letter are not significantly different under DMRT; n=9.

Appendix 14. *M. tanajoa* damage index on the first five leaf positions of potted 'Ratenyi' cassava cultivar grown in pots.

Date	*M.tanajoa* damage index on leaf					F Value	P>F	CV
	1	2	3	4	5			
19 Jan	1.7^{b}	1.7^{b}	2.1^{a}	1.9^{b}	5.0^{a}	7.29	0.0005	29.7
	sd=0.6	0.7	0.6	0.4	1.0			
5 May	1.7^{a}	1.6^{a}	1.4^{a}	1.8^{a}	2.4^{a}	1.96	0.1367	47.6
	sd=0.7	0.5	0.6	0.8	1.2			
2 Mar	1.7^{a}	1.4^{a}	1.4^{a}	1.6^{a}	1.2^{a}	0,98	0.4381	34.1
	sd=0.0	0.4	0.6	0.4	0.2			
25 Mar	2.2^{a}	2.1^{ab}	1.7^{b}	1.9^{ab}	1.7^{b}	3.06	0.0360	22.7
	sd=0.8	0.7	0.6	0.7	0.4			
11 Apr	2.0^{a}	2.0^{a}	1.8^{a}	1.9^{a}	1.7^{a}	1.08	0.3867	26.1
	sd=0.5	0.4	0.5	0.5	0.4			
19 Apr	1.8^{a}	1.9^{a}	1.9^{a}	1.8^{a}	1.7^{a}	0.50	0.7360	21.9
	sd=0.4	0.2	0.2	0.4	0.4			

Means with the same letter are not significantly different under DMRT; n=9.

Appendix 15. Means of *A. teke* per leaf on potted 'Ratenyi' cassava cultivar treated with: H. thompsonii (F); *A. teke* (P) and water (C).

Date	*A. teke* per leaf on treatments		
	F	P	C
*12/01. H. thompsonii =8.0x10^5 CPM; *A. teke*=15 PP.			
19 Jan	0.0	9.4	4.4
	sd=0.0	8.4	7.4
5 Feb	0.5	2.1	2.3
	sd=0.8	2.3	1.7
2 Mar	0.3	11.7	0.5
	sd=1.0	28.2	1.8
25 Mar	0.0	0.5	0.0
	sd=0.0	2.1	0.0
*06/04. H. thompsonii =3.7x10^5 CPM; *A. teke*=10 PP.			
11 Apr	0.6	1.0	2.7
	sd=1.0	1.0	3.5
19 Apr	0.3	0.5	7.5
	sd=0.5	0.9	7.2

* Dates and units of treatment applications; n=15.

Appendix 16. Distribution of *A. teke* on the first five leaves of potted 'Kibandameno' cassava cultivar.

Date	*A. teke* counts on leaf in position				
	1	2	3	4	5
19 Jan	4.0	7.5	6.2	7.1	3.4
	sd=4.6	5.9	7.2	10.3	2.3
5 Mar	0.0	0.0	0.0	0.0	0.0
	sd=0.0	0.0	0.0	0.0	0.0
2 Mar	0.2	0.6	0.3	0.6	0.3
	sd=0.4	1.0	0.5	1.0	0.5
25 Mar	0.0	0.9	0.3	1.0	0.0
	sd=0.0	1.8	1.0	2.0	0.0
11 Apr	1.7	2.5	2.0	4.1	3.2
	sd=2.4	3.5	2.1	5.2	2.0
19 Apr	5.3	3.1	4.9	1.5	5.5
	sd=4.9	2.3	5.4	2.7	6.1
8 May	0.0	0.2	0.8	0.8	1.1
	sd=0.0	0.7	1.7	1.7	3.3

n=15

Appendix 17. Mean number of *A. teke* per leaf on the first five leaves of 'Ratenyi' cassava cultivar grown in pots.

Date	*A. teke* on leaf numbers				
	1	2	3	4	5
19 Jan	4.1	7.2	6.8	3.1	3.3
	sd=5.3	9.2	7.8	6.2	7.9
5 Mar	2.2	1.0	0.4	3.4	1.1
	sd=2.1	1.7	0.8	1.8	1.7
2 Mar	9.3	9.0	1.6	0.0	0.8
	sd=28.0	25.5	4.7	0.0	2.3
25 Mar	0.0	0.9	0.0	0.0	0.0
	sd=0.0	2.7	0.0	0.0	0.0
11 Apr	1.9	2.2	0.5	0.4	2.1
	sd=1.3	2.8	0.8	0.8	3.7
19 Apr	5.0	1.4	1.0	4.7	1.7
	sd=5.5	1.6	0.9	4.3	2.0

n=9.

Appendix 18. *A. teke* eggs per leaf of potted ' Ratenyi' cassava cultivar treated with: *H. thompsonii* (F); *A. teke* (P) alone and water (C).

Date	*A. teke* eggs on treatments F	P	C
*12/01. *H. thompsonii* =8.0×10^5 CPM; *A. teke*=15 PP.			
19 Jan	1.0	5.6	0.0
	sd=2.6	6.3	0.0
5 Mar	0.1	8.7	6.6
	sd=0.2	14.3	11.1
2 Mar	0.0	4.1	0.0
	sd=0.0	7.1	0.0
25 Mar	0.0	0.0	0.5
	sd=0.0	0.0	1.8
*06/04. *H. thompsonii* =3.7×10^5 CPM; *A. teke*=10 PP.			
11 Apr	0.1	2.5	2.4
	sd=0.2	2.6	3.6
19 Apr	0.7	0.1	3.1
	sd=1.2	0.1	3.1

* Dates when the indicated levels of treatments were applied; n=15.

Appendix 19. Mean of *A. teke* eggs per leaf on the first five leaves of potted 'Kibandameno' cassava plants.

Date	A. teke eggs on leaf number				
	1	2	3	4	5
19 Jan	3.1	6.1	3.6	6.2	3.4
	sd=3.6	5.7	2.8	7.0	4.4
5 Mar	0.0	1.1	0.0	0.0	0.0
	sd=0.0	1.9	0.0	0.0	0.0
2 Mar	0.0	1.1	0.0	0.0	0.0
	sd=0.0	1.9	0.0	0.0	0.0
25 Mar	0.0	0.0	1.7	0.3	0.9
	sd=0.0	0.0	4.3	1.0	2.7
11 Apr	1.1	8.9	2.3	2.2	0.8
	sd=1.0	15.0	2.8	3.3	0.8
19 Apr	4.2	3.1	2.8	2.6	8.4
	sd=4.3	5.4	2.5	3.6	9.4
8 May	0.0	0.0	0.6	0.0	0.0
	sd=0.0	0.0	1.7	0.0	0.0

n=9.

Appendix 20. *A. teke* eggs per leaf on the first five leaves of potted 'Ratenyi' cassava cultivar.

Date	*A. teke* eggs on leaf number				
	1	2	3	4	5
19 Jan	4.9	8.1	2.1	3.8	1.0
	sd=7.5	11.9	3.9	5.9	3.0
5 Feb	16.0	6.0	2.6	2.6	0.0
	sd=27.1	7.3	3.7	4.8	0.0
2 Mar	6.2	0.0	0.6	0.0	0.0
	sd=10.8	0.0	1.1	0.0	0.0
25 Mar	0.0	0.8	0.0	0.0	0.0
	sd=0.0	2.3	0.0	0.0	0.0
11 Apr	3.1	2.4	1.2	0.1	2.3
	sd=2.8	3.3	0.4	0.2	3.9
19 Apr	3.0	1.7	0.0	2.0	3.3
	sd=3.1	1.6	0.0	1.4	0.6

n=9.

Appendix 21. Number of *M. tanajoa* eggs per leaf of field cassava treated with *H. thompsonii*; n=75.

Record Date	CGM Egg/Leaf sprayed with *H.thompsonii*	CGM Egg/Leaf sprayed with Water	F value	P>F	CV
25/01/88	(4.389)^{a} 223.7 sd=208.8	(4.173)^{a} 170.8 sd=164.9)	0.53	0.4806	31.3
* 26/01. *H.thompsonii* =8.0x10^{5} CPM.					
8/02/88	(5.229)^{b} 308.8 sd=243.6	(6.178)^{a} 834.7 sd=602.9	28.53	0.0001	15.1
18/02/88	(4.474)^{a} 290.8 sd=313.8	(4.937)^{a} 349.1 sd=329.8	0.78	0.3915	26.0
8/3/88	(1.790)^{a} 17.4 sd=19.1)	(1.691)^{a} 18.4 sd=31.0	0.12	0.7305	60.2
21/3/88	(0.909)^{a} 5.1 sd=8.0)	(0.845)^{a} 9.8 sd=26.6	0.23	0.6350	94.3
4/4/88	(2.5)^{a} 36.2 sd=43.2	(2.2)^{a} 36.7 sd=50.1	1.75	0.2071	58.9
* 06/04· *H.thompsonii* =3.7x10^{5} CPM					
16/4/88	(2.4)^{a} 40.5 sd=3.1	(2.1)^{a} 21.3 sd=6.6	0.74	0.4035	55.8
6/5/88	(2.7)^{a} 45.6 sd=6.9	(3.5)^{a} 100.4 sd=17.4	18.20	0.1267	29.0
21/5/88	(3.5)^{a} 161.7 sd=32.1	(2.3)^{b} 85.2 sd=20.6	4.69	0.0481	33.8

* Dates when the levels of treatments shown were applied; values in parentheses (log(count+1)) bearing the same letter are not different statistically under DMRT; actual counts per leaf and 'sd' are indicated.

Appendix 22. Number of *M. tanajoa* per leaf of field cassava treated with *H. thompsonii*; n=75.

Record Date (1988)	CGM per leaf from treatment		F Value	P>F	CV
	H. thompsonii	Water			
25.Jan.	(4.6)[a] 185.2 sd=149.7	(4.6)[a] 198.5 sd=179.8	0.12	0.7608	23.5
* 26/01. *H. thompsonii* conc.=8.0x10^5 CPM.					
8.Feb	(3.8)[b] 72.3 sd=71.0	(5.3)[a] 307.9 sd=287.1	34.23	0.0001	18.9
18.Feb.	(4.0)[b] 136.4 sd=140.2	(4.7)[a] 203.8 sd=166.1	3.12	0.0001	21.6
8.Mar.	(1.9)[a] 18.5 sd=12.1	(2.3)[a] 20.5 sd=14.3	2.11	0.1680	47.7
21.Mar	(1.0)[a] 4.7 sd=5.4	(1.0)[a] 6.4 sd=14.9	0.01	0.9278	72.6
4.Apr.	(1.9)[a] 13.4 sd=17.6	(1.8)[a] 17.6 sd=28.8	0.16	0.6969	61.8
* 06/04. *H. thompsonii* conc=3.7x10^5 CPM.					
16.Apr.	(2.3)[a] 23.5 sd=3.3	(1.8)[a] 10.0 sd=1.3	1.58	0.2289	41.6
6.May	(2.5)[a] 68.1 sd=12.0	(3.2)[a] 24.6 sd=3.9	19.72	0.1398	28.4
21.May	(3.6)[a] 82.7 sd=3.6	(2.3)[b] 53.0 sd=11.7	16.27	0.0001	27.6

* Dates when the levels of treatment shown were applied; means bearing the same letter are not different statistically under DMRT; values with/without parentheses are log(count+1) and actual mean counts respectively.

Appendix 23. *M. tanajoa* damage index on cassava leaves treated with *H. thompsonii* and and Water; n=75.

Record Date (1988)	CGM damage index on treatment		F Value	P>F	CV
	H. thompsonii	Water			
25/Jan	1.7[a] sd= 0.5	1.8[a] 0.7	4.60	0.0500	20.3
* 26/01. *H. thompsonii* conc=8.0x10^5 CPM.					
8/Feb	1.6[a] d=0.7	1.9[a] 0.8	1.67	0.2166	31.6
18/Feb	1.9[a] sd=0.6	2.0[a] 0.5	0.72	0.4099	20.7
8/Mar	1.9[b] sd=0.4	2.6[a] 0.6	8.31	0.0001	23.3
21 Mar	1.4[b] sd=0.3	1.7[a] 0.6	11.92	0.0039	32.3
4 Apr	1.1[a] sd=0.2	1.1[a] 0.2	0.06	0.8178	29.4
* 06/04. *H. thompsonii* =3.7x10^5 CPM.					
16 Apr	1.0[a] sd=0.2	1.0[a] 0.2	0.32	0.4000	10.5
6 May	1.0[a] sd=0.2	1.0[a] 0.2	0.04	0.8496	12.3
21 May	1.4[a] sd=0.1	1.2[a] 0.1	1.00	0.3343	26.9

* Dates when the levels of treatments shown were applied; values with the same letters are not statistically different under DMRT.

Appendix 24. Mean number of *M. tanajoa* cadavers from field cassava sprayed with *H. thompsonii* and water; n=75.

Record Date (1988)	No. of cadavers on treatments: *H. thompsonii*	Water	F value	P>F	CV
25 Jan	(2.2)[a] 20.1 sd=27.8	(2.6)[a] 25.3sd=27.5	1.32	0.2701	46.9
* 26/01. *H. thompsonii* conc.=8.0x10^5 CPM.					
8 Feb	(5.0)[a] 307.0 sd=346.3	(4.4)[a] 158.5 sd=216.0	3.85	0.0699	20.6
18Feb	(4.3)[a] 137.0 sd=177.3	(4.7)[a] 177.3 sd=170.8	2.97	0.1069	20.7
8 Mar	(1.8)[a] 915.6 sd=22.7	(1.8)[a] 9.76 sd=11.7	0.01	0.9392	57.3
21 Mar	(0.4)[a] 0.8 sd=1.1	(0.5)[a] 1.1 sd=1.5	0.94	0.3479	134.1
4 Apr	(0.5)[a] 1.3 sd=1.9	(0.6)[a] 1.0 sd=4.1	0.35	0.5657	114.1
* 06/04. *H. thompsonii* conc.=3.7x10^5 CPM.					
16 Apr	(0.3)[a] 0.77 sd=1.6	(0.2)[a] 0.45 sd=0.9	0.62	0.4439	146.4
6 May	(1.1)[a] 4.4 sd=0.9	(1.6)[a] 10.0 sd=2.0	2.38	0.1449	60.4
21 May	(1.2)[a] 5.7 sd=1.1	(0.8)[a] 4.2sd=1.0	0.90	0.3595	78.3

* Dates when indicated treatments were applied; values with/without parentheses are log(count+1) and actual counts respectively.

Appendix 25. Regression analyses to show association between weather and CGM infestation levels.

Period	Temp. (°C)	Rain Days	Total Rainfall (mm)	CGM dam	CGM pop[1].	CGM egg[2]
15-31/01	23.7	4	105.6	1.8	198.5	170.8
1-15/02	24.1	1	2.8	1.9	307.9	834.7
16-29/02	24.2	2	37.4	2.0	203.8	349.1
1-15/03	24.9	6	109.8	2.6	20.5	18.4
16-31/03	24.0	4	79.2	1.7	6.4	9.8
1-15/04	22.8	5	136.0	1.1	17.6	36.7
16-31/04	24.3	3	17.7	1.0	10.0	21.3
1-15/05	21.4	8	62.2	1.0	24.6	100.4
16-31/05	23.9	5	59.7	1.2	53.0	85.2
MEAN=	23.7	4.2	67.8	1.6	93.6	180.7
S.D=	1.0	2.1	44.3	0.6	112.5	267.7

[1] and [2] CGM population and egg counts per leaf.

Appendix 26. Mean number of CGM on leaves 1 to 9 of field cassava leaves; n=30.

Record Date (1988)	Mobile CGM on leaf no. 1	3	5	7	9	F Value	P>F	CV
25.Jan	4.7^{bc}	5.3^{a}	5.1^{ab}	4.4^{c}	3.5^{d}	12.6	0.0001	23.5
	165.5	253.6	262.2	201.5	76.3			
sd=	123.9	140.2	224.3	248.3	87.1			
8.Feb	$4.5b^{c}$	4.7^{ab}	5.0^{a}	4.2^{c}	4.2^{c}	4.9	0.0012	18.9
	159.8	194.6	274.2	155.0	167.0			
sd=								
18.Feb	5.0^{a}	5.0^{a}	4.6^{a}	3.9^{b}	3.4^{c}	17.7	0.0001	21.6
	282.1	210.1	401.2	109.6	73.9			
sd=	223.8	166.7	170.8	105.4	89.3			
8.Mar	3.1^{a}	2.7^{a}	2.0^{b}	1.5^{c}	1.1^{c}	21.6	0.0001	47.7
	70.5	20.8	18.2	7.7	6.8			
sd=								
21.Mar.	1.6^{a}	1.7^{a}	1.0^{b}	0.4^{c}	0.4^{c}	19.9	0.0001	72.6
	6.3	10.2	6.5	3.3	1.3			
sd=	5.8	14.3	15.9	11.0	2.7			
4.Apr	2.2^{a}	2.3^{a}	2.3^{a}	1.4^{b}	0.8^{c}	11.0	0.0001	61.8
	20.2	25.9	19.8	7.6	3.9			
sd=	39.4	33.4	21.9	12.0	7.4			
16.Apr	1.8^{a}	2.3^{a}	2.2^{a}	2.0^{a}	1.9^{a}	2.23	0.0705	41.6
	9.9	20.3	16.6	21.7	15.3			
sd=	1.4	2.3	1.7	3.9	2.2			
6.May	3.1^{ab}	3.4^{a}	3.1^{ab}	2.7^{b}	2.1^{c}	9.99	0.0001	28.4
	45.0	58.0	54.5	47.2	27.4			
d=	9.7	12.0	16.5	14.4	10.0			
21.May	3.8^{a}	3.8^{a}	3.2^{b}	2.6^{c}	1.4^{d}	45.4	0.0001	27.6
	101.7	125.6	54.3	43.2	14.5			
sd=	20.3	32.7	12.2	15.2	5.5			

* First row against each record date=log(count+1) used in DMRT; values with same letter are not significantly different.

Appendix 27. *M. tanajoa* egg per leaf in positions 1 to 9 on field cassava plants; n=30.

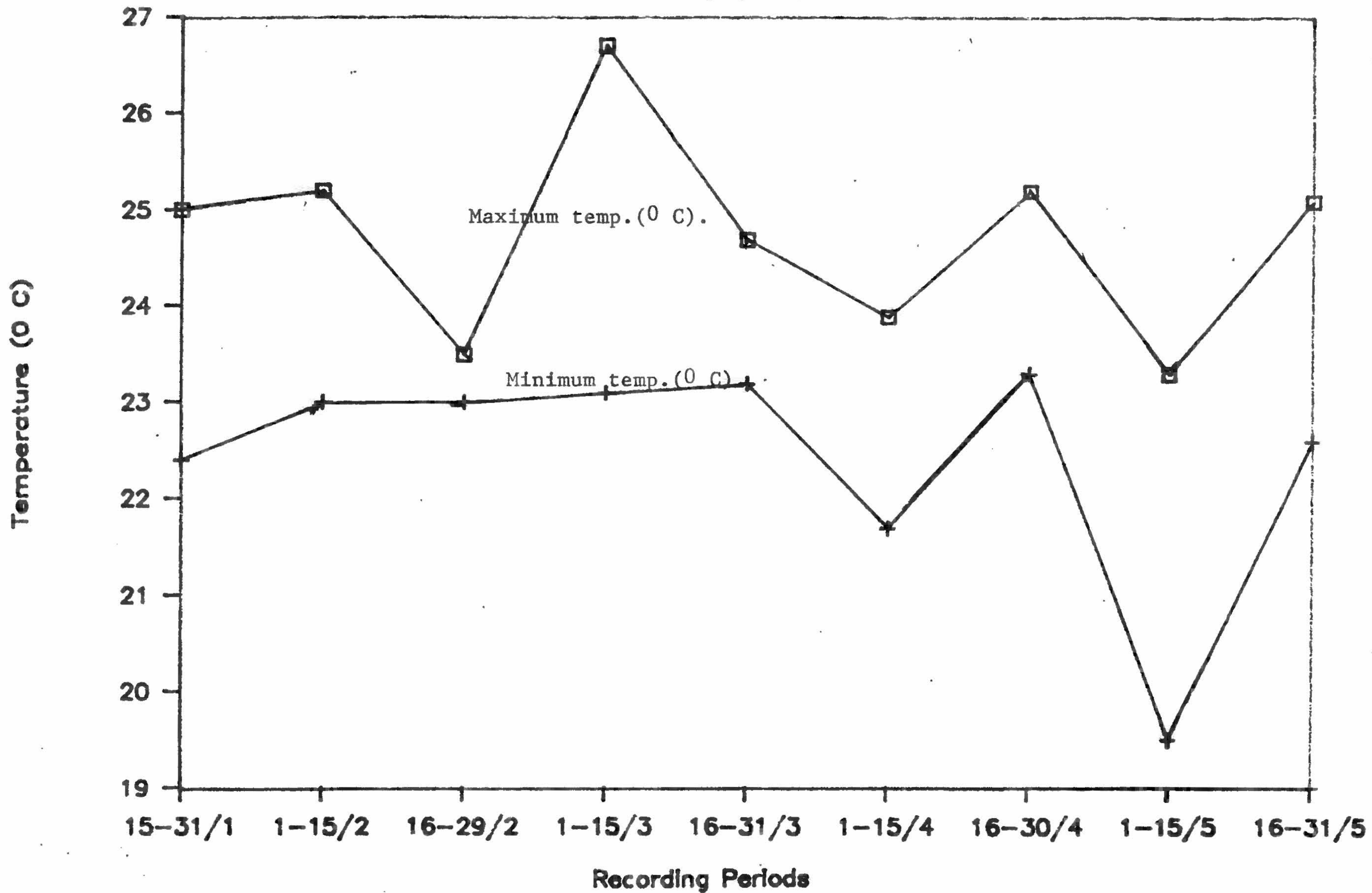
Record Date (1988)	Mean CGM eggs on leaf numbers					F Value	P>F	CV
	1	3	5	7	9			
25.Jan	5.4^{a}	5.3^{a}	4.5^{b}	3.5^{c}	2.6^{d}	23.80	0.0001	31.3
	324.4	293.6	214.1	115.7	38.6			
	sd=213.3	213.1	246.0	203.2	58.7			
8 Feb	6.0^{a}	6.3^{a}	6.2^{a}	4.7^{c}	5.3^{b}	18.00	0.0001	15.1
	550.3	789.2	808.4	198.2	512.8			
	sd=325.9	461.8	606.9	223.3	572.9			
18 Feb	5.6^{a}	5.4^{a}	4.8^{b}	4.0^{c}	3.6^{c}	15.36	0.0001	26.0
	589.2	409.2	303.4	160.2	137.7			
	sd=514.0	335.2	539.8	191.4	194.5			
8 Mar	3.1^{a}	3.4^{b}	1.6^{c}	1.0^{d}	0.6^{d}	27.48	0.0001	60.2
	46.0	28.0	10.0	3.6	1.9			
	sd=55.9	45.5	15.4	5.1	3.4			
21.Mar	1.7^{a}	1.7^{a}	0.7^{b}	0.2^{c}	0.2^{c}	25.40	0.0001	94.3
	13.5	12.2	9.7	1.1	0.9			
	sd=24.6	21.4	33.2	4.2	0.5			
4 Apr	3.1^{ab}	3.3^{a}	2.6^{b}	1.9^{c}	0.8^{d}	16.29	0.0001	58.9
	59.6	61.3	36.7	18.5	6.1			
	sd=80.1	61.3	44.6	27.7	12.4			
16 Apr	2.8^{ab}	3.0^{a}	2.3^{bc}	1.5^{d}	1.7^{cd}	8.18	0.0001	55.8
	39.9	52.5	29.5	14.9	16.5			
	sd=8.4	12.7	8.5	4.3	3.0			
6 May	4.1^{a}	3.9^{a}	3.1^{b}	2.3^{c}	1.8^{d}	36.97	0.0001	29.0
	155.3	114.8	56.9	23.9	14.5			
	sd=39.1	31.1	15.9	5.0	4.4			
21 May	4.5^{a}	3.9^{b}	3.1^{c}	2.1^{d}	0.9^{e}	64.76	0.0001	33.8
	344.0	157.2	71.1	37.7	7.3			
	sd=120.7	41.2	26.6	15.8	7.3			

* First row against each record date =log(count+1) used for analysis of variance (DMRT); values with same letter are not different statistically.

Appendix 28. CGM cadaver counts on cassava leaves 1 to 9; n=30.

Record Date (1988)	CGM Cadaver Counts on leaf number 1	3	5	7	9	F Value	P>F	CV
25 Jan	1.7^{c}	2.2^{bc}	2.7^{ab}	2.9^{a}	2.4^{ab}	5.17	0.0007	46.9
	9.8	21.2	28.4	32.5	21.7			
sd=	14.8	29.1	31.7	35.5	27.1			
8 Feb	4.2^{c}	4.5^{bc}	5.0^{a}	4.8^{ab}	4.9^{ab}	3.73	0.0069	20.6
	170.0	178.4	294.9	272.2	243.7			
sd=	263.3	230.8	279.4	359.9	272.6			
18 Feb	4.4^{a}	4.6^{a}	4.6^{a}	4.7^{a}	4.2^{a}	1.63	0.173	20.7
	140.3	155.9	172.4	214.9	202.3			
sd=	133.9	175.3	196.7	228.2	94.9			
8 Mar	1.3^{bc}	1.2^{c}	1.8^{ab}	2.2^{a}	2.3^{a}	1.33	0.0001	57.3
	8.9	7.8	11.3	13.9	21.5			
sd=	14.2	12.4	14.3	13.5	31.7			
21 Mar	0.2^{cd}	0.1^{d}	0.4^{bc}	0.6^{ab}	0.8^{a}	7.43	0.0001	134.1
	0.4	0.1	0.8	1.4	1.8			
sd=	0.8	0.3	1.3	2.1	2.1			
4 Apr	0.3^{b}	0.5^{b}	0.5^{b}	0.4^{b}	0.8^{a}	3.65	0.0078	114.1
	0.6	1.2	0.9	0.8	2.2			
sd=	1.3	2.2	1.4	1.4	2.9			
16 Apr	0.1^{a}	0.2^{a}	0.4^{a}	0.3^{a}	0.3^{a}	1.18	0.3220	146.4
	0.3	0.6	0.8	0.9	0.6			
sd=	0.5	1.2	1.3	2.2	1.1			
6 May	1.3^{a}	1.4^{a}	1.3^{a}	1.4^{a}	1.3^{a}	0.15	0.9626	60.4
	10.6	5.5	5.5	6.8	7.5			
sd=	4.4	1.2	1.6	1.7	2.2			
21 May	0.9^{a}	1.0^{a}	1.1^{a}	0.9^{a}	1.1^{a}	0.49	0.7404	78.3
	3.7	4.4	6.6	4.7	5.4			
sd=	1.1	1.5	2.4	1.8	1.7			

* Values bearing same letter are log(count+1) and are not significantly different under DMRT statistics.



Appendix 29. Mean minimum and maximum air temperature in the field between January to May 1988.
-Mbita Point Field Station.