

**ASSESSMENT OF *DICHAPETALUM* PLANTS INFESTATION AND ITS  
SUSCEPTIBILITY TO THREE HERBICIDES IN MKURANGA DISTRICT  
TANZANIA.**

**BY**

**NASSORO ATHUMANI MOPEI**

**A DISSERTATION SUBMITTED IN PARTIAL FULFILLMENT OF THE  
REQUIREMENTS FOR THE DEGREE OF MASTER OF VETERINARY  
MEDICINE OF SOKOINE UNIVERSITY OF AGRICULTURE.  
MOROGORO, TANZANIA**

2007



## ABSTRACT

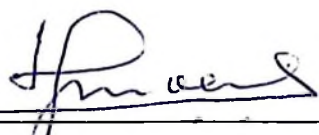
A study was conducted in Mkuranga District between October, 2005 and February, 2006 to investigate *Dichapetalum* plants infestation and effectiveness of selected herbicides on these plants. Specifically, this study intended to identify species prevalence and their coverage in this District. The study further aimed at determining effectiveness of herbicides on the control of *Dichapetalum* plants. The Purposive sampling was used to select farms for the study. The actual areas of infestation for surveys in these farms were chosen on random basis so as to get representative picture for whole area infested with these plants in the District. The survey conducted using Scientific Quadrant Method, established presence of four *Dichapetalum* species; *Dichapetalum stuhlmanii*, *Dichapetalum mossambicence*, *Dichapetalum ruhlandii* and *Dichapetalum arenarium*, with percentage cover of 76%, 42%, 37% and 27% respectively. The plants that were commonly in close association with *Dichapetalum* plants were *Deinbollia borbonica*, *Annona senegalensis*, *Xylothea tetensis* and *Milletia* spp.

*Dichapetalum* plant sprouts were individually sprayed with 2,4-dichlorophenoxy acetic acid (2,4-D), glyphosate (Round up®) and paraquat in the field, followed by observation for responses to herbicides for 60 days. A number of phytotoxic effects were recorded. The efficacies of selected herbicides were assessed in terms of killing percentages. Killing percentages recorded at the end of study (Day 60) were analyzed by MSTAT-C. Overall killing percentage was 16.2 %. On individual herbicide, paraquat recorded the highest killing percentages (17.8 %) among the three tested herbicides. This was followed by 2,4-D (16.3 %). Glyphosate recorded killing

percent of 14.6 % which was the lowest in the study. Analysis of variance (ANOVA) has shown that the degree of control on *Dichapetalum* plants is influenced by herbicide type, dosage rate, and interaction between herbicide and dose. It was concluded that Mkuranga District is heavily infested with *Dichapetalum* plants and application of 2,4-D, Glyphosate and Paraquat in dry conditions gave poor herbicide performance.

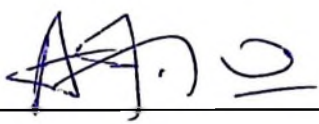
**DECLARATION**

I, Nassoro Athumani Mopei do hereby declare to the Senate of Sokoine University of Agriculture that, this dissertation is my original work and that it has neither been submitted nor concurrently submitted for a degree award at any other University.

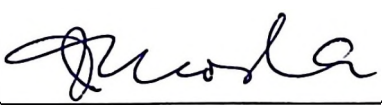
  
\_\_\_\_\_  
Nassoro Athumani Mopei  
(MVM Candidate)

10/05/2007  
Date

The above declaration is confirmed by:

  
\_\_\_\_\_  
Prof. A. J. Ngomuo  
(Supervisor)

10/05/2007  
Date

  
\_\_\_\_\_  
Prof. R. D. Mosha  
(Supervisor)

10/05/2007  
Date

**COPYRIGHT**

No part of this dissertation may be reproduced, stored in retrieval system, or transmitted in any form or by any means without the prior written permission of the author or the Sokoine University of Agriculture in that behalf.

## ACKNOWLEDGEMENT

I am greatly indebted to many individuals and institutions for their support and encouragement throughout the study.

I owe a debt of gratitude to my sponsor Belgian Technical Co-operation (BTC) who financed my studies. I am grateful to my employer, Mkuranga District Council for granting me a study leave so that I could pursue my studies at Sokoine University of Agriculture (SUA).

My sincere appreciation is due to my supervisors; Professors A. J. Ngomuo and R. D. Mosha for their guidance, constructive criticisms and tireless supervision which led to outcome of this work. I am also indebted to acknowledge all academic members of staff of Department of Physiology, Biochemistry, Pharmacology and Toxicology for their assistance and co-operation in this research work.

I would like to thank the Head of Department of Physiology, Biochemistry, Pharmacology and Toxicology, Professor S. T. Balthazar for facilitation throughout my study period.

I wish to thank Mr. H. Selemani, a botany technician at Department of Botany, UDSM for his technical assistance in the field survey and botanical identification of collected plants.

I am indebted to TMA for provision of data on rainfall trend in Mkuranga District and technical assistance on weather interpretation.

I wish to thank Mr. S. O. Mkwachu, a PhD student at Department of Crop Science, Sokoine University of Agriculture for valuable assistance on data analysis using MSTAT-C.

I am indebted to Dr. S. Hoyle and Eng. J. Hoyle for their moral and material support. Their words of encouragement during course of study were very much welcome.

Last, but not least, my special thanks go to my family who gave me support and encouragement.

Since it is virtually impossible to mention all those who in one way or another, played a role in this research work, I simply say, thanks for their kind and/or moral support.

## **DEDICATION**

This work is dedicated to my father Mr. Athumani S. Mopei, my mother Hadija S. Mbullu and my friends; Dr. Sarah Hoyle and Eng. James Hoyle for their immense contribution to my education.

## TABLE OF CONTENTS

<b>ABSTRACT .....</b>	<b>i</b>
<b>DECLARATION .....</b>	<b>iii</b>
<b>COPYRIGHT .....</b>	<b>iv</b>
<b>ACKNOWLEDGEMENT .....</b>	<b>v</b>
<b>DEDICATION .....</b>	<b>vii</b>
<b>TABLE OF CONTENTS .....</b>	<b>viii</b>
<b>LIST OF TABLES .....</b>	<b>xii</b>
<b>LIST OF FIGURES .....</b>	<b>xiii</b>
<b>LIST OF APPENDICES .....</b>	<b>xv</b>
<b>ABBREVIATIONS AND SYMBOLS .....</b>	<b>xvi</b>
<b>ABBREVIATIONS AND SYMBOLS .....</b>	<b>xvi</b>
<b>CHAPTER ONE .....</b>	<b>1</b>
<b>1.0 INTRODUCTION .....</b>	<b>1</b>
<b>CHAPTER TWO .....</b>	<b>7</b>
<b>2.0 LITERATURE REVIEW .....</b>	<b>7</b>
<b>2.1 Animal plant poisoning overview .....</b>	<b>7</b>
2.1.1 Toxic plant constituents .....	7
2.1.2 Defensive mechanisms of animals against poisonous plants .....	7
2.1.3 Predisposing factors for plant poisoning .....	8
2.1.4 Factors determining effects of poisonous plants to animals .....	9
2.1.5 Diagnosis and treatment of plant poisoning .....	9
<b>2.2 Background information of <i>Dichapetalum</i> plants .....</b>	<b>10</b>

2.2.1 Classification.....	10
2.2.3 Fluoroacetate mode of action .....	12
2.2.4 Fluoroacetate metabolism.....	13
2.2.5 <i>Dichapetalum</i> poisoning symptoms .....	13
2.2.6 Dichapetalosis treatment .....	14
<b>2.3 Perspective of weed control in pastures .....</b>	<b>14</b>
<b>2.4 Scope of chemical weed control.....</b>	<b>15</b>
<b>2.5 Herbicides .....</b>	<b>17</b>
2.5.1 Herbicide classification .....	17
2.5.2 Herbicide Families and their injurious symptoms.....	18
2.5.3 Herbicide Sprays Formulation .....	20
2.5.4 Methods of herbicides applications.....	22
2.5.5 Influence of environmental factors on herbicide performances.....	22
2.5.6 Assessment of herbicide performance.....	24
<b>2.6 Details of selected herbicides .....</b>	<b>25</b>
2.6.1 Bipyridyliums: Paraquat.....	25
2.6.2 Phenoxy acetic acids: 2, 4-D compounds.....	28
2.6.3 Phosphono Amino Acids: Glyphosate (Roundup®).....	30
<b>2.7 Weed resistances to herbicides.....</b>	<b>33</b>
2.7.1 Meaning and history of weed resistance .....	33
2.7.2 Herbicide resistance mechanisms.....	34
2.7.3 Diagnosis of herbicide resistance .....	34
2.7.4 Control measures of herbicide resistant weeds .....	34
<b>2.8 Herbicide tolerant plants .....</b>	<b>35</b>

2.8.1 Background information on herbicide tolerance of plants .....	35
2.8.2 Biotechnology in developing herbicide-tolerant crops .....	35
2.8.3 Management of herbicide-tolerant weeds .....	36
<b>2.9 Study for the structure and composition of plant communities.....</b>	<b>37</b>
2.9.1 Sampling for survey research.....	37
2.9.2 Description of plant communities .....	37
2.9.3 Percentage cover as a measure of plant communities .....	38
2.9.4 Quadrant method for study of plant communities.....	39
<b>CHAPTER THREE .....</b>	<b>41</b>
<b>3.0 MATERIALS AND METHODS.....</b>	<b>41</b>
<b>3.1 Description of the study area.....</b>	<b>41</b>
3.1.1 Location of the study site: Mkuranga District.....	41
3.1.2 Climatic condition.....	43
<b>3.2 Sampling procedure and sample size .....</b>	<b>43</b>
<b>3.3 Surveying of infested areas for sample collection.....</b>	<b>44</b>
<b>3.5 Efficacy study of herbicides.....</b>	<b>46</b>
3.5.1 Site of study and experimental design.....	46
3.5.2 Phytotoxicity observation.....	49
3.5.3 Rainfall data collection.....	49
<b>3.6 Data analysis .....</b>	<b>52</b>
<b>CHAPTER FOUR.....</b>	<b>53</b>
<b>4.0 RESULTS.....</b>	<b>53</b>
<b>4.1 Prevalence of <i>Dichapetalum</i> spp. in Mkuranga District .....</b>	<b>53</b>
<b>4.2 Observations for phytotoxic effects on sprayed plants .....</b>	<b>56</b>

4.2.1 General trend of injurious symptoms and leaf killing per cent .....	56
4.2.2 2,4-D treated plants .....	56
4.2.3 Roundup® treated plants.....	57
4.2.4 Paraquat treated plants .....	57
<b>4.3 Evaluation of herbicides efficacy .....</b>	<b>63</b>
<b>4.4 Rainfall trend observations .....</b>	<b>65</b>
<b>5.0 DISCUSSION .....</b>	<b>71</b>
<b>5.1 Discussion on plant prevalence .....</b>	<b>71</b>
<b>5.2 Discussion on botanical changes of sprayed plants .....</b>	<b>74</b>
<b>5.3 Field efficacy evaluation of herbicides on <i>Dichapetalum</i> plants control. ....</b>	<b>76</b>
<b>CHAPTER SIX.....</b>	<b>83</b>
<b>6.0 CONCLUSION AND RECOMMENDATIONS .....</b>	<b>83</b>
<b>6.1 CONCLUSION.....</b>	<b>83</b>
<b>6.2 RECOMMENDATIONS.....</b>	<b>83</b>
<b>REFERENCE .....</b>	<b>85</b>
<b>APPENDICES .....</b>	<b>109</b>

### LIST OF TABLES

Table 1: Spraying volumes (ml) for three herbicides for every ten plants in area of 2.5m <sup>2</sup> .....	52
Table 2: Point Quadrant results for determination of percentage cover for <i>Dichapetalum</i> plants.....	54
Table 3: Trend of leaf killing percentages at a dosage rate of 0.5 kg/ha for three tested herbicides in sixty days of observation.....	57
Table 4: Trend of leaf killing percentages at a dosage rate of 1.0 kg/ha for three tested herbicides in sixty days of observation.....	57
Table 5: Trend of leaf killing percentages at a dosage rate of 1.5 kg/ha for three tested herbicides in sixty days of observation.....	58
Table 6: Trend of leaf killing percentages at a dosage rate of 5 kg/ha for three tested herbicides in sixty days of observation.....	58
Table 7: Trend of leaf killing percentages at a dosage rate of 10 kg/ha for three tested herbicides in sixty days of observation.....	59
Table 8: Trend of leaf killing percentages at a dosage rate of 15 kg/ha for three tested herbicides in sixty days of observation.....	59
Table 9: Analysis of Variance (ANOVA) for Herbicide treatments, Dosage rates and their Interactions on Killing Percentages.....	65
Table 10: Mean killing percentages between herbicides.....	65
Table 11: Mean killing per cent of different dosage rates.....	66
Table 12: Mean killing per cent of dosage rates in three herbicides.....	66
Table 13: Mean killing percents of interactions between herbicide type and dosage rates .....	67

## LIST OF FIGURES

Figure 1:	Molecular Structure of Paraquat Dichloride.....	26
Figure 2:	2, 4-D .....	28
Figure 3:	Glyphosate .....	31
Figure 4:	Map of Tanzania and Mkuranga District, the study area.....	42
Figure 5:	Knapsack hand sprayer .....	50
Figure 6:	The sprayed <i>Dichapetalum</i> plant sprouts .....	51
Figure 7:	Herbicides used for field assessment.....	51
Figure 8:	Frequency distribution of percentage cover of identified <i>Dichapetalum</i> species.....	54
Figure 9:	<i>Dichapetalum mossambicense</i> .....	55
Figure 10:	<i>Dichapetalum stuhlmanii</i> .....	55
Figure 11:	Graphs of trend of leaf killing percentages at a dosage rate of 0.5 kg/ha for three tested herbicides in sixty days of observation .....	60
Figure 12:	Graphs of trend of leaf killing percentages at a dosage rate of 1.0 kg/ha for three tested herbicides in sixty days of observation.....	60
Figure 13:	Graphs of trend of leaf killing percentages at a dosage rate of 1.5 kg/ha for three tested herbicides in sixty days of observation .....	61
Figure 14:	Graphs of trend of leaf killing percentages at a dosage rate of 5 kg/ha for three tested herbicides in sixty days of observation .....	61
Figure 15:	Graphs of trend of leaf killing percentages at a dosage rate of 10 kg/ha for three tested herbicides in sixty days of observation .....	62

Figure 16:	Graphs of trend of leaf killing percentages at a dosage rate of 15 kg/ha for three tested herbicides in sixty days of observation .....	62
Figure 17:	Frequency distribution of killing percentages of herbicides.....	68
Figure 18:	Frequency distribution of killing percentages of dosage rates .....	68
Figure 19:	Frequency distribution of killing percentages of dosage rates for 2,4-D.....	69
Figure 20:	Frequency distribution of killing percentages of dosage rates for Round up@.....	69
Figure 21:	Frequency distribution of killing percentages of dosage rates for Paraquat .....	70
Figure 22:	Graph of Trend of Rainfall between October and February in Mkuranga District. ....	70

**LIST OF APPENDICES**

**Appendix 1: Data on Rainfall Trend in Mkuranga District ..... 109**  
**Appendix 2: Data analysis results from MSTAT-C..... 113**

## ABBREVIATIONS AND SYMBOLS

ae	Acid equivalent
ai	Active ingredient
ATP	Adenoside Triphosphate
BTC	Belgian Technical Co-operation
<sup>14</sup> C	Carbon fourteen
°C	Degree centigrade
Ca <sup>2+</sup>	Calcium ions
CNS	Central Nervous System
DALDO	District Agriculture and Livestock Development Officer
e.g	For example
etc	And so forth: continuing in the same way
<i>et al</i>	And others
Fig.	Figure
g	Grams
g/l	Gram/litre
ha <sup>-1</sup>	Per hectare
i.e	That is
km	Kilometres
kg/ha	Kilogramme/hectare
l	Litre
lb	Pound
LTD	Limited
m <sup>2</sup>	Metre square
mm	Millimetres
®	Trade mark
UDSM	University of Dar es salaam
SUA	Sokoine University of Agriculture
USA	United States of America
(T)	Tanzania
TMA	Tanzania Meteorological Agency

TPRI	Tropical Pesticides Research Institute
v/v	Volume by volume
w/v	Weight by volume

## CHAPTER ONE

### 1.0 INTRODUCTION

Plant poisoning is a worldwide problem and occurs frequently in animals and humans (McKenzie, 2006). Poisonous plants have reduced grazing potential of affected areas in Tanzania (Kidunda, 1996). The presence of poisonous plants in pasture land affects animal performance (Rutgers, 2000). About a thousand species of plants are known to be toxic to livestock and humans in Australia (McKenzie, 2006). Ruminant livestock in developing countries are largely dependent on natural pastures and crop residues for feed (Crowder and Chheda, 1982). The presence of poisonous plants, therefore in pastoral land poses a challenge to animal health and productivity (Ngomuo *et al.*, 1995; Msami, 1999).

Poisonous plants have major economic effect on agricultural enterprises which produce livestock by grazing native pastures. This makes the reduction of the effects of plant poisoning on livestock one of the serious concerns of the pastoral industries (McKenzie, 2006). Losses due to plant poisoning cases include mortality particularly in ruminants, decreased productivity and control measure costs (Pfister, 1988). The average annual range livestock death loss from poisonous plants is estimated at 2% to 5% in most rangelands in Tanzania (Kidunda, 1996). In the Western United States, it has been estimated that yearly cattle and sheep death losses due to poisonous plants are 1% and 3.5%, respectively (Pfister, 1988). In South Africa, high mortality of livestock is caused annually by the plant, *Dichapetalum cymosum* (gifblaar) (Egyed and Schultz, 1986). The toxic factor in *Dichapetalum* plant is monofluoroacetic acid.

This upon ingestion causes a condition called dichapetalosis responsible for livestock losses (Watt and Breyer-Brandwijk, 1962; Vickery and Vickery, 1975).

Plants belonging to the genus *Dichapetalum* occur widely in East Africa and have been responsible for livestock and human deaths (Verdcourt and Trump, 1969; Ngomuo *et al.*, 1995; Msami, 1999). *Dichapetalum* plants are small trees or shrubs, erect or scandent, rarely undershrubs (Hutchinson, 1964). According to Breteler (1990) *Dichapetalum* plants are lianas, lianescent shrubs, shrubs, or small trees with sympodial growth. These plants are widespread in Tanzania, particularly in Coast Region, Dar es Salaam, Lindi, Mtwara, some parts of Tabora region and in Morogoro (Central Veterinary Laboratory, 1970). Millions of hectares of grazing land along the coastal strip are infested by the *Dichapetalum* plants (Breteler, 1990; Meyer and Van Rooyen, 1996). The growth of these plants in coastal areas is favoured by warm, tropical climate and the sandy loamy soil type (Vickery and Vickery 1973). *Dichapetalum* species reported to be found in Tanzania include: *D. deflexum*, *D. braunii*, *D. macrocarpum*, *D. stuhlmannii*, *D. ruhlandii*, *D. edule*, *D. lindicum*, *D. arenarium*, *D. eickii*, *D. crassifolium*, *D. ugandense*, *D. zenkeri*, *D. barbosa*, and *D. mossambicense*. The first five of these listed species are known for their poisonous properties (Verdcourt and Trump, 1969; Vickery and Vickery, 1973; Breteler, 1990; Ngomuo *et al.*, 1995; Msami, 1999 and Ngomuo, 2001).

Mkuranga district is heavily infested with *Dichapetalum* plants; infestation is widespread in virtually all grazing areas of the district. However, majority of livestock keepers in this district are not aware of presence of these plants in their

localities due to lack of knowledge on poisonous plants (Ngomuo *et al.*, 2003). The widespread nature of the plants has to a large extent hindered ruminant livestock production. To solve this problem, there is a need to eradicate the plants. However, the work of eradicating *Dichapetalum* plants is difficult because they have an extensive underground root and stem system, making it a hard-to-kill.

The total land area of Mkuranga District is 2 432 square kilometres with high potential for livestock production; however widespread occurrence of *Dichapetalum* plants is deterrent to livestock production. The records available at District Agriculture/Livestock office for Mkuranga show that the current number of cattle, goats and sheeps in this District is around 3 000, 8 000, and 1 200 respectively (DALDO's office, Personal communication, 2005) . These figures show that the number of ruminant livestock is relatively small compared to other areas of Tanzania with similar size. The widespread occurrence of these plants is most probably, the reason for relatively small number of ruminant livestock. In fact, the number of ruminant livestock in this District is not proportional to its potential and area size.

A number of cases of animals have died of plant poisonings from several livestock farms and have been reported to District Agriculture/livestock office (Mmbaga, S. R. Personal communication, 2005). Most cases occur during dry season and are accompanied with pasture shortages (Mmbaga, S. R. Personal communication, 2005). Currently animals such as cattle, goats and sheep are being introduced to Mkuranga District by several non-governmental organizations such as Heifer Project International (HPI) and Village Oriented Development Programme (VODP).

However, the efforts made by these non governmental organizations are hampered by *Dichapetalum* plant poisonings (Mmbaga, S. R. Personal communication, 2005).

Previous studies on *Dichapetalum* plants have just hinted on widespread infestation in the Mkuranga District. There is no quantification on the problem in terms of exact species present and their densities, a situation that led to many livestock keepers being unaware of the presence of these poisonous plants. The magnitude of infestation is unclear. Consequently, it is difficult to put in place control programmes. As poisonous properties of *Dichapetalum* species vary, knowing prevalent species and their densities can assist on decision regarding where to direct control efforts, set measurable objectives and assess how well those controls have worked.

According to Van Rijn (2000) herbicides might be necessary for controlling hard-to-kill perennial weeds, which can not be removed practically by hand because of their re-growth characteristics. Swarbrick *et al* (1995) reported best performance of herbicides on sprouts. Rana and Singh (1999) eradicated *L. camara* in India by using glyphosate sprayed on to regenerated growth, cut 4 months previously. Herbicides such as Picloram (Tordon®), Glyphosate (Roundup®) and others have been used to eradicate *Dichapetalum* species from pastures in Coast Region but there has been variable success with this technique (Ngomuo *et al.*, 2003). Variable success of herbicides applied is possibly due to inappropriate application techniques and application outside recommended period (Forbes *et al.*, 1980; and Meyer and Van Rooyen, 1996). Ngomuo *et al.* (2003) reported lack of information pertaining to application methods, optimum dosages and effectiveness of herbicides against

*Dichapetalum* plants. Keeler (1977) emphasized the importance of right method of application in order to achieve good herbicide performance.

Generally speaking lack of information on scope and extent of *Dichapetalum* infestation and inappropriate method of herbicides application to these plants are main reasons behind poor control of the plants. This is substantially responsible for unsatisfactory ruminant livestock production in the district. The ruminant livestock production is the one primarily hampered as these animals depend on grazing directly on natural pastures. In view of the aforesaid, there was a great need to conduct research on these plants, so as to assess *Dichapetalum* infestation in this district with a view to establish prevalent species and their coverage in infested areas which is an important stage toward embarking on control programmes; and also doing assessment on how herbicides perform under field conditions in order to investigate the effectiveness of chemical method, as attempts to control *Dichapetalum* plants by manual methods proved unsatisfactory due to regrowth characteristics of the plants.

The general objective of this study was to understand scope and size of the infestation, and establishing effectiveness of herbicides on *Dichapetalum* plants with long term objective of improving ruminant livestock production through laying down basic foundations for future control programmes in Mkuranga District of Tanzania. In order to accomplish this goal, this study was conducted based on the following specific objectives: (a) surveying sites infested by *Dichapetalum* plants by the scientific quadrant method, (b) identifying of *Dichapetalum* plants in herbarium and determination of percentage cover, (c) testing suitable method for herbicide

application and establishing optimum application rates (dosages) and (d) tracing out phytotoxicity, assessing trend and overall killing percentage of tested herbicides.

## **CHAPTER TWO**

### **2.0 LITERATURE REVIEW**

#### **2.1 Animal plant poisoning overview**

##### **2.1.1 Toxic plant constituents**

Plants contain a large number of biologically active chemicals. Some of these have been found to be extremely useful for treating various human and animal diseases (e.g. digitoxin, colchicines and atropine). However, some plant constituents produce adverse health effects following exposure (Tisserand and Balacs, 1995). The known plant toxins are part of a group of chemicals known as secondary compounds or metabolites because they are not essential to the basic biochemistry of the plants. The presence of certain chemicals in plants is believed to confer some degree of protection from plant predators such as insects and ruminants (Tisserand and Balacs, 1995).

##### **2.1.2 Defensive mechanisms of animals against poisonous plants**

Under normal circumstances, plant-eaters (including mammals and insects) have developed ways of avoiding being poisoned. These mechanisms are through modified behaviour and through chemical-based defences (Cheeke, 1998). Mammalian plant-eaters generally use a wider range of plants and depend on taste, smell and learning to avoid toxic species. They have more broadly-based chemical detoxication systems in the chemical mechanisms of their livers and other organs and in the microbes in their stomachs and intestines (Doll, 2006). Because plant-eaters are well adapted to their natural environments, poisoning occurs only when those environments are seriously

disturbed (for example by drought or human interference) and animals are forced to leave their accustomed ecological niches to feed on dangerous plants (Pfister, 1988).

### **2.1.3 Predisposing factors for plant poisoning**

Animal unfamiliar with the plants in the new environment are likely to be poisoned (Auda, 1975; Nwude and Parsons, 1977). According to Ngomuo (2001) factors that predispose animals to plant poisoning include overgrazing of pastures that contain poisonous plants, subjecting animals to hungry situations, improper harvesting of hay may result in small quantities of toxic plants being fed to animals and poor storage of pasture.

Poisonous plant occurrences are more frequent in dry years as droughts create situations in which animals often graze plants they would otherwise not eat and producers may harvest fields or plants that are not usually harvested due to pasture shortage (Mac Dougall, 1996 and Doll, 2006). The accumulation of potentially toxic concentrations of nitrate in forages most often occurs during periods of drought that can lead to plant poisoning once ingested by animals (Pfister, 1988).

Weeds usually not considered toxic might become poisonous under certain conditions. For example, weeds may become more palatable to livestock following an herbicide application in a pasture or fencerow. This may result in animals eating plants they would normally avoid consuming (Doll, 2006).

It is possible for animals to adapt to a potentially toxic plant if exposure is allowed to occur over a period of time. For example, ruminants adapted to oxalate-containing

plants such as *Halogeton glomeratus* can tolerate concentrations that are lethal to non-adapted animals (Cheeke, 1998).

#### **2.1.4 Factors determining effects of poisonous plants to animals**

Severity of plant poisonings is greatly influenced by many factors, they include: the chemical nature of the toxic constituent; amount and time period of the toxin eaten; parts of the plant eaten; the general condition and stage of maturity of the plant; environmental conditions in which the plant is growing. On the other hand, species, age, size, sex and general condition of the animal influence severity of plant poisoning (Pfister, 1988).

#### **2.1.5 Diagnosis and treatment of plant poisoning**

The diagnosis of plant poisonings can be difficult (Minnaar, 2000). Ingestion of many plants produces non-specific clinical signs that must be differentiated from other disease conditions. In addition, death due to toxic plant ingestion often does not result in characteristic post-mortem lesions (Cheeke, 1998).

For proper diagnosis the history, clinical signs, and post-mortem lesions should be investigated. In suspected plant poisoning, animals should be removed from contaminated pastures as quickly as possible. For most cases, there is no effective treatment; however symptomatic treatment may be instituted (Ngomuo, 2001).

Long term control of poisonous plants involves eradication of them from pastures and avoiding exposure of animals to area of infestation (Minnaar, 2000).

## 2.2 Background information of *Dichapetalum* plants

### 2.2.1 Classification

*Dichapetalum* plants fall under phylum Spermatophyta and class Dicotyledons. These plants belong to family Dichapetalaceae. The family was formerly known as Chailettiaceae (Cornell University, 2006). The family comprises about 154 species in 4 genera, and occurs in tropical regions (Hutchinson, 1964). The most interesting morphological feature of this family is the frequent concrescence of the petiole and the peduncle of the inflorescence, the latter then apparently arising from the base of the leaf-blade (Mabberley, 1987).

The genera under family Dichapetalaceae are *Dichapetalum*, *Stephanopodium*, *Falya*, and *Tapura*. The largest and most widely spread genus is *Dichapetalum*, which is found in most parts of the tropics, particularly in Africa. There are about 100 species under this genus, which occurs in tropics and subtropics (Hutchinson, 1964).

### 2.2.2 Toxic constituents of *Dichapetalum* plants

Monofluoroacetic acid is the toxic principle of *Dichapetalum* spp. Monofluoroacetic acid is organofluoride compound. It belongs to fluoroacetic acid derivative group (Chenoweth, 1949). Its synonyms include 2-fluoroacetic acid, acide-monofluoracetique, acido monofluoroacetico, Compound 1080, cymonic acid, fluoroacetate, fluoracetato de sodium, fluoroacetic acid sodium salt, fluoroacetic acid, fluoroethanoic acid, gifblaar poison, monofluorazijnzuur, onofluoressigsauere, monofluoroacetate, monofluoroacetic acid, sodium fluoroacetate, sodium

monofluoroacetate, sodium perfluoroacetate (Chenoweth, 1949; and Meyer *et al.*, 1992).

In 1944, potassium monofluoroacetate ( $\text{CH}_2\text{FCOOK}$ ) was isolated from *Dichapetalum cymosum*, a South African plant, and was the first known example of a naturally occurring organic fluoride (Chenoweth, 1949). Other plants that contain these toxic constituents include *Gastrolobium* species such as *Gastrolobium grandiflorum* and *Acacia* species such as *Acacia georginae* (McKenzie, 2006).

Some species are vulnerable to fluoroacetate, while others, particularly some Australian and African varieties, produce the toxin and possess natural resistance (Lien *et al.*, 1978). According to Meyer *et al.* (1992) the plants contain fluoroacetyl-CoA hydrolase enzyme which protects them from being poisoned by their production of fluoroacetate.

Ingestion of cooked meat from 1080-poisoned animals is not thought to constitute a human health hazard due to the low concentration of toxicant present in muscle tissue, and degradation of fluoroacetate at cooking temperatures (Temple and Edwards, 1984). Poisoned carcasses are a significant risk to dogs due to their susceptibility and feeding habits (Gooneratne *et al.*, 1995). Persistence of fluoroacetate in carcasses is dependent on a number of factors such as temperature, weather conditions and the size and amount of decomposition of the animal (Walker and Lien, 1981).

### 2.2.3 Fluoroacetate mode of action

Fluoroacetic acid is not toxic *de novo*, but when ingested it undergoes transformation in the body to fluoroacetyl-CoA, thereby gaining entry to the citric acid cycle. Citrate synthase then condenses fluoroacetate with oxaloacetate to form fluorocitrate, a process dubbed as lethal synthesis (Buffa and Peters, 1950). The time taken for lethal synthesis is on the order of 30 - 150 minutes, and symptoms will be exhibited at some point after this latent period, depending on the species and dose (Egekeze and Oehme, 1979). The toxic isomer (-)-erythro-2-fluorocitrate exerts its action mainly on the citric acid cycle enzyme aconitase, where it is thought to act as a suicide substrate at the sulphhydryl-bearing active site of the enzyme, causing a blockade at this point of the cycle (Clarke, 1991). ATP is rapidly depleted, disrupting energy-dependent processes. The capacity of cells to restore inactivated aconitase and combat oxidative damage is probably also hindered. Other enzymes affected include pyruvate dehydrogenase kinase (Taylor *et al.*, 1977), succinate dehydrogenase (Mehlman, 1967), phosphofructokinase (Godoy and del Carmen Villarruel, 1974), and possibly ATP-citrate lyase (Rokita and Walsh, 1983). As a result of the blockade at aconitase, citrate levels rise dramatically, causing chelation of divalent metal ions, especially  $\text{Ca}^{2+}$ . Depletion of these ions at a CNS site may be responsible for seizures in certain species (Hornfeldt and Larson, 1990). Serum citrate levels generally provide a reliable biochemical marker of fluoroacetate intoxication (Bosakowski and Levin, 1986).

#### **2.2.4 Fluoroacetate metabolism**

Defluorination of fluoroacetate has been demonstrated in several species, with most metabolism occurring in the liver (Mead *et al.*, 1985). The development of tolerance to increasing doses of fluoroacetate has been reported in rats and mice, whereby a dose of 0.5 mg/kg protects rats against a dose of 5 mg/kg for a period of 48 hours (Chenoweth *et al.*, 1951). The mechanism of fluoroacetate resistance in certain species is not well understood, but rate of defluorination does not appear to play a significant role (Mead *et al.*, 1985). In sheep, there was found to be a total excretion of unchanged sodium fluoroacetate ranging from 7.5% to 33.9%, most of which occurred in the urine within the first 48 hours (Eason *et al.*, 1993). Rats dosed with 0.25 mg/kg <sup>14</sup>C-labelled fluoroacetate excreted the following cumulative percentages of radiolabel in the urine : 6% at 4 hours, 32% at 24 hours, and 45% at 72 hours. After 72 hours various organs each contained between 0.5-1% of the total amount labelled (Teclé and Casida, 1989).

#### **2.2.5 *Dichapetalum* poisoning symptoms**

There may be a latent period of up to 6 hours or more during which minor symptoms are exhibited, including vomiting, tingling of nose and numbness of face. Carnivores such as dogs appear to be more susceptible to CNS effects, while herbivores show predominantly cardiac effects which evoke less prominent symptoms. Omnivores such as humans show mixed symptomology (Norris, 2001).

Signs of *Dichapetalum* poisoning in ruminants include sudden death, dullness, tachycardia, rapid and weak pulse, dyspnea, loss or decreased appetite, excessive salivation, rumen hypomotility, blindness; trembling, chilling, inability to stand,

incoordination, opisthotonus; hyperesthesia, grunt, decreased or absent milk production; increased frequency of urination (Ngomuo *et al.*, 1995; Minnaar, 2000 ).

Diagnosis is usually made on the basis of verified exposure, clinical signs, necropsy findings and chemical analysis. Samples for analysis should include suspected baits, vomitus, stomach contents, liver and kidney (Minnaar, 2000). Elevated citric acid levels in kidney and serum is indicative of fluoroacetate (or fluorocitrate) poisoning when correlated with clinical history (Bosakowski and Levin, 1986). Differential diagnosis must be made amongst compounds such as strychnine, chlorinated hydrocarbons, plant alkaloids and lead (Minnaar, 2000).

#### **2.2.6 Dichapetalosis treatment**

So far no therapeutic measures have been developed for the prevention. Acetamide has demonstrable therapeutic value as an antidote for the prevention of experimental gifblaar poisoning in sheep. Emesis, gastric lavage, and administration of adsorbents such as activated charcoal have been tried to limit absorption of fluoroacetate in the gastrointestinal tract (Egyed and Schultz, 1986).

#### **2.3 Perspective of weed control in pastures**

Weed control in pastures can be a very difficult challenge (Rutgers, 2006). In pastures, the most undesirable traits a plant may have are that it is poisonous or not consumed by animals (Salzer and Holen, 2006). Weeds can become a serious problem in pastures because they compete with desirable pasture species and can reduce the longevity and nutritional value of a pasture stand (Rutgers, 2006). An

effective weed control program is essential to establish and maintain highly productive pastures and animal performance (Salzer and Holen, 2006).

Chemical weed control can be a challenge because of its selective or nonselective means of controlling specific plant species (Beck, 2004). Weeds can be satisfactorily checked by integrated system of control methods, consisting of a combination of some or all of cultural, ecological, manual, mechanical, chemical and biological control (Van Rijn, 2000).

#### **2.4 Scope of chemical weed control**

Chemical control is simply the use of various herbicides to kill weeds. Herbicide is a chemical compound used to kill or control plant growth or algae (Crafts and Robbins, 1962). Herbicides provide a more effective and economical means of weed control than cultivation, hoeing, and hand pulling (Vallentine, 1971).

The first chemicals used in weed control were inorganic compounds such as brine, salt and ashes. These were used by the Romans to sterilize the land as early as biblical times (Ware, 2000). Chemical control of undesirable range plants by the use of herbicides has developed rapidly in recent years and is now the mostly widely used means of removing noxious shrubs and weeds from grazing lands (Vallentine, 1971). Chemical weed control in pastures is much more prevalent in Central and South America and South-East Asia than in Africa and South Asia (Swarbrick and Kent, 1982; Scanlan, 1984; Scanlan and Fossett, 1984).

Studies carried out in the South African Kruger National Park by Erasmus *et al.* (1993) showed that chemical control was cheaper and caused less disturbance resulting in higher biodiversity than mechanical control.

However, disadvantages include: no chemical control has yet proven effective for some species, cost of control may outweigh expected benefits on low-value range, the careless use of chemicals is hazardous to cultivated crops and may contaminate water supplies and herbicides may kill associated forbs shrubs important for grazing (University of Minnesota, 2002).

A number of factors affect the effectiveness of the chemical treatment and they include: plant size, time of application, mode of application, and the use of surfactant. Use of herbicide in uncut stands may not be effective in preventing eventual regrowth. In such cases combination of mechanical and chemical control may be the best (University of Minnesota, 2002). The seasonal response of *Lantana camara* to applications of fluroxypyr, metsulfuron-methyl, glyphosate and dichlorprop has recently been reported by Hannan-Jones (1998).

Herbicides were found to be effective in control of a number of undesirable plants; scattered infestations of *Imperata cylindrical* in pastures, was successfully controlled with repeated applications of glyphosate at 1.5-2 kg ai/ha. Imazapyr at 1 kg/ha is also an effective herbicide. Both herbicides kill foliage and rhizomes (Brook, 1989 and Terry, 1994). According to Swarbrick *et al.* (1995) *Lantana camara* can be controlled by wetting foliage or cut stumps with 0.5 –1 % glyphosate or 0.5% 2, 4-D amine. Recommended rates for application of 2, 4-D for post-emergent control of

weeds in most crops and pastures range from 0.28 to 2.2 kg/ha (Loos, 1975). For poisonous plants, Picloram (Tordon®) is applied at a rate of 0.09-0.90 kg/acre (Ghassemi *et al.*, 1981).

The Australian experience in controlling *L. camara* indicates that some herbicides are more effective on particular lantana forms. The most effective herbicides belong to the phenoxy acid (2, 4-D, 2,4,5-T and dichloroprop ), benzoic acid (dicamba) and pyridine groups. Glyphosat, sulfonyleureas (metsulfuron methyl) and imidazolinones (imazapyr) also show good activity. Photosynthetic herbicides (triazine and urea) are not effective (Swarbrick *et al.*, 1995). A single July application of Imazapyr (Arsenal®) at 1.1 kg ae ha<sup>-1</sup> eradicated (100% control) common bermudagrass {*Cynodon dactylon* (L.) Pers.} (Griffin *et al.*, 1994)

## **2.5 Herbicides**

### **2.5.1 Herbicide classification**

Herbicides are classed as selective when they are used to kill weeds without harming the crop and as nonselective when the purpose is to kill all vegetation. They are also classified as contact or translocated (Crafts and Robbins, 1962).

Contact herbicides kill the plant parts to which the chemical is applied and are most effective against annuals, those weeds that germinate from seeds and grow to maturity each year. Complete coverage is essential in weed control with contact materials (Weed Science Society of America, 1983).

Translocated herbicides are absorbed either by roots or above-ground parts of plants and are then circulated within the plant system to distant tissues. Translocated herbicides may be effective against all weed types; however, their greatest advantage is in the control of established perennials, those weeds that continue their growth from year to year. Uniform application is needed for the translocated materials, whereas complete coverage is not required (Weed Science Society of America, 1983).

Another method of classification is the timing of herbicide application with regard to the stage of crop or weed development. The three categories of timing are preplanting, preemergence, and post emergence. Preplanting applications for control of annual weeds are made to an area before the crop is planted, within a few days or weeks of planting. Preemergence applications are completed prior to emergence of the crop or weeds, depending on definition, after planting. Post emergence applications are made after the crop or weed emerges from the soil (Weed Science Society of America, 1983).

### **2.5.2 Herbicide Families and their injurious symptoms**

Herbicide families are a convenient way of organizing herbicides that share a common chemical structure and have similar herbicidal activity (University of Minnesota, 2002).

The growth regulators include the following herbicide families: phenoxy acetic acids, benzoic acids, and the pyridines. Growth regulator herbicides can act at multiple sites in a plant to disrupt hormone balance and protein synthesis and thereby cause a

variety of plant growth abnormalities. Growth regulator herbicides selectively kill broadleaf weeds; however, they are capable of injuring grass crops. Herbicides in this group can move in both the xylem and the phloem to areas of new plant growth. As a result, many herbicides in this group are effective on perennial and annual broadleaf weeds. Herbicide uptake is primarily through the foliage, but root uptake is possible. Injury symptoms are most obvious on newly developing leaves (University of Minnesota, 2002).

The seedling growth inhibitors include the following herbicide families: dinitroanilines, acetanilides, and thiocarbamates. Seedling growth inhibitors interfere with new plant growth, thereby reducing the ability of seedlings to develop normally in the soil (South African Sugar Association, 2004).

The amino acid synthesis inhibitors include the following herbicide families: sulfonylureas, imidazolinones, sulfonamide, and amino acid derivatives. An example of amino acid Derivatives is Glyphosate (Roundup®). Amino acid synthesis inhibitors act on a specific enzyme to prevent the production of specific amino acids, key building blocks for normal plant growth and development. In general, injury symptoms are slow to develop (1 to 2 weeks) and include stunting or slowing of plant growth and a slow plant death (South African Sugar Association, 2004).

The lipid synthesis inhibitors include the following herbicide families: aryloxyphenoxypropionates and cyclohexanediones. These herbicides prevent the

formation of fatty acids, components essential for the production of plant lipids (University of Minnesota, 2002).

The photosynthesis inhibitors include the following herbicide families: triazines, phenylureas, uracils, benzothiadiazoles, and nitriles. Photosynthesis inhibitors shut down the photosynthetic (food producing) process in susceptible plants by binding to specific sites within the plant's chloroplasts (University of Minnesota, 2002).

The cell membrane disrupters include the diphenylether and bipyridylium herbicide families. These herbicides are postemergence contact herbicides that are activated by exposure to sunlight to form oxygen compounds such as hydrogen peroxide. These oxygen compounds destroy plant tissue by rupturing plant cell membranes. Destruction of cell membranes results in a rapid browning (necrosis) of plant tissue (South African Sugar Association, 2004).

Pigment inhibitors prevent plants from forming photosynthetic pigments. As a result, the affected plant parts become white to translucent. Herbicides falling under this family include clomazone (Command®), and norflurazon (Zorial®) (University of Minnesota, 2002).

### **2.5.3 Herbicide Sprays Formulation**

Formulation is the manner in which the toxicant, carrier and other ingredients are mixed. The combination of formulation, rate of application and method of application

generally determine whether a recommended herbicide will be highly selective and effective or not (Johnson, 2005).

For most hand-pump sprayers, the output can be controlled by adjusting the concentration of the pesticide in the tank (Johnson, 2005). If the sprayer is not accurately calibrated, too little or too much herbicide may be applied, resulting in unsatisfactory weed control or damage or death of the seedlings (Norland and Heiligmann, 2005).

The amount of herbicide required to provide adequate control varies greatly with kind of herbicide, plant species and method of application. Herbicide rate recommendations primarily consider optimum toxic effects; higher rates are rarely more effective and may prove detrimental (Weed Science Society of America, 1983). Many translocated herbicides may be reduced in effectiveness when high rate are used because leaves may die so quickly that the toxicant is not absorbed. However, reducing rates below recommended levels to save chemicals may sharply reduce kills, particularly when less than ideal conditions are encountered (Johnson, 2005). Herbicide mixtures have often resulted in no added benefits, and contact herbicides in mixture with phenoxy herbicides are capable of interfering with their translocation within the plant and reducing the permanent kill (Meyer *et al.*, 1970).

#### **2.5.4 Methods of herbicides applications**

In order to achieve good performance of herbicides, applications should be made with recommended herbicide, at the most susceptible plant growth stage and with the recommended form and rate of the herbicide (Keeler, 1977).

Herbicide application based on the area covered involves four categories: band, broadcast, spot treatments, and directed spraying. A combination of broadcast spraying followed by individual plant treatment is often the best method for such as hard-to-kill chaparral plants (Paulsen and Miller, 1968).

Applying herbicides directly to the soil is another means of killing undesirable plants. Soil-active herbicides are more commonly applied as dry granules or pellets. Soil application is less dependent on the stage of growth than foliage sprays but does require rainfall to dissolve and penetrate into the soil. In some areas excessive losses may result from leaching or adsorption on soil colloids (Keeler, 1977).

#### **2.5.5 Influence of environmental factors on herbicide performances**

Environmental factors, principally climatic and edaphic (soil), materially influence the effectiveness of herbicides. These factors principally affect herbicide penetration or translocation or both (Coupland, 1986).

Cook (1963) reported unsatisfactory big sagebrush kills when soil moisture in the upper two feet of silty clay loam soil was below 12 percentage owing to poor translocation of herbicides. He came up with recommendation that translocated

herbicides such as phenoxy herbicides should not be applied in areas with seasonal or prolonged drought.

Rain four hours or more after foliar application of herbicide seldom reduces herbicidal effects. However, rain shortly after application may wash herbicide off the foliage and reduce control (Coupland, 1986).

Warm but not excessive air temperature promotes the entry and translocation of herbicides, respiration, protoplasmic streaming and plant growth and thereby herbicide effectiveness. Low temperatures during the preceding week tend to slow plant growth and retard herbicide activity (Cook, 1963).

High relative humidity increases the effectiveness of most herbicide sprays by reducing plant water stress, by delaying drying of droplets, favoring stomatal opening and probably increasing the hydration and thus permeability of leaves to polar substances. Low humidity effects can partially be overcome by using oil or oil-water emulsions instead of water as diluent (Coupland, 1986).

Wind above minimum levels frequently causes improper distribution of sprays and increases drift of herbicides onto susceptible crop. Aerial application of herbicides is best in early morning and secondarily late evening because of typically reduced wind velocities, higher humidity, and lower temperatures. Light generally promotes herbicide penetration by stimulating stomatal opening and accentuates photosynthesis and translocation (Currier and Dybing, 1959).

### 2.5.6 Assessment of herbicide performance

The performance of herbicide treatments may be measured in a variety of ways and may be concerned with the degree of the control of the weeds or with the response of the crop to the treatment (Bioenergy, 2005). Measurements of average plant biomass (fresh plant weight) are commonly used to help assess overall biological productivity. This is done by cutting down herbage and measuring plant biomass (Bioenergy, 2005).

According to Dow AgroSciences (2006) determination on the level of performance that a herbicide delivers can be done as follows; first of all, it is important to leave an untreated area as a comparison to treated acres. To determine the level of control, compare weed control in the treated area with the number of weeds in an untreated area. The percentage of control can be determined as follows: Place a one-foot square measure on the ground in a representative treated area of the field, and count the number of live weeds (i.e. weed escapes) in the square (Value A). Place the one-foot square measure on the ground in a representative location of the untreated area and count the number of live weeds in the square (Value B). Then, Determine the level of control using the following formula: Percentage level of control =  $[1 - (A/B)] \times 100$ . For example, if you counted 10 live weeds (weed escapes) in a square foot section of the treated area and 82 live weeds in a square foot section of the untreated area, you would have the following level of control:  $10 \text{ weed escapes} / 82 \text{ live weeds} = 0.12195$ .  $(1 - 0.12195) \times 100 = 87.8\%$  control. Porterfield *et al.* (2001) assessed the injury due to trifloxysulfuron in cotton leaves by visual rating.

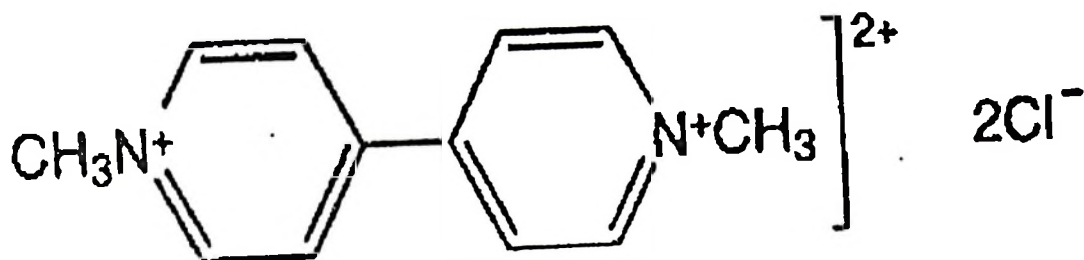
## **2.6 Details of selected herbicides**

### **2.6.1 Bipyridyliums: Paraquat**

The three important herbicides under bipyridyliums are paraquat, diquat and difenzoquat. In each of these compounds there are attachments of two pyridyl rings (Syngenta, 2006). Paraquat has been available to farmers for over 40 years and is currently one of the most widely used herbicides in the world. The herbicidal properties of paraquat were discovered by Syngenta in 1955 and was introduced to world markets in 1962 under the brand name Gramoxone® (Syngenta, 2006).

Paraquat (1,1'-dimethyl-4,4'-bipyridylium) is a broad spectrum, non-selective herbicide. It is very fast-acting, rain-fast within minutes of application, becomes biologically inactive upon contact with soil (Weed Science Society of America, 1983).

Pure paraquat salts are white and the technical products, yellow. They are crystalline solid, odorless, hygroscopic powders, melting and decomposing at 300°C. Paraquat is extremely soluble in water slightly soluble in alcohol and practically insoluble in organic solvents. Paraquat is non-explosive and non-flammable in aqueous formulations. Paraquat is formulated as the dichloride salt. It is corrosive to metals and incompatible with alkylarylsulfonate wetting agents. It is stable in acid or neutral solutions, but is readily hydrolysed by alkali (Weed Science Society of America, 1983).



**Figure 1: Molecular Structure of Paraquat Dichloride**

Paraquat is a foliage-applied contact herbicide. It enters the plant through leaf and green stem tissue and move little once absorbed. Very little translocation occurs so thorough coverage is essential for greatest activity. After application, penetration through the plant leaf surface occurs almost immediately. This absorption is increased by high light intensity, humidity, non-ionic adjuvant in the formulation which ensures good spray retention and wetting of target foliage (Crafts and Robbins, 1962). Herbicide molecules carry a strong positive charge and are tightly bound to soil colloidal matter upon contact, resulting in no soil activity. They require the presence of sunlight for activity and plants treated on cloudy days or in the dark will not express symptoms until placed in the light (Crafts and Robbins, 1962).

The chloroplast is the site of action for paraquat. In the chloroplasts paraquat inhibit the photosynthesis which causes total disruption of cell membranes. The chloroplasts contain the photosynthetic systems of green plants, which absorb light energy that is used to produce sugars. Paraquat is known to act on the photosynthetic membrane system called photosystem I, which produces free electrons to drive photosynthesis.

The free electrons from photosystem I react with the paraquat ion to give the free radical form. Oxygen rapidly reconverts this free radical and in that process produces super oxides. Chemically highly reactive, super oxides attack unsaturated membrane fatty acids, rapidly opening up and disintegrating the cell membranes and tissues. As a result of these dramatic chemical changes, membranes are destroyed, and cell contents leak and mix causing further destruction. It is the rupturing of the cell membranes allowing water to escape from the plant material that leads to the rapid desiccation of the foliage. Chloroplast membranes rupturing give the tissues their frostbitten appearance. The paraquat ion/free radical process then recycles producing further quantities of super oxide until the supply of free electrons ceases. The speed of cell destruction is usually too rapid to allow any measurable translocation from the treated leaf to occur (Syngenta, 2006).

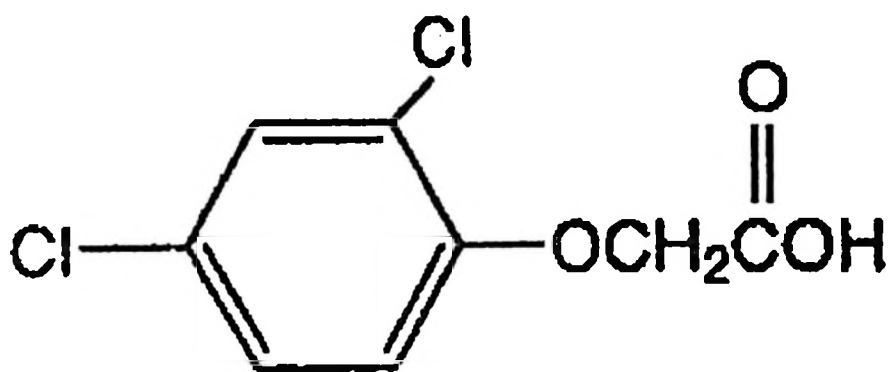
Visible wilting of treated plants is apparent within hours under warm, bright conditions but may take several days under dull, cool conditions. This is quickly followed by the appearance of brown, desiccated or chlorotic tissue (Syngenta, 2006).

As paraquat is not biologically active in the soil it has no adverse effects on soil fauna. Provided spray deposits have dried, there is no health risk to domestic animals entering fields sprayed with paraquat, or to livestock grazing on vegetation sprayed at normal recommended dilutions. Paraquat binds tightly to plant tissues, so is not easily absorbed in animals. Any trace amounts are rapidly excreted (Satchell, 1983).

### 2.6.2 Phenoxy acetic acids: 2, 4-D compounds

Phenoxy acids are a relatively old group of compounds that date back to the 1940s. Several compounds belong to this class, of which 2, 4-D and 2,4,5-T are the most commonly used. Others are 2, 4-DB, MCPA and silvex (Norris, 1981).

The first phenoxy herbicides to be introduced for commercial use was 2, 4-dichlorophenoxy acetic acid (2, 4-D) in 1944. It has been sold under various trade names, which include Agrotect®, Amoxone®, Chloroxone®, Crop Rider®, Weedar® and Weedone® (Meister Publishing Company, 1981). The various commercial formulations of 2, 4-D are generally either salt or ester formulations. Since its introduction as selective weed killer in 1944, 2,4-D has become one of the most widely used herbicides in the world (Meister Publishing Company, 1981). 2,4-D, MCPA and 2,4,5-T have been used for years in very large volume worldwide with no known adverse effects on human or animal health (Ware, 2006).



**Figure 2:** 2, 4-dichlorophenoxy acetic acid (2, 4-D)

Phenoxy herbicides such as 2,4-D are particularly useful, since they are toxic to a wide variety of broadleaved weeds but not to most species of the grass family (Loos, 1975). The mostly widely used herbicide for the herbaceous species is 2,4-D (Keeler, 1977).

Phenoxy herbicides are usually foliarly applied and are translocated within the food stream of plants. In some cases, phenoxy herbicides applied at higher rates may also exhibit soil activity on emerging broadleaf seedlings (Weed Science Society of America, 1983). Uptake of 2,4-D by plants occur through leaves, stems, and roots. Salts forms of 2,4-D are most readily absorbed through plant roots, whereas the ester forms are readily absorbed through foliage. Both forms are readily translocated within the plant where the herbicide tends to accumulate at the meristematic areas of both shoots and roots (Loos, 1975 and Sassman *et al.*, 1984).

Only minimal losses of 2,4-D activity occur due to photodegradation and, for most formulations, due to volatilization (Weed Science Society of America, 1983). The use of surfactants in 2,4-D formulations (acids and ester formulations) often leads to increased efficacy as consequence of increased absorption of the herbicidal compound as the surfactants tend to dissolve the surface waxes, loosen cuticular structure, and in some case, interact with cuticular lipids (Loos, 1975).

Phenoxy herbicides have complex mechanisms of action resembling those of auxins (growth hormones). They affect cellular division, activate phosphate metabolism, and

modify nucleic acid metabolism. This result in excessive cellular growth with symptoms appearing as abnormal growth of the plant (Crafts and Robbins, 1962).

The selectivity of 2,4-D is due to protoplasmic tolerance resulting from inherent ability of certain plants to resist the toxic action of the chemical. Oxidative destruction of 2,4-D in certain plant species may partially explain selectivity to this chemical, but other mechanisms are undoubtedly involved (Luckwill and Lloyd-Jones, 1960).

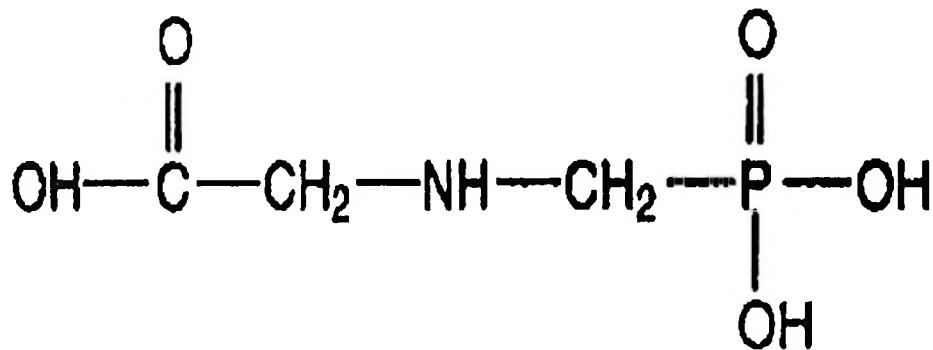
Application of 2,4-D to plants may result in several distinct responses. First, it causes a twisting or bending of the stems and leaves of some sensitive plants. Second, it causes a thickening of leaves and sometimes stems, accompanied by increase in turgor. Third and most important, there is a cessation of growth, followed by death of tissues. This is followed by characteristic browning and drying of stems and leaves and often by decay of roots in the soil (Crafts and Robbins, 1962; Weed Science Society of America, 1983).

### **2.6.3 Phosphono Amino Acids: Glyphosate (Roundup®).**

The best known of this group is glyphosate (Roundup®). Others are glufosinate (Ignite®), fosamine ammonium (Krenite®), and glyphosate trimesium (Touchdown®) (Weed Science Society of America, 1983). Glyphosate [N-(Phosphonomethyl) glycine] (Figure 3) is a nonselective, nonresidual, postemergence material. They are more effective against grasses than broadleaf weeds. They

penetrate rather slowly, thus rainfall shortly after application can reduce effectiveness (Crafts and Robbins, 1962).

Glyphosate is a white, odourless solid with a melting point of 200°C. Pure glyphosate has a density of 0.5 g/cm<sup>3</sup>. At 25°C glyphosate has solubility in water of 1.2 percent (12,000 ppm) but is not soluble in other solvents. The active ingredient of glyphosate is N-(Phosphonomethyl)glycine (Weed Science Society of America, 1983).



**Figure 3: Glyphosate**

In general control of perennials require rates of 1.1 to 4.5 kg/ha and for annuals, the rate is 0.3 to 1.1 kg/ha to achieve control (Weed Science Society of America, 1983). Roundup® offered the best long-term control of *Poa annua* (cool-seasonal annual weeds) in Dormant Bermudagrass Turf (Menn *et al.*, 1999). In India, eradication of *L. camara* from sub-watersheds in the Markanda catchment, Himachal Pradesh, was effective and economical using glyphosate sprayed on to regenerated growth, cut 4 months previously (Rana and Singh, 1999). Glyphosate has been the primary

herbicide for control of common bermudagrass since its introduction in the early 1970s (Dickens and Turner, 1984).

Glyphosate is taken up by plant primarily through the foliage and is then translocated through the plant to roots and other structures. However minimal amounts of material may be taken up by roots under soil conditions that minimize glyphosate adsorption to soil particles (Gottrup *et al.*, 1976). Foliar absorption is greatly increased by increased humidity and by the presence of surfactant in the formulation (Ghassemi *et al.*, 1981).

Glyphosate is readily translocated to underground plant structures and is a strong inhibitor of sprouting in perennial species (Gottrup *et al.*, 1976). Glyphosate is not metabolized in plants, although complete and rapid degradation occurs in both soil and water by microbiological activity but not by chemical activity (Rueppel *et al.*, 1977; Ghassemi *et al.*, 1981).

Its mechanism of action appears to be the inhibition of aromatic amino acid synthesis, which results in the inhibition of nucleic acid metabolism and protein synthesis (Crafts and Robbins, 1962). These herbicides inhibit enzymes critical to the production of certain amino acids. Amino acids are important building blocks in the production of proteins. Proteins are important structural components, constituents of cell membranes and as enzymes are regulators of metabolic processes. When amino acid production stops, plant growth decreases and finally is stopped (Weed Science Society of America, 1983).

Because so many plant processes are affected, many symptoms can be produced including stunting, chlorosis, reddening, and distortion of terminal growth. Treated plants stop growing, wilt, become chlorotic and then necrotic. This is a slow process and may require 10 to 14 days. Trees and shrubs treated with sublethal doses may initiate new leaves which are twisted, curled, or generally malformed (Crafts and Robbins, 1962; Weed Science Society of America, 1983).

## **2.7 Weed resistances to herbicides**

### **2.7.1 Meaning and history of weed resistance**

Herbicide resistance refers to the inherited ability of a weed or crop biotype (A biotype is a group of plants within a species that has biological traits that are not common to the population as a whole) to survive a herbicide application to which the original population was susceptible. In Australia a biotype of annual ryegrass is resistant to at least five different herbicide groups. This is called multiple resistance (Mallory-Smith *et al.*, 2006). Howat (1987) reported resistance of ryegrass (*Lolium rigidum*) to many different herbicides, including diclofop-methyl, fluazifop-methyl, chlorsulfuron, simazine and trifluralin. *Lantana camara* is resistant to triclopyr, a widely used herbicide for woody weed control (Goodall and Naude, 1998).

The first identified herbicide-resistant weed, spreading dayflower (*Commelina diffusa*), which is resistant to 2, 4-D, was identified in 1957 in a sugarcane field in Hawaii. Since then, more than 200 weeds resistant to one or more herbicides have been identified worldwide (Weed Science, 2006). Resistance of weeds to chemical

herbicides is a problem of rapidly growing importance in agriculture (Gorrdard *et al.*, 1995; Gunsolus, 1999).

### **2.7.2 Herbicide resistance mechanisms**

Currently, the three known resistance mechanisms that plants employ are: an alteration of the herbicide site of action, metabolism of the herbicide, and removal of the herbicide from the target site (sequestration) (Gorrdard *et al.*, 1995). Repeated use of a product for more than two years could develop a herbicide resistance problem (Gunsolus, 1999).

### **2.7.3 Diagnosis of herbicide resistance**

Initial suspicion of resistance usually results from unsatisfactory weed control following a herbicide application. However, resistance should not be assumed to be the cause and other reasons should be investigated first (Iowa State University, 2005). Indicators for a rise of herbicide resistance include the presence of living plants adjacent to dead individuals and a gradual decline in control over a period of years (Vitolo, 1999).

### **2.7.4 Control measures of herbicide resistant weeds**

Once herbicide resistance has been diagnosed, the next is to develop a comprehensive weed control program to manage the problem. According to Vitolo (1999) the strategies for avoiding and managing problems with herbicide resistant weed biotypes include Using herbicides only when necessary, herbicide rotation, using short-residual herbicides, rotating crops, particularly those with different life cycles.

Combine, where feasible, mechanical weed control practices such as rotary hoeing and cultivation with herbicide treatments. Clean tillage and harvest equipment before moving from fields infested with resistant weeds to those that are not.

## **2.8 Herbicide tolerant plants**

### **2.8.1 Background information on herbicide tolerance of plants**

Herbicide tolerance (HT) is the ability of a plant to survive the application of a specific herbicide. In these plants, a gene is present in these plants that make them tolerant of a particular herbicide or group of herbicides (Cooperative Research Centre, 2006).

Some plants are naturally tolerant to a specific herbicide, while others develop this tolerance in the evolutionary process of adapting to their environment (sometimes plants develop a tolerance to a specific herbicide because the herbicide was not used properly for weed management). Other herbicide-tolerant plants were developed through biotechnology (Moss and Cussans, 1991). Herbicide-tolerant weed species appeared before the adoption of agricultural biotechnology, mainly due to poor agronomic practices (Cooperative Research Centre, 2006).

### **2.8.2 Biotechnology in developing herbicide-tolerant crops**

Plant breeders and researchers are working to produce crops that are resistant or tolerant to herbicides (Crawley *et al.*, 1993). Simazine-resistant groundsel *Senecio vulgaris*, was discovered in 1978 (Heap, 1997). This was the first herbicide-tolerant weed to arise in agriculture through natural selection (Ryan, 1970). Hall *et al.* (2000) reported on a triple tolerant canola, recovered from a farmer's field in northern

Alberta. Studies have shown that herbicide tolerant plants are no more invasive of cultivated or natural habitats than their herbicide susceptible counterparts, unless the relevant herbicide is used exclusively to eliminate competing vegetation (Downey, 1999). An imidazolinone-tolerant wheat (*Triticum aestivum*) mutant in the winter has been identified and characterized. The mutant was isolated from a population derived through seed mutagenesis of the variety with an aqueous solution containing sodium azide (Newhouse *et al.*, 1992).

There are four ways to create herbicide tolerant plants: Herbicide tolerance created by natural selection, herbicide tolerance created using naturally occurring genes. On the other hand herbicide tolerant plants can be created by mutagenesis and traditional cross breeding (Biotechnologyonline, 2006).

### **2.8.3 Management of herbicide-tolerant weeds**

Farmers have traditionally used a number of methods to manage weeds that have become herbicide-tolerant, whether naturally or through genetic modification. For example, they may use a different herbicide to control the weeds, till the land immediately before they plant their seed, use herbicide mixtures to treat fields, rotate the herbicides they use, use non-chemical weed control methods such as silage and green manure, rotate the crops planted in a field, swath at the optimum crop stage (Hall *et al.*, 2000; Canadian Food Inspection Agency, 2006).

## **2.9 Study for the structure and composition of plant communities.**

### **2.9.1 Sampling for survey research**

Survey research is based on sampling, which involves getting information from only some members of the population. Samples can be drawn in several different ways, such as probability samples, quota samples, purposive samples, and volunteer samples (Greig-Smith, 1983).

Purposive sampling is a sampling method in which elements are chosen based on purpose of the study. In this kind of sampling a surveyor tries to create a representative sample without sampling at random. Purposive sampling may involve studying the entire population of some limited group or a subset of a population (Fowler, 1993). One of the commonest uses of purposive sampling is in selecting a group of geographical areas to represent a larger area; it involves making a planned selection of specific cases (List, 2005). Purposive sampling is very convenient in that it doesn't require much time and resources, however the sample might not be actual representative of the population, and this method is largely limited to exploratory research (Babbie, 1973).

### **2.9.2 Description of plant communities**

The description of vegetation, with or without concurrent recording of factors of the environment, has played a major part in the development of plant ecology and continues to be important (Greig-Smith, 1983).

Two methods of community description predominated in the past and are still used to some extent (Greig-Smith, 1983). The first involves the making of a complete list of species present in a community with assignment of 'frequency symbols' or numerical ratings by inspection. This developed from the subjective assessment of species as rare, occasional, common etc in floras and represents an essentially similar process applied to much smaller area and more closely defined area. The second method depends on the recording of presence or absence of species in small samples of the community under investigation (Raunkiaer, 1934).

### **2.9.3 Percentage cover as a measure of plant communities**

In making detailed studies of sample areas, various measures are available for conveying information about plant communities. One of simplest techniques is to list species in sample quadrants at intervals along a line transect (really a row of point quadrants); the frequency being equal to the proportion of the total number of quadrants in which the species occurs (Greig-Smith, 1983).

Density is a measure of the number of individuals per unit area. The other measure is percentage cover which measure the proportion of the area covered by any species, and is usually expressed as a percentage (Greig-Smith, 1983).

Percentage cover is defined as the proportion of ground occupied by perpendicular projection on to it of the aerial parts of individuals of the species under consideration. Cover is usually expressed as a percentage. It is a convenient measure in short

grassland communities where determination of density would be very laborious or impossible (Smith, 1944).

Cover may be either estimated or measured. Measurement of cover may be made by the point quadrant method, which depends on recording the presence or absence of a species vertically above a number of points in the community being described. The percentage of points above which the species is present represents the percentage cover (Smith, 1944). Percentage cover can be measured using any sampling technique and quadrant of any size. The point quadrant technique is both rapid and accurate for short grassland communities (Smith, 1944).

#### **2.9.4 Quadrant method for study of plant communities**

A quadrant is the main basis of the study for the structure and composition of plant communities (Greig-Smith, 1983).

A quadrant is the sampling unit. It is a square, or less commonly a rectangle or a circle, of defined area, which may be placed either at random or in some regular manner. The number of quadrants that we use must be sufficiently large to give a relatively low figure for standard deviation and thus increase accuracy of information. Accuracy varies with the number of individual counted rather than the area sampled, so that more quadrants are needed for sparse than for abundant species (Greig-Smith, 1983).

Point quadrant is a technique that provides rapid and potentially accurate method of estimating percentage cover (Ashby, 1961). In principle it depends on recording the presence or absence of a species in a large number of very small quadrants. In practice, then all that is necessary is to down a number of pin-points at random on the vegetation, and record the number of hits and misses on the species we are considering. If the sample is large enough, the proportion of hits to the total of hits and misses together will give a reasonably accurate assessment of the percentage cover of that species (Ashby, 1961).

In dense scrub vegetation, where the use of quadrants would be difficult, cover can be estimated reasonably well by using a number of parallel line transects (Greig-Smith, 1983).

## CHAPTER THREE

### 3.0 MATERIALS AND METHODS

#### 3.1 Description of the study area

##### 3.1.1 Location of the study site: Mkuranga District

Tanzania is located in the equatorial zone of East Africa at latitude 1°S and 12°S ; and longitudes 30°E and 39°E. Total area is 945 000 km<sup>2</sup>. According to Kidunda *et al.* (1990) grazing land in Tanzania covers about 51% of the total land area. This research work was conducted in Mkuranga district, which is about 40 km south of Dar es Salaam.

Mkuranga is one of six districts in the Coast Region of Eastern Tanzania. Mkuranga is situated between 38°50' to 39°28' longitudes east and 06°70' to 07°33' latitudes south. Administratively, it is divided into 4 divisions, 15 wards and 101 villages. Mkuranga district has a total area of 2 432 square kilometres with high potential for livestock production in terms of abundant grazing land.

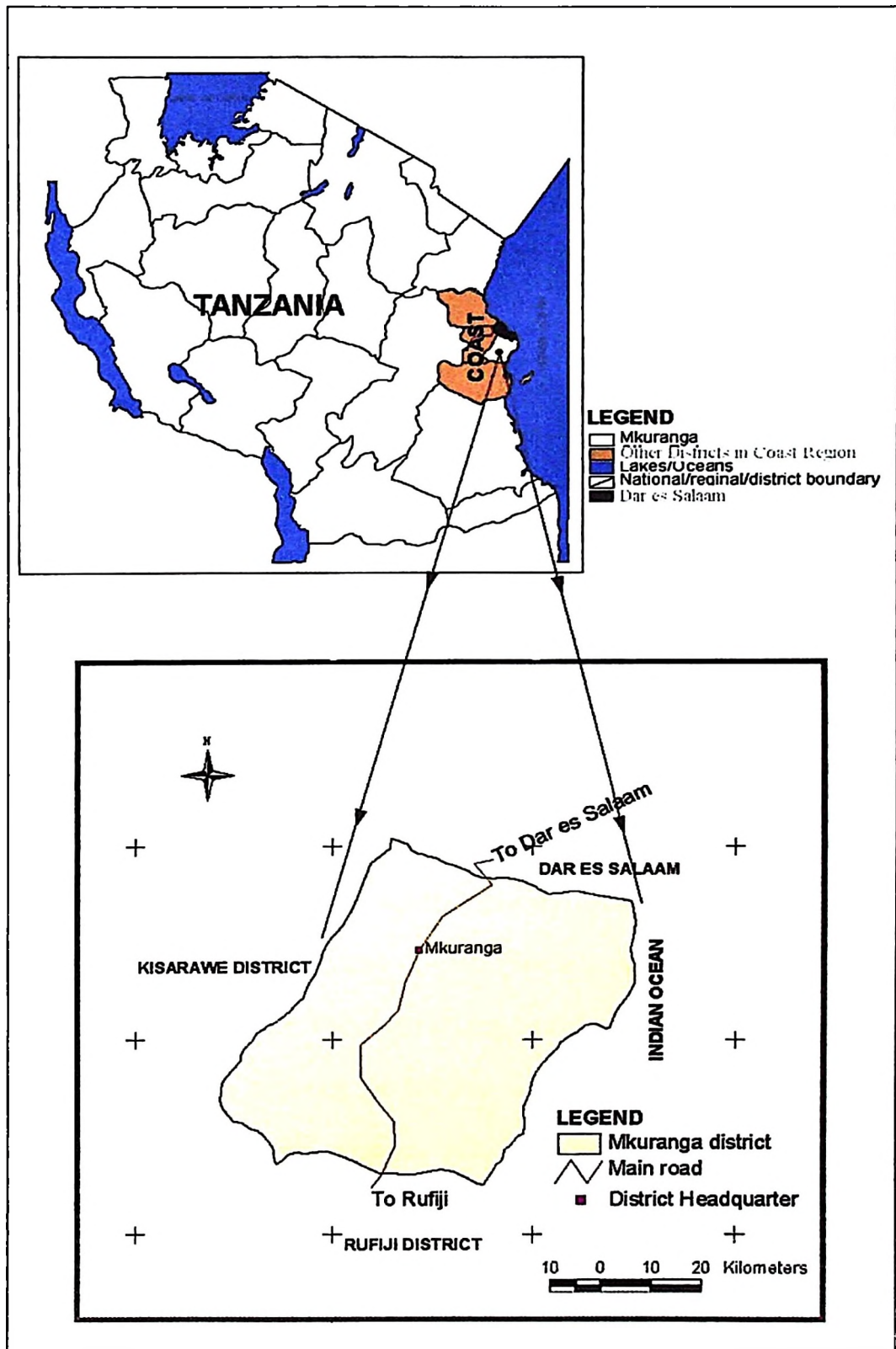


Figure 4: Map of Tanzania and Mkuranga District, the study area.

### **3.1.2 Climatic condition**

Mkuranga has a typical coastal climate, the average temperature is 32°C. There are two rainy seasons per year. The first season includes rains between October and January, while the second begins in March and ends in June. The average rainfall ranges from 80 mm to 12 000 mm per annum (Department of Fund for International Development, 2005).

### **3.2 Sampling procedure and sample size**

Geographical areas in Mkuranga District for this study were selected using Purposive Sampling Method as described by Babbie (1973), Fowler (1993) and List (2005). Purposive sampling was employed as *Dichapetalum* plants are naturally found in some areas, and not uniformly distributed. Under this sampling procedure, samples are chosen based on purpose of the study. As the purpose of this study was to investigate on *Dichapetalum* plants, some geographical areas of Mkuranga District affected with these plants were purposively selected to represent the whole area infested with these plants in the district. Due to budgetary limitations, time constraints and nature of distribution of *Dichapetalum* plants, five neighbouring farms were selected for survey. The neighbouring farms were selected in order to cut down travel costs. The sampling of infested areas for closer studies in each purposively selected farm was done on random basis and therefore the farms surveyed were enough to give representative picture of whole area infested in this District. In each farm, several areas infested with these poisonous plants were visited and studies to assess the infestation in terms of *Dichapetalum* species prevalent and size of infested area occupied by identified species were made. Out of several

infested areas surveyed, one site with relatively large numbers of sprouting *Dichapetalum* plants was selected for field assessment of selected herbicides in order to establish suitable application methods, optimum dosages and general effectiveness against *Dichapetalum* plants.

### 3.3 Surveying of infested areas for sample collection

As it was convenient to conduct survey in neighbouring farms, farms in Mkuranga, Tengelea, Magoza, Kiparang'anda and Mwanambaya were selected for survey. Neighbourhood of these farms and road accessibility were main criteria considered when Purposive Sampling was employed in selection of these farms. The two criteria for selection reduced travel expenses without affecting accuracy of results obtained.

In these farms several areas infested by these plants were visited and collection of *Dichapetalum* plants was done using point quadrant method. Under point quadrant method each area with these plants was evenly divided into small size more or less square areas of land; these land divisions are called quadrants. In the study, quadrants were of different sizes depending on area of infestation. The number of quadrants in one area depended on abundance of plants. The number of quadrants was more in sparsely populated areas than in areas densely infested. A big number of quadrants was created; however only 100 quadrants were chosen randomly using a table of random numbers as reported by Snedecor (1946), for detailed examination of *Dichapetalum* plants they contained. Observation was also done to find out other plants growing in association with *Dichapetalum* plants. In each selected quadrant, *Dichapetalum* plants and associated plants were critically examined and photographs

of some plants in quadrants were taken. Sample plants were collected in a special instrument called press from all 100 quadrants investigated and sent to herbarium for identification.

### **3.4 Botanical identification and percentage cover determination**

The identification of collected plant samples was carried out at the Herbarium of Botany Department of the University of Dar Es Salaam (UDSM). Botanical identification of *Dichapetalum* plants to species level was done using keys described by Polhill (1988). The voucher specimens were kept at herbarium for future reference. The collected associated plants were also identified using appropriate botanical identification keys for them.

After identification, records in terms of presence or absence of identified species in each quadrant were taken for the case of *Dichapetalum* plants. These records were used to determine percentage cover of each *Dichapetalum* species identified. The percentage cover represents proportion at which one species occurred in all quadrants examined. This was calculated as a ratio of number of quadrants with a particular species to total number of quadrants examined (100). This measure of vegetation is important as it indicates proportion of area covered by a particular species.

### 3.5 Efficacy study of herbicides

#### 3.5.1 Site of study and experimental design

The site with Geographical Positioning System (GPS) coordinates of S07\*09.355', E039\*10.574' at altitude of 391 feet was used for efficacy assessment of selected herbicides. It had sprouting *Dichapetalum* plants. The experimental design was a randomized complete block with four repetitions. The chosen site was divided into four replications (plots). In each replication each selected herbicide was sprayed to 60 plants (six doses of each herbicide were used and each dose was tested to ten plants). This means for each replication 180 plants were used as three herbicides were tested. There were four replications for entire study and therefore, a total of 720 plants were treated with herbicides. These herbicides were applied using a 5-litre knapsack hand sprayer with single nozzle (Figure 5). This is a pressure sprayer that has been manufactured by P.T. Plasindo Bhama Prasasta. Apart from spraying herbicides, it can also be used for watering and spraying insecticides. Sprayed plants were identified using tags. The stage of *Dichapetalum* plants sprayed is shown in Figure 6.

University of Minnesota (2002) recommended employing combination of mechanical and chemical control for plants with sprouting tendency in order to prevent eventual regrowth. Basing on this recommendation by University of Minnesota (2002) and studies by Keeler (1977), Swarbrick *et al.* (1995), Rana and Singh (1999); and Van Rijn (2000) a suitable method thought to be effective for these plants was picked up for testing herbicide effectiveness. The study aimed at testing performance of herbicides in sprouts following cutting of *Dichapetalum* plants. However, due to time limitation and period of time to access site given by landowner, the actual stage

of cutting and waiting for sprouting was not practiced in the present study, but instead this study made use of already established *Dichapetalum* sprouts which were 8 weeks old as stated by the landowner. Cutting was done by owners of land in the process of preparing their farms for crop raising. Individual plant spraying was employed as suggested by Paulsen and Miller (1968).

Three herbicides; paraquat, 2, 4-D and glyphosate (Round up®) were selected for testing to demonstrate efficacy of the herbicides on *Dichapetalum* plants. Paraquat used for assessment was manufactured by Gap Chemicals of South Africa and distributed by Balton Tanzania LTD of Arusha, Tanzania. It contained 200 g of paraquat dichloride in one litre. This herbicide was approved by TPRI of Arusha with registration number: HE-0113. Round up® used was manufactured by Monsanto Kenya Limited, Nairobi. It contained 480 g/l IPA salt of N-phosphonomethylglycine equivalent to 360 g/l glyphosate. It is a water soluble formulation. This was approved by the TPRI with registration number HE 0055 and Ugc/94/00014/He/R for Tanzania and Uganda respectively. 2, 4-D tested was manufactured by ATUL LIMITED of Gujarat, India. It was distributed by Twiga Chemical Industries (T) of Dar es salaam, Tanzania. Its concentration was 720 g/l. It contained active ingredient dimethylamine salt of 2, 4 dichlorophenoxyacetic acid at 86.4% w/v, and co-formulants and inerts at 13.6%; all these add up to 100%. It was approved by TPRI, with registration number of HE/0144. All the three herbicides assessed are shown in Figure 7.

Due to lack of labeled field application rates for *Dichapetalum* plants; the dosages tested were based on efficacy studies conducted on plants closely related to

*Dichapetalum* plants (Loos, 1975; Ghassemi *et al.*, 1981; Swarbrick *et al.*, 1995; Hannan-Jones, 1998; and Rana and Singh, 1999). In order to get full information on response of these plants, a range of dosages were applied. These ranged from conventional dosages as suggested by previous works to excessive dosages to rule out unsatisfactory performance attributed to underdosages. At each replication, each herbicide was tested at six different doses (0.5, 1.0, 1.5, 5, 10, 15 kg/ha) and each dose was tested on ten plants. The amount of herbicide in spray volume to be delivered to plants depended on dose and herbicide concentration. The three herbicides tested were water soluble and therefore water was used as diluent. Before determinations of quantity of herbicide, estimations on area to be covered by a plant and quantity of water required for total wetting of a plant were made. One plant was estimated to occupy an area of 0.25 m<sup>2</sup> and therefore the total area to be covered by ten plants is 2.5 m<sup>2</sup>. The quantity of water for each plant was estimated to be 0.5 litre, which amounted to 5 litres for ten plants. Thereafter calculations were made to determine the quantity of herbicide required to spray in an area of 2.5 m<sup>2</sup>. Taking an example of Round up<sup>®</sup> calculations were as follows: at a dose of 1 kg/ha, 1 ha (10,000 m<sup>2</sup>) required 1 kg of Round up<sup>®</sup>, therefore an area of 2.5 m<sup>2</sup> required 0.25 g of Round up<sup>®</sup>. The stock solution in bottle contained Round up<sup>®</sup> at concentration of 360 g/l; therefore to get 0.25 g of this herbicide, 0.69 ml was taken. This amount was mixed with 5 litres of water in knapsack hand sprayer giving concentration of 0.005% w/v. The herbicide at this concentration in the hand sprayer was sprayed on ten plants. The calculations of this kind were also done to determine the amount of herbicides at different doses (0.5, 1.0, 1.5, 5, 10, 15 kg/ha) to be sprayed to other

plants. Table 1 below shows concentrations and volume of herbicides sprayed for every ten plants.

### **3.5.2 Phytotoxicity observation**

Observations on tagged plants in the field were made to trace out phytotoxicity and killing percentages of herbicides. The total number of leaves for each plant at the time of herbicide application was recorded. The field observations were made on the following days after spraying (DAS): 2, 5, 7, 10, 12, 14, 17, 21, 25, 28, 30, 35, 38, 40, 42, 45, 49, 55, 57 and 60. A total of twenty observations were made, with an average of observation at three days interval for the entire study period. During observations injurious symptoms in plants were recorded, special attention was paid to the number of leaves killed in each plant. The trend in killing percentages (KP) and KP at the end of study were recorded for all three tested herbicides. KP for each plant was calculated as a ratio of number of killed leaves to number of leaves at spraying time i.e.  $KP = \text{Killed leaves following herbicide application} / \text{Total number of leaves at the time of herbicide application}$ . This ratio is useful as it indicates degree of the control of herbicide on *Dichapetalum* plants.

### **3.5.3 Rainfall data collection**

Rainfall data was also collected in order to understand the rainfall trend during the study period. Rainfall data for Mkuranga District as recorded by Kongowe Forest Weather Station were collected at TMA (TMA Headquarter weather records, 2006). The focus was on rainfall trend during field efficacy studies. Three categories of

rainfall data were collected; rainfall data at time of present study (October, 2005 to February, 2006); at period of excessive rains, in 1997/98 heavy rains occurred and therefore data in that year was chosen to represent that scenario. The third category was data on mean rainfall trend for that particular period in the Mkuranga District.



**Figure 5: Knapsack hand sprayer**



**Figure 6: The sprayed *Dichapetalum* plant sprouts**



**Figure 7: Herbicides used for field assessment**

**Table 1: Spraying volumes (ml) for three herbicides for every ten plants in area of 2.5m<sup>2</sup>**

Herbicide	Concentration (g/l)	Spraying volumes (ml) at dosage rates (kg/ha)					
		0.50	1.00	1.50	5.00	10.00	15.00
2, 4-D	720.00	0.17	0.35	0.52	1.74	3.47	5.21
Roundup®	360.00	0.35	0.69	1.04	3.47	6.94	10.42
Paraquat	200.00	0.63	1.25	1.88	6.25	12.50	18.75

### 3.6 Data analysis

The overall killing percents recorded at end of the study (day 60), which represents maximum control effect of tested herbicides (2, 4-D, Roundup® and Paraquat) against *Dichapetalum* were, analyzed by MSTAT-C, a statistical procedure developed by Michigan State University (1993). Analysis was done using Randomized Complete Block Design for Factor A (Herbicides), with Factor B (Dosage rates) a split-plot on A.

## CHAPTER FOUR

### 4.0 RESULTS

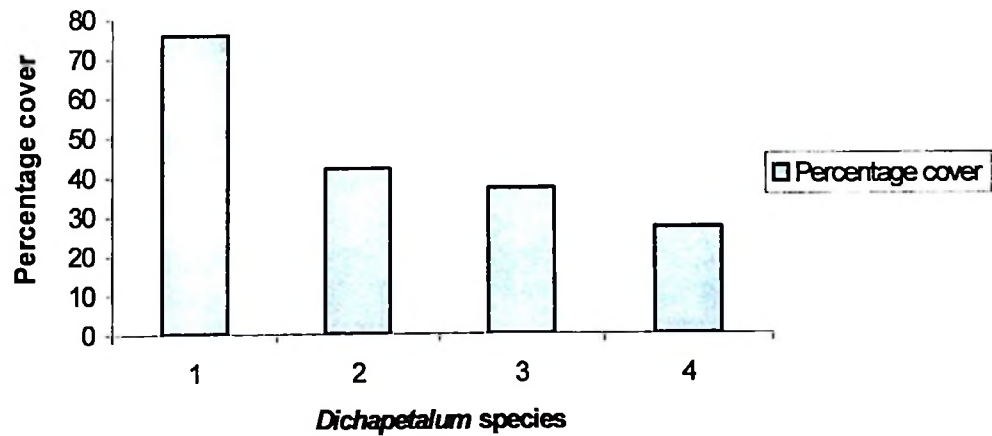
#### 4.1 Prevalence of *Dichapetalum* spp. in Mkuranga District

The botanical survey using Point Quadrant Method carried out in this study established the presence of four *Dichapetalum* species in Mkuranga district. The species identified in the survey were: *Dichapetalum stuhlmanii*, *D. mossambicence*, *D. ruhlandii* and *D. arenarium*. The plants that were identified to be in close association with *Dichapetalum* plants are *Deinbollia borbonica*, *Annona senegalensis*, *Xylothea tetensis* and *Milletia* spp.

The occurrence of *Dichapetalum* species in 100 quadrants examined and percentage cover calculated using Point Quadrant Method for the four species is shown in Table 2. From this table percentage cover for *Dichapetalum stuhlmanii*, *Dichapetalum mossambicence*, *Dichapetalum ruhlandii*, *Dichapetalum arenarium* is 76%, 42%, 37%, and 27% respectively. These observations of percentage cover are presented in terms of frequency distribution in Figure 8. Some photographs of *Dichapetalum* plants taken during the survey are shown in Figures 9 and 10.

**Table 2: Point Quadrant results for determination of percentage cover for*****Dichapetalum* plants**

Quadrant group	Number of quadrants recorded presence of species in quadrant group			
	<i>D. stuhlmanii</i>	<i>D. mossambicence</i>	<i>D. ruhlandii</i>	<i>D. arenarium</i>
1 -10	10	1	0	0
11 - 20	9	6	0	1
21 - 30	9	3	4	8
31 - 40	5	7	7	2
41 - 50	7	2	3	0
51 - 60	9	6	7	1
61 - 70	6	4	2	5
71 - 80	3	3	6	4
81 - 90	8	7	7	1
91 - 100	10	3	1	6
Percentage cover (%)	76	42	37	27

**Figure 8: Frequency distribution of percentage cover of identified *Dichapetalum* species**

1= *D. stuhlmanii* 2= *D. mossambicence* 3= *D. ruhlandii* 4. *D. arenarium*



**Figure 9:** *Dichapetalum mossambicense*



**Figure 10:** *Dichapetalum stuhlmanii*

## **4.2 Observations for phytotoxic effects on sprayed plants**

### **4.2.1 General trend of injurious symptoms and leaf killing per cent**

Botanical observations on sprayed plants for sixty days after spraying have revealed variable responses to herbicide treatments. However, for most herbicides noticeable changes started in the second week after spraying. Changes observed included discoloration, cuts and holes in leaves, scorching (desiccation), leaf wrinkling, and leaf death (necrosis).

Trend of leaf killing percentages throughout the study period at different dosage rates tested for all three tested herbicides is indicated in Tables 3, 4, 5, 6, 7, and 8. The figures for killing percentages shown in these tables represent average values for every ten plants. This trend is presented graphically in Figures 11, 12, 13, 14, 15 and 16. A close look on these tables reveals insignificant killing percent in the first three weeks of observation for Roundup®, at all doses. The trend also shows that 2,4-D and paraquat showed appreciable killing percent just in the first week of the study at all rates tested.

### **4.2.2 2,4-D treated plants**

Significant changes for 2, 4-D treated plants started in the second week; leaves were discolored. Qualitative assessment revealed destruction of leaves in terms of cuts, holes, mild wrinkling and total leaf death. The young leaves at the growing tips (newly developing leaves) were mostly affected; these leaves were destroyed and transformed into dark masses (necrosis); however at doses 5 kg/ha and above old leaves also were destroyed.

#### 4.2.3 Roundup® treated plants

Qualitative assessment for leaves sprayed with Roundup® revealed very slow development of changes; after two weeks leaves showed discolorations, developed cuts and holes. Treated leaves were observed to be stunted. Some leaves had black spots which indicate leaf necrosis. Scorched areas were observed; scorching was prominent at doses 5 kg/ha and above. Roundup® primarily destroyed old leaves, which were transformed into black masses and patches. In all doses tested leaf destruction was prominent in the fourth week of spraying.

#### 4.2.4 Paraquat treated plants

Paraquat produced relatively quick effects; just on second day, scorched areas in leaves dominated especially on leaves treated with dose 5 kg/ha and above. Visual assessment revealed black patches development indicating leaf necrosis in majority of leaves. Discoloration was observed, some leaves developed yellowish coloration, while others had grey and black patches. Some leaves were curled. Paraquat had effect primarily on old leaves in which necrosis of treated leaves dominated.

**Table 3: Trend of leaf killing percentages at a dosage rate of 0.5 kg/ha for three tested herbicides in sixty days of observation**

Herbicide	Killing percentages in days							
	7	14	21	28	35	42	49	60
Paraquat	5.50	5.70	6.34	6.67	7.10	7.25	7.50	7.77
2, 4-D	8.30	8.90	9.11	10.98	11.54	11.68	11.97	12.03
Round up®	0.54	0.98	1.20	1.60	5.56	6.66	7.00	7.24

**Table 4: Trend of leaf killing percentages at a dosage rate of 1.0 kg/ha for three tested herbicides in sixty days of observation**

Herbicide	Killing percentages in days							
	7	14	21	28	35	42	49	60
Paraquat	6.90	7.10	7.89	8.45	8.61	8.99	9.12	9.88
2,4-D	7.20	7.63	8.76	9.15	10.77	11.99	12.95	13.76
Round up®	1.11	1.23	1.98	4.34	6.56	7.88	8.26	9.91

**Table 5: Trend of leaf killing percentages at dosage rate 1.5 kg/ha for three tested herbicides in sixty days of observation**

Herbicide	Killing percentages in days							
	7	14	21	28	35	42	49	60
Paraquat	11.20	13.67	15.54	17.90	18.00	18.67	18.97	19.62
2,4-D	8.76	9.11	10.33	11.35	12.68	13.09	13.95	14.28
Round up®	3.45	5.78	7.05	10.21	11.69	12.23	13.05	13.67

**Table 6: Trend of leaf killing percentages at a dosage rate of 5 kg/ha for three tested herbicides in sixty days of observation**

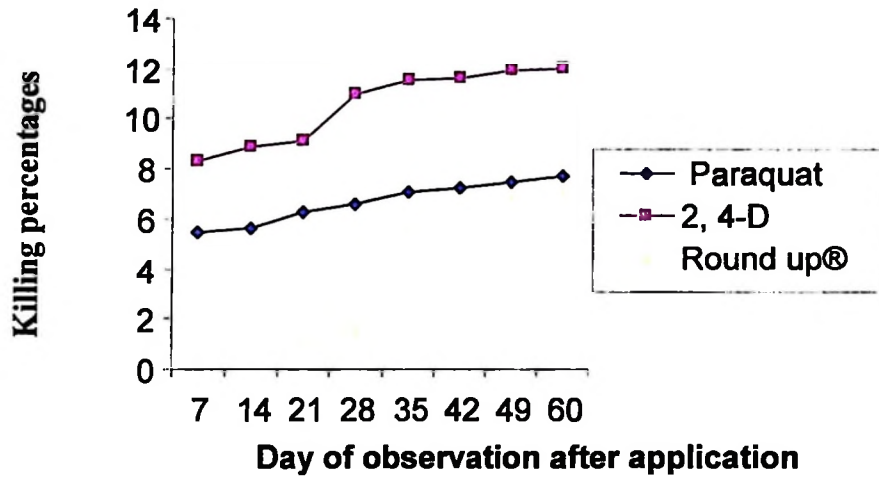
Herbicide	Killing percentages in days							
	7	14	21	28	35	42	49	60
Paraquat	13.46	15.70	17.07	18.08	18.65	19.10	19.56	20.94
2,4-D	8.00	9.15	11.97	12.04	13.24	13.56	14.11	14.91
Round up®	5.68	6.00	7.34	11.08	12.33	13.45	14.88	15.64

**Table 7: Trend of leaf killing percentages at a dosage rate of 10 kg/ha for three tested herbicides in sixty days of observation**

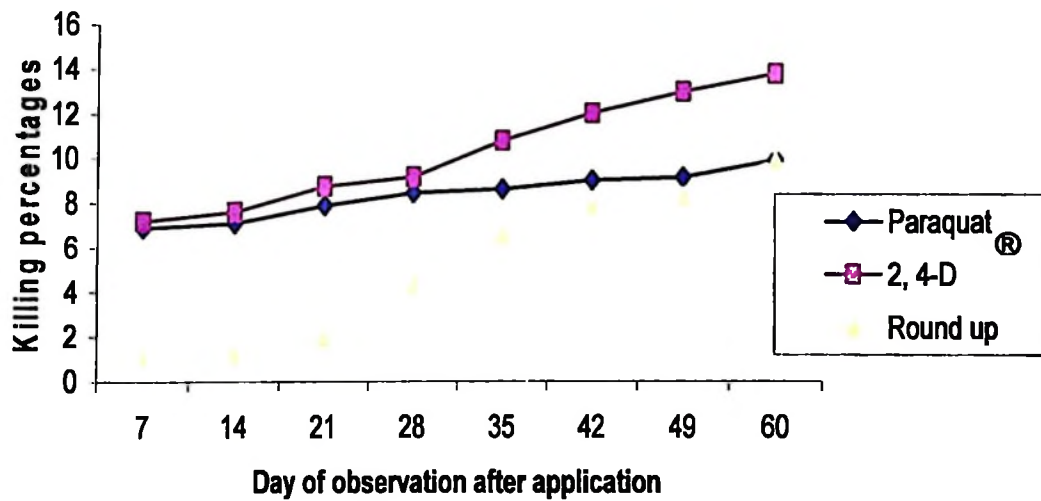
Herbicide	Killing percentages in days							
	7	14	21	28	35	42	49	60
Paraquat	18.19	18.67	19.86	20.65	21.00	21.43	21.86	22.10
2,4-D	9.04	10.46	11.23	13.56	13.78	14.57	14.94	15.24
Round up®	7.99	8.40	8.56	11.67	13.45	15.99	16.96	17.75

**Table 8: Trend of leaf killing percentages at a dosage rate of 15 kg/ha for three tested herbicides in sixty days of observation**

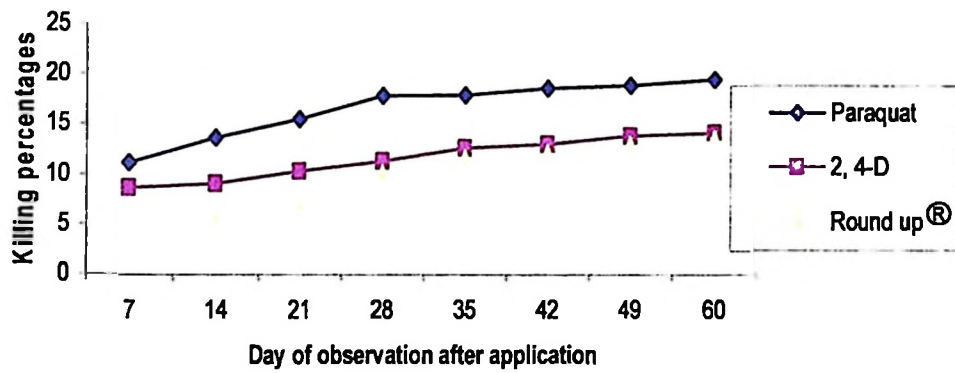
Herbicide	Killing percentages in days							
	7	14	21	28	35	42	49	60
Paraquat	20.20	21.4	22.00	22.70	23.41	24.68	25.90	26.19
2,4-D	12.33	15.78	20.18	23.60	25.61	26.80	27.22	27.57
Round up®	2.10	3.80	4.10	11.60	18.34	20.60	21.18	23.11



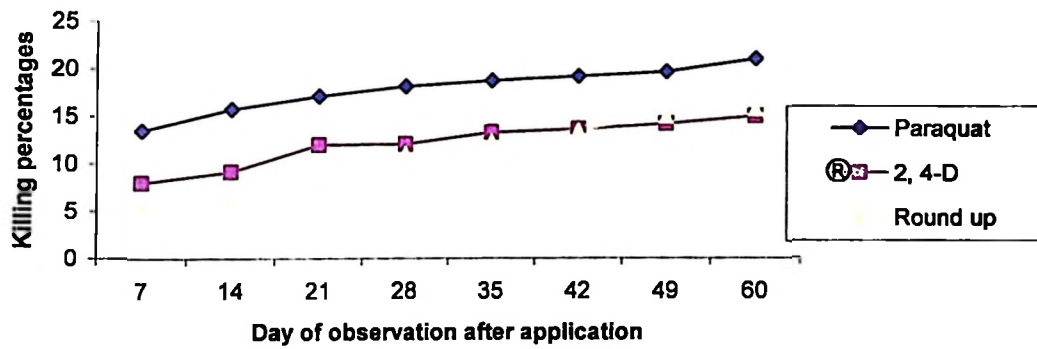
**Figure 11: Graphs of trend of leaf killing percentages at a dosage rate of 0.5 kg/ha for three tested herbicides in sixty days of observation**



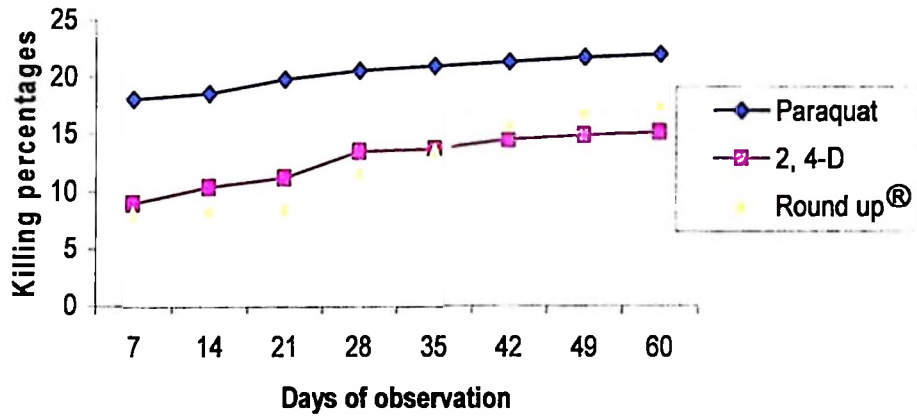
**Figure 12: Graphs of trend of leaf killing percentages at a dosage rate of 1.0 kg/ha for three tested herbicides in sixty days of observation**



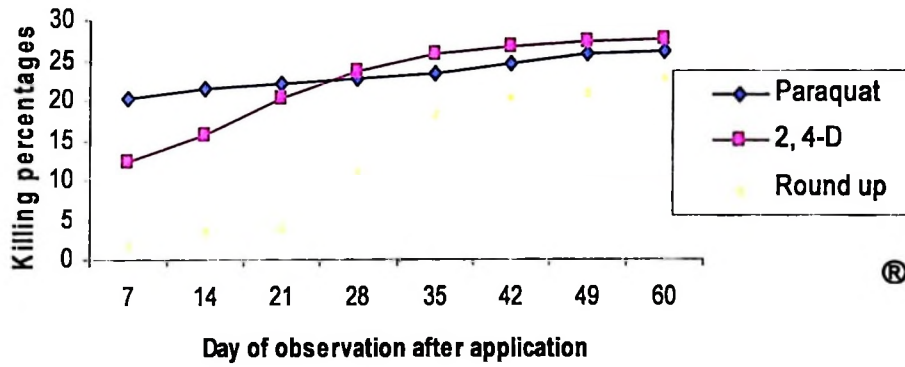
**Figure 13: Graphs of trend of leaf killing percentages at a dosage rate of 1.5 kg/ha for three tested herbicides in sixty days of observation**



**Figure 14: Graphs of trend of leaf killing percentages at a dosage rate of 5 kg/ha for three tested herbicides in sixty days of observation**



**Figure 15: Graphs of trend of leaf killing percentages at a dosage rate of 10 kg/ha for three tested herbicides in sixty days of observation**



**Figure 16: Graphs of trend of leaf killing percentages at a dosage rate of 15 kg/ha for three tested herbicides in sixty days of observation**

### 4.3 Evaluation of herbicides efficacy

MSTAT-C analysis results have shown that overall killing percentage was 16.2 % (Appendix 1). Analysis of variance (ANOVA) in Table 9 has shown herbicides, application rates and interaction of herbicide and rate were highly ( $P < 0.001$ ) significant sources of variations in percentage control of *Dichapetalum* plants.

Mean separation for herbicide treatments revealed that among the three tested herbicides, paraquat performed somewhat better than the other two. According to results; 2, 4-D was followed by Paraquat in terms of extent of control against *Dichapetalum* plants. Of the three, Round up® had the lowest killing percentage (Table 10).

Mean separation was applied to dosage rates without taking into consideration herbicide types, killing percentage was found to increase with increased dosage rates as indicated in Table 11. The highest dose of 15 kg/ha had greatest degree of control, on the other hand smallest killing per cent was recorded with 0.5 kg/ha which was smallest dose tested in field experiment.

Mean separation for dosage rates in individual herbicide was done to find out trend in killing percentages of dosage rates in each herbicide. The observations showed that in case of Round up® and paraquat killing per cents increased with an increase in doses. However, this was not the case with 2, 4-D. Means separation in 2, 4-D has revealed that killing percentages at dosage rates of 10 kg/ha and 5 kg/ha are not significantly

different. The other dosage rates which produced killing percentages not significantly different in 2,4-D are; dosage rates of 1.5 kg/ha and 5 kg/ha; dosage rates of 1 kg/ha and 1.5 kg/ha (Table 12).

Interactions of herbicide type and dosage rate played a role in killing percents; this was closely studied to find out the exact effect of each interaction and results are shown in Table 13 below. There were a total of 18 interactions; the highest killing percentage was recorded in interaction of 2,4-D at dose 15 kg/ha. The two interactions: Round up® at 0.5 kg/ha and Paraquat at the same dosage rate produced lowest killing percents in this experiment. Analysis on mean separation has also revealed that some interactions were not significantly different; interactions of 2,4-D at 1 kg/ha and Round up at 1.5 kg/ha; Round up® at 1 kg/ha and Paraquat at 1 kg/ha. Other interactions which were statistically not different are; Round up® at 5 kg/ha and 2,4-D at 10 kg/ha; 2, 4-D at 10 kg/ha and at 5 kg/ha; 2,4-D at 1.5 kg/ha and at 5 kg/ha.

The general observation for all herbicides is that the effect of interactions increased with an increase in dosage rates. The example for this is with 2,4-D; interaction of this herbicide at 0.5 kg/ha was 12.0 % while at 15 kg/ha, killing percentage was 27.6. However, there are some exceptions to this general observation.

Frequency distributions on herbicides performance are shown in Figures 17, 18, 19, 20 and 21.

#### 4.4 Rainfall trend observations

Data on rainfall trend for the period between October and February for year 1997/1998, 2005/2006 and Mean Rainfall conditions for Mkuranga District is as shown in Appendix 2. These data are represented graphically in Figure 22.

**Table 9: Analysis of Variance (ANOVA) for Herbicide treatments, Dosage rates and their Interactions on Killing Percentages.**

Source of variation	Degree of freedom (df)	Sum Squares (SS)	Mean Square (MS)	F value
Herbicide types (A)	2	122.93	61.46	235.29 <sup>xxx</sup>
Error (a)	6	1.57	0.26	
Dosage rates (B)	5	2056.84	411.37	1927.58 <sup>xxx</sup>
Interactions (AB)	10	282.38	28.24	132.32 <sup>xxx</sup>
Error (ab)	45	9.60	0.21	
Total	71	2474.09		

Note: xxx = Highly significant difference ( $p \leq 0.001$ ).

**Table 10: Mean killing percentages between herbicides**

Herbicide types	Killing percentages
2,4-D	16.30 <sup>b</sup>
Round up ®	14.55 <sup>c</sup>
Paraquat	17.75 <sup>a</sup>
Mean	16.20
CV (%)	3.15
se ±	0.10

Note: Means followed by the same letter do not differ significantly at  $P \leq 0.05$  according to Duncan's New Multiple Range Test (DNMRT).

CV = Coefficient of variation

se = Standard error

**Table 11: Mean killing percentages of different dosage rates**

Dosage rates (kg/ha)	Killing percentages
0.5	9.01 <sup>f</sup>
1.0	11.18 <sup>c</sup>
1.5	15.85 <sup>d</sup>
5	17.16 <sup>c</sup>
10	18.36 <sup>b</sup>
15	25.63 <sup>a</sup>
Mean	16.20
CV(%)	2.85
se ±	0.13

Note: Means followed by the same letter do not differ significantly at  $P \leq 0.05$  according to Duncan's New Multiple Range Test (DNMRT).

CV = Coefficient of variation

se = Standard error

**Table 12: Mean killing percentages of dosage rates in three herbicides.**

Dosage rates	Herbicides		
	2,4-D	Round up®	Paraquat
0.5	12.03 <sup>e</sup>	7.24 <sup>f</sup>	7.77 <sup>f</sup>
1.0	13.76 <sup>d</sup>	9.91 <sup>c</sup>	9.88 <sup>c</sup>
1.5	14.28 <sup>cd</sup>	13.67 <sup>d</sup>	19.62 <sup>d</sup>
5	14.91 <sup>bc</sup>	15.64 <sup>c</sup>	20.94 <sup>c</sup>
10	15.24 <sup>b</sup>	17.75 <sup>b</sup>	22.10 <sup>b</sup>
15	27.57 <sup>a</sup>	23.11 <sup>a</sup>	26.19 <sup>a</sup>
Mean =	16.29	14.55	17.75
CV(%)		2.85	
se±		0.13	

Note: Means followed by the same letter do not differ significantly at  $P \leq 0.05$  according to Duncan's New Multiple Range Test (DNMRT).

CV = Coefficient of variation

se = Standard error

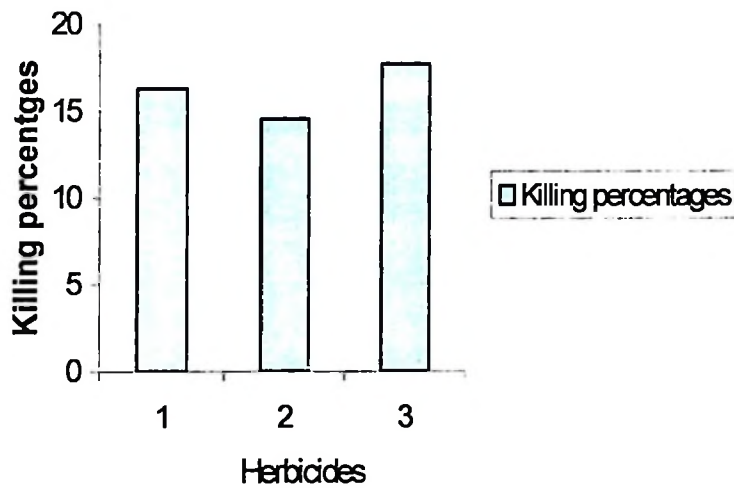
**Table 13: Mean killing percentages of interactions between herbicide type and dosage rates**

Serial no.	Treatment (dosage rates are at kg/ha)	Mean killing percentages
1	2,4-D at 0.50	12.03 <sup>l</sup>
2	2,4-D at 1.00	13.76 <sup>k</sup>
3	2,4-D at 1.50	14.28 <sup>jk</sup>
4	2,4-D at 5.00	14.91 <sup>ij</sup>
5	2,4-D at 10.00	15.24 <sup>hi</sup>
6	2,4-D at 15.00	27.57 <sup>a</sup>
7	Round up at 0.50	7.24 <sup>n</sup>
8	Round up at 1.00	9.91 <sup>m</sup>
9	Round up at 1.50	13.67 <sup>k</sup>
10	Round up at 5.00	15.64 <sup>h</sup>
11	Round up at 10.00	17.75 <sup>g</sup>
12	Round up at 15.00	23.11 <sup>c</sup>
13	Paraquat at 0.50	7.77 <sup>n</sup>
14	Paraquat at 1.00	9.88 <sup>m</sup>
15	Paraquat at 1.50	19.62 <sup>f</sup>
16	Paraquat at 5.00	20.94 <sup>e</sup>
17	Paraquat at 10.00	22.10 <sup>d</sup>
18	Paraquat at 15.00	26.19 <sup>b</sup>
Mean		16.20
CV(%)		2.85
se ±		0.23

Note: Means followed by the same letter do not differ significantly at  $P \leq 0.05$  according to Duncan's New Multiple Range Test (DNMRT).

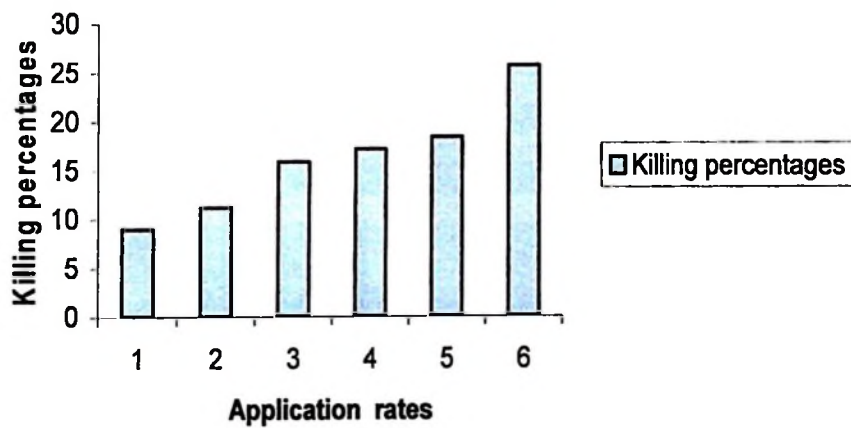
CV = Coefficient of variation

se = Standard error



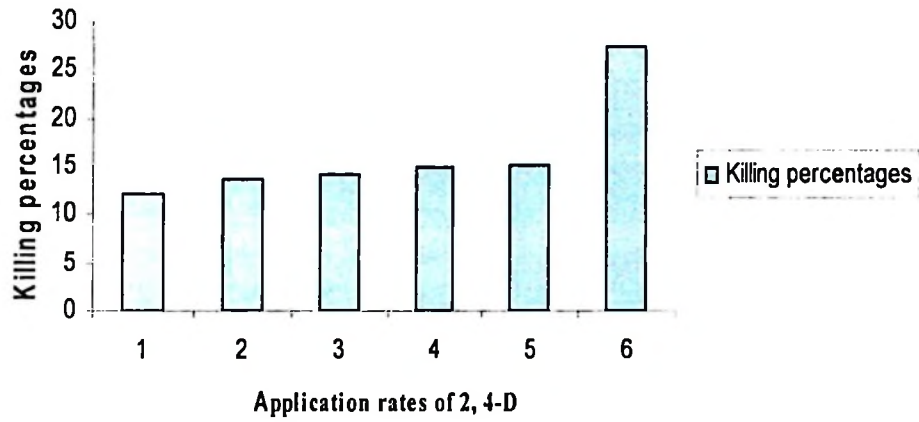
**Figure 17: Frequency distribution of killing percentages of herbicides**

1=2,4-D 2= Round up ® 3= Paraquat



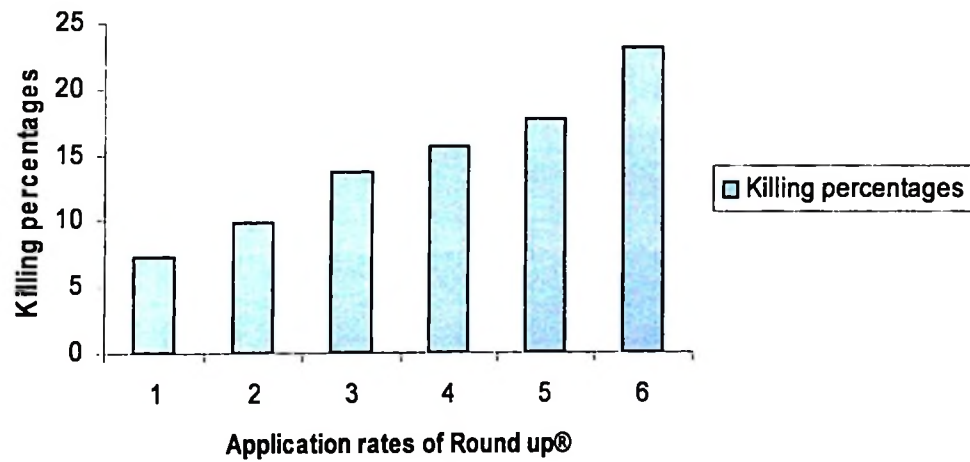
**Figure 18: Frequency distribution of killing percentages of dosage rates**

1=0.5 kg/ha 2=1.0 kg/ha 3=1.5 kg/ha 4= 5 kg/ha 5=10 kg/ha 6=15 kg/ha



**Figure 19: Frequency distribution of killing percentages of dosage rates for 2,4-D**

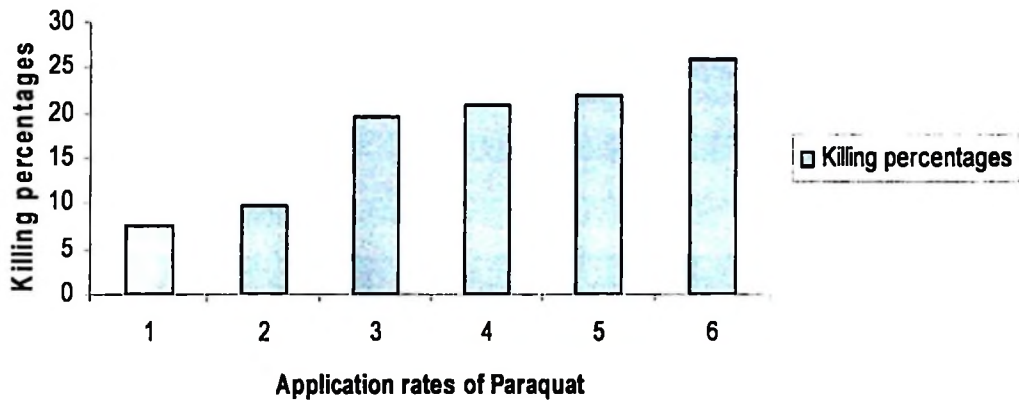
1=0.5 kg/ha 2=1.0 kg/ha 3=1.5 kg/ha 4=5 kg/ha 5=10 kg/ha 6=15 kg/ha



**Figure 20: Frequency distribution of killing percentages of dosage rates for Round up®**

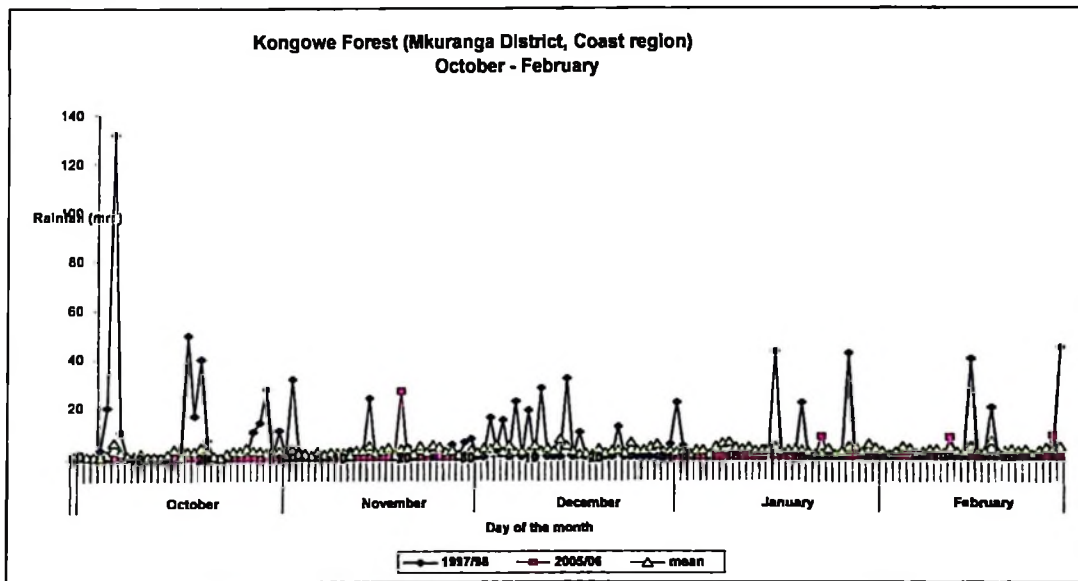
**Round up®**

1=0.5 kg/ha 2=1.0 kg/ha 3=1.5 kg/ha 4= 5 kg/ha 5=10 kg/ha 6= 15 kg/ha



**Figure 21: Frequency distribution of killing percentages of dosage rates for Paraquat**

1=0.5 kg/ha 2=1.0 kg/ha 3=1.5 kg/ha 4=5 kg/ha 5=10 kg/ha 6=15 kg/ha



**Figure 22: Graph of Trend of Rainfall between October, 2005 and February, 2006 in Mkuranga District.**

**Note:**

- 1997/98: Represent rainfall trend in years of excessive rainfall
- 2005/2006: Rainfall trend as it occurred in the present study.
- Mean: Shows mean rainfall trend.

## CHAPTER FIVE

### 5.0 DISCUSSION

#### 5.1 Discussion on plant prevalence

The present study has given valuable information on scope and size of the *Dichapetalum* infestation. The survey carried out has shown and confirmed widespread infestation of *Dichapetalum* plants in Mkuranga district, Coast Region. These findings are in agreement with report by Central Veterinary Laboratory (1970). In that annual report by Central Veterinary Laboratory it was reported that *Dichapetalum* plants are widespread in Tanzania, particularly in Coast Region. According to Breteler (1990); Meyer and Van Rooyen (1996) millions of hectares of grazing land along the coastal strip are infested by the *Dichapetalum* plants and this affects grazing of animals. The study conducted in 2001 revealed that Mkuranga and Kisarawe Districts were heavily infested with deadly plants belonging to the genus *Dichapetalum* (Ngomuo *et al.*, 2003). According to Vickery and Vickery (1973) the growth of *Dichapetalum* in Coastal strip is favoured by warm and tropical climate, temperatures averaging 26.7°C, rainfall ranging from 1016 to 1930mm, and the sandy loamy soil type. Mkuranga District has similar climatic conditions as reported by Department of Fund for International Development (2005). It is therefore, speculated that climate in this district plays a crucial role on growth of these plants.

*Dichapetalum* species identified in this study are in agreement with findings of Verdcourt and Trump (1969); Vickery and Vickery (1973); Breteler (1990); Ngomuo *et al.* (1995); Msami (1999) and Ngomuo (2001).

The plants that were found to be in close association with *Dichapetalum* plants (*Deinbollia borbonica*, *Annona senegalensis*, *Xylothea tetensis*, *Milletia* spp.) can give an indication of presence of *Dichapetalum* plants in areas they occur.

This study has also provided information on percentage cover of identified species. This measure of vegetation gives estimation on proportion of area occupied by particular species. In the present study, it has been revealed that greatest proportion of area infested in Mkuranga District is occupied by *D. stuhlmannii*. This indicates that *D. stuhlmannii* is widespread in the district. *D. stuhlmannii* occupies 10% of total area in Rufiji district, it is highly toxic to animals and has totally or partially inhibited ruminant animal production in areas with widespread distribution (Ngomuo *et al.*, 2003). The present study has shown that next in terms of percentage cover in Mkuranga district is *D. mossambicense*. The toxicity of this species is not well stated, there is conflicting information on toxicity, some reports claim it is non poisonous, while others claim the plants to be toxic to animals. Further research should be done to ascertain toxicity of this species. *D. ruhlandii* identified in survey is a highly poisonous plant. In Tanzania this species has been found in Lunguza Forest reserve in Lushoto District, Kiwanda in Tanga District, Turian and Kimboza Forest reserve in Morogoro District and Lindi (UDSM herbarium specimens, personal communication, 2006). Small proportion of *Dichapetalum* infested area in the district is occupied by *D. arenarium*. This occurs as a climbing shrub; it is poisonous to animals (Ngomuo, 2001). The quantification of problem made in this study is valuable as it has provided a clue on the extent of the problem and troublesome species present in this district. The photographs of *Dichapetalum* plants

taken during this study are instrumental in assisting all those involved in livestock activities to be familiar with these plants. According to Kidunda (1996), 2-5% of all mortalities in Rangeland in Tanzania are due to exposure to poisonous plants. The findings of the study create awareness and have positive impact to livestock keepers as reported losses due to exposure to these plants can be avoided by avoiding exposure of animals to poisonous plants. In addition, if control measures are instituted after successful identification of poisonous plants a large part of grazing land which is now restricted for grazing, can be used as the deterrent would have been removed. This will lead to increase in number of animals in the district, in particular ruminant animals. This is supported in report by Mlay (2001) who reported that a steady rise in the cattle population in Tanzania is due to presence of uninhabited savannah plains and advances in chemoprophylaxis of tsetse-borne diseases. In view of the above, successful control of *Dichapetalum* plants will definitely promote ruminant livestock production in this district as chemoprophylaxis for tsetse-borne diseases are already in place; what is now required is grazing land free of these poisonous plants and measures to avoid or at least minimize animal exposure to them. This view is in agreement with the statement on best control measures of *Dichapetalum* plants by Ngomuo *et al.* (2003) who pointed out that because *Dichapetalum* plants are difficult to eliminate, the best control measure is to prevent access of animals to infested areas. On the other hand, Minnaar (2000) stated that the long term control of poisonous plants involves eradication of them from pastures and avoiding exposure of animals to area of infestation.

## **5.2 Discussion on botanical changes of sprayed plants**

The effects of 2, 4-D and Roundup® were apparent in the second week whereas those of paraquat treated plants were observed only after two days of treatment. These findings tend to agree with what was reported in the literature. Paraquat is a foliage-applied contact which is a very fast-acting herbicide. After application, penetration through the plant leaf surface occurs almost immediately and this lead to its rapid effects (Syngenta, 2006). Roundup® and 2, 4- D are translocated herbicides; these after application are absorbed through leaf and readily translocated within plants to their sites of action. 2, 4-D tends to accumulate at the meristematic areas of both shoots and roots. This process requires a long duration of time resulting into relatively delayed effects (Sassman *et al.*, 1984). Glyphosate is taken up by plant, primarily through the foliage and is then translocated through the plant to roots and other structures. Glyphosate and sulfosate move readily in the phloem tissue with photosynthate or mobilized storage products. Its mechanism of action appears to be the inhibition of aromatic amino acid synthesis. This is a slow process and may require 10 to 14 days and thus its prominent effects are mostly observed after two weeks (Weed Science Society of America, 1983). According to Hager and Sprague (2000) application of translocated herbicides may cause leaf cupping or puckering. Explanation behind this observation is that translocated herbicides move into the apical meristem, the location of hormonal control, and disrupt the hormone balance of the plant. Following the disruption of hormonal balance, the plant exhibits some response such as leaf cupping or puckering.

Porterfield *et al.* (2001) conducted field studies in 1998 and 1999 to evaluate the response of seven cotton cultivars to CGA-362622 (common name: trifloxysulfuron, chemical name: *N*-[(4, 6-dimethoxy-2-pyrimidinyl) carbamoyl]-3-(2, 2, 2-trifluoroethoxy)-pyridin-2-sulfonamide sodium salt) applied postemergence at 7.5 and 15 g ai/ha to three- to five-leaf cotton. Cotton injury symptoms included chlorosis and minor stunting. At 3 to 4 week after treatment (WAT), injury from CGA-362622 at 7.5 and 15 g/ha was 2 to 6% and 7 to 9% by visual rating respectively.

The bending of leaves sprayed with 2, 4-D is in agreement with observation by Crafts and Robbins (1962). Newly developing leaves were mostly affected in 2, 4-D treated plants, this finding agrees with reports by Crafts and Robbins (1962); Sassman *et al* (1984); and Weed Science Society of America (1983). All the investigations stated that 2, 4-D affect primarily areas of active growth as observed in present study. Owen and Hartzler (2002) observed leaf cupping and malformation in soybean, the symptoms seemed to be caused by exposure to plant growth regulator herbicides such as 2, 4-D and dicamba.

The key observations on plants sprayed with Roundup® were slow development and stunting of leaves. This might be explained by the fact Round up® impairs protein synthesis which is an important process for plant growth (Weed Science Society of America, 1983). In general, injury symptoms of amino acids synthesis inhibitors are slow to develop (1 to 2 weeks) and include stunting or slowing of plant growth and a slow plant death (South African Sugar Association, 2004). As a group, these

compounds are more effective on grasses than broadleaf weeds but they are generally considered to be nonselective foliar herbicides with little or no soil activity. Penetration is fairly slow, so rainfall too soon after application may reduce control (Weed Science Society of America, 1983).

Necrosis dominated in leaves treated with paraquat. This is caused by action of paraquat on photosystem I resulting in production of chemically highly reactive, super oxides which attack unsaturated membrane fatty acids, rapidly opening up and disintegrating the cell membranes and tissues. As a result of these dramatic chemical changes, membranes are destroyed, and cell contents leak and mix causing further destruction which lead to gross necrosis of plants. Areas of desiccation observed in this study are also due to chloroplast membranes rupturing allowing water to escape from the plant material that leads to the rapid desiccation of the foliage (Syngenta, 2006).

### **5.3 Field efficacy evaluation of herbicides on *Dichapetalum* plants control.**

A number of studies have been conducted on efficacy of herbicides on plants and findings on this research area have been reported (Hull *et al.*, 1952; Bentley, 1967; Muzik, 1976; Keeler *et al.* 1977; Forbes *et al.* 1980; Oswald, 1980; Gerber *et al.*, 1983; Kudsk and Kristensen, 1992; and Carsky *et al.*, 1994).

In the present study, the observations from field experiments on efficacy evaluations showed that plants sprayed with Roundup® did not show very apparent response to treatment. This is in agreement with report of Weed Science Society of America

(1983), that Roundup® is more effective against grasses than broadleaf weeds. The response of plants to 2, 4-D and Paraquat was relatively noticeable. This can be explained by the fact that action of Paraquat was not very much affected by prevailed drought conditions and stage of plant growth as this is not translocated herbicide, it acts upon coming into contact with plant tissues. *Dichapetalum* plants have broad leaves. According to Loos (1975) phenoxyacetic acids such as 2, 4-D are very effective against broadleaf weeds. Therefore application of 2, 4-D gave somehow satisfactory response despite the dry conditions and advanced stage of plant growth which affected translocation of herbicide within plant.

The findings have also shown that interaction of herbicide and dosage rate has significant effect on the degree of control. Means separation has revealed that some interactions are not statistically significantly different; for example interactions of 2, 4-D at 1kg/ha and Round up at 1.5 kg/ha. It is advised to use the herbicide that produced that degree of control at lower doses. However, current market price and availability of those herbicides should be critically analyzed before making decision. Lower doses are preferred in order to minimize costs and hazardous effects to the environment.

A notable finding is with 2,4-D in which lower doses led to relatively large killing percentages. However the increase in doses did not produce expected increase in killing percentages as shown in Table 12. At a dose of 0.5 kg/ha killing percents for 2,4-D, Round up® and Paraquat were 12.0, 7.2, and 7.8 respectively. The rise in killing percent from dose 1.5 kg/h to 10 kg/ha for 2,4-D, Round up® and paraquat

was 1.0, 4.0, and 2.5 respectively. This is in agreement with the report of Meyer *et al.* (1970) who observed that translocated herbicides such as 2,4-D may be reduced in effectiveness when high rates are used because leaves may die so quickly that the toxicant is not absorbed.

A similar study in northern Cameroon showed that 2,4-D applied at a dose of 2000 g active ingredient per hectare significantly reduced the number of emerged *Striga hermonthica* (a parasitic weed in sorghum crop) plants, resulting in significant sorghum grain yield increases (Carsky *et al.*, 1994). Hull *et al.* (1952) found that the percentage of sagebrush kill varied from 58 to 97 percentage using 1 to 2 lb per acre of 2,4,5-T applied in three to five gallons of diesel oil.

A series of case studies revealed that spraying outside the recommended period and bad spraying techniques were the principal causes of poor control of *Senecio jacobaea*. Sometimes cause of variability in control of *Senecio jacobaea* lies in short-term changes taking place in the plants within a population rather than in differences between populations on different sites (Forbes *et al.*, 1980).

Studies have shown that 100% control is rarely achieved with single herbicide application and therefore sometimes there is a need for additional control strategies. The work of Oswald (1980) suggested that sequential application of herbicides is a potentially effective method of controlling *Poa trivialis* in ryegrass seed crops compared to single application.

In the present study, the herbicide effects in terms of killing percent are unsatisfactory. It is very difficult to explain the poor performance of herbicides as it might be caused by many factors (Iowa State University, 2005). However, in the present study drought conditions and advanced growth stages of treated plants are probable factors for unsatisfactory performance of the herbicides. The spraying of herbicides in the present study was done in December, 2005. Basing on Rainfall data collected by TMA, the quantity of rainfall was zero mm (nothing was recorded) for the whole month in which herbicide spraying was done (Figure 22 and Appendix 2). Even in subsequent months of study, in January and February; rainfall data collected by Kongowe Forest Weather Station showed rainfall recorded was below mean rainfall conditions. This appears to be a very irregular rainfall trend because during this period, Mkuranga District is usually under first rainy season (Department of Fund for International Development, 2005). This might be responsible for poor herbicide performance observed in the present study. According to Kudsk and Kristensen (1992) environmental conditions significantly affect the activity and effect of foliage applied herbicides. These factors principally affect herbicide penetration or translocation or both. Plants under moisture stress do not readily translocate herbicides. According to Iowa State University (2005) variation in weather conditions affected herbicide performance more than weed growth; therefore the statement that small weeds are easier to kill than large weeds may be an oversimplification. However, the limited understanding of how weeds adapt to environmental fluctuations restrict how we can use weather information to optimize herbicide application (Iowa State University, 2005). Cook (1963) reported unsatisfactory big sagebrush kills when soil moisture in the upper two feet of silty

clay loam soil was below 12 percent. Plants in water logged sites have also been found more resistant to herbicides than plants of the same species on well drained sites. Plants grown under low humidity and/or soil water deficit tend to have smaller leaves with thicker cuticles, more epicuticular wax and pubescence than unstressed plants, and this might retard interception, retention and penetration of the herbicide (Muzik, 1976; Caseley, 1989; Wanamarta and Penner, 1989). Herbicide retention, uptake and translocation are affected by climatic conditions (Gerber *et al.*, 1983).

Drought conditions which happened in the present study, impaired growth which had negative impact on performance of herbicides. The importance of weed growth for the positive herbicide effect has been stressed but not quantified by Hammerton (1967), Muzik (1976), Davies *et al.* (1983), Gerber *et al.* (1983), Legg (1983), Coupland (1986;1989) and Wills and McWhorter (1988). Herbicide effects in the field may, therefore be expected to increase with increased growth rate of the seedlings and hence, be increased by all weather and soil factors combinations that would promote growth rate. Results from studies in controlled environments have confirmed that conditions which promote growth rate, such as optimal temperature (Devine, 1989; Wanamarta and Penner,1989), high relative humidity (Nalewaja and Woznica,1985; Caseley, 1989; Kudsk *et al.*, 1990) and optimum water supply (Caseley,1989) enhance the activity of many foliage-applied herbicides. Weed plants growing vigorously will probably absorb a herbicide more readily (Legg, 1983), resulting in a rapid movement of the herbicide along with the photosynthates away from the leaf. This will maintain the gradient in herbicide concentration between the inside and the outside of the leaf, which is necessary for rapid penetration (Muzik,

1976). A high growth rate will probably also enhance the metabolic process induced by herbicides, such as phenoxy-acetic acids (Muzik, 1976) and sulfonyleureas. The former herbicide group stimulates the weed plant to abnormal growth, while the latter group retards the proteins synthesis and thereby inhibits cell elongation and cell division (Cobb, 1992).

The stage of plant growth has influence on performance of herbicides. In the present study *Dichapetalum* sprouts were sprayed at 8-week old, this might be too advanced stage for good response of herbicides, resulting in low killing percentages. Maximum rate of translocation rate is generally toward the end of rapid growth. Paulsen and Miller (1968) found that 0.90 kg of 2,4-D ae per acre killed 82 percentage of the plants when the new shoots were three to six inches long, only 65 percentage up to three inches long. Bentley (1967) found that sprout in chaparral species allowed to reach thirty six inches high become less susceptible hormone herbicides. Third-year sprouts of chaparral species are generally too old for effective kill.

On the other hand, poor performance of herbicides in this experiment might be due to *Dichapetalum* plants being naturally resistant to herbicide treatments. Resistance of weeds to chemical herbicides is a problem of rapidly growing importance in agriculture (Gorrdard *et al.*, 1995). Howat (1987) reported resistance of ryegrass (*Lolium rigidum*) to many different herbicides, including diclofop-methyl, fluazifop-methyl, chlorsulfuron, simazine and trifluralin. Owen (1998) reported an infestation of common waterhemp that has not been controlled by three applications of Roundup Ultra®. Interestingly, there are some plants that have been killed, some that are

injured and may or may not recover, and some that are seemingly not affected. The reason for the lack of control does not appear to be the result of misapplication, rate selection, or environmental conditions. *Lantana camara* was found to be resistant to triclopyr, a widely used herbicide for woody weed control (Goodall and Naude, 1998). According to Iowa State University (2005) resistance should be considered as a possible cause when other factors have been eliminated. In the present study, it is unlikely that these plants were resistant to herbicides as there was no history of herbicides applications against them in the area. The recommended management strategies for herbicide-resistant weed populations include an integrated system of crop rotation and rotation of herbicide with different modes of action (Vitolo, 1999).

Tolerance of *Dichapetalum* plants might be responsible for poor performance; it is suspected that these plants can tolerate herbicide treatment. The tolerance is probably conferred by a gene present in them. Hall *et al.* (2000) reported triple tolerance of Canola. These plants had tolerance to three herbicides, imidazolinone (Pursuit®), glyphosate (Roundup®), and glufosinate (Liberty®).

## CHAPTER SIX

### 6.0 CONCLUSION AND RECOMMENDATIONS

#### 6.1 CONCLUSION

The findings from this study showed that Mkuranga is heavily infested with *Dichapetalum* plants. *Dichapetalum stuhlmanii* which is highly toxic to animals was found to have widespread distribution. On the other hand, herbicides tested for efficacy gave unsatisfactory performance probably due to drought conditions which prevailed when the experiment was conducted and advanced growth stages of sprayed plants. Spraying herbicides in dry conditions has resulted in poor performance; farmers are therefore advised to avoid spraying in drought conditions.

Generally, it can be concluded that the findings obtained in this study will be of great use towards designing proper control measures for *Dichapetalum* plants. In addition, the findings will add to our basic knowledge and understanding of these plants.

#### 6.2 RECOMMENDATIONS

The findings on herbicide performance assessment seem to propose that a follow-up study should be done. The study should be directed especially towards clearing doubts whether drought conditions and stage of growth are reasons behind unsatisfactory performance of herbicides observed in the present study. It is proposed that the three tested herbicides should be assessed for efficacy in non-drought conditions and early growth stage of *Dichapetalum* plant sprouts so as to see disparity in herbicides performance between these two different situations. It will be

also useful to conduct studies so as to understand effects of environmental changes on response of *Dichapetalum* plants to herbicides treatment.

As application of herbicides in drought conditions gave unsatisfactory performance, farmers are advised to avoid applications at this time. Farmers are hereby counseled to consider a number of criteria before deciding on herbicide to use rather than basing only on field performance of herbicides. The other criteria to be considered include; market availability, current price and effects on environment.

As identification is one step toward proper control measures of these plants, it is recommended that special training programmes be put in place focusing mainly on identification skills so as to provide livestock keepers skills on identification of these plants. This will enable livestock keepers avoid exposure of animals to *Dichapetalum* plants. This should be followed by proper control programmes for eradication of plants from grazing areas.

**REFERENCE**

- Adauidi, A. O. (1975). Poisoning in goats caused by *Dichapetalum barteri*. *Tropical Animal Health Production* 7: 56-57.
- Ashby, M. (1961). *Introduction to Plant Ecology*. 2<sup>nd</sup> edition. Macmillan and Co. Ltd. London. pp. 205-221.
- Babbie, E. R. (1973). *Survey Research Methods*. Wadsworth Publishing Company Inc. Belmont, California. 384pp.
- Beck, K.G. (2004). Range and Pasture Weed Management. Colorado State University [<http://www.ext.colostate.edu/Pubs/natres/03105.html>] site visited on 25/04/2006.
- Bentley, J. R. (1967). *Conversion of chaparral areas to grassland*. USDA Agriculture Handbook. 328pp.
- Bioenergy (2005). Average Plant Biomass. [<http://www.eere.energy.gov/RE/bioenergy.html>] site visited on 27/06/2005.
- Biotechnologyonline (2006). A biotechnology solution to weeds. [<http://www.biotechnologyonline.gov.au/foodag/solution.cfm>] site visited on 21/04/2006.

Bosakowski, T. and Levin, A. A. (1986). Serum Citrate As A Peripheral Indicator Of Fluoroacetate And Fluorocitrate Toxicity In Rats And Dogs. *Toxicology And Applied Pharmacology* 85 : 428-436.

Breteler, F. J. (1990). A new species and a new record of *Dichapetalum* from Tanzania. *Kew Bulletin* 45: 721-724.

Brook, R. M. (1989). Review of literature on *Imperata cylindrical* (L.) Raeuschel with particular reference to South East Asia. *Tropical Pest Management* 35: 12-25.

Buffa, P. and Peters, R. A. (1950). The *in vivo* formation of citrate induced by fluoroacetate and its significance. *Journal of Physiology* 110: 488-500.

Canadian Food Inspection Agency (2006). Biotechnology Products: Plants that Tolerate Herbicides.

[<http://www.inspection.gc.ca/english/sci/biotech/enviro/herbice.shtml>] site visited on 23/04/2006.

Caseley, J. C. (1989). Variations in Foliar Pesticide Performance Attributable to Humidity, Dew and Rain effects. *Aspects of Applied Biology* 21: 215-225.

- Carsky, R. J.; Singh, L. and Ndikawa, R. (1994). Effect of herbicide and handweeding on current and subsequent season *Striga hermonthica* density on sorghum. *International Journal of Pest Management* 40: 111-116.
- Central Veterinary Laboratory (1970). *Annual Report*. Ministry of Agriculture and Cooperatives, United Republic of Tanzania, Dar es Salaam. pp. 19-21.
- Cheeke, P. R. (1998): *Natural Toxicants in Feeds, Forages, and Poisonous Plants*. Interstate Publishers, Danville, IL. 528pp.
- Chenoweth, M. B. (1949). Monofluoroacetic acid and related compounds. *Journal of Pharmacology and Experimental Therapeutics* 97(2): 383-423.
- Chenoweth, M. B.; Kandel, A.; Johnson, L. B. and Bennett, D. R. (1951). Factors Influencing Fluoroacetate Poisoning: Practical treatment with glycerol monoacetate. *Journal of Pharmacology and Experimental Therapeutics* 102 : 21-49.
- Clarke, D. D. (1991). Fluoroacetate and Fluorocitrate: Mechanism of Action. *Neurochemistry Research* 16(9): 1055-1058.
- Cobb, A. (1992). *Herbicides and Plant physiology*. Chapman and Hall, London. 532pp.

Cooperative Research Centre (2006). Herbicide Tolerant (HT) Crop. Cooperative Research Centre for Australian Weed Management.

[[http://www.weeds.crc.org.au/documents/fs29\\_faht\\_crops.pdf](http://www.weeds.crc.org.au/documents/fs29_faht_crops.pdf)] site visited on 23/05/2006.

Cook, C. W. (1963). Herbicide Control of Sagebrush on Seeded Foothill Ranges in Utah. *Journal of Range Management* 16(4):190-195.

Cornell University (2006). Taxonomy. [[www.vet.cornell.edu/consultant/consult.asp](http://www.vet.cornell.edu/consultant/consult.asp)] site visited on 14/04/2006.

Coupland, D. (1986). The effects of Environmental Factors on the Performance of Fluazifop-butyl against *Elymus repens*. *Annal Applied Biology* 108: 353-363.

Coupland, D. (1989). Pre-treatment Environmental Factors Effects on the Uptake, Translocation, Metabolism and Performance of fluazifop-butyl in *Elymus repens*. *Weed Research* 29: 289-297.

Crafts, A. S. and Robbins, W. W. (1962). *Weed control*. Third edition. McGraw-Hill Book Company. 514pp.

Crawley, M. J.; Hails, R.S.; Rees, M.; Kohn, D. and Buxton, J. (1993). Ecology of Transgenic oilseed rape in natural habitats. *Nature* 363: 620-623.

- Crowder, L. V. and Chheda, H. R. (1982). *Tropical Grassland Husbandry*. Longman. London and New York. 562pp.
- Currier, H. B. and Dybing, C. D. (1959). Foliar Penetration of Herbicides—review and Present Status. *Weeds* 7(2):195-213.
- Davies, W. J.; Blackman, P. G. and Mansfield, T. A. (1983). Manipulation of Stomatal Behaviour and Plant Water Status to increase Herbicide Effect. *Aspects of Applied Biology* 4: 197-205.
- Devine, M. D. (1989). Phloem Translocation of Herbicides. *Review of Weed Science* 4: 191-213.
- Dickens, R. and Turner, D. L. (1984). Postemergence Herbicide Tolerance Among Warm Season Turfgrasses. *Weed Science Society* 37:20-26.
- Department of Fund for International Development (2005). Child labour and its impact on children's access to and participation in primary education—a case study of Tanzania. [<http://www.dfid.gov.uk>] site visited on 20/05/2005.
- Doll, J. (2006). Poisonous Weeds of Pastures and Forages. University of Wisconsin. [[http://ipcm.wisc.edu/uw\\_weeds/extension/articles/poisonpasture.htm](http://ipcm.wisc.edu/uw_weeds/extension/articles/poisonpasture.htm)] site visited on 21/04/2006.

Dow AgroSciences (2006). Field Performance Assessment - Eastern Canada  
[<http://www.dowagro.com/>] site visited on 25/04/2006.

Downey, R. K. (1999). Risk Assessment of Outcrossing of Transgenic Brassica, with focus on *B. rapa* and *B. napus*. *Proceedings of the 10th International Rapeseed Congress* Canberra, Australia, 10 November, 1998. 112pp.

Eason, C. T.; Gooneratne, R.; Fitzgerald, H.; Wright, G. and Frampton, C. (1993). Persistence of sodium monofluoroacetate in livestock animals and risk to humans. *Human Experimental Toxicology* 13(2) : 119-122.

Egekeze, J. O. and Oehme, W. (1979). Sodium Monofluoroacetate (SMFA, compound 1080): A literature review. *Veterinary and Human Toxicology* 21(6): 411-415

Egyed, M. N. and Schultz, R. A. (1986). The Efficacy of Acetamide for the Treatment of Experimental *Dichapetalum cymosum* (gifblaar) poisoning in sheep. *Onderstepoort Journal of Veterinary Research* 53 (4):231-234.

Erasmus, D. J.; Maggs, K. A. R.; Biggs, H. C.; Zeller, D. A. and Bell, R. S. (1993). Control of *Lantana camara* in the Kruger National Park, South Africa, and subsequent vegetation dynamics. *Proceedings of an International conference*, Brighton, United Kingdom, 22-25 November 1993. Volume. 1:399-404.

Forbes, J. C.; Kilgour, D. W. and Carnegie, H. M. (1980). Some Causes of Poor Control of *Senecio jacobaea* L. by Herbicides. *Proceedings of British Crop Protection Conference-Weeds*, London, United Kingdom, 12 March, 1980. 113pp.

Fowler, F. J. (1993). *Survey Research Method*, Second Edition, SAGE Publication London. 156pp.

Gerber, H. R.; Nyffeler, A. and Green, D. H. (1983). The Influence of Rainfall, Temperature, Humidity and Light on Soil- and Foliage-applied Herbicides. *Aspects of Applied Biology* 4: 1-14.

Ghassemi, M.; Fargo, L.; Painter, P.; Painter, P.; Quinlivan, S.; Scofield, R. and Takata, A. (1981). *Environmental Fates and Impacts of Major Forest Use Pesticides*. Redondo Press, California. 612pp.

Gorddard, R. J.; Pannell, D. J. and Hertzler, G. L. (1995). An Optimal Control Model of Integrated Weed Management Under Herbicide Resistance. *Australian Journal of Agricultural Economics* 39: 71-87.

- Godoy, H. M. and del Carmen Villarruel, M. (1974). Myocardial adenine nucleotides, Hexose Phosphates and Inorganic Phosphate, and the regulation of Phosphofructokinase Activity During Fluoroacetate Poisoning in the Rat. *Biochemistry and Pharmacology* 23 : 3179-3189.
- Goodall, J. M.; and Naudé, D.C.(1998). An Ecosystem Approach for Planning Sustainable Management of Environmental Weeds in South Africa. *Agriculture, Ecosystems and Environment* 68(1/2): 109-123.
- Gooneratne, S. R.; Eason, C. T.; Dickson, C. J.; Fitzgerald, H. and Wright, G. (1995). Persistence of Sodium Monofluoroacetate in Rabbits and Risk to non-target species. *Human Experimental Toxicology* 14(2): 212-216.
- Gottrup, O. O; Sullivan, P. A.; Schraa, R. J.; and Vanden Born, W. H. (1976). Uptake, Translocation, Metabolism and Selectivity of Glyphosate. *Weed Research* 16: 197-201.
- Greig-Smith, P. (1983). *Quantitative Plant Ecology*. 3<sup>rd</sup> edition. Blackwell Scientific Publications, Oxford. 359pp.
- Griffin, K. A.; Dickens, R. and West, M. S. (1994). Imazapyr for Common Bermudagrass Control in Sod Fields. Auburn University. *Crop Science* 34:202-207.

- Gunsolus, J. L. (1999). Herbicide Resistant Weeds. North Central Regional Extension Publication 468. University of Minnesota.  
[<http://www.extension.umn.edu/distribution/cropsystems/DC6077.html>] site visited on 21/04/2006.
- Hager, A. and Sprague, C. (2000). Soybean Leaf Cupping/Puckering. University of Illinois. [<http://www.ag.uiuc.edu/cespubs/pest/articles/200013f.html>] site visited on 21/04/2006.
- Hall, L. M.; Huffman, J. and Topinka, K. (2000). Pollen Flow Between Herbicide Tolerant canola (*Brassica napus*). *Weed Science Society of America* 40: 48.
- Hammerton, J. L. (1967). Environmental Factors and Susceptibility to Herbicides. *Weeds* 15: 330-336.
- Hannan-Jones, M. A. (1998). The Seasonal Response of *Lantana camara* to Selected Herbicides. *Weed Research* 38(6): 413-423.
- Heap, I. M. (1997). The Occurrence of Herbicide-Resistant Weeds Worldwide. *Pesticide Science* 51: 235-243.

Hornfeldt, C. S. and Larson, A. A. (1990). Seizures Induced by Fluoroacetic acid and Fluorocitric. *European Journal of Pharmacology* 179: 307-313

Howat, P. D. (1987). Weeds Resistant to Herbicides in Australia and Contributing Factors leading to their appearance. *Plant Protection Quarterly* 2: 82-85.

Hull, A.C.; Kissinger, N. A. and Vaughn, W. T. (1952). Chemical Control of Big Sagebrush in Wyoming. *Journal of Range Management* 5: 398-402.

Hutchinson, J. (1964). *The Genera of Flowering Plants (Angiospermae). Dicotyledones, Volume 1.* Clarendon Press, Oxford. 516pp.

Iowa State University (2005). Sublethal herbicide effect on weeds [<http://www.weeds.iastate.edu>] site visited on 23/02/2006.

Johnson, S. R. (2005). Small sprayer calibration. Iowa State University of Science and Technology. [<http://www.extension.iastate.edu/Publications/PM1271.pdf>.] site visited on 05/07/2005.

- Keeler, R. F (1977). *Effects of Poisonous plants on livestock*. Academic Press, Inc. New York. In: Proceedings of a joint United States-Australian symposium on Poisonous plants( Edited by Van Kampen, K. R., and James, L. F.), 19-24 June, 1977. Utah State University (US). 600pp.
- Kidunda, R.; Lwoga, A. B. and Mtengeti, E. J. (1990). Utilization of Pasture Research Results in Tanzania. In: *Proceedings of the First Joint Workshop*, 5-9 December 1988, Lilongwe, Malawi, pp.36-56.
- Kidunda, R. S. (1996). Range and Pasture Management: A compendium. Sokoine University of Agriculture, Animal Science and Production Department, Morogoro, Tanzania. 126pp.
- Kudsk, P.; Olesen, T. and Thonke, K. E. (1990). The Influence of Temperature, Humidity, and Simulated Rain on the Performance of Thiameturon-methyl. *Weed Research* 30: 261-269.
- Kudsk, P. and Kristensen, J. L. (1992). Effect of Environmental Factors on Herbicide Performance. In: *Proceedings of the First International Weed Control Congress*, 22 March 1992, Melbourne, Australia, pp.173-186.
- Legg, B. J. (1983). Micrometeorology and the Influence of Local Variations of Environment on Plant Growth and Herbicide Performance. *Aspect of Applied Biology* 4: 15-31.

Loos, M. A. (1975). Phenoxyalkanoic Acids. In: *Herbicides: Chemistry, Degradation, and Mode of Action*. 2<sup>nd</sup> edn. ( Edited by Kearney, P. C. and Kaufman, D. D.) Marcel Dekker, Inc., New York. pp. 70-76.

Lien, B. C; Walker, J. R. L.; Cole, A. L. J. and Peters, J. A. (1978). Effect of Sodium Fluoroacetate ("Compound 1080") on the soil microflora. *Soil Biology and Biochemistry* 11: 13-18.

List, D. (2005). Sampling for Surveys.

[<http://www.sysurvey.com/tips/whitepapers.asp>] site visited on 20/06/2005.

Luckwill, L.C. and Lloyd-Jones, J. (1960). Metabolism of Plant Growth Regulators. I. 2,4-Dichlorophenoxyacetic Acid in Leaves of Red and Black Currant. *Annal Applied Biology* 48: 613-625.

Mabberley, D. J. (1987). *The Plant-Book: A portable Dictionary of the Higher Plants*. Cambridge University Press, Cambridge. 707pp.

Mac Dougall, M. E. (1996). Indiana Plants Poisonous to Livestock and Pets. Purdue University [<http://vet.purdue.edu/depts/addl/toxic/cover1.htm>] site visited on 20/05/2005.

Mallory-Smith, C; Hyslop, G. R., Thill, D. and Morishita, D. (2006). Herbicide-Resistant Weeds and Their Management. University of Idaho. [<http://info.ag.uidaho.edu/resources/PDFs/PNW0437.pdf>.] site visited on 21/04/2006.

McKenzie, R. (2006). Australian Native Poisonous Plants.

[<http://farrer.csu.edu.au/ASGAP/APOL7/sep97-4.html>] site visited on 21/04/2006.

Mead R. J; Moulden, D. L . and Twigg, L. E. (1985). Significance of Sulphydryl Compounds in the Manifestation of Fluoroacetate Toxicity to the Rat, Brush-tailed possum, Woylie and Western grey kangaroo. *Australian Journal of Biological Science* 38: 139- 149

Mehlman, M. A. (1967) Inhibition of Pyruvate Carboxylation by Fluorocitrate in Rat Kidney Mitochondria. *Journal of Biology and Chemistry* 243: 1919-1925

Meister Publishing Company (1981). *Farm Chemicals Handbook*. Meister Publishing Company, Willoughby, Ohio. 612pp.

- Menn, W. G., Hall, M. H., Hale, T. C. and Gaudreau, J. E. (1999). Evaluation of Several Herbicides for Efficacy in Post-emergent Control of Cool- Season Annual Weeds in Dormant Bermudagrass Turf. [<http://aggie-turf.tamu.edu/aggieturf2/publications/progressreports/8postcontrolcoolannu als.pdf>] site visited on 20/06/2005.
- Meyer, R.E.; Morton, H.L.; and Merkle, M. G. (1970). Brush Control on Forest Rangelands in East Texas. *Journal of Range Management* 23(2): 129-132.
- Meyer, J. J. M.; Brobbelaar, N.; Vleggaar, R. and Louw, A. I. (1992). Fluoroacetyl-coenzyme A Hydrolase-like Activity in *Dichapetalum cymosum*. *Journal of Plant Physiology* 139: 369-372.
- Meyer, J. J. M. and van Rooyen, S. W. (1996). Genetically transformed *Bacillus subtilis* with defluorinating ability. *South African Journal of Botany* 62: 65-66.
- Minnaar, P. P. (2000). Investigation of Biological samples for monofluoroacetate and *Dichapetalum cymosum* poisoning in Southern Africa. *Onderstepoort Journal Of Veterinary Research* 67: 27-30.

Michigan State University (1993). *MSTAT-C: A microcomputer for Design, Management and Analysis of Agronomic Research Experiments*. Michigan State University, East Lansing MI

Mlay, Paul N. S. (2001). Enhancement of Smallholder Dairy production under Tropical Conditions Through Supplementation to Optimize Roughage Intake, Digestibility and Microbial Protein Synthesis. PhD Thesis, Royal Veterinary and Agricultural University (RVAU), Copenhagen, Denmark. pp.12.

Moss, S. R. and Cussans, G. W. (1991). The development of Herbicide-resistant Populations of *Alopecurus myosuroides* (Black-grass) in England. In: *Herbicide resistance in weeds and crops* (Edited by Caseley, J.C.; Cussands, G.W. and Atkin, R. K.) Butterworth-Heinemann, Oxford. pp. 45-55.

Msami, H. M. (1999). An Outbreak of Suspected Poisoning of cattle by *Dichapetalum sp.* in Tanzania. *Tropical Animal Health and Production* 31(1): 1-7.

Muzik, T. J. (1976). Influence of Environmental Factors on Toxicity to Plants. In *Herbicides, Physiology, Biochemistry, Ecology* (Edited by Audus, L. J.) Academic Press, London. pp.203-247.

- Nalewaja, J. D. and Woznica, Z. (1985). Environment and Chlorsulfuron Phytotoxicity. *Weed Science* 33: 395-399.
- Newhouse, K. E.; Smith, W. A.; Starrett, M. A.; Schaefer, T. J. and Singh, B. K. (1992). Tolerance to Imidazolinone Herbicides in Wheat. *Plant Physiology* 100: 882-886.
- Ngomuo, A. J.; Kambarage, D. M.; Mwamengele, G. L. M. and Matovelo, J. A. (1995). *Dichapetalum species* Toxicity in Cattle at a Farm in Tanzania. *Veterinary and Human Toxicology* 37(2): 143-144.
- Ngomuo, A. J. (2001). Common Poisonous Plants of Tanzania Compendium. Sokoine University of Agriculture, Morogoro, Tanzania. pp. 65-78.
- Ngomuo, A. J.; Mosha, R. D.; Temu, R. P. C.; Mahunnah, R. L. A.; Mtengeti, E. J.; Minja, M. M. J. and Otari, M. M. (2003). Common Poisonous Plants of Coast Region: A Field Manual. Sokoine University of Agriculture, Morogoro Tanzania. 117pp.
- Norland, E. and Heiligmann, R. (2005). Calibration of Hand Sprayers For Herbicide Application. Ohio State University [<http://ohioline.osu.edu/for-act/0020.html>] site visited on 04/07/2005.

Norris, W. R. (2001). Sodium fluoroacetate. New Zealand National Poisons Centre. [www.intox.org/databank/documents/chemical/sodfluor/pim494.htm] site visited on 20/04/2006.

Norris, L. A. (1981). The Movement, Persistence and Fate of the Phenoxy herbicides in the Forest. *Research Review* 80: 65-135.

Nwude, N. and Parsons, L. E. (1977). Nigerian Plants That May Cause Poisoning in Livestock. *Veterinary Bulletin* 47: 811-817.

Oswald, A.K. (1980). *The Use of Sequential Herbicide Treatments to Control Poa trivialis in two perennial ryegrass crops grown for seed*. Proceedings of British Crop Protection Conference-Weeds, London, United Kingdom, 12 May, 1980. 134pp.

Owen, M. (1998). Weeds not controlled with Roundup Ultra® Department of Agronomy. Iowa State University. [http://www.agron.iastate.edu/] site visited on 21/04/2006.

Owen, M.; and Hartzler, B. (2002). Plant Growth Regulator Herbicide Symptoms in Soybean. Department of Agronomy, Iowa state University. [http://www.ent.iastate.edu/Ipm/Icm/2002/7-22-2002/pgr.html] site visited on 20/04/2006.

- Paulsen, H.A. and Miller, J. C. (1968). Control of Parry rabbitbrush on mountain grasslands of western Colorado. *Journal of Range Management* 21(3): 175-177.
- Pfister, J. A. (1988): Nitrate intoxication of ruminant livestock. In: *The Ecology and Economic Impact of Poisonous Plants on Livestock Production (Edited by James, L. F.; Ralphs, M. H. and Nielson, D. B.)* Westview Press, Boulder, CO. pp. 233-260.
- Polhill, R.M. (1988). *Flora of Tropical East Africa*. Agricultural University, Wageningen. Rotterdam. 19pp.
- Porterfield, D.; Wilcut, J. W.; Clewis, S. B. and Edmisten, K. L. (2001). Weed-Free Yield Response of Seven Cotton (*Gossypium hirsutum*) Cultivars to CGA-362622 Postemergence. [<http://www.bioone.org/perlserv/>] site visited on 21/04/2006.
- Rana, R. S and Singh, L. N. (1999). Eradication of *Lantana camara* and Wasteland utilization in Kandi region of Himachal Pradesh. *Indian Journal of Soil Conservation* 27(2): 137-140.
- Raunkiaer, C. (1934). *The life Forms of Plants and Statistical Plant Geography*. Oxford University Press, Oxford. 632pp.

Rokita, S. E ; and Walsh, C. (1983) Turnover and Inactivation of Bacterial Citrate Lyase with 2-Fluorocitrate and 2-Hydroxycitrate Stereoisomers.

*Biochemistry* 22: 2821-2828

Rueppel, M. L.; Brightwell, B. B.; Schaefer, J. and Marvel, J. T. (1977). Metabolism and Degradation of Glyphosate (Herbicide) in soil and water. *Journal of Agriculture and Food Chemistry* 25(3): 517-528.

Rutgers (2000). Weed Control. The State University of New Jersey.

[[http://www.rcrc.rutgers.edu/horsepastures/weed\\_control.htm](http://www.rcrc.rutgers.edu/horsepastures/weed_control.htm)] site visited on 25/04/2006.

Ryan, G..F. (1970). Resistance of common groundsel to simazine and atrazine. *Weed Science* 18: 614-616.

Salzer, T. and Holen, C. (2006). Weed Control in Pastures. University of Minnesota

[<http://www.extension.umn.edu/Beef/components/homestudy/plesson4.PDF>]. site visited on 25/04/2006.

Sassman, J.; Pienta, R.; Jacobs, M. and Cioffi, J. (1984). Pesticide Backgrounds Statements. Volume 1. Herbicides. Mitre Corporation. Forest Service, U.S. Department of Agriculture hand book No. 633.

Satchell (1983). *Earthworm Ecology*. Institute of Terrestrial Ecology, UK. 567pp.

Scanlan, J. C. (1984). Herbicidal Control of Woody Weeds in Central Queensland. *Tropical Grasslands* 18: 26-32.

Scanlan, J. C. and Fossett, G. W. (1984). Herbicidal Control of Woody Weeds in Central Queensland. *Tropical grasslands* 18: 33-38.

Smith, A. D. (1944). A study of reliability of range vegetation estimates. *Ecology* 25: 441-448.

Snedecor, G.W. (1946). *Statistical Methods*. State College Press, Ames, Iowa. 621pp.

South African Sugar Association (2004). WEEDS: Biology and Control. Unpublished Teaching Manual. 30pp.

Swarbrick, J. T.; and Kent, J. H. (1982). The Status of Weed Control in Tropical Pastures. In: *FAO paper no.44, Improving weed management*, FAO, Rome, Italy. pp. 126-134.

Swarbrick, J. T.; Wison, B. W.; and Hannan-Jones, M. A. (1995). The biology of Australian weeds. *Plant Protection Quarterly* 10: 82-95.

Syngenta (2006). Paraquat. [<http://www.Syngenta.com>] site visited on 15/04/2006.

- Taylor, W. M.; D'Costa, M.; Angel, A. and Halperin, M. L. (1977). Insulin-like Effects of Fluoroacetate on Lipolysis and Lipogenesis in Adipose tissue. *Canadian Journal of Biochemistry* 55: 982-987.
- Tecle, B. and Casida, J. E. (1989). Enzymatic Defluorination and Metabolism of Fluoroacetate, Fluoroacetamide, Fluoroethanol, and (-)-erythro-fluorocitrate in Rats and Mice. *Chemistry Research and Toxicology* 2: 429-435
- Temple, W. A. and Edwards, I. R. (1984). Toxic ducks - 1080 residues in game birds. *Veterinary and Human Toxicology* 27(1): 20-21.
- Terry, P. J. (1994). *Imperata cylindrical* (L.) Raeuschel. In: *Weed Management for developing countries* (Edited by Labrada, R.; Caseley, J. C. and Parker, C.). FAO Plant Production and Protection Paper, no.120. pp. 63-69.
- Tisserand, R. and Balacs, T. (1995): *Essential Oil Safety: A Guide for Health Care Professionals*, Churchill and Livingstone, Edinburgh, United Kingdom. 361pp.
- Undersander, D. J. and Pinkerton, B. W. (1988). Weed control in Bermuda grass. [<http://www.clemson.edu/psapublishing/PAGES/AGRO/FORAGE6.PDF>.] site visited on 25/04/2006.

- University of Minnesota (2002). Herbicide Mode of Action and Injury Symptoms. [<http://www.extension.umn.edu/distribution/cropsystems/DC3832.html>] site visited on 20/06/2005.
- Van Rijn, P. J. (2000). *Weed Management in the humid and sub-humid tropics*. Royal Tropical Institute, Netherlands. 678 pp.
- Vallentine, J. F. (1971). *Range development and Improvements*. 1<sup>st</sup> edition. Brigham Young University Press, USA. 544 pp.
- Verdcourt, B. and Trump, E. C. (1969). *Common Poisonous Plants of East Africa*, Collins, London. 254pp.
- Vickery, B. and Vickery, M. L. (1973). Toxicity for Livestock of Organofluorine compounds present in *Dichapetalum* plant species. *Veterinary Bulletin* 43: 537-542.
- Vickery, B.; and Vickery M. L. (1975). The Synthesis and Defluorination of Monofluoroacetate in some *Dichapetalum* species. *Phytochemistry* 14(2): 423- 427
- Vitolo, D. (1999). Detecting Herbicide Resistance: Guidelines for conducting diagnostic tests and interpreting results. Syngenta Crop Protection. [<http://www.plantprotection.org/HRAC/>] site visited on 25/04/2006.

- Wanamarta, G.; and Penner, D. (1989). Foliar Absorption of Herbicides. *Review of Weed Science* 4: 215-231.
- Walker, J. R. L. and Lien, B. C. (1981). Metabolism of Fluoroacetate by a Soil *Pseudomonas sp.* and *Fusarium solani*. *Soil Biology and Biochemistry* 13: 231-235.
- Ware, G. W. (2000). *The Pesticide Book*. 5th Ed. Thomson Publications, Fresno, California. 415 pp.
- Ware, G. W. (2006). An Introduction to Herbicides. University of Arizona.  
[<http://ipmworld.umn.edu/chapters/wareherb.htm>] site visited on 14/04/2006.
- Watt, J. M. and Breyer-Brandwijk, M. G. (1962). *The Medicinal and Poisonous Plants of Southern and Eastern Africa*. 2nd edition. E and S Livingstone, Edinburgh. 542 pp.
- Weed Science Society of America (1983). *Herbicide Handbook*, Fifth Edition. Champaign, Illinois. 352 pp.
- Weed Science (2006). Herbicide Resistance. [<http://WeedScience.com>] site visited on 14/04/2006.

Wills, G. D. and McWhorter, C. G. (1988). Absorption and Translocation of Herbicides. In: *Pesticide Formulations, Innovations and Developments* (Edited by Cross, B. and Scher, H. B.) American Chemical Society, Washington DC. pp.90-101.

## APPENDICES

## Appendix 1: Data on Rainfall Trend in Mkuranga District

Month	Day of month	1997/98	2005/06	mean
October	1	0	0	0.7
		0	0	0.8
		0	0	0.4
		0	0	0.3
		3	0	0.3
		20.5	0	1.3
		132	0	6.2
		10.5	0	2.3
		0	0	0.8
	10	0	0	0.8
		0	0	1.7
		0	0	0.3
		0	0	0.3
		0	0	0.0
		0	0	1.3
		0	0	3.4
		0	0	0.3
		50	0	2.5
		17	0	2.5
	20	40.3	0	4.1
		7.1	0	2.7
		0	0	0.5
		0	0	0.0
		0	0	1.7
		0	0	2.6
		0	0	2.5
		0	0	3.8
		10.5	0	2.8
		14.2	0	2.7
		28	0	1.9
	31	0	0	3.3
November		11	0	2.7
		0	0	0.9
		32	0	3.4
		2.5	0	1.7
		0	0	1.6
		0	0	0.9
		0	0	2.5
		0	0	1.3
		0	0	2.1
	10	0	0	1.9

		0	0	1.8
		2.8	0	0.8
		0	0	2.8
		0	0	2.8
		24.2	0	4.7
		0	0	2.1
		0	0	2.9
		0	0	3.9
		0	0	0.7
	20	0	27	3.4
		4	0	3.5
		0	0	1.3
		0	0	4.6
		0	0	2.2
		0	3.5	4.8
		3.7	0	4.1
		0	0	2.2
		5	0	2.7
		0	0	0.9
	30	6	0	0.5
December		7.4	0	3.0
		0	0	1.9
		0	0	3.7
		16	0	3.4
		2.6	0	5.1
		15	0	2.1
		0	0	4.8
		22.7	0	2.0
		0	0	2.4
	10	19	0	2.0
		0	0	4.8
		28.1	0	2.5
		0	0	3.7
		0	0	2.6
		0	0	7.2
		32	0	4.6
		0	0	2.3
		10	0	1.1
		0	0	1.6
	20	0	0	1.1
		1.9	0	3.2
		0	0	1.7
		0	0	2.6
		12.2	0	2.2
		0	0	1.0
		0	0	5.1
		0	0	2.0
		0	0	2.2
		0	0	3.4

		0	0	4.4
	31	0	0	1.5
January		5.1	0	1.8
		22.2	0	1.2
		4.3	0	1.8
		0	0	0.6
		0	0	2.0
		0	0	2.2
		0	0	0.2
		0	0	3.5
		0	0	4.9
	10	0	0	5.2
		0	0	3.1
		0	0	2.5
		0	0	3.1
		0	0	1.8
		0	0	2.1
		2.4	3.2	2.7
		43	0	3.3
		0	0	1.6
		0	0	2.1
	20	0	0	2.3
		22	0	1.9
		0	0	1.2
		0	0	0.2
		0	8.1	0.4
		0	0	2.5
		0	0	0.2
		0	0	1.5
		42.1	0	3.2
		0	0	3.4
		0	0	1.0
	31	0	0	4.3
February		0	0	2.7
		0	0	1.5
		0	0	0.1
		0	0	1.3
		0	0	3.2
		0	0	2.5
		0	0	0.5
		0	0	1.0
		0	0	1.9
	10	0	0	2.1
		0	0	0.7
		0	7.8	3.0
		0	0	1.2
		0	0	1.1
		40	0	3.4
		0	0	1.4

		0	0	1.1
		20	0	5.7
		0	0	0.7
	20	0	0	1.4
		0	0	2.0
		0	0	1.5
		0	0	1.9
		0	0	0.1
		0	0	1.6
		0	0	2.7
		0	8.5	2.3
	28	44.6	0	3.3

Source: TMA, weather records (2006).

**Appendix 2: Data analysis results from MSTAT-C****Data file:** NASSA**Title:** EFFECTS OF HERBICIDES, DOSAGES AND THEIR INTERACTIONS ON KILLING PER CENTS**Function:** FACTOR

Experiment Model Number 9:

Randomized Complete Block Design for Factor A, with Factor B a Split Plot on A

Data case no. 1 to 72.

Factorial ANOVA for the factors:

Replication (Var 1: REPLICATIONS) with values from 1 to 4

Factor A (Var 2: HERBICIDES) with values from 1 to 3

Factor B (Var 3: DOSAGE RATES) with values from 1 to 6

Variable 4: KILLING PERCENTAGES

Grand Mean = 16.200 Grand Sum = 1166.410 Total Count = 72

## A. TABLE OF MEANS

1	2	3	4	Total
1	*	*	16.027	288.480
2	*	*	16.299	293.390
3	*	*	16.246	292.430
4	*	*	16.228	292.110
*	1	*	16.297	391.120
*	2	*	14.554	349.290
*	3	*	17.750	426.000
*	*	1	9.013	108.150
*	*	2	11.183	134.190
*	*	3	15.855	190.260
*	*	4	17.163	205.950
*	*	5	18.362	220.350
*	*	6	25.626	307.510
*	1	1	12.030	48.120
*	1	2	13.755	55.020
*	1	3	14.278	57.110
*	1	4	14.910	59.640
*	1	5	15.237	60.950

* 1 6	27.570	110.280
* 2 1	7.237	28.950
* 2 2	9.912	39.650
* 2 3	13.668	54.670
* 2 4	15.640	62.560
* 2 5	17.752	71.010
* 2 6	23.113	92.450
* 3 1	7.770	31.080
* 3 2	9.880	39.520
* 3 3	19.620	78.480
* 3 4	20.938	83.750
* 3 5	22.097	88.390
* 3 6	26.195	104.780

---

**B. ANALYSIS OF VARIANCE TABLE**

K Value	Source	Degrees of Freedom	Sum of Squares	Mean Square	F Value	Prob
1	Replication	3	0.77	0.26	0.98	
2	Factor A	2	122.93	61.46	235.29	0.0000
-3	Error	6	1.57	0.26		
4	Factor B	5	2056.84	411.37	1927.58	0.0000
6	AB	10	282.38	28.24	132.32	0.0000
-7	Error	45	9.60	0.21		
Total		71	2474.09			

Coefficient of Variation: 2.85%

s<sub>y</sub> for means group 1: 0.12      Number of Observations: 18

s<sub>y</sub> for means group 2: 0.10      Number of Observations: 24

s<sub>y</sub> for means group 4: 0.13      Number of Observations: 12

s<sub>y</sub> for means group 6: 0.23      Number of Observations: 4

**C. MEAN SEPARATION****1. MEAN SEPARATION FOR HERBICIDES**

Case Range: 79 - 81 HERBICIDES

Variable 4: KILLING PERCENTAGE

Function: RANGE

Error Mean Square = 0.26

Error Degrees of Freedom = 6

No. of observations to calculate a mean = 24

Duncan's Multiple Range Test

LSD value = 0.36

$s_{\bar{x}} = 0.1043$  at  $\alpha = 0.050$

x

Original Order	Ranked Order
Mean 1 = 16.30 B	Mean 3 = 17.75 A
Mean 2 = 14.55 C	Mean 1 = 16.30 B
Mean 3 = 17.75 A	Mean 2 = 14.55 C

## 2. MEAN SEPARATION FOR DOSAGE RATES (IRRESPECTIVE OF HERBICIDES CONSIDERATION)

Case Range: 84 - 89 DOSAGE

Variable 4: KILLING PERCENTAGE

Function: RANGE

Error Mean Square = 0.21

Error Degrees of Freedom = 45

No. of observations to calculate a mean = 12

Duncan's Multiple Range Test

LSD value = 0.38

$s_{\bar{x}} = 0.13$  at  $\alpha = 0.050$

x

Original Order	Ranked Order
Mean 1 = 9.01 F	Mean 6 = 25.63 A
Mean 2 = 11.18 E	Mean 5 = 18.36 B
Mean 3 = 15.85 D	Mean 4 = 17.16 C
Mean 4 = 17.16 C	Mean 3 = 15.85 D

Mean 5 = 18.36 B      Mean 2 = 11.18 E  
 Mean 6 = 25.63 A      Mean 1 = 9.01 F

### 3. MEAN SEPARATION FOR DOSAGE RATES IN 2,4-D

Case Range : 92 - 97

Variable 4 : KILLING PERCENTAGE

Function : RANGE

Error Mean Square = 0.21

Error Degrees of Freedom = 45

No. of observations to calculate a mean = 4

Duncan's Multiple Range Test

LSD value = 0.66

$s_{\bar{x}} = 0.23$  at  $\alpha = 0.050$

x

Original Order		Ranked Order
Mean 1 = 12.03	E	Mean 6 = 27.57 A
Mean 2 = 13.76	D	Mean 5 = 15.24 B
Mean 3 = 14.28	CD	Mean 4 = 14.91 BC
Mean 4 = 14.91	BC	Mean 3 = 14.28 CD
Mean 5 = 15.24	B	Mean 2 = 13.76 D
Mean 6 = 27.57	A	Mean 1 = 12.03 E

**4. MEAN SEPARATION FOR DOSAGE RATES IN ROUND UP**

Case Range: 98 - 103

Variable 4: KILLING PERCENTAGES

Function: RANGE

Error Mean Square = 0.21

Error Degrees of Freedom = 45

No. of observations to calculate a mean = 4

Duncan's Multiple Range Test

LSD value = 0.66

 $s_{\bar{x}} = 0.23$  at  $\alpha = 0.050$ 

x

□

Original Order		Ranked Order	
Mean 1 = 7.24	F	Mean 6 = 23.11	A
Mean 2 = 9.91	E	Mean 5 = 17.75	B
Mean 3 = 13.67	D	Mean 4 = 15.64	C
Mean 4 = 15.64	C	Mean 3 = 13.67	D
Mean 5 = 17.75	B	Mean 2 = 9.91	E
Mean 6 = 23.11	A	Mean 1 = 7.24	F

**5. MEAN SEPARATION FOR DOSAGE RATES IN PARAQUAT**

Case Range: 104 - 109

Variable 4: KILLING PERCENTAGES

Function: RANGE

Error Mean Square = 0.2130

Error Degrees of Freedom = 45

No. of observations to calculate a mean = 4

Duncan's Multiple Range Test

LSD value = 0.66

 $s_{\bar{x}} = 0.23$  at  $\alpha = 0.050$ 

x

□

Original Order

Ranked Order

Mean 1 = 7.77 F Mean 6 = 26.19 A

Mean 2 = 9.88 E Mean 5 = 22.10 B

Mean 3 = 19.62 D Mean 4 = 20.94 C

Mean 4 = 20.94 C Mean 3 = 19.62 D

Mean 5 = 22.10 B Mean 2 = 9.88 E

Mean 6 = 26.19 A Mean 1 = 7.77 F

□

**6. MEAN SEPARATION INTERACTIONS OF HERBICIDES AND DOSAGE RATES**

Case Range: 92 - 109 INTERACTIONS OF HERBICIDES AND DOSAGE RATES

Variable 4: KILLING PERCENTAGES

Function: RANGE

Error Mean Square = 0.21

Error Degrees of Freedom = 45

No. of observations to calculate a mean = 4

Duncan's Multiple Range Test

LSD value = 0.66

$s_{\bar{x}} = 0.23$  at  $\alpha = 0.050$

X

Original Order			Ranked Order				
Mean	1 =	12.03	L	Mean	6 =	27.57	A
Mean	2 =	13.76	K	Mean	18 =	26.19	B
Mean	3 =	14.28	JK	Mean	12 =	23.11	C
Mean	4 =	14.91	IJ	Mean	17 =	22.10	D
Mean	5 =	15.24	HI	Mean	16 =	20.94	E
Mean	6 =	27.57	A	Mean	15 =	19.62	F
Mean	7 =	7.238	N	Mean	11 =	17.75	G
Mean	8 =	9.913	M	Mean	10 =	15.64	H
Mean	9 =	13.67	K	Mean	5 =	15.24	HI
Mean	10 =	15.64	H	Mean	4 =	14.91	IJ
Mean	11 =	17.75	G	Mean	3 =	14.28	JK
Mean	12 =	23.11	C	Mean	2 =	13.76	K
Mean	13 =	7.770	N	Mean	9 =	13.67	K
Mean	14 =	9.880	M	Mean	1 =	12.03	L
Mean	15 =	19.62	F	Mean	8 =	9.913	M
Mean	16 =	20.94	E	Mean	14 =	9.880	M
Mean	17 =	22.10	D	Mean	13 =	7.770	N
Mean	18 =	26.19	B	Mean	7 =	7.238	N