PREDATION ON LIZARD EGGS BY ANTS: INTERACTION MODIFICATIONS IN AN UNSTABLE PHYSICAL ENVIRONMENT

by

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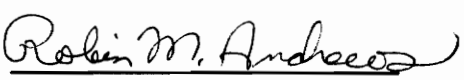

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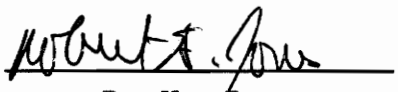

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(ABSTRACT)

The importance of abiotic influences on the strength of biotic interactions is largely unknown. To explain large annual fluctuations in the population size of the tropical lizard, *Anolis limifrons*, on Barro Colorado Island, Panama, I hypothesized that annual variation in lizard population size is the result of modifications in the rate of predation on lizard eggs by *Solenopsis* ants induced by annual variation in wet season rainfall. I tested this hypothesis by manipulating water availability on experimental plots to simulate the wettest (HW) and driest (LW) wet seasons in the last twenty years. The mean time to find and attack eggs by *Solenopsis* ants was significantly shorter on HW plots (range=6.6-21.7 days) than LW plots (range=17.8-30.8 days). Exponential models that regressed time on the cumulative percent mortality indicated that 1) the rate of predation was 3-5 times faster on HW plots than LW plots and 2) the predicted mortality of lizard eggs during their 42 day incubation period was 82.2-95.7% on HW plots and 56.1-58.6%

on LW plots. Thus, the amount of rainfall during the wet season affected the population size of *A. limifrons* by modifying the strength of the interaction between *Solenopsis* ants and the eggs of *A. limifrons*.

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CHAPTER 1

INTRODUCTION

Two important concepts emerged during the early history of ecology. First, Malthus (1798) demonstrated that populations cannot grow without limit. As cited by Pollard (1981), this phenomenon was expressed mathematically by Verhurlst (1838) as the logistic growth model. The logistic growth model predicts that the trajectory of population density follows a sigmoidal path through time. Thus, the maximum population density occurs in the region of the trajectory where the upper arm of the sigmoidal curve levels out. This maximum population size has been termed the population limit or carrying capacity. The limit to population size is a result of some essential factor (e.g., food) that imposes the greatest restriction (e.g., food shortage) on population growth. This process is known as population limitation.

Second, many populations persist for long periods of time despite fluctuations in size (Nicholson 1933). If annual fluctuations are averaged over many years, populations tend to maintain a characteristic density (Howard and Fiske 1911). Today, ecologists speak of populations having an equilibrium density rather than a

characteristic density. The equilibrium density of a population is a result of a balance between processes that promote population increase and population decline (Nicholson 1933). Birth and immigration are processes that contribute to population increase while death and emigration are processes that contribute to population decline.

How do populations maintain equilibrium densities for long periods of time? The answer is that deviations (or fluctuations) in the equilibrium density are corrected by changing the relative strengths (rates) of processes promoting population growth and decline. For example, if the size of the population is less than the equilibrium density, the relative rates of birth and/or immigration increase in relation to the rates of death and/or emigration to promote population growth. If the size of the population is greater than the equilibrium density, the relative rates of death and/or emigration increase in relation to the rates of birth and/or immigration to promote population decline. To maintain an equilibrium density, however, either the rate of population decline or population growth or both must be dependent upon population density (Howard and Fiske 1911, Nicholson 1933).

Before I discuss how the relative strength (effectiveness) of a process can change, the terms "factor"

and "process" must first be distinguished. A factor is any component within the environment that can be measured at one point in time. A process is the act of changing the value or status of a component during a period of time. In other words, processes are described as rates of change in the value of a factor. Thus, the density of a predator is a factor but the act of predation is a process. The distinction between factor and process is very important (Sharov 1992). Factors may influence the strength of a process but they cannot influence the value of other factors directly. Instead, a factor can only influence the value of another factor by changing the strength of the process that links them together. Similarly, the strength of a process can alter the value of a factor but it cannot directly alter the strength of another process. For example, the rate of birth may influence the density of a population but it does not directly influence the rate of death.

As ecologists we would like to know 1) how different factors influence the strength of population regulating processes 2) which factors have the strongest influence on population regulating processes and 3) how do factors influence population regulating processes in a manner that allows populations to persist for long periods of time. Both Malthus and Darwin postulated several mechanisms to

explain the maximum size of populations but it was not until the turn of the century that scientists looked at these questions more closely.

HISTORICAL VIEWS

The relationship between the strength of any given process and the density of a population is either density-independent or density-dependent (Smith 1935). If a process does not vary in strength at different densities it is classified as density-independent. On the other hand, if the process varies in strength at different population densities, it is classified as density-dependent. Graphically, a density-independent relationship would be one in which the line representing the per capita affect on density has either a slope that is undefined or is equal to zero. A density-dependent relationship however, has a slope that is either positive or negative, although the relationship does not have to be linear (Sinclair 1989). Hereafter I will make references to density-dependent and density-independent factors as well as processes. I do not intend to use the terms "factor" and "process" synonymously but rather I want to indicate those factors that influence the strength of a process in a density-dependent or density-

independent manner.

Which factors cause a process to behave in a density-dependent manner and which factors cause a process to behave in a density-independent manner? Density-dependent factors are mainly biotic in nature whereas density-independent factors are mainly abiotic in nature (Smith 1935). Biotic factors include all of the living components in a system whereas abiotic factors include all of the non-living components of the system. The influence of biotic factors can either be internal or external with respect to the population. Internal factors can include the genetic composition (Ford 1931, Chitty 1967), intraspecific competitors (Nicholson 1933, Smith 1935), and behavior (Chitty 1960, Wynne-Edwards 1962). External factors include interspecific competitors (Nicholson 1933), predators (Kenmore et al 1984, Le Cren 1987), disease (Anderson and May 1980, Myers 1988, Bergerud 1988), and parasites (Howard and Fiske 1911, Roland 1988). All of these biotic factors may promote variation in the strength of a process with density. Abiotic factors typically include weather, soil attributes, slope and elevation, and generally affect all individuals in the population the same regardless of density. Both Andrewartha and Birch (1954) and Smith (1935) argue, however, that under some conditions, abiotic factors

may also influence populations in a density-dependent manner.

If an organism's environment can be divided into density-dependent and density-independent components, is one more important than the other in determining population size? This debate has raged for much of this century. Some researchers believed that density-dependent factors were primarily responsible for determining population size whereas others believed that density-independent factors were primarily responsible.

Nicholson (1933) is generally credited as the father of the density-dependent school of thought while Andrewartha and Birch (1954) have been given this role for the density-independent school. However, both schools had their origin before Nicholson's and Andrewartha and Birch's time. In an effort to control gypsy moth and brown-tailed moth populations, Howard and Fiske (1911) proposed that the numbers of moths present is a result of what they termed facultative factors. This term is the pre-cursor of density-dependent factors. According to Howard and Fiske, only biotic processes such as parasitism, disease and competition for food could control the population size. All of these processes operate at different strengths at varying densities. It is these processes which are thus responsible

for lowering the population density if it grows too big or allowing population growth if the population density becomes too small. Population size is thus regulated about an equilibrium density by density-dependent processes. This is the same concept proposed by Nicholson (1933), although Nicholson believed that competition is the most important process that can determine the size of a population.

On the other hand, as cited by Krebs (1994), both Bodenheimer (1928) and Uvarov (1931) stressed the importance of density-independent processes for limiting the size of a population. Bodenheimer recognized that weather affected physiological processes and survivorship of insects. He showed that the rate of egg production and speed of development of larval insects were influenced by temperature. The speed of development is important to population growth because most mortality of insects occurs in the early life history stages. High survival during early stages of development results in a large adult population. Fluctuations in insect population size are also correlated with climate (Uvarov 1931).

The density-dependent school of thought was widely accepted and received little criticism until "The distribution and abundance of animals" by Andrewartha and Birch was published in 1954. Andrewartha and Birch argued

that natural populations do not behave in the manner predicted by current (Nicholson's) theory or in the manner of laboratory populations. They believed that natural populations fluctuate in size but that fluctuations are not necessarily around an equilibrium density and that equilibrium densities are theoretical constructs and can not be measured empirically. Andrewartha (1958) believed that ecologists should focus their efforts on measuring abundances and determining why population size varies temporally and spatially.

Andrewartha and Birch (1954) also proposed that abundance is related to geographic location. Individuals of a species are distributed throughout regions that are suitable for survival. If a region is unsuitable, then no individuals will be found there. Typically there is a gradient from suitable to unsuitable environments. According to Andrewartha and Birch, suitability of an environment refers to the abiotic components of the environment (e.g., weather). Thus, at the core of a distribution, environmental suitability is greatest and this is where abundance is the greatest. On the periphery of a distribution, abundance is low as a result of less suitable but tolerable conditions. Because the distribution and abundance of individuals is based upon suitable abiotic

environments, local fluctuations in abundance must be a result of the same processes.

Members of the density-dependent school of thought did not believe that the size of a population could be determined by the effects of density-independent factors or processes. They argued that if weather (for example) was responsible for determining the size of the population, populations would frequently become extinct. Elton (1955) and others did not believe that populations could be maintained by chance occurrences because weather would not necessarily become more favorable when a population was at low numbers. They believed instead that deviations from the equilibrium density are corrected by processes that are dependent upon the density of the population. The density-independent school of thought countered this argument by stating that the climate as it presently is, undergoes small fluctuations in favorableness (Birch 1958). Most individuals are adapted to surviving the current fluctuations in environmental favorableness and hence the populations will persist. Populations near the edges of the species distribution however, may become extinct because the climate in these regions are naturally less favorable for the organisms than if they were at the core of their distribution. Even small deviations in favorableness at

distribution boundaries will cause extinction. When conditions become favorable again, recolonization can occur as a result of immigration from the distribution core. Birch (1958) believed that it was this process that was responsible for accounting for the low numbers of individuals encountered at the species distribution boundary. Also, environmental conditions change through long periods of time and hence the populations at the core of a species distribution may experience conditions similar to the periphery at some future date and therefore promote their extinction.

These two schools provided very different kinds of evidence to support their claims. Evidence for the density-independent school included extensive field studies on natural populations. Until the 1960's evidence for the density-dependent school was largely from theory with little data on natural populations (e.g., Lack 1954, MacArthur 1958). Despite this, Nicholson's theory had great support from contemporary ecologists who thought it was more deterministic and more robust than Andrewartha and Birch's theory (Sinclair 1989). During the 1960's, however, several methods were developed to detect density-dependent processes in natural populations (e.g., Morris 1963, Varley and Gradwell 1960, 1968).

MODERN VIEWS

The accumulation of evidence for density-dependent processes in natural populations (e.g., Krebs 1970, Larkin 1973, Lidicker 1975), convinced ecologists that density-dependent processes may be common (Sinclair 1989, Harrison and Cappuccino 1995). Recent surveys, however, indicate that the frequency of density-dependence detected in natural populations is often less than 50% (Gaston and Lawton 1987, Stiling 1987). Why are density-dependent processes detected so infrequently in natural populations? One possibility is that density-dependence is difficult to detect (Gaston and Lawton 1987, Hassell 1987, Dennis and Taper 1994). For example, observations of mortality and natality rates at different densities often appear as a cloud of points when graphed. A relationship of this type is called density vague (Strong 1984). Strong believed that this type of relationship is a result of temporal or spatial differences in the strength of density-dependent and density-independent processes. When plotting mortality (or natality) rates against density, variation in the relative strength of density-dependent and density-independent processes causes data points to appear scattered and hence a statistically significant regression can not be detected (i.e, the

variance in data points about a regression line is large). Therefore density-dependence may not be detected in some populations because density-independent processes mask the results of density-dependent processes.

Are there better methods we can use to test for density-dependence? One method is to use long time series (Godfray and Hassell 1992, Woiwood and Hanski 1992, Holyoak 1993, Wolda and Dennis 1993). Woiwood and Hanski (1992) found that if short time series (less than 20 years) were excluded from analyses, the frequency of detection of density-dependence increases from 69% to 84% in moth populations and from 29% to 57% in aphid populations. Another method is to experimentally manipulate densities (e.g., Murdoch 1970, Gaston and Lawton 1987, Stiling 1987, Sinclair 1989, Harrison and Cappuccino 1995).

Density-dependent processes are now generally believed to be essential for population regulation (Murdoch and Walde 1989, Hanski 1990, Godfray and Hassel 1992), however, the detection of density-dependent processes does not necessarily mean that all populations are regulated (Murdoch 1994, Turchin 1995). For regulation to occur, density-dependent processes must have three important properties (Turchin 1995). First, the density-dependent process must work in a manner that promotes the return of the population

to an equilibrium density. Second, the density-dependent process must be strong enough to counteract the influence of density-independent factors. Third, any time lags in the density-dependent response must be relatively short as long time lags produce diverging oscillations (May 1973a).

Since the 1970's, ecologists have become more receptive to the idea that populations may be in a state of non-equilibrium (De Angelis and Waterhouse 1987, Koetsier et al. 1990). A population is in a non-equilibrial state if the density of the population does not fluctuate about an equilibrium density or if there is no tendency to return to an equilibrium density if perturbed. This concept was actually first proposed by Andrewartha and Birch (1954).

Many ecologists (e.g., Elton 1958, Pimental 1961, MacArthur 1955) believed that natural populations were in an equilibrial state as a result of complex interactions between many species (De Angelis and Waterhouse 1987). This connection between stability and complexity, however, may be unfounded. By increasing the number of species or increasing the strength of interactions between species, May (1973b) showed that populations would no longer fluctuate about an equilibrium density. Instead, predicted densities would always fall within a range of points and a constant density was never observed. The probability distribution of

points within this range can be estimated but the actual density a population would have in the future could not be determined. This is known as chaotic dynamics. Non-equilibrium populations may also be a result of either stochastic environmental events or as a result of interactions between species that have positive feedbacks (e.g., a predator population which is not reduced in size as a prey becomes rare) (Wiens 1984, De Angelis and Waterhouse 1987). For many centuries, scientists from a variety of fields held onto the belief that their study systems were in an equilibrial state (Egerton 1973). Many of the theoretical models that were developed were predominantly equilibrial and had no stochastic component to them (Sousa 1979). With the diversity of equilibrium models available, several field ecologists still examine populations (on a local scale) and apply equilibrium models (e.g., population regulation) to them without knowing whether or not their populations are in fact at equilibrium (Connell and Sousa 1983). Ecologists should examine the local population dynamics of the taxa they study more closely because they may find that the local populations are not in fact at equilibrium (Sousa 1979).

The detection of equilibrium densities in natural populations, however, is difficult. Equilibrium models are

merely abstractions based upon ideal conditions with unreal assumptions and therefore may not be applicable to field studies (Wolda 1989). In the natural world, the prevalence of biotic feedback instabilities and stochasticity subjects populations to conditions that are not incorporated in models but may be very important in determining long term population dynamics (De Angelis and Waterhouse 1987). As a result, there is a great need to incorporate both temporal and spatial stochastic elements to models to make predictive models more realistic (Onstad 1991).

Several studies document that local populations can exist in a state of non-equilibrium (list from Sousa 1979). If all local populations happen to be in a state of non-equilibrium, is it worth while to examine population regulation any further?

Recently, Murdoch (1994) stated that the study of population regulation is central to ecology regardless if local populations are in a state of equilibrium or non-equilibrium. The definition that Murdoch employs for regulation however is a modification of the definition I described earlier. Regulation is now defined as all the processes that keep the size of a given population within bounded limits rather than keeping the size of a population at one fixed equilibrium density (Royama 1977, Murdoch

1994). This definition is similar to that of Wolda (1989) and Dennis and Taper (1994) who describe equilibrium as being a cloud of points that represent the probability distribution of observed densities. This broad definition of regulation now includes cyclic and chaotic dynamics, and the arguments between equilibrium and non-equilibrium concepts become semantic (Berryman 1991, Turchin 1995).

Before I explain why a metapopulation approach is important to population regulation I will define what a metapopulation is. A metapopulation is composed of a collection of local populations that are spatially separated but linked together by the dispersal of individuals. Each local population is composed of all individuals living together in a patch within a spatially heterogenous landscape. The metapopulation concept usually assumes that the number of occupied patches (local populations) is constant through time. The equilibrium number of occupied patches is a result of a balance between the extinction and colonization processes.

Does a metapopulation approach help us learn more about population regulation? If each local population is taken in isolation, there is a high probability that a local population will become extinct as a result of demographic and environmental stochasticity. If, however, that same

local population is examined within the metapopulation, the local population will persist. Why is this so? Local populations may persist for longer periods of time as a result of density-dependent migration of individuals from surrounding populations. Rather than forming new populations, migrating individuals may move into populations that have a sparse density and hence prevent extinction. The equilibrium number of populations in existence is still maintained because few local populations are formed (more dispersers move into existing populations rather than creating their own) and few local populations go extinct (populations on the verge of extinction are rescued by immigration).

To test this hypothesis, Murdoch (1994) studied populations of california red scale that appear to be regulated (i.e., the population density fluctuated within narrow bounds). To determine how population density is regulated, eight possible mechanisms were investigated. These mechanisms included predation, a refuge and metapopulation dynamics. None of these mechanisms, however, could account for the stability of red scale populations. Density-dependent parasitism was not even detected in the study populations, but Murdoch (1994) believed it to be present.

Recently, Krebs (1992) suggested that the emphasis on density-dependent regulation may be overplayed, and may actually impede the progress towards understanding how population size is determined. During most of the last half of this century, ecologists devoted their time to detecting density-dependent processes and accomplishing little to resolve the debate for population regulation (Krebs 1992). In fact, Krebs equated the search for density-dependence with the legendary search for the holy grail. A suggested alternative approach is to explain the variability in population size instead of searching for a process that deterministically regulates population size (Shepherd and Cushing 1990).

INDIRECT EFFECTS AND INTERACTION MODIFICATIONS

Both density-dependent and density-independent factors may influence populations either directly or indirectly. Direct effects of a factor on a population are a result of immediate physical interactions between the population and the factor in question (Wootton 1993, 1994a,b). Indirect effects of a factor, on the other hand, are a result of one factor that influences a population through an intermediary step (Wootton 1993, 1994a,b). For example, the predation

of one species upon another species is a direct effect. On the other hand, if some factor positively or negatively alters the predation process, that factor would exert an indirect effect upon the prey species.

The distinction between direct and indirect effects was first made by Beklimishev (1951). According to Beklimishev, direct effects can be produced by a) topic interactions: species one alters the physical or chemical conditions of the environment for species two, b) trophic interactions:species one eats species two, c) phoresic interactions:species one uses species two for transportation and d) fabric interactions:species one uses either the bodies, carcasses or excretions of species two for some constructive purpose. A third species (or factor) could have an indirect effect on species two in all of the above types of interactions by changing the amount of influence species one has on species two.

If the direct relationship between two species can be described by the linear equation:

$$G_2 = m_{12} N_1 + b \qquad (1)$$

where G_2=the amount of population growth of species 2

N_1=the density of species 1

m_{12}=regression coefficient that describes the influence of species 1 on the amount of population

growth of species 2

b=the amount of population growth of species 2 in the absence of species 1

a third species could alter the amount of influence exerted by species one on species two ($m_{12}N_1$) by one of two mechanisms.

First, a factor may indirectly influence a population by altering the abundance of an intermediary species (N_2). Because the indirect influence of a factor operates through a series of direct interactions this type of mechanism is referred to as an interaction chain (Wootton 1993, 1994b). An example of this type of mechanism includes a trophic cascade effect. A carnivore may indirectly influence the density of a plant population by reducing the density of a herbivore population.

Second, a factor may indirectly influence a population by altering the strength (m_{12}) of the direct interaction between a population and another factor. The mechanism responsible for this type of indirect effect is referred to as an interaction modification (Wootton 1993, 1994b). For example, high levels of plant abundance may indirectly influence the density of a prey species by providing more cover for the prey. By providing more cover for the prey,

the plant abundance has effectively lowered the predation rate of a predator species on a prey species. Hence, the changing of plant cover will also change the rate of predation.

The importance of direct effects on populations is well known, but how important are indirect effects on populations? The detection of indirect effects within a community would indicate that the dynamic behavior of a species can not be predicted based upon observations of it and one other species it directly interacts with (Billick and Case 1994). Currently, most models that predict the density of one species are based upon the density of two interacting species (e.g., Lotka-Volterra competition model, Rosenzweig and MacArthur predator prey model 1963). If indirect effects can be detected in natural communities, it would indicate that more complex models are needed.

To account for the influence of additional environmental variables on long term population dynamics, complex models have been developed that are based upon the life system concept (Clark 1964, Geier 1964, Sharov 1992). A life system is defined as a population taken together with all components of the environment that have significant positive or negative effects on this population (Sharov 1992). The life system model is multivariate incorporating

the influence of many different factors on physiological and ecological processes. The life system is therefore a product of all the interactions between environmental and population components. This includes density-independent physiological responses to the environment and intraspecific and interspecific density-dependent processes.

In life system models, both density-dependent and density-independent processes are considered important for population regulation because the influence of one may be modified or compensated by the other (Sharov 1992). This concept has been ignored as a result of the exuberant search for density dependent mechanisms during the latter half of this century. However, the life system concept can reveal important processes and factors in population regulation. Modifications to the structure of a valid life system model can identify important population regulating factors and processes by promoting a significant change in the dynamics of a population (Sharov 1992). Modifications to the life system model include: 1) changing the mean value or variance of some factor; 2) addition or exclusion of some ecological process; and 3) fixation of the rate of an ecological process at the equilibrium level. These modifications can be made during empirical experiments or in computer simulations.

Currently, indirect effects are generally identified by statistical methods and the causal mechanism is seldomly known (Billick and Case 1994). The statistical detection of indirect effects may be unfounded for two reasons. First, models concerning species interactions may be inaccurate. Billick and Case (1994) demonstrated that models differ in the ability to detect indirect effects. For example, a model that assumes constant per capita rates may detect an indirect effect whereas a model that assumes constant populational rates (i.e, constant # of individuals dying/time interval) may not detect an indirect effect. Why is this so?

The difference between the two model types concerns the probability of some particular event occurring to a given individual in the population. If a model assumes a constant per capita rate, the probability of some event (e.g., death) occurring to any given individual remains the same as the number of individuals increases or decreases. The probability of some event occurring to an individual when a constant populational rate is assumed changes as the number of individuals changes. (i.e., assume that during any given time interval 10 individuals are always killed. If the population size initially consisted of 1000 individuals, the per capita survivorship would be 0.99. If the population

size declined to a level of 100 individuals, the per capita survivorship would now be 0.90%.) Thus, if a constant per capita rate is assumed, the total number of individuals killed decreases exponentially with time because there are fewer individuals to be killed. If a constant populational rate is assumed, the total number of individuals killed will not decrease with time but instead increase on a per capita basis in a linear fashion until all individuals in the population are killed.

Second, changes in population density may be a result of some factor that is not measured (e.g., the density of another species). If this factor is not measured, the indirect effect may not be detected. Given these problems, Billick and Case (1994) suggest it would be simpler to search empirically for causal mechanisms that produce changes in population density. This approach emphasizes the importance of studying fluctuations in population size rather than looking for density dependent mechanisms that might maintain a population at a determined equilibrium density. The argument for studying fluctuations in population size rather than looking for density dependent mechanisms is not new (e.g., Howard and Fiske 1911 and Andrewartha and Birch 1954). It does indicate however, that despite strong suggestions during the past century,

ecologists are still trying to identify density-dependent mechanisms that could maintain a population in an equilibrial state rather than identifying what causes the population to fluctuate.

With the accumulation of evidence for global warming there has been a recent surge of interest on how changes in the environment will influence populations (e.g., Kareiva et al. 1993). Clearly, abiotic factors affect biotic interactions (e.g., Wiens 1977, Peterson and Black 1988, Traveset 1990, Dunson and Travis 1991, Werner and McPeek 1994), but evidence that natural variation in an abiotic factor affects the relative strength of biotic interactions is lacking (Karieva et al. 1993, Spiller and Schoener 1995). Variation in the strength of biotic interactions caused by temporal and spatial variation in abiotic factors may have an important influence on population dynamics and community organization (Kingsolver 1989, Dunson and Travis 1991, Hunter and Price 1992, Spiller and Schoener 1995). One reason for the lack of empirical research in this area may be the absence of theoretical models that predict changes in population size as a result of modified biotic interactions.

Recently, Sharov (1985, 1986, 1992), Ives and Gilchrist (1993) and Ives (1995a,b) developed the theoretical framework to predict changes in population size as a result

of modifications to population growth rates imposed by variation in biotic and abiotic factors. Changes in the population size of a species can be described by the linear equation:

$$N_{t+1} = au + b_0 + N_t + CN_t \qquad (2)$$

where N=log population density

t=discrete time

u=value of a given environmental factor that influences the growth of the population in a density-independent manner (e.g., temperature)

a=effect of factor u on the population growth rate

b_0=population growth rate when u is equal to 0 (y intercept from regression of u on the amount of population growth)

C=intraspecific density-dependent influence on population size (regression coefficient derived from a regression of the amount of population growth on the density of the population).

For a community, $\mathbf{N}_t$ becomes a vector in which the elements are log-densities of individual populations, **a** and **b** are described by vectors and **C** is a matrix. Each element in matrix **C** describes the density-dependent influence of other members of the community on the population size of other species (regression coefficient derived from a regression of

the amount of population growth of species *j* on the density of species *i*). If several environmental variables were to be considered, **u** would become a vector and thus **a** would be described by a matrix.

This model makes an important differentiation between the direct and indirect influence of factor *u* on the population density of a species (Ives and Gilchrist 1993, Ives 1995a). First, *u* can directly influence the amount of population growth by influencing the physiology of individuals (e.g., the number of eggs produced by an individual). This effect is determined by the term *au* in equation (2). Secondly, factor *u* can indirectly influence the density of a species by directly altering the amount of population growth (and hence population size) of other species within the community. The mechanism of the indirect effect in this case is an interaction chain because factor u changes the densities of other species in the community vector $\mathbf{N}_t$ (intermediate step) by altering their population growth. By changing the values of vector $\mathbf{N}_t$, *u* indirectly alters the outcome of the term $\mathbf{CN}_t$ in equation (2).

The ability of a population to resist a change in its equilibrium (mean) density as a response to a change in the mean of an environmental factor has been termed buffer ability or mean stability (Sharov 1986, Sharov 1992).

Changes in the mean population density with respect to changes in the mean value of u can be determined by solving equation (2) under the conditions that $N_{t+1}=N_t$:

$$\frac{dN}{du} = aC^{-1} \tag{3}$$

In a community model, the result of equation (3) is a matrix (**S**). Therefore the coefficient of mean stability (MS) is the reciprocal of the elements (partial derivatives of N_t with respect to u_j) of matrix **S**:

$$MS_{ij} = \left(\frac{\partial N_i}{\partial u_j}\right)^{-1} \tag{4}$$

where i=the species number in the community

j=the number of environmental factors considered in the model.

Larger values of MS imply that the mean population density is less sensitive to changes in the mean value of factor u. Density-dependent processes (**C**) can minimize changes in the mean population density resulting from the change in factor u (Sharov 1992, Ives and Gilchrist 1993, Ives 1995a). Changes in the mean population density can therefore be determined by dividing the change in the observed mean value of u by MS.

The ability of a population to resist a change in the

variance (width of a probability distribution) of population density as a response to a change in the variance of some factor u is termed homeostasis or variance stability (Sharov 1986, Sharov 1992). The coefficient of variance stability (VS) is characterized by:

$$VS = \left(\frac{\partial s^2_N}{\partial s^2_u} \right)^{-1} \quad (5)$$

Larger values of VS imply that variation in population density is not sensitive to changes in the variance of u. Changes in the variance of population density can thus be determined by dividing the change in the observed variance of u by VS.

As mentioned above, the influence of density independent mechanisms on population growth may be countered by strong density dependent interactions. Results of simulation models using this mathematical construct indicate that the amount of change in population density depends upon the type of density-dependent interaction occurring between species (Ives and Gilchrist 1993, Ives 1995a). For example,

competitive interactions tend to promote changes in population size when changes in the environment occur whereas predator/prey interactions tend to buffer changes in population density produced by changes in the environment.

An assumption of the models predicting changes in population size associated with environmental variation is that the strength of the interactions between species remains the same as the environment changes. In other words, the elements of matrix **C** are invariant. Wootton (1993) has shown that the presence of one species may modify the interaction between two other species. Therefore elements of matrix **C** may not remain constant.

Assumptions are inherent to all models but this does not mean that they should remain untested. Violations in even the most basic assumptions could possibly alter the outcome of simulations utilizing mathematical models. We should therefore: 1) test for the realism of such assumptions; and 2) determine if violations of these assumptions significantly alter model predictions. In my study, I will show that the assumption of invariant interaction strengths is unrealistic and that the long term dynamics of a population are dependent upon violations of this assumption.

CHAPTER 2

INTRODUCTION

Population density of the lizard *Anolis limifrons* at Barro Colorado Island (BCI) in Panama fluctuates as much as eight fold from one year to the next, and even more widely over decades (Andrews and Rand 1982, Andrews 1991, Andrews and Wright 1994). Several lines of evidence suggest that the amplitude of population fluctuation is more related to variation in the mortality of eggs than the mortality of lizards (hereafter lizard refers to all post-hatching individuals) or to fecundity. First, mortality of eggs exhibits greater temporal and spatial variability than does mortality of lizards (Andrews 1988). Second, the changes in lizard density that followed application of water to large tracts of BCI forest during three successive dry seasons were associated with changes in the survival of eggs, but not with alteration in the survival of lizards, or to the rate of egg production (Andrews and Wright 1994). Lizard densities increased on watered plots after the initial dry season in which water was applied, and decreased thereafter. Andrews and Wright (1994) hypothesized that continued wet conditions permitted the build-up of predators or pathogens

on the watered plots, although these same conditions were initially favorable for egg survival. Third, demographic models that incorporated the observed variability in survival of eggs and lizards, produced population fluctuations similar in amplitude to observed population fluctuations (Andrews, unpublished). Thus, the factor or factors that affect egg mortality are also likely to be responsible for the large fluctuations in population density.

Population density of *A. limifrons* is negatively correlated with the amount of rainfall during the wet season (approximately mid-April to December) (Andrews 1991, Andrews and Wright 1994), and wet season rainfall varies considerably from year to year (Windsor 1990). If the amplitude of population fluctuation is related to variation in egg mortality, then the amount of rainfall during the wet season and egg mortality should be negatively correlated. What mechanism(s) could connect moisture levels in the wet season to egg mortality? One possible mechanism is a direct effect of moisture on egg survival. Such a mechanism would result in population fluctuation because most of the egg production by *A. limifrons* occurs during the wet season (Andrews and Rand 1982). However, above some minimal level, variation in moisture is not related to egg survival

(Andrews and Sexton 1981). Another mechanism is the effect of moisture on the predators of eggs which indirectly affects the rate of egg mortality. Mortality of *A. limifrons* eggs in the field is largely due to predation by various species of *Solenopsis* ants (Subgenus: *Diplorhoptrum;* Kaspari in press). *Solenopsis* were the only ants that attacked eggs during staged encounters in the laboratory and the only ants observed to attack eggs in the field (Andrews 1988).

The distribution and abundance of ants are affected by rainfall. The abundance of ants is higher in the wet than the dry season and higher in dry seasons that have relatively high rainfall than in those which have relatively low rainfall (Levings 1983, Levings and Windsor 1984, Levings and Windsor 1985). In particular, moist microhabitats have both greater abundances of all ants and higher levels of foraging activity by *Solenopsis* and other small ants (Kaspari 1993). Study plots in which litter moisture was artificially enhanced had both greater numbers and higher levels of foraging activity by all ants than in unenhanced study plots (Levings 1983, Levings and Windsor 1984). Rainfall could thus indirectly influence the population density of *A. limifrons* by altering the rate at which *Solenopsis* ants prey upon *Anolis* eggs.

The hypothesis tested was that the amount of moisture during the wet season affects the rate of predation by *Solenopsis* ants on the eggs of *A. limifrons*. I tested this hypothesis by manipulating moisture on experimental plots and by monitoring predation rates on *A. limifrons* eggs. I predicted that variation in the abundance or in the foraging activity of *Solenopsis* ants as a result of these treatments would cause a parallel variation in egg mortality; enhanced moisture should increase egg mortality while reduced moisture should decrease egg mortality. Acceptance of the hypothesis that spatial variation in moisture causes parallel variation in egg mortality would imply that annual variation in the amount of wet season rainfall would also promote annual variation in egg mortality, and thus, annual variation in population density of *A. limifrons*.

MATERIALS AND METHODS

Plot Descriptions

Observations were conducted near Allee Creek on BCI, Panama. BCI is located within Gatun Lake, an artificial lake created during the formation of the Panama Canal. The forest in the Allee Creek watershed is regrowth from agricultural land that is at least 100 years old (Foster and

Brokaw 1982). Rainfall in central Panama is seasonal but the amount of rainfall varies both geographically (Andrews 1991) and annually (Windsor 1990).

All plots were located within a 30 x 18 m study area. The size and boundaries of the study area corresponded to a relatively flat area in the otherwise hilly terrain. The study area had a closed canopy and an open understory. The understory was dominated by the palm *Chrysophyllum panamensis* and by shrubs (e.g., *Diefenbachia* spp, *Eugenia oerstedanii, Garcinia madruno, Hybanthus prunifolium* and *Psychotria limonenesis*).

Ten experimental and two reference plots (hereafter reference plots are referred to as RF), each measuring 2 x 5 m, were established within the study area (Figure 1). Plot size was a compromise: manipulating water on larger plots would have been technically difficult and smaller plots would have had too few experimental eggs if eggs were allocated at normal densities (see below). Each experimental plot was randomly assigned to one of two treatments. These treatments simulated wet seasons that are relatively dry (hereafter referred to as low water plots=LW) or relatively wet (hereafter referred to as high water plots=HW), respectively. Plots were separated by no less than 2 m.

This study was conducted from May through August, 1995. The sequence of events for each experimental plot is outlined in Figure 2. Plots were initiated sequentially as eggs became available, but the sequence of events and the length of the study period was the same for all of them.

Water manipulations

During the manipulation period, I added 440 and 880 l of water per week to each LW and HW plot, respectively. The volume of water added to LW plots each week corresponded to the sum rainfall for the months of June, July and August of 1988 (49.9 cm), the year with the lowest rainfall during these months in the last 20 years The volume of water added to HW plots each week corresponded similarly to 1979 (101.8 cm), the year with the highest rainfall during these months in the last 20 years. The normal frequency of wet season rainfall is approximately 3-4 times a week. Thus, 110 l of water was added to each LW plot and 220 l of water was added to each HW plot on Monday, Wednesday, Thursday and Saturday during the manipulation period.

To ensure that water availability was at prescribed levels, I placed a clear plastic tarp above each experimental plot to intercept normal rainfall. The lowest portion of the tarp was at least 1 m above the ground so

that air movement at ground level would not be impeded. Three 110 l barrels aligned along the lowest edge of the tarp collected the water runoff. Litter that fell on the tarp was scattered evenly on the plot weekly.

Water was distributed to the plots from water collected in the barrels and from water pumped into the barrels from the water supply for the station. Water was sprayed on the plots with a garden hose connected to an electric sump pump located inside the collection barrels. Water pressure was controlled so that it formed a light spray. Each plots' daily allotment of water was sprayed evenly over each plot over a period of 30 to 60 minutes. The volume of water sprayed was monitored with a calibrated measuring stick that was marked with 10 l increments inside the collection barrel.

Ant density and foraging activity

To ensure that changes in predation rates on eggs were a result of change in the abundance or the activity, or both, of *Solenopsis* that were on the plot initially, I surrounded all experimental plots with ant-proof enclosures. The enclosure consisted of 34 cm high corrugated fiberglass sheets that were buried in the ground to a depth of 5-10 cm. The fiberglass was cut to fit over large roots. Gaps

between the root and the barrier were sealed with duct tape. A 5 cm wide strip on both sides of the top of the enclosure was painted with Fluon AD1. Fluon AD1 is a teflon based paint that creates a frictionless surface that prevents ants and other invertebrates from crawling on it (Hölldobler and Wilson 1990). I checked the integrity of the enclosure daily. In the middle of the observation period, the Fluon strips were wiped clean and Fluon was reapplied because the previous painted area began to show signs of weathering. I also raked away all of the litter from a 1 m wide strip around the outside of each plot after ant and colony density was sampled (see below) to serve as a further barrier for ant movement and to remove any nearby colonies or individuals.

The abundance of *Solenopsis* ants was monitored in several ways. To determine the initial and final density (# of ants/0.25 m^2) of individual *Solenopsis*, six 0.25 m^2 litter samples were collected using a stratified sampling scheme (one to two samples from each side of the plot) from each plot before and after the manipulation period. Litter collected initially was taken from the 1 m wide strip surrounding the plot, and litter collected after the manipulation period was collected from within the plots themselves. Litter samples were placed in Berlese funnels

for 48 hrs and heated from above with a 60 W light bulb. Invertebrates were collected in small cups containing 70% ethanol. To prevent other invertebrates from being attracted to the lights and entering the funnel, I placed plastic bags over the tops of the funnels. The invertebrates collected were placed in vials containing 70% ethanol. The dry litter was weighed (± 0.1 g) and used as an index of the quantity of litter on each plot at the start and conclusion of the manipulation period (see below).

In six of the one hundred and twenty berlese samples, the funnel plugged so that arthropods were not extracted. To avoid missing values for data analyses, I used the mean value for the number of *Solenopsis* ants collected from other samples on the same plot during the same time period. No more than one sample was missing for a given plot for a given time period.

To determine the initial and final density (# /0.25 m^2) of *Solenopsis* colonies, I collected ant colonies from the litter in six 0.25 m^2 quadrates using a stratified sampling scheme (one to two samples from each side of the plot) both before and after the experiment. Colonies were collected before the experiment from the 1 m wide strip surrounding the plots while colonies collected after the experiment were collected from within the plots themselves. Ant colonies

were found by carefully searching through the litter by hand as described by Kaspari (1993). Twigs, nuts and leaves containing ant colonies were collected and stored in 70% ethanol.

I monitored ant foraging activity during the manipulation period with pitfall traps. Pitfall traps consisted of a film roll canister (opening width= 32 mm) containing 70% ethanol. Traps were inserted into plastic tubes that had been sunk into the ground. I placed two rows of five pitfall traps approximately 0.5 m away from the edge of the enclosure and approximately 1 m apart. Within each row the traps were separated by an approximate distance of 0.75 m. I began trapping 1 week after traps were placed into the ground to avoid the "digging-in" effect (Greenslade 1973). Traps were only open for 24 hrs one day per week to minimize negative affects on ant population density. I sampled weekly for the duration of the study period. Arthropods collected in pitfall traps were placed in vials containing 70% ethanol. Two indices of the foraging activity of *Solenopsis* ants were derived from pitfall trap data. First, the number of individual *Solenopsis* ants captured on each plot each week represented an index of the number of foraging *Solenopsis* ants. Second, the proportion of traps capturing *Solenopsis* ants represented an index of

the foraging area covered by *Solenopsis* ants.

During the study period, eleven of the 700 traps filled with water. When this occurred, I used the mean value for the number of *Solenopsis* ants captured in other traps on the same plot during the same week and I excluded filled traps in the calculation of the proportion of traps capturing *Solenopsis* ants on a plot. No more than two traps were found filled on a given plot during a given week.

Egg mortality

Eggs were collected from 43 female *A. limifrons* housed in a screened building. Females were caged singly (33 individuals) and in pairs (10 individuals). Cages contained a single potted plant. The soil provided a substrate for eggs to be laid and the plant served as a perch site. I watered the cages twice daily and fed the lizards 6-7 days per week with grasshoppers and moths captured with a sweep net and with crickets that were purchased commercially. Cages were checked weekly for newly laid eggs. Because I did not want hatchlings to escape, I placed all eggs into small nylon mesh bags (mesh diameter=5 mm) before they were put in the plots. The mesh was large enough to allow predators in the litter access to eggs but small enough to prevent hatchlings from escaping.

Ten newly laid eggs were uniformly distributed (@ 1 egg/m^2) in each plot on the ground under the litter, the typical nest site of *A. limifrons* (Andrews 1988). Most, if not all, eggs on a plot would have been produced by different females because individual females lay approximately one egg per week. Eggs were placed by marked flags and checked every two days until five weeks had elapsed. The incubation time of *A. limifrons* eggs is 6 weeks (Andrews and Sexton 1981). Eggs were at most 1 week old when they were collected from the cages and placed into the field. Thus, five more weeks in the field should have allowed sufficient time for most eggs to hatch. At each check, eggs were recorded as present, hatched or predated. I assigned the date of death for predated eggs to the day that the egg was checked. This is the maximum time to predation (hereafter referred to as life span) because the egg may have been attacked at any time during the two day interval between checks. Only four of the eggs hatched before day 35, and these all hatched on day 32. These eggs were classified as having survived to day 35 for subsequent analyses.

To determine the mortality of eggs in the absence of invertebrate predators, I monitored 10 eggs on one HW plot and 10 eggs on one LW plot. I cut the bottom out of plastic

cups and partially buried them to prevent migration of invertebrates under the cup. A 5 cm wide strip around the top outside portion of the cup was painted with Fluon to prevent invertebrates in the litter from crawling into the cup. One egg was placed on the soil in each cup and covered with litter from which all invertebrates were removed. I checked the status of these eggs in the same manner as I checked the experimental eggs, except that these eggs were left in the field until they hatched.

Abiotic measurements

Soil moisture and litter moisture were monitored for the entire study period. Samples were collected after each pitfall trapping session. I took a 2.5 cm wide core sample from the top 5 cm of the **A** soil horizon from each plot per week. This sample was weighed wet and again after it was dried at 60 °C to a constant weight (±0.001 g). Soil moisture was calculated as mass lost divided by the wet mass of the sample. I also collected approximately 15-20 g of litter through the litter profile from each plot each week. Litter was weighed wet and again after it was dried at 60 °C to a constant weight (±0.001 g). Litter moisture was then calculated as mass lost divided by the wet mass of the sample.

To quantify the amount of litter on plots I weighed the litter from Berlese samples collected at the start and conclusion of the manipulation period. Litter was weighed ((± 0.1 g) after Berlese extraction.

Data analysis

To determine if the data sets met the assumptions of parametric analyses, I checked distributions for normality (both skewness and kurtosis) and homogeneity of variances. I pooled observations for each data set within each treatment across times and plots to have sufficient sample sizes for realistic tests. Normality was evaluated by comparing the values of g_1 (skewness) and g_2 (kurtosis) to the null hypothesis that both g_1 and g_2 are equal to 0. F-tests were used to determine if variances were homogeneous. The assumption of normality and homogeneous variances for a data set was rejected when $p<0.025$ because ANOVA's are robust to wide deviations with respect to these parameters (Sokal and Rohlf 1969, Ferguson 1976). A $\log_{10} + 1$ transformation was performed on data sets that had significant deviations from normality or non-homogeneous variances. Unless otherwise mentioned, data sets used in analyses were normal and variances were homogeneous (Appendix 1).

Data from RF plots were not included for statistical analyses because they would have created an unbalanced experimental design (5 replicates per HW and LW compared to 2 replicates per RF). Severely unbalanced data sets reduce the power of statistical tests and can therefore obscure the effect of the experimental manipulation (Shaw and Mitchell-Olds 1993). Data from RF plots are presented for relative comparisons; I expected values intermediate to those obtained from HW and LW plots.

Three of the data sets represented observations made only at the start and conclusion of the manipulation period: litter mass and density of *Solenopsis* ants and colonies. I used a repeated measures nested analysis of variance to compare treatments and treatments through time. Litter mass and the density of *Solenopsis* ants and colonies were the dependent variables (6 replicates/plot), time was the start and conclusion of the manipulation period, and treatment was the class variable. The five plots were nested within treatments.

Four of the data sets represented observations made during each week of the study period: percent litter moisture, soil moisture, number of foraging *Solenopsis* ants, and foraging area of *Solenopsis* ants. I used ANOVA's to determine if these variables differed between the treatments

during the pre-manipulation period. Litter moisture, soil moisture, number of foraging *Solenopsis* ants or the foraging area on each plot were the dependent variables (5 replicates/treatment) and treatment was the class variable. The number of foraging *Solenopsis* ants was the mean number captured on each plot during weeks 1 and 2. The foraging area was the mean of the foraging areas for each plot during weeks 1 and 2. Values of litter and soil moisture on each plot for the pre-manipulation period represent week 2 because I did not collect data on soil or litter moisture during the first week.

To determine if watering during the manipulation period influenced litter moisture, soil moisture, number of foraging *Solenopsis* ants, or the foraging area of *Solenopsis* ants, I used repeated measures analyses of variance. Litter moisture, soil moisture, number of foragers or the foraging area of *Solenopsis* ants during each week of the manipulation period (with no replication per plot) were the dependent variables (5 replicates/treatment/week) and treatment was the class variable.

To compare the life span of *A. limifrons* eggs during the manipulation period on HW and LW plots I used a nested analysis of variance. Life span was the dependent variable, treatment was the class variable, and plot was the nested

variable.

The cumulative percent mortality of *A. limifrons* eggs through time on HW and LW plots was compared with a Kolmogrov-Smirnov two-sample test. The distribution of percent mortality through time was calculated for each treatment by determining the proportion of predated eggs in all plots within a treatment during each two day period. I also performed two exponential regressions on the times series of the cumulative percent mortality for each treatment. Model 1 only considered the slope as a parameter:

$$m = 1 - e^{-st} \tag{6}$$

where m=proportion of eggs predated

s=rate of change in the proportion of mortality with time

t=time measured in days

The second model included both slope and the maximum asymptote (max) as parameters:

$$m = \max(1 - e^{-st}) \tag{7}$$

The proportion of egg mortality for a cohort of eggs during their 42 day incubation period was extrapolated from these

regressions.

RESULTS

Abiotic variables

During the pre-manipulation period, soil moisture did not differ between HW plots (mean=40.6 ± 1.9%) and LW plots (mean=39.2 ± 1.8%)(ANOVA $F_{1,8}$=0.32 $p>0.05$). During the manipulation period, soil moisture did not differ between HW plots (mean range=38.2 to 44.9%) and LW plots (mean range=36.6 to 42.0%), but soil moisture increased in both treatments through time (Table 1, Figure 3).

During the pre-manipulation period, litter moisture did not differ between HW plots (mean=44.9 ± 0.96%) and LW plots (mean=43.3 ± 0.58%)(ANOVA $F_{1,8}$=2.02 $p>0.05$). During the manipulation period, however, litter moisture was higher on HW plots (range of means=46.5 to 57.4%) than on LW plots (range of means=38.3 to 42.3%)(Table 2, Figure 4). Litter moisture did not vary as a function of time on either treatment.

Litter mass did not differ between treatments or between the start and conclusion of the manipulation period. However, a significant plot effect indicated that, within treatments, the plots differed from one another (Table 3,

Figure 5).

Ant density and foraging activity

The density of individual *Solenopsis* ants did not differ between HW and LW plots or between the start and conclusion of the manipulation period. However, a significant plot effect indicates that, within treatments, plots differed from one another (Table 4, Figure 6).

Because I detected significant plot effects for both litter mass and density of *Solenopsis* ants, I regressed the density of *Solenopsis* ants on the mean litter mass to determine if the density of *Solenopsis* ants was related to litter mass. Data for both litter mass and the density of *Solenopsis* ants on each plot, were the plot means of the values at the start and conclusion of the manipulation period. Regressions were performed for HW and LW treatments separately. In both cases, the density of *Solenopsis* ants was independent of litter mass (HW $F_{1,3}=0.01$ $p>0.05$; LW $F_{1,3}=0.28$ $p>0.05$).

Distributions of the density of *Solenopsis* ant colonies did not satisfy parametric assumptions (Appendix 1). Therefore, further comparisons were made with Mann-Whitney U-tests. Treatments did not differ in the density of *Solenopsis* ant colonies at either the start ($U_{5,5}=7.00$

p>0.05) or the conclusion ($(U_{5,5}$=21.50 p>0.05) of the experiment. To determine if the density of colonies changed overall during the experiment, I pooled data from both treatments within times (start and conclusion). The density of *Solenopsis* ant colonies increased during the manipulation period ($U_{10,10}$=17.50 p=0.01) (Figure 7).

Because the density of *Solenopsis* ant colonies increased during the manipulation period and there was no change in the density of *Solenopsis* ants, I tested the hypothesis that the number of *Solenopsis* ants per colony decreased during the manipulation period. This hypothesis was tested with a Mann-Whitney U-test; time was the class variable and the mean number of *Solenopsis* ants per colony for each plot was the dependent variable. Because treatments did not differ in either the density of *Solenopsis* ants or in the density of *Solenopsis* ant colonies, I pooled data from both treatments within time (start and conclusion). The number of *Solenopsis* ants per colony did not change from the start (54.9 ± 16.6) to the conclusion (34.8 ± 6.4) of the manipulation period ($U_{6,10}$=39.00 p=0.33).

During the pre-manipulation period, the number of foraging *Solenopsis* ants captured was lower on HW plots (mean=0.5 ± 0.39 individuals) than LW plots (mean=2.8 ± 0.67

individuals) (ANOVA $F_{1,8}$=8.67 p=0.019). The number of foraging *Solenopsis* ants during the manipulation period was transformed (log_{10}+1) to better satisfy the assumptions of normality and homogeneity of variances (Appendix 1). During the manipulation period, the weekly number of foragers did not differ between HW plots (range of means=1 to 13 foragers) and LW plots (range of means=0 to 20 foragers) or among weeks (Table 5, Figure 8).

During the pre-manipulation period, the mean foraging area of *Solenopsis* ants on HW plots (mean=5.0 ± 3.9 %) was less than the mean foraging area of *Solenopsis* on LW plots (mean=22.7 ± 4.8%) (ANOVA $F_{1,8}$=8.27 p=0.021). During the manipulation period, there was no difference in the foraging area of *Solenopsis* ants between HW plots (mean range=18.0 to 35.2%) and LW plots (mean range=27.0 to 42.0%) or among weeks (Table 6, Figure 9).

Egg mortality

During the entire duration of the study, no eggs were missing or killed by mortality agents other than *Solenopsis* ants. All of the eggs enclosed in plastic cups on HW and LW plots hatched. The mean time to hatching of eggs placed in plastic cups on the HW plot was 37.2 ± 0.6 days and 38.2 ± 0.6 days on the LW plot (T-test p=0.269).

The life span of eggs was lower on HW plots (range of plot means= 6.6 to 21.7 days) than on LW plots (range of plot means=17.8 to 30.8 days; Nested ANOVA treatment effect, $F_{1,90}$=34.3 p=0.001)(Figure 10). In addition, the life span of eggs was heterogenous among plots within treatments (Nested ANOVA nested plot effect $F_{8,90}$=2.02 p=0.05). Thus, to determine if variation in the amount of litter mass or the density of *Solenopsis* ants among plots influenced the life span of eggs within treatments, I used ANCOVA's in which treatments were the class variable, life span was the dependent variable, and litter mass and the density of *Solenopsis* ants were covariates. Values for litter mass and the density of *Solenopsis* ants were the means from the data collected at the start and conclusion of the manipulation period for each plot.

The life span of eggs was not related to the density of *Solenopsis* ants ($F_{1,7}$=0.05 p>0.05). However, the life span was greater on LW than on HW plots ($F_{1,7}$=7.54 p=0.03). The interaction between *Solenopsis* ant density and treatment was not significant ($F_{1,6}$=0.10 p>0.05).

The life span of eggs was negatively related to litter mass ($F_{1,7}$=34.96 p=0.001) (Figure 11) and the life span of eggs was greater on LW than on HW plots ($F_{1,7}$=57.61 p=0.001). The interaction between litter mass and treatment was not

significant ($F_{1,6}$=1.65 p>0.05).

Cumulative percent mortality differed between HW and LW plots (Kolmogrov-Smirnov D=0.813 n_1=16 n_2=16 p=0.001)(Figure 12). Exponential regressions of time on cumulative percent mortality with slope as a parameter indicate that the rate of mortality occurred 3 times faster on HW than on LW plots (Table 7). Model 1 (eq. 6) regressions are significant for both HW (F=1518.6 $F_{1,15[0.001]}$=16.6, R^2=0.875) and LW plots (F=537.0 $F_{1,15[0.001]}$=16.6, R^2=0.914). Parameters of the model 2 (eq. 7) type could not be estimated for LW treatments because the parameter estimates would not converge. As a result, I substituted the maximum asymptote value obtained from the model 2 (eq. 7) regression of HW plots in the model 2 (eq. 7) regression for LW plots. This is a fair assumption to make because it merely stipulates that the maximum proportion of eggs predated upon in both treatments is 1.0. When this modification is made, the model 2 (eq. 7) regressions indicate that the rate of mortality was 5 times faster on HW than on LW plots. Model 2 (eq. 7) regressions are significant for HW (F=1808.0 $F_{1,14[0.001]}$=11.8, R^2=0.960) and LW (F=531.33 $F_{1,15[0.001]}$=16.6, R^2=0.903) plots. The observed distribution and predicted values by each model are presented for HW plots and LW plots (Figure 13). Based upon the estimated parameters from each model, the cumulative

percent mortality of a cohort during the 42 day incubation period was higher on HW plots (model 1=95.7%, model 2=82.2%) than on LW plots (model 1=58.6%, model 2=56.1%).

DISCUSSION

My experiment confirmed the prediction that variation in the amount of moisture during the wet season would change egg mortality such that mortality would be higher under relatively wet than relatively dry conditions, and that mortality would be due to predation by *Solenopsis*. Egg mortality, projected over the six week incubation period, was approximately 50% greater on the HW treatment than on the LW treatment. Differences in mortality occurred early: over 60% of egg mortality occurred within the first 10 d on the HW treatment, while only 20% of the mortality on the LW treatment had occurred by this time. Moreover, all egg mortality was due to predation by *Solenopsis;* experimental eggs that were not killed by ants, survived to the end of the manipulation period (2-3 days short of the time eggs hatched in the ant-proof cups), and eggs that were protected from ants in the ant proof cups all survived to hatching. Thus, by manipulating the moisture content of the litter, the rate of predation on eggs by ants was altered as

predicted.

Can the spatial variation in egg mortality demonstrated here be extrapolated to a temporal scale? Because plots were surrounded by ant proof barriers, the changes in mortality were due to changes in the responses of ants in situ to eggs, not to immigration or emigration of ants. Thus, my experiments support the contention that annual variation in the density of *A. limifrons* is due to annual variation in the intensity of predation on egg by ants, and that this interaction is mediated by annual variation in the amount of wet season rainfall.

Mechanistic basis of variation in egg mortality

Contrary to what I expected, water manipulations apparently did not affect the density of *Solenopsis*. This indicates that differences in mortality between treatments were due to a functional response by ants to litter moisture. Thus, moisture did not directly alter the density of *Solenopsis* ants but rather it altered the rate of predation by *Solenopsis* ants on lizard eggs. Therefore, the interactions between litter moisture, ants, and lizard eggs are described by an interaction modification and not an interaction chain (Wootton 1993, 1994a,b).

The density of *Solenopsis* ants may not have changed in

response to the treatments for two reasons: 1) *Solenopsis* do not exhibit a numerical response to changing moisture conditions; 2) five weeks was not a long enough time for a numerical response by ants. For *Solenopsis invicta*, the time for an egg to develop into a worker ranges from 3 to 6.5 weeks, depending upon temperature (Hölldobler and Wilson 1990).

In contrast to the results of this study, the number of ants increased when water was added to 0.25 m^2 litter plots by Levings (1983) and Levings and Windsor (1984). However, their manipulations were conducted in the dry season, and their small plots were not enclosed. Thus, the increase that they observed may have been due to immigration onto their plots. In my study, movements in or out of the plots were precluded by ant-proof barriers.

Water manipulations also did not influence the number of foraging *Solenopsis* ants or the amount of area foraged by *Solenopsis* ants. This means that pitfall traps either did not sample ants in proportion to their foraging activity or that eggs became more attractive to, or easier to find by, ants under conditions of high litter moisture. Eggs with parchment type shells (like those of *A. limifrons*) readily absorb water from nest sites (Tracy 1980, Andrews and Sexton 1981), and water uptake is associated with the expansion of

eggs and the thinning of their shells. Moreover, for crocodilian eggs, at least, egg shell thickness decreases and porosity increases as the result of the biochemical activities of soil microbes (Ferguson 1981). Thus, *Solenopsis* ants may have been more successful in attacking eggs on the HW treatment than on LW treatment if eggs were thinner or more porous or if increased levels of moisture promote the release of chemicals from eggs into the environment. The negative relationship between the mass of litter on the plots and egg mortality further suggests that moisture per se affects the ability of *Solenopsis* to find and attack eggs; litter mass may influence the survivorship of eggs by maintaining moisture in the litter.

An alternative to the above mechanism, is that changes in litter moisture may affect the abundance or activity patterns of prey other than eggs, predators, or competitors. For example, the number of individuals of non-ant arthropods was higher on unwatered plots than watered plots (Levings and Windsor 1984). Thus, under relatively dry conditions *Solenopsis* ants may prey upon non-ant arthropods rather than lizard eggs because: 1) the non-ant arthropods are more abundant than lizard eggs; or 2) non-ant arthropods are more attractive to ants than eggs. However, all these possible mechanisms are highly speculative because little is known

about the interactions between *Solenopsis* ants, other litter invertebrates, and lizard eggs.

The number of colonies increased on both treatments during the manipulation period. How could the density of *Solenopsis* ant colonies have increased while the density of individual *Solenopsis* ants remained the same? Kaspari (in press) found that *Solenopsis* ants on Barro Colorado Island are polydomous, meaning that satellite colonies can bud from a central colony. Such budding may increase the foraging area of the colony (Hölldobler and Wilson 1990). Satellite colonies are initially founded by worker ants and are typically queenless. Thus, in my study, budding may have increased the number of colonies while the overall density remained constant.

Variable interaction strengths and population models

As this study demonstrates, the influence of rainfall on the interaction between *A. limifrons* and *Solenopsis* ants indicates the importance of interaction modifications on population dynamics. This supports the idea that indirect effects are an important component of community interactions, and that theoretical models that do not consider interaction modifications may not realistically predict population dynamics (Neill 1974, Billick and Case

1994, Karieva 1994).

Typically, models that consider the effect of environmental variability on the growth of a population, do so by incorporating "white noise" into the model. "White noise" is a mathematical term for a variable that fluctuates randomly through time, but has a fixed variance and a mean value of 0. When May (1973b) added a "white noise" term (representing environmental variability) to his models, the predicted population dynamics changed. Models that did not account for "white noise" predicted that the density of populations within a community would converge to a stable or unstable equilibrium point. Models incorporating white noise predicted that: 1) the density of a population would fluctuate through time but some densities will be more probable than others (probability distribution of densities rather than a stable equilibrium point); and 2) the probability distribution of densities has a wider range of densities as environmental variation increases. However, the actual mechanisms through which environmental variation affects population dynamics are poorly understood. My study demonstrated that natural variation in a physical factor, rainfall, modified the interaction between a predator, *Solenopsis* ants, and a prey species, *A. limifrons*. Variation in the amount of rainfall, in this case, may

produce parallel variation in the population density of this lizard through its affect on the rate of predation by ants on lizard eggs.

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FIGURES

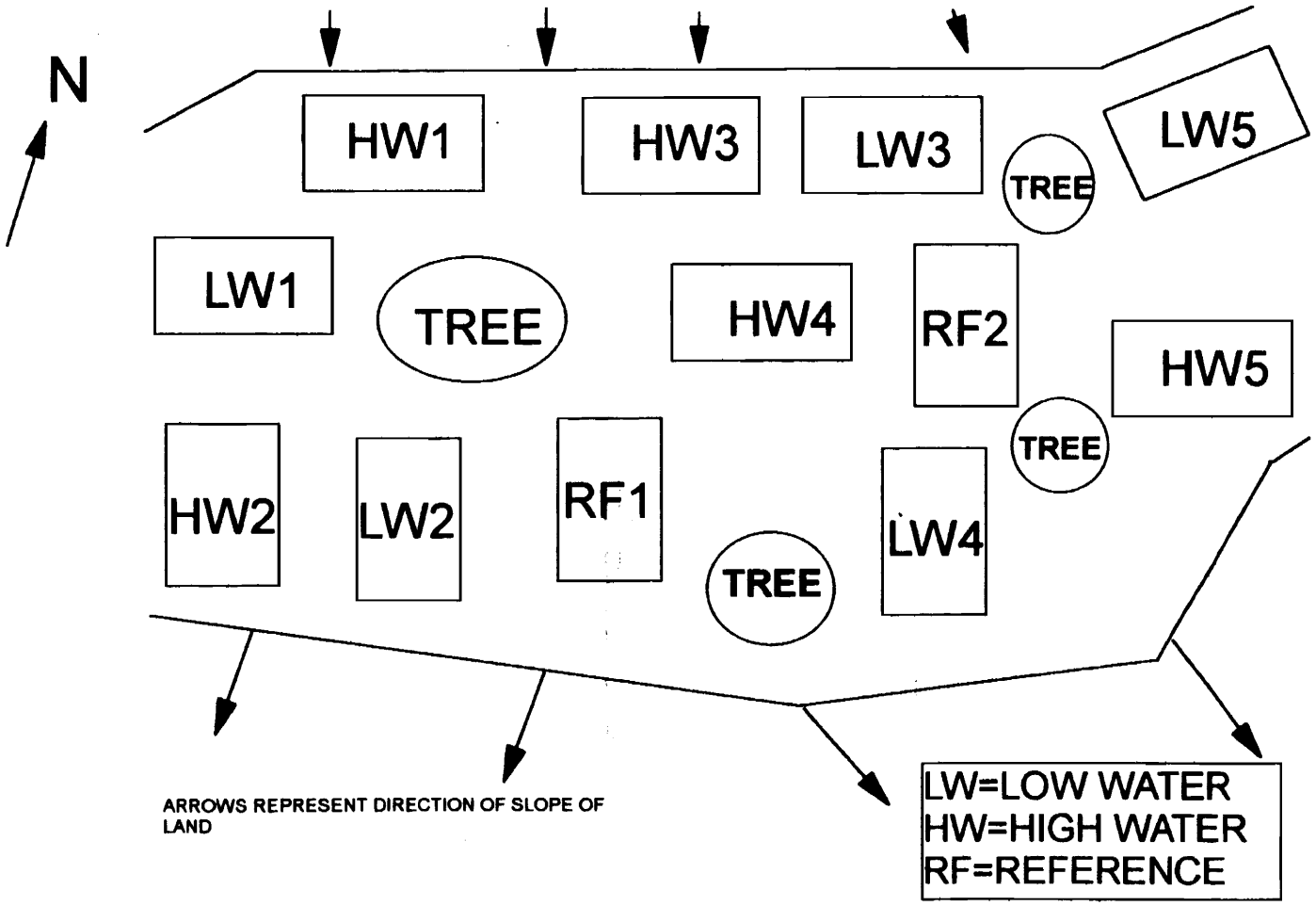

Fig. 1. Map of the study plots in the Allee creek watershed. Plots are 2 x 5 m in width and length.

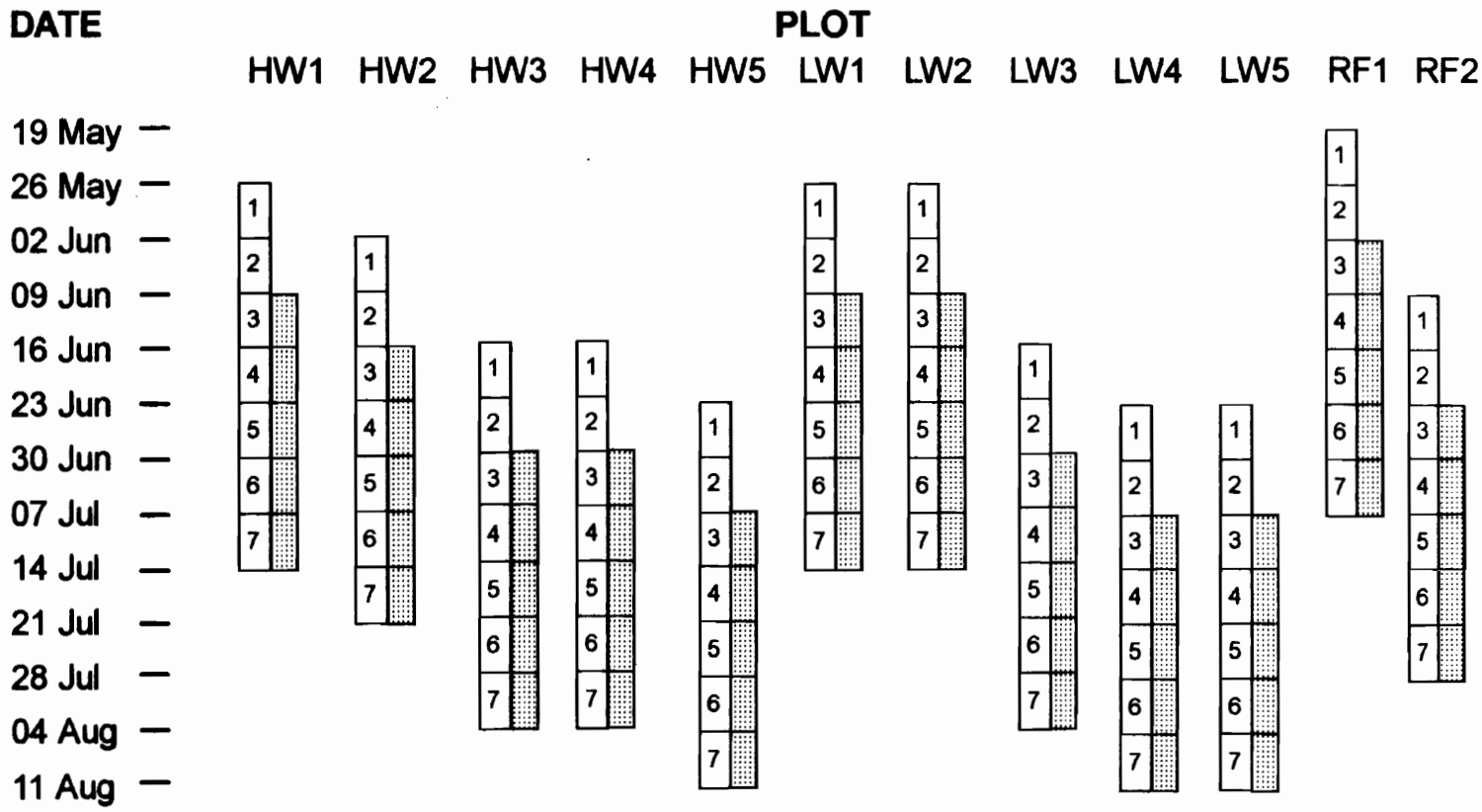

Fig. 2. Sequence of events on each plot. ☐ =Study period (pitfall trapping, soil and litter moisture measurements). ▨ =Manipulation period (watering and monitoring of egg predation). Tarps were raised, the enclosures created, initial litter and colony collections were made, and eggs were placed on plots at the start of the manipulation period. Final litter and colony collections were made at the conclusion of the manipulation period. The divisions within the study period represent the seven weeks of study.

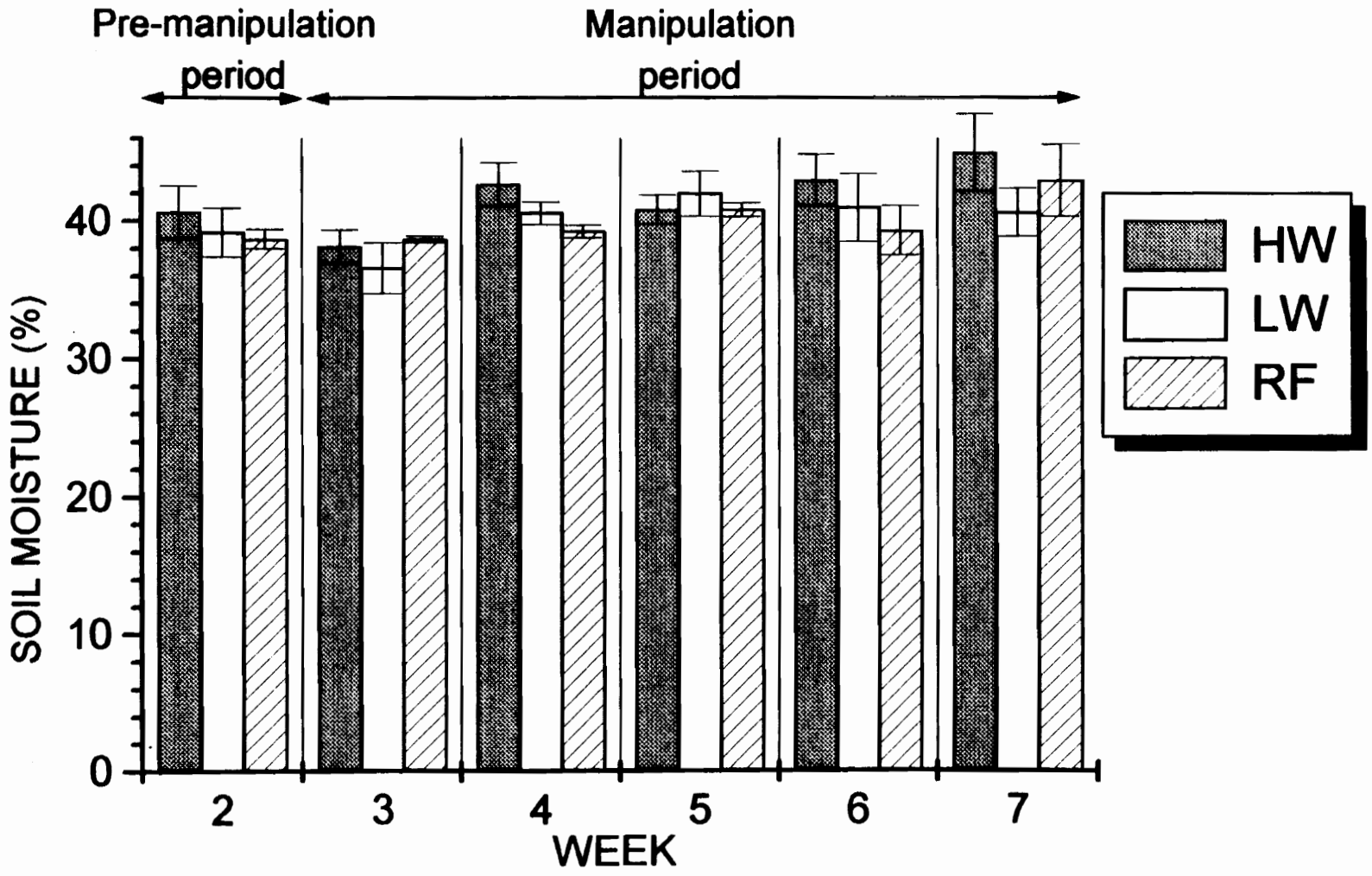

Fig. 3. Mean weekly soil moisture on HW, LW and RF treatments (± SE). Treatments did not differ in either period.

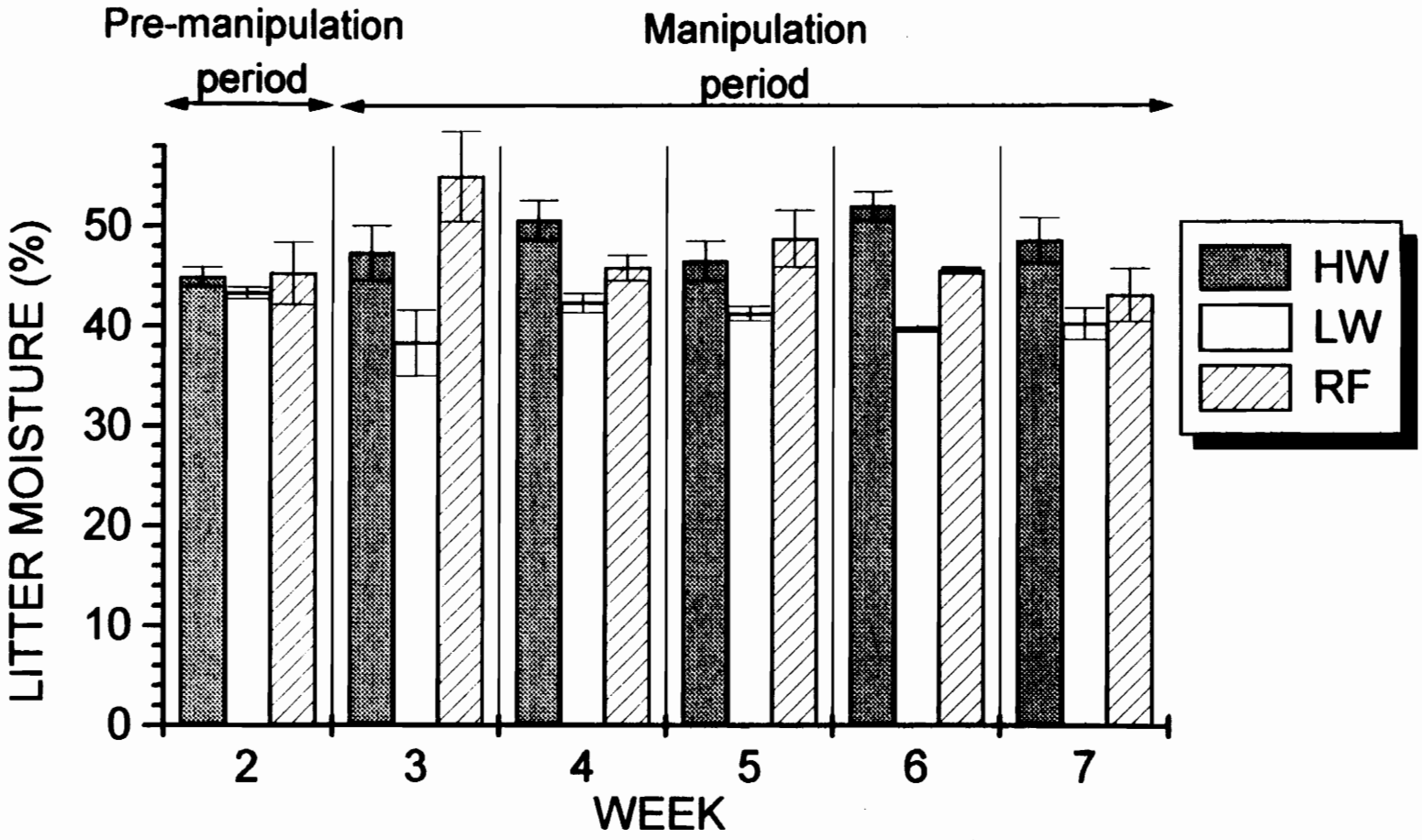

Fig. 4. Mean weekly litter moisture on HW, LW and RF treatments (± SE). Treatments did not differ during the pre-manipulation period. During the manipulation period, the HW treatment had greater litter moisture than the LW treatment.

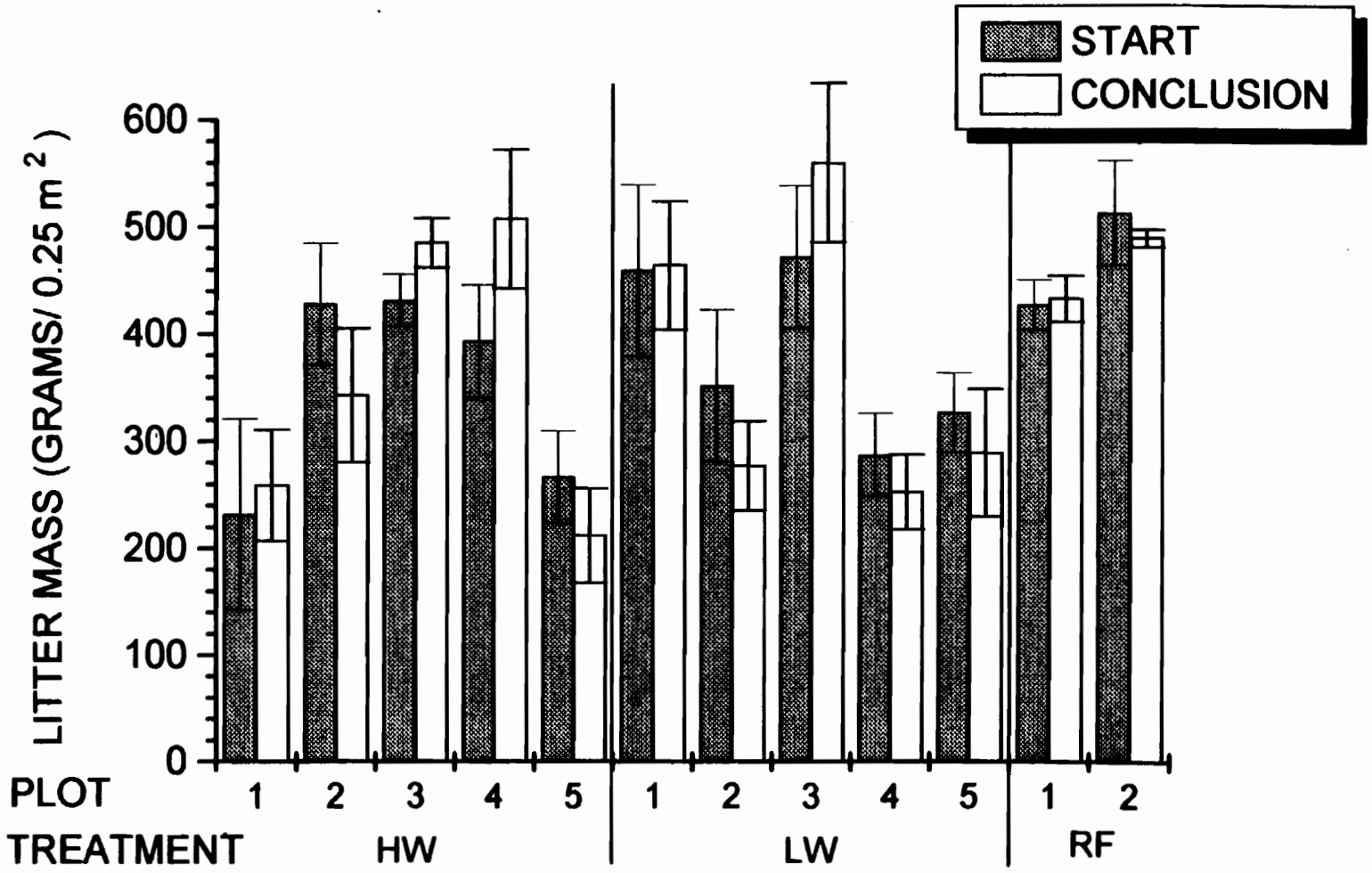

Fig. 5. Mean litter mass on LW, HW and RF treatments at the start and conclusion of the experiment (± SE). Treatments did not differ in litter mass at either time.

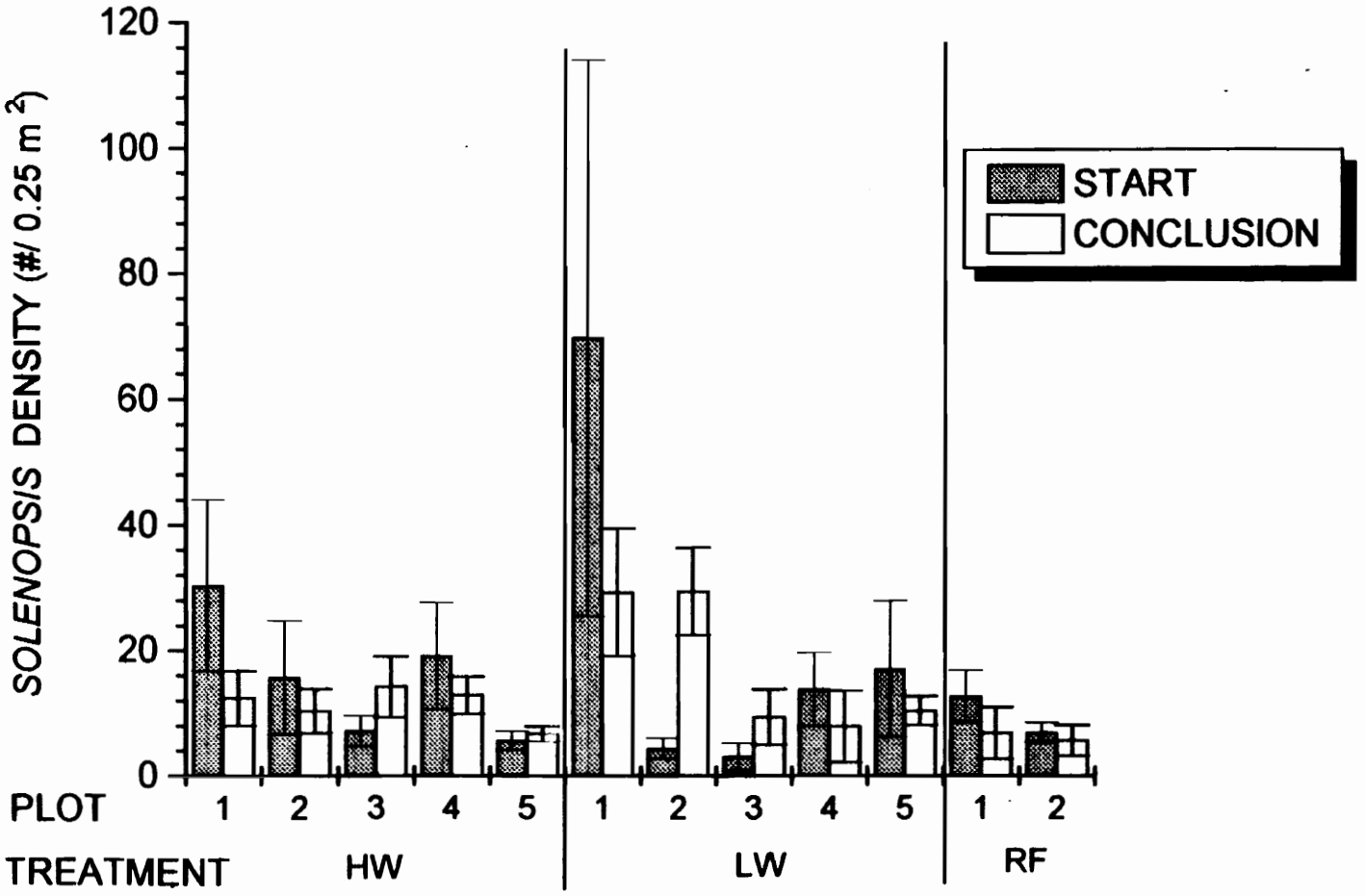

Fig. 6. *Solenopsis* density on LW, HW and RF treatments at the start and conclusion of the experiment (± SE). Treatments did not differ in *Solenopsis* density at either time.

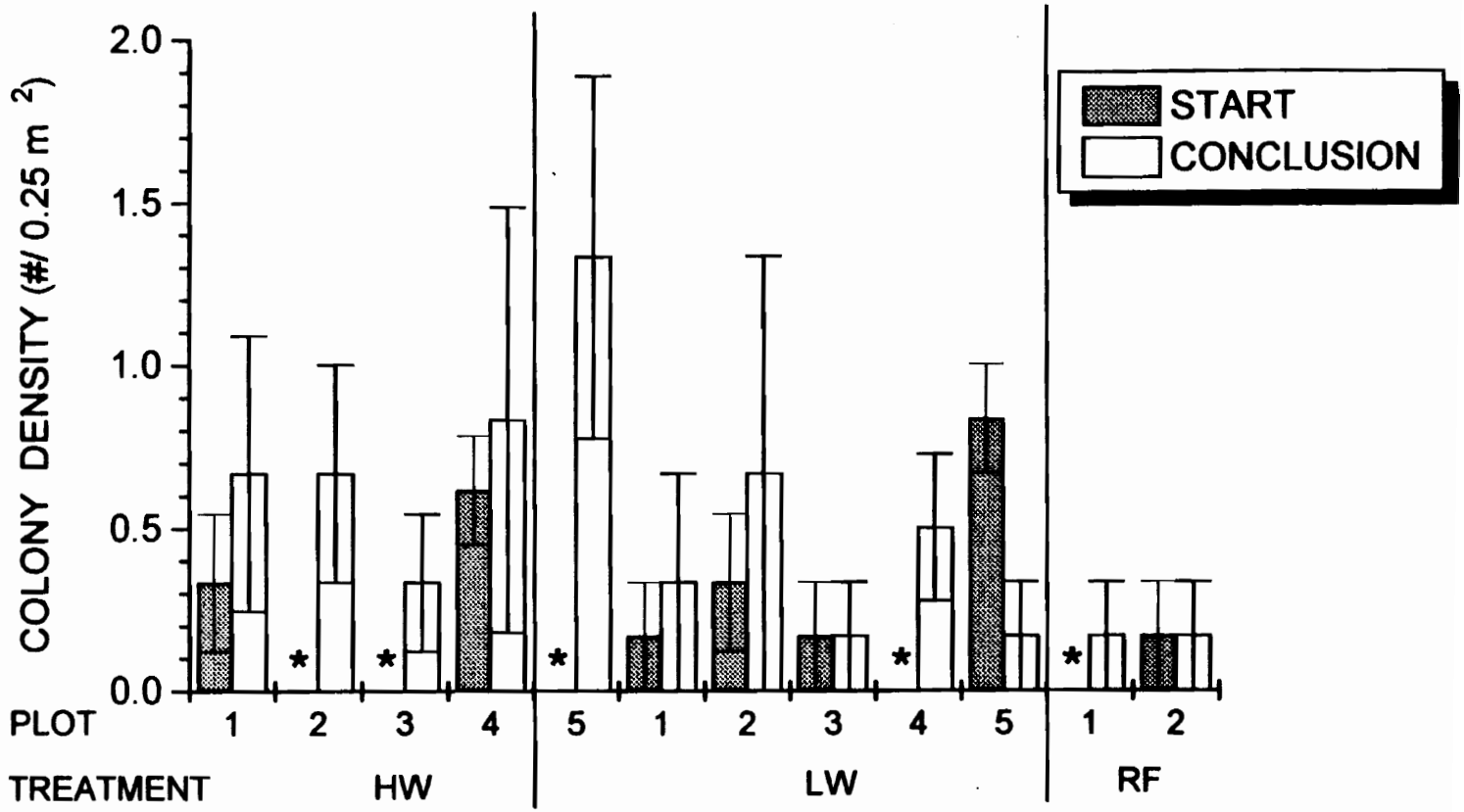

Fig. 7. *Solenopsis* colony density on LW, HW and RF treatments at the start and conclusion of the experiment (± SE). * indicates that no colonies were found. Treatments did not differ in the density of *Solenopsis* colonies at either time but the density of colonies was higher at the conclusion of the manipulation period.

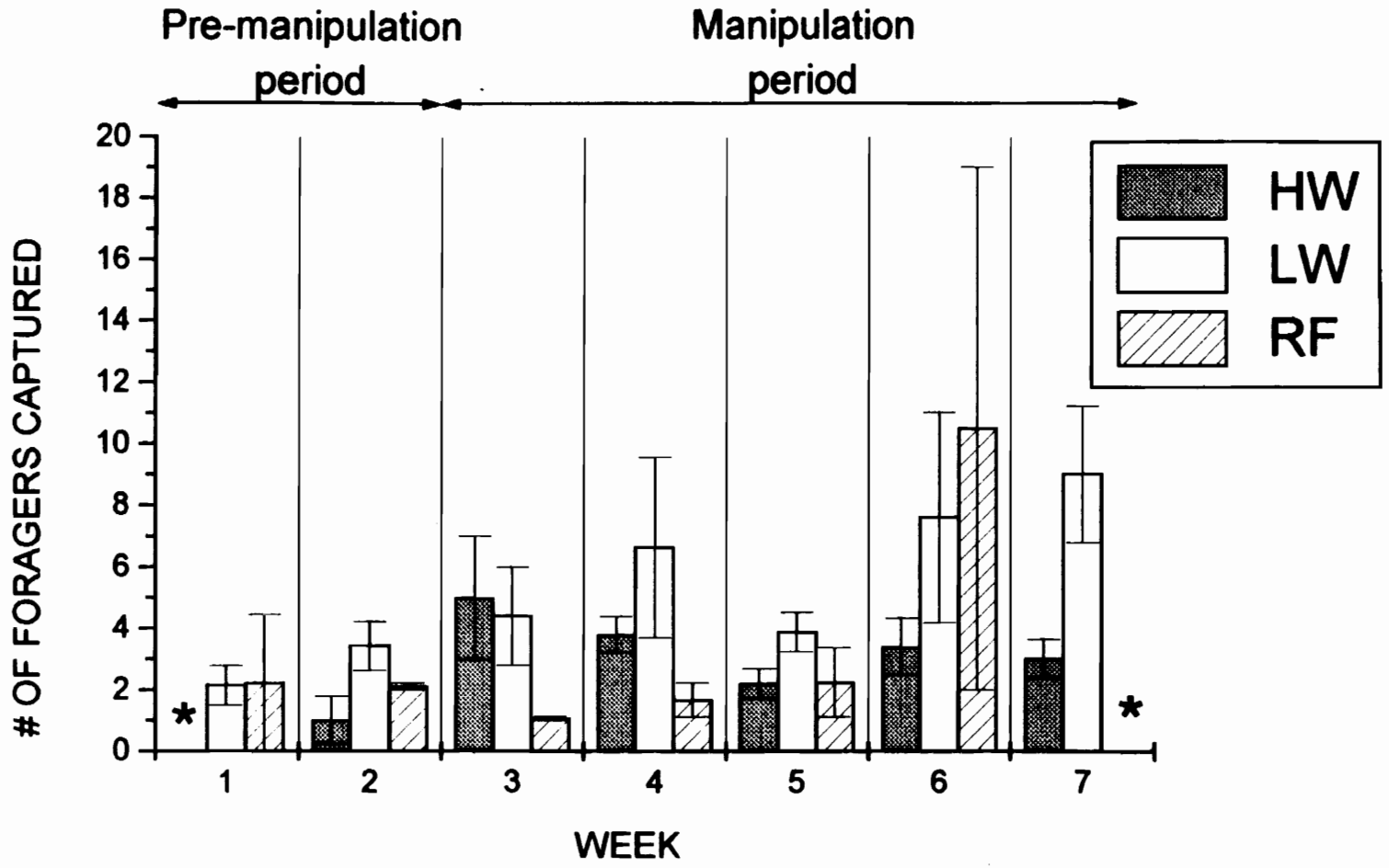

Fig. 8. Number of foraging *Solenopsis* ants captured on LW, HW and RF treatments (± SE). * indicates that 0 ants were captured. The number of foraging *Solenopsis* ants was greater on the LW treatment during the pre-manipulation period. During the manipulation period the number of foraging *Solenopsis* ants did not differ between treatments.

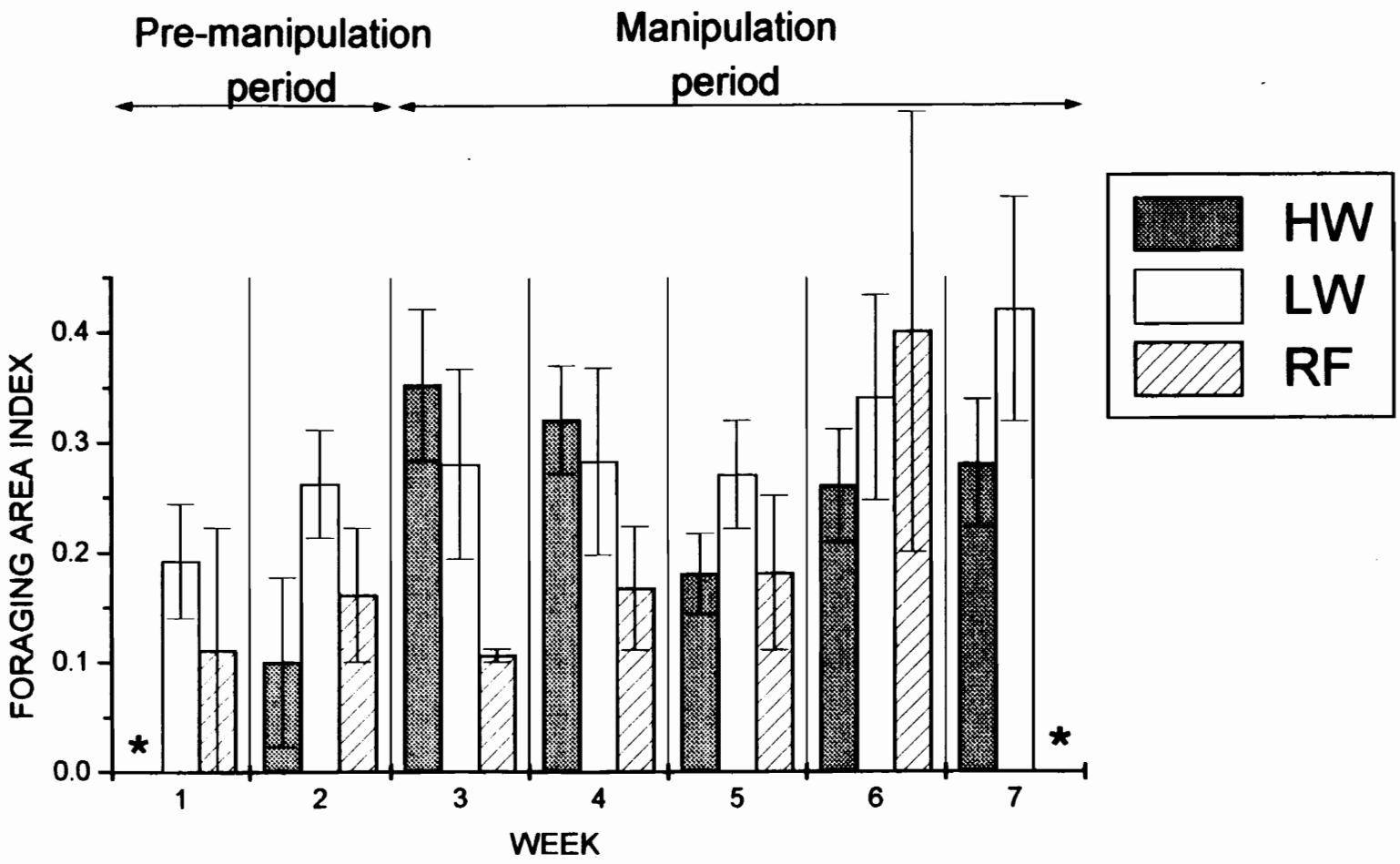

Fig. 9. Foraging area of *Solenopsis* ants on LW, HW and RF treatments (± SE). * indicates that 0 ants were captured. The foraging area of *Solenopsis* ants was greater on the LW treatment during the pre-manipulation period. During the manipulation period foraging area did not differ between treatments.

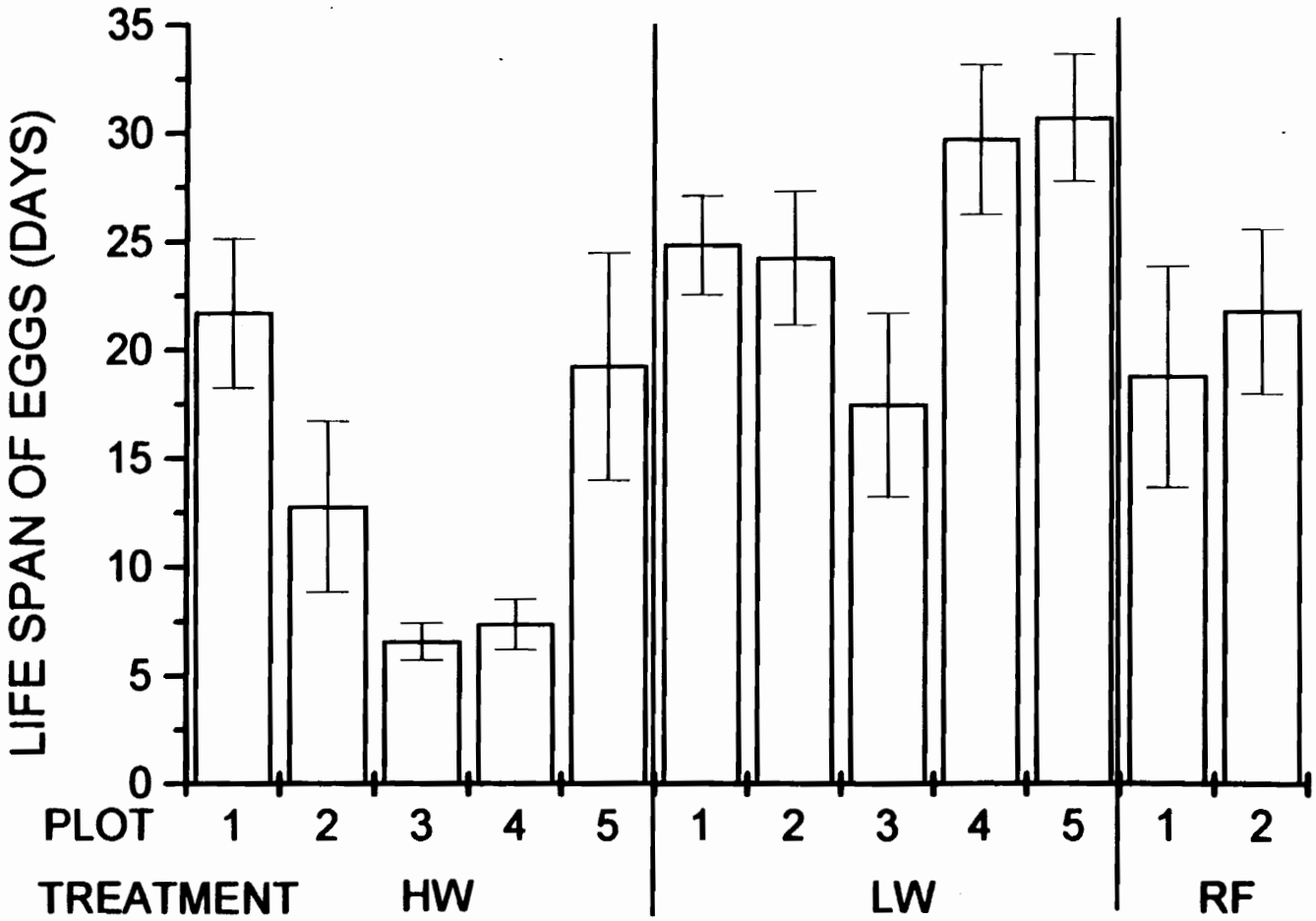

Fig. 10. Mean life span of *A. limifrons* eggs on HW, LW and RF treatments (± SE). The life span of *A. limifrons* eggs was shorter on the HW treatment than onthe LW treatment.

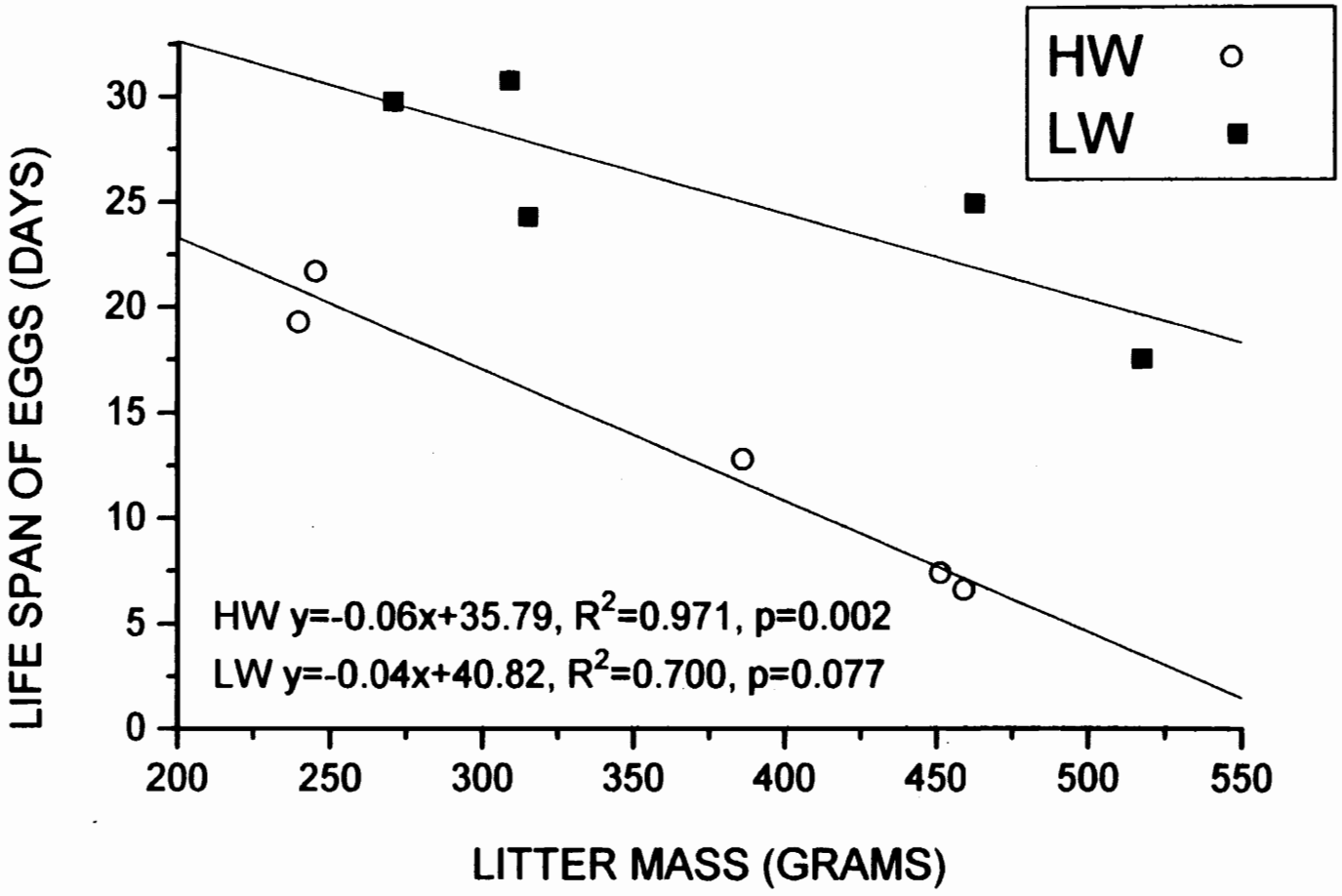

Fig. 11. Regression of the life span of *A. limifrons* eggs on HW and LW treatments as a function of litter mass.

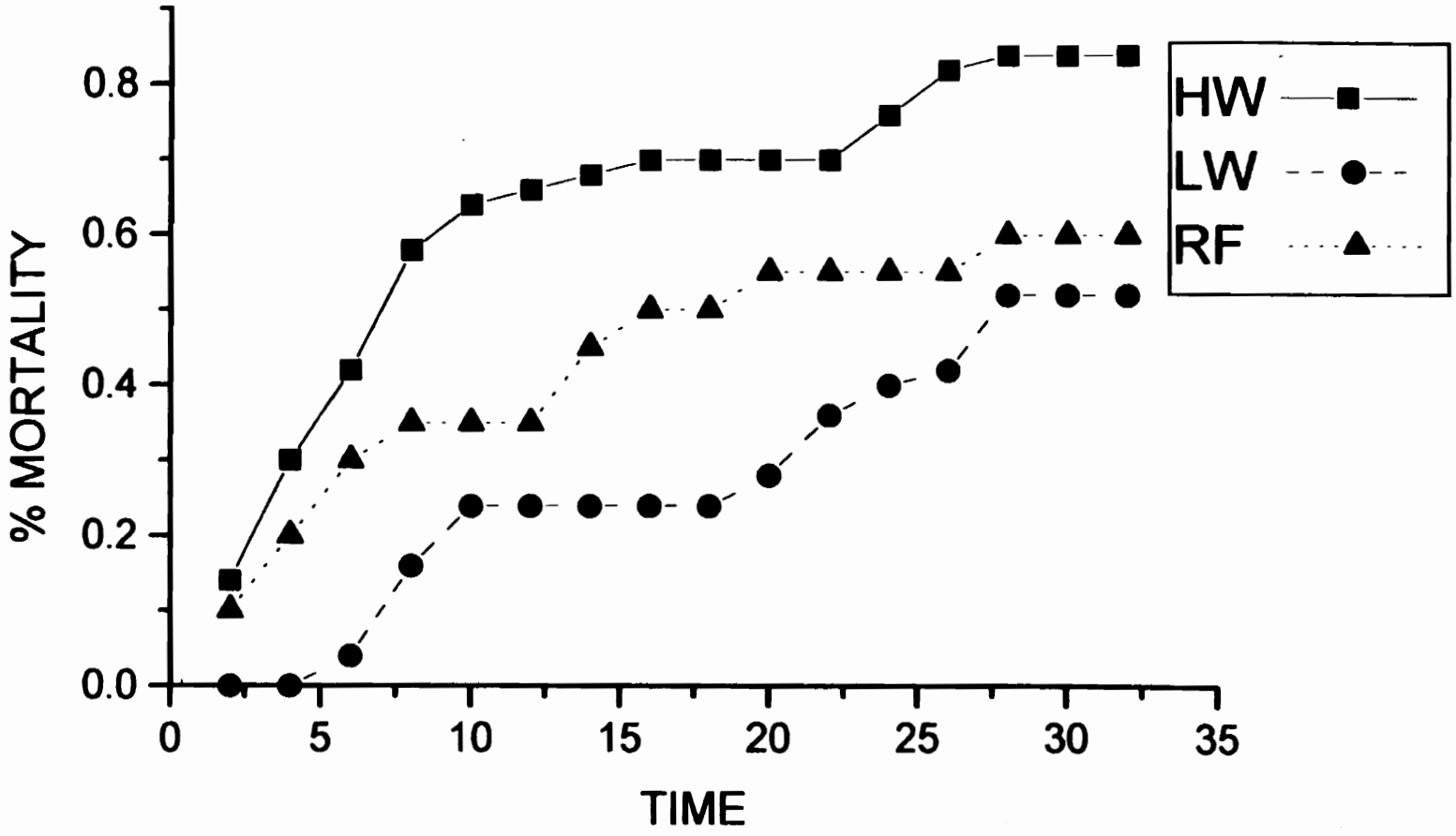

Fig. 12. Cumulative mortality of *A. limifrons* eggs during the incubation period on LW, HW and RF treatments.

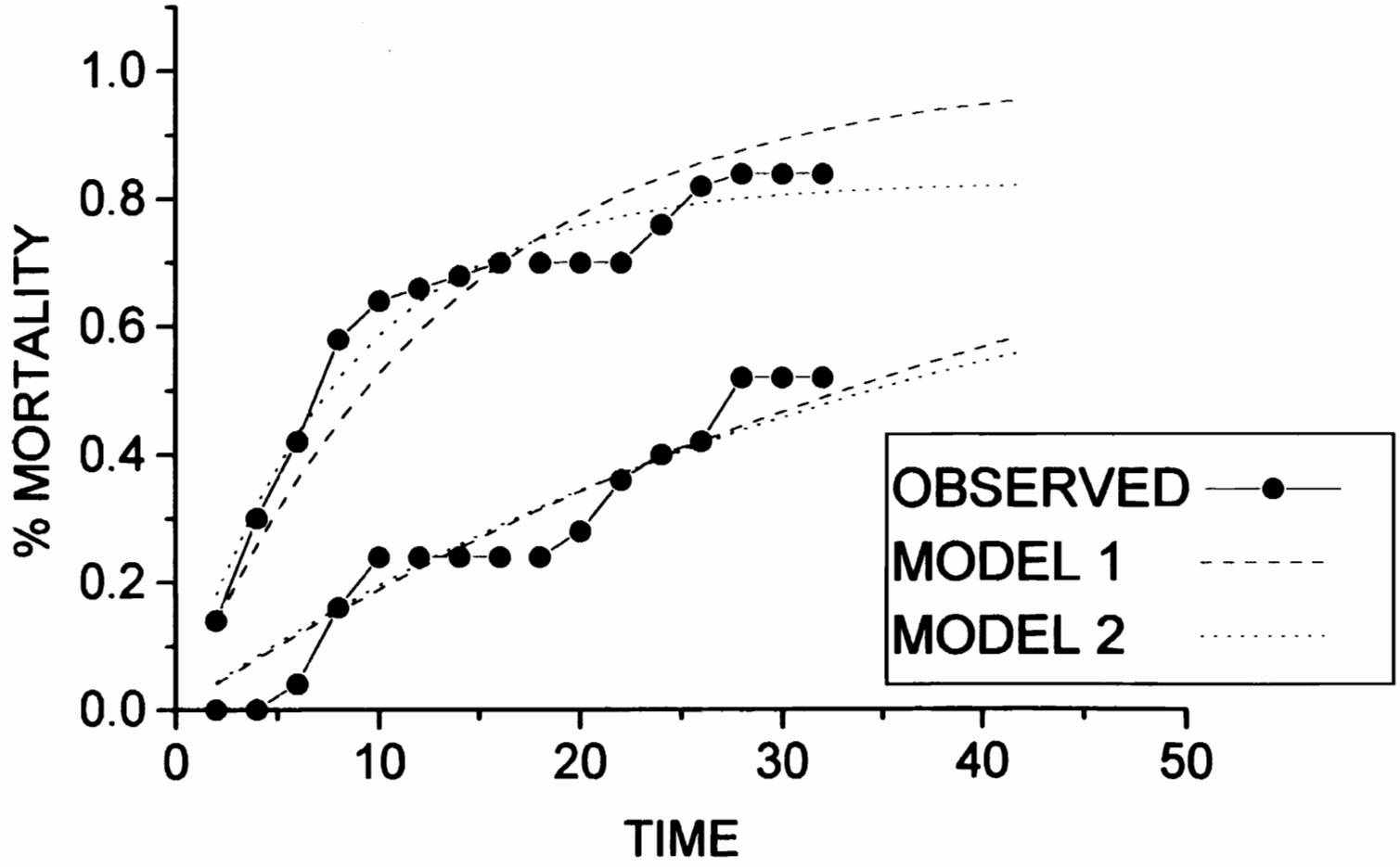

Fig. 13. Observed and simulated projections of cumulative mortality of *A. limifrons* eggs during the 42 day incubation period on the HW and LW treatments. Simulated projections are based on two types of exponential models. R^2 for all models were > 0.88.

TABLES

Table 1. F-ratios and significance for repeated measures analysis of variance of percent soil moisture on HW and LW treatments during the manipulation period.

Source	df	F	P
Between Subjects			
Treatment	1,8	1.24	0.30
Within Subjects			
Time	4,32	3.65	0.02
Time x Treatment	4,32	0.79	0.54

Table 2. F-ratios and significance for repeated measures analysis of variance of percent litter moisture on HW and LW treatments during the manipulation period.

Source	df	F	P
Between Subjects			
Treatment	1,8	22.83	<0.001
Within Subjects			
Time	4,32	1.60	0.20
Time x Treatment	4,32	1.21	0.33

Table 3. F-ratios and significance for repeated measures analysis of variance of litter mass collected at the start and conclusion of the manipulation period on HW and LW treatments.

Source	df	F	P
Between Subjects			
Treatment	1,50	0.64	0.43
Plot(Treatment)	8,50	8.64	<0.001
Within Subjects			
Time	1,50	0.00	1.00
Time x Treatment	1,50	0.16	0.69
Time x Plot(Treatment)	8,50	0.70	0.69

Table 4. F-ratios and significance for repeated measures analysis of variance of the density of *Solenopsis* ants at the start and conclusion of the manipulation period on HW and LW treatments.

Source	df	F	P
Between Subjects			
Treatment	1,50	0.00	1.00
Plot(Treatment)	8,50	3.02	0.01
Within Subjects			
Time	1,50	2.07	0.16
Time x Treatment	1,50	0.90	0.35
Time x Plot(Treatment)	8,50	2.00	0.07

Table 5. F-ratios and significance for repeated measures analysis of variance of the number of foraging *Solenopsis* ants during the manipulation period on HW and LW treatments.

Source	df	F	P
Between Subjects			
Treatment	1,8	3.92	0.08
Within Subjects			
Time	4,32	0.70	0.60
Time x Treatment	4,32	1.03	0.41

Table 6. F-ratios and significance for repeated measures analysis of variance of the foraging area of *Solenopsis* ants during the manipulation period on HW and LW treatments.

Source	df	F	P
Between Subjects			
Treatment	1,8	0.64	0.45
Within Subjects			
Time	4,32	0.88	0.49
Time x Treatment	4,32	0.86	0.50

Table 7. Exponential regression models on the cumulative percent mortality of *A. limifrons* eggs as a function of time.

Model	Treatment	Parameter	Parameter Estimate	df	F	P	R-square
1	LW			1,15	537.00	<0.001	0.91
		Slope	0.02				
	HW			1,15	1518.60	<0.001	0.88
		Slope	0.08				
2	LW			1,15	531.33	<0.001	0.90
		Slope	0.03				
		Maximum	0.83				
	HW			2,14	1808.00	<0.001	0.91
		Slope	0.12				
		Maximum	0.83				

APPENDIX

Appendix 1. Comparisons of g_1 and g_2 of pooled data distributions with the normal distribution and comparisons of variances between treatments. A * denotes a significant deviation from homogeneous variances and normaility ($p < 0.025$).

Data	Transformation	Treatment	g1	g2	t for g1	t for g2	F-test	df
Pre-manipulation period								
Soil moisture	none	HW	0.62	-1.20	0.67	0.60	1.16	4,4
		LW	0.95	-0.33	1.04	0.17		
Litter moisture	none	HW	1.12	-0.60	1.23	0.15	2.75	4,4
		LW	0.45	-0.96	0.37	0.48		
# of foraging ants	none	HW	1.29	-0.83	1.42	0.42	3.00	4,4
		LW	0.23	-1.50	0.25	0.13		
Foraging area	none	HW	1.29	-0.83	1.42	0.42	1.38	4,4
		LW	0.20	-1.42	0.22	0.71		
Manipulation period								
Soil moisture	none	HW	0.73	0.55	0.76	0.76	1.14	24,24
		LW	-0.18	-0.14	0.19	0.19		
Litter moisture	none	HW	0.08	-0.62	0.08	0.64	1.62	24,24
		LW	-0.40	1.57	0.41	2.20		
# of foraging ants	none	HW	2.53	7.88	5.46 *	8.74 *	4.71 *	24,24
		LW	1.17	0.65	2.52 *	0.72		

Appendix 1 cont.)

Data	Transformation	Treatment	g1	g2	t for g1	t for g2	F-test	df
# of foraging ants	log (base 10)	HW	0.58	1.04	0.60	1.45	2.89 *	24,24
		LW	-0.24	-0.33	0.25	0.46		
Foraging area	none	HW	0.67	0.07	1.50	0.07	2.06	24,24
		LW	0.45	-0.65	0.96	0.72		
Maximum life span	none	HW	0.84	-0.89	2.31	1.35	1.17	49,49
		LW	-0.70	-1.10	1.92	1.66		
Pre- vs post- manipulation Solenopsis density	none	HW	2.93	10.14	9.51 *	16.67 *	4.60 *	59,59
		LW	4.42	23.82	14.33 *	39.17 *		
	log (base 10)	HW	-0.34	0.35	0.31	0.57	1.75	59,59
		LW	0.07	-0.59	0.22	0.96		
Colony density	none	HW	2.30	5.07	7.48 *	18.35 *	1.64	59,59
		LW	3.06	12.40	9.93 *	20.40 *		
	log (base 10)	HW	1.57	1.26	5.08 *	2.07	1.44	59,59
		LW	1.62	2.09	5.53 *	3.43 *		
Litter mass	none	HW	0.24	-0.64	0.78	1.06	1.08	59,59
		LW	0.69	-0.17	2.24	0.27		

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PUBLICATIONS

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