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Theodore Lee Grabarz

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THE USE OF A HABITAT QUALITY STRESS INDEX
TO EVALUATE STRESS AS AN ANALOG FOR PROXIMATE FITNESS IN THE
AMERICAN CROW WITHIN A MATRIX OF LANDCOVER CHARACTERISTICS
TO ASSESS ITS POTENTIAL CONTRIBUTION TO DISEASE ETIOLOGIES

A Dissertation

Presented to the Faculty of
Antioch University New England

In partial fulfillment for the degree of
DOCTOR OF PHILOSOPHY

by

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December 2023

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This dissertation, by Theodore Lee Grabarz, has
been approved by the committee members signed below
who recommended that it be accepted by the faculty of
Antioch University New England
in partial fulfillment of requirements for the degree of

DOCTOR OF PHILOSOPHY

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ABSTRACT

THE USE OF A HABITAT QUALITY STRESS INDEX TO EVALUATE STRESS AS AN ANALOG FOR PROXIMATE FITNESS IN THE AMERICAN CROW WITHIN A MATRIX OF LANDCOVER CHARACTERISTICS TO ASSESS ITS POTENTIAL CONTRIBUTION TO DISEASE ETIOLOGIES

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All organisms occur within spatial and temporal environments to maximize proximate fitness (health) and thus life history outcomes. Previous work has examined the temporal and behavioral aspects of proximate fitness on life history outcomes particularly regarding highly perturbed environments (i.e., climate and land use change, resource extraction, agricultural erosion, etc.). My work focuses on the less examined spatial aspect of these perturbed environments. More specifically, this dissertation examines habitat selection and quality as the basis for understanding stress response (negative and positive feedback mechanisms) to environmental stressors within the larger context of regional or gamma (γ) biodiversity. Through the lens of environmental endocrinology, I examine patterns of glucocorticoid (GC) hormone differentiation spatially. I do this to understand how biotic and anthropogenic environmental stressors affect stress response in the American Crow (AMCR). This stress response could have an impact on human disease origins. I examined 13 sites throughout the State of Connecticut between 2019 and 2021, from very rural to very urbanized. I collected 153 opportunistic fecal samples of AMCR, then used radio immunoassay to characterize and quantify the samples as GC hormones, a key chemical constituent that reflects stress response in avian subjects. I then used a geographic information system (GIS) to plot various catchments for each sample centroid as notional representations of AMCR territories. I then overlaid 15 landcover

types as biotic and anthropogenic environmental stressors (ESs). I used ordinary least squares linear regression for my initial analyses to evaluate the degree of validity of the ES–GC relationship at discrete locations where samples were taken and subsequently within varying sized territorial catchments. Finally, I reinterpreted a single constrained gravity model for the development of a habitat quality stress index (HQSI) to understand more dynamically how stress response is affected by movement around AMCR territories. Originally based on Newton’s law of universal gravitation I believe this is the first use of such a model in evaluating stress response via fecal GCs in an ecological setting across a spatial landscape. A major takeaway from these findings is that the historically understood linearly composed landscape gradient has a much greater extracellular or episodic or granular location-specific nature. Examining GIS raster imagery for instance, yields dramatic differentiation of land cover types over very small areas ($< 0.1 \text{ km}^2$) that indicates stress being applied in a highly stochastic manner. This coupled with the dramatic variation in GC levels around roost areas shows AMCR likely traveling significant distances over and through locations with various levels of environmental stressors to arrive at their roost sites each evening. Stress is mediated most effectively when there is consistency or linearity in its application, facilitating a rapid return to equilibrium. The extracellular nature of landcover examined showed a dramatic differentiation that stress response is unable to adjust to over time, without having a pathological response. This results in the extension or lengthening of the negative feedback response culminating in disequilibrium of a positive feedback response, and thereby reduction in proximate fitness and immunological resistance. AMCR, more so than many other taxa, is a highly social and adaptable avian species due to its higher level of cognition and neuroplastic nature (rapid flexibility and adaptation of response via its sophisticated central nervous system [CNS]). The AMCR populations in the roosts I observed

thus favor urban locations. However, AMCR's endocrine system adapts more slowly than their CNS (brain) to higher stress environments. Social cohesion thus outweighs homeostatic balance. In effect we would say that *they are too smart for their own good!* This dissertation is available in open access at AURA (<https://aura.antioch.edu/>) and OhioLINK ETD Center (<https://etd.ohiolink.edu>).

Keywords: habitat quality, stress response, glucocorticoids, habitat quality stress index, immune-competence, proximate fitness, American Crow

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Thanks to my dissertation committee members, Dr. Jim Jordan, Dr. Liz Willey, and Dr. Tony Pappantoniou, for their unwavering support over the years. Thanks also to Dr. Alicia Maltz for encouraging my use of the American Crow as a study subject. It made the years of work that much more interesting and fulfilling. To my friends and family, thanks for supporting my efforts over many years, even prior to my time at Antioch University, when I was developing some initial thoughts on biodiversity, stress response, and the American Crow while at Fordham University. Finally, a special thanks to my parents whose unwavering support of education as the foundation of all good societal outcomes, facilitated my drive to always be learning more. I was always taught that *time goes by no matter what you do*, so that it is important to make that time count. The last 10 years has taught me that statement is more than a truism.

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CHAPTER 1: INTRODUCTION

Habitat Quality, Homeostasis, And Life History

Every organism functions within its external environment subject to the physiological constraints of its internal environment. The external environment is made up of environmental stressors that include both biotic and anthropogenic stressors that I characterize collectively as habitat quality. Habitat quality attains life history significance post habitat selection, as it can set the trajectory for an organisms life development and ultimate death. The internal environment is the basis of an organism's physiology, determined by its genotype and physically expressed by its phenotype. The balancing mechanism between these two often competing environments is homeostasis. The tension between these two opposing systems to maintain an equilibrium condition for homeostasis in an organism is the most profound and far reaching in nature. It is the maintenance of life itself. In so doing, it governs life history, for without it, there is no life, and thus no history. Homeostasis enables birth, life, including predation, competition, habitat selection and quality and the myriad of activities that go into living and ultimately dying—senescence. Homeostatic dysfunction is the root cause of disease pathologies in an organism. The subtlety of the etiology of the unifying foundation of those pathologies though, is hard to quantify. My research focuses on quantification, the measurement of stress response originating in the external environment, which occurs due to the inability to re-achieve homeostatic equilibrium. This disequilibrium is ultimately the result of the stochastic or varying magnitude and duration of stress caused by spatial environmental stressors that are increasingly perturbed in today's ecological setting (Hastings et al., 2018).

Habitat Selection, Quality, and Fitness

Increasing ecological perturbation complicates the axiom—*where and how we live can affect our health*, or relative to other taxa—life history outcomes. For most species, that choice

is based on habitat selection, a hierarchical process of proximate behavioral responses in choosing and/or using habitat (Cody, 1985). The negative consequences of these choices and/or uses come about in habitat quality, and influences fitness and individual survival (Block & Brennan, 1993; Hutto, 1985; J. Jones, 2001) due to organismal stress. Individual survival then is related to habitat conditions (Cody, 1985) that act as habitat quality environmental stressors. Habitat selection is the temporal and spatial framework (catchment) of the interface between habitat and habitat quality. This temporo-spatial point sets the life history trajectory for an organism and determines its proximate fitness, or overall health over time at that spatial location. Thus, there is a direct correlation between the avifauna, the American Crow (AMCR) and its health influenced by the selected habitat and its quality through the environment. The metric for this influencing process is fitness, defined here as individual proximate fitness which influences current or near-term generations. This contrasts with reproductive success regarding fitness, based on evolutionary natural selection.

The Nature of Stress

All organisms inhabit spatial and temporal environments to optimize proximate fitness (health) and thus influence life history outcomes. Significant research has been conducted on stress hormones in evaluating their temporal nature and magnitude in organismal response to seasonality, temperature change, predation, and reproduction etc. (Allen & Hoekstra, 1992; Hastings et al., 2018). No research to my knowledge has characterized those same stress hormones through fecal sampling in three dimensions spatially or derive its origins from multiple landcover characteristics as environmental stressors to represent habitat quality, across multiple sized habitat catchments.

Thus, my research focuses on the spatial aspects impacting proximate fitness of these perturbed anthropogenic environments. More specifically my work entails examining habitat

quality as the basis for understanding stress response in the AMCR to biotic and anthropogenic environmental stressors within the larger context of regional or gamma (γ) biodiversity.

Through the lens of environmental endocrinology, I examine patterns of glucocorticoid (GC) corticosterone hormone spatial differentiation. This stress response can enable pathologies leading to disease, by alteration of negative and at times *in extremis*, positive feedback mechanisms, that can ultimately facilitate transmission to humans in many cases.

Stress response results in the release of stress hormones (in this case GCs) following the inducement of stress from environmental stressors, the disruption of homeostasis and, the equilibrium state between the internal and external organismal environment. The ability to return to, or the prolongation of the return to that equilibrium state through an aberrant negative feedback mechanism is the basis for disease etiology or origins. In a larger context, developing a better understanding of the mechanisms of stress response in various organisms, (in this case AMCR), can establish a more comprehensive understanding of reservoir competence (Littwin et al., 2015), and the ability to reduce the potential for disease transmission including humans, through greater biodiversity.

To minimize extreme negative or positive feedback effects, stress is mediated most effectively when there is consistency or uniformity in its application, facilitating a rapid return to equilibrium. The matrix or extracellular nature of landcover I examined, showed dramatic differentiation. Over time, I hypothesized stress response in AMCR was not able to adjust to this differentiation, resulting in a pathological response. This would cause the expansion or temporal lengthening of the negative feedback mechanism, or in the extreme, positive feedback, resulting in the complete disruption of, and inability to return to homeostasis. This disequilibrium results in alteration to the hypothalamus, pituitary, and adrenal (HPA), gonadal (HPG), liver (HPL), and

thyroid (HPT), HPA/G/L/T axes and can lead to multi-system disruption. These axes are mediated by the central nervous system (CNS) enabling homeostatic function or dysfunction. This dysfunction reduces proximate fitness and immunological resistance through the collective neuro-endocrine axes, across a wide constellation of physiological systems. Specific to AMCR, this dysfunction is manifest in a behavioral disconnect between its preference socially for more urbanized human habitat selection (particularly in traveling to and from winter roosts). This habitat facilitates more social behavior neuroplastically (enhancing neurogenesis) but contributes to AMCR's lack of physiological adaptability to this environment (as exhibited in higher GC levels) through the urban environments habitat quality. However, AMCR's evolutionarily older endocrine system adapts more slowly than their more highly developed CNS, to higher stress environments. Social cohesion thus outweighs homeostatic balance. In effect we could say that *they are too smart for their own good.*

Immunologically, this can lead to a decrease in proximate fitness and thus an increased susceptibility of the immune system to disease. This poor immune response has been examined largely through the lens of AMCR's susceptibility (reservoir competence; Littwin et al., 2015) as a host of West Nile virus (WNV) in the 2000-2001 timeframe in Connecticut (Hadler et al., 2001) through the use of serum samples in the blood. However, it has never been evaluated through the lens of susceptibility due to excessive stress via fecal sampling as I have done. More specifically, it has not been evaluated from the impact of multiple biotic (deciduous and coniferous, wetland, etc.) landcover and anthropogenic (impermeable surface, agricultural surface, turf, etc.) environmental stressors on the AMCR.

Linking susceptibility to disease, habitat selection and quality are also key components of gamma (γ) regional biodiversity, the number of species and quantities of individuals of those

species, found over a range of habitat types (Morin, 2011). Along with species richness (the number of species in an area), biodiversity is related to important functional attributes of communities including the resistance to disturbance, invasion, and alteration of primary production (Loreau, 2004; Loreau et al., 2001). Research over the last several decades has suggested that biodiversity can serve as a valuable ecosystem service (Bernstein, 2008) that can mitigate emerging infectious zoonotic disease in humans (Wood et al., 2014).

Validating the correlation of biodiversity and human disease mitigation, through the mechanism of stress amelioration, as a general principle, would yield significant utility in the public policy realm of health care. My work further seeks to rectify the lack of an aggregated method to evaluate multiple environmental stressors serving as the basis for biodiversity. These stressors that lead to stress response that affects proximate fitness, can affect immunological resistance and thus biodiversity.

Proximate fitness is affected by what I call “extracellular habitat” or in the current literature the *matrix habitat* (“habitat as islands embedded in a matrix of ‘nonhabitat’”; DiMarco et al., 2022). I differentiate this habitat term as in my research relative to AMCR there is no ‘matrix of nonhabitat; there is simply a degree of utilization of that habitat space that differs in its utility. The plastic nature of today’s more urbanized landcover (a patchwork of differentiated landcover types within small physical areas) results in profound cumulative effects on stress and thereby stress response in AMCR. The areas that I examined spatially were almost exclusively an extracellular heterogeneous landcover in contrast to the linear homogeneous landcover continuum that has been described so prevalently in the literature (McDonnell et al., 1997; Rapport et al., 1985). This differentiation in land cover type over a small area leads to dramatic changes in stress level and thus stress response.

The extracellular nature of habitat quality can have significant effects on stress level within a small physical space. This spatial concept also facilitates the “dilution effect” (Civitello et al., 2015). The action of this effect is where vertebrate hosts with a negligible ability to infect vectors (termed incompetent reservoir hosts) dilute the potential for competent reservoirs (hosts with a high probability of infecting a feeding vector), thereby reducing disease risk (Schmidt & Ostfeld, 2001). One of the first studies of this idea looked at the intersection of biodiversity and human infectious disease mitigation. The study examined the transmission of the Lyme disease spirochete bacterium (*Borrelia burgdorferi*) from the tick (genus *Ixodes*) and white footed mouse (*Peromyscus leucopus*; Ostfeld & Keesing, 2000). The work suggests that increasing incompetent reservoir hosts – (a more biodiverse community structure) creates a notional 'firewall', reducing the passage of disease into the human population.

To date though, the physiological basis for the dilution effect or ‘competence’ in reservoir hosts, across the 1400+ known human zoonotic infectious diseases (K. F. Smith & Guégan, 2010) remains inconclusive. That said, the emergence, prevalence and persistence of those human pathogens has been associated with several environmental stressors, the most significant of which are changes in land use which my dissertation focuses on (Taylor et al., 2001; Shochat et al., 2006; Woolhouse & Gowtage-Sequeria, 2005). Freedman (2015) defines environmental stressors as influences that limit the performance (reproductive success or fitness) of individuals, population, or community levels, caused by high or low levels of exposure to that stressor. Significant changes in land use occur in the rural to urban landscape gradient, an area of increasing human density and attendant increase in infrastructure intensity and environmental perturbation.

Those changes in land use also affect an organism's reservoir competence proximately, which is an organism's susceptibility to a pathogen that potentially reduces the species fitness multi-generationally. Work regarding the dilution effect and reservoir competence, has focused on vector's infection of a variety of competent and incompetent hosts, thus "diluting" the ability to spread the disease across a broader population (Ostfeld & Keesing, 2000). Not addressed satisfactorily is the basis for that competence. For instance, does that susceptibility vary based on physical location and thus potentially of differences in habitat quality? If so, this could suggest that there is a stress mechanism invoked within the organism that would increase its susceptibility by weakening its ability to fight disease catalyzed by external stressors that represent habitat quality. In converse, altering the physical mix of those environmental stressors of habitat quality could have a positive effect on stress response, thus reducing susceptibility to disease.

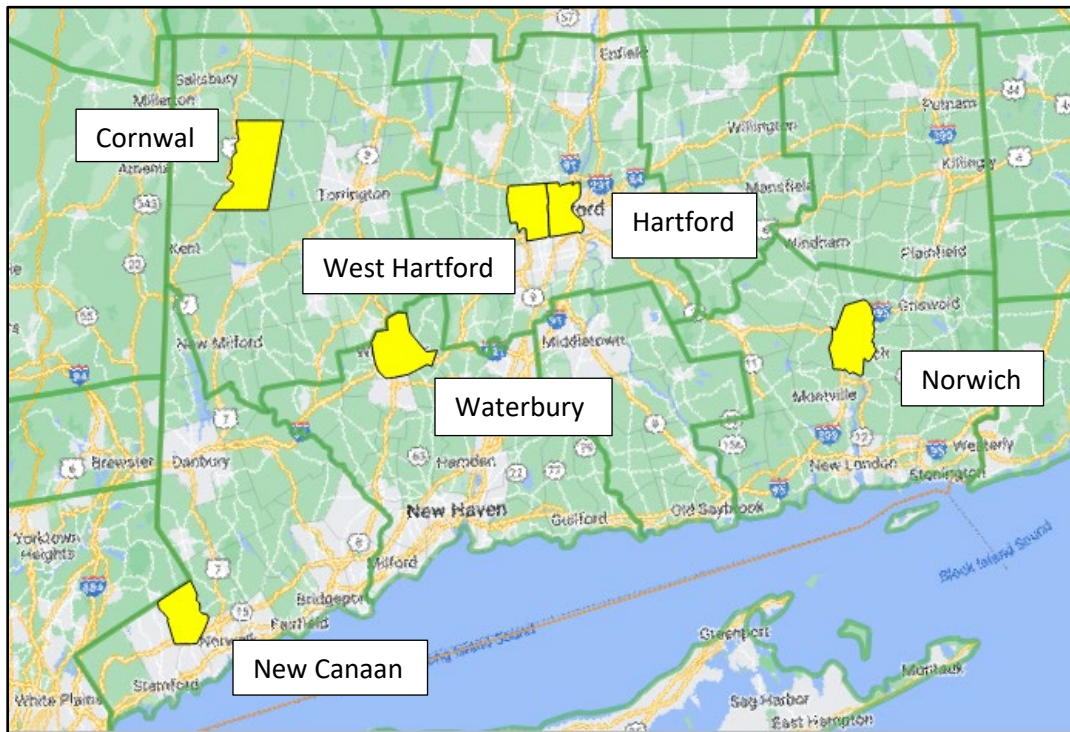
American Crow (AMCR) *Corvus brachyrhynchos*

The American Crow (AMCR; *Corvus brachyrhynchos*) is an ideal avian model to answer these questions of stress response and disease pathologies as it lives within the entire rural to urban landscape gradient. AMCR serves as host for several zoonotic diseases, most notably in the last twenty-five years, West Nile virus (WNV; Loss et al., 2009). An analysis of WNV avian mortality of 40 wild bird species in Connecticut from 1999 to 2005 found that 80% of those fatalities occurred in AMCR (Dickson, 2020). Between 2000 and 2003, 92% of all human WNV infections were preceded by an AMCR fatality within their community (Centers for Disease Control and Prevention [CDC], 2015). In earlier work, I found a strong positive correlation between WNV mortality in AMCR and increasing urbanization, e.g., including impermeable surface (Grabarz, 2013). Impermeable surface represents human made infrastructure such as paving, concrete, and buildings. More importantly, rain-water fluxes from these anthropogenic

surfaces transport chemicals such as Zn, Cu, P, and other toxic constituents that results in a background toxicity to other organisms (Verity & Yasui, 1996). I hypothesized that conducting its life at the urban end of the rural to urban gradient contributed to lowering individual fitness, making AMCR more susceptible to disease. My previous work further examined land cover and AMCR WNV mortality across the eight counties in Connecticut from 2000 to 2012 at a landscape regional scale. I found strong correlation between landcover and AMCR WNV mortality from 2000–2001 ($R^2 = 0.65$ and 2000–2003 of $R^2 = 0.42$ (Grabarz et al., 2015). Using 2001–2005 AMCR mortality for five of the counties currently sampled for GC resulted in an $R^2 = 0.52$ for developed area (impermeable surface) and AMCR mortality.

Study Sites and Catchment Areas

My study location was throughout the State of Connecticut (Figure 1.1), examining the species AMCR, along a notional transect from very rural to very urbanized areas. Previous work examining a single species used only one or two sites (Bonier, 2012). My previous work examined the WNV incidence in AMCR associated with landcover changes at the eight-county political geographic scale in CT (Grabarz, 2013). Building on that previous research in my dissertation I have studied six communities within five of those counties. I collected 153 opportunistic fecal samples of AMCR at 13 sites, then used radio immunoassay to characterize and quantify the samples as GC hormones corticosterone, a key chemical constituent that reflects stress response in avian subjects.

Figure 1.1*County and Site Locations – State of Connecticut*

Using GIS, I then spatially established each of the samples geo-coordinates origin (centroid at winter roost location) with concentric areas (catchments) at a 1, 4, 7, 14 and 18-kilometer (km) areas. These concentric areas represent the maximum extent of published (Caccamise et al., 1997; McGowan, 2001) potential territories of AMCR. I overlaid 15 landcover types characterized as biotic and anthropogenic environmental stressors, converting each of those landcover types to percentages of the area or quantity (in the case of human census or biodiversity data) of the catchments. Catchment area or quantity was then used as the independent variable and GC the dependent variable for all subsequent calculations.

Data Analysis

The analysis included the biotic and anthropogenic environmental stressors that make up the habitat quality of AMCR habitat. The goal of this analysis was to examine stress response to

environmental stressors that could induce AMCR susceptibility to disease. I determined the magnitude and variation of the physiological stress effects by non-invasive means through hormonal fecal sampling. There have been very few if any studies that have looked at the full spectrum of environmental stressors and the physiological (neuro-endocrine) response to them for one species across multiple habitat quality areas.

Impermeable surface or developed area is one of the fifteen biotic and anthropogenic environmental stressors that I examined as representative analogs or components of habitat quality. Characterizing habitat quality as a series of environmental stressors (landcover type percentage and population point data) removes the typically qualitative nature of habitat suitability. It therefore facilitates the quantitative evaluation of stressor level that can then be measured against a stress response emanating from the organism. This stress response to environmental stressors results in alterations to proximate fitness, the overall health of the organism which can be measured against the variation in spatial area of the various stressor components. I used ordinary least squares (OLS) linear regression, and a single constrained gravity model for the development of the habitat quality stress index.

Habitat Quality Stress Indices

The index of habitat quality stress that I have established graphically depicts optimal to suboptimal habitat using a single constrained gravity model (Isard, 1954; Newton, 1687; Stewart, 1947). Based on my review, this is the first time that the single constrained gravity model has been used specifically in ecology to evaluate stress spatially, at a landscape scale across a series of environmental stressors. To further refine the indices noted above, my research questions included the following:

1. What is the magnitude and relationship of GCs (as an analog for stress response in AMCR), at and compared with multiple site locations?
2. How will stress response in AMCR be affected by variation in environmental stressors, at multiple site locations, at multiple size catchments?
3. What is the effect on county wide mortality of AMCR due to GC level stress response at multiple sites.
4. What is the three-dimensional dynamic stress response, due to environmental stressors in AMCR? Further, what is this stress response at multiple catchment sizes, across the geographical studied sites, through a lens that I characterize as a habitat quality stress index (HQSI)?

CHAPTER II: LITERATURE REVIEW

This review is divided into four parts: habitat selection and quality theory, physiological stress response to environmental stressors affecting habitat quality, the American Crow (AMCR), and the rural to urban landscape stress gradient. The interaction of these aspects serves to highlight the basis for and the challenges of AMCR proximate fitness and its relation to the transmission of disease.

Habitat Selection and Quality Theory

An organism's life history is foundational to its proximate or lifetime fitness, codified as habitat selection theory. History is critical to understanding the action of environmental stressors on organism's response, resulting from differing habitat quality. Habitat selection and its influence on organismal communities (thus biodiversity) begin with a determination of physical location. This is a learned or instinctual assessment that a physical space at a point in time meets the species life history requirements (Cody, 1985). Karr (1980) and Block and Brennan (1993) suggested that habitat selection, the observation and analysis of floral and faunal habitat, passed through three eras. These eras include the observational (descriptive biology and the development of systematics), qualitative natural history (abiotic attributes as limiting factors of species range), and quantitative ecology eras that focused increasingly on the organism's interaction with its environment (habitat) leading to an understanding that environmental conditions clearly impact organismal health.

Working Models of Habitat Selection and Quality Theory

The interaction of the organism and its biotic and abiotic environment found increasing resonance in the mid twentieth century in habitat selection and quality theories. As a branch of optimal foraging theory, habitat selection suggests that varying individual and species phenotypes (specific traits) vary in the ability to harvest resources (MacArthur & Pianka, 1966).

This could account for AMCR's broad habitat range as well as its opportunistic diet. This differential accessibility would also hold true for environmental stressors impact on organisms as the basis for habitat quality. Specifically, this means that based on a species phenotype, environmental stressors affect species and potentially individuals differently. This differing ability of physical characteristics could be based on physiology (e.g., beak size, flight range), behavior (e.g., innate methods for foraging sources), or temperament. Implicitly, according to this theory, additional time in a location with sufficient resources will result in greater fitness (Cody, 1985). This occurs as the choice (cost of energy expended versus energy saved) results in less energy required by remaining in the same habitat.

A series of subsequent working models formalized many of these habitat concepts that Block and Brennan (1993) postulated could be a foundational theory of vertebrate biology. These theories included the ideal free distribution (IFD) Model focusing on the "selection" aspect of habitat, including the density of conspecifics, and carrying capacity (Fretwell & Lucas, 1970). Subsequently, the habitat training (HT) model suggested that cultural transmission (verbal or non-verbal behavioral cues) could point to an "ideal" habitat in terms of quantity of resources (Fretwell, 1972). Following this behavioral idea of competitive ability, the ideal despotic distribution (IDD) model was formulated where birds of varying levels of boldness would push other conspecifics into less resource rich habitat (Fretwell, 1972). This led to the concept of settlement time periods in the quitting time (QT) model where the time spent in a habitat patch could overcome otherwise poor resource levels (Charnov, 1976) or poor habitat quality. Finally, the reproductive success matrix (RSM) model viewed habitat as a series of properties, or ecological strategies to survive and reproduce (Southwood, 1977).

These habitat selection theories implicitly assumed adequate habitat quality to ensure reproductive success, as the assumption at the time was largely based on a stable abiotic environment (Hastings et al., 2018). In the current era, our greater understanding of the long-term implications of environmental change recognizes that stable conditions can no longer be assumed accurate.

While the current catalyst for understanding habitat quality is based on our greater recognition of the importance of environmental perturbation, there is a germane historical reason as well. Specifically, the history of natural science has been primarily a focus on descriptive biology of the organism and its needs of settlement (the present) rather than its fitness (the future). As described by M. D. Johnson (2007), Van Horne (1983) was the first to uncouple the concept of habitat quality from successful habitat selection based on subjectively evaluating habitat quality, i.e., there are lots of trees so it must mean it is appropriate habitat. To do that she questioned the foundational tenet of habitat selection, that greater conspecific density necessarily positively correlates with greater habitat quality. Bock and Jones (2004) subsequently found that 20 years later 72% of habitat quality studies still regarded density to be positively correlated with reproductive success. They also allowed though that anthropogenic habitat dysfunction (fractured urbanized environments) would likely confuse many species concept of habitat quality (Bock & Jones, 2004). They also corroborated Van Horne's (1983) concern, that highly territorial species (such as AMCR) behavior would likely be at variance with this density/reproductive success hypothesis. Indeed, due to rapid occupation of space, *aves* may not have the time to learn to recognize ecological traps (seemingly preferred habitat) from genuine opportunities (Bock & Jones, 2004). Attempting to find some common ground while exploring some of these extant issues, M. D. Johnson (2007) found that over 90% of 200 habitat quality

studies from 1984 (post the publication of Van Horne's seminal work) to 2005 used distributional and demographic methods. Both these methods rely upon counting population size as the determination of habitat quality. However, both these techniques assume some degree of an unrealistic static pre-condition such that movement is frozen in time and space. M. D. Johnson then looked at the other 10% of studies and characterized them as "individual condition measures." The first individual condition measure was morphological-descriptive analysis of individual bird condition within the landscape as the basis for habitat quality derivation. The second individual measure was physiological-tissue or biotic fluid samples as a measure of fitness of the individual. He suggested that in these two techniques, an organism's physical condition is a consequence of habitat use. As such, these techniques avoided the problems of the other major categories reliance on count data, and thus were more reflective of organism's biological response to the environment. My research relies upon this second type of individual condition measure and is thus physiological in nature.

Physiological Stress Response to Environmental Stressors Affecting Habitat Quality

The process of habitat selection for AMCR is based on the search for adequate habitat quality wherein a significant amount of time is spent responding to environmental stressors as part of its life history. That stress response has both an internal and external component that likely varies along the stress gradient in response to differences in environmental stressor levels. Internally, the short - and long-term effects are exhibited through the neuro-endocrine axes (HPA/G/L/T; Bonier, 2012; Whittow, 2000), where the struggle to maintain homeostasis in a constantly changing environment is processed. Externally the short-term effect could be revealed as emaciation such as due to starvation. This could ultimately result in the self-determination to locate another foraging territory of potentially higher habitat quality.

Environmental Stressors

Environmental stressors impact organisms throughout their life histories and can be thought of as measurements of habitat quality. They are influences that limit the physiological performance (reproductive success or fitness) of individuals, populations, or communities, caused by varying levels of exposure to those stressors (Freedman, 2015). These stressors consist of two categories relevant to this research: biological (due to organismal interactions including humans), and anthropogenic or chemical (toxic chemical soils and other human induced pollution; Freedman, 1995). In a direct sense, these limits on physiological performance are exhibited in aspects to include altered habitat, habitat fragmentation, loss of predators, changes in land use, niche invasion, and host transfer (Grimm et al., 2008). The result is that these factors often promote reduction in fitness which then intensifies the pathogen-vector-host interactions (McMichael 2004; Patz & Confalonieri, 2004). The list of environmental stressors I examined are listed in Table 3.1 that include specific biotic (fauna and flora), and anthropogenic including human made infrastructure (impermeable surfaces), and human and animal population sources.

Theories of Stress - Stressor Interaction

Physiological (the basis for this dissertation) and behavioral stress response mediates stress that occurs as the result of environmental stressors. Stress relative to this research is a real or perceived threat to homeostasis (S. Smith, 2006). Stress responses to these environmental stressors are conserved physiologically across vertebrate groups (Boonstra, 2004) making its study and findings relevant across many species. These effects are also evolutionarily optimized meaning they are calibrated for a precise degree of fitness (Monaghan, 2014). Therefore, organisms are adapted to predictable stressful events. However, unpredictable stress events and the increasing rate of those perturbations of environmental change challenge our understanding

of how organisms will cope with that increasing rate of change. Stress in the context of habitat theory relates to homeostasis or energy balance. This is energy available in the environment versus energy required to maintain basic survival function internal to the organism (Busch & Hayward, 2009). Stress occurs when a biological control mechanism fails in a fitness critical variable (Del Giudice et al., 2018). The current theoretical framework of stress is based on four somewhat overlapping models: the reactive stress model (Romero et al., 2009) the allostatic stress model (McEwen & Stellar, 1993; McEwen & Wingfield, 2003), the cognitive stress model (Ursin & Eriksen, 2010) and most recently the adaptive calibration stress model (Del Giudice et al., 2011).

- **Reactive stress model:** This is the most well-known stress response mechanism of the HPA Axis (Romero et al., 2009). Originally termed the general adaptation syndrome (GAS) by Selye (1936) it consists of an alarm, (fight, or flight), resistance, and exhaustion stages. To maximize survival, the alarm stage culminates physiologically in the release of glucocorticoids (GC), to attempt a return to homeostasis.
- **Allostatic stress model:** Avian species engage in several behavioral changes in response to altered habitat, which impacts fitness (McEwen & Wingfield, 2003). Multiple pathways of the peripheral nervous system (PNS) affect these behavioral changes resulting in alteration of one of the HPA/G/L/T axes (Martin, 2012; Whittow, 2000). These pathways are manifest physiologically as the allostatic load, the 'wear and tear' on the organism when energy requirements exceed energy availability (Busch & Hayward, 2009). Over time, this impact accentuates neuro-endocrine responses when exposure to repeated or chronic stress, results in debilitating health or fitness affects (McEwen & Stellar, 1993).

- **Cognitive stress model:** Subsequent work on the GAS process involved the discovery that a certain basal, maintenance or intermediate level of GC input through environmental enrichment (sensing external environmental influences including stressors), inspires neurogenesis (nerve cell reproduction) thereby enhancing cognitive ability (DuPret & Fabre, 2007; La Dage, 2015; Ursin & Eriksen, 2010) . The crucial aspect of this environmental enrichment is that it occurs in response to cyclical environmental events. These events could include circadian rhythms (Fairhurst, 2011) as well as a complement of repetitive life history behaviors (Dibner, 2010). Other work has suggested that environmental variability within existing heterogeneous environments may facilitate an organism's further adaptation to environmental change (Gonzalez-Gomez, 2015). In particular, urban environments appear to inspire higher cognition and neural plasticity in avifauna, while little work has been done to understand the physiologic basis for this behavioral acclimation (Grabarz, 2018b; Weaver et al., 2009). Levels of GCs can vary within environment as well as taxonomic group, and life history stage (Busch & Hayward, 2009). Measurements of GCs need to be controlled statistically to accommodate these covariates or caveats in their interpretation and must recognize these various aspects so that measurements are understood within the appropriate context. (McEwen & Wingfield, 2003).
- **Adaptive calibration stress model:** This model combines aspects of allostasis with the idea that repeated stress conveys important information about life history originating from the environment (stressors). In the development of the organism, the stress response system (neuro-endocrine axes) integrates these stress responses into the regulation of key life history tradeoffs. This occurs as the organism attempts to

accommodate these stressors internally to optimize its effect on its fitness that Del Guidice et al. (2018) terms hormesis.

Neuro-Endocrine Complex Intersection

Stress response begins with cell signaling through the nervous system to various targets in the organism. The modalities of these signals can be either specific point-to-point targets electrically through nervous system neurons, or diffusely chemically to distant sites through endocrine system hormones. The neuro-endocrine complex or axes are specialized subsystems of the evolutionarily more recent nervous system and older endocrine system. The nervous system, when stimulated responds more rapidly but with a shorter duration and less fidelity (less signal specificity) than the endocrine system. From a gross anatomic perspective, the nervous system consists of the central nervous system (CNS) and the peripheral nervous system (PNS). The CNS has two major components—the brain and the spinal cord. The brain provides executive function for processing, interpreting, storing information, and “issuing orders” to muscles, glands, and organs. The spinal cord acts as the “bridge” between the brain and the PNS. The PNS is responsible for transmitting information to and from the CNS via nerve pathways. The PNS is further broken down into the voluntary (somatic) nervous system (VNS) and the autonomic nervous system (ANS). The VNS is voluntary, involving self-determination or individual control, generally of voluntary skeletal muscle movement that we could characterize as voluntary behavior or behavioral volition. The ANS operates automatically and controls or regulates the homeostatic (internal balance with the external environment) mechanisms of the glands, blood vessels, and associated organs. The last branch of the ANS consists of the sympathetic nervous system (SNS) and the parasympathetic nervous system (PSNS). Colloquially the SNS is thought of as responsible for the “fight or flight” or emergency response and the PSNS as responsible for the "feed and breed" or rest response (Martin, 2012). Other than

the SNS, the other neuro-endocrine component systems result in involuntary or autonomic behavior. In short, the SNS and the PSNS are the overarching regulator of the neuro-endocrine axes concerned in my research.

Most critically, between the PNS and the VNS/ANS levels, is a mechanism much like a computer router. That “router” has a sensory or “afferent” division of nerve fibers sensing the external environment including stressors, through receptors in the five primary senses back to the CNS for processing a response, as necessary. In parallel to the sensory division is the motor or “efferent” division that conducts impulses back from the CNS that process a response (Whittow, 2000). These impulses pass through the effectors, of muscles of the voluntary SNS and the muscles and glands of the involuntary ANS. It is at this intersection of the motor and sensory pathways "router" where the neuro-endocrine HP/A/C/T axes can have their greatest positive or negative impact on AMCR's fitness.

The endocrine system itself sends chemical signals via hormones (various amino acids, lipids, and steroids) throughout the AMCR's body to regulate processes including homeostasis, growth, metabolism, immunity, sexual development, and emotions such as fear (Bonier, 2012). The endocrine system, when stimulated, responds more slowly but with a longer signal duration and higher fidelity (more signal specificity) than the nervous system. Hormones as chemical messengers are released into the vasculature or blood stream, responding to environmental stressors, and transfer information to different organs to obtain some biological response. Major glands involved in the endocrine system and relevant to the neuro-endocrine HP/A/G/L/T axes of avifauna, include the pituitary, thyroid, thymus, adrenal, gonads and pancreas (Whittow, 2000). The hypothalamus, a small region at the base of the brain above the pituitary, mediates all these glands release of hormones. These hormones ultimately involve releasing, stimulating, or

inhibiting functions that include a cascade of effects that result in a negative or positive feedback signal (Whittow, 2000). These feedback mechanisms can further control and release additional hormones. The inability to restore the equilibrium of homeostasis is the threshold indicator of disease pathologies. The observable responses or behaviors seen in organisms such as AMCR are the direct result of activities internally, within the neuro-endocrine systems and more directly, the axes discussed. Mediated then by the neuro-endocrine system this behavior can be voluntary when originating in the VNS pathway or involuntary when originating within the other autonomic neuro-endocrine pathways.

The typical hormonal process, shortly after the recognition of a stressor causing an internal stress affecting homeostasis, results in a cascade of hormonal release. The hypothalamus as the ultimate mediator of hormonal activation is enervated by sensory input from the SNS relative to sensing environmental stressors. This stimulus results in the release of cortico-tropic releasing hormone (CRH) and arginine vasopressin (AVP) of the paraventricular nucleus (PVN) originating anatomically in the hypothalamus (Bonier, 2012). This induces GC (in avifauna primarily corticosterone) release to occur in the adrenal cortex (superior to the kidneys) which is regulated by adreno-corticotropic hormone (ACTH) located in the anterior pituitary gland below the hypothalamus (Whittow, 2000). Typically, these lipid soluble steroid hormones collect in blood plasma, the circulating media for many of the hormones. In addition, GC is the only known hormone (or its secondary metabolites) that are passed through the gut into feces, relatively unchanged (Del Guidice et al., 2018). Therefore, it is a high-fidelity hormone that can be collected non-invasively which was the basis of my sample collection procedure.

Endocrine-Behavioral Interaction

The endocrine system is a key player in modulating an organism's physiological and behavioral response to unique environments (Bonier, 2012). A few studies have examined the

role of glucocorticoids (GC) and its direct relationship to urbanization or non-urbanization. However, the results of these studies have been frustratingly inconsistent. Equal numbers of studies have found GCs increasing or decreasing in response to the urbanization gradient based on variation in sex, life history, and species (Bonier, 2012). Further as of 2012 only one study had examined GC of one species across multiple environments (Bonier, 2012; Fokidis et al., 2011) which my work rectifies.

Current endocrine ecology literature does not reveal any consistent patterns but has demonstrated that populations of birds in urban habitat often exhibit differences in endocrine traits. For instance, hormone concentrations vary when compared to conspecifics in nonurban habitat (Vitousek et al., 2018). High GC levels within the endocrine system were historically thought to be an automatic marker of pathology resulting from stress (Bonier, 2012). The types of hormones and their signal transduction pathways are similar in most taxa, while the amounts of those circulating hormones vary widely across those same groups, and even individuals (Vitousek et al., 2018). Recent work though has shown that the releases of GCs are an important baseline component of homeostasis (Jimeno et al., 2015). As a mechanism of homeostasis, energetic balance is maintained appropriately through the varying in GC levels in response to environmental cues (e.g., lower temperatures or generally varying environmental conditions; Jimeno et al., 2015). Teasing out the variation caused by the actual negative consequences of stress then from basal level GCs, is an important factor in proving the reliability of the stress level due to anthropogenic environmental stressors and abnormal abiotic conditions.

Further, acute increase in GC post-natally can have long-term effects on stress response. Intracellular down regulation (reduced homeostatic balance) has been reported in mammals perhaps due to reduced capacity for negative feedback (Spencer et al., 2009). This could be

caused by GCs binding first to the higher-affinity mineral corticoid receptor (Whittow, 2000). The binding of the lower-affinity glucocorticoid receptor occurs after mineral corticoid receptors have been saturated (Busch & Hayward, 2009). This leads to the down regulation (decline) of negative feedback controls. This reduction in the ability to maintain homeostasis could have long-term effects on fitness through the iterative negative consequences on the components of the HPA axis. That could affect not only their long-term viability in urban environments, but also be a cause of their increased sensitivity to pathogens such as WNV or immune-competence. Other work suggests that ACTH activates immune response and the production of lymphocytes while other work suggests its suppression (Jimeno et al., 2015; Touma & Palme, 2005). An inverse relationship between protein deficiency and thyroid function results in the production of T3 thyroid hormone and increased production of GCs as part of the HPA axis response (Whittow, 2000). This could point to one of the challenges of AMCR fitness immunologically, where lymphocyte activity is partly tied to stress hormone release and thyroid activation, rather than strictly disease incidence and thus immuno-suppression.

This suggests that AMCR while cognitively superior in a neuro plastic sense (higher cognition and neural plasticity in avia that would favor urban locations) their endocrine system adapts more slowly than their CNS to higher stress environments. Their endocrine system therefore must absorb more stress but responds more slowly and thus negatively to it. This potentially highlights the increased incidence of disease in urban areas due to the lowering of fitness.

Challenging the determination of that stress level is the difficulty in typical field collection. The stress of physically handling the taxa to obtain the blood sample can alter the hormones blood plasma concentrations. Further, the distribution of hormones within the

vasculature is discrete enough that the concentrations can vary at different locations from where it is anatomically collected (Touma & Palme, 2005). Hormones can also vary periodically both in a circadian and diurnal sense episodically in blood plasma studies (Medvedev et al., 2018). As an alternative, collecting fecal matter makes levels of circulating hormone concentrations more consistent over time.

American Crow (AMCR)

As noted previously the American Crow is an ideal species as a subject taxa due to a series of specific traits that mimic hominids (Figure 2.1). Due to its wide-ranging habitat, social cohesion, and susceptibility to various pathologies, there are few animals of observable scale that have adapted as well across the rural to urban landscape stress gradient than the *Corvidae* family (including species of Crows, Ravens, Magpies, and Jays). Members of the Order Passeriformes (perching birds) contain over 6,500 species (Gill et al., 2023). It is part of the order that represents more than half of all bird species and twice as many species as the largest mammal orders (Mayr, 1946). Adaptive radiation or rapidly evolving to adapt to new geographic challenges via speciation (Futuyma, 2009) is thought to be one of the *Corvidae* primary advantages and exceeds many other passerine families (Goodwin, 1986). Specifically, this speciation results in phenotypic or physical characteristics adaptations (e.g., different size and girth of beaks) that can facilitate successful life histories in a diverse range of environments (Schluter, 2000). A recent reorganization of the *Corvidae* family (1990) has now increased that number to 650 passerine species. They are also part of a subfamily *Corvinae* consisting of 297 species and 56 genera. A tribe *Corvini* of which Crow and Jays are now a part consist of 113 species and 25 genera (Madge & Burn, 1999). The *Corvidae* family then has virtually a global and native presence but for the tip of South America and the polar ice caps (Clayton & Emery, 2005).

Figure 2.1

American Crow (Corvus brachyrhynchos)



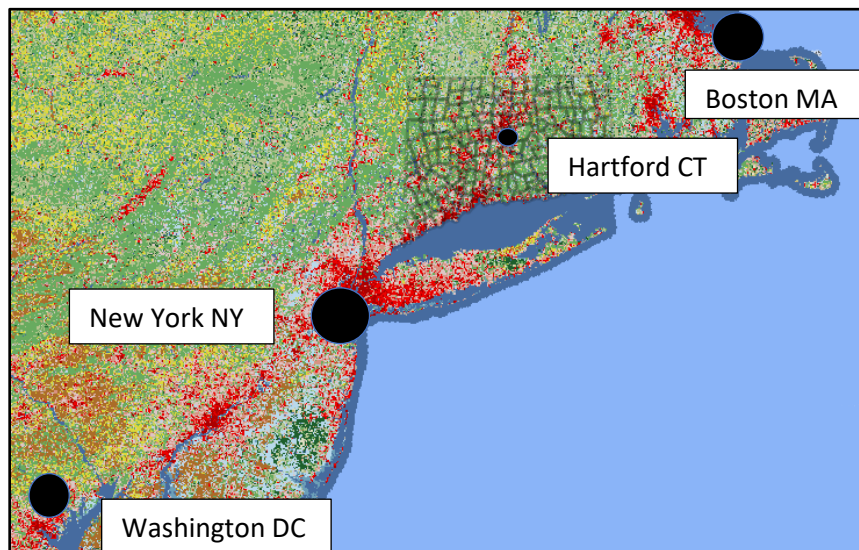
Note. Images by author.

As the most intelligent of birds and one of the most intelligent of all animals, noted for its tool use and self-awareness, (Clayton & Emery, 2005; Emery, 2006) *Corvidae* omnipresence is likely a result of its brain to body mass ratio (encephalization quotient [EQ]; Emery, 2006) on par with primates and cetaceans (Bednekoff et al., 1997; Emery & Clayton, 2004). Through convergent evolution the most recent genera common to *Corvidae* and Primates was likely 5–10 MYA during the Late Miocene to Pliocene period, a time of tremendous climatic and environmental instability which could have fostered several attributes including superior intelligence and adaptation (Emery, 2006). Emery (2006) derived a model of six attributes found in *Corvidae* and species of higher cognition. These attributes include omnivory, opportunistic diet and habitat, high sociality, large relative brain size, innovation (novel approaches to a changing environment), and an extended developmental period (extended rearing within a family unit) contributing to longevity.

Rural to Urban Landscape Stress Gradient

As noted above, key to AMCR's longevity is its adaptability to novel and varying environments, particularly along the rural to urban landscape gradient. This adaptability is also key to understanding AMCR's role in physiological stress response to the environment. More importantly is AMCR as a unique taxa, to understanding the more rapid climate change events and its effect on physiology through ecological function. Hastings et al. (2018) has called this "transient ecological phenomena," the increasingly ephemeral nature of ecosystem behavioral function operating over long time scales but increasingly shorter random amplitudes. This dichotomy affects a variety of environmental stressors. One spatial and temporal aspect of this effect is patchiness, as the essence of the rural to urban landscape gradient is a fractured landscape (McDonnell et al., 1997). Despite this patchiness, most specialist species (due to limiting factors of foraging capacity, competition, reproduction, etc.) still require a certain territorial size regardless of habitat quality. Settlement in areas smaller than required by territorial size, result in settling for suboptimal habitat that increases stress on organism's life history.

AMCR conversely thrives in such fractured habitat. As an example of some of this type of habitat and gradient, I chose the State of Connecticut to evaluate the stress response of AMCR, to habitat quality environmental stressors. Connecticut is part of the New England to Mid-Atlantic coastal area, the densest megapolitan area within the US (Figure 2.2) making it ideal for examining stress characteristics along the rural to urban landscape gradient (Grabarz, 2018). Subject to habitat fragmentation, channelization through human-made infrastructure and impermeable surfaces this area challenges life across the taxonomic spectrum, both profoundly and subtly.

Figure 2.2*New England to Mid-Atlantic Coastal Area*

Megapolitan areas significantly affect habitat quality and biodiversity as the concentration of developed land surface increases (United States [US] Census Bureau, 2012). Between 2000 and 2030 there will be more than a 20% increase in urbanization (impermeable surface area and human population growth) as the continental US coalesces into seven megapolitan areas (Grimm, 2005) where 71% of the US population currently resides. The largest is the New England to Mid-Atlantic region of New York-New Jersey-Connecticut (2,054 persons/km²), almost double the next largest area of Pennsylvania-Delaware-Maryland at 1,060 persons/km² (US Census Bureau, 2010).

Emanating from this fractured landscape, within the New England to Mid-Atlantic region in 2000–2012, Connecticut experienced a significant outbreak of West Nile virus (WNV). Originating in Uganda, WNV was first identified in the continental US in Queens, New York, in 1999 and spread rapidly across the US (CDC, 2015). From 2000 to 2005 there were a total of

2,718 fatal cases of WNV discovered in AMCR across the eight Connecticut counties (CDC, 2015).

A significant factor for the spread of pathogens like WNV are the changes in land use represented by the rural to urban landscape gradient. The gradient can be used as a proxy to represent the levels of stress associated with changes in land use, which on a global scale show a pronounced effect on pathogen abundance (Bradley et al., 2008; K. E. Jones et al., 2008; McDonnell et al., 1997). Those changes have a profound effect on an organism's competence or susceptibility to disease, affecting its fitness. At the scale of the terrestrial biosphere, from the poles to the equator, taxa across the full spectrum of size follow a species richness latitudinal and developmental gradient, correlated with both temperature and rainfall. However, there is a distinct variance in the presentation of emergent and non-emergent infectious diseases. emergent infectious diseases (EID) like WNV, are those diseases that affect host geographic range, and are more prevalent in temperate developed areas. It is also where large human population centers correlated with high drug resistance to those diseases exist (K. E. Jones et al., 2008). In contrast non-emergent infectious diseases are contained in the traditional latitudinal biodiversity hotspots of the tropics. This suggests that unique transmission potential may outweigh traditional disease locations (K. F. Smith & Guégan, 2010). These macro-ecological characteristics are likely to have overpowering effects on the evolutionary, ecological, genetic, and immunological basis of competence or susceptibility of disease, as it affects fitness in such species as AMCR, particularly as these megapolitan areas density increases.

Cadenasso (2007) believes that these increases in density are largely the result within the landscape gradient of spatial heterogeneity of buildings, vegetation, and surface features, related to land-use and zoning. I would submit that the relationship to land use and zoning is relative to

the human scale. However, when measured against the typical organisms (AMCR) physical size the heterogenous aspect is far more significant. Appendix B lists some of the physical landcover environmental stressor characteristics of the counties of the State of Connecticut in which my study takes place. Across these areas, avifauna and humans co-exist within the rural to urban landscape gradient. At the urbanized end of this gradient, the spread of disease increases with the density of the hosts (Lafferty et al. 2002) and appears to increase with the density of impermeable surface. Below a host-density threshold, disease is not sustainable through a population, making it difficult to drive a species to extinction in the absence of host susceptibility (Lafferty et al., 2002). Various stressors including increased conspecific population density, habitat fragmentation, and reliance on anthropogenic food consumption (e.g., fast food dumpster diving; Lafferty et al., 2002) cause this susceptibility, resulting in at least one facet of stress—immuno-suppression (Bradley et al., 2008; Dickinson, 1998; Heiss et al., 2009; Marzluff et al., 2001). Additionally, lower biodiversity and altered community assemblages, changes in interspecific competition, reproductive stress and changes in trophic interactions are shown to be adversely affected in urbanized areas and thus negatively affect biodiversity (Faeth et al., 2005; Shochat et al., 2006).

Historically there have been a minimal number of studies investigating the link between stress response to landscape gradient environmental stressors, and the adequacy of landscape quality tied to population health. Most of those studies have focused on numbers of individuals within that habitat space as a proxy for suitable habitat quality (Bonier, 2012). The earliest studies on the confirmation of the rural to urban stress gradient hypothesis (whereby a reduction in species richness foretold an increase of stress) was by Rapport et al. (1985), who denoted five areas of environmental stressors. First, renewable resources harvesting (foraging); next,

anthropogenic sources (pollutant discharges; (Flather et al., 2008); third, planned physical changes to land use; fourth, invasive species introduction; and finally, natural weather events (Rapport et al., 1985). Evans most recently found that in the rural to urban gradient, species richness and functional diversity declined with increasing impervious surface area as a measure of urbanization intensity (Evans et al., 2018).

Within the landscape gradient, organismal susceptibility to disease due to stress can take several forms. Recent work has shown correlation to oxidative stress caused by the build-up of H_2O_2 (cellular metabolic nitrogenous waste; Salomons & Mulder, 2009). This pathology reduces cell signaling potential. Increased human and non – human populations within the urban gradient mean that stress affects more of these populations (Salomons et al., 2009). A characteristic of this gradient includes heterogeneous habitat composition. This occurs through a patchwork of developed and undeveloped area, lowering the ability of a space to host undivided habitat, thereby influencing potential population establishment and biodiversity. Quantitatively evaluating this density gradient for stress in AMCR (correlating to fitness) has shown the highest levels of corticosterone at 3.68 ± 2.79 ng/ mL for suburban-urban land areas, and half that level at 1.35 ± 2.16 ng/mL for more rural areas (Heiss et al., 2009).

The stress gradient is often highest in the densest spatial areas, where there are the largest number of buildings, impermeable surface, and human density. A further example within the rural to urban gradient is sero (antibody) prevalence as a response to pathogen invaders, positively correlates with increased urbanization. A study in Atlanta, Georgia with 14 different sites with 499 individual birds and seven different songbird species had correlations ranging from 6.3% ($N = 43$) in more rural areas to 30.8% ($N = 52$) in more urbanized areas relative to WNV prevalence (Bradley et al., 2008).

Compounding the effect of stress is the immunological basis of competence. Here the phenotypic or genetic basis of “fast” and “slow” immunity, affect the short and long term “costs” of immunological defense for different species (Hasselquist & Nilsson, 2012; P. T. Johnson et al., 2012). Fast (short lifespan) species (to maximize fecundity) early in its lifespan, rely more on innate immunity which requires less energy investment than adaptive immunity, thus in the near-term using energy for increased reproduction potential (Lee, 2006; Townsend , 2009). Slow (long lifespan) species rely more on adaptive immunity, with longer lives can make additional energy investment, and promote more robust immunity, thereby increasing longer term fecundity (Lee, 2006). In addition, recent work has shown a strong relationship between immunosuppression and adreno-cortical function (GC release) suggesting that stress hormones in avifauna can profoundly affect immune system function (Carsia & Harvey, 2000).

Within the context of habitat selection and quality, a conflict between the selection of a habitat and the individual's immunological response could lead to disruption of a larger community. For instance, an individual with fast (short lifespan) immunity, selecting, or relegated to a poor habitat, could require greater travel distance to obtain food. This could negatively impact its fitness due to the greater energy expenditure shifted to searching for food rather than immunological energy investment resulting in two negative effects. The first would be greater susceptibility to more virulent pathogens. The second would be the lowering of fitness due to greater travel resulting in greater stress.

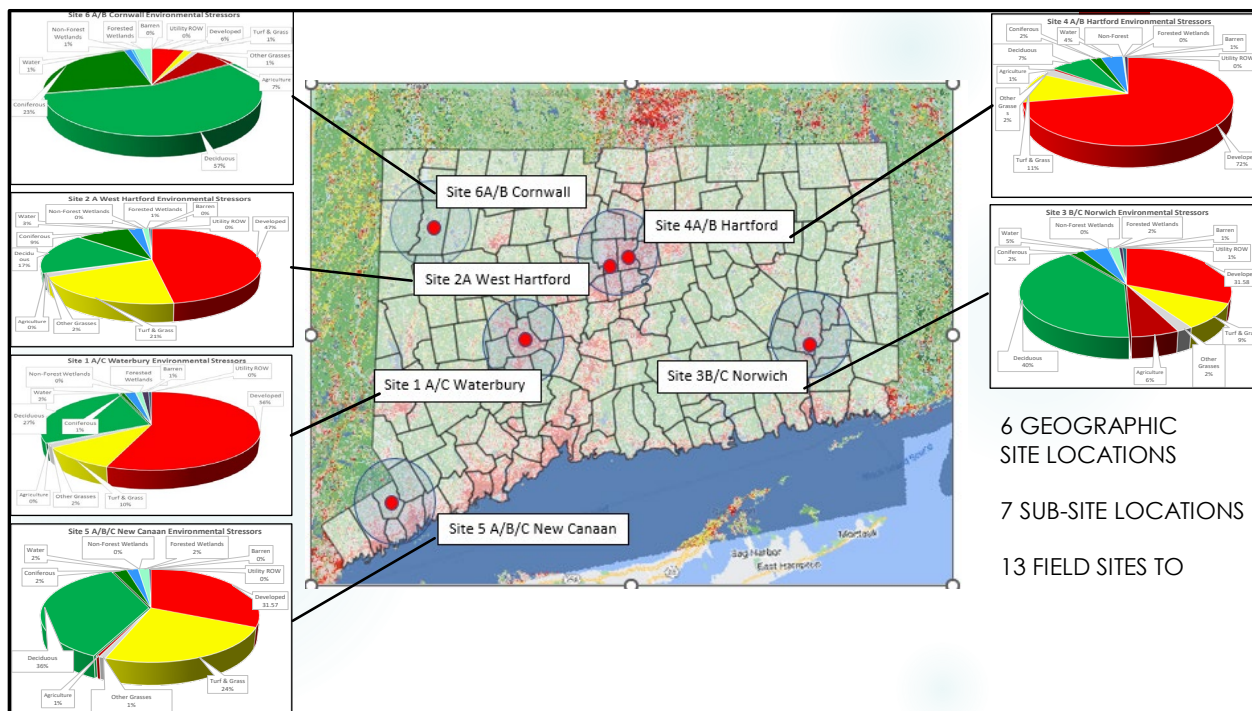
CHAPTER III: METHODS

Study Site Determination

I used eBird (Cornell Lab of Ornithology, 2019), Biodiversity Information Serving Our Nation (Nation, 2019), the web-based survey site of the Connecticut Ornithological Association (Wood, 2023) and my historical field observation, to assess 12 potential study sites throughout the eight counties of Connecticut covering 169 municipalities. Based on that assessment over twelve months, I established six primary sites and seven secondary sites (Figure 3.1) with active AMCR roosts consistently occupied from late September to April that represent my statistical population between 2019 and 2021.

Figure 3.1

Habitat Site Locations and Municipal Land Use Percentages



Each site represented a unique location along the rural to urban gradient (Appendix B).

Sampling Design

Catchment area is the area within which AMCR lives and thus is impacted by stress within its home range (as opposed to travel beyond its home range that I will discuss in the HQSI). It includes both its territorial area, and travel distance from the territory to the roost each night. I based this catchment on the boundary of its territory and travel distance to the point within the roost that a fecal sample is taken. For instance, an 18 km hypothetical territory would be measured from an origin of the roost where the specific fecal sample is taken to the 9 km edge of the hypothetical territory, resulting in a total catchment diameter of 18 km. Historic research of AMCR territory ranges from 0.3 km diameter in urban Utica, New York to 0.61 km diameter in contiguous rural areas (McGowan, 2001); 3.0 to 7.0 km in Northern New Jersey (Caccamise et al., 1997) and 14 km (Stouffer & Caccamise, 1991) to 18 km (Caccamise et al., 1997) for travel from territories to nightly roosts. Therefore, I chose 1, 4, 7, 14, and 18 km distances as hypothetical catchment areas to examine differing stress responses to environmental stressors by the level of GCs fecal sampling at each of these catchment area locations. The basis of this assessment was to calculate within each habitat site and subsite roost area (in Cornwall, West Hartford, Waterbury, New Canaan, Norwich, and Hartford), the concentric areas of catchments at 1.0, 4.0, 7.0, 14.0, and 18.0 km which represented hypothetical territorial areas. Thus, spatially the boundary for these stressors was the catchment area of each of the hypothetical territorial areas (Appendix C, Figure C1). The intensity of the stress response of the stressors was then measured. This was measured by the level of GC hormone found at the origin or centroid of the sample location. That was the basis for the calculation of the environmental stressors percentage area in the individual catchments (Appendix C, Figure C2). For each of the 153 sample locations there were five territorial areas calculated (1, 4, 7, 14, 18 km) and thereby a total of 765 areas of aggregated environmental stressors across the 13 geographic sites.

GIS Data

The geographic information system (GIS) used to visualize the vector and raster layers of these environmental stressor databases and quantify their spatial magnitude was Quantum Geographic Information System (QGIS) 3.12 Bucuresti, open-source GIS platform (Sherman, 2007).

Data derivation began by using GIS to determine the percentage (%) area of each of the biotic floral, faunal and anthropogenic stressors at each of the notional territorial catchments 1, 4, 7, 14, and 18 km for each of the fifteen environmental stressors. These calculations resulted in a (15 environmental stressors x 5 notional catchment areas x 153 GC sample locations) matrix with a total of 11,475 specific environmental stressor quantifications across the 13 geographical sites located in six municipalities from rural to urban. A key assumption in the data derivation and subsequent analysis is that an external environmental stressor induces internal stress in the organism and elicits a stress response, measured by GCs in the organism as it attempts to return to homeostasis. Historic research has not identified the physical boundaries within which stress response is valid. I identified a series of boundaries (territorial catchments) to derive a spatial extent of environmental stressor magnitude more discretely to be able to (in the HQSI) identify the spatial boundaries of stress response that results in a measurable level of habitat quality.

Raster and vector imagery of biotic floral, faunal, and anthropogenic stressors were downloaded from the National Landcover Database (NLCD; United States Geological Survey [USGS], 2019; Multi-Resolution Land Consortium, 2020), the UCONN Center for Land Use Education and Research (CLEAR; Arnold, 2015; US Census Bureau, 2010, 2012) and BISON (Nation, 2019). Imagery was then isolated from the larger NLCD, USCB, or BISON file to capture only the territorial catchments described at the 1, 4, 7, 14 and 18 km distances required using the “buffer” function in QGIS (Appendix C, Figure C1). Finally, a “zonal statistics”

function (Appendix C, Figure C2) was used to calculate the areas or quantities of specific landcover area (environmental stressor) or point data. This data was then transferred onto a spreadsheet in EXCEL for further manipulation.

Habitat Quality Characterization

The overarching goal of this dissertation was to determine proximate fitness outcomes of AMCR at a variety of locations within the rural to urban landscape gradient. This research was done to better understand foundationally how disease pathologies might develop based on stress levels (and its analog GC levels). These GC levels vary within the rural to urban landscape gradient and by extension by environmental stressors (as an analog for habitat quality) spatially. Habitat quality and its result - habitat selection are key components of biodiversity. Biodiversity has been found to alter disease producing capability of various species. I explored this relationship by testing the reliability of GC levels at various locations via F tests, R^2 coefficient of correlation and testing these values (on a county wide basis) against AMCR mortality due to West Nile virus (WNV) from previous research I had conducted. Finally, I developed a Habitat Quality Stress Index (HQSI) of a broader series of characteristics that impact habitat quality which included biotic and anthropogenic environmental stressors across a spatial landscape gradient. The HQSI denotes physical areas of stress based on GC levels resulting from environmental stressors tied to the catchment levels previously described. In addition, the HQSI extends the individual territorial catchments of each sample beyond the home range. This is to show how additional travel distance and the life history interface of AMCR in other habitat areas can significantly impact habitat quality and thus stress level. This HQSI could be potentially used as a tool to determine areas of optimal and suboptimal habitat quality from a conservation and public health perspective to optimize biodiversity and thus act as a notional firewall against

the spread of various disease pathologies. The environmental stressors being evaluated are listed below in Table 3.1.

Table 3.1

Environmental Stressors

A	Biotic Stressors		B	Anthropogenic stressors	
	Floral	Faunal			
1	Other Grasses	1 Non-human population	1	Developed Area (Impermeable Surface - Buildings, Roads)	
2	Deciduous Forest	Mammalia and Aves	2	Agriculture	
3	Coniferous Forest		3	Utility ROW	
4	Water		4	Barren Land	
5	Non-forested Wetland		5	Human population	
6	Forested Wetland		6	Turf and grasses	
7	WWE				
8	WWES				

Biotic Characterization

All 13 sites exist within the Eastern Broadleaf Forest Province and Lower Connecticut River Valley Ecoregion (221A1; Novak, 2023; US Forest Service, 2023). I conducted a characterization of typical flora within this ecoregion of coniferous and deciduous trees and groundcover consisting of a 150-acre site in north central Connecticut (Grabarz, 2018a). Typical associations of coniferous species include Pitch Pine (*Pinus rigida*), Red Pine (*Pinus resinosa*), and Eastern White Pine (*Pinus strobus*). Typical associations of deciduous trees include White Oak (*Quercus alba*), Pin Oak (*Quercus palustris*), Black Oak (*Quercus velutina*), Red Oak (*Quercus rubra*), American Beech (*Fagus grandifolia*), Sugar Maple (*Acer saccharum*), Red Maple (*Acer rubrum*), Norway Maple (*Acer platanoides*), White Birch (*Betula papyrifera*), Silver Birch (*Betula pendula*), River Birch (*Betula nigra*), and White Ash (*Fraxinus americana*). Ground cover associations included: Common mullein (*Verbascum thaspis*), Wintercress (*Barbarea vulgaris*), Field Violet (*Viola Avensis*), Field Peppergrass (*Lampidium capestrium*), Staghorn Sumac (*Rhus typhina*), Sassafras (*Sassafras albidum*), Red Chokeberry (*Aronia*

arbituifolia). Mountain Woodsorrel (*Oxalis montana*), and Cinammon Fern (*Osmunda cinammonea*) et alia.

Numerically the physical area of these stressors was converted to a percentage for biotic floral land cover stressors (Appendix C, Figure C2). These floral landcover environmental stressors include deciduous trees, coniferous trees, grasses other than turf, non-forested wetland, forested wetland, and woody wetlands (WWE). For biotic faunal stressors, I used a whole number value for gamma (γ) biodiversity for non-human animal species within the taxonomic classes Aves and Mammalia using the Biodiversity in Support of Our Nation (BISON) website for presence/absence characteristics (Nation, 2019) for the last 20 years (the maximum age of AMCR (Bent, 1964).

Anthropogenic Characterization

Anthropogenic characteristics (which are also converted to a percentage based on its surface area) included developed (impermeable) area including buildings, roads and other impermeable human made surfaces and structures, turf, agricultural area, and utility ROW (GIS Data, 2020; USGS, 2020).

For human population determination, I used US Census Bureau census blocks from 2020 at each of the sites. The census block is the smallest division of census area. Each block contains 600-3,000 people and is bounded by a physical demarcation such as a road, building or natural feature such as a river, thus making it easier to field locate. Due to its small physical or granular scale, it is the most reliable in terms of population number derivation (US Census Bureau, 2012).

Field Data Collection

I used fecal sampling to determine GC levels within the catchment areas. This sampling was conducted by opportunistic collection which eliminates handling stress on the subject.

Within each site under, around or adjacent to the roosts, samples were collected directly from the ground surface. Samples identified as from AMCR were determined based upon a general size determination of approximately 24.26 mm (approximately equal to US quarter dollar diameter). Entering the site from the same location each time, a consistent zig zag pattern was used for 30 minutes to identify new samples. Along that path, any samples identified were scraped from the ground surface and deposited into a 1.5 mL Eppendorf vial. The Eppendorf vial was stored in a polyethylene bag with ice. A sample number was written on the vial and within a logbook, along with the sample number, GPS coordinates, ambient temperature, weather conditions were recorded and numbers of AMCR present. Returning from the field, samples were packed in dry ice in a Styrofoam container and mailed overnight to the St Louis Zoological Society for characterization by their Endocrinological Laboratory.

Collection Periodicity

Sampling took place from November 2019–April 2020 and October 2020–April 2021.

Each site was visited once per week on a Saturday between 0800 and 1200 (GMT-5).

GC Processing

- Fecal sampling characterization (K. Kozlowski, St Louis Zoo Endocrinology Laboratory, personal communication, 2021).
- Preparation

In the laboratory, the samples were mixed evenly to distribute urates and fecal material. Then, 0.5 grams of the sample was added to a scintillation vial (low potassium borosilicate glass vial for maximal radioactive gamma transference. A 2.5 mL stock extraction buffer (phosphate buffered saline [PBS]) is added to the vial to maintain a constant pH of 7.4. A 25 uL β -Glucuronidase Arylsulfatase (GC enzyme) is added to speed the extraction process by breaking the molecular bonds in the analyte. The sample is then vortexed and incubated overnight at

37°C. Finally, 2.5 mL of methanol is added to the sample and vortexed. The analyte is shaken for 4 hours, decanted, spun, and frozen.

Hormone Immunoassay

Radio immunoassay (RIA) is used to test the levels of circulating GCs in AMCR specimens primarily due to the relatively small size of avifauna fecal matter (detection can be achieved with < 10 uL (MP Biomedicals, 2019). By comparison one drop of water = 50 uL. Many other circulating metabolites in body fluids can alter analytical outcomes of various testing processes through cross reaction by similar chemical properties. RIA uses a highly specific antibody/antigen immune reaction technique with a high specificity to the analyte being tested for, as the analytical process itself is quantifying radioactive decay of one of the compounds' compatible enzyme binding sites, rather than the compound itself (Yalow & Berson, 1960). The decay process uses a gamma counter to convert visible light into an electrical signal that quantifies the concentration of the analyte based on the competition for that enzyme binding site. This procedure is used in a wide variety of analytes, species, and sample types. From an analytical chemistry processing standpoint, RIA achieves this outcome with a minimal amount of other purification steps and laboratory skills, thus making it a highly economical process from a throughput standpoint. The results of this RIA characterization yield levels of glucocorticoids (corticosterone) in ng/g.

Data Analysis

Analysis of the data began with using ordinary least squares (OLS) univariate regression from the statistical program function of EXCEL Office 365 using the Data Analysis Add-On.

In accordance with my first research question, I determined the magnitude of GCs (as an analog for stress response in AMCR) at and compared with multiple site locations using a whisker plot. I then evaluated the magnitude and changes of landcover percentages using

waterfall charts. Using the changes to landcover percentages I compared those to the GC levels using F -tests to isolate specific types of sites as being of high (low stress) versus low habitat quality (high stress). This high versus low habitat quality evaluation was then followed by examining the R^2 coefficient of determination. To perform this analysis, I used the average values of environmental stressors as the independent variable and the average GC levels as the dependent variable for each site to understand the degree of correlation between the two variables and the direct (+) or indirect (-) relationship across each catchment area size (1, 4, 7, 14, 18 km). I did this because based on the number of environmental stressors evaluated, there could be significant differences within sites as well as catchment territories.

The response to my second research question came from the R^2 coefficient of determination obtained from the first question. The purpose of the second question was to examine how stress and thus stress response would be affected by variation in environmental stressors percentages (as analogs for habitat quality) at each site location and catchment size. More specifically, I examined the environmental stressor and GC level R^2 on average across each catchment size on a per site basis.

Previous research has shown conflicting patterns where in some cases the highest stress was apparent in developed areas where in others it was far more episodic and less definitive (Schoenle et al., 2021). Further, previous analyses relied on a much smaller number of sites and single versus the multiple territories that I evaluated (Bonier, 2012).

The response to my third question examined the strength of the relationship of county-wide mortality of AMCR from WNV, correlated with GC level stress response at multiple sites. In previous research I found a strong correlation between impermeable landcover percentage and AMCR mortality (due to WNV). In this dissertation I explored the potential

AMCR mortality with GC level (stress response) relationship strength through R^2 . I did this using the five counties of the municipal locations for which I had GC data.

Gravity Model for Habitat Quality Stress Index

My fourth and last research question dealt with the development of a three-dimensional stress response index and how its responses compared with the GC levels previously evaluated at multiple catchment sizes that I characterize as a habitat quality stress index. The HQSI was developed through an evaluation of the percentages of spatial area of the biotic and anthropogenic characteristics occurring within each of the habitat catchment areas at 1.0, 4.0, 7.0, 14.0, and 18.0 km area using the single constrained gravity model. The single constrained gravity model was used most recently in international economics (Isard, 1954), previously in city and regional planning (Stewart, 1947; Zipf, 1949) and originally in the derivation of the celestial law of universal gravitation (Newton, 1687). This model, in its original formulation by Newton, measures the magnitude of the attraction between two celestial bodies based on their gravitational pull which varies based on their size and distance between them. My reinterpretation of this model uses the magnitude of the stress response (GC level) multiplied over the percentage area of each of two adjoining catchments, then divided by the squared distance between each of the two adjoining catchments. In the model below F = the overall stress level of the area, M_i = the area of catchment 1 the origin of which is the sample GC. M_j = the area of catchment 2. Then $(M_i * GC) * (M_j * GC) / d^2$ = the distance between the origin of each catchment. The original formulation of Newton's law of universal gravitation is the following where M is the mass of a celestial body, the square of the distance between them = their attraction based on F the gravitational constant.

$$F = \frac{M_i M_j}{d^2}$$

My reinterpretation is the following: $\frac{(M_i * GC)}{d^2}$

As the area of the catchment increases directly, the level of stress responses varies inversely by attenuating or reducing the larger the catchment area, representing a larger area in which to conduct life history. This procedure establishes a HQSI, the magnitude of the index measuring the level of stress response to an environmental stressor. In this way stress response can be identified over an entire catchment area of landcover spatially (of multiple environmental stressors) instead of an individual point and recognizes the stress of travel between each of the catchment origins. More specifically, the stress response as represented by GC hormones is evaluated across multiple habitat catchment areas to simulate various habitat size areas. The result of the indices is an analog for habitat quality that is divided into four levels, the highest index (stress) representing poor habitat quality (red), followed by fair habitat quality (orange), good habitat quality (yellow), and excellent habitat quality (green). The distance between each catchment calculates the stress of energy use through travel between these catchment sites which captures as an analog AMCR's life history. Compared to other indices, the importance of this index is that when aggregated together, it will synthesize multiple (15) environmental stressor effects within a discrete physical area at multiple locations. My work in this dissertation focuses on evaluating the individual environmental stressors and stress response at each site.

CHAPTER IV: RESULTS

My dissertation was a quantitative observational study. I collected samples of fecal matter and used them to determine stress hormone levels of AMCR and thus stress response at a variety of different sites in situ. These AMCR habitat sites (territories and roosts at 13 sites) represented the statistical population.

Fecal samples of AMCR were obtained within the territories and roosts at the sites described in Figure 3.1. The total collection sample size was 153 fecal samples over the total collection period of 12 months over two seasons, 2019 and 2020.

The sampling unit consisted of 13 habitat sites, in the six municipalities of Waterbury, West Hartford, Hartford, New Canaan, Cornwall, and Norwich, Connecticut. The 13 site sampling locations were determined using databases, anecdotal information, and my own physical observation in a variety of land use densities. Developed (impermeable surface) was the primary discriminating factor for selection, and where AMCR currently exists within territories and roosts. I used a random number generator to determine the number of samples to be taken for a roost site, each week to collect data independent and representative of the statistical population.

The independent (X) explanatory variable was as per Table 3.1, the environmental stressors. The dependent (Y) predictor variable was the GC level, isolated via radio immunoassay processes and are listed in Chapter IV Results.

- The statistical null hypothesis was: Circulating steroid hormone levels (glucocorticoids) will not be higher in areas of low habitat quality. I used an F statistic to confirm validity.

- The alternative hypothesis was: Circulating steroid hormone levels (glucocorticoids) will be higher in areas of low habitat quality.

The essence of the statistical analysis was to use the coefficient of determination R^2 to determine the percentage of explanatory power of the environmental stressors with an alpha level of 0.05. Bias could occur in land use quality relationships extending over an area beyond the AMCR roosts. My solution was to calculate the prospective area of habitat quality (i.e., the spatial scale) based on historic research of territory size, which was at the 1, 4, 7, 14, and 18 km areas. Thus, habitat quality as an explanatory (X) variable was based on historic territory size rather than specific AMCR location (other than the original roost location and thus sample location as the origin or centroid). I assumed that catchment (area within the range of AMCR using each roost) was representative of the historic territorial size and therefore used as a habitat quality area for all calculations and analyses. Finally, the HQSI shows an overall habitat quality using an overarching stress level at each of the catchment areas: 1–4 km, 1–7 km, 1–14 km, and 1–18 km using the reinterpretation of the single constrained gravity model previously described.

Examining the data over the 12-month period in Figure 4.1 and Table 4.1, the means of the GC levels are generally higher in the more developed or urbanized areas with means across all the sites from 15.57–45.16 ng/g. As a comparison, data from HormoneBase, a wide-ranging database of ‘unmanipulated free-living organisms’ glucocorticoids (Vitousek et al., 2017) shows GC mean levels from 4.03–22.67 ng/g (1982–1984; Johnson et al., 2017) in the southern New England area (40N–42 N. latitude) for avian species which were the most recent data available for this geographical area and aves class. Thus, my GC sample data was 2x–4x higher than historic published data.

Figure 4.1

GC Mean and Median Samples per Site

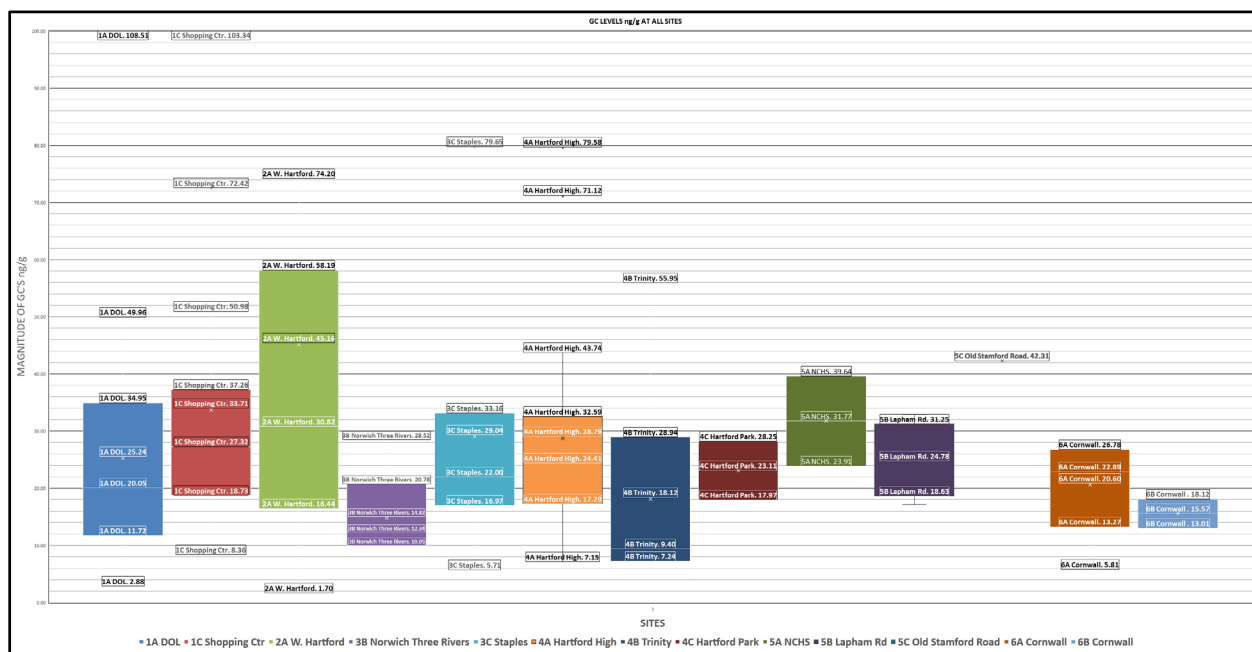


Table 4.1

Descriptive Statistics of GC Samples

Sites	1A DOL	1C Shopping Ctr	2A W. Hartford	3B Norwich Three Rivers	3C Staples	4A Hartford High	4B Trinity	4C Hartford Park	5A NCHS	5B Lapham Rd	5C Old Stamford Road	6A Cornwall	6B Cornwall	
Date Collect	DOL	Shopping Ctr	W. Hartford	Three Rivers	Staples	Hartford High	Trinity	Hartford Park	NCHS	Lapham Rd	Stamford Road	Cornwall	Cornwall	
Sample QU.	23	43.0	17	20	7	18	6	2	2	4	1	5	2	150
MEAN	25.24	33.71	45.16	14.82	29.04	28.79	18.12	23.11	31.77	24.89	42.31	20.60	15.57	MEAN
MEDIAN	20.0	27.32	27.11	12.24	22.00	22.12	9.40	23.11	31.77	24.78	42.31	22.89	15.57	
STDDEV	21.98	26.58	51.56	6.06	22.03	18.53	17.51	5.14	7.86	5.63	0.00	7.78	2.56	

This is more easily grasped when it is equated to one of the levels of environmental stressors that impact on the GC levels. Developed area (impermeable surface) is shown in Figure 4.2, with the average highest percentage environmental stressor on each site. The figure shows the trend of average GC level rising and falling in Developed Area for each of the various catchment areas sizes across all the sites and a trend line slowly decreasing in magnitude as the sites slowly become less stressful (less developed) with GC levels decreasing. A comparison of

the municipal average/community development (impermeable surface %) is shown in Figure 4.3 compared to the specific site and sample locations at the various catchment sizes.

Figure 4.2

Mean % Dev Area 1 km-18 km: GC Mean

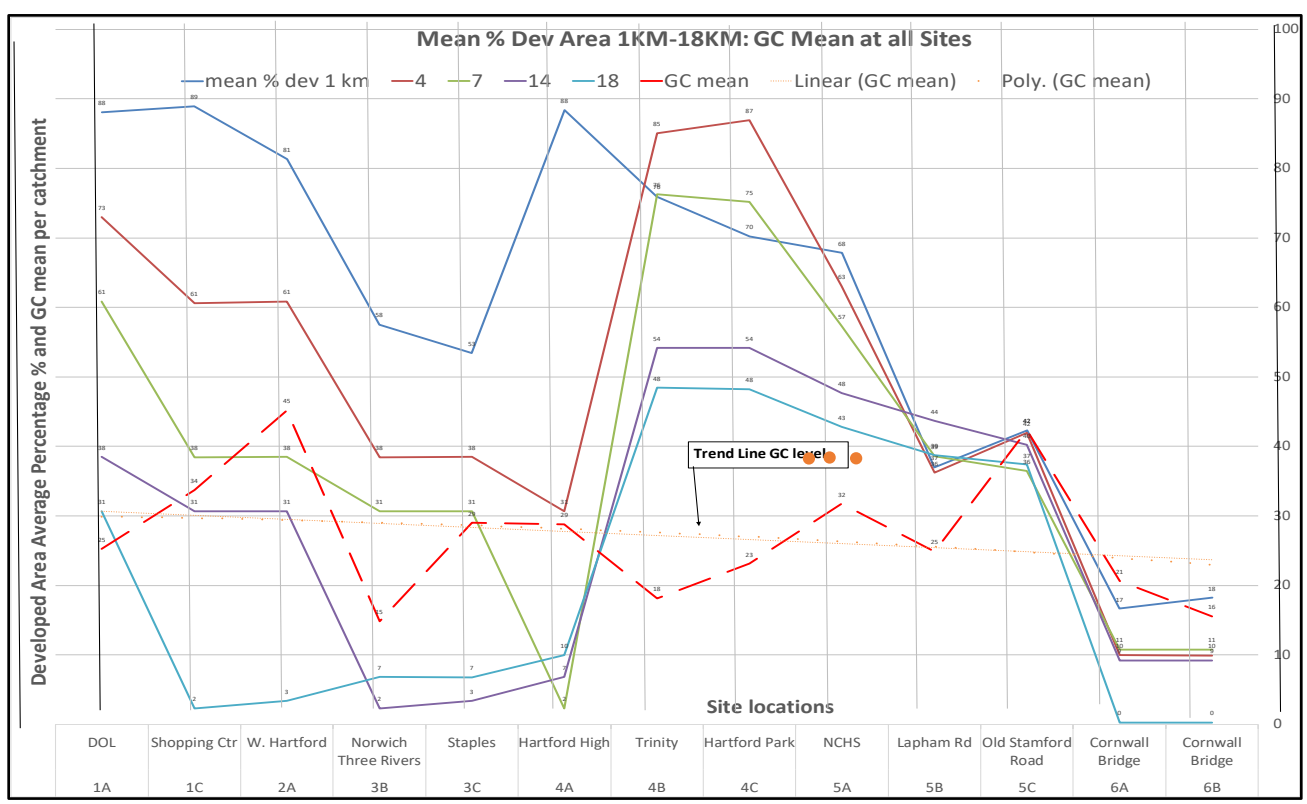


Table 4.2 shows the results of performing *F*-tests of two variances to determine similarities or differences between each of the sites.

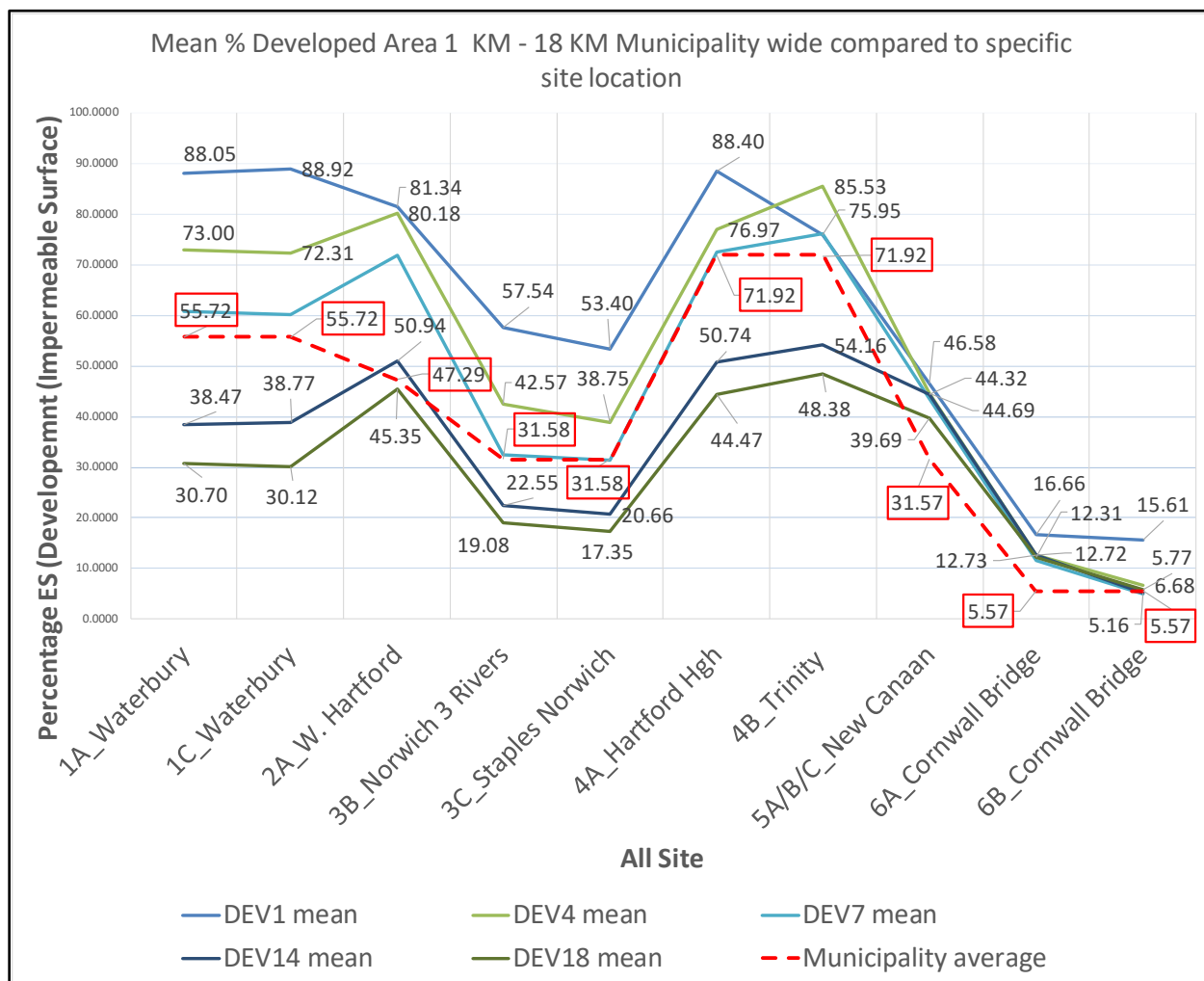
Table 4.2

F-tests of Two Variances Between Each Site

	1A_Waterbury	1C_Waterbury	2A_W. Hartford	3B_Norwich 3 Rivers	3C_Staples Norwich	4A_Hartford High	4B_Trinity	5abc_New Canaan	6ABC_Cornwall Bridge
1A_Waterbury		A	A	R	R	A	R	R	R

Figure 4.3

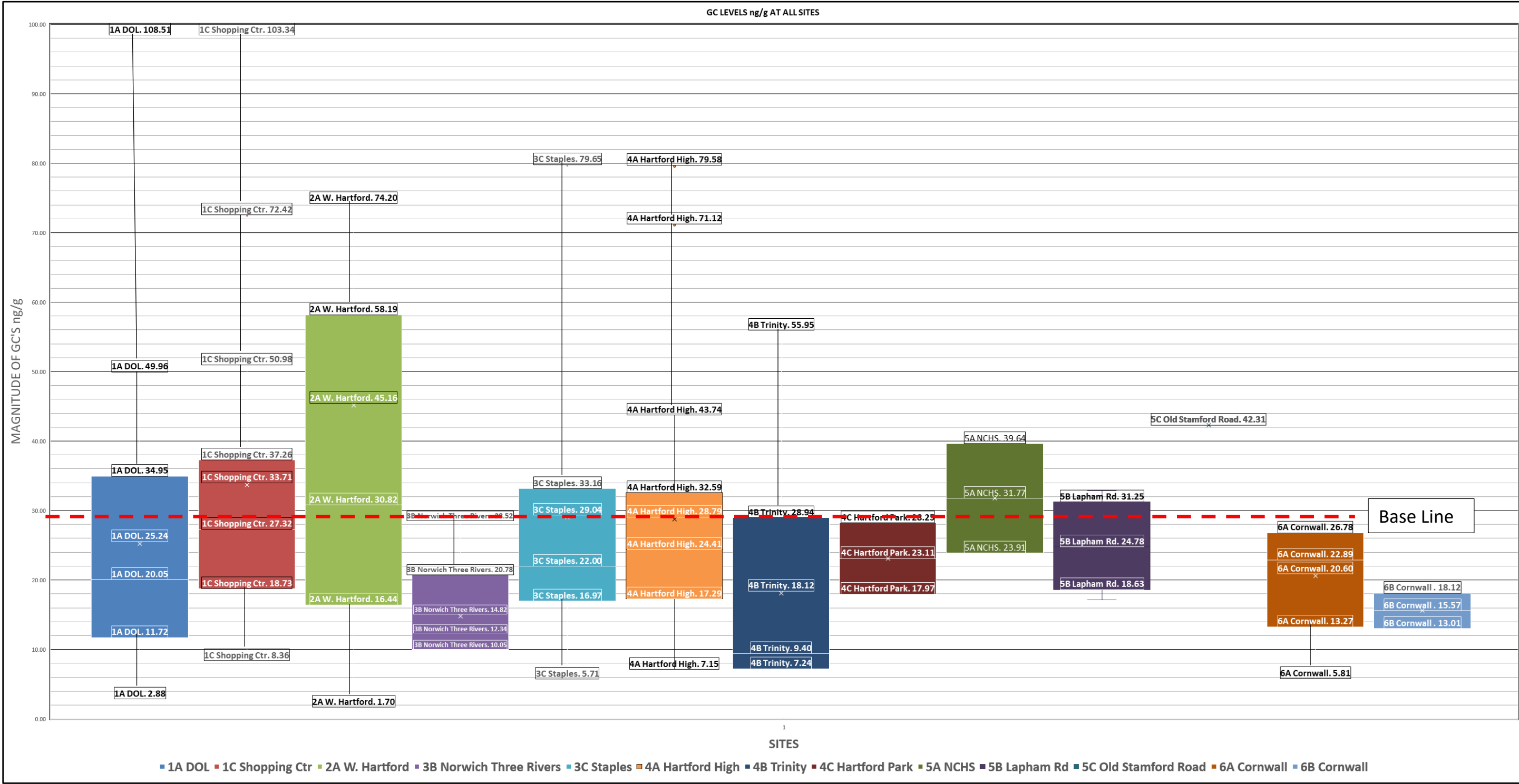
Mean % Dev Area Municipality Wide Compared to Specific Site Locations 1 km – 18 km



A box and whisker plot of all the sites (Figure 4.4) is shown with average GC levels and minimum and maximum outliers. You can also observe a baseline across all sites of between 20–25 ng/g that could be considered baseline homeostasis.

Figure 4.4

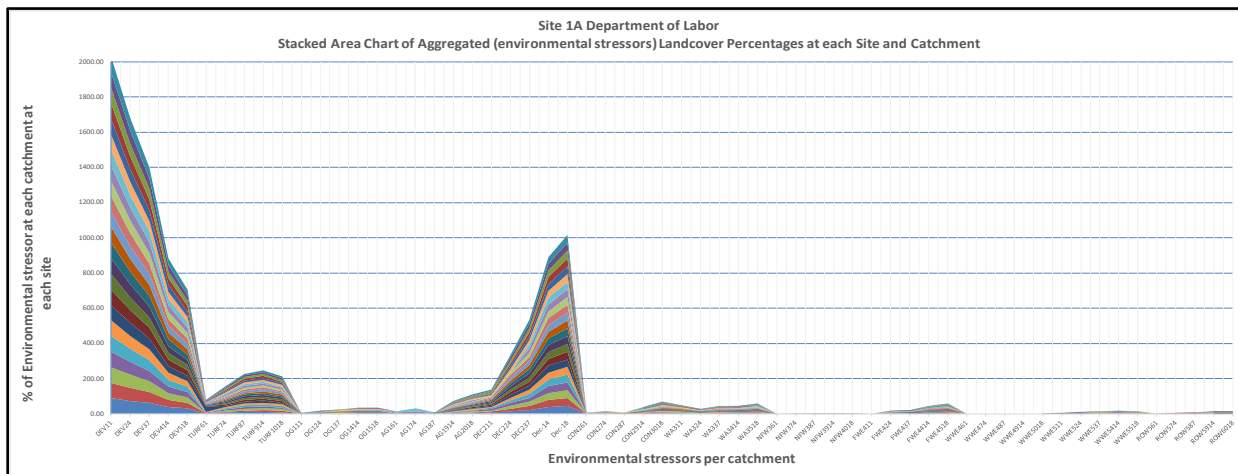
Whisker Plot of All Sites Showing Extent of All GC Levels With Each Sample, as Well as Means and Outliers



The stacked area waterfall charts of Figures 4.5–4.13 and Appendix E collectively show the percentage of land cover for each of the catchment areas for each site 1–10. In general, these waterfall charts show the stochastic nature of the sites environmental stressors (landcover). Note the wavy line indicating variation in each of the environmental stressors in a nonlinear relationship. Further, the abrupt changes between many of the stressors shows the dramatic transition between the landcover stressor types.

Figure 4.5

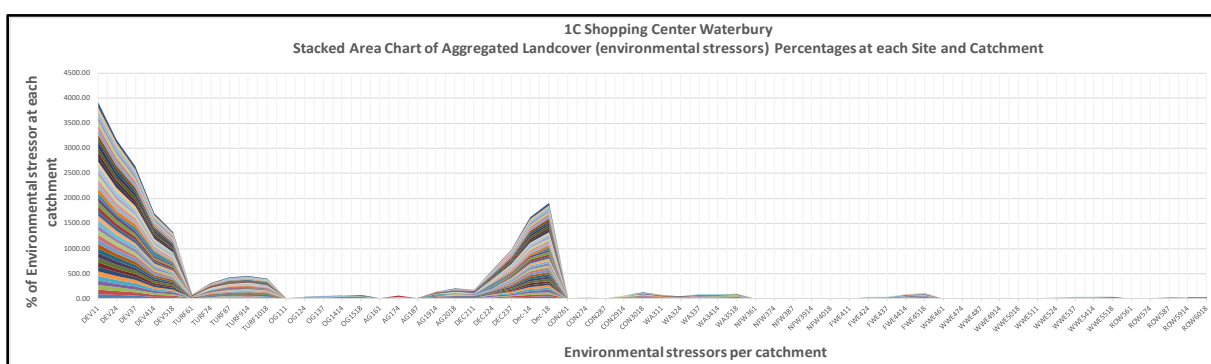
Site 1A Department of Labor Stacked Waterfall



For instance, in Figure 4.5 above, each of the different stacked colored areas on the vertical axis represent a different sample location at the Site 1A Department of Labor location in Waterbury, Connecticut. The thickness of the environmental stressor (landcover characteristic) represents the percentage % magnitude of the stressor. Along the horizontal axis are each of the environmental stressors at the 1–18 km catchment areas. Thus, the figure reduces what would otherwise be a 60 x 23, or 1,380 cell matrix (for Site 1A) into one chart. Specific to this site there were 23 samples taken, and due to the highly urbanized nature of the site, developed area (DEV) is very high measuring between 87%–88% of land cover or totaling 1937% adding all those different site location samples together. The developed area magnitude gradually descends from the 1 km catchment location to the 18 km catchment location. It then increases again somewhat for the next major stressor, turf (TURF). Across other grasses (OG) and agricultural space (AG), the percentage of the area remains negligible until it reaches deciduous tree cover (DEC) where it increases again to its highest point at the line of the 18 km catchment area. The balance of the stressors at this urbanized location are negligible.

Figure 4.6

Site 1B Shopping Center Stacked Waterfall

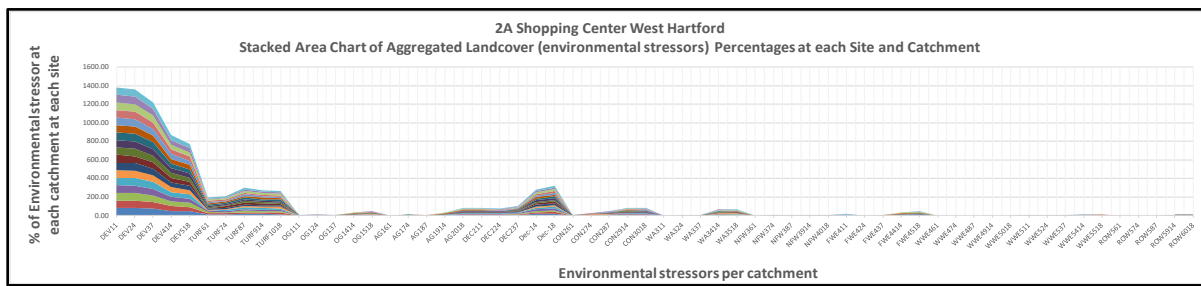


Site 1B Shopping Center, again in Waterbury, Connecticut (Figure 4.6) shows a larger number of samples 43 and thus partly a higher percentage of magnitude along the vertical axis but also a higher percentage of developed area (DEV) from 88%–92% at the 1 km catchment area. Turf (TURF) is also higher partly because of the larger number of samples but also by its width on the horizontal axis that there is a broader range of turf from 1.6%–6.9%. Further, there is a sharper rise and decline in deciduous tree (DEC) area from 0.18% to 0.48% at the 1 km catchment. This indicates a “patchier” or what I call extracellular habitat, abrupt changes in specific

landcover type. Finally, there are modest amounts of coniferous tree (CON) cover 0.09% to 1.9% at the 1 km level; water (WA; the site is along the Naugatuck River), and forested wetland (FWE) 0.74%.

Figure 4.7

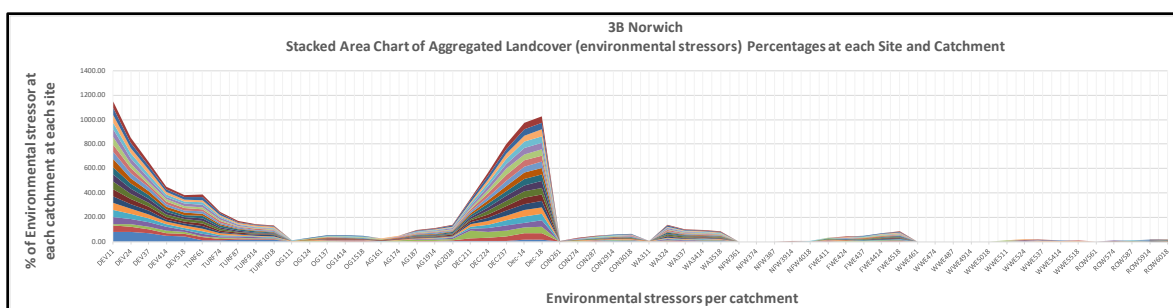
Site 2A West Hartford, CT Stacked Waterfall



Site 2A West Hartford, Connecticut (Figure 4.7) consisted of 17 samples that varied in magnitude for developed area (DEV) from 79%–82%. The connection between developed area and turf (TURF) 11%–13% indicates a gradual transition between the two landcover stressor types. Next, the transition between agricultural (AG) 4.5%–4.7% area and deciduous tree (DEC) covered area 4.3%–5.5%. Finally, coniferous trees (CON) 0.27%–0.64%, lake waterbody (WA) 0.05%–0.31%, and forested wetland (FEW) 1.19%–2.69% are the remaining significant environmental stressors for this site.

Figure 4.8

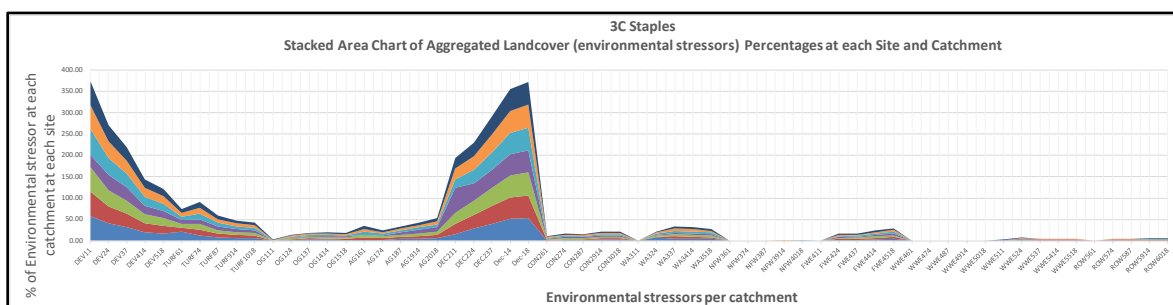
Site 3B Three Rivers College, Norwich, CT Stacked Waterfall



Site 3B Norwich, Connecticut (Figure 4.8) consisted of 20 samples that varied in magnitude for developed area (DEV) from 14%–80%. Turf (TURF) was 8%–23% and gradually transitioned from DEV to TURF. There was also a smooth transition from other grasses (OG) at 0.09%–2% to agricultural use at 0.18%–1.5%. and finally, a sharp increase to deciduous (DEC) cover at 5%–65%. Lastly, minor amounts of coniferous (CON) at 0.09%–2%, water (WA) Thames River 1.2%–2.6%, and forested wetland (FEW) at 0.18%–2.4%.

Figure 4.9

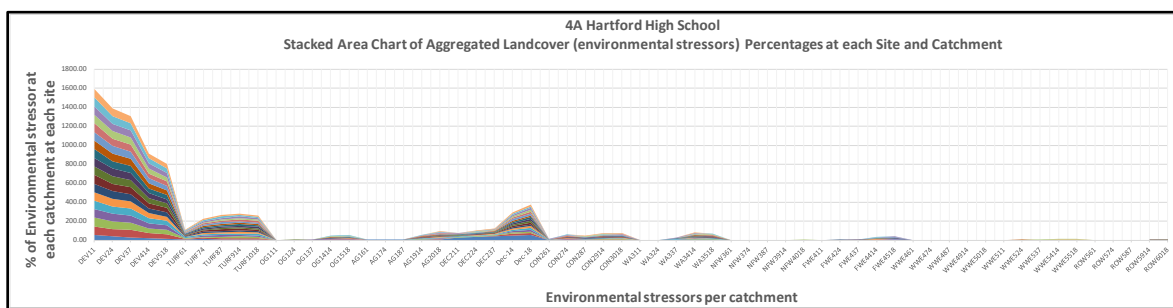
Site 3C Staples, Norwich, CT Stacked Waterfall



Site 3C Staples Norwich, Connecticut (Figure 4.9) consisted of 7 samples that varied in magnitude for developed area (DEV) from 57%–60%. Turf (TURF) was 8%–21% and gradually transitioned from developed area to turf to other grasses (OG) at 0%–0.45%, then agriculture (AG) at 1.5%–7%. Landcover stressors then rapidly transitioned to deciduous trees (DEC) at 15%–53%. Finally, this sites landcover concluded with small amounts of coniferous trees (CON) at 0.2%–1.9%., water (WA) by the Thames River 0.73%, and lastly forested wetlands (FWE) at 1.8%–2.4%.

Figure 4.10

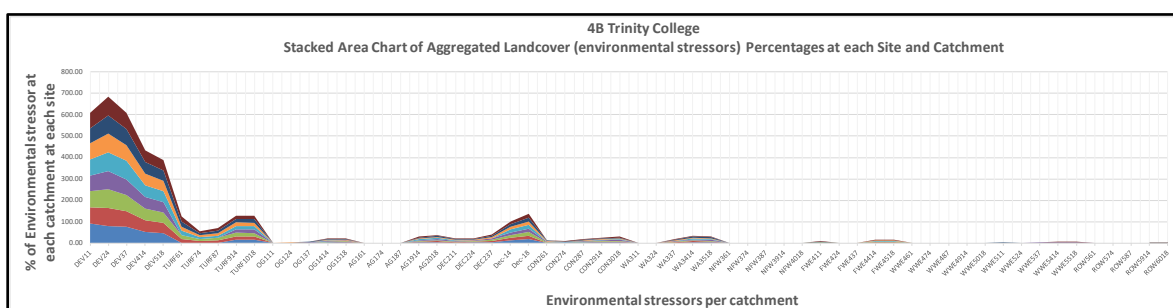
Site 4A Hartford High School, Hartford, CT Stacked Waterfall



Site 4A Hartford High School Hartford, Connecticut (Figure 4.10) consisted of 18 samples that varied in magnitude for developed area (DEV) from 54%–92%. Turf was 4.7%–5.8% with a distinct differentiation from DEV as well as from other grasses (OG) at 0.09%–0.45%. There was a negligible amount of agriculture (AG) in this area starting at 7.3%, followed by a spike in deciduous tree cover (DEC) at 2.5%–27.36%. water (WA) as drainage channels was 0.07%–2.7%, and lastly, forested wetlands (FWE) at 0.24%–2.4%.

Figure 4.11

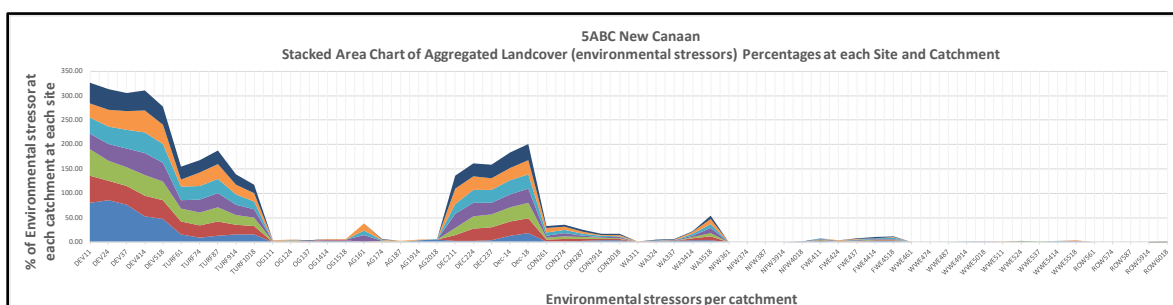
Site 4B Trinity College, Hartford, CT Stacked Waterfall



Site 4B Trinity College in Hartford, Connecticut (Figure 4.11) consists of 8 samples that varied in magnitude for developed area (DEV) from 70%–90%. Turf was 18%–24% with a gradual transition from developed area. There were minimal other grasses (OG) and agriculture (AG). deciduous tree cover (DEC) at 2.4%–3.0%. Finally, forested wetlands (FWE) were 0.45%–2.8%.

Figure 4.12

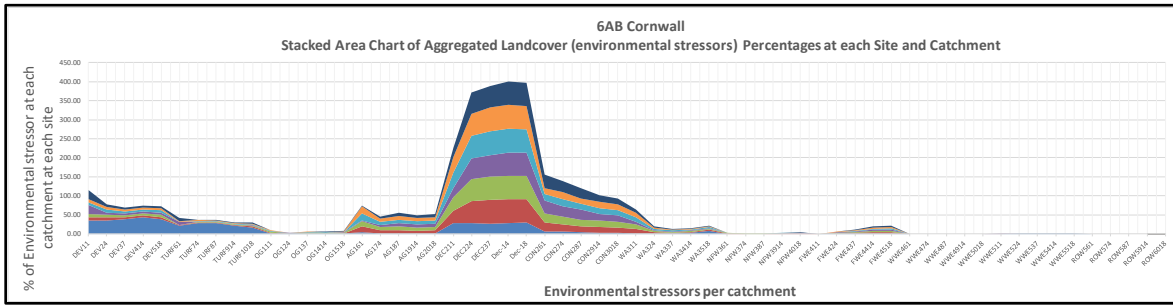
Site 5ABC New Canaan NCHS, Lapham Road, and Old Stamford Road Stacked Waterfall



Site 5ABC – NCHS, Lapham Road, and Old Stamford Road, New Canaan, Connecticut (Figure 4.12) consists of seven samples that varied in magnitude for developed area (DEV) from 28%–51%. Turf was 14%–28% with a gradual transition from developed areas. There were minimal other grasses (OG) and agriculture (AG), and deciduous tree cover (DEC) at 2%–30.0%. Finally, forested wetlands (FWE) were 0.50%–2.8%.

Figure 4.13

Site 6AB River Road, Cornwall Bridge, CT Stacked Waterfall



Site 6 AB Cornwall Bridge, Connecticut (Figure 4.13) consists of 8 samples that varied in magnitude for developed area (DEV) from 7.8%–35% which was minor. Turf was 0.82%–21% with a gradual transition into other grasses (OG) at 0.7%–2.2% and the agricultural area (AG) at 0.98%–9.8%. These blended rapidly into deciduous cover (DEC) at 23%–41% and to coniferous cover at 5%–34%.

To show the disparity by the lack of linearity between environmental stressors, the first series of line graphs below (Figures 4.14–4.21) depicts the difference between each stressor at each of the catchment sizes of 1, 4, 7, 14, and 18 km. This shows that lack of linearity in the magnitude of various stressors as they change in size, that I hypothesize intensifies stress.

Figure 4.14

Environmental Stressor - % Development Mean 1 km – 18 km Catchment versus GC Mean

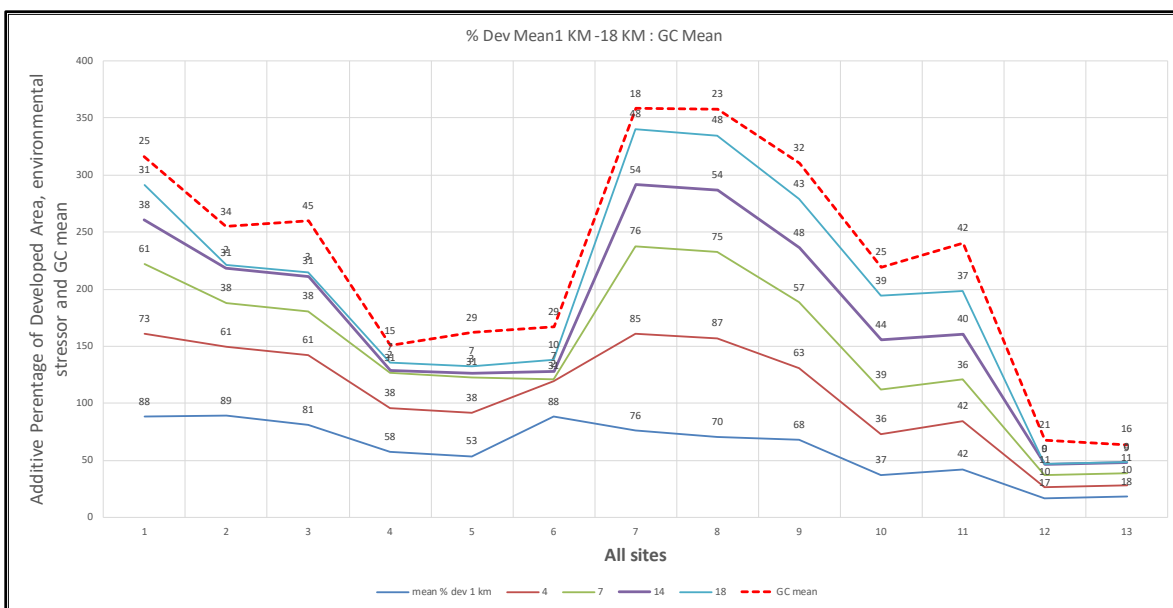


Figure 4.15

Environmental Stressor – % Turf Mean 1 km -18 km Catchment versus GC Mean

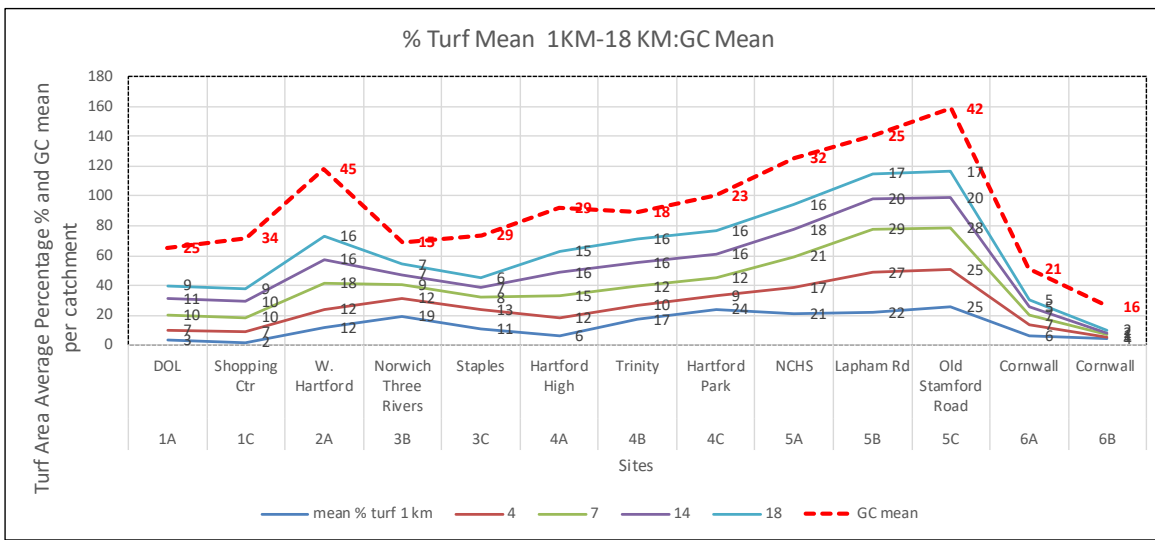


Figure 4.16

Environmental Stressor – % Other Grasses Mean 1 km -18 km Catchment versus GC Mean

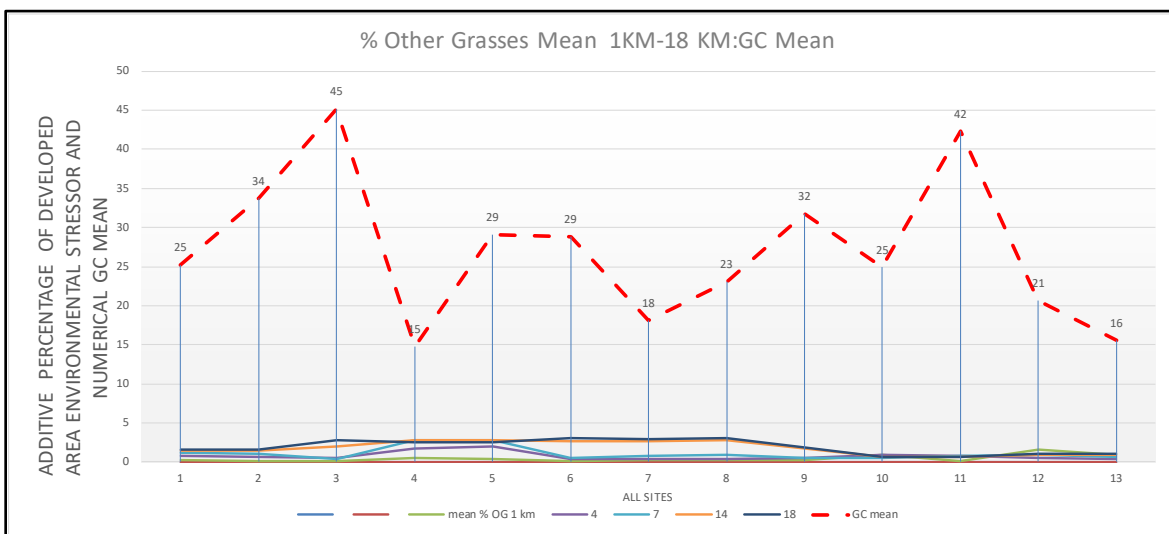


Figure 4.17

Environmental Stressor – % Agriculture Mean 1 km -18 km Catchment versus GC Mean

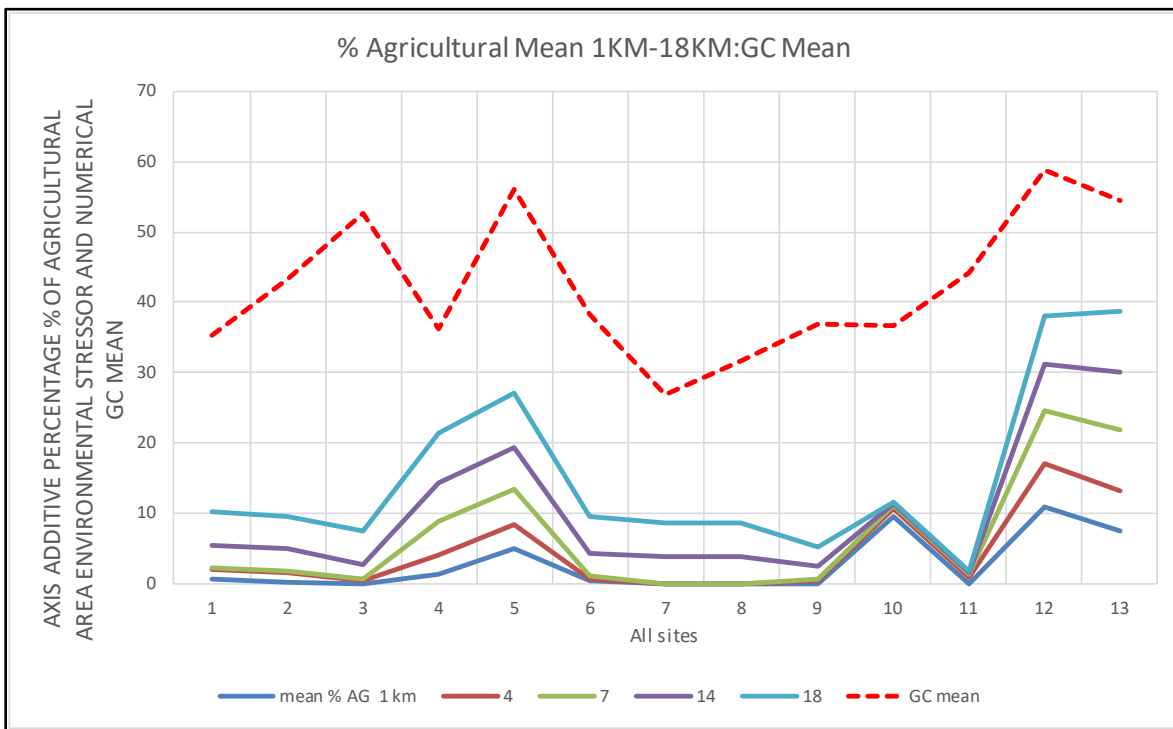


Figure 4.18

Environmental Stressor – % Deciduous Mean 1 km -18 km Catchment versus GC Mean

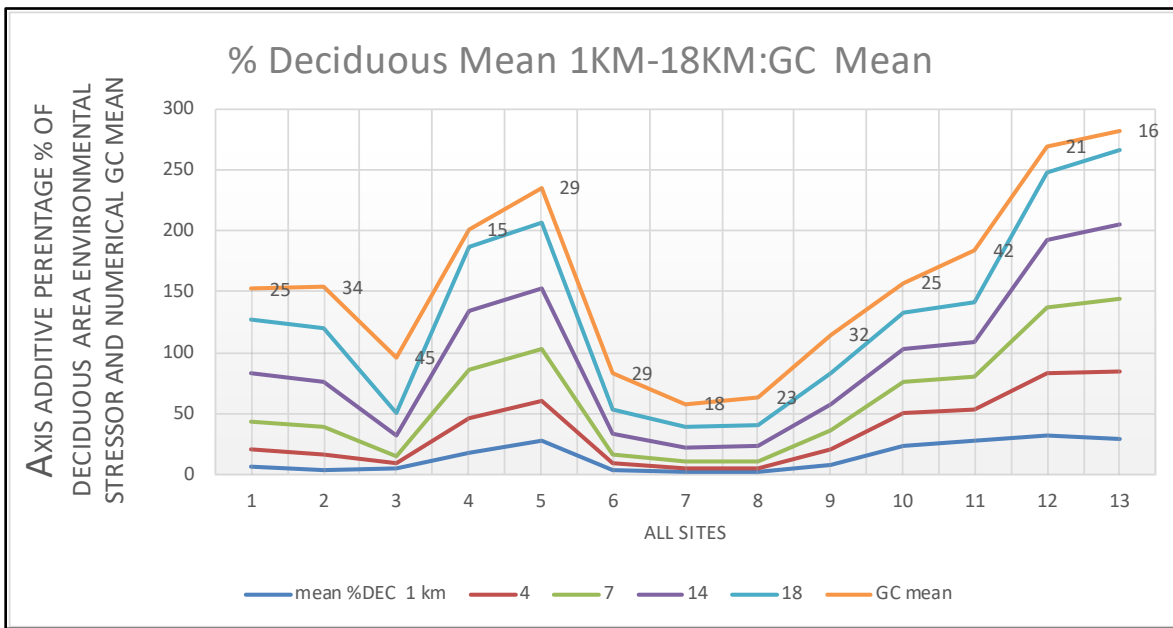


Figure 4.19

Environmental Stressor – % Coniferous Mean 1 km -18 km Catchment versus GC Mean

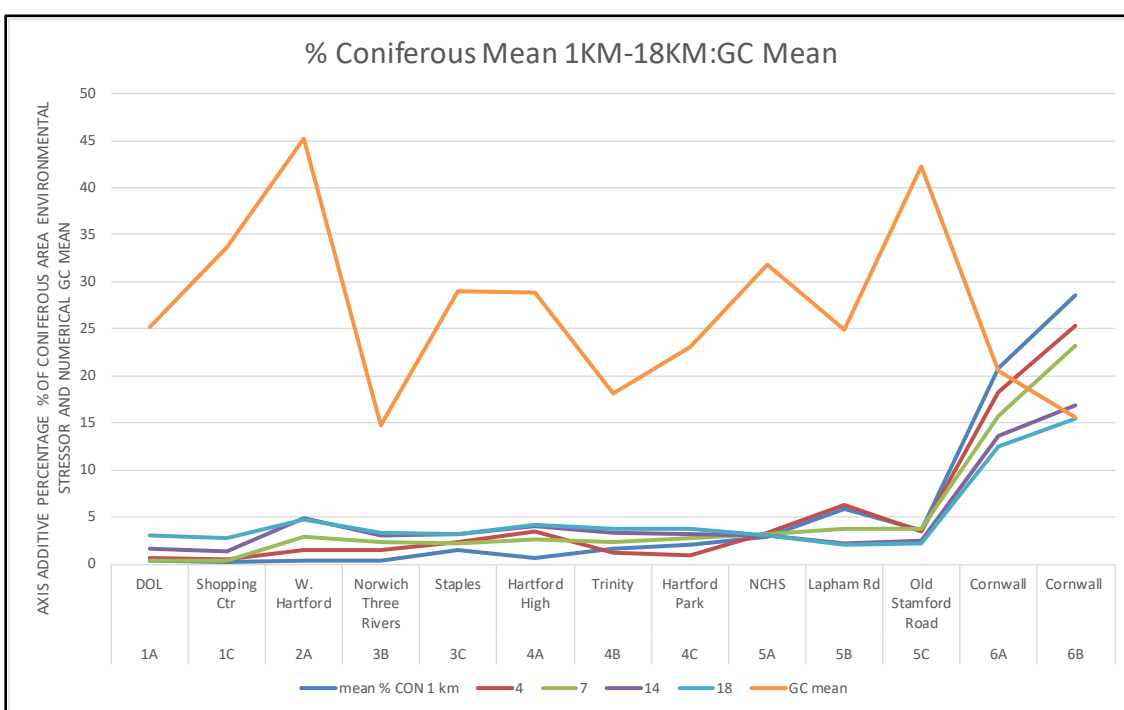


Figure 4.20

Environmental Stressor – % Census Mean 1 km -18 km Catchment versus GC Mean

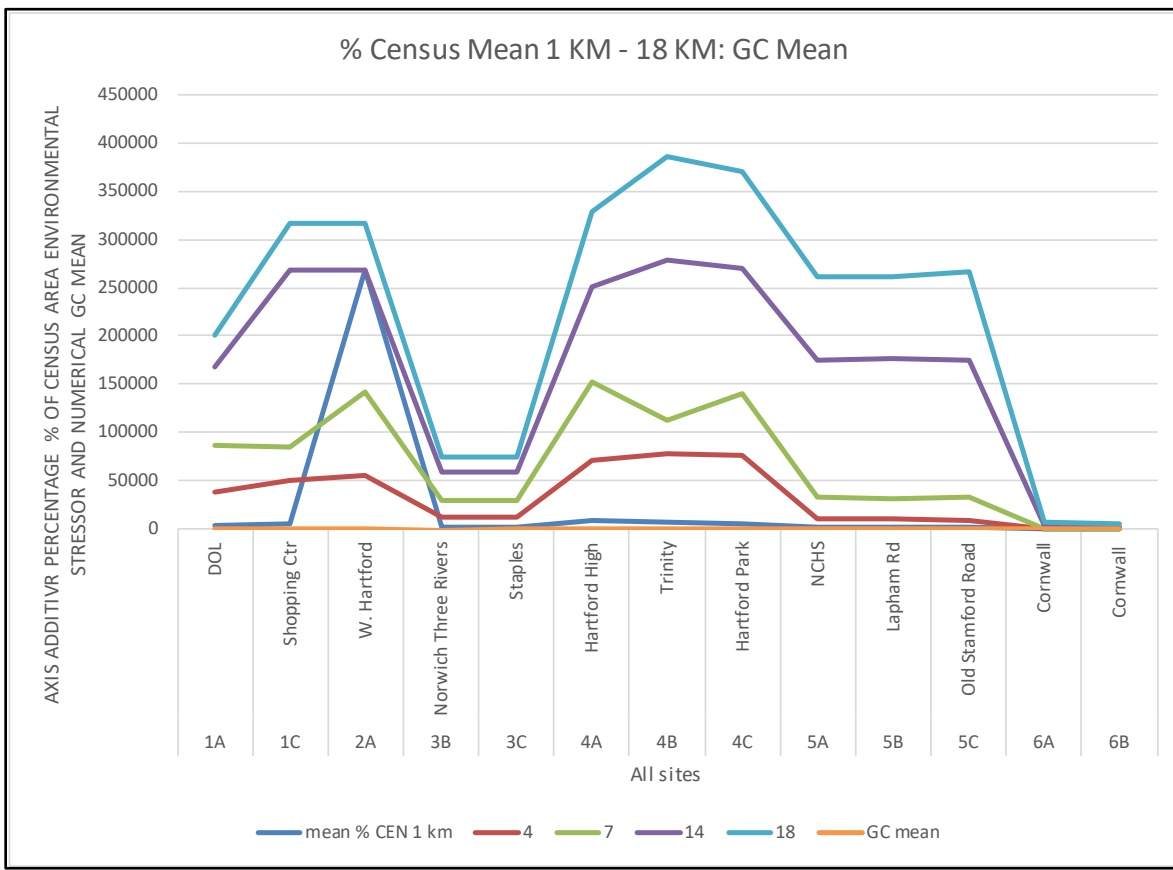
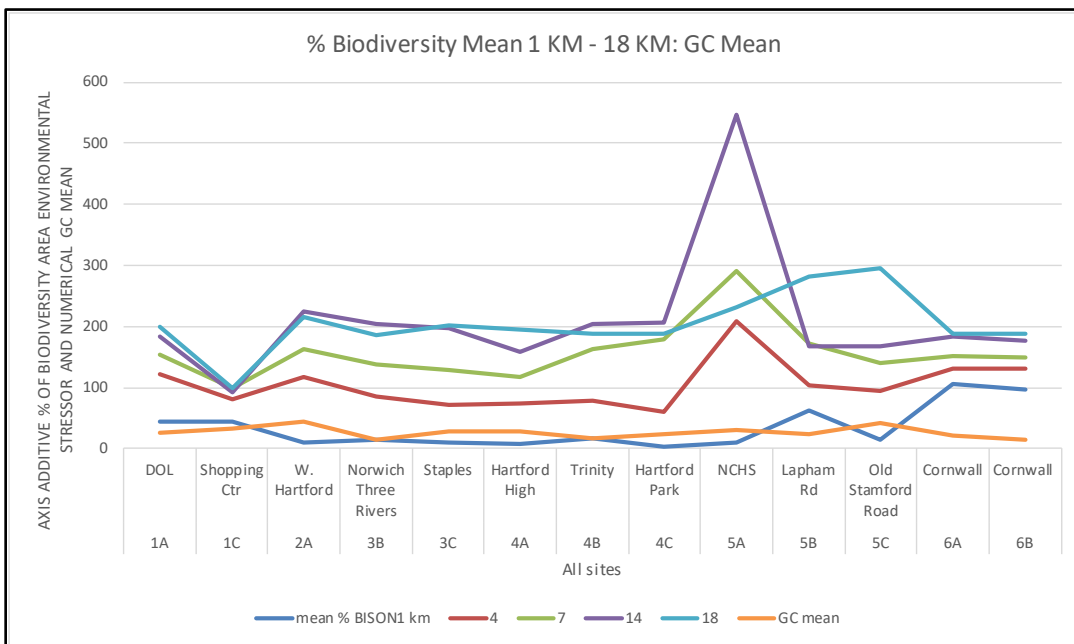


Figure 4.21

Environmental Stressor – % Biodiversity Mean 1 km -18 km Catchment versus GC Mean



Tables 4.3–4.12 below show the average percentage of each environmental stressor/GC sample for each site at each catchment size along with the GC sample mean for each of the sites. Several of the GC samples at the more urbanized sites were spatially close in proximity (< 5 m), aggregating and averaging all the samples at each site gave a more pragmatic response. Lastly, for each environmental stressors, the average R^2 value of all sites for each catchment size is given at the end. (Note: Darkness of blue highlight indicates higher R^2 value).

Table 4.3

Developed - Environmental Stressor Average Percentages per Site at Different Catchment Diameters and Average R²/ Catchment Diameters per Environmental Stressors

Developed	Site	GC mean	% Area				
			DEV1 mean	DEV4 mean	DEV7 mean	DEV14 mean	DEV18 mean
	1A_Waterbury	25.23	88.0491	73.0042	60.851006	38.46768	30.69529752
	1C_Waterbury	32.95	88.9211	72.3057	60.068697	38.76886	30.11654331
	2A_W. Hartford	45.16	81.3410	80.1814	71.848135	50.93501	45.34723035
	3B_Norwich 3 Rivers	14.82	57.5390	42.5729	32.49607	22.5527	19.07709833
	3C_Staples Norwich	29.04	53.4042	38.7482	31.328382	20.65831	17.35221784
	4A_Hartford Hgh	28.79	88.3991	76.9681	72.479836	50.73713	44.46680785
	4B_Trinity	19.37	75.9527	85.5290	76.016314	54.16073	48.37945666
	5A_New Canaan	29.34	46.5827	44.6869	43.618688	44.31981	39.68585261
	5B_Cornwall Bridge	20.60	16.6575	12.7254	11.668809	12.71904	12.30848515
	5C_Cornwall Bridge	15.57	15.6107	6.6779	5.0170132	5.162782	5.765844692
	Site		DEV1 mean	DEV4 mean	DEV7 mean	DEV14 mean	DEV18 mean
	R²_DEV		0.2754	0.2793	0.3051	0.3177	0.2981
	relationship		(+)	(+)	(+)	(+)	(+)

Table 4.4

Turf- Environmental Stressor Average Percentages per Site at Different Catchment Diameters and Average R²/ Catchment Diameters per Environmental Stressors

Turf	Site	GC mean	% Area				
			TURF61	TURF64	TURF67	TURF614	TURF618
	1A_Waterbury	25.23	3.4218	6.7428	9.9269	10.76357	10.7636
	1C_Waterbury	32.95	1.956792908	6.8899	9.7465	10.36562	10.3656
	2A_W. Hartford	45.16	13.02395086	12.3690	17.5511	16.04529	16.0453
	3B_Norwich 3 Rivers	14.82	19.38016529	12.2338	8.5290	7.295187	7.2952
	3C_Staples Norwich	29.04	10.54702873	12.9530	8.4669	6.737132	6.7371
	4A_Hartford Hgh	28.79	5.902458933	12.4743	14.6916	15.63688	15.6369
	4B_Trinity	19.37	18.96235078	9.5718	12.1787	16.01693	16.0169
	5A_New Canaan	29.34	22.03856749	23.9172	26.7929	19.76773	19.7677
	5B_Cornwall Bridge	20.60	6.483011938	6.8271	6.7395	5.427067	5.4271
	5C_Cornwall Bridge	15.57	2.969084787	1.3694	1.1771	1.501797	1.5018
	Site		TURF61	TURF64	TURF67	TURF614	TURF618
	R²_TURF		0.0032	0.1244	0.3100	0.2954	0.2684
	relationship		(-)	(+)	(+)	(+)	(+)

Table 4.5

Other Grasses- Environmental Stressor Average Percentages per site at Different Catchment Diameters and Average R²/ Catchment Diameters per Environmental Stressors

Other Grasses	Site	GC mean	% Area				
			OG111	OG124	OG137	OG1414	OG1518
	1A_Waterbury	25.23	0.2755	0.7833	1.1540	1.521808	1.5594
	1C_Waterbury	32.95	0.1845	0.7000	1.1196	1.471414	1.5615
	2A_W. Hartford	45.16	0.1690	0.5903751	0.4190	2.012011	2.772093514
	3B_Norwich 3 Rivers	14.82	0.5372	1.6706585	2.7925	2.791945	2.478987513
	3C_Staples Norwich	29.04	0.3804	2.0703089	2.7437	2.757927	2.558030436
	4A_Hartford Hgh	28.79	0.0816	0.3441265	0.5658	2.702638	3.115956621
	4B_Trinity	19.37	0.2410	0.4406794	0.8797	2.665345	2.958327249
	5A_New Canaan	29.34	0.6166	0.785835	0.5741	0.946658	0.994246548
	5B_Cornwall Bridge	20.60	1.5611	0.5349339	0.8779	0.867757	1.045232921
	5C_Cornwall Bridge	15.57	0.9183	0.409844	0.6131	0.961327	1.081364288
	Site		OG111	OG124	OG137	OG1414	OG1518
	R²_OG		0.2432	0.0073	0.0976	0.0005	0.0565
	relationship		(-)	(-)	(-)	0	(+)

Table 4.6

Agricultural - Environmental Stressor Average Percentages per Site at Different Catchment Diameters and Average R²/ Catchment Diameters per Environmental Stressors

Agricultural	Site	GC mean	% Area				
			AG161	AG174	AG187	AG1914	AG2018
	1A_Waterbury	25.23	0.6029	1.4173	0.2872	3.107191	4.7549
	1C_Waterbury	32.95	0.2798	1.3341804	0.1849115	3.265543	4.526773312
	2A_W. Hartford	45.16	0.0000	0.5429548	0.1387	2.033725	4.87022638
	3B_Norwich 3 Rivers	14.82	1.4141	2.5630904	4.8733468	5.552732	6.918037823
	3C_Staples Norwich	29.04	4.9456	3.4829805	4.9017013	6.104286	7.595112736
	4A_Hartford Hgh	28.79	0.4081	0.2211086	0.5391724	3.149232	5.157579268
	4B_Trinity	19.37	0.0000	0	0	3.732717	4.778280239
	5A_New Canaan	29.34	5.4834	0.8574259	0.3937348	0.732455	0.964184793
	5B_Cornwall Bridge	20.60	10.8173	6.3131519	7.421172	6.59471	7.027694034
	5C_Cornwall Bridge	15.57	7.4686	5.6853719	8.6514454	8.210676	8.803322635
	Site		AG161	AG174	AG187	AG1914	AG2018
	R²_AG		0.1434	0.2443	0.3544	0.4236	0.2086
	relationship		(-)	(-)	(-)	(-)	(-)

Table 4.7

Deciduous - Environmental Stressor Average Percentages per Site at Different Catchment Diameters and Average R²/ Catchment Diameters per Environmental Stressors

Deciduous	Site	GC means	% Area				
			DEC211	DEC224	DEC237	Dec-14	Dec-18
	1A_Waterbury	25.23	6.0535	14.5414	23.2034	38.76447	44.1731
	1C_Waterbury	32.95	4.1445	13.067413	22.25653	36.95413	43.35534348
	2A_W. Hartford	45.16	4.9547	4.3682155	6.0502889	16.56054	18.8475672
	3B_Norwich 3 Rivers	14.82	17.8926	28.447404	39.944784	48.61735	51.26190751
	3C_Staples Norwich	29.04	27.7581	32.945133	41.653524	50.76599	53.12951054
	4A_Hartford Hgh	28.79	4.331190695	5.5406639	6.8005671	16.28457	20.83685247
	4B_Trinity	19.37	2.697428834	2.8167065	4.8737559	12.63462	16.97955944
	5A_New Canaan	29.34	19.48051948	22.979846	22.722927	26.19607	28.69032588
	5B_Cornwall Bridge	20.60	32.2681359	51.520308	53.930057	55.22672	54.94097875
	5C_Cornwall Bridge	15.57	28.89501071	56.356467	58.637485	61.63283	60.73075833
	R²_DEC		0.1938	0.3051	0.3313	0.2789	0.2711
			(-)	(-)	(-)	(-)	(-)

Table 4.8

Coniferous - Environmental Stressor Average Percentages per Site at Different Catchment Diameters and Average R²/ Catchment Diameters per Environmental Stressors

Coniferous	Site	GC means	% Area				
			CON261	CON274	CON287	CON2914	CON3018
	1A_Waterbury	25.23	0.3394	0.6508	0.3620	1.648131	2.9940
	1C_Waterbury	32.95	0.2344	0.5291921	0.3365661	1.424299	2.817726303
	2A_W. Hartford	45.16	0.3153	1.4459277	2.90717	4.906154	4.766441534
	3B_Norwich 3 Rivers	14.82	0.325987144	1.5473701	2.3824651	3.100915	3.307026767
	3C_Staples Norwich	29.04	1.521710613	2.3641646	2.1766136	3.158397	3.158161235
	4A_Hartford Hgh	28.79	0.663197633	3.44774	2.6063852	4.015655	4.210999803
	4B_Trinity	19.37	1.698806244	1.1253715	2.4511629	3.240207	3.758435185
	5A_New Canaan	29.34	4.643841007	5.0729644	3.6027545	2.508193	2.395247052
	5B_Cornwall Bridge	20.60	20.8815427	18.347416	15.715312	13.63588	12.46978794
	5C_Cornwall Bridge	15.57	28.55831038	25.390906	23.225542	16.85597	15.48142231
	R²_CON		0.2309	0.2149	0.217	0.1669	0.177
			(+)	(+)	(+)	(+)	(+)

Table 4.9

Non-Forested Wetland (NFW) - Environmental Stressor Average Percentages per site at Different Catchment Diameters and Average R²/ Catchment Diameters per Environmental Stressors

Non-Forested Wetland	Site	GC mean	% Area				
			NFW361	NFW374	NFW387	NFW3914	NFW4018
	1A_Waterbury	25.23	0.0319	0.1890	0.1948	0.196353	0.1888
	1C_Waterbury	32.95	0.009306975	0.1593679	0.1724093	0.209244	0.172002693
	2A_W. Hartford	45.16	0.0054	0.218636	0.1678255	0.232064	0.265606445
	3B_Norwich 3 Rivers	14.82	0.013774105	0.1021417	0.1024528	0.204084	0.244332958
	3C_Staples Norwich	29.04	0	0.0882399	0.078855	0.19854	0.234972497
	4A_Hartford Hgh	28.79	0	0.0401427	0.1371561	0.151572	0.278954004
	4B_Trinity	19.37	0.068870523	0.1871977	0.2037773	0.296894	0.259246853
	5A_New Canaan	29.34	0	0.0457849	0.068593	0.080419	0.101841865
	5B_Cornwall Bridge	20.60	0.477502296	0.104889	0.1153119	0.406966	0.55307904
	5C_Cornwall Bridge	15.57	0.122436486	0.0854651	0.1254503	0.474651	0.666607497
	R²_NFW		0.1307	0.1143	0.0217	0.2221	0.2144
			(-)	(+)	(+)	(-)	(-)

Table 4.10

Forested Wetland (FWE) - Environmental Stressor Average Percentages per site at Different Catchment Diameters and Average R²/ Catchment Diameters per Environmental Stressors

Forested Wetlands (FWE)	Site	GC means	% Area				
			FWE411	FWE424	FWE437	FWE4414	FWE4518
	1A_Waterbury	25.23	0.0080	0.7613	0.9235	1.995379	2.5981
	1C_Waterbury	32.95	0.0170	0.7402778	0.7155439	1.918696	2.502732236
	2A_W. Hartford	45.16	0.9669	0.0082357	0.3496693	2.088668	2.606976049
	3B_Norwich 3 Rivers	14.82	1.6345	2.0547574	2.4829605	3.571282	4.279421864
	3C_Staples Norwich	29.04	0.0000	2.331699	2.42101	3.418981	4.25517559
	4A_Hartford Hgh	28.79	0.0000	0.3949524	0.305083	1.893357	2.383213261
	4B_Trinity	19.37	1.2626	0.038605	0.2401146	2.072624	2.096127473
	5A_New Canaan	29.34	1.1544	0.6509777	1.0783149	1.447754	1.695278505
	5B_Cornwall Bridge	20.60	0.0000	0.8962182	1.6937618	2.725163	2.96099702
	5C_Cornwall Bridge	15.57	0.0000	1.1576637	1.8190249	3.114978	3.266710757
	R²_FWE		0.0070	0.1664	0.2603	0.2617	0.114
			(-)	(-)	(-)	(-)	(-)

Table 4.11

Census - Environmental Stressor Average Percentages per site at Different Catchment Diameters and Average R²/ Catchment Diameters per Environmental Stressors

Census	Site	GC mean	% Area				
			CEN611	CEN624	CEN637	CEN6414	CEN6518
	1A_Waterbury	25.23	3701.4783	38132.6957	86038.0870	167570	200429.4783
	1C_Waterbury	32.95	4554.886364	49475.81818	84917.13636	164624.7	192904.5909
	2A_W. Hartford	45.16	5447.411765	55814.11765	141384.4706	268791.8	317465.0588
	3B_Norwich 3 Rivers	14.82	2499.05	12317.6	28803.35	59239.35	74690.05
	3C_Staples Norwich	29.04	1545.428571	12289.14286	29078.42857	58774.57	73876.71429
	4A_Hartford Hgh	28.79	9332.5	70497.61111	153127.0556	250520.8	328929.6111
	4B_Trinity	19.37	7196.625	76708	119620.375	276730	381881.875
	5A_New Canaan	29.34	1617.142857	10990.85714	31923.14286	170975.6	260462.4286
	5B_Cornwall Bridge	20.60	155	495.2	757.2	3432	6914.8
	5C_Cornwall Bridge	15.57	170.6666667	258.6666667	594.6666667	2096.667	4665.333333
	R ² _CEN		0.1237	0.1663	0.3239	0.3248	0.2491
			(+)	(+)	(+)	(+)	(+)

Table 4.12

Biodiversity (BISON) - Environmental Stressor Average Percentages per site at Different Catchment Diameters and Average R²/ Catchment Diameters per Environmental Stressors

Biodiversity (BISON)	Site	GCY1	% Area				
			BIS661	BIS674	BIS687	BIS6914	BIS7018
	1A_Waterbury	25.23	44.7826	121.7391	154.3478	184	200.1304
	1C_Waterbury	32.95	43.9545	126.75	144.9318182	171.9318	184.9318182
	2A_W. Hartford	45.16	16.4118	93.05882353	179	286.3529	247.6470588
	3B_Norwich 3 Rivers	14.82	4.6000	61.45	92.9	138.2	143.1
	3C_Staples Norwich	29.04	14.4286	59.28571429	110.7142857	152.1429	266.1428571
	4A_Hartford Hgh	28.79	11.6111	91.11111111	148.7222222	184.9444	215.1111111
	4B_Trinity	19.37	12.8750	73.625	167	205.25	169.75
	5A_New Canaan	29.34	41.0000	132	201.4285714	278.8571	269.5714286
	5B_Cornwall Bridge	20.60	105.8000	131.2	151.2	182.6	187.8
	5C_Cornwall Bridge	15.57	53.0000	96.66666667	97.66666667	142.3333	168
	R ² _BIS		0.0390	0.0409	0.3212	0.4652	0.4959
			(-)	(+)	(+)	(+)	(+)

Table 4.13 shows each site and its largest number of significant ($R^2 > 0.10$) R^2 values and the breadth or number of significant environmental stressors at each site. In addition, the relationship (+, -) is indicated for the environmental stressor with the largest R^2 value. The catchment (1, 4, 7, 14, or 18 km) where the maximum R^2 is also shown along with the maximum developed area of each of the towns where the sites are located.

Table 4.13

Maximum Significant R²/Site, Environmental Stressor, and Span of Significant R² Across Catchment Area Sizes (1,4,7,14,18 km)

Site	Name	Intensity of Dev % Town	# Sig Env Stressors	High R ²	Env. Stress	Breadth of Env Stress Catch	Relationship	Catchment
4B	Trinity College	71.9 Hartford	10	0.84	DEV	24	(+)	7
5ABC	New Canaan	31.6 New Canaan	12	0.67	AG	45	(-)	4
3C	Staples	31.6 Norwich	6	0.54	CONIF	13	(-)	1
3B	Norwich 3 Rivers	31.6 Norwich	10	0.41	BIS	24	(+)	14
6AB	Cornwall	5.6 Cornwall	10	0.4	DEV	36	(-)	1
2A	W. Hartford	47.3 W. Hartford	11	0.32	NFW	27	(+)	7
1A	DOL	55.7 Waterbury	10	0.27	CONIF	20	(+)	1
4A	Hartford High	71.9 Hartford	5	0.23	DEC	14	(-)	1
1C	Shop Ctr	55.7 Waterbury	4	0.12	DEC/CON	5	(+)	4

Table 4.14 indicates the expected versus the actual relationship found when ordinary least squares (OLS) was conducted for each of the sites and each of the environmental stressors. The first column under the site heading lists the average R^2 value for that ES and in the next column the actual relationship found between the ES and the GC value. The next column labeled Expected was the relationship presumed to be occurring. For instance, for development (DEV) at Site 1A Waterbury, the actual relationship found was that as ES percentage increased GC level decreased. The expected result was that as the ES percentage increased the GC level would increase as well. That expected cell, when it differs from the actual relationship condition is filled in red; if expected matches actual, it is filled in green. The number at the bottom of each Expected column is the percentage occurrence when the expected matched the actual condition.

Table 4.14

R² Relationship Direct or Inverse, Expected vs. Actual for Each Site and Environmental Stressor

Site	1A_Waterbury		1C_Waterbury		2A_W.Hartford		3B_Norwich 3 Rivers		3C_Staples Norwich		4A_Hartford High		4B_Trinity		5A_New Canaan		5B_Cornwall Bridge		5C_Cornwall Bridge		R ²	relationship
	Expected	Actual	Expected	Actual	Expected	Actual	Expected	Actual	Expected	Actual	Expected	Actual	Expected	Actual	Expected	Actual	Expected	Actual				
DEV	0.4026 (-)	(-)	0.0187 (-)	(-)	0.1045 (+)	(+)	0.1022 (+)	(+)	0.2250 (+)	(+)	0.1008 (+)	(+)	0.3012 (-)	(-)	0.1877 (+)	(+)	0.1948 (+)	(+)			0.66	
TURF	0.1282 (+)	(+)	0.0011 (+)	(+)	0.0209 (-)	(-)	0.0439 (+)	(+)	0.2406 (+)	(+)	0.1473 (-)	(-)	0.0249 (+)	(+)	0.4028 (-)	(-)	0.2439 (+)	(+)			0.66	
DG	0.0314 (+)	(+)	0.0311 (-)	(-)	0.1382 (-)	(-)	0.1283 (-)	(-)	0.0463 (-)	(-)	0.0007 (-)	(-)	0.0028 (+)	(+)	0.1159 (-)	(-)	0.1074 (+)	(+)			0.66	
AG	0.0669 (-)	(-)	0.0051 (-)	(-)	0.3666 (-)	(-)	0.2954 (-)	(-)	0.1724 (-)	(-)	0.0656 (+)	(+)	0.4185 (+)	(+)	0.478 (-)	(-)	0.3233 (-)	(-)			0.77	
DEC	0.0016 (-)	(-)	0.0260 (-)	(-)	0.1655 (-)	(-)	0.2426 (-)	(-)	0.2706 (-)	(-)	0.0295 (+)	(+)	0.6185 (+)	(+)	0.0612 (-)	(-)	0.2915 (-)	(-)			0.77	
CON	0.0827 (-)	(-)	0.0266 (-)	(-)	0.0069 (-)	(-)	0.2087 (-)	(-)	0.0888 (-)	(-)	0.0251 (-)	(-)	0.7255 (+)	(+)	0.6138 (-)	(-)	0.0385 (-)	(-)			0.88	
NFW	0.1789 (+)	(+)	0.0260 (-)	(-)	0.1463 (-)	(-)	0.072 (-)	(-)	0.5878 (-)	(-)	0.1774 (-)	(-)	0.2395 (+)	(+)	0.0576 (+)	(+)	0.0021 (-)	(-)			0.55	
FEW	0.0545 (-)	(-)	0.0321 (-)	(-)	0.1482 (-)	(-)	0.1489 (-)	(-)	0.2712 (-)	(-)	0.165 (+)	(+)	0.3119 (+)	(+)	0.3882 (+)	(+)	0.3866 (-)	(-)			0.55	
WHE	0.1388 (+)	(+)	0.0483 (+)	(+)	0.1345 (+)	(+)	0.0552 (+)	(+)	0.227 (+)	(+)	0.0062 (-)	(-)	0.4674 (+)	(+)	0.0093 (+)	(+)	0.0095 (+)	(+)			0.11	
ROW	0.042 (-)	(-)	0.0111 (+)	(+)	0.0847 (+)	(+)	0.1355 (+)	(+)	0.6038 (-)	(-)	0.1477 (+)	(+)	0.302 (+)	(+)	0.3878 (+)	(+)	0.2654 (+)	(+)			0.55	
CEN	0.1287 (-)	(-)	0.0306 (-)	(-)	0.0767 (-)	(-)	0.1696 (-)	(-)	0.4014 (+)	(+)	0.1696 (-)	(-)	0.5015 (+)	(+)	0.288 (-)	(-)	0.7385 (-)	(-)			0.22	
BISON	0.2 (+)	(+)	0.0183 (-)	(-)	0.0283 (-)	(-)	0.1788 (-)	(-)	0.4373 (-)	(-)	0.2476 (-)	(-)			0.0255 (+)	(+)	0.1501 (-)	(-)			0.44	
R2 avg	0.130		0.023		0.119		0.148		0.296		0.105		0.319		0.251		0.232		0.66		0.56	

Tables 4.15 and 4.16 show each site and all the significant R^2 values ($R^2 > 0.10$ highlighted in blue) for each environmental stressor at each catchment at each site. In addition, the relationship (+, -) is indicated for the environmental stressor for each R^2 value.

Table 4.15

Environmental Stressor All Significant R²/Site and Span of Significant R² Across Catchment Area Sizes (1,4,7,14,18 km)

	IND LS all sites		DEV1		DEV4		DEV7		DEV14		DEV18	R2 mean
DEVELOPED	1A_Waterbury	(-)	0.0274	(-)	0.0889	(-)	0.0251	(-)	0.0428	(-)	0.014	0.03964
	1C_Waterbury	(-)	0.0197	(-)	0.0164	(+)	0.0023	(-)	0.0066	(-)	0.021	0.0132
	2A_W. Hartford	(+)	0.2206	(-)	0.1683	(-)	0.0385	(-)	0.0116	(-)	0.1058	0.10896
	3B_Norwich 3 Rivers	(-)	0.0637	(+)	0.0308	(+)	0.0965	(+)	0.0595	(+)	0.075	0.0651
	3C_Staples Norwich	(+)	0.0179	(-)	0.0028	(-)	0.0215	(+)	0.0105	(+)	0.0139	0.01332
	4A_Hartford Hgh	(+)	0.1161	(-)	0.015	(-)	0.0151	(-)	0.015	(-)	0.0149	0.03522
	4B_Trinity	(+)	0.1732	(+)	0.1871	(+)	0.8497	(+)	0.0465	(+)	0.0384	0.25898
	5Abc_New Canaan	(+)	0.1654	(+)	0.2734	(+)	0.212	(+)	0.1097	(+)	0.1724	0.18658
	6ABC_Cornwall Bridge	(-)	0.4062	(+)	0.221	(+)	0.2424	(+)	0.2517	(+)	0.2506	0.27438

			TURF1		TURF 4		TURF 7		TURF 14		TURF 18	R2 mean
TURF	1A_Waterbury	(+)	0.1379	(+)	0.106	(+)	0.0169	(+)	0.0762	(-)	0.041	0.0756
	1C_Waterbury	(-)	0.0089	(-)	0.0372	(-)	0.014	(-)	0.0057	(-)	0.0162	0.0164
	2A_W. Hartford	(-)	0.0219	(+)	0.222	(-)	0.112	(+)	0.0949	(+)	0.0537	0.1009
	3B_Norwich 3 Rivers	(-)	0.0355	(-)	0.0605	(+)	0.1214	(-)	0.2426	(+)	0.0878	0.10956
	3C_Staples Norwich	(+)	0.0062	(+)	0.0207	(+)	0.0262	(+)	0.0093	(+)	0.00021	0.012522
	4A_Hartford Hgh	(+)	0.0433	(+)	0.0218	(-)	0.006	(-)	0.0145	(-)	0.0152	0.02016
	4B_Trinity	(+)	0.82	(-)	0.7069	(-)	0.0565	(+)	0.0419	(-)	0.1319	0.35144
	5A_New Canaan	(-)	0.2473	(-)	0.2715	(-)	0.2665	(-)	0.2162	(-)	0.1905	0.2384
	56ABCornwall Bridge	(+)	0.381	(+)	0.2522	(+)	0.257	(+)	0.258	(+)	0.243	0.27824

			OG1		OG4		OG7		OG14		OG18	R2 mean
OG	1A_Waterbury	(-)	0.047	(+)	0.015	(-)	0.1454	(-)	0.0146	(-)	0.0013	0.04466
	1C_Waterbury	(+)	0.0776	(+)	0.0027	(-)	0.0018	(-)	0.0549	(-)	0.0569	0.03878
	2A_W. Hartford	(+)	0.1292	(-)	0.0435	(+)	0.1798	(-)	0.069	(-)	0.0939	0.10308
	3B_Norwich 3 Rivers	(-)	0.1772	(-)	0.021	(-)	0.0872	(-)	0.1592	(-)	0.0953	0.10798
	3C_Staples Norwich	(+)	0.034	(-)	0.2052	(-)	0.0448	(-)	0.0561	(+)	0.096	0.08722
	4A_Hartford Hgh	(+)	0.0091	(+)	0.0062	(-)	0.0627	(-)	0.0058	(-)	0.0195	0.02066
	4B_Trinity	(+)	0.1688	(+)	0.0029	(-)	0.1982	(+)	0.6196	(+)	0.1537	0.22864
	5A_New Canaan	(+)	0.0127	(-)	0.4251	(+)	0.2565	(+)	0.2357	(+)	0.2461	0.23522
	56ABCornwall Bridge	(-)	0.3774	(+)	0.0762	(-)	0.3165	(-)	0.1436	(-)	0.3726	0.25726

			AG1		AG4		AG7		AG14		AG18	R2 mean
AG	1A_Waterbury	(+)	0		0	(-)	0.0315	(+)	0.1098	(+)	0.0952	0.0473
	1C_Waterbury	(+)	0		0	(+)	0.0873	(+)	0.0007	(+)	0.0006	0.01772
	2A_W. Hartford	(+)	0		0	(+)	0.1669	(-)	0.1163	(+)	0.1572	0.08808
	3B_Norwich 3 Rivers	(-)	0.194	(+)	0.101	(-)	0.0653	(-)	0.1052	(-)	0.0404	0.10118
	3C_Staples Norwich	(-)	0.0668	(-)	0.4463	(+)	0.0125	(+)	0.0182	(+)	0.0023	0.10922
	4A_Hartford Hgh							(+)	0.0008	(+)	0.04	0.00816
	4B_Trinity							(+)	0.1503	(-)	0.0264	0.03534
	5A_New Canaan			(-)	0.6651	(-)	0.409	(+)	0.241	(+)	0.2479	0.3126
	56ABCornwall Bridge	(-)	0.2838	(-)	0.3902	(-)	0.3293	(-)	0.2406	(-)	0.2364	0.29606

			,DEC1		,DEC4		,DEC7		,DEC14		,DEC18	R2 mean
DEC	1A_Waterbury	(-)	0.1195	(+)	0.0438	(+)	0.0402	(-)	0.0819	(-)	0.016	0.06028
	1C_Waterbury	(+)	0.0255	(+)	0.0116	(-)	0.009	(+)	0.0247	(+)	0.0154	0.01724
	2A_W. Hartford	(-)	0.1358	(-)	0.1842	(+)	0.0819	(+)	0.0827	(+)	0.0001	0.09694
	3B_Norwich 3 Rivers	(-)	0.0408	(-)	0.0199	(-)	0.0115	(-)	0.0986	(-)	0.1039	0.05494
	3C_Staples Norwich	(+)	0.0126	(-)	0.0025	(+)	0.2242	(+)	0.4242	(-)	0.0708	0.14686
	4A_Hartford Hgh	(-)	0.2265	(+)	0.108	(+)	0.1081	(+)	0.1064	(+)	0.1276	0.13532
	4B_Trinity	(+)	0.0663	(-)	0.1424	(-)	0.1588	(-)	0.0277	(-)	0.0659	0.09222
	5A_New Canaan	(+)	0.343	(-)	0.2254	(-)	0.1904	(-)	0.1416	(-)	0.12893	0.205866
	56ABCornwall Bridge	(-)	0.2059	(-)	0.2813	(-)	0.3429	(-)	0.2654	(-)	0.2659	0.27228

			CON1		CON4		CON7		CON14		CON18	R2 mean
CON	1A_Waterbury	(+)	0.2703	(-)	0.1339	(-)	0.1159	(-)	0.1007	(-)	0.1123	0.14662
	1C_Waterbury	(+)	0.0071	(+)	0.118	(+)	0.0319	(+)	0.064	(+)	0.037	0.0516
	2A_W. Hartford	(-)	0.0015	(-)	0.306	(+)	0.1331	(+)	0.0745	(+)	0.128	0.12862
	3B_Norwich 3 Rivers	(+)	0.1963	(-)	0.0025	(+)	0.0781	(+)	0.191	(+)	0.2058	0.13474
	3C_Staples Norwich	(-)	0.545	(-)	0.1023	(+)	0.0921	(-)	0.0192	(-)	0.0213	0.15598
	4A_Hartford Hgh	(+)	0.115	(+)	0.1006	(+)	0.038	(+)	0.0225	(+)	0.0325	0.06172
	4B_Trinity	(+)	0.2211	(-)	0.0664	(+)	0.5614	(-)	0.0935	(-)	0.0563	0.19974
	5A_New Canaan	(-)	0.0754	(-)	0.434	(-)	0.1656	(+)	0.2499	(+)	0.2861	0.2422
	56ABCornwall Bridge	(-)	0.0291	(+)	0.1732	(+)	0.1806	(-)	0.2136	(+)	0.1458	0.14846

Table 4.16

Environmental Stressor All Significant R^2 /Site and Span of Significant R^2 Across Catchment Area Sizes (1,4,7,14,18 km)

			FEW1		FEW4		FEW7		FEW14		FEW18	R2 mean
FEW	1A_Waterbury	(-)	0	(-)	0.0162	(-)	0.0008	(+)	0.0282	(+)	0.0407	0.01718
	1C_Waterbury	(-)	0	(+)	0.0046	(-)	0.00005	(-)	0.0058	(+)	0.0105	0.00419
	2A_W. Hartford	(-)	0.1273	(+)	0.0196	(+)	0.0356	(+)	0.0124	(+)	0.2424	0.08746
	3B_Norwich 3 Rivers	(+)	0.0952	(-)	0.1213	(-)	0.1738	(-)	0.0575	(+)	0.0691	0.10338
	3C_Staples Norwich	(-)	0	(+)	0.0009	(+)	0.0064	(-)	0.018	(+)	0.0111	0.00728
	4A_Hartford Hgh	(-)	0	(+)	0.0153	(+)	0.0127	(+)	0.0122	(+)	0.0252	0.01308
	4B_Trinity	(+)	0.008	(-)	0.0751	(-)	0.6069	(+)	0.0187	(-)	0.0559	0.15292
	5A_New Canaan	(+)	0.3714	(-)	0.0217	(-)	0.1666	(+)	0.3287	(+)	0.3298	0.24364
	56ABCornwall Bridge	(-)	0	(+)	0.0134	(-)	0.2614	(-)	0.2006	(-)	0.261	0.14728

			WWES1		WWES4		WWES7		WWES14		WWES18	R2 mean
WWES	1A_Waterbury	(+)	0.0046	(-)	0.0357	(-)	0.1881	(-)	0.036	(-)	0.0199	0.05686
	1C_Waterbury	(+)	0.0291	(+)	0.0505	(+)	0.0007	(-)	0.0004	(+)	0.0006	0.01626
	2A_W. Hartford	(-)	0.0046	(-)	0.0289	(-)	0.0668	(+)	0.1359	(+)	0.2369	0.09462
	3B_Norwich 3 Rivers	(+)	0.0474	(+)	0.128	(+)	0.1536	(-)	0.0209	(-)	0.1081	0.0916
	3C_Staples Norwich	(-)	0.1424	(+)	0.0062	(+)	0.0189	(-)	0.0079	(+)	0.0355	0.04218
	4A_Hartford Hgh	(+)	0.0522	(+)	0.0156	(+)	0.0318	(-)	0.0211	(-)	0.0368	0.0315
	4B_Trinity	(+)	0.0177	(-)	0.175	(-)	0.1375	(+)	0.0637	(-)	0.0776	0.0943
	5A_New Canaan	(-)	0.0288	(-)	0.1373	(+)	0.0094	(+)	0.1588	(-)	0.0153	0.06992
	56ABCornwall Bridge	(-)	0	(+)	0.2272	(+)	0.241	(+)	0.2518	(+)	0.26	0.196

			ROW1		ROW4		ROW7		ROW14		ROW18	R2 mean
ROW	1A_Waterbury	(+)	0	(+)	0.1351	(-)	0.301	(-)	0.1724	(+)	0.0158	0.12486
	1C_Waterbury	(+)	0	(+)	0.0556	(-)	0.0818	(+)	0.1156	(+)	0.0288	0.05636
	2A_W. Hartford	(+)	0	(+)	0	(-)	0	(+)	0.0004	(+)	0.2539	0.05086
	3B_Norwich 3 Rivers	(+)	0	(-)	0.1927	(-)	0.3693	(-)	0.126	(-)	0.1455	0.1667
	3C_Staples Norwich	(+)	0	(+)	0.3435	(-)	0.0117	(-)	0.0171	(+)	0.1082	0.0961
	4A_Hartford Hgh	(+)	0	(+)	0	(+)	0	(+)	0.0981	(+)	0.0141	0.02244
	4B_Trinity	(+)	0	(+)	0	(+)	0	(-)	0.0779	(-)	0.0428	0.02414
	5A_New Canaan	(+)	0	(+)	0	(+)	0	(+)	0.0003	(+)	0.2337	0.0468
	56ABCornwall Bridge	(+)	0	(+)	0	(+)	0	(+)	0	(+)	0.2646	0.05292

			CEN1		CEN4		CEN7		CEN14		CEN18	R2 mean
CEN	1A_Waterbury	(-)	0.2212	(-)	0.173	(-)	0.0895	(+)	0.1039	(-)	0.0816	0.13384
	1C_Waterbury	(+)	0.0722	(+)	0.2195	(-)	0.0111	(+)	0.1171	(+)	0.0594	0.09586
	2A_W. Hartford	(-)	0.2091	(-)	0.285	(-)	0.2556	(-)	0.1144	(-)	0.1448	0.20178
	3B_Norwich 3 Rivers	(-)	0.033	(+)	0.0182	(-)	0.0156	(-)	0.0156	(+)	0.1055	0.03758
	3C_Staples Norwich	(-)	0.0045	(-)	0.0294	(-)	0.0208	(-)	0.0632	(+)	0.022	0.02798
	4A_Hartford Hgh	(+)	0.0673	(+)	0.0076	(-)	0.015	(-)	0.1921	(+)	0.2847	0.11334
	4B_Trinity	(-)	0.61	(-)	0.1124	(+)	0.2125	(-)	0.5123	(-)	0.6417	0.41778
	5A_New Canaan	(-)	0.1062	(-)	0.0273	(-)	0.1944	(-)	0.2915	(-)	0.1596	0.1558
	56ABCornwall Bridge	(-)	0.4784	(+)	0.4718	(-)	0.6875	(+)	0.5161	(-)	0.304	0.49156

			BIS1		BIS4		BIS7		BIS14		BIS18	R2 mean
BIS	1A_Waterbury	(-)	0.0775	(-)	0.0918	(+)	0.008	(-)	0.1096	(-)	0.0613	0.06964
	1C_Waterbury	(-)	0.0001	(+)	0.0011	(-)	0.0202	(-)	0.0058	(+)	0.0463	0.0147
	2A_W. Hartford	(+)	0.3081	(-)	0.0383	(-)	0.1908	(-)	0.2071	(-)	0.0034	0.14954
	3B_Norwich 3 Rivers	(-)	0.0917	(-)	0.0711	(+)	0.055	(+)	0.4145	(+)	0.1643	0.15932
	3C_Staples Norwich	(-)	0.47	(-)	0.3151	(+)	0.1879	(-)	0.8174	(-)	0.1725	0.39258
	4A_Hartford Hgh	(+)	0.1057	(+)	0.1057	(-)	0.0056	(-)	0.3759	(+)	0.116	0.14178
	4B_Trinity	(-)	0.51	(-)	0.3045	(-)	0.0856	(-)	0.0249	(-)	0.259	0.2368
	5A_New Canaan	(-)	0.4581	(+)	0.2601	(-)	0.1383	(-)	0.083	(-)	0.0215	0.1922
	56ABCornwall Bridge	(-)	0.069	(-)	0.0183	(+)	0.0781	(+)	0.0018	(-)	0.3743	0.1083

Tables 4.17–4.25 show the environmental stressor R^2 on average ($R^2 > 0.10$) across each catchment size on a per site basis for each environmental stressor. In the left-hand column, the green cell shows the total number of environmental stressors that have significant values ($R^2 > 0.10$) for the entire site. The blue cell in the left-hand column shows the total number of R^2 values that exceed ($R^2 > 0.10$) for the entire site.

Table 4.17

Site 1A-DOL - Environmental Stressor R^2 on Average Across Each Catchment Size on a per Site Basis

		Site		1	4	7	14	18
DOL	DEV	1A_Waterbury	(-)	0.0274 (-)	0.0889 (-)	0.0251 (-)	0.0428 (-)	0.014
	10	TURF	(+)	0.1379 (+)	0.106 (+)	0.0169 (+)	0.0762 (-)	0.041
	20	OG	(-)	0.047 (+)	0.015 (-)	0.1454 (-)	0.0146 (-)	0.0013
		AG			(-)	0.0315 (+)	0.1098 (+)	0.0952
		DEC	(-)	0.1195 (+)	0.0438 (+)	0.0402 (-)	0.0819 (-)	0.016
		CON	(+)	0.2703 (-)	0.1339 (-)	0.1159 (-)	0.1007 (-)	0.1123
		WA						
		NFW		(+)	0.2055 (+)	0.1661 (-)	0.0426 (-)	0.0284
		FEW		(-)	0.0162 (-)	0.0008 (+)	0.0282 (+)	0.0407
		WWE						
		WWES	(+)	0.0046 (-)	0.0357 (-)	0.1881 (-)	0.036 (-)	0.0199
		ROW		(+)	0.1351 (-)	0.301 (-)	0.1724 (+)	0.0158
		CEN	(-)	0.2212 (-)	0.173 (-)	0.0895 (+)	0.1039 (-)	0.0816
		BIS	(-)	0.0775 (-)	0.0918 (+)	0.008 (-)	0.1096 (-)	0.0613

Table 4.18

Site 1C -Shopping Center - Environmental Stressor R^2 on Average Across Each Catchment Size on a per Site Basis

		Site		1	4	7	14	18
SHOP	DEV	1C_Waterbury	(-)	0.0197 (-)	0.0164 (+)	0.0023 (-)	0.0066 (-)	0.021
	4	TURF	(-)	0.0089 (-)	0.0372 (-)	0.014 (-)	0.0057 (-)	0.0162
	5	OG	(+)	0.0776 (+)	0.0027 (-)	0.0018 (-)	0.0549 (-)	0.0569
		AG			(+)	0.0873 (+)	0.0007 (+)	0.0006
		DEC	(+)	0.0071 (+)	0.118 (+)	0.0319 (+)	0.064 (+)	0.037
		CON	(+)	0.0071 (+)	0.118 (+)	0.0319 (+)	0.064 (+)	0.037
		WA						
		NFW		(-)	0.0044 (-)	0.0008 (-)	0.01 (+)	0.0238
		FEW		(+)	0.0046 (-)	0.00005 (-)	0.0058 (+)	0.0105
		WWE						
		WWES	(+)	0.0291 (+)	0.0505 (+)	0.0007 (-)	0.0004 (+)	0.0006
		ROW		(+)	0.0556 (-)	0.0818 (+)	0.1156 (+)	0.0288
		CEN	(+)	0.0722 (+)	0.2195 (-)	0.0111 (+)	0.1171 (+)	0.0594
		BIS	(-)	0.0001 (+)	0.0011 (-)	0.0202 (-)	0.0058 (+)	0.0463

Table 4.19

Site 2A -Shopping Center - Environmental Stressor R² on Average Across Each Catchment Size on a per Site Basis

				1	4	7	14	18
2A	DEV	2A_W. Hartford	(+)	0.2206 (-)	0.1683 (-)	0.0385 (-)	0.0116 (-)	0.1058
11	TURF	2A_W. Hartford	(-)	0.0219 (+)	0.222 (-)	0.112 (+)	0.0949 (+)	0.0537
27	OG	2A_W. Hartford	(-)	0.0219 (+)	0.222 (-)	0.112 (+)	0.0949 (+)	0.0537
	AG	2A_W. Hartford			(+)	0.1669 (-)	0.1163 (+)	0.1572
	DEC	2A_W. Hartford	(-)	0.1358 (-)	0.1842 (+)	0.0819 (+)	0.0827 (+)	0.0001
	CON	2A_W. Hartford	(-)	0.0015 (-)	0.306 (+)	0.1331 (+)	0.0745 (+)	0.128
	WA							
	NFW	2A_W. Hartford		(=)	0.0177 (+)	0.3241 (+)	0.0308 (+)	0.1528
	FEW	2A_W. Hartford	(-)	0.1273 (+)	0.0196 (+)	0.0356 (+)	0.0124 (+)	0.2424
	WWE							
	WWES							
	ROW	2A_W. Hartford				(+)	0.0004 (+)	0.2539
	CEN	2A_W. Hartford	(-)	0.2091 (-)	0.285 (-)	0.2556 (-)	0.1144 (-)	0.1448
	BIS	2A_W. Hartford	(+)	0.3081 (-)	0.0383 (-)	0.1908 (-)	0.2071 (-)	0.0034

Table 4.20

Site 3B – Three Rivers - Environmental Stressor R² on Average Across Each Catchment Size on a per Site Basis

				1	4	7	14	18
3 RIV	DEV	3B_Norwich 3 Rivers	(-)	0.0637 (+)	0.0308 (+)	0.0965 (+)	0.0595 (+)	0.075
10	TURF	3B_Norwich 3 Rivers	(-)	0.0355 (-)	0.0605 (+)	0.1214 (-)	0.2426 (+)	0.0878
24	OG	3B_Norwich 3 Rivers	(-)	0.1772 (-)	0.021 (-)	0.0872 (-)	0.1592 (-)	0.0953
	AG	3B_Norwich 3 Rivers	(-)	0.194 (+)	0.101 (-)	0.0653 (-)	0.1052 (-)	0.0404
	DEC	3B_Norwich 3 Rivers	(-)	0.0408 (-)	0.0199 (-)	0.0115 (-)	0.0986 (-)	0.1039
	CON	3B_Norwich 3 Rivers	(+)	0.1963 (-)	0.0025 (+)	0.0781 (+)	0.191 (+)	0.2058
	WA							
	NFW	3B_Norwich 3 Rivers		(+)	0.1566 (-)	0.0635 (+)	0.177 (+)	0.1692
	FEW	3B_Norwich 3 Rivers	(+)	0.0952 (-)	0.1213 (-)	0.1738 (-)	0.0575 (+)	0.0691
	WWE	3B_Norwich 3 Rivers			(-)	0.1355 (-)	,083 (-)	0.2136
	WWES							
	ROW	3B_Norwich 3 Rivers		(-)	0.1927 (-)	0.3693 (-)	0.126 (-)	0.1455
	CEN	3B_Norwich 3 Rivers	(-)	0.033 (+)	0.0182 (-)	0.0156 (-)	0.0156 (+)	0.1055
	BIS	3B_Norwich 3 Rivers	(-)	0.0917 (-)	0.0711 (+)	0.055 (+)	0.4145 (+)	0.1643

Table 4.21

Site 3C – Staples - Environmental Stressor R² On Average Across Each Catchment Size on a per Site Basis

			1	4	7	14	18	
STAP	DEV	3C_Staples Norw (+)	0.0179 (-)	0.0028 (-)	0.0215 (-+)	0.0105 (+)	0.0139	
	6	TURF	3C_Staples Norw (+)	0.0062 (+)	0.0207 (+)	0.0262 (+)	0.0093 (+)	0.00021
	13	OG	3C_Staples Norw (+)	0.034 (-)	0.2052 (-)	0.0448 (-)	0.0561 (+)	0.096
		AG	3C_Staples Norw (-)	0.0668 (-)	0.4463 (+)	0.0125 (+)	0.0182 (+)	0.0023
		DEC	3C_Staples Norw (+)	0.0126 (-)	0.0025 (+)	0.2242 (+)	0.4242 (-)	0.0708
		CON	3C_Staples Norw (-)	0.545 (-)	0.1023 (+)	0.0921 (-)	0.0192 (-)	0.0213
		WA						
		NFW	3C_Staples Norwich	(-)	0.0104 (-)	0.0305 (-)	0.0231 (-)	0.0358
		FEW	3C_Staples Norwich	(+)	0.0009 (+)	0.0064 (-)	0.018 (+)	0.0111
		WWE	3C_Staples Norwich		(+)	0.0829 (-)	0.0071 (-)	0.0108
		WWES						
		ROW	3C_Staples Norwich	(+)	0.3435 (-)	0.0117 (-)	0.0171 (+)	0.1082
		CEN	3C_Staples Norw (-)	0.0045 (-)	0.0294 (-)	0.0208 (-)	0.0632 (+)	0.022
		BIS	3C_Staples Norw (-)	0.47 (-)	0.3151 (+)	0.1879 (-)	0.8174 (-)	0.1725

Table 4.22

Site 4A – Hartford High - Environmental Stressor R² on Average Across Each Catchment Size on a per Site Basis

			1	4	7	14	18		
HH	DEV	4A_Hartford Hgh	(+)	0.1161 (-)	0.015 (-)	0.0151 (-)	0.015 (-)	0.0149	
	5	TURF	4A_Hartford Hgh	(+)	0.0433 (+)	0.0218 (-)	0.006 (-)	0.0145 (-)	0.0152
	14	OG	4A_Hartford Hgh	(+)	0.0091 (+)	0.0062 (-)	0.0627 (-)	0.0058 (-)	0.0195
		AG	4A_Hartford Hgh			(+)	0.0008 (+)	0.04	
		DEC	4A_Hartford Hgh	(-)	0.2265 (+)	0.108 (+)	0.1081 (+)	0.1064 (+)	0.1276
		CON	4A_Hartford Hgh	(+)	0.115 (+)	0.1006 (+)	0.038 (+)	0.0225 (+)	0.0325
		WA							
		NFW	4A_Hartford Hgh	(+)	0.0204 (-)	0.0664 (+)	0.0168 (-)	0.0144	
		FEW	4A_Hartford Hgh	(+)	0.0153 (+)	0.0127 (+)	0.0122 (+)	0.0252	
		WWE							
		WWES							
		ROW	4A_Hartford Hgh			(+)	0.0981 (+)	0.0141	
		CEN	4A_Hartford Hgh	(+)	0.0673 (+)	0.0076 (-)	0.015 (-)	0.1921 (+)	0.2847
		BIS	4A_Hartford Hgh	(+)	0.1057 (+)	0.1057 (-)	0.0056 (-)	0.3759 (+)	0.116

Table 4.23

Table 8G- Site 4B – Trinity College - Environmental Stressor R^2 on Average Across Each Catchment Size on a per Site Basis

				1	4	7	14	18
TRI	DEV	4B_Trinity	(+)	0.1732 (+)	0.1871 (+)	0.8497 (+)	0.0465 (+)	0.0384
10	TURF	4B_Trinity	(+)	0.82 (-)	0.7069 (-)	0.0565 (+)	0.0419 (-)	0.1319
24	OG	4B_Trinity	(+)	0.1688 (+)	0.0029 (-)	0.1982 (+)	0.6196 (+)	0.1537
	AG	4B_Trinity				(+)	0.1503 (-)	0.0264
	DEC	4B_Trinity	(+)	0.0663 (-)	0.1424 (-)	0.1588 (-)	0.0277 (-)	0.0659
	CON	4B_Trinity	(+)	0.2211 (-)	0.0664 (+)	0.5614 (-)	0.0935 (-)	0.0563
	WA							
	NFW	4B_Trinity		(+)	0.002 (+)	0.2162 (+)	0.0193 (-)	0.0051
	FEW	4B_Trinity	(+)	0.008 (-)	0.0751 (-)	0.6069 (+)	0.0187 (-)	0.0559
	WWE							
	WWES							
	ROW	4B_Trinity				(-)	0.0779 (-)	0.0428
	CEN	4B_Trinity	(-)	0.61 (-)	0.1124 (+)	0.2125 (-)	0.5123 (-)	0.6417
	BIS	4B_Trinity	(-)	0.51 (-)	0.3045 (-)	0.0856 (-)	0.0249 (-)	0.259

Table 4.24

Site 5ABC – New Canaan - Environmental Stressor R^2 on Average Across Each Catchment Size on a per Site Basis

				1	4	7	14	18
NC	DEV	5Abc_New Canaan	(+)	0.1654 (+)	0.2734 (+)	0.212 (+)	0.1097 (+)	0.1724
12	TURF	5Abc_New Canaan	(+)	0.1654 (+)	0.2734 (+)	0.212 (+)	0.1097 (+)	0.1724
45	OG	5A_New Canaan	(+)	0.0127 (-)	0.4251 (+)	0.2565 (+)	0.2357 (+)	0.2461
	AG	5A_New Canaan			(-)	0.6651 (-)	0.409 (+)	0.241 (+)
	DEC	5A_New Canaan	(+)	0.343 (-)	0.2254 (-)	0.1904 (-)	0.1416 (-)	0.12893
	CON	5A_New Canaan	(-)	0.0754 (-)	0.434 (-)	0.1656 (+)	0.2499 (+)	0.2861
	WA							
	NFW	5A_New Canaan		(-)	0.1004 (+)	0.3098 (+)	0.2604 (+)	0.2516
	FEW	5A_New Canaan	(+)	0.3714 (-)	0.0217 (-)	0.1666 (+)	0.3287 (+)	0.3298
	WWE	5A_New Canaan				(-)	0.3561 (-)	0.3562
	WWES							
	ROW	5A_New Canaan				(+)	0.0003 (+)	0.2337
	CEN	5A_New Canaan	(-)	0.1062 (-)	0.0273 (-)	0.1944 (-)	0.2915 (-)	0.1596
	BIS	5A_New Canaan	(-)	0.4581 (+)	0.2601 (-)	0.1383 (-)	0.083 (-)	0.0215

Table 4.25

Site 6AB – Cornwall Bridge- Environmental Stressor R^2 on Average Across Each Catchment

Size on a per Site Basis

				1	4	7	14	18
CORN	DEV	6ABC_Cornwall Bridge	(-)	0.4062 (+)	0.221 (+)	0.2424 (+)	0.2517 (+)	0.2506
11	TURF	56ABCornwall Bridge	(+)	0.381 (+)	0.2522 (+)	0.257 (+)	0.258 (+)	0.243
36	OG	56ABCornwall Bridge	(-)	0.3774 (+)	0.0762 (-)	0.3165 (-)	0.1436 (+)	0.3726
	AG	56ABCornwall Bridge	(-)	0.2838 (-)	0.3902 (-)	0.3293 (-)	0.2406 (+)	0.2364
	DEC	56ABCornwall Bridge	(-)	0.2059 (-)	0.2813 (-)	0.3429 (-)	0.2654 (+)	0.2659
	CON	56ABCornwall Bridge	(-)	0.0291 (+)	0.1732 (+)	0.1806 (-)	0.2136 (+)	0.1458
	WA							
	NFW	56ABCornwall Bridge	(-)	0.3018 (-)	0.2951 (-)	0.345 (-)	0.3021 (-)	0.2594
	FEW	56ABCornwall Bridge		(+)	0.0134 (-)	0.2614 (-)	0.2006 (-)	0.261
	WWE							
	WWES							
	ROW	56ABCornwall Bridge					(+)	0.2646
	CEN	56ABCornwall Bridge	(-)	0.4784 (+)	0.4718 (-)	0.6875 (+)	0.5161 (-)	0.304
	BIS	56ABCornwall Bridge	(-)	0.069 (-)	0.0183 (+)	0.0781 (+)	0.0018 (-)	0.3743

Evaluating all aggregated environmental stressors together for each of the catchments for each site results in Table 4.26 below showing the R^2 value for each of those locations using the aggregated environmental stressor as the independent variable and GC level as the dependent variable. A rough measure of the aggregate R^2 for each of the sites across all the catchment sizes is shown in Table 4.27. It sums the values for the coefficient of determination and shows the impact of all environmental stressors acting in unison, at a particular catchment area size for each of the sites.

Table 4.26

All Individual Environmental Stressors for Each Sample for Each Catchment and Sample GC

Average R^2 Intensity

Site		1 KM	4 KM	7 KM	14 KM	18 KM	1-18 KM		
1A DOL	(-)	0.26 (-)	0.1491 (-)	0.0492 (+)	0.329 (-)	0.1864 (-)	0.192		R2 range
1C Shop Ctr	(-)	0.1122 (-)	0.1043 (+)	0.0011 (-)	0.0609 (+)	0.033 (+)	0.1401		0.10-0.19
2A	(-)	0.2064 (-)	0.2812 (-)	0.2552 (+)	0.0126 (-)	0.1358 (-)	0.4443		0.20-0.29
3B Norwich	(-)	0.162 (-)	0.1421 (-)	0.0111 (-)	0.1022 (+)	0.1083 (-)	0.3559		0.30-0.39
Staples	(-)	0.0044 (+)	0.0044 (+)	0.0101 (-)	0.1574 (+)	0.0514 (-)	0.0295		0.40-0.49
4A HH	(+)	0.1959 (+)	0.424 (+)	0.1232 (+)	0.2529 (+)	0.2696 (+)	0.1964		0.50-0.59
4B TRINITY	(-)	0.2406 (-)	0.1145 (+)	0.123 (-)	0.0634 (-)	0.651 (-)	0.5431		0.60-0.69
5 ABC	(-)	0.2137 (+)	0.4709 (-)	0.205 (-)	0.2983 (-)	0.1593 (-)	0.2679		
6A/6B	(-)	0.1715 (+)	0.3779 (-)	0.1258 (+)	0.507 (-)	0.7544 (-)	0.1925		

Table 4.27

Summative Mean R^2 Ranking for All Environmental Stressors for Each Site for Each Catchment

From Table 8A-I Above

Site	1A_Waterbury	1C_Waterbury	2A_W. Hartford	3B_Norwich 3 Rivers	3C_Staples Norwich	4A_Hartford Hgh	4B_Trinity	5Abc_New Canaan	6ABC_Cornwall Bridge
Catchment									
1 KM	0.9054	0.2402	1.1581	0.9748	1.2994	0.7352	2.5951	1.8083	2.5326
4 KM	1.0449	0.5216	1.3135	0.9236	1.4853	0.4162	1.7747	2.8413	2.4199
7 KM	1.1285	0.26095	1.5851	1.2908	0.6975	0.3614	3.0833	2.3185	3.3419
14 KM	0.9187	0.4113	0.95	1.6676	1.4842	0.8812	1.6963	2.3168	2.6453
18 KM	0.5275	0.3165	1.5729	1.3699	0.58961	0.7409	1.5547	2.28343	3.2376
GC R2 mean	0.905	0.35011	1.31592	1.24534	1.111202	0.62698	2.14082	2.313666	2.82342

Figures 4.22–4.33 show the mean R^2 values at each of the catchment sizes for each of the environmental stressors.

Figure 4.22

Environmental Stressor – Development - Mean R² All Sites, Landcover vs. GC Ng/G

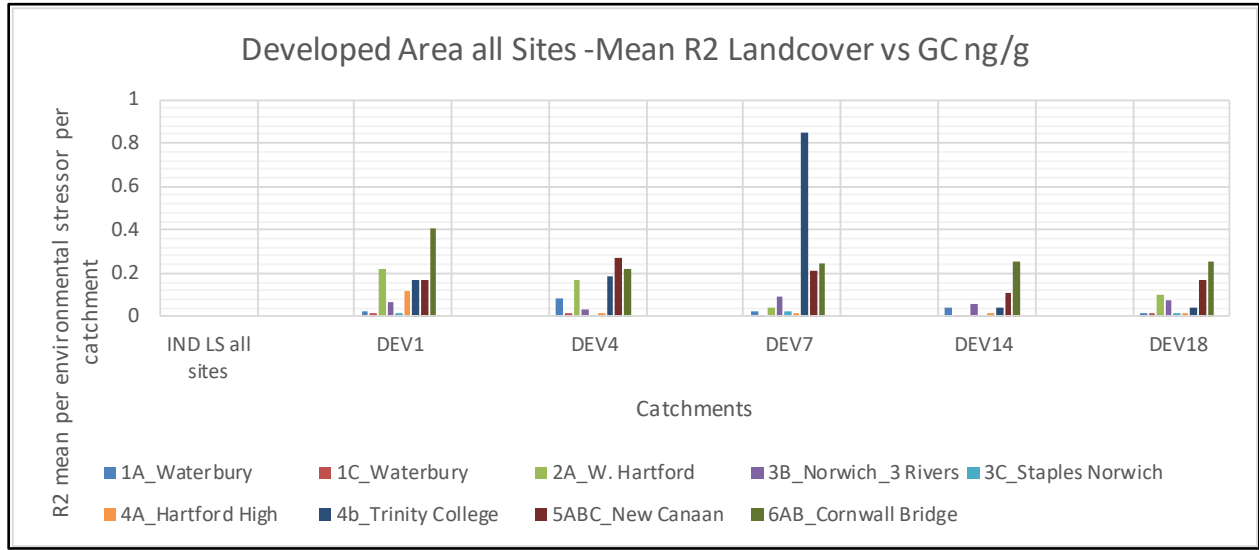


Figure 4.23

Environmental Stressor – Turf- Mean R² All Sites, Landcover vs. GC Ng/G

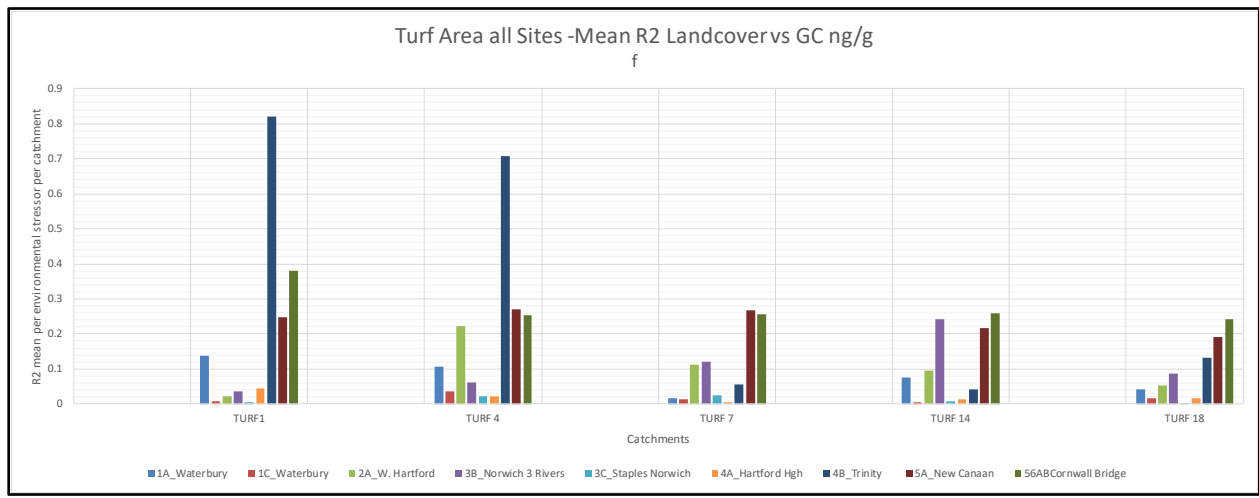


Figure 4.24

Environmental Stressor – Other Grasses - Mean R² All Sites, Landcover vs. GC Ng/G

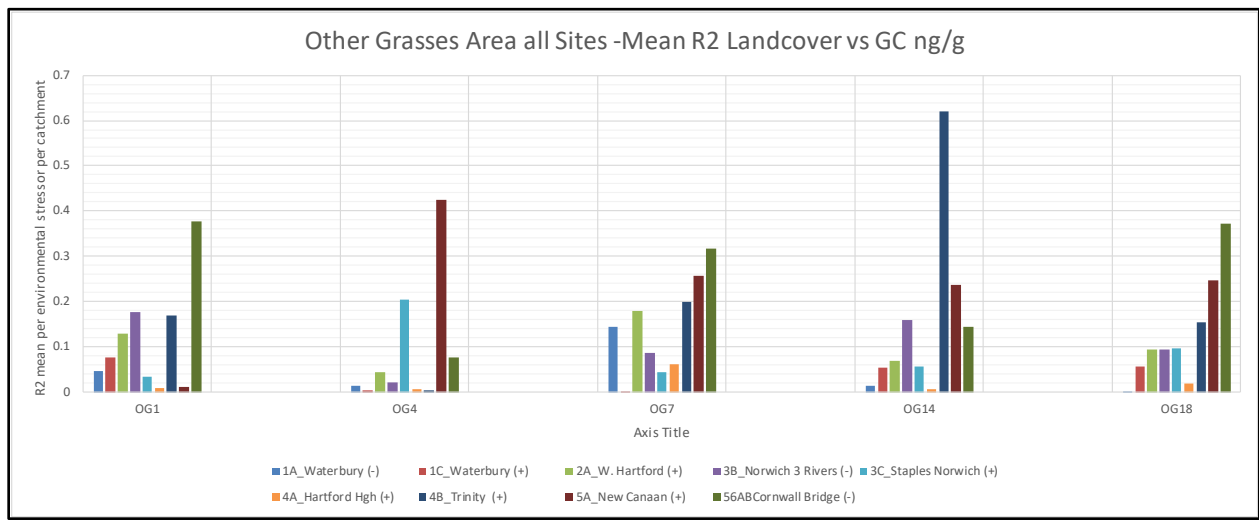


Figure 4.25

Environmental Stressor – Agriculture - Mean R² All Sites, Landcover vs. GC Ng/G

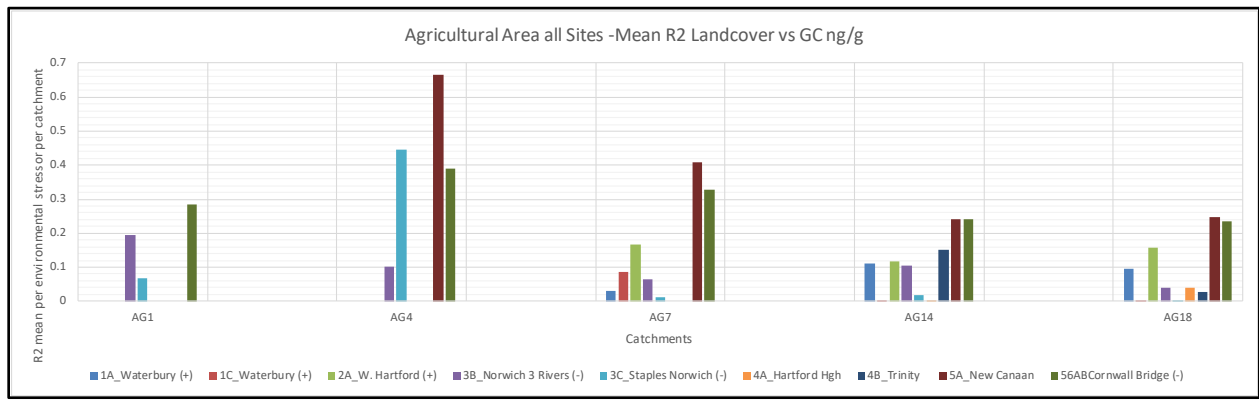


Figure 4.26

Environmental Stressor – Deciduous - Mean R² All Sites, Landcover vs. GC Ng/G

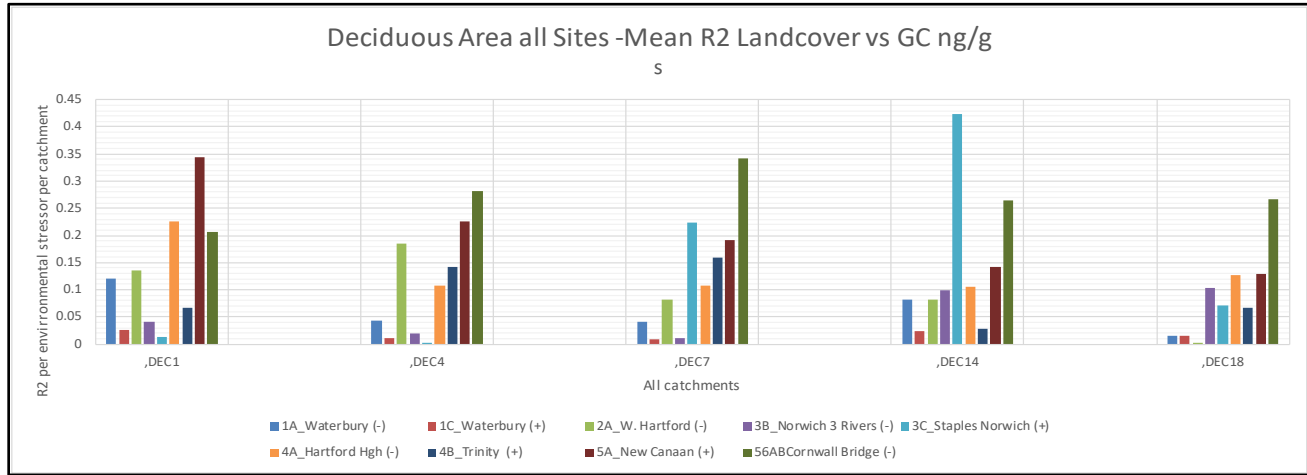


Figure 4.27

Environmental Stressor – Coniferous - Mean R² All Sites, Landcover vs. GC Ng/G

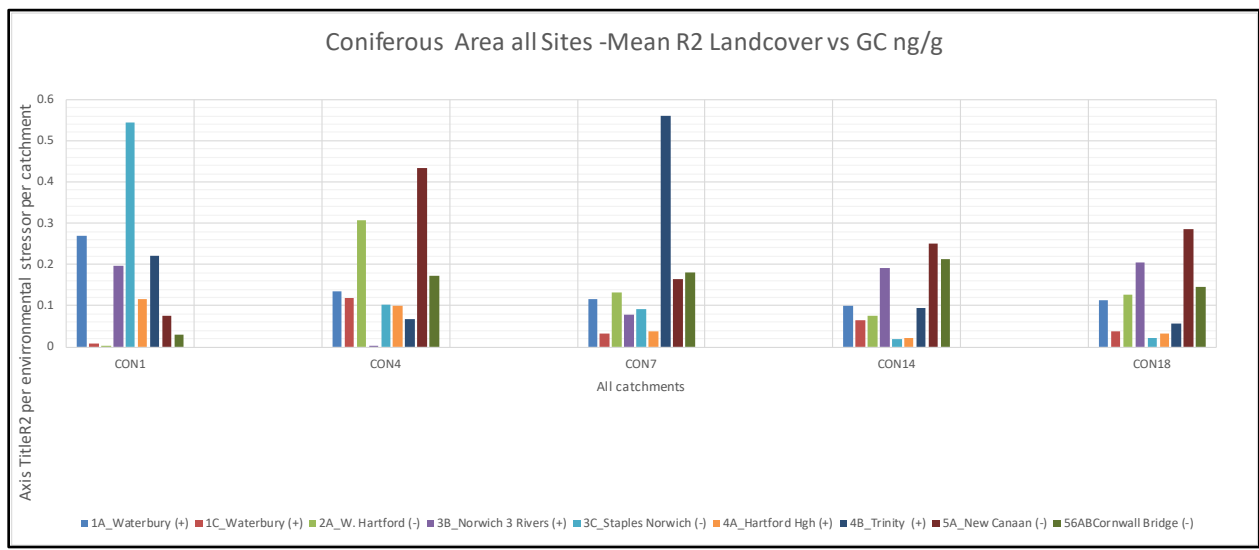


Figure 4.28

Environmental Stressor – Non Forested Wetlands - Mean R² All Sites, Landcover vs. GC Ng/G

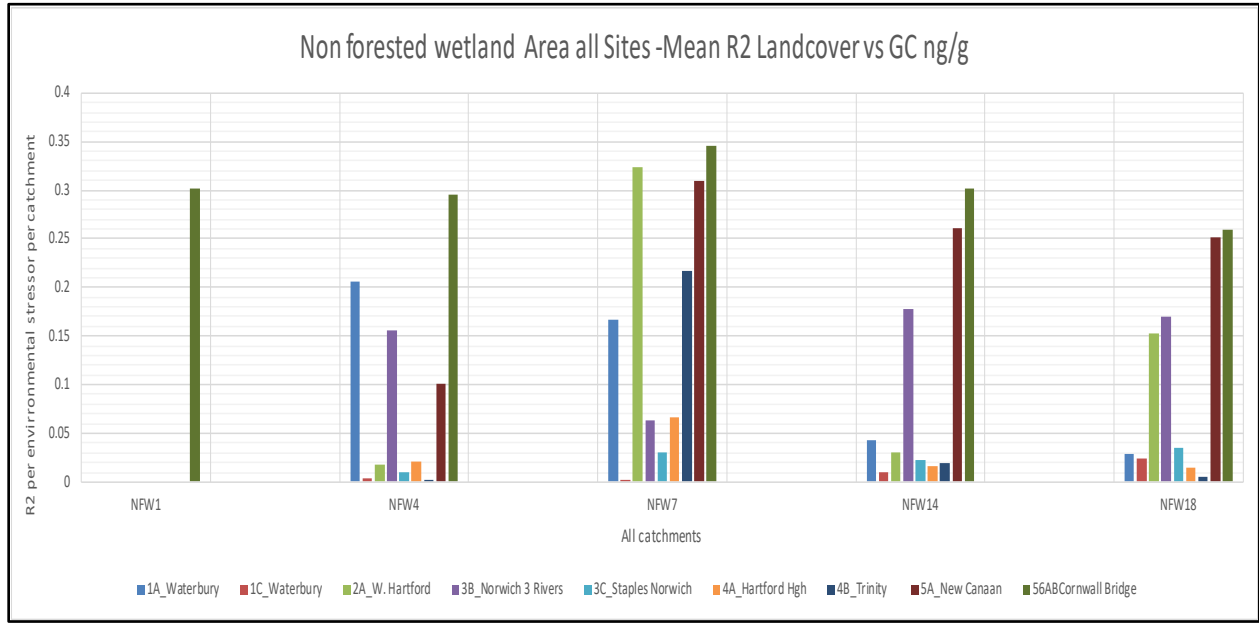


Figure 4.29

Environmental Stressor – Forested Wetlands - Mean R² All Sites, Landcover vs. GC Ng/G

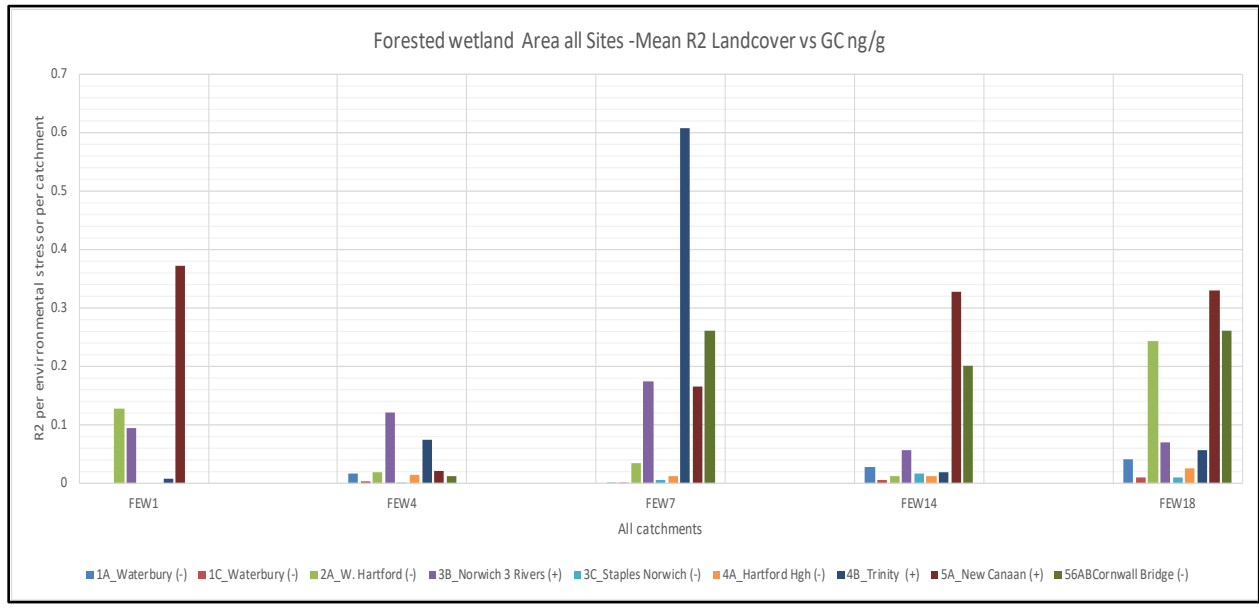


Figure 4.30

Environmental Stressor – Woody Wetlands - Mean R² All Sites, Landcover vs. GC Ng/G

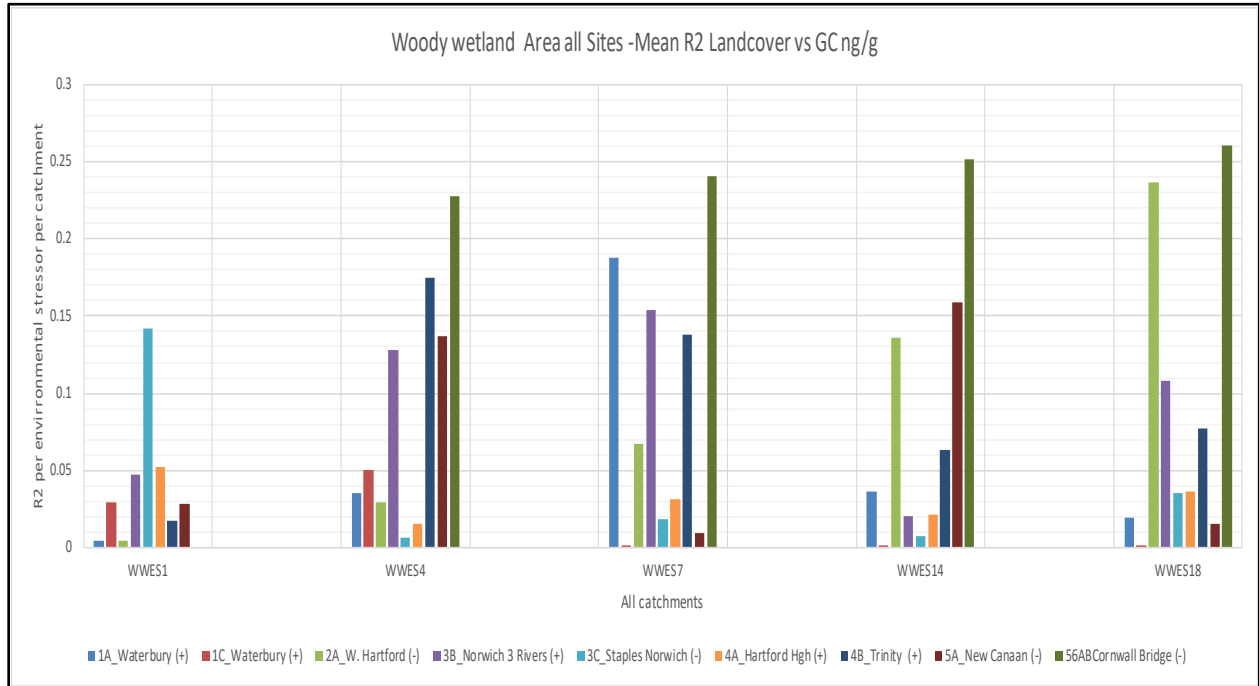


Figure 4.31

Environmental Stressor – Right of Way (ROW) - Mean R² All Sites, Landcover vs. GC Ng/G

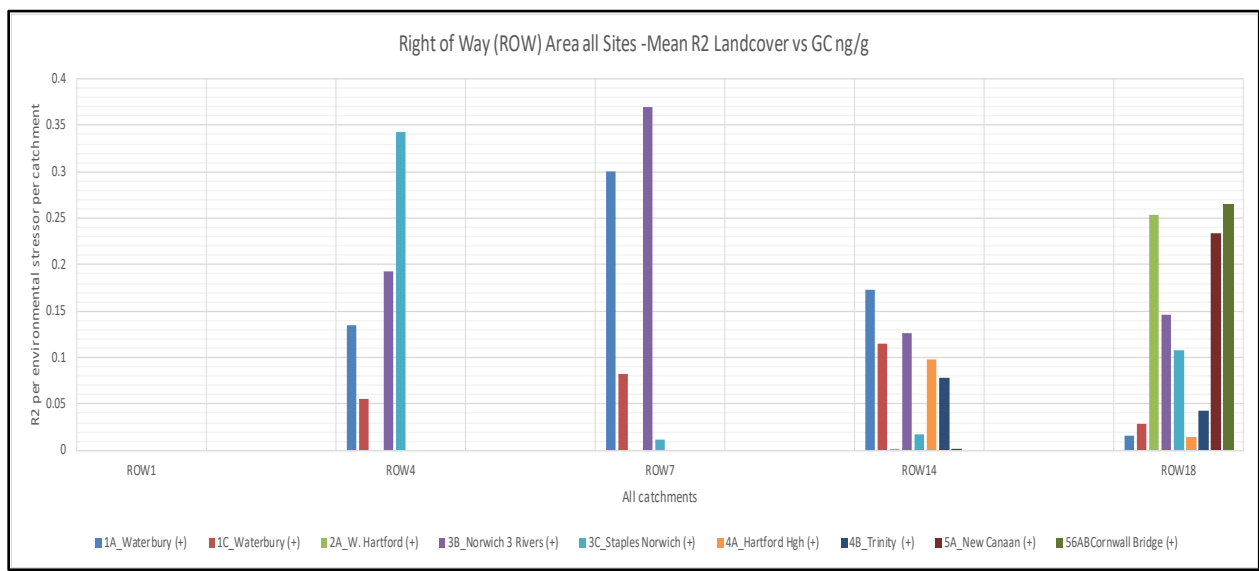


Figure 4.32

Environmental Stressor – Census - Mean R² All Sites, Landcover vs. GC Ng/G

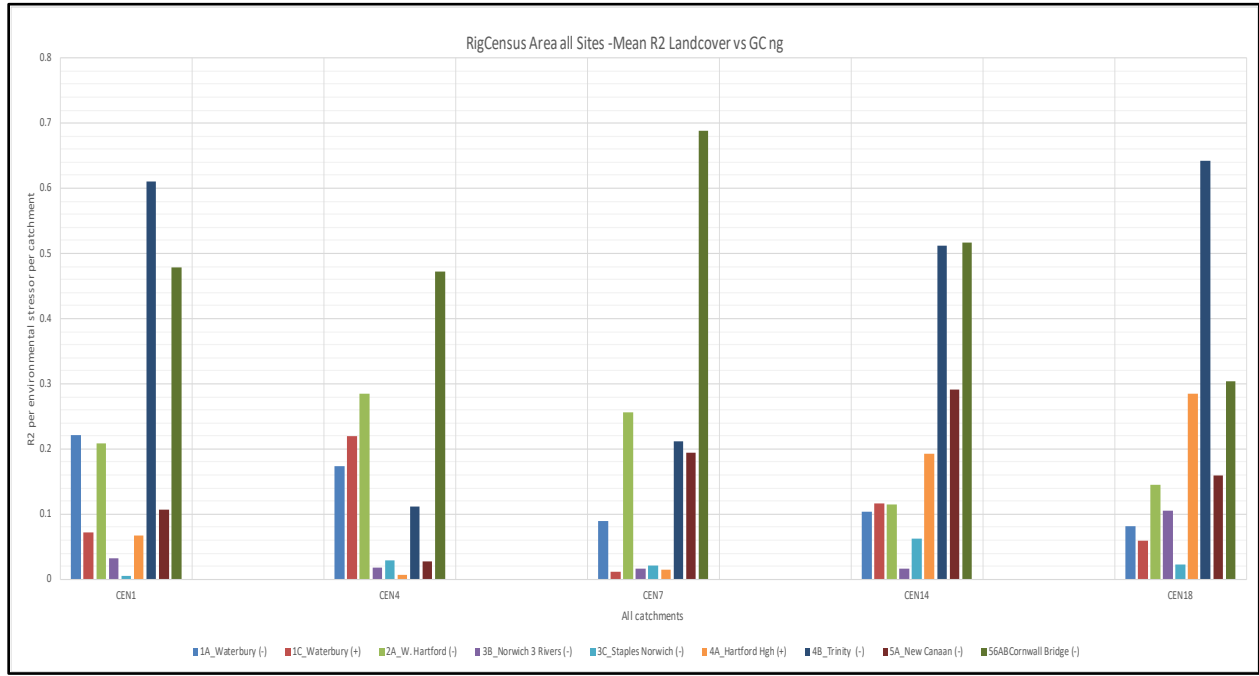
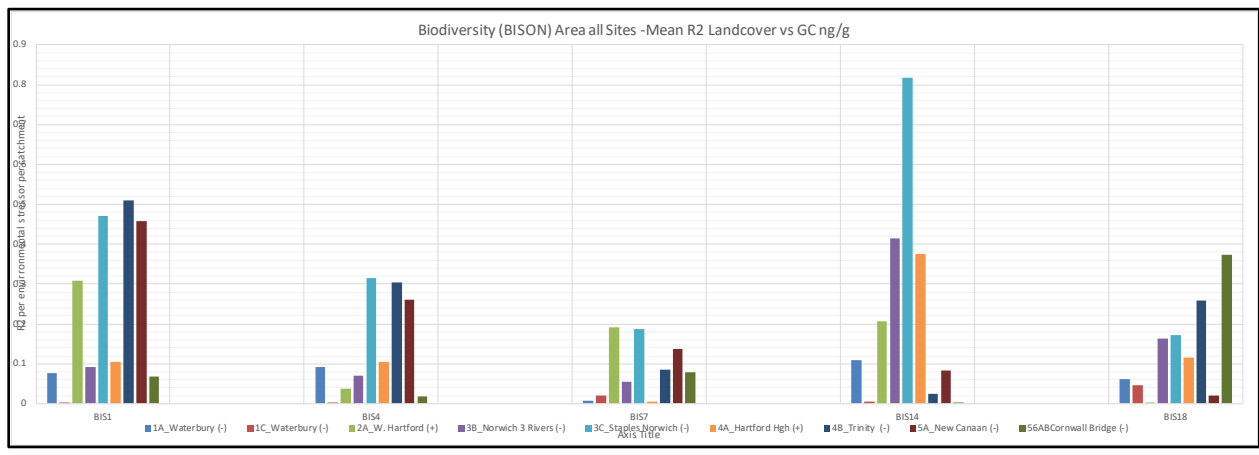


Figure 4.33

Environmental Stressor – BISON - Mean R² All Sites, Landcover vs. GC Ng/G



Figures 4.34–4.41 depict the landcover percentage at each of the sites for each of the catchment sizes compared to the GC level in ng/g.

Figure 4.34

Developed Landcover Percentage for Each Site for Each Catchment vs. GC Level

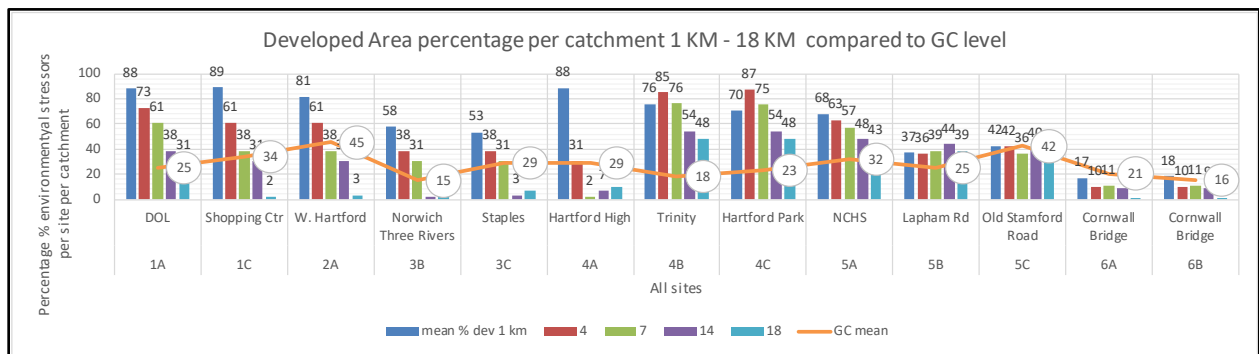


Figure 4.35

Turf Landcover Percentage for Each Site for Each Catchment vs. GC Level

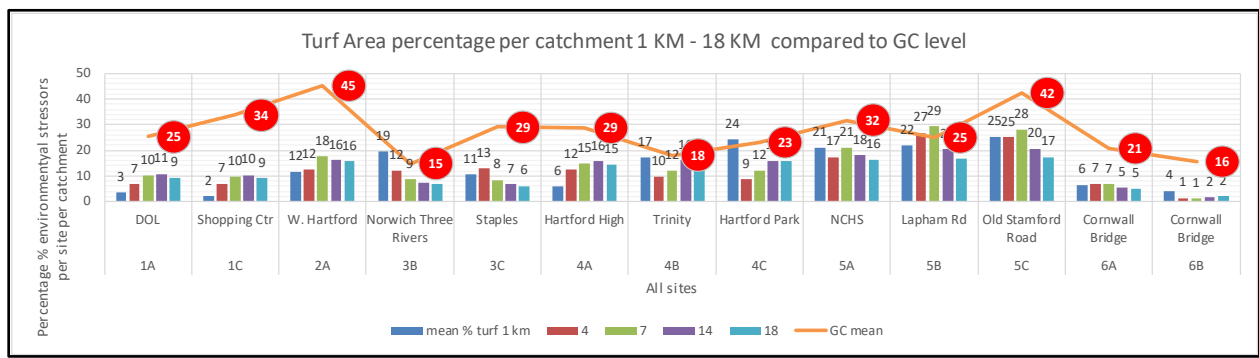


Figure 4.36

Other Grasses Landcover Percentage for Each Site for Each Catchment vs. GC Level

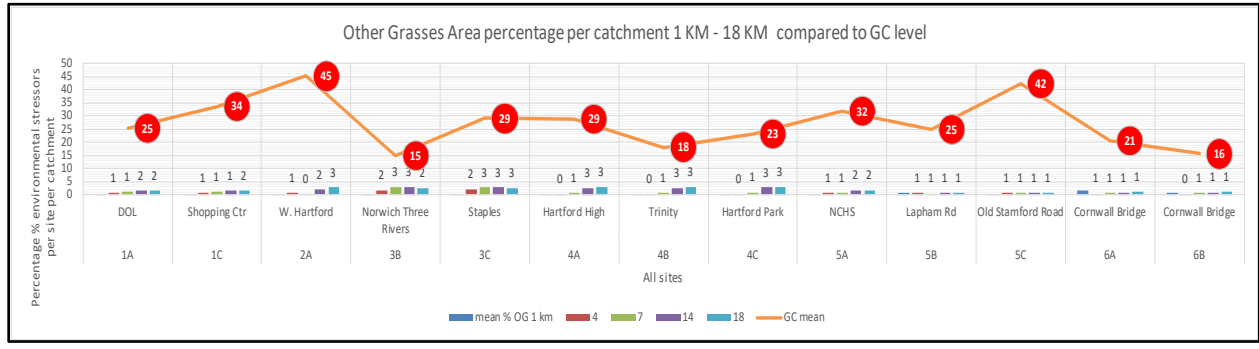


Figure 4.37

Agricultural Landcover Percentage for Each Site for Each Catchment vs. GC Level

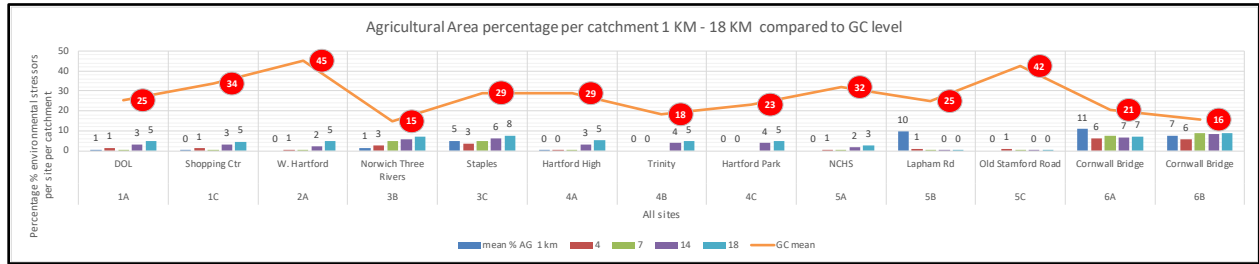


Figure 4.38

Deciduous Landcover Percentage for Each Site for Each Catchment vs. GC Level

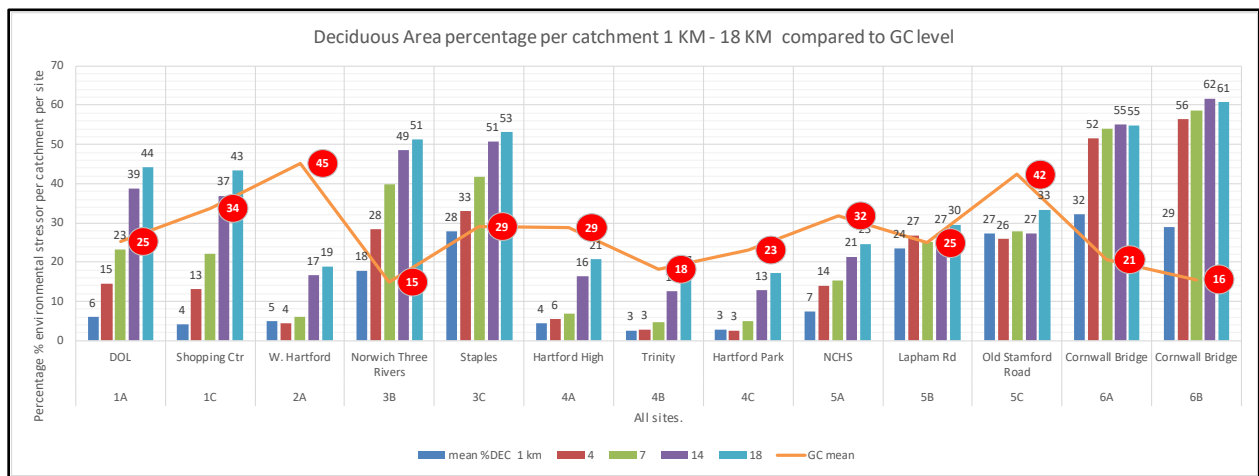


Figure 4.39

Coniferous Landcover Percentage for Each Site for Each Catchment vs. GC Level

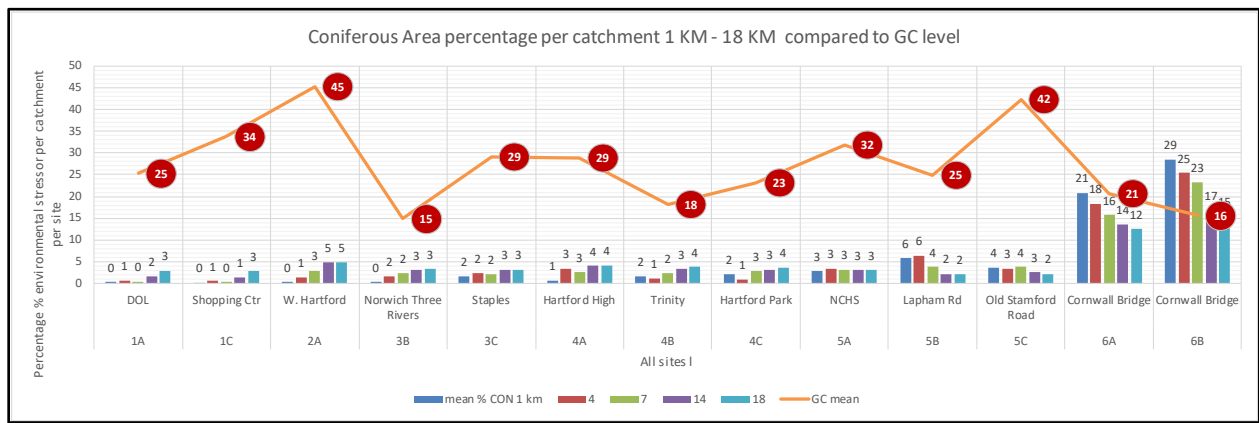


Figure 4.40

Human Census Level for Each Site for Each Catchment vs. GC Level

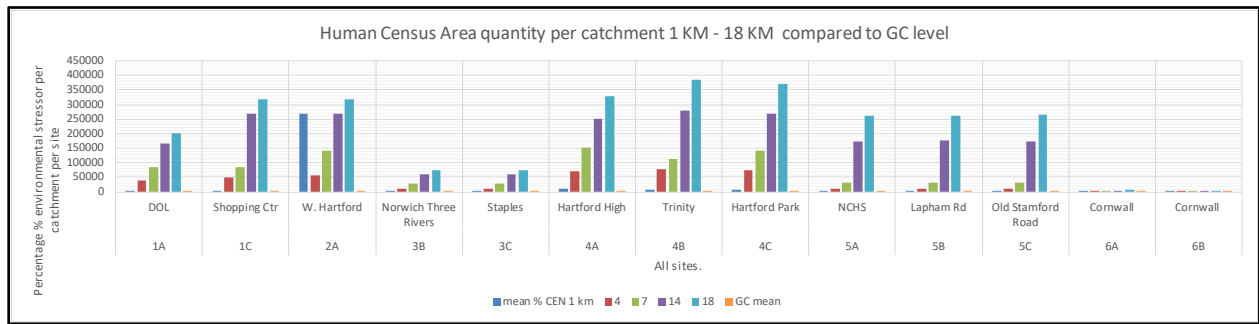
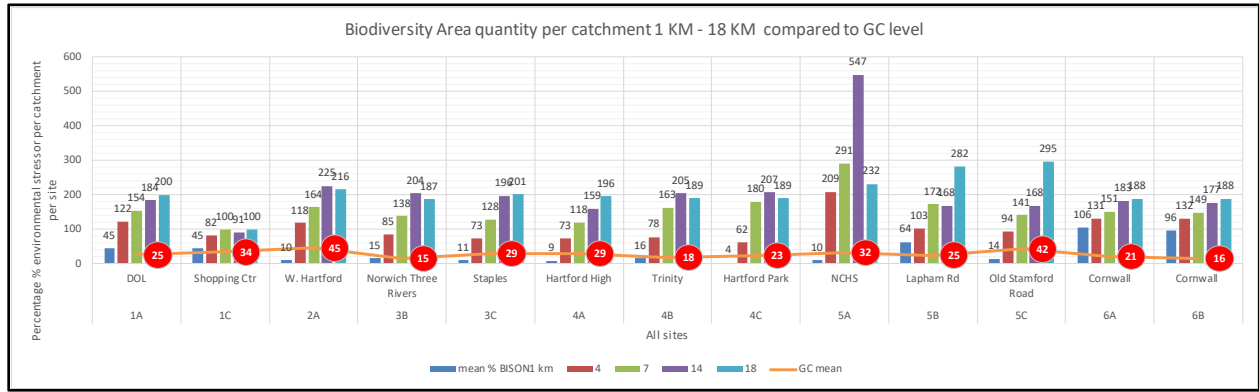


Figure 4.41

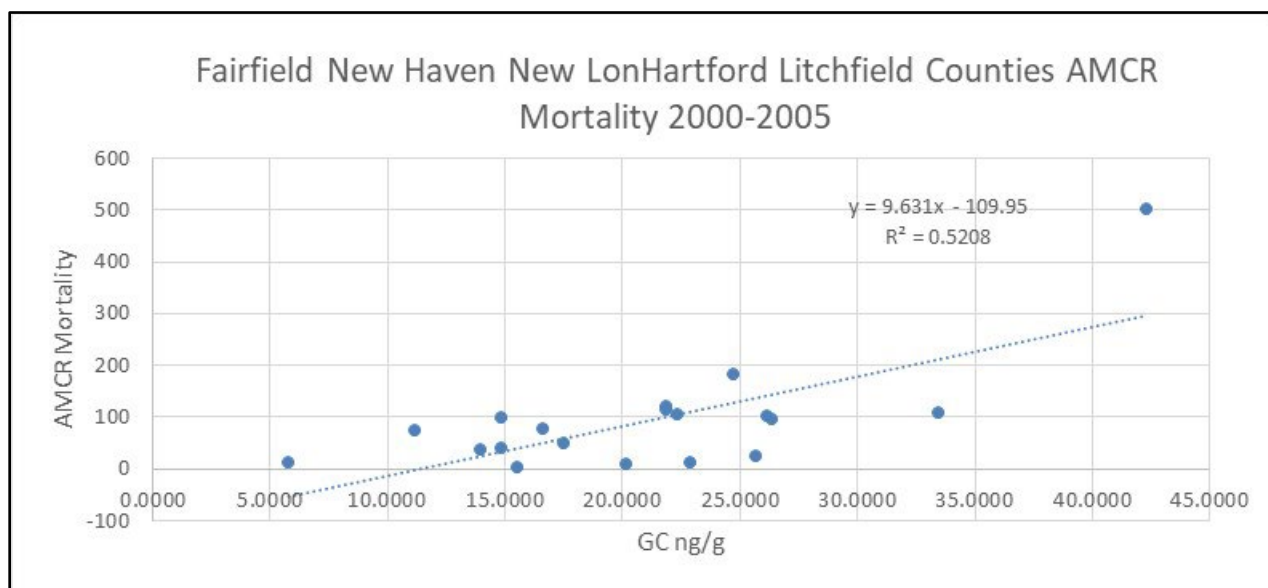
Biodiversity Level for Each Site for Each Catchment vs. GC Level



Considering all the information provided above on the correlation between environmental stressors and their relation to GC levels and stress, those are still statistical analyses, necessarily simplifications of reality. The ultimate question is whether there is a biological basis linking environmental stressors to GC levels to avian stress, in this case leading to AMCR mortality. Figure 4.42 uses data obtained by the author (Grabarz, 2013) and compares AMCR mortality from WNV within five counties in which current GC samples were available from this dissertation research. Other previous work by the author (Grabarz, 2015) found a strong correlation between higher levels of developed area (impermeable surface) and covid mortality.

Figure 4.42

GC Level and Crow Mortality Across 5 CT Counties 2000–2005



The analyses above examined all the information provided on the correlation between environmental stressors and their relation to GC levels and stress, and its biological basis.

The last area of research examines a habitat quality stress index that unites the above findings into a more interactive matrix and as a tool for evaluating various environmental stressor, and stress conditions in the spatial environment. Stress is most often related to the individual species member through serum or less frequently feather or fecal sampling (Vitousek et al., 2018). What these methods miss are the potential direct contribution of the defined physical site area catchment to the environmental stressor itself to the stress response GC level. In addition, this HQSI accounts for the energy expended from the travel distance from each catchment to the central roost area. Building on the previous data then, the index below applies the area of each succeeding physical site area catchment (1, 4, 7, 14, and 18 km) to the specific environmental stressor (developed, turf, deciduous, etc.), and multiplies these catchment areas by the GC level. This provides a bounded area (spatially measurable) environmental stressor with a specific stress value. This stress response area is then divided by the square of the distance between each succeeding catchment to in addition to physical area habitat area, to evaluate the stress of movement and therefore life history from each catchment 1–4, 1–7, 1–14, and 1–18 km as well. A portion of the HQSI is shown below in Table 4.28. The full HQSI is shown in Appendix E which shows total magnitude of stress induced by each environmental stressor for each GC sample for each site.

Generally, the most stressful area is in the 1–4 km catchment areas for developed area (shown in red or terra cotta or brown–poor habitat). Stress gradually reduces moving to the right as the site catchment becomes larger and less stressful due to developed area becoming less concentrated (orange-fair, yellow-good, excellent-green).

The mathematical basis for this relationship is Newton's law of universal gravitation, where gravitational force between two objects is based on their masses and inversely proportional to the square of the distance between them (Cohen, 1999).

$$F = \frac{M_i M_j}{d^\alpha}$$

In subsequent studies, M usually represents the population scale or magnitude of two bodies, such as cities, planets or in my case physical two-dimensional area identified with a specific GC level. The letter " d " represents the Euclidean (or straight line) distance, or in my case the distance between two adjoining catchment areas measured from the origin or centroid of each catchment. Alpha α is the distance attenuation coefficient, that as the square of the distance between the two centroids increases, the magnitude of the force (or in my case) stress decreases. F represents the potential intensity of population movement between the two cities (Zipf, 1949) or in my case the sum Σ of the stress response from the stress exerted from the environmental stressors. In later studies, researchers consider that the intensity of interaction is determined not only by the population magnitude but also by the comprehensive strength (GDP) of the city (Wang et al., 2019). Thus, different researchers have separately set M according to the needs of their own research.

Any geometric area spreading its effect equally in all directions without a limit to its range will follow the inverse square law (Kepler, 1619). Being strictly geometric in its origin, the inverse square law applies to many different types of phenomena (Nave, 2016). In the derivation I have developed the metabolic energy twice as far from the source is spread over four times the area, hence $\frac{1}{4}$ the intensity or for 7x7 or 14x14 or 18x18 it is reduced by that amount linearly except for the differences in GC level for each sample which could vary dramatically.

Table 4.28

Portion of Habitat Quality Stress Index for Developed Area (See Appendix E for Complete HQSI)

Site Name	Sample	Hab Index 1-4	Hab Index 1-7	Hab Index 1-14	Hab Index 1-18
1A DOL	1	218957	59364	9215	3746
1A DOL	2	3478	935	152	60
1A DOL	3	54946	14996	2371	944
1A DOL	4	11231	3050	481	194
1A DOL	9	25806	6902	1086	441
1A DOL	75	25806	35111	5536	2224
1A DOL	76	130451	73857	11651	4715
1A DOL	84	272146	192549	30444	12139
1A DOL	85	708007	133587	21108	8384
1A DOL	86	489006	1287119	203381	81036
1A DOL	87	4711589	169727	26819	10686
1A DOL	88	621299	201739	31877	12728
1A DOL	89	738481	43643	6896	2752
1A DOL	90	159758	28631	4523	1820
1A DOL	91	104781	45852	7245	2890
1A DOL	92	167846	120882	19118	7626
1A DOL	93	442885	269642	42582	17123
1A DOL	94	985932	16762	2651	1057
1A DOL	95	61411	47163	7455	2962
1A DOL	122	172004	28221	4463	1779
1A DOL	136	103311	3175	501	202
1A DOL	137	11698	9027	1428	570
1A DOL	138	33074	21645	3428	1367

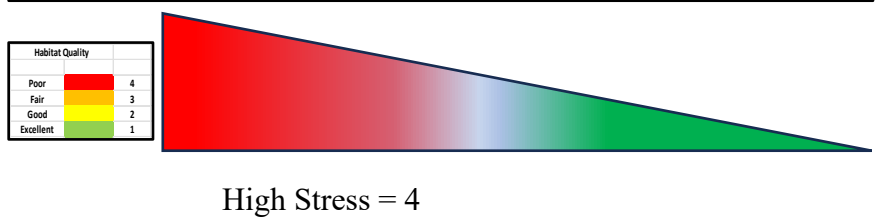


Table 4.29 breaks down the HQSI based on the quantity of catchment combinations totals (1–4, 1–7, 1–14, and 1–18 km) and percentage per site, and their total values per site of habitat quality value of poor, fair, good, and excellent.

Table 4.29

Aggregated % and # of Habitat Quality Stress Indices (HQSI) per Site 1 km - 18 km Catchments

Aggregated % and # of Habitat Quality Stress Indices (HQSI) per Site 1KM - 18 KM Catchments											
SITE	NAME	TOWN	Poor	%	Fair	%	Good	%	Excellent	%	
1A	DOL	Waterbury		65	0.09	102	0.14	130	0.18	439	0.60
1C	Shopping Centert	Waterbury		103	0.07	214	0.16	298	0.22	761	0.55
2A	Shopping Center	W. Hartford		40	0.07	42	0.07	114	0.21	348	0.64
3B	Three Rivers CC	Norwich		15	0.02	70	0.11	119	0.19	436	0.68
3C	Staples	Norwich		21	0.09	38	0.17	42	0.19	123	0.55
4A	Hartford High	Hartford		90	0.06	69	0.12	91	0.16	326	0.57
4B	Trinity	Hartford		15	0.06	41	0.16	47	0.18	153	0.60
5ABC	New Canaan	New Canaan		7	0.03	42	0.19	59	0.26	116	0.52
6AB	Cornwall	Cornwall		9	0.04	22	0.10	44	0.20	158	0.71
				365		640		944		2860	

Table 4.30 shows what I call the “entrained stress level” per environmental stressor per site. Simply, it is the total stress indices for each environmental stressor. To the right of the table is the sequence of entrained stress level per site. This represents the magnitude of the entrained stress and how the environmental stressors vary in severity at each site.

Table 4.30

Entrained Stress Level per Environmental Stressor per Site

Entrained Stress level per environmental stressor per site											Sequence of Entrained Stress level per site							
SITE	NAME	TOWN	1	2	3	4	5	6	7	8	1	2	3	4	5	6	7	8
1A	DOL	Waterbury	13,689,336.65	54,693.48	388.42	258,632.51	12,250.23	33.08	15,579,915.69	5,873,853.97	7.00	1.00	8.00	4.00	2.00	5.00	3.00	6.00
1C	Shopping Centert	Waterbury	44,641,483.10	111,806.71	8,373.61	1,189,313.95	301,976.75	80.40	27,530,941.89	46,979,182.30	8.00	1.00	7.00	4.00	5.00	2.00	3.00	6.00
2A	Shopping Center	W. Hartford	495,408,412.90	1,378,720.17	2,499.81	204,697.46	4,935.66	33.08	15,579,915.69	5,873,853.97	1.00	7.00	8.00	2.00	4.00	5.00	3.00	6.00
3B	Three Rivers CC	Norwich	1,122,646.55	97,143.49	1,827.11	344,613.28	472.90	-	4,385,953.09	13,865.04	7.00	1.00	4.00	2.00	8.00	3.00	4.00	-
3C	Staples	Norwich	1,656,607.28	103,480.31	18,684.08	845,064.84	3,158.46	0.35	4,385,953.09	4,825,752.69	8.00	7.00	1.00	4.00	2.00	3.00	5.00	6.00
4A	Hartford High	Hartford	12,454,392.99	153,965.00	2,998.57	159,449.99	4,433.90	0.35	190,012,777.93	7,372,847.78	7.00	1.00	8.00	4.00	2.00	5.00	3.00	6.00
4B	Trinity	Hartford	2,754,257.97	287,310.71	57,214.17	4,636.51	1,004.89		3,831,216.67	2,880,366.05	7.00	8.00	1.00	2.00	3.00	4.00	5.00	-
5ABC	New Canaan	New Canaan	1,629,446.98	287,310.71	1,401.71	300,864.76	2,390.19		54,115.60	899,450.66	1.00	8.00	4.00	2.00	7.00	5.00	3.00	-
6AB	Cornwall	Cornwall	108,660.88	41,966.41	6,797.36	413,990.13	4,677.42		24,453.42	2,129,745.59	8.00	4.00	1.00	2.00	7.00	3.00	5.00	-

Finally, Table 4.31 below shows the ordinal ranking (4 = poor to 1 = excellent).

Table 4.31

HQSI Ordinal Ranking at Specific Environmental Stressors

		Poor	4_11,000,000.00-500,000,000.00												
		Fair	3_6,000,000.00-10,999,999.00			Developed	Developed								
Total	Total Stress	Good	2_3,000,000.00-5,999,999.00			Stress	Stress								
Stress	HQSI	Excellent	1_0-2,999,999.00			Total	HQSI								
HQSI	Ordinal	Total				HQSI	Ordinal	Developed	Turf	Agriculture	Deciduous	Coniferous	Census	Biodiversity	
Index	Ranking	Stress Value	SITE	NAME	#	Index	Ranking	Dev 1-18	Turf 1-18	AG 1-18	DEC1-18	CON 1-18	CEN1-18	BIS 1-18	
4	4	35,469,070.94	1a	DOL		4	3	13,689,336.65	54,693.48	388.42	258,632.51	12,250.23	15,579,915.69	5,873,853.97	
4	3	120,763,077.32	1C	Shop Ctr		4	2	44,641,483.10	111,806.71	8,372.61	1,189,313.95	301,976.75	27,530,941.89	46,979,182.30	
4	1	497,536,728.21	2A	W.Hart		4	1	495,408,412.90	1,378,720.17	2,499.81	204,697.46	4,935.66	269,919.84	267,542.37	
2	7	5,966,521.46	3B	Norwich		1	8	1,122,646.55	97,143.49	1,827.11	344,613.28	472.90	4,385,953.09	13,865.04	
3	5	10,593,385.83	3C	Staples		1	6	1,656,607.28	103,480.31	18,656.08	845,064.84	3,158.46	3,140,666.17	4,825,752.69	
4	2	210,160,866.16	4A	HH		4	4	12,454,392.99	153,965.00	2,998.57	159,449.99	4,433.90	190,012,777.93	7,372,847.78	
3	6	9,619,812.83	4C	Trinity		1	5	2,754,257.97	91,116.57	57,214.17	4,636.51	1,004.89	3,831,216.67	2,880,366.05	
2	8	3,174,980.59	5ABC	New Canaan		1	7	1,629,446.98	287,310.71	1,401.71	300,864.76	2,390.19	54,115.60	899,450.66	
1	9	2,730,291.22	6AB	Cornwall		1	9	108,660.88	41,966.41	6,797.36	413,990.13	4,677.42	24,453.42	2,129,745.59	

CHAPTER V: DISCUSSION

The preservation and enhancement of biodiversity is a major issue today, not simply for the axiomatic sake of preserving, but rather as a necessary and vital ecosystem service. As an ecosystem service, biodiversity is a critical component of human health and wellbeing (Bernstein, 2008). However, it is not only a factor in clean air and water. It is also a potential buffer against the spread of disease into the human population based on the theory of immuno-competence (Lafferty & Gerber, 2002). A key component of biodiversity is habitat selection and its corollary habitat quality, consisting of fauna, flora, and anthropogenic environments including landcover, that I characterize collectively as environmental stressors (ESs). There has not been any research on the identification, derivation, spatial extent, or evaluation of the efficacy of multiple ES's that can initiate a stress response in AMCR. Research characterizing stress levels in organisms generally makes no mention of the number of different types of environmental stressors nor the multiple quantitative spatial boundaries (that I describe as catchments) from which stress response should be evaluated. Without these definitive spatial boundaries and an evaluation of a taxonomy (classification) of ESs it is difficult to establish the root cause and effect of habitat quality on stress response. Further, this lack of boundary definition prevents broader understanding of its impact on proximate fitness and how that could modulate disease transmission. This stressor-stress-stress response model needs to begin with the identification and measurement of the environmental stressor and then the stress/stress response via GCs in the spatial environmental landscape and bounded and quantified spatially. This spatial bounding becomes more crucial when scaling responses of the individual organism up to the population, the communities, and the interspecific interactions therein. This continuum then establishes the larger context of stressors, stress and stress response related to biodiversity.

More specifically, the magnitude of a healthy biodiversity at a landscape scale is likely related to the magnitude of ESs and their effect on organisms' fitness. Delineating stressors at a defined landscape scale from 1 –18 km within the framework of my HQSI, reveals the areas of high and low habitat quality. The mechanism for this delineation is the quantitatively derived stress response, rather than the historic solution of floral and faunal qualitative visual indicators defining habitat quality. In so doing, this shows the cumulative life history impacts of individuals and populations more concisely, which facilitates the ability to create biodiverse conservation areas more effectively. Creation of those more biodiverse conservation areas will be based on the quantitatively derived buffers to potentially reduce the spread of disease within these environments.

The advent of increasingly detailed geographic information systems (GIS) due to more comprehensive remote sensing global navigation satellite systems (GNSS) data (National Oceanic and Atmospheric Administration, 2021), has dramatically increased the ability to unite detailed spatial extent to precise location for the establishment of these buffers. This GNSS enterprise-wide specificity has increased the ability to track point data such as stress level that even ten years ago was not possible. Measured by the GC level, my research shows the varying intensity of 15 ESs at specific locations at various spatial area sizes and their respective efficacy statistically.

Measured by the R^2 coefficient of determination, these data show the strength of relationship between GC levels and those ESs. This characteristic shows the importance of focusing on the higher R^2 value areas as the determinants of reliability as the basis of habitat quality and its relationship to stress. The lack of a baseline standard environmental stressor level of R^2 at multiple sites precludes the derivation of this intensity and efficacy that varies between

sites. This points to the problem of the potential “one size fits all” solutions in the establishment of biodiversity buffers.

I have defined five specific catchment area sizes as notional territories of AMCR based on historical data at 13 sites at six different geographical locations. No previous studies have created an overall index linking all the catchment sizes and stressors together as a habitat quality stress index (HQSI).

To further explore these issues, my research questions and responses include the following:

1. What is the magnitude and relationship of non-invasive GC hormones, (as an analog for stress response in AMCR), at and compared with multiple site locations?

The size and relationship of the GCs could be due to differences in habitat quality (based on biotic and anthropogenic ESs) that could alter proximate fitness outcomes, potentially changing its susceptibility (reservoir competence) to disease.

I hypothesized that stress hormone level differences or circulating steroid hormone levels (glucocorticoids) would be higher in areas of low habitat quality. I define areas of low habitat quality as represented by % developed (impermeable) surface areas (Bonier, 2012) and relative to my research sites from high to low habitat quality as: 6A/B, Cornwall (5.57%); 5 A/B/C, New Canaan (31.57%); 3B/3C, Norwich (31.58%); 2A, West Hartford (47.29%); 1A/C, Waterbury (55.72%); and 4A/B, Hartford (71.92%; Appendix B). These can be correlated to the rural to urban landscape gradient.

Examining the box and whisker plot in Figure 4.4, I established a baseline of GC levels that was able to cross the maximum number of sites. That baseline is observed at approximately the level of 27 ng/g GC across all the sites, except for Site 3B (Three Rivers Community

College) and Site 6B (Cornwall). These sites both have GC levels consistently below the baseline level. Looking at the waterfall charts in Figures 4.8 and 4.13 you can see graphically the reason for this phenomena. In both cases the level of ES particularly at the developed area end of the scale is very shallow, suggesting a very smooth transition from developed (historically more highly stressed) to lesser stress.

More specifically and using the calculated ES (landcover) percentage from the exact location of the average GC sample, the sites with the lowest to highest GC mean (highest to lowest habitat quality using 1 km mean developed environmental stressor %) is: 3B Three Rivers Norwich, 14.82 ng/g (59.44% developed); 6B Cornwall, 15.57 ng/g (15.61% developed); 4B Trinity, 18.12 ng/g (77.86% developed); 6A Cornwall, 20.60 ng/g (16.65% developed); 4C Hartford Park, 23.11 ng/g, (70.20% developed); 5B Lapham Road New Canaan, 24.89 ng/g (37.00% developed); 1A DOL Waterbury, 25.24 ng/g (88.04% developed); 4A Hartford High School, 28.79 ng/g (93.59% developed); 3C Staples Norwich, 29.04 ng/g (53.40% developed); 5A New Canaan High School, 31.77 ng/g (67.86% developed); 1C Shopping Center Waterbury, 33.71 ng/g (93.15% developed); 5C Old Stamford Road, 42.33 ng/g (42.33% developed); 2A West Hartford, 45.16 ng/g (86.42% developed; Table 4.1, Figures 4.3, 4.4, and 4.14–4.21). I confirmed these findings statistically (that biologically GC hormones as an analog for stress were higher at more developed sites by performing *F*-tests (Table 4.2). These were done on pairs of sites with an $\alpha = 0.05$ to validate similarities or differences between each site. Those tests resulted in the following:

- 1A_Waterbury – 1C_Waterbury $H_0 = H_1$
- 1A_Waterbury - 2A_W. Hartford $H_0 = H_1$
- 1A_Waterbury – 3B_Norwich $H_0 \neq H_1$
- 1A_Waterbury – 3C_Staples $H_0 \neq H_1$
- 1A_Waterbury – 4A_Hartford High $H_0 = H_1$
- 1A_Waterbury – 4B_Trinity College $H_0 \neq H_1$
- 1A_Waterbury – 5ABC_New Canaan $H_0 \neq H_1$
- 1A_Waterbury – 6AB_Cornwall $H_0 \neq H_1$

Both 1A/1C Waterbury sites as well as 2A West Hartford and Hartford High are highly urbanized with significantly developed permeable surface levels at 56%, 47% and 72% respectively, and the GC level is consistent with those high percentages of impermeable surface.

In addition, there are abrupt changes in the ESs from developed area to turf to deciduous trees at Site 1A DOL, Site 1C Shopping Center, and Site 2A West Hartford where significant and rapid changes of the stressors over the catchment distances bring about significant alteration to homeostatic mechanisms (Figures 4.5–4.13). Finally, looking at the actual level of GC and ESs for developed area percentage is shown graphically on (Figure 4.14). The dramatic difference from what the average developed (impermeable area) percentage is at a community (political boundary) level (Appendix B) compared to the specific sample locations (Figure 4.14) is exceptional. Part of this difference is due to the statutory framework where zoning laws preceded planning laws to separate human activities rather than plan for their growth more organically with other uses in the 1920s (Mandelker, 1982). This establishes an illusory sense of homogeneity where one size fits all visually and thus spatially. In fact, the stress and stress response effects in response to ESs created by this natural heterogeneity (Figure 4.14 for

development (impermeable surface) but for all other ES as well) of human and non-human development has remained far more elusive. These three sites show abrupt changes compared to the gradual conditions seen at the other sites (e.g., Site 3B Three Rivers, Site 5 ABC New Canaan, or Site 6AB Cornwall), indicating a challenge in returning to baseline homeostasis. This is further described in Figures 4.32–4.41 which shows each relevant environmental stressor per site at each of the catchment areas and the attendant GC mean at that location.

The type of relationship, direct (+), or inverse (-) varies for all sites for all catchment sizes as is shown in Table 4.14. The common belief has been that the more urbanized end of the landscape gradient being more impermeable, is necessarily directly related to more stress (+) and the more rural end of the gradient being less impermeable is less stressful (-) (McDonnell et al., 1997). However, looking at a larger number of types of stressors at a variety of spatial extents (catchments) a more complex picture emerges. Perhaps there is a degree of what I would characterize as *entrained stress*, developed from stressors located further away from the GC sample location stress response. Thus, stress would be the result of AMCR living in multiple catchments, some more stressful than others, that maintain a latent (embedded) stress level in AMCR that is exhibited in differing statistical relationships. Recall that previous research on avian GC's from HormoneBase, a wide-ranging database of unmanipulated free-living organisms' glucocorticoids (2017) shows GC mean levels from 4.03 ng/g to 22.67 ng/g (1982–1984; Johnson et al., 2017) in the Southern New England area (40N–42 N. latitude) for avian species. This is significantly lower than the baseline of 27 ng/g mean that I found in the same southern New England area.

2. How will stress response in AMCR be affected by variation in environmental stressors (representing habitat quality), at multiple site locations, at multiple size catchments?

I hypothesized that as the catchment area increases, the magnitude of the stress effect as measured by GC will vary in direct proportion to the catchment size. I would then evaluate the efficacy of this relationship with the R^2 coefficient of determination. I further hypothesized that if developed area decreases as the catchment size increases the level of stress should be reduced while still maintaining the same alpha level, i.e., having the same explanatory power.

Looking at the coefficient of determination or R^2 at each of these sites where the independent X variable is landcover (environmental stressor) and the dependent Y variable GC level (stress response), the degree of association between landcover and GC level begins to emerge. Figures 4.22–4.33 and Table 4.26 shows the average R^2 value of each of the sites at each of the catchments individually graphically and tabularly. As the table shows when averaging the R^2 on a per site basis including all catchments (1–18 km) the highest value is at Site 4B Trinity College Hartford at 0.5431, followed by Site 2A West Hartford at 0.4443, followed by Site 3B Three Rivers Community College Norwich at 0.3559. The lowest level is at Site 3C Staples at 0.0295. Individually per site per catchment the highest values were at: Site 4B Trinity 1 km: 0.2406; Site 5 ABC New Canaan 4 km: 0.4709; Site 2A Shopping Center 7 km: 0.2552; Site 1A DOL 14 km: 0.3290; Site 6AB 18 km: 0.7544.

Examining the highest level of R^2 per catchment size, the highest level was at Site 4B Trinity College Hartford at 18 km and $R^2=0.6510$, followed by Site 5 New Canaan at 4 km and $R^2=0.4709$, then Site 4A Hartford High School at 4 km and $R^2=0.4240$. This was followed by Site 6 at Cornwall at 4 km and $R^2=0.3779$, then Site 5 New Canaan at 14 km and $R^2=0.2983$,

followed by Site 1A DOL Waterbury at 1 km and $R^2=0.2600$ and Site 2A West Hartford at 7 km and $R^2=0.2552$.

Cumulatively on a per catchment size basis the highest R^2 occurred at the 18 km level = 0.7544 at Site 6A/B, followed by 14 km level = 0.507 at Site 6 A/B, then 4 km = 0.4709 at Site 5A/B/C, then 7 km at 0.2552 at Site 2A, and lastly 1 km at 0.2600 at Site 1A. A key takeaway when examining the sites on an average per catchment basis is the significant variation of average R^2 values of all environmental stressors, and the direct (+) and indirect (-) relationships of environmental stressor to GC level. Across the board as Table 4.26 shows, each site location has a specific catchment size where the R^2 value has the most efficacy. Equally important is that the more highly developed the site (as in 1A or 1B) does not necessarily signify a higher R^2 value in terms of efficacy, even at the 1 km catchment level. This is noteworthy as much of the literature (Bonier, 2012; Bradley et al., 2008;Muellar et al., 2011) has often suggested that the most developed areas (most impermeable surface), being the most stressful.

Looking at Tables 4.3–4.12 on average, the developed ES for all the sites does indeed have one of the higher average R^2 value at each catchment level. These R^2 values range from 0.2754 at 1 km to 0.3177 at 14 km suggesting that the developed landcover percentage offers some reasonable explanatory power for the stress response represented by the GC level. Interestingly though, looking at Tables 4.4 through 4.10, the agricultural ES has R^2 of 0.1434 to 0.4326, while the Deciduous ES has values of 0.1938 to 0.3313. Further, the Census ES, which is arguably closest to the Developed ES in anthropogenic impact has values from 0.1237 to 0.3248. Indeed, looking at Figures 4.22-4.33, each of the comprehensive list of environmental stressors individually has a maximum R^2 values that is intrinsic to each ES for stress response for various catchments. This shows a degree of reliability of environmental stressor compositional

validity beyond the traditional evaluation of developed site as the primary location for higher stress response. While these R^2 are low by historic standards, they do show variational differences between environmental stressors that can act as guideposts for future research. The maximum significant R^2 value > 0.10 for each site is shown in Table 4.13, the number of significant environmental stressors and the breadth of environmental stressors (number of ES for each site having significant value ($R^2 > 0.10$)). This number is an indication per site of how many ESs R^2 exceed 0.10 to show the importance of ES percentage and GC value on average. Overall, this table provides a detailed breakdown of the contribution of other environmental stressors to the level of stress relative to habitat quality which is more granularly described on Tables 4.17–4.25. The average R^2 for each site, catchment, and ES is shown in Tables 4.17–4.25. Notably, the sites with the highest number of significant value catchments and breadth of ES's are Site 5A/B/C New Canaan at breadth (B) = 12 and significant value (SV) = 48; Site 6A/B Cornwall at B = 10 and SV = 36; Site 4B Trinity B = 10 and SV = 24; and Site 2A West Hartford B = 10 and SV = 23. This is important again, as the historic way of evaluating stress has been for the most part as a singular reliance on developed or impermeable surface as being responsible for all stress and thus stress response. At a much finer scale level, there are several other environmental stressors that either contribute to or mediate such stress, that these tables begin to show. This means then that in two cases Site 4B Trinity and Site 2A West Hartford, the efficacy of the R^2 averages is significant across multiple ESs and catchment sizes at the urbanized end. However, the magnitude of this finding occurs equally at the opposite more rural end of the scale. The average GC level and the most significant ES level is shown more fully in Figures 4.34–4.41. More specifically, this is in terms of the largest percentage spatially at each

site and catchment area. Thus, its importance to overall ES and stress response along the entire landscape gradient is shown.

3. What is the effect on county wide mortality of AMCR due to GC level stress response at multiple sites.

While statistical methods can provide a degree of understanding of the correlation of landcover to stress response, this process lacks the biological underpinnings of a true correlation to an immunological process. To test the efficacy of the multiple ES's and its impact on fitness that could affect immune response I have previously evaluated a series of landcover relationships for eight Connecticut counties. The landcover relationship also included human population, human pathology, vector and AMCR mortality due to West Nile virus (WNV) with R^2 results in the 0.0718 – 70.39 range ($n = 104$ and $P = 0.00$ and $\alpha = 0.05$, 2000–2012; Grabarz, 2013). Narrowing this analysis to more specific years and landcover ESs and AMCR mortality, I obtained R^2 results in the 0.18 to 0.82 range ($n = 16$ –48 and $P = 0.00$ and $\alpha = 0.05$, 2000–2003, 2001–2002; Grabarz, 2015). Further as part of a pilot study I examined the observable social behaviors of AMCR that might differ between rural and urban sites in response to stress induced by differences in habitat quality, characterized as ESs. Using chi-square, I found validity at both ends of the rural to urban landscape gradient for social behavior as part of stress response that could facilitate WNV due to changes in habitat quality (Grabarz, 2017). Building on that previous research, in this dissertation I evaluated the five counties within which I had more refined municipal (town-wide) ES landcover data and now stress hormone (GC) data. These counties and communities are Fairfield County (New Canaan); New Haven County (Waterbury), Hartford County (Hartford and West Hartford), New London County (Norwich); and Litchfield County (Cornwall). I analyzed GC levels as the independent (X) variable and AMCR mortality

as the dependent (Y) variable which resulted in $R^2 = 0.52$ where $n = 19$, $P = 0$, $\alpha = 0.05$, 2000–2005) as Figure 4.42 shows. While linked spatially and not temporally, this still provides a degree of confirmation of the validity of the concept of stress response through the analog of landscape quality using GC levels. Thus, there is a positive correlation between various environmental stressors (landcover percentage/type) and AMCR mortality as there is between GC stress response level and AMCR mortality. Tying these three variables, (landcover percentage, GC level and AMCR mortality) together moves this concept of stress response and landscape quality out of the realm of purely statistical significance to biological significance.

4. What is the three-dimensional dynamic stress response, due to environmental stressors in AMCR, at multiple catchment sizes, across the geographical studied sites, through a lens that I characterize as a habitat quality stress index (HQSI)?

I hypothesized that stress response (GC magnitude) should be highest at Hartford, (71.92% developed) followed by Waterbury (55.72% developed), then West Hartford (47.29% developed) followed by Norwich (31.58% developed), New Canaan (31.57% developed), and finally Cornwall the least developed (5.57% developed).

As noted previously in Appendix B, the differences of ES at a community political boundary level view versus a GNSS remotely sensed ground level view with GIS, highlights the need for a unifying framework for the evaluation of stress and stress response from ESs that is representative of an indicator of habitat quality. Such a framework sets boundaries a priori to measure the effects of stress resulting in stress response due to ESs within that bounded space. The bounded space is three dimensional as it presumes organisms within that space either move two dimensionally on the ground, fly in the air, or are affected by other organisms on an interspecific basis (competition, predation, foraging) that can thus operate in that

three-dimensional space. This movement, as an analog for life history processes, and as an example of energy utilization, is thereby a form of stress, and is accommodated through the algorithm in terms of distance between catchments. The analog for stress level and stress response are GC levels and are presumed to operate within the confines of the spatial boundaries set by the catchments established. The ES area, in conjunction with the GC level and the distance between various catchments collectively aggregates to a Habitat Quality Stress Index (HQSI) that along with an ordinal value 1–4 establishes the level of habitat quality (excellent = 1, poor = 4).

Differences between static levels of GC stress and stress response (as previously described), and the HQSI would likely differ based on movement within the catchments, as part of habitat selection and dispersal, competition, predation, foraging and mating. Examining those potential differences, looking at Figures 4.1 and 4.4 on average Site 2A West Hartford for instance, is more than double the stress response (highest average GC level at 58.19 ng/g) than the next highest location. Site 1 C Waterbury Shopping Center was next in GC magnitude at 37.26 ng/g and Site 1A Waterbury DOL at 34.95 ng/g. Next was Site 3C Staples in Norwich which was 33.16. ng/g followed closely by Site 4A Hartford High at 32.59 ng/g. The next level was Site 5B Lapham Road New Canaan at 31.25 ng/g. This was followed by Site 4B Trinity College Hartford at 28.94 ng/g, and Site 4C Hartford Park at 28.25 ng/g. Lastly, Site 6A and 6 B Cornwall with 26.78 ng/g and 18.12 ng/g respectively, then Site 3B Three Rivers at 20.78 ng/g.

Why the disparity? The top four sites in GC levels (stress response) were surrounded by commercial parking lots (impermeable surfaces) with a significant level of human impact, vehicles and people passing in and out, and significant amounts of artificial light. Hartford High School while located in the densest part of the city, was a large expanse of turfed lawn and trees.

Being an urban high school, most transportation to and from the school was public conveyance, which required little impermeable surface. Additionally, few students or staff were present after about 1600 Eastern time. Lapham Road in New Canaan while rural appearing and surrounded by the 300-acre Waveny Park is within several miles of the highly dense urban area of Stamford Connecticut and the I-95 transportation corridor of New York City and Westchester County New York. Therefore, from a catchment and travel perspective for AMCR, flying within this area of up to 9 km for an 18 km catchment for territory, it could be a very stressful condition. Trinity College, like Hartford High School while surrounded by very dense areas, is located within an essential refuge, and topographically separated vertically by a 100.0' elevation difference from the surrounding area. Lastly, Cornwall, with the lowest GC levels in Litchfield County, is located within the least dense area of Connecticut. and unlike New Canaan - Lapham Road (within its 18 km catchment area being close to an urban center) remains very isolated. Many years ago, potential transportation arteries connecting lower Fairfield County Connecticut to I-90 in Massachusetts via State Route 7 running through Cornwall were averted by sensitive environmental concerns.

The consistency for the highest GC values within these sites was confirmed using Fisher tests. Those results show the same population mean and variances for Sites 1A/1C/2A/4A. This reinforces that these four sites GC levels (32.59–58.19 ng/g) are consistently more urbanized sites. At the other end of the stress gradient Fisher tests show that 3B/3C/4B/5ABC/6AB are not part of the same population mean and variance as the other sites stress gradient (18.12–31.25 ng/g).

Now, to examine how the HQSI would evaluate these same sites as above, after performing the calculations for the gravity model in Chapter IV, there were eight significant

(ESs; Table 4.13). The significance was based on stress index values exceeding ≥ 0.10 . Those eight ES values are: developed, turf, agriculture, deciduous, coniferous, non-forested wetlands (not included in the final HQSI due to low physical representation), Human Census, and Biodiversity. In the HQSI itself the physical extent of the stress index spatial area is divided into four catchment units: 1–4, 1–7, 1–14, and 1–18 km for each of the sites and for each of the sample locations resulting in a 4,864-cell stress index covering 49,248 square kilometers at 13 different site locations. Examining the stress values of each of the sites in the HQSI (Table 4.28) at an $\alpha = 0.05$ (for a portion of the HQSI Site 1A DOL whereas the full HQSI is shown in Appendix E), and evaluating for independent means via an F test, all site stress values rejected the null, indicating independent means. Further all the magnitudes of the stress values are consistent with the overall GC values at the individual sites as per Figures 4.1 and 4.4.

Examining the ranking of the sites in the HQSI, I summed the stress value for each of the ES for each site. I then developed a numerical ranking of habitat quality from 1 = excellent habitat quality (green - 0–2,999,999.00), 2 = good habitat quality (yellow - 3,000,000.00–5,999,999.00), 3 = fair habitat quality (orange - 6,000,000.00–10,999,999.00) and 4 = poor habitat quality (red - 11,000,000.00–500,000,00.00). Analyzing the HQSI more closely, Table 4.30 shows the quantities and percentages of the index's metrics of excellent, good, fair, and poor, aggregating or adding all the ES for each site. From a total of 4,864 cells, each of which representing stress indices, there were 60% excellent locations, 20% good locations, 13% fair locations, and 7% poor locations. Further, F tests performed had similar results to those found in Table 4.2, namely the null was rejected for sites 3B, 5ABC, and 6AB and accepted for all other sites, suggesting a degree of efficacy between GC values and location and stress index magnitude. Table 4.30 shows what I call the entrained stress level per environmental stressor per site. It establishes a

total or “entrained” stress level derived from the GC level at each sample location for each environmental stressor for each site. A key finding in my research refutes the notion that the developed area environmental stressor induces the most stress in organisms. As Table 4.31 shows it has been the human population census environmental stressor and the biodiversity environmental stressor (BISON) that is the primary ES. This is the case at all sites except Site 2A West Hartford and interestingly 5ABC New Canaan. This condition holds also for the secondary and tertiary condition as well as per the right side of Table 4.30 entrained stress level per environmental stressor per site - sequence of entrained stress level per site.

Now reviewing the evaluation process using the HQSI model examining Table 4.31 HQSI Ordinal Ranking at Specific Environmental Stressors Total Stress HQSI Ordinal Ranking that were extracted from the HQSI levels in Appendix E, the rankings of individual sites for stress were: 1-Site 2A West Hartford, 497,536, 728; 2-Site 4A Hartford High School, 210,160,866; 3-Site 1C Shopping Center, 120,763,077; 4-Site 1A DOL, 35,469,070; 5-Site 3C Staples, 10,593,381; 6-Site 4B Trinity College Hartford, 9,619,8 12; 7-Site 3B Norwich, 5,966,521; 8-Site 5A/B/C New Canaan, 3,174,981; 9-Site 6A/B Cornwall, 2,730,291.

Further between Figure 4.1 specific GC levels per site location and Table 4.31 the total stress as derived from the HQSI while the individual ordinal ranking does not seem very similar, in both cases the top five in ranking are all the most highly stressed environments. Further, the lower four sites are the least stressed in both Figure 4.12 and Table 15, strengthening the efficacy of the HQSI with its relationship to GC levels and habitat quality. This is additionally shown clearly in the HQSI Ordinal Ranking at Specific Environmental Stressors (Table 4.31) where the highest stressed sites are 1A DOL, 1C Shopping Center, 2A W. Hartford, and 4A Hartford High School.

Waterfall charts Figures 4.5–4.13 show a fundamental tenet of this nonlinear aspect of these individual sites namely increased stress response, due to abrupt and irregular environmental stressor (landcover) changes. These changes coincide with the premise of increased stress levels requiring more rapid amelioration of homeostasis via the negative feedback mechanism. Initiating this feedback leads to a cascade of hormonal changes required to re-establish equilibrium.

CHAPTER VI: CONCLUSION

A key aspect of this study was to validate the correlation of biodiversity and human disease mitigation, as a general principle, that could yield significant utility in the public policy realm of health care. The basis of that research was to develop a better understanding of stress response due to environmental stressors as an analog for habitat quality and thus disease formation. A key question concerning this topic is the efficacy of the relationship between the magnitude of the ES of habitat quality (a critical component of biodiversity) and stress response within organisms. A significant percentage of human diseases originate in other taxa, and as habitat quality is a key component of stress in these other taxa, and stress contributes to disease, understanding more of that mechanism's origin is helpful in ameliorating human disease transmission. My findings showed that stress response as measured by GC does indeed increase with developed area (as well as other ESs). However, it varies in intensity depending on the percentage of development and other stressors though the efficacy of those findings as measured by R^2 vary dramatically, based on site location and catchment (territorial size).

This study evaluated the magnitude of ESs and its effect on an avian taxa's health (proximate fitness). I found varying relationship (direct and inverse) between certain ES and hormonal stress that varies across a landscape gradient and occur at multiple catchment sites, often at variance with the ubiquitous use of developed area as the primary environmental stressor. I expected and found that this stress will impact proximate fitness in a common avian species, AMCR within the rural to urban landscape gradient—a species that is recognized as a key vector for the transmission of human disease, including West Nile virus. I confirmed this by comparing the GC levels obtained in the same counties as the AMCR mortality was obtained and found R^2 results in the 0.52 range.

Further I found three overarching findings in my research. The first involved heterogeneity versus homogeneity. The historically understood linear and homogeneously composed landscape gradient has a much greater extracellular or episodic specific, and highly heterogenous nature. Examining GIS raster imagery for instance, yields dramatic differentiation of land cover types over very small areas ($< 0.1 \text{ km}^2$) that indicates stress being applied in a highly stochastic manner. This is at variance with the prevailing views in landscape ecology that there is a baseline homogeneity based on the “magnification of the lens used.” This extracellular nature is highly site specific. This coupled with the dramatic variation in GC levels around roost areas shows AMCR likely traveling significant distances over and through locations with various levels of environmental stressors to arrive at their roost sites each evening. This results in a highly stress entrained aspect per site as a combination of those environmental stressors. Stress is mediated most effectively when there is consistency or linearity in its application, facilitating a rapid return to equilibrium. The extracellular nature of landcover examined showed a dramatic differentiation of these entrained aspects that over time, stress response is unable to adjust too, without having a pathological response. This results in the extension or lengthening of the negative feedback response, and thereby reduction in proximate fitness and immunological resistance. AMCR, more so than many other taxa, is a highly social and adaptable avian species (due to its higher level of cognition and neuroplastic nature (rapid flexibility and adaptation of response via its sophisticated central nervous system (CNS)). The AMCR populations in the roosts I observed thus favor urban locations. However, AMCR’s endocrine system adapts more slowly than their CNS (brain) to higher stress environments. Social cohesion thus outweighs homeostatic balance. In effect we would say that *they are too smart for their own good!*

The second finding deals with the magnitude of the environmental stressors and their variance at each of the sites and catchment sizes that I characterize as entrained stress, which is highly site specific and resists a 'one size fits all' characterization. There is clearly an interplay whereby some may be ameliorated while others enhanced by the presence of others and affects stress response. These relationships are deserving of further study.

Lastly the habitat quality stress index that I developed is the first use of its kind as a single constrained gravity model originating with Newton's law of universal gravitation to evaluate stress response over a spatial area. Follow-on work should involve further evaluating the efficacy or strength of the relationship in the HQSI to each environmental stressor.

These findings collectively will strengthen the understanding of the key aspects of habitat quality as ESs based on internal (physiological) stress response. That information can be used to enhance the knowledge of biodiversity as a key component of landscape quality and thus from a policy perspective suggests the need to preserve and enhance that valuable ecosystem service.

Finally, as a very specific follow-on study, combining the HQSI for each of the sites for all the environmental stressors and plotting them spatially in GIS would yield a landscape scale form of spatial biology. Spatial biology deals with modeling macromolecules of tissues and systems down to the atomic scale. Analogously, the HQSI when combined with all its constituent environmental stressors and GC stress levels in GIS, would yield a geographic spatial landscape tied to specific quantitative data that clear policy guidance of biodiversity significance could be derived. Similar to Ian McHarg's work (1969) in the synthesis of a variety of landcover elements for planning and preservation, the HQSI would add an empirical level of categorization for biodiversity not previously seen, based upon the addition of specific stress response level indices in situ. Lastly, generalizing the HQSI as a software program could provide practitioners

with an easy to grasp tool for the evaluation of stress across a variety of spatial landscape, stressors, and catchment areas.

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APPENDIX B: SITE LOCATIONS

Figure B1

Waterbury, CT Site 1A Department of Labor (DOL) and Site 1C Shopping Center

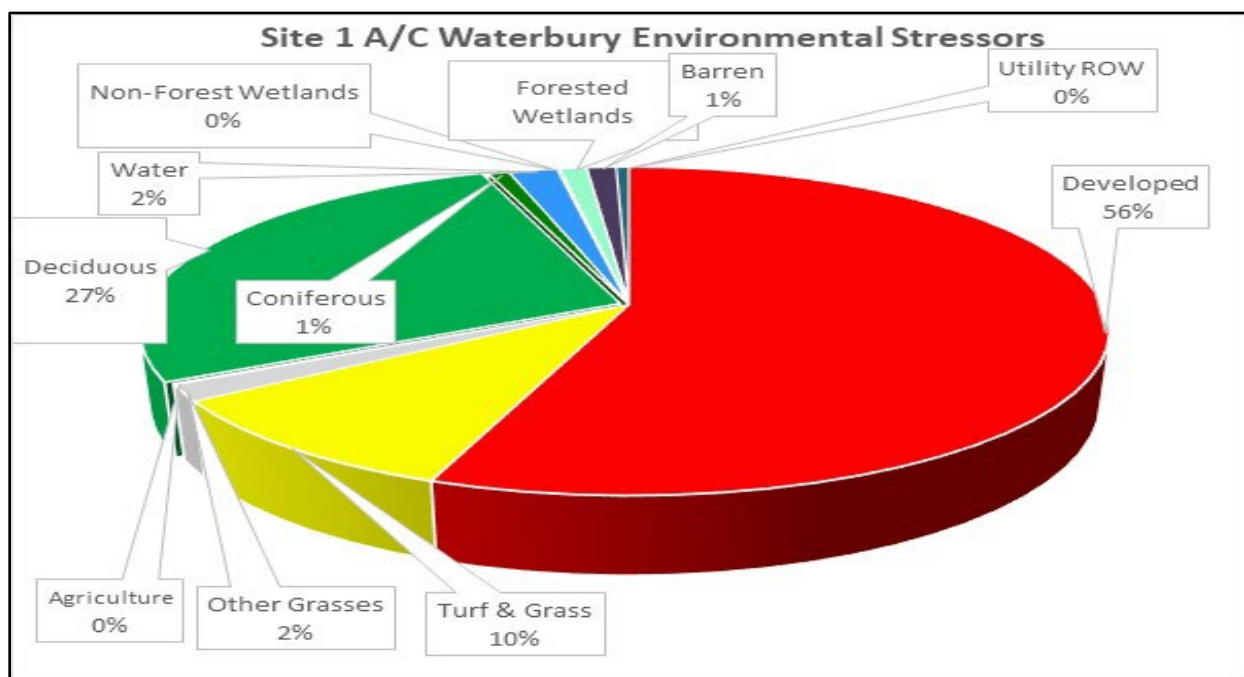
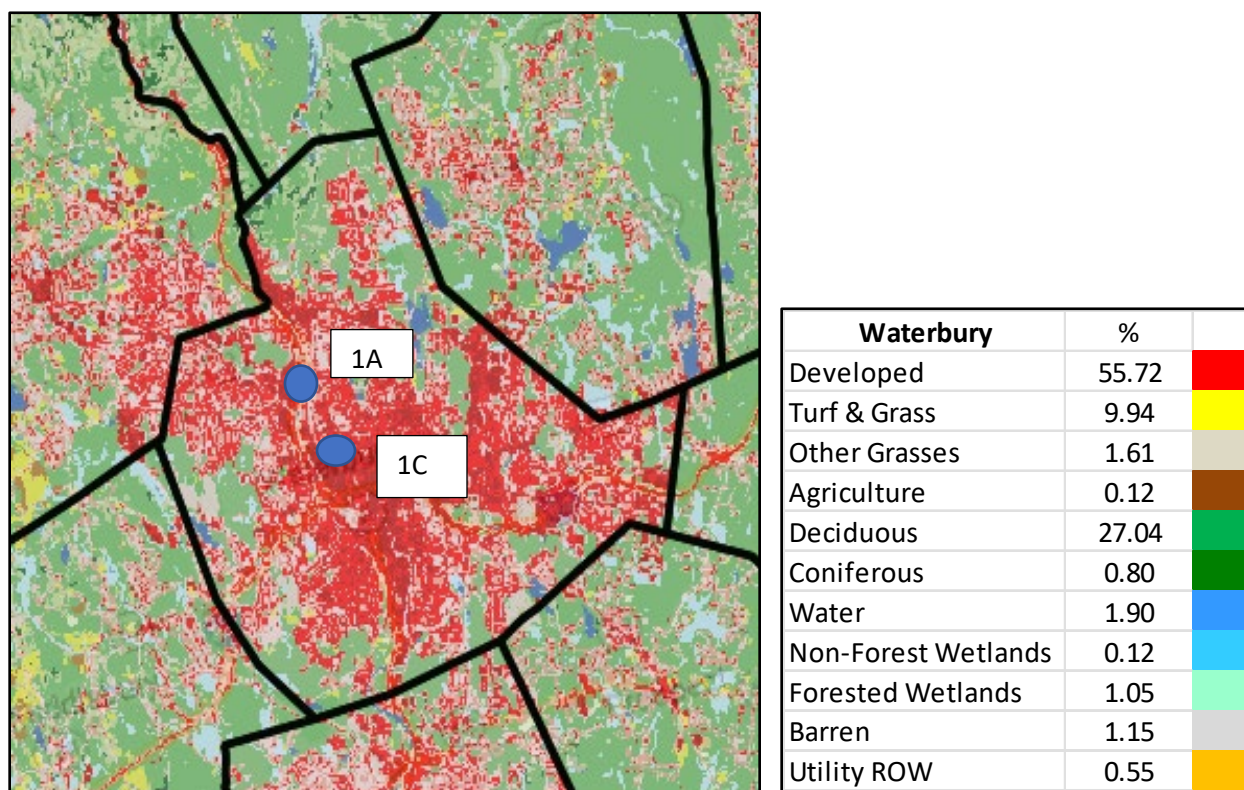


Figure B2

West Hartford, CT Site 2A Shopping Mall

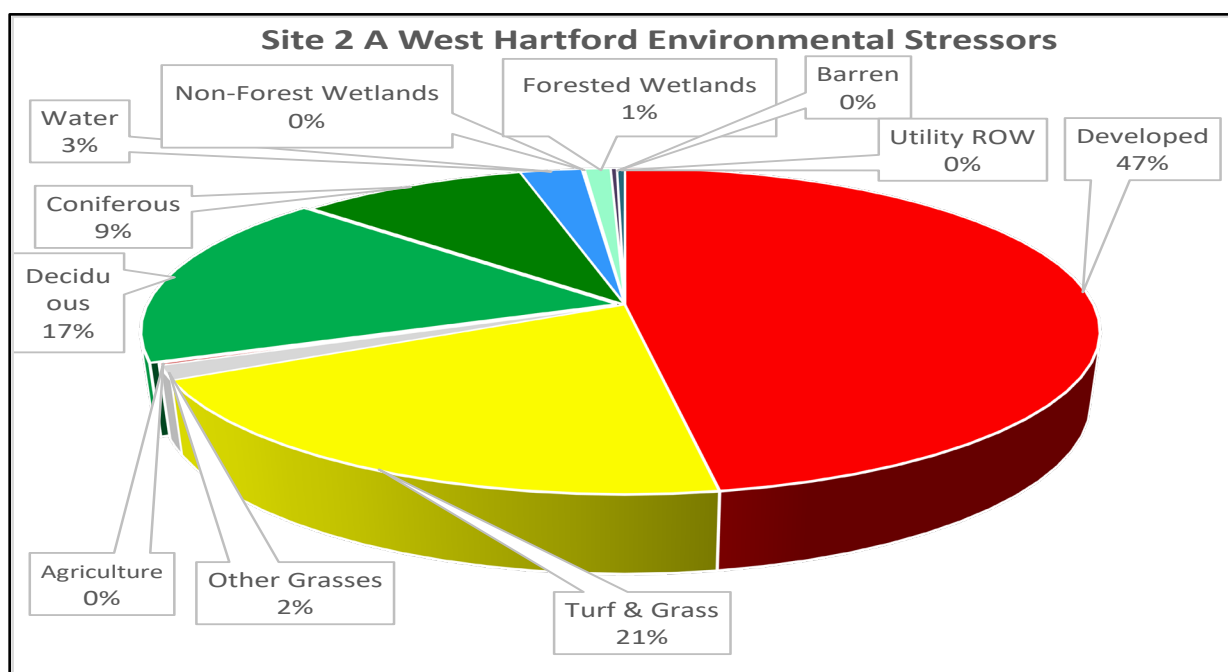
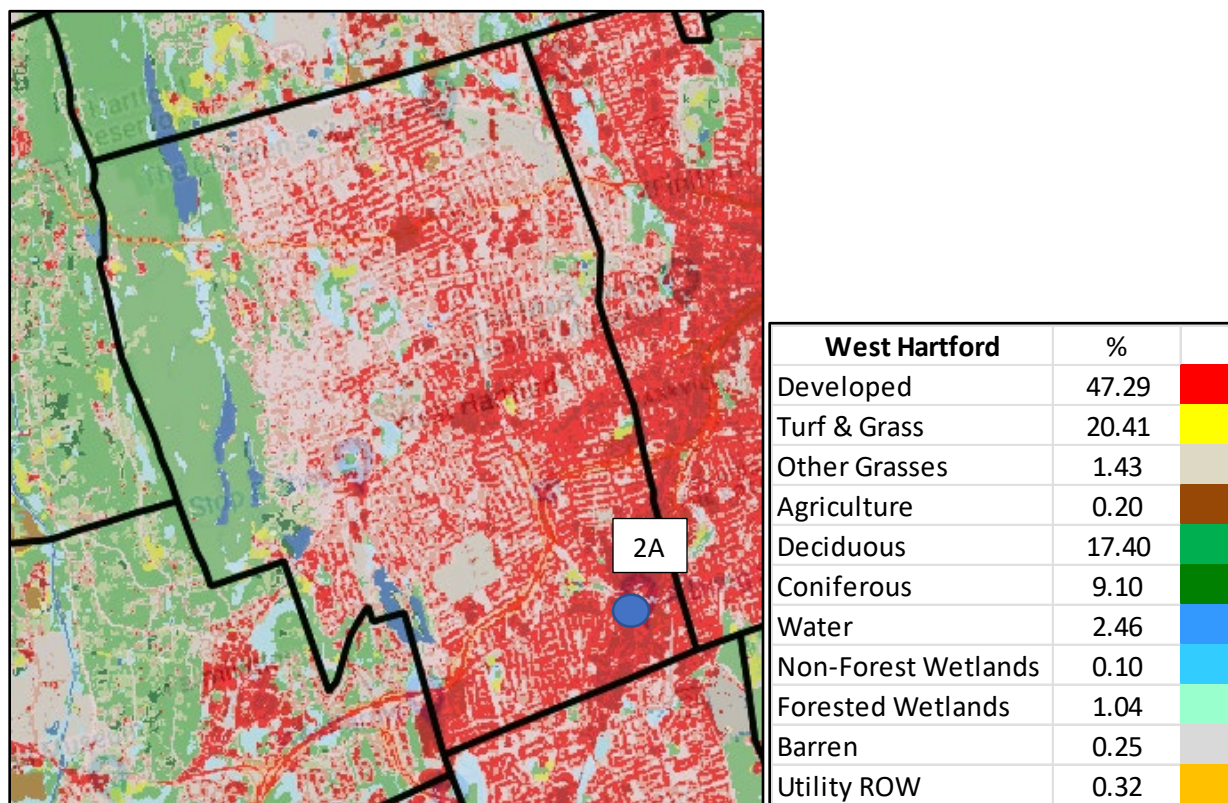


Figure B3

Hartford, CT Site 4A Hartford High School and Site 4B Trinity College

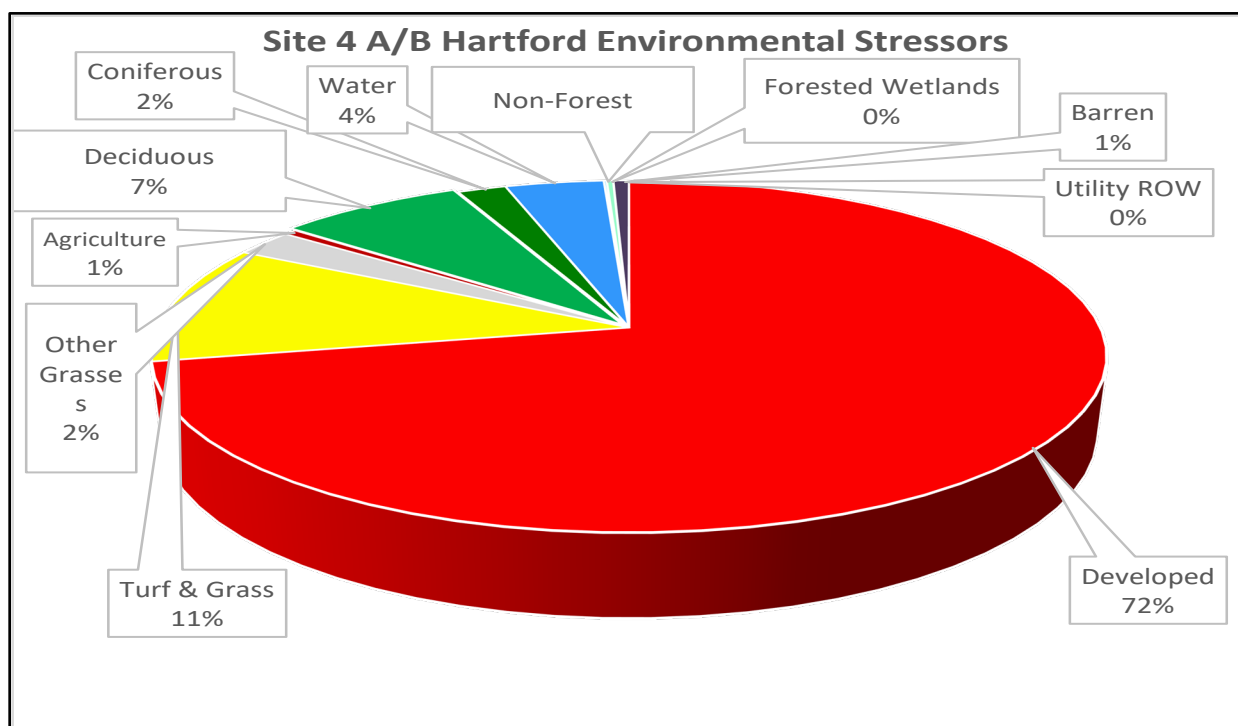
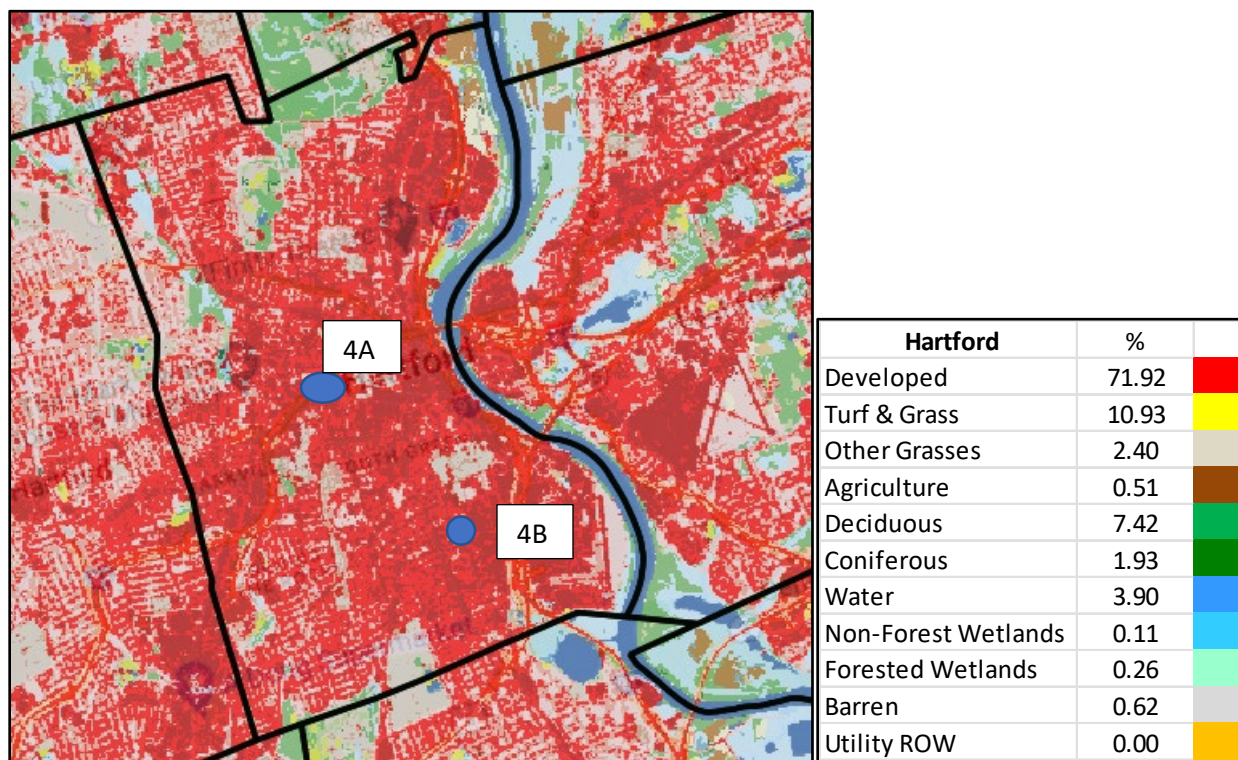


Figure B4

Norwich, CT Site 3B Three Rivers Community College and 3C Staples

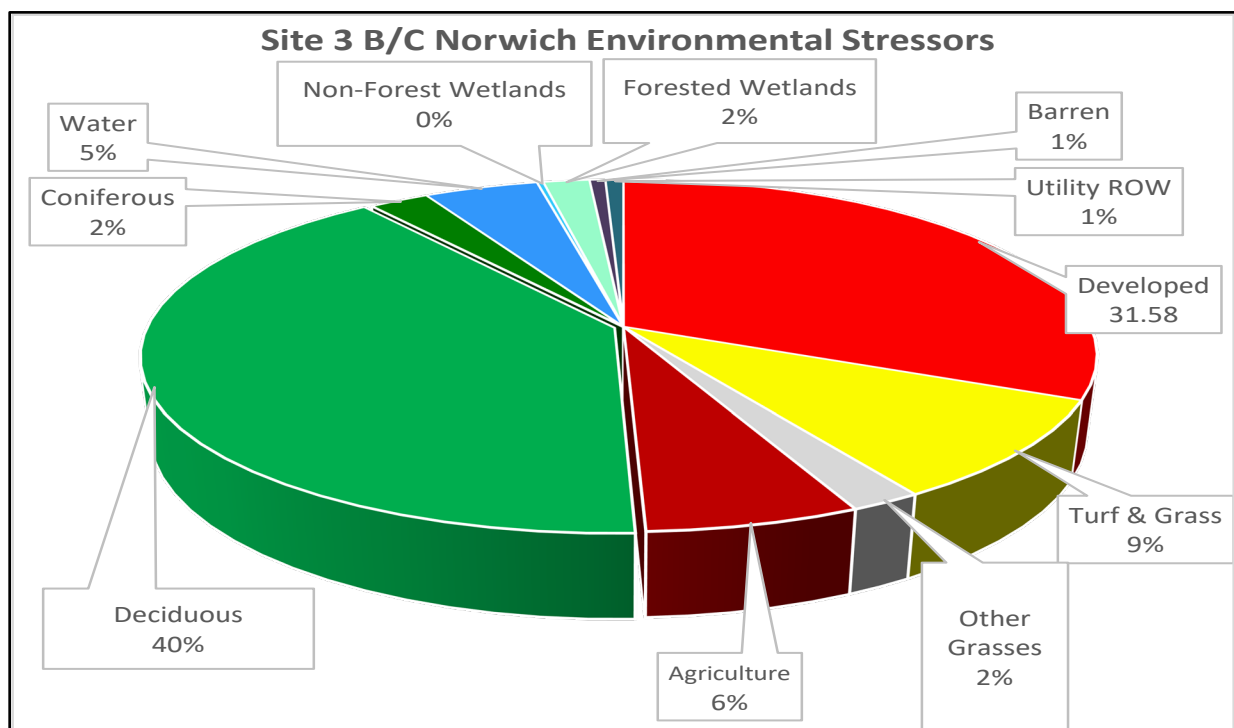
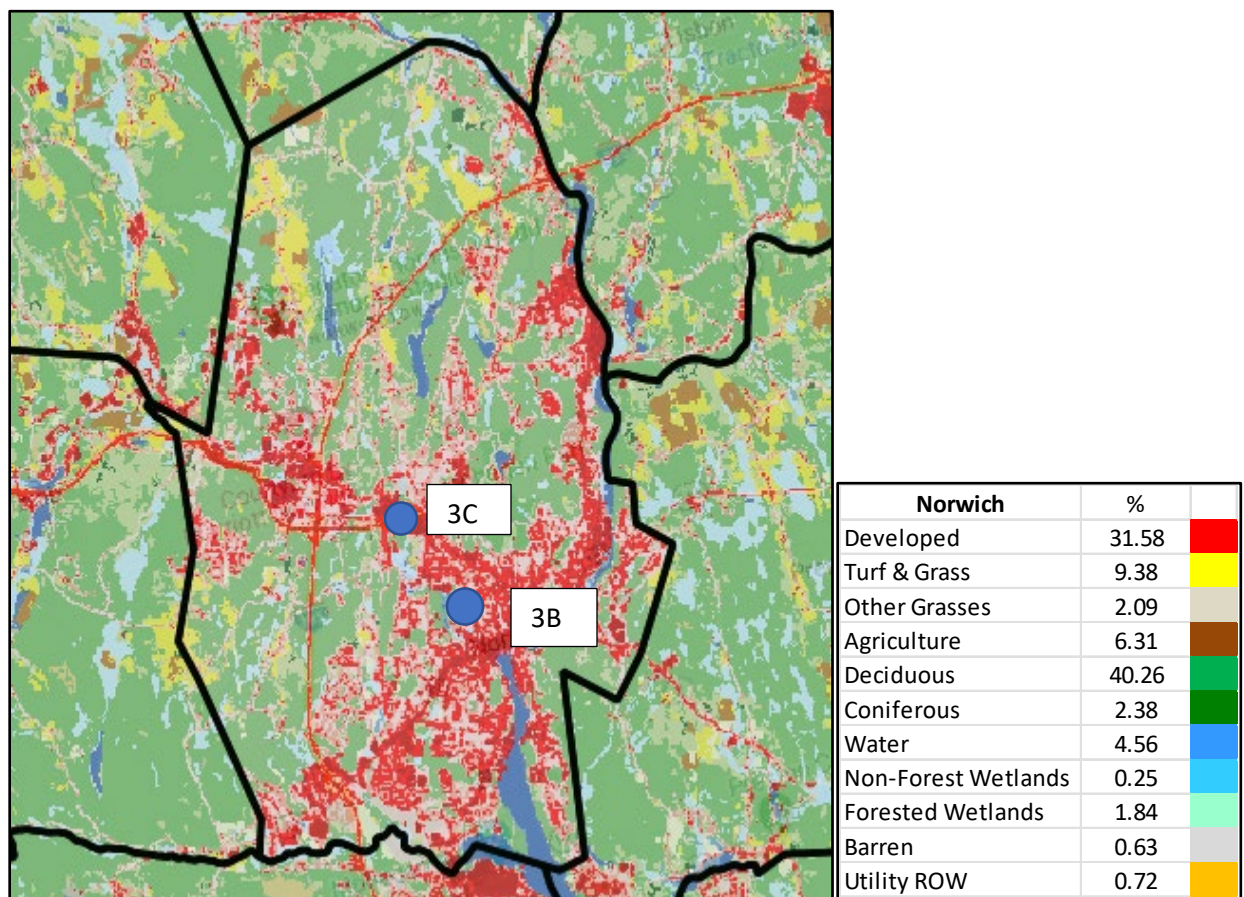


Figure B5

New Canaan, CT Site 5A New Canaan High School, Site 5B Lapham Road and Site 5C Old

Stamford Road

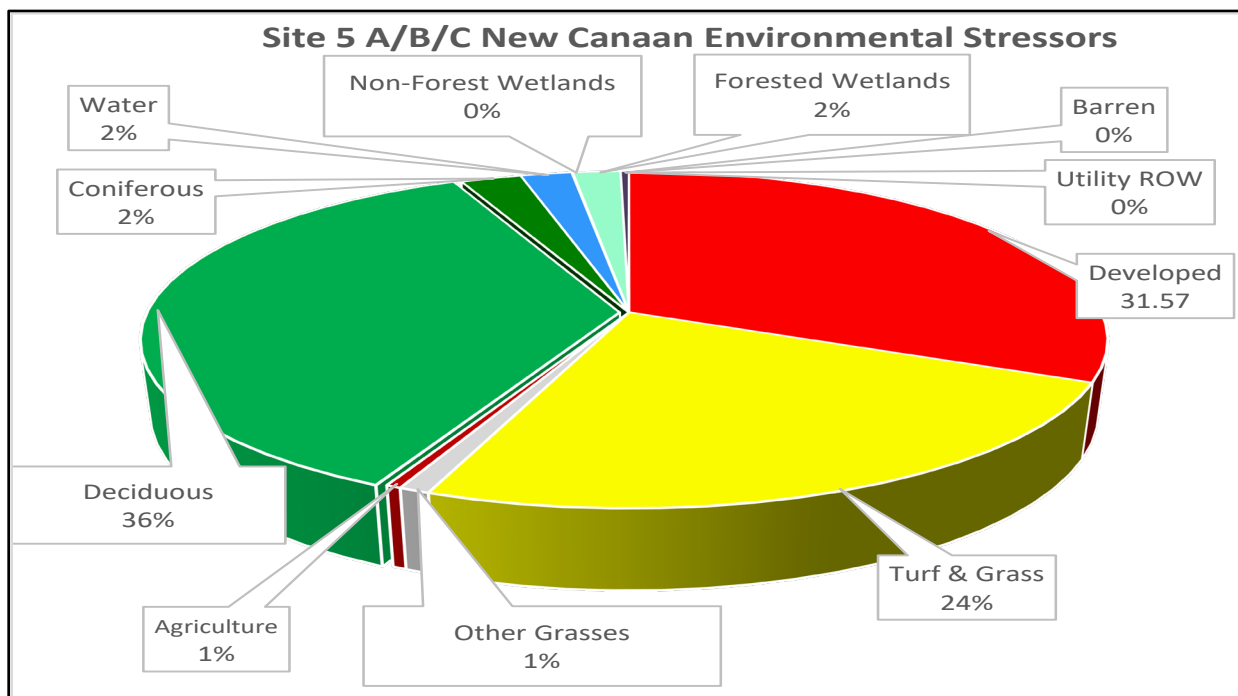
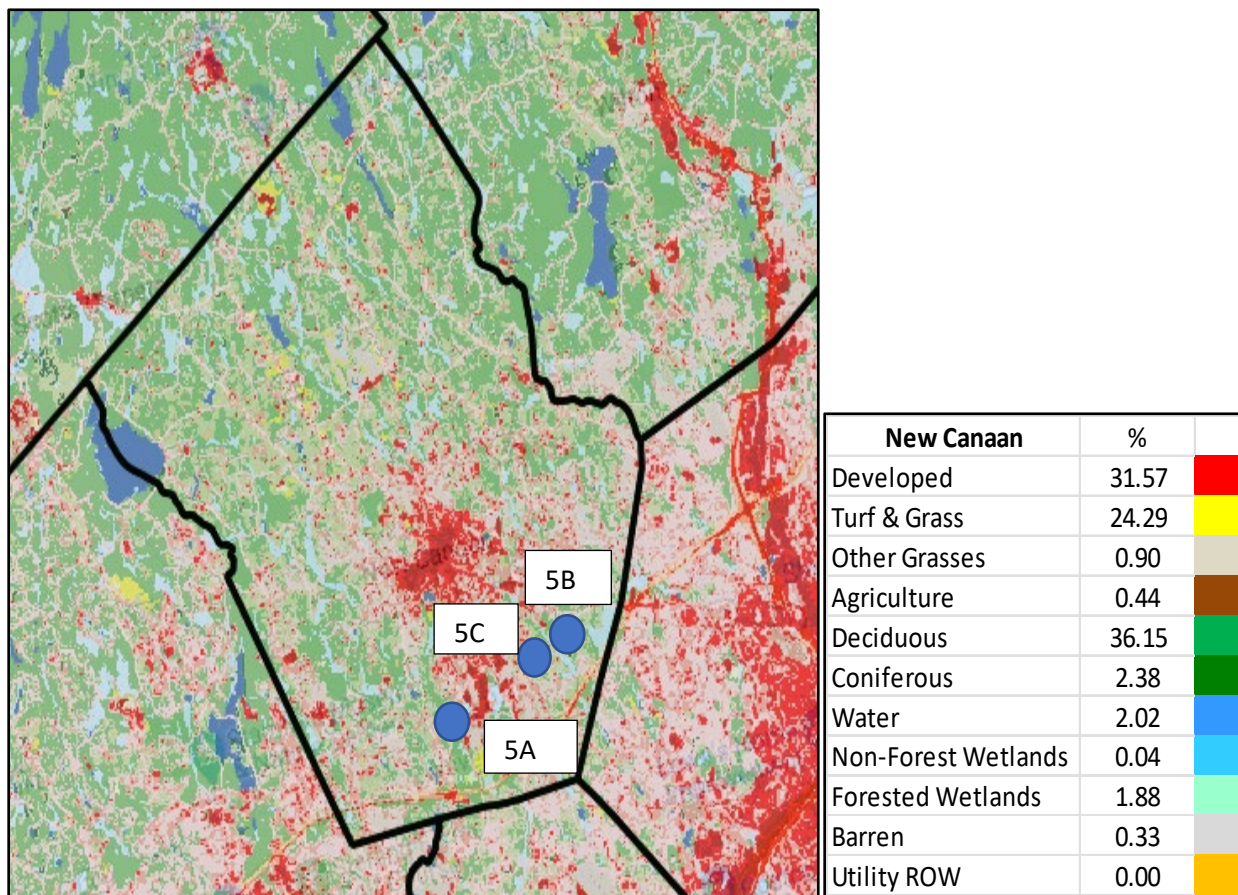
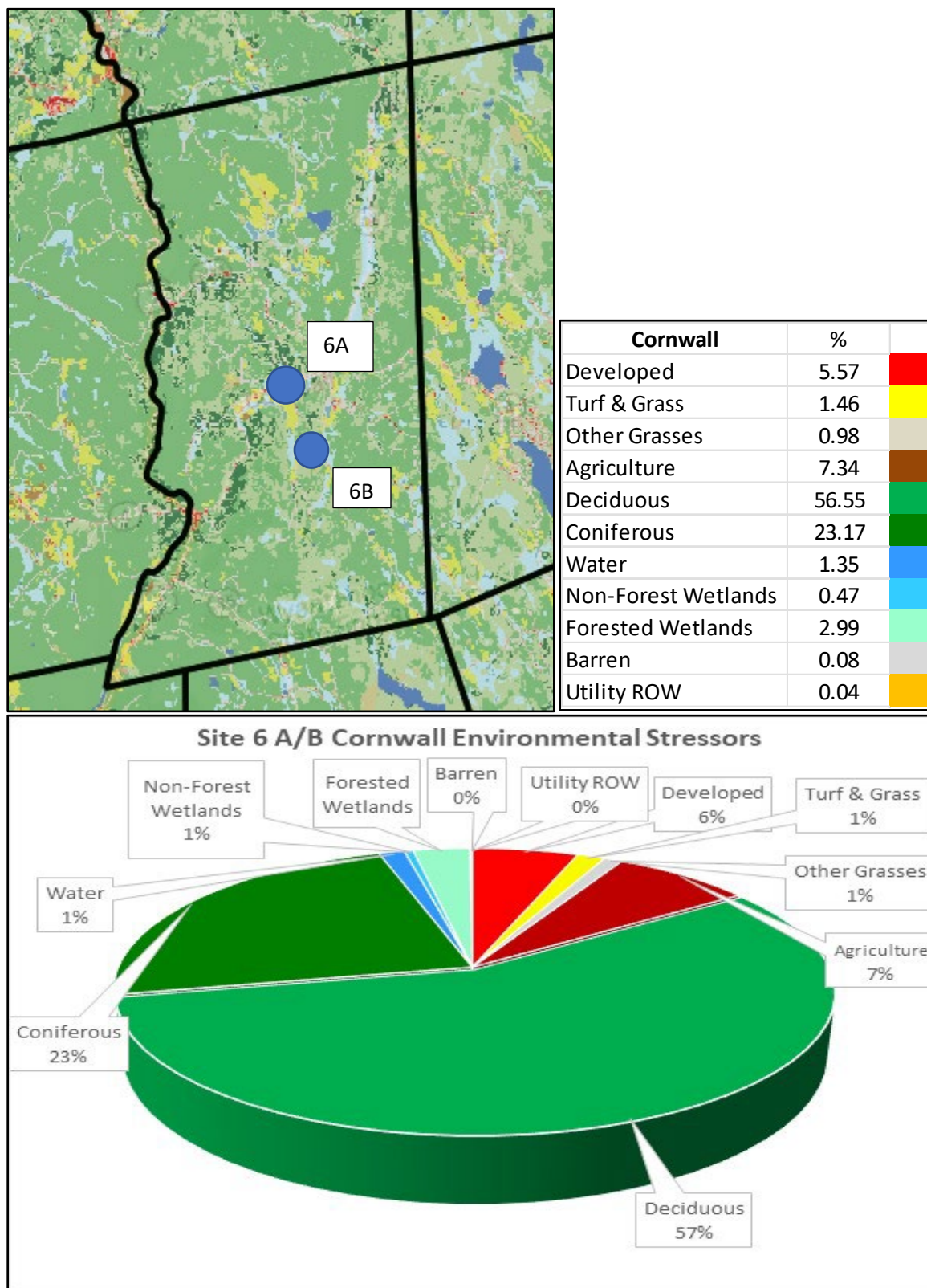


Figure B6

Cornwall, CT (Sharon) Site 6A and Site 6B



APPENDIX C: QGIS CATCHMENT LOCATIONS

Figure C1

Screen Shot in QGIS West Hartford, CT Site 2A Shopping Mall Buffer Establishment Technique

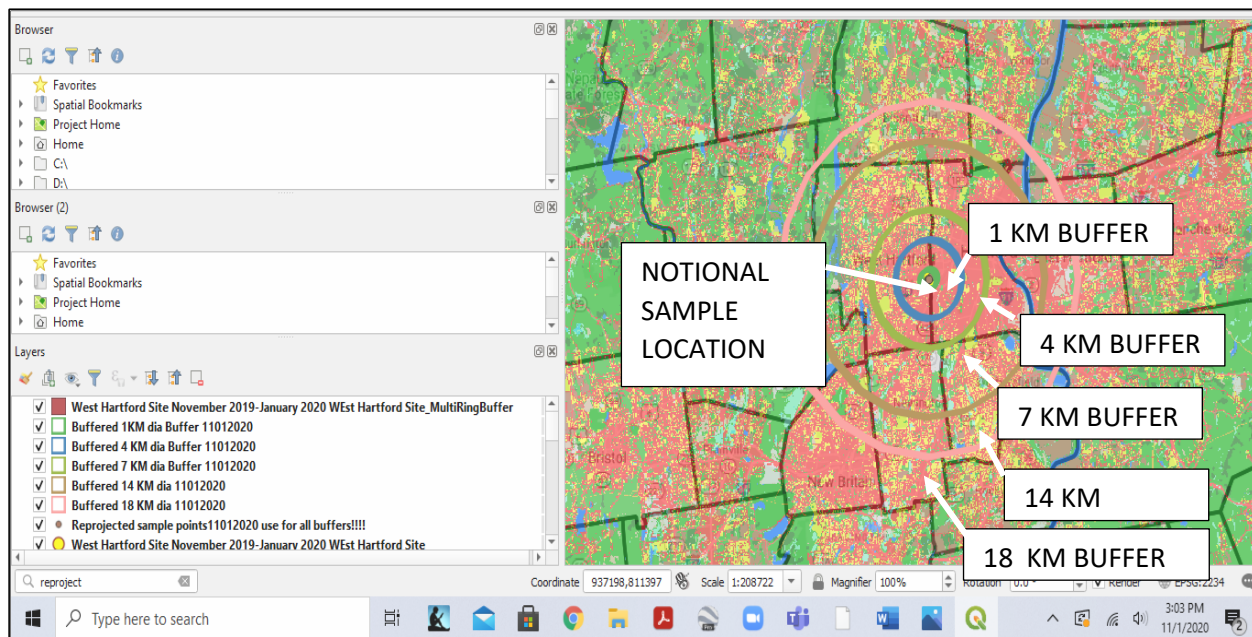
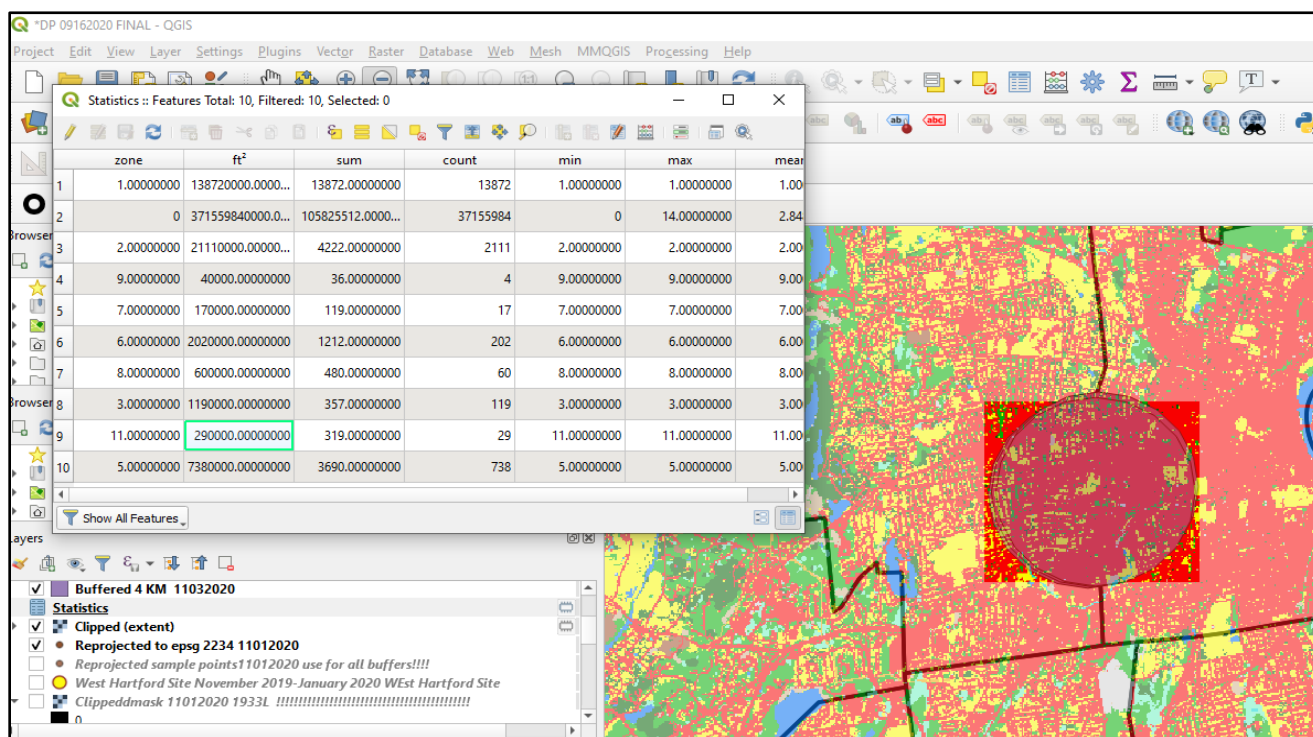


Figure C2

Zonal Statistics Function to Derive Area of Each Landcover (Environmental Stressor)



APPENDIX D: WATERFALL CHARTS

