BIOCOMPLEXITY XI

The Evolution of Cooperation

December 3-December 5 2010
Bloomington, Indiana

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main sessions
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Chris Adami (Keck Graduate Institute)

"Evolution of Cooperation from Reliable Communication"

The observed cooperation between genes, cells, tissues, and higher organisms represents a paradox for Darwinian evolution, because the individual success of cheating is rewarded before its long-term detrimental consequences are felt. The tension between cooperation and defection can be represented by a simple game (the "Prisoner's Dilemma"), which has been used to study the conflicts between decisions to cooperate or defect. Here, we encode these decisions within genes, and allow them to adapt to environments that differ in how well a player can predict how an opponent is going to play. We find that evolutionary paths end at strategies that cooperate if the environment is sufficiently predictable, while they end in defection in uncertain and inconsistent worlds because inconsistency favors defection over cooperation. This work shows that cooperation or defection, in populations of players that use the information from previous moves to plan future ones, can be influenced by changing environmental parameters that affect a player's capacity to reliable communicate to other players their intentions, encoded in the history of their plays.

C. Athena Aktipis (Arizona)

"The Evolution of Metastasis"

Resource limitation is a fundamental ecological pressure that limits survival and reproduction across all ecological systems, exerting evolutionary pressures that favor those who can escape from resourcelimitation. These same evolutionary forces are likely at work inside neoplasms, favoring cells that can gain access to limiting resources in spite of local scarcity. The evolution of cell motility is likely to be driven by these fundamental ecological forces. If so, somatic evolution should favor cells that are capable of dispersal, especiallywhen cells are consuming local resources at high rates. Here we investigate whether the disregulated metabolism characteristic of cancer cells may play a causal role in selection for cell motility, and thus to the tissue invasion and metastasis that define cancer. Using an agent-based computational model, we demonstrate that cells with higher metabolism rates evolve to have higher rates of movement and that 'neoplastic' cells with higher metabolism rates are able to persist in a population of 'normal' cells with low consumption rates, but only if increased metabolism is accompanied by increased motility. These findings suggest that higher rates of cellular metabolism in neoplastic cells may coevolve with higher motility. This has important implications for understanding the progression of cancer from less invasive to more invasive cell types.

John F. Allen (School of Biological and Chemical Sciences, Queen Mary, U London)

"Why cytoplasmic inheritance? Genes in chloroplasts and mitochondria"

Chloroplasts and mitochondria are energy-converting organelles in the cytoplasm of eukaryotic cells. Chloroplasts in plant cells perform photosynthesis; the capture and conversion of the energy of sunlight. Mitochondria in both plant and animal cells perform respiration; the release of this stored energy when work is done. Chloroplasts and mitochondria also contain small, specialised genetic systems to make a few of their own proteins. Both the genetic and the energy-converting machineries of chloroplasts and mitochondria are descended, with little modification, from those of the free-living bacteria that these organelles once were. Today, almost all genes for proteins of chloroplasts and mitochondria are found on chromosomes in the nuclei of eukaryotic cells. There they code for protein precursors that are made in the cytosol for import into these two bioenergetic organelles, there to be trimmed down into their mature, functional forms. So why are any characters at all still inherited through cytoplasm? In other words, why do just a few genes remain steadfastly within chloroplasts and mitochondria as vestiges of ancestral, bacterial DNA? Why did this tiny genetic minority fail to join the emigrant, nuclear majority?

Tommy Angelini (University of Florida)

"Biofilm multicellularity and molecular concentration gradients"

Bacteria move cooperatively for many reasons: to invade host tissues, to seek out nutrients, or to erect structures for spore spreading. Swarming is a type of cooperative surface-associated motility, in which each cell is self-propelled by its own flagella. Twitching and social gliding also depend on a motor driven cellular appendage; type IV pili are extended toward a surface, adhered, then retracted, pulling the individual cell forward toward the adhesion. Intriguingly, many types of collective motility do not require the use of known motor-driven appendages; these include gliding, adventurous gliding, sliding, and spreading. The physical mechanisms underlying these kinds of cooperative motility are not yet clear, yet a clue comes from the physical chemistry of their excretions; all of these motor-independent types of motility involve amphiphilic molecules. In this talk we show that spatial concentration gradients of these amphiphilic molecules give rise to surface tension gradients that drive biofilm spreading. Moreover, we show that polymer production also drives bacterial biofilm spreading, and is triggered by a gradient of nutrient depletion within the colony. Thus, these two types of multicellular spreading depend not only the types of molecules excreted by bacteria, but gradients in their concentration.

Daniel ben-Avraham (Dept of Physics, Clarkson University)

"Selection pressure in the Kleinberg navigation model: A cautionary tale"

The burgeoning study of complex networks offers several examples of selection pressures that give rise to broad (universal) traits. In this talk, I will survey briefly some of the main ideas. I will then tell of a particular model for navigating complex nets, due to Kleinberg, where the pressure for optimal navigation seems to select a specific kind of connectivity. Yet, upon deeper consideration of other relevant physics (possible losses in transmission), the pressure for optimization looses its selective power.

Neil W. Blackstone (Northern Illinois U)

"Programmed death in mitochondria"

A major innovation of eukaryotes was moving chemiosmotic membranes internally during the endosymbiosis of mitochondria. Large and complex eukaryotic cells thus are not necessarily limited in their capacity for energy conversion. As is perhaps typical in the history of life, however, the solution to one problem resulted in the creation of a host of new ones. In this case, a clever engineering solution to surface-to-volume constraints—small, energy-converting cells within a larger cell—became a levels-of-selection nightmare. How could the emerging higher level unit (the eukaryotic cell) keep selfish lower-level units (the mitochondria) from plundering the group's supply of food when it was these very lower-level units that carried out the processes of energy conversion and allocation? Most answers to this question focus on information, i.e., moving the bulk of the mitochondrial genome to the nucleus. Nevertheless, mitochondria retain a small genome and thus also retain the capacity for heritable variation. Often ignored in this context are the mechanisms by which mitochondrial biogenesis is actually regulated. Cell-level metabolic parameters (e.g., NADH/NAD+, ATP/ADP, reactive oxygen species or ROS) are detected by transcriptional coactivators (e.g., PGC-1α) and biogenesis is enhanced or suppressed. Arguably, such regulation functions as a "chromosome rule": mitochondria are replicated together or not at all. Since these cell-level metabolic parameters emerge at the level of the group, a single variant mitochondrion cannot subvert this signaling. Consider an ancient eukaryote in which each mitochondrion still retains a full genetic complement, but biogenesis is regulated by a nuclear equivalent of PGC-1α. A loss-of-function mutation in, for example, the adenine nucleotide translocator gene would immediately create a selfish mitochondrion that no longer exported ATP to the cell. The additional ATP might allow the selfish mitochondrion to replicate at a higher rate. This advantage is unlikely to be realized, however, because the variant mitochondrion cannot influence the metabolic signature of the cell sufficiently for the equivalent of PGC-1α to initiate mitochondrial biogenesis. Meanwhile, the variant mitochondrion would likely convert all of its ADP to ATP. Membrane potential would become maximal, electron carriers would become highly reduced, and ROS formation would be maximal. This variant mitochondrion would likely incinerate in its own ROS and then be consumed by autophagy. Metabolic regulation at the cell level can thus effect a kind of "programmed death" for selfish variant mitochondria. Such metabolic regulation was the crucial step in the prokaryote to eukaryote transition.

José Costa (Yale University Medical School)

"The Cancer Paradox: Innovating through Atavism"

After providing a concise primer on cancer, I will focus on the conceptual models that inform medical cancer research. Interpreting clinical and experimental data in light of ecological and evolutionary dynamics provides an effective way to generate hypotheses that both explain and enable prediction of the natural history of tumors. Metapopulation dynamics (Levins) is well suited to describe the variational changes in tissues undergoing neoplastic transformation and the effects of intermediate disturbance in communities (Tillman) provide a "top down" mechanism to understand how the environment (internal and external) causes tumors. Cooperation is known to play a role in tumor formation/progression and its impact on the competition implicit in the microevolutionary process of tumor formation can be explored with agent-based models based on minor modifications of the Lotka-Volterra equation.

The inescapable conclusion of the current understanding of the mechanism of tumor progression is that evolvability is the fundamental property of both preneoplastic and tumor cells with metastatic potential. The capacity of somatic cells to evolve is an atavism, as this property is lost in the transition of uni- to multicellularity. Whereas unicellular organisms evolve to find new solutions to environmental challenges, somatic cells in multicellular organisms die when the environment imposes a stress beyond their physiologic capacity to adapt. For most tumors of the adult, cancer cells result from the rewiring of control circuits in response to a hostile environment. It is of interest that the coordinated expression of "cancer genes" (tumor supressors and oncogenes) coincides with the appearance of multicellularity in the filogenetic scale. Mutation of these genes disrupts the cooperation imposed by multicellularity and underlies many of the hallmarks of cancer (Hanahan and Weinberg) including novel ways to cooperate, but they fail to explain evolvability. Recent data (Spakowski, et al.) suggest that the rewiring required to transform normal cells into cancer may be accomplished by using repetitive sequences in our genomes, which are normally repressed, thus enlisting "genes in conflict" (Burt and Trivers) to cooperate as capacitators of the evolution of somatic cells.

Iain Couzin (Princeton)

"Information transfer and the evolution of collective behavior"

Cassandra Extavour (Harvard)

"Evolution of Germ Lines"

Ariel Fernández (Rice)

"Nonadaptive origin of Interactome complexity"

A comparison of orthologous proteins shows that while retaining the fold, proteins become structurally more vulnerable in species with low population. This implies that complexation is promoted in inverse relation with population size. The mild structural degradation is the outcome of higher exposure to the vagaries of random genetic drift which prevails in species with small population size, suggesting a nonadaptive origin of interactome complexity. Is there a nonadaptive route opening up a selectivity niche to foster protein cooperativity/allostery?

Robert Gatenby (Moffitt Cancer Center)

"The Darwinian Dynamics of Carcinogenesis"

Invasive cancer emerges following a complex, multistep process often described as "somatic evolution." Models of carcinogenesis are typically based on the Darwinian principle that evolution requires genetic and/or epigenetic changes that generate new phenotypes. However, the models do not typically address why these specific phenotypic/genotypic changes are necessary for carcinogenesis or why they occur in the observed sequence. I propose carcinogenesis is a sequence of successful adaptations to six distinct microenvironmental proliferation barriers that arise in the adaptive landscapes generated by normal and premalignant populations growing on epithelial surfaces. The genotypic and phenotypic heterogeneity of cancer populations is explained by an "ecological equivalence" in which multiple strategies can successfully adapt to the same barrier. This model provides a theoretical framework in which the diverse cancer geno-/phenotypes to be understood according to their roles in overcoming specific microenvironmental growth constraints.

Sergey Gavrilets (NIMBioS, U of Tennessee)

"Rapid Transition towards the Division of Labor via Evolution of Developmental Plasticity"

Biological organisms are highly complex and are comprised of many different parts that function to ensure the survival and reproduction of the whole. How and why the complexity has increased in the course of evolution is a question of great scientific and philosophical significance. Biologists have identified a number of major transitions in the evolution of complexity including the origin of chromosomes, eukaryotes, sex, multicellular organisms, and social groups in insects. A crucial step in many of these transitions is the division of labor between components of the emerging higher-level evolutionary unit. How the division of labor was achieved in the face of selfishness of lower-level units is controversial. Here I study the emergence of differentiated cell colonies in which one part of the colony's cells (germ) specializes in reproduction and the other part of the colony's cells (soma) specializes in survival. Using a mathematical model I show that complete germ-soma differentiation can be achieved relatively easily and fast (with a million generations) via the evolution of developmental plasticity. My approach is expandable in a number of directions including the emergence of multiple cell types, complex organs, or casts of eusocial insects.

Owen Gilbert (Rice U)

"Why do organisms limit social interactions to kin?"

High genetic relatedness among interactants was a prerequisite for natural selection to build adaptive complexity of new levels of biological organization. Social evolutionary theory seeks to explain the role of historical contingencies in the evolution of kin population structure, but it has not previously sought to explain the existence of adaptive behaviors that limit social interactions to kin. Using inclusive fitness and game theory, I show why organisms limit social interactions to kin. I assume a situation where organisms have evolved to discriminate in their behavior in a nepotistic manner. I then show that blocking interactions with non-kin is an evolutionary stable strategy for avoiding conflict. I furthermore show that this behavior can exert a stable selective pressure for genetic polymorphism of its own cue locus. The result of this evolutionary process is the evolution of kin-limited interactions, which reduces the expression of conflict, increases mean fitness, and increases of relatedness among interactants. The increased relatedness may help favor the long-term stability of complex cooperative behavior.

Jeff Gore (Dept of Physis, MIT)

"Cooperation and cheating in microbes "

Understanding the cooperative and competitive dynamics within and between species is a central challenge in evolutionary biology. Microbial model systems represent a unique opportunity to experimentally test fundamental theories regarding the evolution of cooperative behaviors. In this talk I will describe our experiments probing cooperation in microbes. Model systems include the cooperative growth of yeast in sucrose and the cooperative inactivation of antibiotics by bacteria. I will also discuss recent modeling efforts directed towards understanding the evolution of host-symbiont mutualisms, where we find that the more slowly evolving partner can often claim the majority of benefits.

César Hidalgo (MIT)

"The Emergence of Economic Diversity"

Paulien Hogeweg (Utrecht)

“Evolution of molecular cooperation and functional complexity”

We study multilevel processes in the evolution of cooperation. Rather then predefining cooperative interactions and study their robustness and evolvability we study the evolution of emergent cooperation in multilevel systems. We show that various forms of direct and indirect cooperationreadily evolve. This is demonstrated in models in which, given structured entities, interactions evolve (1) In an RNA world, where we predefine a secondary structure of RNA which has replicase activity, we show that an robust intricate network of cooperative and parasitic interactions evolves (2) In a universe of structured resources we show how strong direct competition leads to indirect cooperation (cross-feeding) (3) In a universe of ``tasks'' we show that high mutation rates lead to cooperative problem solving.

Thomas Julou (ENS, Paris)

"Variability of public goods in a clonal population"

Quorum sensing (involving signaling molecules) and phenotypic variability (leading to differentiation) have led to revise the notion of unicellularity in microorganism populations. Genetic innovations drive the evolution of the colony over long time scales. They are selected by the environmental pressures and must also be compatible with the existing interactions between individuals. In this context, understanding the maintenance of cooperative traits remains one of the open challenge in evolutionary biology. We address this question experimentally by monitoring public goods dynamics in microorganism populations.

In Pseudomonas aeruginosa, low iron availability in the environment triggers the synthesis of large amounts of pyoverdin, a high-affinity iron chelator recaptured by bacteria once bound to iron. Though the synthesis of these molecules involves many enzymes and imputes a metabolic cost to the producer, they are secreted to the environment and can be recaptured by other individuals. Taking advantage of the intrinsic fluorescence of pyoverdin and using mutants of pyoverdin synthesis and uptake, we study at the single cell level the dynamics of this public goods in a clonal population. Using lineage and spatial correlation analysis, we can distinguish the relative contributions of phenotypic inheritance and neighbours interaction.

Isaac Klapper (Montana State)

"Discrete Speciation in a Continuous Environment"

Though the idea of "species" seems evident, there is as yet not a clear consensus on how to define and distinguish species in microbial systems. On the one hand, microbes reproduce asexually in most cases (and so lack the cohesion of sexual reproduction); on the other hand, in many instances microbes are able to exchange genetic material even with other microbes that are apparently not closely related (which would seem to have dispersive consequences). Yet, recent advances in genetic analyses suggest that discrete speciation in microbial communities occurs. A mathematical model attempting to address this issue will be presented.

Herbert Levine (UCSD)

"Indirect selection during Darwinian Evolution"

Motivated by experiments on laboratory-scale evolution in both microorganisms and biomo-ecules, we introduce and study a class of multi-locus evolution models. For these models, the population advances via being dragged forward by its most fit members and can be quantitatively studied using ideas from the theory of non-equilibrium spatially-extended processes. A key finding is the anomalously large dependence on population size and the related anomalously large usefulness of genetic recombination. Using this approach, insight can be obtained regarding the indirect selection for mechanisms which speed up adaptation, including becoming mutator-like and going into a state competent for genetic exchange.

Michael Lynch (Indiana University, Bloomington)

"Somatic Mutation and The Cost of Multicellularity"

Carlo Maley (UCSF)

"Cooperation in the evolution and ecology of tumors"

Cancer is the legacy of the evolution of multicellularity in animals. It represents a breakdown in the cooperation between somatic cells. Selection due to cancer mortality has resulted in the evolution of a variety of mechanisms (that are not yet fully understood) for enforcing cooperation among somatic cells. At the cellular level, cancers evolve by a process of natural selection among somatic cells. This drives both progression to malignancy and resistance to therapy. Recent attention has turned to studying the ecology of interactions of cancer cells with each other as well as other cells in their microenvironment. There are clear examples of cooperation, in which one cell type provides a benefit to others. There are even a few documented cases of mutualisms where the benefits are reciprocated. There are theoretical reasons to believe that targeting these cooperative mechanisms for therapeutic effect may delay the evolution of resistance compared to traditional cancer therapies. I will review the evolutionary theory of cancer as well as the nascent field of cellular cooperation in cancer.

William Martin (Düsseldorf)

“The Origin of Mitochondria and Eukaryotes: Cooperation Can Beget Complexity”

Natasha Mehdiabadi (Smithsonian Institute)

"Cooperation and Conflict in Social Systems: From Cellular Slime Molds to Fungus-growing Ants"

Cooperation is central to many of the major transitions in evolution, from the emergence of chromosomes via the assembly of independent genes to the origin of multicellular organisms from single cells. Understanding cooperation is therefore fundamental to understanding the history of life. I study a variety of social systems to understand why and how cooperation is maintained despite the potential for conflict. In today's talk, I will present work on both social microbes and social insects addressing these fundamental questions in evolutionary biology.

Roeland Merks (Amsterdam)

“Emergence of microbial cooperation in the human gut”

Complex sugars from plant cell walls are important sources of energy in our food, but we cannot digest them by ourselves. Fortunately, each of us hosts a diverse population of microorganisms that convert the indigestible complex sugars into short-chain fatty acids that the intestinal wall absorbs. I will introduce our recent multiscale models of the gut microbiota, that will help us explain the emergence, maintenance, and pathological collapse of microbial diversity in the intestine.

Richard Michod (Arizona)

"Cooperation, conflict, and sex in the evolution of individuality"

Risto Miikkulainen (UT Austin)

"Coevolution of cooperative behaviors in simulated predator and prey teams"

In a series of computer simulations, artificial neural networks were evolved (through genetic algorithms) to control predators and prey in a simulation of a hyena clan and its prey. The type of reward (individual vs. team), communication (stigmergic vs. direct), and rank distribution (equal vs. varied) was found to affect the types of behaviors that emerged. Further, under certain conditions, the predator and prey populations engaged in an evolutionary arms race, constracting gradually more complex behaviors in response to those of their opponents. In this manner, computer simulations can be used to demonstrate how cooperative behaviors can emerge through evolution in interacting groups of agents.

Aurora Nedelcu (U of New Brunswick)

"From single-celled to multi-celled individuals: Co-opting environmentally-induced responses for group advantage"

David Nelson (Harvard)

"Competition & Cooperation at Frontiers"

Elinor Ostrom (Indiana University, Bloomington)

"The Evolution of Rules that Enhance Cooperation in Diverse Settings"

Rules can be analyzed as instructions for actors in positions regarding the actions they may or may not take and the benefits and costs of these actions. One can conceptualize them as a set of instructions that affect the structure of action situations similar to how genotypes affect phenotypes. I will present an initial method for analyzing rule strings over time and how they may evolve

Sidney Redner (Boston University)

"Can Consensus Ever Be Achieved by Social Interactions Alone?"

Iñaki Ruiz-Trillo (U Barcelona)

"'Multicellular' genes in unicellular lineages: A comparative genomics perspective to the origin of metazoan multicellularity"

Recent genome data has recently identified a rich genetic repertoire of "multicellular" genes in unicellular relatives of metazoans. For example, the amoeba Capsaspora owczarzaki, a close relative to metazoans, encodes the two major metazoan cell-cell and cell-matrix machineries, i.e., cadherins and integrins, both of them absent in plants and fungi and previously believed to metazoan-specific. Moreover, the genome of Capsaspora encodes transcription factors that are key to animal development, such as T-box genes or Runx, as well as around a hundred protein tyrosine kinases. These findings challenge previous views in which the origin of Metazoa was thought to be a consequence of several evolutionary innovations. Not all, but most of the machineries for cell-adhesion, cell-communication and cell differentiation, were already present in the unicellular ancestors of Metazoa. This opens new questions, such as the role of those "multicellular" genes in an unicellular context, how were they co-adapted into the new (multicellular) functions, or the the importance of having a more complex network, and the possibility that new gene combinations triggered the emergence of multicellularity.

Eugene Shakhnovich (Dept of Chemistry and Chemical Biology, Harvard)

"Evolution on physics-based fitness landscapes: how protein folding determines selection and vice versa.

In this talk I will present a new bottom up approach to derive and analyze evolution of populations of viruses and bacteria on physics-based fitness landscape which originates from fundamental biophysical requirement that in order to function structured proteins must be in their folded states. We consider and contrast ''strictly neutral'' landscapes which posit that proteins having negative free energy of folding are fully folded and the ones which have positive free energy of folding are fully unfolded and near-neutral fitness landscape which represents a continuous function of folding free energy where we assume that organismal fitness is governed by an AND function over the "foldedness" of each essential protein, which, according to statistical-mechanics of protein folding, is given by a "Fermi-function" of folding free energy (ΔG). Most mutations in our model affect fitness by altering ΔG, while, based on simple estimates, $\approx$10% are unconditionally lethal. In contrast to the "survival of the flattest" principle, which is apparent on strictly neutral fitness landscape, we find that, in mutation/selection/drift steady-state, high mutation rates (m) lead to less stable proteins and a more dispersed Distribution of Fitness Effects of mutations, i.e. less mutational robustness. Small population size (N) also decreases stability and robustness. Compensatory mutations are more common and potent in small populations with high mutation rates. In the second (experimental) part of the talk I will present results of our recent experiments, which systematically probe fitness landscape by making controllable biophysical changes in proteins (by chromosomal introduction of stability-changing mutations) and their abundances (by genetic manipulation in the upstream regions) and evaluating their fitness consequences. Our experiments confirm basic features of physics-based fitness landscape and add important new insights on how to make them more comprehensive and accurate.

Kim Sneppen (Niels Bohr Institute)

"Why Do Phage Play Dice?"

When phage lambda infect a bacterium it goes either into the lysis, where the phage particles rapidly replicate and kill the cell, or into a dormant lysogenic state where the phage replicates along with the cell. Experimental observations by P. Kourilsky show that single phage infection deterministically chooses lysis whereas double infection result in a stochastic choice. We argue that the phage are playing a "game" of minimizing the chance of extinction and that the shift from determinism to stochasticity is due to a shift from a single-player to a multi-player game. Crucial to the argument is the clonal identity of the phage.

M. Avlund, IB. Dodd, S. Semsey, K. Sneppen, and S. Krishna, JOURNAL OF VIROLOGY, Nov. 2009, p. 11416–11420

Corina Tarnita (Harvard)

"The evolution of eusociality"

Eusociality, in which some individuals reduce their lifetime reproductive potential to raise the offspring of others, underlies the most advanced forms of social organization and the ecologically dominant role of social insects. For the past four decades, kin selection theory, based on the concept of inclusive fitness, has been the major theoretical attempt to explain the evolution of eusociality. In this talk I propose that standard natural selection theory in the context of precise models of population structure represents a simpler and superior approach, allows the evaluation of multiple competing hypotheses, and provides an exact framework for interpreting empirical observations.

Gregory Velicer (Indiana University, Bloomington)

"Social Bacteria"

Jody Westbrook (UC Berkeley)

"Gene structure and alternative splicing in the unicellular ancestor of animals"

The evolution of multicellular animals from their unicellular progenitors required the evolution of coordinated cell behavior, growth and differentiation. Gene regulation is critical to all of these processes, and two important and inter-related aspects of metazoan gene regulation are alternative splicing and gene structure. To investigate how gene regulation evolved during the origin of metazoans, we have studied gene structure and alternative splicing in choanoflagellates, close unicellular relatives of animals. The genome sequences of two choanoflagellates showed that their genes, on average, have a density of introns comparable to the most intron-rich animal genomes. Comparison of orthologous genes show that many intron positions are conserved between animals and choanoflagellates, suggesting that these introns were present in their common ancestor. The presence of intron-rich genes in this ancestor raises the possibility that alternative splicing was employed as a form of gene regulation prior to the evolution of animals. To investigate this possibility, we analyzed the transcriptome of Monosiga brevicollis by RNA-seq. We find that at least twelve percent of M. brevicollis genes have alternatively spliced isoforms under normal growth conditions. Interestingly, the frequency of different types of alternative splicing were different from what is typically observed in metazoans. Exon-skipping was rarely detected in M. brevicollis genes while it is the most common type of alternative splicing in many animal genomes. Although most choanoflagellategenes are intron-rich and therefore have ample potential for alternative splicing, we found that the longest transcripts are encoded by genes with remarkably few introns, and rather consist of unusually long exons (>10,000 bp). One M. brevicollis gene, which we name Gargantua, contains the longest exon (59,595 bps) known in eukaryotes. Genes with unusually long exons were also found many additional eukaryotic genomes, showing that this gene structure is a widespread feature of eukaryotic genomes. Although widespread, the frequency of unusually long exons in the longest genes differs between choanoflagellates, sponges, and eumetazoans. Extremely long genes in the choanoflagellate M. brevicollis and the sponge Amphimedon queenslandica contain remarkably few introns whereas the longest eumetazoan genes are primarily intron-rich. Thus we have found that the intron-exon structure of genes evolved in unexpected ways during the early evolution of metazoans, and that these changes may have implication for how genes are regulated by alternative splicing.

sound bites
titles and abstracts

Pierre Durand (University of Witwatersrand)

"Cooperation and conflict in the evolutionary origins of life and death programs"

Cooperation and its nemesis conflict play a key role in the multi-level selection of new levels of complexity and organization. Recently, conceptual advances and empirical data have emerged suggesting that genetic programs promoting life (the proto-genome) and death (programmed cell death or PCD) originated during the multi-level selection cycles of cooperation and conflict early in the history of life. The origin of the genome is based on cooperation and conflict between self-replicating genetic elements, which lead to a new functionally integrated group of genes or gene-network: the proto-genome. The adaptive value of programmed death also lies at the group level where the avoidance of the harmful effects of necrosis and the provision of benefits is beneficial to neighbors. It is intriguing that while life and death may be considered the antithesis of each other, the two hypotheses presented here suggest that both life and death programs have adaptive origins in the cycles of conflict and cooperation which drive evolutionary transitions to higher levels of individuality. In life as well as in death, cooperation and multi-level selection take an unexpected central role.

Carey Nadell (Princeton)

"Emergence of Spatial Structure in Cell Groups and the Evolution of Cooperation"

On its own, a single cell cannot exert more than a microscopic influence on its immediate surroundings. However, via strength in numbers and the expression of cooperative phenotypes, such cells can enormously impact their environments. Simple cooperative phenotypes appear to abound in the microbial world, but explaining their evolution is challenging because they are often subject to exploitation by rapidly growing, non-cooperative cell lines. Population spatial structure may be critical for this problem because it influences the extent of interaction between cooperative and non-cooperative individuals. It is difficult for cooperative cells to succeed in competition if they become mixed with non-cooperative cells, which can exploit the public good without themselves paying a cost. However, if cooperative cells are segregated in space and preferentially interact with each other, they may prevail. Here we use a multi-agent computational model to study the origin of spatial structure within growing cell groups. Our simulations reveal that the spatial distribution of genetic lineages within these groups is linked to a small number of physical and biological parameters, including cell growth rate, nutrient availability, and nutrient diffusivity. Realistic changes in these parameters qualitatively alter the emergent structure of cell groups, and thereby determine whether cells with cooperative phenotypes can locally and globally outcompete exploitative cells. We argue that cooperative and exploitative cell lineages will spontaneously segregate in space under a wide range of conditions and, therefore, that cellular cooperation may evolve more readily than naively expected.

Darren Pais (Princeton)

"Role of the Interaction Graph Topology in the Evolution of Collective Migration"

We use the perspective of evolution by natural selection to investigate the collective migration problem, where individuals in a group can respond to social information and to a costly environmental cue. We study the role of the social interaction topology on evolutionary outcomes and demonstrate a minimum connectivity threshold for random interconnection graphs to yield speciated outcomes in responsive behavior. We study the adaptation of nodes on fixed graphs and how topology affects emergent results.

Mauricio Gonzalez-Forero (U of Tennessee)

"Eusociality through manipulation"

I consider a simple model for eusociality through maternal manipulation with the possibility of resistance. A mother induces her offspring to stay in the nest as adults and raise siblings. Manipulation evolves when b>c and hence Hamilton's rule does not need to be satisfied. If Hamilton's rule is not satisfied, manipulated individuals evolve resistance to manipulation. The evolution of resistance eliminates effective manipulation. Here I find that the evolution of resistance may be prevented if the benefit of altruism has a genetic component expressed in the recipient. Both spontaneous altruism and maternal manipulation create selection pressure for increased benefits to recipients. However, manipulated altruism can create such pressure even when Hamilton's rule is not satisfied if the benefit is controlled by the recipient. One way in which the recipient may control the benefit is if the recipient may choose to develop poorly as in altricial species. I obtain that the evolution of increasing recipient-controlled benefit as a result of manipulation may lead to a point where Hamilton's rule is eventually satisfied and hence resistance fails to evolve. Manipulation may thus successfully initiate eusociality under parameter values that seem more feasible for a presocial stage than the parameter values necessary for Hamilton's rule.

Kirill Korolev (MIT)

"Population Genetics in a Petri Dish"

Spatial structure plays an important role in the evolution and maintenance of cooperation. Spontaneous development of spatial structure is especially pronounced during geographic expansions when demographic stochasticity is significantly enhanced due to a small number of individuals colonizing new territories. Such expansions are common in nature, a disease outbreak being a familiar example, yet remain poorly understood. We used both theory and experiments with microbes to better understand the evolutionary dynamics of expanding populations. In addition, we developed a set of methods to estimate important biological parameters of microbial colonies (migration rates, selective advantage, and effective population size responsible for the fluctuations). These methods could be useful for future studies of evolutionary dynamics in populations with spatial structure.

Aditya Rawal (UT Austin)

"Emergence of Cooperation and Competition in Multi-agent systems"

In nature, multiple agents in teams collaborate and compete with one another at the same time. Replicating such agent interactions in games can make for realistic opponent teams. Yet cooperation and competition have mostly been studied separately so far. This work focuses on simultaneous cooperative and competitive coevolution in a complex predator-prey domain. A cooperative genetic algorithm architecture consisting of multiple cooperating neural networks within each agent is introduced. This architecture successfully results in hierarchical cooperation and competition in teams of prey and predators: In sustained coevolution, high-level pursuit-evasion behaviors emerge. In this manner, coevolution of neural networks is shown to scale up to an arms race of multiple competing and cooperating agents, more closely modeling coevolution of complex behavior in nature.

This talk/poster would be accompanied by video demonstration of cooperative behaviors that such artificial agents have successfully discovered.

Deborah Shelton (University of Arizona)

"The Evolution of Colonial Reproduction in Undifferentiated Volvocine Algae"

During an evolutionary transition in individuality, Darwinian principles shift from applying to a lower-level unit to applying to a higher-level unit (e.g. from cells to groups of cells). The ability to reproduce, to create new individuals from an existing one, is a Darwinian principle. That is, reproduction is necessary for evolution by natural selection, and understanding how reproduction can come to apply at a new level of organization is a major challenge. In this context, we examine the evolution of asexual reproduction in a model empirical system, the volvocine green algae. The asexual life cycle (i.e. asexual reproduction) in these relatively simple, photosynthetic freshwater algae consists of patterns of cell growth, cell division, and cell separation. All of these features are present in unicellular and multicellular (colonial) species. However, cell growth, cell division, and cell separation are organized differently in colonial compared to unicellular species. We review the among-species differences in volvocine life cycle organization and present preliminary results concerning the conditions under which life cycles can be shifted to a more "unicellular" or to a more "colonial" outcome.

Jeff Smith (Rice U)

"Microbial cooperation: quantitative tests and alternative hypotheses"

Kin selection is a prominent theoretical framework for understanding social evolution, but does it translate to the microbial world? How do we test kin selection hypotheses for cooperation in asexual organisms? How do we distinguish microbial adaptations to social life from "just-so stories"? I describe a generalization of Hamilton's rule that allows quantitative empirical tests and clarifies the meaning of "relatedness" in these species. I also show that non-adaptive processes can create the appearance of facultative cheating -- even in the absence of any cooperative trait. Experiments with Myxococcus bacteria show that facultative exploitation can be caused by both social and nonsocial effects on sporulation rate.

Sid Goyal, Madhav Mani and Boris Shraiman (KITP, UCSB)

"Multicellularity and Evolvability?"

Mutations arising in different individuals compete with each other and in large populations most deleterious mutations are lost. However, evolution of complex traits -- which involve interaction between different genetic loci -- may require populations to acquire deleterious or neutral intermediate mutations to eventually obtain a beneficial genotype. Can multicellularity facilitate evolution of such complex traits?

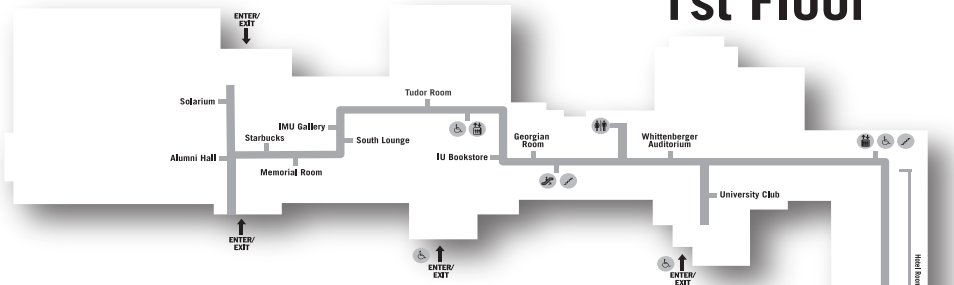
We consider here examples of organisms that have both multicellular and free-living life cycles. (1) Neurospora is a multinucleate filamentous fungi which also forms conventional spores with single nuclei. In the filamentous stage, the various nuclei flow inside the filaments and the gene products from different nuclei mix. Such mixing of gene products from different nuclei could increase the chances of accumulation of deleterious mutation due to buffering from other nuclei. (2) Biofilm is an aggregate of cells that behaves like a multicellular organism. In comparison to free-floating planktonic cell, cells in biofilms experience different environment. Could this allow deleterious mutations to accumulate in cells in a biofilm?

maps and miscellanea

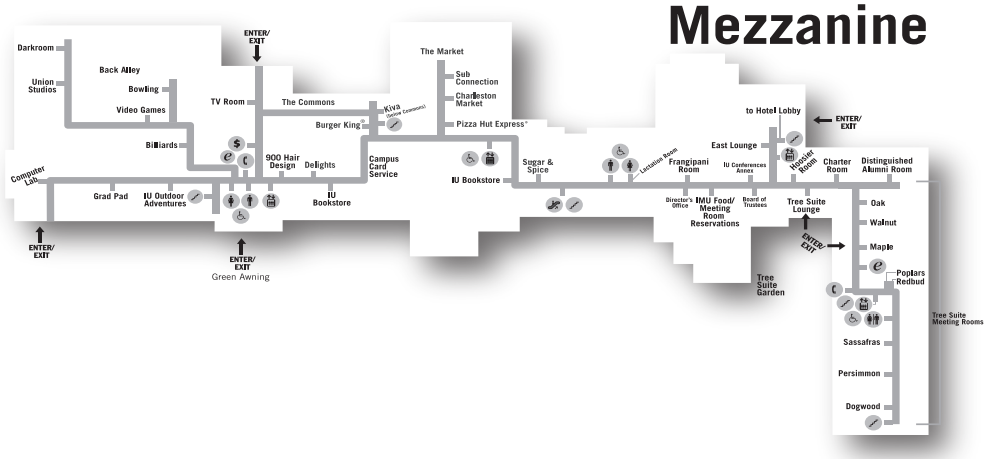
INDIANA MEMORIAL UNION GUIDE

2nd Floor

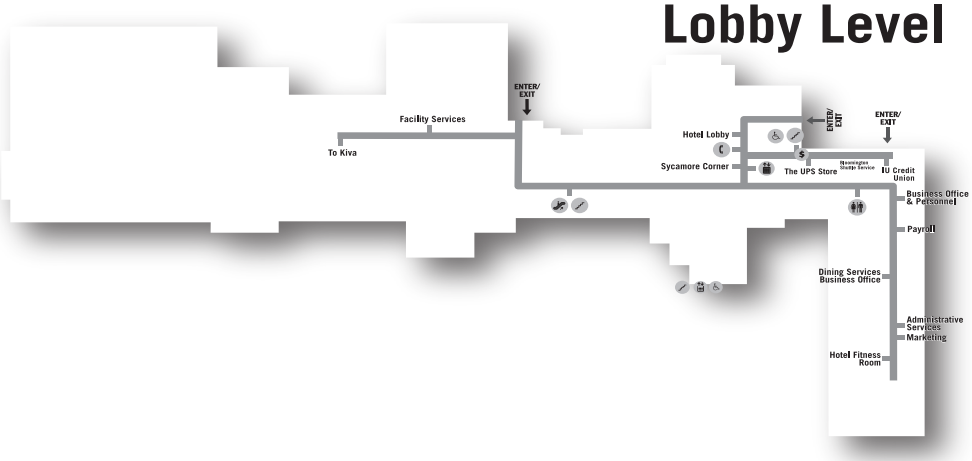
1st Floor



Mezzanine



Lobby Level



Legend

- Handicap accessible
- Men's Restroom
- Women's Restroom
- Elevator
- Public telephone
- Stairs
- ATM machine
- E-mail station
- Escalator
- Enter/Exit

INDIANA MEMORIAL UNION
INDIANA UNIVERSITY
Biddle Hotel & Conference Center
Bloomington

ACTIVITIES & ENTERTAINMENT

The Back Alley | MEZZANINE | (812) 855-2328

Check out "Moonlight Madness," glow-in-the-dark bowling on the weekends! Bowling, billiards, air hockey, and more.

Union Studios | (812) 855-2328

Ceramics and photography studios. Take a class or design-your-own workshop.

IU Outdoor Adventures | (812) 855-2231

Rent outdoor equipment, take a class or workshop, or design-your-own adventure.

DINING & SNACK SHOPS

Burger King | MEZZANINE | *WI-FI*

The Market | MEZZANINE | *WI-FI*

- **Charleston Market:**
  Hot breakfast and lunch bar.
- **Pizza Hut Express®**
  - **Sub Connection**

Delights | MEZZANINE

Bloomington-famous popcorn, bulk snacks, and drinks

Sugar & Spice | MEZZANINE

Green Mountain Coffee, baked goods, local goodies, organic snacks and much more!

Starbucks | FIRST FLOOR

Starbucks coffee and products.

Tudor Room | FIRST FLOOR | (812) 855-1620

Casual dining in an elegant setting. Grand buffet with an extensive salad and dessert bar. Reservations recommended.

Kiva | MEZZANINE

Healthy goodness from grilled paninis to a fresh salad bar.

WI-FI LOUNGES & PUBLIC SPACES

East Lounge | MEZZANINE

A large, comfortable lounge perfect for meeting friends, catching up on the news, or just sitting back and relaxing. The East Lounge is located directly above the hotel lobby.

IMU Gallery | FIRST FLOOR

An eclectic combination of a reading room, entertainment space, and gallery for students and local artists to showcase their talent. Now serving Starbucks coffee!

South Lounge | FIRST FLOOR

Often referred to as the fireplace lounge, the South Lounge is full of couches, antiques, and even a fireplace that burns year-round.

Tree Suite Lounge | MEZZANINE

A small lounge located adjacent to the Commemorative Garden. The perfect place to relax during conferences and other events taking place in the Tree Suites.

SERVICES

900 Hair Design | MEZZANINE | (812) 855-2633

The latest hair styling trends: cuts, highlights, and color. Plus manicures and quality hair care products. Walk-ins welcome. Expanded hours by appointment.

ATMs

MEZZANINE
Chase, Fifth Third, IU Credit Union, Key Bank, Old National

HOTEL LOBBY BREEZEWAY
IU Credit Union

UITS public computer lab | MEZZANINE

E-mail/Web computers | MEZZANINE, CampusLink Information Center; MEZZANINE, Tree Suites

IU Credit Union | HOTEL LOBBY BREEZEWAY

UPS Store | HOTEL LOBBY BREEZEWAY

Campus Card Service | FIRST FLOOR IU BOOKSTORE

SHOPPING

Computer Connection | MEZZANINE

Computers, CDs, cables, software, and more.

IU Bookstore | MEZZANINE AND FIRST FLOOR

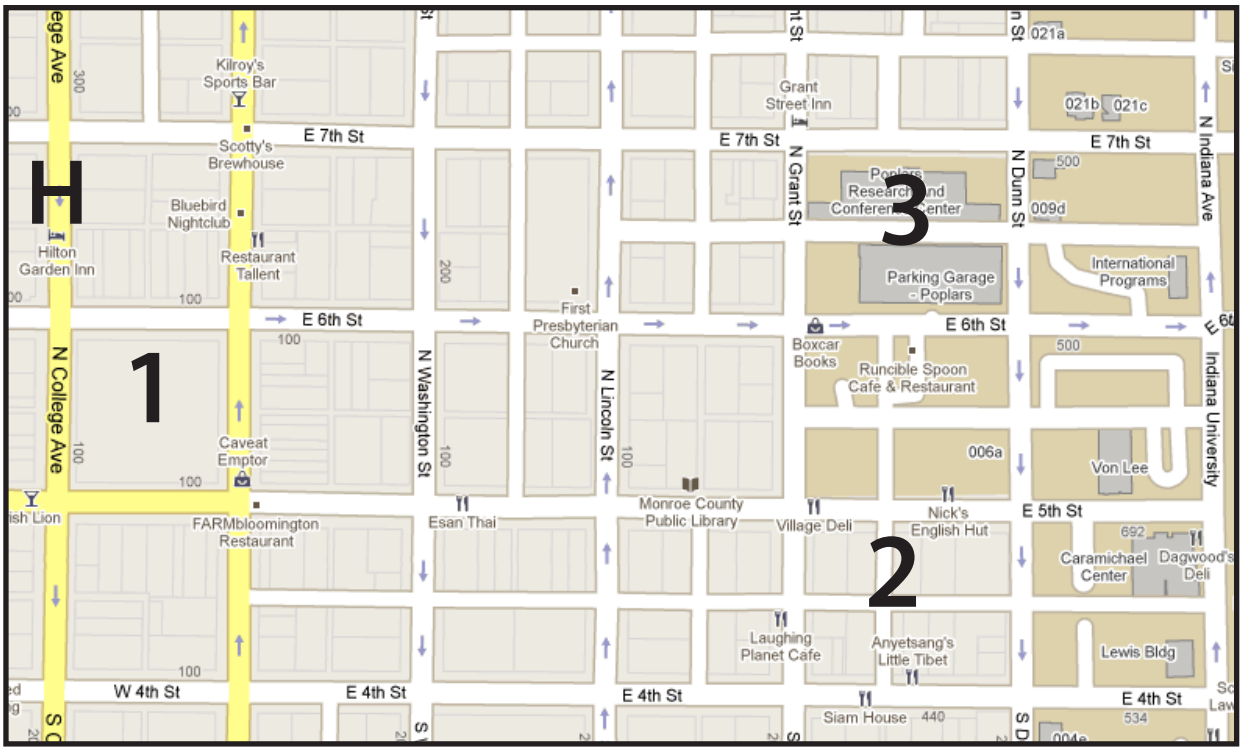
The place for textbooks, other reading, and IU souvenirs.

Sycamore Corner | MEZZANINE

The Union's convenience store. A little bit of everything: munchies, grab-n-go foods, drinks, newspapers, and magazines.

please see **www.imu.indiana.edu** for more information and up-to date hours

Downtown Map



Legend

1	Downtown
2	4th Street Area
3	6th Street Area

Dining

1 Downtown

Crazy Horse
214 W. Kirkwood Avenue
336-8877
Bar/Grill
L D V B W

Bloomingfoods.
316 W. 6th Street
Grocery Store, Buffet
R L D V B W

Grazie!
106 W. 6th Street
323-0303
Italian/Pizza
L D B W

Irish Lion
212 W. Kirkwood Avenue
336-9076
Irish Casual Dining
L D V B W

Janko's Little Zagreb
223 W. 6th Street (at Morton)
332-0694
Steakhouse/Casual Dining

Le Petit Café
308 W. 6th Street
334-9747
French
D V B W

Malibu Grill
106 N. Walnut Avenue
332-4334
Bar/Grill
L D V B W

Michael's Uptown Café & Bakery
102 E. Kirkwood Avenue
339-0900
American Café
R L D V B W

Opie Taylor's Burger Works
110 N. Walnut Avenue
333-7287
American Casual Dining
L D V B W

Samira's Restaurant
100 W. 6th Street
331-3761
Afghani Casual Dining

Scotty's Brewhouse
302 N. Walnut Street
333-5151
Bar/Grill
L D B W

Shanti Indian Cuisine
221 E. Kirkwood Avenue
Suite G
333-0303
Indian
L D V

Soma Coffee House
322 E. Kirkwood Avenue
331-2770
Specialty
R L V

Trojan Horse
100 E. Kirkwood Avenue
332-110
Greek
L D V B W

FARMbloomington
108 East Kirkwood Avenue
323-0002
Casual American Dining
L D V B W

2 4th Street Area

Anatolia
405 E. 4th Street
334-2991
Turkish
L D

Anyetsang's Little Tibet
415 E. 4th Street
331-0122
Tibetan
L D V

Bombay House
416 E. 4th Street
323-8962
Indian
L D

Puccini's
420 E. 4th Street
333-5522
Italian
L D V B W

Siam House
430 E. 4th Street
331-1233
Thai
L D V B W

Turkuaz Café
301 E. Third Street
333-7908
Italian
L D V B W

3 6th Street Area

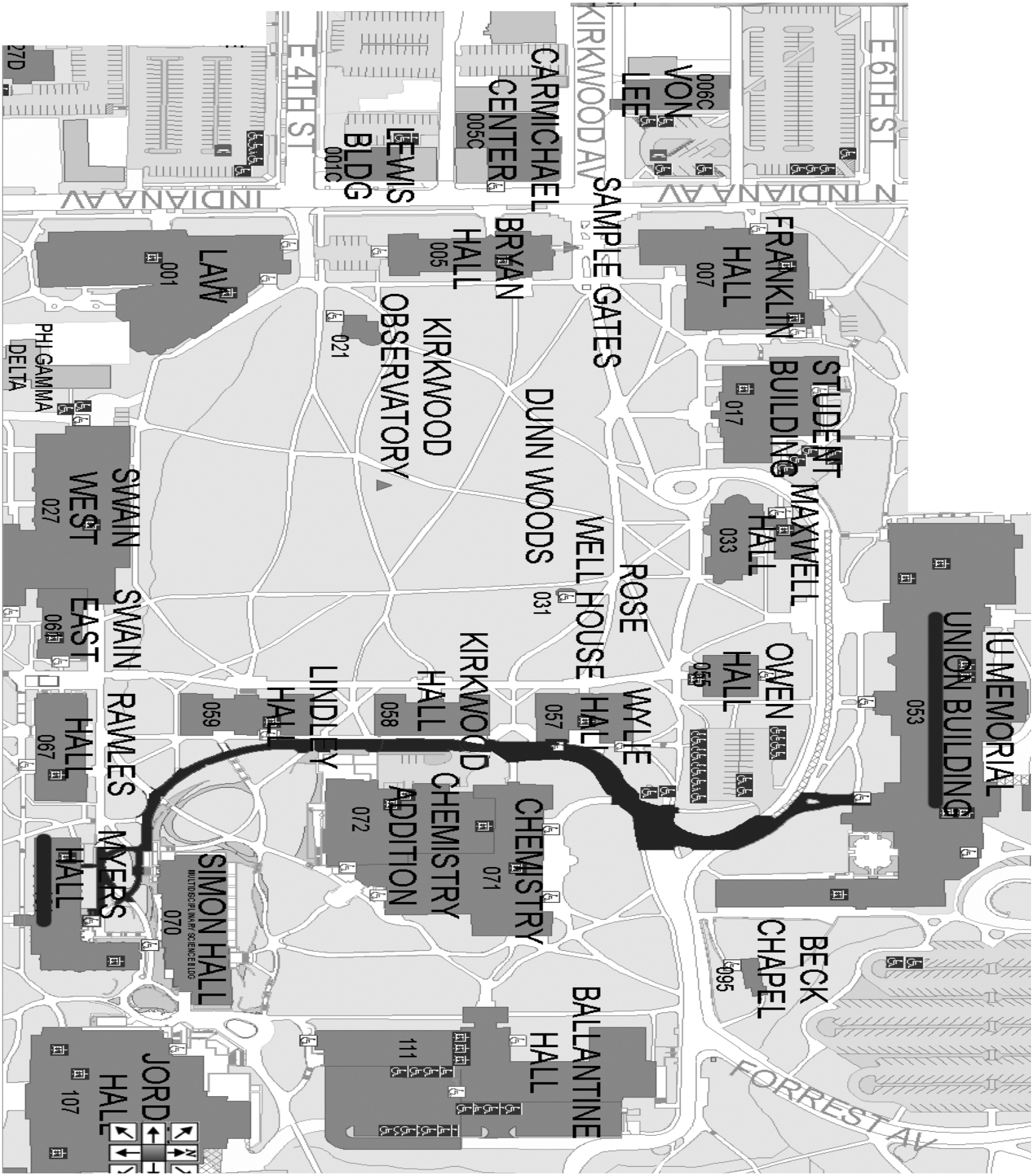
Café Django
116 N. Grant Street
335-1297
Specialty Café
R L D W

Runcible Spoon
412 E. 6th Street
334-3997
Specialty Café
R L V

Legend

R ..... Serves breakfast
L ..... Serves lunch
D ..... Serves dinner
B ..... Serves beer
W ..... Serves wine
V ..... Has vegetarian dishes

Getting to Myers Hall



Getting to the Tree Suites