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BLAKE LEON

Using Phenotyped But Ungenotyped Relatives in Genetic Association Tests

NewPath Learning

Sparsity is one of the intrinsic properties of real-world data, thus sparse representation based learning models have been widely used to simplify data modeling and discover predictive patterns. By enforcing properly designed structured sparsity, one can unify specific data structures with the learning model. We proposed several novel structured sparsity learning models for multi-modal data fusion, heterogeneous tasks integration, and group structured feature selection. We applied our new structured sparse learning methods to the emerging imaging genetics studies by integrating phenotypes and genotypes to discover new biomarkers which are able to characterize neurodegenerative process in the progression of Alzheimer's disease and other brain disorders. Different to traditional association studies, our new structured sparse learning models can elegantly take advantage of the useful information contained in biomarkers, cognitive measures, and disease status, where, crucially, the interrelated structures within and between both genetic/imaging data and clinical outcomes are gracefully exploited by our newly designed convex sparse regularization models. We empirically evaluate our new methods on the Alzheimer's Disease Neuroimaging Initiative (ADNI) cohort to identify Alzheimer's disease (AD) risky biomarkers, where we have achieved not only clearly improved prediction performance for

cognitive measurements and diagnosis status, but also a compact set of highly suggestive biomarkers relevant to AD.
Dyslipoproteinemia GRIN Verlag
Whereas genetic studies have traditionally focused on explaining heritance of single traits and their phenotypes, recent technological advances have made it possible to comprehensively dissect the genetic architecture of complex traits and quantify how genes interact to shape phenotypes. This exciting new area has been termed systems genetics and is born out of a synthesis of multiple fields, integrating a range of approaches and exploiting our increased ability to obtain quantitative and detailed measurements on a broad spectrum of phenotypes. Gathering the contributions of leading scientists, both computational and experimental, this book shows how experimental perturbations can help us to understand the link between genotype and phenotype. A snapshot of current research activity and state-of-the-art approaches to systems genetics are provided, including work from model organisms such as Saccharomyces cerevisiae and Drosophila melanogaster, as well as from human studies.
Investigation of Methods for Machine Learning Associations Between Genetic Varations and Phenotype National Academies Press
Herpes simplex virus type 1 (HSV-1) is the primary source of infectious blindness in the United States, and animal studies have shown the severity of infection is influenced by several factors, including viral strain. We perform a viral genotype-phenotype association analysis by applying machine-learning approaches to identify genetic determinants of host disease phenotypes. Given a dataset composed of genome sequences with different genetic variations, and the phenotypes quantifying the severity of ocular diseases in various dimensions, we (i) establish feature vectors representing genetic variations acquired from a multiple sequence alignment of the genotypes, (ii) learn several types of regression models, (iii) evaluate the predictive accuracy of these learning approaches by conducting cross-validation accompanied with a Monte Carlo methodology. In many application domains where black-box models provide the best predictive accuracy, it is important to characterize how learned models make their decisions across the distribution of instances. We present a model-agnostic approach to this task with large, structured feature spaces that makes the following specific contributions. Our approach (i) tests feature groups, in addition to base features, and tries to determine the right level of resolution at which important features can be identified, (ii) uses hypothesis testing to assess the effect of each feature on the model's loss, (iii) employs a hierarchical approach to control the false discovery rate when testing feature groups and individual base features for importance, and (iv) uses hypothesis testing to identify important interactions among features and feature groups. We evaluate our approach by analyzing random forest models learned in the viral genotype-phenotype association analysis, as well as another challenging biomedical application. Through the application of our method, additional insights into virulence genes and epistatic interactions in HSV-1 are gained. This approach lays the groundwork for future virulence studies. Additionally, we apply

computational methods to characterize sequence properties of genetic recombination in HSV-1, which is a highly recombinogenic virus. We perform recombination breakpoint analysis to test whether the occurrence of recombination events (i) reveals any bias toward specific regions of HSV-1 genome, (ii) appears to be responsible for spontaneous mutations, (iii) indicates recombination hot spots. *Statistical Models for High Dimensional Screening of Genetic and Epigenetic Effects* National Academies Press Evolutionary biology is challenged with understanding how genotypes shape phenotypes, how and when selection acts on phenotypes, and how those selective forces translate to changes in the underlying frequencies of those genotypes: the genotype-phenotype-fitness map. Theoretical and technological advances have facilitated progress connecting genotypes to phenotypes and phenotypes to fitness, yet we have few cases of a complete genotype-phenotype-fitness map, limiting our ability to accurately predict evolutionary outcomes. This dissertation describes my work on the genotype-phenotype-fitness map of an adaptive and pleiotropic haplotype in threespine stickleback (Gasterosteus aculeatus), a powerful model of adaptive evolution. In the introduction (Chapter 1) I provide background relevant to the genetics of adaptation and my work, including progress on answering outstanding questions in the field. In Chapter 2 I describe my work on the genetic architecture of adaptation within a pleiotropic haplotype containing the developmental signaling gene, Ectodysplasin (Eda). Specifically, I present the results of fine-mapping multiple traits in two stickleback populations aimed at disentangling the roles of pleiotropy and linkage. I find that the 16 kilobase Eda haplotype is significantly associated with three phenotypes in both populations of stickleback, and that all three phenotypes show the same pattern of association with the genetic markers within a small 1.4 kb region of the haplotype, suggestive of a pleiotropic mutation. In Chapter 3, I present the results of an empirical test of a leading hypothesis about selection on Eda; namely I test the effect of an abiotic agent of selection (dietary phosphorus) on a component of fitness (juvenile growth rate) in experimental crosses between fish that differ in their genotype at the Eda haplotype. The results of this experiment suggest that phosphorus limitation is not the agent of selection acting on the Eda haplotype, and highlight the importance of testing hypotheses of selection by experimentally connecting genotypes to fitness through selection on phenotypes. I end in Chapter 4 with my interpretation of these results, the implications of these findings, and my suggestions for future work in this system on the genetics of adaptation.

Crumbling Genome Springer Background and Aims:CADASIL is the most common monogenic cerebral small vessel disorder, caused by distinctive cysteine-altering mutations affecting the 34 EGFr domains of the NOTCH3 protein. A recent report suggests that mutations outside EGFr domains 1-6 are mainly paucisymptomatic and have lower MRI lesion loads. We investigated the genotype-phenotype correlation in a CADASIL cohort.Methods:We reviewed clinical and imaging features of CADASIL patients who attended a Neurovascular Genetics Clinic, between January 2001 and

October 2018. The cohort was divided into two groups: proximal-genotype (EGFr domains 1-6) and distal-genotype (EGFr domains 7-34). Leukoaraiosis, microbleeds and lacunes were manually measured using MANGO software. The relationships between genotype, clinical phenotype and imaging phenotypes were explored by linear regression, co-varying for age, sex and risk factors. Log-rank tests were performed for time to event analysis for clinical end-points.Results:We included 165 CADASIL patients with cysteine missense mutations, 140 of whom were proximal-genotype and 25 distal-genotype. Compared to proximal-genotype cases, distal-genotype patients experienced their first stroke 6 years later (mean 52.1, SD 12.13; p=0.05); onset of cognitive impairment (5 years later, mean 53.6, SD 14.3) and neuropsychiatric disorders (3 years later, mean 48.4, SD 15.2) was not significantly different between genotypes. Volumes of subcortical white matter hyperintensities (mean 112 ml, SD 37), lacune counts (mean 2.5, SD 3.5) and microbleeds (0) were significantly less in the distal genotype patients.Conclusions:We confirmed a genotype-phenotype correlation in CADASIL, mutations outside the classic EGFr domains 1-6 being associated with later onset of symptoms and lower MRI structural lesion loads.

Understanding Racial and Ethnic Differences in Health in Late Life John Wiley & Sons

Despite its centrality to Darwin's theory of evolution by natural selection, the process of adaptation is still not fully understood. In particular, the dynamics of the genotypes and phenotypes associated with an adaptive response remain to be fully elucidated. In my dissertation, I utilized laboratory evolution experiments to study how the genotypes and phenotypes of Escherichia coli change over time as they adapted to high temperature.Chapter 1 explored how metabolic phenotypes of 115 evolved E. coli clones changed as a result of 2,000 generations of adaptation to 42.2°C. Using phenotypic microarrays (Biolog plates), I quantified 94 phenotypes of these evolved clones, as well as their ancestor under stressed (42.2°C) and unstressed (37.0°C) conditions. Comparing the evolved phenotypes to the ancestral phenotypes revealed that adaptation was predominantly restorative, shifting evolved phenotypes from the stress state toward the unstressed state. I also uncovered associations among common genotypic changes found in the evolved clones and their phenotypes.Chapter 2 investigated the different mutational dynamics in populations traversing two different adaptive pathways typified by mutations in the rpoB and rho genes, respectively. These genes were predicted to be differentially pleiotropic, and were therefore expected to create differences in compensatory evolution when mutated. I used temporal sequencing data of four rpoB and four rho populations to reconstruct their mutational trajectories over the course of adaptation to 42.2°C. These trajectories revealed that rpoB and rho mutations occurred early on during adaptation, canalizing the adaptive process. Furthermore, rpoB populations accumulated more mutations and experienced more clonal interference over the course of adaptation than rho populations.Chapter 3 was a study of the Lazarus effect, a phenomenon of population recovery under lethal selection

conditions. I evolved ~300 E. coli populations to the lethal temperature of 43.0°C and measured their cell density over five days. I sequenced those populations that recovered and found mutations in two operons---hslUV and rpoBC---to be the major drivers of Lazarus events. These mutations differed in their frequency in the experiment, degree of parallelism within and between weeks, and fitness tradeoffs at 37.0°C, suggesting different origins and adaptive dynamics between them.

Molecular Biology of the Cell CRC Press

This timely text presents a comprehensive guide to genetic association, a new and rapidly expanding field that aims to elucidate how our genetic code (genotypes) influences the traits we possess (phenotypes). The book provides a detailed review of methods of gene mapping used in association with experimental crosses, as well as genome-wide association studies. Emphasis is placed on model selection procedures for analyzing data from large-scale genome scans based on specifically designed modifications of the Bayesian information criterion. Features: presents a thorough introduction to the theoretical background to studies of genetic association (both genetic and statistical); reviews the latest advances in the field; illustrates the properties of methods for mapping quantitative trait loci using computer simulations and the analysis of real data; discusses open challenges; includes an extensive statistical appendix as a reference for those who are not totally familiar with the fundamentals of statistics.

Principles of Biology Cambridge University Press

The fundamental goal of genetics is to understand the functional effect of DNA sequence variations on a wide range of phenotypes, from basic biology to genetic diseases. Broadly, there are two major strategies to approach this goal: the first one is to find natural genetic variants underlying the trait of interest through linkage or association studies; the other is experimentally introducing genetic perturbations and assaying the effects of the perturbations in a high-throughput manner. In this dissertation, both approaches were employed to understand the effect of genetic variants. Following the first approach, we used linkage analysis to find the genetic basis of mutation rate variation in yeast. We developed a high-throughput fluctuation assay to enable quantification of spontaneous mutation rate in hundreds of yeast for the first time. We measured the mutation rate of 1040 yeast segregants from a cross between two diverge yeast strains, BY and RM. Combined with the genotype data, we performed linkage analysis in the segregants and identified four quantitative trait loci (QTLs) that contribute to the mutation rate variation in the cross. We fine-mapped two QTLs to the underlying causal genes, RAD5 and MKT1, that contribute to mutation rate variation. For the second approach, we developed three different systems to study the effect of natural variants using the genetic engineering tool CRISPR-Cas9. We constructed ten different CRISPR-Cas9 base editor systems for yeast, aiming to expand the targetable regions and the base converting types by using different base editors. We measured the efficiency of ten base editors in yeast from amplicon sequencing results at ten different sites along the genome and found one base

editor that recognized the protospacer adjacent motif (PAM) site NGA with high efficiency. In addition to CRISPR base editor, we constructed a precise genome editing system with trackable genome integrated barcode using CRISPR-Cas9 with gRNA and donor DNA pairs. The integrated barcode enables precise tracking of edited strains with sequencing, ensuring robust downstream phenotyping. We also worked toward developing a CRISPR-directed mitotic recombination mapping panel in human cell lines to narrow down mapped out regions to causal genes by targeted creation of DNA double strand breaks along the chromosome.

Scientific Frontiers in Developmental Toxicology and Risk Assessment Univ of California Press

Essay from the year 2002 in the subject Biology - Genetics / Gene Technology, grade: 2.1 (B), Oxford University (New College), 6 entries in the bibliography, language: English, abstract: Ultimately, the goal of genetics is the analysis of the genotype of organisms. But the genotype can be identified – and therefore studied – only through its phenotypic effect. This means that two genotypes are recognised as different from each other because the phenotypes of their carriers are different. A problem can be seen with this approach as the actual variation between organisms is usually quantitative, not qualitative. Many different genotypes may have the same average phenotype. At the same time, because of environmental variation, two individuals of the same genotype may not have the same phenotype. This lack of a one-to-one correspondence between genotype and phenotype obscures underlying Mendelian genetics. I am going to explore the use of various statistical techniques for studying quantitative traits with application to behavioural traits. I am also going to examine whether there are behavioural traits with sufficiently high heritabilities to give hope for gene searches and I am going to discuss the difficulties that confront molecular geneticists regarding psychiatric genetics.

Interrogating the Genotype-phenotype-fitness Map of an Adaptive Haplotype in Threespine Stickleback

Principles of BiologyThe Principles of Biology sequence (BI 211, 212 and 213) introduces biology as a scientific discipline for students planning to major in biology and other science disciplines. Laboratories and classroom activities introduce techniques used to study biological processes and provide opportunities for students to develop their ability to conduct research.Genetics: The Study of Heredity Science Learning Guide

The sequencing of genomes has been completed for an increasing number of crop species, and researchers have now succeeded in isolating and characterising many important QTLs/genes. High expectations from genomics, however, are waving back toward the recognition that crop physiology is also important for realistic improvement of crop productivity. Complex processes and networks along various hierarchical levels of crop growth and development can be thoroughly understood with the help of their mathematical description – modelling. The further practical application of these understandings also requires quantitative predictions. In order to better support design, engineering and breeding for new crops and cultivars for improving agricultural production under global warming and climate change, there is an

increasing call for an interdisciplinary research approach, which combines modern genetics and genomics, traditional physiology and biochemistry, and advanced bioinformatics and modelling. Such an interdisciplinary approach has been practised in various research groups for many years. However, it does not seem to be fully covered in the format of book publications. We want to initiate a book project on crop systems biology - narrowing the gaps between genotypes and phenotypes and the gaps between crop modelling and genetics/genomics, for publication in 2013/2014. The book will be meant for those scientists and graduate students from fundamental plant biology and applied crop science who are interested in bridging the gap between these two fields. We have invited a group of scientists (who have very good track records in publishing excellent papers in this field or in a closely related area) to contribute chapters to this new book, and they have agreed to do so.

Phenotypic and Genetic Studies of Grapevine Springer

Predicting quantitative traits from genomic data provides considerable information in studies of human diseases and livestock breeding. However, phenotype prediction is usually confronted with genetic relatedness among individuals. It is even more challenging when there are quite a number of factors associated with traits under study (the problem of p > n). In this study, we propose to incorporate pedigree structure as a polygenic random effect into a mixed linear model to predict phenotypes. The beauty of our approach is that we can include all available markers in the prediction model, even in the situation of p > n. A penalized maximum likelihood method is derived to estimate parameters in the mixed model. The performance of predictions is evaluated through 95% coverage rate and mean squared prediction error. We also investigate the robustness of our proposed prediction model in some common scenarios. Specifically, we explore the effect of several main factors on the performance of our method. These factors include sample size, trait heritability, magnitude of additive effects, and complexity of pedigree structures. Simulation results show that our method achieves high 95% coverage rates with mean square prediction error well controlled in most of scenarios. The performance is consistent across all pedigree structures, which indicates that our method may be applied for individuals with arbitrary unknown relationships. Predictions can be improved with increased sample size or for a trait with higher heritability as we expected. To evaluate the performance of our method in real data analysis, we apply the proposed method to predict traits of laboratory mice with unknown pedigrees. The results of comparison between the predicted and true laboratory measured traits are satisfactory.

Biology for AP ® Courses

Plant breeding is the science of altering a plant's genetics to attain a desired phenotype. In this dissertation, I explore what phenotypes to measure when breeding for downy mildew resistance and improved floral scent and how to measure these phenotypes accurately and efficiently. Traditionally, downy mildew resistance has been measured by visually rating sporulation and hypersensitive response on leaves or leaf discs. However, such manual ratings become intractable

when dealing with thousands of samples. Therefore, to measure sporulation on leaf discs, I developed a computer vision system that reduced phenotyping time by more than 90% when compared to manual ratings, and also was found to work well for phenotyping leaf trichomes. If phenotypes are collected in the vineyard, spatial variation from inoculum, soil, and microclimate might have an effect on these phenotypes. Testing this assumption, spatial processes explained some variance in vineyard phenotypes, but accounting for the spatial variance might not lead to significantly more accurate phenotypes. Quantitative phenotyping of floral scent for large numbers of grapevines using headspace analysis is not economically feasible, so I evaluated the robustness of a hexane extraction followed by gas chromatography-mass spectrometry to identify floral volatiles and found that it was robust regardless of extraction time when flowers were sampled from the same inflorescence. After obtaining phenotypes and genotypes of vines, quantitative trait loci are found, traditionally using one phenotype at a time. In our case, understanding how sporulation, HR, and leaf trichomes affected each other was of interest, in addition to how genetic markers affected the phenotypes, so I used Bayesian networks to explore these interactions. In one of two F1 families studied, HR had a positive effect on sporulation, and leaf trichomes had a negative effect on both HR and sporulation, suggesting that leaf trichome density can be selected for in breeding for downy mildew disease resistance. A breeding project was started with the intention of creating a dwarf grapevine with an attractive floral scent. With a complementary interest to understand what volatile compounds were responsible for the various floral scents in grapevine, a diverse set of genotypes from various Vitis spp. were phenotyped for floral scent and volatiles, and it was found that similar scents were generated from different sesquiterpene profiles. Overall, this dissertation spans key concepts in the science of plant breeding, from parental selection and hybridization, to phenotyping by computer vision and chemical analysis, to statistical analyses of interacting phenotypes, genotypes, and spatial variability, with the findings possibly enhancing grapevine breeding strategies and execution.

Statistical Methods for Inferring Correlation and Causation Between Genotypes and Phenotypes

Abstract: In some longitudinal studies, there are individuals for whom rich phenotypic data have been collected, but who died before providing DNA for genetic studies. Genotypes of their relatives are often available. The main question we address is how and when one should incorporate phenotyped but ungenotyped relatives into genetic association tests. For genotypes missing completely at random (MCAR) and a quantitative outcome, Visscher and Duffy (2006) inferred the power increase due to the inclusion of ungenotyped individuals using information from relatives' genotypes for the case of a single genotyped single-nucleotide polymorpherm (SNP) and a single type of relative. We derive a theoretical formula for the power gain for a dichotomous outcome. We verify and extend the theoretical result with simulations of small or moderate sized pedigrees assuming a MCAR, missing at random (MAR), or not

missing at random (NMAR) missingness mechanism. For quantitative and binary outcomes, we observe biased effect estimates in data sets that exclude subjects with MAR genotypes and in data sets that include imputed NMAR genotypes. For most situations, power increases when ungenotyped individuals are included using imputed genotypes. The missingness mechanism, heritability, minor allele frequency, and SNP-specific heritability are important factors in the change in power for dichotomous or quantitative outcomes.We find that the increase in the test statistic from including individuals with genotypes imputed based on relatives' genotypes compared to omitting these individuals is about half of what could be attained using the true genotypes if they were available. Therefore, we propose a phenotypically enriched genotypic imputation (PEGI) method to impute missing genotypes using observed phenotypes in addition to genotypes. Our simulations with MCAR genotypes show that, for a SNP with moderate to strong effect on a phenotype, PEGI improves power more than imputation based solely on genotypes without excess type I errors. The effect estimate is often biased when the outcome is used for imputation while it is unbiased when a phenotype unrelated with the outcome is used. Compared to using only the observed genotypes for imputation, the PEGI method may improve power for MCAR, MAR, or NMAR genotype data.

Genotypic and Phenotypic Dynamics of Adaptation in Experimentally Evolved Escherichia Coli

Illuminating the processes and patterns that link genotype to phenotype, epigenetics seeks to explain features, characters, and developmental mechanisms that can only be understood in terms of interactions that arise above the level of the gene. With chapters written by leading authorities, this volume offers a broad integrative survey of epigenetics. Approaching this complex subject from a variety of perspectives, it presents a broad, historically grounded view that demonstrates the utility of this approach for understanding complex biological systems in development, disease, and evolution. Chapters cover such topics as morphogenesis and organ formation, conceptual foundations, and cell differentiation, and together demonstrate that the integration of epigenetics into mainstream developmental biology is essential for answering fundamental questions about how phenotypic traits are produced.

Systems Genetics

Understanding the relationship between genotypes and phenotypes is one of the major aims in biology and medicine. An important foundation to understand this relationship is the clear understanding of the pattern of genetic diversity in different human populations and how it correlates with complex genetic diseases. This would facilitate determining why there are differences in susceptibility to common disease among individuals and populations from different continental ancestry groups. In chapter one, I review the critical background for studying the genetics of complex disease. This includes recent studies for ascertaining the genetic structure of human populations using genetic markers, the importance of genetic variations and how it affects the development of specific phenotypes and specific methods to incorporate genetic

substructure in association tests. In chapter two, I introduce our ancestry informative markers (AIMs) set for assessing the continental ancestry and admixture proportions in common populations in America. This set of 128 SNPs can correct for populations stratification in admixed population sample sets. In chapter three, I present additional studies validating these AIMs in multiple population groups from Oceana, South Asia, East Asia, Sub-Saharan African, North and South America and Europe. In addition, a subset of this AIM's, which consists of 93 AIM's are effective in identifying the continental subject groups from the Human Genome Diversity Panels. In chapter Four, I present the application of these AIMs to evaluate the association of genetic admixture in African American and Hispanic populations in Women Health Initiative study (WHI) with different measurements of obesity including Body Mass Index (BMI) and Waist-Hip Ratio (WHR). In chapter five, I further present results of the association of genetic admixture in the same populations and different measurements of hypertension. In chapter six, I discuss some perspective and the direction for the future of this research.

Genetic Background and Environmental Effects on Single Nucleotide Polymorphisms in the NADPH Pathway

Concepts of Biology is designed for the single-semester introduction to biology course for non-science majors, which for many students is their only college-level science course. As such, this course represents an important opportunity for students to develop the necessary knowledge, tools, and skills to make informed decisions as they continue with their lives. Rather than being mired down with facts and vocabulary, the typical non-science major student needs information presented in a way that is easy to read and understand. Even more importantly, the content should be meaningful. Students do much better when they understand why biology is relevant to their everyday lives. For these reasons, Concepts of Biology is grounded on an evolutionary basis and includes exciting features that highlight careers in the biological sciences and everyday applications of the concepts at hand.We also strive to show the interconnectedness of topics within this extremely broad discipline. In order to meet the needs of today's instructors and students, we maintain the overall organization and coverage found in most syllabi for this course. A strength of Concepts of Biology is that instructors can customize the book, adapting it to the approach that works best in their classroom. Concepts of Biology also includes an innovative art program that incorporates critical thinking and clicker questions to help students understand--and apply--key concepts.

Alleles Responsible for ABO Phenotype-Genotype Discrepancy and Alleles in Individuals with a Weak Expression of A Or B Antigens

Biology for AP® courses covers the scope and sequence requirements of a typical two-semester Advanced Placement® biology course. The text provides comprehensive coverage of foundational research and core biology concepts through an evolutionary lens. Biology for AP® Courses was designed to meet and exceed the requirements of the College Board’s AP® Biology framework while allowing significant flexibility for instructors. Each section of the book

includes an introduction based on the AP® curriculum and includes rich features that engage students in scientific practice and AP® test preparation; it also highlights careers and research opportunities in biological sciences.

Temperature-dependent Patterns of Gene Expression in Caenorhabditis Briggsae

As the population of older Americans grows, it is becoming more racially and ethnically diverse. Differences in health by racial and ethnic status could be increasingly consequential for health policy and programs. Such differences are not simply a matter of education or ability to pay for health care. For instance, Asian Americans and Hispanics appear to be in better health, on a number of indicators, than White Americans, despite, on average, lower socioeconomic status. The reasons are complex, including possible roles for such factors as selective migration, risk behaviors, exposure to various stressors, patient attitudes, and geographic variation in health care. This volume, produced by a multidisciplinary panel, considers such possible explanations for racial and ethnic health differentials within an integrated framework. It provides a concise summary of available research and lays out a research agenda to address the many uncertainties in current knowledge. It recommends, for instance, looking at health differentials across the life course and deciphering the links between factors presumably producing differentials and biopsychosocial mechanisms that lead to impaired health.

Epigenetics

The Genetics: The Study of Heredity Student Learning Guide includes self-directed readings, easy-to-follow illustrated explanations, guiding questions, inquiry-based activities, a lab investigation, key vocabulary review and assessment review questions, along with a post-test. It covers the following standards-aligned concepts: How Trait are Inherited; Chromosomes & Karyotypes; Gregor Mendel; Mendel?s Experiments; Dominant and Recessive Traits; Punnett Squares; Phenotypes & Genotypes; Codominance; and Making a Pedigree. Aligned to Next Generation Science Standards (NGSS) and other state standards.

From Genotype to Phenotype

A major focus in modern genomics is determining the connection between genotypes and quantifying phenotypes. In this connection, many factors come into play including different genetic backgrounds, genetic variation at a locus, and environmental conditions. Genetic variation in Drosophila melanogaster, and specifically the simple polymorphisms within Malic enzyme (Men), can provide insight into the pathways between genotypes and phenotypes. Globally, there are two polymorphic sites in the malic enzyme gene. On site is near the protein (MEN) active site and found at an allelic frequency of 50% glycine amino acid and 50% alanine amino acid. The second polymorphism is buried within the protein and found at an allelic frequency of 90% methionine amino acid and 10% leucine amino acid. To determine the complexity of the pathway between genotypes and phenotypes, multiple genetic backgrounds for each genotype, using multiple D. melanogaster lines, were included to explore and quantify genetic background effects, and paraquat was used to induce oxidative stress. The biochemical

characteristics of the alleles varied significantly between the genotypes under benign conditions and both polymorphic sites effected some phenotypes. The first site played a role in the MEN Vmax and Km; the glycine allele had 14% higher Vmax activity than the alanine allele and the glycine allele had 8% higher Km than the alanine allele. The second site influenced the Km and Vmax/Km ratio (relative activity); the methionine allele had 34% higher malate Km than the leucine allele the leucine allele had 52% higher relative activity than the methionine allele. Interestingly, the protein product encoded by the rarer allele, leucine, had a higher relative activity and lower Km concentration, having a large impact on the enzymatic phenotype. These extreme phenotypes of that allele may be an indication of the why the allele is maintained at 10% across populations. Different lines with the same genotype had different biochemical phenotypes, indicating the importance of backgrounds effects influencing the final phenotype. Further, the flies' phenotypes differed between benign and oxidative stress conditions. Flies exposed to paraquat had a decrease in MEN Vmax, and the MEN alleles did not significantly differ from each other. Overall, the findings from this study suggest that the final phenotype are strongly influenced by the polymorphisms found in MEN, the interactions between genetic background and environmental conditions.

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