

Mitosis

How do living things grow and repair themselves?

Why?

Living things must grow and develop. At times they suffer injuries or damage, or cells simply wear out. New cells must be formed for the organism to survive. What process must occur to make a new, properly-functioning cell?

Model 1 – Mitosis as Part of the Cell Cycle



1. Refer to Model 1. List the four phases in the mitosis process.

Pro phase , Metaphase , Anaphase , Telophase

2. Where is mitosis in the cell cycle? Before G₁ and after G₂.

3. What three phases of the cell cycle are considered interphase?

G₁ , Synthesis , G₂ .

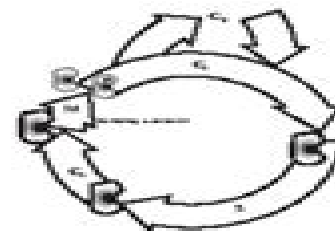
4. Refer to the cell cycle shown.

- a. How many cells are present at the beginning of mitosis?

one

- b. How many cells are present at the end of mitosis?

Two



Hspi The Pogil Project Genetic Variation

Peipei Pang



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Human Genetic Variation ,1999 *Global Views of Human Genetic Variation* Coleman A. Mosley,2006 **Genetic Variation in Gp120** Isaac Ang'Ang'A. Choge,2003 Interrogating Genetic Variation in the Major Histocompatibility Complex Xiaoming (Sherman). Ja,2012 Functional Basis of Genetic Variation in Polymorphic Self-incompatibility Genes Thanat Chookajorn,2004 *Genetic Variation in Drug Response* Tristan M. Victoroff,2010 **From GWAS to Epigenetics** Sophia C. Kamran,2013 **Translating inter-individual genetic variation to biological function in complex phenotypes** , Characterizing Rare Genetic and Epigenetic Variation in Complex Phenotypes Enrique Ivan Ramos,2015

Common genetic variability has failed to explain a large fraction of the heritability of complex disease In contrast rare genomic variation plays an important role in novel Mendelian phenotypes and complex traits and is the most common type of genetic variation in populations Tennesen et al 2012 International large sequencing initiatives have demonstrated that individual genetic variability exceeds prior estimates Fu et al 2013 and complex traits are mediated through additive genetic variation Hill WG 2008 Identification of these rare variant profiles is relevant to pinpoint potential disease related mechanisms in complex disease however due to the substantial number of possible targets in the genome and the variability between individuals this task was not easily attainable until the advent of next generation sequencing NGS and the subsequent ability to target any portion of the genome These analyses require processing extensive amounts of NGS data using robust and accurate bioinformatics To improve the ability to quantitatively characterize rare variability in complex phenotypes I have participated in the development of bioinformatic pipelines that enable detection of rare substitutions insertions and deletions from a few target genes in anonymous pools of DNA Vallania Ramos et al 2010 Vallania Ramos et al 2012 and extended the capability of these pipelines to allow highly sensitive specific and efficient quantification of rare variation from targeted capture of hundreds of candidate genes up to entire exomes in individually indexed bar coded NGS data Ramos et al 2012 I have demonstrated the utility of these algorithms through the identification of disease causing genes in complex phenotypes such as fatal extreme microcephaly Ramos et al 2014 and congenital anomalies of the kidney or lower urinary tract Chatterjee Ramos et al 2012 Additional types of rare epigenetic variation such as DNA methylation may further influence complex phenotypes and biological processes Hernandez et al 2012 Leung et al 2012 Bell et al 2011 However outside of costly whole genome bisulfite sequencing methods for large scale targeted DNA methylation analysis e g methylated DNA immunoprecipitation MeDIP reduced representation bisulfate sequencing RRBS methylation sensitive restriction enzyme MSRE digestion have demonstrated conflicting results To develop more robust bioinformatics for the quantification of rare epigenomic variation in complex traits I have developed a novel genome wide murine methylome hybridization capture array and adapted my previously established pipeline to enable highly sensitive methylation analysis Using this array and bioinformatic pipeline I have identified a discrete number of age and tissue specific methylation changes

in the mouse genome manuscript in preparation as well as identification of novel tissue specific DNA methylation patterns of genes involved in neurodevelopment Hing Ramos et al Taken together the goal of this study is to facilitate the quantification of different forms of rare variation which will lead to novel insights in complex phenotypes and diseases Immunological Significance of Genetic Variation in Structural Proteins and the Genetic Determinants for Cross Protection of Porcine Reproductive and Respiratory Syndrome (PRRS) Virus Won-Il Kim,2007 In conclusion genetic variation of PRRSV negatively impacts cross neutralization among PRRS viruses The similarity of specific amino acid determinants in GP3 GP5 and M proteins may significantly contribute to the level of cross protection between two viruses Detection of Genetic Variation in the Human [alpha] and [beta] Globin Loci Yan-Tat Liu,2006 The Distribution of Single Nucleotide Polymorphisms in Pyoderma Gangrenosum Heather Milliken Mercer,OhioLINK Electronic Theses and Dissertations Center,2013 Pyoderma gangrenosum PG is a rare inflammatory disease marked by reactive neutrophilic dermatosis While PG is non infectious it is painful only diagnosed by exclusion of other disorders Bhat 2012 and challenging to treat Thus there exists a need for discovery of biomarkers that can aid in faster and more reliable diagnosis Whole genome genotyping platforms such as Affymetrix SNP 6 0 chip offer rapid and comprehensive ways to survey patients genomes to identify shared genetic polymorphisms as part of the biomarker discovery In this work we test the hypothesis that PG is caused by genetic variants within the genome that 1 inhibit neutrophil clearance and immune inflammatory pathways and 2 that a combination of Single Nucleotide Polymorphisms SNPs across multiple loci in this pathway may contribute to this dysfunction We examined a list of 64 997 SNPs found in 9 831 genes that were shared across six PG patients These genes were examined to determine whether an over enrichment of immune and or inflammation and or apoptosis variants exists across patients using Gene Ontology tools Gene Ontology tools provided insight into the functional classifications of these genes and possible connections to PG etiology The genes found in common between the PG data set and those known to be active in apoptotic and immunity inflammatory pathways were further analyzed for the potential effects of genetic variation Those genetic variants located within exons and 5 and 3 untranslated regions in addition to those that cause missense or nonsense were viewed as the most likely to be related to PG as mutations in those regions are most likely to alter protein products In addition to the aforementioned genetic regions those SNPS located in non coding genic regions potentially regulatory as well as those from the inter genic regions were analyzed The latter category while currently lacking known functional annotations may provide insight into the genome function as putative regulatory regions and or non coding RNA as suggested by recent endeavors such as the ENCODE project Analyses resulted in the discovery of an over enrichment of SNPs within genes functioning within both apoptotic and immune inflammatory pathways within the PG Data Set in addition to SNPS located in genes that may be integral to the development of PG

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Table of Contents Hspi The Pogil Project Genetic Variation

1. Understanding the eBook Hspi The Pogil Project Genetic Variation
 - The Rise of Digital Reading Hspi The Pogil Project Genetic Variation
 - Advantages of eBooks Over Traditional Books
2. Identifying Hspi The Pogil Project Genetic Variation
 - Exploring Different Genres
 - Considering Fiction vs. Non-Fiction
 - Determining Your Reading Goals
3. Choosing the Right eBook Platform
 - Popular eBook Platforms
 - Features to Look for in an Hspi The Pogil Project Genetic Variation
 - User-Friendly Interface
4. Exploring eBook Recommendations from Hspi The Pogil Project Genetic Variation
 - Personalized Recommendations
 - Hspi The Pogil Project Genetic Variation User Reviews and Ratings
 - Hspi The Pogil Project Genetic Variation and Bestseller Lists
5. Accessing Hspi The Pogil Project Genetic Variation Free and Paid eBooks
 - Hspi The Pogil Project Genetic Variation Public Domain eBooks
 - Hspi The Pogil Project Genetic Variation eBook Subscription Services
 - Hspi The Pogil Project Genetic Variation Budget-Friendly Options
6. Navigating Hspi The Pogil Project Genetic Variation eBook Formats
 - ePub, PDF, MOBI, and More
 - Hspi The Pogil Project Genetic Variation Compatibility with Devices
 - Hspi The Pogil Project Genetic Variation Enhanced eBook Features
7. Enhancing Your Reading Experience
 - Adjustable Fonts and Text Sizes of Hspi The Pogil Project Genetic Variation
 - Highlighting and Note-Taking Hspi The Pogil Project Genetic Variation
 - Interactive Elements Hspi The Pogil Project Genetic Variation

8. Staying Engaged with Hspi The Pogil Project Genetic Variation
 - Joining Online Reading Communities
 - Participating in Virtual Book Clubs
 - Following Authors and Publishers Hspi The Pogil Project Genetic Variation
9. Balancing eBooks and Physical Books Hspi The Pogil Project Genetic Variation
 - Benefits of a Digital Library
 - Creating a Diverse Reading Collection Hspi The Pogil Project Genetic Variation
10. Overcoming Reading Challenges
 - Dealing with Digital Eye Strain
 - Minimizing Distractions
 - Managing Screen Time
11. Cultivating a Reading Routine Hspi The Pogil Project Genetic Variation
 - Setting Reading Goals Hspi The Pogil Project Genetic Variation
 - Carving Out Dedicated Reading Time
12. Sourcing Reliable Information of Hspi The Pogil Project Genetic Variation
 - Fact-Checking eBook Content of Hspi The Pogil Project Genetic Variation
 - Distinguishing Credible Sources
13. Promoting Lifelong Learning
 - Utilizing eBooks for Skill Development
 - Exploring Educational eBooks
14. Embracing eBook Trends
 - Integration of Multimedia Elements
 - Interactive and Gamified eBooks

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