CURRICULUM VITAE

PETKO M. PETKOV

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EDUCATION

1993, PhD, Higher Attestation Commission, Specialized Scientific Council of Molecular Biology and Genetics. Diploma for Candidate of Biological Sciences (Ph.D. equivalent). Thesis: Purification and Characterization of the Main Diaphorases from Several Drosophila Species. Mentor: Assoc. Prof. K. Ralchev, PhD.

1981-1986, **BS/MS**, University of Sofia, Bulgaria. Diploma for Higher Education (BS/MS equivalent) in Molecular and Functional Biology. Specialization: Genetics. Thesis: Cytogenetical and Electrophoretic Investigations on Lacerta vivipara Jacq. and Lacerta muralis Laur. from Bulgaria. Mentor: Assoc. Prof. R. Belcheva, PhD

TRAINING COURSES

January-April 1991. Institute of Bioorganic Chemistry, Moscow, Russia. Mentor: Prof. T.V. Ovchinnikova, PhD. Theme: Structural investigations of proteins**.**

September - December 1993. Free University Berlin, Institute of General Genetics. Mentor: Prof. G. Korge, PhD. Theme: Regulation of Gene Activity in Drosophila.

SCIENTIFIC AND TEACHING RECORD

June 2006 – present
Research Scientist. The Jackson Laboratory, Ken Paigen's lab. Topic: Chromosome-wide mapping of mouse recombination. Genetic control of hotspot activity. Recombination initiation complex and PRDM9 function in it.

March 2004 – June 2006
Associate Research Scientist. The Jackson Laboratory, Ken Paigen's lab. Topic: Investigation on recombination hotspots and LD blocks in the mouse genome.

July 2001-March 2004
Research Associate R&D. Technology Development Department, The Jackson Laboratory. Topic: Investigation on SNPs in different mouse strains.

March 1997-July 2001
Research Associate. Liver Research Center, Albert Einstein College of Medicine of Yeshiva University, Bronx, NY, 10461. Mentor: Prof. D.A. Shafritz, MD/PhD. Topic: Investigation of genes expressed in liver stem/progenitor cells.

March 1993-March 1997
Major Assistant Professor (Assistant Professor equivalent), Department of Genetics, Faculty of Biology, University of Sofia "St. Kliment Ohridski". Topic: Structure and expression of NAD(P)H oxidoreductase genes in Drosophila.

Courses taught
General Genetics (1993-1997, BS students, ~120 students per year)
Molecular Genetics (1994-1997, MS students, ~35 students per year)

Mentoring record
5 students with diploma work for MS graduation

RESEARCH HISTORY

University of Sofia, Bulgaria 1990-1997. NAD(P)H oxidoreductase genes in Drosophila. I started my research as an Assistant Professor in the early 1990's in Bulgaria as a *Drosophila* developmental biologist studying the role of NAD(P)H oxidoreductases. I purified two of these enzymes to homogeneity, studied their substrate specificity, spatial and temporal expression, raised antibodies against the purified proteins, and determined that they were products of different genes (*Biochem.Genet*., 1992). Subsequently, I sequenced peptides deriving from each of the two proteins, which helped us to clone the two genes. My colleagues and students later identified the genes as DLD (dihydrolipoamide dehydrogenase) and a new cytosolic NADH/NADPH dehydrogenase. It is important to note that this work was done at a time when Bulgaria was in a deep economic crisis complicated by hyperinflation, which resulted in total lack of research funding.

Albert Einstein College of Medicine, 1997-2001. Liver progenitor cells and liver repopulation. Because the research opportunitites in Bulgaria were so limited, I came to the U.S. in 1997, accepting a postdoctoral position in Dr. Shafritz' lab at the Albert Eindten College of Medicine, where I worked on liver development, characterization of stem/progenitor cells in rat embryonal liver and their potential for liver regeneration. My research there was instrumental for the overall progress of the lab in the following manner:

1) I developed a method for isolation of highly enriched fetal liver cells and successfully used them to repopulate damaged adult livers.

2) I created a subtracted library of genes highly expressed in fetal liver cells and determined their developmental expression.

3) I introduced the usage of microarrays at the lab and determined the developmental profile of gene expression in fetal rat liver.

Major contributions stemming from my postdoctoral research:

1) Hepatocytes and liver progenitor cell can repopulate damaged livers when endogenous hepatocytes are proliferationally impaired by retrorsine.

2) Thyroid hormone stimulates proliferation of transplanted liver cells.

3) Three subpopulations of fetal liver epithelial cells exist at ED 13-15.

4) These three types of cells differed in their ability to repopulate normal or damaged adult liver.

5) Fetal hepatoblasts undergo a major gene expression switch at ED16-17 leading to their differentiation.

6) Fetal liver cells continued to proliferate more than six months after transplantation into adult liver.

7) Transplanted fetal liver cells have stem cell properties and were able to differentiate both into hepatocytes and bile duct cells.

8) We found 643 genes induced in hepatoblasts compared to adult hepatocytes by suppression substraction hybridization and cloned the full-length transcripts of 31 of them using 5'- and 3'-RACE.

9) As a consequence of this project, we were one of the first laboratories to clone *PCSK9*, a gene coding for a protein involved in cholesterol homeostasis, and characterized its biochemical properties and subcellular distribution.

My work resulted in eight publication in high impact journals such as *Cancer Research, Hepatology, American Journal of Pathology, Biochemistry and Cell Biology*, and *Genomics*.

The Jackson Laboratory, 2001-2004. SNP genotyping platforms and panel development. Realizing that the future really lay in mammalian genetics, I applied for and obtained position here at JAX, initially working on development of a SNP genotyping platform and marker panels to be used for genetic quality control and association studies. My work on this field was highly successful, as the SNP genotyping panels we developed are still used at the lab, ten years after the initial work was done. We were the first to test a moderate number of markers on a high number of mouse strains representing different genetic backgrounds, enabling us to create a mouse family tree which resolved some of the outstanding controversies in the mouse strain phylogeny. The work on these panels was descrined in two papers, in *Genomics* (Petkov et al., 2004) and *Genome Research* (Petkov et al., 2004), the latter being cited more than 200 times as of present.

One unexpected result of my research on mouse strain phylogeny was the finding that classical mouse strains have unusually high levels of linkage disequilibrium, which was difficult to explain solely by their shared history. Trying to find an explanation to this finding led me to collaborate with Drs. Ken Paigen, Gary Churchill, and Joel Graber. Our analysis provided evidence for large-scale functional organization of mammalian chromosomes and was published in *PLoS Genetics* (Petkov et al., 2005). This collaboration was later extended and had the ultimate result of establishing the Center for Genome Dynamics at The Jackson Laboratory.

Ken Paigen lab, The Jackson Laboratory, 2004-2014. **Recombination hotspots and mechanisms of recombination initiation.** When I was offered the opportunity to move to full time genetics research in Ken Paigen's lab, studying mechanisms of genetic recombination, I accepted immediately and have focused my efforts in that area ever since. At that time, it was becoming increasingly evident that mammalian recombination was contained at short genomic regions, 1-2 kb long, termed hotspots. Since data about mouse hotspots were anecdotal, the first question we addressed was where were recombination hotspots positioned along the chromosomes. I receinved my first two grants, a project under CGD P50 and my own R01, to study chromosome-wide recombination patterns. We bred large progeny of C57BL/6J x CAST/Ei/J backcrosses and mapped recombination events separately in female and male meiosis, using reciprocal parental combinations. The results of this investigation were published in *PLoS Genetics* (Paigen et al., 2008). We found that:

1) Recombination rates were evolutionarily conserved on a regional scale, but not at the local level.

2) There was a clear negative-exponential relationship between the relative activity and abundance of hotspot activity classes, such that a small number of the most active hotspots account for the majority of recombination.

3) Females had higher overall recombination than males, although the sex ratio showed considerable regional variation.

4) Locally, entirely sex-specific hotspots were rare.

5) Analysis of reciprocal crosses indicated that parental imprinting has subtle effects on recombination rates.

6) Importantly, the initiation of recombination at the most active hotspot was regulated independently on the two parental chromatids.

This last conclusion emphasized the influence of genetic background on hotspot activity. We argued that if the local sequence has such strong effect on hotspot activity, there must be some *trans*-acting facror (presumably protein) that recognizes and binds to specific sequences to regulate hotspot activity. To test this hypothesis, we bred a congenic cross in which the sequence at a segment of Chr 1 where several hotspots were mapped was kept heterozygous B6/CAST, but the rest of the genome was entirely B6. We found

that, compared to the interstrain B6xCAST cross, some hotspots disappeared, new hotspots appeared, and the activity of other hotspots changed significantly. Mapping the trans-acting factor responsible for hotspot activation pinpointed a single hotspot regulator gene, *Prdm9*. This work was published in two consecutive papers in *PLoS Biology* (Parvanov et al., 2009) and *Science* (Parvanov et al., 2010), the latter accompanied by papers from two other groups who independently reached the same conclusion. Finding the role of *Prdm9* as hotspot regulator was widely recognized as the most significant breakthrough in recombination biology after cloning of *Spo11,* the gene whose product makes double-strand breaks, 13 years before that.

FUTURE RESEARCH PLANS

1. *What is the specificity of PDRM9 binding to DNA?* Zinc finger proteins play key roles in nucleic acid binding and protein-protein interactions. About 4% of all human or mouse genes code for zinc finger proteins, and 3% have long zinc finger array (6 or more fingers). PRDM9, with its DNA binding domain of 8-15 zinc fingers in different human and mouse alleles, is a representative of this large group of genes.Recently, we characterized the DNA binding sites of four hotspots, each activated by a single PRDM9 allele – either Dom2 found in C57BL/6J (12 fingers) or Cst present in CAST/EiJ (11 fingers) – and found that all binding sites required the full array of 11 or 12 contiguous fingers, depending on the allele. I am now extending this study at the genome-wide level using an approach we called Affinity-seq. *This work is supported by funding from CGD P50 awarded to my Project B.*

2. *How is PRDM9 posttranslationally modified to activate its DNA binding function?* This is an important question because it will elucidate the mechanisms of recombination initiation at the onset of meiosis, and might have implications for the carcinogenic potential of ectopically expressed PRDM9. A surprizing finding of our study of PRDM9 binding to DNA was that *E.coli* expressed and purified full length PRDM9 did not bind DNA. We were able to locate the structural feature responsible for this regulation. It turned out to be a separate zinc finger, positioned 100 AA upstream of the array of tandemly repeated zinc fingers with a DNA binding activity. I plan to use mutational analysis in bacterial and mammalian cells, changing the amino acid composition of the finger, potential modification sites, and the zinc finger structure, to determine what is the nature of this regulating change – is it imposed in cytoplasm or after its importation into the nucleus, and how it affects other activities of PRDM9 such as histone methylation and protein binding. *This work is supported by funding from CGD P50 awarded to my Project B.*

3. *We have found that PRDM9 functions as part of a Recombination Initiation Complex; what is the molecular composition of this complex and what are its functions at the onset of meiosis*? Answering these questions will be as important as having identified PRDM9 itself. The is an increasing body of evidence that PRDM9 has additional functions in meiosis, ensuring proper initiation of recombination by possibly bringing potential double-strand break sites to chromosomal axes and enabling homolog search. We detected more than 20 strong binders and were able to provide evidence that at least five of them bind to PRDM9 in mouse spermatocytes. Using them, I plan to further study the function of these proteins in meiosis, their interaction with PRDM9, and the spatial and temporal dynamics of their complexes in recombination initiation. *I have submitted an R01 grant proposal to fund this project.*

4. *How is sex specificity of recombination established*? Recombination hotspots are active in both male and female meiosis, however, their recombination frequencies differ

along the chromosomes. We have extensive data about the early events in male meiosis including the frequencies of PRDM9 binding H3K4 trimethylation and double strand break formation in male meiosis. I plan to evaluate recombination initiation frequencies in female meiosis by doing ChIP-seq for H3K4me3, marking PRDM9-dependent hotspots, and with ChIP-seq for H3K9me2/H3K9me3 marks imposed by EHMT2, in mice differing only by their *Prdm9* allele on the same genetic background, and in *Ehmt2* KO mice bred into the same PRDM9 backgrounds. *This work is supported by my current R01 grant.*

5. Is PRDM9 involved in carcinogenesis? This question extends the scope of my work in different direction with potentially high impact. In normal tissues, *Prdm9* expression is restricted only to germ cells, and only in a narrow time window at the onset of meiotic prophase I. Recent findings have pointed to the possibility that this gene might be involved in tumorigenesis. Familial studies have implicated some of its rare alleles in acute lymphocytic leukemia (ALL); whole-genome sequencing studies have found mutations of this gene in several types of tumors, and gene expression studies have detected its ectopic expression in kidney, lung, liver and colon cancer at statistically significant levels. I propose to study whether *Prdm9* plays a role in tumorigenesis by using two well-known genetic models of carcinogenesis. *This work is supported by an internal Cancer Center pilot funds.*

6. *What is the role of genetic background on epigenetic landscape of mouse liver and how does it change after corticosteroid stimulation?* I am a joint PI on this project which was recently funded by The Jackson Laboratory Director's Fund. In this project we propose to use a panel of genetically diverse, inbred mouse strains to test the hypothesis that molecular epigenetics are under genetic control in a robust, replicable experimental system. Our studies will establish the extent of genetic variability in molecular epigenetics, thus enabling future studies in transgenerational inheritance of epigenetic marks, disease risk, aging, and many other fields.

RESEARCH GRANTS

Ongoing Research Support

5 P50 GM076468-07 Churchill (PI) 7/15/11-6/30/16
NIH/NIGMS
Center for Genome Dynamics - Project B: Systems Genetics of Meiotic Recombination
The goal of this project is to identify epistatic genes that interact in networks to control the location and activity of recombination hotspots.
Role: Project Leader

2 R01 GM078452-06A1 Petkov (PI) 8/1/12-4/30/16
NIH/NIGMS
Sex-Specific Regulation of Meiotic Recombination Hotspots
The goal of this project is to determine the factors regulating sex specificity of meiotic recombination, both in its regional distribution along the chromosomes and the activity of sex-specific recombination hotspots.
Role: Principal Investigator

TJL-CCSG-Pilot-PMP01 Petkov (PI) 12/01/12-10/30/13
The Jackson Laboratory Cancer Center
The Jackson Laboratory Cancer Center, 2012-2013 Pilot Feasibility Studies - Does PDRM9 Play a Role in Cancer?
The goal of this project is to determine whether the expression of Prdm9 influence the incidence of tumor formation and progression in Apc+/- mice.
Role: Principal Investigator

TJL DIF FY14 GWC Collab Carter (PI) 05/01/14 - 04/30/16
The Jackson Laboratory Director's Innovation Fund
Genetics of Molecular Epigenetics
The goal of this project is to understand the consequences of genetic variation on genome-wide transcript regulation
Role: Joint PI

Completed Research Support

5 R01 GM078452-05 Petkov (PI) 3/1/07-7/31/12
NIH/NIGMS
Chromosome-wide Mapping of Recombinational Activity
The goal of this project is to achieve understanding of the principles that determine the existence and activity of recombinational hot spots across the genome.
Role: Principal Investigator

3 R01 GM083408-02S1Z Paigen (PI) 8/17/09-7/31/11
NIH/NIGMS
Transacting Genes Regulating Recombination Hotspot Activities - ARRA Administrative Supplement
The aim of this project is to search for genes suppressing and modulating recombination activity
Role: Co-Investigator

5 P50 GM076468-05 Churchill (PI) 4/1/06-7/14/11
NIH/NIGMS
Genome Dynamics: Evolution, Organization and Function - Project 5: Chromosome-wide Mapping of Recombinational Activity
The goal of this project is to create high-resolution genetic maps of Chr 11 in six mouse crosses and study how genetic background, sex and imprinting affect hotspot positioning and usage. This study will provide the necessary information to start a comprehensive mapping of genes regulating recombination activity.
Role: Project Leader

5 P50 GM076468-05 Churchill (PI) 4/1/06-7/14/11
NIH/NIGMS
Genome Dynamics: Evolution, Organization and Function - Core 3: Molecular Biology
The main task of this core is to provide high-quality DNA and RNA samples for Projects 1, 3 and 5, and to perform expression studies using Affymetrix microarrays and real-time PCR..
Role: Core Leader

2 P40 RR016049-06A1 Donahue (PI) 9/1/06-6/30/11
NIH/NCRR
Special Mouse Strains Resource
The major goal of this project is to maintain, characterize and distribute recombinant inbred and consomic strains of mice.
Role: Research Scientist

5 R21 AR055181-02 Petkov (PI) 7/1/08-5/31/11
NIH/NIAMS
Search for Genes Involved in Arthritis Pathogenesis
The specific aims of this project are: (1) Map the region on chromosome 6 responsible for arthritis resistance using a backcross between B6-Chr6PWD and B6, and select candidate genes. (2) Evaluate arthritis susceptibility and clinical parameters of arthritis development using the entire set (n=28) of ChrNPWD consomics and the serum-transfer model of arthritis.

Role: Principal Investigator

5 R01 GM078643-03 Paigen (PI) 1/1/06-12/31/10
NIH/NIGMS
Genomic Organization of Recombination Hot Spots
The major goal of this grant is obtain a detailed map at one Kb resolution of all recombinational hotspots, including their sex and haplotype specificity, for five different five Mb regions chosen for their particular biological interest on separate chromosomes using 6000 meioses.
Role: Co-Investigator

AG-SS-1631-06 Paigen (PI) 10/2/06-10/1/10
Ellison Medical Foundation
New Genetic Strategies in the Study of Aging
The goal of this project is the development of new genetic strategies in the study of human aging using RNAi directed mutagenesis in somatic cell cultures to identify previously unknown genes participating in the aging process.
Role: Co-Principal Investigator

MEMBERSHIPS AND HONORS

2008 – Member of International Mammalian Genome Society
1999 – Member of American Association for Advancement of Science
1986 – Magna Cum Laude, MS graduation

Professional Services

2004	Ad hoc reviewer, Trends in Biotechnology
2005	Ad hoc reviewer, Clinical Chemistry, Comparative Medicine, PLoS Genetics
2006	Ad hoc reviewer, PLoS Genetics
2008	Ad hoc reviewer, Experimental Hematology
2009	Ad hoc reviewer, PLoS Genetics, Genome Research
2010	Ad hoc reviewer, Mutation Research, Genetics, BMC Bioinformatics
2011	Ad hoc reviewer, BMC Genomics, Animal Genetics, Grant Reviewer for Israel Science Foundation, Genetics, PLoS Genetics
2012	Ad hoc reviewer, PLoS Genetics, Am.J.Human Genetics, J. Heredity
2013	Ad hoc reviewer, Genetics, Proc. Royal Society
2013	Study section member, Genomic Variation and Evolution section, NIGMS, NIH
2014	Study section member, Genes, Genomes and Genetics section, NIGMS, NIH

PUBLICATION LIST

Papers:

1. Baker CL, Kajita S, Walker M, **Petkov PM**, Paigen K. (2014). PRDM9 binding organizes hotspot nucleosomes and limits Holliday junction migration. Genome Res. 2014 Mar 6. 24(5):724-732. PMID: 24604780.
2. Billings T, Parvanov ED, Baker CL, Walker M, Paigen K, **Petkov PM**. (2013). DNA binding specificities of the long zinc finger recombination protein PRDM9. Genome Biol. 2013 Apr 24;14(4):R35. PMCID: 23618393.
3. Paigen K, **Petkov P**. 2012. Meiotic DSBs and the control of mammalian recombination. Cell Res 22(12):1624-1626. PMCID: Commentary
4. Billings, T., Sargent, E.E., Szatkiewicz, J.P., Leahy, N., Kwak, I.-Y., Bektassova, N., Walker, M., Hassold, T., Graber, J.H., Broman, K.W., **Petkov, P.M.** (2010)**.** Patterns of recombination activity on mouse chromosome 11 revealed by high resolution mapping. **PLoS One, 5**(12), e15340.

5. Paigen, K., **Petkov, P.** (2010). Mammalian recombination hot spots: properties, control and evolution. **Nat Rev Genet, 11(3),** 221-33.
6. Parvanov ED, **Petkov PM**, Paigen K. (2010). *Prdm9* controls activation of mammalian recombination hotspots. **Science**, **327**(5967), 835.
7. Parvanov ED, Ng SHS, **Petkov PM**, Paigen K. 2009. Trans-regulation of mouse meiotic recombination hotspots by *Rcr1*. **PLoS Biol, 7**, e1000036.
8. Ng SH, Madeira R, Parvanov ED, Petros LM, **Petkov PM**, Paigen K. (2009). Parental origin of chromosomes influences crossover within the Kcnq1 transcriptionally imprinted domain of *Mus musculus*. **BMC Molec Biol**, **10**, 43-52.
9. Ng SH, Maas SA, **Petkov PM**, Mills KD, Paigen K. (2009). Co-localization of somatic and meiotic double strand breaks near the *Myc* oncogene on mouse chromosome 15. **Genes Chromosomes Cancer 48**(10):925-930.
10. Ng SH, Parvanov E, **Petkov PM,** Paigen K. (2008). A quantitative assay for crossover and noncrossover molecular events at individual recombination hotspots in both male and female gametes. **Genomics**. **92(4):**204-9.
11. Paigen K, Szatkiewicz JP, Sawyer K, Leahy N, Parvanov ED, Ng SH, Graber JH, Broman KW, **Petkov PM.** (2008). The recombinational anatomy of a mouse chromosome. **PLoS Genet**. 4(7):e1000119.
12. **Petkov PM,** Broman KW, Szatkiewicz JP, Paigen K. (2007). Crossover interference underlies sex differences in recombination rates. **Trends Genet**. 23(11):539-42.
13. **Petkov PM,** Graber JH, Churchill GA, DiPetrillo K, King BL, Paigen K. (2007). Evidence of a large-scale functional organization of Mammalian chromosomes. **PLoS Biol.** 2007 May;5(5):e127; author reply e128.
14. Grozdanov PN, **Petkov PM**, Karagyozov LK, Dabeva MD. (2006). Expression and localization of PCSK9 in rat hepatic cells. **Biochem Cell Biol. 84(1):**80-92.
15. Ishimori N, Li R, Walsh KA, Korstanje R, Rollins JA, **Petkov P**, Pletcher MT, Wiltshire T, Donahue LR, Rosen CJ, Beamer WG, Churchill GA, Paigen B. (2006). Quantitative Trait Loci That Determine BMD in C57BL/6J and 129S1/SvImJ Inbred Mice. **J Bone Miner Res. 21(1):**105-12.
16. Graber JH, Churchill GA, Dipetrillo KJ, King BL, **Petkov PM,** Paigen K. (2006). Patterns and mechanisms of genome organization in the mouse. J Exp Zoolog A Comp Exp Biol. 305(9):683-8. Review.
17. **Petkov PM,** Graber JH, Churchill GA, Dipetrillo K, King BL, Paigen K. (2005). Evidence of a Large-Scale Functional Organization of Mammalian Chromosomes. **PLoS Genet., 1(3):**e33.
18. Kelmenson, P.M**, P.M. Petkov,** X. Wang, D.C. Higgins, B.J. Paigen, K. Paigen (2005). A Torrid Zone on Mouse Chromosome 1 Containing a Cluster of Recombinational Hotspots. **Genetics, 169 (2),** 833-841.
19. **Petkov**, **P.M.** Yueming Ding, Megan A. Cassel, Weidong Zhang, Gunjan Wagner, Evelyn E. Sargent, Steven Asquith, Victor Crew, Kevin A. Johnson, Phil Robinson, Valerie E. Scott, Michael V. Wiles (2004). An Efficient SNP System for Mouse Genome Scanning and Elucidating Strain Relationships. **Genome Research, 14,** 1806-1811.
20. **Petkov, P.M.,** Megan A. Cassell, Evelyn E. Sargent, Charles J. Donnelly, Phil Robinson, Victor Crew, Steven Asquith, Raymond Vonder Haar, Michael V. Wiles (2004). Development of a SNP Genotyping Panel for Genetic Monitoring of the Laboratory Mouse. **Genomics 83(5):902-911.**
21. **Petkov**, **P.M.,** Jiri Zavadil, David Goetz, Tearina Chu, Robert Carver, Charles E. Rogler, Erwin P. Bottinger, David A. Shafritz, Mariana D. Dabeva (2004). Gene Expression Pattern In Hepatic Stem/Progenitor Cells During Rat Fetal Development Using cDNA Microarrays**. Hepatology 39(3):617-627.**
22. Sandhu, J., **P.M. Petkov,** M.D. Dabeva, and D.A. Shafritz (2001). Stem Cell Properties and Repopulation of the Rat Liver by Fetal Liver Epithelial Progenitor Cells. **Am. J. Pathol., 159 (4),** 1323-1334.

23. **Petkov, P.M.**, K. Kim, J. Sandhu, D.A. Shafritz, and M.D. Dabeva (2000). Identification of differentially expressed genes in epithelial stem/progenitor cells of fetal rat liver. **Genomics, 68 (2),** 197-209.
24. Dabeva, M.D., **P.M. Petkov,** J. Sandhu, R. Oren, E. Laconi, E. Hurston, and D.A. Shafritz (2000). Proliferation and differentiation of fetal liver epithelial progenitor cells after transplantation into adult rat liver. **Am.J.Pathol., 156(6),** 2017-2031**.**
25. Oren, R., M.D. Dabeva, A.N. Karnezis, **P.M. Petkov**, R. Rosencrantz, J.P. Sandhu, S.F. Moss, S. Wang, E. Hurston, E. Laconi, P.R. Holt, S.N. Thung, L. Zhu, D.A. Shafritz (1999). Role of thyroid hormone in stimulating liver repopulation by transplanted hepatocytes. **Hepatology, 30,** 903-913.
26. Oren, R., M.D. Dabeva, **P.M. Petkov,** E.Hurston, E. Laconi, D.A. Shafritz (1999). Restoration of serum albumin levels in Nagase analbuminemic rats by hepatocyte transplantation. **Hepatology,29,** 75-81.
27. Dabeva, M.D., E. Laconi, R. Oren, **P.M. Petkov**, E. Hurston, D.A. Shafritz (1998). Liver regeneration and α-fetoprotein messenger RNA expression in the retrorsine model for hepatocyte transplantation. **Cancer Res., 58,** 5825-5834.
28. Dabeva, M.D., Laconi, E., Oren, R., **Petkov, P.,** Hurston, E., Shafritz, D.A. (1998). AFP mRNA expression in regenerating liver: Dedifferentiation of hepatocytes vs maturation of liver progenitor cells. **FASEB J.,** 12(4), A468.
29. Ralchev, K.H., **P.M. Petkov,** A.V. Valevska, (1994). A new method for purification of diaphorase-1 and diaphorase-2 from Drosophila virilis, and structural investigations. **C.R. Acad. Bulg. Sci., 47**, 3, 91-93.
30. **Petkov, P.M**., A.V. Valevska, K.H. Ralchev (1994). Comparative characterization of the molecular weights of diaphorase-1 and diaphorase-2 from several Drosophila species. **C.R. Acad. Bulg. Sci., 47**, 6, 69-71.
31. Ralchev, K.H., **P.M. Petkov**, B.C. Dunkov (1992). Purification and characterization of diaphorases from some Drosophila species. **Biochem. Genet., 30,** (5-6), 305-315.
32. **Petkov, P.,** K. Ralchev, B. Dunkov (1990). Immunochemical and electrophoretic characterization of diaphorase-2" from three Drosophila species. **Genetics and Breeding, 23,** 285-291 (in Bulgarian).
33. Belcheva, R.G., **P.M.Petkov**, I.R.Kehaiov (1989). Cytogenetic and isoenzyme investigations on two lizard species from genus Lacerta - Lacerta vivipara Jacq. and Lacerta muralis Laur. (Reptilia, Lacertidae). **Acta Zoologica Bulgarica, 37,** 34-42 (in Bulgarian).
34. Belcheva, R.G., V.I. Bisserkov, H.L. Ilieva, V.A. Beshkov, **P.M. Petkov** (1986). Karyological studies on Lacerta vivipara (Jacq.) Collected in Bulgaria. **Cytologia, 51,** 567-570.
35. Belcheva,R.G., V.I. Bisserkov, H.L. Ilieva, V.A. Beshkov, **P.M.Petkov** (1984). A comparative study of the karyotype of eight lizard species from genus Lacerta (Sauria, Lacertidae). **Third Natonal Conference of Cytogenetics, vol. II,** 436-449 (in Bulgarian).

Scholarly books:

1. Fox, R.R., Wiles, M.V., and **Petkov, P.M.** (2007). Chapter 8**,** Genetic monitoring. In: Mouse in Biomedical Research. Fox J, Barthold S, Davisson MT, Newcomer C, Quimby F, Smith A (eds.), Elsevier Press.
2. Ralchev, K.H., **Petkov, P.M.,** Harizanova, N.T., Gueorguieva, T.G., Dunkov, B.C., Ivanova, P.M., Modreva, M.M. A guide for Demonstrations in Gene Engineering. Sofia University, Sofia, Bulgaria, 1997 (in Bulgarian).

Scientific Meetings Participations:

1. **Petko M. Petkov**, Boucher, James, Baker, Christopher L., Billings, Timothy, Sargent, Evelyn, Parvanov, Emil D., Paigen, Kenneth (2013). The Y-chromosome histone demethylase KDM5d modifies recombination hotspot activity. **27th International Mammalian Genome Conference**, 15–18 September 2013, Salamanca, Spain
2. Petkova, Pavlina M.; Baker, Christopher L.; Walker, Michael; **Petkov, Petko M**.; Paigen, Kenneth (2013). Suppression and quantitative control of meiotic recombination hotspot activity. **27th International Mammalian Genome Conference**, 15–18 September 2013, Salamanca, Spain

3. Baker, Christopher L.; Walker, Michael; Kajita, Shimpei; **Petkov, Petko M**.; Paigen, Kenneth (2013). PRDM9-dependent modification organizes hotspot chromatin structure. **27th International Mammalian Genome Conference**, 15–18 September 2013, Salamanca, Spain.
4. Pavlina M. Ivanova, Christopher E. Baker, **Petko M. Petkov**, Ken Paigen (2012). Initiation of meiotic recombination in humans by direct binding of PRDM9A to hotspots. **26th International Mammaliand Genome Conference**, October 23-25, St Pete's Beach, FL, USA
5. Timothy Billings, Emil D. Parvanov, Christopher E. Baker, Michael Walker, Kenneth Paigen, **Petko M. Petkov** (2012). DNA Binding Specificities of the Zinc Finger Recombination Protein PRDM9. **26th International Mammaliand Genome Conference**, October 23-25, St Pete's Beach, FL, USA
6. Cristopher L. Baker, Michael Walker, **Petko M. Petkov**, Kenneth Paigen (2012). Specificity of PRDM9 DNA binding determines Histone H3 Lysine 4 trimethylation at human hotspots. 11th Gordon Research Conference on Meiosis, June 2-6, New London, NH.
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