GOVINDA LENKA, *M.Sc., Ph.D.*

Assistant Professor, Dept. of Biotechnology, M.R PG College, Andhra University.

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Profile

Detail-oriented and performance driven professional, poised to utilize gained knowledge and experience in, cancer genomics, cell and molecular biology, in-vitro and in-vivo experimentation to further my career in research and development in the field of Life sciences and Biotech. Organized and resourceful with effective project management, time handling and problem solving skills. Collaborative team player with strong interpersonal and communication skills, adept in establishing and maintaining positive working relationships with diverse individuals as well as working on own initiative.

Key Skills and Experience

Molecular and Cell Biology

Proficient in Animal cell culture, Transformation, Transfection, Gene expression arrays, PCR, RT-PCR &qPCR, DNA sequencing, Bisulfite sequencing, Pyrosequencing, Gene knockdown, Gene knockout mouse, DNA cloning, EMSA, Immunohistochemistry, EMSA, Gel electrophoresis, Western blot, Genomic DNA isolation and purification, Tissue RNA isolation and purification, Co-immunoprecipitation, GST-Pulldown assay, Functional assays including Cell viability assay, Migration and Invasion assays, Apoptosis assays, Clonogenic assay, Cell cycle analysis, the conduct of Microscopy, ELISA and FACS.

Computational Analysis

Analyzing the Homology and Phylogeny of genes, TFBS and RE sites, CpG Islands, Phosphorylation sites, Design of primers, Methprimers & Expression system, Expression & Methylation array data analysis: Principal component analysis, retrieval of Volcano plots & Starburst plot, Hierarchical cluster analysis, Ingenuity pathway analysis, Kaplan–Meier survival analysis.

Teaching Skills

During doctoral study, I taught Molecular Biology and Polysaccharides for Biomass Energy courses for the master and undergraduate students of National Taipei University of Technology.
In addition, assisted lab members with the troubleshooting of conceptual, technical and experimental queries. Currently, I am teaching Genetic Engineering, Environmental Microbiology and their lab activities for the Master students.

Technical Skills

Highly knowledgeable in utilizing diverse software such as MS Office, ImageJ, GraphPad, and EndNote. Interpretation and analysis of the results using statistical tests, protocol writing and record keeping.

Work Ethics and Professionalism
Practices professional and quality standards at all times. Punctual with good time management skills. Able to complete tasks within time and budgetary limits.

Professional Experience

Assistant Professor, 2017 – Till Date
Dept. of Biotechnology,
M.R PG College, Phool bagh, 535002
Andhra University.
Subjects Taught: Genetic Engineering and Environmental Microbiology

Post-Doctoral Fellow 2014 – 2016
Supervisor: Prof. Liang-Chuan Lai **&** Eric Y. Chaung,
National Taiwan University.
Area of Research: Lung Cancer Genomics.

Project Associate,
Prof. Nitish R Mahapatra, 2007 –2009
Dept. of Biotechnology,
Indian Institution of Technology Madras, India
Field of Study: Cardiovascular Genetics

Education

2009-2014 **Ph.D.** National Taipei University of Technology, Taiwan
Functional Cancer Genomics
Dissertation Title: Characterization of biomarkers in cancer and Development of nanoparticle based PCR platform; **GPA:** 4/4
Adviser: Dr. Wen-Hui Weng

2004-2006 **M.S.** Pondicherry Central University
Life Sciences (Advanced Biology)
Dissertation Title: Study on the incidence of bacteria in the urinary tract Infections; **CGPA:** 7.54/10

2001-2004 **B.S.** Silver Jubilee Govt. Degree College (APRDC), Sri Krishnadevaraya University.
Triple Major: Microbiology, Botany and Chemistry; **Percentage:** 69.5

1999-2001 **Intermediate (10+2).** Andhra Pradesh Social Welfare Residential Junior College, Duppalavalasa, Srikakulam (APSWRJC).
Subjects: English, Telugu, Botany, Zoology, Physics and Chemistry;
Percentage: 85.8

1999 **SSC (10th).** Z.P.H School, Alugolu, Vizianagaram, SSC Board, Andhra Pradesh
Subjects: English, Telugu, Hindi, Mathematics, Science and Social studies
Percentage: 79.5

Projects in Progress: Post-Doctoral Research

- Molecular characterization to uncover the interacting proteins associated with *SEMA5A* function in lung cancer.
- Creation of *SEMA6A* Knockout mouse model to uncover the functional significance of gene in lung cancer.

Publications

- **Lenka G,** Ko PH, Tsai MH, Lai LC. Integrated analysis of gene expression and methylation reveals tumor suppressive role of semaphorin 5A in lung cancer cells, 2017 (**In preparation**).
- **Lenka G,** Tsai MH, Lin HC, Hsiao JH, Lee YC, Lu TP, Lee JM, Hsu CP, Lai LC, Chuang EY. Identification of methylation-driven differentially expressed *STXBP6* as a novel biomarker in lung adenocarcinoma. **Scientific Reports, 2017; 7: 42573**. **(SCI, IF: 5.228)**
- **Lenka G,** Tsai MH, Hsiao JH, Lai LC, Chuang EY. Overexpression of methylation-driven *DCC* suppresses proliferation of lung cancer cells. **Translational Cancer Research,** 2016; 5(2):169-175. **(SCI, IF: 1.757)**
- **Lenka G**, Weng WH, Chen YT, Chuang CK, Pang ST. Aberrant expression of *PRAC* gene in prostate cancer. **International Journal of Oncology**, 2013; 43(6):1960-6. **(SCI, IF: 3.018)**
- Yeh CN, Weng WH, **Lenka G**, Tsao LC, Chiang KC, Pang ST, Chen TW, Jan YY, Chen MF. cDNA microarray profiling of rat cholangiocarcinoma induced by thioacetamide. **Molecular Medicine Reports,** 2013; 8(2):350-60. **(SCI, IF: 1.559)**
- **Lenka G**, Weng WH. Nanosized particles of titanium dioxide specifically increase the efficiency of conventional polymerase chain reaction. **Digest Journal of Nanomaterials and Biostructures,** 2013; 8(4):1435-1445. **(SCI, IF: 1.123)**

- Weng WH, Yeh CN, Cheng YF, **Lenka G**, Liaw YW. Phosphorylated T567 ezrin is associated with merlin expression in KIT-mutant gastrointestinal stromal tumors. **Molecular Medicine Reports.** 2012; 5(1):17-21. **(SCI, IF: 1.559)**

- Sonawane PJ, Sahu BS, Sasi BK, Geedi P, **Lenka G**, Mahapatra NR. Functional promoter polymorphisms govern differential expression of HMG-CoA reductase gene in mouse models of essential hypertension. **PLoS One,** 2011; 31; 6(1):e16661. **(SCI, IF: 3.057)**

Conference Proceedings

- Hwa KY, Subramani B, **Lenka G**, Chang SM. Sequence motif analysis of phycobiliproteins in Cyanobacterial genome. 2011 International Conference on Bioscience, Biochemistry and Bioinformatics; ***IPCBEE*** (2011), **Singapore.**

- **Lenka G,** Weng WH, Hwa KY. Computational Analysis: Towards a better knowledge of the molecular evolution of phosphoenolpyruvate carboxylase among Flaveria species. Convergence of Biomedical Science and Information Technologies; **ISBN: 978-1-4244-7584-1.** ***IEEE Xplore*** **(**2010), **Cebu, Philippines (Engineering Indexed Article)**.

- Mahapatra NR, Sonawane PJ, Sahu BS, **Lenka G,** Sasi BK. Molecular basis of altered expression of the HMG-CoA reductase gene in genetically hypertensive mice. **Int. J. Cardiol.137: S107** (2009), Beijing, **China**.

Poster Presentations

- **Lenka G,** Weng WH. Comparative analysis of TiO2 effect on DNA amplification reactions: in vitro. 2013 Annual Meeting of the Polymer Society, National Chung Cheng University (2013), Chiayi, Taiwan.

- Weng WH, Yeh CN, Chen TW, Jan YY, Chen MF, **Lenka G,** Pang ST. Gene expression profiling of cholangiocarcinoma induced by thioacetamide in a rat model. XGen Congress & Expo (2012), San Diego, CA.

- **Lenka G,** Weng WH, Chuang CK, Pang ST. Aberrant expression of PRAC gene in prostate cancer. Federation of Asian and Oceanian Biochemists and Molecular Biologists (FAOBMB) Conference (2011), Singapore.

- Weng WH, Yeh CN, Cheng YF, **Lenka G,** Liaw YW. The Role of Various hosphorylations of Ezrin and Merlin in KIT Mutant Gastrointestinal Stromal Tumors. The 25th Joint Annual Conference of Biomedical Sciences (2010), Taipei, Taiwan.

- **Lenka G,** Sahu BS, Sonawane PJ, Sasi BK, Mahapatra NR. Functional promoter polymorphisms regulate the expression of the HMG-CoA reductase gene in mouse.

77th Annual meeting of Society of Biological Chemists (India), Indian Institute of Technology Madras (2008), India.

- Sonawane PJ, Sasi BK, Sahu BS, **Lenka G,** Mahapatra NR. Transcriptional regulation of the human renalase gene. 77th Annual meeting of Society of Biological Chemists (India), Indian Institute of Technology Madras (2008), India.

- Sasi BK, Sonawane PJ, Sahu BS, **Lenka G,** Mahapatra NR. Molecular basis of differential expression of the Hspa1a gene in mouse models of human essential hypertension. 77th Annual meeting of Society of Biological Chemists (India), Indian Institute of Technology Madras (2008), India.

- Sahu BS, Geedi P, **Lenka G,** Sonawane PJ, Sasi BK, Mahapatra NR. Identification and characterization of gene polymorphisms in a Mouse model of Hypertension" in Genomics model organisms and diseases. An international satellite meeting to the 13th Human genome meeting, National center for Biological sciences (NCBS) (2008), Bangalore, India.

Travel Grants

- Received financial support from National Taipei University of Technology, Taiwan to attend the International Conference, Federation of Asian and Oceanian Biochemists and Molecular Biologists (FAOBMB) (2011), Singapore.

- Received financial support from National Science Council, Taiwan to attend the International Conference, Convergence of Biomedical Science and Information Technologies, IEEE Xplore (2010), Cebu, Philippines.

Research Fellowship

- Awarded International student scholarship offered by National Taipei University of Technology for the academic year of 2012 September to 2013 August.

- Awarded **International Scholarship offered by Taiwan Government** (Ministry of Education and National Science Council) for doctoral studies (Aug2009 to Aug2012).

- Received assistantship from (Jan 2009 – Aug2009) the Department of Biotechnology (DBT), New Delhi, Govt. of India, funded project to IITM.

- Received assistantship from (Nov2007 – Dec2008) Indian Institute of Technology Madras (IIT-M).

Academic Achievements

- Received **Merit Scholarship** for securing the first rank in all India entrance test (2004) for admission into Pondicherry University.
- Qualified in the All India Entrance exam conducted for the admission to Advanced PG Diploma in Bioinformatics by Pondicherry University.
- Secured admission into the Silver Jubilee Govt. Degree College (APRDC) by state-level competitive entrance exam.
- Received **Prathibha Scholarship** awarded by Ministry of Women and Social welfare department, Andhra Pradesh Government, India for the best performance in intermediate education (Biology, Physics and Chemistry, Languages) .
- Secured admission into APSWRJC for intermediate education by zonal (North Andhra) level entrance exam.

Personal Details

Name	**:** Govinda Lenka
Father's Name	**:** Mahalakshmi Naidu
Father's Profession	: Farmer
Date of Birth	**:** 15-07-1984
Marital Status	**:** Married
Wife	**:** Chittimma Chandaka (M.S in Organic Chemistry & BME)
Children	**:** Induja and Dusyanth
Languages Known	**:** English, Telugu, Hindi, Tamil and Basic Chinese
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References

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Identification of Methylation-Driven, Differentially Expressed *STXBP6* as a Novel Biomarker in Lung Adenocarcinoma

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DNA methylation is an essential epigenetic marker associated with the silencing of gene expression. Although various genome-wide studies revealed aberrantly methylated gene targets as molecular biomarkers for early detection, the survival rate of lung cancer patients is still poor. In order to identify methylation-driven biomarkers, genome-wide changes in DNA methylation and differential expression in 32 pairs of lung adenocarcinoma and adjacent normal lung tissue in non-smoking women were examined. This concurrent analysis identified 21 negatively correlated probes ($r \leq -0.5$), corresponding to 17 genes. Examining the endogenous expression in lung cancer cell lines, five of the genes were found to be significantly down-regulated. Furthermore, in tumor cells alone, 5-aza-2′-deoxycytidine treatment increased the expression levels of *STXBP6* in a dose dependent manner and pyrosequencing showed higher percentage of methylation in *STXBP6* promoter. Functional analysis revealed that overexpressed *STXBP6* in A549 and H1299 cells significantly decreased cell proliferation, colony formation, and migration, and increased apoptosis. Finally, significantly lower survival rates ($P < 0.05$) were observed when expression levels of *STXBP6* were low. Our results provide a basis for the genetic etiology of lung adenocarcinoma by demonstrating the possible role of hypermethylation of *STXBP6* in poor clinical outcomes in lung cancer patients.

Lung carcinoma is one of the most commonly diagnosed cancer types, and it is characterized by poor survival rates. According to recent global cancer statistics, it accounts for 18% of all cancer-related deaths worldwide[1]. Despite decades of research efforts to improve the clinical outcomes of lung cancer patients, the overall survival rates remain dismal. The mortality rates of lung cancer were the highest in Taiwan[2,3].

A wide variety of risk factors, such as genetic, epigenetic, and environmental factors, may cause lung cancer. Nearly 70–90% of lung cancers in Western countries are caused due to cigarette smoking, but only 7% of female lung cancer cases are associated with smoking[4–6]. Furthermore, adenocarcinoma is much more common than other subtypes of non-small cell lung carcinoma seen in non-smokers. These statistics emphasize the necessity of a better understanding of the molecular mechanisms that mediate the development of lung cancer in non-smoking female patients.

Advanced high-throughput technologies have had an important role in identifying the genetic abnormalities that drive the development and growth of various cancers[7,8]. In addition to genetic changes, epigenetic changes, such as hyper- or hypomethylation, lead to the aberrant expression of tumor suppressor genes or oncogenes[9]. Hypermethylation within the promoter region was responsible for the inactivation of approximately half of

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Original Article

Overexpression of methylation-driven *DCC* suppresses proliferation of lung cancer cells

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Contributions: (I) Conception and design: G Lenka, LC Lai; (II) Administrative support: EY Chuang, LC Lai; (III) Provision of study materials or patients: G Lenka, MH Tsai; (IV) Collection and assembly of data: G Lenka, JH Hsiao; (V) Data analysis and interpretation: G Lenka, JH Hsiao, LC Lai; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

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Background: DNA methylation is an epigenetic marker associated with regulation of gene expression and gene silencing. The active role of DNA methylation has been thoroughly studied in a number of cancer types. *The deleted in colorectal carcinoma* (*DCC*) gene, located at chromosome 18q21, plays an important role as a tumor suppressor and is associated with hypermethylation in head and neck squamous cell carcinoma. However, the methylation patterns and functional significance of *DCC* in lung cancer are still not known.

Methods: RT-PCR was used to examine the endogenous expression levels of *DCC* in two lung cancer cell lines (A549, H1299) and their normal counterpart (Beas-2B). The demethylating agent, 5-aza-2'-deoxycytidine (5-aza), was used to examine the role of methylation in regulating expression of *DCC* in lung cancer cell lines. *DCC* was also overexpressed to evaluate its role in proliferation and colony formation. Finally, the gene expression signature of public dataset GSE68456 was used to elucidate the prognostic roles of *DCC* in lung adenocarcinoma patients.

Results: Endogenous expression of *DCC* was significantly decreased in lung cancer compared to the normal cells ($P<0.0001$). Furthermore, treatment with 10 μM 5-aza significantly up-regulated *DCC* in cancer cell lines ($P\leq0.001$), but not in Beas-2B cells. Overexpression of *DCC* significantly decreased cell proliferation ($P<0.05$) and colony formation ($P\leq0.001$). Finally, significantly lower survival rates ($P<0.001$) were observed when expression levels of *DCC* were decreased.

Conclusions: Our results indicate that *DCC* is regulated by methylation in lung cancer cell lines, and may be associated with cell proliferation, colony formation, and prognoses of lung cancer in patients.

Keywords: *The deleted in colorectal carcinoma* (*DCC*) netrin 1 receptor; lung adenocarcinoma; methylation; proliferation

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Introduction

Lung carcinoma is the leading cause of cancer-related deaths worldwide (18% of all cancer deaths) (1,2). Most lung cancers arise from epithelial cells and their etiologies are attributed to both genetic and environmental factors. Non-small cell lung cancer (NSCLC) is the most common type of lung cancer and accounts for at least 80% of all lung cancer cases (3). Despite recent scientific advances in diagnosis and treatment, the 5-year survival rate of NSCLC is still poor (4).

The advent of high-throughput technologies has helped uncover genetic abnormalities that drive the development and growth of various cancers. In addition to genetic

Aberrant expression of the *PRAC* gene in prostate cancer

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[1]Department of Chemical Engineering and Biotechnology, Institute of Biochemical and Biomedical Engineering, National Taipei University of Technology, Taipei; [2]Division of Urology, Department of Surgery, [3]Department of Pathology, Chang Gung Memorial Hospital, LinKou, School of Medicine, Chang Gung University, Tao-Yuan, Taiwan, R.O.C.

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Abstract. Identification of aberrant expression patterns of genes in prostate cancer (PCa) is a key step towards the development of effective therapies. Prostate-specific antigen (PSA) levels are commonly measured for the early detection of PCa, but which itself is still not an ideal biomarker. We analysed the expression patterns of prostate cancer susceptibility candidate (*PRAC*) in prostate cancer. The *PRAC* gene is known to be commonly expressed in prostate tissue, rectum and colon. To provide clear insights into the expression patterns of *PRAC* in PCa, we examined the gene expression by quantitative real-time PCR (qRT-PCR), western blot analysis and immunohistochemistry (IHC). The results showed that *PRAC* expression levels in androgen-insensitive cells (DU145 and PC3) are lower than those in androgen-sensitive cell lines (LNCaP, LNCaP-R and CW22R). However, treatment of the LNCaP cell line with androgen and anti-androgen demonstrated that *PRAC* is expressed in an androgen-independent manner. Further, PRAC expression was restored upon treatment of DU145 and PC3 cells with the methyltransferase inhibitor, 5-aza-2'-deoxycytidine (5-aza-CdR), which indicates the effect of methylation in the control of *PRAC* expression. In addition, IHC analysis revealed a significantly decreased immunoreactivity of PRAC protein in PCa tissues compared to benign prostatic hyperplasia (BPH) ($p<0.0001$). Thus, our findings suggest that the pathogenesis of PCa may be due to the expression levels of PRAC protein, and this protein can serve as a potential biomarker for the management of PCa.

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Key words: prostate cancer susceptibility candidate, immunohistochemistry, aberrant expression, methylation, prostate cancer

Introduction

Prostate cancer is a heterogeneous disease due to which the search for genetic causes involved in the pathogenesis remains a challenge. Deregulated expression of several genes such as *EZR* and *OCT1* were identified, and the involvement of the aberrant expression of these genes was reported in the pathogenesis of PCa (1,2). The variety in biological behaviour of PCa demands identification of biomarkers that may distinguish a slow growing cancer from a more aggressive cancer with a potential to metastasize (3).

The androgen receptor is a ligand-dependent zinc finger DNA-binding protein that is involved in the regulation of transcription of a variety of gene derivatives (4). The unique feature of PCa is its dependency of androgen for its growth and survival. Several novel androgen-regulated genes have been identified, some of which may be important in the regulation of prostate cell invasiveness (5). In general, androgens activate the androgen receptors which in turn control the expression of androgen receptor response elements (ARE) containing genes due to which current research targets androgen-based therapies for PCa.

Epigenetic factors are also known to mediate the expression of several genes. DNA methylation is one of the epigenetic mechanisms (6) and it occurs in mammals mostly at cytosines within CpG dinucleotide. Several studies have been proposed that DNA hypomethylation can cause activation of oncogenes and genetic instability, whilst hypermethylation is associated with inappropriate gene silencing (7). For instance, Lin *et al* reported the role of hypermethylation in the silence of glutathione-S-transferase P1 (*GSTP1*) expression in PCa (8). It is reported that *GSTP1* is hypermethylated in nearly all human prostate cancers and its promoter DNA methylation level is able to differentiate between BPH and different grades of prostate adenocarcinoma (9-11).

PRAC is a novel gene encoding for the 382 nucleotide RNA, and it specifically expressed in prostate tissue, rectum and colon. The sequence tag database is a potential source for discovery of new genes (12,13), and it was used to find the *PRAC* gene (14). The *PRAC* gene is located on chromosome 17 at position 17q21, 4 kbp downstream from the homeodomain *Hoxb-13* gene. To date, there is no specific study on the prognostic role and regulatory factors that govern the expression of

cDNA microarray profiling of rat cholangiocarcinoma induced by thioacetamide

CHUN-NAN YEH[1*], WEN-HUI WENG[2*], GOVINDA LENKA[2], LEE-CHENG TSAO[2], KUN-CHUN CHIANG[3], SEE-TONG PANG[4], TSUNG-WEN CHEN[1], YI-YIN JAN[1] and MIIN-FU CHEN[1]

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Abstract. Cholangiocarcinoma (CCA) is a malignant neoplasm affecting thousands of individuals worldwide. CCA develops through a multistep process. In the current study, an oral thioacetamide (TAA)-induced model of rat CCA was established which generates the histological progression of human CCA, particularly the mass-forming type. Seven male Sprague-Dawley rats were treated with TAA for 24 weeks to induce CCA. Following the generation of the rat CCA model, whole rat genomic oligo microarray was performed to examine gene expression profiles in CCA and non-cancerous liver samples. In brief, 10,427 genes were found to be differentially expressed (8,318 upregulated and 3,489 downregulated) in CCA compared with non-tumor liver tissue. The top 50 genes (upregulated or downregulated) were selected and their functional involvement in various pathways associated with cancer progression was analyzed, including cell proliferation, apoptosis, metabolism and the cell cycle. In addition, increased expression of CLCA3, COL1A2, DCN, GLIPr2 and NID1, and decreased expression of CYP2C7 and SLC10A1 were validated by quantitative real-time PCR. Immunohistochemical analysis was performed to determine the protein expression levels of GLIPr2 and SLC10A1. The gene expression profiling performed in this study provides a unique opportunity for understanding the carcinogenesis of TAA-induced CAA. In addition, expression profiling of a number of specific genes is likely to provide important novel biomarkers for the diagnosis of CCA and the development of novel therapeutic strategies for CCA.

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E-mail: pst64lab@gmail.com

*Contributed equally

Key words: cholangiocarcinoma, thioacetamide, carcinogenesis, cDNA expression array, pathway analysis, diagnostic markers

Introduction

Cholangiocarcinoma (CCA) is a lethal malignancy derived from the epithelial cells (i.e. cholangiocytes) of the bile duct. CCA exhibits a considerable variety of symptoms commonly at the later stages of disease and therefore treatment for CCA is extremely difficult. CCA is grossly divided into mass forming (MF), periductal infiltrating and intraductal papillary subtypes (1). Gross pathological classifications of CCA are important in clinical practice and further translational investigations due to the distinct characteristics and outcomes following hepatectomy (2). The incidence of CCA exhibits considerable geographical variation but generally accounts for 5-30% of primary liver cancer (3). Previous studies have reported that the incidence and mortality rates of CCA have been increasing worldwide, particularly intrahepatic CCA (4-6). CCA is caused by a number of risk factors, including parasitic infections, primary sclerosing cholangitis, choledochal cysts, hepatolithiasis and carcinogen exposure, which leads to the significant variance in incidence rates of CCA worldwide (7-9).

Clinically, CCA remains extremely challenging as patients do not typically exhibit clear symptoms until the disease is quite advanced and therefore it is difficult to diagnose in its early stages. In addition to surgical treatments (2,10-14), radiation therapy and current chemotherapeutic protocols have not been found to significantly improve the long-term survival rates of CCA patients (8,15). In our previous study, a thioacetamide (TAA)-induced CCA rat model was established to analyze the molecular and morphological behavior of CCA, aiming to generate a powerful preclinical platform to provide insights into therapeutic and chemopreventative strategies for human CCA (16). Since the model recapitulates the dysplasia-carcinoma sequence of human CCA, it is likely

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NANOSIZED PARTICLES OF TITANIUM DIOXIDE SPECIFICALLY INCREASE THE EFFICENCY OF CONVENTIONAL POLYMERASE CHAIN REACTION

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In recent years, the use of nanoparticles (NPs) for improving the specificity and efficiency of the polymerase chain reaction (PCR) and exploring the PCR enhancing mechanism has come under intense scrutiny. In this study, the effect of titanium dioxide (TiO_2) NPs in improving the efficiency of different PCR assays was evaluated. Transmission electron microscopy (TEM) results revealed the average diameter of TiO_2 particles to be about 7 nm. Aqueous suspension of TiO_2 NPs was included in PCR, reverse transcription PCR (RT-PCR) and quantitative real time PCR (qPCR) assays. For conventional PCR, the results showed that in the presence of 0.2 nM of TiO_2 a significant amount of target DNA ($P<0.05$) could be obtained even with the less initial template concentration. Relative to the larger TiO_2 particles (25 nm) used in a previous study, the smaller TiO_2 particles (7 nm) used in our study increased the yield of PCR by three or more fold. Sequencing results revealed that TiO_2 assisted PCR had similar fidelity to that of a conventional PCR system. Contrary to expectation, TiO_2 NPs were unable to enhance the efficiency of RT-PCR and qPCR. Therefore, TiO_2 NPs may be used as efficient additives to improve the conventional PCR system.

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Keywords: Nanosized TiO_2 particles, Conventional PCR, Reverse transcription PCR, Quantitative real time PCR, Efficiency, Fidelity

1. Introduction

The molecular biology techniques including conventional PCR, qPCR and RT-PCR have been recognised to be the standard industrial techniques for the qualitative and quantitative analysis of nucleic acids due to their high sensitivity and specificity. PCR and qPCR mimic the in vivo molecular process, DNA replication [1]. Both of these techniques require single or few copies of DNA template, the primer specific for targeting the sense and antisense strands, dNTPs, heat stable Taq polymerase, and magnesium ions in the buffer for the synthesis of target DNA sequence. Usually, the PCR assays are performed by cycling of denaturation (94^0C), annealing (~$50\text{-}60^0$C) and extension (72^0C) temperatures. The high temperature is applied to denature the strands of the double helical DNA by destroying the hydrogen bonds. Then, temperature is lowered to let primers anneal to the template, and finally the temperature is set around 72^0C which is optimum for the heat stable polymerase that extends the new DNA copies by incorporating the dNTPs [2].

PCR technology is emerged to specifically amplify a target DNA from an undetectable amount of starting materials. In conventional PCR at the end of the amplification, the products (also known as amplicons) can be run on a gel for detection. Later, qPCR was developed to eliminate the necessity of PCR product gel analysis and for the simultaneous detection of a specific target DNA amount in a sample by monitoring the reaction product in real time.

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Phosphorylated T567 ezrin is associated with merlin expression in KIT-mutant gastrointestinal stromal tumors

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Abstract. Membrane-cytoskeleton linker organizer ezrin is a member of the ERM (ezrin-radixin-moesin) protein family. It has been suggested as an important element in the oncogenic process, particularly in conferring a metastatic ability on tumor cells. We hypothesized that the KIT oncogenic form is one of the proteins that modulates expression of the ezrin protein via phosphorylated ezrin at different residues; furthermore, it may interact with the protein merlin, and promoting tumor development via the PI3K or MAPK pathway. In the present study, we observed that differential expression of ezrin was a common feature in gastrointestinal stromal tumors (GISTs). We further demonstrated that cases exhibiting expression of phosphorylated Thr567 in the ezrin protein were associated with immunoactivities of KIT and merlin expression (p=0.039 and 0.013, respectively). In conclusion, GISTs harbor activation of KIT protein may induce phosphorylation of the downstream protein ezrin at certain residues, thereby triggering subsequent signal transduction cascades and driving downstream pathways of tumor progression. However, a larger series of tumor samples should be analyzed in future studies, as well as the identification of phosphorylated sites to determine the role of ezrin in tumor progression thus shedding light on clinical outcomes.

Introduction

Investigations spanning almost a decade reveal that the membrane-cytoskeleton linker protein ezrin plays an important role in promoting tumor metastasis (1,2). It is therefore generally considered to be one of the predictive prognostic biomarkers in various cancer types, including osteosarcoma, breast cancer, colorectal carcinoma, soft tissue sarcoma and serous ovarian carcinoma (3-10). Ezrin protein is a member of the ERM (ezrin-radixin-moesin) group of proteins that are produced from the *VIL2* (Ctytovillin) gene, which is a cyclic AMP-dependent protein kinase anchoring protein. Activation of ezrin is known to be caused by phosphorylation at certain residues, in turn interacting directly with the actin by the C-terminus, and connecting with several transmembrane proteins or membrane-associated partners via the amino-terminal FERM (four-point-one, ezrin, radixin, moesin) domain (11). There are reports indicating that the functions of ezrin particularly operate in the regulation of epithelial cell morphogenesis, cell-cell and cell-matrix adhesion proceed through various pathways (12). Furthermore, the unexpected phosphorylation at certain residues of ezrin is vital for tumor progression. Monni *et al* observed that phosphorylated ezrin at the Tyr353 and Tyr146 residues may induce tumor cell apoptosis and promote cell proliferation in murine erythroleukemia, respectively (13). In addition, the binding of phosphatidylinositol 4,5-bisphosphate (PIP2) with ezrin N-terminal ERM association domain is necessary for the subsequent phosphorylation at Thr567, which is involved in the subsequent activation process to unmask both membrane and actin binding sites (14), which then extend to interact with CD43 and CD44 (15-18). Moreover, some of the studies further indicated that a mutant form of ezrin by mimic a phosphorylated residue ezrin T567D may maintain the protein in an open conformation that will further trigger the activity of the Rac1 pathway (but not RhoA or Cdc 42) (19,20).

Conversely, merlin acts as an inhibitor of small G-protein activation, a role more like a 'gatekeeper', in many types of tumors. The protein merlin is encoded by the tumor-suppressor gene *Neurofibromin 2 (NF2)*, from which multiple isoforms are generated after transcripts undergo alternative splicing. However, only isoform I functions as a tumor-suppressor protein, and the activitity is according to the phosphorylated status (21,22). The location of the merlin protein in cells is rather similar to ezrin. They are commonly present at the membrane-cytoskeleton interface underneath the plasma membrane, cell-cell junctions, as well as actin-rich sites (23); thus the structure of merlin shares N-terminal

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Functional Promoter Polymorphisms Govern Differential Expression of HMG-CoA Reductase Gene in Mouse Models of Essential Hypertension

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Abstract

3-Hydroxy-3-methylglutaryl-coenzyme A [HMG-CoA] reductase gene (*Hmgcr*) is a susceptibility gene for essential hypertension. Sequencing of the *Hmgcr* locus in genetically hypertensive BPH (blood pressure high), genetically hypotensive BPL (blood pressure low) and genetically normotensive BPN (blood pressure normal) mice yielded a number of single nucleotide polymorphisms (SNPs). BPH/BPL/BPN *Hmgcr* promoter-luciferase reporter constructs were generated and transfected into liver HepG2, ovarian CHO, kidney HEK-293 and neuronal N2A cells for functional characterization of the promoter SNPs. The BPH-*Hmgcr* promoter showed significantly less activity than the BPL-*Hmgcr* promoter under basal as well as nicotine/cholesterol-treated conditions. This finding was consistent with lower endogenous *Hmgcr* expression in liver and lower plasma cholesterol in BPH mice. Transfection experiments using 5′-promoter deletion constructs (strategically made to assess the functional significance of each promoter SNP) and computational analysis predicted lower binding affinities of transcription factors c-Fos, n-Myc and Max with the BPH-promoter as compared to the BPL-promoter. Corroboratively, the BPH promoter-luciferase reporter construct co-transfected with expression plasmids of these transcription factors displayed less pronounced augmentation of luciferase activity than the BPL construct, particularly at lower amounts of transcription factor plasmids. Electrophoretic mobility shift assays also showed diminished interactions of the BPH promoter with HepG2 nuclear proteins. Taken together, this study provides mechanistic basis for the differential *Hmgcr* expression in these mouse models of human essential hypertension and have implications for better understanding the role of this gene in regulation of blood pressure.

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Introduction

Essential hypertension, the chief risk factor for cardiovascular and renal diseases, is often associated with and complicated by dyslipidemia [1,2]. 3-Hydroxy-3-methylglutaryl-coenzyme A [HMG-CoA] reductase gene (*Hmgcr*/*HMGCR*) is a candidate gene for hypertension; it translates to the rate-limiting enzyme in the cholesterol biosynthesis pathway and cholesterol is the precursor of glucocorticoid steroid hormones that play a profound role in blood pressure homeostasis and hypertension [3–7]. Consistently, the G allele of *HMGCR* rs17238540 (G/T) single nucleotide polymorphism [SNP] was associated with higher blood pressure [BP] and higher stroke risk in an European population of ~23,000 participants [8]. Moreover, this SNP was associated with the BP response to urinary sodium: potassium ratio [9] and response to statin (inhibitor of HMGCR enzyme) therapy in terms of total cholesterol and triglyceride lowering [10]. Two common and tightly linked *HMGCR* SNPs were also significantly associated with reduced efficacy of pravastatin therapy [11]. Additionally, investigations on gene expression pattern in adrenal glands of two independent, inbred, homozygous rodent models of human essential hypertension (viz. spontaneously hypertensive rat and blood pressure high [BPH] mice) revealed ~2- to 3-fold over-expression of *Hmgcr* in these strains as compared to their corresponding controls (viz. Wistar/Kyoto rat and blood pressure low [BPL] mice) [12,13]. These findings suggested the possibility that an altered *Hmgcr* expression might be a systematic facet of hereditary hypertension in mammals, perhaps even contributing to diverse metabolic abnormalities associated with this common disorder. However, molecular basis of the differential *Hmgcr* expression in these animal models has not been studied. *Hmgcr* levels in other tissues (e.g., liver) of BPL and BPH mice also remain unknown.

The hypertensive mouse strain BPH was developed in a breeding program based solely on selection by elevated BP and it parallels human hypertension [14]. The BPH strain exhibits many of the co-morbidities observed in human hypertension, such as higher heart rate, larger hearts and kidneys, higher left ventricular weight and early mortality than the hypotensive BPL strain [14]. During generation of the BPH and BPL strains, the normotensive inbred strain BPN (blood pressure normal) was derived from the unselected control population and this strain serves as a control for hypertensive BPH and hypotensive BPL mice [14].

Computational Analysis: Towards a Better Knowledge of the Molecular Evolution of Phosphoenolpyruvate Carboxylase among Flaveria Species

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***Abstract*- *Flaveria* is a genus of plants in the sunflower family (Asteraceae). Within the close species of *Flaveria*, there are several different photosynthetic metabolisms including C3, C4 and C3-C4 mixed metabolism. In C4 metabolism, phosphoenolpyruvate carboxylase (PEPC) catalyses the primary fixation of atmospheric CO_2. In order to elucidate the discrete steps in PEPC evolution computational analysis was made for the PEPC protein sequences of C3, C3–C4 and C4 species of the dicot genus *Flaveria*. The predicted key amino acid residue changes and putative phosphorylation sites can advance our knowledge on plant photosynthesis metabolism, especially on the regulation of PEPC activity. One of the most notable amino acid residue changes found at 123 contains serine in C4 *Flaveria* species and occupied by arginine in C3, C3-C4, and C4 like *Flaveria* species and also the serine residue at this position was predicted as putative phosphorylation site. Functional expression and characterization of the C3, C3-C4 intermediate and C4 PEPC of *Flaveria* species enzymes can reveal that these molecules exhibit diverse kinetic properties despite their relatively high degree of sequence similarity.**

Keywords- Evolution, Flaveria, Phosphoenol pyruvate carboxylase, Phosphorylation, Photosynthesis

I. INTRODUCTION

The largest current use of renewable energy sources is associated with plant biomass. Although the primary aim is food production, increasing amounts of residues are made useful for energy purposes and as feedstock in manufacturing industries. How to increase the biomass energy production from plant is an important issue. Study on regulatory aspects of the key metabolic enzymes, in related to photosynthesis is one of the best strategies to increase the biomass production. In this article, we have used bioinformatics tools to study the significant changes of Phosphoenolpyruvate carboxylase (PEPC; EC 4.1.1.31) among the *Flaveria* species including C3, C3-C4 and C4 plants.

PEPC catalyses the β-carboxylation of phosphoenolpyruvate in the presence of HCO_3 to yield oxaloacetate and inorganic phosphate (Pi) [1, 2]. In C4 plants, C4 PEPC is located in the mesophyll-cell cytosol. L-malate, the end-product of the corresponding pathway, is transported to bundle sheath cells where it undergoes subsequent decarboxylation by chloroplastic malic enzyme. The reaction feeds CO_2 to the Calvin cycle [3]. PEPC plays a pivotal role in photosynthesis by C4 and Crassulacean acid metabolism plants, in addition to its many anaplerotic functions.

The comparison study of organisms on the level of molecular characters has become a powerful and now indispensable approach to elucidate the function of an enzyme. Because of the wide distribution of PEPC and the high functional diversity, it has been proposed that the nucleotide sequences of the PEPC genes and the amino acid sequences of the gene products should provide powerful markers in molecular taxonomic and phylogenetic investigations [4]. We have analyzed PEPC sequences in species including *Flaveria* and *Zea mays*. *Flaveria* is a genus of plants in the sunflower family (Asteraceae). Some members of this genus exhibit C3 carbon fixation, while others are C4 plants and some are intermediate [5]. Since members of the same genus exhibit different carbon fixation mechanisms by C3 and C4, it will be very interesting to look at the evolutionary relationship of key enzyme C4 Phosphoenolpyruvate Carboxylase to understand the role in enhancing photosynthetic efficiency.

In previous works, the *in vitro* dual regulation of *Sorghum* C4 PEPC activity by opposing metabolites, G-6P and L-malate, and phosphorylation was investigated [6]. Our goal is to describe how to use computational analysis to find key aminoacid residue changes and putative phosphorylation sites which might be contributed to the regulation of plant PEPC function. Owing to its location at a pivotal branch point in primary plant metabolism, PEPC is tightly controlled by a combination of fine metabolic controls, including allosteric effectors and reversible phosphorylation [7]. Furthermore, Phosphorylation was found to modify the kinetic properties of the enzyme and most notably, this led to a reduced sensitivity

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Sequence Motif Analysis of Phycobiliproteins in Cyanobacterial Genome

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***Abstract*—Phycobiliproteins are a group of pigmented proteins commonly present in cyanobacteria and red algae which has potential application in different fields. Based on the color and absorbance they are grouped in to three important types. The sequence motif was analyzed for all the important Phycobiliproteins by using its protein sequence obtained from the non-redundant database. 39 sequence motifs were predicted by MEME, among them 11 were found significant with less e-value. MAST analysis showed the motifs showed high similarity with Synechococcus species and Thermococcus species in the Cyanobase.**

Keywords-Phycobiliproteins; Cyanobacteria; MEME; MAST; motif

I. Introduction

Phycobiliproteins are a group of proteins referred as light-harvesting macromolecules that function as components of the photosynthetic apparatus in cyanobacteria and several groups of eukaryotic algae [1-5]. Three major types of phycobiliproteins are phycoerythrins, phycocyanins and allophycocyanins which are present in red algae and cyanobacteria. The absorption maxima are 540–570nm for Phycoerythrin, 610–620nm for Phycocyanin, and 650–655 nm for Allophycocyanin. These proteins spans over a 200-nm portion of the visible spectrum (470–670 nm) which enable the organism to extend the range of their spectral absorption to collect light which are not absorbed completely by chlorophyll *a* and through fluorescence energy transfer, it conveys the energy to chlorophyll at the photosynthetic reaction center. The phycobiliproteins are water soluble, very stable at physiological pHs, and highly fluorescent proteins [6,7] and constitutes about 60% of the soluble protein content [8]. These are oligomeric proteins, built up from chromophore-having polypeptides belonging to two families (α and β) which may be originated from a common ancestor [4]. The phycobiliproteins are composed of a number of subunits, each having a protein backbone to which linear tetrapyrrole chromophores are covalently bound. The phycobiliproteins involved in an extremely efficient energy transfer chain in the photosynthetic reaction. These proteins transfer the excitation energy with less radiation processes to the reaction centers in the photosynthetic membranes [6].

Cyanobacteria, which produce phycobiliproteins, are recognized as an important and widespread component of marine picophytoplankton that contributes significantly to total carbon biomass and primary productivity of the oceans. The phycobiliproteins obtained from cyanobacteria have gained commercial importance, as they got several applications. The main applications of these molecules are as natural dyes, colorants in food and it is most widely as fluorescent labels for cells and macromolecules in highly sensitive fluorescence techniques [9], but various studies showed on their health-promoting properties and broad range of pharmaceutical applications [10].

In this study, the available protein sequences for phycobiliproteins from the non-redundant protein database were used to find sequence motif patterns among the cyanobacterial species. Basically, motif is a region or portion of a protein sequence that has a specific structure and is functionally significant. Protein families are often characterized by one or more such motifs. For motif prediction, MEME tool was used to analyze the sequences and produce a description (motif) for each pattern it discovers. This study was carried out to find key functional motifs among the different cyanobacterial species (Fig. 1). Detection of sequence motifs in protein is an important approach since motifs carry out and regulate various functions, and the presence of specific motifs may help to classify proteins. The motifs which are involved in regulating the synthesis of phycobiliproteins can be helpful in overproduction of the protein in commercial scale. After

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