

CURRICULUM VITAE

NAME: **Robert MacGregor Gemmill**

EDUCATION:

1975 B.A. (Biology), University of Connecticut, Storrs, CT
1981 Ph.D. (Biochemistry), Cornell University, Ithaca, NY
1981-82 Postdoctoral Training (*Aedes aegypti* Molecular Biology), Cornell University, Ithaca, NY
1982-83 Postdoctoral Training (*Drosophila* Molecular Genetics), Arizona State University, Tempe, AZ

DISSERTATION:

"Attenuation Control: The Leader and the Promoter of the Salmonella Leucine Operon." Dr. Joseph M. Calvo, Advisor.

PROFESSIONAL EXPERIENCE:

1975-1976 Research Assistant, Cornell University, Ithaca, NY.
1976-1977 Teaching Assistant, Cornell University, Ithaca, NY.
1978-1980 Research Assistant, Cornell University, Ithaca, NY, (Predoctoral trainee).
1980-1981 Postdoctoral Associate, Cornell University, Ithaca, NY.
1982-1983 Postdoctoral Associate, Arizona State University, Tempe, AZ.
1983-1984 Faculty Research Associate, Department of Zoology, Arizona State University, Tempe, AZ.
1984-1989 Assistant Professor, Adjunct, Department of Zoology, Arizona State University, Tempe, AZ.
1984-1989 Director of Molecular Genetics, The Genetics Center of Southwest Biomedical Research Institute, Scottsdale, AZ.
1989-2007 Institute Fellow, Eleanor Roosevelt Institute, Denver CO.
1989-1999 Assistant Professor, Adjunct, Department of Biochemistry, Biophysics and Genetics, University of Colorado Health Sciences Center, Denver, CO.
1996-1997 Assistant Professor, Department of Medicine, Division of Medical Oncology, University of Colorado Health Sciences Center, Denver, CO.
1997-2006 Associate Professor, Department of Medicine, Division of Medical Oncology, University of Colorado Health Sciences Center, Denver, CO.
2000-2007 Joint appointment in Cellular & Structural Biology, University of Colorado Health Sciences Center, Denver, CO.
2007-2007 Professor of Medicine, Department of Medicine, Division of Medical Oncology, University of Colorado Health Sciences Center, Denver, CO.
2007- Melvin Berlinsky Chair in Cancer Research, Professor of Medicine, Department of Medicine, Division of Hematology/Oncology, Medical University of South Carolina, Charleston, SC.

ADMINISTRATION/COMMITTEES

1990-1996	Director, Cytogenetics Core Laboratory, Colorado Cancer Center, UCHSC.
1996-1997	Leader, Chromosomes and Mutations Program, Colorado Cancer Center, UCHSC.
1997-2003	Leader, Cancer Genetics Program, Colorado Cancer Center, UCHSC.
1994-1995	Member, Chancellor's Genetics Steering Committee, UCHSC.
1995-1997	Member, Human Medical Genetics Program Director Search Committee, UCHSC.
1998-	Member, HMGP Executive Committee
2003	Member, Cancer Center Assoc. Director for Basic Research Search Committee.
1989-1991	Major Advisor and Member of Jury for Sylvie Paulien, Doctoral Candidate, University VII, Paris, France.
1995-1997	Member of Graduate Committee for Yuri Pekarsky, Doctoral Candidate, UCHSC.
1997-2001	Member of Graduate Committee for Brym Grimason, Doctoral Candidate, UCHSC.
2001-	Major Advisor for Jason Lee, Graduate Student
2004-	Chair, Division of Medical Oncology Research & Space Committee
2005-	Member, Dept of Medicine Research Committee
2006-	Member, Division of Medical Oncology Fund Raising Committee

SERVICE

1. NIH Study Sections and Ad-Hoc Review Committees

1987	Genome special study section
1988-1998	Ad-hoc member of various Program Project Review Committees
1995	Breast SPORE Review Committee (March 1995)
1995-2000	Member, Cancer Research Manpower Review Committee; Certificate of Appreciation issued June 2000.
2005	NIH Intramural Review, Lab of Cellular Carcinogenesis and Tumor Promotion
2007	NIH Program Project Review Committee

2. Reviewer for the following journals

Cancer Genetics & Cytogenetics
Cancer Research
Clinical Cancer Research
Cytogenetics & Cell Genetics
Genomics
PNAS
Science
Development

3. Organized 2nd International Workshop on Human Chromosome 3 mapping (1990).
Organized 1st International Workshop on Human Chromosome 12 mapping (1992).

AWARDS

2006 "Best of Show". Univ. of Colorado @ Denver and Health Sciences Center, Dept. of Medicine Research Day, Oct 21, Award for Best Research Poster "The TRC8 hereditary kidney cancer gene is a sterol-regulated E3 ubiquitin ligase linking lipid status with growth control, protein translation and hypoxia".

2007 Excellence in Teaching Award. Univ. Colorado at Denver and Health Sciences Center, Dept. of Medicine, Div. of Medical Oncology.

TEACHING EXPERIENCE:

- 1) "Cloning and analysis of large DNA". Course at Cold Spring Harbor, New York. Two week course covering YAC and P1 cloning with pulsed field gel electrophoresis. April 1989 and April 1990.
- 2) Molecular Genetics Course IDPT. Taught "Mammalian cell and human molecular genetics". Spring 1993 - 1997.
- 3) Established new course on Molecular Biology of Cancer (MED 6626) with Dr. Chris Hogan. Course has been taught 6 semesters, Spring 1998 and Fall 1998 - 2002. Will be taught in Fall quarter 2007.
- 4) Section on Cancer Genetics for HMGP7600 Graduate Survey of Human Genetics. Spring 2002, 2003.
- 5) Section on Size Controls in Developmental Biology CSBI-7605 (2002, 2003).
- 6) Participation in Oncology Fellows Conference (twice per week), in-lab Journal Club.
- 7) Organized on-going series of didactic lectures in Cancer Molecular Biology for Medical Oncology Fellows (2002-).
- 8) Graduate student Core Course IDPT 7802, taught section on Human Genetics (Fall 2003-present).

PRESENTATIONS:

(invited = *)

2006

"A Lung Cancer Suppressor, SEMA3F: Mechanisms of Activity and Downregulation in Tumors", Lung Cancer SPORE Winter Workshop, Los Angeles, CA, Jan 25-26, 2006.

"Linking Lipid Metabolism to Neoplasia through TRC8, a Kidney Cancer Tumor Suppressor and E3 Ubiquitin Ligase" Presentation to MSTP students, Feb 8, 2006.

* “TRC8 is a lipid-regulated E3 ubiquitin ligase that links cancer with cholesterol biosynthesis” Professor’s Rounds for Pediatric Oncology, The Children’s Hospital, Denver CO, Feb. 17, 2006.

“TRC8, the t(3;8) Hereditary RCC Gene, Is a Polytopic RING Protein whose E3 Ubiquitin Ligase Activity is Necessary for Tumor Suppression” Poster @ AACR Annual Meeting, Washington, DC, April 2006.

“Preclinical analysis of rapamycin plus EGFR or MEK1/2 inhibitors in renal cell carcinoma (RCC)” Poster @ AACR Annual Meeting, Washington, DC, April 2006.

* “TRC8, a Polytopic E3 Ubiquitin Ligase linking growth control with lipid biosynthesis through protein translation initiation and oxygen sensing” UCHSC Cancer Center Developmental Therapeutics Program Retreat, April 21, 2006

* “TRC8 is a lipid-regulated E3 ubiquitin ligase that links cancer with cholesterol biosynthesis” Hormone Related Malignancies and Endocrinology Seminar, UCHSC, April 26, 2006.

* “The Skinny on Fat and Cancer: A molecular link between lipid status and growth control through the TRC8 tumor suppressor” UCDHSC Cancer Center Symposium, May 23, 2006.

“The TRC8 Hereditary Kidney Cancer Gene is a Sterol-regulated E3 Ubiquitin Ligase that Links Protein Translation and Growth Control with Sterol and Lipid Status”. ZOMES-IV The Fourth International Symposium on COP9 Signalosome, Proteasome and eIF3 – at the Interface between Signaling & Proteolysis, Yale Univ. June 18-21, 2006.

“The TRC8 Hereditary Kidney Cancer Gene is a Sterol-regulated E3 Ubiquitin Ligase that Links Protein Translation and Growth Control to Sterol and Lipid Status”. Cold Spring Harbor Meeting on “Translational Control”, Sept 6-10, 2006, Cold Spring Harbor, NY.

2005

“E-cadherin regulation, apoptosis & inhibitor interactions in lung cancer cells” SPORE Winter Workshop, San Diego CA., Feb 2005.

“Inhibition of lung tumorigenesis by SEMA3F” SPORE Winter Workshop, San Diego CA., Feb 2005.

“Growth Suppression Induced by the TRC8 Hereditary Kidney Cancer Gene is Dependent upon JAB1/CSN5”. Poster @ AACR Annual Meeting, Anaheim, CA, March 2005.

* “Growth Suppression by the TRC8 Kidney Cancer Gene is Dependent Upon Interactions with the COP9 Signalosome Subunit JAB1/CSN5” Invited talk at Medical University of South Carolina, Charleston, SC, Mar 18, 2005.

* “Renal Cancer and the Polytopic RING protein TRC8” Invited lecture, NCI-Frederick, MD, Laboratory of Protein Dynamics and Signaling, June 30, 2005.

* “Renal Cancer and the Polytopic RING protein TRC8” Invited talk at Medical University of South Carolina, Charleston, SC, Aug 15, 2005.

“Pathways to Renal Cancer” Lecture to Medical Oncology Fellows, Div. Medical Oncology, UCHSC, Sept 2, 2005

2004

* “Role of Growth Factor Receptors (GFR) in Lung Tumorigenesis”. Lecture given to the First Latin American Conference on Lung Cancer, Guaruja, Brasil, March 2004.

* “Tumor microenvironment”. Lecture given to the First Latin American Conference on Lung Cancer, Guaruja, Brasil, March 2004.

“Growth Inhibition by Iressa and Rapamycin in Renal Cell Carcinoma with wild-type VHL”. RM. Gemmill and HA. Drabkin, Synergistic Third International Kidney Cancer Symposium, Oct. , 2004, Palmer House, Chicago, IL.

“VHL Mutations in RCC Confer Differential Resistance to Gefitinib and Rapamycin” Poster at DOM Research Day, UCHSC, Nov. 2004.

“GROWTH SUPPRESSION INDUCED BY THE T(3;8) HEREDITARY RENAL CANCER TRC8 GENE IS DEPENDENT UPON JAB1/CSN5” . ZOMES III. The COP9 Signalosome, Proteasome and eIF3: at the Interface between Signaling and Proteolysis, Berlin, Germany 9-12 May, 2004.

2003

* “TRC8: A novel pathway to kidney cancer” 36th Annual Meeting, Am. Society of Nephrology, Renal Week, San Diego, CA, Nov.12-17, 2003. Invited Symposium Lecture.

“Growth suppression induced by the t(3;8) hereditary kidney cancer TRC8 gene is dependent upon JAB1/CSN5” Cell & Developmental Biology Program Retreat, Breckenridge, CO, Sept. 5-7, 2003.

“Genetic analysis of growth suppression induced by the t(3;8) hereditary kidney cancer gene TRC8”, American Association for Cancer Research Annual Meeting,, Washington DC., July 11-14, 2003. Poster Discussion with Platform Presentation.

“Reversal of E-cadherin loss in Lung Cancer”, American Association for Cancer Research Annual Meeting,, Washington DC., July 11-14, 2003. Poster Presentation.

“WNT7a Induces E-cadherin in lung cancer cells”, 11th SPORE Investigator’s Workshop Baltimore, MD, July 8-10, 2003

“Growth suppression induced by the t(3;8) hereditary renal cancer gene TRC8 is dependent upon interaction with the COP9 signalosome subunit CSN5”. Proteasome Workshop, Clermont-Ferrand, France, April 28-May 1, 2003.

* “Growth suppression induced by the t(3;8) hereditary renal cancer gene TRC8 is dependent upon interaction with the COP9 signalosome subunit CSN5”. Centre Etude du Polymorphisme Humain (CEPH), Paris, France, May 3, 2003.

* “Reversal of E-cadherin loss in lung cancer” Centre Etude du Polymorphisme Humain (CEPH), Paris, France, May 3, 2003.

* “Growth suppression induced by the t(3;8) hereditary renal cancer gene TRC8 is dependent upon interaction with the COP9 signalosome subunit CSN5”. Presented at Cleveland Clinic, Cleveland OH, April 25, 2003.

2002

* “The hereditary kidney cancer TRC8 gene functions in parallel with VHL and affects *Drosophila* development”. ZOMES II: The COP9 Signalosome, Proteasome and eIF3 at the Crossroads of Signaling, Antalya, Turkey 28 April - 2 May, 2002.

“Regulation of E-cadherin by β -catenin in lung cancer” UCHSC Human Medical Genetics Program Retreat, Denver, CO, Sept. 14, 2002.

“Regulatory interactions affecting E-cadherin expression in lung cancer”, 10th SPORE Investigator’s Workshop, Chantilly, VA, July 13-16, 2002.

2001

* “The Hereditary Kidney Cancer TRC8 Gene and VHL Function within a common pathway” Univ. Florida, Gainesville, FL, with Harry Drabkin, Oct. 2001.

* “Exploring the TRC8 hereditary kidney cancer gene through *Drosophila* developmental biology”, Colorado State Univ., Cell & Molec. Biol. Program, May 3, 2001.

2000

“TRC8 and hereditary kidney cancer: Inhibition of a *Drosophila* homolog causes defects in early cuticular development” UCHSC Human Medical Genetics Program Retreat, Denver, CO, Sept. 16, 2000.

“TRC8 and hereditary kidney cancer: Inhibition of a *Drosophila* homolog causes defects in early cuticular development”. UCHSC, Developmental Therapeutics Seminar. Sept. 19, 2000.

“HOX gene expression in human lung cancer” 8th SPORE Investigators Workshop, Chantilly, VA, July 9-11, 2000.

“TRC8 and kidney cancer” UCHSC, Dept of Cellular and Structural Biology, April 18, 2000.

“Ubiquitination, protein degradation and kidney cancer” UCHSC, Dept. of Medicine, Div. Medical Oncology, Fellows Conference, Jan 24, 2000.

1999

“TRC8 and Kidney cancer” UCHSC, Dept. of Medicine, Div. Medical Oncology Seminar, June 8, 1999.

* “TRC8, a multiple membrane spanning protein implicated in hereditary kidney cancer and early development” World Health Organization, International Agency for Research on Cancer, Lyon, France, Oct. 12, 1999

1998

* “The hereditary renal cell carcinoma 3;8 translocation is functionally distinct from deletions involving FRA3B” Am. Soc. Human Genetics, 48th Annual Meeting, Workshop on Common Fragile Sites, Denver, CO, October 27-31, 1998.

“The hereditary renal cell carcinoma 3;8 translocation fuses FHIT to a novel Patched related gene, TRC8” Platform Presentation, Am. Soc. Human Genetics, 48th Annual Meeting, Denver, CO, October 27-31, 1998.

* “Molecular Genetic alterations of chromosome 3 in gynecologic tumors”. Invited Keynote Lecture, Japanese Soc. Of Gynecologic Pathology and Colposcopy, Osaka, Japan, July 12 –16, 1998.

* “The hereditary renal cancer (3;8) translocation fuses FHIT to TRC8, a novel gene related to the hedgehog receptor, Patched. Cancer Institute Hospital, Tokyo, Japan, July 9 1998.

* “Molecular Genetics of Gynecological Tumors” Chiba Univ., Chiba, Japan, July 10, 1998.

PAST and CURRENT GRANT SUPPORT:

1. NIH, P50 CA58187 (SPORE in Lung Cancer: Bunn-PI; Component: Gemmill-PI, Drabkin-CoPI) 09/01/92-04/30/03, “SPORE in Lung Cancer Component Title: The Role of Chromosome 3 in Lung Tumorigenesis”, Continuing.
2. NIH, 1RO1 CA76035-05 (Co-PI with Harry Drabkin), “Molecular-Genetic Analysis of 3p14 Genomic Stability”. Continuing.
3. Department of the Army, DAMD17-94-J-4391 (Gemmill, Co-PI with H. Drabkin), “Isolation of a Breast Cancer Tumor Suppressor Gene from Chromosome 3p” Completed in 2000.

4. NIH, 5 P30 CA46934 (Paul Bunn, Jr., M.D.), “Cancer Center Core Grant”, Gemmill - Cancer Genetics Program Leader. Completed.
5. NIH, 1U01 CA85070 (Franklin-PI, Drabkin-CoPI), “Biomarkers in Lung Carcinoma and Premalignancy” Completed 2003.
6. Colorado Cancer League, “Structure Function analysis of DTrc8” 2000-2001, completed.

PROFESSIONAL SOCIETIES:

American Association for Cancer Research
American Association for the Advancement of Science

RESEARCH INTERESTS

The close relationship between developmental signaling pathways and genetic alterations underlying cancer provides a basic theme for the investigations carried out in my lab. For many years my research was driven by the desire to identify, isolate and study genes involved in cancer. Using the crude molecular genetics approaches of the time (mid 1980's), this process was slow and painful. Involvement in the genome project led to our development of a YAC-based physical map for human chromosome 3. This map became the foundation for multiple further studies to identify genes undergoing consistent alterations in cancer, particularly affecting the lung and kidney. Together with my long term collaborator, Dr. Harry Drabkin, we have isolated several genes from 3p and from 8q implicated in these cancers. In each case, these altered genes supported the existence of close relationships between developmental processes and cancer.

A 3p21 homozygous deletion removing β -catenin led to the discovery that E-cadherin expression was regulated in lung cells by the WNT developmental signaling pathway. In contrast to colorectal cancers, where WNT signaling is activated and drives tumor growth, our data indicated that WNT signaling was lost or down-regulated in lung tumors. This alternative effect of the WNT pathway represented a paradigm shift in cancer biology, since the pathway had previously only been understood to drive neoplastic proliferation. Loss of WNT signaling resulted in loss of E-cadherin accompanied by an epithelial-mesenchymal transition and progression of tumors to a metastatic state. We searched for small molecules that could re-establish E-cadherin expression and found success with both lithium (an activator of WNT signaling) and HDAC inhibitors. Effects of the latter agents were consistent with E-cadherin loss occurring through transcriptional repression mediated by histone de-acetylase co-repressors. In fact, we found that the Zn-finger repressor ZEB1 was clearly involved in suppression of E-cadherin in lung cancers. We are now pursuing studies in collaboration with Drs. Dan Chan and Paul Bunn examining the effects of these agents on tumor growth *in vivo* in an orthotopic nude rat model. In addition, we are exploring the regulatory mechanisms that contribute to E-cadherin repression and expression to increase understanding and potentially identify new agents suitable for inducing expression.

A second homozygous deletion in 3p21 led to the identification of the novel semaphoring, SEMA3F, as a potential tumor suppressor gene in lung cancer. We have recently shown that re-expression of SEMA3F indeed blocks tumor formation and appears to interfere with integrin-mediated activation of ILK and downstream effectors. We are currently searching for inhibitory agents that will mimic the SEMA3F effects and that will have efficacy in patients.

A family described in 1979 with hereditary renal cell and non-medullary thyroid cancer forms the foundation for a second major project in our lab group. Members of this family are segregating an apparently balanced translocation t(3;8)(p14.2;q24.1) and there is tight

concordance between the inheritance of the translocation and development of cancer. Moreover, the kidney cancers in this family are early onset, and frequently both bilateral and multi-focal, all hallmarks of hereditary disease. After many years of reverse genetics efforts, we cloned the chromosome 3 breakpoint using a YAC clone contig generated by us. Ultimately, we sequenced several 100 kb of this region and demonstrated its instability in cancer cells, but could not identify a gene. The FHIT gene was subsequently described from this region, but many aspects of its biology led us to suspect that the chromosome 8 breakpoint might also harbor an important gene in kidney cancer. Using RACE technology, we discovered that in cells bearing the t(3;8), FHIT was fused to a novel 8q24 gene we named TRC8. TRC8 encodes an ER-resident, multi-membrane spanning E3-ubiquitin ligase whose coding sequence was interrupted by the (3;8) translocation (in contrast to FHIT, where the break resides in the 5' UTR). TRC8 is an evolutionarily conserved protein with a close homologue in *Drosophila*. In a collaborative effort with Drs. Drabkin and Joan Hooper (Cellular and Structural Biology), we exploited the *Drosophila* model to gain an understanding of TRC8 functions. When expressed ectopically, TRC8 suppressed growth in a dose-dependent manner, consistent with a role as a tumor suppressor. We found that *Drosophila* Trc8 directly interacted with DVhl, a major gene in hereditary and sporadic kidney cancer, and these two proteins appeared to function in a common pathway in at least some tissues. We also found that both human and *Drosophila* TRC8 proteins interacted with the COP9 Signalosome subunit, JAB1/CSN5. Genetic manipulation of CSN5 demonstrated that Trc8-induced growth suppression was CSN5 dependent. Moreover, a mutation which reduced binding to Trc8 also restored growth. We discovered that the Trc8 binding site resided in the MPN domain of CSN5, leading us to test other MPN domain genes for interactions with Trc8. Of 10 MPN genes in *Drosophila*, 5 were able to genetically interact by affecting the DTrc8 phenotype. Several of the corresponding proteins have been shown to bind DTrc8. We subsequently developed Tet-inducible cell lines that express wild type and mutant human TRC8 proteins. Using these HEK-293 FlpIn cell lines, we have found that TRC8 blocks cell growth and appears to greatly slow or arrest cells at the G2/M phase of the cell cycle. This growth blockade is dependent upon the RING domain in TRC8's C-terminus, which is able to stimulate polyubiquitylation reactions similar to other RING proteins. In collaboration with Dan Chan, we have shown that tumorigenicity of these lines in *nude* mice is abrogated by induction of wild type but not RING mutant TRC8.

Remarkably, we have discovered over the past year that TRC8 is regulated by sterols and has the ability, in turn, to regulate cholesterol and lipid biosynthesis. It binds and destabilizes the INSIG proteins, which are negative regulators of lipid biosynthesis. In addition, it destabilizes the SREBP transcription factors directly responsible for inducing lipid biosynthesis. Knock-down of endogenous TRC8 using siRNA methods has yielded the opposite effects, as predicted if TRC8 were truly a physiological regulator of lipid homeostasis. Our data suggest that TRC8 has the potential to link two fundamental aspects of cellular physiology: cholesterol biosynthesis/metabolism and growth/cell cycle. Thus TRC8 is unique in the mammalian genome as a lipid-regulated E3 ubiquitin ligase and tumor suppressor. A major implication of this work is that drugs such as the statins, that starve cells for sterols, need to be tested in combination with chemotherapy or targeted biological therapies in cancer patients. Such drugs should reactivate TRC8 and help to inhibit tumor growth. Ongoing analyses in the mammalian system include investigation of the G2/M blockade, elucidation of the details of TRC8 regulatory mechanisms and identification of further substrates for its E3 ubiquitin ligase activity. Already we have identified mutations that abrogate some but not all the effects of TRC8. The investigation of these mutations will allow us to dissect the pathways through which growth inhibition is

achieved. Genetic analysis in fruit flies using a large set of overlapping chromosomal deficiencies has identified 14 regions with strong suppressors or enhancers. Identification of these genes will provide important insights into the upstream and downstream components of the TRC8 pathway. Among the human homologues of these genes, we may find some with mutations in kidney cancers. One overall goal is to shuttle between the fly and mammalian systems to define the TRC8 pathway and identify new targets for alteration in kidney cancer.

PUBLICATIONS:

1. Gemmill, R.M., Wessler, S.R., Keller, E.B., Calvo, J.M. leu operon of Salmonella typhimurium is controlled by an attenuation mechanism. Proc. Natl. Acad. Sci. USA, 76:4941-4945 (1979).
2. Hertzberg, K.M., Gemmill, R.M., Jones, J., Calvo, J.M. Cloning of an EcoRI-generated fragment of the leucine operon of Salmonella typhimurium. Gene, 8:135-152 (1980).
3. Gemmill, R.M., Jones, J.W., Haughn, G.W., Calvo, J.M. Transcription initiation sites of the leucine operons of Salmonella typhimurium and Escherichia coli. J. Mol. Biol., 170:39-59 (1983).
4. Doane, W. W., Treat-Clemons, L. G., Gemmill, R. M., Levy, J. N., Hawley, S. A., Buchberg, A. M., and Paigen, K. (1983). Genetic mechanism for tissue-specific control of alpha-amylase expression in Drosophila melanogaster. Isozymes Curr Top Biol Med Res 9, 63-90.
5. Gemmill, R.M., Tripp, M., Friedman, S., Calvo, J.M. Promoter mutation causing catabolite repression of the Salmonella typhimurium leucine operon. J. Bact., 158:948-953 (1984).
6. Gemmill, R.M., Levy, J.N., Doane, W.W. Molecular cloning of a-Amylase genes from Drosophila melanogaster. I. Clone isolation by use of a mouse probe. Genetics, 110:299-312 (1985).
7. Levy, J.N., Gemmill, R.M., Doane, W.W. Molecular cloning of a-Amylase genes from Drosophila melanogaster. II. Clone organization and verification. Genetics, 110:313-324 (1985).
8. Hecht, F., Morgan, R., Gemmill, R.M., Hecht, B.K., Smith, S.D. Translocations in T-cell leukemia and lymphoma. New Engl. J. Med., 313:758-759 (1985).
9. Gemmill, R.M., Hamblin, M., Glaser, R.L., Racioppi, J.V., White, B.N., Calvo, J.M., Hagedorn, H.H. Isolation of mosquito vitellogenin genes and induction of expression by 20-hydroxyecdysone. Insect Biochemistry, 16:761-774 (1986).
10. Racioppi, J.V., Gemmill, R.M., Kogan, P.H., Calvo, J.M., Hagedorn, H.H. Expression and regulation of vitellogenin messenger RNA in the mosquito, Aedes aegypti. Insect Biochemistry, 16:255-262 (1986).

11. Haughn, G.W., Gemmill, R.M., Wessler, S.R., Calvo, J.M. High AT base pair content conserved in DNA sequences upstream of leu ABCD in Escherichia coli and Salmonella typhimurium. *J. Bacteriology*, 166:1113-1117 (1986).
12. Glover, T.W., Coyle-Morris, J., Pearce-Berge, L., Berger, C., Gemmill, R.M. DNA demethylation induced by 5-azacytidine does not affect fragile X expression. *Am. J. Hum. Genet.*, 38:309-318 (1986).
13. McPeck, F.P., Coyle-Morris, J., Gemmill, R.M. Separation of large DNA molecules by modified pulsed field gradient gel electrophoresis. *Anal. Biochem.*, 156:274-285 (1986).
14. Sandberg, A.A., Gemmill, R.M., Hecht, B., Hecht, F. The Philadelphia chromosome: A model of cancer and molecular cytogenetics. *Cancer Genetics Cytogenet.*, 21:129-146 (1986).
15. Gemmill, R.M., Schwartz, P.E., Doane, W.W. Structural organization of the Amy locus in seven strains of Drosophila melanogaster. *Nucleic Acids Res.*, 14:5337-5352 (1986).
16. Gemmill, R.M., Pearce-Birge, L., Bixenman, H., Hecht, B.K., Alanson, J.E. Y chromosome specific DNA sequences in Turner syndrome mosaics. *Am. J. Hum. Genet.*, 41:157-167 (1987).
17. Gemmill, R.M., Coyle-Morris, J.F., McPeck, F.D., Jr., Ware-Urbe, L.F., Hecht, F. Construction of long-range restriction maps in human DNA using pulsed field gel electrophoresis. *Genet. Anal. Tech.*, 4:119-131 (1987).
18. Doane, W. W., Gemmill, R. M., Schwartz, P. E., Hawley, S. A., and Norman, R. A. (1987). Structural organization of the alpha-amylase gene locus in Drosophila melanogaster and Drosophila miranda. *Isozymes Curr Top Biol Med Res* 14, 229-266.
19. Allanson, J.E., Gemmill, R.M., Hecht, B.K., Johnsen, S., Wenger, D.A. Deletion mapping of the β -glucuronidase gene. *Am. J. Med. Genet.*, 29:517-522 (1988).
20. Smith, D.S., Morgan, R., Gemmill, R.M., Amylon, M.D., Link, M.P., Linker, C., Hecht, B.K., Warnke, R., Glader, B.E., Hecht, F. Clinical and biologic characterization of T-cell neoplasias with rearrangements of chromosome 7 band q34. *Blood*, 72:395-402 (1988).
21. Sandberg, A.S., Turc-Carel, C., Gemmill, R.M. Perspectives in Cancer Research. Chromosomes in solid tumors and beyond. *Canc. Res.*, 48:1049-1059 (1988).
22. Tajara, E.H., Berger, C.S., Hecht, B.K., Gemmill, R.M., Sandberg, A.S., Hecht, F. Loss of common 3p14 fragile site expression in renal cell carcinoma with deletion breakpoint at 3p14. *Canc. Genet. Cytogenet.*, 31:75-82 (1988).
23. Glover, T.W., Coyle-Morris, J.F., Li, F.P., Brown, R.S., Berger, C.S., Gemmill, R.M., Hecht, F. Translocation of t(3;8)(p14.2;q24.1) affects expression of the common fragile site at 3p14(FRA3B) in lymphocytes. *Canc. Genet. Cytogenet.*, 31:69-73 (1988).

24. Gemmill, R.M., Coyle-Morris, J., Ware-Uribe, L., Pearson, N., Hecht, F., Brown, R.S., Li, F.P., Drabkin, H.A. A 1.5 megabase restriction map surrounding c-MYC does not include the translocation breakpoint in familial renal cell carcinoma. *Genomics*, 4:28-35 (1989).
25. Drabkin, H.A., Sage, M., Helms, C., Greer, P., Gemmill, R.M., Smith, D., Erickson, P., Hart, I., Ferguson-Smith, A., Ruddle, F., Tommerup, N. Regional and physical mapping studies characterizing the Greig Polysyndactyly 3;7 chromosome translocations, t(2;7)(p21.1;p13). *Genomics*, 4:518-529 (1989).
26. Drabkin, H.A., Smith, D., Jones, C., Jonsen, M., Sage, M., Gold, S., Glover, T., Dobrovic, A., Bradley, W.E.C., Gemmill, R. Regional and physical mapping studies involving rearrangements of human chromosome 3. In: Cancer Cells, Molecular Diagnostics of Human Cancer, Furth, M. and Greaves, M. (eds.). Cold Spring Harbor Press (1989).
27. Decker, H.J., Gemmill, R.M., Neumann, H.P., Walter, T.A., Sandberg, A.A. Loss of heterozygosity on 3p in a renal cell carcinoma in von Hippel-Lindau syndrome. *Cancer Genet. Cytogenet.* 39:289-293 (1989).
28. Paulien, S., Turc-Carel, C., Cin, P.D., Jani-Sait, S., Sreekantaiah, C., Leong, S.P.L., Vogelstein, B., Kinzler, K.W., Sandberg, A.A., Gemmill, R.M. Myxoid liposarcoma with t(12;16)(q13;p11) contains site-specific differences in methylation patterns surrounding a zinc-finger gene mapped to the breakpoint region on chromosome 12. *Canc. Res.*, 50:7902-7907 (1990).
29. Mendez, M.J., Klapholz, S., Brownstein, B.H., Gemmill, R.M. Rapid screening of a YAC library by pulsed-field gel southern blot analysis of pooled YAC clones. *Genomics*, 10:661-665 (1991).
30. Gemmill, R.M., Varella-Garcia, M., Smith, D.I., Erickson, P., Golembieski, W., Miller, Y., Coyle-Morris, J., Tommerup, N., Drabkin, H. A 2.5-Mb physical map within 3p21.1 spans the breakpoint associated with Greig cephalopolysyndactyly syndrome. *Genomics*, 11:93-102 (1991).
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