BIOGRAPHICAL SKETCH

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NAME: James M. Ford, M.D.

eRA COMMONS USER NAME (credential, e.g., agency login): FORD.JAMES

POSITION TITLE: Professor of Medicine, Pediatrics and Genetics

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Yale University	B.A.	1984	Biology
Yale University School of Medicine	M.D.	1989	Medicine
Stanford University School of Medicine	Fellow	1994	Medical Oncology
Stanford University	Postdoc	1997	Biological Sciences

A. Personal Statement

My overall research goals are to understand the role of genetic changes in cancer genes in the risk, development and treatment of solid tumors. My laboratory research focuses on how DNA repair and DNA damage response pathways are critical to tumorigenesis and are potential candidates for targeted therapeutics and prevention. A major focus is the characterization of DNA repair defects in solid tumors, and the synergistic activity of DNA damaging chemotherapy drugs and radiation with PARP inhibitors in breast and GI cancers. We are highly engaged in the translation of these ideas to the clinic through clinical trials in patients with high genetic risk for cancer. I direct the Stanford Cancer Genetics Clinic, where with a team of cancer genetic counselors, we see patients for genetic counseling and germline testing of hereditary cancer syndromes, and enter patients on clinical research protocols for prevention, early diagnosis and treatment of cancer in high–risk individuals. I also direct the Stanford Molecular Tumor Board for analysis and targeted therapy of cancer patients through somatic tumor genomic profiling. I have extensive experience in training and mentoring students, fellows and junior faculty members in biomedical sciences and translational research, and many former students and fellows have gone onto faculty positions at prestigious academic centers. In summary, I have a demonstrated record of successful laboratory and translational clinical research, and a track record of mentoring physician–investigators into independent academic careers.

B. Positions and Honors

Professional Positions

1989 – 91 Intern and Resident, Internal Medicine, Stanford University Medical Center
1991 – 94 Clinical Fellow, Medical Oncology, Stanford University Medical Center (under Saul Rosenberg, MD)
1993 – 97 Research Fellow, Dept. of Biological Sciences, Stanford University (with Prof. Phil Hanawalt)
1998 – 06 Assistant Professor of Medicine (Oncology) and Genetics, Stanford Univ. Medical School
1999 – Director, Stanford Cancer Genetics Program and Clinic
2002 – 15 Director, Stanford Medical Oncology Fellowship Training Program
2003 – 06 Assistant Professor of Pediatrics (by courtesy), Division of Medical Genetics
2006 – Associate Professor of Medicine (Oncology), Pediatrics (Medical Genetics) and Genetics (tenured)
2013 – Director, Stanford Clinical Cancer Genomics Program
2016 – Professor of Medicine (Oncology), Pediatrics (Medical Genetics) and Genetics (tenured)

Other Experience and Professional Activities

2003 – 10 California Breast Cancer Research Program (Council Member 03 – 06; 08 – 11; Chair 09 – 10)
2003 – V Foundation Scientific Review Committee
2006 – 14 ASCO Education Committees, Tumor Biology Track Leader (08); Cancer Genetics (11 – 14)
2011 – 14 ASCO Scientific Program Committee, Tumor Biology Track
2011 – 14 Conquer Cancer Foundation (ASCO) Grants Selection Committee
2003 – 16 Reviewer, DOD Breast Cancer Research Program, Chair (12); Gastric Cancer Research Chair (2015)
2002 – 10 Reviewer, Susan G. Komen Breast Cancer Foundation Research Grant Program (Chair 08 – 10)
2008 – 10 NIH/NCI Sub–Committee E – Epidemiology/Prevention Program Project Grant Study Section
2009 – 13 NIH/NCI Sub–Committee I – Career Development Grant Study Section (Member)
2010 – 16 NIH/NCI Sub–Committee A – Cancer Centers Site Visits
2014 – 15 NIH/NCI Omnibus Grants (R03/R21); NIH/NCI SPORE Grants
2003 – Editorial Boards: *Cancer Research*, *DNA Repair*, *PLoS Genetics*
2016 – Founding Editor–in–Chief, JCO Precision Oncology
2005 – NCCN Guidelines Committees: Colorectal Cancer Screening; Genetics/Familial High Risk Assessment
2010 – 13 Chair, ASCO Oncology Training Program Directors Subcommittee
2013 – 16 Member, ASCO Cancer Prevention Committee, Cancer Research Committee

Honors and Awards

1984 B.A., *Magna Cum Laude*, Yale University
1995 NIH K08 Clinical Investigator Award
1999 Sidney Kimmel Foundation for Cancer Research Scholar Award
1999 Doris Duke Foundation Clinical Scientist Award in Cancer Etiology and Pathogenesis
2000 Burroughs Wellcome Fund New Investigator Award in Toxicological Sciences
2002 V Foundation Translational Research Award
2005 Evelyn Lauder Breast Cancer Research Foundation Scholar
2007 Member, Western Society for Clinical Investigation
2009–13 Visiting Professorships: Oxford (07); Hong Kong Univ. (09)

C. Contribution to Science (Out of a total of 160 Publications)

1. ***Pharmacology of Cancer Drug Resistance*:** My early work in cancer research focused on the pharmacology of multidrug resistance to cancer chemotherapy in tumor cell lines. I defined a number structural rules and identified small–molecules that inhibit the MDR P–glycoprotein pump and sensitize drug resistant cancers to chemotherapy.

    - Ford JM, Prozialeck WC, and Hait WN. Structural features that determine activity for phenothiazines and related drugs for inhibition of cell growth and reversal of multidrug resistance. *Mol. Pharmacol.* 35: 105 – 115 (1989). PMID: 2563302
    - Ford JM and Hait WN. Pharmacology of drugs that alter multidrug resistance in cancer. *Pharmacol. Rev.* 42:155 (1990). PMID: 2217530
    - Ford JM, Bruggeman EP, Pastan I, Gottesman MM, and Hait WN. Cellular and biochemical characterization of thioxanthenes for reversal of multidrug resistance in human and murine cell lines. *Cancer Res.* 50: 1748 – 1756 (1990). PMID: 1968358

2. ***DNA Repair:*** As a postdoctoral research fellow with Professor Phil Hanawalt at Stanford, I discovered that the p53 tumor suppressor gene regulates global genomic nucleotide excision repair (NER) but not transcription–coupled DNA repair. During my early faculty years, I showed that p53 acts as a transcription factor to induce expression of XPE and XPC NER genes following DNA damage, and these factors directly bind damage and induced the repair response, providing a mechanistic basis for this observation.

    - Ford JM and Hanawalt PC. Li–Fraumeni syndrome human skin fibroblasts homozygous for p53 mutations are deficient in global nucleotide excision repair, but exhibit normal transcription–coupled repair and UV resistance. *Proc. Natl. Acad. Sci.* 92: 8876–8880 (1995). PMCID: PMC41070

- Ford JM and Hanawalt PC. Expression of wild–type p53 is required for efficient nucleotide excision repair in UV–irradiated human fibroblasts. *Journal of Biological Chemistry* 272: 28073–28080 (1997). PMID: 9346961
- Hwang BJ, Ford JM, Hanawalt PC and Chu G. Expression of the p48 xeroderma pigmentosum gene is p53–dependent and is involved in global genomic repair. *Proc. Natl. Acad. Sci.* 96:424–428 (1999). PMCID: PMC15152
- Adimoolam S and Ford JM. p53 and DNA damage inducible expression of the xeroderma pigmentosum group C gene. *Proc. Natl. Acad. Sci.* 99: 12985–12990 (2002). PMCID: PMC130573
- Fitch ME, Cross I and Ford JM. p53 responsive nucleotide excision repair gene products p48 and XPC, but not p53, localize to sites of UV–irradiation induced DNA damage. *Carcinogenesis* 24: 843 – 850 (2003). PMID: 12771027

3. ***DNA Repair Pathways and Targeted Therapies:*** My faculty research has focused on genes involved in hereditary cancer susceptibility and their role in DNA repair. We identified specific DNA repair deficiencies associated with mutations in *BRCA1, BRCA2, p53* and others, and spearheaded translational studies to target these with DNA damaging agents and inhibitors of DNA repair pathways, such as PARP inhibitors. Furthermore, we have performed high–throughput screens that identified small–molecules that enhance DNA repair in a *BRCA1*–deficient background, and are developing candidate molecules in preclinical models for prevention of breast and ovarian cancer.

   - Hartman AR and Ford JM. BRCA1 induces DNA damage recognition factors and enhances nucleotide excision repair. *Nature Genetics* 32:180 – 184 (2002). PMID: 12195423
   - Alli E, arma VB, Sunderesakumar P and Ford JM. Defective repair of oxidative DNA damage in triple–negative breast cancer confers sensitivity to inhibition of poly(ADP–ribose) polymerase. *Cancer Research* 69: 2589 – 96 (2009). PMCID: PMC2681413
   - Hastak K, Alli E and Ford JM. Synergistic chemosensitivity of triple–negative breast cancer cell lines to PARP inhibition, gemcitabine and cisplatin. *Cancer Res.* 70: 7970 – 80 (2010). PMCID: PMC2955854
   - Alli E, Solow–Codero DE, Casey SC and JM Ford. Therapeutic targeting of BRCA1–mutated breast cancers with agents that activate DNA repair. *Cancer Research* 74: 6205 – 15 (2014). PMCID: PMC4388430
   - Alli E and JM Ford. BRCA1: Beyond double–strand break repair. *DNA Repair* **32:** 165 – 171 (2015). 32:165–71. PMID: 25956865
   - Telli ML, Jensen KC, Vinayak S, Kurian AW, Lipson JA, Flaherty P, Timms K, Abkevich V, Schackmann EA, Wapnir I, Carlson RW, Sparano JA, Head B, Goldstein LJ, Haley B, Dakhil SR, Reid JE, Hartman AR, Manola J and JM Ford. Phase II study of gemcitabine, carboplatin and iniparib as neoadjuvant therapy for triple–negative and *BRCA1/2* mutation–associated breast cancer with assessment of a tumor–based measure of genomic instability: PrECOG 0105. *J Clin Oncol* **33:** 1895 – 1901 (2015). PMCID: PMC4451172

4. ***Clinical Cancer Genetics:*** I direct the Stanford Cancer Genetics Clinic, where with a team of cancer genetic counselors, we see patients for genetic counseling and germline testing of hereditary cancer syndromes, and enter patients on clinical research protocols for prevention, early diagnosis and treatment of cancer in high–risk individuals. We have identified and reported on many clinical cancer genetics syndromes and contributed to the diagnosis, screening and early–detection of hereditary breast, ovarian, gastric, colon cancer and others.

   - Ford JM and Whittemore A. Prediction and prevention of hereditary colorectal cancer. *JAMA* 296:1521–1523 (2006). PMID: 17003401
   - Norton JA, Van Dam J, Jeffrey RB, Longacre TA, Huntsman DG, Kurian AW, Chun N and Ford JM. CDH1 truncating mutations in the E–cadherin gene: an indication for total gastrectomy to treat hereditary diffuse gastric cancer. *Annals of Surgery* 245: 873 – 879 (2007). PMCID: PMC1876967
   - Kurian AW, Gong GD, Chun NM, Mills MA, Staton AD, Kingham KE, Crawford BA, Lee R, Chan S, Donlon SS, Ridge Y, Panabaker K, West DW, Whittemore AS, and Ford JM. BRCA1 and BRCA2 mutations and predictive model performance in Asian–Americans. *J. Clin. Onc.* 26: 4752 – 58 (2008). PMCID: PMC2653135
   - Ladabaum U, Wang G, Terdiman J, Blanco A, Kuppermann M, Boland R, Ford J, Elkin E and K Phillips. Strategies to identify the Lynch syndrome among patients diagnosed with colorectal cancer: a cost–effectiveness analysis. *Annals of Internal Medicine* 155: 69 – 79 (2011). PMCID: PMC3793257

- Robson ME, Bradbury AR, Arun B, Domchek SM, Ford JM, Hampel HL, Lipkin SM, Syngal S, Wollins DS, Lindor NM. American Society of Clinical Oncology Policy Statement Update: Genetic and Genomic Testing for Cancer Susceptibility. *J Clin Oncol* **33:** 3660 – 7 (2015). PMID: 26324357

5. ***Cancer Genomics and Precision Oncology:*** I am highly involved in clinical research studying the role of next–generation DNA sequencing in germline and somatic tumor analyses of individuals with personal and family histories of cancers. I published the first report of using cancer gene panels to identify significant additional risk alleles in high–risk breast cancer families lacking a BRCA1/2 mutation. I am involved in whole–genome sequencing projects to define risk in healthy and syndromic families.
   - Kurian AW, Hare EE, Mills MA, Kingham KE, McPherson L, Whittemore AS, McGuire V, Ladabaum U, Kobayashi Y, Lincoln SE, Cargill M and JM Ford. Clinical evaluation of a multiple–gene sequencing panel for hereditary cancer risk assessment. *J Clin Oncology* **32:** 2001 – 09 (2014). PMCID: PMC4067941
   - Dewey FE, Grove ME, Pan C, Goldstein BA, Bernstein JA, Chaib H, Merker JD, Goldfeder RL, Enns G, David SP, Pakdaman N, Ormond KE, Caleshu C, Kingham K, Klein TE, Whirl–Carillo M, Sakamoto K, Wheeler MT, Butte AJ, Ford JM, Boxer L, Ioannidis JPA, Yeung AC, Altman RA, Assimes TL, Snyder M, Ashley EA and T Quertermous. Clinical interpretation and implications of whole–genome sequencing. *JAMA* **311:** 1035 – 1044 (2014). PMCID: PMC4119063
   - Watkins JA, Weekes D, Shah V, Gazinska P, Joshi S, Sidhu B, Gillett C, Pinder S, Vanoli F, Jasin M, Mayrhofer M, Isaksson A, Cheang MCU, Mirza H, Frankum J, Lord CJ, Ashworth A, Vinayak S, Ford JM, Telli ML, Grigoriadis A and ANJ Tutt. Genomic complexity profiling reveals that HORMAD1 overexpression contributes to homologous recombination deficiency in triple–negative breast cancers. *Cancer Discovery* **5:** 488 – 505 (2015). PMCID: PMC4490184
   - Haslem DS, Van Norman SB, Fulde G, Knighton AJ, Belnap T, Butler AM, Rhagunath S, Newman D, Gilbert H, Tudor BP, Lin K, Stone GR, Loughmiller DL, Mishra PJ, Srivastava R, Ford JM, Nadauld LD. A Retrospective Analysis of Precision Medicine Outcomes in Patients With Advanced Cancer Reveals Improved Progression–Free Survival Without Increased Health Care Costs. J Oncol Pract. 2016 Sep 6. pii: JOPR011486. [Epub ahead of print]

Complete List of Published Work (160) in MyBibliography:
http://www.ncbi.nlm.nih.gov/sites/myncbi/1tUvqjs3oz6Az/bibliography/47772914/public/?sort=date&direction=descending

D. Additional Information: Research Support and/or Scholastic Performance

Ongoing Research Support

Goldberg Ford, Sidow, Diehn (Co–PI) 08/01/15 – 07/31/17
BRCA Gene Foundation
Understanding BRCA1– and BRCA2–Associated Cancers
Role: Co–PI

The Breast Cancer Research Foundation Ford (PI) 10/01/05 – 09/30/17
Improving Breast Cancer Genetic Risk Assessment
Major Goal: Explore the use of multigene panels in identifying hereditary breast cancer susceptibility genes
Role: PI

Stanford Cancer Institute Rao, Ford (Co–PI) 01/05/15 – 01/02/17
Translational Research Award
Pre–clinical PET Imaging and validation of PARP–1 activity as the biomarker for patient stratification and treatment monitoring
Role: Co–PI

B027952 Ford (PI) 11/09/12 – 11/30/16
Genentech, Inc.
A Randomized, Multicenter, Adaptive Phase II/III Study to Evaluate the Efficacy and Safety of Trastuzumab Emtansine (T–DM1) Versus Taxane (Docetaxel or Paclitaxel) in Patients with Previously Treated Locally

Advanced or Metastatic HER2–Positive Gastric Cancer, Including Adenocarcinoma of the Gastroesophageal Junction
Role: PI

Myriad Genetics, Inc. Ford (PI) 05/15/14 – 05/14/17
Hereditary Cancer Panel Study
Major Goal: Determine the utility of multigene panels for diagnosing hereditary cancer risk in a cancer genetics clinic.
Role: PI

D081FC00001 Ford (PI) 02/11/15 – 02/28/17
Astra Zeneca
A Phase III, Randomised, Double Blind, Placebo Controlled, Multicentre Study of Maintenance Olaparib Monotherapy in Patients with gBRCA Mutated Metastatic Pancreatic Cancer whose Disease Has Not Progressed on First Line Platinum Based Chemotherapy.
Role: PI

ML28897 Ford (PI) 06/18/15 – 03/31/20
Genentech, Inc.
My Pathway: an open–label phase IIA study evaluating trastuzumab/pertuzumab, erlotinib, vemurafenib, and vismodegib in patients who have advanced solid tumors with mutations or gene expression abnormalities predictive of response to one of these agents.
Role: PI

D2015017 Ford (PI) 09/01/15 – 08/31/17
The V Foundation
Sequencing the Gastric Cancer Tumor Genome
Role: PI

D2015005 Ford (PI) 09/01/15 – 08/31/17
The V Foundation
Gastric Cancer Registry
Role: PI

Natera, Inc. Ford (PI) 09/24/15 – 09/23/19
Prospective collection of samples to enable the validation of circulating DNA biomarkers for the early detection of ovarian cancer.
Role: PI

Completed Research Support

3U54CA13646505S2 Contag (PI) 09/22/08 – 08/31/14
National Institutes of Health
Multimodality Imaging of GI Cancers for Diagnosis and Directed Therapy
Role: Co–PI

Myriad Genetics Inc. Ford (PI) 01/01/13 – 12/31/13
Myriad Homologous Recombination Deficiency Assay
Role: PI