BIOGRAPHICAL SKETCH

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NAME: Cole, Peter David

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POSITION TITLE: Professor of Pediatrics; Chief, Division of Pediatric Hematology and Oncology

EDUCATION/TRAINING

INSTITUTION AND LOCATION	DEGREE	Completion Date	FIELD OF STUDY
Cornell University, Ithaca, NY	BS	05/1989	Neurobiology & Behavior
Cornell Univ. Medical College, New York, NY	MD	05/1993	Medicine
Mount Sinai Medical Center, New York, NY		06/1996	Pediatric Residency
New York Hospital/Memorial Sloan Kettering Cancer Center, New York, NY		06/1999	Fellowship, Pediatric Hematology & Oncology

A. Personal Statement

As Chief of the Division of Pediatric Hematology and Oncology, and Director of the New Jersey Center for Pediatric Cancer and Blood Disorders Research at the Rutgers Cancer Institute of New Jersey, I am committed to improving outcomes for children with cancer or blood disorders through a coordinated program of clinical and laboratory-based research. My current clinical research program focuses on improving curative therapy for children and young adults with hematologic malignancies (leukemia or lymphoma). I am the chair of the Children's Oncology Group Hodgkin Disease (HD) Salvage Therapy Subcommittee, charged with developing novel therapeutic approaches for patients with relapsed or refractory HD, in order to increase cure rates while decreasing long-term toxicity. My laboratory and translational research focuses on reducing the neurotoxic effects of cancer therapy. Our translational studies of children being treated for acute lymphoblastic leukemia are identifying biomarkers within cerebrospinal fluid that relate to clinical toxicity and genetic varients that confer increased susceptibility. In addition, we developed a rat model of chemotherapy-related cognitive impairment to shed light on the underlying pathophysiology and to test protective interventions. I am also fully committed to the academic development of the next generation of innovative scientists and clinicians, and am specifically eager to support trainees from underrepresented backgrounds. In addition to mentoring my junior faculty, I have mentored a diverse group of students, residents, and fellows in clinical and laboratory research. While at Albert Einstein College of Medicine, I developed the curriculum for the Pediatric Fellows Research Course, and was named mentor of the year by the Clinical Research Training Program. Previous trainees have continued on to successful careers in both industry and academic medicine, indicated by manuscript publication, grant funding, and faculty appointments.

Ongoing and recently completed projects that I would like to highlight include the following:

NIH/NCI R01CA240360 Cole (MPI) 02/01/2020-01/31/2025
Characterization of brain dysfunction during development in survivors of childhood acute lymphoblastic leukemia

NIH/NCI R01- CA220568 Cole (MPI) 09/17/2018-08/31/2023
Identifying children with neurocognitive decline and susceptibility to oxidative damage during therapy for ALL

NIH/NCI R01- CA182284 Cole (MPI) 06/01/2014-06/30/2020
Pathophysiology of Chemotherapy-Induced Cognitive Deficits in Juvenile Rats

Citations:

1. Wen J, Patel C, Diglio F, Baker K, Marshall G, Li S, Cole PD. Cognitive impairment persists at least 1 year after juvenile rats are treated with methotrexate. Neuropharmacology. 2022. Available online January 2, 2022; https://doi.org/10.1016/j.neuropharm.2021.108939 PMCID: PMC8792316
2. Wen J, Maxwell RR, Wolf AJ, Spira M, Gulinello ME, Cole PD. Methotrexate causes persistent deficits in memory and executive function in a juvenile animal model. Neuropharmacology. 2018; 139:76-84. https://doi.org/10.1016/j.neuropharm.2018.07.007. PMID: 29990472

3. Cole PD, Finkelstein Y, Stevenson KE, Blonquist TM, Vijayanathan V, Silverman LB, et al. Polymorphisms in Genes Related to Oxidative Stress Are Associated with Inferior Cognitive Function after Therapy for Childhood Acute Lymphoblastic Leukemia. Journal of Clinical Oncology. 2015; 33(19):2205-11. PMID: 25987702
4. Cole PD, Vijayanathan V, Ali NF, Wagshul ME, Tanenbaum EJ, Price J, Dalal V, Gulinello ME. "Memantine protects rats treated with intrathecal methotrexate from developing spatial memory deficits," Clinical Cancer Research 2013: 19(16):4446-54. PMID: 23833301.

B. Positions, Scientific Appointments, and Honors

Academic Appointments

1999-2007 Assistant Professor, Departments of Pediatric Hematology/Oncology and Pharmacology; Damon Runyon-Lilly Clinical Investigator. University of Medicine and Dentistry of New Jersey, New Brunswick, NJ

2007–2018 Associate Professor of Pediatrics, Section of Pediatric Hematology/Oncology, Albert Einstein College of Medicine, The Children's Hospital at Montefiore, Bronx, NY

2018-present Professor of Pediatrics, with tenure; Embrace Kids Endowed Chair; Chief, Division of Pediatric Hematology/Oncology, Rutgers Cancer Institute of New Jersey, Robert Wood Johnson Medical School; Director of the New Jersey Center for Pediatric Cancer and Blood Disorders Research at the Rutgers Cancer Institute; Director of Pediatric Hematology, Oncology, and Cellular Therapies at the Bristol Myers Squibb Children's Hospital at Robert Wood Johnson University Hospital, New Brunswick, NJ

Selected Awards and Other Professional Activities

2006-present Dana Farber Cancer Institute ALL Consortium, Member and Site PI
2009 Henry L Moses Award, Clinical Research Paper, Montefiore Medical Center
2009-2018 Pediatric Fellows' Research Conference, Co-Coordinator
2010-2018 Einstein Institutional Review Board, Vice Chair
2010-present Children's Oncology Group, Hodgkin Disease Retrieval Study Subcommittee, Chair
2012 Einstein Clinical Research Training Program Mentor of the Year
2016-present Children's Oncology Group, Hodgkin Disease Committee Vice-Chair
2018-present Embrace Kids Foundation Endowed Chair in Pediatric Hematology and Oncology
2021-present Director, New Jersey Center for Pediatric Cancer and Blood Disorders Research at RCINJ

C. Contributions to Science

C. Contributions to Science

1. Through both investigator-initiated studies and participation in the Dana Farber Cancer Institute ALL Consortium, I have contributed to clinical research improving therapy for children with acute lymphoblastic leukemia.
    a. Burns MA, Place AE, Stevenson KE, Gutiérrez A, Forrest S, Vrooman LM, Harris MH, Hunt SK, O'Brien JE, Asselin BL, Athale UH, Clavell LA, **Cole PD**, Gennarini LM, Kahn JM, Kelly KM, Laverdierre C, Leclerc J-M, Michon B, Schorin MA, Sulis ML, Welch JJG, Neuberg DS, Sallan SE, Silverman LB. Identification of Prognostic Factors in Childhood T-cell Acute Lymphoblastic Leukemia: Results from DFCI ALL Consortium Protocols 05-001 and 11-001. Pediatric Blood & Cancer. 2021; 68:e28719. PMID: 33026184
    b. Song Y, Zhu Q, **Cole PD**, Stevenson K, Harris M, Schultz E, Kahn JM, Ladas EJ, Athale UH, Clavell L, Laverdiere C, Leclerc J-M, Michon B, Welch JJG, Sallan SE, Silverman LB, Kelly K. Genetic Ancestry and Skeletal Toxicities among Childhood Acute Lymphoblastic Leukemia Patients in the DFCI 05-001 Cohort. Blood Advances 2020; 5(2):451-8.
    c. Vrooman LM, Blonquist TM, Harris MH, Stevenson KE, Place AE, Hunt SK, O'Brien JE, Asselin BL, Athale UH, Clavell LA, **Cole PD**, Kelly KM, Laverdiere C, Leclerc J-M, Michon B, Schorin MA, Sulis ML, Welch JJG, Neuberg DS, Sallan SE, Silverman LB. Refining Risk Classification in Childhood B Acute Lymphoblastic Leukemia: Results of DFCI ALL Consortium Protocol 05-001. Blood Advances, 2018; 2 (12), 1449-1458. PMID: 29941458
    d. **Cole PD**, Drachtman RA, Masterson M, Smith AK, Glod J, Zebala JA, Lisi S, Drapala D-A, Kamen BA. Phase IIB Trial Of Aminopterin In Multiagent Therapy For Children With Newly Diagnosed Acute Lymphoblastic Leukemia at High Risk of Relapse. Cancer Chemotherapy and Pharmacology, 2008; 62:65-75. PMID: 26879921

2. Through the Children's Oncology Group, I have led clinical trials to improve therapy for children, adolescents, and young adults with relapsed or refractory Hodgkin Lymphoma. Through successive clinical trials, we have demonstrated the efficacy and tolerability of novel approaches that maintain high response rates while using agents chosen to minimize long-term toxicity. I served as the study chair for two trials (AHOD0321 and AHOD1221) and vice-chair for three (AHOD0831, AHOD0521, and AHOD1721).
    a. Kelly KM, **Cole PD**, Pei Q, Bush R, Roberts KB, Hodgson DC, McCarten KM, Cho SY, Schwartz S. Response Adapted Therapy for the Treatment of Children with Newly Diagnosed High Risk Hodgkin Lymphoma

(AHOD0831): A Report from the Children's Oncology Group. British Journal of Haematology. 2019;187(1): 39-48. HTTPS://doi.org/10.1111/bjh.16014.

b. **Cole PD**, McCarten KM, Pei Q, Spira M, Metzger ML, Drachtman RA, Horton TM, Bush R, Blaney SM, Weigel BJ, Kelly KM. Brentuximab vedotin with Gemcitabine for Pediatric and Young Adult Patients with Relapsed or Refractory Hodgkin Lymphoma: A Children's Oncology Group, Single-arm, Phase 1/2 Trial. The Lancet Oncology, 2018; 19(9):1229-1238. PMID: 30122620

c. Harker-Murray P, Drachtman RA, Hodgson DC, Chauvenet AR, Kelly KM, **Cole PD**. Stratification of treatment intensity in relapsed pediatric Hodgkin lymphoma. Pediatric Blood & Cancer, 2014; 61(4): 579-586. PMID: 24504790

d. **Cole PD**, Schwartz CL, Drachtman RA, de Alarcon PA, Chen L, Trippett T. Phase 2 Study of Weekly Gemcitabine and Vinorelbine for Children with Recurrent or Refractory Hodgkin Disease, A Children's Oncology Group Report. Journal of Clinical Oncology. 2009; 27(9): 1456-61.

3. Spurred by the complaints of patients about the toxic sequelae of leukemia therapy, I have conducted translational studies of biomarkers in blood and spinal fluid collected during leukemia therapy. The objective of these studies is to define models predictive of treatment-related toxicity and to shed light on the responsible pathophysiology. We demonstrated the changes in folate physiology within the central nervous system during therapy for leukemia. We also described an effective intervention, oral dextromethorphan, to improve symptoms among patients experiencing severe neurotoxicity. Most recently, we identified variants in genes related to oxidative stress and neuroinflammation that are associated with increased risk of cognitive impairment after treatment for childhood leukemia and a gene variant associated with bone toxicity. I served as the primary investigator or co-investigator in all of these studies.

   a. Williams ALM, **Cole PD**. Biomarkers of Cognitive Impairment in Pediatric Cancer Survivors. Journal of Clinical Oncology. 2021; in press.

   b. **Cole PD,** Finkelstein Y, Stevenson KE, Blonquist TM, Vijayanathan V, Silverman LB, et al. Polymorphisms in Genes Related to Oxidative Stress Are Associated with Inferior Cognitive Function after Therapy for Childhood Acute Lymphoblastic Leukemia. Journal of Clinical Oncology. 2015: published online 18 May 2015; doi: 10.1200/JCO.2014.59.0273

   c. **Cole PD,** Beckwith KA, Vijayanathan V, Roychowdhury S, Kamen BA. CSF Folate Homeostasis During Therapy for Acute Lymphoblastic Leukemia. Pediatric Neurology 2009; 40(1): 34-41.

   d. Drachtman RA, **Cole PD**, Golden CB, James SJ, Melnyk S, Aisner J, Kamen BA. Dextromethorphan is effective in the treatment of subacute methotrexate neurotoxicity. Pediatric Hematology and Oncology, 2002; 19(5): 319-327.

4. To further understand chemotherapy-induced cognitive decline, we established a rodent model of methotrexate-induced memory deficits. Using clinically relevant doses of systemic or intrathecal methotrexate, we have characterized the resulting memory deficits with a battery of behavioral tests. Using this model, we have demonstrated the impact of methotrexate on normal folate physiology in the central nervous system and the contribution of pathologic signaling through glutamate receptors to methotrexate-induced neurotoxicity. More importantly, we identified interventions to alleviate and prevent methotrexate-induced memory deficits. I served as the primary investigator or co-investigator in all of these studies.

   a. Wen J, Patel C, Diglio F, Baker K, Marshall G, Li S, **Cole PD**. Cognitive impairment persists at least 1 year after juvenile rats are treated with methotrexate. Neuropharmacology. 2022. Available online January 2, 2022; https://doi.org/10.1016/j.neuropharm.2021.108939

   b. Wen J, Maxwell RR, Wolf AJ, Spira M, Gulinello ME, **Cole PD**. Methotrexate causes persistent deficits in memory and executive function in a juvenile animal model. Neuropharmacology. 2018; 139:76-84. https://doi.org/10.1016/j.neuropharm.2018.07.007. PMID: 29990472

   c. Thomsen AM, Gulinello ME, Vijayanathan V, Wen J, Schmiegelow K, **Cole PD**. Liposomal Cytarabine Induces Less Neurocognitive Dysfunction than Intrathecal Methotrexate in an Animal Model. Journal of Pediatric Hematology / Oncology. 2017; published online July 17 2017. PMID: 28654460.

   d. **Cole PD**, Vijayanathan V, Ali NF, Wagshul ME, Tanenbaum EJ, Price J, Dalal V, Gulinello ME. "Memantine protects rats treated with intrathecal methotrexate from developing spatial memory deficits," Clinical Cancer Research 2013: 19(16):4446-54. PMID: 23833301.

Complete list of published works: http://www.ncbi.nlm.nih.gov/pubmed?term=cole%20peter%20d[Author]