John P. Murnane

John P. Murnane, Ph.D.

Professor
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Professional Focus

John Murnane?s interests are the study of the mechanisms of DNA damage, DNA repair, and chromosome instability, and their relationship to cancer. His early work was the first to demonstrate that cell cycle regulation is important in protecting cells against DNA damage (Nature 285:326, 1980). His work was also the first to demonstrate that human cells can become immortal by maintaining telomeres through a process other than telomerase, and to propose that this mechanism involves recombination (EMBO J 13:4953, 1994). Telomeres are the caps on the ends of chromosomes, and acquiring the ability to maintain telomeres is an important step in cancer progression. His laboratory was also the first to establish a model

system to demonstrate the ability of mammalian cells to stabilize broken chromosomes through the addition of new telomeres, called chromosome healing (PNAS 96:6781, 1999). He is currently investigating whether the regulation of chromosome healing can be used as a method for preventing chromosome instability in cancer cells, and selectively sensitize cancer cells to ionizing radiation. More recently his work has demonstrated that regions near telomeres are highly sensitive to DNA double-strand breaks, and has proposed that this sensitivity of telomeric regions to DNA double-strand breaks is an important factor in chromosome instability, which is important in tumor cell progression and resistance to classical and targeted therapies (Cancer Res 70:4255, 2010). His laboratory is now involved in the analysis of the mechanisms and proteins responsible for the sensitivity of telomeric regions to DNA double strand breaks, and how this sensitivity can be exploited to prevent chromosome instability or selectively kill cancer cells demonstrating chromosome instability.

Education

1971	California State University, Northridge	BS	Chemistry
1977	UCLA		
1980	UCLA	PhD	Microbiology

Professional Experience

2007-present	UCSF	Professor in Residence, Vice Chair	Department of Radiation Oncology
1998-2007	UCSF	Adjunct Professor	
1996-1998	UCSF	Associate Adjunct Professor	Department of Radiation Oncology
1991-1996	UCSF	Associate Adjunct Professor	
1984-1991	UCSF	Assistant Adjunct Professor	Laboratory of Radiobiology & Environmental Health
1983-1984	UCSF	Asst. Research Biochemist with Dr. Robert Painter	Laboratory of Radiobiology & Environmental Health
1980-1983	UCSF	Postdoctoral Fellow with Dr. Robert B. Painter	Laboratory of Radiobiology & Environmental Health

Recent Significant Publications :

Murnane, J. P. **Telomere loss as a mechanism for chromosome instability in human cancer**. *Cancer Res.,* 70:4255-4259 (2010).

Reynolds, G.E., Gao, Q., Miller, D., Snow, B.E., Harrington, L.A., and Murnane J.P. **The role of PIF1 and NBS1 in chromosome healing and fusion resulting from double-strand breaks near telomeres in murine embryonic stem cells**. *DNA Repair*, 10:1164-

1173 (2011).

Miller, D., Reynolds, G.E., Mejia, R., Stark, J.M., and Murnane, J.P. **Subtelomeric regions in mammalian cells are deficient in DNA double-strand break repair**. *DNA Repair*10:536-544 (2011).

Murnane, J.P. **Telomeric dysfunction and chromosome instability**. *Mutat. Res*. 730:28-36 (2012).

Muraki, K., Nyhan, K., Han, L., and Murnane, J.P. **Mechanisms of telomere loss and their consequences for chromosome instability**. *Front. Oncol.* 2:135 (2012).

Muraki, K., Han, L., Miller, D., and Murnane, J.P. **The role of ATM in the deficiency in nonhomologous end-joining near telomeres in a human cancer cell line**. *PLoS Genetics*, 9:e1003386 (2013).

Li, Z., Hudson, F.Z., Wang, H., Wang, Y., Bian, Z., Murnane, J.P., and Dynan, W.S. **Increased mutagenic joining of enzymatically-induced DNA double-strand breaks in high-charge and energy particle irradiated human cells**. *Radiation Res.* 180:17 (3013).

Li, Z., Wang, H., Wang, Y., Murnane, J.P., and Dynan, W.S. **Effect of radiation quality on mutagenic joining of enzymatically-induced DNA double-strand breaks in previously irradiated human cells**. Radiation Res. In press (2014).

Bakhoum, S.F., Kabeche, L., **Murnane, J.P.**, Zaki, B.I., and Compton, D.A. **DNA-damage response during mitosis induces whole-chromosome missegregation**. *Cancer Discov.* 4:1281-9 (2014).

Bakhoum, S.F., Kabeche, L., Wood, M.D., Laucius, D., Qu, D., Laughney, A.M., Reynolds, G.E., Louie, R.J., Phillips, J., Chan, D.A., Bassem, I.Z., **Murnane, J.P.**, Petritsch, C., and Compton, D.A. **Numerical chromosomal instability mediates susceptibility to radiation treatment**. *Nature Commun.* 6:5990 (2015).

Muraki, K., Han, L, Miller, D., and Murnane, J. P. **Processing by MRE11 is involved in the sensitivity of subtelomeric regions to DNA double-strand breaks**. *Nucleic Acids Res.* doi: 10.1093/nar/gkv714 (2015).

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