BIOGRAPHICAL SKETCH

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NAME	POSITION TITLE
May, James M.	Professor of Medicine & Molecular Physiology & Biophysics
eRA COMMONS USER NAME (credential, e.g., agency login) mayjamesm	

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	YEAR(s)	FIELD OF STUDY
Yale College, New Haven, CT	B.S.	1965-1969	Biology
Vanderbilt Univ. Med. School, Nashville, TN	M.D.	1969-1973	Medicine
Vanderbilt University, Nashville, TN	Internship	1973-1974	Medicine
Johns Hopkins Hospital, Baltimore, MD	Residency	1974-1975	Medicine
University of Washington, Seattle, WA	Fellowship	1975-1978	Endocrinology/Metabolism

A. Positions and Honors

Positions and Employment

1978-1983 Assistant Professor of Medicine, Medical College of Virginia, Richmond, VA
1983-1986 Associate Professor of Medicine, Medical College of Virginia, Richmond, VA
1986-2000 Associate Professor of Medicine, Vanderbilt University School of Medicine, Nashville, TN
1993-2000 Associate Professor of Molecular Physiology and Biophysics, Vanderbilt Univ. School of Medicine
2000-present Professor of Medicine and Molecular Physiology and Biophysics, Vanderbilt Univ. School of Med.
2006-present Section Chief, Endocrinology and Diabetes, VA Hospital, Nashville, TN

Other Experience and Professional Memberships

1973 Alpha Omega Alpha Medical Society
1985-1986 American Diabetes Association; President, Virginia Affiliate
1983-1987 AFCR - Southern Section; Sec.-Treas., 1983-86; President-Elect, 1986; President, 1987
1986-present American Society for Clinical Investigation
1996-present Associate Editor, Metabolism, 1996-present; Editorial Board, Free Radic. Biol. Med., 2001-present
1997-2001 NIH, Chemistry and Related Science Special Emphasis Panel (ZRG3 SSS Z01), 1997, Chair, 2000
2004-2007 NIH, EMNR E(10) B-SBIR/STTR study section member
2007-present NIH, Integrative Nutrition and Metabolic Processes (INMP) study section, Member

Honors

1979-1982 NIH Clinical Investigator Award
1983-1988 NIH Research Career Development Award

B. Selected peer-reviewed publications (in chronological order)

1. Li, X., Hill, K.E., Burk, R.F. and May, J.M. Selenium spares ascorbate anḍ α-tocopherol in cultured liver cell lines under oxidant stress. FEBS Letts. 508:489-492, 2001.
2. May, J.M. Recycling of vitamin C by mammalian thioredoxin reductase, Methods Enzymol. 347:327-332, 2002.

3. Hardy, T.A. and May, J.M. Coordinate regulation of L-arginine uptake and nitric oxide synthase activity in cultured endothelial cells. Free Radic. Biol. Med., 32:122-131, 2002.
4. May, J.M., Morrow, J.D. Burk, R.F. Thioredoxin reductase reduces lipid hydroperoxides and spareș -tocopherol. Biochem. Biophys. Res. Commun., 292:45-45, 2002.
5. Li, Xia, and May, J.M. Catalase-dependent measurement of H2O2 in intact mitochondria. Mitochondrion, 1:447-453, 2002..
6. Jones, W., Li, X., Qu, Z.-c., Perriott, L., Whitesell, R.R., and May, J.M. Uptake and recycling oḟ -lipoic acid by endothelial cells, Free Radic. Biol. Med., 33:83-93, 2002.
7. Li, X., Cobb, C.E, and May, J.M. Mitochondrial recycling of ascorbic acid from dehydroascorbic acid: dependence on the electron transport chain. Arch. Biochem. Biophys., 403:103-110, 2002.
8. May, J.M., Li, X., and Qu, Z.-C. Ascorbic acid blunts oxidant stress due to menadione in endothelial cells. Arch. Biochem. Biophys., 411:136-144, 2003.
9. Li, X., Huang, J., and May, J.M. Ascorbic acid spares α-tocopherol and decreases lipid peroxidation in neuronal cells. Biochem. Biophys. Res. Commun., 305:656-661, 2003.
10. Hill, K.E., Montine, T.J., Motley, A.K., Li, X., May, J.M., and Burk, R.F. Combined deficiency of vitamins E and C causes paralysis and death in guinea pigs. Am. J. Clin. Nutr., 77:1484-1488, 2003.
11. May, J.M., Qu, Z., Neel, D.R., and Li, X. Recycling of vitamin C from its oxidized forms by human endothelial cells. Biochim.Biophys. Acta, 1640:153-161, 2003.
12. Li, X., and May, J.M. Location and recycling of mitochondrial α-tocopherol. Mitochondrion, 3:29-38, 2003.
13. May, J.M**.** Qu, Z.-c., and Cobb, C.E. Human erythrocyte recycling of ascorbic acid: Relative contributions from the ascorbate free radical and dehydroascorbic acid. J. Biol. Chem., 279:14975-14982, 2004.
14. May, J.M. Qu, Z.-c., and Cobb, C.E. Reduction and uptake of methylene blue by human erythrocytes. Am. J. Physiol. (Cell Physiol.), 286:C1390-C1398, 2004.
15. May, J.M. and Qu, Z.-C. Ascorbate protects against nitrite-induced oxidant stress in endothelial cells. Free Radic. Res., 38:581-589, 2004.
16. May, J.M. Qu, Z.-c., and Li, X., May, J.M., and Qu, Z.-C. Nitrite generates an oxidant stress and increases nitric oxide in EA.hy926 endothelial cells. Free Radic. Res., 38:581-589, 2004.
17. May, J.M., and Qu, Z.-C. Nitric-oxide induced oxidant stress in endothelial cells: amelioration by ascorbic acid. Arch. Biochem. Biophys. 429:106-113, 2004.
18. May, J.M., and Qu, Z.-C. Redox regulation of ascorbate transport: role of transporter and intracellular sulfhydryls. Biofactors 20:199-211, 2004.
19. May, J.M. and Qu, Z.-c. Transport and intracellular accumulation of ascorbic acid in endothelial cells: relevance to collagen synthesis, Arch. Biochem. Biophys., 34:178-186, 2005.
20. May, J.M., Qu, Z.-c., Juliao, S., and Cobb, C.E. Oxidant stress induced by thenitroxide Tempol in endothelial cells. Free Radic. Res., 39:195-202, 2005.
21. May, J.M**.,** Li, L., Qu, Z.-c., and Huang, J. Ascorbate uptake and antioxidant function in peritoneal macrophages, Arch Biochem. Biophys., 440: 165-172, 2005.
22. May, J.M., Huang, J., and Qu, Z.-c. Macrophage uptake and recycling of ascorbic acid: Response to activation by lipopolysaccharide, Free Radic. Biol. Med., 39:1449-1459, 2005. PHS 398 (Rev. 04/06) 8
23. May, J.M., Qu, Z.-c., and Nelson, D.J. Cellular disulfide reducing capacity: An integrated measure of cell redox capacity. Biochem. Biophys. Res. Commun., 344:1352-1359, 2006.
24. Huang, J**.** and May, J. M. Ascorbic acid protects SH-SY5Y neuroblastoma cells from apoptosis and death Induced by β-amyloid, Brain Res., 1097:52-58, 2006.
25. May, J. M., Li, L., and Qu, Z.-c. Ascorbate transport and recycling by SH-SY5Y neuroblastoma cells: response to glutamate toxicity, Neurochem. Res., 31:785-794, 2006.
26. Su, D.; May, J.M.; Koury, M.J.; Asard, H., Human erythrocyte membranes contain a cytochrome b561 that may be involved in extracellular ascorbate recycling, J. Biol. Chem. 281:39852-39859, 2006.
27. Qiu, S., Li, L., Weeber, E. J., May, J. M. Ascorbate transport by primary cultured neurons and its role in neuronal function and protection against excitotoxicity, J. Neurosci. Res., 85:1046-1056, 2007.
28. May, J.M., Qu, Z.-C., Qiao, H., Koury, M.J. Maturational loss of the vitamin C transporter in human erythrocytes. Biochem. Biophys. Res. Commun. 360:295-298, 2007.

29. May, J.M., Li, L., Qu, Z.-C., Cobb, C.E. Mitochondrial recycling of ascorbic acid as a mechanism for regenerating cellular ascorbate. Biofactors 30:35-48, 2007.

30. Sabharwal, A.K., May, J.M. α-Lipoic acid and ascorbate prevent LDL oxidation and oxidant stress in endothelial cells. Mol. Cell. Biochem. 309:125-132, 2008.

31. Qiao, H., May, J.M. Development of ascorbate transporters in brain cortical capillary endothelial cells in culture. Brain Res., 1208:79-86, 2008.

32. Harrison, F.E., Yu, S.S, VanDenBossche, K.L., Li, L., May, J.M, McDonald, M.P. Elevated oxidative stress and sensorimotor deficits but normal cognition in mice that cannot synthesize vitamin C. J. Neurochem., in press, 2008.

33. Qiao, H., may, J.M. Development of ascorbate transporters in brain cortical capillary endothelial cells in culture. Brain Res. 1208: 79-86, 2008.

34. Harrison, F.E., Yu, S.S., VanDenBossche, K.L., Li, L., May, J.M., mcdonald, M.P. Elevated oxidative stress and sensorimotor deficits but normal cognition in mice that cannot synthesize vitamin C. J. Neurochem. 106: 1198-1208, 2008.

35. Qiao, H., Bell, J., Juliao, S., Li, L., May, J.M. Ascorbic acid uptake and regulation of type 1 collagen synthesis in cultured vascular smooth muscle cells. J. Vasc. Res., in press.

C. Research Support

Ongoing Research Support

5 R01 AG023138-04 1/15/04 -12/31/08
NIH/NIA
Antioxidant Vitamins in Models of Alzheimer's Disease
This project uses cell culture and mouse models of Alzheimer's disease to test the hypotheses that tissue damage occurs as a result of oxidant stress, and that the antioxidant vitamins C and E can decrease this damage. This project will be completed 12/31/08 and no renewal will be submitted.
Role: PI

5 RO1 AG016236-09 5/15/04 - 3/31/09
NIH/NIA
Antioxidant Interactions of Selenium and Vitamins C and E
The goal of this project is to define a role for selenium interactions with vitamins C and E in preventing oxidant damage, both *in vivo* and *in vitro*. The impact of dietary deficiencies of selenium, ascorbic acid, and alpha-tocopherol, alone and combined, will be studied in guinea pigs, which cannot synthesize vitamin C. This project will be completed 3/31/09 and no renewal will be submitted.
Role: PI

2 R01 DK050435-12 4/15/06 – 3/31/11
NIH/NIDDK
Ascorbic Acid Function and Metabolism
The goal of this project is to study the role of ascorbate in the development of atherosclerosis. Aim 1 evaluates vitamin C function in cultured endothelial cells, vascular smooth muscle cells, and macrophages. Aim 2 utilizes the ApoE-deficient mouse model of atherosclerosis to assess effects of vitamin C on the progression, severity, and nature of atherosclerotic lesions. Aim 3 tests whether macrophages prepared from these animals have changes in function or antioxidant defenses with varying intracellular vitamin C concentrations.
Role: PI

2 T32 DK07061-33 7/1/06-3/31/11
NIH/NIDDK
Research Training in Diabetes and Endocrinology
Institutional post-doctoral training grant in diabetes and endocrinology
Role: PI

1 R01 NS057674-01A2 7/01/08-6/30/12
NIH/NINDS
Vitamin C Transporters in the Brain
The project will examine the role of the neuronal SVCT2 transporter in maintaining brain ascorbate concentrations and in protecting against oxidant stress generated in mouse models of combined deficiencies of vitamins C and E. This project does not involve ascorbate and its transporter in pregnancy or in the placenta.
Role: PI

Completed Research Support
None