CMIIT Sponsors Cardiovascular Symposium at NIH

The centerpiece of SNM Center for Molecular Imaging Innovation and Translation (CMIIT; formerly the Molecular Imaging Center of Excellence) activities each year is an annual multimodality molecular imaging symposium at the National Institutes of Health (NIH). In 2012, CMIIT will sponsor the "Multimodality Cardiovascular Molecular Imaging Symposium," to be held April 19–21 at the Natcher Auditorium on the NIH campus in Bethesda, MD. The proceedings of the last cardiovascular symposium in 2009, led by the CMIIT in conjunction with several other cardiovascular imaging societies, were published as a supplement to *The Journal of Nuclear Medicine* (http://jnm.snmjournals.org/content/vol51/Supplement_1/index.dtl).

The CMIIT program committee has designed a 2.5-d symposium to bring together individuals from multiple scientific disciplines, including chemistry, engineering, physics, molecular biology, cardiovascular physiology, and imaging scientists, with the goal of promoting cardiovascular molecular imaging. The speaker roster includes experts in new imaging probes and technology, imaging of cardiovascular receptors, stem cell therapy, vascular biology, myocardial metabolism, and other biological processes relevant to the cardiovascular system. The symposium will emphasize interaction among speakers and registrants to stimulate further growth in the field.

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Sessions will review the current state of imaging in cardiovascular disease, from novel probes for evaluation and treatment of cardiovascular disease to evaluation of stem cell therapy. Challenges to translation of molecular imaging and therapy also will be addressed. These lectures will provide an overview of the potential of molecular imaging for improving understanding and management of critical cardiovascular pathophysiological processes, such as atherosclerosis, angiogenesis, cardiomyopathies, ischemia, and infarction.

The call for abstracts opens on November 14, 2011. Those abstracts accepted for presentation will be published in *The Journal of Nuclear Medicine*. Travel awards will be available to young investigators. For additional information, visit www.snm.org/cvmi2012.

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Symposium Program Committee

receptor decoy protein, and reimaged over the course of 3 mo. They saw an increase in serotonin transporter binding associated with improved therapeutic results.

Hirvonen et al. from the University of Turku (Finland), the National Institute of Mental Health (Bethesda, MD), and the National Institute on Drug Abuse (Baltimore, MD) reported on "Reversible and regionally selective down-regulation of brain cannabinoid CB_1 receptors in chronic daily cannabis smokers" [10]. Being a daily cannabis smoker was shown to have measurable effects throughout the brain (Fig. 37), with CB_1 receptors region-specifically downregulated. This downregulation also correlated with years of abuse. However, CB_1 receptors were found to increase after abstinence.

Neuroinflammation ligands have been an area of both great promise and disappointment. Studies with ^{11}C-PK11195, the first ligand for the peripheral benzodiazepine receptor that is expressed on activated inflammatory response cells, have been hindered by high nonspecific white matter uptake and poor image quality. This receptor is now referred to as the transporter protein (TSPO). Recently developed TSPO ligands have better imaging characteristics but have revealed that marked heterogeneity exists in the degree of binding in normal persons. Hannestad et al. from Yale University School of Medicine (New Haven, CT) reported on "Experimental endotoxemia and microglial activation: a PET study in nonhuman primates" [499]. Their aim was to measure whether binding of a new TSPO ligand, ^{11}C-PBR28, increases after systemic endotoxin administration in baboons. The baboons underwent PET imaging at baseline and 1, 4, and 24 h after endotoxin infusion. Figure 38 shows that an acute reaction was readily detected by PET imaging. Much work continues in this field because of the many potential applications for imaging neuroinflammation.

Finally, another animal study posed the question of whether there is a back door to the brain for biomolecules. Belov et al. from Massachusetts General Hospital, Harvard Medical School, and Shriners Hospitals for Children (all in Boston, MA) looked at "Iodine-124 for quantitative PET imaging of macromolecule transport to CSF" [1195]. Tracer was administered into the lumbar space in nonhuman primates (Fig. 39). The brain was imaged over various time periods (0.5, 2.5, 5, and 24 h), and the initial CSF distribution of the tracer was subtracted from later images. Results show that the tracer made it into the brain tissue. This group concluded that intrathecal administration of enzyme replacement therapeutics may be beneficial for the therapy of the central nervous system component of lysosomal storage diseases.

Conclusion

Molecular imaging continues to have an important role in drug development and basic discovery in neuroscience. Clinical applications in neurodegenerative disease are set to expand substantially with better use of FDG, wide availability of ^{18}F-labeled β-amyloid ligands, and a DAT SPECT ligand now available in the United States and many other parts of the world. Clinical demand will rise greatly once an effective treatment for AD is found. We are likely to see an evolving role for PET imaging in brain tumor management. The future is bright, but clinical acceptance will depend on comparative effectiveness research, which has yet to be fully embraced by the molecular imaging community.

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