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# Decreased Serotonin Transporters in the Hypothalamus and Midbrain in Patients with Multiple Systemic Atrophy: A Study with [$^{123}$I]-FP-CITA

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**Purpose:** We investigated quantification of dopaminergic transporter (DAT) and serotonergic transporter (SERT) for differentiating between multiple systemic atrophy (MSA) and idiopathic Parkinsons disease (IPD). **Methods:** Nfluoropropyl- 2β -carbomethoxy-3β-4-[$^{123}$I]-iodophenylnortropane SPECT ([123I]-FP-CIT SPECT) was performed in 6 patients with MSA, 18 with early IPD, and 6 healthy controls. Standard ROIs (region of interests) of striatal regions to evaluate DAT, and hypothalamus and midbrain for SERT were drawn on standard template images and applied to each image taken 4 hours after radiotracer injection. Striatal V3? for DAT and hypothalamic and midbrain V3? for SERT were calculated using region/reference ration based on the transient equilibrium method. Group differences were tested using ANOVA with the postHoc analysis. **Results:** DAT in the putamen was significantly decreased in both patients groups with MSA and early IPD, compared with healthy control (p=0.03, p=0.05, respectively). A reduction of DAT in the caudate was significant in MSA patients (p=0.05) and showed a trend in early IPD patient. This implied least involvement of caudate in early IPD. Regarding SERT, MSA patients showed significant reduction of SERT in hypothalamus compared with controls as well as early IPD patients (p=0.05, 0.01, respectively), and also showed a tendency of decrease in SERT of the midbrain (p=0.058 vs. control). In patients with IPD, there was no significant reduction of SERT in the hypothalamus or midbrain when compared with controls. **Conclusion:** In this study, the decreased SERT in the hypothalamus and midbrain could be demonstrated in MSA patients using [$^{123}$I]-FP-CIT SPECT. We suggest that the quantification of SERT as well as DAT in [$^{123}$I]-FP-CIT SPECT is helpful to differentiate parkinsonian disorders.

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# Molecular Photonic Imaging of Cancer using Light-Emitting *E. Coli*

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**Purpose:** Cancer research has long sought a magic bullet that would selectively target and destroy malignant cells. In this study, we exploited that *E. coli* injected into tumor-bearing mice selectively target and proliferate in solid tumors by employing optical imaging technique. **Methods:** Lux operon or GFP has been cloned into pUC19 plasmid to engineer pUC19Lux or pUC19gfp which was transformed into varying kinds of wild type (MG1655) or mutant *E.coli* strains. For stable expression, lux operon was cloned with asd (aspartate β-semialdehyde dehydrogenase) gene and transformed into asd defective *E. coli* (MG1655asd-/asd+lux). These bacteria were i.v. injected into tumor mice or directly into central necrosis of tumor. **Results:** The imaging signal from wild type E.coli was detected initially at liver (20min), then migrated to and shine in the tumor mass until 2 weeks of injection which was consistently observed in immuno-defective (nude) and -competent (Balb/c) mice. Imaging signal of stbaly transformed strain (MG1655asd-/asd+lux) was stronger and longer-lasting than that of transiently transformed strain (MG1655lux). Flagella defective E. coli strain failed to reach tumor loci. Only a few amounts of stress regulatory defective E. coli strain arrived at but couldnt survive at the tumor loci. *E. coli* colonies expressing GFP was mostly observed at the border of central necrosis and peripheral proliferative areas in immunofluorescence studies. Directly injected MG1655asd-/asd+lux was transiently observed at central necrosis followed by spreading to the peripheral tumor mass which was consistent with the finding by tail vein injection. **Conclusion:** We successfully engineered *E. coli* strain stably expressing lux reporter gene. *E. coli* strongly targeted solid tumor regardless of host immune status. Our results support that the targeting of tumor by E.coli is an active process and would be applied as a delivery vehicle of varying imaging markers or therapeutic molecules.