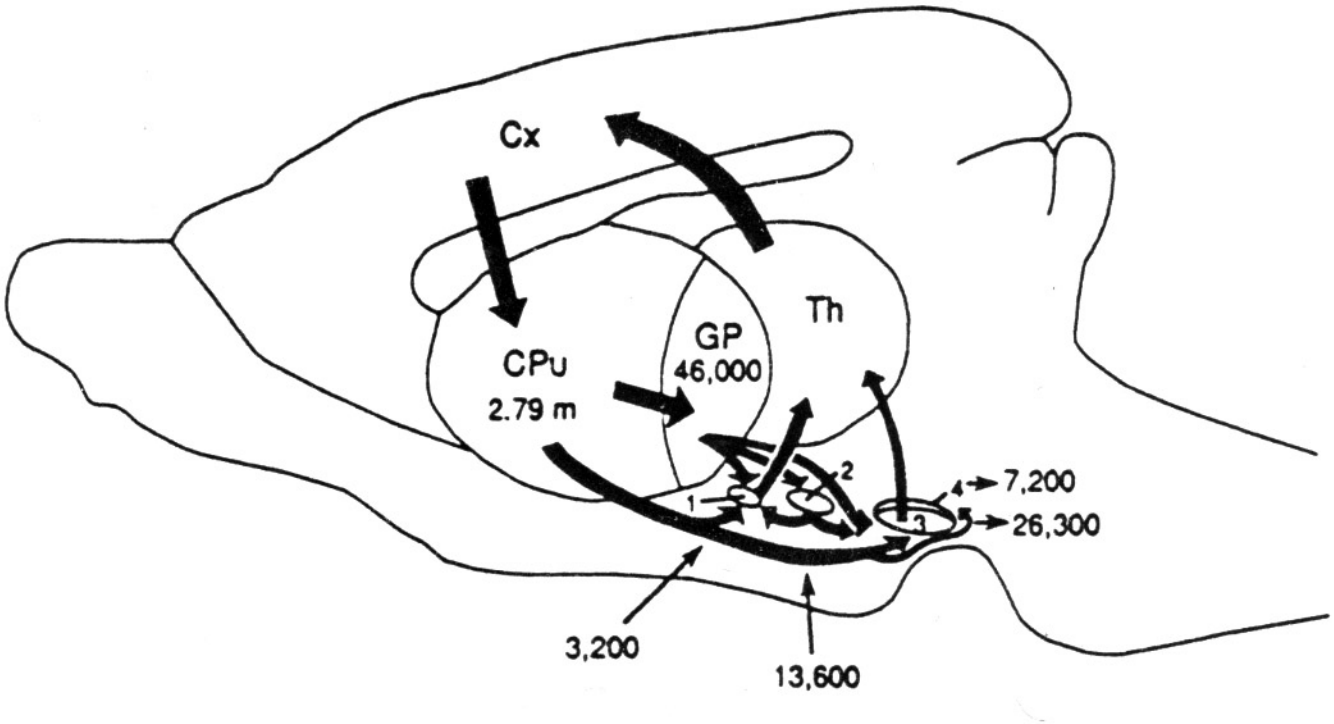
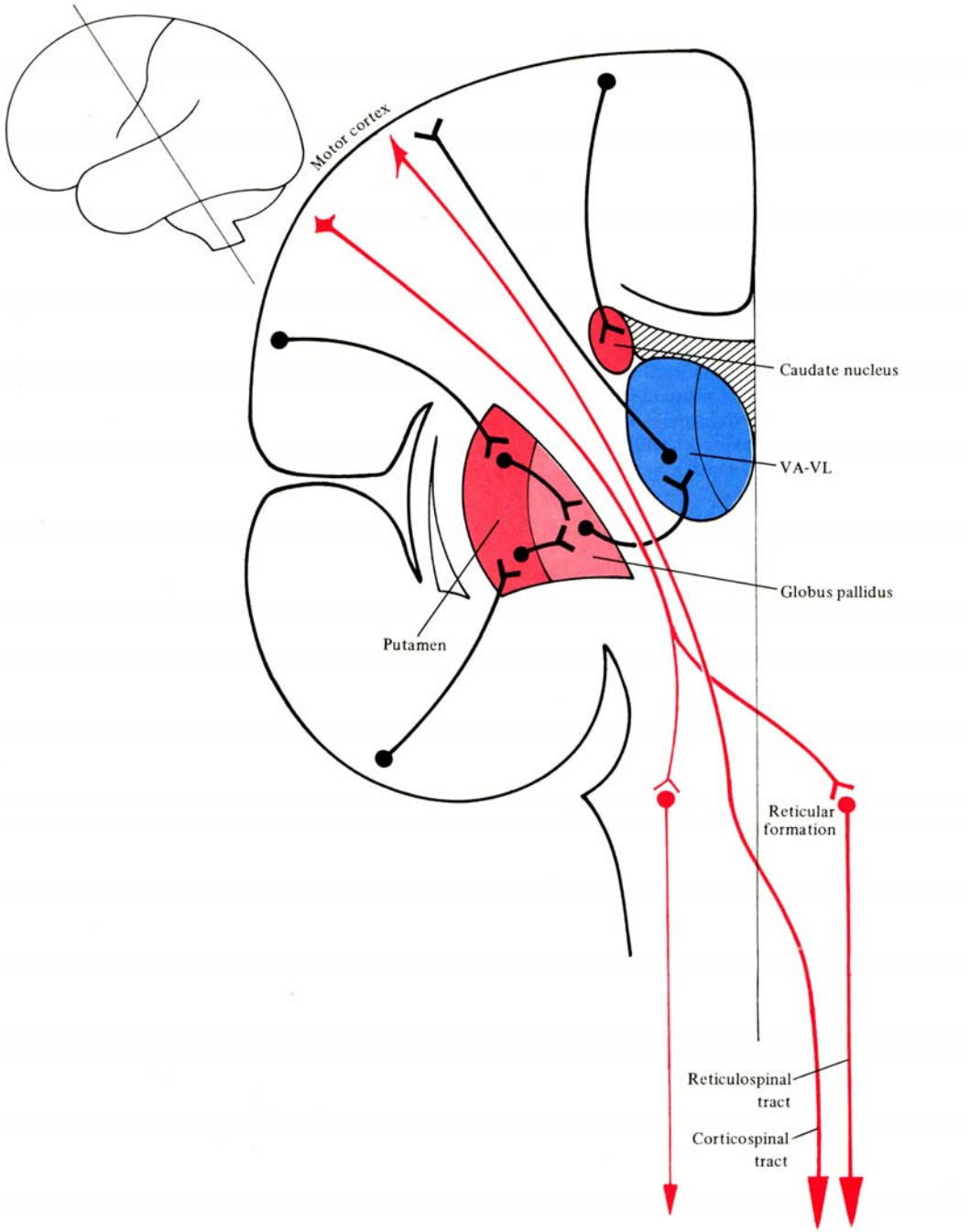
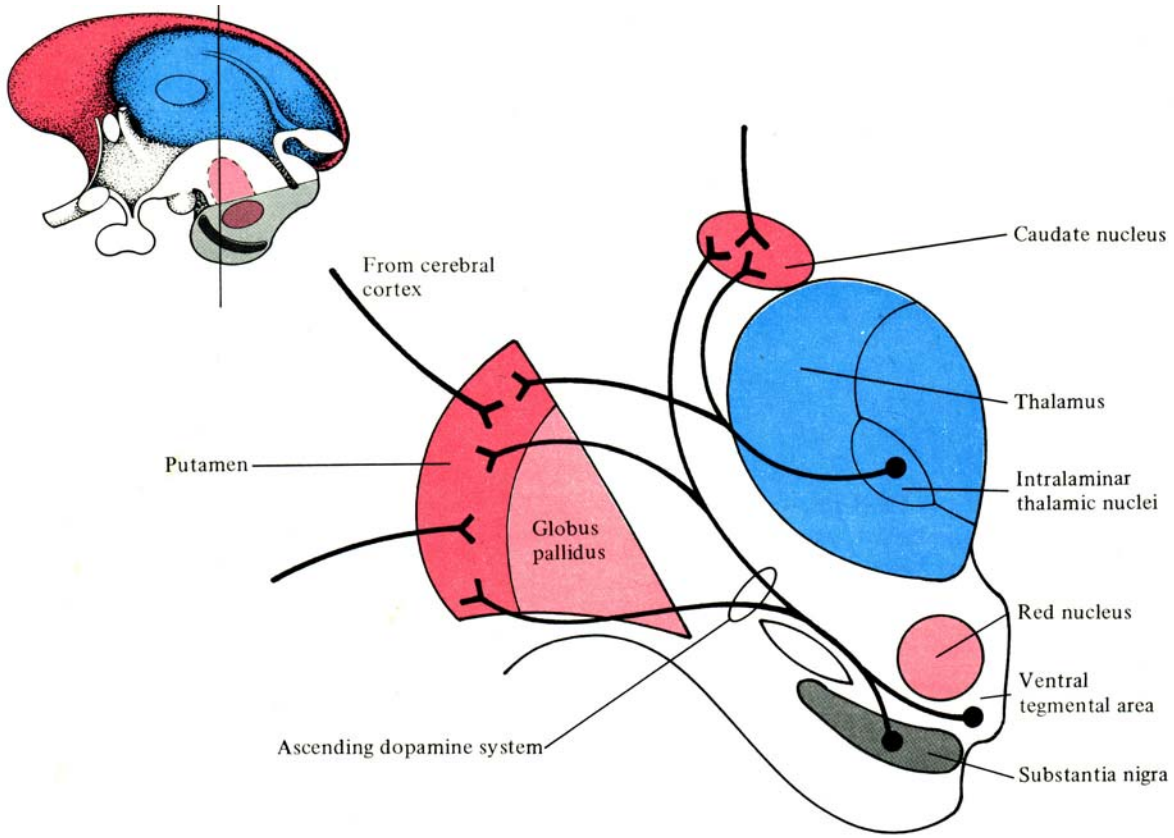
# BASAL GANGLIA



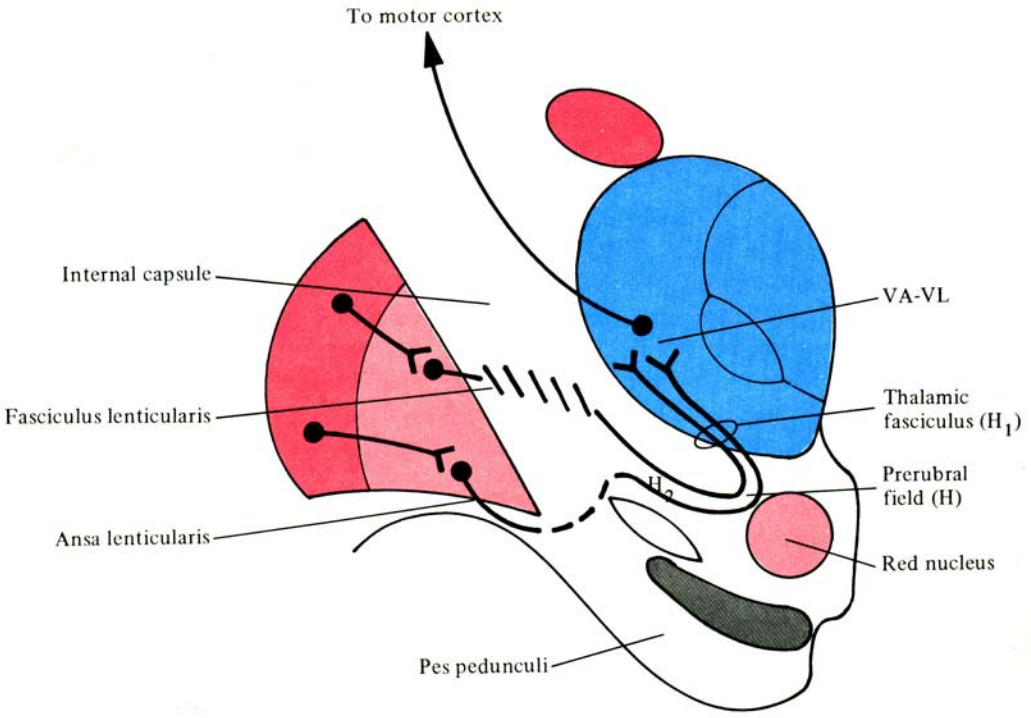

Schematic diagram illustarting the total number of neurons within each subdivisions of the rat basal ganglia. 1=entopeduncular n. (internal pallidal segment); 2=subthalamic nucleus; 3=substantia nigra reticulata; 4=substantia nigra compacta



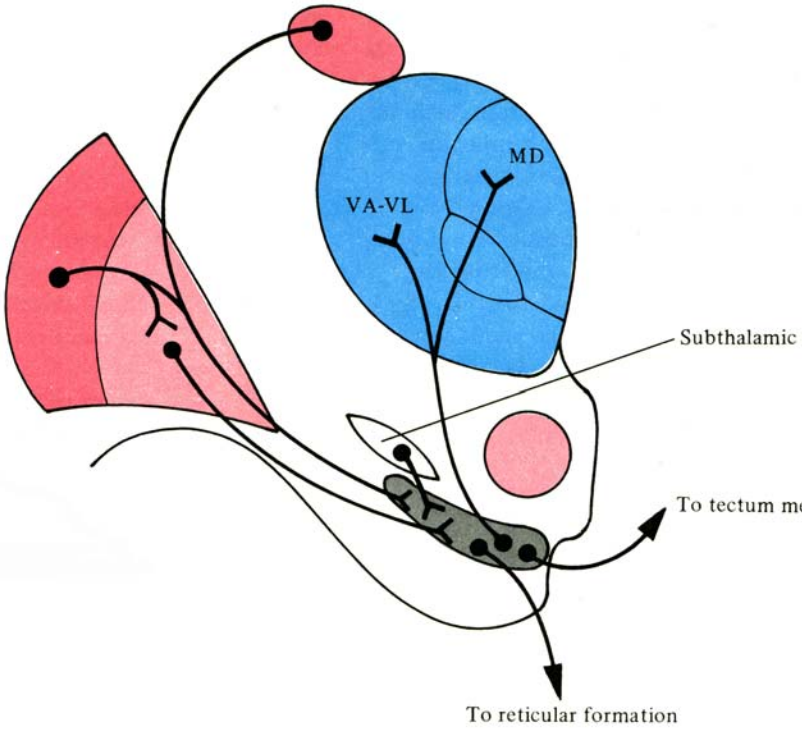

Schematic figure showing the cortico-strio-pallido-thalamic loop and its relationship to the descending corticospinal (pyramidal) and cortico-reticulo-spinal pathways (Heimer)



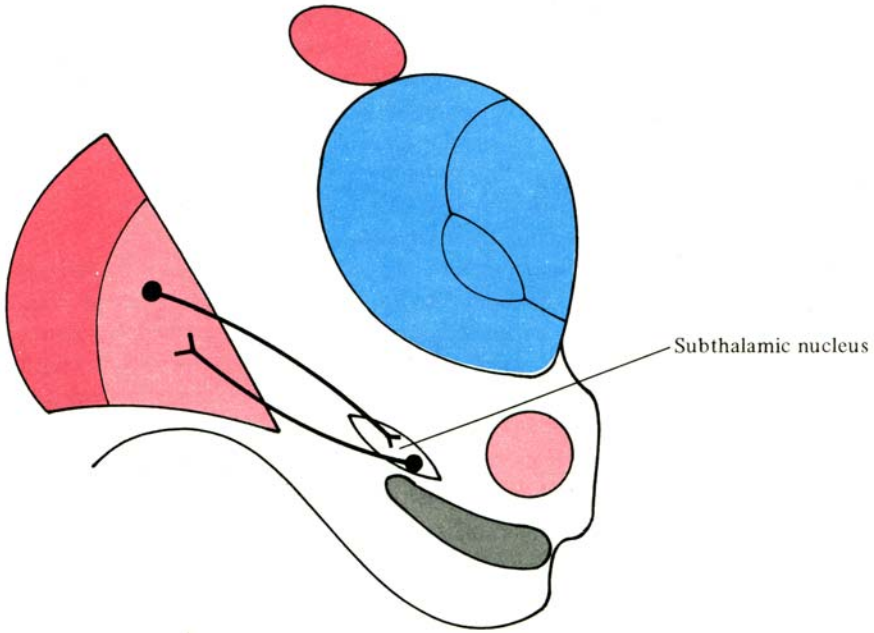

Afferent connections of the striatum (from cortex, thalamic intralaminar nuclei and the dopaminergic neurons of the SN)



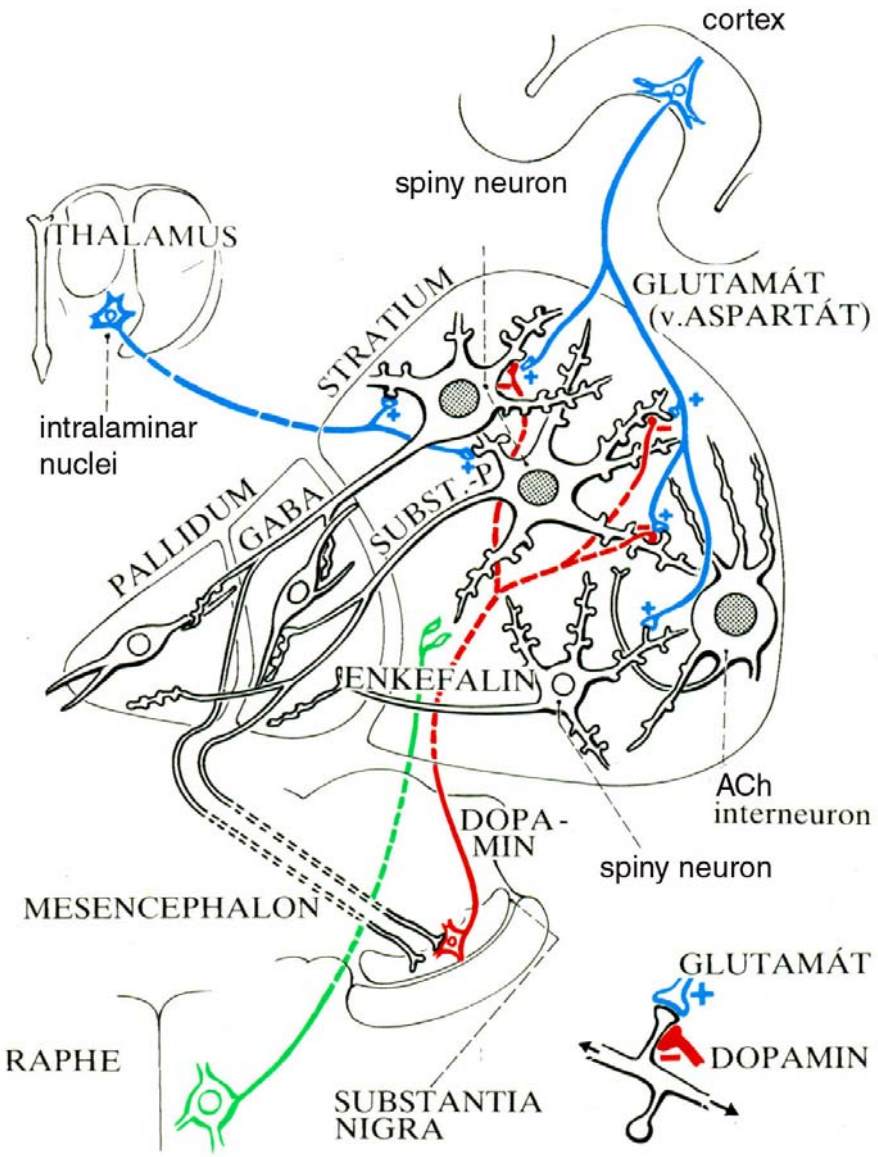

The striato-pallido-tahalmic loop (Heimer)



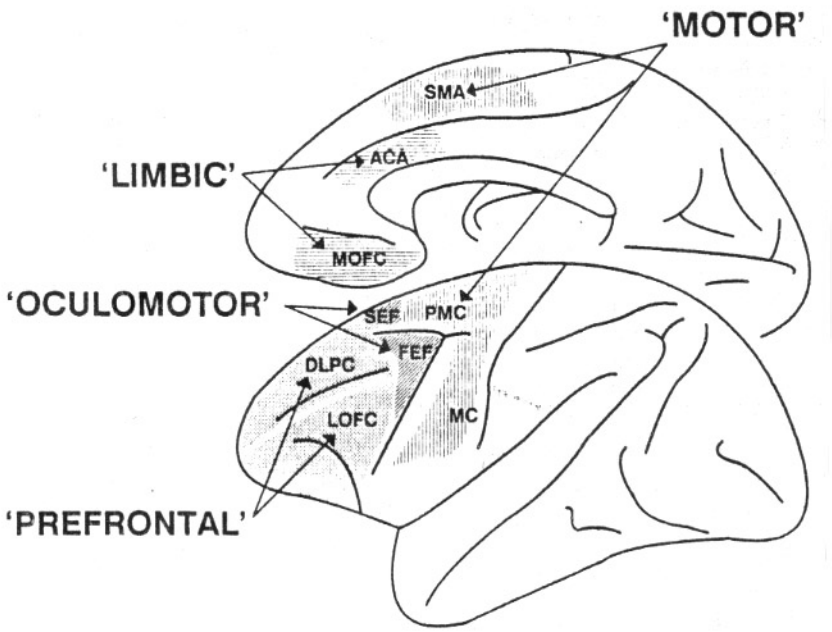

Some nigral afferents (Heimer)



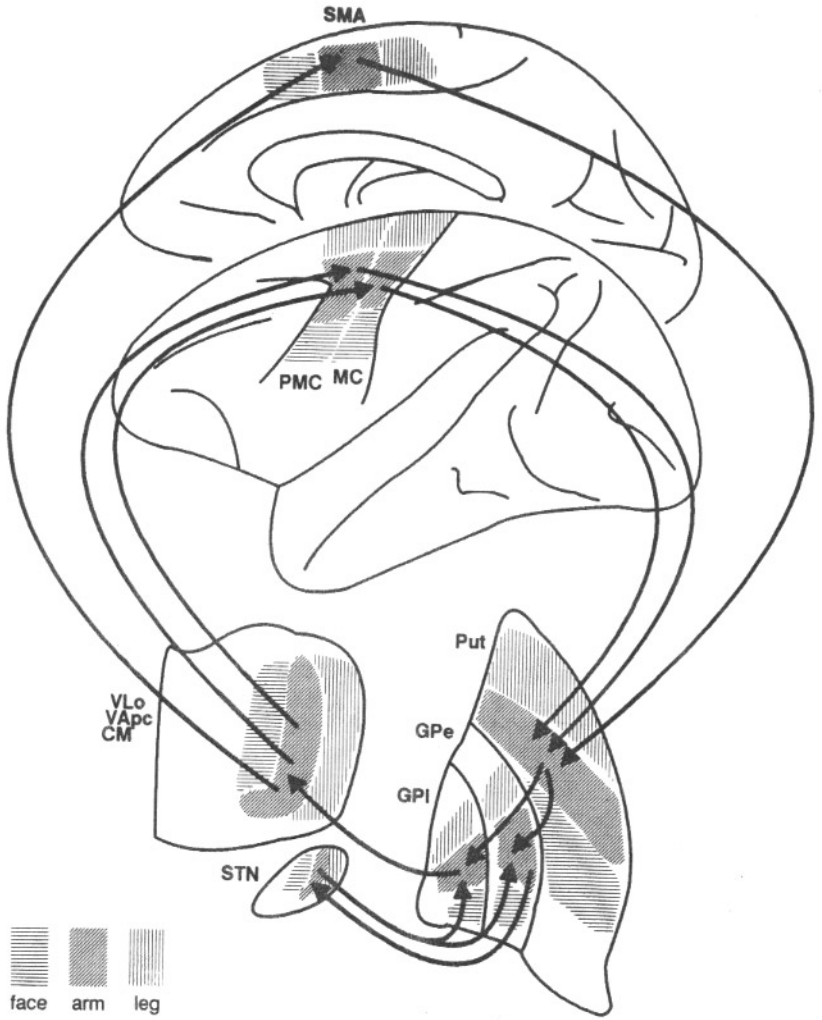

The pallido-subthalamic-pallido loop (Heimer)



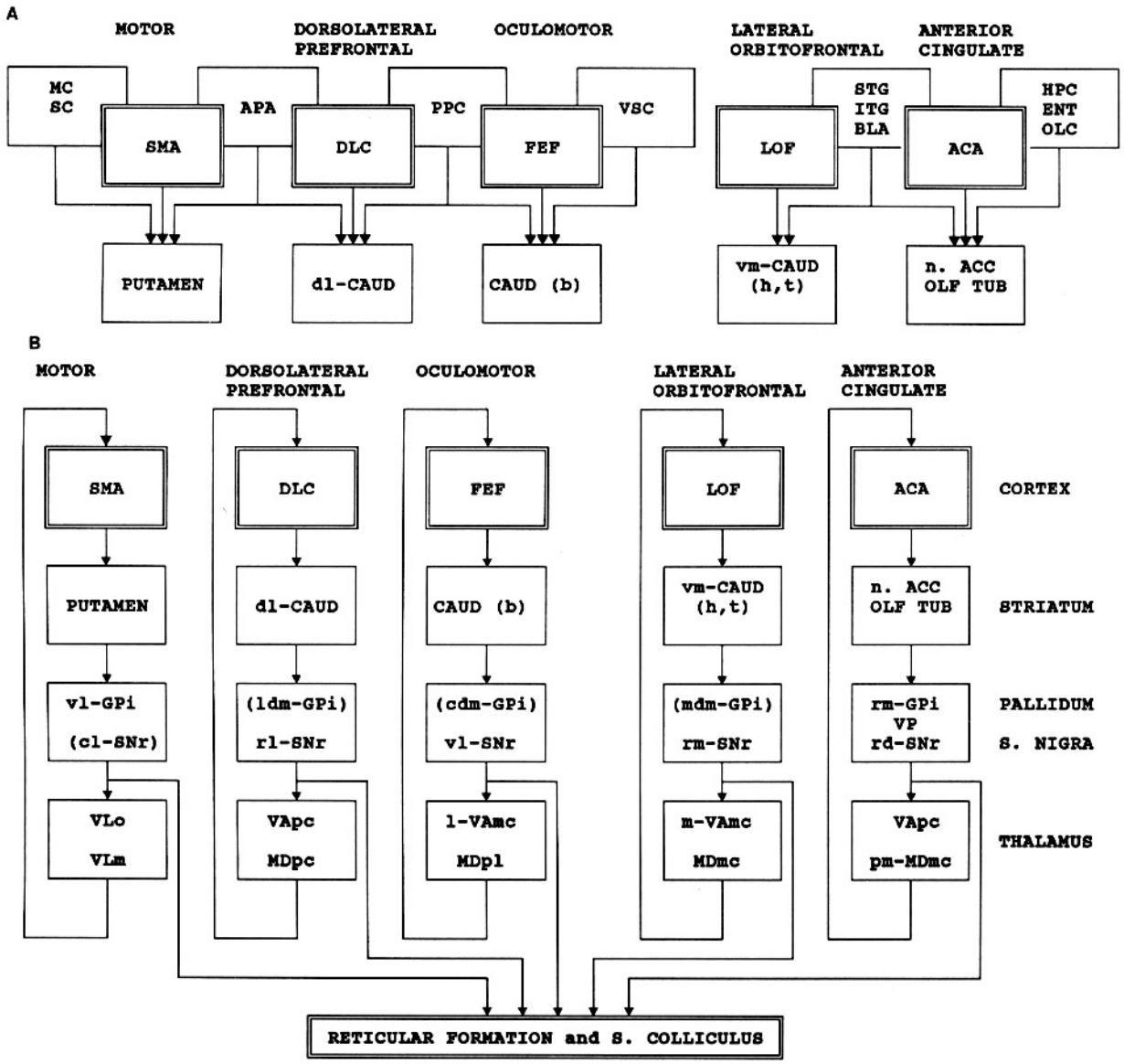

Synaptology and transmitters in some of the basal ganglia circuits (Szentagothai)



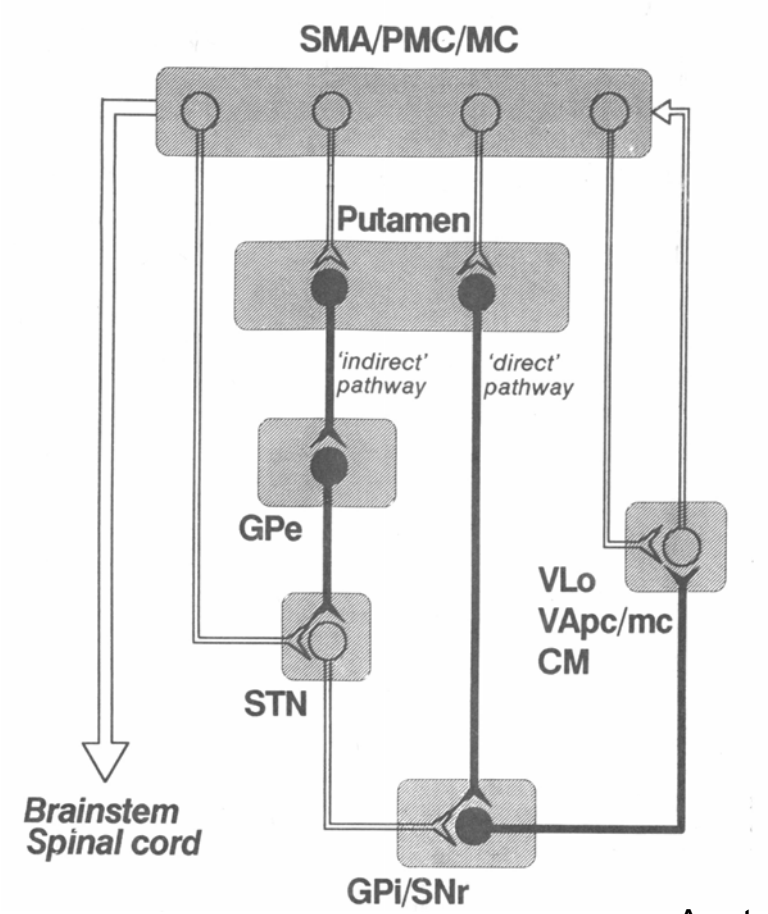

Schematic illustartion of the cortical areas that receive the output of the separate basal ganglia-tahalamo-cortical circuits. ACA=anterior cingulate area; DLPC=dorsolateral, LOFC, lateral prefrontal, MOFC= medial orbitofrontal cortex. FEF= frontal; SEF=supplementary eye field



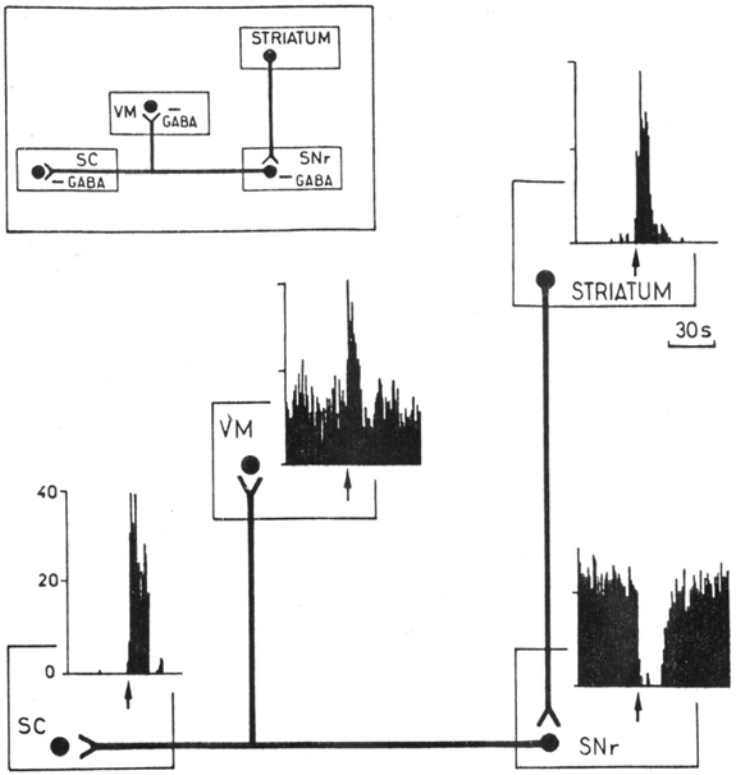

Somatiotopic organizaation of the 'motor' circuit. The arrows indicate the topographycally organized pathways that link the respective 'arm' representations at different stages of the circuit (Alexander and Crutcher, 1990)



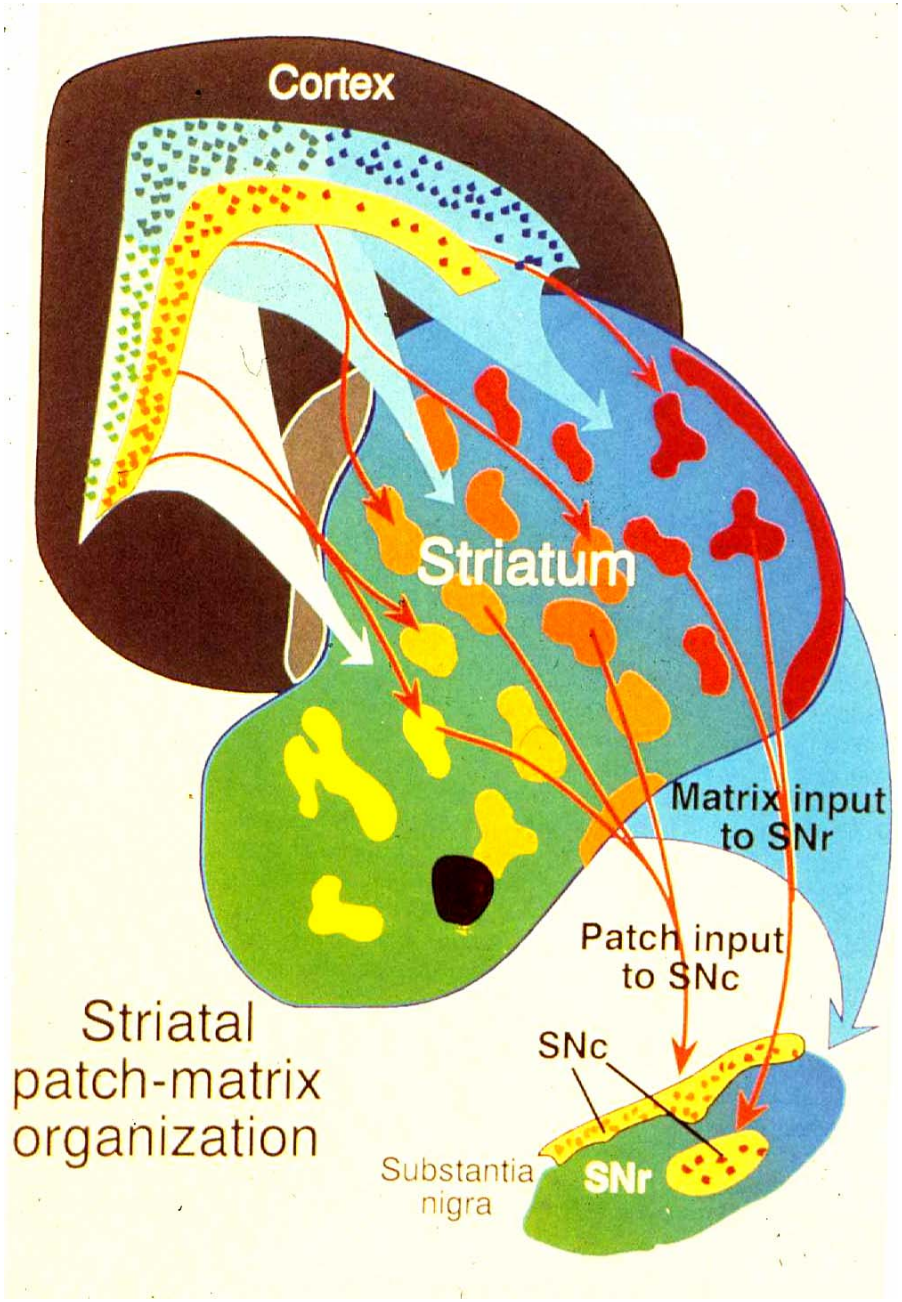

Macaque corticostriatal (A) and cortico-striato-pallidal (B) circuits based on the scheme proposed by Alexander et al (1986) (Alheid and Heimer, 1990).



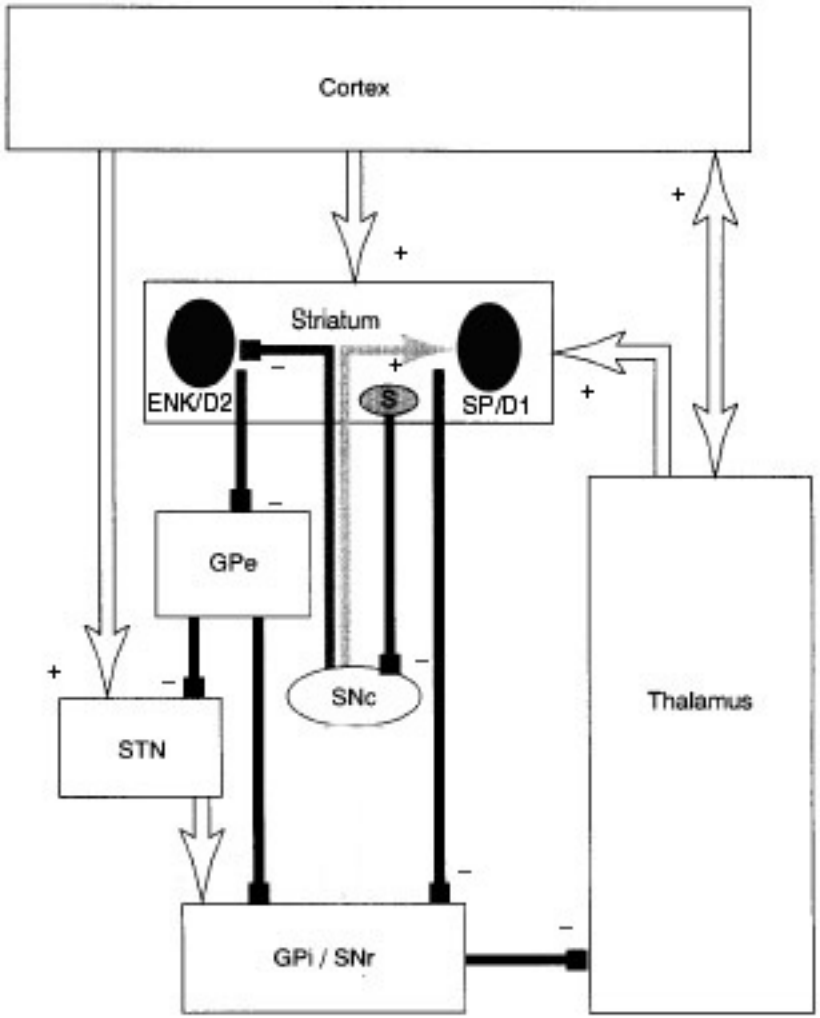

Simplified diagram of the 'motor' circuit. Inhibitory neurons are filled, excitatory neurons are open (Alexander and Crutcher, 1990).




Anatomico-physiological organization of the striato-nigrofual ('direct')pathways to the ventromedial thalamic (VM) and to the superior colliculus (SC). The frequncy histograms illustrate the sequence of electrophysiological events underlying the disinhibitory influence of the striatum. A striatal spike discharge, evoked by local application of glutamate, readily induces a clearcut silencing of the tonically active nigral neurons (SNr). Released from the potent nigral inhibition, collicular and thalamic celkls are vigorously discharged (The arrow in each histogram indicates the onset of Glu injection in the striatum (Chevalier and Deniau, 1990)




Corticostriatal neurons in the deep parts of layer 5 of the cortex provide inputs to the striatal patch compartment, whereas superficial layer V neurons provide inputs to striatal matrix. Patch neurons provide inputs to dopaminergic neurons of the SNc. Matrix neurons provide inputs to locations of GABAergic neurons in the SNr (Gerfen)



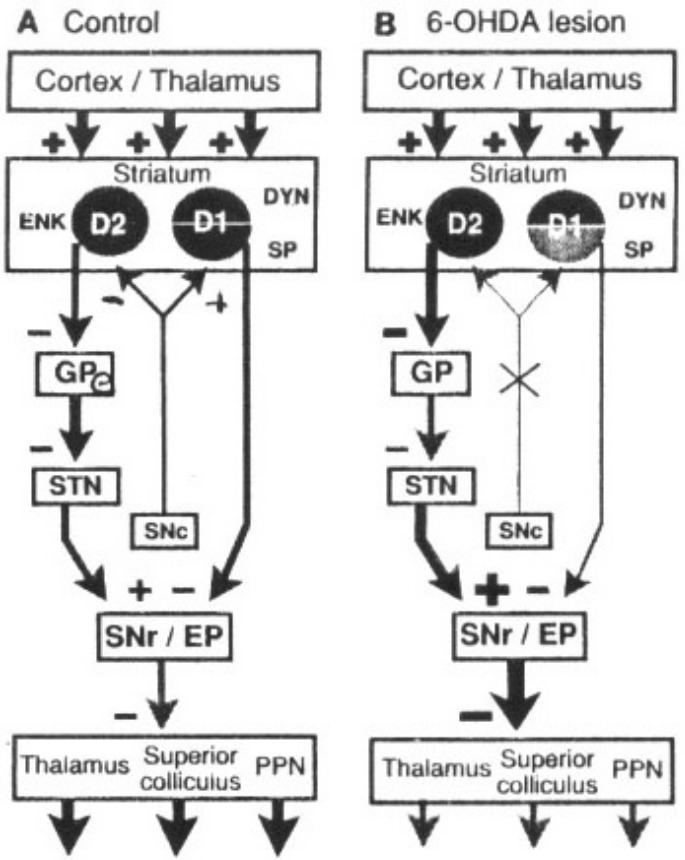
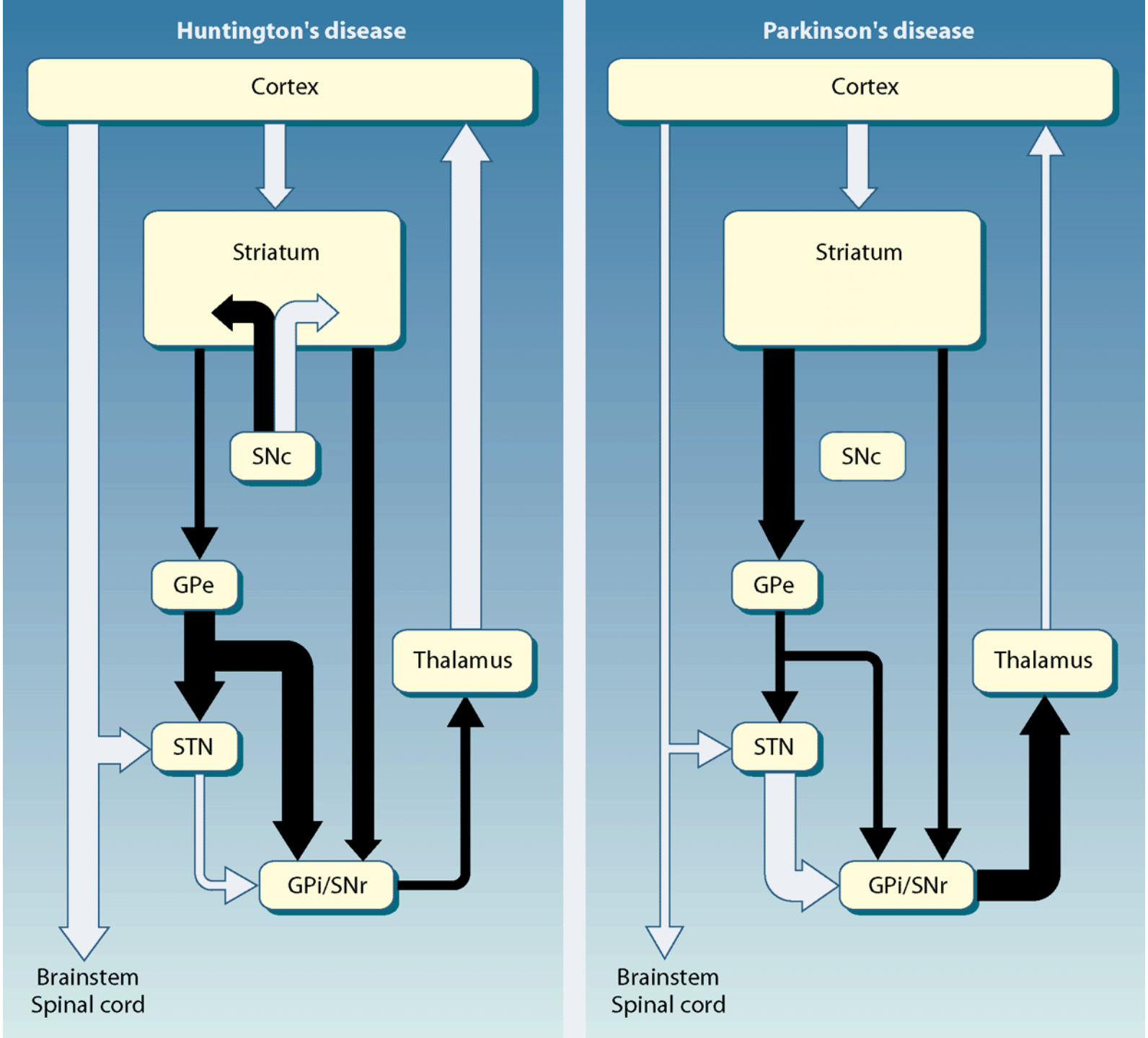
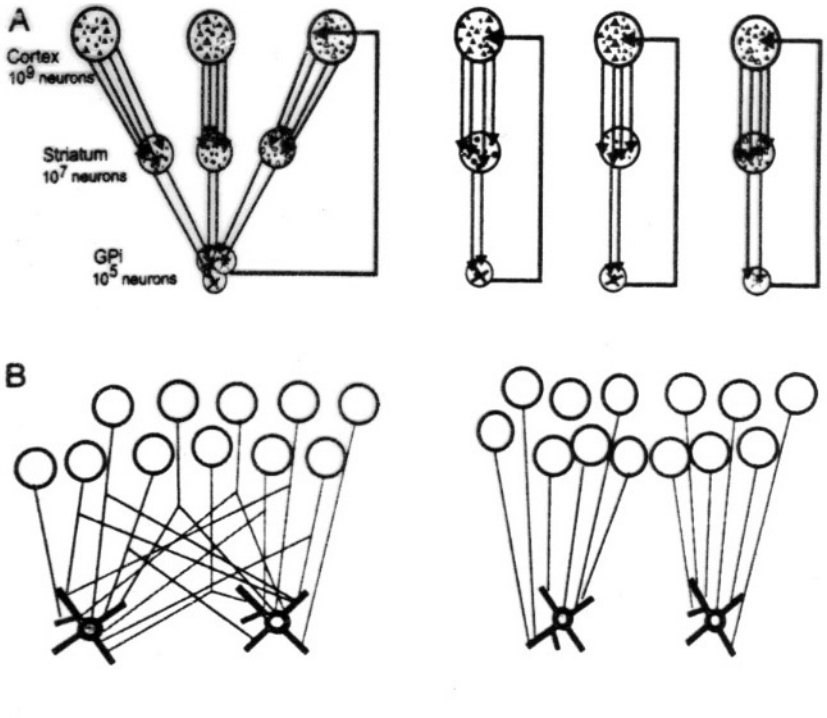
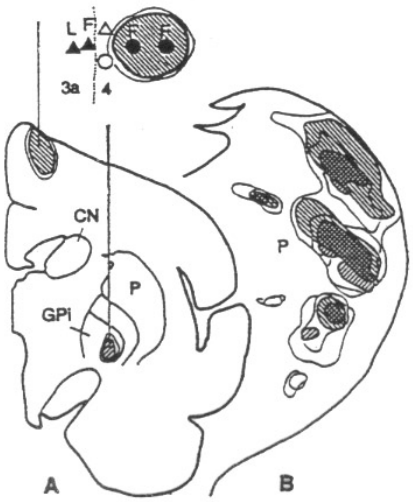




Diagram to explain changes of gene expression in basal gangila circuitry after 6-OHDA-lesion of the dopaminergic cells of the SN. This lesion result in increased ENK expression and activity of striatopallidal neurons. This results in increased firing of SNr-GABAergic neurons and diminished activity in thalamo-cortical axons.

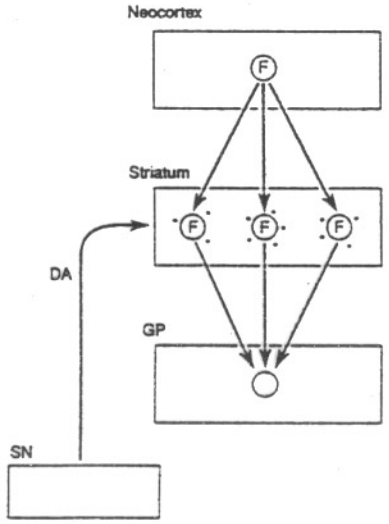

Huntington's disease
Cortex
Striatum
SNc
GPe
STN
Thalamus
GPi/SNr
Brainstem
Spinal cord
Parkinson's disease
Cortex
Striatum
SNc
GPe
STN
Thalamus
GPi/SNr
Brainstem
Spinal cord



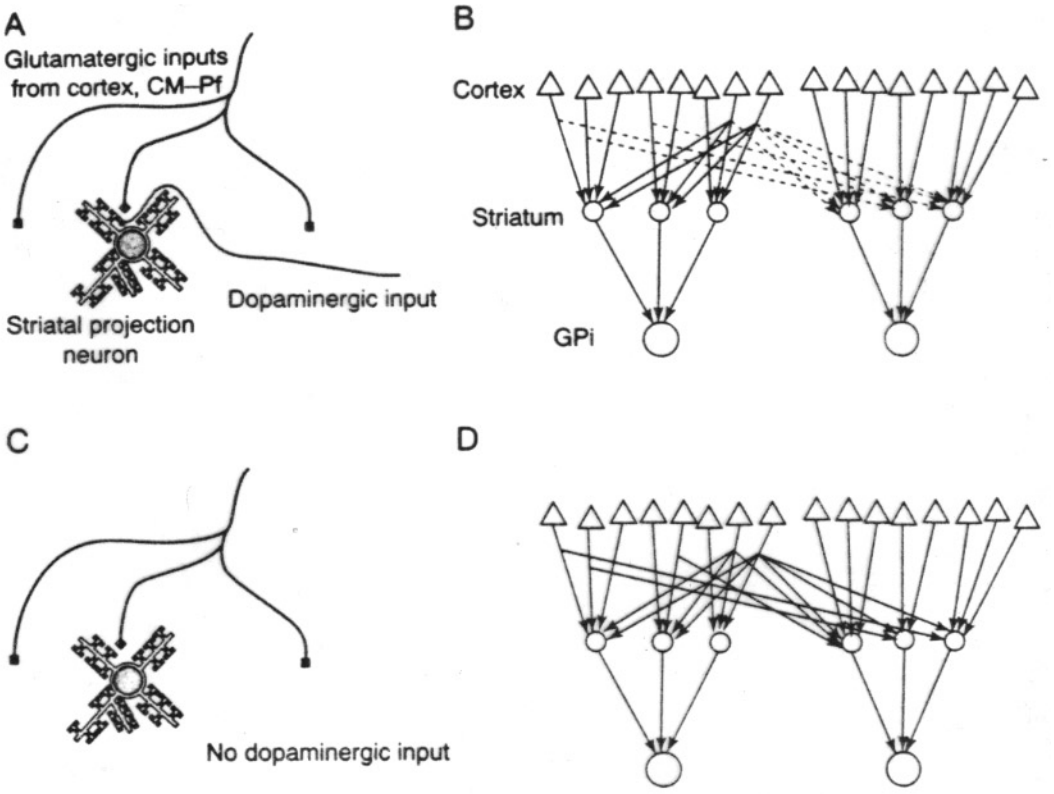

Conflicting views of information processing in the basal ganglia. Left old (convergent); Right: parallel processing. B: Zooming in the striatum-GPi connections according to the two models. According to the info-sharing, the two cells integrate the same information from many input sources. According to the segregared parallel model, there is no overlap in the incoming information to the two cells (Bergman et al., 1998).



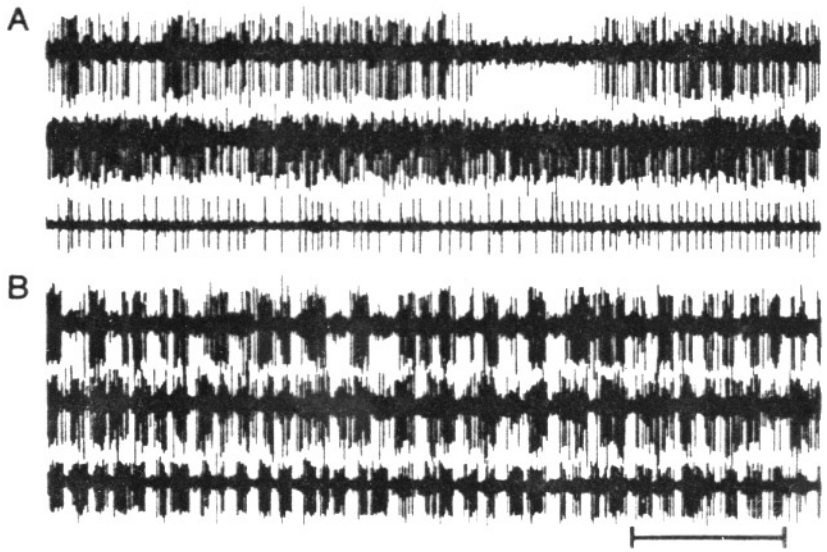

A: An injection of antergograde tracer was made in a small site in the motor cortex (area 4) representing the foot. In the same hemisphere , a small site in the pallidum was injected with retrograde tracer. Both the labeled axon projections from the cortex to terminal sites in the striatum and the labeled stratopallidal output cells are organizeds as sets of patches in the striatum. B The input clusters and output clusters overlap extensively (cross-hatching in B). Experiments using multiple-electrode recording suggest that during sensorimotor learning such distributed networks maybe coordinated by widely spaced striatal interneurons (possible cholinergic neurons) that acquire response properties on the basis of experienced reward (Graybiel et al., 1994).



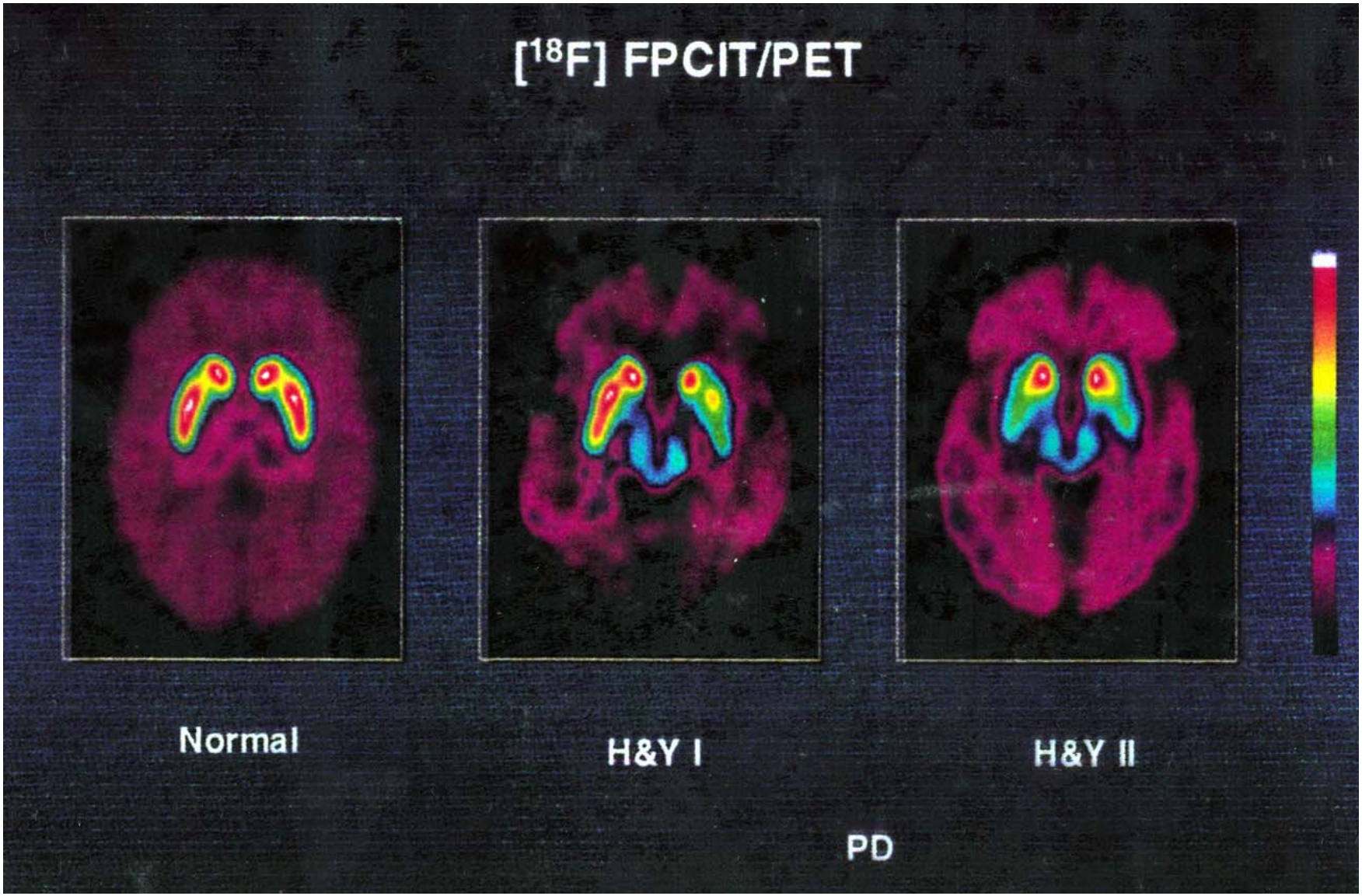

Model of divergent-reconvergent processing in basal ganglia pathways. Experimental evidence favors the divergence of cortical inputs to modules in the striatum. Any given module can receive somatotopically matched inputs (labeled F=foot) from different S1 areas (3a, 3b, and 1) and from M1. This divergence can be followed by reconvergence onto sets of basal ganglia output cells in the pallidum. Inputs from the midbrain SN-DA cells modulate this processing, as do local interneurons (small dots) (graybile et al., 1984)



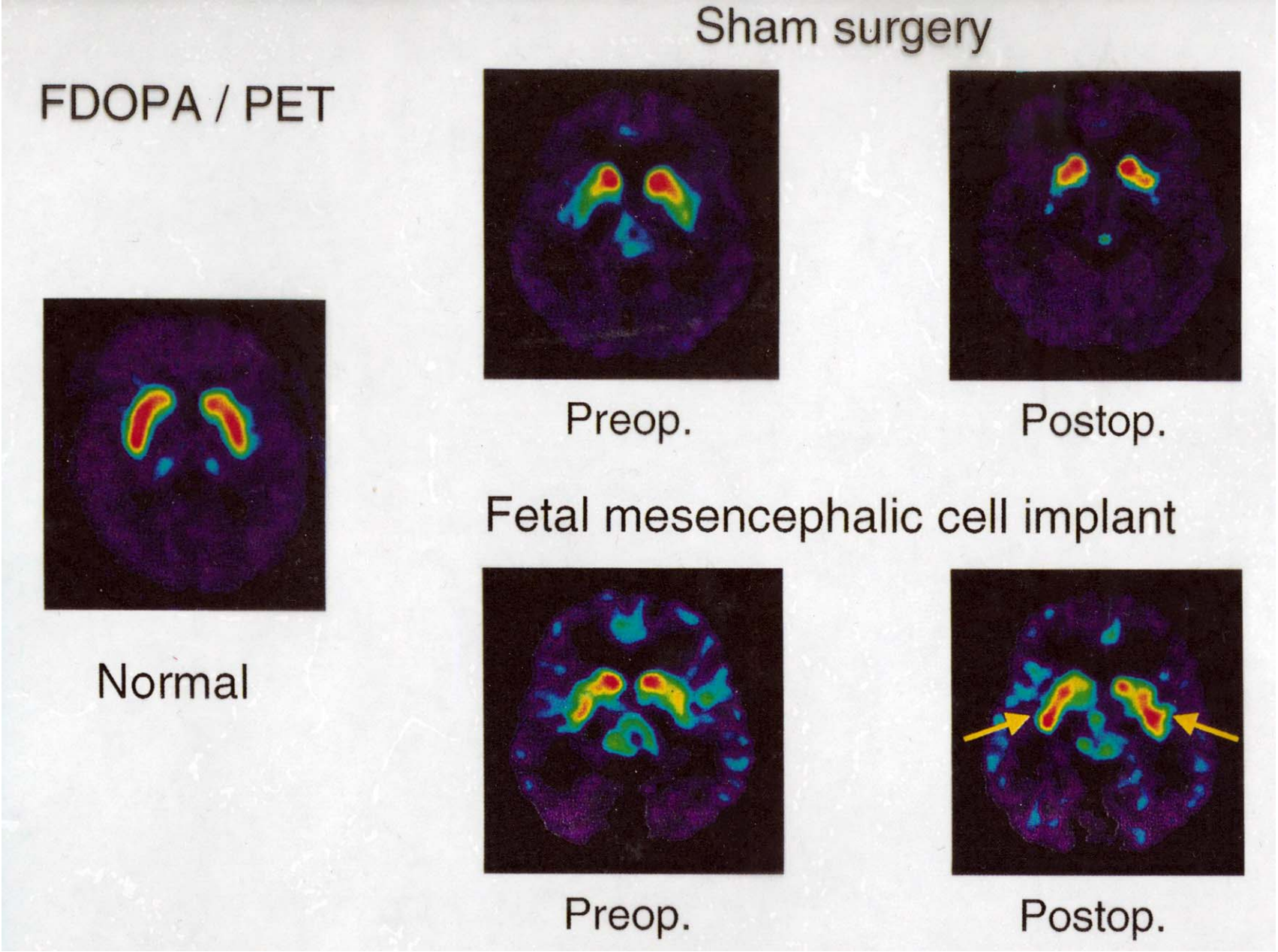

Dopamine modulation of functional connectivity in the basal ganglia. It is hypothesized that the main action of DA is to regulate the coupling level between the different subcircuits in the BG. In the normal state (A) DA endings on striatal spines can veto divergent glutamatergic inputs to the striatum thereby reducing the efficacy of cross-connections between channels. B: Diagramatic model of the resulting segregated channels in the normal state. Broken arrows represent cross-channle connectiuon with reduced efficacy. Following DA depletion © this segregation of afferent channels is lost, resulting in synchronized activation of pallidal cells (Bergman et al., 1998).



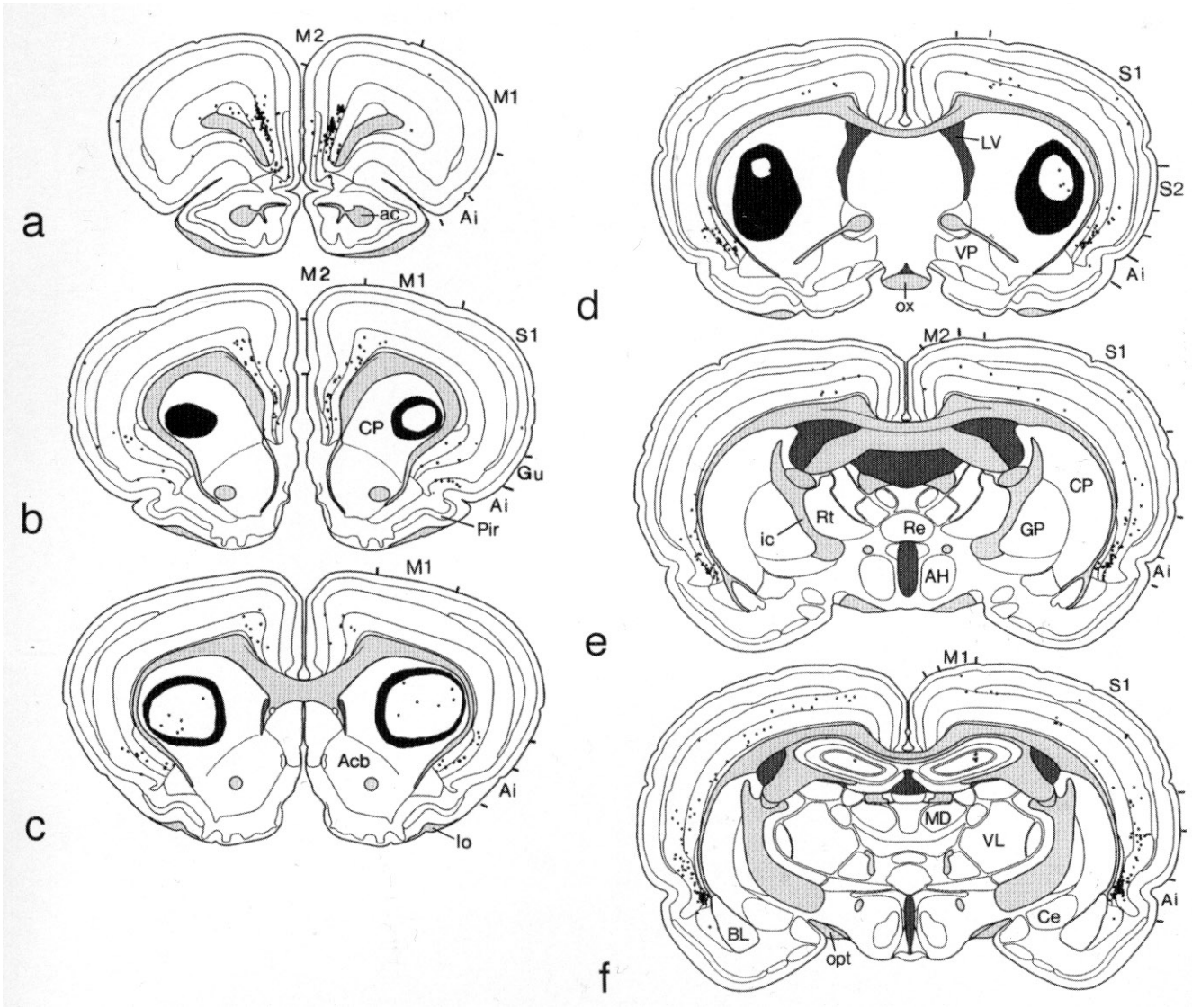

Multiple-electrode recordings in the globus pallidus of normal (A) and parkinsonian (B) monkeys. An example of 2.5 s of the simultaneous output of 3 electrodes. A: The upper two traces are from GPe, the lower one from GPi. B: in MPTP monkey intermittent episodes of synchronous, periodic bursting are seen in about one thrid of the recorded pallidal neurons, but never in normal monkeys (Bergman et al., 1998)

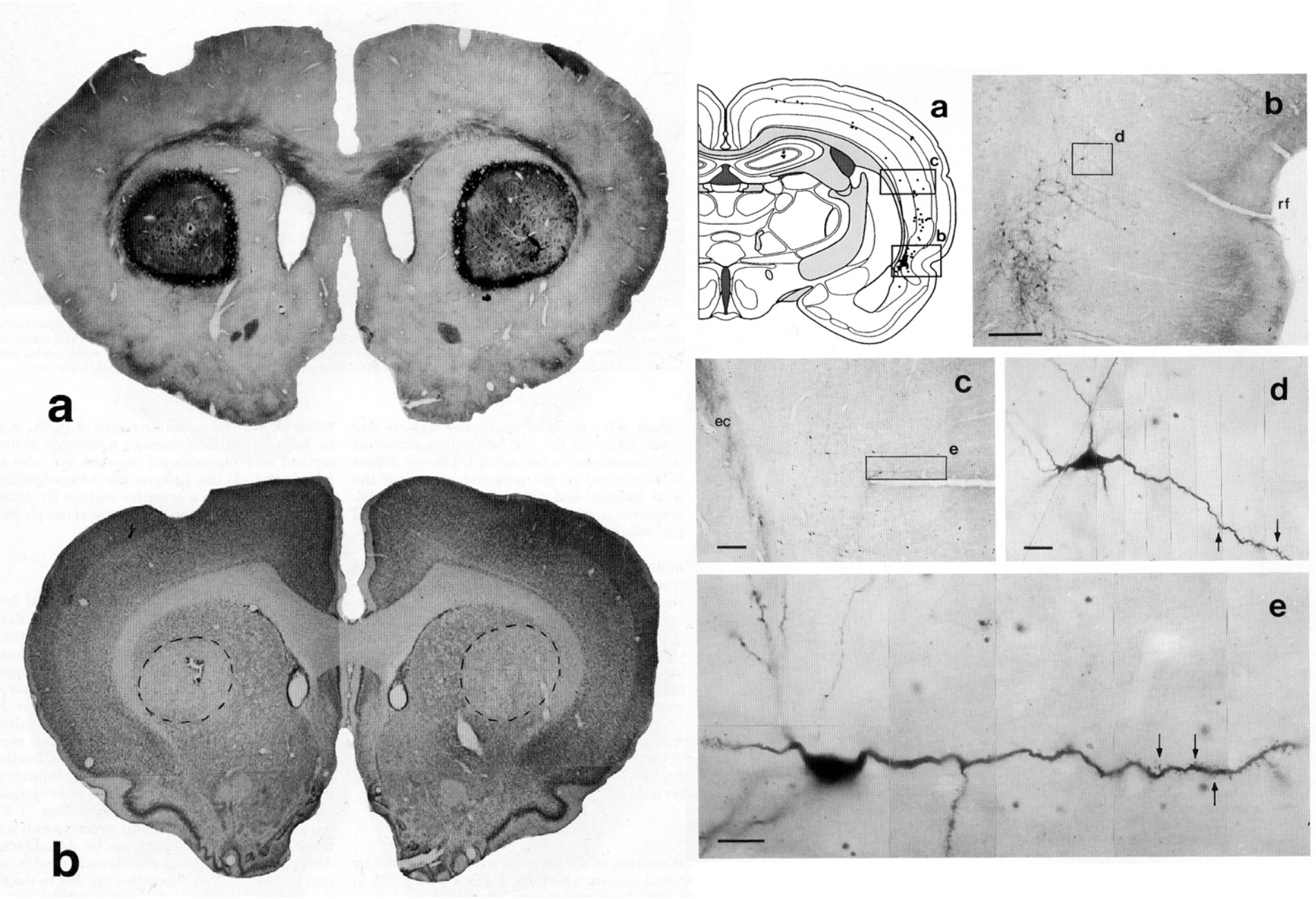



PET images obtained with [18F]FP-B CIT in a normal volunteer (left), in a patient with Hoehn-Yahr sage I, and in a patient with H-Y IIPD

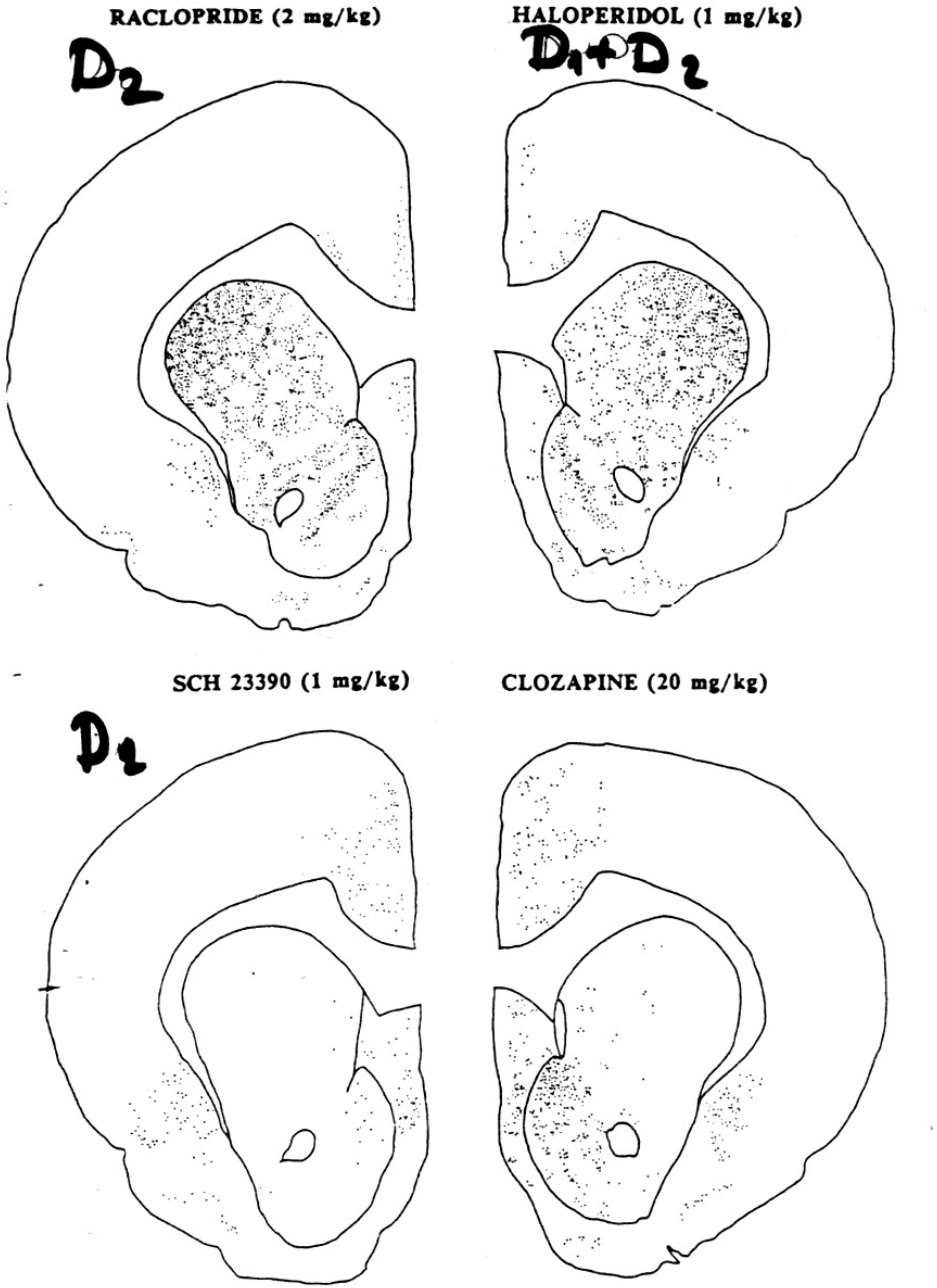



FDOPA/PET images from a normal volunteer (left) and from two patients of a sham and fetal nigral dopamine cell implantation in the putamen for advanced PD treatment. Baseline and 15 –month postoperative scans.

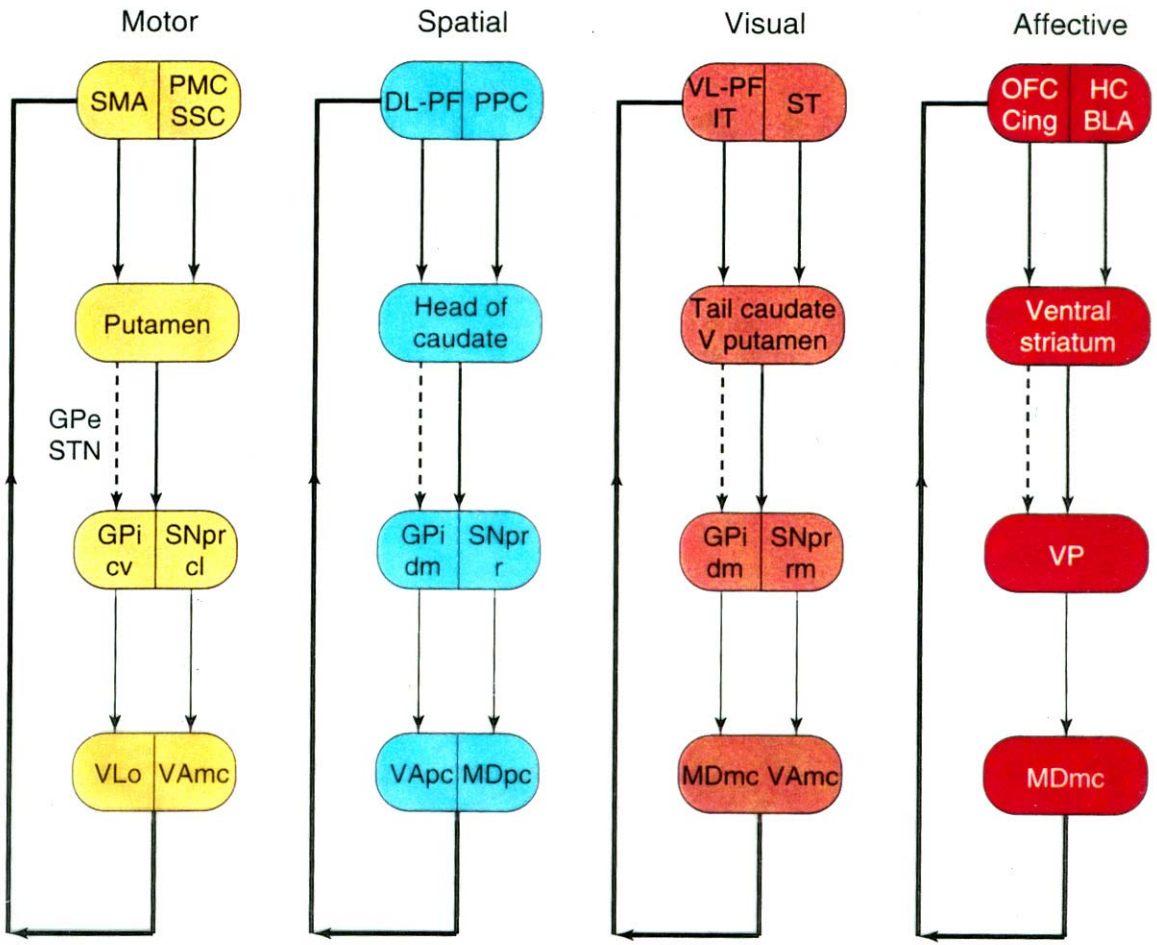

a
M2
M1
ac
Ai
b
M2
M1
S1
CP
Gu
Ai
Pir
c
M1
Acb
Ai
Io
d
S1
LV
S2
VP
Ai
ox
e
M2
S1
CP
Rt
ic
Re
GP
AH
Ai
f
M1
S1
MD
VL
Ce
Ai
BL
opt



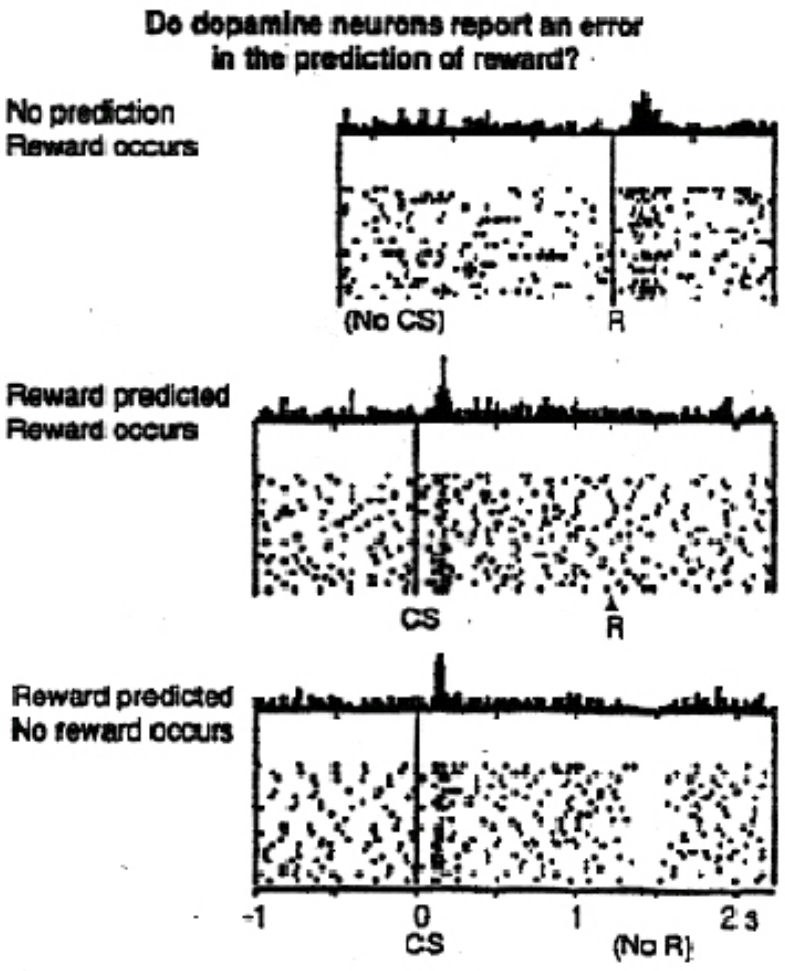

3-NPA induced degeneration in a Huntingon-model in rats. (Miller and Z, 1997)




Camera lucida drawings representative of the effects of the injection of raclopride, haloperidol, SCH23390 and clozapine on the distribution of Fos-positive neurons in the nucleus accumbens and striatum.




Corticostriatal loops, modified from the original scheme of Alexander et al. Four of the putative segregated, paralell loops are shown with possible functions labeled . Dashed lines indicate net inhibitory influences of the so-called ‘indirect’ striatal output pathways. (Lawrence et al, 1998)


Do dopamine neurons report an error in the prediction of reward?
No prediction
Reward occurs
(No CS)
R
Reward predicted
Reward occurs
CS
R
Reward predicted
No reward occurs
-1
0
1
2 s
CS
(No R)