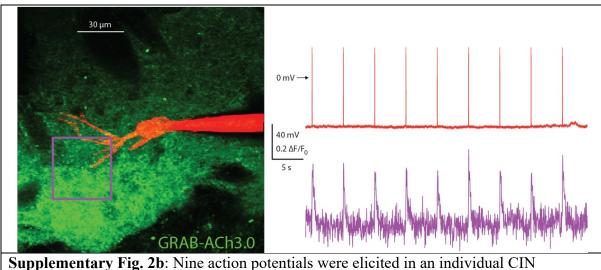
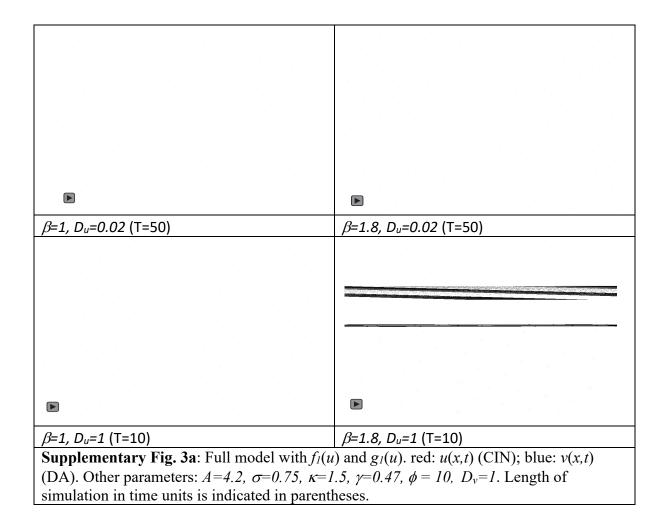
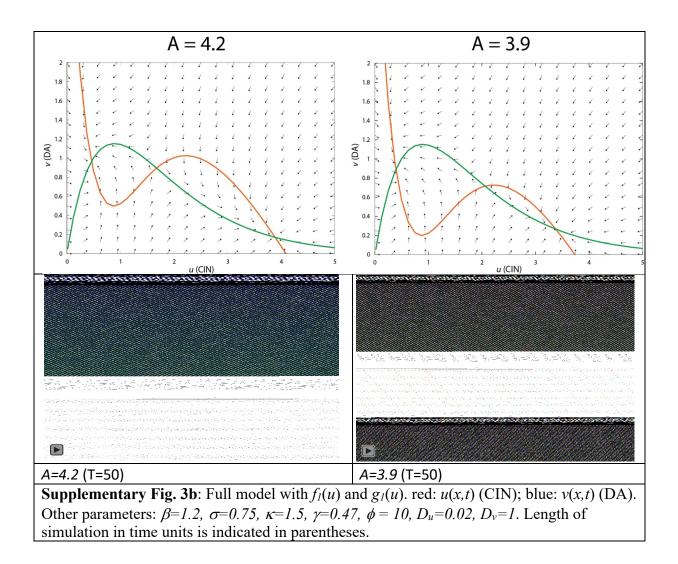


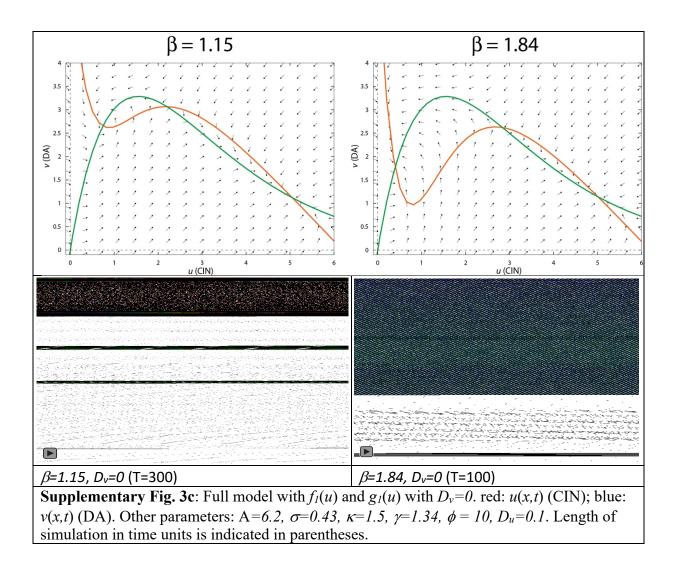
**Supplementary Fig. 2a**: Synchronous optogenetic activation (with a 1-ms long, 470 nm pulse) of CINs in an acute slice from a ChAT-ChR2 mouse whose dorsal striatum was inoculated with AAVs harboring GRAB-DA2m, causes DA release that is eliminated in the presence of 10  $\mu$ M mecamylamine, a nAChR antagonist. DA release was evoked in 8 slices from 4 mice, and the mecamylamine block was tested and confirmed in 3 slices from two of these mice. The artifact which is removed is explained in the Methods section.

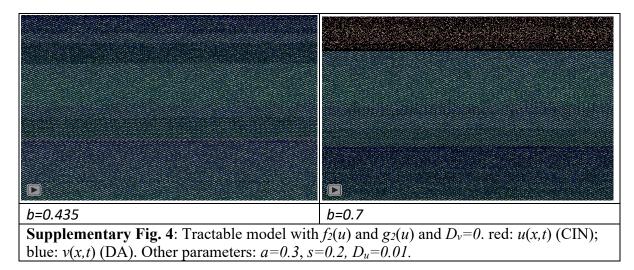


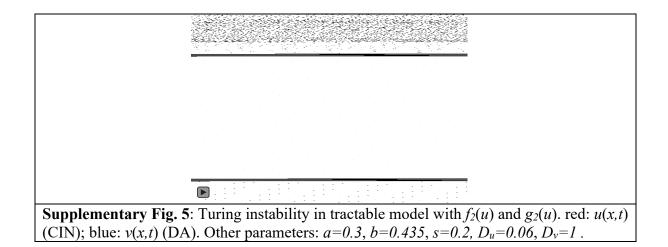
**Supplementary Fig. 2b**: Nine action potentials were elicited in an individual CIN hyperpolarized to quiescence (each with a 35 ms-long, 220 pA current pulse). The action potentials triggered ACh release (averaged from the color coded box) as visualized in wild-type mice whose dorsal striatum was inoculated with GRAB-ACh3.0. At this frequency (0.2 Hz) the median decrease of the amplitude of the 2<sup>nd</sup> to 9<sup>th</sup> ACh responses was no more than 4% relative to the 1<sup>st</sup> response (n = 4 CINs from N = 3 mice).

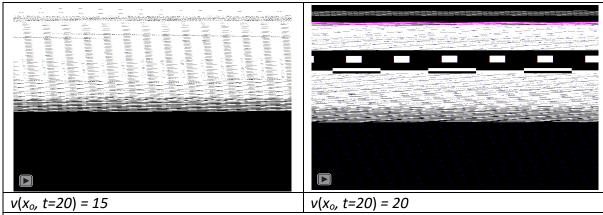




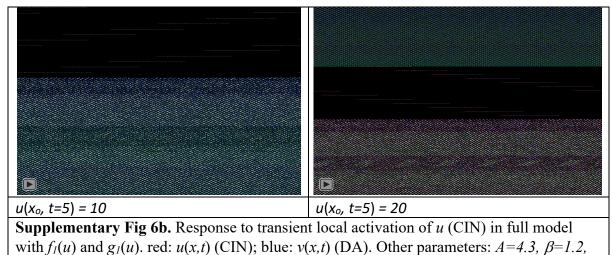




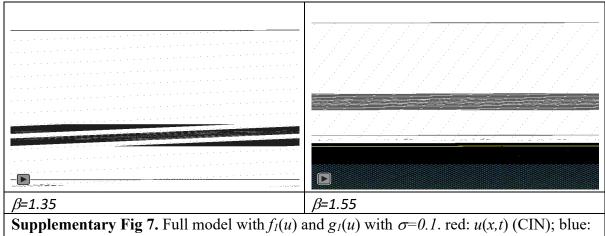




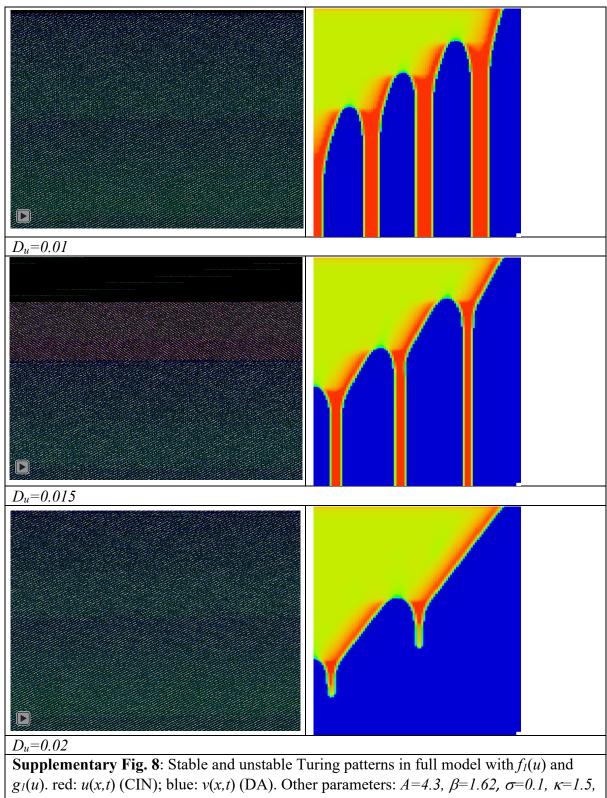
**Supplementary Fig 6a.** Response to transient local activation of v (DA) in full model with  $f_1(u)$  and  $g_1(u)$ . red: u(x,t) (CIN); blue: v(x,t) (DA). Other parameters: A=3.9,  $\beta=1.2$ ,  $\sigma=0.75$ ,  $\kappa=1.5$ ,  $\gamma=0.47$ ,  $\phi=10$ ,  $D_u=0.01$ ,  $D_v=1$ .



 $\sigma = 0.75, \kappa = 1.5, \gamma = 0.47, \phi = 10, D_u = 0.01, D_v = 1.$ 



v(x,t) (DA). Other parameters: A=4.3,  $\kappa$ =1.5,  $\gamma$ =0.47,  $\phi$  = 10,  $D_u$ =0.2,  $D_v$ =1.



 $\gamma = 0.5, D_v = 1.$ 

s=0.1	s=0.25
<b>Supplementary Fig 9.</b> Tractable model with $f_3(u)$ and $g_3(u)$ . red: $u(x,t)$ (CIN); blue: $v(x,t)$	
(DA). Other parameters: $b=0.14$ , $D_u=0.1$ , $D_v=1$ .	

