Hippocampal neurons, as well as dopaminergic cells in the compact zone of the substantia nigra and ventral tegmental area, are known to respond to the appearance of new stimuli [1, 36]. Increases in the activity of both hippocampal neurons and dopaminergic cells are evoked by the same experimental conditions [35]. It has been hypothesized that the hippocampus facilitates activation of dopaminergic cells via excitation of the nucleus accumbens, which is the ventral part of the input structure of the basal ganglia/striatum [26]. As GABAergic cells of the nucleus accumbens project, via the GABAergic cells of another nucleus of the basal gangli, i.e., the ventral pallidum, to dopaminergic cells of the ventral tegmental area (Fig. 1, a), discharges of hippocampal neurons in response to new stimuli can facilitate disinhibition of dopaminergic cells and facilitate their responses to new stimuli. The neuronal hippocampal-nucleus accumbens-ventral pallidum-ventral tegmental area loop functions not only in the descending direction, but also in the ascending [26]. Dopamine released in response to a new stimulus, increasing the efficiency of the excitatory synaptic input from neurons in field CA3 to neurons in hippocampal field CA1, increases their activity. Thus, on the one hand, the activity of dopaminergic cells depends on the responses of the hippocampus to new stimuli and, on the other, it influences the functioning of the hippocampus (Fig. 1, a). It has been suggested that the hippocampus and dopaminergic structures are activated independently [35]. Another hypothesis holds that the dopaminergic influence facilitates really production of hippocampal neuron responses to new stimuli [35]. This is supported by data showing that dopaminergic activity also occurs in the absence of novelty, and is dependent on the expectation of reinforcement, for which hippocampal activity is not required.
These hypotheses do not explain the fact that hippocampal neuron responses become weaker after 8–20 presentations of sensory stimuli [1]. This effect, subsequently termed response extinction, is also seen in dopaminergic cells [36], as well as in neurons of the visual and temporal areas of the cortex [12]. Extinction of responses to unreinforced sensory signals is also seen in striatal neurons, the extent of the effect depending on the dopaminergic input to the striatum [5]. As the causes of the extinction of responses to repeated stimuli are unclear, the aim of the present work was to analyze the mechanisms which might underlie this phenomenon.

Widely used models consider only the excitatory input to the hippocampus from the entorhinal cortex. Our model considers the point that the hippocampal formation also receives excitation from the thalamic nucleus reuniens and the adjacent ventral part of the median complex [43, 51]. The nucleus reuniens projects to field CA1 and the subiculum [47] (Fig. 1, b). The fact that neurons in field CA1/subiculum project predominantly to the shell of the nucleus accumbens, which projects via the ventral pallidum to the midline thalamic nuclei, while the latter have recurrent projections to the nucleus accumbens and hippocampus [16], results in the formation of closed hippocampus–basal ganglia–thalamus–hippocampus loops (Fig. 1, b). We have previously demonstrated that in closed neuronal loops whose component parts are the basal ganglia and thalamus, dopamine-dependent modulation of the excitatory inputs to the striatum leads to reorganization of activity in the structure exciting the striatum, particularly the cortex [2, 4, 39]. The existence of this mechanism provides grounds for suggesting that the altered responses of neurons in the hippocampus and structures connected to it may take part in dopamine-dependent changes in the efficiency of synaptic transmission between the hippocampus and striatum.

**Characteristics of the functional organization of the neuronal hippocampus–basal ganglia–thalamus–hippocampus loop.** Understanding of the functional mechanism of the hippocampus–basal ganglia–thalamus–hippocampus loop requires consideration of several characteristics of the organization of connections in this loop. As the nucleus reuniens receives inputs from the lateral geniculate body, the superior colliculi, and the pedunculopontine nucleus [29, 43], sensory (and particularly visual) information can reach hippocampal field CA1 bypassing the cortex (Fig. 2). The subiculum projects not only to the ventral striatum, but also to the medial, ventral, rostral, and caudal parts of the caudate nucleus and putamen of the striatum [19]. These associative parts of the striatum receive excitation from the visual areas of the cortex, whose activity in turn is regulated by the basal ganglia via the thalamus [4, 39]. Hippocampal terminals from the fimbria/fornix converge on the dendrite branches and spines of projection cells in the nucleus accumbens with dopaminergic fibers from the ventral tegmental area, as well as with axon terminals from the nucleus reuniens, entorhinal, and prefrontal areas of the cortex [17, 33]. Stimulation of the fornix/fimbria evokes monosynaptic responses with latent periods of 8–10 msec in the nucleus accumbens [11]. Cross-correlation analysis of simultaneously recorded pairs of cells in the hippocampus and nucleus accumbens showed that hippocampal neurons usually fire earlier than spiny cells [40].

The mechanism of plasticity in the striatum which we suggested previously [2, 38] was based on the differential nature of the modulation of inputs to functionally diverse spiny neurons in the dorsal striatum, which has a heterogeneous structure. The dorsal and associative parts of the striatum (the caudate nucleus and putamen) contain clusters termed striosomes. Strionigral neurons of striosomes project to the compact zone of the substantia nigra [31, 32]. The other part of the striatum, termed the matrix, contains two functionally differing types of spiny neuron. Strionigral neurons give rise to the “direct” disinhibitory...