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## Shaping of STDP curve by interneuron and Ca<sup>2+</sup> dynamics Lynsey McCabe<sup>\*1</sup>, Paolo Di Prodi<sup>1</sup>, Bernd Porr<sup>1</sup> and Florentin Wörgötter<sup>2</sup>

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Spike-timing-dependent-plasticity (STDP)[1,2] is a special form of Hebbian learning [3] where the relative timing of post- and presynaptic activity determines the change in synaptic weight. More familiarly, the postsynaptic and presynaptic activity correspond respectively to the derivative of the membrane potential V<sub>m</sub> and the NMDA channel activation [4]. We present a model where the postsynaptic activity is modelled by the derivative of the Ca<sup>2+</sup> concentration. Using a model of a pyramidal cell, attached interneuron and detailed Ca2+ dynamics, we show that the classical STDP curve is greatly altered, in particular, that long term depression (LTD) is markedly reduced [5] while LTP remains close to the original expected weight-change curve. In addition to this we have shown that by reducing the NMDA activity in the circuit model there is a noticeable change in the LTD/LTP magnitude in the STDP weight-change curve. This modification causes two effects; it reduces plasticity in the excitatory neuron but also reduces inhibition on the excitatory neuron. Therefore we show that by decreasing NMDA activity there is a clear reduction in LTD and LTP. This appears much like the "classical" STDP curve albeit scaled down in ratio to the reduced NMDA activity. In this study we have shown that the inhibitory interneuron reduces the LTD part of the STDP weight change curve. The more inhibition seen, the less LTD in the excitatory neuron. Thus, a hypofunction of inhibitory neurons will lead to more LTD in cortical structures and ultimately to less cortical activity. This hypofunction could be a possible mechanism of how administration of the NMDA antagonist PCP causes cortical hypoactivity[6] after a time lapse of a few days,

and is already a topic of interest in the research of schizophrenia.

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