Memory reconsolidation is the process in which reactivated long-term memory (LTM) becomes transiently sensitive to amnesic agents that are effective at consolidation. The phenomenon was first described more than 50 years ago but did not fit the dominant paradigm that posited that consolidation takes place only once per LTM item. Research on reconsolidation was revitalized only more than a decade ago with the demonstration of reconsolidation in a well-defined behavioral protocol (auditory fear conditioning in the rat) subserved by an identified brain circuit (basolateral amygdala). Since then, reconsolidation has been shown in many studies over a range of species, tasks, and amnesic agents, and cellular and molecular correlates of reconsolidation have also been identified. In this talk, I will first define the evidence on which reconsolidation is based, and why the evidence for specific impairments in consolidation, reconsolidation and LTM maintenance always lead to memory erasure. Last, I will refer to the potential clinical implications of reconsolidation. These include the ability to cause the synaptic circuits of a variety of psychopathologies to become transiently un-stored for a short period of time. If the mechanisms mediating restabilization of this circuit are prevented from being restored by a behavioral or pharmacological intervention, then that individual’s psychopathology would be reduced within a single session. To date this has been shown to be a fact, for psychopathologies ranging from PTSD, drug-cue induced craving, and Phobias.

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