Stochastic models of viral infection
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We develop biophysical models of viral infections from a stochastic process perspective. The entry of enveloped viruses is treated as a stochastic multiple receptor and coreceptor engagement process that can lead to membrane fusion or endocytosis. The probabilities of entry via fusion and endocytosis are computed as functions of the receptor/coreceptor engagement rates. Since membrane fusion and endocytosis entry pathways can lead to very different infection outcomes, we delineate the parameter regimes conducive to each entry pathway. After entry, viral material is biochemically processed and degraded as it is transported towards the nucleus. Productive infections occur only when the material reaches the nucleus in the proper biochemical state. Thus, entry into the nucleus in an infectious state requires the proper timing of the cytoplasmic transport process. We compute the productive infection probability and show its nonmonotonic dependence on both transport speeds and biochemical transformation rates. Our results carry subtle consequences on the dosage and efficacy of antivirals such as reverse transcription inhibitors.