Models of the Subduction Interface Motivated by Field Observations

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Cellular automaton modeling of the subduction interface shows that many of the features of subduction seismicity such as earthquake clustering at a variety of frequencies, an updip limit to the seismogenic zone, and temperature dependence on earthquake size distributions can result from the failure of asperities that nucleate during interseismic periods at rates determined by silica kinetics. Modeling is motivated by observations of ancient fault zones that were accreted within the temperature range of the seismogenic zone. In such cases, two types of structural fabrics are recognized that suggest different mechanisms of plate boundary shear. Type 1 is characterized by pervasive scaly fabrics and veins related to stratal disruption within a shear zone 10’s- 100 meters wide. Microstructures indicate local silica redistribution from scaly microfaults to veins, with periodic cracking and partial sealing. These observations are interpreted as evidence for slow shearing by linear viscous flow within a system that is buffered at fluid pressures that exceed s_3 and experiences periodic fluctuations in crack porosity, possibly due to slow earthquakes. Type 2 is defined by a discrete 1-3 meter thick fault zone characterized by gouge and in many cases pseudotachylite, an indicator of partial melting. Type 2 structure is interpreted as a record of coseismic slip along the plate interface after any footwall fracture network is healed by dissolution and precipitation. These two types of behavior, observed at any given fault zone, could occur side by side along the plate interface any given time. If there is heterogeneous effective stress within the seismogenic zone, these variations in porosity and normal stress would lead to gradients in chemical potential and silica redistribution/healing during the interseismic period. To evaluate the effects of these processes, we construct a cellular automaton model where the plate interface is represented by a layer of cells linked via springs to nearest neighbors and to a rigid plate that drags the cells at a constant rate. Cells slip according to two different failure criteria that correspond to type 1 and 2 slip behavior. We consider three different cases: a) no asperities and uniform interface properties, b) asperities whose nucleation rate increases linearly with temperature, and c) asperities whose nucleation rate increases exponentially with temperature, as expected based on Arrhenius-law silica kinetics. Only in the case of thermally activated healing related to silica kinetics are there supercycles of buildup and release of an overall slip deficit, with 1) increasing asperity area during the build up of elastic strain followed by clusters of earthquakes, 2) a temperature-based updip limit to genesis of earthquakes, and 3) a power law size distribution of earthquakes and a b-value that varies as a function of temperature.

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