

Evolution of structural phase states of 08Cr18Ni10Ti steel subjected to high-cycle fatigue with electrostimulation

Molotova K. Ye.¹, Konovalov S. V.¹, Kozlov E. V.², Ivanov Yu. F.², Gromov V. E.¹

(1. Siberian State Industrial University, Novokuznetsk 654007 Russia; 2. Tomsk State Architecture-Construction University, Tomsk 634003, Russia)

This work contains analysis results from the study of defective substructure and phase state of steel subjected to fatigue loading and pulse current action in order to increase its fatigue life. Steel fatigue loading for $N_1 \sim 100000$ cycles resulted in higher scalar density of dislocations, formation of stacking faults and microtwins; coagulation of TiC particles and their size increase up to 121.6 ± 9.3 nm; formation of microcracks along carbide-matrix phase boundary. Fatigue failure of samples after $N_2 \sim 170000$ stress cycles resulted in evolution of dislocation substructure with reticular dislocation substructure being dominant in this case. Steel fatigue loading and subsequent failure lead to a substantial (1.6 times) average size growth of carbide phase particles. It was determined that for particles with average size over $0.5 \mu\text{m}$ microcracks form under fatigue along the particle/matrix phase boundary.

Fatigue failure of electrostimulated steel after $N_3 \sim 250000$ cycles has lead to the presence of a large number of grains a few microns in size in the failure area. Large size grains characteristic for initial state of the steel are practically absent. Therefore, dynamic recrystallization processes accompanied by considerable grain structure atomization develop in steel as a result of fatigue loading. Current-induced failure has increased the scalar density of dislocations 1.6 times (relative to the electrostimulated state at the intermediate fatigue stage); from $3.2 \times 10^{10} \text{ cm}^{-2}$ to $5.4 \times 10^{10} \text{ cm}^{-2}$. This resulted in subsequent steel dislocation substructure evolution; cellular substructure volume fraction increased up to 92% at the expense of coil-reticular substructure (the rest is occupied by cellular-reticular dislocation substructure).