**eTable 4.** Summary of the Key Molecular Properties of Aflibercept, Ranibizumab, Bevacizumab, and Brolucizumab

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Anti-VEGF Agent** | **Molecular Weight, kDa97** | **Potency (IC50), pM\*,69** | **Binding Affinity for VEGF-A165 (KD), pM†** | **Intravitreal Half-life, days‡** |
| Aflibercept | 115 | 18 | 0.4963 | 9.14–11 (2.0 mg)64 |
| Ranibizumab | 48 | 45 | 4663 | 7.19 (0.5 mg)68 |
| Bevacizumab | 149 | 116 | 5863 | 9.82 (1.25 mg)67 |
| Brolucizumab | 26 | 54 | 28.466 | 5.1 (6.0 mg)70 |

\*IC50 was determined by monitoring the inhibition of VEGF-induced human retinal microvascular endothelial cell proliferation over 2 days.69

†Kinetic binding to the VEGF family of molecules was determined in vitro by measuring the KD in a single study for aflibercept, ranibizumab, and bevacizumab.63 The kinetic binding affinity of brolucizumab was determined in a separate in vitro study.66

‡The presented intravitreal half-life is specific to the stated concentrations of aflibercept (mean = 9.14 days; median = 11 days), ranibizumab, brolucizumab, and bevacizumab.

IC50 indicates half maximal inhibitory concentration; KD, equilibrium dissociation constant; VEGF, vascular endothelial growth factor.