Two strains of genetically epilepsy-prone rats (GEPRs) have been derived from Sprague Dawley stock. One strain, known by the acronym GEPR-9, has a more pronounced epileptic condition than the other strain, known by the acronym GEPR-3. Only a small fraction of commercially available Sprague Dawley rats exhibits evidence of epilepsy. GEPRs are similar to most humans with epilepsy in that their general behaviors appear normal. GEPRs also share other traits with their non-epileptic counterparts. They are susceptible to forebrain and brainstem seizures produced by convulsant drugs and electrical currents. Because GEPRs and normal rats share these seizure non-epileptic brain rather than to an understanding of epilepsy. However, humans with epilepsy, the GEPR and other mammalian models of genetic epilepsy are distinctive because they are characterized by seizure predisposition. They exhibit seizures in response to stimuli that do not cause such episodes in normal mammals. This property appears to represent the fundamental distinction between epilepsy and normality. Therefore, when GEPRs are used in protocols that compare their biology with that of non-epileptic rats, the results can be used to elucidate the underlying causes of epilepsy. Because seizure predisposition in GEPRs exists in the seizure naïve state, these rats provide a means for determining the pristine mechanisms underlying epilepsy in brains that are unadulterated by seizure-induced seizure propensity. The GEPR model is especially useful in determinations of epileptic abnormalities in interacting neuronal networks. Three manifestations of seizure predisposition are evident in GEPR-3s and GEPR-9s. Both exhibit a low incidence of spontaneous seizures, a highly evident exaggerated seizure responsiveness to stimuli which also provoke seizures in non-epileptic rats, and susceptibility to seizures induced by stimuli that do not cause seizures in normal rats. Seizure predisposition in GEPRs is subject to developmental influences. Thus, they are useful models of ontogenetically determined epileptogenesis. Since seizure induced aggravation of epileptogenesis has been documented in GEPRs, they are also a model of stimulus-exacerbated genetically-determined predisposition epileptogenesis. Because of these epileptic traits, GEPRs are useful for studies of the etiology of seizure predisposition and of antiepileptic drug development. In the field of applied pharmacology, GEPRs are applicable to the development of novel drugs that selectively ameliorate innately determined seizure predisposing abnormalities.