**Supporting Information**

***Data S1***

***Electrode implantation and video electrocorticography (ECoG)***

Anesthesia was induced by volatile isoflurane (4% induction, 1–2% maintenance) to implant epidural electrodes in frontal (bregma 0 mm, 3.5 mm lateral from midline) and occipital cortices (bregma −6.5 mm, 3.5 mm lateral from midline), bilaterally. The reference electrode was in bregma -10.0 mm, 2.0 mm lateral on the left. Filtered (0.3 Hz high-pass, 500 Hz low-pass) signals were acquired at 1 kHz/channel and stored after subtraction by traces of reference electrode (PowerLab/30, ADInstruments; Dunedin, Otago, New Zealand). Recordings were offline digitally filtered (band-pass: high 50 Hz, low 1 Hz) and analyzed by hand scroll using LabChart 8 pro software (ADInstruments). Videos were digitally captured by a camera connected to the computer and synchronized to ECoG traces by LabChart 8 pro internal trigger. All seizures were defined as ECoG segments with minimum duration of 10 s, continuous synchronous high-frequency activity, and amplitude at least twice as the previous baseline. Seizures and their durations were determined in the ECoG traces, and then investigated for a behavioral correlate by using the synchronized video recordings. Particularly, seizures were scored as stage 0 (or subclinical) if a clear epileptiform ECoG signal was present without corresponding evident behavior in the video; stage 1-2 in presence of absence-like immobility, “wet-dog shakes”, facial automatisms, and head nodding; stage 3, when presenting with forelimb clonus and lordosis posture; stage 4, corresponding to generalized seizures and rearing; and stage 5, when seizures consisted of rearing with loss of posture and/or wild running, followed by generalized convulsions. SE was defined as the period of time in which rats either did not recover normal behavior between one seizure and the other, or in which they displayed continuous shaking for more than 5 min. In our animal model, SE was allowed to self-terminate.

***Quantitative analysis of neurosteroids by liquid chromatography-electrospray tandem mass spectrometry (LC***–***MS/MS)***

Twenty-four rats treated with trilostane (n=12) or sesame oil (n=12) were used. Brains were carefully removed after euthanasia (isoflurane) and chilled on ice to dissect both hippocampi and neocortices for the LC–MS/MS analysis. Details about chemicals, reagents andstandard solutions were published previously.2

All the samples were spiked with internal standard solution, vortexed and added with acetonitrile/methanol (70/30; +1.0% formic acid). The samples were then sonicated, centrifuged and purified on Phree–SPE cartridges to remove endogenous phospholipids. Eluates were concentrated, derivatized with Amplifex Keto Reagent and transferred to autosampler vials for LC–MS/MS analysis, which was performed on a Kinetex XB–C18 column (100x2.1 mm; 2.6 µm particle size) preceded by a C18 Security Guard cartridge (2.1 mm) (Phenomenex). Mass spectrometric detection was performed using an Agilent QQQ–MS/MS (6410B) triple quadrupole operating in electrospray positive ionization mode.

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**Figure S1:** **Video electrocorticographic (ECoG) recordings in rats pretreated with trilostane or vehicle and exposed to kainic acid (KA)-induced *status epilepticus*.** Upward and downward arrowheads respectively indicate onset and termination of the ictal discharges. From panel (**A**) to panel (**D**), ECoG traces illustrating visually identified stage 4 and stage 5 seizures obtained 90 minutes after KA treatment are shown. It can be appreciated a higher seizure occurrence in trilostane-treated rats. However, 90 min later (panels **E**-**H**) stage 4 and 5 seizures occurred at a similar extent in both treatment groups but presented a longer duration in vehicle-treated rats.