Figure 2: Inflammation is a controlled process with an initiation, resolution and termination phase. After microbial invasion, lesion or chemical injury, the initiation phase starts with the production of pro-inflammatory mediators like LTB4 and PG2. These mediators increase inflammation until the Eicosanoid Switch, the end of the initiation phase, takes place. This occurs when the level of PGE2 plus PGD2 is equal to the LTB4 level. The resolution phase is entered, triggering the generation of anti-inflammatory mediators like LK, resolvins, protectins, maresins, PGD2 and PGF2α. When the total level of anti-inflammatory mediators exceeds the level of LTB4 the Stop Signal takes place. This is the last phase, the inflammation will be terminated by clearing the affected area [11].

The stress hormones produced by the systemic stress axes have a direct effect on the inflammation phases. A microbial invasion, lesion or injury sends off an alarm in the body, setting off the systemic stress system which produces NE as response and tunes the system to insulin and cortisol resistance [30]. The Eicosanoids Switch to resolution can only take place when NE is equal to the level of cortisol plus insulin and when cortisol sensitivity is recovered. The Stop Signal requires a low level of NE and normalized cortisol sensitivity. The termination phase is entered when the stress axes are switched off.