Supplementary Table 1: Identified possible search terms

[diet]	[target]
Ketone	Neuro* (Neuroinflammation, Neuron, Neurotoxicity,
Ketosis	neurodegeneration)
Ketogenic	Astrocyte
ketogenic diet	Glia
High-fat diet	Cortex
Low-carbohydrate diet	Brain
Carbohydrate-restricted diet	Mitochondria*
Medium-chain triglyceride	oxidative stress
Beta-hydroxybutyrate" [or 3-Hydroxybutyric Acid/],	anticonvulsant
Acetoacetate	Antiepileptic
metabolic therapy	Inflammation
	Anti-inflammatory

Ovid search strategy

1. ketogenic diet.mp. [mp=ab, hw, ti, ot, sh, kw, tn, dm, mf, dv, fx, dq, nm, kf, ox, px, rx, ui, sy]
2. high-fat diet.mp. [mp=ab, hw, ti, ot, sh, kw, tn, dm, mf, dv, fx, dq, nm, kf, ox, px, rx, ui, sy]
3. low-carbohydrate diet.mp. [mp=ab, hw, ti, ot, sh, kw, tn, dm, mf, dv, fx, dq, nm, kf, ox, px, rx, ui, sy]
4. carbohydrate-restricted diet.mp. [mp=ab, hw, ti, ot, sh, kw, tn, dm, mf, dv, fx, dq, nm, kf, ox, px, rx, ui, sy]
5. medium-chain triglyceride.mp. [mp=ab, hw, ti, ot, sh, kw, tn, dm, mf, dv, fx, dq, nm, kf, ox, px, rx, ui, sy]
6. rat.mp. [mp=ab, hw, ti, ot, sh, kw, tn, dm, mf, dv, fx, dq, nm, kf, ox, px, rx, ui, sy]
7. mouse.mp. [mp=ab, hw, ti, ot, sh, kw, tn, dm, mf, dv, fx, dq, nm, kf, ox, px, rx, ui, sy]
8. mice.mp. [mp=ab, hw, ti, ot, sh, kw, tn, dm, mf, dv, fx, dq, nm, kf, ox, px, rx, ui, sy]
9. animal.mp. [mp=ab, hw, ti, ot, sh, kw, tn, dm, mf, dv, fx, dq, nm, kf, ox, px, rx, ui, sy]
10. astrocyte.mp. [mp=ab, hw, ti, ot, sh, kw, tn, dm, mf, dv, fx, dq, nm, kf, ox, px, rx, ui, sy]
11. 3-Hydroxybutyric Acid.mp. [mp=ab, hw, ti, ot, sh, kw, tn, dm, mf, dv, fx, dq, nm, kf, ox, px, rx, ui, sy]
12. Beta-hydroxybutyrate.mp. [mp=ab, hw, ti, ot, sh, kw, tn, dm, mf, dv, fx, dq, nm, kf, ox, px, rx, ui, sy]
13. acetoacetate.mp. [mp=ab, hw, ti, ot, sh, kw, tn, dm, mf, dv, fx, dq, nm, kf, ox, px, rx, ui, sy]
14. ketone.mp. [mp=ab, hw, ti, ot, sh, kw, tn, dm, mf, dv, fx, dq, nm, kf, ox, px, rx, ui, sy]
15. ketosis.mp. [mp=ab, hw, ti, ot, sh, kw, tn, dm, mf, dv, fx, dq, nm, kf, ox, px, rx, ui, sy]
16. neuroinflammation.mp. [mp=ab, hw, kw, ti, ot, sh, tn, dm, mf, dv, fx, dq, nm, kf, ox, px, rx, an, ui, sy]
17. neurone.mp. [mp=ab, hw, kw, ti, ot, sh, tn, dm, mf, dv, fx, dq, nm, kf, ox, px, rx, an, ui, sy]
18. neurotoxicity.mp. [mp=ab, hw, kw, ti, ot, sh, tn, dm, mf, dv, fx, dq, nm, kf, ox, px, rx, an, ui, sy]
19. neurotransmission.mp. [mp=ab, hw, kw, ti, ot, sh, tn, dm, mf, dv, fx, dq, nm, kf, ox, px, rx, an, ui, sy]
20. *glia/
21. 1 or 2 or 3 or 4 or 5
22. 6 or 7 or 8 or 9
23. 10 or 11 or 12 or 13 or 14 or 15 or 16 or 17 or 18 or 19 or 20
24. 21 and 22 and 23

Supplementary Table 2: Individual study characteristics and outcomes
(*Reference list in the main manuscript*)

Disease (n) [diet length]	Mechanistic theme/s presented	Positive outcome reported; (*Neutral or negative outcome reported in italics*)
Age-related degeneration (5) [4 weeks to 6 months]	Cellular energetics /metabolism	Decrease glucose transporters and increased MCT (ketone) transporters [42,43]. Improved cerebral metabolic rate of glucose metabolism in aged brain via restored glutamate levels [46].
	Synaptic transmission	Reversal of age-related decline in hippocampal vesicular transporters for GABA and glutamate [42] and post synaptic excitation and plasticity [44].
	Neurotransmitters	Upregulated GABA(A) receptor subunits a1 in the hippocampus [45].
	Mitochondria	Increased mitochondrial mass in hippocampus and upregulated mitochondrial antioxidant defences *(but impaired mitochondrial dynamics and function)* [45].
	Structural integrity	*(Accelerated atrophy, neurodegeneration, and reactive astrogliosis in the hippocampus [45]).*
	Epigenetic regulation	Upregulation of several genes involved in presynaptic glutamate regulation and postsynaptic excitation and plasticity in the hippocampus and dentate gyrus [44].
Alzheimer's (4) [2 weeks to 4 months]	Cellular energetics	Increased cerebral metabolism of glucose and ketones [49].
	Structural integrity	*(No effect on beta-amyloid or precursors [47]),* Reduced beta-amyloid [50].
	Signalling pathways	Reduced mTOR and increased eNOS [48].
	Vascular supply	Significant increases in cerebral blood flow [48].
Autism (7) [10 days to 8 months]	Mitochondria	Improved bioenergetic profile and improved oxygen consumption, [51] improved function and morphology with reduced phosphorylation of key protein regulators [52].
	Cortical / neuronal excitability	Reduced seizure events via presynaptic mechanisms [53] and balance of excitation and inhibition restored towards more normal levels of inhibition [57].
	Signalling pathways	cAMP/GPR: effector substrates for glutamate, serotonin, nNOS, and dopamine [54], *(O-GlcNAc: integrates energy supply with changes seen in the liver but not in the brain [56]).*
	Structural integrity	Improved myelin formation and white matter development [54].
	Epigenetic regulation	Differences in mitochondrial gene expression [55].
Cerebral ischemia (4) [21 days to 25 days]	neuroprotection	Reduced likelihood of seizure and severity of myoclonic jerks with cerebral hypoxia [58] and elimination of post ischemia hippocampal neurodegeneration [59].
	Epigenetic regulation	Upregulated HIF-1a/HIF-2a and HIF regulated genes [61].
	Signalling pathways	K_{ATP} channels not demonstrated to be involved in neuroprotection [60] and A1R activation which increases phosphorylation of Akt and ERK1/2 providing neuroprotection [61].
	Vascular supply	Reduced infarct volume, increased regional cerebral blood flow and adenosine levels [61].
CNS general (24) [1 week to 3 months]	Cellular energetics / metabolism	NAD+ elevation via efficient ketone metabolism for substrate for other neuroprotective processes [63]. Increased MCT1 and GLUT1 in brain endothelial cells [68], increase blood-brain barrier MCT1 expression with increased AcAc and glucose uptake in brain [72]. Decreased neuronal glycolysis with increased astrocytic metabolism [70] and improved metabolic efficiency in the brain [74]. Increase intracellular BOHB in hippocampus [75], correlating with serum BOHB levels [83]. Increase in brain PGC1β mRNA suggesting enhanced brain aerobic infrastructure/respiratory efficiency [76]. Oxidative metabolism derived from AcAc, with glucose contribution to Acetyl-CoA decreased by 30% [85].
	Cortical excitability	Elevated blood ketone level and seizure threshold [67].
	Neurotransmitters	Hippocampal expression of AMPA-type GluR1 was significantly increased [64]. Increased valine, leucine and isoleucine, *(no change in GABA and decreased amount of glutamate [70]).* Increased GABA/glutamate ratio [74]. GABA concentration constant but derived from ketone bodies [85].
	Signalling pathways	NAD+ driven increase in sirtuins with broad neuroprotective effects [63]. Kynurenine (tryptophan metabolite) downregulated in hippocampus and plasma [66]. Kynurenic acid upregulated in hippocampus and striatum but not cortex [84]. Lipid metabolism gene expression in the hippocampus altered potentially via dietary lipid signalling [69]. Nrf2 detoxification pathway activated via mild oxidative stress to induce glutathione synthesis [71]. *(BDNF reduced in the striatum*) but not in the hippocampus [82] BDNF mRNA increased in the brain and showed a 12-hour phase shift in its circadian timing [65].
	Synaptic transmission	*(Unaltered basal synaptic transmission and long-term potentiation in the hippocampus [67]).*
	Epigenetic regulation	MCT1 upregulation on blood-brain barrier [72]. Altered hippocampal mRNA expression of genes related to lipid and energy metabolism [69] Increase in brain PGC1β mRNA (bioenergetic function) and decreased TNF-α mRNA (inflammation) [76]
	Redox balance	Reduced hippocampal oxidative stress markers that correlated with reduced PARP-1 requirement [63]. Acute production of H_2O_2 and 4-HNE activating Nrf2 and improving mitochondrial redox state [71] and lowered Oligomycin-induced ROS production [78]. *(Decreased antioxidant capacity in the cerebellum, no change in the cortex),* 400% increase of GPx in the hippocampus [86].
	Structural integrity	No evidence of negative morphologic or histochemical alterations in the brain [73] Variable neuroanatomical differences with prenatal exposure to ketogenic diet with altered neurobehavior in adulthood [79].

	Mitochondrial	*(Decreased mitochondrial DNA levels)* without a reduction in mass [76] and increased maximum mitochondrial respiration rates in the hippocampus [78].
	Neuroplasticity	No evidence of negative impact on neurogenesis in the dentate gyrus [77]. *(fEPSPs paired-pulse potentiation unchanged suggesting no change in short-term plasticity [80]).*
	Vascular supply	Reduced capillary density linked to reduced tumour growth and prevention of epilepsy when combined with calorie restriction [81].
Diabetes (2)	Cellular energetics	*(Blunted glucagon release to hypoglycaemia and neuroglycopenia [87]).*
[1 to 3 weeks]	Neuroprotection	Reduced neuronal death [88].
Epilepsy / seizures (91) [1 week to 9 weeks]	Neuronal / cortical excitability	Increase threshold for seizure induction [89-92,94,107,115,118,142,150,154,156,167,172] and abolished correlation with firing rate [171]. Increased after discharge threshold [118,124], and after-discharge duration [124,127]. *(No increase in after-discharge threshold [151]).* Reduced incidence of convulsions [93,110,113,116,124,125,141,158,161-163,179] including after KD cessation [135]. Decreased intensity and duration of seizure [104,110,113,141,173,177], *(no reduction in severity once the seizure has commenced [154]).* Increased latency to seizure [107,110,113,114,144,155,164,167,170,172], *(no increase in latency [150]).* Reduction of the cortical spreading depression velocity of propagation for short-term KD [152]. Reduced pathologic neuronal activity [108,160] and dampened hyperactive mossy fiber synapses [160]. Delayed progression of seizure stage [124,127,159] and increased lifespan [155,159]. *(Increased severity of seizure evoked by maximal electric shock [91,166,175] and kainite [93]).* *(No correlation between BOHB and seizure threshold [92] or latency to seizure [114]).* Increased number of seizures required to reach status epilepticus (single seizure >5 mins) [109]. Suppression of drug-resistant manifestations [111]. Reduced glucose levels required to maintain reduced excitability [125]. Attenuation of cortical sensitivity induced by a variety of neurotoxins [129]. Restoration of normal circadian rhythms [158]. Alterations in the type of dietary fat affect seizure resistance [131]. *No change in baseline excitability [178] or seizures [140].*
	Cellular energetics / metabolism	Calorie restricted KD increased seizure resistance [91]. Decreased glycogen levels and elevated glutamate levels as an energy source [95]. Increased energy reserves [97] and ATP levels [142] promoting neuronal stability. Increased transport capacity for ketones and lactate in cortical astrocytes [108]. Improved glucose sensitivity [125], supplementation of glucose reduced the anticonvulsant action [136].
	Epigenetic regulation	Upregulation of differentially regulated transcripts encoding energy metabolism enzymes [95] Upregulation of transcripts encoding mitochondrial proteins [95,145].and energy metabolism enzymes [97] Upregulation of intracellular signal transduction pathways [145]. Increased IGF system gene expression that regulates brain glucose utilisation [99]. Increased expression of GAD the rate limiting enzyme in GABA production [100]. Increased expression mHS mRNA, the key enzyme converting acetyl coenzyme A to ketones [105]. Decreased hippocampal mRNA levels for IL-1β modulating inflammation [106]. Increased MCT1 expression [108]. Downregulation of cathepsin E related to neuronal apoptosis induced by KA [122]. Ameliorated seizure-induced DNA methylation [127]. Abnormal expressions of Scn1a and Scn3a reduced by weakening GAPDH's binding to the element [132]. Decreased DNA hypermethylation [135]. Increased expression of $Ca2^{+}$ binding proteins in the interneurons of the hippocampus and astrocytes [146]. Decreased PENK gene expression in the hippocampus [149]. Transient upregulation of GFAP (S100B) expressed by astrocytes which plays a neurotrophic role on neighbouring cells [157], *(no SB100 change [177]).* Increased KCC2 protein expression pre-seizure and inhibited the upregulation of NKCC1 expression post seizure [172], increased basal protein phosphorylation [176]. Increased expression GluR6 mRNA [174]. *(No effect on brain expression of anticonvulsant peptides neuropeptide Y or galanin that are regulated by energy states [165]).*
	Mitochondria	Increased of mitochondria in neuronal processes [97]. Improved markers of mitochondrial biogenesis, dynamics and function [117] Decreased percentage of damaged mitochondria post seizure with increased expression of autophagy proteins and decreased apoptosis [173]. The mitochondrial level of UCP2 increased in the perikarya and axon terminals of hippocampus [117]. Improved mitochondrial redox status [120]. Decreased cytochrome c release from mitochondria, attenuated activation of casepase-9 and caspase-3 following seizures [134].
	Neurotransmitters	*(Glutamate transporters were not changed in hippocampus, cerebral cortex, or cerebellum [96]).* Increased GABA levels but not glutamate [98]. Increased dopamine activity in the motor and somatosensory cortex [101]. Altered gut biome resulting in systemic GABA and elevated hippocampal GABA/glutamate levels [153].
	Neuroinflammation	Suppression of COX-2 pathway and terminal enzyme mPGES-1.[123] Reduced cytokine TNF-α levels in the hippocampus [123]
	Neuroplasticity	Reduced supragranular mossy fiber sprouting [141,143].
	Signalling pathways	*(NRSF (targets genes such as BDNF) not shown to be essential in anti-epileptic effect [119]).* *(No change in cation chloride cotransporters (NKCC1 and KCC2) that regulate the polarity of GABAergic transmission in the hippocampus [112]).*

		Increased AMPK phosphorylation with reduced hippocampal cell apoptosis [121]. Reduced hippocampal TNF-α levels and nuclear factor (NF)-κB translocation into the nucleus [123]. PPARγ upregulation / activation (via fatty acids [161]) suppressing neuroinflammation via COX-2 pathways [123] and increased hippocampal catalase expression [126]. Increased adenosine [135] and purinergic pathways (such as A1R or K_{ATP} channels) enhancing glucose-based regulation of excitability [125] and seizure reduction [138]. Norepinephrine signalling partially involved in anti-seizure effects [137,164]. *(Seizure protection does not improve with higher levels of ketosis [131]).* mTOR activation reduced in the hippocampus [139]. Restored lipid membrane peroxidation and autophagy-associated pathway [143]. Increased nNOS with increased NO in hippocampus [148]. Suppresses KA-induced activation of JNK signalling pathways [149]. down-regulated expression of zinc and lipid transporters in hippocampus [168] and cortex [169].
	Structural integrity	Prevention of neuronal loss in ipsilateral hippocampus [124]. *(Altered hippocampal development with decreased neuronal density in young rat [124]).* Increased proliferation rate of neuronal progenitor cells after KA-induced seizures [130]. Prevention of hippocampal neuronal loss or change in density [133]. Reduction of nuclear clusterin accumulation [147] and preservation of pyramidal neurons [144] from caspase-3 mediated apoptosis. Decreased neuronal death in the ipsilateral hippocampus [134].
	Synaptic transmission	Synaptic transmission in hippocampal slices resistant to low glucose [95] and metabolic stress [97]. Reduced long-term potentiation consistent with decreased excitability [128] with concomitant maintenance of baseline excitability levels [178]
	Redox balance	No detected neurotoxic effects [92]. Increase in hippocampal mitochondrial glutathione [120].
	Biochemical	*(Increased calcium, decreased phosphorus, potassium & zinc areas of hippocampus [102,103]).*
Metabolic syndrome (2) [1 week to 8 Months]	Redox balance	*(No effect on brain antioxidant gene expression in short- or long-term diet [180]).* Improved brain oxidative stress responses [181].
	Mitochondria	*(No effect on brain mitochondrial function in short- or long-term diet [180]).*
	Epigenetic regulation	Downregulation of brain amyloid protein precursor, APOE and caspase-3 mRNA expression [181].
MCI (1)	Epigenetic regulation	Upregulated MCT1 mRNA after 10-90 days KD [182].
Multiple Sclerosis / demyelination (2) [1-12 weeks]	Structural integrity	Reversal of hippocampal atrophy and periventricular lesions [183]. Restored oligodendrocyte integrity and increased CNS myelination, ameliorated axonal degeneration and facilitated repair [184]
	Mitochondria	Ameliorates mitochondrial abnormalities in axons [184].
	Neuroinflammation	Suppression of inflammatory cytokines/chemokines and ROS [183].
	Neuroplasticity	Hippocampal synaptic plasticity (long-term potentiation)[183].
Nerve Toxin (1)	Neuroprotection	Attenuated toxicity from a neurotoxin after 4 weeks of KD [185].
Optic Nerve (4) [21 days to 8 weeks]	neuroinflammation	Reduced inflammation of optic nerve through inhibition of AMPK activation and stimulation of HCAR1 signalling which mediates inhibition of the NLRP3 inflammasome [188].
	Cellular energetics	Reversal of axonal metabolic decline by MCT transporter upregulation [187] Reduction in chronic glaucoma driven by low energy facilitated inflammation [188].
	Mitochondria	Increased mitochondria number and surface area in optic nerve axon [187].
	Signalling pathways	BDNF increased in the ganglion cell layer of the retina and optic nerve [187].
	Redox balance	Prevents increase in IL-1α and superoxide [186].
	Structural integrity	Preserves axons and visual evoked potentials [186] and reduced retinal ganglion cell loss [189].
Pain (2) [3-11 weeks]	Nociception	Decreased thermal pain sensitivity [16,62], that was not dependent on lowered glucose levels [62]
Parkinson's Disease (2) [1-2 weeks]	Neuroprotection	Protected dopaminergic neurons in the substantia nigra against 6-OHDA neurotoxicity [190] and degeneration [191].
	Neurotransmitters	Inhibited the decrease of striatal dopamine and metabolites [190]
	Neuroinflammation	Decreased glial activation and inhibition of proinflammatory cytokines [191]
	Redox balance	Inhibited glutathione decreases in the substantia nigra and striatum from 6-OHDA [190] .
Peripheral nerve dysfunction (3) [6 to 21 weeks]	Cellular energetics	Reduced (more efficient) oxidative respiration in sciatic nerve [192].
	Redox balance	Reduced H_2O_2 emission in sciatic nerve [192].
	Mitochondria	Reduced ROS mitochondrial production [192]
	Epigenetic regulation	Mitochondrial RNA expression for NADH dehydrogenase complex and complex IV altered potentially reducing ROS [192].

	Nociception	Protection from allodynia, reversal of allodynia induced by high fat + high carbohydrate diet [193].
	Structural integrity	Increased epidermal axon density and protection of nerve when on KD prior to injury [193]. Improved nerve regeneration when ketogenic diet commenced pre-injury [194] *(Unable to improve regeneration when KD provided after injury [194]).*
Spinal cord injury (4) [2 to 14 weeks]	Signalling pathways	Nrf2 activation supressing oxidative stress in KD started post injury [196]. NF-κB suppression resulting in reduced expression of proinflammatory cytokines TNF-α, IL-1β, and IFN-γ in KD started post injury [196]. HDAC inhibitor which protects against oxidative stress with KD started preinjury [198].
	Structural integrity	Reduced lesion size and sparing of grey matter in KD started post injury [197]. *(No enhancement of corticospinal tract plasticity [197]).*
	Cellular energetics	Upregulation of transporters GLUT1 and MCT1 in the blood vessels adjacent to the lesion [197].
	Redox balance	Reduced oxidative stress markers [196]. Downregulated NADPH, and oxidase (NOX2 and NOX4) with KD preinjury [195]. Upregulated FOXO3a, MnSOD and catalyse with KD preinjury [195].
Stroke (3) [3 weeks]	Structural integrity	Reduced infarct size [199-201], blood-brain barrier permeability and cellular apoptosis showing Improved ischemic tolerance [199].
	Neuroinflammation	Reduced NLRP3 inflammasome activation, capsase-1 and IL-1β [199].
	Signalling pathways	TXNIP expression which is required for NLRP3 activation [199] and HIF 1α upregulation via ketone utilisation causing the elevation of succinate [200]. HCAR2 activated on macrophages within the brain by BOHB exerting neuroprotection [201].
	Mitochondria	Decreased ROS production and endoplasmic reticulum stress [199].
Traumatic brain injury (9) [variable pre & post TBI]	Cellular energetics / metabolism	Injury induced brain energy deficits reduced in younger rats through shift in fuel source [202]. Reduction in cerebral metabolic rates for glucose while on KD after injury age-dependent with alternate substrate aiding recovery [207].
	Cortical excitability	Higher threshold to seizure [209].
	Redox balance	Increased protein expression of cytosolic and mitochondrial antioxidants [203] and improved neurochemical metabolite ratios [210].
	Mitochondria	Preserved mitochondrial Complex II-III activity [203] and reduced cytochrome c release reducing cellular apoptosis [204,205].
	Neuroinflammation	Reduced oedema [204,206].
	Epigenetics	mRNA changes in expression of genes involved in neuroplasticity, neuroinflammation, mitochondrial function [208].

4-HNE: 4-Hydroxy-2-nonenal, 6-OHDA: 6-hydroxydopamine, A1R: Adenosine 1 receptor, AcAc : Acetoacetate, Akt: protein kinase B, AMPK: 5' adenosine monophosphate-activated protein kinase, AMPA: alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid, APOE: apolipoprotein E, ATP: Adenosine triphosphate, BDNF: Brain derived neurotrophic factor, BOHB: beta hydroxybutyrate, cAMP: cyclic adenosine monophosphate, CNS: Central nervous system, COX: Cyclooxygenase, DNA: Deoxyribonucleic acid, eNOS: endothelial nitric oxide synthase, ERK1/2: extracellular signal-regulated kinase 1 and 2, fEPSP: Field excitatory postsynaptic potentials, FOXO: forkhead box transcription factors, GABA: g-aminobutyric acid, GAD: Glutamic acid decarboxylase, GAPDH: Glyceraldehyde 3-phosphate dehydrogenase, GFAP: glial fibrillary acid protein, GluR: Glutamate receptor, GLUT: Glucose transporter, GPR: G-coupled protein receptor, GPx: glutathione peroxidase, H_2O_2: Hydrogen peroxide, HCAR: Hydroxycarboxylic Acid Receptor, HDAC: Histone deacetylases, HIF: hypoxia-inducible factor, IL: Interleukin, IFN: Interferon, IGF: Insulin-like growth factor, JNK: c-jun amino-terminal kinase, KA: Kainic acid, K_{ATP}: ATP sensitive potassium channel, KCC: Potassium-chloride transporter, KD: Ketogenic diet, KYN: kynurenic acid, MCI: Mild cognitive impairment, MCT: monocarboxylic acid transporter, mHS: mitochondrial 3-hydroxy-3-methylglutaryl-CoA synthase, MnSOD: manganese superoxide dismutase, mPGES-1: prostaglandin E_2 synthase-1,mRNA: Messenger ribonucleic acid, mTOR: mechanistic target of rapamycin, NAD: nicotinamide adenine dinucleotide, NADH: nicotinamide adenine dinucleotide + hydrogen, NADPH: nicotinamide adenine dinucleotide phosphate, NKCC: Sodium-potassium-chloride transporter, NLRP3: NOD-, LRR- and pyrin domain-containing protein 3, nNOS: neuronal NO synthase, NO: Nitric oxide, NF-κB: Nuclear factor-κB, Nrf2: NF-E2 p45-related factor 2, NRSF: Neuron-Restrictive Silencer Factor, O-GlcNAc: O-linked-β-N-acetyl glucosamine, PARP: poly(ADP-ribose) polymerases, PPAR: Peroxisome proliferator-activated receptor, PENK: proenkephalin, PGC1β: peroxisome proliferator activated receptor gamma coactivator 1β, ROS: Reactive oxygen species, Scn: Sodium voltage-gated channel, SSADH -Succinic semialdehyde dehydrogenase, Sirt: Sirtuin, TXNIP: Thioredoxin Interacting Protein, UCP: Uncoupling protein ZnT: Zinc transporter,