Neuroscience and Biobehavioral Reviews 45 (2014) 46–62

Contents lists available at ScienceDirect

Neuroscience and Biobehavioral Reviews

journal homepage: www.elsevier.com/locate/neubiorev

Review

Oxidative & nitrosative stress in depression: Why so much stress?

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ARTICLE INFO

Article history:
Received 3 February 2014
Received in revised form 17 April 2014
Accepted 13 May 2014

Keywords:
Depression
Bipolar disorder
Oxidative & nitrosative stress
Inflammation
Antioxidants
Autoimmune

ABSTRACT

Many studies support a crucial role for oxidative & nitrosative stress (O&NS) in the pathophysiology of unipolar and bipolar depression. These disorders are characterized inter alia by lowered antioxidant defenses, including: lower levels of zinc, coenzyme Q10, vitamin E and glutathione; increased lipid peroxidation; damage to proteins, DNA and mitochondria; secondary autoimmune responses directed against redox modified nitrosylated proteins and oxidative specific epitopes. This review examines and details a model through which a complex series of environmental factors and biological pathways contribute to increased redox signaling and consequently increased O&NS in mood disorders. This multi-step process highlights the potential for future interventions that encompass a diverse range of environmental and molecular targets in the treatment of depression.

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Contents

Abbreviations: 5-HT, 5-hydroxytryptophan; 5-HTTLPR, serotonin transporter linked polymorphic region; 8-iso, 8-iso-prostaglandin F2; 8-OHdG, 8-hydroxy-2′-deoxyguanosine; ATP, adenosine triphosphate; BH_4, 5,6,7,8-tetrahydrobiopterin; CMI, cell mediated immune; CRH, corticotrophin releasing hormone; DAMP, damage-associated molecular pattern; DNA, eeoxyribonucleic acid; GPX, glutathione peroxidase; GSH, glutathione; HDL, high density lipoprotein; HPA, hypothalamic–pituitary–adrenal axis; IDO, indoleamine 2,3-dioxygenase; IFNα, interferon-alpha; IFNγ, interferon-gamma; Ig, immunoglobulin; IgG, immunoglobulin G; IgM, immunoglobulin M; IL-1, interleukin-1; IL-10, interleukin-10; IL-12, interleukin-12; IL-1β, interleukin-1β; IL-2, interleukin-2; IL-4, interleukin-4; IL-6, interleukin-6; iNOS, inducible nitric oxide synthase; KYNA, kynurenic acid; LDL, low-density lipoprotein; LPS, lipopolysaccharide; MAPK, mitogen-activated protein kinases; MDA, malondialdehyde; NAC, N-acetylcysteine; NDMA, N-methyl-D-aspartate; NF-κB, nuclear factor (NF)-κB; nNOS, neuronal nitric oxide synthase; NO, nitric oxide; NOS, nitric oxide synthase; NOX, NADPH oxidase complex.; Nrf2, nuclear factor erythroid 2-related factor; NSE, nitrosative specific epitopes; O&NS, oxidative & nitrosative stress; ONOO-, peroxynitrite; OSA, obstructive sleep apnoea; OSE, oxidation specific epitope; Ox-LDL, oxidized low density lipoprotein; Ox-PLP, oxidized phospholipids; PAMP, pathogen-associated molecular pattern; PIC, pro-inflammatory cytokine; PON1, paraxonase 1; PRR, pattern recognition receptor; QUIN, quinolinic acid; RNS, reactive nitrogen species; ROS, reactive oxygen species; SOD, Superoxide dismutase; SSRI, selective serotonin reuptake inhibitor; TCA, trycyclic antidepressant; TLR, Toll-like receptor; TNF-α, tumor necrosis factor-alpha; TRYCAT, tryptophan catabolite.

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http://dx.doi.org/10.1016/j.neubiorev.2014.05.007
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1. Introduction

Many studies support dysregulated redox signaling as being crucial in the pathophysiology and neuroprogressive nature of major depression (Maes et al., 2011a). Reactive oxygen and nitrogen species (ROS and RNS), including peroxynitrite, superoxides, peroxides and nitric oxide (NO), are produced during normal physiologic processes and, through interacting with proteins, fatty acids and DNA, perform numerous roles in regulation of cellular function. When present in excess however, ROS/RNS can lead to structural and functional changes that produce cellular injury. These potentially toxic effects are offset under normal conditions by intrinsic antioxidant mechanisms that participate in the physiologic and/or pathologic metabolism of ROS/RNS (Maes et al., 2011a). Increased oxidative and nitrosative stress (O&NS), which can arise as a consequence of raised production of ROS and RNS and/or decreased availability of antioxidant defenses, may cause damage to cellular components, induce harmful autoimmune responses, and ultimately facilitate failure of normal cellular processes.

People with unipolar and bipolar depression display dysregulated redox signaling (Lee et al., 2013; Maes et al., 2011a; Moylan et al., 2013c; Scapagnini et al., 2012). Studies using clinical and animal models have demonstrated that depression is associated with increased levels of redox products such as malondialdehyde (MDA, a marker for lipid peroxidation) and 8-iso-prostaglandin F2 (8-iso) (a marker of arachidonic acid peroxidation) (Dimopoulos et al., 2008; Forlenza and Miller, 2006; Galecki et al., 2009; Yager et al., 2010). Additionally, other studies have reported oxidative damage to DNA, as measured by increased levels of 8-hydroxy-2′-deoxyguanosine (8-OHdG) in serum (Forlenza and Miller, 2006) oxidative damage to RNA in post-mortem hippocampus in depression (Che et al., 2010) and telomere shortening (Shalev et al., 2014).

Studies conducted in depressed populations demonstrate sustained increases in O&NS. These effects result in depleted levels of n-3 fatty acid concentrations (Peet et al., 1998), a lowered oxidative potential index of serum (Maes et al., 1999), reduced functioning of antioxidant systems represented by lower levels of plasma concentrations of vitamin E (Maes et al., 2000; Owen et al., 2005) and C (Khanzode et al., 2003), decreased albumin levels (Van Hunsel et al., 1996), lowered levels of antioxidants including zinc, glutathione (GSH) and coenzyme Q10 (Maes et al.), and lower levels of amino acids, such as tryptophan and tyrosine (Maes et al., 2000). Similarly, alterations of antioxidant-enzyme levels have been reported. For example, levels of superoxide dismutase (SOD) and glutathione peroxidase (GpX) are lower in depressed patients (Maes et al., 2011a). Paraoxonase 1 (PON1), an antioxidant enzyme bound to high-density lipoprotein (HDL), was significantly reduced in unipolar, but not bipolar, depression (Bortolasci et al., 2014a). Impairment of these aforementioned antioxidant systems contributes to the pathophysiology of depression via lowered protection to ROS and RNS, which may result in increased risk of sustained O&NS damage (Forlenza and Miller, 2006; Maes et al., 2011a).

NO is an important mediator in many neural processes. Rodents subjected to acute and chronic immobilization stress exhibit increased levels of inducible nitric oxide synthase (iNOS). Although NO levels, iNOS and neuronal NOS (nNOS) expression are increased in depression, recent studies have indicated that NOS participates in the mechanisms underlying antidepressant efficacy (Galecki et al., 2012; Maes et al., 2008b). This suggests that NO may have differential effects at different sites during the course and treatment of depression. Persistently increased levels of NO and O_2^- may lead to the formation of peroxynitrite (ONOO—) and subsequent oxidation, nitration and nitrosylation of proteins, thereby contributing to cellular injury (Maes et al., 2008b, 2011d).

Major depression and bipolar depression are also accompanied by increased autoimmune responses against newly formed oxidation specific epitopes (OSEs), following structural damage by O&NS (Maes et al., 2007, 2011d, 2013b). Immunoglobulin (Ig)G and IgM-mediated immune responses against OSEs of membrane fatty acids, like oxidized low density lipoprotein (LDL), oleic acid, MDA and azelaic acid, and anchorage molecules, such as phosphatidyl inositol, palmitic acid, myristic acid and farnesyl-L-cysteine, can be seen in depression (Maes et al., 2007, 2011d, 2013b). This may have profound functional consequences as oxidative damage to membranes, especially to the major anchorage molecules, may affect the operation of hundreds of functionally “anchored” proteins that regulate basic cellular processes, including cell survival, growth, apoptosis, cell-signaling, neuroplasticity and neurotransmission (Maes et al., 2011a).

Chronically increased NO, following iNOS activation, can nitrosylate (NSO or NO) proteins and amino acids yielding new NO-adducts (NO-neoepitopes) like NO-tyrosine, NO-tryptophan, NO-arginine, NSO-cysteine and NO-albumin. The consequent hyper-nitrosylation may cause dysfunction to intracellular signaling, as well as competitively inhibit the palmitoylation of anchored proteins to the membrane (Maes et al., 2008b, 2011d, 2012a). Moreover, some of these NO adducts can be immunogenic and therefore contribute to further autoimmune responses

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