REVIEW ARTICLE

From Oxidative Stress to Genomic Instability: A Comparative Overview of Hemodialysis and Peritoneal Dialysis

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ABSTRACT

Chronic kidney disease (CKD) is linked to elevated oxidative stress (OS) due to impaired renal function, inflammation, and uremic toxin accumulation. In end-stage renal disease, renal replacement therapies—hemodialysis (HD) and peritoneal dialysis (PD)—further increase oxidative burden, contributing to DNA damage and genomic instability. HD triggers acute oxidative bursts through blood contact with bio-incompatible membranes and complement activation, whereas PD causes sustained oxidative stress via continuous exposure to glucose-based dialysate and its degradation products. Excess reactive oxygen species induce DNA base oxidation, strand breaks, chromosomal alterations, and telomere shortening. Biomarkers such as 8-hydroxy-2'-deoxyguanosine (8-OHdG), comet assay, and micronucleus test consistently show higher DNA damage in individuals undergoing dialysis. Impaired DNA repair mechanisms and polymorphisms in base excision repair pathways exacerbate genomic instability. Long-term consequences include increased risk of cardiovascular disease, cancer, and cellular senescence. Preventive strategies, including antioxidant supplementation, biocompatible or vitamin E-coated membranes, lifestyle modifications, and optimization of dialysis modality and dialysate quality, may mitigate genotoxic risk. Relevant literature was identified through structured searches of databases such as PubMed, Scopus, Web of Science, and Google Scholar. This review compares HD and PD in relation to oxidative DNA damage, highlighting mechanisms, biomarker evidence, and current preventive approaches

Keywords: Chronic kidney disease, hemodialysis, peritoneal dialysis, DNA damage, genomic instability.

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1. INTRODUCTION

Oxidative stress (OS) refers to tissue and systemic damage that occurs when the balance between oxidant molecules and the body's antioxidant defense mechanisms is disrupted. Among the numerous oxidative by-products, reactive oxygen species (ROS) and nitric oxide (NO) are the most prevalent, while antioxidants may be produced within the body or obtained from external sources. The primary targets of OS-induced damage include proteins, carbohydrates, lipids, and nucleic acids[1]. OS plays a lead role in the progression of chronic diseases such as diabetes mellitus, atherosclerosis, inflammation, and chronic kidney disease (CKD), and is also linked to renal anaemia, malnutrition, β2-microglobulin amyloidosis, and cardiovascular disease[2].

In CKD, this oxidative burden becomes more pronounced as declining renal function leads to the accumulation of uremic toxins, chronic inflammation, and impaired antioxidant defenses[3]. More than two million people worldwide are currently suffering from CKD[4]. As the disease advances to end-stage renal disease (ESRD), many patients require renal replacement therapy, most commonly hemodialysis (HD) or peritoneal dialysis (PD). Although these modalities are lifesaving, they can further intensify oxidative stress—HD through blood–membrane interactions and PD through continuous exposure to glucose- and GDP-rich dialysate[5]. Genomic instability, marked by heightened DNA strand breaks, chromosomal abnormalities, and compromised DNA repair mechanisms, has become a significant issue for patients receiving renal replacement therapy. Patients undergoing HD and PD

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exhibit elevated genomic instability levels relative to healthy individuals, attributed to ongoing oxidative stress, inflammation, and the accumulation of uremic toxins. The degree and pattern of genetic damage, however, may vary between the two modalities[6].

2. Oxidative stress and inflammation in dialysis patients

People suffering from chronic kidney disease (CKD) often exhibit oxidative stress and inflammation, which are significant factors in the advancement of CKD and the emergence of cardiovascular disease. Usually, oxidative stress triggers the activation of the transcription factor, nuclear factorerythroid 2–related factor 2 (Nrf2), which serves as a key regulator of genes that produce antioxidant and detoxifying enzymes, as well as protective proteins[7]. CKD impairs antioxidant defense mechanisms and enhances the generation of reactive oxygen species (ROS), a condition that is further intensified by the dialysis process[8]. During hemodialysis, exposure of blood to bio-incompatible membranes and endotoxins in the dialysate activates immune cells, leading to additional ROS production and inflammatory mediator release[3]. As a result, patients experience endothelial impairment, accelerated development of atherosclerosis, heightened protein and lipid oxidation, and a consequent rise in cardiovascular mortality[9].

2.1. Overview of oxidative stress in ESRD

Chronic kidney disease (CKD) involves a gradual deterioration of kidney function over time. As GFR decreases, CKD progresses. The deterioration in renal function disrupts overall body homeostasis, leading to both biological and clinical complications. These include impaired cellular energy metabolism, protein malnutrition, imbalance in nitrogen intake and excretion, insulin resistance, and a significant rise in the production of inflammatory and oxidative stress mediators[10]. Ultimately, it advances to end-stage renal disease (ESRD), resulting in the need for renal replacement therapy (such as hemodialysis or peritoneal dialysis) or a kidney transplant[11]. Patients with ESRD experience increased oxidative stress due to weakened antioxidant defense such as deficiencies in vitamin C and selenium, lower levels of intracellular vitamin E, and reduced glutathione activity and increased production of pro-oxidant factors, including older age, high prevalence of diabetes, chronic inflammation, uremic toxins, and the use of dialysis materials that may not be fully biocompatible.

Oxidative stress and inflammation are closely related because inflammatory stimuli activate phagocytic cells, which generate free radicals. Both processes contribute to endothelial dysfunction, as the endothelium both produces and is damaged by oxidants and is actively involved in inflammation. Growing experimental and clinical evidence suggests that oxidative stress plays a role in the development of atherosclerosis and other complications in ESRD patients, including dialysis-related amyloidosis, malnutrition, and anemia. Since free radicals exist only for a few seconds, oxidative stress is assessed indirectly by measuring stable oxidized molecules (e.g., lipid peroxidation products, advanced glycation and oxidation products of lipids and proteins, or nucleic acid oxidation markers) or by detecting antibodies against these oxidized molecules (such as anti-oxidized LDL antibodies)[12]. Usually the generation of reactive species and their localization within cells is balanced by the presence of antioxidant enzymes such as cytosolic catalase (CAT), superoxide dismutase (SOD), and glutathione peroxidase (Gpx). Both elements need to be at suitable levels for cells to function normally. Oxidative stress occurs when reactive oxygen species (ROS) are produced at a rate that outstrips the ability of antioxidant systems to neutralize them[11,13].

Increased oxidative stress has been observed even during the initial stages of CKD. Multiple studies have shown that patients with severe kidney dysfunction experience a significant rise in oxidative stress, and this condition becomes even worse with hemodialysis[14]. Some researchers, based on in vivo evidence, argue that the increased oxidative damage is mainly due to a decline in antioxidant enzyme levels rather than an increase in ROS production[15]. Uremic toxins are known to be crucial in triggering oxidative stress. Studies have shown that uremic toxins contribute to inflammation and oxidative stress by activating acute inflammatory polymorphonuclear leukocytes and inducing the release of interleukin (IL)-1β and IL-8[16].

Furthermore, hemodialysis itself promotes oxidative stress. This occurs because the procedure removes antioxidant components—particularly those of low or very low molecular weight—and triggers leukocyte activation

through interaction with the dialysis membrane and dialysate. These mechanisms intensify inflammation and result in elevated ROS production[17]. The rise in oxidative stress observed in HD patients depends on various factors, including aging, deterioration of residual renal function and the resulting uremic state, as well as the HD procedure itself[18]. The increased oxidative stress observed in hemodialysis (HD) patients is attributed to the depletion of antioxidants during the treatment and the accumulation of oxidized metabolites. Additionally, oxidative stress was found to be greater in ESRD patients receiving PD compared to those with uremia who are not on dialysis, although it remains lower than in patients undergoing HD[19]. This observation is further supported by findings showing that resting levels of superoxide anion in whole blood are elevated after each HD session[20]. Several studies have confirmed the presence of oxidative stress and weakened antioxidant defense mechanisms in individuals with CKD or ESRD. Kinugasa E observed elevated levels of oxidative stress markers, such as advanced glycation end products (AGEs), malondialdehyde (MDA), and 8-hydroxydeoxyguanosine (8-OHdG), in the blood and/or tissues of CKD patients[21-22]. Additionally, Colombo et al. established a strong connection between uremia and oxidative stress in ESRD patients receiving maintenance hemodialysis(HD). as evidenced by marked oxidative damage to proteins, particularly elevated levels of plasma advanced oxidation protein products[23].

2.2. Role of dialysis modality in ROS generation and inflammatory cytokine release

The choice of dialysis modality depends on factors such as healthcare policy, physician judgment, patient suitability, availability of resources, reimbursement systems, patient preference, quality of life, and awareness of treatment options[24]. Age is also influential, with data from 2013 showing an average starting age of 61 years for PD and 65 years for HD. Dialysis modalities are vital in the production of ROS and the release of inflammatory cytokines, with HD typically linked to a more significant rise in oxidative stress and inflammation than PD, though contemporary methods are lowering these impacts[25].

Hemodialysis (HD) involves circulating a patient's blood through an external dialysis system that removes metabolic waste products and excess fluid before returning the blood to the body. Conventionally, HD is prescribed three times per week, with each session lasting approximately three to four hours. However, this standard schedule can be modified to increase the frequency or duration of treatments in order to provide more physiological solute clearance and better control of extracellular fluid volume, while also minimizing interference with patients' daily activities[25]. Intensified HD regimens include short-daily HD, typically delivered six to seven times per week for around two to three hours per session, and frequent nocturnal HD, which is often administered five to seven nights per week for six to nine hours, usually during sleep. In practice, nocturnal HD performed at home may be provided three to four times per week to align with patient convenience and resource availability[26].

HD is further classified by the setting in which treatment is provided. In-CenterHemodialysis (ICHD) is conducted in a hospital or dialysis facility under the supervision of trained healthcare personnel. A variation of this is self-care ICHD, where patients independently perform treatment-related tasks within the clinical setting, requiring minimal staff intervention. In contrast, Home Hemodialysis (HHD) is carried out in the patient's home environment, either independently (by the patient or caregiver) or aided by a healthcare professional. HHD is increasingly associated with frequent dialysis schedules such as short-daily or nocturnal HD, as the home setting offers greater flexibility to tailor the dialysis prescription to individual patient needs[27].

In peritoneal dialysis (PD), a permanent catheter is inserted into the abdomen, allowing dialysate to be introduced into the peritoneal cavity. The dialysis fluid, known as the dwell, remains inside the cavity for several hours. During this time, waste products and excess fluid gradually pass from the blood across the peritoneal membrane into the dialysate. After the dwell period, an exchange occurs, during which the used solution (effluent) is drained and replaced with fresh dialysate[28-29].

PD produces less systemic oxidative stress but can cause local ROS generation in the peritoneal cavity due to repeated exposure to glucose-rich, acidic dialysate, leading to glucose degradation product formation and peritoneal inflammation. Online hemodiafiltration (HDF) appears to cause lower oxidative stress than conventional HD, likely because it removes middle molecules more effectively and reduces complement activation[29]. Persistent ROS

contributes to endothelial damage, protein and DNA oxidation, atherosclerosis, and higher cardiovascular risk in dialysis patients. Inflammatory cytokines are proteins released by immune cells like monocytes, macrophages, and T cells in response to stress, injury, or infection. They play a key role in triggering and sustaining inflammation by activating and attracting other immune cells. Important pro-inflammatory cytokines include IL-6, TNF-α, IL-1β, and IL-8[28,30]. These molecules amplify inflammation through pathways such as NF-κB and JAK/STAT. In chronic kidney disease and dialysis, cytokine release is stimulated by factors such as uremic toxins, oxidative stress, complement activation, and immune responses to the dialysis procedure. During hemodialysis, blood contact with dialysis membranes activates complement proteins (C3a, C5a), which increase cytokine release from immune cells[30]. Peritoneal dialysis can also trigger local cytokine production due to the effect of glucose-rich, acidic dialysate on the peritoneal membrane[31]. High levels of pro-inflammatory cytokines over time contribute to endothelial damage, atherosclerosis, malnutrition-inflammation-atherosclerosis (MIA) syndrome, oxidative stress, protein and DNA damage, anemia, and faster progression of kidney disease. Anti-inflammatory cytokines like IL-10 help control this response, but in dialysis patients, pro-inflammatory cytokines often dominate[30-31]. Using biocompatible membranes, ultrapure dialysate, and tailored dialysis schedules can help reduce cytokine-related inflammation and improve patient outcomes[31].

2. Mechanism linking oxidative stress to DNA damage

Oxidative stress occurs when ROS production exceeds the body's antioxidant defense, leading to damage of cellular macromolecules, particularly DNA. DNA damage may arise from multiple sources, including external factors like UV radiation and ionizing radiation, and internal causes such as replication errors and oxidative stress [32].

Pathway of ROS-induced genotoxicity

ROS comprise of a family of short-lived molecules like O_2^-, H_2O_2 and OH, first described in skeletal muscles as free radicals. Reactive oxygen species (ROS) contain at least one oxygen atom per molecule but exhibit greater reactivity than molecular oxygen[33]. The ROS-induced genotoxicity pathway begins with the generation of reactive oxygen species, which oxidize DNA bases and produce lesions like 8-oxo-guanine, potentially causing mutations if left unrepaired. In addition, ROS activate signalling pathways, including MAPK and NF-κB, which, together with DNA damage, stimulate the p53 tumor suppressor. Activated p53 can pause the cell cycle to allow DNA repair or trigger apoptosis to remove severely damaged cells; however, persistent or excessive ROS can surpass these protective responses, leading to genomic instability [34].

Mitogen-Activated Protein Kinases (MAPKs) are a family of serine/threonine kinases that transmit signals from the cell surface to the nucleus, regulating key processes such as cell growth, stress response, and apoptosis[35]. MAPK is activated through mitogenicsignalling cascades involving various cell-associated kinases such as ERK1/2, c-Jun N-terminal kinases (JNK), p38 kinase (p38), and the prominent MAP kinase 1 pathway (BMK1/ERK5). Members of the MAPK family share structural and functional similarities. Consequently, numerous studies have focused on identifying the molecules that specifically regulate different MAPK signaling pathways[36]. The MAPK signaling cascade consists of three sequential kinase types: mitogen-activated protein kinase kinase kinases (MAPKKKs), mitogen-activated protein kinase kinases (MAPKKs), and mitogen-activated protein kinases (MAPKs) which regulates different cellular functionalities[37]. Conversely, in the JNK pathway, Apoptosis Signal-Regulating Kinase 1 (ASK1), an upstream protein, also contributes to the activation of the p38 pathway. Oxidative stress has been shown to affect ASK1, mitogen-activated protein kinase kinases 1–4 (MEKK1–4), and mixed-lineage kinase 3 (MLK3), either directly or indirectly, thereby sequentially activating the p38 signaling pathway[38,39]. MAPK activation can also increase ROS production, forming a feedback loop that amplifies cellular stress signals. The balance between MAPK activation and MKP-mediated deactivation is essential to determine whether a cell survives, repairs damage, or undergoes apoptosis [40].

Often referred to as the "guardian of the genome," p53 is crucial for preserving genomic stability and coordinating cellular reactions to stress[41]. Chronic or excessive ROS, often arising from persistent stress or inflammation, can induce severe DNA damage, prompting p53 to halt the cell cycle or initiate apoptosis to prevent propagation of

damaged cells. ROS also affect the balance between p53 and its negative regulator, MDM2, by oxidizing MDM2 cysteine residues, which prevents p53 degradation and increases its activity[42]. At basal levels, p53 maintains ROS at non-toxic concentrations through antioxidant gene induction, whereas elevated p53 can act as a pro-oxidant by enhancing ROS production or repressing antioxidant genes. The redox-sensitive cysteine residues in p53 allow ROS to finely tune its function, determining whether a cell undergoes repair, senescence, or apoptosis[43].

NF-κB signaling and reactive oxygen species (ROS) are closely interconnected in the regulation of cellular functions and gene expression[44]. The NF-κB family of transcription factors regulates genes involved in cell survival, proliferation, and inflammation, and relies on a Rel-homology domain (RHD) for DNA binding and dimerization. The NF-κB family consists of the subunits p52/p100 (NF-κB2), RelA (p65), RelB, p50/p105 (NF-κB1), and c-Rel. NF-κB activation primarily occurs via two mechanisms: the canonical and non-canonical pathways[45]. The canonical pathway is stimulated by factors such as viral infection, cellular stress, and pro-inflammatory cytokines. These signals induce phosphorylation-dependent processes that result in the proteasomal degradation of IκBα, thereby releasing NF-κB. This enables its translocation into the nucleus, where it activates the transcription of downstream target genes [46,47].

3. Comparative analysis of DNA damage in hemodialysis and peritoneal dialysis

The buildup of uraemic toxins, oxidative stress mediators, and other endogenous chemicals with genotoxic qualities may be the cause of the genomic instability and consequent widespread genetic damage seen in patients with CKD[48]. Patients on dialysis have higher levels of DNA damage than healthy individuals, largely due to increased oxidative stress and inflammation[49]. This is reflected in elevated DNA strand breaks, oxidative base modifications, and chromosomal aberrations observed in both HD and PD patients. In HD, this damage is driven by repeated blood–membrane interactions that trigger oxidative bursts, whereas in PD, continuous exposure to glucose-based dialysate and its degradation products results in a sustained oxidative insult. Some data suggest that HD may induce more acute, episodic damage, while PD may contribute to persistent low-grade damage; however, direct comparative findings remain inconsistent[50]. Common forms of DNA damage observed in dialysis patients include single- and double-strand breaks, base modifications (8-OHdG), and chromosomal aberrations along with mitochondrial DNA damage —[51].

Persistent and inadequately repaired DNA damage in ESRD patients promotes genomic instability, which in turn is strongly in HD, the interaction between blood and synthetic dialysis membranes activates immune cells and complement pathways, leading to sudden increases in ROS that can damage DNA[52]. This is often demonstrated by higher concentrations of 8-OHdG, a marker of oxidative DNA injury, particularly when bio-incompatible membranes are used and is linked with long-term complications such as cardiovascular disease and cancer[53]. Moreover, comet-assay studies show that HD patients have significantly increased percentages of DNA strand breaks and oxidative lesions compared to healthy controls.

Evidence from biomarkers

Biomarker assessment plays a pivotal role in understanding the extent of oxidative stress, inflammation, and genomic instability associated with dialysis therapy in end-stage renal disease (ESRD) patients. 8-OHdG, the comet assay, and the micronucleus test, are the key indicators of DNA damage in uremic conditions. In, ESRD, the accumulation of reactive oxygen species leads to increased genomic instability, making these biomarkers highly relevant[54].

3.1.1. 8OHdG

DNA damage is usually repaired, and the oxidized products are excreted in urine. 8-hydroxy-2-deoxyguanosine is considered a biomarker for oxidative damage of DNA.8-OHdG is regarded as a biomarker due to its specificity as a product of oxidative DNA damage induced by ROS[55]. Guanine is the DNA base most susceptible to oxidative attack. The resultant 8-OHdG is excreted and can be found in urine or serum, serving as an indicator of the body's total oxidative stress. There is a correlation between heightened 8-OHdG levels and the increased risk of various diseases, such as cancer and cardiovascular disease[56]. To effectively utilize 8-OHdG as a marker of excessive oxidative stress, it is important to establish baseline reference values for normal levels systematic review and meta-analysis were conducted to establish reference ranges for urinary 8-OHdG in healthy individuals. From 1,246 studies, 84 were

included and categorized by BMI, gender, and smoking status. In adults with BMI ≤ 25, the pooled geometric mean measured by chemical methods was 3.9 ng/mg creatinine (IQR: 3–5.5). Smoking showed a significant increase in 8-OHdG levels, while no gender effect was observed[57]. 8-OHdG is among the most extensively investigated oxidized DNA metabolites and is widely recognized as an indicator of oxidative DNA damage. Its generation through the action of oxygen-derived free radicals was initially documented in 1984 by Kasai and Nishimura[58]. Elevated levels of 8-OHdG have been linked to several disorders, including cardiovascular disease and chronic obstructive pulmonary disease (COPD)[59,60].

A study assessing oxidative DNA damage in patients with chronic renal failure (CRF)/chronic kidney disease (CKD) measured 8-hydroxy-2'-deoxyguanosine (8-OHdG), a widely recognised biomarker of oxidative DNA injury due to the high vulnerability of guanine bases to oxidative attack[61]. The findings showed significantly elevated 8-OHdG levels in CKD patients compared to healthy controls, indicating increased DNA oxidation. The authors suggested that excess reactive oxygen species contribute to heightened DNA damage in individuals with CKD[62,63].

3.1.2. Comet assay

The alkaline comet assay is a specialized technique used to assess DNA damage by analysing single nucleoids through electrophoresis[64]. During the process, damaged DNA migrates, producing a characteristic "comet-like" appearance, where the intensity and length of the tail reflect the extent of strand breaks'[65]. This standard assay effectively detects DNA strand breaks and alkali-labile sites. Its diagnostic capability is further enhanced when combined with lesion-specific endonucleases such as Formamidopyrimidine DNA glycosylase (FPG), 8-oxoguanine DNA glycosylase 1 (OGG1), and endonuclease III, which allow for the identification of additional types of lesions, including oxidized purines, oxidized pyrimidines, and pyrimidine dimers. Thus, by incorporating these enzymes, the comet assay becomes a versatile tool for detecting a broader spectrum of DNA damage[66]. Most case-control studies have evaluated DNA damage in white blood cells, revealing that individuals with coronary artery disease, diabetes, kidney disease, chronic obstructive pulmonary disease (COPD), and Alzheimer's disease exhibit approximately twice the level of DNA strand breaks compared to healthy controls[67].

Additionally, patients with coronary artery disease, diabetes, kidney disease, and COPD show 2- to 3-fold higher levels of oxidative DNA damage in white blood cells, though variations between diseases are not clearly defined. Elevated DNA strand breaks have also been reported in breast cancer patients, while evidence for colorectal and lung cancer is limited[67,68]. A cross-sectional study comparing CKD patients in pre-dialysis, PD, and HD stages with healthy controls using comet and CBMN assays reported significantly higher DNA and chromosomal damage in pre-dialysis and PD groups, while the HD group did not exhibit a notable increase. The authors suggested that factors such as male sex, diabetes, and treatment modality contribute to genomic instability, likely driven by oxidative stress and comorbid conditions[6].

3.1.3. Micronucleus test

The Cytokinesis-Block Micronucleus (CBMN) assay also serves as a useful biomarker for assessing DNA damage in dialysis patients, as it detects chromosomal alterations such as breaks and losses, which are commonly increased in this population[69]. It is a highly sensitive and comprehensive tool for evaluating genetic instability caused by factors like oxidative stress and uremic toxicity—both prevalent in patients undergoing dialysis[70]. Therefore, the CBMN assay helps in determining the extent of genomic instability linked to the disease itself as well as its treatment[71].

A case–control study using the micronucleus assay on exfoliated buccal cells compared patients undergoing HD and PD with healthy controls. HD patients exhibited a significantly higher frequency of micro nucleated cells, whereas PD patients did not show a comparable increase ""__'[72,73]. Importantly, the rise in genotoxicity was observed only in HD patients receiving treatment for seven years or more, indicating that long-term HD may contribute to genomic instability and that PD could present a relatively safer alternative[74].

Conversely, another study using the cytokinesis-block micronucleus assay in peripheral lymphocytes of dialysis patients reported significantly higher genomic damage in both HD and PD groups compared to healthy controls[75]. No significant difference was observed between the two

dialysis modalities. Among the variables analyzed, elevated low-density lipoprotein (LDL) levels were identified as the only independent predictor of genomic damage[76-79].

3.2. Influence of membrane type, dialysate composition and duration of therapy

Dialysis membranes were initially categorized based on their material (cellulosic vs synthetic)[80] and the sieving coefficient, which reflects molecular weight retention characteristics. Hemodialysis membranes are generally classified into three main types based on their composition. Early dialysis membranes were made from flat cellulose sheets, such as Cuprophan, which was derived from cotton and efficiently removed small solutes with molecular weights below 500 Da[81]. Later, hollow-fiber membranes made from modified cellulose were introduced, and today, dialysis membranes are predominantly produced from synthetic polymers[82,83]. Synthetic membranes gained preference due to superior physical, chemical, and structural properties. Recent innovations include medium cut off, graphene oxide, mixed-matrix membranes, bio artificial kidneys, and membranes enhanced with agents like vitamin E, lipoic acid, and neutrophil elastase inhibitors. Dialysis membranes were historically classified based on their material composition as either cellulosic or synthetic. As synthetic membranes have become widely used and cellulosic ones are now rare, a new classification system based on the ultrafiltration coefficient (Kuf) has been introduced[80]. Kuf, expressed in mL/hour/mmHg, reflects the membrane's water permeability and is calculated by dividing the ultrafiltration rate by the transmembrane pressure[83].

3.2.1. Membrane types

3.2.1.1. Membrane type for HD

A. Cellulose based membranes

The first cellulose-based dialysis membrane, Cuprophan®, was made from cotton in Wuppertal, Germany, and efficiently removed small solutes (<500 Da)[84]. However, it was less effective than synthetic membranes for hemodialysis due to low biocompatibility. This was mainly caused by free hydroxyl groups on the cellulose surface, which could activate complement by binding to C3b. To improve compatibility, these hydroxyl groups were chemically modified, mainly through acetylation, producing cellulose acetate, cellulose diacetate (CDA), and cellulose triacetate (CTA) membranes. The number of acetate groups determines the membrane type, and these modifications reduced free hydroxyl groups and complement activation[85]. Among these, CTA is currently the most biocompatible cellulosic membrane[86].

Another modification of cellulose-based dialysis membranes involves attaching aromatic benzyl groups and incorporating ether bonds into the cellulose chains to create hydrophobic regions[87]. Additionally, Hemophan® represents a further advancement, where functional tertiary amines are introduced during membrane production. In this process, hydroxyl groups on the surface are replaced with large diethylaminoethyl groups, which sterically prevent interaction between the membrane and blood cells, resulting in improved biocompatibility[88].

B. Synthetic polymer membranes

Synthetic polymer membranes are made from materials such as polysulfone (PSU), polyethersulfone(PES), polymethylmethacrylate, polyester polymer alloy, polyacrylonitrile (PAN), polycarbonate(PC), polyamide, and polyethylene-co-vinyl alcohol. Compared to cellulosic membranes, they offer larger pore sizes, better hydraulic permeability, higher filtration capacity, and improved solute clearance[89]. The outer layer provides mechanical support for the fiber and usually has a sponge-like or finger-like structure. In contrast, the inner layer forms a dense, selective barrier that comes into direct contact with blood and controls the passage of molecules across the membrane[90]. Structurally, cellulose membranes are symmetrical with uniform pore size, while synthetic membranes are asymmetrical. The primary drawback of synthetic membranes compared to cellulose-based ones is their high hydrophobicity, which leads to greater protein adsorption, membrane fouling, and decreased biocompatibility[91].

3.2.1.2. Membrane type for PD

The membrane used in peritoneal dialysis (PD) is the peritoneal membrane, also known as the peritoneum, which lines the abdomen and its organs, covering a surface area approximately equal to the body surface area[92]. A healthy and functional peritoneal membrane is key to achieving sufficient ultrafiltration (UF) and restoring fluid balance, both vital components of high-quality PD prescription[93]. Dysfunction of this membrane contributes significantly to

fluid overload and associated complications[94]. Structurally, the peritoneum is composed of the visceral peritoneum which covers the abdominal organs and the parietal peritoneum which lines the abdominal wall and diaphragm[95]. It includes a single layer of mesothelial cells, an interstitium, and a dense microvascular network. Crucially, the endothelium of peritoneal capillaries serves as the main barrier regulating the movement of solutes and fluids across the membrane[96-99].

The membrane facilitates the fundamental dialysis processes of diffusion, osmosis, and convection[100]. Peritoneal transport is often described using a three-pore model. Small pores (radius 4–5 nm) facilitate the diffusion of small solutes such as urea and creatinine, whereas ultra-small pores (radius <0.3 nm) are essential for free-water transport[101,102]. This water movement is mediated by Aquaporin water channels (AQP1), which are expressed in endothelial cells lining the micro vessels and facilitate water flow according to an osmotic gradient[103]. Osmotic agents like glucose (used in concentrations such as 1.36%, 2.27%, or 3.86% anhydrous glucose) in the dialysis solution drive UF across the membrane[102]. Membrane function and solute transfer rates (PSTR) are often characterized using the Peritoneal Equilibration Test (PET)[104].

3.2.2. Dialysate composition

Sodium (Na): Sodium (Na) is transported across the dialysis membrane by both diffusion and convection, and the kinetics of these mechanisms are vital for defining the appropriate dialysate sodium concentration [Na]. The major goal of intermittent hemodialysis (HD) is to normalize total body water by achieving a zero balance, which requires removing the Na and water accumulated during the interdialytic period. Because the interdialytic Na load and the Na/water ratio vary significantly among and within patients, the amount of Na removal ideally requires individualization by adjusting the dialysate [Na] for each session; however, this approach is unsuitable for routine clinical application. Selecting the appropriate dialysate sodium concentration is vital for maintaining hemodynamic stability[104]. A high dialysate sodium level supports osmotic refilling, counteracting intravascular volume loss due to ultrafiltration. This can help prevent cardiovascular instability but may reduce net sodium removal. As a result, patients may experience excessive thirst, fluid overload, refractory or intradialytic hypertension, and pulmonary edema[105].

In contrast, low dialysate sodium enhances sodium loss by diffusion and lowers plasma osmolarity. This leads to fluid movement into cells, causing cellular over hydration and potentially contributing to disequilibrium syndrome, presenting with fatigue, cramps, and headache. Insufficient intravascular refilling can also result in hypotension and cardiovascular instability. Additionally, low sodium levels may paradoxically trigger hypertensive crises through renin–angiotensin system activation[104,105].

Potassium (K): Hemodialysis eliminates potassium from the extracellular fluid, which accounts for only about 2% of total body potassium, while the majority is located within cells[106]. The high dialysate potassium concentrations to restore body stores is generally discouraged. For patients with pre-dialysis serum potassium levels between 4 and 5 mEq/L, a dialysate potassium concentration of 3 mEq/L is typically recommended and widely adopted as the standard of care. In cases where pre-dialysis serum potassium consistently exceeds 5 mEq/L, dietary modification and review of medications should be considered first. If these measures are insufficient, reducing the dialysate potassium concentration to 2 mEq/L may be appropriate. Very low dialysate potassium levels should only be used in exceptional circumstances, as large serum-to-dialysate potassium gradients can increase the risk of mortality. Individualizing dialysate potassium and other electrolyte prescriptions requires more frequent monitoring, as monthly blood tests may not accurately reflect potassium fluctuations during each dialysis session. Implementing point-of-care serum electrolyte measurements could help address this limitation[107].

Calcium (Ca): The optimal approach to maintaining calcium balance in hemodialysis patients remains controversial. Calcium mass balance studies indicate that in patients with normal pre-dialysis serum calcium, a dialysate calcium concentration of 1.25 mmol/L (Low Concentration) produces a negative calcium balance, whereas 1.75 mmol/L (High Concentration) results in a positive balance[108]. A positive calcium balance—where calcium intake exceeds removal—can help control hyperparathyroidism but may also increase the risk of soft tissue calcification and cardiovascular mortality. Therefore,

current KDOQI hemodialysis guidelines recommend lowering dialysate calcium concentration to promote a neutral or negative calcium balance and reduce vascular calcification. They also advise keeping corrected serum calcium levels within the low to normal range[109].

Excessive reduction of calcium in dialysate or serum during hemodialysis may increase the risk of cardiac arrhythmias and sudden cardiac arrest (SCA), which accounts for about one in four deaths in this population[110]. Low calcium dialysate has been associated with hypotension and QT interval prolongation, indicating higher arrhythmic risk[111,112]. Patients receiving dialysate calcium levels below 2.5 mEq/L were previously shown to have a greater risk of SCA[113].

Magnesium (Mg): Magnesium removal during dialysis largely depends on pre-dialysis plasma magnesium levels, as the diffusion gradient between plasma and dialysate ($Mg^{2+}D$) drives magnesium movement. Both high (>0.75 mmol/L) and low (<0.25 mmol/L) dialysate magnesium levels can have benefits and risks. Higher concentrations may help reduce parathyroid hormone (PTH) release and slow arterial calcification but may also cause altered nerve conduction, pruritus, and increase the risk of osteomalacic renal osteodystrophy. Lower dialysate magnesium levels may support bone mineralization and prevent magnesium overload in patients taking oral magnesium (e.g., as a phosphate binder), but may also lead to muscle cramps and elevated PTH levels. Commercially available dialysate solutions typically contain 0.25–0.75 mmol/L of magnesium, though magnesium-free and 1.0 mmol/L formulations also exist[114].

3.2.3. Duration of therapy

In hemodialysis, blood is withdrawn from the body through a system of tubes, passed through a dialyzer (filter) to remove impurities, and then returned to the patient. As blood moves through the dialyzer, it comes into contact with dialysate—a fluid similar to body fluid but without waste products—to facilitate toxin removal. This procedure is commonly conducted in dialysis centres within medical or commercial facilities. It also requires establishing and maintaining a vascular access—such as a dialysis catheter or an arteriovenous (AV) fistula or graft in the arm or groin to provide the high blood flow necessary for treatment[115].

Peritoneal Dialysis (PD) is considered one of the simpler dialysis modalities, offering minimal disruption to daily life and greater flexibility. It involves placing a synthetic catheter into the abdominal cavity, allowing dialysis to occur through regular exchanges of dialysate fluid. PD can be customized to suit individual needs—patients may perform it overnight using a cycler machine while asleep, or during the day through approximately four manual exchanges, each taking around 15–30 minutes. Due to its convenience and adaptability, PD is often preferred by individuals with busy schedules, active family roles, or significant time limitations[115].

4. DNA repair and genomic instability in Dialysis-Related Oxidative Stress

4.1. Alterations in DNA repair pathways

Damage repair (DDR) pathways are crucial for preserving accurate genetic information. However, genomic stability is continuously threatened by both internal and external factors. Alterations in DDR mechanisms primarily occur through genetic inactivation or epigenetic regulation[116]. Individuals receiving dialysis are exposed to persistent oxidative stress, systemic inflammation retained uremic toxins and dialysis-related bioincompatablity, all of which collectively increases genetic instability by promoting excessive ROS forming the cellular DNA damage[117,118].

4.1.1. BER - Base Excision Repair

Genetic polymorphism, occurring within the DNA repair genes have the potential to compromise the capacity for DNA repair consequently result in the accumulation of DNA damage.For humans, Base Excision Repair (BER) pathway incorporates the gene *hoGG1, MTH1,MUTYH* acts as the primary defence mechanism against DNA damage. Furthermore, a specific C>G polymorphism at position 1245 in exon 7 of the *hoGG1*gene is linked to the replacement of serine with cysteine at codon 326[119,120]. A population that has reduced hOGG1 enzyme activity is more likely to build up 8-OHdG in their nuclear DNA, since oxidatively damaged DNA cannot be fully repaired. In a study involving patients on long-term haemodialysis, those with the 1245 GG genotype showed even higher leukocyte 8-OHdG levels than individuals carrying the 1245 CG or CC genotypes. In a study conducted on a Chinese population, BER pathway polymorphisms specifically, MUTYH c.972GG (rs3219489) and AluYb8MUTYH (rs10527342) were found to elevate the risk of developing

ESRD, particularly when combined with the OGG1 c.977GG variant. This suggests that individuals who are homozygous or heterozygous for these BER polymorphisms may have a higher genetic susceptibility to ESRD[121].

4.1.2. NER- Nucleotide excision repair

Nucleotide excision repair (NER) is a key mechanism used by all cells to remove bulky DNA adducts caused by environmental toxins. The nucleotide excision repair (NER) pathway is the most versatile DNA repair system, capable of correcting a broad variety of helix-distorting DNA lesions. NER targets DNA damage such as UV-induced lesions like cyclobutane pyrimidine dimers (CPDs) and pyrimidine-pyrimidine (6-4) photoproducts as well as intrastrand crosslinks and bulky base adducts formed by reactive metabolites of certain chemical carcinogens or chemotherapy drugs. These types of DNA lesions are handled by two overlapping NER branches; global genome NER (GG-NER) and transcription-coupled NER (TC-NER) which differs mainly in how the damage is detected. In GG-NER, specialized sensor proteins continuously scan the entire genome throughout the cell cycle to locate damage[122,123]. Unlike GG-NER, TC-NER specifically and quickly removes lesions that block transcription, but only from actively transcribed DNA strands. In this pathway, damage is recognized when it causes RNA polymerase to stall on the template strand[124,125]. In summary, NER involves three main stages: identifying the damage, which includes both initial detection and confirmation; making dual cuts around the lesion and removing the damaged fragment; and filling in the resulting gap through repair synthesis followed by DNA ligation.

4.2. Long-term consequences:

4.2.1. Genomic instability & cancer risk

Failures in DNA repair pathways that contribute to genome instability can severely affect an organism's health and longevity, ultimately giving rise to various diseases such as cancer and neurodegenerative disorders. Genomic instability (GI), characterized by abnormal chromosome structure or number, along with changes to DNA such as nucleotide deletions, insertions, or substitutions, is a fundamental feature of most solid tumors and adult-onset leukaemia. Chromosome instability (CIN) is considered a major facilitator of tumorigenesis, supported by various mouse models that are either tumor-prone or display accelerated tumor development[126]. These resulting DNA changes, particularly those affecting oncogenes or causing the loss of tumor suppressor genes such as *p53*, *PTEN*, *SIRT6*, and *VHL*, provide a selective advantage for cell survival and growth, ultimately leading to malignancy. Alterations to DNA copy number are routinely observed, exemplified by amplification of *EGFR* in gliomas, *MYCN* in neuroblastoma, and *ERBB2* in breast and ovarian cancer[127].

4.2.2. Cellular senescence

Senescent cells stop dividing permanently, develop a distinct secretory profile, and build up in the kidneys and other organs as a result of ageing or tissue damage. Senescent cells are not only dysfunctional themselves but also influence surrounding cells by releasing a wide range of pro-inflammatory and pro-fibrotic molecules known collectively as the senescence-associated secretory phenotype (SASP). This includes factors such as TGF-β, PAI-1, IL-6, and CTGF. Because these signals amplify through humoral pathways, even a small number of senescent cells can negatively affect both nearby and distant tissues. Therefore, targeting senescent cells either by removing them or reducing SASP production offers significant potential for new treatments for kidney diseases. In this review, we outline the current understanding of the complex mechanisms and defining features of cellular senescence, highlight newly discovered regulatory factors, and discuss possible therapeutic approaches for kidney disorders[128]. Major hallmark and types of cellular senescence includes telomere shortening, the DNA damage response (DDR), cyclin-dependent kinase (CDK) inhibitors, SASP, mitochondrial dysfunction, and autophagy dysfunction.

5. Therapeutic and preventive strategies to mitigate DNA damage

Therapeutic and preventive approaches to mitigate DNA damage focus on reducing oxidative stress through antioxidants, biocompatible membranes, and lifestyle changes. Oxidative stress (OS) refers to an imbalance in which the generation of reactive oxygen species exceeds the capacity of antioxidant systems. To protect against the harmful effects of these pro-oxidant free radicals, all

aerobic organisms rely on antioxidant defense that work to neutralize excess oxidants and repair the damage they cause. Antioxidants may be produced within the body (endogenous) or obtained from external sources such as food and supplements (exogenous). Endogenous antioxidants include both enzymatic and non-enzymatic types, which can be either water-soluble or fat-soluble molecules[129].

5.1. Antioxidant supplementations

To begin with, Vitamin E consists of eight fat-soluble molecules, comprising four tocopherols and four tocotrienols. Among the eight isomers, alpha-tocopherol is the only form of vitamin E that is widely available in foods, efficiently retained in the human body, and exhibits the highest biological activity. Vitamin E is recognized as the only specific physiological scavenger of free reactive oxygen species (ROS), capable of preventing cellular and molecular lipid peroxidation by interrupting oxidative chain reactions[127]. Patients with ESRD frequently experience deficiencies in small, water-soluble vitamins like vitamin C because these are removed rapidly during dialysis and dietary intake is often limited. In contrast, larger fat-soluble vitamins, such as vitamin A, tend to accumulate in the body. Vitamin E's antioxidant properties support human health and may help slow the progression of chronic diseases such as Alzheimer's, cancer, atherosclerosis, cardiovascular disease, allergies, and non-alcoholic fatty liver disease[128,129]. Although evidence from studies using oral vitamin E to attenuate oxidative stress and inflammation has been inconsistent, findings from research conducted in dialysis patients consistently indicate a reduction in DNA damage. This protective effect is likely attributable to the antioxidant activity of vitamin E, with improvement observed regardless of the administration route, whether delivered orally or via a vitamin E–coated dialyzer[129-132].

Similarly, the B vitamins are a group of related essential nutrients, with B1 (thiamine), B6 (pyridoxine), B12 (cobalamin), and B9 (folic acid) being among the most active forms. Folic acid, a key member of this group, plays an important role in DNA synthesis and amino acid metabolism. In both the general population and individuals with CKD, folic acid together with vitamins B6 and B12 is commonly used to lower homocysteine levels; a marker of protein oxidation that is associated with cardiovascular disease[133,134]. Adding folic acid and vitamin B12 has been shown to greatly reduce genomic damage in the lymphocytes of chronic haemodialysis patients[135].

In addition, coenzyme Q10 is a fat-soluble, vitamin-like compound that plays a crucial role in energy production. Because the liver, heart, and kidneys have high energy demands, they naturally contain the highest levels of CoQ10[136]. Coenzyme Q10 functions as an electron carrier, which enables it to act as a potential scavenger of reactive oxygen species (ROS)[137,138]. During hemodialysis, substantial amounts of vitamin C are lost, and the formation of ascorbate free radicals further contributes to increased oxidative stress[139]. Although interventions using intravenous vitamin C or ascorbate-rich dialysate have shown potential in reducing oxidative markers and DNA damage in some studies, overall findings remain inconsistent. In certain cases, supplementation failed to attenuate oxidative stress and, when other antioxidants such as vitamin E are depleted, may even exert prooxidant effects. Therefore, routine vitamin C supplementation for oxidative stress management in dialysis patients remains inconclusive and cannot be generally recommended[140].

5.2. Biocompatible membrane

Biocompatible membranes provide a key strategy to mitigate DNA damage by addressing the underlying oxidative stress, which is postulated as a risk factor for the high levels of genetic damage reported in CKD patients. The most direct intervention involves the use of Vitamin E-coated membranes (ViE-m), which utilize alpha-tocopherol as a lipid-soluble reactive oxygen species (ROS) scavenger on the blood surface. Clinical studies confirm that ViE-m use leads to a significant decrease in the levels of oxidative DNA damage in hemodialysis patients[141,142]. Further strategies focus on enhanced biocompatibility: the hydrophilic polysulfone neovascular(NV) membrane helps relieve inflammation and oxidative stress by suppressing platelet and leukocyte activation, maintaining stable levels of oxidative stress markers like pentosidine[143,144], while Ethylene-Vinyl Alcohol Copolymer (EVOH) membranes achieve a greater oxidative stress reduction and significantly reduce plasma levels of oxidation protein products compared to cellulose membranes[145].

5.3. Life style measures.

A balanced, kidney-friendly diet is essential for maintaining kidney health and slowing the progression of chronic kidney disease (CKD). Limiting sodium intake is particularly important, as excessive salt can increase blood pressure and place additional strain on the kidneys. Including nutritious foods such as fresh fruits, vegetables, and whole grains, along with guidance from a dietician, helps ensure adequate nutrition while managing dietary restrictions. Equally important is staying properly hydrated, as dehydration can impair kidney function; therefore, consuming an adequate amount of water daily is crucial. Maintaining a healthy weight also plays a significant role in reducing the risk of kidney disease, since obesity increases the likelihood of developing diabetes and high blood pressure, both of which are major contributors to CKD. Regular physical activity, including walking, yoga, or swimming, supports weight management while improving cardiovascular health. Finally, lifestyle factors such as chronic stress and smoking can negatively affect kidney function; incorporating stress-reduction techniques like meditation or deep breathing and quitting smoking can protect the kidneys and promote overall health[146].

6. Future direction and clinical implications.

Future research and clinical strategies for addressing oxidative stress in dialysis patients are focused on more precise monitoring and the development of targeted therapies. A primary future direction is the use of cell-specific biomarkers, specifically the redox state of peripheral blood polymorphonuclear (PMNs) and mononuclear (MNs) leukocytes, to monitor CKD progression and the specific impact of hemodialysis (HD) versus peritoneal dialysis (PD). Because these cells are easily obtained and analyzed, their oxidant and antioxidant parameters offer a practical clinical tool for tailoring dialysis prescriptions to a patient's unique cellular profile[147]. Pharmacological targeting represents a major area of exploration, with a focus on activating the NRF2 pathway (using compounds like sulforaphane) to stimulate endogenous antioxidant genes and stabilizing the HIF pathway (e.g., Roxadustat) to help cells adapt to chronic oxidative conditions. Novel metabolic interventions, such as inhibiting eIF5A hypusination with the compound GC7, are being investigated to protect tissues by "silencing" mitochondria and reducing the production of reactive oxygen species (ROS) during stress[148]. Additionally, targeting pro-oxidant enzymes like xanthine oxidase (XO) and NADPH oxidase (NOX) with specific inhibitors remains a key clinical implication for reducing baseline oxidative damage. Optimization of dialysis protocols is also a priority, specifically through the use of citrate-buffered dialysates and advanced biocompatible membranes that minimize leukocyte activation and preserve the antioxidant balance during treatment. Medical nutrition therapy will play an increasing role in future strategies to address micronutrient depletion and the accumulation of uremic toxins that further exhaust extracellular antioxidant defenses[147]. Ultimately, the transition toward large-scale, prospective clinical trials is essential to validate these redox biomarkers and confirm the long-term safety and efficacy of combining these pharmacological and nutritional approaches[147,148].

7. CONCLUSION

Chronic kidney disease (CKD) and end-stage renal disease (ESRD) establish a foundation of heightened oxidative stress (OS) due to chronic inflammation and the retention of uremic toxins, a condition that is drastically intensified by renal replacement therapies. This imbalance, characterized by excessive reactive oxygen species (ROS) production, directly targets and damages nucleic acids, leading to genomic instability observed as DNA base oxidation, strand breaks, chromosomal alterations, and accelerated telomere shortening. The degree of genotoxic injury is consistently documented using key biomarkers, such as 8-hydroxy-2′-deoxyguanosine (8-OHdG), the comet assay, and the micronucleus test, which demonstrate significantly higher DNA damage levels in individuals undergoing both hemodialysis (HD) and peritoneal dialysis (PD) compared to healthy controls. Moreover, the failure to effectively repair this damage, particularly through compromised pathways like Base Excision Repair (BER) involving genetic polymorphisms, further exacerbates genomic instability in the dialysis population.

A comparative analysis shows that while both dialysis modalities induce DNA damage, they operate through distinct mechanisms that affect the pattern of oxidative injury. Hemodialysis often results in acute, episodic oxidative bursts, primarily triggered by the activation of complement proteins and immune cells upon blood contact

with bio-incompatible membranes and the extracorporeal circuit. In contrast, peritoneal dialysis promotes sustained, low-grade oxidative stress, fueled by continuous exposure to acidic, glucose-based dialysate and its degradation products, leading to local ROS generation and peritoneal inflammation.

To counteract this persistent genotoxic risk, therapeutic and preventive strategies are primarily aimed at reducing the oxidative burden associated with dialysis. Key interventions involve optimizing the dialysis hardware, such as employing Vitamin E-coated membranes (ViE-m) in HD, which clinical data confirms can significantly decrease markers of oxidative DNA damage. Additionally, targeted nutritional support with antioxidant supplementation, including folic acid and Vitamin B12, has proven effective in reducing genomic damage in chronic HD patients. Furthermore, comprehensive lifestyle modifications—including a kidney-friendly diet, regular physical activity, and stress management—contribute essential support for mitigating risk factors associated with CKD progression. Future clinical research must focus on leveraging reliable biomarkers, like 8-OHdG, to guide personalized dialysis management and investigate novel inhibitors to overcome resistance mechanisms within DNA damage response pathways, ultimately improving the long-term prognosis and quality of life for ESRD patients

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S No.	ABBREVATION	FULL FORM
1.	8-OHdG	8-hydroxy-2'-deoxyguanosine
2.	AGEs	Advanced glycation end products
3.	AQP1	Aquaporin water channels
4.	ASK1	Apoptosis Signal-Regulating Kinase 1
5.	AV	Arteriovenous
6.	BER	Base Excision Repair
7.	BMI	Body Mass Index
8.	BMK1/ERK5	MAP kinase 1 pathway
9.	C3a, C5a	Complement proteins activated during dialysis
10.	CAT	Cytosolic catalase
11.	CBMN assay	Cytokinesis-Block Micronucleus assay
12.	CDA	Cellulose diacetate
13.	CDK	Cyclin-dependent kinase
14.	CIN	Chromosome instability
15.	CKD	Chronic kidney disease
16.	COPD	Chronic obstructive pulmonary disease
17.	CoQ10	Coenzyme Q10
18.	CPDs	Cyclobutane pyrimidine dimers
19.	CRF	Chronic renal failure
20.	CTA	Cellulose triacetate
21.	CTGF	Connective tissue growth factor
22.	Da	Dalton
23.	DNA	Deoxyribonucleic acid
24.	DDR	DNA damage response
25.	ERK1/2	Extracellular signal-regulated kinases 1 and 2
26.	ESRD	End-stage renal disease
27.	EVOH	Ethylene-vinyl alcohol copolymer
28.	FPG	Formamidopyrimidine DNA glycosylase
29.	GDP	Glucose degradation product
1.	GFR	Glomerular filtration rate
2.	GG-NER	Global genome nucleotide excision repair
3.	GI	Genomic instability
4.	Gpx	Glutathione peroxidase
5.	HD	Hemodialysis
6.	HDF	Online hemodiafiltration
7.	HHD	Home hemodialysis
8.	hOGG1	Human 8-oxoguanine DNA glycosylase 1
9.	HR	Homologous recombination
10.	IQR	Interquartile Range
11.	ICHD	In-center hemodialysis
12.	IL	Interleukins
13.	JAK/STAT	Janus kinase/signal transducer and activator of transcription
14.	JNK	c-Jun N-terminal kinases
15.	KDIGO	Kidney Disease: Improving Global Outcomes
16.	KDOQI	Kidney Disease Outcomes Quality Initiative
17.	Kuf	Ultrafiltration coefficient
18.	LDL	Low-density lipoprotein
19.	MAPK	Mitogen-activated protein kinases
20.	MAPKKs	Mitogen-activated protein kinase kinases
21.	MAPKKKs	Mitogen-activated protein kinase kinase kinases
22.	MDA	Malondialdehyde
23.	MDM2	A negative regulator of the p53 protein
24.	MEKK1–4	Mitogen-activated protein kinase kinases 1–4
25.	MIA	Malnutrition-inflammation-atherosclerosis syndrome
26.	MKP	MAP kinase phosphatase
27.	MLK3	Mixed-lineage kinase 3
28.	MMR	Mismatch repair (DNA repair pathway)
29.	mmol/L	millimoles per liter
30.	mEq/L	milliequivalents per liter.
31.	MTH1	MutT homolog 1
32.	MUTYH	MutY homolog
33.	NER	Nucleotide excision repair
34.	NF-êB	Nuclear factor-kappa B
35.	NHEJ	Non-homologous end joining
36.	NO	Nitric oxide
37.	Nrf2	Nuclear factor erythroid 2–related factor 2
38.	NV	Hydrophilic polysulfone membrane
39.	OH	hydroxyl radical
40.	O_2^-	superoxide anion
41.	OGG1	8-oxoguanine DNA glycosylase 1.
42.	QT	Q wave to the end of the T wave.
43.	OS	Oxidative stress
44.	PAI-1	Plasminogen activator inhibitor-1
45.	PAN	Polyacrylonitrile
46.	PC	Polycarbonate
47.	PD	Peritoneal dialysis
48.	PES	Polyethersulfone
49.	PET	Peritoneal Equilibration Test
50.	PSTR	Peritoneal solute transfer rates
51.	PSU	Polysulfone
52.	PTH	Parathyroid hormone
53.	RHD	Rel-homology domain
54.	RelA	v-rel avian reticuloendotheliosis viral oncogene homolog A.
55.	RelB	v-rel avian reticuloendotheliosis viral oncogene homolog B.
56.	ROS	Reactive oxygen species
57.	SASP	Senescence-associated secretory phenotype
58.	SCA	Sudden cardiac arrest
59.	SOD	Superoxide dismutase
60.	TC-NER	Transcription-coupled nucleotide excision repair
61.	TGF-â	Transforming growth factor-beta
62.	TNF-á	Tumor necrosis factor-alpha
63.	UV	Ultraviolet
64.	UF	Ultrafiltration
65.	ViE-m	Vitamin E-coated membranes