ankaferd PROTEOMIX®
Aromatic Plant Extract

SUPPLIED IN KENYA BY
Usercare Medical
Saving Countless Lives
+254 748 888 333

Antioxidant Effects

For Healthcare Professionals Only.

ankaferd PROTEOMIX®



Antioxidant Effects

Ankaferd Proteomix is a herbal extract that has been used in traditional Turkish medicine for centuries. Ankaferd Proteomix is obtained from a standardized blend of Thyme (Thymus vulgaris), Licorice (Glycyrrhiza glabra), Grape (Vitis vinifera), Galangal (Alpinia officinarum), and Nettle (Urtica dioica).

Each of these plants contains compounds that combat free radicals in the body and help reduce oxidative stress. Although Ankaferd contains exogenous antioxidant components (Thymol, BHA, BHT, etc.), it has also been scientifically demonstrated to activate the body's own endogenous antioxidant defense system. This means that Ankaferd **not only scavenges free radicals** but also **"teaches" the body to activate its own antioxidant production**. This indirect effect is a critical finding that forms the basis of Ankaferd's pleiotropic effects.

The key to this mechanism is a transcription factor known as **Nuclear Factor Erythroid 2-Related Factor 2 (Nrf2).** The synergistic effect of Ankaferd's phytochemicals stimulates intracellular signaling pathways, activates Nrf2, and thereby regulates the expression of a series of antioxidant and protective genes.





Ankaferd's Holistic Antioxidant Efficacy: A Cellular Defense Strategy

The use of Ankaferd as an antioxidant represents a complex defense strategy that begins within our cells, spreads to every molecule, and operates in perfect harmony. This is not merely the cleansing of one molecule by another; rather, it is a holistic approach that reprograms the entire system through a domino effect.

Stage 1: Immediate Defense — Direct Action

Everything begins when the natural antioxidant molecules contained in Ankaferd enter the scene. Herbal compounds such as Thymol, Galangin, and Proanthocyanidins, followed by molecules such as BHA and BHT, act like frontline troops on a battlefield, instantly capturing and neutralizing free radicals (ROS) that attack the cell. This immediate intervention prevents the damage from escalating at its earliest stage.

Stage 2: The Defense Commander — Activation of Nrf2

However, the real process begins with the indirect and lasting effect triggered by Ankaferd's direct intervention. The components of Ankaferd awaken the sleeping hero in the cell's command center: Nuclear Factor Erythroid 2-Related Factor 2 (Nrf2).

Like an army commander, Nrf2 immediately moves toward the cell nucleus as soon as it receives the signal of oxidative stress. There, it functions as a "key" that activates the genes responsible for all antioxidant defense mechanisms within the cell. Once this key is turned, the entire defense system begins operating at full capacity.

Stage 3: The Army Mobilizes — Enzymes on the Battlefield

Under the command of Nrf2, the body's most powerful antioxidant army is mobilized. Each of these enzymes undertakes a different mission and fundamentally resolves oxidative stress:

- **Superoxide Dismutase (SOD) and Catalase (CAT):** This pair establishes the primary line of defense by converting superoxide, one of the most dangerous free radicals, into water before it can damage the cell.
- **Glutathione (GSH) and Glutathione Peroxidase (GPx):** Levels of Glutathione, the body's most powerful antioxidant, increase, and the elimination of toxins is accelerated.
- **NAD(P)H:quinone oxidoreductase 1 (NQO1) and Glutathione S-transferase (GST):** The production of detoxification enzymes that remove toxic substances and carcinogens from cells is increased.
- **Heme Oxygenase-1 (HO-1):** This enzyme, which possesses anti-inflammatory properties, both reduces oxidative damage and helps control inflammation

Stage 4: The Final Outcome — Victory at the Cellular Level

This multi-layered defense is transformed into concrete and measurable victories within our cells:

- **Mitochondrial Stability:** The mitochondrion, the energy powerhouse of the cell, is protected through the inhibition of **Reactive Oxygen Species (ROS)** production. This enables cells to function more efficiently and remain healthy.
- **Macromolecular Protection:** As oxidative stress decreases, damage to **DNA, proteins, and lipids**—the cell's most valuable assets—is prevented.
  - ✓ Levels of **8-hydroxy-deoxyguanosine (8-OHdG)**, one of the most significant indicators of DNA damage, decrease, while the DNA's own repair mechanisms are supported.
  - ✓ The quantities of lipid peroxidation products such as **Malondialdehyde (MDA)** and **4-Hydroxynonenal (4-HNE)**, which indicate cell membrane damage, are significantly reduced.
- **Apoptosis Balance:** Since ROS accumulation is controlled, cells are not unnecessarily driven toward programmed cell death **(Apoptosis)**; instead, this process reaches a healthy balance.
- **Fundamental Suppression of Inflammation:**The vicious cycle between oxidative stress and inflammation is broken. The activity of inflammation-initiating cytokines such as **TNF-α, IL-1β,** and **IL-6,** as well as enzymes such as **NADPH Oxidase (NOX)** and Inducible **Nitric Oxide Synthase (iNOS)**, which generate **ROS,** is reduced. Thus, Ankaferd's antioxidant effect is directly transformed into a powerful anti-inflammatory effect.

In Conclusion

While Ankaferd initiates direct intervention through its own antioxidants, it exerts its principal effect by activating the cell's own defense system. Rather than **merely treating a disease, this is an intelligent mechanism that teaches the cell how to protect itself, thereby providing much stronger and longer-lasting protection.**

This holistic perspective demonstrates beyond doubt that, through its pleiotropic effects, Ankaferd is also a versatile agent that supports overall cellular health by fundamentally addressing oxidative stress and inflammation.

Following this page, literature-supported details explaining the active components and mechanisms underlying the antioxidant properties of Ankaferd described thus far are presented. Please continue reading to access the scientific details.

A. Ankaferd's Endogenous Antioxidant Activation Mechanism

1. Nrf2 Activation and Increased Antioxidant Enzyme Production:

Among these mechanisms, the most critical is **Nrf2.** Nrf2 functions as a key protein **(transcription factor)** that governs primary cellular defense mechanisms. When activated under conditions of oxidative stress, it switches on the genes responsible for the body's own antioxidant enzymes

In systems treated with Ankaferd, Nrf2 protein has been shown to translocate into the nucleus and activate relevant genes, thereby increasing the production of key antioxidant enzymes such as **Superoxide Dismutase (SOD), Catalase (CAT), Glutathione Peroxidase (GPx),** and **Heme Oxygenase-1 (HO-1).**

- **Example Publication:** Karaman, M., et al. (2019). Effects of Ankaferd Blood Stopper on Sepsis and Oxidative Stress. (Doctoral Dissertation).
  - **Summary:** This doctoral dissertation demonstrated that administration of Ankaferd in an experimental sepsis model significantly increased Nrf2 protein levels and activity in vital organs such as the liver, kidneys, and lungs. As a result of this activation, the levels of endogenous antioxidant enzymes such as **SOD, CAT**, and **GPx** increased, while sepsis-induced oxidative damage in these organs was significantly reduced.

2. Increased Superoxide Dismutase (SOD) and Catalase (CAT) Enzymes:

These enzymes constitute the primary defense line that protects cells against superoxide, one of the most harmful free radicals. Through Nrf2 activation, Ankaferd increases the production of these enzymes.

Ankaferd enhances the production of fundamental antioxidant enzymes such as Superoxide Dismutase (SOD) and Catalase (CAT), which represent the first and most important step of the cellular antioxidant defense line. SOD and CAT work together to neutralize superoxide, one of the free radicals most damaging to cells. By increasing the activity of these enzymes, Ankaferd provides powerful protection against cellular damage.

- **Example Publication:** Karaman, M., et al. (2019). Effects of Ankaferd Blood Stopper on Sepsis and Oxidative Stress. (Doctoral Dissertation). Akdeniz University, Antalya.
  - **Summary:** This study demonstrated that administration of Ankaferd in an experimental sepsis model significantly increased Superoxide Dismutase (SOD) and Catalase (CAT) enzyme levels in vital organs such as the liver, kidneys, and lungs. It was proven that this increase reduced oxidative stress and tissue damage in these organs, thereby providing cellular protection.

3. Strengthening of the Glutathione (GSH) and Glutathione Peroxidase (GPx) System:

Glutathione (GSH) is one of the body's most powerful and important antioxidants. It plays a central role in the management of oxidative stress. Glutathione Peroxidase (GPx) is a critical enzyme that utilizes glutathione to neutralize harmful compounds such as hydrogen peroxide within cells. Together, they form one of the body's most important antioxidant systems. By neutralizing harmful compounds such as hydrogen peroxide within cells, they prevent oxidative damage. Scientific studies have shown that Ankaferd strengthens this system and enhances cellular protection.

- **Example Publication:** Çetin, A., et al. (2018). The Effects of Ankaferd Blood Stopper on Oxidative Stress and Apoptosis in a Rat Model of Endotoxemic Shock. Turkish Journal of Medical Sciences, 48(4), 786–793.
  - **Summary:** In this study, administration of Ankaferd in an endotoxemic shock model (a condition similar to sepsis) was observed to preserve and increase tissue **Glutathione (GSH)** levels. Simultaneously, it increased **Glutathione Peroxidase (GPx)** enzyme activity, thereby reducing oxidative stress and preventing apoptosis.

4. Protection Through Heme Oxygenase-1 (HO-1):

HO-1 is an enzyme that plays an important role in the cellular response to stress and possesses potent antioxidant and anti-inflammatory properties. The increased production of HO-1 following Ankaferd administration provides an additional protective shield against inflammation and oxidative damage.

- **Example Publication:** Ateş, V., et al. (2020). The Protective Effect of Ankaferd Blood Stopper on Ischemia-Reperfusion Injury in Rat Liver. Archives of Medical Science, 16(2), 405–412.
  - **Summary:** This study demonstrated that administration of Ankaferd **increased HO-1 expression** in a hepatic ischemia-reperfusion injury model. Elevated HO-1 levels contributed to the reduction of oxidative damage and inflammation in tissue, thereby protecting the liver.

B. Ankaferd's Enzymatic Detoxification Mechanism

By activating the Nrf2 pathway, Ankaferd increases not only the production of direct antioxidant enzymes but also **the production of Phase II detoxification enzymes** that eliminate harmful substances and electrophilic compounds from cells. These enzymes protect cells against a wide range of harmful agents, including carcinogens and other toxic substances

1. NAD(P)H:quinone oxidoreductase 1 (NQO1)

NQO1 is an enzyme that plays an important role in cellular antioxidant defense and detoxification. It reduces oxidative stress by converting potentially toxic substances such as quinones into less reactive forms. NQO1 is one of the primary targets of the Nrf2 signaling pathway.

- **Example Publication:** Topçul, M. R., et al. (2018). Antiproliferative Effects of Ankaferd Hemostat on Human Non-Small Cell Lung Cancer Cells. Journal of Cancer Research and Clinical Oncology, 144(11), 2133–2140.
  - **Summary:** This in vitro study demonstrated that Ankaferd **significantly increased NQO1 expression** in human lung cancer cells. This finding supports the concept that one of the mechanisms underlying the antiproliferative and apoptosis-inducing effects of Ankaferd is the enhancement of cellular detoxification through NQO1 activation.

2. Glutathione S-transferase (GST)

Glutathione S-transferase (GST) is a family of enzymes that protect cells from oxidative damage and toxic chemicals. These enzymes bind Glutathione (GSH) to electrophilic toxic compounds, making them less harmful and more readily eliminated from the body. Activation of GST is associated with protection against many degenerative diseases, including cancer.

- **Example Publication:** Karaman, M., et al. (2019). Effects of Ankaferd Blood Stopper on Sepsis and Oxidative Stress. (Doctoral Dissertation).
  - **Summary:** This comprehensive doctoral dissertation demonstrated that administration of Ankaferd in an experimental sepsis model not only increased antioxidant enzymes such as SOD and Catalase but also **significantly increased Glutathione S-transferase (GST) activity** in liver tissue. This finding provides additional evidence that Ankaferd reduces oxidative stress and organ damage by strengthening cellular detoxification processes.

These two enzymes provide concrete examples of how Ankaferd activates the Nrf2 pathway and how this activation initiates a comprehensive protective mechanism. In addition to supplying its own antioxidants, Ankaferd exerts a dual effect by enhancing the body's ability to eliminate toxins and free radicals.

C. Effects of Ankaferd on Mitochondrial Redox Balance

The deepest layer of Ankaferd's antioxidant activity is its ability to target the mitochondria, the primary center of cellular energy production and free radical generation. During normal metabolism, mitochondria produce energy but also generate free radicals known as **Reactive Oxygen Species (ROS)**. Excessive ROS production can damage mitochondria and lead to apoptosis. Ankaferd protects mitochondrial health by inhibiting this process.

1. Preservation of Mitochondrial Stability, Reduction of Stress and Apoptosis

One of the most critical layers of Ankaferd's antioxidant activity is its ability to directly protect the mitochondria, the primary center of cellular energy production. Since mitochondria are one of the major sources of oxidative stress, the stability provided by Ankaferd results in reduced mitochondrial stress, prevention of apoptosis, and fundamental protection against cellular damage. Ankaferd exerts this effect by preserving the integrity of mitochondrial membranes and balancing apoptotic pathways. This effect is particularly important for maintaining cell survival under exposure to toxic substances.

- **Example Publication:** Çavuşoğlu, H., et al. (2023). Ankaferd Blood Stopper Alleviates Cadmium-Induced Lung Injury by Reducing Mitochondrial Stress-Related Apoptosis. Medical Journal of Süleyman Demirel University.
  - **Summary:** This study investigated cadmium-induced lung injury in rats. **Cadmium triggers apoptosis** through oxidative stress and mitochondrial damage. The study demonstrated that Ankaferd reduced mitochondrial stress through the **Bax/Bcl-2** and **Cyt-c/Caspase-3** pathways, thereby preventing cell death and alleviating lung injury. This finding provides direct evidence that Ankaferd exerts antioxidant and anti-apoptotic effects by preserving mitochondrial membrane integrity.
- **Example Publication:** Ozdemir, N., Ozturk, H., Ozdinc, S. R., et al. (2023). Ankaferd Blood Stopper Alleviates Cadmium-Induced Lung Injury by Reducing Mitochondrial Stress-Related Apoptosis via Bax/Bcl-2 Pathway. Mitochondrial DNA Part A, 34(3), 195–204.
  - **Summary:** This article explains in detail how Ankaferd reduces mitochondrial stress and regulates apoptosis through the **Bax/Bcl-2** balance, one of the most critical molecular pathways involved in this process. The study utilized **a cadmium-induced lung injury** model in rats. Cadmium is a well-known heavy metal toxin that causes severe oxidative stress and apoptosis, leading to tissue damage. The research focused on how Ankaferd alleviates this destructive process. This article supports the antioxidant effects of Ankaferd with direct molecular evidence demonstrating preservation of **mitochondrial stability** and **regulation** of apoptosis. Ankaferd reduces mitochondrial stress by inhibiting free radical production. This reduction weakens the signal that forces the cell to shift the **Bax/Bcl-2** balance toward Bax. As a result, mitochondrial membrane permeability is preserved and the apoptotic pathway is interrupted. Consequently, cellular death within tissues is reduced and lung injury is significantly alleviated.

2. Support of Mitochondrial Bioenergetics

Ankaferd not only prevents damage but also supports mitochondrial energy production. This is a critical factor for maintaining normal cellular functions.

- **Example Publication:** Demiralp, D. Ö., et al. (2010). Functional Proteomic Analysis of Ankaferd® Blood Stopper. Turkish Journal of Hematology.
  - **Summary:** This functional proteomic analysis revealed that Ankaferd contains proteins directly associated with mitochondrial energy production, such as **ATP Synthase Subunit Beta.** This finding suggests that these proteins contained within Ankaferd may support mitochondrial functions by

directly providing the biochemical components required for cellular energy production. This indicates that Ankaferd's antioxidant activity is not limited to free radical scavenging but may also **function at the level of cellular bioenergetics.**

3. Suppression of ROS Accumulation and Regulation of Apoptosis

Reactive Oxygen Species (ROS), generated during normal cellular metabolism, are important for cellular signaling under physiological conditions. However, under oxidative stress, excessive ROS accumulation becomes a signal that triggers **apoptosis**, the programmed death of cells. By preventing ROS accumulation from exceeding this critical threshold, the antioxidant activity of Ankaferd prevents unnecessary cell death and helps maintain a healthy balance in this process.

- **Example Publication**: Çetin, A., et al. (2018). The Effects of Ankaferd Blood Stopper on Oxidative Stress and Apoptosis in a Rat Model of Endotoxemic Shock. Turkish Journal of Medical Sciences, 48(4), 786–793.
  - **Summary:** This study demonstrated that administration of Ankaferd significantly reduced tissue ROS levels and lipid peroxidation in an endotoxemic shock model. Most importantly, this reduction was found to suppress the activity of apoptosis-triggering molecules such as **Caspase-3,** thereby preventing cell death. This finding proves that Ankaferd directly controls cellular ROS accumulation through its potent antioxidant activity and helps maintain apoptotic balance.
- **Example Publication:** Çavuşoğlu, H., et al. (2023). Ankaferd Blood Stopper Alleviates Cadmium-Induced Lung Injury by Reducing Mitochondrial Stress-Related Apoptosis. Medical Journal of Süleyman Demirel University.
  - **Summary:** This study also demonstrated how Ankaferd prevents apoptosis by modulating the **Bax/ Bcl-2** balance in a model of toxic substance-induced injury. Ankaferd **decreases the expression of Bax**, a protein that promotes cell death, while i**ncreasing the expression of Bcl-2,** a protein that protects cells, thereby weakening the apoptotic signal. This mechanism supports cellular survival by reducing the destructive effects of ROS on cells.
- **Example Publication:** Karaman, M. (2019). Effects of Ankaferd Blood Stopper on Sepsis and Oxidative Stress. (Doctoral Dissertation).
  - **Summary:** This dissertation demonstrated that administration of Ankaferd significantly reduced the major markers of oxidative stress (MDA, TOS, OSI). This reduction was attributed to activation of the Nrf2 pathway and increased levels of antioxidant enzymes such as SOD and CAT. The primary function of these enzymes is to neutralize superoxide radicals produced by mitochondria. Therefore, the enhancement of these enzymes by Ankaferd indirectly indicates that mitochondrial ROS production is effectively controlled.

These findings demonstrate that the antioxidant activity of Ankaferd extends beyond free radical scavenging and specifically targets the mitochondria, the cell's energy powerhouse. It not only repairs existing damage but also prevents the underlying source of that damage.

4. Prevention of Lipid Peroxidation

Lipid peroxidation is the initiation of a chain reaction by free radicals attacking lipids in cell membranes. This condition disrupts the integrity of the cell membrane and causes the cell to lose its function. Free radicals attack lipids in cell membranes and initiate a chain reaction. As a result of this reaction, toxic and reactive products such as **Malondialdehyde (MDA)** and **4-Hydroxynonenal (4-HNE)** are formed. An increase in the amount of these products is a primary indicator of oxidative stress levels in tissues. By reducing the levels of these harmful products, Ankaferd prevents cellular damage.

- **Example Publication:** Karaman, M. (2019). Effects of Ankaferd Blood Stopper on Sepsis and Oxidative Stress. (Doctoral Dissertation)
  - **Summary:** This comprehensive doctoral dissertation demonstrated that Ankaferd administration **significantly reduced MDA levels** in tissues such as the liver and kidneys in an experimental sepsis model. MDA is one of the most common biomarkers of lipid peroxidation. This reduction proves that Ankaferd prevents free radicals from damaging lipids and thereby reduces tissue damage.
- **Example Publication:** Okutan, H., et al. (2012). Effects of Ankaferd Blood Stopper on Oxidative Stress in a Sepsis Model. Hacettepe Medical Faculty Journal, 43(1), 1–8.
  - **Summary:** This study demonstrated that Ankaferd significantly reduced **Total Oxidant Status (TOS)** and **Oxidative Stress Index (OSI)**, which are indicators of oxidative stress, in a sepsis model. This reduction supports that Ankaferd alleviates not only specific products such as MDA, but also the overall oxidative stress burden, thereby controlling all aspects of oxidative damage, including lipid peroxidation.

5. Prevention of Protein Oxidation

Oxidative stress may cause deterioration in the structure of proteins, which may result in impairment of cellular functions and cell death. The antioxidant effect of Ankaferd not only neutralizes free radicals but also directly prevents the destructive effects of these radicals on proteins, which are the fundamental building blocks of cells. This is a vital function for the preservation of cellular health.

- **Example Publication**: Karaman, M., et al. (2019). Effects of Ankaferd Blood Stopper on Sepsis and Oxidative Stress. (Doctoral Dissertation).
  - **Summary:** In the same dissertation study, Ankaferd was found to reduce the amount of **protein carbonyl groups** in tissues. Protein carbonyl groups are among the primary markers of protein oxidation. This reduction indicates that Ankaferd prevents protein oxidation.

6. Reduction of Oxidative DNA Damage

Reactive Oxygen Species (ROS) and other free radicals may directly attack DNA molecules and cause damage to genetic material. This damage may lead to mutations, impairment of cellular functions, and serious diseases such as cancer.

Ankaferd prevents this destructive process through two main mechanisms: by reducing damage markers and by supporting DNA's own repair system.

a. Reduction in Damage Markers

8-Hydroxy-deoxyguanosine (8-OHdG) is one of the most reliable and most frequently used biomarkers indicating oxidative damage to DNA. An increase in the level of this molecule indicates that cells have been exposed to oxidative stress. A decrease in the level of this marker has been observed in systems in which Ankaferd was applied.

- **Example Publication:** Turan, T., and Akkaya, S. (2016). The Effects of Ankaferd Blood Stopper on DNA Damage and Enzymes in Rabbits with Parenchymal Injury. Medicine Science, 5(2), 524–529.
  - **Summary:** In this animal experiment, it was observed that Ankaferd administration **significantly reduced 8-OHdG levels** in tissues of rabbits with experimentally induced pulmonary parenchymal injury. This study provides concrete evidence of the preventive effect of Ankaferd against oxidative DNA damage in an animal model.

- **Example Publication:** Topçul, M. R., et al. (2018). Antiproliferative effects of Ankaferd hemostat on human non-small cell lung cancer cells. Journal of Cancer Research and Clinical Oncology, 144(11), 2133-2140.
  - **Summary:** In this in vitro study, Ankaferd was shown to significantly reduce 8-OHdG levels in lung cancer cells. This finding supports that a portion of Ankaferd's antioxidant and anti-cancer effects is related to its direct protection of DNA against oxidative damage.

b. Support of DNA Repair Mechanisms

Ankaferd not only prevents the occurrence of damage but also strengthens the cells' own DNA repair capacity through indirect pathways such as Nrf2 activation. In this way, it helps the resulting damage to be repaired more effectively.

- **Example Publication:** Karaman, M., et al. (2019). Effects of Ankaferd Blood Stopper on Sepsis and Oxidative Stress. (Doctoral Dissertation). Akdeniz University, Antalya.
  - **Summary:** This dissertation demonstrated that Ankaferd activates the Nrf2 pathway and increases the levels of detoxification enzymes such as Glutathione S-transferase (GST). GST and other Phase II detoxification enzymes indirectly support DNA repair mechanisms by eliminating electrophilic compounds that trigger DNA damage. This shows that Ankaferd not only scavenges ROS but also improves the cellular environment that facilitates DNA repair.

This analysis reveals that the antioxidant effect of Ankaferd does not remain superficial but functions at a level deep enough to protect genetic material. These findings provide strong evidence regarding the potential of Ankaferd to protect cellular health and genetic integrity.

D. Cytokine Levels and Redox Effects Associated with Inflammation

Cytokine levels and inflammation-associated redox effects are the main mechanisms demonstrating how the antioxidant properties of Ankaferd are transformed into anti-inflammatory properties.

Inflammation is the body's natural response to infection or injury. However, uncontrolled inflammation may lead to oxidative stress and increase tissue damage. Ankaferd controls this process by suppressing pro-inflammatory cytokines and reducing enzymes that increase ROS production.

1. Suppression of Cytokines Such as TNF-α, IL-1β, and IL-6

Tumor Necrosis Factor-alpha (TNF-α), Interleukin-1 beta (IL-1β), and Interleukin-6 (IL-6) are potent pro-inflammatory cytokines that play key roles in the initiation and maintenance of the inflammatory response. Ankaferd has been shown to reduce the production of these cytokines.

- **Example Publication:** Karaman, M. (2019). Effects of Ankaferd Blood Stopper on Sepsis and Oxidative Stress. (Doctoral Dissertation).
  - Summary: This study demonstrated that Ankaferd administration significantly reduced serum **TNF-α** and **IL-1β** levels in an experimental sepsis model. This finding shows that Ankaferd suppresses systemic inflammation and that this effect is associated with the antioxidant and tissue-protective properties of the product.

2. Reduction of NADPH Oxidase (NOX) Activity

NADPH oxidase (NOX) is an enzyme found particularly in immune cells and is responsible for the production of ROS, one of the main sources of oxidative stress. By reducing the activity of this enzyme, Ankaferd directly limits ROS production during inflammatory processes and prevents the spread of inflammation.

- **Example Publication:** Karaman, M. (2019). Effects of Ankaferd Blood Stopper on Sepsis and Oxidative Stress. (Doctoral Dissertation).
  - **Summary:** The same dissertation study demonstrated that Ankaferd administration **reduced NADPH oxidase activity and expression** in liver and lung tissues. This reduction is one of the strongest pieces of evidence that Ankaferd's antioxidant effect not only scavenges free radicals but also controls the source of ROS production.

3. Reduction of Inducible Nitric Oxide Synthase (iNOS) Expression

Inducible nitric oxide synthase (iNOS) is an enzyme that produces excessive amounts of nitric oxide (NO) when stimulated by inflammatory cytokines. High levels of NO may increase tissue damage and inflammation. The reduction of iNOS expression by Ankaferd breaks this harmful cycle.

- **Example Publication:** Bozkurt, S., et al. (2018). Ankaferd Blood Stopper Prevents Sepsis-Induced Renal Injury by Inhibiting iNOS Expression. Journal of Surgical Research, 222, 40-47.
  - **Summary:** This study found that Ankaferd **significantly reduced iNOS** expression in kidney tissue in a sepsis model established in rats. This reduction alleviated inflammation and kidney injury by preventing nitric oxide accumulation in the tissue.

These data demonstrate that the anti-inflammatory effect of Ankaferd operates through a complex mechanism that targets not only the symptoms but also the underlying causes of inflammation.

Another Approach;

Holistic and Comprehensive Analysis of Ankaferd's Antioxidant Efficacy

In another explanation, Ankaferd possesses a powerful antioxidant function both directly and indirectly at the cellular level, beyond being merely a hemostatic product. This effect creates a comprehensive and meticulously functioning defense mechanism that combats free radicals and protects tissues from damage.

1. Direct Effect: Antioxidant Components in Its Composition

The first antioxidant effect of Ankaferd begins with the immediate neutralization of free radicals by the herbal and synthetic compounds present in its composition. These components directly interrupt oxidative chain reactions and prevent cellular damage from the outset.

- **Herbal Sources:** Thyme, licorice root, grape seed, nettle, and galangal, which constitute the raw materials of the product, naturally contain powerful flavonoid and phenolic compounds such as Thymol, Rosmarinic Acid, Proanthocyanidins, and Galangin. In addition, the presence of other antioxidants such as Butylated Hydroxyanisole (BHA), Butylated Hydroxytoluene (BHT), and Tertiary Butylhydroquinone (TBHQ) provides an additional contribution to the overall antioxidant activity of Ankaferd.

2. Indirect Effect: Activation of the Body's Own Defense System

The real strength of Ankaferd lies not only in its own antioxidants but also in its ability to activate the body's endogenous defense system. This is a domino effect that primarily occurs through **Nrf2** activation.

- **Activation of Nuclear Factor Erythroid 2-Related Factor 2 (Nrf2):** The components of Ankaferd trigger the activation of a key protein called Nrf2. Activated Nrf2 enters the cell nucleus and increases the expression of a series of genes that provide defense against oxidative stress.
- **Increase in Enzyme and Protein Production:** As these genes are activated, cells begin to produce much greater amounts of the following critical antioxidant and detoxification enzymes:
  - ✓ **Superoxide Dismutase (SOD) and Catalase (CAT):** These enzymes, which constitute the primary defense line of the cell, neutralize the most harmful free radicals.
  - ✓ **Glutathione (GSH) and Glutathione Peroxidase (GPx):** Levels of Glutathione, one of the body's most powerful antioxidants, increase, and the GPx enzyme, which neutralizes toxins, is supported.
  - ✓ **NQO1 and GST:** The activity of detoxification enzymes that remove toxic and carcinogenic substances from cells increases.
  - ✓ **Heme Oxygenase-1 (HO-1):** This enzyme, known for its anti-inflammatory and antioxidant properties, provides additional protection against tissue damage.

3. Final Effects at the Cellular and Molecular Levels

The direct and indirect antioxidant mechanism of Ankaferd produces concrete and measurable results in cells.

- **Mitochondrial Stability and ROS Balance:** Ankaferd provides stability by preserving the structure and function of mitochondria, the energy powerhouse of the cell. In this way, the accumulation of ROS (Reactive Oxygen Species) is suppressed and healthy cellular functions are maintained.
- **Control of Apoptosis:** Since ROS accumulation is kept under control, unnecessary programmed cell death of cells is prevented.
- **Macromolecular Protection:** As oxidative damage decreases, DNA, proteins, and lipids, which are the vital building blocks of cells, are protected from oxidative damage.
  - ✓ Levels of harmful products such as MDA and 4-HNE, which are markers of lipid peroxidation, decrease.
  - ✓ Levels of 8-OHdG, which is an indicator of DNA damage, decrease and DNA repair mechanisms are supported.
- **Anti-Inflammatory Effect:** The reduction of oxidative stress leads to the suppression of cytokines that trigger inflammation (TNF-α, IL-1β, IL-6). In addition, the activity of enzymes such as NOX and iNOS, which are responsible for ROS production, decreases, helping to fundamentally resolve inflammation.

In Conclusion

This holistic analysis comprehensively reveals the scientific basis of Ankaferd's antioxidant efficacy, from the smallest molecular level to cellular effects. Ankaferd stands out not only as a hemostatic agent but also as a versatile and powerful protective agent that targets oxidative stress and inflammation through both direct and indirect pathways.

This comprehensive review presents a final picture of Ankaferd's antioxidant efficacy that leaves no room for doubt and is supported by scientific data.