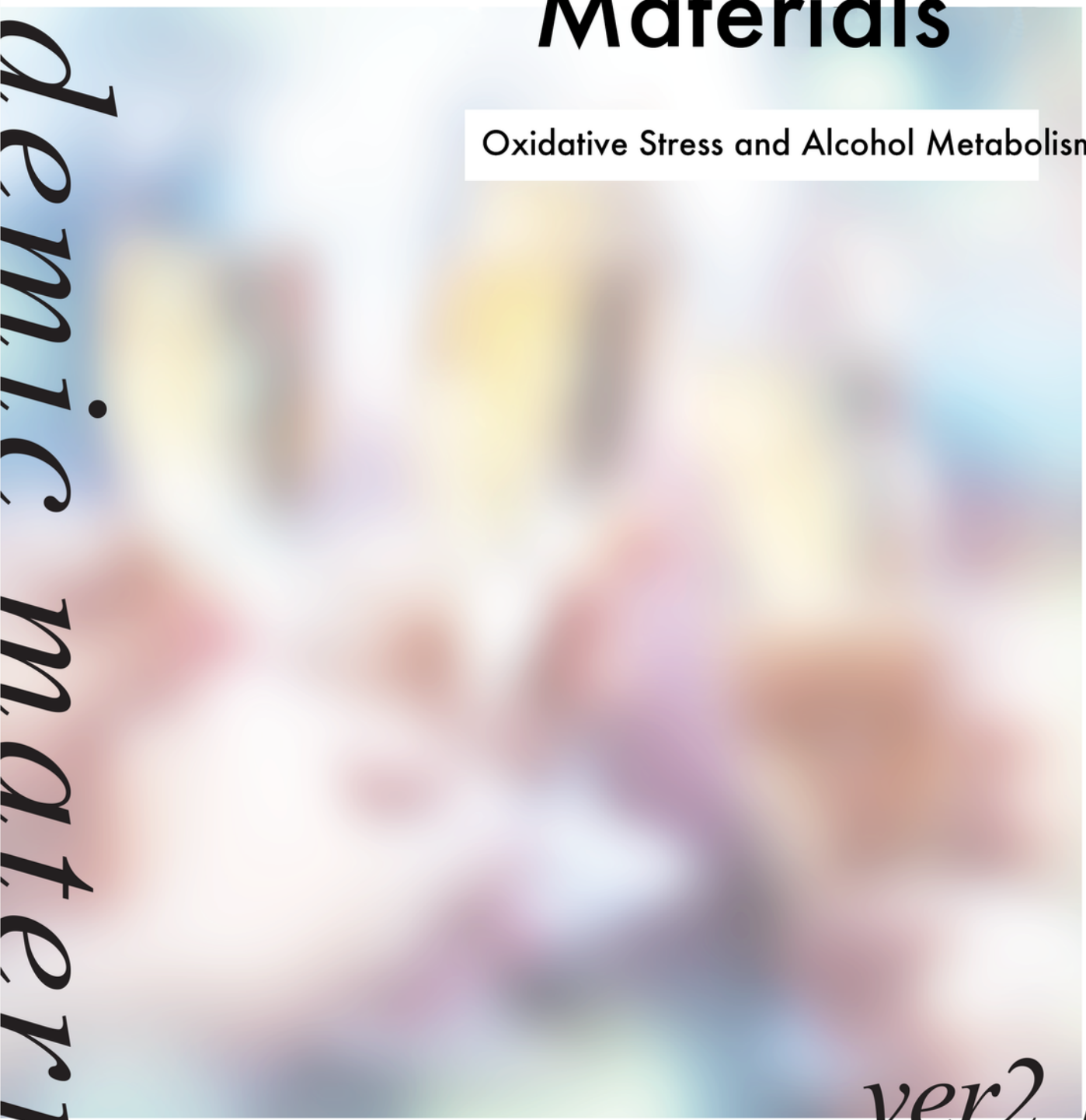
Academic Materials

# Academic Materials

## Oxidative Stress and Alcohol Metabolism



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Tokai National University Organization Gifu University Antioxidant Laboratory
Gifu University Faculty of Advanced Studies Center for Scientific Research Infrastructure
Joint Research Division Antioxidant Research Section

Antioxidant Research
Louis Pasteur Center for Medical Research

# Research Objectives

Oxidative stress is associated with sunburn, hay fever, and acne that occur throughout the year, especially from early spring to summer, and can cause significant emotional stress because it affects the quality of life and appearance. Oxidative stress is also associated with age-related odors and dementia, which can unintentionally affect the loved ones around us.

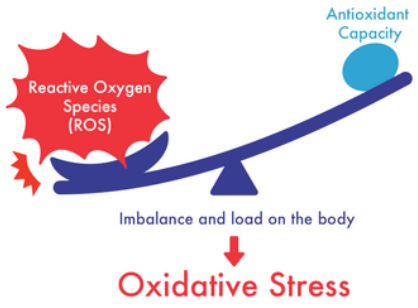
Lifestyle factors such as alcohol consumption and tobacco smoking also contribute to oxidative stress. Thus, oxidative stress can be caused by a variety of factors and is associated with many diseases. Therefore, it is beneficial not only to the individual but also to those around him/her to reduce oxidative stress on a daily basis. Most diseases are related to oxidative stress, or in other words, reducing oxidative stress on a daily basis can help prevent and treat many diseases.

Our ultimate goal is to contribute to a "healthy and long-lived society" with a large amount of evidence through various experiments to explain how suppressing oxidative stress can lead to disease prevention and treatment, and to explain the mechanism of oxidative stress. The ultimate goal is to contribute to a "healthy and long-lived society" with a large amount of evidence.

# What is Oxidative Stress?

The active energy necessary to move the body is produced in organelles called mitochondria in cells using the oxygen taken in through daily food and respiration.

However, at the same time, highly reactive oxygen species (ROS) are produced as by-products. In addition to energy production, it is said to be produced by various factors such as ultraviolet rays, radiation, infection by bacteria and viruses, external factors such as air pollution, lifestyle habits such as smoking and large amounts of alcohol, illness, and stress.



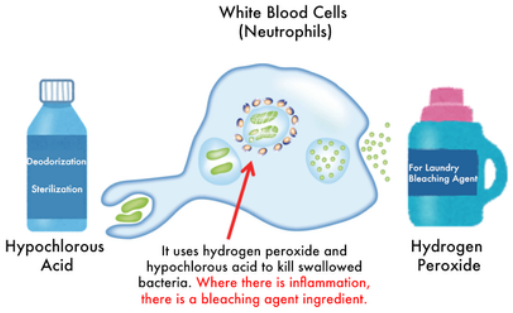

This active oxygen has the function of damaging DNA, lipids, proteins and enzymes that are essential for maintaining health in the body. Therefore, in order to protect the body, this active oxygen must be eliminated. This erasing power is called "antioxidant power", and in fact it is already present in our bodies. However, active oxygen cannot be completely eliminated. The important thing is the balance between the active oxygen that is constantly produced, the power to protect against it and the antioxidant power. When the active oxygen increases and this balance becomes imbalanced, the body will be upset. This state is called “oxidative stress”.
You have probably seen rusted nails and discolored apples. These are all "oxidation phenomena" caused by exposure to oxygen in the air. In a state of oxidative stress, exactly the same phenomenon is occurring in the body.

• Discoloration of Apples

• Rusty Nails

# What are Reactive Oxygen Species?

Active oxygen that causes oxidative stress is defined as "oxygen that is more activated than atmospheric oxygen, that is, oxygen that is highly reactive". Because of its high reactivity, it damages DNA, lipids, proteins, etc. in the body and causes inflammation. But it's not all bad. This powerful force is also used to exterminate bacteria and viruses that have invaded the body, so it also plays an important role in immunity. Let's think about something familiar.

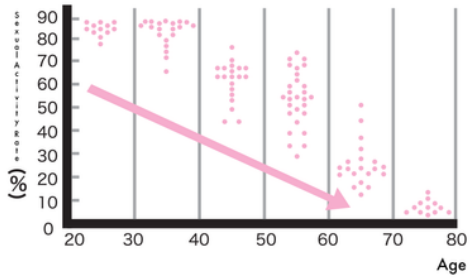



There are bleaches commonly used in laundry, the ingredient of which is Hydrogen Peroxide. There are also deodorizers and sanitizers used in daily life, the raw material of which is hypochlorous acid. In fact, both are a type of reactive oxygen species, which are also produced in our bodies. Bleaching agents, deodorizers, and sanitizers are made using this powerful bacteria-destroying property, and the body's immune system, the white blood cells, makes use of this property.

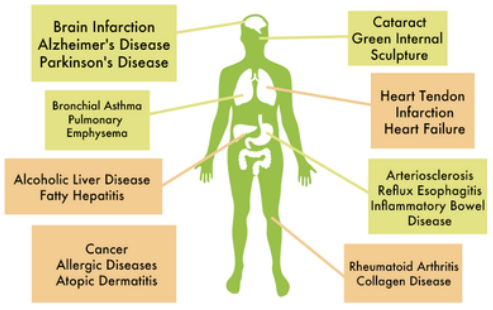
When they find bacteria that have invaded the body, the white blood cells wrap them up and swallow them, then release hydrogen peroxide or hypochlorite to kill them. When you get a wound, does it become red, swollen and painful? This is an inflammatory reaction that occurs when a large number of white blood cells gather to repair the wound and release a large amount of active oxygen and other substances at the site of the wound. If we think of it as a large number of white blood cells all releasing bleach and sanitizers into the bloodstream at the same time, then the body begins to "rust," as mentioned above. As you can imagine, this can lead to a whole body of diseases and other horrible consequences.

# Relationship to Disease

As mentioned earlier, our bodies are equipped with "antioxidant capacity" to fight oxidative stress. Antioxidant capacity begins at birth and gradually declines with age, peaking in the 20s. This increased oxidative stress causes the body to become rusty, leading to inflammation and cell damage. This rusting of the body is also known as "aging" and is linked to a variety of diseases. In fact, more than 150 diseases have been reported to be associated with oxidative stress.




Source: Blood,76835(1990)




Antioxidant therapies and research are currently being conducted around the world to treat many diseases. However, despite in vitro and animal studies detailing the mechanisms and preventive effects, there are still only a few drugs that prevent or treat diseases through antioxidant action. We have been waiting for an antioxidant that is versatile, shows no side effects, and is effective in the prevention and treatment of many oxidative stress diseases. If we can increase antioxidant capacity and reduce oxidative stress, we can prevent and improve aging and various diseases.

# Antioxidant Combination Products Developed

## Compound “SUPALIV”

To relieve alcohol metabolism and reduce the risk of alcohol-induced disease composition" (Japan Patent Office, Patent No. 5785581). A formula developed from research on the promotion of alcohol metabolism and the health hazards caused by alcohol.
The company has been conducting clinical trials and safety testing by a third-party organization. After undergoing clinical and safety testing by a third-party organization, the product is now available at convenience stores and drugstores nationwide. It is on sale to the general public at grocery stores and other retailers.

Active ingredients of "SUPALIV" (raw materials for production)

All are originally found in our bodies and are all natural ingredients.

Ingredients: Maltose (manufactured in Japan), Coenzyme Q10/Vitamin C (sugar), L-Glutamine (rice, corn), L-Cystine (chicken), Crystal cellulose, Hydroxypropyl cellulose, Magnesium stearate, Fumaric acid (aromatic hydrocarbon) Succinic acid (fumaric acid), Microdioxide Silicon dioxide, Vitamin B2 (dextrose), Niacin (amino acid)

## Safety of “SUPALIV”

Safety testing was commissioned to Ina Research Corporation (http://www.ina-research.co.jp/) in 2007.

Practice Tests

Effects of 1 - week repeated dosing in rats (INARESEARCH Study No: GL43080)
Chromosome Aberration Test Using Cultured Mammalian Cells (INARESEARCH Test No: BV07158)
4 - week repeated oral administration toxicity study in rats (INARESEARCH Test No: BV07156)
Reverse mutation test using bacteria (excluding cystine) (Ina Research Test No: BV07352)
Adverse drug reaction study in human clinical research (INARESEARCH Study No: NRP07-001)

In both studies, safety was confirmed. The maximum safe daily intake is reported to be at least 2 grams per kilogram of body weight.

## Patents Acquired

Based on the empirical data, we applied for patents in various countries around the world, and received a patent from the Japan Patent Office, Patent No. 5785581, "Composition for moderating alcohol metabolism and reducing the risk of alcohol-induced diseases" in Japan and all over the world (36 EU countries including Germany, USA, Canada, China, Australia, South Korea, and other countries in Eurasia).

## How "SUPALIV" works

SUPALIV contains eight active ingredients in optimal proportions: niacin, riboflavin, CoQ10, vitamin C, cystine, glutamine, succinic acid, fumaric acid, and fumaric acid, which are used to (1) accelerate alcohol and acetaldehyde metabolism and (2) act as antioxidants, SUPALIV contains eight active ingredients in optimal proportions: niacin, riboflavin, CoQ10, vitamin C, cystine, glutamine, succinic acid and fumaric acid.

1)Promotes alcohol and aldehyde metabolism

**Niacin (NAD)**
Acts as a coenzyme that aids alcohol-degrading enzymes

**Riboflavin, CoQ10**
Acts as a coenzyme that aids alcohol-degrading enzymes

**Fumaric Acid, Succinic Acid**
Acetic Acid Decomposition

**Glutamine**
Send Coenzymes to Mitochondria

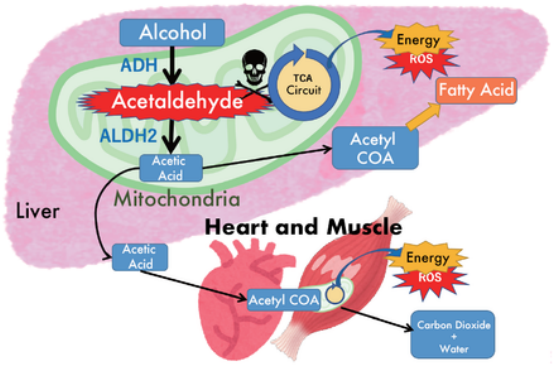
(2) Antioxidant action (neutralizes acetaldehyde toxicity)

**Vitamin C**
Direct Antioxidant Action

**Cystine**
Direct Antioxidant Action

**Glutamine**
Acts as glutathione to inhibit oxidation

## Alcohol Metabolism and Oxidative Stress




Alcohol entering the body is absorbed 20% from the stomach and 80% from the small intestine, and most of it is processed in the liver. In the liver, alcohol is immediately decomposed into acetaldehyde by the enzyme ADH (alcohol dehydratase) and MEOS (microsomal ethanol oxidizing system). The acetaldehyde produced is highly toxic and damages the liver if it remains in the liver for a long period of time. This is because in addition to the toxicity of acetaldehyde, acetaldehyde itself causes oxidative stress.

To detoxify it quickly, the liver breaks it down into acetic acid by an enzyme called ALDH2 (aldehyde dehydratase type 2) in intracellular mitochondria, which is then transported to the heart and muscles and finally released from the body in the form of oxygen dioxide and water.

Mitochondria not only metabolize alcohol, but also produce energy for our daily activities using the oxygen we take in through food and breathing. Mitochondria themselves produce reactive oxygen species (ROS), so when exposed to excessive oxidative stress, mitochondria are damaged and are unable to function as they should. Therefore, protecting mitochondria from oxidative stress will help maintain our health and prevent the harmful effects of alcohol.

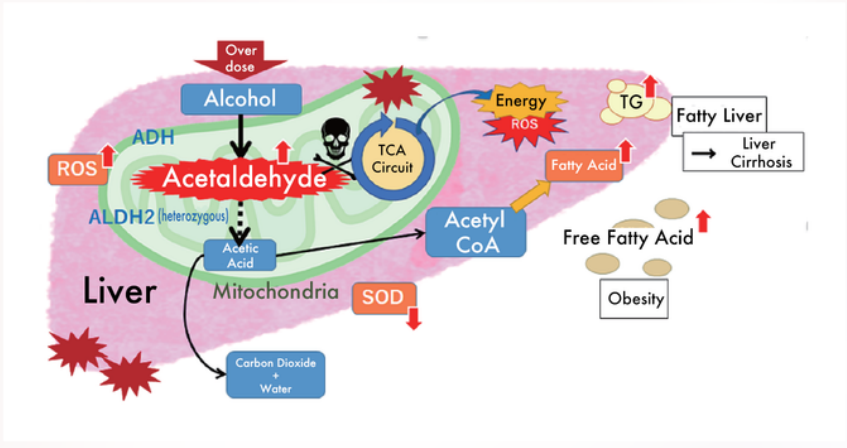
## Differences in Ability to Metabolize Alcohol - Alcohol Metabolizing Genes

The ability to metabolize alcohol is divided into three types, depending on the enzyme ALDH2, which breaks down acetaldehyde as described above: normal homozygous for full function of ALDH2, heterozygous for weak function of this enzyme, and abnormal homozygous for no function at all. Most Europeans and Africans are homozygous for normal ALDH2, while half of the Japanese are heterozygous or heterozygous for abnormal ALDH2. This is why half of the Japanese are said to be weak drinkers. This is due to the action of MEOS (microsomal ethanol oxidase), which is a pathway for alcohol degradation. This enzyme is activated when the amount of alcohol increases, so it seems that frequent drinking makes one stronger to a certain extent. However, since the ability to decompose acetaldehyde created by drinking does not change, people who are weak drinkers (especially those with abnormal homophobia) should be aware that no amount of training will make them drink more.

Racial Differences in ALDH2 Gene

| Genotype | Enzyme Activity | Japanese | Chinese | Korean | European | African |
|---|---|---|---|---|---|---|
| Normal Homozygous | 100% | 50% | 53% | 66% | 100% | 100% |
| Heterozygous | 6% | 44% | 41% | 27% | 0% | 0% |
| Abnormal Homozygous | 0% | 6% | 6% | 6% | 0% | 0% |

## Alcohol and its Relationship to Disease




Health damage caused by alcohol is caused by alcohol itself, but most of it is caused by acetaldehyde, a highly toxic intermediate metabolite. Continuing excessive drinking also activates alcohol metabolism in the liver, and this process increases free fatty acids and triglycerides in the liver, leading to obesity and fatty liver.

Also, if you drink more than your metabolic capacity, your metabolism will not be able to catch up, and acetaldehyde will stay in your body for a long time. Therefore, liver mitochondria are damaged by the toxicity and the resulting oxidative stress. As a result, mitochondrial function does not work properly, which leads to a vicious cycle in which metabolic function declines and oxidative stress further increases, causing cell damage and inflammation, leading to disease.
In heterozygotes with weak ALDH2 function, the rate at which acetaldehyde is broken down is slow, so excessive drinking increases the risk of liver damage.

# Research Results to Date

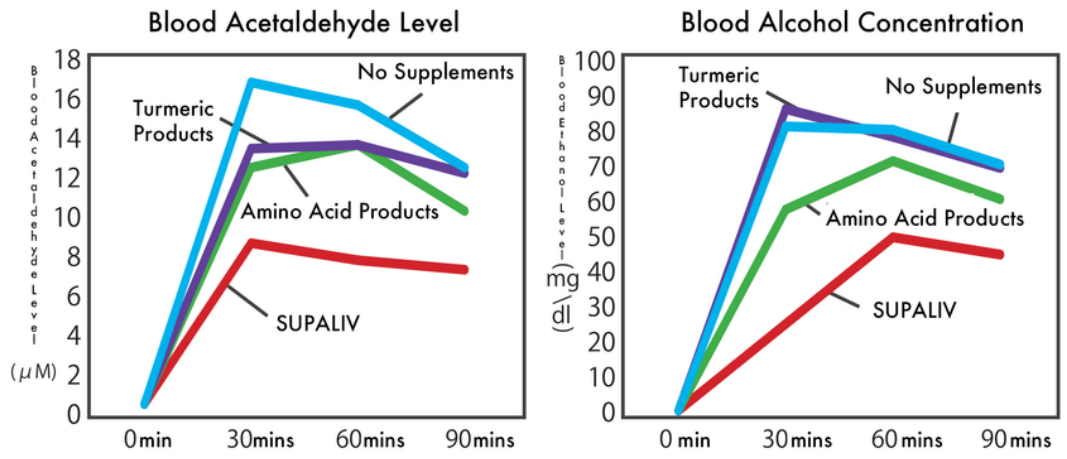
## Alcohol Metabolic Effects

Due to the increased opportunities for alcohol consumption, various types of anti-alcohol supplements are now available on the market. Therefore, we compared SUPALIV's ability to metabolize alcohol with that of typical supplements.

Tested on: Total of 6 men and 6 women
Test Method: Blood ethanol and acetaldehyde concentrations.

Test Subject: Supplements
Turmeric Products
Amino Acid Products Amino Acid Products

Evaluation Methods: Supplement non administered, SUPALIV, turmeric products, and amino acid products. All doses to be taken in a single serving as recommended by each product. Each anti-alcohol supplement was taken with 200 ml of water, followed 20 minutes later by drinking 400 ml of red wine over 30 minutes. Blood samples were drawn 0 minutes before drinking the wine and 30, 60, and 90 minutes after drinking the wine, and measurements were performed by BML Co. The effects of each supplement were compared with those of the control blood concentration when the supplement was not taken.



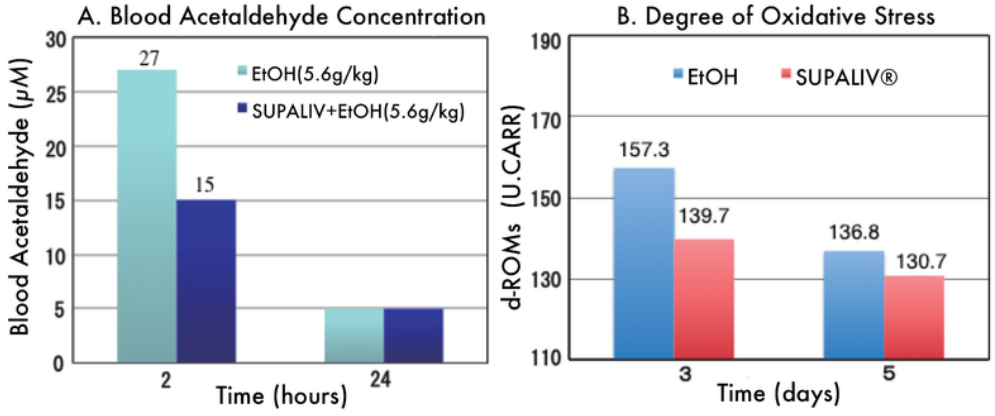

Quoted in Nikkei Trendy, February 2014 issue.

Results: Turmeric products and amino acid products were more effective than the control, but no effective data were obtained. On the other hand, SUPALIV effectively reduced both blood alcohol and acetaldehyde concentrations ( significant difference, P<0.05, t-Test). ( Significant difference, P<0.05, t-Test)

# Acetaldehyde and Oxidative Stress changes after Alcohol Administration

It is said that oxidative stress is increased by alcohol consumption. Therefore, we actually overdosed mice with alcohol and verified changes in blood acetaldehyde concentration and oxidative stress using mice, respectively.

| | |
|---|---|
| Implementation Under Test: | Non-administered, SUPALIV (30~60 mg/kg) forced oral administration. |
| Alcohol (EtOH) concentration: | 7 g/kg (specific gravity calculation 5.6 g/kg). Dilute to 500 µl with distilled water and administered intraperitoneally. |
| Experimental Method A: | Changes in blood acetaldehyde concentration:<br>Mice were given SUPALIV (60 mg/kg) and EtOH 30 minutes later. Blood samples were drawn twice, once at 2 hours and once at 24 hours after EtOH administration, and the results were analyzed by BML Co. |
| Experimental Method B. | Change in oxidative stress level:<br>Mice were administered SUPALIV (30 mg/kg) at 24-hour intervals for 4 days. EtOH was administered 1 hour after SUPALIV administration twice on days 3 and 4. Blood samples were collected twice on days 3 and 5 after the second day of alcohol administration and measured with Wismer's FREE Carrio Duo (d-ROMs test). In both studies A and B, the effects of SUPALIV were controlled for blood concentrations in the absence of alcohol administration. |
| Evaluation Method: | Ethanol and acetaldehyde concentrations in blood. |



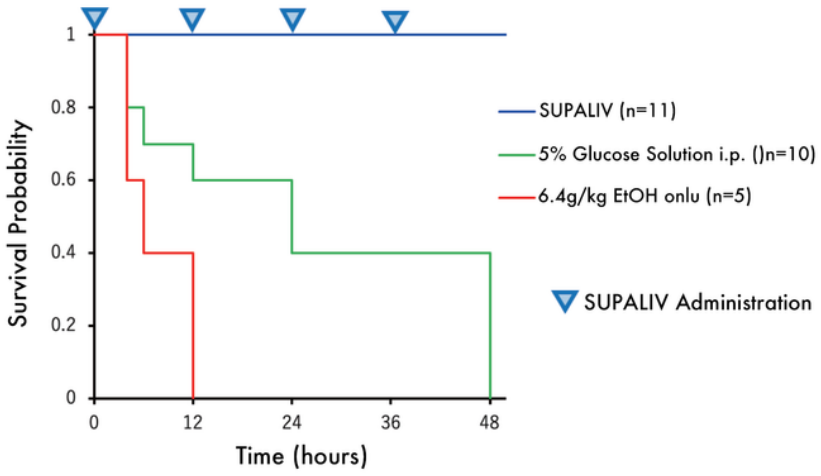

Results: Acetaldehyde concentration in the blood increased after 2 hours of EtOH administration, but almost disappeared after 24 hours, showing a level below the limit of measurement. On the other hand, the oxidative stress level did not change significantly from the first day when acetaldehyde was present in the blood to the next day when it disappeared, but the oxidative stress level increased from the third day (72 hours later) and remained high on the fifth day. In contrast, SUPALIV suppressed not only acetaldehyde metabolism but also oxidative stress better than the non-treated group.

The results of this experiment show that oxidative stress remains high for about a week after drinking a large amount of alcohol, even though the alcohol is metabolized. We often hear that people get sick after drinking too much, but since oxidative stress remains so high, it is not surprising that the immune system is weakened.

# Survival of Mouse Models of Acute Alcohol Intoxication

Acute alcohol intoxication is common during the season of welcome and farewell parties, year-end parties, and New Year's parties. This is because a large amount of alcohol enters the body in a short period of time and the body's metabolism cannot keep up, resulting in acute alcohol intoxication. The symptoms of alcohol intoxication are proportional to the level of alcohol in the blood and can lead to loss of consciousness and even death. Treatment for acute alcohol intoxication is limited to gastric lavage, intravenous fluids, and hemodialysis. If dialysis is delayed or not started, it is not possible to save the patient's life. Therefore, we have created a mouse model of acute alcohol intoxication.
The effect of SUPALIV on acute alcohol intoxication was tested.

| | |
|---|---|
| implementation under test: | Non-administered, intraperitoneal administration of 5% glucose solution, forced oral administration of SUPALIV (60 mg/kg) |
| Alcohol (EtOH) Concentration: | 8 g/kg (specific gravity calculated 6.4 g/kg). Diluted in distilled water to 500 µl and administered intraperitoneally. |
| Experimental Procedure: | Mice were treated with 500 µl of 5% glucose solution or SUPALIV, followed 30 minutes later by EtOH. SUPALIV was also administered 12, 24, and 36 hours after EtOH administration, and the survival of all mice was subsequently observed. The effect of SUPALIV was compared by the difference in survival rate between the non-treated group and the 5% glucose solution group. |
| Evaluation Method: | Survival |

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## Alcohol Metabolic Effects in Humans (Clinical Study)

In 2008, a clinical trial of SUPALIV on alcohol metabolism was contracted to Allegro, Inc.

| | |
|---|---|
| Test Subject: | 10 males each with normal and partially deficient ALDH2 gene. |
| Alcohol: | The type of wine was red wine (alcohol content: 13.5°), and the loading dose was 0.70 mg/kg of ethyl alcohol equivalent for ALDH2-normal subjects and 0.35 mg/kg for partially deficient subjects. |
| Test Procedure: | SUPALIV was ingested with 100 ml of water 20 minutes before the start of red wine loading, followed by ingestion of one-third of the prescribed amount of red wine every 10 minutes for 30 minutes. Blood samples were drawn 4 times at 30, 60, 90, and 120 minutes after the start of red wine consumption. All food and beverages on the day of the study were standardized from 9:00 p.m. the night before. The red wine load test was conducted under the SUPALIV administration in the first period, and the red wine load test without SUPALIV administration was conducted in the second period after a one-week interval. |

Test results with dynamic parameters of Supaliv

Blood Acetaldehyde Concentration

| | | No Supaliv Intake | | | Supaliv Intake | | | Test |
|---|---|---|---|---|---|---|---|---|
| | Parameter | n | Average | Standard | n | Average | Standard | |
| Overall | Cmax | 20 | 13.1 | 9.6 | 20 | 7.6 | 4.4 | 0.0005 |
| | Tmax | 20 | 55.5 | 32.7 | 20 | 114.0 | 20.9 | 0.0000 |
| | AUC | 20 | 1093.5 | 778.4 | 20 | 509.2 | 400.7 | 0.0000 |
| Normal | Cmax | 10 | 7.1 | 3.0 | 10 | 4.3 | 0.7 | 0.0082 |
| | Tmax | 10 | 63.0 | 35.9 | 10 | 120.0 | 0.0 | 0.0007 |
| | AUC | 10 | 537.4 | 112.4 | 10 | 241.9 | 42.2 | 0.0000 |
| Defect | Cmax | 10 | 19.1 | 10.3 | 10 | 10.8 | 4.1 | 0.0055 |
| | Tmax | 10 | 48.0 | 29.0 | 10 | 108.0 | 29.0 | 0.0004 |
| | AUC | 10 | 1649.7 | 761.0 | 10 | 776.5 | 422.4 | 0.0002 |

Cmax: Maximum blood concentration of measured species

Tmax: Time to reach peak blood concentration

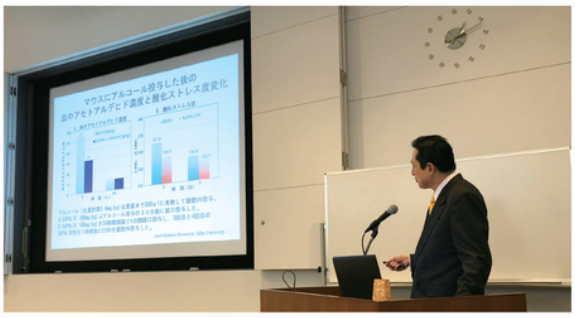
Conclusion: When wine, which is believed to contain many impurities that interfere with alcohol metabolism, is consumed, the blood acetaldehyde concentration tends to increase even 120 minutes after drinking without taking SUPALIV, and alcohol metabolism is not accelerated. However, when SUPALIV was taken, the metabolism of alcohol was accelerated, as confirmed by the analysis of Cmax and Tmax. However, SUPALIV did not promote the metabolism of alcohol, but it did promote the metabolism of alcohol when SUPALIV was administered.

## Conference Presentation

### International Conferences

1. October 2020, The 22nd World Conference on Oxidative Stress Reduction, Redox Homeostasis & Antioxidants.
**"COVID-19 infection is oxidative stress disease. Twendee will be the best solution to prevent and avoid severe symptoms."**

2. October 2020, The 22nd World Conference on Oxidative Stress Reduction, Redox Homeostasis & Antioxidants.
**"Does continuous OS reduction prevent and ameliorate through species diversity of intestinal bacteria?"**



### Domestic Academic Societies

| | |
|---|---|
| 1. June 2021 | The 10th Annual Meeting of the Japanese Society for Dementia Prevention "Cutting Edge of Dementia Prevention" |
| 2. September 2020 | 61st Annual Meeting of the Japan Neurological Society, "Antioxidant Therapy of Neurological Diseases -Twendee X Infinite Potential-" |
| 3. October 2019 | 1st Annual Japanese Brain Supplement Conference, "Antioxidant Supplement Twendee X Clinical Effects" |
| 4. October 2019 | 1st Annual Japanese Brain Supplements Conference, "Does the Antioxidant Supplement Twendee X Act as a Body Regulator?" |
| 5. October 2019 | 9th Annual Meeting of the Japanese Society for the Prevention of Dementia, "Action of Twendee X in the Cranial Nerves." |

### Education/Seminars, etc.

| | |
|---|---|
| October 2020 | 2nd Annual Meeting of the Japanese Society for Brain Supplements "Antioxidant Supplement TwendeeX for Dementia Prevention" |
| November 2019 | Anti-Aging Fair in Okayama "Suppressing Oxidative Stress for Health and Aim for Longevity" |
| September 2019 | 2nd Antioxidant Summit - Dementia and Healthy Longevity - . Public Seminar, Tokyo, Japan |
| September 2019 | Oxidative Stress and Disease. Special Lecture. |
| August 2019 | 2nd Antioxidant Summit - Dementia and Healthy Longevity - . Public Seminar, Osaka, Japan |
| January 2019 | Life Span and Oxidative Stress. Antioxidant Research Public Seminar - Healthy Longevity and Allergic Disease. |

### Paper Presentation

Yang, F., Tanaka, S., Marcus Matuska-Greifenclau, T. Yoshikawa, N. Okada, and H. Inufusa. The World's First Antioxidant for the Prevention of Dementia -Antioxidant Twendee X for a Super-aging Society-. BIO Clinica Vol. 35 No. 10 Sep. 2020.

Koh Tadokoro, Yasuyuki Ohta, Haruhiko Inufusa, Alan Foo Nyuk Loon, Koji Abe. Prevention of Cognitive Decline in Alzheimer's Disease by Novel Int J Mol Sci. 2020 Mar; 21(6)

Yang, F., Tanaka, S., Marcus Matuska-Greifenclau, T. Yoshikawa, N. Okada, and H. Inufusa. World's First Achievement of Dementia Prevention with Twendee X, a Combination Product of the Antioxidant Research Division. Medical Science Digest, Vol 45(13), 2019.11.

Yang, F., Tanaka, S., Marcus Matuska-Greifenclau, T. Yoshikawa, N. Okada, and H. Inufusa. The world's first achievement in dementia prevention - Relationship between oxidative stress, inflammation and immunity. BIO Clinica Vol. 35 No. 4 Apr. 2020.

# About our laboratory

## Profile

### Haruhiko Inufusa

Chief Researcher, Antioxidant Research Laboratory, Louis Pasteur Medical Research Center/
Specially Appointed Professor, Antioxidant Research Division, Center for Scientific Research Infrastructure, Gifu University

Graduated from Kinki University School of Medicine in 1982, and received his M.D. in 1988 from the Department of Surgery, Graduate School of Medicine, Kinki University. D. in 1988 from the Department of Surgery, Graduate School of Medicine, Kinki University, specializing in gastrointestinal surgery, laparoscopic surgery, and research on cancer metastasis. After retiring from Kinki University, he started research on alcohol metabolism, glucose and lipid metabolism, and oxidative stress as a chief researcher at TIMA establishment in 2007. In 2013, he was appointed as a specially-appointed professor at the establishment of the Antioxidant Research Division, Joint Research Department, Center for Scientific Research Infrastructure, Gifu University. Currently, he is working on oxidative stress and anti-oxidative agents.

Since March 2020, he has been a chief researcher at the Antioxidant Research Laboratory of the Louis Pasteur Medical Research Center, a public interest incorporated foundation.

Videos are available on the Antioxidant Channel on YouTube

## Advisor

Toshikazu Yoshikawa (President, Louis Pasteur Medical Research Center, Honorary President, Japanese Society of Oxidative Stress, Honorary President, Japanese Society of Anti-Aging Medicine ) / Taku Nakajima (Assistant Professor, Graduate School of Molecular Medicine, Hiroshima University ) / Helmut Durschlag (PhD, Institute of Biophysics and Physical Biochemistry, Regensburg University ) / Christian Amatore ( Member of the French Academy of Sciences, Institut Pasteur, France ) / Naomi Okada (Director, Naomi Clinic) / Kaori Tanaka (Professor, Department of Anaerobic Bacteriology, Gifu University) / Koji Fukui (Professor, Laboratory of Molecular Cell Biology, Department of Life Sciences, Faculty of Systems Science and Engineering, Shibaura Institute of Technology )

## Contact

For joint research, interviews, etc., please contact us by e-mail.
We also welcome questions from the general public regarding our antioxidant research.

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https://www1.gifu-u.ac.jp/~lsrc/staff/staff_anti.html

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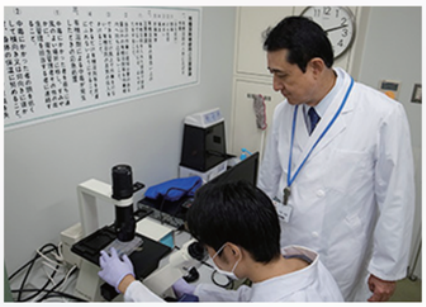
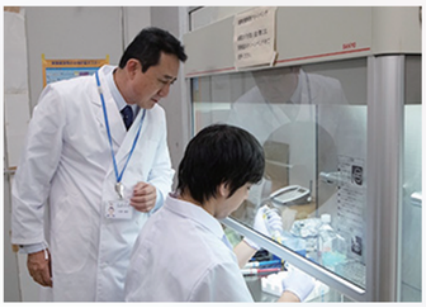
Office Manager: Toshikazu Yoshikawa (M.D., M.PhD.) / Chief Researcher: Haruhiko Inufusa (M.D., M.PhD.)





Produced September 2022