

## **Effectiveness of Pentavalent Vanadium Ion Water in Treating Alzheimer's-Type Cognitive Disease (2)**

### **1 Introduction**

For Alzheimer's Disease (AD), in addition to providing a constant supply of  $\text{Ca}^{2+}$  ions to nerve cells explained generally in "Effectiveness of Pentavalent Vanadium Ion Water in Treating Alzheimer's-Type Cognitive Disease (1)", pentavalent vanadium Ion Water ( $\text{V}^{5+}\text{H}_2\text{O}$ ) is thought to greatly improve the symptoms of AD by treating diabetic hyperinsulinemia and suppressing progression of AD through increased HDL cholesterol. These 3 effects may provide a scientific explanation for reports of a water supplier experiencing improvements in AD through vanadium Ion Water uptake, I encountered by chance 14.5 years ago.

### **2 Hyperinsulinemia and AD**

(1) The correlation between diabetes and AD is becoming clear through the results of many recent studies. Some studies report a 4.6-fold higher rate of AD onset in elderly diabetic patients compared to control groups. The rapid rise in geriatric diabetes and AD makes the correlation between the two conditions undeniable.

The basis for the onset of diabetes accompanied by advanced age is characterized by reduced ability to secrete insulin with age and obesity as well as reduced insulin sensitivity. Even now, many physicians hastily associate diabetes with insulin insufficiency and over-prescribe drugs that promote insulin secretion, resulting in inappropriate treatment, etc. Many such cases not only produce zero therapeutic benefit, but also worsen diabetes. This results in many diabetic patients developing hyperinsulinemia and invites a host of other severe conditions. Representative conditions include hypertension, atherosclerosis of blood vessel endothelia, and other circulatory system disease in addition to AD.

(2) Hyperinsulinemia leads to excess insulin being used in vascular endothelium, hardening the vessels and reducing the ability for them to expand. This process is thought to result in impaired blood flow, negatively impact the entire brain (which demands a large amount of oxygen), and speed the progression of AD.

(3) Insulin delivers glucose signaling for sugar metabolism. Once that role has

been fulfilled, it is broken down by insulin-degrading enzyme and eliminated in the blood. Insulin-degrading enzyme possess the capability to break down other unnecessary products as well, can degrade the amyloid  $\beta$  protein ( $A\beta$ ) that is leaked from nerve cells, and is central to the development of AD. As diabetes progresses, blood insulin concentrations become continually elevated, resulting in monopolization of insulin-degrading enzyme by insulin degradation and lessening the degradation of  $A\beta$ . The amount of  $A\beta$  deposition increases, and AD subsequently progresses.

- (4)  $V^{5+}H_2O$  has been shown in several past clinical studies to drastically improve symptoms in patients with hyperlipidemic insulin resistance that progresses into diabetes (more than 50% of all patients) upon 1 to 3 months of administration (1-2L/day).

(Refer to attached “Wonders of Vanadium”)

$V^{5+}H_2O$  normalizes blood sugar levels and alleviates hyperinsulinemia in patients with hyperlipidemic diabetes. Furthermore, AD is improved through the promotion of  $A\beta$  degradation and increased blood flow resulting from healthier blood vessels.

### 3 HDL Cholesterol and AD

- (1) Currently, there is little knowledge of cholesterol metabolism as it occurs within the organ richest in cholesterol: the brain (central nervous system). However, recent studies have shown us that it is related to AD in many ways. The central nervous system is separated from the general circulatory system by the blood-brain barrier, and is therefore thought to have an independent cholesterol metabolization system. Moreover, brain cholesterol is limited to only the HDL cholesterol that can pass the blood-brain barrier. In light of this AD connection, multiple reports indicate a decreased amount of serum HDL cholesterol and brain HDL cholesterol in AD patients. There are also reports of low serum HDL cholesterol (and possibly low brain HDL cholesterol given the HDL formation mechanism) as a risk factor for the progression of AD.
- (2) HDL cholesterol also collects deposited  $A\beta$ , meaning a reduction in HDL can

promote the progression of AD.

- (3) A sufficient quantity of HDL cholesterol is necessary to maintain the plasticity of synapses, which plays an important role in information transmission. Accordingly, we have come to learn that this required quantity is produced by cholesterol metabolism mechanisms that take place near the intracranial terminus.
- (4)  $V^{5+}H_2O$ , through the Aoki Hypothesis, has demonstrated a mechanism of action as well as clinical trial results (refer to attached “Wonders of Vanadium”). According to this, almost all people have elevated HDL cholesterol (by an average of 11%). The HDL cholesterol that plays a major role in the onset and progression of AD can be raised with  $V^{5+}H_2O$ .

It is thought that these properties of  $V^{5+}H_2O$  also offset the onset and improve the symptoms of AD.

#### 4 Conclusion

- (1) Kin and Gin, the nationally popular sisters that came to popularity through television, kept a sharp wit and did not show signs of age-related cognitive decline even after passing 100 years of age. When a group of interested researchers autopsied the sisters’ brains after death, to the research group’s surprise, they found tightly-packed deposits of  $A\beta$ , the fundamental cause of AD. Thinking this strange, the research group investigated the sisters’ eating habits when they were alive and found that both Kin and Gin ate a lot of blue-skin fish rich in DHA and EPA every day. This left the question of whether DHA and EPA possess some action that suppresses  $A\beta$  deposition and AD onset. This, combined with 13 consecutive failures in AD drug development carried out based on the basis of the  $A\beta$  theory, strengthened the case for an explanation other than  $A\beta$  theory in regards to the true onset of AD.
- (2) Although only observed in 3 cases, we think the fact that  $V^{5+}H_2O$  drastically improves AD is significant. Moreover,  $V^{5+}H_2O$  is theorized to possess physiological functions that produce various results of AD onset suppression and symptom improvement by (i) constant  $Ca^{2+}$  supply to the central intracranium, (ii) alleviation of diabetic hyperinsulinemia, and (iii) HDL

cholesterol elevation.

- (4) In addition, another physiological function of  $V^{5+}H_2O$  is the unique trait of promoting fat catabolism ( $V^{5+}H_2O$  increases nuclear and endoplasmic reticular  $Ca^{2+}$  concentrations, activating peroxisomal proliferator response elements, PPARs, thereby promoting an increase of ultra-long fatty acid chains and other fatty acid catabolism activity). Including the case of Kin and Gin's DHA and EPA consumption, the sense that the link between fat metabolism and AD holds some great key persists to this day despite an exceedingly small amount of knowledge that prevents theorization. Conducting detailed clinical studies of the effects and abilities of  $V^{5+}H_2O$  on AD will clarify our overall understanding of the onset and progression of AD. Moreover, such studies may elucidate the unique pharmacological action of  $V^{5+}H_2O$  on AD.

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#### References

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