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Review Article

Thirst sensation and oral dryness following alcohol intake

Kiyotoshi Inenaga*, Kentaro Ono, Suzuro Hitomi, Ayu Kuroki, Izumi Ujihara[1]

Division of Physiology, Kyushu Dental University, 2-6-1 Manazuru, Kokurakita, Kitakyushu 803-8580, Japan

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Summary Substantial acute and chronic intakes of alcohol or ethanol (EtOH) severely influence oral sensations, such as thirst and oral dryness (dry mouth, xerostomia). Thirst sensation and oral dryness are primarily caused by the activation of neurons in brain regions, including the circumventricular organs and hypothalamus, which are referred to as the dipsogenic center, and by a decrease in salivary secretion, respectively. The sensation of thirst experienced after heavy-alcohol drinking is widely regarded as a consequence of EtOH-induced diuresis; however, EtOH in high doses induces anti-diuresis. Recently, it has been proposed that the ethanol metabolite acetaldehyde induces thirst via two distinct processes in the central nervous system from EtOH-induced diuresis, based on the results of animal experiments. The present review describes new insights regarding the induction mechanism of thirst sensation and oral dryness after drinking alcohol.

Contents

* Corresponding author at: Division of Physiology, Kyushu Dental University, 2-6-1 Manazuru, Kokurakitaku, Kitakyushu, Fukuoka, 803-8580, Japan. Fax: +81 93 582 8288.
E-mail address: inenaga2211@yahoo.co.jp (K. Inenaga).
[1] Present address: 3-6-3, Hanatachibana, Shingu, Kasuya, Fukuoka, 811-0103, Japan.

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1. Introduction

Thirst sensation is not only induced by an increase in plasma osmolality and/or a decrease in body fluid volume but also neurally and humorally related neurotransmitters, hormones and cytokines [1,2]. The thirst sensation induced by increased plasma osmolality causes water intake, whereas the thirst sensation induced by a decreased body fluid volume results in both water and salt intakes. To induce this thirst sensation, it is important to activate neurons in the circumventricular organs (CVOs), including the organum vasculosum of the lamina terminalis (OVLT) and the subfornical organ (SFO), and the hypothalamus, which are referred to as the dipsogenic center. Oral dryness, which comprises a feeling of dryness in the oral cavity, is produced by a decrease in salivary secretion [3—5] and is distinguished from thirst sensation [6—9]. In the present review, thirst sensation and oral dryness after alcohol drinking or administration are differentiated.

It is widely believed that the thirst sensation after acute alcohol intake may be attributed to a decrease in the body fluid volume via an alcohol- or ethanol (EtOH)-induced diuresis [10,11]. This hypothesis is supported by findings that EtOH reduces vasopressin (AVP) release from the nerve terminals of the posterior pituitary, which results in increased urine formation [12,13]. Low doses of EtOH induce diuresis; however, the urine volume is decreased rather than increased by substantial doses in animal experiments [14—16]. In addition, alcohol intake that is sufficient to induce a hangover in humans causes diuresis immediately afterwards and gradually shifts to anti-diuresis [17]. In the condition referred to as a hangover, in which individuals experience nausea, vomiting and dizziness, as well as thirst, the former symptoms are thought to be elicited by acetaldehyde, which comprises a metabolite of EtOH and a toxic substance [18]. Acetaldehyde is also considered to have an important key role in alcohol addiction [19]. Recently, it has been reported that acetaldehyde elicits the intake of water and salt without diuresis [16]. Moreover, a study has demonstrated that acetaldehyde has no effect on AVP release from the posterior pituitary [20]. Thus, the hypothesis of ''EtOH-induced diuresis'' must be reconsidered.

In addition to acute alcohol intake, chronic alcohol intake induces thirst sensation [21]. Acute [22] and chronic alcohol intake [23—25] also induces hyposalivation, which is a cause of oral dryness. There are many unknown points. The purpose of this review is to provide new insights regarding the induction mechanism of thirst sensation and oral dryness following acute and chronic alcohol intake, with a focus on the involvement of EtOH and acetaldehyde and their effects on the dipsogenic center in the brain and salivary secretion.

2. Heavy-alcohol induces thirst sensation: can it be explained by EtOH-induced diuresis?

On the subsequent morning after heavy-alcohol drinking, many individuals experience thirst sensation and oral dryness as well as other unpleasant feelings [18,26]. It is widely believed that the thirst sensation induced by alcohol drinking causes alcohol-induced diuresis [12]. This idea is based on a suppression of AVP release from the posterior pituitary [27] and a decrease in plasma AVP [12] by EtOH. EtOH inhibits calcium currents in neurosecretory neurons in the hypothalamus [28] and the terminals of the posterior pituitary [10,11,29], and it potentiates voltage-gated potassium channels [30]. Carney et al. have reported that EtOH-induced diuresis is not a result of the inhibition of AVP secretion; instead, it results from an alteration of AVP-induced water permeability within the proximal tubule in the kidney [31]. In the case of relatively heavy-alcohol drinking or administration, which may cause a hangover, the urine volume is decreased with an increase in plasma AVP or remains unchanged [14—16,21,32]. One study indicates the biphasic responses of early alcohol-induced diuresis and late anti-diuresis following alcohol drinking in humans [17]. Immunocytochemical studies indicate an increase in c-Fos immuno-positive neurons in the supraoptic (SON) and paraventricular nuclei (PVN) of the hypothalamus, which include AVP neurons, following EtOH administration [33—35]. A recent study has reported that the AVP-enhanced green fluorescent protein (eGFP) expression levels were increased in the SON and PVN but decreased in the posterior pituitary in transgenic rats, which suggests that AVP was released from the posterior pituitary by EtOH administration [16]. To date, there is no decisive conclusion regarding whether EtOH elicits diuresis or anti-diuresis. However, it is clear that EtOH-induced diuresis is not always the cause of thirst sensation following heavy-alcohol drinking.

3. Acetaldehyde induces thirst sensation

3.1. Activation of renin—angiotensin system

Following ingestion and absorption, EtOH is metabolized into acetaldehyde via the enzymes alcohol dehydrogenase

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