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The effects of real and placebo alcohol on deaffrication

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Abstract

The more alcohol a person has consumed, the more mispronunciations occur. This study investigates how deaffrication surfaces in Bernese Swiss German when speakers are moderately intoxicated (0.05-0.08 % Vol.), whether these effects can be hidden, and whether a placebo effect interacting with mispronunciation occurs. Five participants reading a text were recorded as follows. In stage I, they read the text before and after drinking placebo alcohol, and finally again after being told to enunciate very clearly. 3-7 days later, the same experiment was repeated with real alcohol. The recordings were then analysed with *Praat*. Despite interspeaker variation, the following generalisations can be made. The most deaffrication occurs in the C_C context both when speakers are sober and inebriated; affricates in _#, V_C, and V_V position encounter more deaffrication in the alcohol stage; and /t͡ʃ/ and k͡x are deaffricated more when the speaker is intoxicated, with /t͡ʃ/ being the most susceptible to mispronunciation. Moreover, when alcohol is consumed, more deaffrication occurs, which cannot consciously be controlled. Furthermore, a statistically significant difference between the pre- and the post-placebo-drinking experiment could be found, which implies that a placebo effect takes place. Nevertheless, the effects of real alcohol are considerably stronger.

Index Terms: forensic phonetics, drunk speech, deaffrication, placebo effect

1. Introduction

The ways in which alcohol (i.e. ethanol) influences speech are numerous; they can be lexical/pragmatic, segmental, or suprasegmental [1-4]. Thus, the present study's focus has been narrowed down to deaffrication as it is one of the most salient features of drunk speech. Before starting, however, the question as to why it is important to conduct research on this topic must be addressed. Knowing how a common substance like alcohol affects speech is of great value, especially in a forensic setting. When *Exxon Valdez*, a U.S. oil tanker, grounded in March 1989 spilling over 41 million litres of crude oil in the Pacific Ocean, for instance, forensic phoneticians provided evidence that its captain had been intoxicated before running the ship aground by examining and contrasting the emergency call recordings and the television interview the captain gave some 30h later [1, pp. 284-312]. The aim of this study is, however, not only to investigate how deaffrication surfaces in Bernese, but also whether other factors exist that influence the speech of an inebriated speaker. The study hence includes placebo alcohol in the design to explore this question.

Three questions guided the research. To see the general effects of alcohol, research question 1 has been created:

- How does alcohol-related deaffrication surface?

This question will focus on the phonological environment (PE) and the place of articulation (PoA) of the respective affricates. Moreover, to check whether speakers' expectations influence their speech, the 2nd research question has been formulated, which is linked to the underlying 3rd research question:

- Can alcohol-related effects be counteracted when trying to enunciate as clearly as possible?
- Does a placebo effect regarding deaffrication take place?

2. Literature summary

2.1. Bernese Swiss German

Bernese Swiss German, spoken in western Switzerland in and around the canton of Bern, has four affricates in its phoneme inventory that can be affected by deaffrication: /p͡f/, /t͡s/, /t͡ʃ/, and /k͡χ/ [5, pp. 33-34]. These can occur word-initially (#_), word-finally (_#), interconsonantally (C_C), between a vowel and a consonant (V_C; C_V), and intervocalically (V_V).

2.2. Alcohol's mechanism of action

Alcohol can have a negative influence on speech performance even at a small dosage. As speaking involves a highly coordinated system of muscle control, visual stimuli, and cognitive functions, it is little surprising that errors in pronunciation occur when alcohol interferes with this complex interplay [6, 7]. This part will briefly describe the articulation process and will outline the effects of alcohol on the body and brain.

Articulation in the mouth happens at a velocity of 5 to 20 cm/s, and occasionally, if movement occurs very fast as e.g. in the case of consonant clusters, even at 30 cm/s [8]. Despite this speed, it is quite uncommon for articulators, specifically active ones, to move more than 1 cm. Movements of 1.5 cm can happen, but they are relatively rare. At the same time, the articulator placements are very precise, varying less than 1 mm for certain vowels [9, 10]. Over 100 muscles need to be controlled and coordinated at a speed of 14 phones per second for the tongue alone [11]. These muscles are organized in a 3D structure, i.e. there are longitudinal, vertical, and transverse muscle fibres, allowing for movements in all directions. Insufficient control of these muscles, provoked by e.g. alcohol, inevitably leads to pronunciation errors. The next paragraph will elucidate why this is the case.

Ethanol has sedative effects on the central nervous system [1, p. 18] due to it changing the way in which neuronal-specific proteins behave. Most importantly, it affects gamma-aminobutyric acid (GABA), which is responsible for muscle tone, in that "there is increased activity of the neuronal chloride ion channel that is linked to the A-type GABA

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receptor ($GABA_A$)" [1, pp. 18-19]. This higher permeability for chloride ions results in a hyperpolarization, i.e. the cells become more negative, making it harder for them to fire, which results in a lower frequency of action potentials. As these are the neurons' only way of communication, less information can be conveyed. Applied to muscle control, this phenomenon is responsible for the anxiolytic and sedative effects as motor neurons excite the muscles to which they are attached less frequently, rendering them relaxed and hence impeding their accurate control [12].

The part of the brain directly linked to coordination, motor control and hence also language production, is the cerebellum [13]. It contains a great number of a specific kind of neuron, namely Purkinje cells, which are sensitive to ethanol since they use GABA as a neurotransmitter [14, 15]. If due to a higher level of intoxication less GABA is available for communication between cells, motor control becomes increasingly difficult. These effects of alcohol surface as follows. "Between 0.03 and 0.05%, judgement and visual acuity are impaired. Between 0.05 to 0.08%, muscle control is affected, as evidenced by increased [reaction time] and mild incoordination. As [blood alcohol level] rises, there is an exaggeration of these effects, as well as new ones. Around 0.10%, … incoordination becomes more prominent" [16, p. 22]. Therefore, pronunciation errors should be observable from a blood alcohol level of 0.05 % Vol.

2.3. Interspeaker variation in the context of alcohol

Previous research has reported high amount of interspeaker variation regarding the effects of alcohol apart from those considering the different vocal tract and glottis shapes as well as the speakers' psychological and physical state, i.e. stress, emotions, hunger or thirst [17, 18]. This section will attempt to link these to the placebo effect, stating that the variation is due to speakers' different mental representations of how alcohol influences speech.

If an action has pleasant consequences, it will happen more often, and if it has unpleasant consequences, its frequency will decrease. In other words, an individual learns how to react to a certain stimulus due to social reinforcement and how it is evaluated. Alcohol can alleviate pain, diminish social or general fear, and foster sexual arousal, which are all positive consequences and are associated with good experiences. It can therefore be inferred that for a specific individual, alcohol provokes positive feelings, actions or states that have occurred before, so called "response expectancies" [19, p. 1189]. These, however, can differ from person to person as they are dependent on previous experiences. If the alcohol is secretly substituted, some drinkers will still show that behaviour due to the expected positive effects. This would then be called 'placebo effect' as its consumers know of these desired effects, and they will hence show the associated behaviour more frequently [20].

A placebo effect certainly occurs as well when real alcohol is consumed. Kirsch states that "[p]lacebo and drug effects are widely assumed to be additive" [19, p. 1192]. In other words, the total effect of a drug it the sum of both the drug's ingredients plus the expected effect of these ingredients. Alcohol can therefore be regarded as an amplifier of the "response expectancies", contributing to interspeaker variation.

3. Methodology

3.1. Participants

Five Bernese students (age: 20-24) have been recruited for this study (2 male/3 female). They drink alcohol on a rare to fairly regular basis (1-3 standard drinks per instance; 1-3 times a week). None of them were paid to participate, but they were given free drinks. They had been explicitly told not to assimilate any food for 4-6 hours prior to the experiments in order not to skew the alcohol digestion. None of the female participants were pregnant, and no one stood under any kind of medication nor had reported an alcohol addiction.

3.2. Testing procedure

The experiment was conducted in two stages, separated by 3-7 days. Stage I involved a placebo (non-alcoholic beer labelled with 4.8 % Vol. beer labels), stage II real alcohol (4.8 % Vol. beer). To make it harder for the participants to detect the placebo, spicy wasabi nuts were served before the test, and the beer was cooled down to 3°C. In each stage, the participants were asked to read a text containing 121 affricates once before drinking (PreDE), and twice after drinking (post-drinking (PDE)), whereas in the last round they were told to imagine being controlled by the police and hence trying to hide their alcohol consumption (henceforth referred to as post-post drinking experiment (PPDE)). They were given time to familiarise themselves with the text before each recording to reduce possible pronunciation errors due to a lack of training. The recordings took place in a quiet furnished room at the University of Bern. As a recording device, the Zoom H2n was used in unidirectional microphone mode that records in a 90°-angle in stereo at a sampling rate of 96 kHz and a 24-bit sample size. It was placed on a piece of cloth about 50 cm in front of the subjects. The sessions lasted between 55 to 150 minutes. It was attempted to make the experiment phase as short as possible to reduce the risk of fatigue, which could distort the results [4, p. 554]. After the last experiment, the participants were debriefed; the purpose of the experiment was elucidated, and the participants were asked to give a short feedback. None of them reported that they knew that non-alcoholic beer had been served in stage I.

After drinking and after the last recording of each stage, the breath alcohol level (BrAL) of the participants was measured with a Breathalyzer (Dräger Alcotest 6820). As it is required by law to have a tolerance value of 10% in Switzerland, the actual BrAL was calculated by adding 10% of the BrAL to the displayed value. This has been proven to be reliable, and as accurate as blood or urine tests [4, p.554]. Prior to measuring, the participants rinsed their mouths thoroughly to avoid any residual alcohol skewing the analysis. During the placebo stage, the participants were told an imaginary number and were not granted a glimpse on the display. The results of the stage II are summarised in table 1.

Table 1: *Breath alcohol level in % Vol.*

	Before PDE-II	**After PPDE-II**
Subject 1	0.077	0.074
Subject 2	0.062	0.070
Subject 3	0.064	0.062
Subject 4	0.036	0.029
Subject 5	0.072	0.069
Mean	**0.062**	**0.061**

Subject 4 did not reach the threshold of 0.05% Vol. However, pronunciation errors occurred even at this stage, which is why no exclusion from the analysis resulted.

3.3. Analysis

The recordings were then carefully examined in *Praat* [21]. First, they were played twice in their normal speed while simultaneously noting down suspicious-sounding affricates. Subsequently the speed was slowed down threefold to hear whether a pre-burst closure took place. Once all the ill-pronounced affricates had been identified, the waveform and the spectrogram were inspected. If no clear occlusion phase, burst, and release phase were visible, the affricates were considered deaffricated. For the statistical analysis, McNemar's χ^2 tests with continuity correction were applied.

4. Results

Table 2 summarises the deaffrication rates in the alcohol stage (II) by PE. C_C affricates experience deaffrication the most, both when the speakers are sober and inebriated. On the other hand, V_V affricates are pronounced the most reliably when sober, and #_ ones when drunk.

Table 2: *Mean deaffrication rates (with p-value and number of tokens per experiment) by PE.*

	Tokens/Exp	PreDE-II	PDE-II	p
#_	235	1.3%	3.0%	n. s.
_#	75	4.0%	13.3%	0.0455
C_C	20	25.0%	30.0%	n. s.
C_V	20	10.0%	10.0%	n. s.
V_C	130	6.9%	25.4%	0.0001
V_V	125	0.8%	6.4%	0.0455

The deaffrication increase of the _# ($\chi^2(1) = 4.0$, $p = 0.0455$), the V_C ($\chi^2(1) = 17.633$, $p = 0.0001$) and the V_V ($\chi^2(1) = 4.0$, $p = 0.0455$) affricates are statistically significant.

Stage II's deaffrication rates by PoA can be found in table 3. Without alcohol, p͡f is pronounced most reliably, whereas the other three are mispronounced at rates between 4.0-4.4%. When inebriated, t͡ʃ was deaffricated the most often.

Table 3: *Mean deaffrication rates (with p-value and number of tokens per experiment) by PoA.*

	Tokens/Exp	PreDE-II	PDE-II	p
p͡f	90	1.1%	7.8%	n. s.
t͡s	280	4.3%	7.1%	n. s.
t͡ʃ	100	4.0%	23.0%	0.0001
k͡x	135	4.4%	11.9%	0.0339

The deaffrication increases of t͡ʃ ($\chi^2(1) = 15.429$, $p = 0.0001$) and k͡x ($\chi^2(1) = 4.5$, $p = 0.0339$) are statistically significant.

The mean deaffrication rates are summarised in table 4.

Table 4: *Mean deaffrication rates (605 tokens per experiment).*

	Placebo (I)	Alcohol (II)
PreDE	3.5%	3.8%
PDE	5.8%	10.9%
PPDE	4.8%	9.9%

The differences between PreDE-I and PDE-I ($\chi^2(1) = 4.024$, $p = 0.0449$), PreDE-II and PDE-II ($\chi^2(1) = 27.138$, $p < 0.0001$), and PDE-I and PDE-II ($\chi^2(1) = 12.0$, $p = 0.0005$) are statistically significant. The changes in the deaffrication rate between PDE-II and PPDE-II, however, are not ($\chi^2(1) = 0.391$, $p = 0.532$).

Interspeaker variation, as reported in previous studies, has also been found to some extent though less than anticipated based on the literature review, which might be due to the limited number of participants. Whilst four subjects behaved in a similar pattern throughout the experiment, subject 3 consistently made comparatively few errors both when sober and intoxicated. This can be explained by the subject trying to perform well and thereby monitoring his/her speech more than the rest, who used a more casual style.

5. Discussion

5.1. How does alcohol-related deaffrication surface?

To answer this question, the results of PreDE-II were compared to the ones of PDE-II (see table 4). First, the different PE are discussed, followed by the PoA.

The best indicator for an auditory assessment of a speaker's intoxication level is listening to V_C affricates. They are articulated the worst under the influence of alcohol. In fact, they experience the greatest increase in deaffrication, which is supported by their p-value of 0.0001. Less reliable indicators are _# and V_V affricates, whose deaffrication rates in the inebriated state were just statistically significant. Although C_C affricates are the most difficult to pronounce when speakers are inebriated, they experience only a little less deaffrication when speakers are sober. Therefore, they do not allow for a valid conclusion about a speaker's intoxication level by auditory assessment. Interestingly, the environment that experienced the least deaffrication in this study, i.e. #_, had been mispronounced the most in Lester and Skousen's study [22].

A reason for C_C being hard to enunciate can be found when considering articulation. Interlinguistically, deaffrication (or spirantisation) is not an uncommon sound change [23, p. 38], implying that affricates are somewhat harder to pronounce than fricatives. When an already complex consonant is in between consonants, articulation becomes even harder. This stands in objection to our body's aim to reduce labour-intensive muscle activities to save energy and time, i.e. in the context of speech production, tongue movement [24, p. 413]. In this study, the cluster /nt͡sʂ/ has been reduced to /nsʂ/ one in four times. When the sonority hierarchy is considered, it becomes clear why this has happened. Fricatives possess a higher sonority than stops yet in the cluster, the fricative part is preceded by a stop. If deaffrication occurs, the sonority hierarchy is satisfied again, and the articulation effort is thus reduced.

A similar reason can be found for both V_C and _# affricates. In the V_C context, the consonants after the affricate are predominantly stops or sonorants, with both constellations violating the sonority hierarchy. If the affricate is deaffricated in front of a stop, the hierarchy is intact again, which increases the ease of articulation. As for following sonorants, even with deaffrication the hierarchy is violated; however, the degree to which this violation occurs is lower, which results in a less hard articulation. In the _# context, the fricative part comes after the stop part, violating the sonority

hierarchy. This phonotactically marked order is easier to master when control over the vocal tract is maintained. However, when muscle control becomes more difficult due to alcohol, the tongue will choose the articulation that demands less effort, which results in deaffricated consonant clusters that do not violate the sonority hierarchy. In conclusion, the concept of 'ease of articulation' plays a crucial role in the pronunciation of affricates as they are harder to pronounce than their fricative counterparts.

Regarding PoA, the results suggest that the best indicator for a speaker's alcohol level is t͡ʃ. Its deaffrication rate increases about six-fold after the speakers drink alcohol. A similar pattern is observed for the affricate k͡x, whose alcohol-related deaffrication rate increases about three-fold. In other words, if speakers seem to experience more problems than usual with the articulation of t͡ʃ and k͡x, it could be evidence for them being under the influence of ethanol. When the deaffrication rates for the remaining two are considered, no statistically significant differences can be observed. The deaffrication rates of the affricates p͡f and t͡s can thus not be regarded as reliable indicators for an auditory assessment of a speaker's intoxication level. Nevertheless, a tendency for more deaffrication exists as well in that in both cases the deaffrication rate is higher after the consumption of alcohol.

5.2. Can alcohol-related effects be counteracted?

Comparing PDE-II to PPDE-II (see table 4), it becomes evident that alcohol-related effects cannot be counteracted or suppressed. Although the deaffrication rate decreased by 1.0% when the speakers try to monitor their speech, it is still statistically significantly higher than in the sober state. This is not surprising as alcohol affects the body on a biochemical level that is beyond the central governance of the human mind.

5.3. Is there a placebo effect regarding deaffrication?

The answer to this question is a "yes, but". The differences between the results of PreDE-I and the ones of PDE-I were found to be just statistically significant, implying that there is a placebo effect. When the results of PDE-I are compared to the statistically significantly different results of PDE-II, however, it becomes evident that the effects of real alcohol are much stronger. Nevertheless, these two insights do not contradict each other. In stage I, the speakers' pronunciation was only influenced by the placebo effect, i.e. by what Kirsch [19] has called "response expectancy". Put differently, the speakers had an idea about how they were supposed to be sounding when drunk in their minds, which added a certain amount of deaffrication instances to their speech performance from PreDE-I to PDE-I. This was the case for stage II as well. However, the pronunciation of PDE-II was influenced not only by the speakers' mentally stored "response expectancies", but also by the bio-chemical effects of ethanol on the speakers' brains and muscles, which is an indication for the additive effects described before. This provides evidence that a placebo effect regarding intoxicated speech exists indeed.

By implication, this would mean that the placebo effect could be eradicated when the participants are asked to enunciate as clearly as possible if we assume that the effects are additive. Accordingly, the amount of deaffrication should statistically significantly decrease from PDE-II to PPDE-II. However, this was not the case despite a lower number of mispronunciations providing evidence against a placebo effect. The higher amount of deaffrication instances can arguably be accredited to fatigue phenomena. The same pattern is observable in stage I, in that the results of PreDE are better than the ones of PPDE. Although no alcohol has interfered with the speakers' muscle control, the amount of mispronunciations increased towards the end of the stage. This could at least partially account for the statistical insignificance regarding the differences between PreDE and PPDE.

Given these circumstances it is sensible to state that the speaker-specific expectations of how one sounds when intoxicated do have an influence on the pronunciation of affricates. However, a placebo effect could only be identified in one way, i.e. from PreDE-II to PDE-II, but it did not pass the inverse test, i.e. the comparison of PDE-II to PPDE-II.

It is important to understand that this study's insights regarding a placebo effect solely apply to deaffrication. Theoretically multiple placebo effects can occur at the same time, each one for a specific feature. Therefore, there might also occur one for changes in voicing or intonation patterns.

6. Conclusions

In this paper, I have explored how alcohol-related deaffrication surfaces in Bernese Swiss German, whether the effects of alcohol can be suppressed, and whether a placebo effect interfering with speech production occurs. We have seen that deaffrication takes place more often in consonant clusters (C_C and V_C) and in _# position due to violation of the sonority hierarchy. Moreover, the results showed that t͡ʃ and k͡x become statistically significantly more difficult to pronounce when inebriated. These effects cannot be hidden when articulation is monitored as alcohol relaxes the muscles, which makes it difficult to execute precise muscle movements. Finally, we have seen that a placebo effect does in fact occur. Its influence on deaffrication is, however, not as strong as the one of real alcohol. These insights make it possible to auditorily evaluate speakers regarding their intoxication status, which can be a useful tool for in situ assessments of drivers or laboratory analyses of audio files. Future research with more participants is necessary to validate the findings of this study. Additional analyses of other salient features such as voicing differences or changes in nasality might also be interesting to test for placebo effects. Furthermore, given that this study has found mispronunciations occurring mostly in complex syllables, conducting experiments with speakers of phonotactically simpler languages such as Hawaiian to see how they behave when intoxicated might add to the discussion. Research in this area is, however, not limited to ethanol. To use forensic phonetic analyses in court, it is crucial that the effects of other substances, legal or not, be investigated.

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