Intelligence 71 (2018) 66–75

Contents lists available at ScienceDirect

Intelligence

journal homepage: www.elsevier.com/locate/intell

Faster, but not smarter: An experimental analysis of the relationship between mental speed and mental abilities☆

Anna-Lena Schubert[a,*], Dirk Hagemann[a], Gidon T. Frischkorn[a], Sabine C. Herpertz[b]

[a] Institute of Psychology, Heidelberg University, Heidelberg, Germany
[b] Department of General Psychiatry, Center for Psychosocial Medicine, Heidelberg University, Heidelberg, Germany

ABSTRACT

Individual differences in the speed of information processing may contribute to individual differences in general intelligence by enhancing the efficiency of information processing. So far, this hypothesis is based on correlational data, and thus a causal relationship between mental speed and mental abilities has not yet been established. In the present study, we used transdermal nicotine administration in a double-blind design to increase the speed of information processing and tested whether this increase in information processing speed affected performance in intelligence tests. While nicotine administration decreased both reaction times and P3 latencies in the Sternberg memory scanning task, there was no effect of nicotine on intelligence test performance. These results contradict theories proposing that a greater speed of information processing causes greater intelligence. Instead, they suggest that structural properties of the brain may affect both the speed of information processing and general intelligence and may thus give rise to the well-established association between mental speed and mental abilities.

More intelligent individuals show a greater speed of information processing in numerous cognitive tasks (Sheppard & Vernon, 2008). Most intriguingly, this association can still be found between extremely simple tasks with little to no variance in response accuracies that do not rely on any kind of strategic processing. Such tasks typically require participants to react to the appearance of a stimulus as fast as possible, to classify words into categories, or to decide whether a number was included in a previously presented memory set. Due to the simplicity of these elementary cognitive tasks, individual differences in reaction times are not prone to be affected by individual differences in strategic decision making.

In a review of 172 studies published between 1955 and 2005, Sheppard and Vernon (2008) reported an average correlation of $r = -0.24$ between different measures of information processing speed in elementary cognitive tasks and cognitive abilities. Several studies have tried to describe this association in terms of mechanistic processes using mathematical models of information processing (e.g., Schmiedek, Oberauer, Wilhelm, Süss, & Wittmann, 2007; Schmitz & Wilhelm, 2016; Schubert, Hagemann, Voss, Schankin, & Bergmann, 2015; Schulz-Zhecheva, Voelkle, Beauducel, Biscaldi, & Klein, 2016; van Ravenzwaaij, Brown, & Wagenmakers, 2011), or functional neuroscience methods (e.g., Euler, McKinney, Schryver, & Okabe, 2017; Kievit et al., 2016; Penke et al., 2012; Pineda-Pardo, Martínez, Román, & Colom, 2016; Schubert, Hagemann, & Frischkorn, 2017; Troche, Houlihan, Stelmack, & Rammsayr, 2009; Troche, Indermühle, Leuthold, & Rammsayer, 2015). Although these research efforts have revealed several neural and cognitive process candidates that may give rise to the association between the speed of information processing and general intelligence, all of these studies have been correlational in nature. To overcome this problem, we experimentally manipulated the speed of information processing in the present study by transdermal nicotine administration and investigated whether this increase in information processing speed translated to fluid intelligence.

1. Proposing a causal relationship between mental speed and mental abilities

Decades of research on the relationship between the speed of information processing and general intelligence have produced several theories suggesting how a greater speed of information processing may give rise to individual differences in general intelligence. We will be shortly discussing three theoretical perspectives that try to explain this association in terms of neuro-cognitive processes: Mental speed as *g*, mental speed as an intermediate endophenotype of *g*, and mental speed as one of many overlapping processes underlying *g*.

☆ Funding: This work was supported by the Excellence Initiative of the German Research Foundation (DFG) [grant number ZUK 49/2 4.1.054].
* Corresponding author at: Institute of Psychology, Heidelberg University, Hauptstrasse 47-51, D-69117 Heidelberg, Germany.
E-mail addresses: anna-lena.schubert@psychologie.uni-heidelberg.de (A.-L. Schubert), dirk.hagemann@psychologie.uni-heidelberg.de (D. Hagemann), gidon.frischkorn@psychologie.uni-heidelberg.de (G.T. Frischkorn), sabine.herpertz@uni-heidelberg.de (S.C. Herpertz).

https://doi.org/10.1016/j.intell.2018.10.005
Received 24 January 2018; Received in revised form 11 October 2018; Accepted 11 October 2018
Available online 22 October 2018
0160-2896/

1.1. Mental speed as g

The most straight-forward way of explaining the relationship between the speed of information processing and general intelligence is that the speed of information processing *is g*. The most famous proponent of this idea was Arthur Jensen, who suggested that the speed of neural oscillations underlies individual differences in general intelligence (Jensen, 2006). Given the consistently moderate correlations between reaction times and general intelligence (Sheppard & Vernon, 2008), this theory may seem far-fetched. However, recent diffusion modeling and psychophysiological approaches have revealed that measures of mental speed and cognitive abilities share > 50% of their variance and that the moderate correlations of earlier studies may have been underestimated due to measurement problems (Schmiedek et al., 2007; Schubert et al., 2017; Schubert, Frischkorn, Hagemann, & Voss, 2016).

In particular, drift diffusion modeling approaches, which decompose reaction time distributions into distinct cognitive process parameters of a decision process, have shown that the drift rate, quantifying the velocity of and signal-to-noise ratio in evidence accumulation, is most strongly related to general intelligence and working memory capacity (e.g., Schmitz & Wilhelm, 2016; Schubert et al., 2015; van Ravenzwaaij et al., 2011). Being able to accumulate more information per time unit may facilitate the maintenance and subsequent processing of information in working memory. It has even been suggested that the age-related decline in cognitive abilities may be mainly driven by an age-related decline in the speed of information-processing that impairs performance in a wide range of cognitive tasks by limiting the quality and amount of simultaneously available information due to slower information accumulation (Salthouse, 1996).

In addition, psychophysiological research has suggested that general intelligence is strongly related to the latency of event-related potential (ERP) components associated with higher-order cognitive processes such as memory and context updating (Bazana & Stelmack, 2002; Schubert et al., 2017; Troche et al., 2009). In particular, the latency of the P3, which is a component occurring about 300 ms after stimulus onset with a maximum deflection at parietal electrode sites, has been associated with general intelligence (Bazana & Stelmack, 2002; Schubert et al., 2017; Troche et al., 2009). Shorter P3 latencies have been shown to reflect a faster inhibition of extraneous processes and may thus facilitate information transmission in working memory (Polich, 2007). Hence, faster P3 latencies may reflect a facilitation of information processing in working memory and may thus contribute to a better performance in intelligence tests by enhancing information storage and updating in working memory.

Taken together, both mathematical modeling and electrophysiological approaches studying the relationship between mental speed and general intelligence suggest that a greater speed of information processing affects the efficiency of information processing in working memory and may thus give rise to individual differences in intelligence. Hence, the speed of information processing may directly influence all kinds of cognitive processes involving working memory in a substantial way and may thus be a candidate for *g*.

1.2. Mental speed as an intermediate endophenotype of g

A different theory about the relationship between the speed of information processing and general intelligence has recently been proposed in the watershed model of fluid intelligence (Kievit et al., 2016). The model suggests that general intelligence is an observable phenotype that is affected by many small, independent genetic factors that exert their influence on general intelligence through a series of intermediate neural and cognitive endophenotypes. In detail, multiple fractional anisotropy measures of white matter integrity explained unique amounts of variance in a smaller number of behavioral processing speed measures, and these processing speed measures predicted over 58% of variance in fluid intelligence in a many-to-one way. In addition to processing speed, other properties of cognitive processes may thus contribute independently to individual differences in general intelligence, giving rise to the positive manifold. In the view of the watershed model of fluid intelligence, mental speed is a property of one of a limited number of independent elementary cognitive processes contributing to individual differences in fluid intelligence.

1.3. Mental speed as one of many overlapping processes underlying g

A similar perspective has been suggested by Process Overlap Theory (POT; Kovacs & Conway, 2016), which explains the emergence of *g* with the idea that each cognitive test taps several of a limited number of independent elementary cognitive processes, which contribute to performance in these tests in a multiplicative way. One of the main differences between the watershed model and POT consists of the fact the former assumes that properties of cognitive processes contribute to individual differences in an additive way, whereas the latter proposes that they do so in a multiplicative way.

As such, POT proposes that certain domain-general cognitive processes such as executive processes may serve as central bottlenecks constraining performance in a large number of cognitive tests. According to POT, the speed of information processing might be seen as a domain-general property constraining performance in a wide number of cognitive tests. For individuals with a high speed of information processing, mental speed would not be predictive of their cognitive test performance (as other cognitive processes would instead constrain performance), whereas for individuals with a low speed of information processing, cognitive test performance would be strongly related to mental speed. Moreover, slowest reaction times should be more strongly related to general intelligence than fastest or mean reaction times if they reflect limitations in attentional control. This prediction of POT is supported by the *worst performance rule*, which describes the phenomenon that reaction times in the slowest percentile of the reaction time distribution are more strongly related to general intelligence than mean or fastest reaction times (Coyle, 2003; Larson & Alderton, 1990).

In addition, POT's assumption that executive processes may play a central role in explaining individual differences in general intelligence is supported by psychophysiological results showing that the latency of the P3 component is strongly related to general intelligence (Bazana & Stelmack, 2002; Schubert et al., 2017; Troche et al., 2009). Individuals with shorter P3 latencies may profit from a more efficient transmission of information from frontal attentional to parietal memory-related areas and may be less prone to be affected by irrelevant information (Polich, 2007). In this way, executive functions may mediate the link between neural processing speed and general intelligence and may affect attentional control as a central bottleneck in the POT framework. This tight link between executive functions and processing speed may, however, only emerge after adolescence, as developmental research has suggested that developmental cognitive change during childhood may be largely driven by executive functions, whereas the speed of information-processing contributes to stable individual differences in children's cognitive processing (Anderson, 2017).

2. Testing a causal relationship between mental speed and mental abilities

Taken together, all three of the above-mentioned theories suggest that mental speed is causally related to mental abilities in terms of neuro-cognitive processes. However, because all research on this topic has been correlational in nature, the direction of this association cannot be empirically determined, although there are substantial theoretical reasons to believe that processing speed serves as an endophenotype of intelligence. Moreover, it cannot be ruled out that the association is caused by third variables such as structural or organizational properties

of the brain affecting both mental speed and mental abilities.

A causal test of the association in question can be achieved by experimentally manipulating the speed of information processing as an independent variable and assessing the effect of this manipulation on an intelligence test. Ample psychopharmacological research has suggested that easily accessible psychostimulants such as caffeine, nicotine, or theanine can facilitate cognitive processing by enhancing the speed of information processing (e.g., Foxe et al., 2012; Haskell, Kennedy, Milne, Wesnes, & Scholey, 2008; Heishman, Kleykamp, & Singleton, 2010; Kahathuduwa, Dassanayake, Amarakoon, & Weerasinghe, 2017). In the present study, we used nicotine to achieve this experimental manipulation of the speed of information processing. Nicotine acts as an agonist at nicotinic acetylcholine receptors (nAChRs), which show a high density in the prefrontal cortex, parietal cortex, hippocampus, and thalamus (Azizian, Monterosso, O'Neill, & London, 2009; Brody, 2006; Levin, McClernon, & Rezvani, 2006). Nicotine administration has been associated with both increased and decreased neural activity (e.g., Hahn et al., 2007, 2009; Kumari et al., 2003), suggesting that nicotine modulates neural network activity depending on the types of neurons expressing nAChRs and the location of these neurons in a specific network (Hong et al., 2009; Mansvelder, van Aerde, Couey, & Brussaard, 2006).

A recent meta-analysis of 48 experiments testing the effects of nicotine on cognitive processing reported consistent effects of nicotine administration on the speed of information processing in a variety of experimental tasks (Heishman et al., 2010). On *N*-weighted average, the administration of nicotine led to an increase in reaction times of $g = 0.33$ in attention- and memory-related tasks. Effect sizes ranged from $g = -0.16$ to $g = 1.42$ for alerting attention RTs ($g_{mean} = 0.34$), from $g = 0.05$ to $g = 0.61$ for orienting attention RTs ($g_{mean} = 0.30$), and from $g = 0.00$ to $g = 1.05$ for short-term and working memory RTs ($g_{mean} = 0.34$). Despite the substantial variance in effect sizes, results from this meta-analysis suggest that nicotine administration overall has a small-to-medium positive effect on the speed of information processing.

Moreover, there has been some evidence that nicotine administration decreases P3 latencies in comparison to a placebo condition (Edwards, Wesnes, Warburton, & Gale, 1985; Houlihan, Pritchard, & Robinson, 1996). For example, Houlihan et al. (1996) administered either a placebo cigarette with a nicotine yield of only 0.05 mg or a treatment cigarette with a nicotine yield of 1.1 mg to 32 overnight-abstaining regular smokers in a repeated-measures design. They found that smoking decreased both reaction times, $\omega^2 = 0.40$, and P3 latencies, $\omega^2 = 0.13$.

However, the effect size of these pharmacological effects on P3 latencies were smaller than the effects on reaction times and some studies failed to find any effect on P3 latencies (e.g., Ilan & Polich, 1999; Knott, Bosman, Mahoney, Ilivitsky, & Quirt, 1999). In a study on 21 regular smokers, smoking a cigarette with a nicotine yield of 1.1 mg decreased N2, but not P3 latencies in a Sternberg short-term memory scanning task in comparison to smoking a placebo cigarette with a nicotine yield of only 0.05 mg (Houlihan, Pritchard, & Robinson, 2001). This inconsistency in results is not surprising (Houlihan et al., 2001) given the rather small sample sizes typical for electrophysiological studies, the probably small-to-moderate actual effect size, and the unreliability of ERP latencies (Cassidy, Robertson, & O'Connell, 2012; Schubert et al., 2017). In a review of the existing literature on the effects of nicotine on ERP components, Pritchard, Sokhadze, and Houlihan (2004) concluded that there probably were small effects of nicotine administration on P3 latencies, but that the existing literature was too heterogeneous in terms of sample composition, nicotine administration, and experimental procedure to allow for a systematization of boundary conditions.

Taken together, there is clear evidence that nicotine has an effect on reaction times that is not just a reversal of withdrawal-related symptoms (Heishman et al., 2010), and some evidence that this effect may at least in part be due to an acceleration of stimulus-evaluation on a neural level (Pritchard et al., 2004). If nicotine administration increases the speed of information processing, and if a greater speed of information processing positively affects general intelligence, then nicotine administration should enhance performance on standard intelligence tests. In fact, a first study supports this notion: In a study on 16 regular smokers who had to abstain from smoking two hours prior to their participation in the experiment, taking six puffs of a cigarette yielding 0.80 mg of nicotine prior to completing Raven's Advanced Progressive Matrices and two additional puffs ten minutes into the intelligence test, led to an average increase of six IQ points in comparison to a placebo condition, corresponding to a medium effect size of about $d = 0.50$ (Stough, Mangan, Bates, & Pellett, 1994).

However, the study does not allow a clear conclusion regarding the nature of the relationship between mental speed and mental abilities due to four limitations. First, because the speed of information processing was not measured in this study, it cannot be concluded if nicotine effects on intelligence test scores were mediated by changes in mental speed. Second, because only regular smokers participated in the study who had to abstain from smoking for two hours prior to participation, it is not clear if the results may not at least in part reflect the recovery from withdrawal symptoms. Third, the sample size was rather small, which may have led to an overestimation of the effects of nicotine on intelligence test performance and warrants replication of the study. Fourth, instead of a sham-smoking in the control condition, participants did not smoke at all in the control condition. Hence, the design was not double-blind and participant and experimenter expectancy effects may have biased the outcome (Rosenthal & Jacobson, 1966). Moreover, the experimental condition differed from the control condition not only in terms of nicotine administration, but also in the sensory and motoric components of smoking. Nevertheless, this study can be seen to yield the first preliminary evidence that nicotine administration might enhance performance in intelligence tests.

3. The present study

Bridging the fields of correlational and experimental psychology (Cronbach, 1957), the present study used a double-blind design to assess the effects of nicotine administration on the speed of information processing and intelligence test performance in occasional smokers. The hypotheses of the present study are based on the idea that individual differences in the speed of information processing cause individual differences in general intelligence. We assume that nicotine administration decreases reaction times and ERP latencies, and that this facilitation of information processing positively affects performance in an intelligence test.

4. Method

4.1. Participants

We recruited a sample of $N = 55$ participants. Sample size was determined based on the meta-analysis about effects of nicotine on reaction times by Heishman et al. (2010), assuming an average effect size of $g = 0.33$ of nicotine on the speed of information processing across different attention- and memory-related cognitive tasks, an alpha level of $\alpha = 0.05$, a power of $1\text{-}\beta = 0.80$, and a dropout-rate of 10% due to the longitudinal design (two measurement occasions).

Of these 55 participants, five had to be excluded because they reported side effects from the nicotine patch, and one had to be excluded because the participant reported side effects from the placebo patch. In addition, two participants did not show up in the second week of the experiment, resulting in a final sample size of $N = 47$ participants (26 females, 20 males, one not reported) between 19 and 63 years old ($M = 24.93$ years, $SD = 9.48$ years).

All participants were at least 18 years old, were not pregnant, had

normal or corrected to normal vision, and did not suffer from any cardiovascular, skin, neurological, or psychiatric diseases. Moreover, all participants identified themselves as "occasional smokers", which we defined as a) smoking only on some days, but not every day; b) having smoked at least one and not > 20 cigarettes in the last month; c) having smoked at least 15 cigarettes in their lifetime. On average, participants had smoked 10 cigarettes (SD = 11) in the previous months. At the first laboratory session, participants signed an informed consent. They received 50€ or course credit as a reward for their participation. The study was approved by the ethics committee of the faculty of behavioral and cultural studies, Heidelberg University.

4.2. Materials

4.2.1. Sternberg memory scanning task (SMT)

In the SMT (Sternberg, 1969), participants were shown memory sets consisting of five digits between 0 and 9, and they had to indicate whether an immediately afterwards presented probe stimulus was part of the previously presented memory set. Participants completed ten practice trials with immediate feedback followed by 100 test trials without feedback.

At the beginning of each trial, a white fixation cross was shown in the middle of a black screen for 1000–1500 ms. Digits were presented sequentially for 1000 ms with a blank screen shown for 400–600 ms between each digit. A black screen with a white question mark was shown for 1800–2200 ms after the last digit was presented, followed by a black screen showing the probe stimulus. Participants had to press one of two keys with their index fingers to indicate whether the digit was part of the memory set, which was the case in 50% of the trials. The position of keys was counterbalanced across participants. After their response, the screen remained unchanged for 1000 ms, followed by an ITI of 1000–1500 ms. Please see Fig. 1 for an illustration of the task procedure.

4.2.2. Hagen Matrices Test (HMT)

The HMT (Heydasch, 2014) is a freely available, web-based fluid intelligence test that consists of 20 items. Each item consists of a 3 × 3-matrix with geometric figures that follow certain logical rules and symmetries. The last element of the matrix is missing and must be chosen out of eight alternatives within two minutes. We divided test items in half based on an odd-even split to create a different version for each of the two laboratory morning sessions. Test versions were counterbalanced across laboratory sessions and participants. Participants' performance was calculated separately for each laboratory session as the percentage of correctly solved items. Cronbach's alpha for the version containing even items was α = 0.68 and for the version containing odd items α = 0.70. Participants' mean IQ score based on aggregated performance in the HTM across both laboratory sessions was M = 117.69, SD = 11.93.

4.2.3. Advanced Progressive Matrices (APM)

We used a computer-adapted version of the APM (Raven, Court, & Raven, 1994). The APM is a fluid intelligence test consisting of 36 items. Each item consists of a 3 × 3-matrix with geometric figures that follow certain logical rules and symmetries. The last element of the matrix is missing and must be chosen out of eight alternatives without time limit. We divided test items in half based on an odd-even split to create a different version for each of the two laboratory afternoon sessions. Test versions were counterbalanced across laboratory sessions and participants. Participants' performance was calculated separately for each laboratory session as the percentage of correctly solved items of the second test set. Cronbach's alpha for the version containing even items was α = 0.80 and for the version containing odd items α = 0.74. Participants' mean IQ score based on aggregated performance in the APM across both laboratory sessions was M = 100.03, SD = 14.53.

4.3. Procedure

Participants were told to not consume alcohol 24 h and nicotine and caffeine 12 h prior to their participation in the study. When participants entered the laboratory for their first session, they were informed extensively about the procedure and risks of the study. After signing an informed consent, they completed the SMT, Attention Network Test (ANT; Fan, McCandliss, Sommer, Raz, & Posner, 2002; data not reported here), and HMT in one of six possible orders. The order of tasks remained the same for each participant across all four laboratory sessions, but was counterbalanced across participants. After completing the experimental tasks, participants were asked to put on a sleeping mask while another experimenter – not running the actual experiments – applied the nicotine or placebo patch on the inner side of the non-dominant upper arm. The nicotine patch contained either 7 mg of nicotine (lower dose, 2/3 of the participants) or 14 mg of nicotine (higher dose, 1/3 of the participants). Nicotine dose was varied because of the unclear dose-response relationship reported by Heishman et al. (2010). The administration of nicotine vs. placebo patches in the first or second week was counterbalanced across participants. The patch was then covered with a larger, opaque patch and a colored bandage. The morning session took about one hour. Participants were asked to refrain from consuming alcohol, nicotine, and caffeine until the afternoon session and were asked to return to the laboratory about seven hours later.

In the afternoon session, participants completed the SMT, ANT, and APM in the same order as in the morning while their EEG was recorded. Afterwards, they were asked if they had adhered to the instructions to refrain from nicotine, caffeine, and alcohol consumption during the previous day and had to guess which patch had been applied.

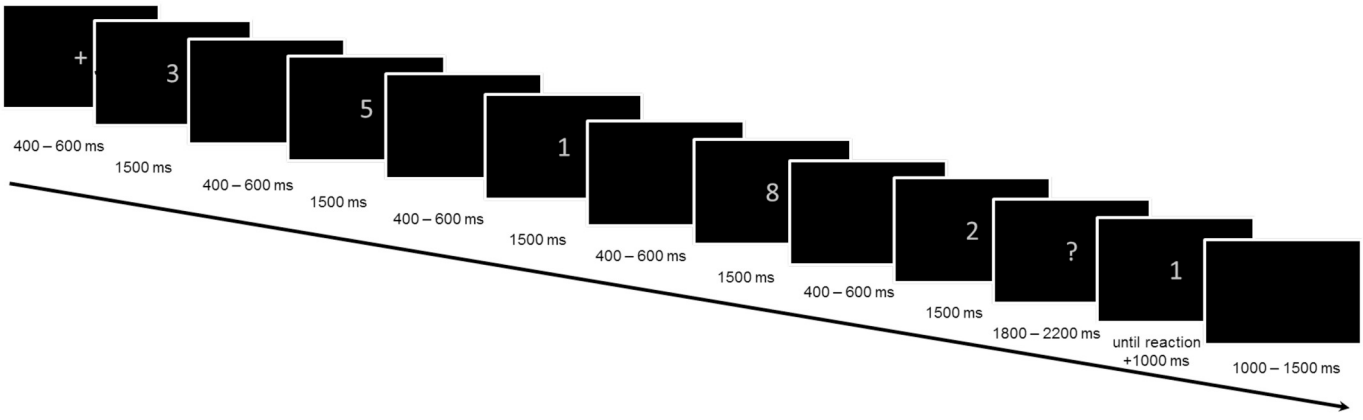

Fig. 1. Task procedure of the Sternberg memory scanning task.

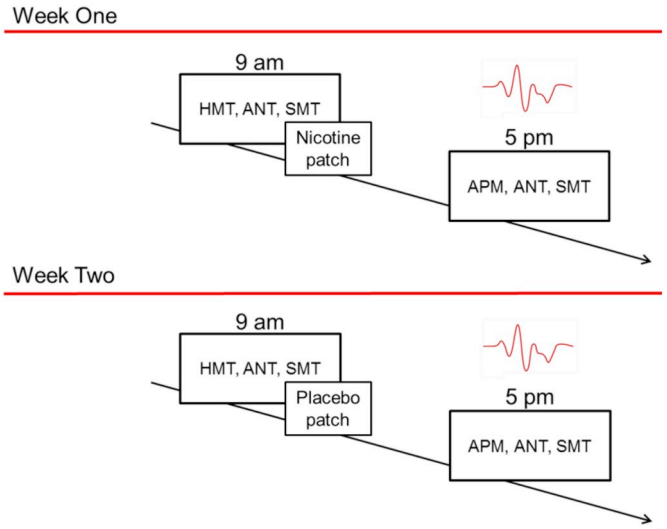

Fig. 2. Overview over the experimental procedure for a sample participant. The order of tasks remained the same for each participant across all four laboratory sessions, but was counterbalanced across participants. The nicotine or placebo patch was applied on the inner side of the non-dominant upper arm. The patch was then covered with a larger, opaque patch and a colored bandage. Whether the nicotine patch was administered in the first or second week was counterbalanced across participants. ANT = Attention Network Test; APM = Raven's Advanced Progressive Matrices; HMT = Hagen Matrices Test; SMT = Sternberg memory scanning task.

Participants were not able to correctly identify when the nicotine patch had been applied above chance level (45% of correct guesses). Patches were removed by the experimenter while participants wore a sleeping mask and averted their eyes. The afternoon session took 1.5–2 h including EEG preparation.

Participants returned to the laboratory a week later. The procedure was identical to the week before, except that they were administered the placebo patch if they had worn the nicotine patch in the previous week and vice versa. They met the same experimenter as the week before in the morning session, as this experimenter was not aware which patch had been applied the previous week, but a different experimenter in the afternoon session, as the experimenter of the previous week had removed the patch at the end of the afternoon session and thus knew about participants' allocation to experimental conditions. At the end of the afternoon session, participants completed two additional questionnaires about demographic data and their nicotine and drug consumption and were debriefed. An overview over the experimental procedure is shown in Fig. 2.

4.4. EEG recording

The EEG was recorded with 12 Ag/AgCl electrodes (F3, Fz, F4, C3, Cz, C4, P3, Pz, P4, O3, Oz, O4) that were positioned according to the international 10–20 system. We used the aFz electrode as the ground electrode. Electrodes were initially referenced to Cz and offline re-referenced to linked mastoids (TP9, TP10). To correct for ocular artifacts, we recorded the electrooculogram (EOG) bipolarly with two electrodes positioned above and below the left eye and two electrodes positioned at the outer canthi of the eyes. All electrode impedances were kept below 5 kΩ. The EEG signal was recorded continuously with a sampling rate of 1000 Hz (band-pass 0.1–100 Hz), and filtered offline with a low-pass filter of 30 Hz with a slope of 12db/octave.

4.5. Data analysis

4.5.1. Behavioral data

Behavioral data (RTs and accuracies) stem from the Sternberg memory scanning task. To remove outliers in the reaction time data, we discarded any reaction times faster than 100 ms or slower than 3000 ms. In a second step, we discarded any trials with logarithmized reaction times exceeding ± 3 SDs of an individual participant's RT distribution. We subsequently calculated median reaction times separately for each of the four laboratory sessions. Additionally, we created an odd-even split of RTs for each of the laboratory sessions to calculate odd-even correlations as estimates of reliability. Odd-even correlations ranged from $r = 0.95$ to $r = 0.99$.

4.5.2. Electrophysiological data

Event-related potentials (ERPs) were calculated by averaging all experimental trials in the SMT, time-locked to the onset of the probe stimulus, with windows of interest that were 1000 ms long with a preceding baseline of 200 ms. Ocular artifacts were corrected for with the regression procedure suggested by Gratton, Coles, and Donchin (1983). Windows of EEG data with amplitudes exceeding ± 70 μV, with amplitude changes exceeding 100 μV within 100 ms, or with activity ongoing lower than 0.5 μV in an interval of 100 ms were discarded as artifacts.

We determined N2 and P3 latencies for each participant in the Sternberg memory scanning task at the Pz electrode, because an inspection of the grand average (see Fig. 4) revealed that they were largest at this electrode site and because of the previously reported association between peak latencies at parietal electrode sites and general intelligence (Schubert et al., 2017). We calculated N2 amplitudes by averaging across electrode activity in a time window from 280 to 325 ms, and we calculated P3 amplitudes by averaging across electrode activity in a time window from 340 to 470 ms separately for the F3, Fz, F4, C3, Cz, C4, P3, Pz, and P4 electrodes. Reliabilities of ERP data were estimated as correlations between laboratory sessions separately for each of the two ERP components and ranged from $r = 0.60$ to $r = 0.90$.

4.5.3. Statistical analyses

All statistical analyses were conducted both with null hypothesis significance testing (NHST) and with Bayesian analyses. The Bayes Factors (BFs) reported quantify the evidence in favor of including the relevant effect in the model in comparison to a model that does not contain the relevant effect. BF_{10} quantifies evidence in favor of the alternative hypotheses, and BF_{01} ($BF_{01} = 1/BF_{10}$) quantifies evidence in favor of the null hypotheses. All statistical analyses were conducted with JASP (Version 0.8.6; JASP Team, 2018) with a Type I error-level of $\alpha = 0.05$ for NHST, a non-informative JZS default prior for Bayesian ANOVAs, and a uniformly distributed prior ranging from −1 to 1 for correlational analysis, unless otherwise noted. We first calculated separate ANOVAs testing if nicotine administration enhanced performance in each dependent variable (reaction times, ERP latencies, intelligence test scores). Subsequently, we *z*-standardized all values separately for each measure and calculated an overall ANOVA to test if the effect of nicotine on performance differed across measures.

5. Results

5.1. Behavioral effects of nicotine

Irrespective of treatment condition, participants showed shorter reaction times in the Sternberg memory scanning task in afternoon sessions than in morning sessions, $F(1, 43) = 27.00$, $p < .001$, $\omega^2 = 0.37$, $BF_{10} = 2911.37$. In addition, participants showed greater improvements in reaction times under the application of nicotine than under the application of placebo as indicated by the significant interaction between Treatment and Session, $F(1, 43) = 5.29$, $p = .026$, $\omega^2 = 0.08$, $BF_{10} = 0.41$ (see Fig. 3). Participants in the placebo condition showed an average decrease in reaction times of $M_{decrease} = 59.02$ ms ($SD_{decrease} = 119.80$ ms), $F(1, 43) = 7.75$, $p = .008$, $\omega^2 = 0.13$, $BF_{10} = 17.44$. In comparison, participants in the nicotine condition showed an average decrease in reaction times of

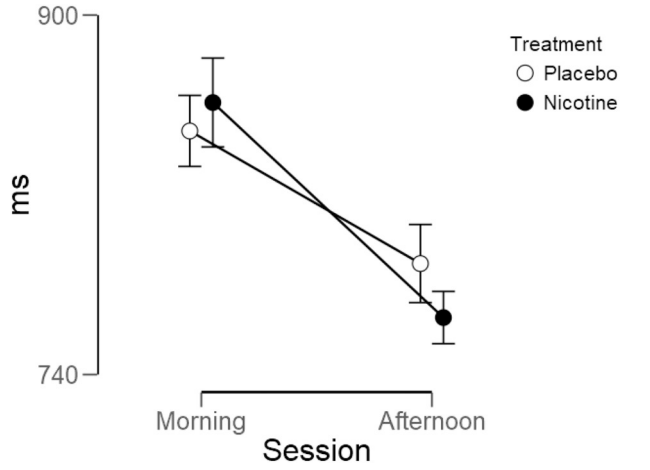

Fig. 3. Median reaction times as a function of Session (Morning vs. Afternoon) and Treatment (Placebo vs. Nicotine). Error bars indicate standard errors.

$M_{decrease}$ = 95.72 ms ($SD_{decrease}$ = 112.00 ms), $F(1, 43) = 32.16$, $p < .001$, $\omega^2 = 0.40$, $BF_{10} = 13{,}106.70$. These effects were not moderated by nicotine dose, all Fs ≤ 2.62, all ps ≥ 0.113, all ω^2s ≤ 0.03, all $BFs_{01} \geq 4.01$. Taken together, these results suggest from a Bayesian perspective that while there was insufficient evidence for an interaction between Treatment and Session ($1/3 < BF_{10} < 3$), we observed substantially more evidence for a decline in reaction times in the nicotine than in the placebo condition (13,106.70/ 17.44 = 751.53). Accuracies did not change as a function of Session, Treatment, or Dose, all Fs ≤ 2.36, all ps ≥ 0.132, all ω^2s ≤ 0.03, $BFs_{01} \geq 1.70$. This pattern of results indicates that the experimental effects of nicotine administration cannot be attributed to a shift in speed-accuracy tradeoffs, which was also supported by a supplementary diffusion model analysis (not reported here) that found no effect of nicotine administration on the response criterion.

5.2. Electrophysiological effects of nicotine

5.2.1. ERP latencies

Grand averages of event-related potentials are shown separately for the placebo and the nicotine condition in Fig. 4. We found no evidence for an overall effect of Treatment on N2 latencies, $F(1, 43) = 2.55$, $p = .118$, $\omega^2 = 0.03$, $BF_{10} = 0.39$, nor for a moderation of this effect by nicotine dose, $F(1, 43) = 2.70$, $p = .107$, $\omega^2 = 0.04$ $BF_{10} = 0.88$. See Fig. 5 for effects of Treatment on ERP latencies.

P3 latencies decreased under the application of nicotine, $F(1, 43) = 7.32$, $p = .010$, $\omega^2 = 0.12$, $BF_{10} = 21.74$. On average, participants showed $M_{decrease}$ = 29.29 ms (SD = 57.26 ms) shorter latencies in the nicotine than in the placebo condition. Again, this effect was not moderated by nicotine dose, $F(1, 43) = 3.32$, $p = .075$, $\omega^2 = 0.04$, $BF_{10} = 1.39$.

5.2.2. ERP amplitudes

We also investigated if nicotine affected ERP amplitudes in an exploratory manner. We observed no significant main effect or interaction including Treatment on N2 amplitudes, all Fs ≤ 2.57, all ps ≥ 0.089, all ω^2s ≤ 0.01, all $BFs_{01} \geq 1.39$. We observed a marginally significant Treatment x Caudality interaction on P3 amplitudes, $F(2, 86) = 3.37$, $p = .054$, $\varepsilon = 0.75$, $\omega^2 = 0.02$, $BF_{10} = 0.46$. This interaction suggested that there may be an amplifying effect of nicotine on P3 amplitudes that may be most pronounced at frontal electrode sites.

5.3. Nicotine and fluid intelligence

We observed a negative correlation between reaction times and HMT scores at baseline, $r = -0.49$, $p < .001$, $BF_{10} = 41.17$, but no evidence for an association between N2 latencies, $r = 0.22$, $p = .144$, $BF_{10} = 0.52$, or P3 latencies, $r = 0.12$, $p = .430$, $BF_{10} = 0.25$, with HTM scores at baseline. The lack of associations between neural processing speed and intelligence test scores is, however, not surprising given the low consistency of single ERP latency measures (see Schubert et al., 2017).

Irrespective of treatment condition, participants performed better in the APM that was conducted at all afternoon sessions than in the HMT that was conducted in all morning sessions, $F(1,43) = 205.89$, $p < .001$, $\omega^2 = 0.81$, $BF_{10} = 2.24e + 14$. This improvement was, however, not moderated by Treatment, $F(1,43) = 3.25$, $p = .078$, $\omega^2 = 0.05$, $BF_{10} = 0.39$, nor by an interaction between Nicotine Dose and Treatment, $F(1,43) = 1.37$, $p = .249$, $\omega^2 = 0.01$, $BF_{10} = 0.55$. Fig. 6 illustrates the effect of Session and Treatment on intelligence test scores and shows that even the small non-significant interaction effect pointed in the wrong direction, i.e. that participants' session-related increase in performance was smaller in the nicotine than in the placebo condition.

To determine if the data contained sufficient evidence to assume the null hypothesis that nicotine administration did not lead to an increase in intelligence test scores, we conducted a post-hoc Bayesian t-test on the difference between intelligence test scores in morning and afternoon sessions with a one-sided Cauchy prior truncated at zero. Truncating the prior distribution at zero was necessary to test the one-sided hypothesis that nicotine administration should lead to a greater increase in IQ scores in comparison to placebo administration. According to the criteria suggested by Jeffreys (1961), we found strong evidence in favor of the null hypothesis that nicotine administration did

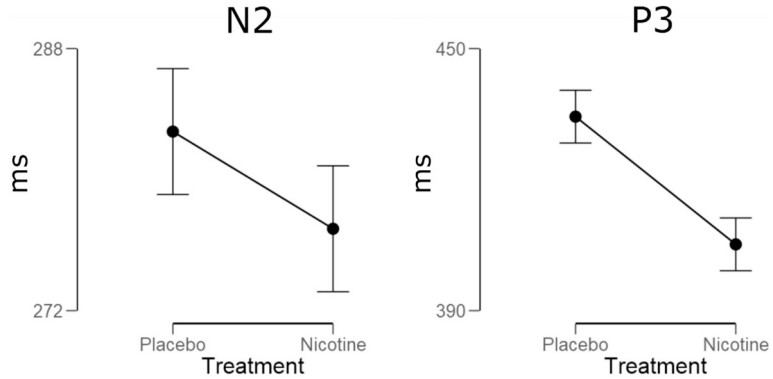

Fig. 4. Grand averages of event-related potentials as measured at Fz, Cz, and Pz electrode sites for the placebo condition (solid lines) and the nicotine condition (dashed lines). ERPs were elicited by the onset of the probe stimulus.

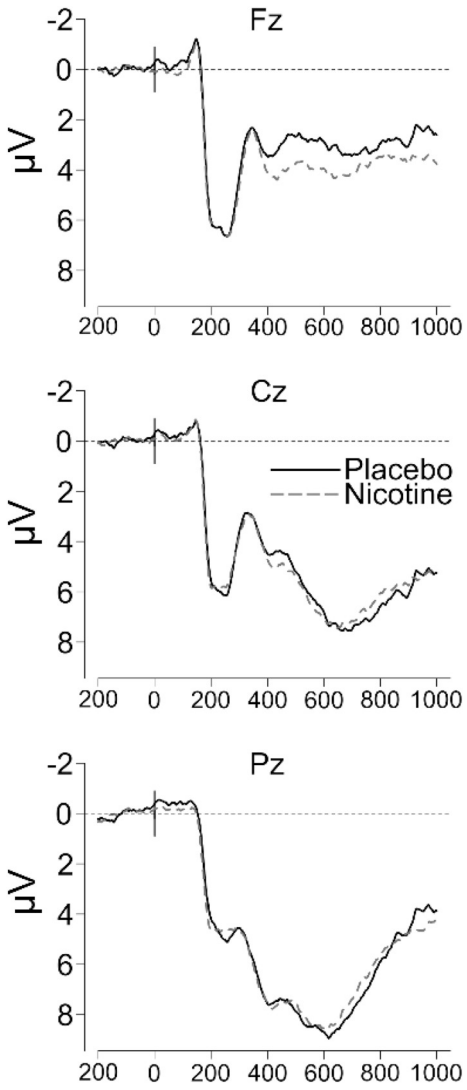

Fig. 5. ERP latencies as a function of Treatment (Placebo vs. Nicotine). Error bars indicate standard errors.

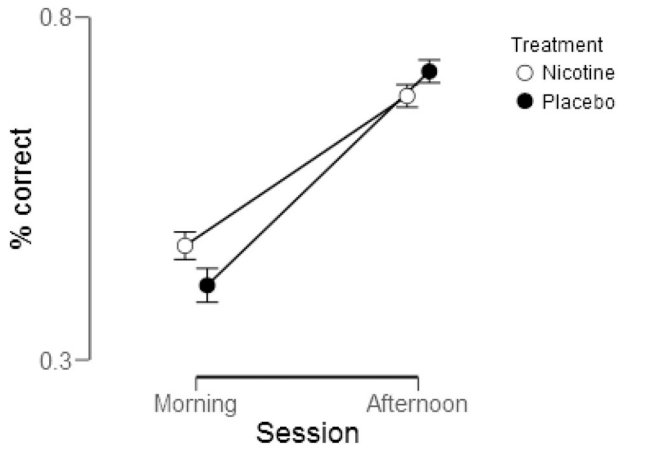

Fig. 6. Percent of correctly solved trials in intelligence tests as a function of Session/Test (Morning/HMT vs. Afternoon/APM) and Treatment (Placebo vs. Nicotine). Error bars indicate standard errors. Note that the HMT was conducted at all morning sessions, whereas the APM was conducted at all afternoon sessions.

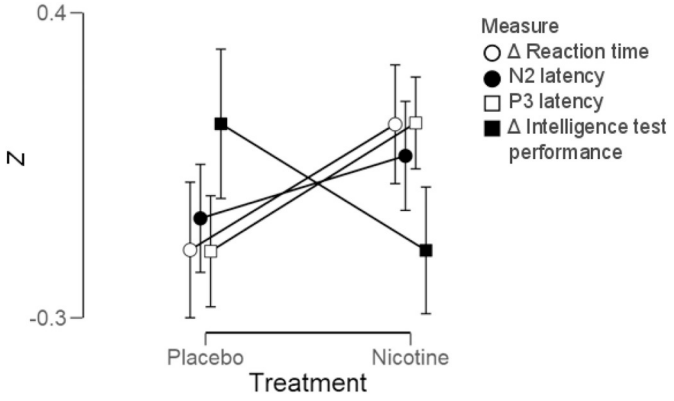

Fig. 7. *Z* scores of different performance measures (N2 latency, P3 latency, and morning-to-afternoon differences in reaction time and intelligence test performance) as a function of Treatment (Placebo vs. Nicotine). Greater *z* values indicate better performance or respectively a greater increase in performance for all dependent variables. Error bars indicate standard errors.

not lead to an increase in intelligence test scores in comparison to the placebo condition, $BF_{01} = 13.17$.

To test if nicotine only enhanced fluid intelligence in participants with lower processing speed, we introduced averaged baseline RTs in the STM across the two weeks and P3 latencies in the placebo condition as covariates. However, including baseline processing speed variables as covariates did not change the result that nicotine administration had no significant effect on APM test scores, all $Fs \leq 2.12$, all $ps \geq 0.153$, all $\omega^2 s \leq 0.02$, all $BFs_{01} \geq 1.74$.

5.4. Comparing the effect of nicotine on all performance measures

Although there was a significant nicotine-related decrease both in reaction times and in P3 latencies and no significant effect on intelligence test performances, this does not imply that the difference between these effects itself is significant. Therefore, we *z*-standardized each measure so that greater *z*-values reflected better performance or a greater increase in performance, respectively. Table 1 shows effect sizes and 95% Bayesian credibility intervals around these effect sizes for the effect of nicotine in comparison to placebo administration on each measure. An inspection of the credibility intervals of pairwise comparison effect sizes indicates that there was a positive nicotine-related effect on P3 latencies, $CI_{\delta P3}$ 95% = [0.22; 1.11] that was substantially larger than the nicotine-related effect on intelligence test score performance, $CI_{\delta IQ}$ 95% = [−0.65; 0.16], as indicated by the non-overlapping credibility intervals around the effect sizes δ.

Table 1
Median effect sizes and 95% credibility intervals of the effect sizes for the effect of nicotine in comparison to placebo administration on N2 latency, P3 latency, and morning-to-afternoon differences in reaction time and intelligence test performance. Variables were coded in such a way that greater δ values indicate better performance or respectively a greater increase in performance after nicotine administration for all dependent variables.

	Median effect size δ	95% credibility interval
Δ Reaction time	0.31	−0.10; 0.71
N2 latency	0.23	−0.18; 0.64
P3 latency	0.67	0.22; 1.11
Δ Intelligence test performance	−0.24	−0.65; 0.16

Subsequently, we submitted these *z*-standardized values into a two-factorial ANOVA with the factors Treatment (Placebo vs. Nicotine) and Measure (RT difference, N2 latencies, P3 latencies, Intelligence test performance difference). We found a significant interaction between

Treatment and Measure (see Fig. 7), $F(3,123) = 3.12$, $p = .046$, $\omega^2 = 0.02$, $BF_{10} = 0.37$, suggesting that nicotine may affect the four performance measures differentially. However, the BF yielded weak evidence in favor of the null hypothesis ($BF_{01} = 2.70$), indicating that more data would be needed for a conclusive test of the interaction hypothesis either way.

6. Discussion

The present study analyzed the effects of transdermal nicotine administration on the speed of information processing and intelligence test scores in occasional smokers. Consistent with our hypotheses and previous research, we found that nicotine administration caused an increase in both neural and behavioral information processing speed. However, we did not observe any effect of nicotine on intelligence test scores despite the drug-related increase in the speed of information processing. These results contradict theories proposing that a greater speed of information processing causes greater general intelligence.

Consistent with previous research, we found that nicotine administration decreased reaction times in the Sternberg memory scanning task in comparison to a placebo condition by about 35 ms, $\omega^2 = 0.08$ (Heishman et al., 2010; Pritchard et al., 2004), resulting in a greater behavioral processing speed. In addition, nicotine administration also decreased P3 latencies by about 30 ms and accounted for 12% of the variance in P3 latencies, which is consistent with previous studies reporting effects on P3 latencies after nicotine inhalation (e.g., Edwards et al., 1985; Houlihan et al., 1996). Contrary to one study investigating nicotine effects on performance in the Sternberg memory scanning task (Houlihan et al., 2001), we did not observe any conclusive evidence for or against a nicotine-related decrease of N2 latencies. This result suggests that nicotine enhanced information processing most strongly by affecting the efficiency of neurocognitive processes related to the P3, such as the inhibition of neural activity related to non-target stimuli in the memory set and subsequent memory updating (Polich, 2007). The unclear effect on N2 latencies compared to Houlihan et al. (2001) may suggest that nicotine affects information processing differently in abstaining regular than in occasional smokers, or that our sample size was too small to detect more subtle effects on N2 latencies.

In spite of these effects of nicotine on both the neural and behavioral speed of information processing, we failed to observe any positive effect of nicotine on intelligence test scores. Unless nicotine simultaneously increased the speed of information processing and impaired some other cognitive processes fundamental to general intelligence, these results directly contradict theories proposing that a greater speed of information processing causes greater fluid intelligence. In particular, they contradict the hypothesis that a greater speed of information processing positively affects intelligence test performance by enhancing the efficiency of memory updating, as measured by P3 latencies and performance in the Sternberg memory scanning task (Jensen, 2006; Schubert et al., 2017). In addition, they challenge the idea that the speed of information processing is the neural basis or an endophenotype of *g* (Jensen, 2006; Kievit et al., 2016).

Instead, our results suggest that some additional variables that affect both the speed of information processing and intelligence test performance, but are themselves unaffected by nicotine administration, may give rise to the association between mental speed and mental abilities. If this were the case, nicotine administration would have affected some properties of the latent P3-generating process, resulting in shorter latencies under nicotine administration, but not those properties of the generating process associated with cognitive abilities. The covariance between neural processing speed and mental abilities might thus arise due to individual differences in those properties of the P3-generating process that cannot be temporarily enhanced by nicotine administration. However, it is entirely possible that a longer and more intense administration of nicotine might affect these properties of the P3-generating process. As high intensity longitudinal cognitive trainings have been shown to change the white matter microstructure (Lövdén et al., 2010), high intensity longitudinal nicotine administration might affect structural brain properties in a similar way. In fact, there is some evidence that smokers show a greater white-matter tract integrity than non-smokers in the first years of nicotine consumption (Hudkins, O'Neill, Tobias, Bartzokis, & London, 2012; Jacobsen et al., 2007; Paul et al., 2008).

Candidate variables underlying the association between mental speed and mental abilities that are not affected by a one-time nicotine administration may be structural brain properties or other cognitive process parameters such as attentional control that have been shown to be related to both neural information processing speed and cognitive abilities: Organizational properties of the salience network responsible for the detection and evaluation of goal-relevant information have been shown to be reflected in the timing of ERP components associated with higher-order cognition (Menon & Uddin, 2010; Soltani & Knight, 2000). Moreover, individual differences in the nodal efficiency in the right anterior insula and the dorsal anterior cingulate cortex, which are core regions of the salience network, have been shown to be related to individual differences in general intelligence (Hilger, Ekman, Fiebach, & Basten, 2017). Hence, a more efficient neural organization of the salience network may be reflected in shorter P3 latencies and may also give rise to greater performance in intelligence tests by facilitating goal-driven information selection.

Alternatively, greater attentional control may act as a third variable that increases both the speed and consistency of information processing as well as performance in intelligence tests. Attentional lapses have been shown to be specifically associated with longer reaction times in the slowest quantiles of the reaction time distribution (McVay & Kane, 2012), and individual differences in slowest reaction times have been shown to be more predictive of cognitive abilities than fastest or mean reaction times (Coyle, 2003; Larson & Alderton, 1990). An experimental manipulation aimed at increasing attentional control either pharmacologically (e.g., by modafinil administration; Marchant et al., 2009; Turner et al., 2003) or through meditation exercises (e.g., Brewer et al., 2011; Sahdra et al., 2011) would allow a direct test of this hypothesis.

In the light of Process Overlap Theory (Kovacs & Conway, 2016), the speed of information processing may be seen as one of several independent domain-general properties constraining performance in a wide number of cognitive tests. Hence, enhancing the speed of information processing should only positively affect intelligence test scores if mental speed acted as a central bottleneck constraining intelligence test performance to begin with. However, including baseline processing speed as a covariate in our analyses did not change the results regarding the effects of nicotine on intelligence test scores in any way. Nevertheless, given the average to above average intelligence of the student sample in the present study, it can be argued that the speed of information processing did not act as a central bottleneck constraining intelligence test performance even in those participants with the slowest processing speeds in the present study.

6.1. Limitations

One limitation of the present study is that the sample size may not have been large enough to detect more nuanced effects of nicotine on information-processing. In particular, although NHST indicated a significant effect of nicotine on the improvement in processing speed between morning and afternoon sessions, Bayesian analyses indicated that the data contained insufficient evidence to make strong claims regarding the presence or absence of an interaction effect on reaction times. If anything, the Bayes Factor suggested that the data contained weak evidence in favor of the null hypothesis instead ($BF_{01} = 2.43$). Moreover, the sample size or the population effect may have been too small to find an effect of nicotine dose on ERP latencies as indicated by Bayes factors close to one. Increasing the sample size in future studies might shed more light on dose-response-effects on information processing speed variables and may provide more robust estimates of

nicotine-related effects on reaction times. Nevertheless, we found clear evidence for a substantial effect of nicotine on P3 latencies, and also strong evidence against a nicotine-related increase in intelligence test performances – a conclusion that was further supported by non-overlapping credibility intervals of the two effects. Moreover, our results suggested that the effect of nicotine may not be equal for all performance measures, although Bayesian analyses indicated that more data are needed to draw firm conclusions.

Moreover, most participants had an academic background and average to above average cognitive abilities. Although this sample composition is ideal to minimize between-person variance with regard to the experimental design, we could not observe any effects of nicotine on cognitive abilities specific to individuals with below average intelligence. Previous research has shown that the beneficial effects of nicotine are most pronounced in individuals with low baseline performance in experimental tasks (e.g. Behler, Breckel, & Thiel, 2015; Knott et al., 2014; Newhouse, Potter, & Singh, 2004).

In a similar vein, there is some evidence that the large inter-individual variability in response to nicotine can at least in part be accounted for by cholinergic and dopaminergic genetic variations (Ahrens et al., 2015; Heishman et al., 2010). Although genotyping study participants was not feasible in the present study, it might have been illuminating with regard to individual differences in the effect of nicotine administration. However, given the substantial effects of nicotine on both behavioral and neural processing speed, it is unlikely that including information about genetic variation would substantially alter the main conclusions.

Moreover, we did not use the same fluid intelligence test in the baseline morning sessions as in the experimental afternoon sessions. Hence, the large difference between morning- and afternoon-treatment test scores can either be accounted for by training effects, by circadian effects, or by differences in test difficulty. However, this limitation does not affect the main conclusions of the present study, as we failed to observe any significant interaction between session and treatment.

6.2. Conclusion

The present study used transdermal nicotine administration in a double-blind design to enhance the speed of information processing and tested whether this increase in information processing speed affected performance in intelligence test scores. While we observed nicotine-related increases in both the neural and behavioral speed of information processing, there was no effect on intelligence test scores. These results contradict the idea that the association between mental speed and mental abilities can be explained by mental speed having a direct causal effect on performance in intelligence tests. Instead, they suggest that structural properties of the brain may affect both the speed of information processing and general intelligence and may thus give rise to the association between mental speed and mental abilities.

References

Ahrens, S., Markett, S., Breckel, T. P. K., Behler, O., Reuter, M., & Thiel, C. M. (2015). Modulation of nicotine effects on selective attention by DRD2 and CHRNA4 gene polymorphisms. *Psychopharmacology, 232*, 2323–2331. https://doi.org/10.1007/s00213-015-3869-2.

Anderson, M. (2017). Binet's Error: Developmental Change and Individual differences in Intelligence are Related to Different Mechanisms. *Journal of Intelligence, 5*. https://doi.org/10.3390/jintelligence5020024.

Azizian, A., Monterosso, J., O'Neill, J., & London, E. D. (2009). Magnetic resonance imaging studies of cigarette smoking. *Handbook of Experimental Pharmacology, 192*, 113–143. https://doi.org/10.1007/978-3-540-69248-5_5.

Bazana, P. G., & Stelmack, R. M. (2002). Intelligence and information processing during an auditory discrimination task with backward masking: An event-related potential analysis. *Journal of Personality and Social Psychology, 83*, 998–1008.

Behler, O., Breckel, T. P. K., & Thiel, C. M. (2015). Nicotine reduces distraction under low perceptual load. *Psychopharmacology, 232*, 1269–1277. https://doi.org/10.1007/s00213-014-3761-5.

Brewer, J. A., Worhunsky, P. D., Gray, J. R., Tang, Y.-Y., Weber, J., & Kober, H. (2011). Meditation experience is associated with differences in default mode network activity and connectivity. *Proceedings of the National Academy of Sciences of the United States of America. Vol. 108. Proceedings of the National Academy of Sciences of the United States of America* (pp. 20254–20259). . https://doi.org/10.1073/pnas.1112029108.

Brody, A. L. (2006). Functional brain imaging of tobacco use and dependence. *Journal of Psychiatric Research, 40*, 404–418. https://doi.org/10.1016/j.jpsychires.2005.04.012.

Cassidy, S. M., Robertson, I. H., & O'Connell, R. G. (2012). Retest reliability of event-related potentials: Evidence from a variety of paradigms. *Psychophysiology, 49*, 659–664. https://doi.org/10.1111/j.1469-8986.2011.01349.x.

Coyle, T. R. (2003). A review of the worst performance rule: Evidence, theory, and alternative hypotheses. *Intelligence, 31*, 567–587. https://doi.org/10.1016/S0160-2896(03)00054-0.

Cronbach, L. J. (1957). The two disciplines of scientific psychology. *American Psychologist, 12*, 671–684. https://doi.org/10.1037/h0043943.

Edwards, J. A., Wesnes, K., Warburton, D. M., & Gale, A. (1985). Evidence of more rapid stimulus evaluation following cigarette smoking. *Addictive Behaviors, 10*, 113–126.

Euler, M. J., McKinney, T. L., Schryver, H. M., & Okabe, H. (2017). ERP correlates of the decision time-IQ relationship: The role of complexity in task- and brain-IQ effects. *Intelligence, 65*, 1–10. https://doi.org/10.1016/j.intell.2017.08.003.

Fan, J., McCandliss, B. D., Sommer, T., Raz, A., & Posner, M. I. (2002). Testing the efficiency and independence of attentional networks. *Journal of Cognitive Neuroscience, 14*, 340–347. https://doi.org/10.1162/089892902317361886.

Foxe, J. J., Morie, K. P., Laud, P. J., Rowson, M. J., de Bruin, E. A., & Kelly, S. P. (2012). Assessing the effects of caffeine and theanine on the maintenance of vigilance during a sustained attention task. *Neuropharmacology, 62*, 2320–2327. https://doi.org/10.1016/j.neuropharm.2012.01.020.

Gratton, G., Coles, M. G. H., & Donchin, E. (1983). A new method for off-line removal of ocular artifact. *Electroencephalography and Clinical Neurophysiology, 55*, 468–484. https://doi.org/10.1016/0013-4694(83)90135-9.

Hahn, B., Ross, T. J., Wolkenberg, F. A., Shakleya, D. M., Huestis, M. A., & Stein, E. A. (2009). Performance effects of nicotine during selective attention, divided attention, and simple stimulus detection: An fMRI study. *Cerebral Cortex, 19*, 1990–2000. https://doi.org/10.1093/cercor/bhn226.

Hahn, B., Ross, T. J., Yang, Y., Kim, I., Huestis, M. A., & Stein, E. A. (2007). Nicotine enhances visuospatial attention by deactivating areas of the resting brain default network. *The Journal of neuroscience: the official journal of the Society for Neuroscience, 27*, 3477–3489. https://doi.org/10.1523/JNEUROSCI.5129-06.2007.

Haskell, C. F., Kennedy, D. O., Milne, A. L., Wesnes, K. A., & Scholey, A. B. (2008). The effects of l-theanine, caffeine and their combination on cognition and mood. *Biological Psychology, 77*, 113–122. https://doi.org/10.1016/j.biopsycho.2007.09.008.

Heishman, S. J., Kleykamp, B. A., & Singleton, E. G. (2010). Meta-analysis of the acute effects of nicotine and smoking on human performance. *Psychopharmacology, 210*, 453–469. https://doi.org/10.1007/s00213-010-1848-1.

Heydasch, T. (2014). *The Hagen Matrices Test (HMT) (part of doctoral dissertation thesis). University of Hagen.* https://doi.org/10.13140/RG.2.2.31433.75361.

Hilger, K., Ekman, M., Fiebach, C. J., & Basten, U. (2017). Efficient hubs in the intelligent brain: Nodal efficiency of hub regions in the salience network is associated with general intelligence. *Intelligence, 60*, 10–25. https://doi.org/10.1016/j.intell.2016.11.001.

Hong, L. E., Gu, H., Yang, Y., Ross, T. J., Salmeron, B. J., & Buchholz, B. (2009). *Association of nicotine addiction and nicotine's actions with separate cingulate cortex functional circuits. Archives of general psychiatry. Vol. 66*, 431–441. https://doi.org/10.1001/archgenpsychiatry.2009.2.

Houlihan, M. E., Pritchard, W. S., & Robinson, J. H. (1996). Faster P300 latency after smoking in visual but not auditory oddball tasks. *Psychopharmacology, 123*, 231–238.

Houlihan, M. E., Pritchard, W. S., & Robinson, J. H. (2001). Effects of smoking/nicotine on performance and event-related potentials during a short-term memory scanning task. *Psychopharmacology, 156*, 388–396.

Hudkins, M., O'Neill, J., Tobias, M. C., Bartzokis, G., & London, E. D. (2012). Cigarette smoking and white matter microstructure. *Psychopharmacology, 221*, 285–295. https://doi.org/10.1007/s00213-011-2621-9.

Ilan, A. B., & Polich, J. (1999). Tobacco smoking and memory scanning: Behavioral and event-related potential effects. *Nicotine & Tobacco Research, 1*, 233–240.

Jacobsen, L. K., Picciotto, M. R., Heath, C. J., Frost, S. J., Tsou, K. A., & Dwan, R. A. (2007). Prenatal and adolescent exposure to tobacco smoke modulates the development of white matter microstructure. *The Journal of Neuroscience, 27*, 13491–13498. https://doi.org/10.1523/JNEUROSCI.2402-07.2007.

Jeffreys, H. (1961). *The Theory of Probability* (3rd ed.). Oxford: Oxford University Press.

Jensen, A. R. (2006). *Clocking the mind: Mental chronometry and individual differences* (1st ed.). Amsterdam, Boston, London: Elsevier.

Kahathuduwa, C. N., Dassanayake, T. L., Amarakoon, A. M. T., & Weerasinghe, V. S. (2017). Acute effects of theanine, caffeine and theanine–caffeine combination on attention. *Nutritional Neuroscience, 20*, 369–377. https://doi.org/10.1080/1028415X.2016.1144845.

Kievit, R. A., Davis, S. W., Griffiths, J., Correia, M. M., Cam-CAN, & Henson, R. N. (2016). A watershed model of individual differences in fluid intelligence. *Neuropsychologia, 91*, 186–198. https://doi.org/10.1016/j.neuropsychologia.2016.08.008.

Knott, V., Bosman, M., Mahoney, C., Ilivitsky, V., & Quirt, K. (1999). Transdermal nicotine: Single dose effects on mood, EEG, performance, and event-related potentials. *Pharmacology, Biochemistry, and Behavior, 63*, 253–261.

Knott, V., Choueiry, J., Dort, H., Smith, D., Impey, D., La, S., ... Philippe, T. (2014). Baseline-dependent modulating effects of nicotine on voluntary and involuntary attention measured with brain event-related P3 potentials. *Pharmacology, Biochemistry, and Behavior, 122*, 107–117. https://doi.org/10.1016/j.pbb.2014.03.020.

Kovacs, K., & Conway, A. R. A. (2016). Process Overlap Theory: A Unified Account of the General factor of Intelligence. *Psychological Inquiry, 27*, 151–177. https://doi.org/10.1080/1047840X.2016.1153946.

Kumari, V., Gray, J. A., Ffytche, D. H., Mitterschiffthaler, M. T., Das, M., Zachariah, E., ... Sharma, T. (2003). Cognitive effects of nicotine in humans: an fMRI study. *NeuroImage, 19*(3), 1002–1013.

Larson, G. E., & Alderton, D. L. (1990). Reaction time variability and intelligence: A "worst performance" analysis of individual differences. *Intelligence, 14*, 309–325. https://doi.org/10.1016/0160-2896(90)90021-K.

Levin, E. D., McClernon, F. J., & Rezvani, A. H. (2006). Nicotinic effects on cognitive function: Behavioral characterization, pharmacological specification, and anatomic localization. *Psychopharmacology, 184*, 523–539. https://doi.org/10.1007/s00213-005-0164-7.

Lövdén, M., Bodammer, N. C., Kühn, S., Kaufmann, J., Schütze, H., & Tempelmann, C. (2010). Experience-dependent plasticity of white-matter microstructure extends into old age. *Neuropsychologia, 48*, 3878–3883. https://doi.org/10.1016/j.neuropsychologia.2010.08.026.

Mansvelder, H. D., van Aerde, K. I., Couey, J. J., & Brussaard, A. B. (2006). Nicotinic modulation of neuronal networks: From receptors to cognition. *Psychopharmacology, 184*, 292–305. https://doi.org/10.1007/s00213-005-0070-z.

McVay, J. C., & Kane, M. J. (2012). Drifting from slow to "D'oh!": Working memory capacity and mind wandering predict extreme reaction times and executive control errors. *Journal of Experimental Psychology, 38*, 525–549. https://doi.org/10.1037/a0025896.

Menon, V., & Uddin, L. Q. (2010). Saliency, switching, attention and control: A network model of insula function. *Brain Structure & Function, 214*, 655–667. https://doi.org/10.1007/s00429-010-0262-0.

Marchant, N. L., Kamel, F., Echlin, K., Grice, J., Lewis, M., & Rusted, J. M. (2009). Modafinil improves rapid shifts of attention. *Psychopharmacology, 202*(1-3), 487–495. https://doi.org/10.1007/s00213-008-1395-1.

Newhouse, P. A., Potter, A., & Singh, A. (2004). Effects of nicotinic stimulation on cognitive performance. *Current Opinion in Pharmacology, 4*, 36–46. https://doi.org/10.1016/j.coph.2003.11.001.

Paul, R. H., Grieve, S. M., Niaura, R., David, S. P., Laidlaw, D. H., Cohen, R., ... Evian, G. (2008). Chronic cigarette smoking and the microstructural integrity of white matter in healthy adults: a diffusion tensor imaging study. *Nicotine & tobacco research: official journal of the Society for Research on Nicotine and Tobacco, 10*(1), 137–147. https://doi.org/10.1080/14622200701767829.

Penke, L., Maniega, S. M., Bastin, M. E., Valdes Hernandez, M. C., Murray, C., & Royle, N. A. (2012). Brain white matter tract integrity as a neural foundation for general intelligence. *Molecular Psychiatry, 17*, 1026–1030. https://doi.org/10.1038/mp.2012.66.

Pineda-Pardo, J. A., Martínez, K., Román, F. J., & Colom, R. (2016). Structural efficiency within a parieto-frontal network and cognitive differences. *Intelligence, 54*, 105–116. https://doi.org/10.1016/j.intell.2015.12.002.

Polich, J. (2007). Updating P300: An Integrative Theory of P3a and P3b. *Clinical Neurophysiology: Official Journal of the International Federation of Clinical Neurophysiology, 118*, 2128–2148. https://doi.org/10.1016/j.clinph.2007.04.019.

Pritchard, W., Sokhadze, E., & Houlihan, M. (2004). Effects of nicotine and smoking on event-related potentials: A review. *Nicotine & tobacco research: official journal of the Society for Research on Nicotine and Tobacco, 6*, 961–984.

Raven, J. C., Court, J. H., & Raven, J. (1994). *Manual for Raven's progressive matrices and mill hill vocabulary scales. Advanced progressive matrices.* Oxford: Oxford University Press.

van Ravenzwaaij, D., Brown, S., & Wagenmakers, E.-J. (2011). An integrated perspective on the relation between response speed and intelligence. *Cognition, 119*, 381–393. https://doi.org/10.1016/j.cognition.2011.02.002.

Rosenthal, R., & Jacobson, L. (1966). Teachers' expectancies: Determinants of pupils' IQ gains. *Psychological Reports, 19*, 115–118. https://doi.org/10.2466/pr0.1966.19.1.115.

Salthouse, T. A. (1996). The processing-speed theory of adult age differences in cognition. *Psychological Review, 103*, 403–428. https://doi.org/10.1037/0033-295X.103.3.403.

Schmiedek, F., Oberauer, K., Wilhelm, O., Süss, H.-M., & Wittmann, W. W. (2007). Individual differences in components of reaction time distributions and their relations to working memory and intelligence. *Journal of Experimental Psychology: General, 136*, 414–429. https://doi.org/10.1037/0096-3445.136.3.414.

Schmitz, F., & Wilhelm, O. (2016). Modeling Mental speed: Decomposing Response Time Distributions in Elementary Cognitive Tasks and Correlations with Working memory Capacity and Fluid Intelligence. *Journal of Intelligence, 4*, 13.

Schubert, A.-L., Frischkorn, T. G., Hagemann, D., & Voss, A. (2016). Trait Characteristics of Diffusion Model Parameters. *Journal of Intelligence, 4*, 7.

Schubert, A.-L., Hagemann, D., & Frischkorn, G. T. (2017). Is general intelligence little more than the speed of higher-order processing? *Journal of Experimental Psychology: General, 146*, 1498–1512. https://doi.org/10.1037/xge0000325.

Schubert, A.-L., Hagemann, D., Voss, A., Schankin, A., & Bergmann, K. (2015). Decomposing the relationship between mental speed and mental abilities. *Intelligence, 51*, 28–46. https://doi.org/10.1016/j.intell.2015.05.002.

Schulz-Zhecheva, Y., Voelkle, C. M., Beauducel, A., Biscaldi, M., & Klein, C. (2016). Predicting Fluid Intelligence by Components of Reaction Time Distributions from simple Choice Reaction Time Tasks. *Journal of Intelligence, 4*, 8.

Sheppard, L. D., & Vernon, P. A. (2008). Intelligence and speed of information processing: A review of 50 years of research. *Personality and Individual Differences, 44*, 535–551. https://doi.org/10.1016/j.paid.2007.09.015.

Soltani, M., & Knight, R. T. (2000). Neural origins of the P300. *Critical Reviews in Neurobiology, 14*, 199–224.

Sternberg, S. (1969). Memory-scanning: Mental processes revealed by reaction time experiments. *American Scientist, 57*, 421–457.

Stough, C., Mangan, G., Bates, T., & Pellett, O. (1994). Smoking and Raven IQ. *Psychopharmacology, 116*, 382–384.

JASP Team (2018). *JASP (Version 0.8.6) [Computer software]*.

Troche, S. J., Houlihan, M. E., Stelmack, R. M., & Rammsayer, T. H. (2009). Mental ability, P300, and mismatch negativity: Analysis of frequency and duration discrimination. *Intelligence, 37*(4), 365–373. https://doi.org/10.1016/j.intell.2009.03.002.

Troche, S. J., Indermühle, R., Leuthold, H., & Rammsayer, T. H. (2015). Intelligence and the psychological refractory period: A lateralized readiness potential study. *Intelligence, 53*, 138–144. https://doi.org/10.1016/j.intell.2015.10.003.

Turner, D. C., Robbins, T. W., Clark, L., Aron, A. R., Dowson, J., & Sahakian, B. J. (2003). Cognitive enhancing effects of modafinil in healthy volunteers. *Psychopharmacology, 165*(3), 260–269. https://doi.org/10.1007/s00213-002-1250-8.