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Dual Process Theory and Smoking: Evidence for independent System 1-2 pathways in predicting smoking frequency

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Abstract

The study develops a novel method to measure System 1 (fast, cue-based, thought-light) smoking frequency as an outcome measure to overcome the limitations of existing measures. It also examines its relationship to System 2 (slow, deliberative) measures. 116 participants met in two sessions set at 48 hours apart. System 2 measure of smoking frequency was measured via self-reported 24-hour smoking frequency. System 1 smoking frequency was measured by observing the number of cigarettes participants carried and asking about the number of cigarette packs consumed between the two sessions. System 2 measure of smoking frequency underestimated System 1 smoking frequency by 47.4%. Results also revealed that System 1 smoking attitudes only predicted the newly developed System 1 smoking frequency significantly, while System 2 smoking attitudes predicted only System 2 smoking frequency significantly. Hence, interventions that modify explicit attitudes (System 2) might have a limited impact on observed (System 1) smoking behaviours.

Dual Process Theory and Smoking: Evidence for independent System 1-2 pathways in predicting smoking frequency

Smoking frequency has a dose-dependent effect on mortality risk (Thun et al., 2013). Hence, the examination of smoking frequency is important. Dual process theory postulates that two pathways contribute to behavioural outcomes: System 1, which is faster and 'thought-light', while System 2 is slower and deliberative (Hagger, 2016). This study describes the development of a System 1 outcome measurement of smoking frequency and examines its relationship with System 2 measures. The differentiation between System 1 and 2 measurements of smoking frequency has important implications for research and behavioural change interventions (Stacy and Wiers, 2010).

Evans (2008) articulated the differences between System 1 and 2 measures in four clusters – Consciousness, Evolution, Functional Characteristics, and Individual Differences. Under the Consciousness cluster, System 1 measures tend to have one or more properties: low awareness, implicit, low effort, and fast processing (Van Gestel et al., 2021). In contrast, System 2 measures have the contrasting properties of high awareness, explicit, high effort and slow processing. An example of a System 1 measure that encapsulates low deliberative mental effort in this cluster would involve using computer software on mobile phones to scan and process people's grocery checkout receipts for a week, and have the computer software compute their weekly healthy food purchases. In contrast, a System 2 version that involves high deliberative mental effort would require people, instead of software, to engage in mental arithmetic and memory recall by asking them how many times they have purchased a sample of healthy foods over the week. In this example, the System 1 measure only requires people to remember to scan their receipts using their mobile phones. In contrast, the System 2 measure requires people to exert greater mental effort in mental arithmetic and memory recall (i.e., recall, identification, tallying, and averaging for a week's data for the purchasing behaviour of specific items).

The existing System 1 measures for smoking frequency are mainly measured using biological markers. Biological measures do not involve high mental effort in memory recall or arithmetic and rely on medical technology to translate biological data to estimate smoking frequency. Hence, they are a System 1 measure of smoking frequency. However, these biological measures of smoking frequency have several limitations. Biological assessment's half-life (i.e., 16-20 hours) does not have a high discriminant value for high smoking frequency (Gorber et al., 2009). Biological data processing might also involve costly specialised equipment (e.g., McKee et al., 2011). Breath analysers that measure carbon monoxide during exhalation might also require participants to be able to hold their breath, which might be difficult for people with chronic obstructive pulmonary disease, a health risk among people who smoke (Adeloye et al., 2022). In addition, biological measures differentiate smoking status better than smoking frequency (Rubinstein et al., 2007). This is because biological measures do not translate well into the same metric used in self-reported measures of smoking frequency (e.g., daily cigarettes consumed). For instance, carbon monoxide (CO) breath analysers report CO data in parts per million units rather than daily cigarettes consumed. Hence, concluding the accuracy of self-reported smoking frequency is difficult compared to biological measures when both methods' metric scales differ.

To assess the validity of the newly developed System 1 measure of smoking frequency in this study, existing System 1 and 2 measures of smoking attitudes as predictors were used. Most research has found that attitudes towards smoking, such as the perceived benefits and harms of smoking, are a significant factor in predicting smoking behaviours (Heris et al., 2020). Research on differentiating System 1 and 2 measures for attitude towards smoking has involved behavioural response times via the Implicit Association Test (IAT) as System 1 and self-reported smoking attitudes as System 2 measures (see Greenwald and Lai, 2020 for a review). This System 1-2 operationalisation capitalises on response speed differences, where participants' responses to IAT are faster (three to one second) than the more deliberate responses to self-reported smoking attitude questionnaires. A meta-analysis by Hofman et al. (2005) reported that the corrected

population correlation between System 1 and 2 measures was .24, indicating that these measures assess different aspects of attitudes. There is also evidence for the incremental predictive validity of these System 1 and 2 measures (Kurdi et al., 2019). However, most research does not differentiate System 1 and 2 outcomes and hence, did not examine the System 1 to System 1 predictor-outcome relationship but instead, examined a System 1 to 2 predictor-outcome relationship. For instance, a System 1 reaction time measure might be used to predict self-reported (System 2) behaviour (Meissner et al., 2019). The study reported here attempts to address this gap by developing a new System 1 outcome measure for smoking frequency.

The System 1 outcome measure of smoking frequency developed in this study requires less deliberative mental effort and is more implicit than the System 2 measure. Specifically, the System 1 measure of smoking frequency asks participants to recall the number of additional cigarette packs consumed over the last 48 hours rather than the number of cigarette sticks consumed. For most smokers in the UK, the number of cigarette packs consumed typically ranged from one to two packs over two days (Jackson et al., 2025), a number that is easy to recall accurately. This recall is an indirect (implicit) way to compute each participant's daily cigarette consumption (Greenwald and Lai, 2020). On the other hand, the System 2 measure of smoking frequency in our study requires participants to recall every cigarette stick smoked over 48 hours and then mentally average this to derive and report the daily average of cigarette sticks smoked in the same period. This measure requires more deliberative mental effort than the System 1 measure, and is more transparent (i.e., explicit) in its data collection. Current System 2 measures of smoking frequency utilise this form of explicit self-reported data, where participants are asked to report their total or average cigarettes smoked over a specified period, ranging from days, as done in this study, to a participant's lifetime (e.g., Volk et al., 2020).

Differentiating Systems 1 and 2 pathways for smoking behaviours has important implications for dual process theory and designing behavioural change interventions (Stacy and Wiers, 2010). An

implication for the effectiveness of interventions in changing smoking behaviours is the crossover of System 1-2 pathways between the intervention strategy and its outcome. Specifically, a system crossover occurs when a System 2 intervention that focuses on changing smokers' understanding of the health risks of smoking (i.e., an intervention that involves high deliberative mental effort) is aimed at influencing a System 1 (low deliberative mental effort) smoking behaviour outcome. If System 1 and 2 pathways are independent, such system crossovers in smoking cessation strategy might be less effective than using a System 1 intervention (behavioural nudge) to change System 1 smoking behaviours (Marteau et al., 2012).

Currently, there is a lack of research evidence testing the unique System 1 and 2 pathways predicting smoking frequency. However, a meta-analysis on intergroup behaviour found that System 1 measures have incremental predictive validity over System 2 measures, which provided evidence of the independence of these two Systems (Kurdi et al., 2019). This is consistent with research that reported observed smoking behaviours sometimes misestimate self-reported smoking behaviours by 10% to 30% (Why et al., 2021). In addition, a meta-analysis found that when compared to biological measures, self-reported smoking status can underestimate smoking status by 25%, with the underestimation as high as 45% for studies of better quality (Gorber et al., 2009).

One obstacle to examining this underestimation of smoking frequency is the lack of a System 1 measure that uses the same metric scale (i.e., number of cigarettes smoked per day rather than carbon monoxide in one's breath measured in parts per million). This study contributes to the existing research by developing an accessible method of measuring System 1 smoking frequency as an outcome. In addition, it uses existing System 1-2 measures of smoking attitudes (IAT and self-reported questionnaire) to predict System 1-2 outcome measures of smoking frequency to test the independence of System 1-2 pathways. That is, whether the System 1 predictor only predicts System 1 outcome and similarly for the System 2 predictor and outcome.

Method

Participants

Posters were placed in various locations on a UK university campus to recruit unpaid volunteers. Participant requirements were that they had to smoke cigarette sticks daily (i.e., at least one stick daily) and could come to our experimental room located on the same university campus twice over two days. One hundred sixteen participants were recruited. The average age was 23.49 years (SD = 6.08). Sixty-nine (59.5%) participants were women. Data was missing for four participants due to software malfunction for the IAT task. Hence, data from 112 were analysed. We sampled only participants who smoked cigarette sticks for two reasons. Firstly, the System 1 assessment of smoking frequency developed in this study required a standardisation of tobacco products used between participants. Secondly, it is the most prevalent tobacco product consumed worldwide (WHO, 2023). This study has been approved by the Institutional Review Board.

Measures

System 1 and 2 measures of smoking attitudes are used as predictors to assess the validity of our newly developed System 1 smoking frequency as an outcome variable. System 2 measure of smoking attitude was measured using the brief Questionnaire of Smoking Urges, QSU Brief, which measures smoking urges as two factors – Smoking as Reward and Smoking as Relief (Cox et al., 2001). The QSU Brief is a valid measure of participants' motivation to smoke, as smokers who were experimentally abstained from smoking for 24 hours reported significantly higher urges to smoke in this questionnaire (West and Ussher, 2010). In this sample, the two subscales were combined because they were highly correlated, r(115) = .70, p < .001. The mean (SD) QSU Brief score was 29.98 (17.84). Internal consistency of the QSU Brief was excellent, Cronbach's $\alpha = .94$. The Implicit Association test was the System 1 measure of smoking attitude (Swanson et al., 2001). The mean (SD) IAT score for smoking in this sample was 0.017 (0.59). D, the measure derived from this IAT task, was computed using two blocks of response trials. The Ds derived from each block were correlated to estimate the IAT's internal consistency, which was good: r(110) = .86, p < .001. QSU-Brief and IAT

Smoking were negatively correlated, r(110) = -.28, p = .003 (see raw data in Supplementary Material).

System 2 measure of smoking frequency was measured via self-report by asking each participant about their daily consumption of cigarettes for the last two days. The average (SD) daily cigarette consumption in this sample was 4.41 cigarettes/day (4.03). A System 1 measure of smoking frequency was done by observing the number of cigarettes participants carried in two sessions separated by 48 hours. In the second session, participants were asked whether any additional packs were consumed and not seen by the researcher. These observations and questions enabled the estimation of daily cigarette consumption for each participant. For example, if a participant was observed to have six cigarettes in session 1, was observed to have 10 cigarettes in a 12 cigarette pack in session 2, and had reported consuming another pack of 12 cigarettes not observed by the researcher between both sessions, the 24hr smoking frequency for this participant would be estimated by [6 + 12 + (12-10)]/2 = 20/2 = 10 cigarettes/24hr. Participants were told not to share their cigarettes with anyone between these two sessions. The average (SD) System 1 smoking frequency was 8.39 cigarettes/day (4.84), 47.4% over that derived from the System 2 measure of smoking frequency. The correlation between System 1 and 2 measures of smoking frequency was r(115) = .29, p = .002. Procedure

Participants were met twice, with the second session occurring 48 hours after the first session. Informed consent was obtained from all participants in the first session, and this study has acquired the relevant institutional ethical approval. All self-reported and attitude assessments were done in the first session, while the System 1 measurement of smoking frequency was done in sessions 1 and 2. At the end of session 2, participants were debriefed.

Results

The significance level was set at .05. MANCOVA in SPSS (version 29, IBM) was used. Gender and age were entered as covariates. System 1 (i.e., IAT) and 2 (i.e., QSU) measures of smoking

attitudes were entered as predictors for two outcome measures: System 1 and System 2 measures of smoking frequency. If the multivariate result was statistically significant, the univariate result was examined.

For the covariates, the multivariate result for Age was significant, Wilks' Λ = .84, F(2, 106) = 9.84, p < .001, partial η^2 = .16. Examination of its univariate results indicates that Age was a significant predictor for both System 1, F(1, 107) = 13.50, p < .001, partial η^2 = .11, B = 0.27, and System 2, F(1, 107) = 9.91, p = .002, partial η^2 = .085, B = 0.20, smoking frequency measures. Gender was non-significant, Wilks' Λ = .998, F(2, 106) = 0.91, p = .91, partial η^2 = .002.

The multivariate result for System 1 measure of smoking attitude, IAT, was significant, Wilks' Λ = .94, F(2, 106) = 3.34, p = .039, partial η^2 = .059. The univariate results indicated that IAT predicted the System 1 measure of smoking frequency, F(1, 107) = 5.01, p = .027, partial η^2 = .045, B = 1.72, but not for the System 2 measure, F(1, 107) = 2.88, p = .09, partial η^2 = .026, B = 1.12. The multivariate result for System 2 measure of smoking attitude, QSU, was also significant, Wilks' Λ = .93, F(2, 106) = 3.98, p = .02, partial η^2 = .07. The univariate results indicated that QSU predicted System 2 measure of smoking frequency, F(1, 107) = 7.97, p = .006, partial η^2 = .069, B = 0.061, but not for our System 1 measure, F(1, 107) = 0.55, p = .46, partial η^2 = .005, B = 0.019.

Discussion

The results support the Dual Process theory for two independent System 1 and 2 pathways predicting smoking frequency. In addition, System 1 and 2 measures were weakly correlated. The newly developed System 1 smoking frequency measure revealed an underestimation of self-reported smoking close to the upper boundary reported in Gober et al.'s (2009) meta-analysis for smoking status (i.e., 45%). However, it is important to point out that Gober et al.'s (2009) meta-analysis was on smoking status rather than smoking frequency. As reviewed earlier, biological measures of tobacco use do not translate into per cigarette consumed metric (i.e., self-reported smoking frequency). The System 1 formulation of smoking frequency in this study permits using the

same metric (i.e., daily cigarettes consumed) to compare with System 2 (self-reported) smoking frequency.

While Rooke et al. (2008) examined System 1 predictors of substance use, they did not differentiate System 1 or 2 outcome measures of substance use. Three of our four variables in the current study are typical operationalisations of System 1 and 2 variables: IAT, QSU, and self-reported smoking frequency. With the development of a new System 1 outcome measure of smoking frequency, evidence was found for only the IAT (System 1) and not the QSU (System 2), predicting this outcome. A recent study also reported this lack of crossover of System 1-2 pathways: Tibboel et al. (2024) found that self-reported smoking attitude (System 2) predicted self-reported smoking frequency (System 2), but behavioural reaction time measures of smoking attitude (System 1) did not predict their System 2 outcome variable. This System 1-2 independence also provided convergent and discriminant validity of the new System 1 measure of smoking frequency detailed in this study; That is, System 1 (IAT) smoking attitude predicts only the new System 1 smoking frequency, while System 2 (QSU) smoking attitudes predict only System 2 (self-reported) smoking frequency.

Using a behavioural reaction time task, Motschman and Tiffany (2016) found that regular smokers use less deliberative mental processes for their smoking behaviours compared to occasional smokers. This suggests that System 1 processes maintain smoking behaviours among regular smokers. If these two systems have independent pathways, it might explain why interventions that modify explicit attitudes (System 2) might have limited impact on observed (System 1) smoking behaviours. That is, an intervention strategy that involves a system crossover might be less effective than an intervention strategy that involves only one system. Many behavioural change interventions tend to involve such a system crossover, and this might explain their low success rates for behavioural change (Marteau et al., 2012): A recent systematic review found that only nine interventions of the 32 studies reviewed were effective in smoking reduction or cessation among

U.S. young adults (Villanti et al., 2020). Hence, System 1 interventions, such as implicit primes and nudges, might be more effective in changing regular smoking behaviours maintained by System 1 processes.

While the results in this study showed that System 1 and 2 predictors predict the outcome variable within the same System, researchers do not uniformly agree that this is convincing evidence for dual process theory (see Evans and Stanovich, 2013 for an overview of this debate). A rule-based single-process theory can also explain the results. For example, a single rule-based cognitive system could be stated as: if the goal is to predict a System 1 outcome, then a System 1 predictor will be used, else System 2 variables will be used (i.e., if 'a', then use 'b', else use 'c'). This is an actively debated issue that some researchers have pointed out might not be constructive; among other things, its resolution, even if possible, might have little impact on applied research (De Neys, 2021).

This study has a few limitations. Firstly, the test-retest of the new method of measuring System 1 smoking behaviours was not investigated. Secondly, the time-lapse for observed smoking behaviours was set at 48 hours. An examination of the predictive validity of this measure and/or its relationship with System 2 measures could be investigated if the time lapse was greater than 48 hours. Results from this study reveal that System 1 processes have low correlations with System 2 processes, and there are independent pathways influencing different measures of smoking behaviours. Overall, the novel System 1 measurement of smoking frequency developed in this study shows good potential as a research tool: it is valid, cost-effective, easy to implement, and could supplement existing smoking frequency assessments.

References

- Adeloye D, Song P, Zhu Y, et al. (2022) Global, regional, and national prevalence of, and risk factors for, chronic obstructive pulmonary disease (COPD) in 2019: a systematic review and modelling analysis. *The Lancet Respiratory Medicine* 10(5). Elsevier: 447–458.
- Cox LS, Tiffany ST and Christen AG (2001) Evaluation of the brief questionnaire of smoking urges (QSU-brief) in laboratory and clinical settings. *Nicotine & Tobacco Research: Official Journal of the Society for Research on Nicotine and Tobacco* 3(1): 7–16.
- De Neys W (2021) On Dual- and Single-Process Models of Thinking. *Perspectives on Psychological Science* 16(6). SAGE Publications Inc: 1412–1427.
- Evans JSBT (2008) Dual-processing accounts of reasoning, judgment, and social cognition. *Annual Review of Psychology* 59: 255–278.
- Evans JStBT and Stanovich KE (2013) Dual-Process Theories of Higher Cognition: Advancing the Debate. *Perspectives on Psychological Science* 8(3): 223–241.
- Gorber SC, Schofield-Hurwitz S, Hardt J, et al. (2009) The accuracy of self-reported smoking: A systematic review of the relationship between self-reported and cotinine-assessed smoking status. *Nicotine & Tobacco Research* 11(1): 12–24.
- Greenwald AG and Lai CK (2020) Implicit Social Cognition. *Annual Review of Psychology* 71(Volume 71, 2020). Annual Reviews: 419–445.
- Hagger MS (2016) Non-conscious processes and dual-process theories in health psychology. *Health Psychology Review* 10(4). Routledge: 375–380.
- Heris CL, Chamberlain C, Gubhaju L, et al. (2020) Factors Influencing Smoking Among Indigenous Adolescents Aged 10–24 Years Living in Australia, New Zealand, Canada, and the United States: A Systematic Review. *Nicotine & Tobacco Research* 22(11): 1946–1956.
- Hofmann W, Gawronski B, Gschwendner T, et al. (2005) A meta-analysis on the correlation between the implicit association test and explicit self-report measures. *Personality & Social Psychology Bulletin* 31(10): 1369–1385.
- Jackson SE, Tattan-Birch H, Buss V, et al. (2025) Trends in Daily Cigarette Consumption Among Smokers: A Population Study in England, 2008–2023. *Nicotine & Tobacco Research* 27(4): 722–732.
- Kurdi B, Seitchik AE, Axt JR, et al. (2019) Relationship between the Implicit Association Test and intergroup behavior: A meta-analysis. *American Psychologist* 74(5). US: American Psychological Association: 569–586.
- Marteau TM, Hollands GJ and Fletcher PC (2012) Changing Human Behavior to Prevent Disease: The Importance of Targeting Automatic Processes. *Science* 337(6101). American Association for the Advancement of Science: 1492–1495.
- McKee SA, Sinha R, Weinberger AH, et al. (2011) Stress decreases the ability to resist smoking and potentiates smoking intensity and reward. *Journal of Psychopharmacology* 25(4). SAGE Publications Ltd STM: 490–502.

- Meissner F, Grigutsch LA, Koranyi N, et al. (2019) Predicting Behavior With Implicit Measures:

 Disillusioning Findings, Reasonable Explanations, and Sophisticated Solutions. *Frontiers in Psychology* 10. Frontiers.
- Motschman CA and Tiffany ST (2016) Cognitive regulation of smoking behavior within a cigarette: Automatic and nonautomatic processes. *Psychology of Addictive Behaviors* 30(4). US: American Psychological Association: 494–499.
- Rubinstein ML, Thompson PJ, Benowitz NL, et al. (2007) Cotinine levels in relation to smoking behavior and addiction in young adolescent smokers. *Nicotine & Tobacco Research: Official Journal of the Society for Research on Nicotine and Tobacco* 9(1): 129–135.
- Stacy AW and Wiers RW (2010) Implicit Cognition and Addiction: A Tool for Explaining Paradoxical Behavior. *Annual review of clinical psychology* 6: 551.
- Swanson JE, Swanson E and Greenwald AG (2001) Using the Implicit Association Test to investigate attitude-behaviour consistency for stigmatised behaviour. *Cognition and Emotion* 15(2). Routledge: 207–230.
- Thun MJ, Carter BD, Feskanich D, et al. (2013) 50-Year Trends in Smoking-Related Mortality in the United States. *New England Journal of Medicine* 368(4). Massachusetts Medical Society: 351–364.
- Tibboel H, Van Bockstaele B, Spruyt A, et al. (2024) Implicit beliefs and automatic associations in smoking. *Journal of Behavior Therapy and Experimental Psychiatry* 83: 101925.
- Van Gestel LC, Adriaanse ,M. A. and and De Ridder DTD (2021) Do nudges make use of automatic processing? Unraveling the effects of a default nudge under type 1 and type 2 processing. *Comprehensive Results in Social Psychology* 5(1–3). Routledge: 4–24.
- Villanti AC, West JC, Klemperer EM, et al. (2020) Smoking-Cessation Interventions for U.S. Young Adults: Updated Systematic Review. *American Journal of Preventive Medicine* 59(1): 123–136.
- Volk RJ, Mendoza TR, Hoover DS, et al. (2020) Reliability of self-reported smoking history and its implications for lung cancer screening. *Preventive Medicine Reports* 17: 101037.
- West R and Ussher M (2010) Is the ten-item Questionnaire of Smoking Urges (QSU-brief) more sensitive to abstinence than shorter craving measures? *Psychopharmacology* 208(3): 427–432.
- WHO (2023) Tobacco. Available at: https://www.who.int/news-room/fact-sheets/detail/tobacco (accessed 10 December 2024).
- Why FYP, Undarwati A and Nuzulia S (2021) The sociodemographic context of observed solitary and social smoking behaviours using a behavioural ecological approach. *Journal of Health Psychology* 26(9): 1318–1323.